# Demography and Nutrition: Evidence from Historical and Contemporary Populations

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**Blackwell Science**

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# Contents

# Preface

We have presented in our two previous books, *Human Demography and Disease* and *Biology of Plagues*, an overview of the interaction of population dynamics and infectious diseases, with a particular emphasis on preindustrial England.

*Demography and Nutrition* offers the view that the change from the hunter-gatherer to an agricultural lifestyle had a major impact on our demography which still has repercussions today. The abandonment of a nomadic way of life led eventually to the emergence of cities and civilisations, but the new diet was unsatisfactory and malnutrition caused a reduction in stature which was fully corrected only in the twentieth century.

Our approach, again, has been interdisciplinary and has involved time-series analysis, mathematical modelling, aggregative analysis and family reconstitution as well as analysis of data series from Third World countries in the twentieth century. The thesis that is developed is that malnutrition, from which the bulk of the population suffered, acting in a way that is not readily detectable, was the major factor that regulated human demography in historical times. Chronic malnutrition caused this effect mainly via three interacting mechanisms: (i) direct and indirect effects on the mortality of children from infectious diseases, (ii) down-regulating the levels of body fat and, hence, reducing a woman's fertility and, most importantly, (iii) raising the levels of infant mortality because of inadequate nutrition of the foetus during critical periods in pregnancy. Thus, the key to the regulation of human demography, both in historical times and in developing countries today, lies in the nutrition of the mother before, during and after pregnancy. These adverse effects of malnutrition were exacerbated by a regular oscillation in the supply of grains and by the annual hungry season which was imposed on human populations when they adopted an agricultural lifestyle.

We hope that this integrated approach to demography in our three books will be of value to readers from a variety of disciplines.

Once again we thank Dr S.R. Duncan of the University of Oxford for introducing us to the intricacies of time-series analysis and for developing the mathematical models that we have used.

We are grateful to Professor Rose Frisch, Harvard Center for Population and Development Studies, and Dr Brian Merry, University of Liverpool, for many helpful suggestions and comments.

Susan Scott
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Chapter 1

# Introduction

Nutrition is big business today. Almost every week the daily papers carry articles and research reports concerning our diet and health. Everything is discussed in detail: daily calorific intake, obesity, anorexia, oxygen radical scavengers, low density lipoproteins, lycopene. Fortunately, two admirable compendia on nutrition have recently been published: *The Cambridge World History of Food* (Kiple and Ornelas, 2000) and *Encyclopedia of Human Nutrition* (Sadler *et al.*, 1999). They have proved to be invaluable sources of references.

In this book, we are concerned with the interactions between malnutrition and demography. Consequently, the most important effects of nutrition considered here are on fertility and mortality, with deaths in infancy and childhood having the greatest demographic impact. Once a woman has ceased to bear children, her health and survival are of secondary importance demographically. From this it follows that the current interest in dietary modifications and additives (including oxygen radical scavengers) to combat heart disease and cancer, which are mainly operative in later life, are of little direct relevance to our thesis. Nor are the manifold afflictions of old age or the long-term chronic effects of malnutrition, such as markedly reduced stature. However, as we shall see, stature has proved to be a useful measure of nutritive levels in both prehistoric and modern (1600–1900) times (Fogel *et al.*, 1983; Floud *et al.*, 1990; Mokyr & O'Grada, 1996).

The dramatic demographic effects of the major famines in the Third World in the twentieth century are described in Chapter 4, but our thesis will be, in contrast to the opinion of many historians, that famines and outright starvation were generally of minor importance in pre-industrial England. Rather, we shall suggest that the long-lasting effects of chronic malnutrition were apparently subliminal and not readily detectable. Nevertheless, a suboptimal level of nutrition became a dominant factor in regulating the population dynamics of pre-industrial England.

For 300 years, from the Black Death in 1347 to the plague of London in 1665, the demography of the towns and cities of Europe was dominated by the awful mortality of the epidemics which broke out at irregular intervals (Scott & Duncan, 2001). The death rate among those infected was very high, particularly in the Black Death. The suboptimal nutrition of the bulk of the population

probably did little to exacerbate either the susceptibility or the death rate in this terrible disease, probably the worst ever to strike in Europe (Scott & Duncan, 2001).

After the mysterious disappearance of the plague in 1670, malnutrition became one of the most important factors that regulated the population dynamics of many communities in England, particularly in the north, where conditions were more severe. Many individual populations existed in steady-state, with numbers maintained by density-dependent conditions such as the availability of sufficient food for the whole community (see Chapter 5).

## 1.1 The history of human diet

Larsen (2000) writes

> 'Virtually every major anatomical change ... can be related in one way or another to how foods are acquired and processed by the human body. Indeed, the very fact that our humanlike ancestors had acquired a bipedal manner of walking by some five to eight million years ago is almost certainly related to how they acquired food.'

Humans have evolved by adaptation to acquire a wide means of securing their food and Gordon (1987) recognised three sequential phases in the evolution of human diet:

(1) The shift from a diet comprised primarily of unprocessed plant foods to one that incorporated deliberate food-processing techniques and included significant amounts of meat probably occurred 1.5 million years ago. The evidence suggests that the meat component of the diet was probably acquired through a strategy involving scavenging rather than deliberate hunting.

(2) The second phase (the hunter-gatherers) began about 700 000 years ago and was characterised by the deliberate hunting of animal food sources, although their degree of success is unclear (Larsen, 2000). The evidence indicates that by late Pleistocene times (25 000 to 11 000 years ago) human populations had developed strategies and means by which larger species of animals were successfully hunted. Late Stone Age peoples in southern Africa fished extensively and hunted dangerous animals like wild pigs and buffalo with considerable success (Klein, 1989). It is probable that plant foods contributed substantially to the diets of earlier, premodern hominids, although the evidence is virtually non-existent.

(3) The third phase began about 10 000 years ago when the essentially modern patterns of climate, vegetation and fauna began. The transition from a diet

of hunted and collected foods to one based on varying degrees of food production was gradual. The disappearance of megafauna, such as the mastodon and the mammoth, in many parts of the world at about this time may have been an impetus for human populations to develop new means of food acquisition in order to meet protein and fat requirements, i.e. necessity, born of increasing food scarcity, may have been the mother of agricultural invention (Larsen, 2000). By about 7000 years ago, a transition to a plant-based economy was well established in some areas of the Middle East. Agriculture spread from this region into Europe, and thence to Africa, Asia and the New World (Larsen, 2000).

## 1.2 The diet of the hunter-gatherers

Studies of contemporary African hunter-gatherers provide valuable insights into the diets and nutrition enjoyed by those following this lifestyle and suggest the conditions under which primitive humans may have lived. Cohen (2000) concludes that primitive hunter-gatherers frequently enjoyed an adequate and well-balanced nutrition, but also a relatively light workload.

The diet of the !Kung San hunter-gatherers of the Kalahari Desert in southern Africa consists of an eclectic, yet selective, collection of wild foods (mostly vegetable) eaten fresh. The San consume 23 of 85 plant species that they know to be edible in their environment and 17 of 55 edible animal species. San hunter-gatherers obtain an adequate and well-balanced diet for a relatively small investment of time. Each individual in the group receives sufficient protein, vitamins and minerals and there is no medical evidence of malnutrition. In contrast to modern diets in the developed world, what seems to be the limiting factor is the number of calories it delivers; the caloric intake is estimated to be about 2140 kcal per person per day.

Contemporary hunter-gatherers also receive a relatively large part (20–40%) of their diet from animal products. Daily animal protein intake among the San is approximately 30 to 50 g per person per day, which greatly exceeds an estimated average of 7 to 10 g of animal protein per person per day in modern developing countries. Populations such as the Hadza of Tanzania, who live in a richer foraging area, are estimated to get 3000 kcal and 50 to 250 g of meat protein per person per day. Groups like the Hadza appear to be a better model for prehistory than the San because they live in the same kinds of environments as early human beings (Cohen, 2000).

Palaeopathological evidence and a comparison of the environments in which prehistoric hunter-gatherers chose to live with those in which their modern counterparts are confined by the pressures of competition suggest that the populations of prehistory were at least as well nourished as those of today. Prehistoric, but biologically modern, human hunter-gatherers seem to have

expanded first through relatively game-rich environments, savannas, steppes and open forests. Occupation of the deserts and jungles in which most hunting and gathering groups are now found is a relatively recent phenomenon (Cohen, 2000).

Interestingly, the first biologically modern human populations of hunter-gatherers throughout Europe and areas of Asia including India seem to have been relatively tall. These Paleolithic hunters were taller than the Mesolithic foragers and the Neolithic farmers that came after them, and the populations of eighteenth century Europe were among the shortest human groups that have ever lived (Fogel, 1984). Cohen (2000) concludes that evidence from archaeology, from prehistoric skeletons and from the study of contemporary populations indicates that small, mobile human groups living on wild foods enjoy relatively well-balanced diets and relatively good health. Indeed, the available evidence suggests that hunter-gatherer diets remain well balanced even when they are low in calories. The data also show that *per capita* intake of calories and of protein has declined rather than increased in human history for all but the privileged classes. The predominant direction of prehistoric and historic change in human stature has been a decline in size, despite the 'secular trend' among some Western populations of the last century. Good surveys are provided by *The Cambridge Encyclopaedia of Hunters and Gatherers* (Lee & Daly, 2000) and by Panter-Brick *et al.* (2001).

## 1.3 Demographic change linked to the beginnings of agriculture

Some time after 12 000 BC, sedentary hunter-gatherer communities in the Levant, followed slightly later by inhabitants of Anatolia and parts of the Zagros Mountains, embarked on a path that led to the domestication of plants and animals and, ultimately, to food production. This shift in subsistence (the 'Neolithic Revolution') was arguably the most profound change in human history and one that still has a far-reaching demographic impact on the planet.

Current consensus is that plant domestication in the Near East began in the Jordan Valley around 9500 BC and animal husbandry started a little later in the Zagros Mountains and, possibly, in North Africa. Even with advances in agriculture, however, gathering and hunting continued to play an important role in the economy; in fact it is probable that the ancestors of the first domesticated plants in the Near East (emmer, einkorn and barley) had been important food plants for local foraging populations.

Thus, the change from a hunter-gatherer to an agriculture-based economy began in the Near East and Egypt and involved the demographic development of larger and more complex communities and, eventually, led to the emergence of civilisations. Improvements in food production and food storage technologies led to the accumulation of a surplus and permitted the growth of large, dense

populations and urban centres. With the advent of a complex society, people no longer had equal access to all types of food; an élite class ultimately controlled the production and distribution of much of the food supply. Some foods even became their sole prerogative, particularly exotic imports and those requiring extensive labour to produce. Most of the population, however, subsisted mainly on grain and grain products as earned wages in kind. In both Egypt and the Near East, the diet was based on plants, primarily cereal products like bread and beer, supplemented with vegetables, fish and meat. For the lower classes, meat was probably a rare commodity except for the pigs that households could raise without interference from state authorities (Miller & Wetterstrom, 2000).

## 1.4 To which diet is modern man adapted?

Thus, man followed and became adapted to a hunter-gatherer diet over a period of some 1.5 million years before changing to an agricultural lifestyle only some 10–12 000 years ago, a very short time in evolutionary terms. To which diet is our digestive system and underlying biochemistry best adapted? This is an important question for vegetarians and vegans.

Our diets were probably programmed at least 40 000 years ago. Eaton and Konner (1985) and Eaton *et al.* (1997) proposed that humans evolved to consume a Palaeolithic diet and, therefore, that we are genetically determined to eat diets quite different from those of today. Humans have not evolved much in the past 15 000 years or so, but our diets have changed dramatically and have done so in parallel with a shift in disease patterns. Whereas the leading causes of death in the nineteenth century in England used to be infections and diseases associated with nutrient deficiencies, they now comprise chronic degenerative diseases associated with an excessive and unbalanced intake of energy and nutrients. These workers suggest that the Paleolithic diet must have included 37% of energy from protein, 41% from carbohydrates and 22% from fat, along with amounts of vitamins, minerals and fibre that seem unimaginably high and unachievable through modern-day diets. Cordain *et al.* (2000) go further. They estimate from their survey of environmentally-diverse hunter-gatherer populations that, whenever and wherever it was ecologically possible, 45 to 65% of the energy in their diets came from animal foods.

The marked differences between the estimated Palaeolithic diet and the US and UK recommendations have been summarised by Nestle (2000) (see Table 1.1). The !Kung San hunter-gatherers of the Kalahari desert take in much more meat and, hence, more cholesterol than is recommended by modern medicine and yet they have very low blood-cholesterol levels and virtually no heart disease (Kiple, 2000), probably because they are eating free-range animals.

It has been popularly assumed that this change in basic lifestyle and diet was advantageous to mankind. However, in game-rich environments, regardless of the

**Table 1.1** Comparison of estimated Paleolithic diet with US and UK dietary recommendations.

| | Paleolithic diet | US dietary recommendations | UK dietary reference values |
|---|---|---|---|
| Protein (% energy) | 37 | 12 | |
| Carbohydrate (% energy) | 41 | 58 | 50 |
| Fat (% energy) | 22 | 30 | 35 |
| Cholesterol (mg/d) | 480 | 300 | |
| Fibre (g/d) | 104 | 20–35 | 20–24 |
| Vitamin C (mg/d) | 604 | 60 | 40 |
| Vitamin E (mg/d) | 32.8 | 8–10 | |
| Iron (mg/d) | 87.4 | 10–15 | 8.7–14.8 |
| Energy | | | |
| (kJ) | 12 558 | 9209–12 139 | 8100–10 600 |
| (kcal/d) | (3000) | (2200–2900) | (1940–2550) |

*Source:* Nestle (2000).

strategy employed, hunters may obtain between 10 000 and 15 000 kilocalories per hour. Subsistence cultivators, in contrast, average between 3000 and 5000 kilocalories per hour (Cohen, 1989). Furthermore, the shift from hunter-gathering to agriculture was characterised by a change from generally high-quality to low-quality foods. Animal sources of protein contain all essential amino acids in the correct proportions; they are a primary source of vitamin $B_{12}$, are high in vitamins A and D and contain important minerals. Moreover, animal fat is a critical source of essential fatty acids and fat-soluble vitamins. Thus, relative to plant foods, meat is a highly nutritional food resource. Plant foods used alone generally cannot sustain human life, primarily because of a deficiency in essential amino acids. Moreover, in circumstances where plant foods predominate, a wide variety of them must be consumed in order to fulfil the basic nutritional requirements.

Humans are not perfectly adapted to either dairy products or grains in their diet, witness lactose intolerance in southern Europeans, allergies to cow's milk in children and coeliac disease caused by an intolerance to the proteins in wheat and some other grains (Kiple, 2000).

## 1.5 Consequences of an agricultural life-style

Agriculture, particularly in Europe, has a strongly seasonal basis, which results in a pronounced annual variation in the availability of food, leading for example to the hungry season in pre-industrial England before the harvest was gathered (see Chapter 13). Periodic food shortages have been observed in contemporary human populations that depend on subsistence agriculture. These can result from variations in a number of factors, such as rainfall, temperature and the prevalence of insects and other pests; rust is a serious fungal disease of wheat (see section

3.11). Consequently, the availability of grains in pre-industrial England fluctuated markedly and autoregressive factors combined to generate a regular short wave oscillation in grain prices. The cause of this cycle of malnutrition and hardship is described in Chapter 3; it had profound effects on mortality (see Chapter 10) and fertility (see Chapter 6) and hence has been a dominant factor in regulating population dynamics.

Archaeological evidence from prehistoric agriculturalists, together with observations of living peasant agriculturalists, indicates that their diets tended to be dominated by a single cereal staple: rice in Asia, wheat in temperate Asia and Europe, millet or sorghum in Africa and maize in the New World. Inevitably, a diet based almost exclusively on one food source leads to a number of nutritional deficiencies.

Rice is remarkably deficient in protein and inhibits the activity of vitamin A, even if it is available through other food sources (Larsen, 2000).

Wheat, a wild grass that flourished in the wake of the retreating glaciers some 12 000 years ago, was (apparently) deliberately planted for the first time in the Middle East about 2000 years later. By the first century BC, Rome required some 14 million bushels per year just to feed the people of that city, leading to a programme of expansion that turned much of the cultivable land of North Africa into wheat fields for the Romans. Wheat is deficient in two essential amino acids, lysine and isoleucine, although most human populations that have dairy animals provide products (e.g. cheese) that make up for these missing amino acids. The history of wheat breeding is described in detail in Bonjean and Angus (2001).

Old World wheat gave back only 5 grains for every 1 planted, whereas maize returned 25 to 100 (a single ear of modern maize yields about 1000 grains) and, by the middle of the seventeenth century, had become a staple of the peasants of northern Spain, Italy and, to a lesser extent, southern France. From there, maize moved into much of the rest of Europe and, by the end of the eighteenth century, cornmeal mushes had spread via the Ottoman Empire into the Balkans and southern Russia. Maize was domesticated in Mesoamerica and formed the economic basis for the rise of civilisations and complex societies. By comparing 13 strains of maize and 17 strains of its wild ancestor teosinte (*Zea* spp.), a weedy grass that grows in Mexico and Guatemala, Wang *et al.* (1999) propose that Neolithic Central Americans turned grass into corn by selecting a gene for branch size. They suggest that early farmers altered the control region of a gene called *tb1* by always resowing teosinte crops with the biggest seeds. As a result, *tb1* is much more active in maize than in grass, causing the branches to grow fat ears of corn rather than their tassels of seed.

Maize is deficient in a number of important nutrients: its constituent protein, zein, is deficient in lysine, isoleucine and tryptophan and if maize consumers do not supplement their diets with foods containing these amino acids, such as beans, significant growth retardation is an outcome. Moreover, maize, although not deficient in niacin (vitamin $B_3$), contains it in a chemically-bound form that,

untreated, will withhold the vitamin from the consumer. Consequently, human populations eating untreated maize frequently develop pellagra (Larsen, 2000).

Maize nourished humans indirectly as well. With maize available to help feed livestock, it became increasingly possible to carry more animals through the winters and to derive a steady supply of whole protein in the forms of milk, cheese and eggs, in addition to year-round meat. Thus, it has been argued that it is scarcely a coincidence that, beginning in the eighteenth century, European populations began to grow and, by the nineteenth century, had swollen to the point where Europeans began migrating in vast numbers to the lands where the plants that had created the surplus were grown.

In the seventeenth century, invasion, famine and evictions forced Irish peasants to adopt the potato as a means of getting the most nourishment from the least amount of cultivated land and, during the eighteenth century, it was introduced in Germany and France because of the frequent failures of other crops. From there, use of the potato spread towards the Ural Mountains, where rye had long been the only staple that would ripen during the short, often rainy, summers. Potatoes not only did well under such conditions, they provided some four times as many calories per acre as rye and, by the first decades of the nineteenth century, were a crucial dietary element in the survival of large numbers of northern Europeans, just as maize had become indispensable to humans in some of the more southerly regions.

Ortner and Theobald (2000) conclude that humans have experienced nutritional problems from the Mesolithic period. They find palaeopathological evidence of deficiencies of vitamins C and D and of iron, anaemia, fluorosis and protein-energy malnutrition. However, because the conditions that caused malnutrition may be sporadic or random, they vary in expression in both space and time. In summary, Ortner and Theobold (2000) believe that the evidence shows that the level of health *declined* with the change from the Mesolithic hunter-gatherer way of life to the later period of developed agriculture. Cohen and Armelagos (1984) agree that the evidence is consistent with the view that farming was accompanied by a decline in the quality of nutrition. As we shall see, this chronic malnutrition became a subtle regulator of human population dynamics.

In contrast, Meiklejohn and Zvelebil (1991) found variability in the health status of populations connected with the Mesolithic–Neolithic transition. Part of this variability was related to diet, and they concluded that, for Europe, no significant trends in health were visible in the skeletons of those populations that made the transition from hunter-gathering to a greater dependence on agriculture, and from mobile to relatively sedentary communities.

## 1.6 Domestication of animals

In the early Neolithic, as the glaciers receded and the climate softened, herbivorous animals began to multiply, and, in the case of sheep and goats, their

growing numbers probably found easy meals in the grains that humans were raising. It is suggested that the new farmers ceased chasing the animals away and captured them instead. The next step from capture was domestication. Tribesmen in the Zagros mountains of Iran and Iraq began to keep goats in captivity about 10 000 years ago, making the species the earliest known to have been domesticated by man. Sheep were tamed and farmed about 7000 years ago in lowland areas of Iraq and Turkey and were soon being herded, with the herdsmen and their flocks spreading out far and wide.

The domestication of bovines began around 6000 BC. Pictorial records from the Sahara to Mesopotamia show that dairy farming began there between 4000 BC and 2900 BC, and pieces of pottery from an Iron Age site at Stanwick, Northamptonshire, England, dating from around 1000 BC, have been found which bear evidence of the traces of milk. However, it must have been much more difficult to domesticate bovines than to subjugate sheep and goats, although the rewards were great; in addition to the meat and milk and hides provided by the cattle, the ox was put to work along with sheep and goats at stomping seeds into the soil, threshing grain, pulling carts and wagons and (later) the plough.

## 1.7 Interactions between demographic pressures and diet

Boserup (1965) proposed a new model of economic growth in human history and argued persuasively that population growth rather than technological progress had been the main stimulus for economic change. Primitive behaviour, although usually considered to be a function of ignorance, might be seen as an efficient adjustment to a small social scale. So-called progress might simply be a necessary demographic adjustment to increasing population size, scale and density, and might be associated with declining rather than improving labour efficiency, and declining rather than improving individual welfare.

It was proposed that the initial adoption of farming by prehistoric hunter-gatherer groups which began about 10 000 years ago (the Neolithic Revolution) might also have been a grudging response to ecological stress or population pressure on resources, i.e. the Neolithic Revolution might not have been the result of technological progress as had previously been assumed (Cohen, 2000). This argument was extended by suggesting that much of what had passed for progress in prehistory might, like the adoption of farming, have been a response to the pressure of a growing population, rather than the result of new inventions, since the new 'progressive' techniques seemed to represent the input of extra effort for relatively little output. These 'improvements' would include the adoption of diets based on small seeds and the development of grindstones to process them and also the development of small projectiles for hunting small game (Cohen, 2000).

## 1.8 Height and nutrition

It has long been recognised that there is a close association between the height of a population and its nutritional status, and Europeans living in Europe have been the subject of a large number of anthropometric surveys (Harris, 2000). Research suggests that heights are a good indicator of nutritional status in childhood (Schofield & Reher, 1991). Much of this work has been surveyed by Floud *et al.* (1990) who concluded that, in broad outline, the average nutritional status has markedly improved in Britain since 1750, but the increase has not been smooth nor uninterrupted. Height grew in the period of the Industrial Revolution, fell back in the middle of the nineteenth century, gradually climbed back to its previous peak by the time of the first World War, grew slowly between 1918 and 1939 and then accelerated after World War II.

The data were derived, in the main, from medical examinations of military recruits who, almost exclusively, were drawn from the working class and so were very important from a demographic standpoint. Comparable results, with a steady rise in height at conscription, were found in other European armies from 1860 to 1980. Thus, the mean heights of working class males in European countries before 1750 were substantially below those of hunter-gatherer communities today – good, but indirect, evidence for a suboptimal diet in England before 1750, a continuing legacy from the adoption of an agricultural way of life with a diet weighted towards grains. These findings were reinforced by the study of Fogel *et al.* (1983) who found that the mean height of boys aged from 14 to 16 from families of London labourers, 1770–1865, correlated well with Tucker's index of the real wage of London artisans. Floud and Wachter (1982) suggest that certain portions of the late eighteenth century population of London could properly be described as stunted.

We have no comparable data sets for the seventeenth century, but anecdotal evidence suggests (see section 1.9) that the bulk of the population were of small stature and hence we conclude that they suffered from a suboptimal diet.

## 1.9 The working class diet in pre-industrial England

Rotberg (1983) concludes that 'Despite the importance of food in human history, we have few reliable records on which to base a sensible analysis of nutrition and malnutrition in the past'. Henry VIII consumed gargantuan (but not necessarily well-balanced) meals, with a high intake of meat, a practice that was followed by royalty and the nobility. Drummond and Wilbraham (1991) conclude that all the indications are that the tables of the rich were heavily laden with meat and fish from the earliest times, but these people were of little demographic significance. Poorer families had a different type of diet, consisting mainly of bread, cheese, salt meat (predominantly pork) and pulses. Because many cottagers kept a cow in

the sixteenth century, milk was more likely to be drunk by these families than by the wealthy, although milk drinking was rare in towns because of poor supplies, a situation that was not remedied until the late eighteenth and early nineteenth centuries. Until at least the late eighteenth century there were regional differences in milk-drinking habits; the north of England drank milk more than was usual in the southern counties. However, as Eden (1797) makes clear, grain was the dominant form of food for all except the rich; the lower the income available to a household, the greater was the dependence on grains (Walter & Schofield, 1989).

Eden (1797) assembled a mass of data relating to the expenditure and diets of the poor in different counties in England in the late eighteenth century, and Drummond and Wilbraham (1991) presented a careful appraisal of diets in England over five centuries, together with a biochemical analysis of their nutritive value. Their findings are summarised in the following paragraphs.

The Englishman of every class in medieval and Tudor times fed better than his counterpart on the continent. Of course, the peasants suffered badly when the harvest failed, as they still do today in primitive agricultural countries, but in good years they were relatively well nourished. The general diet of the ordinary country people of England was of coarse 'black bread' (maslin, barley, rye or beanflour), milk, cheese, eggs and occasionally bacon or a fowl. Dairy produce was generally known as 'white meat'. The peasant seldom ate meat in the Middle Ages and his diet is summarised in Table 1.2, column (i); this would have been partially supplemented by vegetables, fruit and wild berries in season. The general character of the food of a poor countryman seems to have steadily improved and the villagers were better fed during the fifteenth and early sixteenth centuries. Drummond and Wilbraham (1991) conclude that this must have been in most ways a good diet, although much would have depended on the amount of white meats eaten by the peasant and his family. The proteins of milk, whey, cheese and eggs are of high biological value, i.e. they contain all the amino acids essential for growth and maintenance. These proteins of animal origin, when eaten with proteins of lower biological value, such as those of cereals and pulses, supplement the value of the latter, so that the resultant mixture has much the same protein value as that of the animal proteins alone.

Protein deficiency is not nowadays considered to be a widespread problem among adults, even in the Third World, but the children, particularly in ultra-poor families, run a considerable risk of reaching a critical condition because of a negative protein balance during the hungry season. They need a higher proportion of protein in their diet for growth and, furthermore, the standards of child care are apt to decline during the busy agricultural season, and infants tend to be weaned on poor quality foods (Gill, 1991).

The second half of the sixteenth century in England saw a sharp turn for the worse when prices rose alarmingly and there was widespread unemployment. Many of the villagers were no longer able to keep a cow to provide them with

**Table 1.2** The estimated constituents of different diets from the fifteenth to the eighteenth centuries.

| Fifteenth century peasant diet (per day) | Fifteenth century diet, meat-eating classes (per day) | Seventeenth century diet when white meat became scarce (per day) | Longshore sailors' diet, 1615 (per day) | Diet of St Bartholomew's Hospital, 1687 (per day) | Navy ration, 1745 (per week) |
|---|---|---|---|---|---|
| (i) | (ii) | (iii) | (v) | (vi) | (vii) |
| 1 pt milk | 4 oz cheese | 3½ oz cheese | 8 oz cheese | 1½ oz cheese | 12 oz cheese |
| 1 pt whey | 1½ lb meat | 2 lb bread | 4 oz bacon | 1 pt milk pottage[2] | 4 lb salt beef |
| 2 oz cheese | 6 oz herring | 9 oz pease | 4 oz butter | 4 oz beef or mutton | 2 lb salt pork |
| 1 oz bacon | 1 oz fat | | 1 lb biscuit | 1 pt broth | 8 oz butter |
| 2 lb maslin bread[1] | 1 lb bread | (iv) | 3 oz oatmeal | 1 oz butter | 7 lb biscuit |
| 2 oz pease | 1 pt wine | Without cheese | 1 gall beer | 10 oz bread | 2½ lb oatmeal |
| | 1 qt ale | | | 3 pt beer | 2 pt pease |
| | | | | | 7 gall beer |

*Source:* Drummond and Wilbraham (1991).
*Note:* pt = pint, oz = ounce, lb = pound, qt = quart (=2 pints), gall = gallon (= 8 pints).
[1] Bread made with mixed grains, usually wheat and rye.
[2] A porridge of oatmeal or of peas.

dairy products and meat soon reached a price beyond their slender means. They had to fall back on the simple diet of earlier times, bread, peas and beans, together with what they could gather or trap in wood or field, river or pond.

People with comfortable incomes fed well in the seventeenth century, but bread, beef, beer and cheese were the food of the working class. When money was short or prices were high, meat was replaced by cheese or by thick broths prepared with dried peas and beans (see Table 1.2, column (iii)). Garden vegetables were being grown in greater quantities by the cottagers but were rather despised by the townspeople, probably because they came to be regarded as the food of poverty. A longshore sailor's recommended diet in 1615 and the diet of St Bartholomew's Hospital in 1687 are given in Table 1.2. Drummond and Wilbraham (1991) suggest that the diets of the seventeenth century were deficient, particularly in vitamin A and iron, which would have had severe consequences in pregnancy (see section 7.14).

There was an improvement in England in agricultural output and a rise in the standard of living of the working people during the first half of the eighteenth century, although the smaller tenant farmers felt the effects of a rather long period of low returns and some fell on bad times and were forced to give up their farms. The conditions of most villagers improved and they were able to keep a cow, a pig and a few ducks and chickens so that dairy products came to be included in their diets. They could also cultivate a variety of vegetables. Although in the south of England the staple diet was bread, butter and cheese, most were able to eat meat once or twice a week. They ate less meat but drank more milk in the north, where potatoes became the most important vegetable.

Conditions in England changed completely in the second half of the eighteenth century when the rapid growth of the population outstripped the production of grains, with the consequent soaring of prices of wheat and other staple foods. A large proportion of the population faced dearth, depression and distress by the end of the century. The contrast between south and north became greater because the effects of enclosure were much more marked in the south and because the potato gave the poorer people of the north a measure of protection against the shortage of corn which affected the whole country. Eden (1797) reported that, in 1795, the village labourers in the south of England were living almost exclusively on bread and cheese, while milk did not form a substantial part of their diet because the value of grass land in the vicinity of large towns was too high for the labourers to rent it to advantage. Practically all the milk produced on large farms was converted into butter or cheese and the whey was used for feeding pigs.

Thus, surprisingly, at the end of the eighteenth century, the inhabitants of northwest England, who had been suffering from marginal living conditions for over 200 years and had suffered on occasion from outright famine, enjoyed a better-balanced and more nutritious diet than their counterparts in southern England. The nutritive values of the diets of the subsistence, tradesmen and elite classes in northwest England are discussed in detail in section 9.5.

A summary of the constituents of some of these diets in different centuries in southern England is given in Table 1.2 and their analysis by Drummond and Wilbraham (1991) is presented in Table 1.3. Of note are the estimated calorific values of diets (i) and (iii), representative of working-class people in the fifteenth and seventeenth centuries, respectively. Shammas (1984) estimates the daily calorific content of the diet per adult to be 2823 in the north and 2109 in the south of England at the end of the eighteenth century, which she compares with a value of 2000 needed by a woman today, unless she is pregnant or lactating when the requirement rises to about 2500 calories per day. In addition, she considers that the manual labour performed by early modern men and women may have raised the optimum considerably. Often 3500 to 4000 calories are recommended for those engaged in heavy physical labour, such as working in the fields when both sexes helped with the harvest. Also, children from the age of 10 need greater amounts than their elders in order to grow. In this context, the 2100–2500 daily calories available to people in the south would not seem to provide the energy for hard labour or for growth in children and the 2800–3200 calories in the daily diet of northerners was, at best, barely adequate. Moreover, by adopting an agricultural way of life, the availability of food and energy fluctuated on an annual basis, resulting in severe deprivation at certain times of the year (e.g. the hungry season, see Chapter 13).

There is an additional point that has been overlooked by nutritionists and historians. Humans, being homeotherms, have a high basal metabolic rate and use a high proportion of their energy intake in maintaining their body temperature. The weather in Europe during the Little Ice Age of the seventeenth

**Table 1.3** Analysis of the approximate nutritional values of the diets listed in Table 1.2, together with the daily requirement of a moderately active man in the twentieth century.

| Twentieth century | | | Diet number | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | (i) | (ii) | (iii) | (iv) | (v) | (vi) | (vii) |
| Energy value | (cal) | 3000 | 3200 | 4750 | 2850 | 2450 | 5800 | 2350 | 5500 |
| Protein | (g) | 87 | 100 | 200 | 110 | 90 | 150 | 70 | 160 |
| Fat | (g) | NA | 60 | 190 | 40 | 10 | 250 | 80 | 180 |
| Calcium | (mg) | 0.8 | 1.6 | 1.3 | 1.1 | 0.3 | 2.6 | 0.9 | 1.9 |
| Phosphorus | (g) | NA | 2.8 | 4.2 | 2.3 | 1.7 | 3.7 | 1.7 | 3.7 |
| Iron | (mg) | 12 | 17 | 39 | 25 | 24 | 24 | 14 | 36 |
| Vitamin A | (IU) | 2500 | 1450 | 2800 | 1300 | trace | 6350 | 3200 | 1750 |
| Vitamin $B_1$ | (mg) | 1.2 | 1.9 | 1.5 | 2.3 | 2.3 | 1.9 | 1.1 | 2.6 |
| Riboflavin | (mg) | 1.8 | 2.8 | 3.7 | 1.3 | 0.8 | 3.9 | 1.7 | 4.0 |
| Nicotinic acid | (mg) | 12 | 22 | 68 | 33 | 33 | 84 | 40 | 100 |
| Vitamin C | (mg) | 20 | ? | ? | ? | ? | ? | ? | ? |
| Vitamin D | (IU) | NA | 9 | 950 | 14 | 0 | 100 | 25 | 26 |

Approximate nutritional value of diets expressed per day.
NA not available.
From Drummond and Wilbraham (1991).

century was very much colder than today, particularly during winter. The dwellings of the bulk of the population were cold and draughty and poorly insulated, fuel was in short supply and clothing lacked the sophistication of outdoor wear today. Drummond and Wilbraham (1991) describe their plight at the end of the seventeenth century:

> 'By that time their condition had deteriorated to such an extent that they were lucky if they had meat once a week, and when they did they usually had to send it to the baker to be cooked owing to their own lack of fuel.... It was of course the system of enclosures that had taken away their pasturage and the land where they collected the fuel for cooking their hot meals'.

We conclude that the energy intake overall was clearly suboptimal and it is not surprising that the children, who have high protein requirements, grew up to be small of stature. We describe in section 6.3 the importance of leptin in establishing the build-up of fat reserves in the adipose tissue which are essential for fertility and a successful pregnancy. With a suboptimal intake of energy, few working-class women would have been able to develop copious fat stores, so that fertility and neonatal life would often have been compromised.

There is a considerable body of evidence that the labouring population in England at various times before 1900 suffered from a deficiency of vitamins A, C and D and of iron (Drummond & Wilbraham, 1991). However, the incidence of scurvy, rickets, small stature in children, xerophthalmia and 'green-sickness', although further indicators of malnutrition and ill health, would have had little demographic impact. Much more important from our viewpoint is that they show that the diet of women during their childbearing years must have been deficient not only in energy, but also in a range of vitamins and minerals.

Indeed, it is difficult to understand how the working classes (the bulk of the population) succeeded in maintaining their numbers when suffering from such chronic malnutrition. But, from 1600 to 1750, the rural communities in England remained in a steady state or increased very slowly. These were often saturated habitats balanced on a knife-edge; if the population numbers rose, density-dependent constraints (operating via exacerbated malnutrition) reduced fertility and increased child mortality, thereby returning the community to steady-state conditions (see Chapter 5). We conclude that people must have become at least partially adapted over many years to these clearly suboptimal conditions.

The subtle effects of malnutrition on fertility, infant mortality and childhood susceptibility to lethal infectious diseases are not readily detected but, as we shall see in the following chapters, they indirectly controlled the population dynamics of rural England for over three centuries.

Chapter 2

# Mortality Oscillations in 404 English Parishes – a Metapopulation Study

Wrigley and Schofield (1981) studied 404 rural parishes in England during the period 1539 to 1871 and determined simple aggregated totals of burials and baptisms. These parishes were concentrated in the Midlands, whereas the Northern Territory and Cornwall were under-represented. The corrected, total, annual burials and baptisms aggregated for the 404 parishes are shown in Figs 2.1 and 2.2, respectively. It is evident from both figures that the overall rural population was rising only very slowly over the first 200 years, from 1550 to 1750, during which time the mean annual number of baptisms doubled and burials increased some 1.5-fold. The population numbers in rural England rose much more sharply after 1750.

The aggregation of data from 404 parishes tends to obscure any regular oscillations in individual populations. Nevertheless, the burial data series reveals evidence of major plague epidemics up to 1670 with outbreaks of unknown lethal diseases over the following 80 years. The scale of the ordinate also hides low-amplitude oscillations; the baptism series of Fig. 2.2 for the years 1539 to 1759, when the trend was only gently rising, is shown as an inset. Apparent irregular oscillations are now evident by eye.

## 2.1 Use of time-series analysis techniques

Time-series analysis is a computer-based statistical technique that is of great value to demographers. It is based on the fast Fourier transform, which regards any complex waveform or oscillation as the sum of sine waves. It is based on the concept of least squares, which, briefly, involves an estimation of the departure from a linear relationship in terms of sums of the squares of the deviations from values predicted by the regression line. It can be used for the analysis of demographic data series (such as annual baptisms, marriages or Bills of Mortality) to detect regular oscillations that occur which are otherwise masked by the noise and the overall trend. We use the technique frequently in this book, particularly in Chapters 3, 5, 10, 15 and 16.

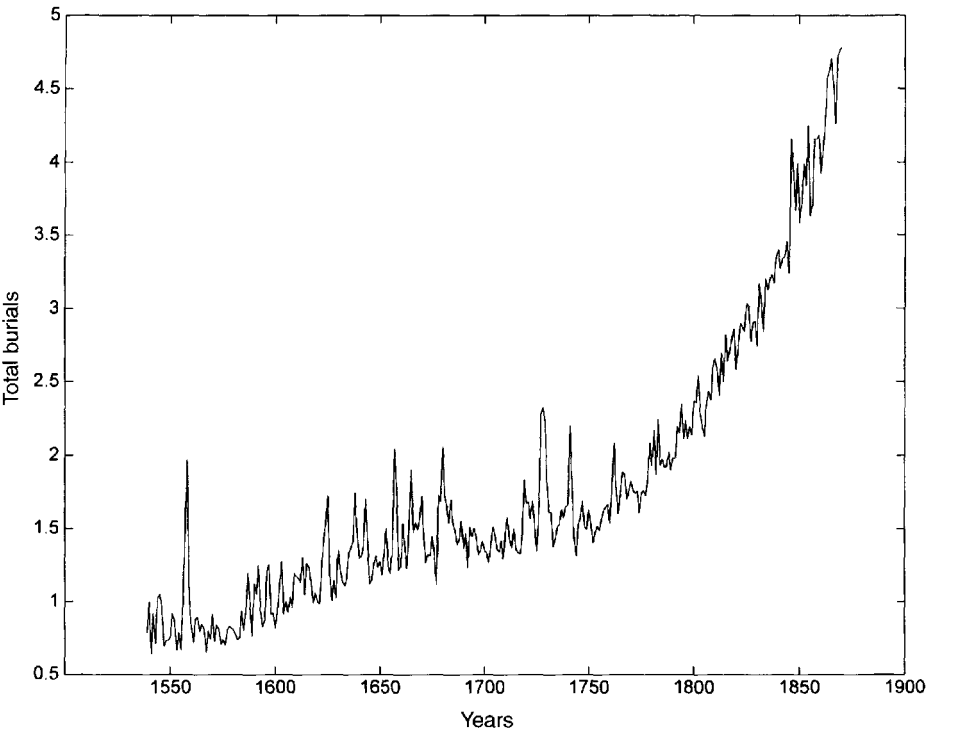


**Fig. 2.1** Annual burials in the 404 rural parishes in England, 1539–1873. Note the major mortality crises before 1750 and the change in the slope of the line thereafter. Ordinate: total burials $\times$ $10^{-5}$. Data from Wrigley and Schofield (1981).

We have described the technique of time-series analysis in detail previously, giving worked examples (Scott & Duncan, 1998). In brief, there are three main programmes of use to demographers:

(1) *Spectral analysis.* The data series is fed into the computer program which analyses the relative importance (or strength) of the different cycles contained within the series and identifies their period (i.e. the number of years for a complete cycle or oscillation). The significance of these cycles can be tested on the program in MATLAB.

(2) *Filtering.* It is difficult to observe specific cycles in data by visual inspection because of random variability and, under such circumstances, smoothing the white noise series by a moving average, or by the more sophisticated process of filtering, removes unwanted oscillations and also, if desired, the trend. In this way, the use of linear filters can enhance the interesting part of the data. Filters can be designed to pass oscillations of any desired frequencies (the filter window) that have been identified by spectral analysis.

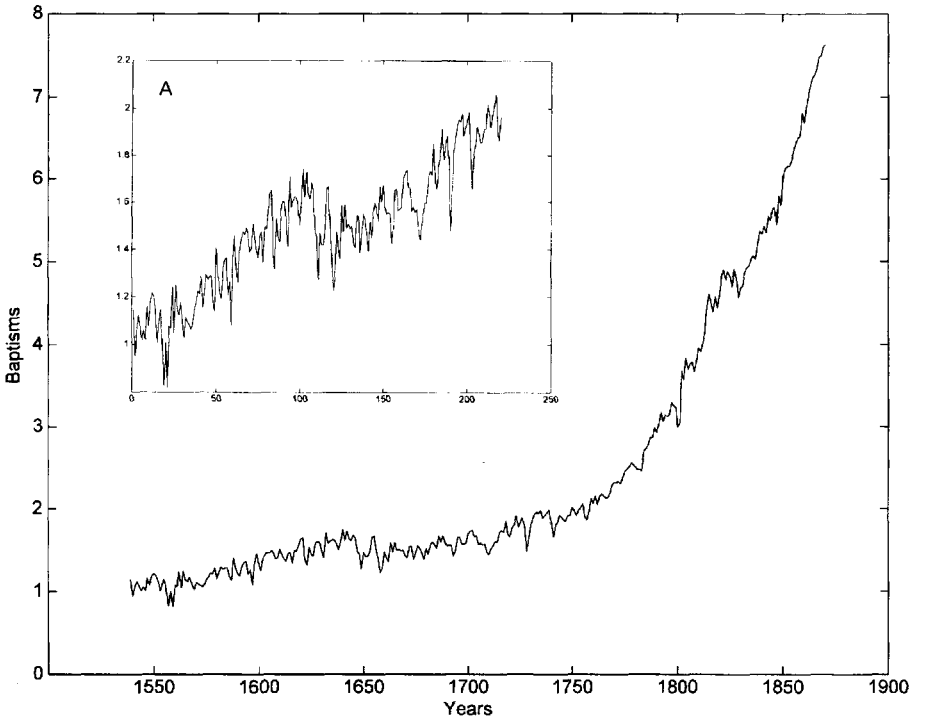


**Fig. 2.2** Annual baptisms in the 404 rural parishes in England, 1539–1873. Ordinate: baptisms $\times 10^{-5}$. Inset (A): baptisms 1539–1759; ordinate $\times 10^{-5}$; abscissa = years after 1539. Data from Wrigley and Schofield (1981).

(3) *Cross-correlation function (ccf).* This program compares two filtered data series over a standard time period and provides an estimate of the significance of the correlation between them (the larger the cross-correlation value, the stronger the relationship) and of the delay (or lag) between the two cycles. A significant correlation does not necessarily indicate causality.

Time-series analysis of the baptism and burial series of the parish registers of England reveals (Duncan *et al.*, 1992) three types of oscillations:

(1) Endogenous, long wavelength; period 43–44 years
(2) Endogenous, medium wavelength; period 30–33 years
(3) Exogenous, short wavelength; period about 5–6 years.

Endogenous population oscillations are discussed in Chapter 5; we describe here the exogenous oscillations that can be detected in aggregation of the 404 parishes of rural England.

## 2.2 Exogenous oscillations in 404 parishes

The use of time-series analysis is illustrated in the following study of the data series contained in Figs 2.1 and 2.2. Spectral analysis of the burial series for the years 1539 to 1719 reveals a complex output (see Fig. 2.3) in which long wavelength elements of the trend dominate and swamp other oscillations. The period of each peak can be determined as the reciprocal of the frequency (shown on the abscissa). A 13.8-year ($P < 0.05$) and (most importantly for our purposes) a 6.7-year ($P = 0.05$) oscillation are evident. This latter short wavelength oscillation in burials persisted through into the nineteenth century, although it then became weaker. It is shown after filtering in Fig. 2.3A.

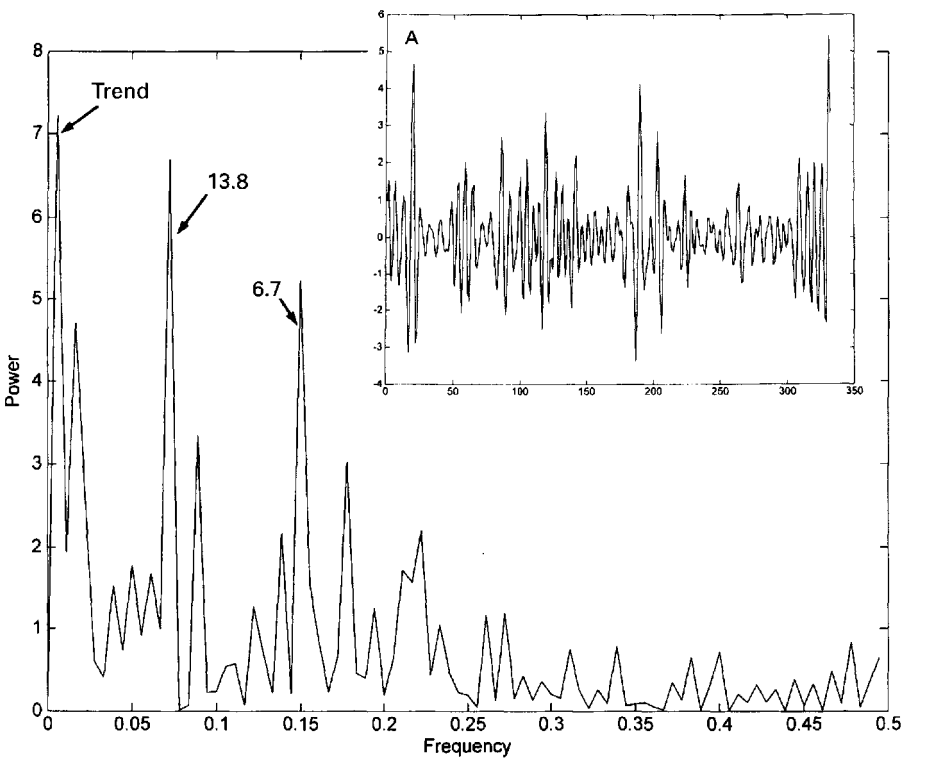


**Fig. 2.3** Spectral analysis of the annual burials shown in Fig. 2.1 for the period 1539–1719. The oscillations with a period of 6.7 and 13.8 years are indicated. Inset (A): filtered annual burials, 1539–1873 (ordinate); filter window = 4 to 10 years; abscissa = years after 1539.

In summary, this short wavelength oscillation in total burials (i.e. infants plus children plus adults) persisted for 300 years and is detectable even when the data from 404 parishes are combined. We conclude that not only must this oscillation in mortality have been present in the majority of these small, separate, rural populations but that (most importantly) *they must have been synchronous.*

## 2.3 The role of wheat prices in driving exogenous population oscillations

The oscillation in burials in the 404 parishes has all the hallmarks of being exogenous, i.e. driven by external factors. We have suggested (see section 1.5) that when annual wheat prices were high they caused hardship, famine and, ultimately, mortality among the bulk of the population of England. We describe in Chapter 3 how different cycles can be detected by time-series analysis in the annual grain prices series of England and suggest the different external factors that may have caused them.

The filtered wheat price series and burial series (filter window = 4 to 10 years) can be compared by using the cross-correlation program. The ccf for the 220-year period, 1539–1759, is excellent and, overall, the two series correlate at a lag of +1 years (i.e. they were synchronous). A ccf (wheat series versus burial series) for the period 1639–89 is shown in Fig. 2.4 (note zero lag for this period). In spite of all the variability that must have existed between the constituent populations and over this long time period, the two series were strongly, positively cross-

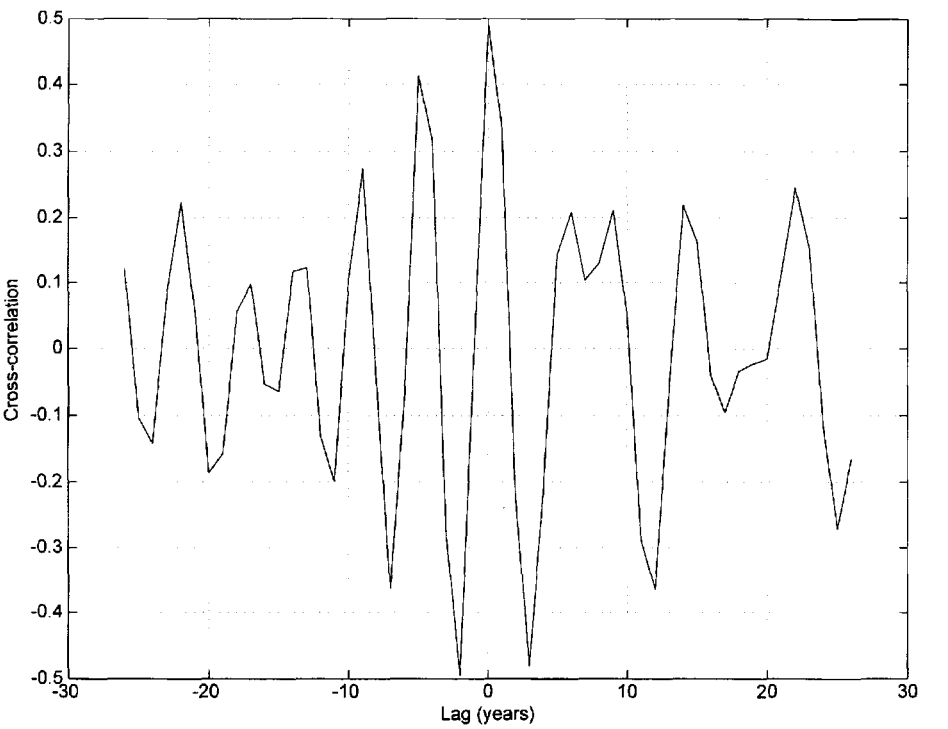


**Fig. 2.4** Cross-correlation function: wheat prices versus annual burials in the 404 rural parishes, 1639–89. Ccf = 0.5 at zero lag.

correlated, as evidenced by the marked central peak. The analysis of the cross-correlation in 50-year cohorts gave the following results:

1539–1588: ccf = +0.68; +2 years lag
1589–1638: ccf = +0.5; lag = 0 to +1 years
1639–1688: ccf = +0.5; zero lag (see Fig. 2.4)
1689–1738: ccf = +0.33; lag = +1 year
1739–1788: ccf = +0.38; zero lag
1789–1838: ccf = +0.34, lag = +1 year

The two oscillations were either synchronous (zero lag) or a year of high wheat prices was followed by a high number of burials in the next year (lag = +1 year).

We conclude that the short wavelength oscillation in the total burial series of rural England over a 300-year period was exogenous and was driven by regular periods of malnutrition and hardship which can be measured by the oscillation in wheat prices. This sensitivity to fluctuating levels of malnutrition is detectable even in an aggregated series that is predominantly composed of parishes in the Midlands in England where conditions were much more favourable than in the Northern Territory (Thirsk, 1967). Thus, time-series analysis can be used to elucidate the not otherwise readily-detectable effect of regular cycles of malnutrition on mortality and hence on population dynamics. We shall show that the *fluctuating* availability of grain was of great importance for some 300 years in regulating the demography of England, particularly via its direct and indirect effect on infant mortality (see Chapter 10). We begin, in Chapter 3, with an analysis of the oscillating grain prices, identifying the periods of the different cycles, tracing their evolution and determining their different causes.

## 2.4 Short wavelength oscillation in baptisms in 404 parishes

A short wavelength oscillation can also be found by time-series analysis in the aggregated baptism series from the 404 parishes (see Fig. 2.2) and this is displayed after filtering in Fig. 2.5. Time-series analysis shows that the filtered burial and baptism series (filter window = 4 to 10 years) are strongly cross-correlated *negatively* for the period 1539–1759, i.e. there was a low number of baptisms in the years of high mortality. Analysis of the cross-correlation between the two series in 50-year cohorts gave the following results:

1539–1588: ccf = −0.56; lag = 0 to +1 years
1589–1638: ccf = −0.68; lag = −1 to 0 years
1639–1688: ccf = −0.72; zero lag
1689–1738: ccf = −0.72; zero lag
1739–1788: ccf = −0.68, zero lag

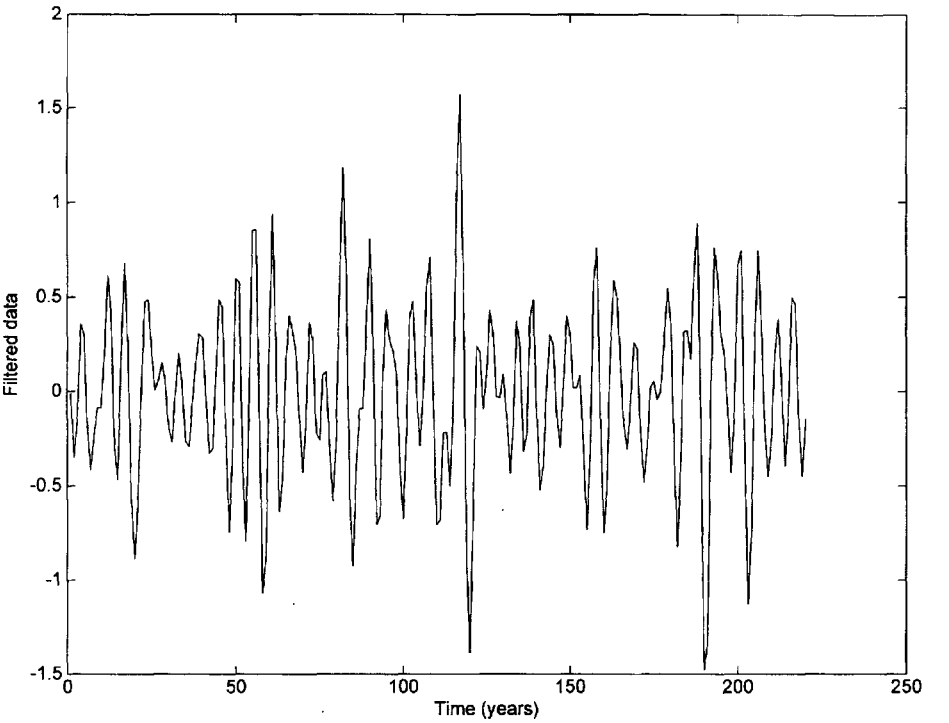


**Fig. 2.5** Filtered annual baptisms (ordinate $\times 10^{-4}$) in the 404 rural parishes, 1539–1759 (i.e. before the population boom). Filter window = 4 to 10 years. Abscissa = time after 1539.

The ccf for the years 1640 to 1740 is shown in Fig. 2.6.

This short wavelength oscillation in baptisms is discussed in greater detail in Chapter 5, after the description of a matrix model of the population dynamics of a community. It is an exogenous cycle that is driven both directly and indirectly by malnutrition caused by high wheat prices:

(1) Directly: malnutrition suppresses births and raises stillbirths.
(2) Indirectly: a mortality cycle generates a births cycle at the same period, but 180° out of phase (see section 10.5).

## 2.5 Conclusions

The 404 parishes studies by Wrigley and Schofield (1981) represent the meta-population of rural England during the period 1539–1873, i.e. a population of individual populations (parishes). Each maintained its own individuality while being in close communication with its neighbours via trade and migration. Aggregation of such a large number of individual series usually smoothes out the

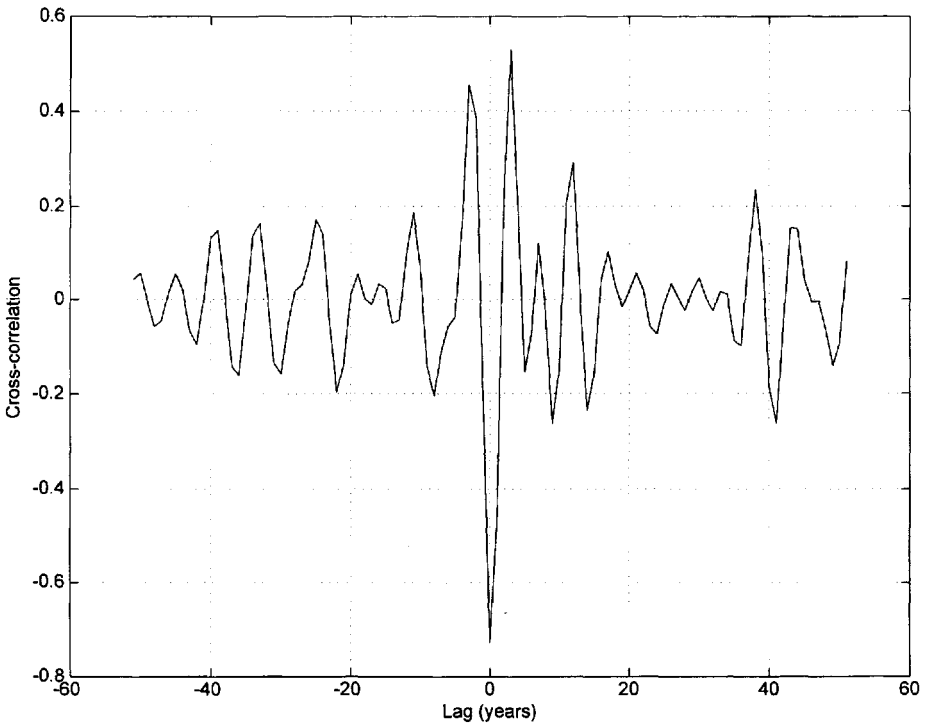


**Fig. 2.6** Cross-correlation function: annual burials versus annual baptisms in the 404 rural parishes, 1640–1740. Filter window = 4 to 10 years. Ccf = −0.72 at zero lag.

results and eliminates the noise, but time-series analysis of the burial series shown in Fig. 2.1 reveals a significant short wavelength oscillation. Since it is detectable in an aggregated data series, we conclude that this oscillation in mortality was present and was synchronous in most communities in the meta-population of rural England. This short wavelength oscillation was probably non-stationary (i.e. its period tended to vary slightly) but had an average period of about 6 years. It cross-correlates with a comparable cycle in wheat prices and we conclude that this mortality cycle was exogenous, i.e. that it was directly driven by an oscillation in the levels of malnutrition. The élite classes would not show this sensitivity to grain prices, but they were of little demographic consequence. The bulk of the population were managing to exist and to reproduce and maintain their population numbers with difficulty on a suboptimal (and doubtless boring) diet. Any annual (or even seasonal) reductions in the availability of grains (their staple food) was reflected demographically in raised levels of mortality (particularly of infants, see Chapter 9). The working classes in England had, therefore, to contend with a general suboptimal level of nutrition, superimposed on which was a marked oscillation in the price (and hence in the availability) of their food.

In the chapters that follow, we analyse which sections of the rural population of

England were most sensitive to *fluctuating* nutritive levels, and show that the most important demographic effects, which effectively controlled the population dynamics, were via the mothers before, during and after pregnancy. We analyse the oscillations in the national prices of grains in the next chapter and seek to determine the underlying causes of these fluctuating levels of malnutrition which had such a profound effect on human demography after the Neolithic Revolution.

Chapter 3

# The Staple Food Supply: Fluctuating Wheat Prices and Malnutrition

We have seen in Chapter 2 that a short wavelength oscillation can be detected by time-series analysis in the aggregated burial series for the 404 parishes of rural England and that this correlated with a comparable oscillation in the wheat price index. This does not necessarily mean that there is a causal relationship between the two series, but it is strongly suggestive of a cycle of mortality in the meta-population that was driven by a cycle of malnutrition. In Chapter 10, we explore the effects of the oscillations in grain prices on the infant, child and adult burial series in individual populations and show how these mortality series reacted differently to the cycles of malnutrition.

First, in this chapter, we trace the origins of the different oscillations in the national grain price series in England that can be detected by time-series analysis, and suggest what may have been the factors on which they were dependent. It has been suggested that the annual grain prices provide a good measure of the fluctuations in nutritive levels, particularly among the poorer sections of a community, and that, in certain circumstances, the annual price of grains interacted with the price of other commodities to produce years of particular hardship (see section 4.10). Communities living under marginal farming conditions would be particularly susceptible to such years of hardship.

## 3.1 Hypotheses to account for fluctuating grain prices

Grain prices in past centuries were subject to violent fluctuations from one year to another, although some regularity in the longer-term movement of prices has been discerned. The prime cause of these annual fluctuations has been ascribed to the weather which, in the short run, largely determined the supply of agricultural produce and this dependence meant that prices fluctuated more markedly than the prices of other commodities. Modern economic systems are provided with a number of built-in stabilising influences, but these conditions were largely absent in England in the sixteenth and seventeenth centuries and the

dramatic fluctuations in grain prices had to be endured as one of the penalties of a backward economic system, in particular the consequence of the inelasticity in demand and supply conditions which distinguished arable agriculture from most other economic activities. Drought was normally advantageous to the wheat harvest in England and, unless prolonged, was seldom a cause of failure among the other cereal crops because of the predominance of heavy moisture-retaining clay land over lighter sandy soils. The main danger to cereal crops is believed to be prolonged rainfall in summer accompanied by an almost total absence of sunshine when crops failed to ripen and yields were poor. An excessively cold winter and spring were also said to be harmful to the wheat harvest but had little effect on spring-sown barley, oats and pulses (Thirsk, 1967).

Any study of the fluctuations in grain prices in England is dependent on the pioneering work of Hoskins who provided an analysis, decade by decade, from 1480 to 1759. He suggested that really bad harvests came one year in six, agreeing closely with the popular belief in the sixteenth century that a bad harvest came once every seven years. It also corresponds with the short wavelength oscillations in wheat prices and burials described in Chapter 2. His examination of harvest sequences revealed no discoverable rhythm and he suggested that good or bad runs were not basically because of weather cycles and, although short-term climatic fluctuations may well have been implicated, he believed that the underlying factor was that of yield ratios in an agrarian economy that worked normally on a very fine margin between sufficiency and shortage (Hoskins, 1964). Hoskins concluded that, for the vast majority of the population, these short-term fluctuations in the cost of food 'were infinitely more important than the long-term movements to which economic historians have always paid such assiduous attention' (Hoskins, 1968).

Pfister (1988) studied the significance of meteorological variables in determining grain prices in Europe for the period between the mid-sixteenth and mid-nineteenth centuries, using a model based on monthly estimated measurements of temperature and precipitation, and distinguished between weather and non-weather effects. Subsistence crises were found to be triggered by the combination of a cold spring and a rainy midsummer whereas, in the long run, grain prices were more closely related to the intensity and duration of trade cycles. Spectral analysis and autoregressive models of agricultural prices in pre-nineteenth century Europe have shown that the prices of all cereals and potatoes rose simultaneously in subsistence crises and that regional fluctuations in the prices of cereals existed in Germany because of differences in production, commercialisation and consumption practices (Roehner, 1991).

The existence of long wavelength cycles in Europe, with a period greater than 30 years, in the prices of cereals from the sixteenth to the twentieth century has been studied by Bos *et al.* (1986); such cycles are clearly different from the short-run fluctuations described by Hoskins. Only weak indications of such long waves were found, although there was strong evidence of cycles with 10–13 or 15-year

periods, which seemed to be caused by clusters of unfavourable events in population, climate, structures of production or modes of behaviour. There was no evidence of a relation, as had been previously suggested, between these medium wavelength oscillations and sunspot activity.

Two theories have been advanced to explain the fluctuations of grain prices: first that demand remains constant but rigid and cannot adapt itself to fluctuations in the harvest, and second, that the amount of grain offered to the market is subject to fluctuations by the producers, who consume a certain amount of their own harvest. Subsistence farming and the instabilities of supply and demand must be included in these models of the grain market. During good harvests, subsistence farmers reduce their purchases in the market and increase the amount of grain they offer to it; during bad harvests, they increase their purchases in the market and reduce the amount of grain offered. It is concluded that the perceived demand on the market does not reveal as much as the total demand including susbsistence farming (Simonin, 1990).

## 3.2 Sources for the data series

Seasonal mean temperatures in central England (°C) from 1659 to 1811 were taken from Manley (1974), and seasonal rainfall at Kew, England from 1697 to 1811 (expressed as percentages of the 1916–50 averages) were taken from Wales-Smith (1971). Winters include the month of December from the previous year. The national grain price indices, 1450–1749 (wheat, barley and oats) were taken from Bowden (1967, 1985); the baseline for 1450–1649 was the mean grain price for 1450–99; the baseline for 1650–1749 was the mean value over these years. Absolute wheat prices (shillings) for 1600–1812 were taken from Stratton (1970).

## 3.3 Cycles in the wheat price index

The English national wheat price index for the early years, 1450–1599, is shown in Fig. 3.1, and the genesis of the oscillations can be clearly seen. The cycles were of low amplitude before 1550 and spectral analysis of this period shows a dominant oscillation with a wavelength of 8 years. This low amplitude, short wavelength oscillation can be seen after filtering in Fig. 3.1A. A weak, medium wavelength oscillation in the wheat price index for these early years before 1550 is also detectable; its wavelength is 10 years. The earlier stability is lost after 1550, the index shows a clear rising trend (Fig. 3.1) and oscillations at relatively greater amplitude emerge: spectral analysis of the period 1550–1600 shows a short wavelength oscillation and a weaker medium wavelength oscillation with a periodicity of 10 years. The emergent, short wavelength oscillation after 1550 has a wavelength of 5–6 years and can be seen after filtering in Fig. 3.1A. In summary,

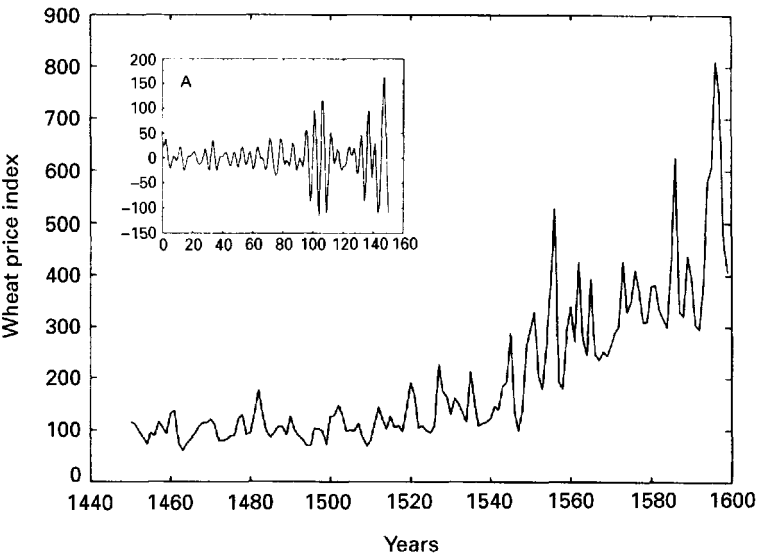


**Fig. 3.1** Annual national wheat prices index (ordinate) for the early years, 1450–1599. Note rising trend and the emergence of a short wavelength oscillation after 1550. Inset (A): wheat price index, 1450–1599, filtered to reveal the short wavelength oscillation (filter window = 4 to 10 years); abscissa = time in years after 1450. Data source: Bowden (1967).

the low amplitude, short wavelength oscillation with a period of 8 years during the period 1450–1550 changed sharply after 1550 into a 5–6-year oscillation of much greater amplitude, which was superimposed on a rising trend, as is clearly shown in Figs 3.1 and 3.1A.

This rising trend levelled out and the two types of oscillation in the wheat price index (short- and medium-wavelength) became more regular and were well established after 1600 (Fig. 3.2A). Spectral analysis for the period 1600–1749 (i.e. before the population boom in rural England, Fig. 2.2) shows the short wavelength (period = 6 years; $P < 0.005$) and the medium wavelength (fluctuating periodicity, predominantly 12-year wavelength, but changing to a strong 16-year cycle which developed after 1660; $P < 0.005$) cycles. The short wavelength oscillation during this period is shown in Fig. 3.2B after filtering; a more detailed analysis of the series shows that the wavelength was predominantly 8 years during the early years but stabilised thereafter at 6 years. The strong medium wavelength cycle, also found during 1600–1749 and continuing until 1812, is described in greater detail below and these different oscillations in the wheat index are summarised in Table 3.1.

## 3.4 Oats and barley price indices

The English national indices for oats and barley prices for the period 1450–1749 are given by Bowden (1967) and the trends and their pattern of oscillation cor-

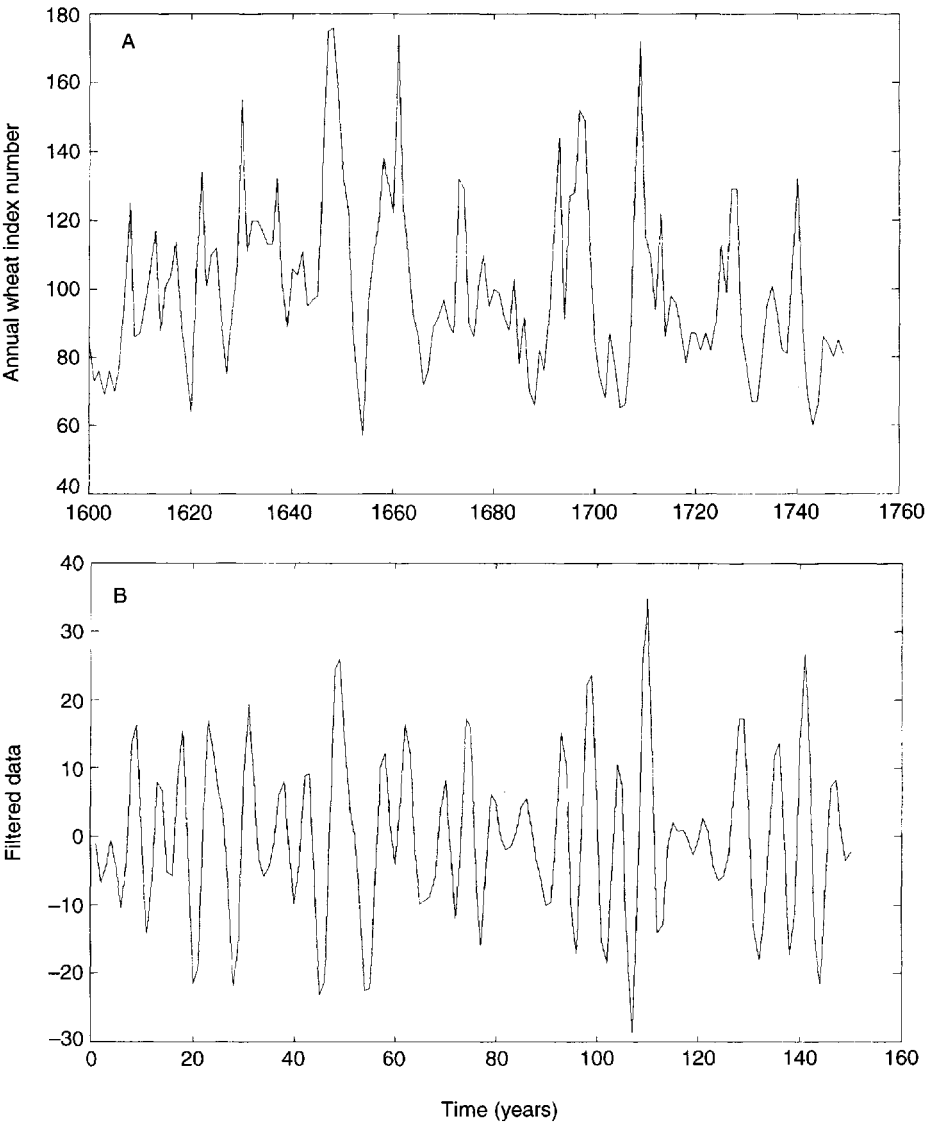


**Fig. 3.2** Annual national wheat price index, 1600–1749 (A). Filtered to reveal the short wavelength oscillation (B); filter window = 4 to 10 years.

respond closely with that of the wheat prices and with each other. The two series are compared in Fig. 3.3. Thus, the indices of both barley (Fig. 3.3A) and oats (Fig. 3.3B) also remained steady for the first 100 years (1450–1550), with short wavelength oscillations of very low amplitude (see insets, Fig. 3.3); after 1550, both series also showed a rising trend with the short wavelength oscillations superimposed (periods = 5 years for barley and 6 years for oats), which developed strongly over the next 50 years, corresponding to the pattern in wheat prices.

**Table 3.1** Characteristics of the cycles in the English national wheat index, 1450–1749.

| Cohort | 1450–1550 | 1550–1600 | 1600–1749 |
|---|---|---|---|
| Short wavelength | 8 | 5–6 | 6 |
| Medium wavelength | 10 | 10 | 12 and 16 |

Periodicity of different cycles was determined by spectral analysis of the unfiltered and filtered data series.

Filtered series are shown in the insets of Fig. 3.3. A medium wavelength oscillation with a period of 12–13 years was also detectable in the barley index during 1450–1550 and this changed to 10 years during 1550 to 1600. No significant medium wavelength oscillation was detected in the oats index (see Table 3.2).

The short wavelength oscillations became firmly established in both the barley and oats indices during the period 1600–1749 and are clearly seen after filtering (see Fig. 3.4). The wavelength of the oscillation in the barley index was 6 years during 1600–80, but changed to 5.5 years thereafter. Oats showed a 5-year periodicity throughout. A strong, medium wavelength oscillation with a periodicity of 12 years ($P < 0.05$) was clearly shown by spectral analysis in both the barley and oats indices.

## 3.5 Correspondence between the grain price indices in England

The apparent correspondence between the different grain price indices was tested on the cross-correlation program and the results are summarised in Table 3.2, which shows that both the oats and barley indices synchronise closely with the wheat series. They are strongly coherent or synchronise in the short and the medium wavebands ($P < 0.01$) over a period of 300 years (see Table 3.2). An example of the cross-correlation function, wheat series versus barley series, for 1600–1749 is shown in Fig. 3.5 and a similar analysis shows that the barley and oats series are also significantly and strongly cross-correlated throughout the period 1450–1749.

We conclude that the annual price indices of the three grains exhibit clear short and medium wavelength oscillations that are strongly interdependent and oscillate together in both wavebands.

This finding confirms previous suggestions. Bowden, (1967) concluded that the short-term movements in the prices of wheat, barley, oats and rye were broadly similar because each grain was in some degree a substitute for the others, while any extremes of climate in spring or summer affected the yield of all. The graph of the prices of wheat, beans, peas and oats at Lincoln, England, 1543–1620, shows that these commodities also moved in close sympathy with each other (Hoskins, 1964).

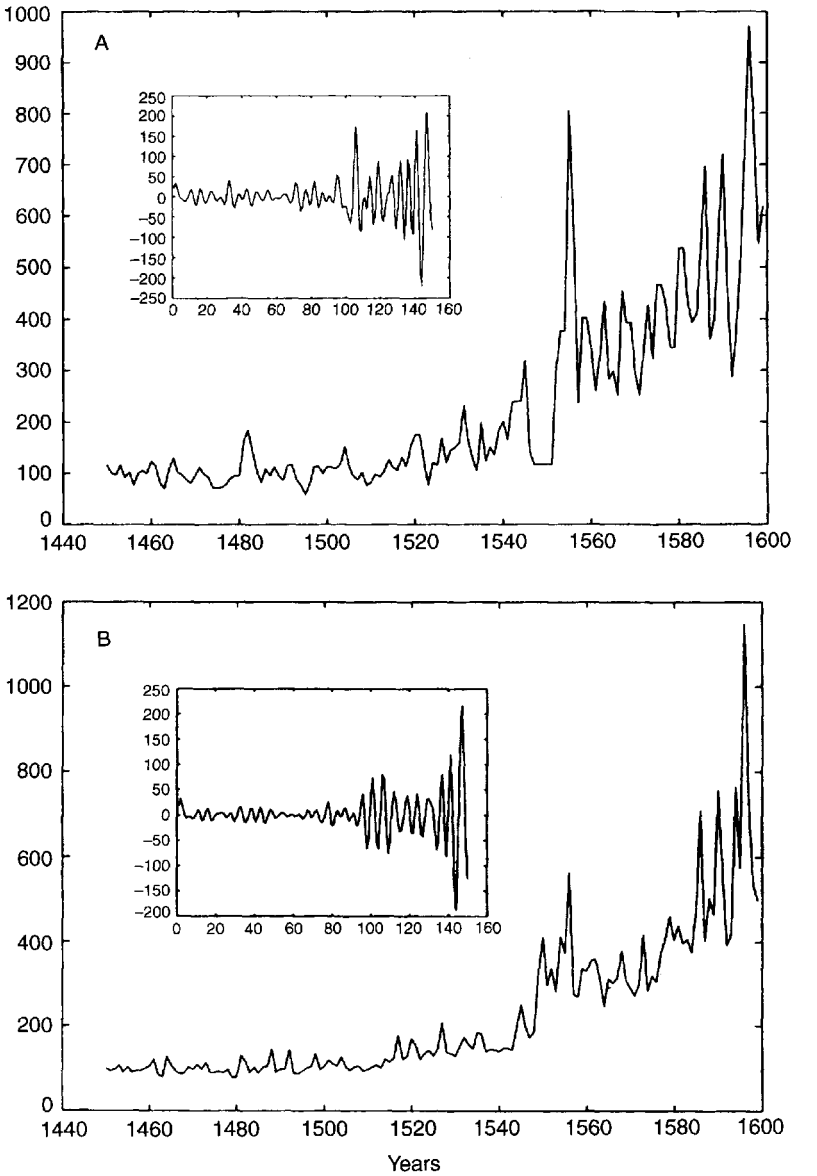


**Fig. 3.3** Annual grain prices, 1450–1599 (ordinates). A: Barley prices; B: oats prices. Insets: data series filtered to reveal the short wavelength oscillation; filtered window = 4 to 10 years; abscissae = time (years) after 1450. Data source: Bowden (1967).

Since the prices of the different grains in England were so closely linked, we have used annual wheat prices as a measure of the fluctuating levels of nutrition in pre-industrial England in the following chapters, and in the next section we explore the different factors that may have caused the clearly-defined cycles in the price of this grain.

**Table 3.2** Synchrony of short and medium wavelength oscillations in the grain prices indices.

| Cohort | 1450–1550 | | | | 1550–1600 | | | | 1600–1750 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Ccf with wheat: | Periodicity (y) | Lag (y) | ccf | *P* | Periodicity (y) | Lag (y) | ccf | *P* | Periodicity (y) | Lag (y) | ccf | *P* |
| Barley: short | 6 | 0 | 0.6 | 0.001 | 5 | 0 | 0.7 | 0.001 | 6→5 | 0 | 0.8 | 0.001 |
| Oats: short | (4–6) | 0 | 0.8 | 0.001 | 6 | 0 | 0.8 | 0.001 | 5 | 0 | 0.6 | 0.001 |
| Barley: medium | 12→13 | 0 | 0.7 | 0.01 | 10 | 0 | 0.7 | 0.01 | 12 | 0 | 0.9 | 0.001 |
| Oats: medium | ND | 0 | 0.6 | 0.001 | ND | 0 | 0.8 | 0.001 | 12 | 0 | 0.9 | 0.001 |

*Note:* Table shows cross-correlation and coherence between wheat versus oats or barley indices. Note high significance of coherence in both the short and medium wavelength oscillations. Arrow indicates progressive change of wavelength during the 150-year cohort. Ccf, cross-correlation function. ND (oats) indicates no medium wavelength oscillations were detectable by linear filtering, although good ccf and *P* values were obtained between the two series.

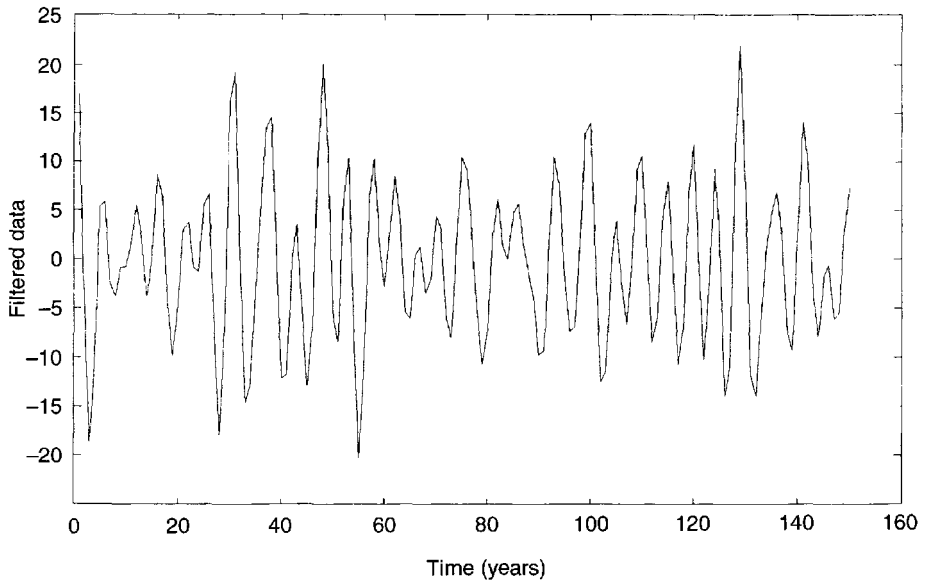


**Fig. 3.4** Annual national oats price index, 1600–1749, filtered to reveal the short wavelength oscillation (filter window = 4 to 10 years). Data source: Bowden (1985).

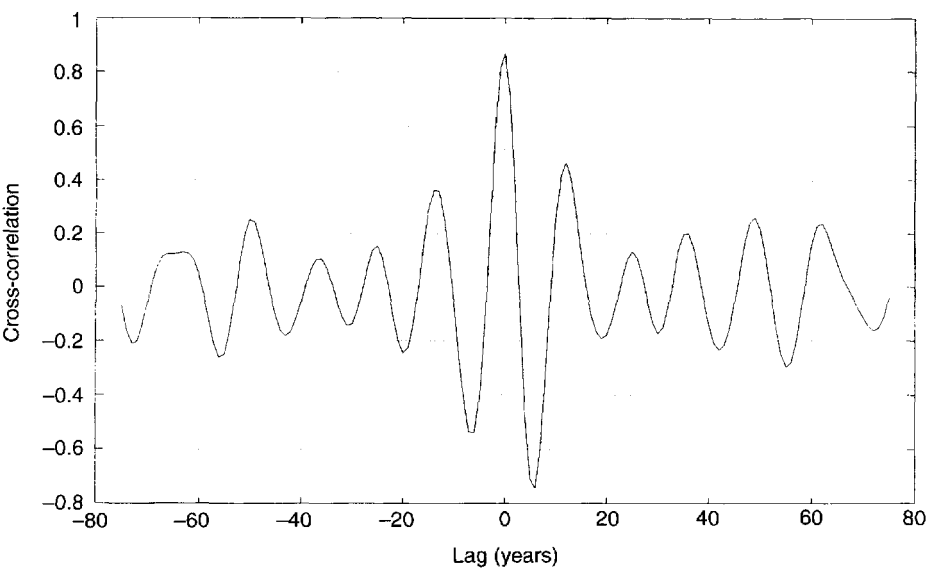


**Fig. 3.5** Cross-correlation function, wheat index versus barley index, 1600–1749. Filtered data (filter window = 8 to 20 years). Maximum peak of ccf (+0.87) is at zero lag.

## 3.6 The effect of seasonal temperatures on wheat prices

The weather and British agriculture are both such varied quantities that the interplay between them is certain to be complex. There is a range of interactions stemming from the diversity of the weather and, except for extremes, the temperature and rainfall requirements of agricultural crops are seldom the same, so that the conditions that are ideal for one may be unsatisfactory for another (Bowden, 1985). Extreme weather events tend to cluster and the recurrence of clustered years of meteorological extremes appears to be the outstanding feature of 1200–1850. There were three main epochs of rises in grain prices in Europe, i.e. 1300, 1600 and 1800, and the biggest of these changes was a many-fold increase between 1550 and 1650 (see section 3.3 and Fig. 3.1), a phenomenon too widespread to be attributable to any of the wars of the period. The price rise in 1800 was largely because of the influence of the Napoleonic War, but it also coincided with the latest period of advance of the glaciers and of the Arctic sea ice surrounding Iceland. Although it has been concluded that the changes around 1300 and 1600 were largely climate-induced (Lamb, 1978), the rising trend of wheat prices between 1550 and 1650 and between 1731 and 1811 were also times when the population of England almost doubled and, for each period, the rising demand caused an increase in the price index that outstripped the rise in population.

The period 1550–1700 in England was also characterised by variability of temperature; the mid-sixteenth century saw the start of a period of cooling and there were persistent cold intervals particularly during 1605–15, 1674–82 and 1695–8. Severe winters were experienced in the 1560s and 1600s, and wet, cool summers occurred in 1570, the 1590s and 1620s (Lamb, 1978). The first half of the eighteenth century was relatively warm, although 1709 and 1740 were cold over most of the northern hemisphere. From 1770 onwards, there was a fairly steady drop in temperatures until 1820 (Landsberg, 1981). The milder climatic circumstances of 1652 and 1654 afforded a brief respite from periods of high prices during 1645–51 and 1656–63. However, the weather conditions up to 1691 were conducive to the production of grain and this period was noted for a number of warm, dry summers culminating in a drought in 1684. Unusual cold and exceptional rainfall in the 1690s led to crop failures in 1692–9 and high prices.

These changes in mean temperature are summarised in Table 3.3 and it can be seen that there was a rise in temperature from the seventeenth to the eighteenth centuries. A degree does not sound impressive, but small changes in the mean temperature may reflect significant differences in the frequency of occurrence of extreme values. In Iceland, a decrease in annual temperature of 1°C reduces the growing days by 27%, illustrating that small changes may be critical in marginal areas (Bryson & Padoch, 1981). There is little doubt that harvest yields in the period under study reacted more sensitively to weather fluctuations before the advent of hybrid seed, artificial fertiliser, mechanised harvesting and other agricultural advances of the twentieth century (DeVries, 1981).

**Table 3.3** Average temperatures for England, 1659–1812.

| Cohort | Average | Winter | Spring | Summer | Autumn |
|---|---|---|---|---|---|
| 1659–1699 | 8.6<br>(−0.027) | 2.9<br>(−0.021) | 7.5<br>(−0.028) | 14.9<br>(−0.026) | 9.1<br>(−0.034) |
| 1700–1749 | 9.2<br>(0.003) | 3.7<br>(0.004) | 8.0<br>(0.0004) | 15.4<br>(0.0003) | 9.8<br>(0.01) |
| 1750–1812 | 9.1<br>(0.003) | 3.3<br>(−0.004) | 8.1<br>(0.001) | 15.5<br>(0.002) | 9.4<br>(0.002) |

*Seasonal temperature data taken from Manley (1974). Figures in brackets are the slopes of the line during the cohort.*

The weather affects cereal crops in several different ways, some of which are closely linked to the season of the year or the stage of growth. Lawes and Gilbert (1880) examined the history of a long run of seasons and concluded that the most abundant wheat harvests had been preceded by above-average temperature during most of the winter and early spring. Most of these years had low winter and spring rainfall and slightly less than average summer rainfall. In contrast, the seasons of lowest wheat production had been characterised by severe, or at least changeable, conditions in the winter and spring with, at the same time, above average rainfall (Lawes & Gilbert, 1880). However, the most powerful determinant of the condition of the cereal harvest was believed to be the state of the weather at sowing, because the cereal suffered if the weather was cold and backward; hard wind, frosts and cold rain could damage young shoots (Holderness, 1989).

We examine the English wheat data series using different statistical techniques below and show that only the medium wavelength oscillation in wheat prices correlates significantly with weather conditions, in contrast with the short wavelength oscillation where the association is weak.

The possibility that wheat yields and hence prices were influenced by weather conditions during the growing and harvesting seasons has been explored by conventional multivariate regression analysis; data series for seasonal temperature are known from 1659 and for seasonal rainfall from 1697. The English wheat price series (taken from Stratton, 1970) rather than the wheat index has been used for this analysis because comparisons with oats and barley indices are not necessary here. Analysis of seasonal temperatures (winter, spring, summer and autumn) versus wheat prices for the complete period 1659–1811 shows that it is only winter temperatures ($P = 0.046$) that had a significant effect; i.e. low winter temperatures are associated with high wheat prices. When this effect of temperature is studied in different periods, the importance and significance of the effect of winter temperature rises: 1659–1780, $P < 0.01$ and in 1700–1750, $P < 0.002$. Additionally, a large negative coefficient was obtained from the analysis for summer temperature, suggesting that low temperatures in this season may

also affect wheat prices. Multivariate analysis for the period 1659–1780 of winter and summer temperatures shows that both have a significant effect on wheat prices (winter temperatures, $P = 0.01$; summer temperatures, $P = 0.005$). These findings have been confirmed by time-series analysis and, in particular, the correlation with seasonal temperatures in the different wavelength oscillations has been further explored in the following sections.

### *3.6.1 Effect of winter temperatures*

Spectral analysis of the winter temperature data series for the period 1659–1812 shows a dominant medium wavelength oscillation which has a period of 12–13 years. The winter temperature and wheat price series cross-correlate negatively at zero lag when they are filtered to reveal medium wavelength oscillations, i.e. low winter temperatures are associated with high wheat prices in the same year in the *medium* waveband.

### *3.6.2 Effect of summer temperatures*

Spectral analysis of the annual summer temperature series shows a peak in the *medium* waveband at 14 years. The cross-correlation between summer temperatures and wheat prices shows that the two series correspond negatively, i.e. low summer temperatures are associated with high wheat prices in the medium wavelength oscillation. The association is significant throughout the period 1659–1812 ($P < 0.05$), but is particularly significant during the earlier years, 1659–1780 ($P< 0.01$). Figure 3.6 shows the wheat price series (1659–1812) filtered to reveal the medium wavelength cycle period, which is predominantly 16.7 years ($P < 0.005$); it cross-correlates negatively with the filtered summer temperatures series (see Fig. 3.7).

We conclude tentatively that these results suggest that, in the *medium* waveband (period = 14–16 years), both low winter and low summer temperatures are associated with poor yields and high wheat prices, with summer temperatures probably having a more significant effect.

### *3.6.3 Principal component analysis of the seasonal temperatures*

Low temperatures in winter may well affect subsequent spring and summer temperatures, so producing generally cold growing and harvest seasons, and this possible interactive climatic effect has been explored by principal component analysis (see Scott & Duncan, 1998). In brief, the spring temperature series during the period 1659–1812 was found to be significantly correlated with the mean temperature in the preceding winter. Likewise, summer temperatures were also significantly correlated with those of the preceding winter, whereas there was no correlation with autumn temperatures. This study suggests, therefore, that

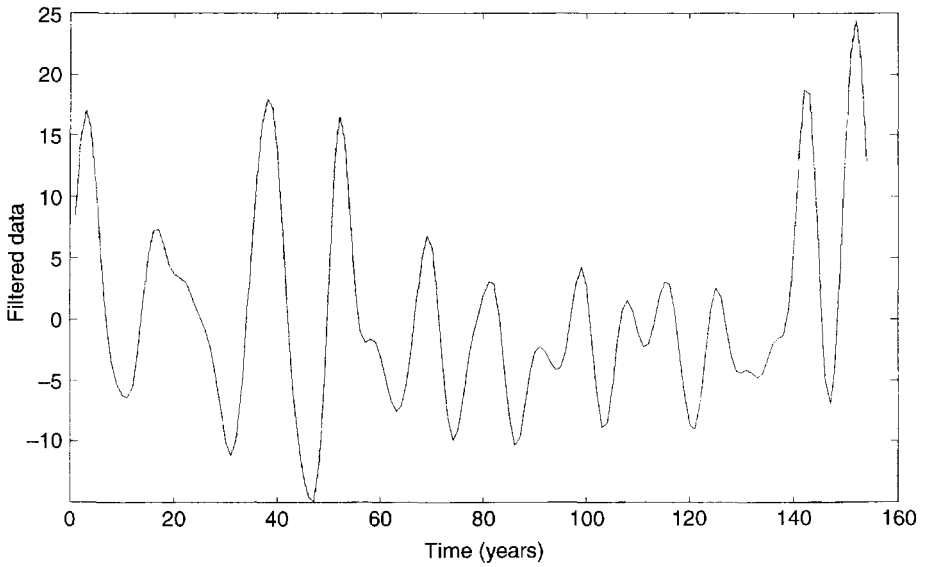


**Fig. 3.6** Annual national wheat prices (shillings), 1659–1812 (ordinate), filtered to reveal the medium wavelength oscillation (filter window = 8 to 20 years). Abscissa = time after 1659. Data source: Stratton (1970).

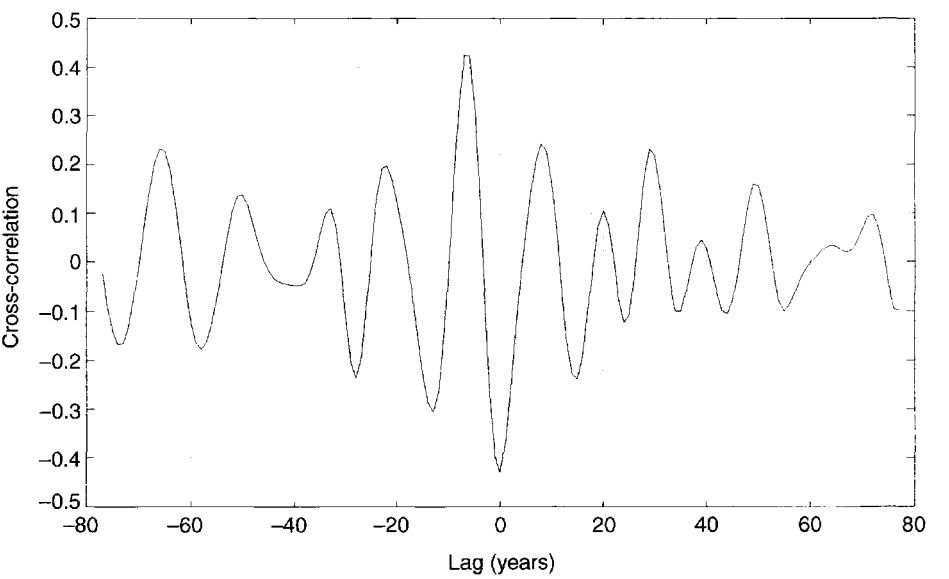


**Fig. 3.7** Cross-correlation function, wheat prices versus mean summer temperatures, 1659–1812. Filter window = 8 to 20 years. Minimum ccf (−0.43) at zero lag. Data sources: Stratton (1970) and Manley (1974).

the annual wheat price in the medium waveband was correlated with the weather conditions from winter to summer in that year. Multivariate analysis confirms that low winter and low summer temperatures have the dominant effect, but principal component analysis indicates that low winter temperatures may also be acting indirectly by causing low summer temperatures which then probably affect harvest yield directly by causing adverse growing conditions. Thus, the results of time-series analysis suggest that these effects of seasonal temperature are detectable in the medium wavelength oscillations of the wheat price series (period about 12–16 years; see Fig. 3.6) but are less strongly associated in the short wavelength oscillations.

## 3.7 The effect of rainfall on wheat prices

The multivariate regression analysis programme was then rerun including the data on seasonal rainfall as well as temperature over the period 1697–1812, but, again, only low winter temperatures showed significance ($P = 0.016$). When the analysis is restricted to the early years, 1697–1781, the overall analysis of variance shows that the regression is significant ($P = 0.005$), the significance of winter temperature is increased ($P = 0.001$) and the effect of winter rainfall now becomes significant ($P < 0.05$). However, there was no significant correlation between winter rain and wheat prices in the medium and short wavebands, but there is correspondence ($P = 0.025$) at high frequencies. We conclude that winter rainfall did not have a cyclical effect and acted only on a simple annual basis.

Wigley and Atkinson (1977) have suggested that it is not the level of precipitation, but the soil moisture deficit that may have the greater effect on plant growth. A dry soil and a continuing high level of evapotranspiration prevent the crop from withdrawing water from the soil, leading to stunted growth and even wilting of the plants. Their data series for mean soil moisture deficits during the growing season (May–August) at Kew, 1698–1812, was tested by time-series analysis and, as would be expected, high spring or summer rainfall correlated with low water deficits at zero lag. There is cross-correlation with wheat prices, both overall and in the filtered medium waveband, but at 1-year lag and with the two series being *negatively* correlated, i.e. most surprisingly, low wheat prices (and presumably good yields) were associated with drought conditions, the converse of the expectations of Wigley and Atkinson (1977). We conclude that this analysis confirms that rainfall and soil moisture deficit had only limited effects on the medium wavelength oscillation in wheat prices in England.

## 3.8 Wheat prices and short wavelength temperature cycles

Spectral analysis of the data series for winter or summer temperatures also reveal smaller and secondary peaks of wavelength 5 years in both. These short wave-

length cycles also cross-correlate negatively with the corresponding cycle in wheat prices (shown in Fig. 3.2B), i.e. again low temperatures synchronise with high wheat prices. We conclude that the short wavelength cycle in wheat prices correlates with these low seasonal temperatures, but the effect is less marked than in the medium wavelength cycles.

Spectral analysis of the data series for mean soil moisture deficits, 1698–1812 (Wigley & Atkinson, 1977), described above, reveals major peaks at wavelengths of 3 and 5 years, and the 5-year oscillation shows weak cross-correlation and coherence at 1-year lag with wheat prices but, paradoxically, a good growing season (reflected in low wheat prices) was again apparently correlated with drought conditions. We conclude that rainfall and moisture deficits in the soil are of only limited significance on wheat prices in the short waveband.

## 3.9 Use of a predicted wheat price series

Multivariate analysis has shown that certain seasonal weather conditions had strong effects on the annual wheat price, and time-series analysis has confirmed that the medium wavelength cycles were particularly sensitive. Using multivariate analysis, it is possible to construct a regression equation which predicts, theoretically, the wheat price in any year, based on the seasonal climatic conditions for that year, if it were influenced only by weather. Thus, a *predicted* annual wheat price during the period 1697–1811 can be calculated from the following equation, which uses the coefficients given in the multivariate analysis:

$$\text{Wheat price (shillings)} = 79.9 + (0.027 \times \text{summer rain}) + (0.0262 \times \text{autumn rain}) + (0.0783 \times \text{winter rain}) - (2.05 \times \text{summer temperature}) - (0.48 \times \text{autumn temperature}) - (3.61 \times \text{winter temperature})$$

Note the high coefficients for summer and winter temperature, indicating their dominant influence. Spectral analysis of this theoretical wheat price series shows the medium wavelength oscillation, confirming that this cycle in wheat prices can be explained as the consequence of weather conditions. However, the 6-yearly, short wavelength cycle, which is characteristic of the actual wheat price series (see Fig. 3.2B and Table 3.1), is of very low power. This confirms the suggestion that the short wavelength cycle may not have been *directly* linked to climatic conditions and that there were other factors producing this oscillation in the wheat price series. When the predicted annual wheat prices are deducted from the real prices, the resulting data series (the residuals) should represent those factors that were less related to the climatic factors on which the predicted price series was solely based. Spectral analysis of this residual series now shows that the only oscillation in the short waveband was of period 5.3 years, confirming that the

short wavelength cycle in wheat prices was not strongly driven by weather conditions (see Scott & Duncan, 1998).

One possibility is that wheat prices in the *short term* were influenced for economic and agricultural reasons by the prices declared in the preceding year, the so-called autoregressive effects. This hypothesis was confirmed by rerunning the multiple regression of winter and summer temperatures versus wheat prices in the preceding year. The temperatures now have reduced significance, whereas the autoregressive term is significant at $P < 0.001$, indicating the 'carry-over' effect of prices into the subsequent year.

In summary, there are, therefore, two different types of cycles in the English national wheat price series, medium wavelength (12–16 years) and short wavelength (5–6 years); it is the former that is most closely affected by weather conditions, whereas the short wavelength cycle in grain prices is primarily driven by other factors.

## 3.10 What drove the different cycles in wheat prices?

It has been shown that the colder the winter, the greater the month-to-month temperature variability (Tromp, 1980). Bergthorsson (1985) also found a strong relationship between hay yield and winter temperatures in Iceland between 1901 and 1975, and noted that cold winters were more effective than cold summers in restricting grass growth. Similarly, Eckstein *et al.* (1984) found that the annual Swedish harvest index was significantly related to annual winter temperatures from 1756 to 1869, but no significant correlations were found between the quality of the harvest and spring, summer or autumn temperatures or annual rainfall. DeVries (1981) found a significant negative correlation between the annual price of rye and annual winter temperatures in the Netherlands during 1635–1839. He also found that a critical factor influencing the level of arable crop yields in northwestern Europe was the amount of precipitation, particularly in winter; high rainfall levels between November and March delayed spring planting. These are short-term, irregular effects.

However, the results of multiple regression and principal component analysis suggest that weather (cold growing and harvesting seasons) had a significant effect on prices in the *medium* waveband. The effect of seasonal rainfall appears to be of secondary importance and is effective only on a simple annual basis, i.e. heavy rain at a specific period in summer could ruin a harvest. As we have seen, the short wavelength oscillation in English wheat prices is less significantly related to weather conditions.

What then are the other causes of the short wavelength cycles in English wheat prices? An index of prices is partly artificial, being driven by economic factors as well as by grain yields at harvest time. Cycles in oats, barley and wheat prices were synchronous and oscillated together; if one grain was in poor supply the

prices of the others were driven up also. The poor were prevented from substituting cheaper grains and the failure of the crops had a ripple effect that was felt throughout the economy. Appleby (1979) also noted that, because all grains in England rose in price together, it prevented the labouring poor from switching from their usual bread grains to cheaper grains and thereby staying alive. Harrison (1971) also recognised that all prices rose in years of high prices, although not to the same extent. Obviously, major transient (non-cyclical) weather events can contribute to a bad harvest in any one year (e.g. scorching of the oats crop in Lancashire in 1762). Hoskins (1968) suggested that the cyclical disposition of grain prices was not basically because of weather cycles but was related to the underlying factor of yield ratios.

One bad harvest, by reducing the yield ratio to dangerously low levels, tended to generate another because of the scarcity of seed corn, and it has been suggested that this effect was cumulative until a change in the weather pattern broke the sequence and restored the normal balance between bread corn and seed corn (Hoskins, 1968; Bowden, 1967). Wrigley (1989) noted the relationship between grain prices and the size of the harvest and suggested that fluctuations in price may reflect the level of grain stocks carried over, varying with the abundance of the harvest, and not the runs of deficient and abundant harvests.

These are so-called autoregressive effects in which the grain price is strongly influenced by the price in the preceding one or even two years. An autoregressive term included in the multiple regression in the present study is highly significant ($P < 0.001$) and a second autoregressive term is also significant ($P = 0.012$), showing that the wheat prices in the two preceding years have detectable effects.

The closer the farmer operates to subsistence level, the larger the number of years in which he will have no surplus to sell and must therefore enter the market as a buyer at high prices. For the sixteenth and seventeenth centuries, the English agrarian economy operated on a very fine margin between sufficiency and shortage and, where a society consumed almost all of its annual output within a year, thereby causing a dangerous shortage of seed corn, fluctuations in the harvest must have had over-riding economic significance. Such economic factors could have made a major contribution to the clear short wavelength oscillation in grain prices that emerged strongly at the end of the sixteenth century, but it seems that other regulatory mechanisms were probably implicated in generating such a regular oscillation, as described in the next section.

## 3.11 Rust and other parasitic infestations of grain crops

'To the farmer of this period pests and diseases were rather like the poor: he had them with him always. It was almost certain that he would lose some part of his crops to an attack by some fungus or insect. How much he lost is not told' (Brown & Beecham, 1989). Fungal diseases, such as mildew, loose bunt and rust, were the

major scourges of cereals and frequently destroyed whole crops. Rust diseases of cereals were the most damaging and, periodically, mankind has been plagued by severe outbreaks.

Could these fungal diseases be a contributory factor to the short wavelength oscillations observed in English grain prices? It is now known that rust had two alternative life cycles in one of which the fungus existed and underwent sexual reproduction during the winter on the barberry (*Berberis vulgaris*) which was found in woods and used in field hedges and boundaries and hence, once this fact was known, barberry bushes were destroyed in the late nineteenth century. Rust has the ability to cycle between these alternate hosts and, like other fungal diseases, can display endemic and epidemic conditions; for instance, a fungal parasite can be endemic with a long interdependent association with the plant host, when disease levels are low and vary little over time.Two kinds of rust have been identified: the orange leaf-rust, caused by *Puccinia rubigo-vera*, and the black stem rust, caused by *Puccinia graminis*. These parasitic fungi grow on a range of host plants, including wheat, oats and barley (Large, 1958).

The epidemic state of rust describes explosive outbreaks associated with an increase of intensity as well as of extensity, and a progressive epidemic occurs when the disease spreads to new regions and, after having reached a high level of severity during the early years of conquest, settles once again to endemic status. If the disease develops explosively, affecting a large number of individuals, it may spread to a new region where it will have the character once again of a progressive epidemic. Population bursts of fungi occur when diseased seeds are planted, or the fungus that has invaded the alternate host emerges. Shoots become diseased and the fungus spreads throughout the field from these primary diseased shoots; the fungus must increase about a billion-fold in order to destroy all the fields in the vicinity. Conditions that favour infection are susceptible host plants, a virulent pathogen and suitable weather conditions that are conducive to the disease. A lower than usual rate of infection suggests that some condition is adverse for the fungus, i.e. there are more resistant than susceptible host plants, or that the weather is too dry for a disease that needs moisture. If conditions remain constant from year to year, the epidemic will settle down and the infection rate will remain constant, but any departure from uniformity, whatever the source, could cause an increase in infectivity and the threat of an epidemic. For instance, depleted stocks of seed corn following a deficient harvest would decrease the amount of plant host available to the pathogen during the next season. Each year without an epidemic would increase the proportion of seed corn available for planting and, consequently, the number of susceptible host plants growing until conditions were suitable for an explosive rust epidemic, resulting in a much reduced crop yield. In this way, the process would be self-perpetuating and these 'boom-and-bust' cycles account for many of the most damaging epidemics of cereal diseases (Priestley, 1978).

Even during the early periods of agriculture in the seventeenth and eighteenth

centuries, the outbred wheat would show wide genetic variability with differing degrees of susceptibility to fungal attack; one of the major ways of developing this protection is by the development of a thickened cuticle in the host plant to prevent fungal entry. The farmer would, unconsciously, use a form of selective breeding in that the seed corn from a harvest during a severe fungal attack would, inevitably, tend to come from the resistant varieties of wheat plants that had survived to maturity. Consequently, a greater proportion of resistant grains would be sown and resistant plants would be harvested in the following year. However, the fungus which produces astronomical numbers of spores would, in turn, have been developing more active penetrant strains and so the boom-and-bust cycle of fungal epidemics would tend to develop. This cycling process is further complicated because at times when no significant rust infestations were present, the rust-resistant strain of wheat might be at a selective disadvantage to the non-resistant strains (the resistant gene would be at an advantage only when there is a major fungal infestation), so promoting the latter and exacerbating the development of another rust epidemic. Consequently, a 'technological arms race' between wheat and fungus develops in which successive resistant strains are overcome by more potent fungal strains, which succeed in overwhelming the defences of the host plant. Severe rust epidemics would be less likely to recur in the year following an outbreak and they would have an inherent periodicity dependent upon the time taken to build up a sufficient number and density of susceptible hosts.

We suggest, therefore, that the regular short wavelength cycle in grain prices was not greatly affected by cycles in the weather (unlike the medium wavelength oscillation) and that the peaks of the oscillations were sharpened by economic and autoregressive factors and possibly by regular epidemics of fungal diseases.

## 3.12 Conclusions

Conventional time-series analysis of the English wheat price series, 1450–1812, reveals a short wavelength (period 6 years) and a medium wavelength (period 12–16 years) cycle throughout this time, although they developed strongly only at the end of the sixteenth century. The comparable cycles revealed in the series of oats and barley prices are strongly coherent with wheat prices, i.e. the different grain prices moved in synchrony and, consequently, the wheat price series has been used in the following chapters as an index of nutritive levels. The medium wavelength oscillation in wheat prices correlates with weather conditions: low winter and summer temperatures are the most significant factors. Both may have a direct effect on the growth and harvesting of the crop, but cold winters may also have indirect effects by establishing a generally cold spring and summer. High winter rainfall is of secondary importance. However, the short wavelength cycle in wheat prices is not significantly correlated with weather and it is suggested that

it is driven by economic factors, the short-term effects of a good or bad harvest (autoregressive effects) and possibly regulated by regular epidemics of fungal pathogens of grain. These regular oscillations in the prices of the staple food of the bulk of the population had a dominant effect on the demography of rural England for over 200 years. The effects of this short wavelength cycle in grain prices in driving exogenous population cycles and the subliminal (and not readily detectable) effects of oscillating levels of malnutrition on fertility, infant mortality and the epidemiology of lethal infectious diseases are described in the following chapters.

Chapter 4

# Famine

## 4.1 Major famines in world history

There are a number of definitions of famine but all contain the necessary elements of widespread starvation with dramatically increased mortality. All formal definitions tend to lack precision, yet they clearly distinguish famine from other aspects of deprivation, such as chronic food insecurity, extreme poverty, high prevalence of wasting malnutrition and mortality, which characterise many regions of the developing world (West, 1999). On this definition, famine is catastrophic, distinct and a human tragedy of unparalleled proportion, with young children and women suffering the greatest risk of starvation. The demographic consequences are severe and obvious: heavy mortality, mass movements, reduced fertility and social upheaval.

Famines on these scales have afflicted mankind from antiquity and have had major effects on demography. Dando (1980) has identified a system of world famine regions for the period from 4000 BC to AD 1980. The first region is northeast Africa and West Asia (4000–500 BC): records of famines in Egypt in the third and second millenia BC describe the devastation wrought on the land and society. China experienced frequent famines from 108 BC to AD 500 (and also during the nineteenth century and again in 1959–60, when up to 30 million people perished). In the thousand years after 500 BC, the greatest concentration of accounts mentioning famine is found in the region that became the Roman Empire. Imperial Rome may have brought order and organisation to subjugated peoples, but Mediterranean Europe still experienced at least 25 major famines during this period. Eastern Europe experienced more than 150 recorded famines between AD 1500 and 1700 and there were 100 hunger years and 121 famine years in Russia between AD 971 and 1974 (Murton, 2000).

Asia emerged as the major famine area from AD 1700 to 1975 and the Bangladesh famine is described in section 4.4. The Great Irish Famine in the 1840s caused 1.5 million deaths and an equal number of migrations, mostly to America but also to England. Famines in the late twentieth century in Africa inflicted catastrophic mortalities, particularly in the Horn (Watkins & Menken, 1985; West, 1999).

There is a general assumption that these major famines were linked to acute food shortage in the region, but the causes, in reality, were more complex. The famines in the Horn of Africa resulted from the interacting effects of deteriorating crop production, droughts, failures in development and commerce, increasing use of agricultural land for growing export crops, the impact of repressive regimes, civil unrest and war. T. Dyson (unpublished report of a conference at Les Treilles, 1999) believes also that so-called 'bang-bang' famines in the past may well have been reinforced by epidemics, and these synergistic effects restricted population growth.

The Great Irish Famine (about 1844–8) is regarded as the greatest in Europe in the nineteenth century; it was triggered by a potato blight that stripped the country of the only staple food that Irish peasantry could afford to grow on their small parcels of land. However, during these years, there were substantial exports of wheat, barley, oats and animal products by landowners to English markets.

The Great Bengal Famine of 1943, when about 1.5 million people died, was originally considered to be because of a shortage in rice supply. However, later analysis showed that the famine occurred in a year during which rice production was only 5% lower than the average of the previous five years and when most economic indicators of Bengal were showing a wartime boom in growth. Rural food stocks, however, were being procured by the government to support military needs and to subsidise rations for civil servants in Calcutta, so driving up the price of rice in rural areas (West, 1999).

It is anticipated that global warming in the twenty-first century will lead to further major disruptions in the weather systems. Keys (2000) believes that exceptionally severe weather events have claimed between 50 000 and 100 000 lives since 1997 and up to 300 million people have been displaced and made homeless. These problems are predicted to get worse with a steadily rising world population and resources will become ever more scarce, leading to enormous demographic changes with mass migrations, poverty, starvation and famine.

## 4.2 The demographic impact of famine

> 'The only way that famines ... could have been a major deterrent to long-run population growth when the underlying normal mortality and fertility rates would have led to even moderate growth is if they occurred with a frequency and severity far beyond that recorded for famines in history' (Watkins & Menken, 1985).

For this reason, we have to look more deeply to determine the subtle demographic effects of famine, in both the short and long terms. Alamgir (1978) concluded that famine is

> 'a general state of prolonged food grain intake decline per capita giving rise to a number of sub-states (symptoms) involving individuals and the community as a whole which ultimately lead, directly or indirectly, to excess deaths in the country or region'.

It is this demographic impact of famine that has been explored by Hugo (1984) and we have relied heavily on his review in preparing this section. Although the scattered literature is predominantly of empirical case studies, the complex interrelationships are of interest to demographers seeking to develop theories of changes in mortality and also to geographers attempting to explain changes over space–time in population growth and composition. As Chen and Chowdhury (1977) have pointed out, famine 'is a complex syndrome of multiple interacting causes, diverse manifestations, and involving all three demographic variables – mortality, fertility and migration', although most commentators have followed Sorokin (1942) in concentrating upon theories that relate famine to mortality. These studies have been extended in premodern and modern populations by demonstrating how they undergo social and economic changes (the 'epidemiologic transition') in passing from high and fluctuating mortality to relatively stable low mortality in three well-defined stages, each characterised by particular levels of mortality and specific patterns of the cause of death: (i) an age of pestilence and famine, (ii) an age of receding pandemics with progressive decline in mortality when epidemic peaks become less frequent, (iii) an age when degenerative diseases dominate (Omran, 1971). Famine, as defined on this scale, is a chronic, long-standing process that is inextricably mixed with lethal infectious diseases and is not strictly applicable to this chapter.

While the effects of periodic famines on mortality are obvious, it has been shown (Hugo, 1984) that population growth rates in the modern period are more often affected by the impact of famine on population movement. The possible socio-cultural, economic, physiological, political and demographic responses in Third World countries, both short and long term, are summarised in Table 4.1 (see Hugo, 1984). Some of these adjustments and responses to famine are applicable to communities in pre-industrial England, whereas, for example, those listed under outside assistance are obviously not.

## 4.3 Changes in fertility

A decline in fertility is the rule during a famine (Watkins & Menken, 1985); the effects are the same as those experienced during extended periods of malnutrition except that they are more immediate, on a greater scale and more obvious. Bongaarts (1980) concludes in his summary of the evidence that 'malnutrition can impair the function of the human reproductive process. This effect is strongest and most evident in famine and starvation...'. Most studies show an initial

**Table 4.1** Community and individual responses to famine, with the emphasis on demographic effects.

| | Short-term adjustments | | | | | |
|---|---|---|---|---|---|---|
| | | *In situ* adjustments | | | *Ex situ* adjustments | |
| Dietary | Increased mortality | Fertility | Employment | Outside assistance | Temporary movement | Permanent movement |
| Lack of food<br>Less nutritious food<br>Different types of food<br>Kill wild animals or domestic animals<br>Malnutrition<br>Weight loss<br>Organised fasting within households | Infanticide<br>Greater incidence of infectious disease<br>Malnutrition<br>Excess deaths among vulnerable groups (aged, infants)<br>Socio-economic and spatial differentials | Abortion<br>Abstinence<br>Delayed marriage<br>Increased divorce<br>Prolonged lactation<br>Reduced fecundity<br>Lowered frequency of intercourse | Seek off-farm work<br>Farm and graze new areas | Remittances from family living elsewhere<br>Government and international aid | Nomadic herding<br>Commuting<br>Seasonal migration<br>Rural to urban migration | Emigration<br>Rural to urban migration<br>Settlement in new agricultural area |
| | Long-term adjustments | | | | | |
| | | *In situ* adjustments | | | *Ex situ* adjustments | |
| Lack of food<br>Less nutritious food<br>Different types of food<br>Smaller stature | Infanticide<br>Greater incidence of infectious disease<br>Malnutrition<br>Excess deaths among vulnerable groups (aged, infants) | Increased to insure against crisis mortality<br>Reduced because lower fecundity | Seek off-farm work<br>Farm and graze new areas | Remittances from family living elsewhere<br>Government and international aid | Establish regular seasonal migration pattern<br>Establish regular cyclic migration pattern | Emigration<br>Rural to urban migration<br>Settlement in new agricultural areas |

*Source:* Hugo (1984).

continuation of birth rates at prefamine levels, with a decline occurring some nine months after the onset of the famine. Some part of the change in fertility is probably because of the postponement of marriages that would otherwise have occurred (see Sorokin, 1942, 1975).

The reasons for the short term decline in births during times of famine are fairly well understood. As with mortality change, malnutrition plays a critical role. There is a decrease in fecundity when the nutritional value and quantity of food consumed fall below minimal levels: women stop ovulating and male sperm mobility and longevity are reduced (Bongaarts, 1980; Zeitlin *et al.*, 1982; see Chapter 6). Moreover, in times of famine there is likely to be increased foetal wastage because of the deteriorating condition of mothers, while women may breast-feed children for longer periods and so prolong post-partum amenorrhoea (Hugo, 1984). The evidence on stillbirth rates is contradictory: increases during periods of scarcity have been found by some researchers (Antonov, 1947; Smith, 1947) but not by others (Stein *et al.*, 1975; Chen & Chowdhury, 1977).

Sometimes there is evidence of a rise in fertility following a famine or other crisis, a rebound after the disaster. Two sources of such a rise may be the increase in nuptiality because of marriages delayed during the famine, and the remarriages of those whose spouses had died. Other explanations are primarily physiological. The death of a nursling that interrupts lactation, or a stillbirth during a crisis shortens post-partum amenorrhoea, and women who fail to conceive are not pregnant when the crisis is over. Either would leave a larger proportion of the female population of reproductive age at risk of another pregnancy. Fertility rose after the famine in China, perhaps by as much as 25% for the first post-crisis fiscal year (1962–3) and then declined again (Ashton *et al.*, 1984). Registered births in Madras rose for several years after the famine of 1876–8 (Lardinois, 1985), but it is impossible to separate an increase in the birth rate from increases in the number of births because many people migrated into the area (Watkins & Menken, 1985).

However, Hugo (1984) concludes that the limited data available suggest that in the short term famine has little impact on fertility 'partly because the deviations from normal levels are smaller than changes in the death rate and partly because the birth rate deficit is compensated by a period of excess births' (Bongaarts & Cain, 1981).

## 4.4 The Bangladesh famine of 1974–5: a case study

The Bangladesh famine of 1974–5 had severe demographic consequences when the people not only sold or mortgaged land, but also sold cattle and agricultural implements, cooking pots, plates, mugs, spoons and even toilet jugs (Alamgir, 1980). The crude death rates rose by nearly 60%, from 15.6 (1973–4) to 24.6 (1974–5), but deaths did not rise equally at all ages. As can be seen in Table 4.2,

**Table 4.2** Infant mortality rates (per 1000 births) and age-specific death rates (per 1000 population) in Matlab thana, Bangladesh, 1966–75.

| Year | Infant mortality rate | | | Age-specific death rates[1] | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Total | Neonatal[2] | Post-neonatal[3] | 1–4 | 5–9 | 10–14 | 15–44 | 45–64 | 65+ |
| 1966 | 110.7 | 59.5 | 51.2 | 24.9 | 4.1 | 1.7 | 4.1 | 15.3 | 67.9 |
| 1967 | 125.4 | 67.8 | 57.6 | 29.4 | 5.0 | 2.1 | 4.4 | 17.9 | 79.3 |
| 1968 | 123.8 | 82.9 | 40.9 | 23.8 | 3.9 | 1.7 | 3.7 | 17.4 | 74.4 |
| 1969 | 127.5 | 87.5 | 40.0 | 23.1 | 3.3 | 1.0 | 3.8 | 17.9 | 71.1 |
| 1966 | 131.3 | 89.9 | 41.4 | 27.9 | 2.3 | 1.3 | 2.7 | 14.4 | 72.9 |
| 1971 | 146.6 | 86.9 | 59.7 | 25.8 | 3.7 | 1.6 | 3.7 | 16.6 | 73.1 |
| 1972 | 129.2 | 71.9 | 57.3 | 36.9 | 11.4 | 2.2 | 5.1 | 20.0 | 119.1 |
| 1973 | 128.8 | 81.1 | 47.7 | 22.7 | 14.1 | 2.2 | 2.9 | 14.7 | 96.5 |
| 1974 | 167.2 | 74.8 | 92.4 | 29.7 | 6.5 | 1.6 | 4.4 | 23.8 | 109.3 |
| 1975 | 150.4 | 71.0 | 79.4 | 32.7 | 12.3 | 1.2 | 3.8 | 25.1 | 100.1 |

*Source:* Watkins and Menken (1985).
[1] Age in years.
[2] Age 0–29 days.
[3] Age 30 days–11 months.

compared with the averages for the previous five years, the greatest rises during the famine years occurred among children aged 1–11 months followed by children aged 5–9 years and persons over 45, in that order. Children 1–9 years experienced even higher mortality in the year after the famine than they suffered during the crisis. Because the neonatal death rate was virtually unchanged, infant mortality rose less than in the age groups already noted. These findings are corroborated by less detailed data from the Mysore famine of 1876–8, in which it was found that the greatest deficits in the census of 1881 occurred in numbers of children aged under 10 years and, to a lesser extent, among those over 50 (Lardinois, 1985).

Neonates, whose death rate was virtually unchanged (Table 4.2), may not be affected by sudden changes in the availability of food nor by varying cultural practices. In most historical populations, as well as most developing countries, they are likely to take most if not all of their nourishment from breast milk. Although the evidence is incomplete, it may be that the quantity and quality of breast milk are sufficient for the very young until the mother is nearly starved, so that they will be protected long after their elders are affected (Watkins & Menken, 1985).

Death rates for females in Bangladesh are normally higher than those for males at nearly all ages; the disadvantage of young females (age under 10 years) appears to have been exaggerated during the 1974–5 famine, but disaster diminished or even reversed the disadvantage of older women in most age groups by raising male death rates more than female death rates. Mortality in 1975 was higher for men than for women from the age of 25 years (Ruzicka & Chowdhury, 1978a).

In Bangladesh, the total fertility rate declined by about 33% between 1974 and 1975 (Chowdhury & Curlin, 1978; Ruzicka & Chowdhury, 1978a; see Table 4.3),

**Table 4.3** Age-specific fertility rate (per 1000 women) in Matlab thana, Bangladesh, 1974 and 1975.

| Age (years) | 1974 | 1975 | Percentage decline from 1974 to 1975 |
|---|---|---|---|
| 10–14 | 5.6 | 3.7 | 34 |
| 15–19 | 161.3 | 116.4 | 28 |
| 20–24 | 311.8 | 223.3 | 28 |
| 25–29 | 323.3 | 200.3 | 38 |
| 30–34 | 253.8 | 179.4 | 29 |
| 35–39 | 163.4 | 95.0 | 42 |
| 40–44 | 55.8 | 36.3 | 35 |
| 45–49 | 16.3 | 7.1 | 56 |

*Source:* Watkins and Menken (1985).

falling by nearly the same proportion in all age groups, so that there was no change in the age pattern of fertility as there had been in the age pattern of mortality. The decline in fertility in Bangladesh was not because of an increase in contraceptive use, since only 2.4% of married women used contraception in a survey conducted in October 1975 (Chowdhury & Curlin, 1978). Neither was it because of delayed marriage; in 1974 only 2.7% of those aged 20–24 were still single (Ruzicka & Chowdhury, 1978b). Thus the most likely explanations for fertility decline in Bangladesh are the effect of severe food deprivation on reproductive physiology, both male and female, and the effect of decreased coital frequency, whether induced biologically or resulting from increased stress or separation (Watkins & Menken, 1985). There was no fertility rebound after the famine in Bangladesh, where total fertility was virtually the same in 1976 as it had been before the famine (Ruzicka & Chowdhury, 1978b).

The effect of the Bangladesh famine has been examined by comparing the mortality rates of three cohorts of children born before, during and after the famine (Razzaque *et al.*, 1990). Mortality was higher in those born during the famine up to the second year of life, whereas it was higher only in the first year in those conceived during the famine. Thereafter, mortality in these two cohorts was low and the difference eventually disappeared after the twenty-fourth month. Post-neonatal mortality in the cohort born in the famine was higher among the poor than among the better off. The mortality of children of younger mothers (below the age of 20) born and conceived during the famine was particularly high in their second year.

## 4.5 The Dutch famine of 1944–5: a case study

The Dutch Hunger Winter of 1944–5 is a unique episode in human history: a well-documented, severe famine in an industrialised population which was clearly

delineated in place and time and occurring in a society with a well-developed administrative structure and where food supplies had previously been generally adequate (Trienekens, 1985). It affected the large cities of western Holland. The people of rural areas and small towns were better off than those in the cities because they could reach food-producing areas: in the Netherlands south of the Rhine, the Allied armies were in occupation and the east and the north had better access to food. The famine was the result of an embargo on the transport of food supplies imposed by the German occupying forces in early October 1944 in reprisal for a wave of partisan activity. Before 1944, the population of the West Netherlands had a daily ration of 1800 calories, but thereafter official rations, which by the end of the famine consisted almost exclusively of bread and potatoes, declined rapidly, dropping below 1000 kcal/day by January 1945 and reaching as low as 500 kcal/day by April 1945. Although pregnant women were allocated some additional food rations over those available to non-pregnant women, the extent to which the redistribution of these additional rations occurred within families is not known. The famine ceased immediately with the liberation in May 1945, when Allied food supplies became widely available (Stein *et al.*, 1995).

In this study (Stein & Susser, 1975; Stein *et al.*, 1995) fertility is defined as the demonstrated capacity to reproduce and is described by the age-specific birth rates of women of childbearing age. Fecundity is defined as a predisposition or latent capacity for reproducing, a precondition for fertility.

Births during 1944–6 were grouped into cohorts by the stage of gestation at exposure to famine as follows (see Fig. 4.1):

- Cohorts A1 (births between January and July 1944) and A2 (births between August and October 1944) were conceived and born before the famine.
- Cohorts B1 (births between November 1944 and January 1945) and B2 (births between February and April 1945) were conceived before the famine and born during the famine; B1 was exposed for the third trimester of gestation, and B2 was exposed for the second trimester as well as the third.
- Cohort C (births in May or June 1945), conceived before and born after the famine, was exposed during the middle 6 months of gestation.
- Cohorts D1 (births between July and September 1945) and D2 (births between October 1945 and January 1946) were conceived during the famine; D1 was exposed during the first and second trimesters of gestation, D2 was exposed for only the first.
- Finally, cohorts E1 (births between February and May 1946) and E2 (births between June and December 1946) were never exposed to famine.

The famine brought an immediate increase in mortality. The cohort exposed during the last trimester of pregnancy and then after birth (see Fig. 4.1) were the most vulnerable, with higher levels of neonatal mortality. Conversely, the chil-

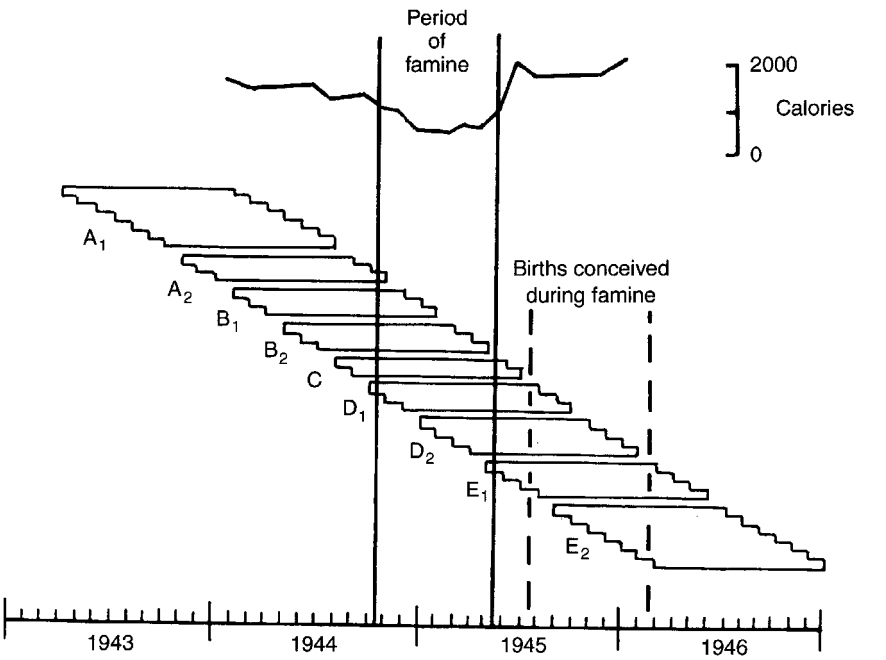


**Fig. 4.1** Diagrammatic representation of the pregnancies in the different cohorts during the Dutch famine winter and the mean daily caloric intake (right hand ordinate). The period of the famine and births conceived during the famine period are indicated. See text for an explanation of the different cohorts. From Stein and Susser (1975).

dren of the famine who survived infancy did not appear to be at special risk and it was found that they had better survival during the first two decades of life.

Fertility ran parallel with food rations through most of the period 1944–5 (Fig. 4.2A). In the famine area, nine months after the onset of acute starvation, a distinct fall in the number of births began; this did not return to prefamine levels until the end of the famine. There was a small decline in the number of children conceived in November and December 1944, a moderate decline in those conceived in January 1945 and a marked decline in those conceived from February to April 1945. In May 1945, with the relief of the famine midway through the month, the number of births conceived at that time rose at once, and by June the number was at the prefamine level.

Although the fall in the number of births coincided with conceptions at the onset of famine, a decline in the official ration to below 1500 calories per day had begun about two months before. This asynchrony of onset suggests that it took some time to exhaust nutritional reserves that maintained the fertility of couples. By contrast, recovery of fertility was immediate and sensitive to an increase of rations, even at very low caloric levels, which indicates that with the provision of nutrients the preconditions of fertility, that is sexual activity and fecundity, were met at once.

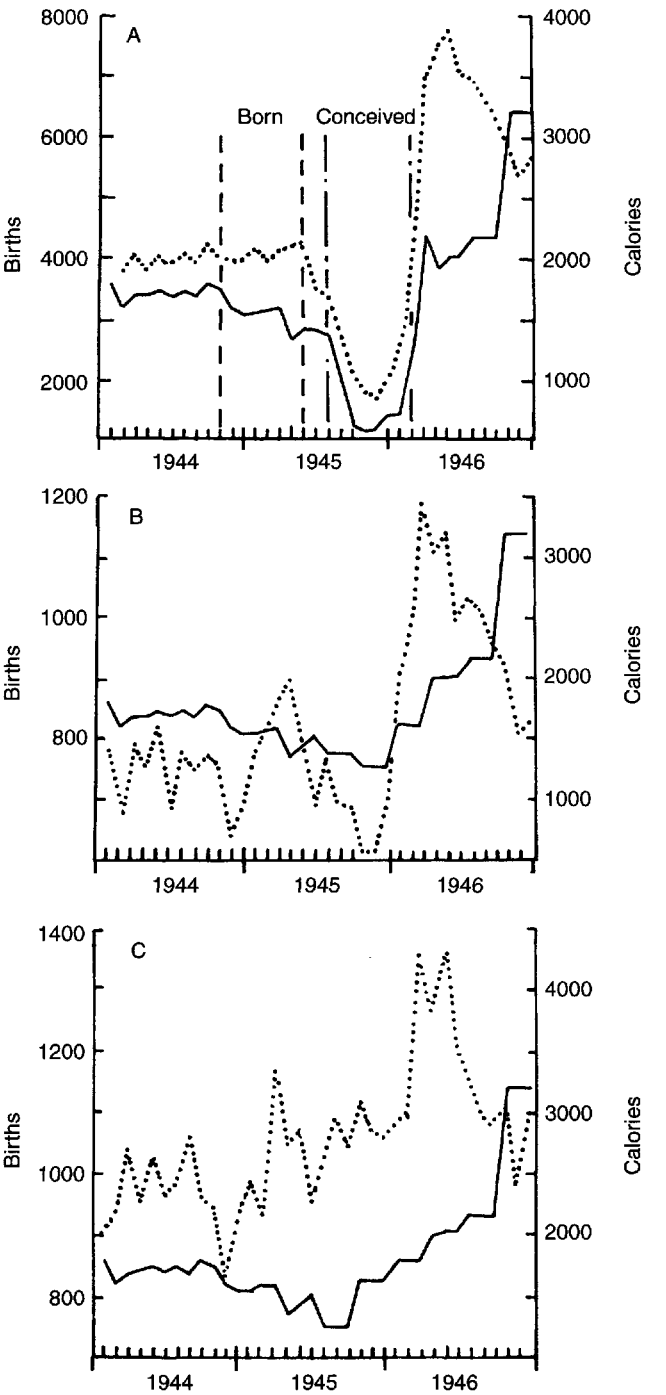


**Fig. 4.2** The relationship between fertility and caloric ration during the Dutch hunger winter. Number of births (left hand ordinate; dotted line) and official mean daily caloric ration at estimated time of conception (right hand ordinate; continuous line). A: Famine cities; B: northern (control) cities; C: southern control cities. Births during the famine and births that were conceived during the famine are indicated. From Stein and Susser (1975).

In the control northern cities (Fig. 4.2B), numbers of births each month exhibited the same sensitivity to local rations when they fell below about 1500 calories per day. Again there was asynchrony, with a lag of two months between the onset of a decline in rations and births, and immediate recovery as rations increased. In the control southern cities, (Fig. 4.2C), numbers of births did not exhibit the same sensitivity to the level of food rations. It is suggested that this was because in these smaller cities, many of which were already freed from the occupation, additional food supplies were available from the surrounding countryside and the liberating armies.

There was a threshold value for the nutritional effect: no definite effect was apparent above the caloric threshold, whereas, below the threshold, the estimated number of conceptions resulting in births correlated with average official daily rations ($r = 0.92$). The reduction in fertility was greater among the manual than among the non-manual occupational classes, and this difference altered the social class composition of the affected birth cohorts.

Babies who were exposed to the famine during the first half of gestation and who were born after the famine was lifted, had normal birthweight. Those exposed during the second half of gestation had lower birth weight, being 327 g lighter than babies born before the famine. Stein *et al.* (1995) conclude that acute maternal nutritional deprivation affects foetal growth only below a threshold and that, conversely, even after a famine, offspring birth size does not respond in a linear fashion to *ad libitum* maternal feeding. The Dutch famine has been the subject of many studies which have yielded a series of interesting results. For example, men who were conceived during the famine had higher rates of obesity at age 19 years (Ravelli *et al.*, 1999) and the offspring who were exposed to maternal malnutrition during early gestation suffered from raised levels of coronary heart disease in later life (Roseboom *et al.*, 2000).

In conclusion, Stein *et al.* (1975) have identified a daily ration of 1500 calories as the threshold that sustained fecundity in the West Netherlands; furthermore the nutritional threshold for conception was clearly lower than the nutritional threshold for foetal viability. It was concluded that famine exerts its greatest effect just before, or just after birth, rather than early in the prenatal period.

## 4.6 The siege of Leningrad, 1941–4

The recent release of archives by the Russian government has allowed an analysis of the events during the siege of Leningrad. Some 800 000 people died and it became the largest-scale famine ever seen in the industrial world. An account of this famine (*Wellcome News*, Issue 26, 2001) suggests that 50 000 were killed by direct military action and that 750 000 died from hunger or related diseases. A welfare state had been created in the 1930s as part of Stalin's modernisation drive and Leningrad had an excellent health system on the eve of World War II. The

city had some 20 medical institutes and a sophisticated civic and public health infrastructure which enables detailed pictures of the events of 1941–4 to be uncovered. Remarkably, the registry office kept reliable statistics throughout the siege, apart from the months between December 1941 and April 1942 when starvation was at its height. During this time, records become more patchy; the statisticians and officials were dying and relatives were too ill to be able to report deaths.

The authorities were worried about cannibalism, not only the eating of those who starved or died of natural causes, but also murder for the consumption of human meat. Over 300 people were shot for committing this crime, and the statistics show that by far the largest numbers of those caught for this offence were ration-less refugees. More than a thousand others were imprisoned.

Medical records show that the population exhibited severe symptoms of stress: a massive increase in high blood pressure and many women stopped menstruating.

Rations reached their lowest in November 1941. They consisted of 700 calories a day for manual workers; non-manual workers and dependants were allowed 473 calories a day; children received 423 calories a day, less than a quarter of an infant's normal requirement. These values can be compared with the allowances in the Dutch hunger winter (see section 4.5). Rations were increased significantly in March 1942, in part because there were fewer people to feed, and in part because some supplies began to come in via the highway across the frozen Lake Ladoga.

The Russians coined the term 'alimentary dystrophy' to describe the effects of starvation. The symptoms were progressive loss of body weight and muscular strength, weakening of the heart, exhaustion and lethargy. The first deaths from starvation came in mid-December 1941, and these peaked in March 1942 but continued until well into the summer of 1942.

The most common actual cause of death was failure of the heart or other organs. One of the remarkable aspects of the siege is that there were no epidemics of disease. There was no running water, sanitation, light or heating. At one point 20 000 unburied bodies were piled in the Piskariovskoye cemetery alone; 3500 people were dying every day. Yet while pneumonia and tuberculosis did increase, the incidence of typhoid and dysentery was remarkably low. This was probably partly because of the low winter temperatures which reached $-40°C$ – the corpses were frozen and contagion was prevented to some extent.

A remarkable number of people survived what seemed to be insufferable deprivations; people's bodies gradually adapted by reducing basic functions, limiting the expenditure of energy to adopt a sort of 'hibernating' state.

## 4.7 Why do women survive famine better than men?

It is generally believed that women survive famine better than men and K. MacIntyre (unpublished report of a conference at Les Treilles, 1999) has

addressed the problem of why this should be so. She outlines six possible mechanisms that could explain this mortality advantage, using the Great Irish famine (see section 4.1) as a case study:

(1) A spurious reading of poor data, which exacerbates the problem of 'missing' women so that their deaths are less likely to be reported than deaths of men.
(2) A concurrent decline in fertility which lowers the risk of pregnancy and, hence, maternal mortality.
(3) A biological argument that says women are less likely to die from starvation because of higher body fat levels, lower energy consumption or possibly higher resistance to disease.
(4) Differential individual and group survival strategies, in particular in relation to migration and to the acquisition of knowledge of famine foods.
(5) The particular political and social structures which may discriminate against women in 'normal' times, but act in their favour in times of great stress.
(6) Emotional or moral factors associated with differential notions of pride, which may mean that women reach places of assistance (the workhouse or the refugee camp, for instance) in a better state than men.

MacIntyre suggests from a study of the Irish Famine that option (6) is the most probable explanation.

## 4.8 Famines in pre-industrial England

Laslett (1965) first posed the question 'Did the peasants really starve?', highlighting the point that pre-industrial England did not suffer famines on the scale listed above, although a number of mortality crises have been reported in different areas. Laslett (1965) concluded that 'the relation between the amount and cost of food and the variations in the level of mortality, of men and women as well as children, must remain an open question for the time, along with that of whether crises of subsistence were a present possibility in the English town and countryside'. Twenty years later, Watkins and Menken (1985) came to a similar conclusion: 'On the basis of the historical record, it would seem that famines in which death rates doubled for two years were rare, even for small populations, and that famines of greater intensity were highly unusual if they occurred at all'. Wrightson and Levine (1979), in their study of the village of Terling, Essex, UK, also conclude that famine seems to have been of no demographic significance:

> 'While it is remarked by the scribe in the 1590s that poor, wayfaring strangers were being found dead in the yeomen's barns, the overall effect of the harvest disasters of these years is muted. Other "famous" dearths likewise seen to have little effect on the life chances of the villagers. To be sure, there was depri-

vation and a crying need in such years, but we have no evidence that there was death by starvation among the settled population of the village'.

These doubts concerning the reality of serious famines in pre-industrial England fired Appleby (1973, 1978) to explore the parish registers of Cumbria in northwest England, searching for evidence of mortality crises in specific areas where the inhabitants were living under marginal conditions. He identified the year 1623, when several parishes in that area showed mortality levels substantially above normal. In the parish of Greystoke, 161 people were buried, over four times the expected number; only 20 babies were baptised, half the average number, and there were only six marriages. The registers of Greystoke explicitly give starvation as the cause of some of the deaths (Appleby, 1973). The following are extracts from the parish burial registers of 1623:

29 January: 'A poore fellowe destitute of succour and was brought out of the street in Johnby into the house of Anthony Clemmetson, constable there, where he died.'
27 March: 'A poore hungersterven beger child – Dorethy, d. of Henry Patteson, Miller.'
28 March: 'Thomas Simpson a poore, hungerstarved begger boye.'
19 May: 'At night, James Irwin, a poore begar striplinge borne upon the borders of Ingland. He died at Johnbye in great miserie.'
21 May: 'A poore man destitute of meanies to live.'
20 June: 'Willm Robinson – single man – borne in Matterdaill a poore fellowe destitute of all meanes to live.'
12 July: 'Thomas, c. of Richard Bell a pore man wch child dyed for verie want of food and maintenance to live.'
18 August: 'Willm Bristoe a poore man destitute of any meanes to live.'
2 September: 'John Tompson a poore labouringe man of Penruddock having no means at all to live by.'
11 September: 'Leonard, s. of Anthony Cowlman, of Johnby, late deceased, wch child died for want of food.'
12 September: 'Jaine, wife of Anthony Cowlman, late deceased, which woman died in Edward Dawson's barn of Greystoke for want of maintenance.'
27 September: 'John, son of John Lancaster, late of Greystoke, a waller by trade, wch childe [4 years old] dyed for want of foode and meanes.'
4 October: 'Agnes w. of John Lancaster, late of G. a waller by his trade wch woman dyed for want of means to live.'

There were many other burial entries of persons 'destitute' and 'poor' or 'without the means to live'. These examples do not exhaust the cases of starvation and the registers reflect a long year of misery for the inhabitants of the parish. Some, but by no means all, of those dying were from outside the parish. The roads must

have thronged with unfortunates, many of them children, seeking a bit of food. The registers show that a large number of the dead were children, widows, and the elderly – those least able to sustain themselves in times of shortage (Appleby, 1973).

Armstrong (1994) has provided an account of the parish of Greystoke during the years 1560 to 1630, so including the 1623 famine. He points out that the parish is huge, covering 65 square miles. Armstrong found good evidence for teenage subfecundity or adolescent sterility, probably indicating that the general level of nutrition was rather poor. He also found that age-specific marital fertility was low and the rate of stillbirths (which, exceptionally, were recorded in the registers) was high at 9%.

Most unfortunately, the parish registers are missing for the critical period from December 1620 to June 1622, immediately before the famine. We have determined the infant mortality at Greystoke from 1615 to 1627 and the results are presented in Table 4.4. Mean total annual burials were 35, both before and after the famine. During the famine, this value rose to 164, an increase of 4.7-fold. Infant mortality has been subdivided into stillbirths, neonates, post-neonates and those not baptised, and then each has been expressed as a percentage of the baptisms in that year. It is evident that the percentage infant mortality rose sharply in 1623, the year of the famine (Table 4.4, column 6): births fell to 23 in that year (as would be expected) and, of these, 43% died.

Following his analysis of events in Cumbria in 1623, Appleby (1973) developed his thesis of two Englands, one subject to trade depression and harvest failure but able to avoid widespread starvation, whereas the other was pushed to the edge of subsistence by the same dislocations. He suggested that the economy of the northern territory was more like that of Scotland, Ireland and parts of continental Europe than lowland England.

When the data for the 404 parishes were published (Wrigley & Schofield, 1981), the results confirmed Appleby's suggestions: subsistence crises were largely absent from the south-east and, while the north was vulnerable, this ceased to be the case after the mid-seventeenth century. However, in the time before 1650 the situation was more complex than a simple north–south dichotomy. Not only did some northern parishes escape mortality crises in years of dearth, but there were also clusters of communities that were affected in an otherwise immune south (Walter & Schofield, 1989). This apparent paradox is explored in more detail below (section 4.10).

Walter and Schofield (1989) surveyed the geographical distribution of local crises that occurred in England from October 1596 to June 1598 and from November 1622 to December 1923. They concluded that during the two worst crisis years of the late sixteenth century (1596/7 and 1597/8) mortality rates were 21% and 26% above the trend. Famine-related mortality crises persisted into the seventeenth century, but famine became almost exclusively a problem of the northern uplands.

**Table 4.4** Analysis of mortality in the first year of life at Greystoke, Cumbria, UK, 1615–27.

| Year | No of baptisms* (1) | No of stillborns (2) | % | No of neonates (3) | % | No of post-neonates (4) | % | No of infants buried but not baptised (5) | % Infant mortality (6) | No of total burials (7) |
|---|---|---|---|---|---|---|---|---|---|---|
| 1615 | 45 | 2 | 4 | 1 | 2 | 4 | 9 | | 16 | 29 |
| 1616 | 42 | 2 | 5 | 3 | 7 | 3 | 7 | | 19 | 32 |
| 1617 | 49 | 2 | 4 | 2 | 4 | 1 | 2 | | 10 | 35 |
| 1618 | 40 | | 0 | 3 | 8 | 5 | 13 | | 20 | 37 |
| 1619 | 40 | | 0 | 5 | 13 | 6 | 15 | | 28 | 43 |
| 1620 | 54+ | 1+ | 2+ | 3+ | 6 | 5+ | 9+ | 2+ | 20+ | 33+ |
| 1621 | | | | | | | | | | |
| 1622 | 16+ | | | | | | | 1+ | | 24+ |
| 1623 | 23 | 3 | 13 | 2 | 9 | 5 | 22 | | 43 | 164 |
| 1624 | 24 | 2 | 8 | 1 | 4 | 3 | 13 | 1 | 29 | 46 |
| 1625 | 41 | 1 | 2 | 5 | 12 | 3 | 7 | 1 | 24 | 41 |
| 1626 | 45 | 0 | 0 | 2 | 4 | 4 | 9 | | 13 | 35 |
| 1627 | 30 | 2 | 7 | 6 | 20 | 0 | 0 | | 27 | 28 |

Data from Greystoke parish registers. Note: deficiencies in the registers 1620–22.
* Includes stillborns plus infants buried but not baptised.

The conclusion that southern England was subject to harvest failure but was able to avoid widespread starvation (Appleby, 1973; Walter & Schofield, 1989) is confirmed by an analysis of events at Ludlow in Shropshire, where there is believed to be evidence of famine. High infant mortality is regarded as an indicator of a famine crisis (Schofield & Wrigley, 1979; Watkins & Menken, 1985). The annual burial totals for Ludlow for the period 1577–1608 are shown in Table 4.5. Mean annual burials were 78.5 and the famine years of 1587 and 1597 stand out as having a high total mortality. Burials more than doubled in 1587 and increased 1.7-fold in 1597. Burials also nearly doubled in the last famine year of 1623. This suggests that Ludlow, with a larger population size, suffered from the effects of famine, but the

**Table 4.5** Annual burials in Ludlow by age, 1577–1608.

| | Total burials (i) | Infant burials (ii) | Baptisms (iii) | % Infant mortality (iv) | Child burials (v) | Adult burials (vi) |
|---|---|---|---|---|---|---|
| 1577 | 50 | 12 | 68 | 18 | 7 | 31 |
| 1578 | 58 | 14 | 81 | 17 | 16 | 28 |
| 1579 | 55 | 14 | 102 | 14 | 11 | 30 |
| 1580 | 92 | 20 | 72 | 28 | 23 | 49 |
| 1581 | 53 | 10 | 81 | 12 | 17 | 26 |
| 1582 | 75 | 27 | 89 | 30 | 19 | 29 |
| 1583 | 59 | 21 | 78 | 27 | 10 | 28 |
| 1584 | 82 | 24 | 84 | 29 | 17 | 41 |
| 1585 | 84 | 22 | 85 | 26 | 18 | 44 |
| 1586 | 94 | 21 | 82 | 26 | 18 | 55 |
| 1587 | 191 | 13 | 55 | 24 | 27 | 151 |
| 1588 | 97 | 12 | 72 | 17 | 8 | 77 |
| 1589 | 76 | 29 | 92 | 32 | 13 | 34 |
| 1590 | 53 | 12 | 66 | 18 | 12 | 29 |
| 1591 | 95 | 12 | 69 | 17 | 14 | 69 |
| 1592 | 78 | 16 | 85 | 19 | 12 | 50 |
| 1593 | 88 | 22 | 68 | 32 | 9 | 57 |
| 1594 | 67 | 14 | 68 | 21 | 16 | 37 |
| 1595 | 109 | 22 | 76 | 29 | 41 | 46 |
| 1596 | 79 | 19 | 68 | 28 | 16 | 44 |
| 1597 | 133 | 19 | 59 | 32 | 36 | 78 |
| 1598 | 54 | 8 | 65 | 12 | 11 | 35 |
| 1599 | 69 | 19 | 91 | 21 | 14 | 36 |
| 1600 | 64 | 20 | 75 | 27 | 12 | 32 |
| 1601 | 79 | 16 | 73 | 22 | 20 | 43 |
| 1602 | 69 | 12 | 65 | 18 | 15 | 42 |
| 1603 | 62 | 18 | 81 | 22 | 11 | 33 |
| 1604 | 75 | 12 | 71 | 17 | 12 | 51 |
| 1605 | 54 | 12 | 80 | 15 | 6 | 36 |
| 1606 | 70 | 12 | 78 | 15 | 16 | 42 |
| 1607 | 86 | 20 | 86 | 23 | 33 | 33 |
| 1608 | 63 | 18 | 72 | 25 | 12 | 33 |

*Source:* Schofield & Wrigley (1979).

percentage mortality was less severe than at Greystoke or Penrith (see section 4.9). Both Schofield and Wrigley (1979) and Watkins and Menken (1985) draw attention to the low levels of infant mortality in 1587 and 1597 (Table 4.5, column (ii)), but if infant burials are expressed as a percentage of baptisms (Table 4.5, column (iv)), it can be seen that they were about average (or increased in 1597) during the crises. Nevertheless, these infant mortalities were clearly lower than at Penrith in 1623 when 50% of the infants baptised died (Table 4.6). We conclude that there are qualitative as well as quantitative differences between southern/central parishes and those living under marginal conditions in Cumbria. Infant mortality is strongly dependent on the status of the mother's health before conception and during pregnancy (see Chapter 9) and, in the non-crisis years, was at broadly comparable levels in Ludlow and Penrith. The difference, as Appleby (1973) pointed out, was that although both areas were subject to harvest failure, Ludlow was an example of a community able to avoid widespread starvation, whereas the population at Penrith was pushed to the edge of subsistence.

**Table 4.6** Age-specific mortality for children baptised at Penrith during the period 1615–32.

| Year | No of Baptisms | Age-specific mortality (% of baptisms) classified by year of baptism | | | | | Wheat prices per qtr (s) | Total deaths |
|---|---|---|---|---|---|---|---|---|
| | | Infant | 1–2 y | 2–5 y | 6–14 y | Total 1–14 y | | |
| (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
| 1615 | 52 | 21.2 | 11.5 | 7.7 | 3.8 | 23.0 | 39 | 35 |
| 1616 | 41 | 22.0 | 4.9 | 2.4 | 7.3 | 14.6 | 40 | 57 |
| 1617 | 62 | 21.0 | 0 | 8.1 | 4.8 | 12.9 | 49 | 57 |
| 1618 | 48 | 22.9 | 4.2 | 14.6 | 0 | 18.8 | 47 | 46 |
| 1619 | 51 | 17.6 | 3.9 | 9.8 | 3.9 | 17.6 | 35 | 40 |
| 1620 | 42 | 21.4 | 2.4 | 11.9 | 7.1 | 21.4 | 30 | 43 |
| 1621 | 54 | 22.2 | 1.9 | 3.7 | 1.9 | 7.5 | 30 | 50 |
| 1622 | 48 | 18.8 | 16.7 | 4.2 | 6.3 | 27.2 | 59 | 49 |
| 1623 | 40 | 50.0 | 2.5 | 0 | 0 | 2.5 | 52 | 241 |
| 1624 | 40 | 20.0 | 2.5 | 5.0 | 2.5 | 10.0 | 48 | 39 |
| 1625 | 53 | 20.8 | 1.9 | 7.5 | 3.8 | 13.2 | 52 | 34 |
| 1626 | 55 | 23.6 | 3.6 | 9.1 | 3.6 | 16.3 | 49 | 43 |
| 1627 | 68 | 10.3 | 5.9 | 1.5 | 1.5 | 8.9 | 36 | 20 |
| 1628 | 56 | 16.1 | 12.5 | 1.8 | 8.9 | 23.2 | 28 | 43 |
| 1629 | 49 | 20.4 | 4.1 | 4.1 | 2.0 | 10.2 | 42 | 43 |
| 1630 | 50 | 18.0 | 2.0 | 4.0 | 2.0 | 8.0 | 56 | 37 |
| 1631 | 54 | 25.9 | 5.6 | 5.6 | 1.9 | 13.1 | 68 | 41 |
| 1632 | 55 | 34.5 | 0 | 12.7 | 5.4 | 18.1 | 53 | 80 |
| Mean | 51 | | | | | | 45.2 | 55.4 |
| SD | 7.42 | | | | | | 10.96 | 47.9 |

The fate of the children (age at death or surviving) born in each year can be traced along the respective row. Qtr, quarter; s, shillings; SD, standard deviation.

## 4.9 Famine at Penrith, Cumbria, 1623: a case study

Following the identification of 1623 as a year of famine in northwest England (Appleby, 1973) we have studied infant mortality at Penrith, a market town close to Greystoke in the Eden Valley, Cumbria during the years 1615 to 1632. The data were extracted from the published records of the parish (Haswell, 1938) and a family reconstitution over the period 1557–1812 was carried out (Duncan *et al.*, 1992; Scott, 1995; Scott & Duncan, 1997a).

As Appleby (1973, 1978) has pointed out, the conditions at Penrith, close to the Scottish borders, during the seventeenth and eighteenth centuries were severe; much of the northern territory was remote from large trading centres and, seen (unjustly) through the eyes of southerners, was a wild and savage country, its inhabitants without understanding of law-abiding behaviour and harbouring a characteristic frontier society (Thirsk, 1967). The main hazard was the meagre corn harvest, the crops being bigg (a poor variety of barley) and oats. Even as late as 1790, a Cumberland correspondent wrote 'At the beginning of this century, the inhabitants were in a state bordering on extreme indigence and ignorance ... they lived barely on the product of their little farms' (Bouch & Jones, 1961). In addition, the landlords gradually increased the entry fines demanded of their tenants during the sixteenth century, and 'the miserable tenant, who is going to pay an arbitrary fine and a heriot, is perpetually impoverished ... those customary tenures are a national grievance. From this tenure is chiefly to be attributed the vast and dreary wastes that are found in Cumberland' (Hutchinson, 1794). Conditions had not improved by 1800:

> 'There are probably few counties, where property in land is divided into such small parcels as in Cumberland; and those small properties so universally occupied by the owner; by far the greatest part of which are held under the lords of the manor, by a species of vassalage, called customary tenure; subject to the payment of fines and heriots, on alienation, death of the lord or death of tenant' (Bailey & Culley, 1794).

The mean annual number of deaths at Penrith in the seventeenth century was 60 and this rose to 241 in 1623; this 4-fold increase is indicative of a major mortality crisis associated with the famine in the area in this year. Infant mortality also rose sharply and we have followed the fate of the children born in each year at Penrith during the period of 1615–32. The results are summarised in Table 4.6, which shows the age-specific mortality of infants and children classified by the year of birth, so that it is possible to trace the fates of the children born in each year by following along the respective lines. For example, 21.2% of the children born in 1615 died as infants and 11.5% died as 1 year olds in 1616. Some 7.7% died between 2 and 5 years of age between 1617 and 1620 (see Table 4.6).

There were 48 births in 1622 (Table 4.6, column 2), close to the mean annual

value of 51 (derived from family reconstitution studies for that period). Although wheat prices (column 8) rose sharply in that year and were high for 1622–3, infant mortality (18.8%, column 3) remained low. Infant mortality in the following year rose dramatically, however, and 50% of those born died in the first year of life, but none of those who survived the first year of life died in the parish up to age 14 years. This was also a feature of the Dutch winter famine (see section 4.5). The results for 1623 illustrate the deleterious effects of high wheat prices during pregnancy in 1622, which subsequently caused severe infant mortality. High wheat prices also had a *direct* effect on the mortality of very young children: 16.7% of the children born in 1622 died in their second year of life (Table 4.6, column 4), i.e. during the famine of 1623.

Wheat prices were again very high (68 shillings per quarter) in 1631 and total deaths rose to 80 in the following year; some 34.5% of those born in 1632 died in infancy (Table 4.6, column 3). Conversely, 1627–8 was a period of low wheat prices: total mortality was very low, whereas births and marriages in the parish were at a peak. Infant mortality (expressed as a percentage of baptisms) was particularly low in 1627 and 1628 (Table 4.6, column 3).

Although poor nutrition during pregnancy was apparently associated with a noticeable increase in infant mortality, columns 4–6 and 7 in Table 4.6 show that the mortality of the surviving children had little correlation with the annual wheat price index in either the year of their birth or the year preceding their birth. We conclude that Table 4.6 does not provide evidence that famine conditions during pregnancy have indirect effects on the subsequent mortality of the *surviving* children over the age of one year. However, wheat prices in later years do have a *direct* effect on childhood mortality. Thus, the high mortality for children at age one year who were born in 1622 (Table 4.6, column 4) reflects the high total of 1 year olds who died in the disastrous following year of 1623. The higher values of mortality in the age groups 2–5 years (Table 4.6, column 5) for the children born in the years 1618–20 illustrate how these children were directly affected by the famine conditions of 1622–3 and died then.

The possible effects of malnutrition on infant mortality has been assessed further, and the records for Penrith for the period 1621–4 have been divided into 12 overlapping groups, each containing the total conceptions for three consecutive months, and the number of subsequent neonatal and post-neonatal infant deaths for each group are shown in Fig. 4.3. For example, group C included 15 children who were conceived during the period December 1621 to February 1622 (i.e. prefamine conceptions who were born before there was a noticeable rise in mortality); of these, one child died as a neonate and four as post-neonatal infants. Groups D, E and F children were conceived before the famine, but were exposed to malnutrition and famine conditions during the second and third trimesters of pregnancy. Group G was exposed to famine *in utero* and after birth, and groups H, I and J were exposed to famine conditions *in utero* but not after birth. Control groups A, B, C, K and L were either prefamine conceptions and

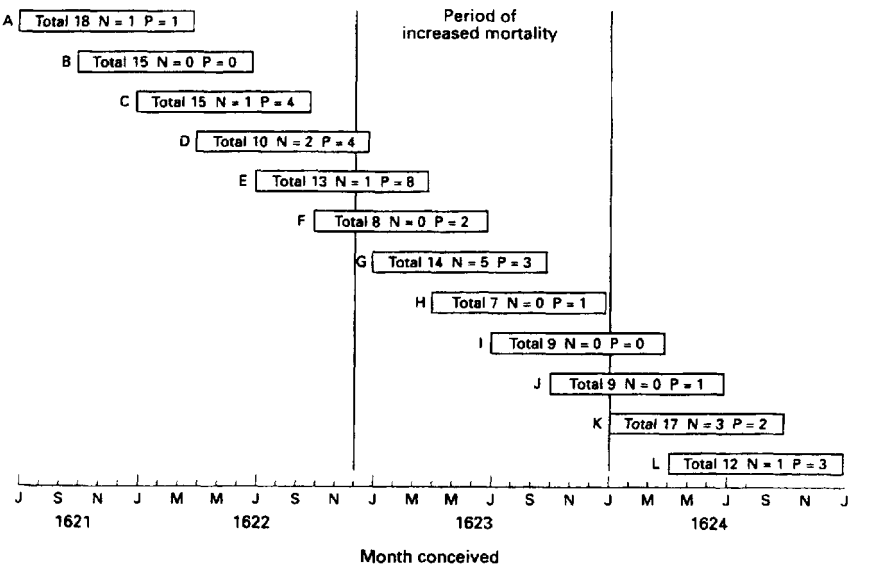


**Fig. 4.3** Infant mortality at Penrith in 1621 and 1624, illustrating the effects of the famine of 1623. N, neonatal deaths; P, post-neonatal deaths. The births are divided into month cohorts, reflecting whether conception and pregnancy were before, during or after the period of famine. The cohorts are separated by 3-monthly intervals. Thus, groups A, B, C, K and L are control groups; D, E and F were infants conceived before, but born during the famine; G infants were conceived and born during the famine; H, I and J were conceived during, but born after the famine. The results of the $\chi^2$ test are tabulated as follows:

| Groups | Survivors | Neonate deaths | Post-neonate deaths | Total |
|---|---|---|---|---|
| ABCKL | 61 | 6 | 10 | 77 |
| DEF | 14 | 3 | 14 | 31 |
| G | 6 | 5 | 3 | 14 |
| HIJ | 23 | 0 | 2 | 25 |
| Total | 104 | 14 | 29 | 147 |

$\chi^2 = 32.4$; 6 df; $P < 0.001$

births, or post-famine conceptions and births. It is evident that the number of baptisms fell considerably during the period of famine. The number in each group is necessarily small, but the results provide a good indication of the effects of severe malnutrition during pregnancy.

Overall, a highly significant departure from random expectation was established ($P < 0.001$) and three main tendencies become apparent (Fig. 4.3). First, groups D, E and F, conceived before the famine but born when food shortages were acute, displayed a large rise in post-neonatal burials whereas neonatal mortality was not raised. Group E infants (exposed during the last trimester of pregnancy and then after birth) were the most vulnerable, with the highest level of post-neonatal mortality. Group D also had a high level of infant mor-

tality with twice as many post-neonates dying as neonates. These foetuses were exposed during the last trimester to conditions that were probably less severe than those experienced by the later group. Second, equally significant, are the increased mortality rates for neonatal infants conceived and born during the famine (group G); this group included five neonates who died as unbaptised infants. Third, conversely, the children of the famine (groups H, I and J) showed remarkably low infant mortality and, although the numbers were small, there were no neonatal deaths and only two post-neonatal deaths out of a total of 25.

We conclude that these studies of the population at Penrith, which lived under very difficult conditions and hence was susceptible to fluctuations in the quality and quantity of the food supply, provide supporting historical evidence for the hypothesis that infant mortality is particularly sensitive to poor nutrition of the mother during pregnancy. Table 4.6 and Fig. 4.3 provide tentative evidence that neonatal mortality is related to such endogenous causes as malnutrition during pregnancy (there is a lag of +1 years with the wheat price index), whereas post-neonatal mortality (zero lag with wheat prices) is directly dependent on exogenous causes such as the food supply during the first year of life, which affected both lactation and weaning. Since famine conditions would frequently overlap pregnancy and infancy, however, a clear separation of the lag period for the two components of infant mortality would not be expected.

The famine at Penrith in 1623 can be compared with the Dutch hunger winter. The highest neonatal mortality was recorded in the cohort conceived and born during the famine, whereas the highest post-neonatal mortality was found in cohorts conceived before and born during the famine, particularly those that were exposed to the severest effects of the famine during the last trimester.

These conclusions concerning the effects of malnutrition during pregnancy and infancy at Penrith in the early seventeenth century are, of necessity, based on indirect evidence, but they fully support the classic studies in historical epidemiology of Hertfordshire in the early twentieth century by Barker (1998), who has shown that nutrition in pregnancy has a profound effect on neonatal mortality (see Chapter 7), even though this population was not existing at subsistence level (and at times on the edge of starvation), as at Penrith.

## 4.10 Interacting economic factors causing famines in northwest England

As we have seen, Cumberland and Westmorland suffered three mortality crises at the end of the sixteenth century and the start of the seventeenth century, in 1587/8, 1596/7 and 1623. Events during the 1623 crisis at Penrith and Greystoke have been described in sections 4.6 and 4.7. Although Appleby (1973) concluded that the mortality crisis of 1587/8 was caused by typhus, there is no evidence for

this view and we suggest that famine and hardship were responsible for the clear rises in the number of burials in all three years in Cumbria. There are two unanswered questions. First, although annual national wheat prices were at a peak of the short wavelength oscillation in 1623, they were not markedly higher than in the preceding or following peaks, raising the question why the crisis occurred in that year. Second, why were these mortality crises largely confined to the northwest when the remainder of England was subject to the same national grain prices? These questions are addressed below and the role of the prices of other commodities, particularly that of wool, is examined in a range of communities in the northern territory.

The main object of sheep farming in Tudor and Stuart times was the production of wool, the quality of which varied considerably in different parts of the country. Cumberland and Westmorland had always produced short and very coarse wool and in 1638 the northern wools were regarded as being among the worst grown in England; these short-staple wools were manufactured into only the most inferior types of woollen cloth (Bowden, 1971).

Among livestock farmers, the adverse effects of a harvest failure were felt with particular severity by the wool growers, because clothing was the principal consumer good on which income might be saved in times of food scarcity. Thirsk (1967) compares the statistics relating to cloth exports, wool prices and English and continental grain prices during the sixteenth and seventeenth centuries and concludes that good harvests were fundamental to the prosperity of the wool industry both at home and abroad, leading to a buoyant demand for wool. Thus, the sheep-farming industry was sensitive to the level of grain prices because the income elasticity of demand for clothing was high: a long run of good harvests meant a rising demand for clothing and the need for more wool, so that cloth exports increased, wool prices rose and the sheep-farming industry expanded (Thirsk, 1967).

The unfiltered series of the wool price index for the critical period 1557–1643 is shown (together with the wheat price index) in Fig. 4.4 and two features are detectable by eye. First, there is a strongly-rising trend in wool prices over this period which runs in parallel with the corresponding rise in wheat prices. Second, there is a clear medium wavelength oscillation in wool prices, and spectral analysis shows that its period is 12 years ($P < 0.005$).

The mortality crises suffered in northwestern England in 1586/7, 1596/7 and 1623 are indicated on Fig. 4.4, a study of which lends support to the thesis advanced by Thirsk (1967) (see above), that in years when high wheat prices synchronised with low wool prices, as in the crisis years indicated, economic conditions were hard and mortality was high. With a 5 to 6-year oscillation in wheat prices and a 12-year oscillation in wool prices, there is potential for a synergistic interaction leading to a mortality crisis every 12 years. This possible interacting effect between the 5 to 6-year oscillation in wheat prices and the 12-year oscillation in wool prices is illustrated in Fig. 4.5, where the two series,

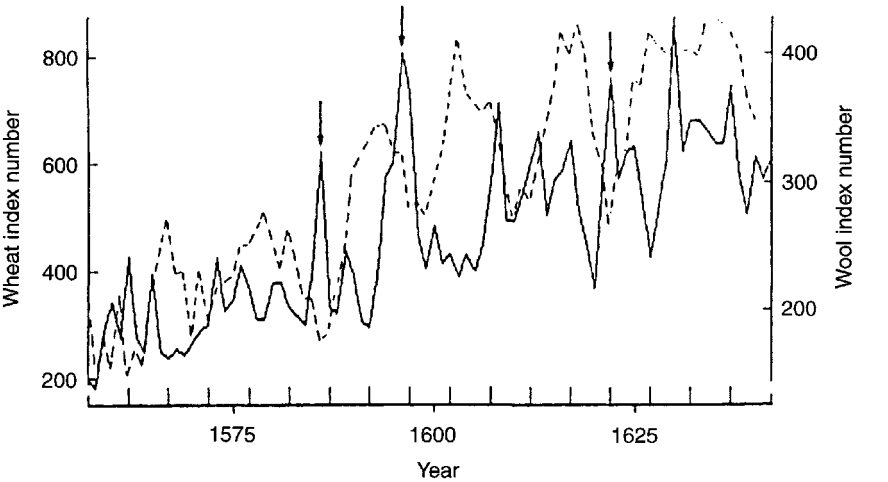


**Fig. 4.4** Comparison of the series of wheat (solid line) and wool (dashed line) price indices, 1557–1643. Arrows indicate the mortality crises in the northwest in 1586/7, 1596/7 and 1623, when high wheat prices coincided with low wool prices. Data sources: Bowden (1967, 1985).

appropriately filtered, are linked together and the years of potential mortality crises (very low wool prices coupled with high wheat prices) are indicated.

These completely opposite effects of wool and wheat prices on the economy at Penrith were further explored by studying the cross-correlations between infant mortality versus these two series separately during the period of the famines in Cumbria, 1587 to 1643. Infant mortality was chosen as the most sensitive demographic indicator of famine conditions and was strongly associated with both *high* wheat prices and *low* wool prices.

This study illustrates how the exogenous short wavelength mortality cycle at Penrith (period 5–6 years) that is driven by wheat prices (see section 10.2) is clearly modulated by wool prices. Thus, the three severe mortality crises experienced at Penrith in 1586/7, 1596/7 and 1623 were not solely because of high wheat prices and famine in these years, but the hardship was exacerbated by sharply falling wool prices. In 1630, when the price of corn rose to an excessive level and there was an extraordinary dearth in England (Hoskins, 1968), the price of wool dipped only slightly (Fig. 4.4) and there was no accompanying rise in mortality in the northwest.

The pattern underlying the economic events in Cumberland and Westmorland at that time is probably more complex. Two (probably independent) cycles in wheat prices can be detected by time-series analysis (see Chapter 3), a short (5 to 6-year) and a medium (11-year) wavelength cycle. When the peaks in these two oscillations in wheat prices coincided, food was scarce with no money to spare for clothing and, consequently, wool prices fell sharply, producing a double blow for communities that were dependent on sheep farming. In these years, one of the

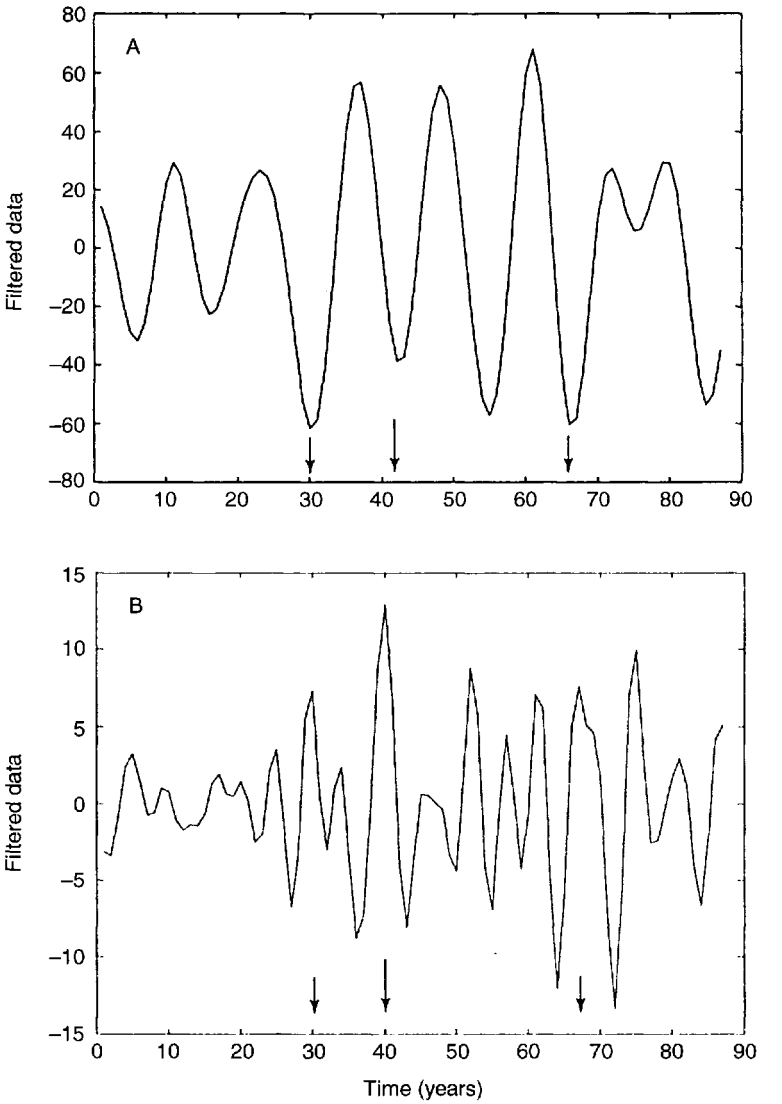


**Fig. 4.5** Filtered series of the wheat and wool price indices, 1557–1643. Arrows indicate the years of potential mortality crises when low wool prices could interact synergistically with high wheat prices, culminating in the actual mortality crises in the northwest 1 or 2 years later. A: Wool index; filter window = 10 to 16 years to show the 12-year oscillation. B: Wheat index; filter window = 4 to 10 years to show characteristic short wavelength oscillation. Abscissae: time (years) after 1557.

severe mortality crises suffered in Cumbria in the late sixteenth and early seventeenth centuries could readily be triggered. We conclude that the three major mortality crises were triggered in northwestern England in those communities where the economy was dependent on rearing sheep and growing grain

in years when wheat prices were particularly high (i.e. when both the short and medium wavelength oscillations in prices were in synchrony) and wool prices were low, the two commodities interacting synergistically.

By the end of the seventeenth century, the Cumberland and Westmorland farmer had moved towards more cattle and fewer sheep, and Marshall (1980), in his analysis of inventories, found that, between 1667 and 1750, cattle were the largest single item of possession and of more value than were the sheep. This is supported by an analysis of mortality at Penrith during later periods, 1650–1750; although high wheat prices still exerted a depressing effect on mortality, wool prices were less important and, instead, high beef prices were now significantly associated with the burials of infants ($P = 0.03$) and adults ($P = 0.04$). Therefore, the synergistic interaction of low wool and high wheat prices no longer affected mortality so dramatically, and famine crises, such as occurred in 1623, were never again experienced in the northwest of England.

## 4.11 The mortality crisis of 1623 in northwestern England

The mortality crisis of 1623 was confined mainly to the north and northwest of England, with only a few scattered outbreaks elsewhere in the country (Wrigley & Schofield, 1981); in this section we present the results of a survey of a range of communities in the northwest. Was the mortality experienced at Penrith typical of neighbouring parishes? An analysis of over 80 parishes in Lancashire showed that burials in 1623 were more than twice the average of the decade (Rogers, 1975) and a study of Stockport, a large Cheshire parish of 14 townships, also revealed a substantial increase (Millward, 1983). The crisis was widespread in Cumberland and Westmorland and was also severe in Scotland, where losses were estimated to be in excess of 10% of the total population of Dumfries and over 20% of Dunfermline, which is considerably more than the 5% calculated for Lancashire (Appleby, 1973; Rogers, 1975; Flinn, 1977). In contrast, only average, or slightly above average, numbers of burials were found for some West Yorkshire and Mid-Wharfedale areas (Drake, 1962; Long & Pickles, 1986).

However, communities in northwest England were not affected with equal severity in 1623. An aggregative analysis of 25 parishes between 1613 and 1624 has been undertaken; they were chosen because they represent a reasonable cross-section: Penrith, Cartmel, Lancaster, Poulton-le-Fylde, Whalley, Brough and Kendal were all market towns (Everitt, 1967) and Newbiggin and Cliburn were small parishes. North Meols is situated on the coast, and Ingleton and Thornton-in-Lonsdale are in the uplands, on the western side of the Pennines. The infant mortality rate has been calculated from the aggregative analysis data as the number of deaths occurring within a year of baptism and expressed as a percentage of baptisms. The crisis mortality ratios (CMR) were calculated as the number of burials in 1623 as a ratio of the mean for the preceding decade and the

results are shown in Table 4.7. The parishes in Cumberland and Westmorland suffered most severely: Bridekirk and Crosthwaite experienced a rise in mortality that was 6–7 times greater than that for the previous decade, whereas five parishes (the upland settlements of Thornton-in-Lonsdale and Ingleton and three south Lancashire communities) appeared to have escaped relatively unscathed.

The adult to child burial ratio showed that adult burials usually exceeded child burials in the decade preceding the crisis period. However, there was a change to this pattern in 1623 and, particularly for the northern parishes in the study, the proportion of children dying increased markedly. Two determining factors that have been suggested to explain the occurrence of crisis mortality were, first, the density of settlement, with market towns and the adjacent parishes experiencing higher than average incidence and, second, altitude because lower crisis rates were found in parishes more than 300 feet above sea level (Wrigley & Schofield, 1981). However, regression analysis shows that there was no significant difference between the estimated size of the parish and its CMR, nor was there any correlation with altitude, a finding that supports the work of Long and Pickles (1986), who found that parishes of high altitude in Yorkshire did not suffer particularly high mortality at this time.

As the two northernmost counties displayed particularly high mortality, the CMR for total burials was also analysed with latitude and, although there was a positive, significant association between all parishes and the degree of latitude ($P = 0.04$), it was evident that there was a dividing line, located approximately between southern Westmorland and northern Lancashire, north of which the relationship with latitude was particularly significant (total burials CMR, $P = 0.002$; adult CMR, $P = 0.004$; child CMR, $P = 0.006$). In addition, the percentage fall in baptisms during the crisis experienced by these northern parishes also showed the same response ($P = 0.035$). On the other hand, there was no significant correlation between the mortality ratios and baptisms and latitude for the parishes to the south in Lancashire, nor was there any relationship between latitude and the change in the ratio of adult to child burials.

Of most interest, perhaps, is the infant mortality rate which showed that, for the decade preceding the crisis year, Lancashire parishes experienced higher rates than the communities in Cumberland and Westmorland, i.e. 15.4% and 12.3%, respectively. This is supported by regression analysis: infant mortality was negatively associated with latitude ($P = 0.017$) but, again, this relationship was most noticeable for the communities of Cumberland and Westmorland, i.e. the survival rate for infants in *normal* years significantly improved moving from south Westmorland to northern Cumberland ($P = 0.003$). In 1623, however, as we have seen, there was a complete reversal of this trend and the risks to infants living in the same region now *increased* from south to north ($P = 0.04$).

Could the synergistic interaction between high wheat prices and low wool prices, described in section 4.10, account for the varying severity of the crisis, with

**Table 4.7** Comparison of events in 1623 with events in the preceding decade in 25 parishes in northwestern England.

| Parish | County | Average No of events pa for decade preceding 1623 | | | Events in 1623 | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baptisms | % Infant mortality* | Adult:child ratio | CMR Total | CMR Adult | CMR Child | % Fall in baptisms | % Rise in infant mortality | Adult:child ratio | Change in adult:child ratio |
| Bridekirk | Cumb | 31 | 13 | 3.5 | 6.2 | 5.9 | 6.8 | 45 | 300 | 1.7 | 0.5 |
| Greystoke | Cumb | 44 | 21 | 1.6 | 3.9 | 5.1 | 2.6 | 54 | 120 | 2.6 | 1.6 |
| Penrith | Cumb | 57 | 20 | 3.6 | 5.1 | 5.4 | 4.6 | 33 | 230 | 2.2 | 0.6 |
| Crosthwaite | Cumb | 92 | 10 | 5.2 | 6.9 | 6.2 | 8.8 | 44 | 320 | 1.8 | 0.4 |
| Newbiggin | West | 5 | 9 | 2.3 | 3.4 | 3.0 | 4.2 | 60 | 730** | 1.4 | 0.6 |
| Cliburn | West | 4 | 7 | 5.4 | 4.1 | 3.9 | 4.7 | 25 | 0 | 3.2 | 0.6 |
| Morland | West | 32 | 9 | 4.3 | 4.4 | 4.4 | 4.4 | 41 | 67 | 2.7 | 0.6 |
| Lowther | West | 14 | 5 | 7.6 | 3.8 | 3.8 | 4.0 | 43 | 120 | 4.6 | 0.6 |
| Warcop | West | 21 | 10 | 2.0 | 4.1 | 5.9 | 1.7 | 53 | 170 | 9.0 | 4.5 |
| Brough | West | 39 | 13 | 3.3 | 4.2 | 5.1 | 2.9 | 44 | 100 | 2.7 | 0.8 |
| Crosby Ravenswoirth | West | 32 | 10 | 2.5 | 4.1 | 4.2 | 4.8 | 35 | 100 | 1.5 | 0.6 |
| Kendal | West | 257 | 21 | 2.4 | 3.1 | 3.7 | 2.5 | 40 | 52 | 2.2 | 0.9 |
| Ingleton | W Yorks | 29 | 17 | 2.4 | 1.8 | 1.7 | 2.1 | 28 | 29 | 1.4 | 0.6 |
| Cartmel | Lanc | 75 | 19 | 2.5 | 2.8 | 3.8 | 1.5 | 50 | 32 | 3.9 | 1.6 |
| Urswick | Lanc | 17 | 18 | 4.9 | 4.3 | 3.8 | 3.3 | 0 | 0 | 3.1 | 0.6 |
| Thornton in Lonsdale | Lanc | 13 | 15 | 1.9 | 1.1 | 1.5 | 0.6 | 27 | 39 | 3.9 | 1.6 |
| Caton | Lanc | 19 | 16 | 4.4 | 3.0 | 3.5 | 2.1 | 37 | 56 | 2.8 | 0.6 |
| Lancaster | Lanc | 101 | 10 | 2.6 | 3.1 | 3.2 | 3.2 | 26 | 40 | 2.0 | 0.6 |
| Cockerham | Lanc | 52 | 10 | 2.3 | 4.5 | 4.7 | 4.2 | 52 | 168 | 1.9 | 0.8 |
| Poulton-le-Fylde | Lanc | 77 | 18 | 1.4 | 3.4 | 4.3 | 2.8 | 53 | 89 | 1.4 | 0.0 |
| Whalley | Lanc | 52 | 21 | 3.4 | 2.3 | 2.7 | 1.7 | 17 | 80 | 3.5 | 1.5 |
| North Meols | Lanc | 21 | 8 | 3.1 | 1.9 | 2.3 | 1.1 | 47 | 125 | 6.3 | 2.0 |
| Prestwich | Lanc | 48 | 15 | 2.8 | 3.1 | 3.5 | 2.5 | 25 | 62 | 2.9 | 1.0 |
| Sefton | Lanc | 53 | 21 | 2.3 | 1.5 | 1.4 | 1.5 | 58 | 114 | 1.6 | 0.7 |
| Walton-on-the-Hill | Lanc | 42 | 14 | 3.4 | 1.3 | 1.6 | 1.0 | 43 | 122 | 5.9 | 1.7 |
| Mean | | | 14.0 | 3.2 | 3.5 | 3.8 | 3.2 | 39.2 | 130.6 | 3.0 | 1.0 |
| SD | | | 5.0 | 1.4 | 1.4 | 1.4 | 1.9 | 14.1 | 149.3 | 1.8 | 0.9 |

* Infant mortality expressed as percentage of live births in that year.
** Rise in infant mortality occurred in 1624.
CMR, crisis mortality ratio; SD, standard deviation.

changes in the pattern of infant mortality depending on the basic economy of the community? An aggregative analysis was made for six parishes (including Penrith, described above) whose records are extant for the period 1587–1643, so encompassing the three major mortality crises. Table 4.8 shows the parishes listed in order of decreasing latitude and, consequently, in order of the severity of the crisis in 1623, and gives the results of simple and multiple regression analysis for adult and child mortality.

**Table 4.8** Regression analysis of wheat and wool index numbers with mortality in northwestern England.

| Parish | Simple regression | | Multiple regression | | |
|---|---|---|---|---|---|
| | High wheat index Nos *P* | Low wool index Nos *P* | High wheat index Nos *P* | Low wool index Nos *P* | Overall *P* |
| Penrith, Cumberland, 1587–1643 | | | | | |
| Adult mortality | 0.002 | 0.017 | <0.001 | <0.001 | <0.001 |
| Total child mortality | 0.025 | NS | 0.004 | 0.018 | 0.005 |
| Infant mortality | 0.009 | NS | 0.001 | 0.004 | <0.001 |
| Crosthwaite, Cumberland, 1587–1643 | | | | | |
| Adult mortality | NS | 0.011 | 0.008 | 0.002 | 0.001 |
| Total child mortality | NS | 0.007 | NS | 0.002 | 0.005 |
| Crosby Ravensworth, Westmorland, 1587–1643 | | | | | |
| Adult mortality | NS | 0.008 | 0.019 | 0.002 | 0.002 |
| Total child mortality | NS | 0.009 | 0.019 | 0.002 | 0.002 |
| Cartmel, Lancashire, 1592–1643 | | | | | |
| Adult mortality | 0.028 | 0.013 | 0.010 | 0.004 | 0.002 |
| Total child mortality | NS | NS | NS | NS | NS |
| Thornton-in-Lonsdale, West Yorkshire, 1587–1643 | | | | | |
| Adult mortality | 0.007 | NS | 0.003 | NS | 0.009 |
| Total child mortality | NS | NS | NS | NS | NS |
| Walton-on-the-Hill, Lancashire, 1587–1641 | | | | | |
| Adult mortality | NS | NS | NS | NS | NS |
| Total child mortality | 0.007 | 0.002 | 0.05 | 0.015 | 0.001 |

NS, not significant.

For the upland parishes of Crosthwaite in Cumberland and Crosby Ravensworth in Westmorland, low wool prices had the most important influence but, like Penrith, the combined effect with high wheat prices is very marked (Table 4.8). Crosthwaite suffered major mortality crises in 1587 and 1623 and Crosby Ravensworth suffered in all three crisis years, 1587, 1597 and 1623, suggesting the synergistic effect of wheat and wool prices. The evidence from these studies suggests, therefore, that the economy of these farming communities on the fells was strongly dependent on sheep rearing, as suggested by Thirsk (1967).

However, only adult mortality showed a significant response at Cartmel (Lancashire) and Thornton-in-Lonsdale (Lancashire–Yorkshire borders); the former to both wheat and wool prices, the latter only to wheat prices. Children at Walton-on-the-Hill (Lancashire) were adversely affected by the price movements of both commodities. Further regression analysis (data not shown) has revealed that adult burials at Thornton-in-Lonsdale were also significantly associated with variations in cattle prices ($P = 0.013$) and all mortalities at Walton-on-the-Hill were linked to the prices of hides (adults $P = 0.006$; children $P < 0.001$). These factors were not significantly associated with mortality at Penrith, Cartmel, Crosthwaite or Crosby Ravensworth. The parishes of Cartmel, Walton-on-the-Hill and Thornton-in-Lonsdale, lying to the south of Cumbria, did not suffer from the mortality crises of 1587, 1597 or 1623, again suggesting that the economy of these parishes was less sensitive to wool prices, although Cartmel was a market town specialising in sheep and corn (Everitt, 1967). This type of analysis is a valuable one for historical demographers; by correlating the mortality series with a range of commodity indices in multiple regression analysis, it is possible to suggest which synergistically interacting factors governed the economy of an identified community.

In summary, therefore, this analysis of parishes in the northwest has shown that the severity of the mortality in the late sixteenth and early seventeenth centuries was linked to the relative dependence of the community on wool production. Small farmers and husbandmen were numerous amongst the wool growers and they often existed at the margin of subsistence, requiring an adequate price for their wool to purchase grain on the market, to pay rents and to meet normal, everyday expenses (Bowden, 1967). For such regions, specialising predominantly in wool production, periods of high wool and low wheat prices (as during the decade 1613–22, preceding the 1623 mortality crisis; see Table 4.7) were times of relative plenty, and infant mortality for Cumberland and Westmorland was at a *lower* level than for the communities further south in Lancashire. In 1623, however, the double impact of high grain and low wool prices proved disastrous, resulting in chronic food shortages, malnutrition and even famine, with the inevitable rise in mortality. In Lancashire, the principal occupation was cattle rearing and fattening, and fewer farmers kept flocks of sheep (Thirsk, 1967). Consequently, the farmers there may not have experienced the comparative halcyon periods when wheat prices were low and wool prices high, but neither did they suffer so grievously when the reverse scenario occurred.

To conclude, time-series analysis of the mortality crises at the end of the sixteenth century and the start of the seventeenth century, described by Appleby as characteristic of the northwest, shows that hardship and malnutrition occurred only in years when the fluctuations in two commodity indices synchronised and interacted synergistically. Examination of the statistical intercorrelation between the mortality cycles (both child and adult) in a population and the different commodity prices allows the historical demographer to explore the underlying

economic factors that were of most importance in controlling the dynamics of the exogenous cycles in that community.

## 4.12 Conclusions

Famines in the Third World in the twentieth century were on a catastrophic scale and the demographic consequences were obvious. Usually the problems of severe food shortage were exacerbated by economic, political, social or demographic factors. Hugo (1984) has reviewed and integrated current knowledge relating to the demographic consequences of famine and these are summarised in Table 4.1.

In contrast, England has largely escaped from severe and overt famine – most of the mortality crises in the pre-industrial period that the population suffered were the result of a plague epidemic (Scott & Duncan, 2001). The clearest examples of famine are found in small communities in northwest England at the end of the sixteenth and the start of the seventeenth centuries when the number of burials in a single year in some communities increased some 4 to 6-fold above the normal mean levels – far below the mortality experienced in the catastrophic famines of Africa and Asia in the twentieth century. We have shown that these mortality crises were confined locally and were not only the result of high grain prices and the resulting food shortages, but occurred in communities that were also hit by low wool prices.

The thesis that will be developed in this book will be that outright famine had limited demographic effects in England, whereas malnutrition had serious subliminal and diverse consequences over many centuries. Analysis of events during the identified famines at Penrith in 1623 and during the Dutch winter is particularly useful to us because it exacerbates and concentrates into a short time span the effects of malnutrition which were normally experienced subliminally. A study of famine reveals that malnutrition has its most serious effects during pregnancy, which result in high infant mortality. The demographic importance of malnutrition before, during and after pregnancy is described in Chapters 6 to 8.

Chapter 5

# Long-term Demographic Effects of even a Small Famine

As we have seen, England was rarely struck by outright famines and certainly not after 1623. At worst, the CMR rarely rose above 4 to 6 and the northwest was the worst affected region. The major mortality crises in rural England were the result of plague epidemics which struck erratically at medium-sized and large towns and which ceased after 1670 (Scott & Duncan, 2001). Penrith was no exception; in addition to the famines of 1587, 1596/7 and 1623 there was a catastrophic plague epidemic in 1597/8. In this chapter, we show how even a small mortality crisis in a population can have long-term demographic effects, triggering oscillations in the annual number of births and deaths.

## 5.1 Endogenous oscillations in the population at Penrith, Cumbria, England

The famine at Penrith in 1623 and its consequences are the best documented that we have found and the parish registers record that 241 people died in 1623 (see Table 4.6). During the seventeenth century and the first half of the eighteenth century, the population at Penrith existed in a steady state: mean annual baptisms equalled mean annual burials and were constant at 60 per annum. After 1750, the population escaped from steady-state conditions and a population boom began. Although *mean* annual events remained constant over a 150-year period, analysis of both the baptisms and burials series shows clear evidence of regular oscillations. This suggests that the population at Penrith was demographically maintained in a steady state by density-dependent constraints, of which the food supply was an important factor. Thus, as the population increased in size by immigration and new births, so the pressure increased, resulting in raised infant mortality and emigration. In this way, the baptism and burial series oscillated within strict limits about the steady-state level.

This pattern is illustrated in Figs 5.1A and B. Annual baptisms at Penrith from 1615 to 1750 are shown in Fig. 5.1A and spectral analysis of this series shows (i) a

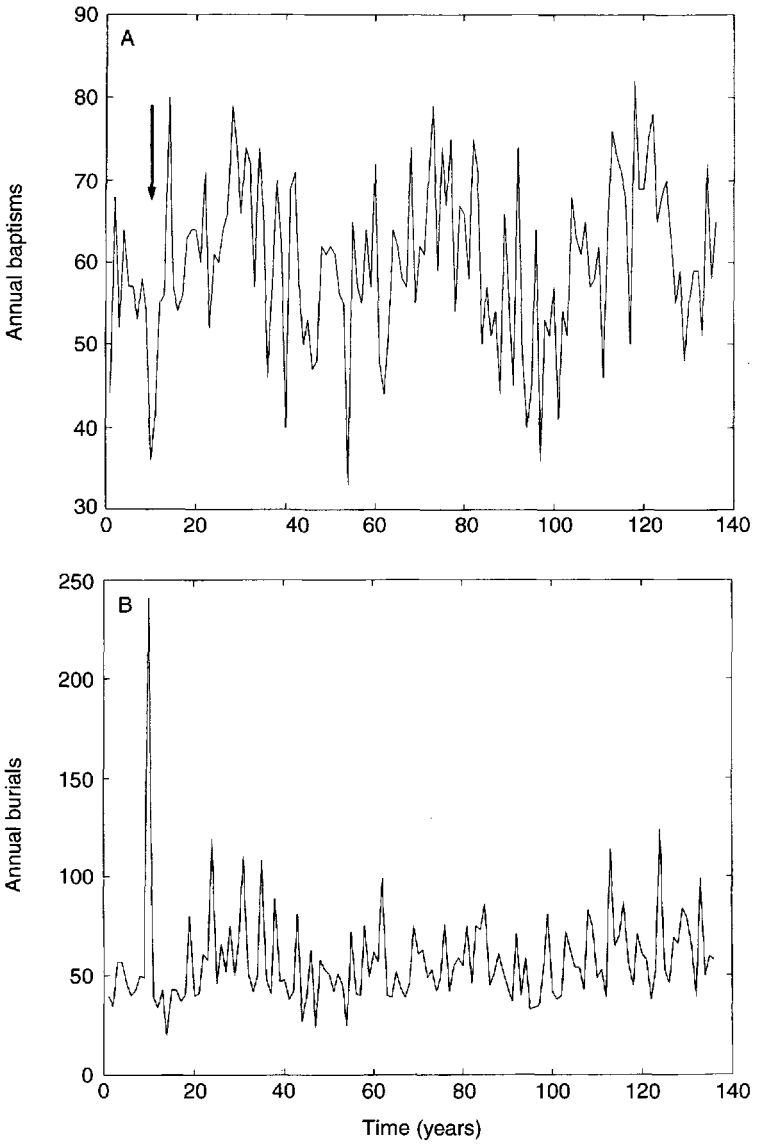


**Fig. 5.1** Parish register series at Penrith, Cumbria, UK, 1615–1750. A: Annual baptisms; note fall in baptisms during the famine of 1623 (arrow) and long wavelength oscillation that followed it. B: Annual total burials; note marked rise in deaths during the famine of 1623.

strong 43-year oscillation ($P < 0.005$), which persisted for the whole period, and (ii) a secondary oscillation of period 33 years, which was detectable for only 65 years after the 1623 famine and disappeared after 1688 – an example of a decaying oscillation. The 43-year oscillation after filtering is shown in Fig. 5.2, with three peaks between 1623 and 1750. It is also noteworthy, as can be seen in

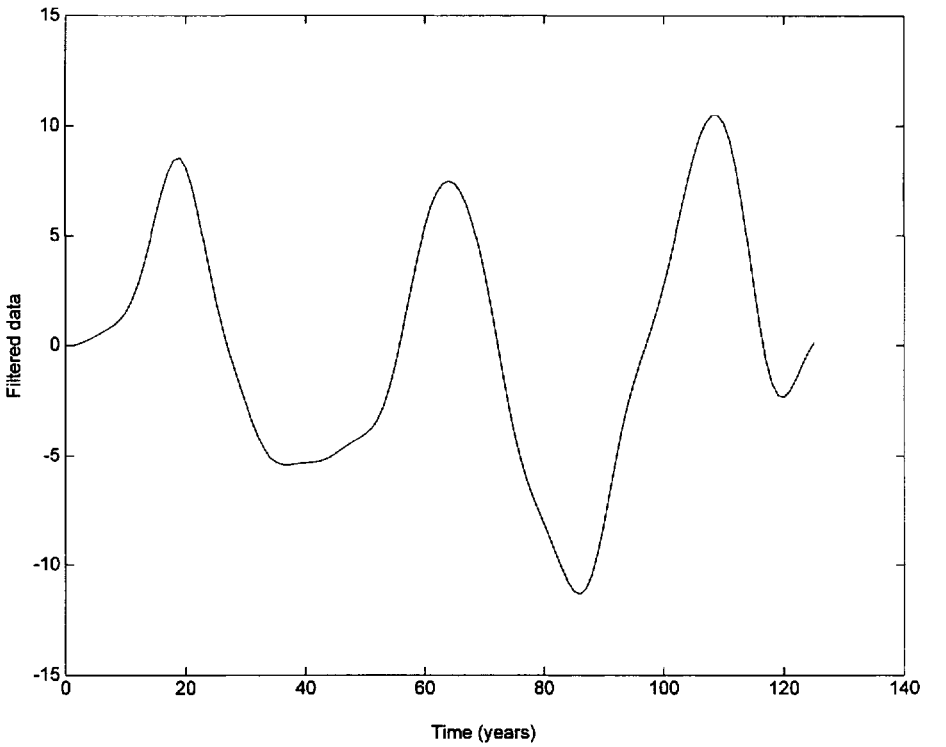


**Fig. 5.2** Filtered baptisms at Penrith (see Fig. 5.1A), 1626–1750, after the famine, to show the 43-year oscillation. Filter window = 33–50 years.

Figs 5.1A and 5.2, that mean annual baptisms rapidly returned to the prefamine, steady-state level.

The annual burial series at Penrith for 1615 to 1750 is shown in Fig. 5.1B and the dominating effect of the famine can be clearly seen. It overshadows any endogenous oscillations that it triggers, but time-series analysis reveals the following oscillations:

(1) An *exogenous* short-wavelength, non-stationary oscillation (period 5–6 years), driven by an oscillation in wheat prices (see Chapter 3), the importance of which is described in Chapter 10.
(2) An *endogenous* oscillation with a wavelength of 43 years which persists throughout the steady-state period, 1623 to 1750, and is directly comparable with the endogenous oscillation in baptisms (Fig. 5.1A). It is revealed after filtering (see Fig. 5.3).

The 43-year, non-decaying oscillations in both baptisms and burials follow the famine of 1623 and are apparently initiated by it. As we shall see, the characteristics of these long wavelength oscillations are determined by the demo-

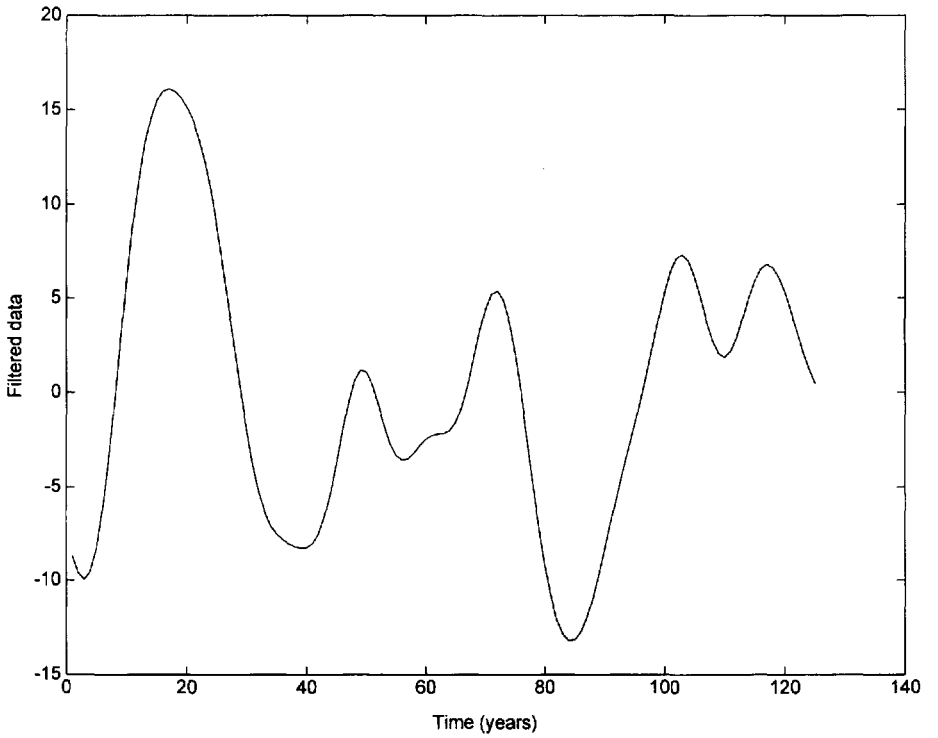


**Fig. 5.3** Total burials at Penrith (see Fig. 5.1B), 1626–1750, after the famine, filtered to show the 43-year oscillation. Filter window = 33–50 years.

graphic properties of the population and, so, are described as endogenous, in contrast with exogenous population cycles, which are directly driven by external forces (e.g. cycles in the availability of food).

The cross-correlation function (Fig. 5.4) shows that the 43-year oscillations in baptisms and burials were synchronous (i.e. cross-correlated at zero lag). We conclude that, after the 1623 famine at Penrith, synchronous, endogenous 43-year oscillations in baptisms and burials were generated and that these persisted until 1750, as long as the population was in steady state, maintained by density-dependent constraints.

## 5.2 Modelling the population dynamics

The population dynamics at Penrith and the impact of the famine are clearly complex. Why is it suggested that these long wavelength oscillations were endogenous, i.e. dependent on the inherent properties of the system and not driven by external forces? The human reproductive cycle is characterised by the very long delay of maturation before a woman starts child-bearing. The

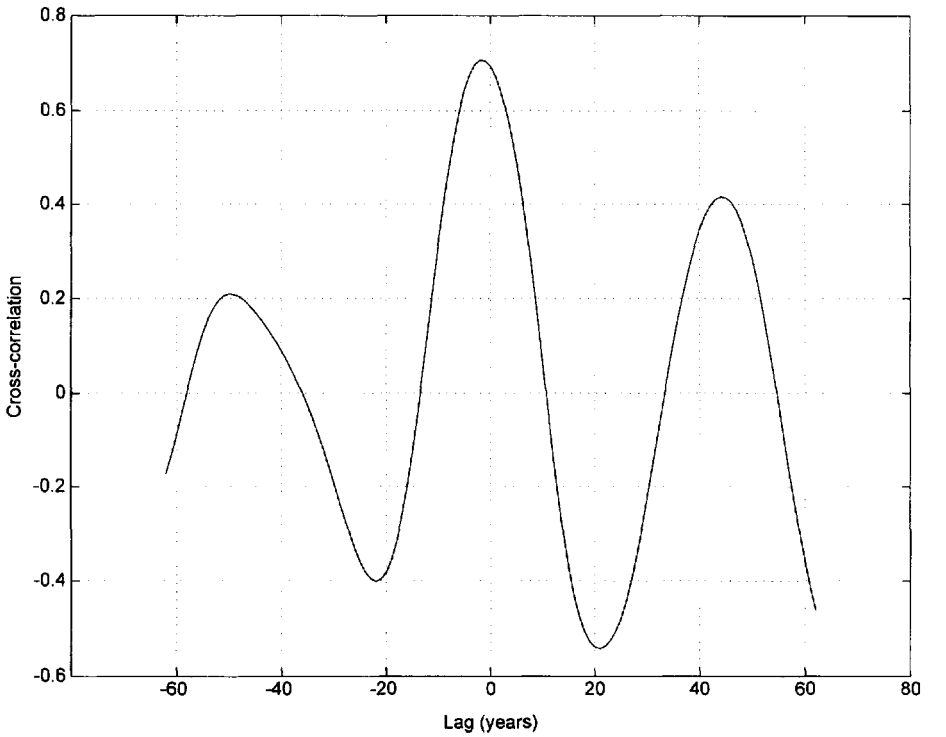


**Fig. 5.4** Cross-correlation of total burials versus baptisms at Penrith, 1626–1750. Ccf = 0.7 at zero lag.

mean age at which a woman had her median child at Penrith was 33 years, a value broadly comparable with pre-industrial, rural England. When, as at Penrith, the mean annual number of births equalled the mean annual number of burials, the system can be represented (Fig. 5.5) by a simple births loop with a gain of 1.0. Such a system is inherently unstable and will tend to oscillate in response to a perturbation, such as a mortality crisis, generating endogenous oscillations.

These conclusions can be demonstrated by a Leslie matrix model of the population at Penrith. The basis of the initial model is a state vector ($\boldsymbol{x}(t)$) with 50 entries which describes the number of women in each age group (i.e. 0–1, 1–2, ... 49–50) for each year.

$$\boldsymbol{x}(t) = \begin{bmatrix} x_1(t) \\ x_2(t) \\ \cdot \\ \cdot \\ \cdot \\ x_{50}(t) \end{bmatrix}$$

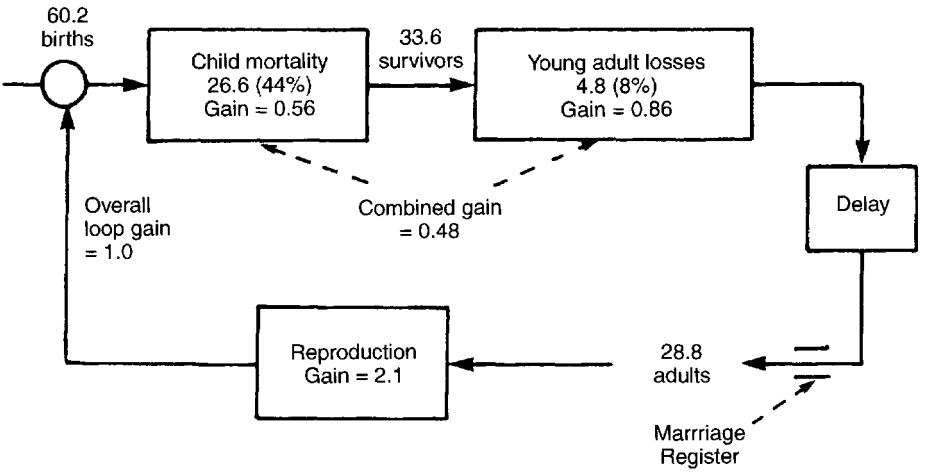


**Fig. 5.5** Diagram to illustrate the births loop at Penrith, 1557–1750. The mean annual births remained constant during this time. The child and preadult mortality (total gain = 0.48) is followed by the long delay before reproduction began, which can be measured by inspection of the marriage register. Reproduction is estimated as 2.1 children/woman. Such a system with an overall loop gain of 1.0 and a long delay is inherently unstable and will tend to oscillate in response to a perturbation.

where $x_1(t)$ is the number of people of age 0–1 at time $t$. This vector characterises the state of the population at time $t$. There are two factors, mortality and fertility, that will influence the movement from $x(t)$ to $x(t+1)$. Proceeding from $t$ to $t+1$, the 0–1 year olds will become 1–2 year olds, except for those that die. Thus

$$x_2(t+1) = p_1 x_1(t)$$

where $p_1$ is the fraction of 0–1 year olds surviving to become 1–2 year olds. Similarly

$$x_3(t+1) = p_2 x_2(t)$$

$$x_4(t+1) = p_3 x_3(t)$$

and so on. The number of births in year $t$ and hence the number of 0–1 year olds in year $t+1$ is equal to

$$f_1 x_1(t) + f_2 x_2(t) + \ldots\ldots + f_{50} x_{50}(t)$$

where $f_1, f_2 \ldots f_{50}$ represent the fertility for each age group. Fertility will equal zero for the young (prepuberty) and the old (post-menopause) women.

The state vector at time $t+1$, $\boldsymbol{x}(t+1)$, is given by

$$\begin{bmatrix} x_1(t+1) \\ x_2(t+1) \\ x_3(t+1) \\ \cdot \\ \cdot \\ \cdot \\ x_{50}(t+1) \end{bmatrix} = \begin{bmatrix} f_1 & f_2 & f_3 & \cdots & f_{50} \\ p_1 & 0 & 0 & & 0 \\ 0 & p_2 & 0 & & 0 \\ & & & \ddots & \cdot \\ & & & & \cdot \\ & & & & \cdot \\ 0 & 0 & \cdots & p_{49} & 0 \end{bmatrix} \begin{bmatrix} x_1(t) \\ x_2(t) \\ x_3(t) \\ \cdot \\ \cdot \\ \cdot \\ x_{50}(t) \end{bmatrix}$$

where $f_i$ (for $i = 1$ to 50) = the probability of a woman in that age group giving birth, and $p_i$ = probability of a woman of age $i$ surviving to age $i+1$ (Pielou, 1969). Therefore, the state vector at time $t+1$, $\boldsymbol{x}(t+1)$, is calculated by multiplying the state vector at time $t$, $\boldsymbol{x}(t)$, by a matrix, which is denoted by $A$. Since the matrix $A$ describes the evolution of the system from the state at time $t$ to the state at time $t+1$, it is referred to as the state evolution matrix and

$$\boldsymbol{x}(t+1) = A\boldsymbol{x}(t)$$

where $\boldsymbol{x}(t)$ and $\boldsymbol{x}(t+1)$ are vectors with 50 elements and $A$ is a 50 by 50 matrix. The $A$ matrix is a linear model of what is almost certainly a non-linear process.

Given the state of the system, i.e. the profile of the population, at any time, $t_0$, the model can predict the female population at all future times $t > t_0$, by repeated application of this equation. The total female population at time $t$, $s(t)$, is given by

$$s(t) = \sum_{i=1}^{50} x_i(t)$$

the number of births, $b(t)$, in year $t$, is given by

$$b(t) = x_1(t)$$

and the number of deaths, $d(t)$, in year $t$, can be calculated from

$$d(t) = s(t+1) - s(t) - b(t)$$

The known fertility and mortality functions at Penrith, determined from a family reconstitution study (Duncan *et al.*, 1992), are fed into the model which is then run over time in which the population is hit by a mortality crisis in which 20% die indiscriminately, a situation equivalent to the 1623 famine at Penrith. Figure 5.6A illustrates the results of the modelling: the number of births falls immediately to 80% of the precrisis level and a decaying oscillation with a wavelength equal to the mean of the fertility function is established (i.e. 33 years). This is in agreement with the predictions above (Duncan *et al.*, 1994a).

The population and the steady-state level of the number of births predicted by

the model does not recover to its pre-crisis level (Fig. 5.6A). Figure 5.6B illustrates a 50% mortality, such as might be caused by a major plague epidemic; the population continues at its new, lower level. Even a very small famine mortality can trigger large-amplitude oscillations in the model. Figure 5.6C illustrates a 10% mortality, which might be the result of a smaller famine crisis anywhere in rural England. The first peak of the oscillation now rises above the original steady-state level and the population immediately establishes a new steady-state level above the 90% level, but still below the precrisis level. Even a 1% mortality is sufficient to perturb the system and to initiate oscillations (Fig. 5.6D); the population immediately establishes a new steady-state level *above* the old pre-crisis level.

The model also predicts that a famine crisis will trigger a comparable oscillation in deaths which will be synchronous and at the same wavelength as the oscillation in births, although at slightly lower amplitude (see Figs 5.7A and B). This compares to the situation at Penrith where the long wavelength oscillations were synchronous (see Fig. 5.4).

## 5.3 Incorporation of density-dependent constraints into the matrix model

Thus, the computer modelling confirms theoretical predictions that synchronous *decaying* oscillations in births and deaths with a wavelength equal to the mean of the fertility function can be triggered in human populations by a famine mortality crisis. However, the modelling shows three major differences with the situation at Penrith after 1623:

(1) The major oscillations in baptisms and burials at Penrith had a period of 43 years (see Figs 5.2 and 5.3) and not at 33 years, as predicted by the modelling.
(2) These long wavelength oscillations in baptisms and burials at Penrith did not decay.
(3) The population numbers recovered almost immediately to the precrisis, steady-state level.

The third observation suggests strongly that the population at Penrith was living under strict density-dependent constraints and, when these were lifted after a mortality crisis, more food was available (with fewer mouths to feed), which led to improved nutrition and lower levels of infant mortality and, consequently, to a return to the original steady-state level (see Figs 5.1A and B). Consequently, the basic matrix model needs to be augmented by a feedback mechanism that drives the system to a 'target' steady-state value that equals the optimum level of population that is sustainable with the resources available. If this target value is

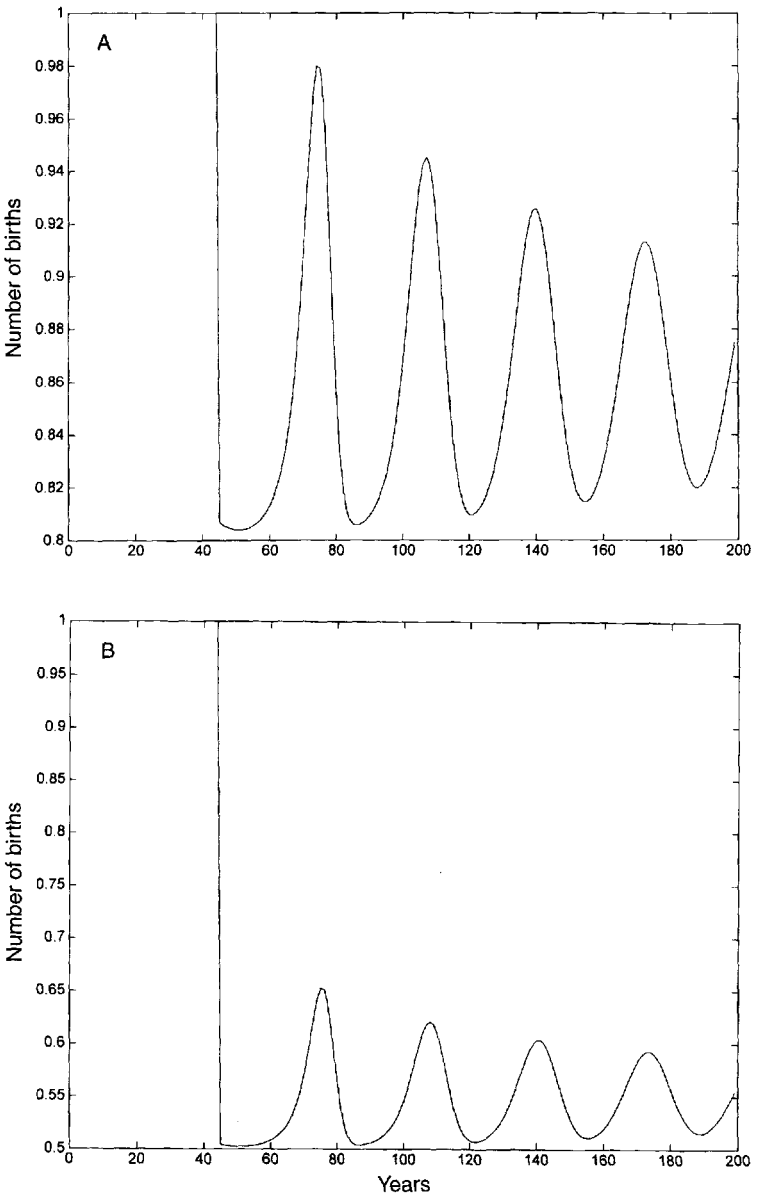


**Fig. 5.6** Number of births per annum shown as a proportion of the initial population (ordinates) predicted by the model with no feedback. The model is run for 43 years before the population is hit by a mortality crisis produced by a famine. Mean and standard deviation of the fertility function = 33 and 3 years, respectively. Abscissae: time (years) of running the model. Proportion of the population dying in the famine, A: 20%; B: 50%; C: 10%; D: 1%.

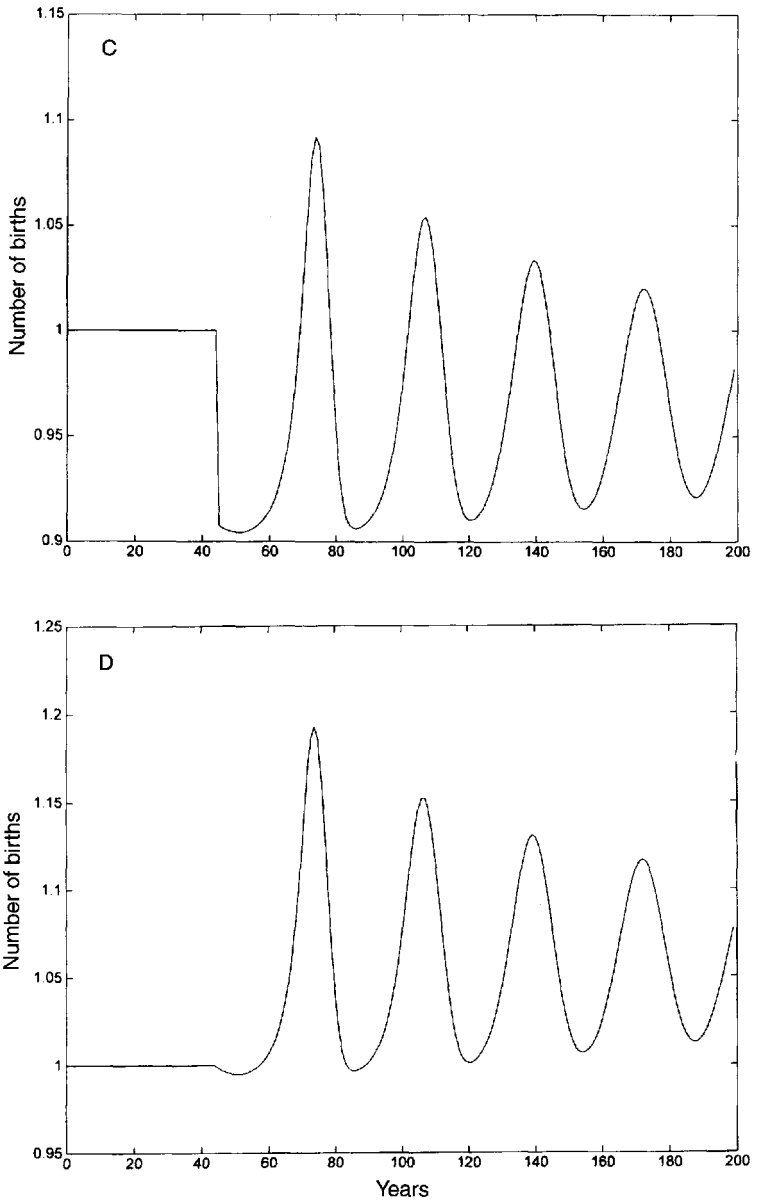


**Fig. 5.6** Continued

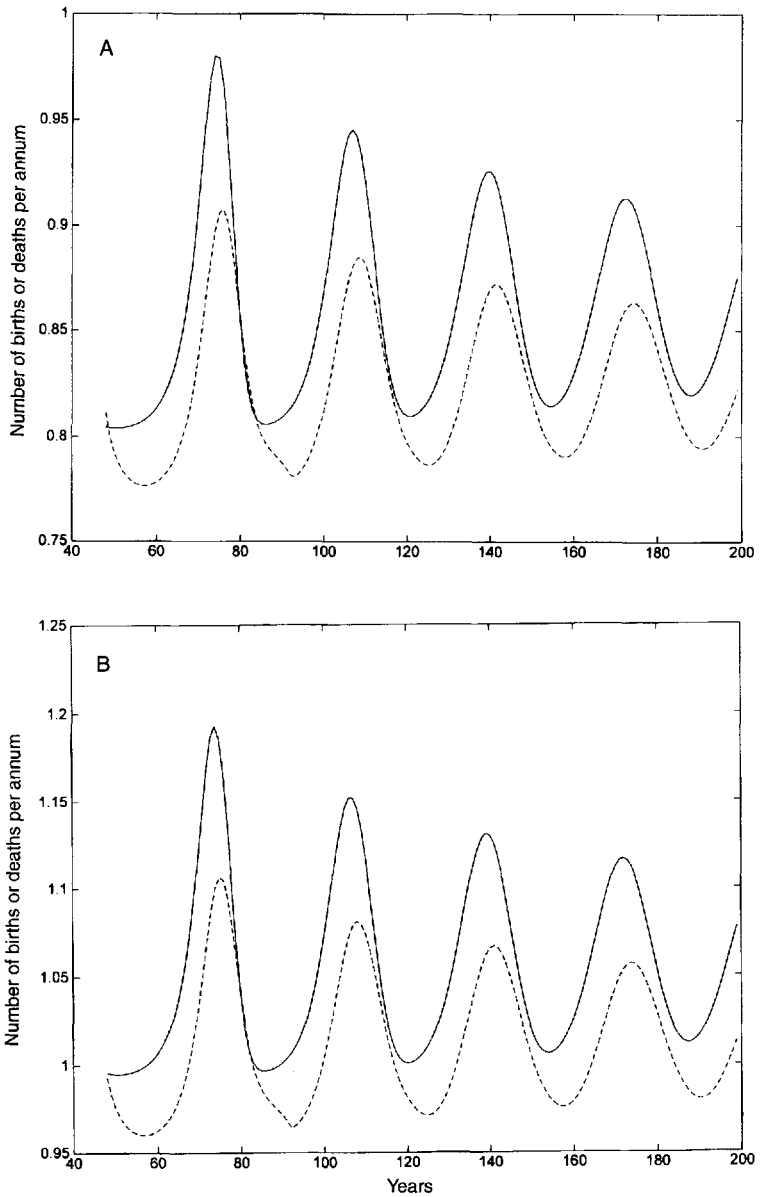


**Fig. 5.7** Number of births (continuous line) and deaths (dashed line) per annum shown as a proportion of the initial population (ordinates) predicted by the model with no feedback. Mean and standard deviation of the fertility function = 33 and 3 years, respectively. Abscissae: time (years) of running the model. Proportion of the population dying in the famine which initiates the oscillations, A: 20%; B: 1%.

denoted as $s(0)$ then, at each time step in the model calculation, the current level of the population, $s(t)$, is compared with the 'target' value to generate a fractional discrepancy, $e(t)$, which is given by

$$e(t) = \frac{s(t) - s(0)}{s(0)}$$

This discrepancy is multiplied by the gain and is used to adjust the $A$ matrix (see Fig. 5.8).

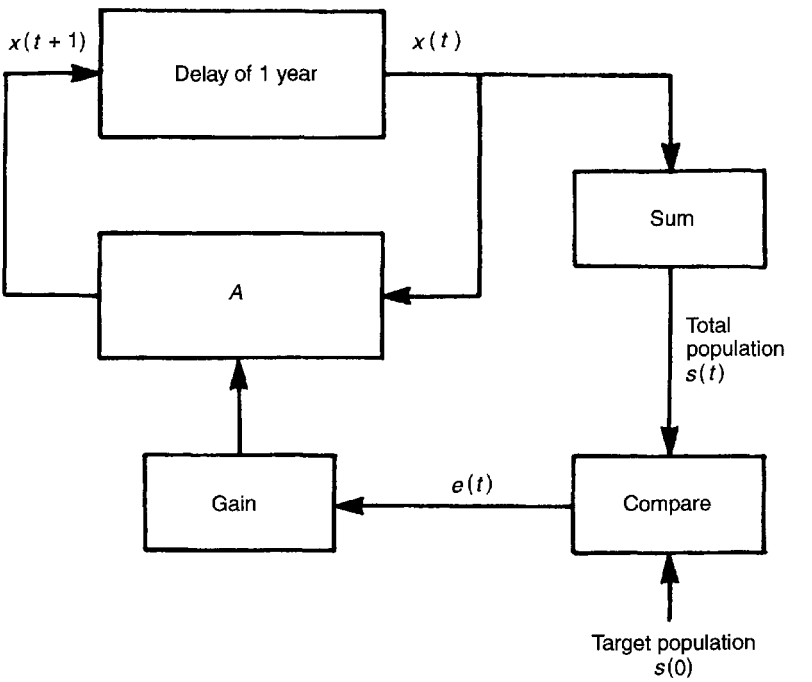


**Fig. 5.8** Diagram to illustrate the feedback mechanism that is added to the matrix model so that the system is driven to its 'target value', $s(0)$. The model now simulates a population under density-dependent constraints, where the total amount of food and other resources available effectively determines the pressure on the individuals within the community.

If there are no external influences, only two properties of the modelled system can be adjusted to drive the birth rate back to a fixed level, namely fertility and mortality. The results of a family reconstitution study suggest that there were no major changes in fertility at Penrith during the years 1623–1750 (see section 6.9) and it is therefore probable that changes in mortality (see Chapter 10) and not fertility are the basis of the feedback mechanism that drove the birth rate to its fixed, precrisis level.

The feedback mechanism is incorporated into the model by making the mor-

tality function, denoted by $m_i$ (where the probability of survival $(p_i) = 1 - m_i$, for $i = 1$ to 50), time-varying and adjusting it on the basis of the fractional discrepancy $e(t)$ between the 'target' total population, $s(0)$, and the current level of the population, $s(t)$, according to

$$m_i(t) = m_i(0)[1 + g\ e(t)]$$

where $m_i(t)$ is the probability of a woman of age $i$ dying before she reaches age $i + 1$ in year $t$; $m_i(0)$ is the probability of death between the ages $i$ and $i + 1$ for the stable age distribution, $\boldsymbol{n}_s(t)$, and $g$ is the gain. The gain is the size of the correction in response to a given discrepancy between the current level of the population and its target level. The gain is defined such that a 1% fractional discrepancy results in a 10% increase in mortality when $g$ is 10.

The new, extended model can now be run, setting the gain on the feedback at 5. When the population suffers a 20% famine mortality, the response of the model is now completely different, as shown in Fig. 5.9. The population now recovers to its precrisis steady-state level driven to this position by feedback, and a *large* amplitude oscillation is generated, the wavelength of which is 43 years. Thus, the inclusion of feedback in the model drives the steady-state condition back to its predetermined level, as would be expected, and also, surprisingly, changes the wavelength of the oscillation from 33 years (the mean of the fertility function fed into the model) to 43 years, the wavelength observed at Penrith. When the model is run with different gains on the feedback, 43-year oscillations are generated at gains from 2 to 7. The higher gains produce a greater amplitude of the oscillations which decay more slowly. At gains below 0.6, the population recovers to its prefamine steady-state level, but the feedback is inadequate to generate a 43-year oscillation and the wavelength of the cycle remains at 33 years.

## 5.4 Conclusions: endogenous population oscillations

Long-term, endogenous oscillations in births and deaths can be generated in human populations in response to a famine mortality crisis, even when only a small proportion of the community died. A 43-year oscillation is predicted to occur in a human population when a typical fertility function is coupled with feedback. The modelling suggests that the nature of the response will be dependent on the underlying characteristics of the population and, particularly, on whether the system was existing under density-dependent constraints, such as the available food supply and other resources. The modelling explains the triggering of the 43-year oscillation at Penrith (Figs 5.2 and 5.3), a population that existed under marginal conditions, was malnourished and was under density-dependent constraints. We can see from the foregoing that any of the following different dynamics would have existed in the parishes of pre-industrial rural England:

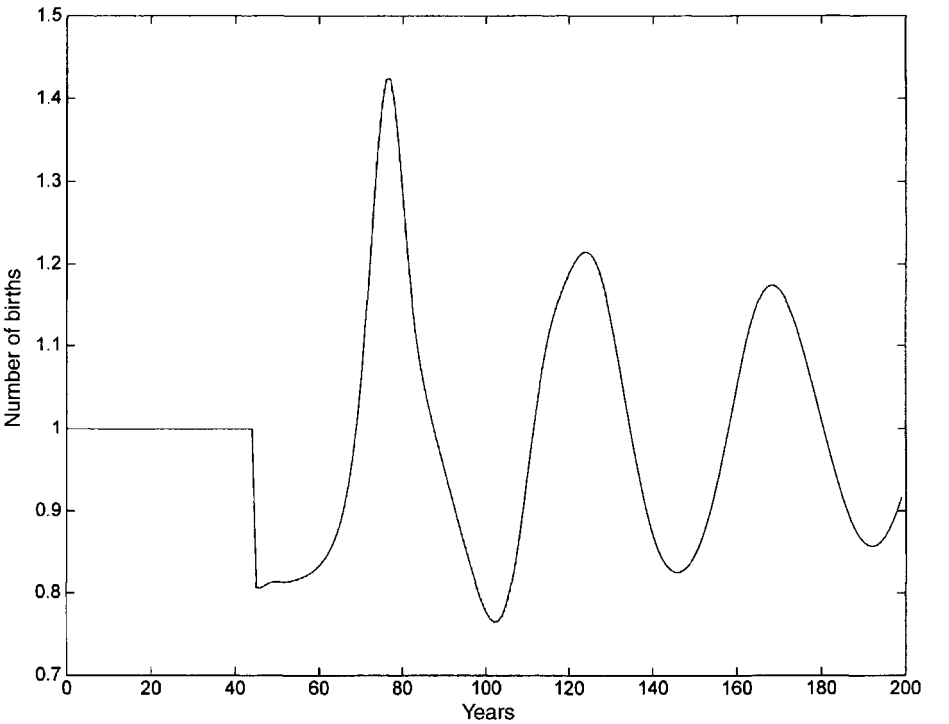


**Fig. 5.9** Number of births per annum shown as a proportion of the initial population (ordinate) predicted by the model when a gain of 5 in the feedback is included. The model is run for 43 years before the population is hit by a mortality crisis in which 20% of the inhabitants die. Mean and standard deviation of the fertility function = 33 and 3 years, respectively. Abscissa: time (years) of running the model. Note that with the incorporation of feedback, the mean annual number of births recovers to its original, precrisis level.

(1) Oscillations in births and deaths of either about 33 years or about 43 years, consequent upon the specific density-dependent constraints.
(2) Decaying or persistent oscillations.
(3) Different amplitudes of the oscillations under different conditions.
(4) Either a rapid recovery to the prefamine steady state as a result of feedback or a continuation at the new, lower level to which the population has been 'knocked back'.

With these different scenarios in mind, we have studied the 404 parishes in rural England described by Wrigley and Schofield (1981) using time-series analysis to reveal any significant oscillations in the data series. Of course, reality is vastly more complex than that predicted by computer modelling and most parishes suffered from a sequence of mortality crises, some small and some catastrophically severe. The major crises were usually the result of plague epidemics lasting about 8 to 9 months, which did not occur in communities of low

population density (Scott & Duncan, 2001). Smaller mortalities lasting about 3 months were probably the result of smallpox epidemics in children. Again, these did not occur in low-density populations (Scott & Duncan, 1998). There were years in the eighteenth century when epidemics of an unknown lethal disease were widespread (see Wrigley & Schofield, 1981). However, the majority of the other crises would be the result of minor famines with a CMR of about 1.5 to 3, a sufficient amplitude to generate endogenous oscillations.

The series of annual total burials at Bridgwater, Somerset, UK is shown in Fig. 5.10 and is an example of apparently chaotic population dynamics and a completely different situation from that at Penrith. The population was in an overall steady state from 1565 to 1800, with a small and late boom thereafter. There were, to what must have seemed to the community, a never-ending series of mortality crises, some of which may have been because of plague epidemics. The maximum CMR was about 2.5 but there were many smaller crises, particularly between 1680 and 1720. Some long wavelength, endogenous oscillations are detectable by eye in this series and time-series analysis reveals a 42-year oscillation, although this is not statistically significant. The irregular series of mortality crises would disrupt the establishment of a long-term, regular oscillation.

However, Bridgwater was exceptional in suffering from such a long succession

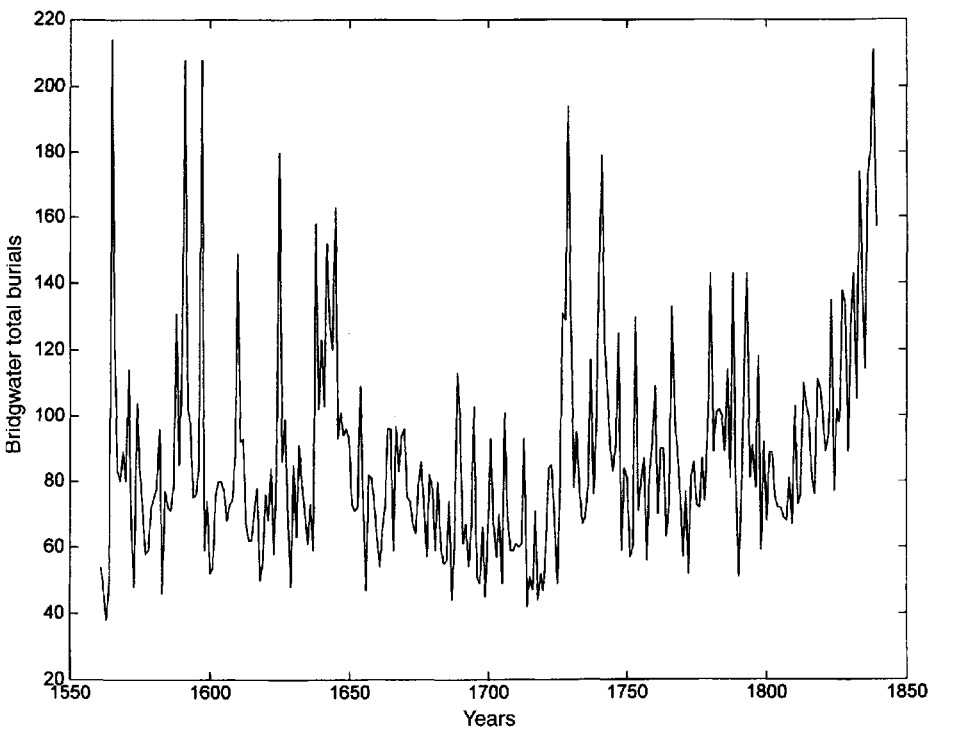


**Fig. 5.10** Annual total burials at Bridgwater, Somerset, UK, 1561–1839.

of crises, even after 1750. Time-series analysis of the data series for the 404 parishes reveals numerous examples of all four of the conditions of population dynamics listed above. Many populations show either 32/33- or 42/43-year oscillations in the baptisms series, all triggered by mortality crises. We see that a modest CMR produced by famine conditions can have subtle, but profound effects on the population dynamics of the community which may persist for many years: endogenous oscillations in births and/or deaths may be initiated and the population may be set back, or it may recover rapidly to its precrisis level, being driven back by density-dependent constraints such as improved nutrition (which led to reduced infant mortality) and immigration.

Chapter 6

# Fertility

Considerable confusion exists concerning the meaning of the term fertility. A biologist defines fertility as the potential capability of an organism to reproduce itself, in contrast with fecundity which is defined as the number of offspring produced by a female (*A Dictionary of Biology*, Oxford University Press). A demographer, therefore, is primarily interested in fecundity, which is a major factor in population dynamics and is, in part, dependent on a woman's fertility. A physiologist or a nutritional biochemist is primarily interested in fertility.

On the other hand, Wrigley *et al.* (1997) define fecundity as the capacity of an individual or couple to reproduce, and fertility as the scale of reproduction that actually occurs. As a consequence, demographers frequently seem to use the term fertility for both biological parameters.

## 6.1 The importance of body fat

Frisch has made a special study of the role of body fat in fertility and we have found her comprehensive reviews (1990; 1999) of particular help in preparing this chapter. At the completion of growth, between the ages of 16–18 years, the body of a well-nourished woman contains about 26–28% fat and about 52% water, whereas the body of a man at the same stage of development contains about 14% fat and 61% water. The main function of the 16 kg of stored female fat, which is equivalent to over 600 MJ (144 000 kcal), is to provide energy for a potential pregnancy and for about 3 months' lactation. In earlier times, when the food supply was scarce or fluctuated seasonally, stored fat would have been necessary for successful reproduction. Fat is the most labile component of the body weight and body fat therefore would reflect environmental changes in food supplies more rapidly than other tissues (Frisch, 1999).

Women who are underweight experience disruption of their reproductive ability; moderate weight loss, in the range of 10–15%, results in amenorrhoea because of hypothalamic dysfunction. This level of weight loss is equivalent to a loss of one-third of body fat. If the excessive leanness occurs before menarche, it may be delayed until as late as the age of 19–20 years. These findings have led to

the prediction of a minimum weight for height for the onset and maintenance of regular ovulatory menstrual cycles (Frisch, 1999).

The idea that relative fatness is important for female reproductive ability followed findings that the menarche in girls is related to the *average* critical body weight of the population. Observations of earlier menarche are associated with attaining the critical weight more quickly. For example, there has been a long term trend in an earlier menarche in Europe of about 3-4 months per decade in the last 100 years. Frisch's explanation is that, today, children are bigger sooner and therefore girls, on average, reach the mean weight at menarche (46–47 kg) in the USA and many European populations more quickly. Theoretically, this demographic trend should end when the weight of children of successive cohorts remains the same because of the attainment of maximum nutrition and child care; this has now happened in the USA.

## 6.2 Adipose tissue

The major function of white adipose tissue is the storage of fat, which is a highly efficient energy reserve. It has a high energy content and is hydrophobic – 1 g of adipose tissue may contain about 800 mg of triacylglycerol and about 100 mg of water. In contrast, protein and glycogen not only have a lower energy content per unit weight than fat, but are also much more hydrated. The development of copious stores of fat was probably very important for the evolution of homeothermy in birds and mammals. Homeotherms have a much higher basal metabolic rate and so need more substantial energy reserves than poikilotherms. Additional reserves of adipose tissue are accumulated during pregnancy in most species to help support the development of the foetus during the later stages of pregnancy and, to a lesser extent, to facilitate milk production (Vernon & Flint, 1999).

Postnatal development of adipose tissue occurs in phases: there is a rapid increase in adiposity after birth reaching a transient peak at about 12 months post-partum. Subsequently, adiposity declines to a low at about 4 years of age before increasing slowly at first and then more rapidly during adolescence, achieving adult levels in the late teens in both males and females, although the deposition sites differ. Females accumulate relatively large amounts of subcutaneous adipose tissue on the thighs and buttocks whereas, in males, accumulation occurs predominantly at abdominal and intra-abdominal sites (Vernon & Flint, 1999).

## 6.3 The role of leptin in the control of fertility

Leptin is a 16-kDa protein that is secreted by adipocytes and has an important role in the control of appetite and energy balance. It is a product of the obese (*ob*)

gene and resistance to leptin occurs in obesity. When it is released into the blood it travels to the hypothalamus, where there are leptin receptors involved in the control of appetite and energy expenditure. Low levels of leptin in the blood increase appetite whereas administration of high doses inhibits. It is now believed that leptin is central to the control of body mass. Leptin synthesis is regulated by insulin, glucocorticoids and catecholamines but, most importantly, the concentration of leptin in the blood is proportional to the amount of fat in the body and so it could act as a 'lipostat' (or adipostat), matching appetite to adiposity. Leptin not only modulates appetite, it also stimulates thermogenesis in brown adipose tissue, modulates changes in secretion of several pituitary hormones during fasting and is important for normal reproductive function. This makes good physiological sense teleologically because it ensures that females do not become pregnant unless they have adequate reserves of adipose tissue lipid to support a growing foetus.

Ahima *et al.* (1996) have shown that the level of plasma leptin falls rapidly in starved mice and this is accompanied by a range of neuroendocrine changes, including switching off reproductive function. When leptin is injected into these mice, the neuroendocrine and reproductive effects of starvation are blunted. These experiments confirm that leptin could be the signal that is used by the brain to modulate reproductive function according to nutritional state – a suggestion that was originally advanced by Frisch (1980). In practice, the body-mass index (BMI) is used clinically to assess potential fertility: over 27 BMI (obese), greatly reduced fertility; 25–27 (overweight), reduced fertility; 20–25, normal fertility; 18–19 (underweight), reduced fertility; less than 17 (seriously underweight), fertility essentially zero (R.E. Frisch, personal communication). Thus, fertility is reduced in both under- and overweight females.

In practice, leptin circulates as a monomer in plasma; its crystal structure indicates that it is a member of the cytokine family and has four or possibly five helical segments. The receptor-binding domains of leptin that are predicted by its structure are consistent with studies of the activity of mutant forms of the protein. It circulates in a free form and is also bound to other proteins. These plasma leptin-binding proteins have not yet been identified, but are likely to include a secreted form of the leptin receptor (Friedman, 1998).

Leptins may have other subtle and far-reaching demographic effects. Birth weight has been found to be a determinant of blood leptin concentration in the mother. Since nutrition during pregnancy can affect the birth weight of the neonate, Ekert *et al.* (2000) suggested that food intake during pregnancy alters the leptin expression in the progeny. Leptin mRNA was measured in subcutaneous adipose tissue and leptin protein was measured in blood plasma from 59-day-old female pigs whose mothers were fed at the same restricted rate, except that half were permitted to consume 35% more feed during the second quarter of pregnancy. Leptin mRNA abundance in adipose tissue ($P = 0.015$) and plasma leptin concentration ($P = 0.01$) were higher in progeny from mothers

provided with more feed. Body weight at birth was negatively correlated with the abundance of leptin mRNA in subcutaneous fat at 59 days of age ($P = 0.01$). These results demonstrate that maternal nutrition during pregnancy programmes postnatal leptin expression in offspring.

Leptin is a member of the cytokine family of hormones that regulate the immune response. The T-cells of the immune system have leptin receptors on their surface and if the amount of stored fat falls below a critical level, the level of circulating leptin also drops to a point where the T-cells cannot operate. Leptin is also capable of inducing the expression and secretion of interleukin 1 receptor antagonist by human monocytes, indicating that it may have immunomodulatory functions *in vivo* (Gabay *et al.*, 2001). It is suggested that this is the reason that malnourished people are more susceptible to infection (see section 15.4).

In summary, the body fat content is regulated by a negative feedback loop centred on the hypothalamus. Leptin, the protein product of the *ob* gene, functions as an afferent signal in this system: increasing leptin levels result in a negative energy balance whereas decreasing levels lead to a positive energy balance.

## 6.4 Menarche

The average age at menarche in the contemporary Western world is about 13 years. Averages in developing countries are typically higher and vary substantially – for example, 12.4 in Cuba, 13 to 14 in India, 13.4 in Sri Lanka, 15.0 among the South African Bantus, 15.7 in Bangladesh, and 18.8 among the Bundi in New Guinea. Estimates for nineteenth century European populations are also high – around 16 years (Bongaarts, 1980). It is generally believed that nutrition has an important effect in determining the age at menarche and so modulates fertility and Bongaarts (1980) summarises the evidence as follows:

(1) A direct link between nutritional intake and age at menarche was found in a US study in which well-nourished girls reached menarche 2 years earlier than undernourished girls. Similarly, an Indian study concluded that girls whose diets were higher in calories and proteins had an earlier menarche.
(2) Nutritional status, as measured by anthropometric indicators, is correlated with age at menarche. A number of reports from different societies has established that the probability of reaching menarche by a given age is positively related to body size and weight. Frisch (1999) has predicted a minimum weight for height necessary for the onset of menarche.
(3) In Western societies, where there are relatively reliable historical data, a reduction in the age of menarche of about 3 years has taken place since the end of the nineteenth century. This decline is believed to be associated with an increase in body size and an improved diet.

(4) Socioeconomic status and age at menarche are negatively related in a number of countries. Differences ranging from a few months to about 2 years have been found between urban and rural populations and between high and low income groups.

However, Bongaarts (1980) has sounded a warning lest we translate these interesting physiological findings into demographic theory. Age at menarche signals the beginning of potential childbearing, but actual reproduction normally starts at marriage. The mean age at marriage is almost always higher than the mean age at menarche, the difference ranging from about 2 years in some traditional societies to more than 10 years in a number of contemporary European populations. In populations where the mean age at marriage is near 20 or higher, one cannot expect a fertility effect of variation in age at menarche driven by nutritional standards.

This conclusion can be demonstrated by considering a hypothetical, but not atypical, poorly nourished population, where the mean age at menarche is 15 years. It is assumed, first, that a large improvement in nutrition can lower menarche to 13 years and, second, that marriage is very early and that there is a significant correlation between menarche and marriage, so that the mean age at marriage will also decrease – say from 17 to 16 years. This 1-year reduction in age at marriage will lengthen the actual reproductive lifespan by about the same amount, thus increasing completed fertility. With a marital fertility rate of 250 per 1000 for 16-year-old women, this additional time would add 0.25 births per woman on average. Since average completed fertility is typically six or seven births per woman, this implies a fertility increase of about 4%. Clearly, substantial changes in the age at menarche following improvements in nutrition can be expected to raise fertility by, at most, a small percentage.

## 6.5 Is leptin needed for the initiation of puberty?

It has now been established that, in addition to its role in the control of adipose tissue mass (and hence in the regulation of fertility), leptin is needed for the initiation of puberty and the establishment of secondary sexual characteristics. Clément *et al.* (1998) describe a large consanguineous family of Kabilian origin, in which three morbidly obese sisters are homozygous for a mutation in the leptin receptor. This mutation leads to a truncated form of the receptor with no signalling function, and it affects all isoforms of the receptor. This is the first description of a human genetic defect at this locus. In two of the affected sisters (ages 13.5 and 19 years) there were no signs of pubertal development, and the endocrine measurements were again consistent with a hypothalamic defect.

Strobel *et al.* (1998) describe a Turkish family in which a homozygous mutation in the leptin gene results in low plasma leptin levels and morbid obesity in three

affected members, two of whom are adults. The 22-year-old male had clearly failed to go through puberty, and endocrine measurements indicated impaired hypothalamic activation of the pituitary gonadotroph. Little information is available on the 34-year-old female, other than that she has never menstruated.

We conclude that leptin has two important roles in relation to nutrition that can contribute to the regulation of the demography of a population: (i) the control of the body fat content and (ii) the initiation of puberty. The foregoing also suggests that the major effect of an adequate nutrition on a woman's fertility is via the establishment of a suitable level of fat reserves, but micronutrient deficiency can also have negative effects. In particular, zinc and selenium deficiency in both sexes is common in infertility problems today. Folic acid supplementation has also been found to increase fertility.

## 6.6 Nutrition and fertility in the twentieth century

There is general agreement that severe malnutrition has an effect on reproductive capacity and that famines cause a reduction in birth rates (Bongaarts, 1980) and yet good evidence concerning the effect of nutrition on human reproduction is hard to find (Keys *et al.*, 1950). It has been suggested that chronic malnutrition in twentieth century populations is not a limiting factor on fertility; fertility rates among humans depend in the first instance on the proportion of fecundable women, second on their opportunities for mating and, ultimately, on social norms expressed in the decisions of couples about birth control and family size.

However, Malcolm (1970), in a study of the Bundi, an upland tribe in New Guinea living under conditions of poor nutrition, compared their mean growth curves with those of British children. The Bundi diet consisted almost entirely of starch. His curves show the following features:

(1) The British boys and girls grew much faster than the Bundi children.
(2) They reached sexual maturity about 5 years earlier. The Bundi girls in fact did not begin to menstruate till their mean age was between 18 and 19 years.
(3) The British children reached their maximum height and weight some time before the Bundi children.
(4) The mean heights and weights of Bundi of the same age were then considerably below those of the British, but the former went on growing for some years, until they were about 24 years of age. They were, however, still below the British in height and weight when growth ceased.

Riley (1994) also concluded that in developing countries in the twentieth century (and he has worked in Bangladesh in particular), the situation is very different from industrialised nations enjoying a satisfactory diet. First, early marriage and childbearing are desired and common across most segments of

society. Second, malnutrition is widespread and is sufficiently severe to delay the adolescent growth spurt and to raise the average age at menarche by two to three years compared with populations in developed countries. This is certainly the case in Bangladesh. In this setting, he makes several observations regarding the relationship of nutritional status, adolescent development and reproduction. First, undernutrition delays growth and reproductive maturation, and women who mature early (i.e. women of young age at menarche) marry at younger ages than later maturers. In addition, body weight appears to have an independent effect on age at marriage (irrespective of the age at menarche), such that relatively heavy women marry at younger ages than their lighter counterparts. Explanations for this finding include correlation between body weight and the development of secondary sex characteristics and, perhaps, a cultural perception that heavier (i.e. normal body weight) women are more attractive. As we have seen, they would have a higher fertility.

Although chronic malnutrition can have demographic effects, operating via depressed fertility and fecundity, these physiological effects will be detected more readily and have greater importance in situations of extreme famine. We have described the hunger winter in the Netherlands during World War II in more detail in section 4.5; Stein and Susser (1975) conclude that nutritional deprivation of sufficient severity causes infertility in humans.

However, it must be emphasised that the effects of malnutrition on fertility are much more subtle than these studies of famines suggest, even in the twenty-first century. A recent conference report shows that women suffering from fertility problems are twice as likely to conceive if they take a multivitamin pill each day. In the research, 215 women attending an *in vitro* fertilisation clinic in Leeds, UK, completed a questionnaire about general diet, lifestyle and the use of vitamin supplements. Eating fruit had no effect, but women who took a multivitamin pill had a 40% chance of pregnancy compared with only 19% for those not taking any supplements. It is not yet known which of the components of the pills was having this effect (although vitamins C and E are probable candidates), but it is obvious that, even in apparently well-nourished societies today, micronutrients can modulate fertility. In pre-industrial Europe, suffering from a chronic deficiency of many vitamins and minerals, malnutrition would have an impact on fertility levels.

## 6.7 Hutterite women: the upper limit of fertility?

The upper limit of fertility is set by the proportion of all women who are engaged in childbearing, the length of the fertile period in women's lives and the speed at which they produce babies during the fertile period. A population that is maximising fertility will have all of the women married all of the time during their reproductive years, will have early menarche and late menopause, and will have

the shortest possible intervals between births (Howell, 1976). The contemporary Hutterites of Western Canada and the USA (Eaton & Mayer, 1953) come closest to these conditions among contemporary populations. They are a well-nourished, healthy group of people with super-abundant food resources and modern medical care who desire maximally large families for religious and practical reasons. The total fertility rate is defined as the sum of the age-specific fertility rates and represents the number of children who would be born to the average woman who survives through the child-bearing years. The married Hutterite women achieve a total fertility rate of 10.4, the world record. The Hutterite record might be slightly higher if more of their children died in infancy, since the interval to the next birth tends to be shortened when a baby dies. The maximum fertility that can be expected for historic and prehistoric populations must, therefore, be close to 11 children per woman who survives to the end of her childbearing period (Howell, 1976).

## 6.8 Fertility in the bushmen of the Kalahari Desert

Howell (1976) has made a detailed study of the demography of the !Kung-speaking San (or Bushmen) of the Kalahari Desert, a hunting and gathering nomadic people who live in small groups (or who did until the very recent past; see section 1.2). The San arrange marriage for all their young women before the beginning of the reproductive period, and permit remarriage for all women who divorce or who are widowed, usually within a year of the end of the earlier marriage. The !Kung do not practise contraception or abortion, and actively desire to have more children. However, the total fertility rate is close to 5, less than half that of Hutterite women. This difference is explained by Howell (1976) on Frisch's 'critical fatness' hypothesis (described in section 6.1) as follows. The San are extreme by contemporary standards in their small stature and thinness, especially those who still subsist by hunting and gathering rather than depending on cattle raising, like their neighbours, the Tswana and Herero peoples. Birth-weights are normal, but children fall behind European and African standards for height and weight by the age of 2, remaining smaller throughout life. Children are uniformly thin, but lively and apparently well nourished (Trusswell & Hanson, 1968). Cross-sectional height and weight curves indicate that growth continues until the late teens for women and the early twenties for men. No substantial work effort in food production is expected from young people before the ages at which growth is complete. The girls conspicuously build up fat deposits, especially in breasts and buttocks, during their teens, reach menarche at a mean age of 16.5 and bear their first child, on average, around the age of 20. If the child survives, the young woman is necessarily engaged in an activity schedule which demands extra fuel: between lactation, carrying the baby, and providing a substantial share of the increased food requirements of the family, she may need an

extra 1000 calories per day, in addition to her maintenance requirements. If she fails to obtain additional food, she will lose her fat deposits. After two or three years, when the baby's lactational demands decrease, she will be able to rebuild her fat deposits gradually, resuming ovulation when she reaches the critical level and re-entering the risk of pregnancy. The baby will be totally weaned from the breast during the next pregnancy, if not before. If the second child is one of the approximately 25% who fail to survive, the mother will soon be at risk of pregnancy again, having already established the necessary fatness level. Most mothers who have an infant death are pregnant again within a year. If the second baby survives, the cycle of fat loss below the critical level of ovulation and gradual recovery of fat deposits will recur, continuing until menopause. The major factor in reducing San fertility below the levels of the Hutterite women, it is suggested, is the suppression of ovulation through loss of fat deposits caused by the increased activity inherent in caring for a baby.

Howell (1976) concludes that the feature of the environment that determines whether or not the critical fatness mechanism will be operative is not the scarcity of food but its caloric cost to the people. Food is also available out in the bush for groups like the San, but they have to use a large proportion of the energy in the effort to obtain the food and so may be relatively limited in their capacity to build fat deposits, whereas those who live where food supplies are relatively more concentrated and easier to obtain may have an easier time maintaining or rebuilding fat deposits, and therefore have shorter birth intervals.

A well-nourished hunting and gathering group might be modelled on the Yanomama (Neel & Weiss, 1975), who enriched their diet with the banana in the last century and who currently have a total fertility rate of 8, which Howell (1976) suggests is the maximum attainable for prehistoric populations.

## 6.9 Effects of chronic malnutrition on fertility: a case study

We have described in more detail elsewhere our demographic studies of the population at Penrith, Cumbria, UK, during the period 1550–1812 (see Chapters 5, 9 and 10). In brief, this community was living under marginal conditions in which the population was maintained in steady state by density-dependent constraints for over 200 years. The propensity of this population to respond significantly to variations in the price of their staple food suggests that this community is an ideal population to ascertain whether nutrition was an important determinant of the level of fertility. The results are assessed by social class, which is used as an indicator of different economic constraints and hence differences in nutrition.

Of course, it is not possible to assign individuals with certainty to their social class when dealing with populations that were living over 300 years ago. We have made every effort, by using the information that is available, particularly the

designations given in the parish records (baptisms, marriages and burials), to determine the appropriate classification. This assignment to social classes was supplemented by an inspection of 150 wills and inventories, the majority of which were made by the trades and elite groups. From these documents, it was possible to determine the relative wealth of the inhabitants. Categorisation of the social backgrounds of the male head of each household was taken either from the occupations listed for vital events in the parish registers or from that given on a will or inventory. From this information, we have identified three separate social groups: first, the elite of the parish, who included the gentry, substantial landowners, merchants and clergy and the mean value of their inventories at the time of death was £293. Second, the tradesmen, who included blacksmiths, skinners, saddlers, glovers, butchers, shoemakers and tanners; the mean value of the inventories for this group amounted to only £44. These tradespeople were sometimes yeomen who were often compelled to seek other income by employment in a craft or trade, and this integration of by-employments into the household economy was the usual response of a peasant society to the paucity of its economic resources. Finally, the third group covered the remainder of the population, of whom the great majority were subsistence farmers with very small landholdings. These were livestock farmers who had to grow enough grain to feed themselves and their animals.

The family reconstitution study allows the calculation of marital fertility rates for this population in the seventeenth and eighteenth centuries. Only marriages that occurred in Penrith and where the age of the mother is known were analysed. The criterion for the analysis of the age at which a women had her last child was that only those marriages for which there is evidence that both spouses survived until the wife reached 45 years of age were included. The data obtained from the family reconstitution study was used to determine age-specific marital fertility, which is defined as the number of children born in relation to a woman's age and is obtained by dividing the total number of live births by the number of woman-years lived.

The total marital fertility rate for women at Penrith aged both 15 to 49 and 20 to 49 for the different cohorts is shown in Table 6.1. Mean values for the whole period, 1557–1812, were 7.4 and 6.4, respectively.

Age-specific marital fertility rates for the population at Penrith have been calculated and are shown in Fig. 6.1 by lines B (the elite), E (the tradesmen) and G (the subsistence group). For comparison, Fig. 6.1 also shows the fertility rates for other populations:

(1) The Hutterites, see section 6.7 (line A).
(2) The mean of 26 English parishes during the period 1600–1824 (line D), which, it has been suggested, is broadly representative of the marital fertility of pre-industrial England (Wrigley *et al.*, 1997; see section 11.3), although there were no parishes from the northwest of England included in the sample.

**Table 6.1** Age-specific marital fertility at Penrith, Cumbria (per 1000 woman-years lived).

| | Age (years) | | | | | | | Total marital fertility ratio | |
|---|---|---|---|---|---|---|---|---|---|
| | 15–19 | 20–24 | 25–29 | 30–34 | 35–39 | 40–44 | 45–49 | 15–49 years | 20–49 years |
| 1557–99 | 187 | 319 | 312 | 309 | 256 | 77 | 93 | 7.8 | 6.8 |
| 1600–49 | 186 | 270 | 321 | 263 | 201 | 112 | 34 | 6.9 | 6.0 |
| 1650–99 | 196 | 320 | 321 | 230 | 229 | 94 | 14 | 7.0 | 6.0 |
| 1700–49 | 308 | 299 | 317 | 301 | 249 | 164 | 64 | 8.1 | 7.0 |
| 1750–1812 | 211 | 269 | 286 | 272 | 223 | 128 | 35 | 7.1 | 6.1 |
| 1557–1812 | 211 | 291 | 307 | 275 | 230 | 114 | 45 | 7.4 | 6.4 |

(3) The mean of four Waldeck parishes in Germany for 1662–1849 (Wilson, 1984) (line C).
(4) Nepal in 1977 (Wilson, 1984), the fourth poorest nation in the world today (line F).

The well-nourished, non-contracepting Hutterites clearly stand out from the other populations shown in Fig. 6.1, with a greater fertility rate for women throughout their reproductive span, but especially for those aged below 32 years. In sharp contrast, the level of fertility for the malnourished Third World population of Nepal is considerably lower at all ages, but particularly for women in their twenties and early thirties and, whereas the Hutterites show a peak of fertility at age 23 years, the Nepalese have a peak at the older age of 27 years. The German and English parishes have a similar pattern to each other, with a peak of maximum fertility at the age of 23 years and, although neither approached the high level achieved by the Hutterites, nor did they have the very low levels of fertility that are apparent for the poorly-nourished population of Nepal. All the populations shown in Fig. 6.1 have rates of marital fertility above the age of 40 that approximate to one another and all show a marked and progressive fall in the level thereafter.

Marital fertility for the subsistence group at Penrith (line G) had very similar characteristics to that for Nepal (line F): in addition to low overall fertility, both showed a characteristic period of subfecundity for women in their early twenties and a peak of fertility in the late twenties. The women of the tradesmen group (line E), had a pattern of fertility above the age of 32 years that closely resembled the English parishes (line D), but the differences between these two groups lay in the markedly lower level of fertility for the women at Penrith, particularly for those in their early twenties.

The elites (line B) were clearly different from the other two social groups at Penrith in that their age of maximum fertility was five years later and they had a considerably higher rate of fertility during the ages of 27 to 40 years. Over the age

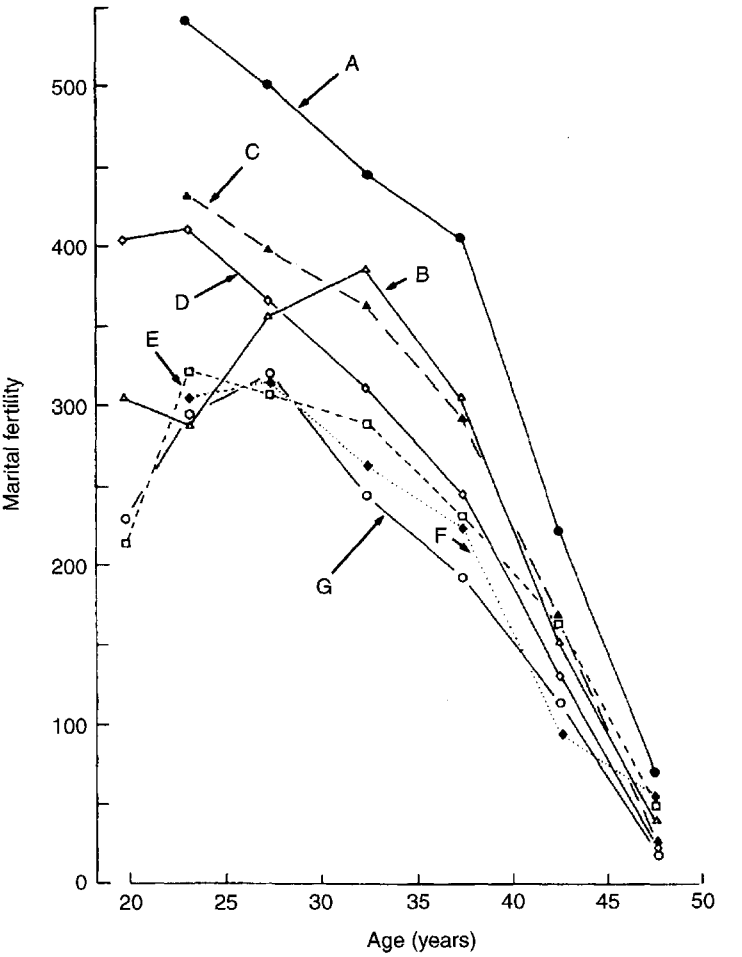


**Fig. 6.1** Age-specific marital fertility rates (children born per thousand women-years lived) for seven populations (ordinate). Line A (closed circles): Hutterites, 1921–1930 (from Wilson, 1983). Line B (open triangles): elites at Penrith, 1600–1812. Line C (closed triangles): average of four Waldeck villages of pre-industrial Germany, 1662–1849 (from Wilson, 1984). Line D (open diamonds): average of 25 reconstituted English parishes, 1600–1824 (from Wrigley *et al.*, 1997). Line E (open squares): tradesmen at Penrith, 1600–1812. Line F (closed diamonds): Nepal, 1977 (from Wilson, 1984). Line G (open circles): subsistence class at Penrith, 1600–1812.

of 30 years, their fertility closely approximated to that for the women of the German parishes but, below the age of 27 years, the elites displayed a subfertility that was characteristic of all the social groups at Penrith and was markedly in contrast with the pattern shown by the Hutterites (line A) and by the English (line D) and German (line C) parishes. Indeed, it is noteworthy that the elites had a fertility level at the age of 23 years that was less than for the tradesmen and was even lower than that of the subsistence group.

It is evident, therefore, that age-specific marital fertility for all social groups at Penrith was low and a characteristic feature was the very clear subfertility in the teen years and in the early twenties. Maximum marital fertility which, for the other populations shown in Fig. 6.1 peaked in the early twenties, occurred at the later age of 27 years for the subsistence group and at the age of 32 years for the elites. Subfecundity in the early part of their reproductive lives is also a feature of the pattern of fertility for the women of Nepal, and this suggests that the majority of the community at Penrith experienced conditions that were indicative of unsatisfactory nutrition and a compromised society.

The mean birth intervals between children for each of the three social groups are shown in Table 6.2. A very similar pattern of intervals between births is evident for the tradesmen and the subsistence farmers: a first child was baptised, on average, 14 to 15 months after marriage, a second child 27 to 28 months after the first-born, and there was a longer interval of 35 to 36 months between the penultimate and the last child. In contrast, elite mothers produced their first child after a longer interval of 18 months after marriage and, after the second child, there was a tendency to shorter intervals between children.

There are significant differences in the number of children produced by the different social groups. Analysis of variance (Table 6.2) shows that the completed family size was significantly different between the social groups; the elite group had a mean of 5.3 baptisms per family, significantly higher than the 4.5 for the tradesmen, which was significantly higher than the 3.8 for the subsistence group. These results may be compared with those found for the historic population of Germany, 1720–50 (Voland, 1990), where the wealthiest group had 5.9 children per completed family and the smallholders (mostly tradespeople) had 4.3. The lowest social group in Germany, the agricultural workers (mostly day labourers), had 4.8 births, which exceeds the completed family size for the comparable group at Penrith, probably reflecting the more severe underlying poverty in northern England at this time.

The age at marriage can have a significant influence on family size; in pre-industrial Europe, marriage age varied from 24 to 29 years and the result was a completed family size of 4 to 6 children. This may be compared with colonial New England where women married between the ages of 20 to 22 years, and had 7 to 9 children (Marcy, 1981). Table 6.2 shows that, at Penrith, the ages at first marriage for each of the social groups were very similar and so this cannot explain the significant differences in their family sizes.

Demographic data for historical populations on the age of women at the birth of their last child show a narrow range of 39 to 41 years (Frank *et al.*, 1994). Comparable values were obtained at Penrith (37 to 40 years); the subsistence women ceased child-bearing two years earlier than the women of the tradesmen group, a difference that is significant (Table 6.2).

Figure 6.2 shows the mean family size for the offspring of each socio-economic group who either stayed in the same class of their fathers or who moved up or

**Table 6.2** Mean age at marriage, family size, age of woman at the birth of her last child and birth intervals between successive infants at Penrith, Cumbria, 1600–1812.

| Social Category | Mean age at marriage | | Mean completed family size | Mean age of women at last birth (years) | Successive birth intervals (months) | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Male (years) | Female (years) | | | M–1 | 1–2 | 2–3 | 3–4 | P–L |
| Elite | 28.6 (75) | 25.1 (73) | 5.3 (120) | 38.9 (54) | 18.7 (57) | 29.2 (61) | 26.6 (58) | 24.4 (55) | 31.8 (52) |
| Tradesmen | 27.5 (291) | 25.4 (276) | 4.5 (451) | 39.5 (176) | 14.8 (169) | 27.8 (171) | 29.8 (167) | 29.9 (159) | 36.3 (139) |
| Subsistence | 27.3 (240) | 25.3 (252) | 3.8 (508) | 37.7 (135) | 14.2 (144) | 27.3 (136) | 29.1 (124) | 28.8 (117) | 35.4 (110) |
| Mean | 27.5 | 25.3 | 4.2 | 38.7 | | | | | |

Sample sizes given in parentheses. No significant differences between the social groups in the mean age at marriage.
Completed family size significantly different in the social groups (analysis of variance; $P < 0.001$) and the difference between the means of all pairs is significant (Scheffe test; $P < 0.05$).
Mean age of women at birth of last child significantly different in the social groups (analysis of variance; $P = 0.026$); Scheffe test shows that only the tradesmen and subsistence groups are significantly different ($P < 0.05$).
Mean birth intervals between successive infants of completed families for each social group. M–1 = mean birth interval between marriage and first birth. P–L = mean birth interval between penultimate and last birth.

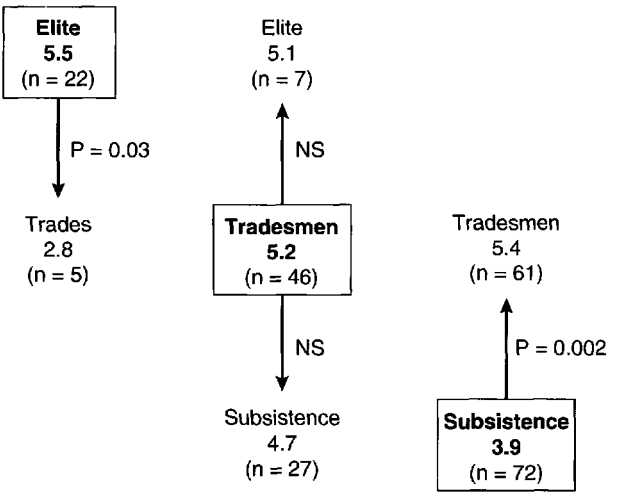


**Fig. 6.2** Mean number of children produced by the offspring (sexes combined) of each social group who remained in the social group of their father (values in boxes) and by those who had moved to another social group at Penrith, 1600–1750.

down the social scale at marriage. The offspring of the elite families who moved down the social hierarchy had the largest fall in family size but, although this effect is significant, the numbers in the sample are small and should be treated with caution. There are no significant changes in the number of children produced for the offspring of the tradesmen group who changed class at marriage. However, the subsistence class showed the most marked change when marrying up into the tradesmen group, and the completed family size rose significantly from 3.9 children to 5.4 children per family, probably because of a better diet and an improved standard of living (see section 9.11).

After 1750, the population escaped from the density-dependent constraints and a population boom began. The analysis of marital fertility at Penrith in this chapter has, so far, concentrated on the differences between the social groups for the period 1600 to 1812, i.e. the steady-state period *and* the boom. We now assess the population as a whole during the steady-state period 1600–1749 (divided into three cohorts as follows: 1600–49, 1650–99 and 1700–49) and a fourth cohort, the boom of 1750–1812, and ask whether there are differences in the level of fertility between the steady-state period and the boom. Is the characteristic subfecundity shown in Fig. 6.1 maintained throughout both periods? Are there differences in the age at marriage, the age at the birth of the last child and the intervals between births?

Figure 6.3 shows the age-specific marital fertility for the four cohorts and it is apparent that the marital fertility rates did not change markedly, either during the steady-state period (1600–1749) or during the boom (1750–1812). Subfecundity showed a slight improvement through the steady-state period to reach a

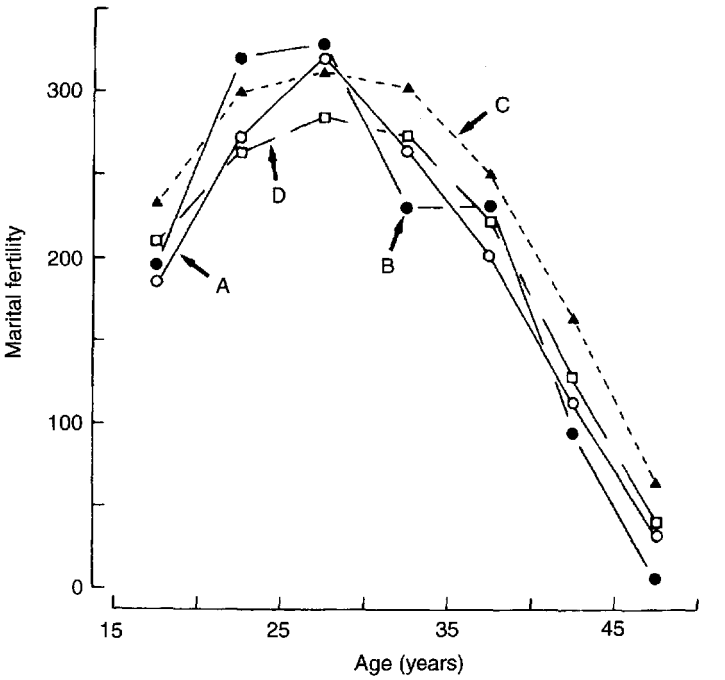


**Fig. 6.3** Age-specific marital fertility (children born per thousand women-years lived) at Penrith in different cohorts (ordinate). A: 1600–49; B: 1650–99; C: 1700–49; D: 1750–1812.

modest peak during 1700–1749, but fell again during 1750–1812. In summary, therefore, marital fertility at Penrith did not rise during the boom and the only period when there appeared to have been an improvement was during the preceding 50 years, i.e. 1700–50.

A reduction in the age at marriage for females is believed to have been one of the main forces behind the growth of population in England in the eighteenth century, falling from a mean of 26 years in the second half of the seventeenth century to 23 years by the beginning of the nineteenth century (Wrigley *et al.*, 1997). However, Table 6.3 shows that there were no changes over time in the ages at first marriage, or in the mean family size of the population at Penrith. There were slight changes in the age of the mother at the birth of her last child, being at a lower age of 38 years for women during the steady-state period and rising to 41 years during the boom (Table 6.3).

The mean birth interval at Penrith varied between 26 and 28 months and the shortest interval is seen during the period 1700–49 (Table 6.3). This may be compared with the reconstitution data for 26 parishes of rural England, where marital fertility was higher; the mean birth interval was 33.3 months in 1640–69, falling to 30.5 months by 1790–1818 (Wrigley *et al.*, 1997).

In summary, all three social groups at Penrith had low marital fertility, small family sizes, a marked subfecundity during the teen years which extended into

**Table 6.3** Demographic characteristics at Penrith by cohort, 1600–1812.

| Cohort | Age of mother at birth of last child (years) | Age at marriage | | Completed family size | Mean birth interval (months) |
|---|---|---|---|---|---|
| | | Male (years) | Female (years) | | |
| 1600–1649 | 38.8 (52) | 28.3 (211) | 24.7 (229) | 4.8 (52) | 27.8 (536) |
| 1650–1699 | 38.1 (67) | 27.8 (187) | 26.2 (228) | 4.1 (77) | 27.3 (422) |
| 1700–1749 | 39.3 (133) | 28.7 (251) | 26.7 (327) | 4.7 (134) | 25.8 (529) |
| 1750–1812 | 41.3 (148) | 27.5 (906) | 26.5 (1017) | 4.1 (138) | 26.1 (620) |

Sample sizes given in brackets.

the twenties and, furthermore, gave birth to their last child at an early age. In populations with natural fertility, age-specific fertility rates typically peaked for women during the ages of 20 to 24 years (Henry, 1961; Woods, 1989) whereas at Penrith this peak occurred at a later age of between 27 and 32 years (Fig. 6.1). Thus, the overall pattern of marital fertility was markedly below that of the well-nourished Hutterites and, although the elite and tradesmen groups after the age of 32 years displayed levels that approximated to the fertility schedules for comparable historic societies (Fig. 6.1), the subsistence group, who formed the largest part of the community, had a fertility rate that closely resembled that shown by a present-day, poorly-nourished population in the Third World.

## 6.10 Procreative power

Procreative power is defined as the variation in the rate of child-bearing with age and the reproductive span of a well-nourished woman who is not using contraception is, on average, longer than that of the undernourished female (Frisch, 1975, 1978, 1982). Figure 6.4 illustrates this point by comparing the level of procreative power for the Hutterite population (line A), who exemplify the highest reliably recorded marital fertility rates, with that for women of mid-nineteenth century England and Scotland (line B), (Frisch, 1978). An early age at menarche for the Hutterites of 12–13 years is followed by a completed physical growth by the age of 16 years, and their best physical fitness for procreation is reached by the end of their teen years, after which there is a steady decline in fertility until the menopause, which occurs in the fifth decade of life. In contrast, the curve representing the procreative power of the women of mid-nineteenth century England and Scotland displays an overall suboptimal level of fertility; menarche was at the later age of 15 to 16 years and there was a longer period of adolescent sterility than for the Hutterites. The best fitness for procreation was during the mid to late twenties and there was a steeper decline in fertility to an age of last birth at 41 to 42 years, followed by a much earlier age at menopause.

The suggested procreative curve for the population at Penrith is given by line C

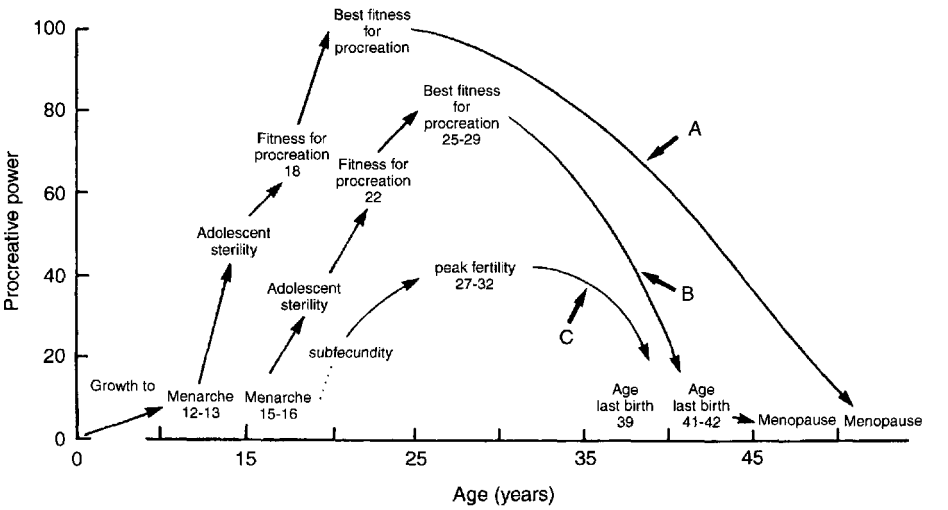


**Fig. 6.4** Variation of procreative power (the rate of childbearing) with age. Line A: Hutterite women, 1930–50. Line B: women in England and Scotland, 1850–70. Line C: approximate curve for women in Penrith, 1600–1812. The maximum procreative power for the well-nourished, non-contracepting Hutterites is 100. Data for lines A and B from Frisch (1978).

in Fig. 6.4. Here, the overall age at the best fitness for procreation has been assumed to be between the ages of 27 to 32 years (data taken from Fig. 6.4), although we know that this peak was different in the social classes and the age of a mother at her last birth was 39 years (see Table 6.2). This postulated curve is well below that for the mid-nineteenth century population in England and Scotland and it confirms that the age of best fitness for procreation was 2 to 3 years later and that the age of mother at her last birth was 2 to 3 years earlier. It is not possible to determine the age at either menopause or menarche, but the long period of adolescent sterility, as shown in Fig. 6.4, would suggest that menarche is unlikely to have occurred earlier than the age of 17 or 18 years.

The earlier and higher level of the peak of fertility for the Hutterites coupled with their longer reproductive span – the consequence of an early menarche and late menopause – has been explained by the effects of improved nutrition (Frisch, 1978) and this population, as we have seen, produced an average of 10 to 12 children per family. Undernutrition, acting via a long period of adolescent subfecundity, reduced fertility for the mid-nineteenth century British population and a mean family size of only six to eight children was the result. A later age at sexual maturity and a shorter reproductive span among the more undernourished women of Penrith meant fewer births and a considerably smaller family size of 4 children (Table 6.2).

In Greystoke, a parish some 5 miles to the west of Penrith, where the scattered community was living under similar marginal conditions, Armstrong (1994) also finds good evidence for teenage marital subfertility during the period 1595–1610.

Fertility was low and declined very rapidly and women, even at their most fertile, were producing fewer than one child every two years; stillbirths were at the high rate of 9% – all features indicative of a population suffering from the effects of chronic malnutrition.

## 6.11 Fertility in pre-industrial England

Wrigley *et al.* (1997) have provided an exhaustive study of marital fertility in England during the period 1600–1824, basing it on the 26 parishes in which a full family reconstitution had been carried out. As we say in section 11.3, these parishes are largely confined to the Midlands and the south of England.

Overall, their findings for the age-specific marital fertility rates for the period 1600–1824 are shown in line D, Fig. 6.1. The mean total marital fertility rate (ages 20 to 49) was 7.4, which compares well to the Yanomama and lies midway between the Hutterites and the San. It is above the value (6.4) estimated for Penrith. Figure 6.5 shows the data from Wrigley *et al.* (1997) plotted for each cohort and it is striking that, from the 20–24 year age group to the age of 49, there is no significant difference in the marital fertility rates over the whole period 1600–1824. This is reflected in the relative constancy of the total marital fertility rates (ages 20 to 49 years) which ranged with no discernible pattern from 6.9 (1650–74) to 7.7 (1800–24). Thus, even in the early nineteenth century the rate had not improved on the value of 7.6 for 1600–24, 200 years earlier. If the nutrition of the mother had improved during this time, it had no detectable effect on fertility above the age of 20 in the Midlands and southern England.

However, Fig. 6.5 shows that teenage marital fertility in the 25 English parishes changed sharply after 1750. From 1600 to 1750, these populations suffered from teenage subfertility, like the populations at Penrith and in present-day Nepal (Fig. 6.1). Unlike Penrith, where teenage subfertility persisted in the early nineteenth century (Fig. 6.3), teenage fertility and age-specific fertility in the parishes of the Midlands and southern England after 1750 (Fig. 6.5) began to approach that of the Hutterites (Fig. 6.1).

We conclude that the improved nutrition after 1750 brought about a marked rise in teenage fertility in the 26 parishes, probably because young women were now able to build up their fat reserves. Since relatively few women married below the age of 20, this would have had only a small demographic impact. In contrast, nutritional levels of young women at Penrith, even in the early nineteenth century, were still inadequate, in spite of a general amelioration in the conditions and a clear improvement in infant mortality (see Fig. 11.3). Indeed, Fig. 6.3 shows that the women at Penrith did not reach their peak fertility in any cohort until after the age of 25 years, suggesting that they built up their fat reserves only slowly.

Wilson (1984) has also carried out an analysis of marital fertility in 14 English parishes, 1600–1799, most of which have been included in the survey of 26

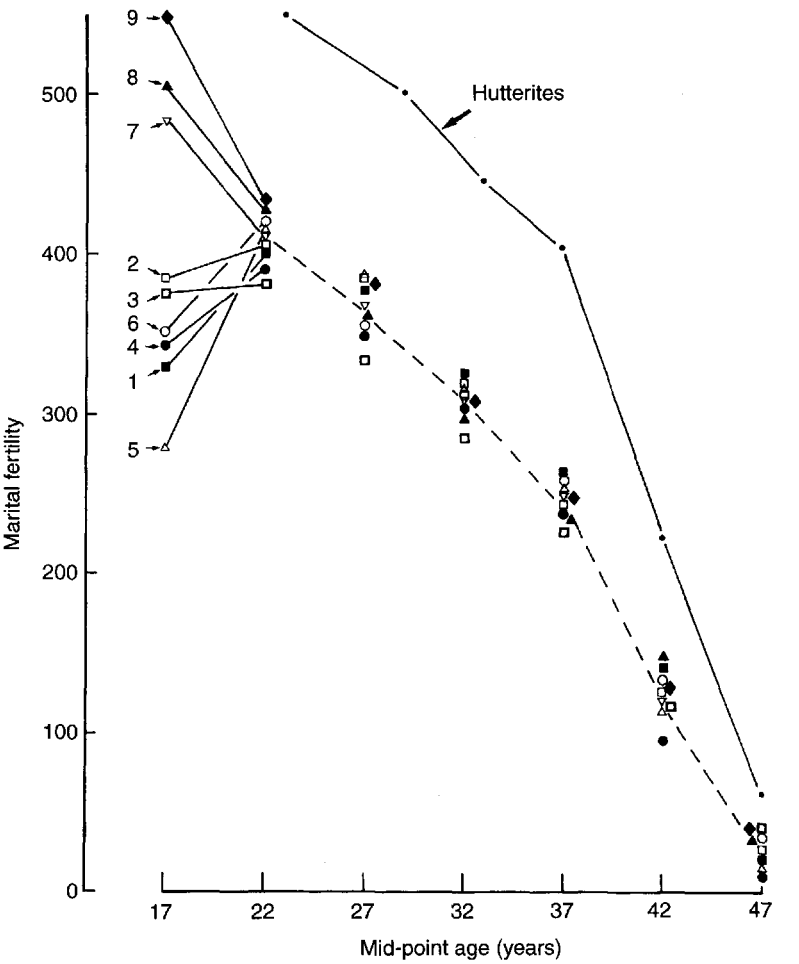


**Fig. 6.5** Age-specific marital fertility rates (per 1000 women-years lived) in the 26 rural parishes of England (ordinate). Abscissa: midpoint of each age group (years). Cohorts: 1, 1600–24; 2, 1625–49; 3, 1650–74; 4, 1675–99; 5, 1700–24; 6, 1725–49; 7, 1750–74; 8, 1775–99; 9, 1800–24. The cohorts differ in age-specific marital fertility only in the 15–19 years age group. There is no significant difference between the lines for the different cohorts after the age of 20 years. Age-specific marital fertility of the Hutterites is shown for comparison. Data from Wrigley *et al.* (1997).

parishes by Wrigley *et al.* (1997). His mean fertility curve shows only a small drop in teenage fertility, with a total marital fertility rate of 7.2, agreeing with the value of 7.4 for the 26 parishes.

Table 6.4 is taken from the data provided by Wilson (1984), where he compares the fertility for his 14 aggregated parishes with that of other communities in Europe in the eighteenth century and populations in Asia in the twentieth century. The total marital fertility rate in the 14 English parishes was, in general,

**Table 6.4** Age-specific fertility rates and total marital fertility ratios (TMFR) for combined set of 14 English parishes and selected historical and contemporary populations.

| Population | Age of mother | | | | | | TMFR |
|---|---|---|---|---|---|---|---|
| | 20–24 | 25–29 | 30–34 | 35–39 | 40–44 | 45–49 | |
| 14 English parishes (1550–1849) | 383 | 350 | 304 | 243 | 134 | 28 | 7.21 |
| Alskog, Sweden (1741–69) | 363 | 318 | 261 | 260 | 114 | 23 | 6.70 |
| 3 Bavarian villages (1648–1849) | 499 | 486 | 449 | 371 | 171 | 28 | 10.02 |
| 4 Waldeck villages (1662–1849) | 433 | 399 | 361 | 287 | 166 | 23 | 8.35 |
| Wardum, Ostfriesland (1662–1849) | 449 | 376 | 322 | 250 | 118 | 17 | 7.66 |
| Thezels-St. Sernin, Quercy (1700–91) | 385 | 335 | 290 | 242 | 67 | — | 6.60 |
| Northwest France (1670–1769) | 447 | 426 | 380 | 293 | 150 | 10 | 8.53 |
| Northeast France (1670–1769) | 515 | 458 | 405 | 323 | 158 | 13 | 9.36 |
| Blankenberghe, Flanders (1650–1849) | 520 | 480 | 431 | 352 | 182 | 19 | 9.92 |
| Crulai, Normandy (1674–1742) | 428 | 431 | 359 | 319 | 119 | 10 | 8.33 |
| Tourouvre au Perche (1667–1714) | 412 | 425 | 378 | 330 | 164 | 11 | 8.60 |
| French Canada (1700–1730) | 509 | 495 | 484 | 410 | 231 | 30 | 10.80 |
| Bengal (mc 1945–6) | 323 | 288 | 282 | 212 | 100 | 33 | 6.19 |
| Punjab (bc 1900–14) | 370 | 357 | 346 | 259 | 113 | — | 7.23 |
| Nepal (1977) | 306 | 314 | 261 | 226 | 93 | 33 | 6.17 |
| Jordan (1978) | 486 | 429 | 357 | 259 | 125 | 55 | 8.56 |
| Kuwait (1969–71) | 453 | 419 | 349 | 253 | 75 | 23 | 7.86 |
| Philippines (1965) | 434 | 388 | 314 | 237 | 110 | 27 | 7.55 |
| Senegal (1967–71) | 389 | 372 | 389 | 231 | 104 | 21 | 7.53 |
| Martinique (bc 1914–28) | 481 | 440 | 333 | 211 | 114 | 11 | 7.95 |

mc, marriage cohort; bc, birth cohort.
*Source:* Wilson (1984).

below that of communities in France and Germany, and the three Bavarian villages (1648–1849) with a rate of 10 stand out. French Canada, also with a rate of 10, is equally conspicuous. Some Asian countries in the twentieth century (Bengal, Nepal) still had a lower fertility rate than that of pre-industrial England, whereas other countries in Africa and Asia, presumably enjoying a better level of nutrition, were showing an improvement.

## 6.12 Breast-feeding, fertility and population growth in the twentieth century

Evidence of a trend toward the abandonment of breast-feeding among the urban poor in a number of less developed countries in the twentieth century has stirred speculation concerning the demographic impact of a massive shift from the breast to the bottle as the primary infant feeding method in populations where the

practice of birth control is far from universal. It is possible that such a change would increase fertility, because of the contraceptive effect of breast-feeding, with the obvious knock-on demographic effects of raised population growth. Prolonged lactation protects against pregnancy, mainly by delaying the return of ovulation. The underlying mechanism is believed to operate through the anovulatory effect of prolactin and other hormones secreted in response to the infant's suckling, and there are now a number of studies that confirm that lactation prolongs the period of post-partum amenorrhea.

The more prolonged, frequent and intense the suckling, the longer the period of amenorrhoea. The ovulation-suppressing effect of lactation appears to be considerably weaker in women who supplement breast-feeding with bottle-feeding, probably because infants on mixed feeding regimes suckle less frequently and less intensely. The nutritional status of the mother may also have an indirect effect because poorly-nourished mothers may produce less satisfactory breast milk and hence more prolonged and intense suckling is required of their infants (Knodel, 1977).

We describe the nutritional and demographic aspects of breast-feeding versus weaning in pre-industrial populations in more detail in Chapter 9. We summarise below various case studies of breast-feeding in the twentieth century.

### *Central Hospital in Hillerød, Denmark, 1986*

In a sample of 361 women, the median duration of amenorrhoea was 17 weeks. A significant correlation was found between the duration of post-partum amenorrhoea and breast-feeding. However, lactation for more than 9 months did not extend the duration of amenorrhoea. Menstruation before weaning occurred in 57% of the women, and 43% terminated breast-feeding before the first menstruation. Four weeks after weaning menstruation had returned in 79%, and after 8 weeks in 93% of the mothers (Vestermark *et al.*, 1994).

### *Hoima district, Uganda, 1992–3*

The introduction of supplementary food by Ugandan women does not replace or substitute for breast-feeding and this study was designed to determine whether lactational amenorrhoea was effective, irrespective of supplementation of the infant's diet. The study began with 154 women, of whom 134 completed the sixth month of the study. At the end of the period, 63% of women were amenorrhoeic, of whom only 39% were exclusively breast-feeding, and no woman had dropped out of the study because of pregnancy or the use of other family planning methods, other than lactational amenorrhoea. The study confirmed that lactational amenorrhoea would be applicable to the majority of the breast-feeding women in Uganda; the return of menses was irrespective of whether supplements had been introduced or their frequency (Ravera *et al.*, 1995).

### *Nomadic society in the late twentieth century*

Measures of suckling frequency and intensity were used to compare the effects of breast-feeding practices on the duration of lactational amenorrhoea and on the length of the birth interval in three prospective studies undertaken during the 1980s, among Quechua Indians of Peru, Turkana nomads of Kenya and the Gainj of Papua New Guinea. In all three societies, lactation is prolonged well into the second year post-partum and frequent, on-demand breast-feeding is the norm. However, the duration of lactational amenorrhoea and the length of birth intervals vary considerably. Breast-feeding patterns among the Gainj and Turkana are similar, but Turkana women resume menses some 3 months earlier than do the Gainj. The average birth interval among the Gainj exceeds that of the nomadic Turkana by over 15 months. Suckling activity decreases significantly with increasing age of nurslings among both the Gainj and Quechua, but not among the Turkana. Earlier resumption of menses among the Turkana women may be linked to the unpredictable demands of the pastoral system, which increase day-to-day variation in the number of periods of on-demand breast-feeding. The short birth intervals of the Turkana women, relative to those of the Gainj, may be related to early supplementation of Turkana nurslings with butterfat and animals' milk, which reduces energetic demands on lactating women who are at risk of a negative energy balance (Gray, 1994).

### *Assam and Eastern Uttar Pradesh, India, 1987–9*

Two retrospective surveys in India investigated the effects of continued breast-feeding on fertility after the resumption of menses, using life table and multivariate time-dependent hazards analyses. Breast-feeding, even after the return of menses, was found to be associated with longer birth intervals. The interaction of breast-feeding duration after resumption of menses and post-partum amenorrhoea had a significant effect on the risk of conception after the return of menses (Nath *et al.*, 1994).

### *Zaire in the 1970s*

Two very different ecological regions in Zaire, the tropical forest and the highlands, were chosen for a retrospective and cross-sectional study of the main components of birth intervals. A well-balanced diet is enjoyed in the tropical forest, but the highlands suffer from a moderate protein energy malnutrition. The populations have certain socio-economic aspects in common, namely an economy dominated by self-subsistence, natural lactation and natural fertility. In rural environments, prolongation of *postpartum* amenorrhoea by 7 to 9 months in lactating women is induced by a moderate chronic malnutrition, characterised by unbalanced supplies of protein and lipid. Birth intervals for the highland popu-

lation are superior to those for the tropical forest population (39 months and 36 months, respectively) because of the length of post-partum amenorrhoea and the absence of post-partum sexual taboos (Carael, 1978).

### *Sub-Saharan Africa at the end of the twentieth century*

These analyses used the data collected in seven Sub-Saharan countries between 1990 and 1994, and the objective was to examine the association between maternal nutritional status and lactational amenorrhoea among breast-feeding women. Women who were not pregnant, who were breast-feeding, who were not using hormonal contraceptives and who had a child less than, or equal to 2 years old at the time of the survey were included in the analyses. Within-country analyses consistently showed the trend of low maternal body mass index associated with a higher likelihood of being amenorrhoeic: women with BMI < 18.5 were more likely to remain amenorrhoeic at the time of the survey than their better-nourished counterparts (see section 6.3). The differential probability of being amenorrhoeic between undernourished and better-nourished women increased with time post-partum and the results suggest that maternal nutritional status plays an independent role in the return of ovulation after delivery (Peng *et al.*, 1998).

## 6.13 The menopause

The menopause marks the end of a woman's fertile period and is defined as the permanent cessation of menstruation resulting from a loss of ovarian follicular activity. It is preceded, in the majority of women, by a period of approximately 12 months of perimenopause when cycles are irregular.

Ginsburg (1991) quotes Aristotle as follows: 'The menstrual discharge ceases in most women about their fortieth year; but with those in whom it goes on longer it lasts even to the fiftieth year, and women of that age have been known to bear children. But beyond that age there is no case on record' and his assessment was similar to those of Hippocrates and also Roman authors. It seems, therefore, that 2000 years ago most women entered the menopause in their early 40s (Amundsen & Diers, 1970). Mediaeval authors, however, gave 50 years as the age when menses ceased (Amundsen & Diers, 1970) very close to the timing for twentieth century women.

In spite of methodological problems, studies show a striking agreement that the median age at the menopause is currently around 50 in Western industrialised societies. In Britain it is 50.8, in the United States 49.8, in Finland 50 and in white South Africans 48.7, with little apparent change over the past century (McKinlay *et al.*, 1972; Ginsburg, 1991; Luoto *et al.*, 1994). Thus, there is little evidence of a significant change in the median age of women in Europe at the start of the menopause between the fifteenth and twentieth centuries.

The few reported estimates from developing countries are much less consistent: a median age of 44 years in India compares with a relatively high mean of 50.7 years among the Bantu in South Africa.

However, malnutrition can bring forward the age of the menopause in the twentieth century and it is reasonable to suppose that it could have had similar effects in pre-industrial England. In the Melanesian population of New Guinea, the median age at the menopause (43.6 years) is lower in women suffering protracted malnutrition with a mean height of only 144.5 cm and mean weight of 40.22 kg. In contrast, the group with a satisfactory nutrition had a median age of menopause of 47.3 years, a mean height of 153.8 cm, and a mean weight of 51.14 kg (Scragg, 1973). Identifiable malnutrition appears to be the important factor for determining early onset menopause because a comparison of three socio-economic urban groups in Karachi, Pakistan (poor slum dwellers, middle-class attendees at a clinic and wives of retired military officers) revealed no significant difference in the mean age (47 years) at menopause (Wasti *et al.*, 1993). Thinner women, particularly with small stature, tend to have an earlier menopause (Van Keep *et al.*, 1979) and in well-nourished women, age at menopause is associated with the percentage of body fat in early adulthood (Sherman *et al.*, 1979; Frisch, 1982; Merry & Holehan, 1994a).

We conclude that the mean age of the start of the menopause has been largely unchanged from the sixteenth to the twentieth centuries in Western Europe. Malnourished women in communities living under marginal conditions may have had their fertile period attenuated by up to 7 years by an advancement of the menopause.

## 6.14 Does malnutrition really affect fecundity?

Frisch (1978) originally advanced the view that nutrition had a direct effect on a woman's reproductive ability, summarising the evidence from historic populations when couples, on average, produced only six or seven children, well below the theoretical maximum. The fact that undernourished human beings and animals are less fecund than well-nourished populations can be regarded as an ecological adaptation to reduced food supplies of the environment, and as being of obvious advantage to the population. It is a less wasteful mechanism than the regulation of overpopulation by mortality. In this chapter, we have described the underlying physiological mechanism whereby malnutrition exerts a subtle, but important, control over fertility, confirming Frisch's original suggestion.

Bongaarts (1980) concedes that malnutrition can impair the function of the human reproductive process; the effect is strongest and most evident in famine and starvation, when both fecundity and fertility are significantly reduced. However, he believes that moderate, chronic malnutrition has only a minor effect on fecundity, and that the resulting decrease in fertility is very small. Among the

fecundity components that he examines, age at menarche and the duration of post-partum amenorrhoea appear to be most affected but, in each case, the effect would make a difference of only a few per cent between the fertility levels of poorly and well-nourished women with caloric intake differences of the order of several hundred calories a day. Breast-feeding is the principal determinant of post-partum amenorrhoea, and unrestricted breast-feeding is associated with lower fertility.

He suggests that other fecundity components, such as age at menopause, prevalence of permanent sterility, regularity of ovulation and quality and quantity of sperm, and the probability of intra-uterine death are even less influenced by chronic malnutrition than are the age at menarche and amenorrhoea. He therefore concludes that a large improvement in the nutrition of mothers in underdeveloped countries would, at most, result in only a slight increase in fertility.

It is now well established that the periconceptual use of folic acid supplements is important during pregnancy and reduces the incidence of neural tube defects, such as spina bifida (see section 7.14). A recent study of 2569 women carried out in Lund, Sweden has shown that the rate of twin births in women who had taken folic acid supplements was 2.8%, almost twice that in the general population where 1.5% of births were twins. Moreover, these children were more likely to be premature, have low birthweights or suffer from cerebral palsy. It is suggested that folic acid may cause a rise in fertility by increasing the probability of multiple ovulation or the implantation of more than one egg.

## 6.15 Overview of the fertility levels in England during a 400-year period

Wilson and Woods (1991) have provided what they describe as a long-term perspective (1551–1975) of fertility in England. They have constructed long time-series of fertility indices and they point out that this is possible only in England where appropriate data sources are available. Fertility in England fluctuated considerably between the sixteenth and nineteenth centuries, whereas marital fertility varied little over this period, so that changes in the levels of nutrition had, overall, relatively little effect on a woman's physiology. Wilson and Woods (1991) conclude that the overwhelming influence on fertility trends came about through variations in the intensity and timing of marriage.

Family reconstitution studies show that there were few marked regional or urban/rural differences in fertility levels in England over a 400-year period. This homogeneity of experience in England is in marked contrast with other European countries, where regional variations in fertility were often considerable (Wilson & Woods, 1991).

The most important point to emerge from these studies is that $I_g$, the index of

marital fertility, remained virtually constant in England for 300 years, 1550–1850, at about 0.66 until it fell progressively after 1875 to a value of 0.24 in 1975.

The reasons for this marked national decline in marital fertility that began after 1870 are unclear, but it seems most probable that it was not because of changing levels of nutrition. Woods (2000) assumes that by the 1890s a majority of couples must have been engaging in some form of behaviour to reduce the number of children born.

Chapter 7

# Nutrition and Pregnancy

Chronic malnutrition has its major demographic effects in both the short and medium terms via the nutrition of the mother during pregnancy. As we shall show, it can even have long-term, intergenerational effects. Any sharp oscillations, either seasonal (see Chapter 13) or in the short waveband (see Chapter 10), in nutritive levels can potentially exacerbate these effects. Furthermore, the results suggest that malnutrition has different effects on the health and survival of the infants, depending on which trimester of pregnancy the mother is exposed to adverse conditions (see Chapter 13).

The long-term importance of the quality of the harvest during the different trimesters of pregnancy has been further demonstrated in a recent study in the parish of Skelleftea, Sweden. Bygren *et al.* (2000) followed a sample of 7572 individuals born between 1805 and 1849 and who were still alive at age 40 and the main outcome measures were overall mortality and mortality from sudden death in the age range 40–70 years. Variations in food availability during their prenatal life were determined from historical sources. The risk of sudden death was almost doubled for those whose mothers were struck by a poor harvest during the early stages of pregnancy, but who experienced a good harvest toward the end. However, almost the same risk was evident for the converse case: a plentiful food supply in early pregnancy followed by a poor harvest towards the end. These authors conclude that a stable maternal access to food during pregnancy is important for the offspring's risk of sudden death from cerebro- and cardiovascular disease as an adult.

These clues concerning the long-term effects of maternal undernutrition during pregnancy have recently been extended by further and more detailed studies of the Dutch winter famine (see section 4.5). There was an increase in placental weight, but not in birthweight, in infants whose maternal nutrition was compromised around conception or in the first trimester of pregnancy. The increase in placental weight is interpreted as being compensatory for the reduction in maternal energy intake. Whereas this suggests that pregnancy undernutrition can stimulate compensatory placental growth, the latter was seen only after first trimester undernutrition, which does not affect infant size at birth (Lumey, 1998). Maternal malnutrition during early gestation was associated with

a higher body mass index and waist circumference in 50-year old women, but not in men. The findings suggest that perturbations of central endocrine regulatory systems established in early gestation may contribute to the development of abdominal obesity in later life (Ravelli *et al.*, 1999).

We have become accustomed to the idea that the major disorders of adult life, such as coronary heart disease, stroke and diabetes, arise through an interaction between influences in our adult lifestyle and a genetically determined susceptibility. Research suggests, however, that growth *in utero* may also play an important role (Barker, 1998). The complex and apparently paradoxical effects of malnutrition of the mother before, during and immediately after pregnancy have been elucidated in a seminal series of studies by Barker and his colleagues (see Barker *et al.*, 1990; Barker & Martyn, 1992; Barker, 1998, 1999a, b). They greatly increased our understanding of the factors contributing to chronic diseases in later life and these workers have presented evidence from British populations that low birthweight at term and, in some cases, low weight at 1 year of age are associated with an increased adult risk of hypertension, coronary heart disease, non insulin-dependent diabetes, and autoimmune thyroid disease. The importance of these findings is that they provide overwhelming evidence that malnutrition at a very early age (*in utero* and in infancy) in Britain in the twentieth century resulted in earlier and more severe adult chronic disease. These studies initially met with scepticism because of numerous obvious confounders (Paneth *et al.*, 1996). However, the same observations have now been made with several data sets, allowing many of the confounders to be controlled (Klebanoff *et al.*, 1999). Equally convincing are the accumulating experimental findings that indicate that nutritional restriction in pregnant animals results in hypertension and insulin resistance in the offspring in later life (Gluckman & Harding, 1997; Langley-Evans *et al.*, 1999).

In brief, many human foetuses, particularly in pre-industrial times, have had to adapt to a limited supply of nutrients and, in so doing, they permanently change their structure and metabolism which can result in a neonate of smaller size and disproportionate morphology and also establish the origins of certain diseases in later life (Osmond & Barker, 2000).

## 7.1 Clues from the geographical distribution of infant mortality rates

A demographic study of the geography of coronary heart disease in England and Wales gave surprising results: standardised mortality ratios are twice as high in the poorer areas of the country and in the lower income groups (Ryle & Russell, 1949). Yet, paradoxically, the steep rise of coronary heart disease in Britain has been shown to be associated with rising prosperity. The explanation came when it was found that the distribution of rates of death from coronary heart disease in

different areas of England and Wales today paralleled the distribution of infant mortality rates in the early twentieth century (Barker & Osmond, 1986). It was concluded that coronary heart disease in the adult may be linked to impaired foetal growth (Barker 1998), a suggestion that was confirmed (see Kramer, 2000) by studies in Norway (Forsdahl, 1977), Finland (Notkola, 1985) and the USA (Buck & Simpson, 1982).

## 7.2 The data series

Barker and his colleagues at Southampton had access to a wonderful data series which began in the early twentieth century in Hertfordshire, where Miss Burnside, the Chief Health Visitor and Lady Inspector of Midwives, established a group of trained nurses to attend women in childbirth. The midwife recorded the birthweight of neonates from 1911 onwards and then informed the local health visitor, who went to the home at intervals throughout infancy and recorded development and illnesses. These visits stopped at the age of 1 year and the records were collated by the county health visitor; the ledgers in which they were kept have survived. After 1923, the visits were continued until the child was 5 years old and the records were maintained until 1945 (Barker, 1998).

The Southampton group then followed the subsequent health records, illnesses and causes of death of these children who were born in the earlier part of the twentieth century. Some were still alive and could be traced and examined. Thus, the Hertfordshire records made it possible to relate an individual's early growth and illness to their health in later life: malnutrition of the foetus not only causes smaller babies and higher neonatal mortality, but also predisposes the adult to serious inter-related illnesses in later life, namely stroke, coronary heart disease, raised blood pressure and diabetes. This is to state the findings bluntly; subsequent work and analysis by Barker and his colleagues have revealed that the effects of malnutrition of the mother during pregnancy are subtle and much more complex.

For example, the mother may suffer throughout her child-bearing years from a steady level of malnutrition to which she may become partially adapted, or she may suffer from regular periods in which her nutrition is inadequate in some way. These fluctuations may be associated with the oscillation in grain prices (Chapter 3) or with the seasonal cycle of the availability of food (i.e. the hungry season; Chapter 13), which would be felt more severely by the poorer members of the population. It is the fluctuating levels of food supply for the mother that can potentially have the most damaging effects on the health of the baby and the subsequent demography of the population. Malnutrition can exert its effects before pregnancy, or when the placenta is being established, or during any of the 9 months of the life of the foetus *in utero*. Its specific effects on development, as we shall see, are different for each of these windows. The renal nephrons, for

example, are laid down during the last trimester of pregnancy, whereas the pancreatic B cells continue to differentiate during infancy; undernutrition at different times in gestation has different effects.

## 7.3 The placenta

Most low birthweight babies (i.e. those most at risk) have a small placenta, the growth of which precedes that of the foetus (Barker *et al.*, 1990; Godfrey *et al.*, 1996). The embryo is composed of two groups of cells in the earliest stages of development; the inner cell mass develops into the embryo proper, whereas the outer cell mass develops into the placenta. The distribution of the cells between these two masses is influenced by nutrition and hormones, as shown by animal experiments. Undernutrition in early pregnancy in sheep leads to placental enlargement, which is probably an adaptation to extract more nutrients. However, this enlargement occurs only if the ewe was well nourished before mating, illustrating the importance of the mother's level of nutrition before conception (Barker, 1998, 1999b).

The abundant macrophage populations present in the endometrium are implicated in the tissue remodelling events and immunological changes necessary for pregnancy. Moderately reduced food consumption can dramatically alter the number of endometrial macrophages and their immunoaccessory function in mice (Hudson *et al.*, 1999) and these workers conclude that endometrial macrophage populations are influenced by nutritional status and this may be mediated through both steroid hormone dependent and independent mechanisms. Nutritionally-induced aberrations in the number or behaviour of endometrial macrophages during early pregnancy could have important implications for the quality of the pre- and peri-implantation environment and the maternal immune response to pregnancy.

Maternal undernutrition generally has limited effects on placental growth in early and late gestation, whereas its effects vary in mid-gestation, causing a reduction or an increase in placental weight in different mammalian species. Good nutrition around the time of conception followed by a restricted diet in mid-pregnancy stimulated placental growth in sheep, whereas mid-pregnancy undernutrition in an already poorly nourished ewe restricted placental growth (DeBarro *et al.*, 1992). These observations on the effect of changing levels of nutrition during pregnancy are consistent with empirical practices in sheep farming, whereby ewes are moved from a rich to a poor pasture after mating. If they are then returned to rich pasture in late pregnancy the lambs are heavier than those whose mothers were on rich pasture throughout (McCabb *et al.*, 1991; Robinson *et al.*, 1994; Barker, 1998). High intakes of carbohydrates by women in mid-pregnancy suppressed placental growth, an effect that was especially marked if followed by a low dairy protein intake in late pregnancy (Godfrey *et al.*, 1996).

However, in contrast, anaemia during pregnancy was found to be associated with increased placental size (Beischer *et al.*, 1970; Godfrey *et al.*, 1991) and life at high altitude or vigorous exercise by the mother in early pregnancy both also increase the volume of the placenta. It is suggested that this placental enlargement may be an adaptive response to a reduced oxygen or nutrient content of the maternal blood. Hypoxia may stimulate blood vessel formation in the developing placenta by increasing the expression of angiogenic growth factors (Barker, 1998).

However, this adaptational expansion of the placenta can exact a long-term price. Blood pressure in adult men fell with increasing birthweight but, at any birthweight, blood pressure *rose* as placental weight increased, i.e. the highest blood pressures were recorded in adults whose mothers had, *in utero*, allocated a greater proportion of their resources to placental development rather than to the growth of the foetus (Barker, 1999b). Furthermore, there is a U-shaped relationship between the death rates from coronary heart disease among men and the ratio of placental weight to birthweight; either a high or a low ratio is associated with increased death rates (Martyn *et al.*, 1996). Other studies have shown that placental enlargement is followed in adult life by impaired glucose tolerance.

An enlarged placenta seems, therefore, to be a general marker of undernutrition of the foetus. There are, however, contradictory studies: raised blood pressure was associated with low placental weight (Campbell *et al.*, 1996) and death from stroke was associated with a low placental weight in relation to head size at birth (Martyn *et al.*, 1996). Other studies have failed to find any associations between blood pressure and placental size (Whincup *et al.*, 1992). One explanation for this is that placental responses to maternal undernutrition vary according to the stage of gestation at which undernutrition occurs and to the nutrition of the mother before pregnancy (Barker, 1998). The effects of maternal malnutrition on placental development are clearly more complex than appears at first sight.

## 7.4 Programming

The studies described above lead to the hypothesis that malnutrition *in utero* causes, in a complex way, permanent changes in the metabolism, morphology and physiology of the embryo and infant, which predispose the adult to specific and serious diseases. During the first 8 weeks after conception the embryo does not increase greatly in size, but the basic form of the baby is laid down in miniature. Thereafter, a period of rapid growth begins and continues until after birth. Different growth rates in different parts of the body mould the form of the baby and the variation in size at birth is essentially determined by the intra-uterine environment rather than the foetal genome (Barker, 1998).

The main feature of foetal growth is cell division. The tissues of the body grow

during periods of rapid cell division and the timing of these differs for different tissues. Growth depends on the availability of nutrients and oxygen, and the main adaptation of the foetus to a lack of these essentials is to slow its rate of cell division, either as a direct effect of undernutrition on the cell or through altered concentrations of growth factors or hormones, of which insulin and growth hormone are particularly important. Even brief periods of undernutrition may permanently reduce the numbers of cells in particular organs and this is one of the mechanisms by which undernutrition may permanently change or 'programme' the body. Many systems in the body seem to be more susceptible to programming during periods when they are growing rapidly, so that their sensitive periods coincide with times of rapid cell replication (Child, 1941; Widdowson & McCance, 1971). In some animals, such as the pig, cell numbers increase most rapidly after birth rather than before, and the animal can therefore largely recover from undernutrition *in utero*. Humans, however, accomplish a greater proportion of their growth before birth than do pigs, and they are more sensitive to the effects of intra-uterine growth failure (Widdowson, 1971). Thus, tissues develop in a predetermined sequence from conception to maturity, with different organs and tissues undergoing periods of rapid cell division and, therefore, passing through sensitive periods at different times. In general, the earlier in the embryonic life that undernutrition occurs, the more likely it is to have permanent effects on body weight and length (Barker, 1998).

## 7.5 Proportionate small size at birth

Some babies are proportionately small at birth; their length, head size and weight are reduced in the same proportion. Such babies are thought to have established a slow trajectory of growth in early gestation and thereby avoided becoming disproportionate. Early slowing of the growth trajectory is a major adaptation to undernutrition because it reduces the subsequent demand for nutrients (Barker, 1999b). Thus, in early intra-uterine life, undernutrition tends to produce small but normally proportioned animals, whereas at later stages of development it leads to selective organ damage (McCance & Widdowson, 1974). During periods of undernutrition those tissues in which maturity is more advanced have a greater priority for growth and may continue to grow at the expense of other tissues (McCance & Widdowson, 1962). The results are similar if growth is restricted by a reduced blood supply.

The timing of the insult is the factor that determines which tissues and systems are damaged, and hence the *disproportion* in the size and function of the neonate. The pattern of disproportion will also be influenced by the relative sensitivity of different organs: growth of the thymus, for example, is markedly influenced by nutrition.

The way in which the body proportions of an animal are modified by under-

nutrition is also related to its growth trajectory. In sheep, the response of the foetus to maternal undernutrition in late pregnancy depends on the growth rate; slow-growing foetuses are unaffected whereas the growth of rapidly growing foetuses ceases abruptly. One possible explanation is that slowly growing foetuses have previously encountered undernutrition in early pregnancy and adapted to it, so protecting them from restricted nutrition in later gestation (Barker, 1998).

It is the neonates who are disproportionate at birth who are most at risk of developing serious illnesses in later life. Specifically, it is thinness at birth, measured by a low ponderal index (birthweight/length$^3$) that is associated with resistance to insulin. A very important point is that the growth trajectory of boys is more rapid than that of girls in early embryonic life and, consequently, boys may be more vulnerable to undernutrition, which may therefore modify sex ratios at birth (Barker, 1998; see Chapter 14).

In brief, plasticity is necessary in early development. The foetus *in utero* experiences continual variations in the supply of nutrients and oxygen and has to adapt to them. Adaptations to environmental change in adult life are generally reversible, whereas in foetal life they tend to be permanent.

Today, there is a difference between foetal growth rates worldwide which has important demographic consequences: proportionate growth retardation is common in the less industrialised countries, while disproportionate growth retardation prevails in Westernised countries. This distinction is paralleled by the incidence of coronary heart disease, which was rare in Britain before the twentieth century and Barker (1999b) suggests that this illness occurs in Westernised populations where the level of nutrition lies between chronic malnutrition (with early down-regulation of foetal growth) and nutrition at a level that allows adequate foetal nutrition. Pre-industrial England, with its generally impoverished diet, probably more nearly approximated to conditions in Third World countries today and their babies were small-sized (with a high risk of infant mortality), but were probably proportionate with a higher ponderal index and hence were not necessarily predisposed to serious illnesses later in adult life.

## 7.6 The mechanisms that underlie programming of the embryo

The following mechanisms have been suggested to explain how permanent changes can be induced by undernutrition of the mother during pregnancy (Lucas, 1991, 1994):

(1) The nutrient environment may permanently alter gene expression, as has been shown in animal experiments.
(2) Early malnutrition may permanently reduce cell numbers. The small, but normally proportioned rat produced by undernutrition before weaning has

been shown to have fewer cells in its organs and tissues and it has been suggested that the animal does not regain its full size when adequately nourished after weaning. Growth-retarded human babies have reduced numbers of cells in their organs and in some instances this can be directly linked to limitations of function.

(3) Undernutrition in pregnancy changes the balance of, for example, liver cells and lymphocytes.

(4) Undernutrition in pregnancy may produce changes in foetal hormones that are essential for normal foetal growth and development. Growth-retarded babies have an altered endocrine profile, with low insulin, insulin-like growth factor 1 and thyroid-stimulating hormone concentrations and high levels of growth hormone and cortisol.

## 7.7 Maternal–foetal conflict

It has been suggested that the relationship between a mother and her foetus can usefully be viewed as genetic conflict: the effects of natural selection on genes expressed in the foetus may be opposed by the effects of natural selection on genes expressed in the mother. What is best for the foetus need not be best for its mother. The theory of parent–child conflict proposes that children are selected to demand more resources from parents than parents are selected to give. Three sets of genes have different interests: the mother's genes, the genes of the foetus derived from the mother, and the genes of the foetus derived from the father. If the genes of the foetus make excessive demands on the mother, it will prejudice the ability of the mother to pass her genes on to other offspring. It has been argued that genes derived from the father have been selected to take more resources from the mother's tissues than the genes derived from the mother.

Maternal–foetal conflict may be enhanced when animals breed before they are mature. Whereas the hormonal responses to pregnancy in adult women seem to be geared to optimising the flow of nutrients to the foetus, the opposite seems to occur when adolescent girls are pregnant. Paradoxically, feeding young, pregnant, adolescent lambs leads to a selective channelling of nutrients to the mother, who thrives at the expense of the foetus (Barker, 1998).

## 7.8 Foetal adaptations to malnutrition

The supply of nutrients to the foetus depends on the composition and size of the body of the mother, her nutrient stores, what she eats during pregnancy, transport of nutrients to the placenta and transfer across it. Undernourishment follows either when the supply is low (for example when the mother is thin or starving or

when the placenta fails) or when demand is high because the foetus is growing rapidly.

As we have seen (section 7.3), and as has been confirmed by animal experiments, the allocation of cells between the foetus or placenta in the early embryonic stages is influenced by both hormones and nutrition. Cell allocation alters the trajectory of growth that is established around this time and which thereafter tracks through gestation. Better periconceptual nutrition is thought to raise the growth trajectory. A high growth trajectory, established in early gestation when the absolute requirement for nutrients by the foetus is small, leads to an increased demand for nutrients in late gestation, when requirements are relatively large and when the progressive reduction in the ratio of placental to foetal size reduces placental reserve capacity (Barker, 1998).

The human foetus is plastic, able to adapt to undernutrition and its responses include metabolic changes, redistribution of blood flow and changes in the production of foetal and placental hormones which control growth, as summarised in Fig. 7.1. The first response of the foetus to undernutrition is the consumption of its own energy reserves. Prolongation of the period of undernutrition leads to a slowing of the growth rate, so enhancing survival by lowering the metabolic rate and the use of substrates. Slowing of growth in late gestation leads to a disproportion in the size of organs that are growing rapidly at this time. Thus, a variety of different patterns of foetal growth may result in similar size at birth. For example, a foetus that grows slowly throughout gestation may have the same size at birth as a foetus whose growth was arrested for a period and then caught up. However, different patterns of foetal growth will have different effects on the relative size of different organs at birth, even though the overall body sizes may be the same (Barker, 1998).

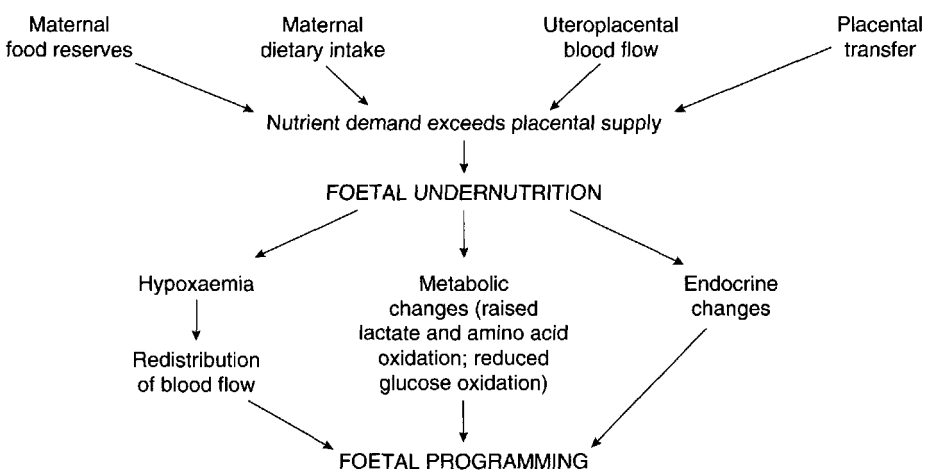


**Fig. 7.1** Diagrammatic summary of foetal adaptations to undernutrition. From Barker (1998).

## 7.9 Overview of the effects of maternal undernutrition on the three stages of gestation

In this section we summarise the differential effects of undernutrition, as given by Barker (1998).

### *Early pregnancy*

Suboptimal nutrition before implantation retards growth and development, the one-cell embryo being particularly sensitive. The early embryo is selective in its use of nutrients and uses amino acids rather than glucose for its respiratory substrate. Before implantation, it switches to a glucose-based metabolism, and low glucose concentrations now retard its growth and development.

### *Mid-pregnancy*

The placenta grows faster than the foetus in mid-pregnancy and nutrient deficiency may therefore affect foetal growth by changing the interaction between the foetus and the placenta. Whereas maternal undernutrition restricts the growth of the foetus and placenta, mild undernutrition may lead to increased placental, but not foetal, size, probably as an adaptation to sustain nutrient supply from the mother. During undernutrition, foetal growth may be sacrificed to maintain placental function, resulting in growth-retarded foetuses and thin neonates.

### *Late pregnancy*

Acute undernutrition results in an immediate slowing of foetal growth, but this rapidly resumes when nutrition is restored. In contrast, prolonged undernutrition may irreversibly slow the rate of foetal growth and so lead to reduced length at birth.

## 7.10 The effects of maternal nutrition on foetal growth and development

The studies on famine conditions (Chapter 4) suggest that even extreme restrictions in food intake of the mother during pregnancy may have only modest effects on birthweight, suggesting that this may depend more on the nutritional status of the mother *before* pregnancy, particularly on the deposition of fat (see section 6.1). This is apparently an important point when considering the demographic effects of malnutrition in pre-industrial England – long-term and persistent malnutrition throughout large sections of the population had more serious demographic consequences than did occasional famines. However, birthweight is a crude measure of foetal growth because, as we have seen, neonates of the same

weight may be proportionate or disproportionate and may be markedly different in organ size, structure, physiology and metabolism.

The mother stores fat in the first half of pregnancy (about 3 kg in an average woman in England today) and mobilises it in the second half under the influence of placental growth hormone and other hormones. In this way she spares glucose for the foetus by switching to fat as her primary energy source. Observations on weight gain in obese women point to the importance of prepregnant weight in determining the birthweight of their babies. Those who gain little weight during pregnancy, or even lose weight, still have babies of average or above average weight. Thus, it is established that a woman who is underweight before becoming pregnant has a higher risk of delivering a low birthweight infant who, in turn, has a greater risk of perinatal death (Bjerre & Bjerre, 1976). This was confirmed in a study of 112 underweight women patients in a hospital in Blackpool, UK. They had significantly higher rates of anaemia, a greater frequency of premature babies and the mean birthweight was 376 g less than that of the infants of the control group (Greiss *et al.*, 1996). Similarly, an increase in the prepregnant weight of 5 kg of women in rural Bangladesh decreases the risk of foetal mortality by 25–33% (Pebley *et al.*, 1985).

In a study in Los Angeles, USA, underweight status before pregnancy nearly doubled the likelihood of delivering preterm, and inadequate weight gain in the third trimester increased the risk by a similar magnitude (Siega-Riz *et al.*, 1996).

A maternal intake of less than 1800 calories per day during pregnancy was associated with babies who weighed 240 g less than those of mothers who ate more than 3000 calories. Low dairy protein intake in late pregnancy is associated with thin (disproportionate) babies, and reduced protein intake in pregnancy is associated with shortness at birth.

The subtle effects of being underweight at birth are illustrated in a well-controlled study of 3500 babies which shows that an increase in one kilogramme in birth weight correlated with a mean rise in the IQ score at age seven years of 4.6 for boys and 2.8 for girls.

Recent studies on rats and sheep have suggested that pregnant women today who consume extra saturated fats in the mistaken belief that they are helping their developing child may be inducing a legacy of ill-health. The cholesterol level in the blood of the baby may be raised, creating an increased risk of heart disease, high blood pressure and diabetes.

Even the frequency of eating during pregnancy can affect preterm delivery. A study carried out in North Carolina, USA, suggests an association between decreased frequency of eating and preterm delivery (Siega-Riz *et al.*, 2001).

## 7.11 Fingerprints

Thus, the original suggestions of Barker and his colleagues that low birthweight infants, particularly those of disproportionate size, were predisposed to, and at

risk of, serious illnesses in later life, including coronary heart disease, hypertension and diabetes, have now been confirmed in an extensive series of studies. The underlying mechanisms between foetal undernutrition and coronary heart disease are suggested in Fig. 7.2 (Barker, 1998).

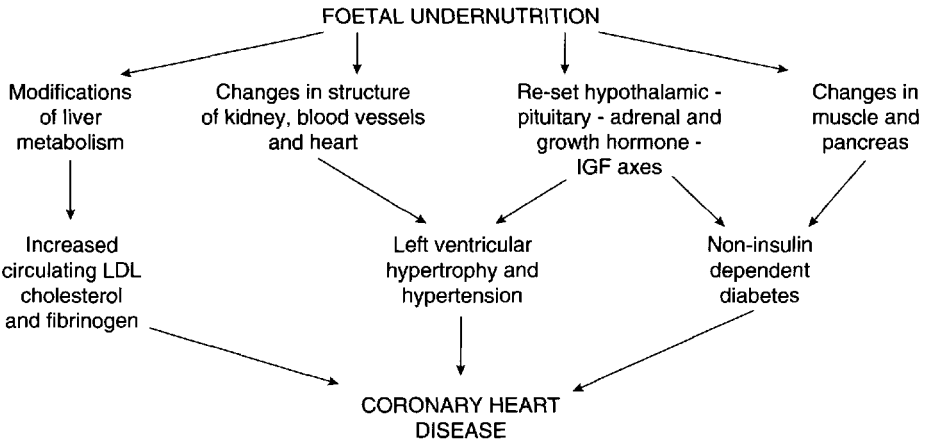


**Fig. 7.2** Diagrammatic summary of the possible mechanisms linking foetal undernutrition and coronary heart disease in adult life. See Fig. 7.1 for the causes of foetal undernutrition. From Barker (1998).

More surprisingly, it has now been shown that fingerprint patterns, which are not genetically determined, are indelible markers of impaired foetal development at different stages in pregnancy (Godfrey *et al.*, 1993). Fingerprint ridge counts remain constant from the nineteenth week of pregnancy (Kahn *et al.*, 2001). People who were thin at birth, with a low ponderal index, had more whorl patterns on their fingers, whereas those who were short at birth in relation to their head circumference had longer hands and a narrower palmar angle. Both fingertip whorls and a narrow palmar angle were significantly associated with raised blood pressure in adult life.

## 7.12 Relationship between foetal growth and adult lung function

Studies in Britain have shown that low rates of foetal growth are followed by reduced lung function in adult life, independent of smoking and social class. It has been suggested that foetal adaptations to undernutrition *in utero* result in permanent changes in lung structure, which in turn lead to chronic airflow obstruction. India today has high rates of intra-uterine growth retardation, but no study has examined the association between foetal growth and adult lung function in Indian people. Size at birth has been related to lung function in an urban

Indian population of 286 men and women aged 38–59 years and born in one hospital in Mysore City, South India, during 1934–1953. Mean forced expiratory volume and mean forced vital capacity fell in both men and women with decreasing birthweight. Stein *et al.* (1997) suggest that malnutrition *in utero* causes a restriction in airway growth in early gestation and that adult lung function is programmed in foetal life. Efficient lung function is particularly important in post-neonates if they are to survive the first year of life.

## 7.13 Maternal diet and the immune function of the offspring

Infant mortality improved slowly during the seventeenth century and the first half of the eighteenth century in England (see section 11.2) but, from about 1630, smallpox emerged as a serious lethal infectious disease of young children and had a major impact on the demography of the communities in towns and cities (see section 15.8). There is evidence that cycles of malnutrition could act as a driver for epidemics and that the general nutritive level could modulate the endemic level of certain lethal diseases (Scott & Duncan, 1998).

Is there evidence that the immune system of children could be modified by the nutritional status of the mother in pregnancy?

Female rats were fed throughout pregnancy on diets that differed in the level of protein and the type of fat they contained. They were transferred at birth to standard laboratory chow. Lower protein intake during pregnancy impaired neonatal thymocyte proliferation by up to 80%; thymic and spleen lymphocyte proliferation also remained impaired at weaning for the offspring of dams fed a lower protein diet. Spleen killer cell activity at weaning was lower following prenatal exposure to the low protein diet. Thus, some aspects of immune function are programmed *in utero* by factors related to the diet of the mother. The effects of the nutrients received during suckling and of the developmental changes in the immune system which occur post-partum appear to be unable to overcome the impact of the diet received *in utero* (Calder & Yaqoob, 2000).

Barker (1998) summarises the studies of the effects of malnutrition on the immune system of human babies as follows:

(1) The thymus seems to be particularly sensitive to foetal and neonatal undernutrition. In the rat, the size and DNA content of the thymus are permanently reduced by transient maternal undernutrition during pregnancy. Undernutrition also impairs the development of thymic-derived T lymphocytes and humoral immunity.

(2) Reduced immune responses in animals can be produced by maternal protein-energy malnutrition or by the deficiency of specific nutrients, such as iron and selenium, in the mother's diet.

(3) Impaired foetal nutrition throughout gestation in humans leads to growth-retarded babies who have low birthweight, global impairment of thymic development and increased susceptibility to infection.

(4) Undernutrition in late gestation may be associated with the diversion of blood and nutrients to the brain at the expense of the trunk. The weight of the thymus may be severely reduced in these babies, who tend to have disproportionately small body size at birth in relation to head size.

(5) Middle-aged men and women who had disproportionately small body size at birth have persistently raised serum IgE concentrations, possibly because of an increase in the numbers of thymic derived TH-2 lymphocytes in relation to TH-1 lymphocytes.

(6) Men and women born during the hunger months in the Gambia were found to have increased death rates, usually from infection, after the age of 15 years. This was attributed to reduced immune competence as a result of undernutrition in mid-gestation.

(7) Most importantly, undernutrition in one generation may affect immunity in the next two generations (Chandra, 1975).

## 7.14 Micronutrients and foetal growth

It is well known that certain vitamins and minerals play a key role in foetal growth and differentiation, in addition to their general functions in regulating the biochemistry during childhood and adult life. Several micronutrients interact in their effects in modulating cell differentiation. Even before clinical symptoms are apparent, marginal deficiencies may be manifest as impaired fertility, or reduced foetal and neonatal viability, all with important demographic consequences.

During the process of cell growth, DNA is transcribed to RNA, which is then translated to proteins which provide the enzymes and structures of the cells. Micronutrients are essential at every stage in the process, either as signals (e.g. retinoic acid), or structural (e.g. zinc in transcription factors) or catalytic (e.g. copper) elements. As we have seen, different organs develop at different times in pregnancy so that there are critical periods when particular organs are more susceptible to alterations in the supply of micronutrients.

In summary, cell proliferation is most affected by deficiencies of vitamins A and $B_{12}$, folic acid, copper and zinc. Subsequent organ differentiation is most affected by micronutrient deficiencies as follows:

- Muscle: iron, copper, zinc
- Eye: vitamin A, zinc
- Bone: vitamins D and K, copper, zinc

- Heart: vitamin E, iron, copper, zinc
- Kidney: vitamin A, zinc

(McArdle & Ashworth, 1999).

The important micronutrients for embryonic development and hence in the maternal diet in pregnancy are evident from this summary and we can now highlight features of certain key vitamins and minerals (see McArdle & Ashworth, 1999):

(1) *Vitamin A* Animal models have shown that severe vitamin A deficiency leads to placental dysfunction, foetal loss and congenital malformations. Recent studies, using more modest ($\approx$ 50%) reductions in maternal retinol concentrations during late pregnancy in the rat, have shown reductions (by about 50%) in neonatal survival and in the relative weights of foetal lungs, liver and heart. Vitamin A deficiency during a woman's pregnancy has particularly serious effects on foetal lung development; damage caused to the lungs by such deficiency can be irreversible, affecting lung function throughout adult life. Such compromised lung function has particularly serious consequences during post-neonatal life.

(2) *Vitamin E* Deficiency during pregnancy in experimental animals results in malformed embryos and foetal death but, in a study of 289 women, no relationship was found between birthweight and serum vitamin E concentrations at 18 and 30 weeks of gestation.

(3) *Folic acid* Because of its role in nucleic acid synthesis, the need for folate increases during the rapid tissue growth in pregnancy. It is now well established that periconceptual use of folate-containing vitamin and mineral supplements reduces the incidence of neural tube defects. The beneficial effects of folate are not confined to events in early pregnancy. Marginal levels of maternal folate throughout gestation impair cellular growth and replication, and mothers with low folate levels at 28 weeks of gestation have an approximately 2-fold greater risk of preterm delivery and low infant weight at birth.

(4) *Copper* This element is essential for proper development *in utero* and maternal deficiency increases the risk of miscarriage. Severe copper deficiency causes foetal resorption while milder deficiencies result in skin, neuronal and hair abnormalities, presumably as a result of reduced cuproenzyme activity.

(5) *Zinc* This mineral is a central part of more than 300 enzymes and proteins (Solomons, 1999) and, consequently, maternal deficiency results in a wide spectrum of extremely severe problems, exacerbated by the fact that the first symptom is loss of appetite. Zinc deficiency is teratogenic and even

marginal changes can cause long-term problems. For example, mild deficiency during pregnancy in monkeys causes immunodeficiency in the offspring which does not improve post-natally. Deficiency also causes complications of labour, premature rupture of membranes and an increased risk of pre-term delivery.

It was generally thought that oxygen levels rise gently throughout pregnancy, but monitoring of the blood entering the umbilical cord revealed that there were very low oxygen levels in the placenta during the first trimester, but that these tripled between the eighth and fifteenth weeks of pregnancy. This change is a relatively sudden and stressful event for the foetus and, if combined with any additional factors, may be sufficient to cause a spontaneous miscarriage. Cells called cytotrophoblasts, which anchor the placenta in the womb and invade the blood vessels to limit the oxygen supply to the foetus, dissipate at around 8 to 10 weeks, so allowing more oxygen in. It has been suggested that if the mother takes antioxidants, such as vitamins C and E or β carotene, it might protect the foetus at this critical time.

## 7.15 Supplementation of the maternal diet during pregnancy

In the light of the foregoing, considerable efforts today are directed at supplementing the diet of pregnant women in Third World countries, with the aim of improving birthweight and hence perinatal mortality, which would have obvious social and demographic advantages. Chronic maternal undernutrition in developing countries is a prime contributor to the birth of over 25 million low birthweight babies annually and to high rates of neonatal mortality. Research conducted in the Gambia suggests, however, that food supplementation exerts a significant effect on infant health when administered to at-risk women during late pregnancy, whereas supplementation during lactation is not as effective (Prentice, 1991a). It is argued that supplementation of lactating women would not be a logical use of scarce resources because this practice is most unlikely to increase the output of breast milk or to improve its composition significantly.

This study in rural Gambia was later extended with a 5-year randomised controlled trial. A diet chronically marginal for many nutrients is exacerbated by a hungry season during the wet weather from June to October, when food stocks from the previous harvest are depleted. These food shortages are compounded by arduous farm work at this time, resulting in rapid weight loss averaging 3–6 kg in adults. These periods of negative energy balance in pregnant women are associated with a decrease in average birthweight and an increase in the proportion of low birthweight babies. Supplementation was started as close as possible to 20 weeks after conception, as judged by clinical examination. The supplement biscuits contained roasted groundnuts, rice flour, sugar and groundnut oil, and they

provided a maximum possible daily intake (two biscuits) of 4250 kJ energy, 22 g protein, 56g fat, 47 mg calcium and 1.8 mg iron.

Supplementation increased weight gain in pregnancy and significantly increased birthweight, particularly during the hungry season ($P < 0.001$). Head circumference of the neonate was significantly increased ($P < 0.01$), but only by 3.1 mm. Supplementation significantly reduced perinatal mortality, both stillbirths ($P < 0.05$) and all deaths in the first week of life ($P < 0.01$). However, it is noteworthy that mortality after 7 days was unaffected. Supplementation was able to reverse the retardation of foetal growth in the hungry season when provided for an average of only 82 days in the second half of pregnancy, suggesting that overall foetal growth is most sensitive to nutritional deprivation in the last trimester (Ceesay *et al.*, 1997). Extrapolating these results to conditions experienced by the bulk of the population in pre-industrial England suggests that the worst predictor for neonatal mortality would have been for the second and third trimesters of pregnancy to fall in the hungry season (see Chapter 13).

Since folate deficiency in pregnancy has been identified as a cause of congenital neural tube defects, a number of studies have concentrated on periconceptual supplementation of this vitamin. These studies confirm that folate supplementation can reduce neural tube defects, but has no other significant effects on pregnancy outcomes (Czeizel *et al.*, 1994; Frelut *et al.*, 1995; Czeizel, 1998).

In contrast, perhaps surprisingly, small weekly doses of vitamin A (7 mg retinol equivalents as retinyl palmitate) or β carotene (42 mg) given to women in the Sarlahi district of Nepal before conception, during pregnancy and through 24 weeks post-partum did not improve foetal or early infant survival (Katz *et al.*, 2000).

## 7.16 Protein nutrition in the perinatal period

Asian babies born at Sorrento Maternity Hospital, Birmingham, UK, have a mean birthweight that is 120 g lighter than the average European baby at the hospital and the incidence of low birthweight babies (12%) is almost twice the national average. The patients come from the poorer parts of Birmingham and their social class distribution is skewed towards the lower groups. Moslem mothers eat meat only if it has been killed in the halal fashion. Hindu mothers from Gujerat are usually vegetarian; they dislike European vegetables and rely mainly on imported ones. Intakes of less than 50 g of protein in a day and less than 1500 kcal were recorded in some mothers.

The following studies add to the anthropometric evidence that Asian mothers of poorly-grown babies have a poorer nutritional status than the Asian mothers of well-grown babies:

- In the mothers of well-grown babies, the triceps skinfold thickness increased from a mean of 16 mm at booking to 19 mm in the third trimester, but there was very little change in the triceps skinfold thickness of mothers of the babies who were light for gestational age. During the second trimester, none of the women who had light for dates infants increased her skinfold thickness at a rate greater than 0.1 mm per week, but this rate of fat accumulation was exceeded by two-thirds of the mothers having well-grown babies. The Asian mother of a poorly-grown baby puts on less fat than the Asian mother of a well grown baby. European mothers of light for gestational age babies behaved quite differently; they were heavier at booking, and became even heavier and fatter as the pregnancy proceeded, almost the reverse of that seen in the Asian mother of the poorly-grown infant.
- The Asian mothers of the poorly-grown babies had a lower plasma valine:glycine ratio and a reduced urinary urea nitrogen:total nitrogen ratio.
- The mothers also had a higher plasma ribonuclease activity. A raised ribonuclease activity occurs in situations where protein intake is insufficient for requirements and its activity is raised in the placentas of malnourished mothers.

It is concluded from these studies of malnutrition of the mother during pregnancy that deficiencies may be of three types:

- Protein and energy deficiency
- Protein deficiency alone in the presence of an adequate energy intake
- Lower quality protein in the presence of an adequate quantity of both protein and energy

From the foregoing, it is suggested that three aspects should be considered:

(1) The possibility of protein energy deficiency in certain pregnant women
(2) Evidence that the rapidly-growing, low birthweight baby requires more of its energy as protein than is commonly given at present and
(3) The importance of protein *quality* when the *quantity* is reduced.

Desai and Hales (1997) have provided a summary of the role of foetal and infant growth in programming metabolism in later life. Foetal growth and development is dependent upon the nutritional, hormonal and metabolic environment provided by the mother and any disturbance can modify early foetal development, with possible long-term outcomes. Growth restriction resulting from a deficit in tissue/organ cell number is irrecoverable. However, when the cell size (or cell protein content) is reduced, the effects on growth may not be permanent.

Epidemiological studies, using archival records of anthropometric measure-

ments related to early growth in humans, have shown strong statistical associations between these indices of early development and diseases in later life, as shown above. It has been hypothesised that the processes explaining these associations involve adaptive changes in foetal organ development in response to maternal and foetal malnutrition. These adaptations may permanently alter adult metabolism in a way that is *beneficial* to survival under continued conditions of malnutrition, as in compromised populations in pre-industrial England and Third World countries today, but *detrimental* when nutrition is abundant.

This hypothesis is being tested in a rat model that involves studying the growth and metabolism in the offspring of rat dams fed a low-protein diet during pregnancy and/or during lactation. It has been demonstrated that there is:

(1) Permanent growth retardation in offspring nursed by dams fed a low-protein diet.
(2) Permanent and selective changes in organ growth. Essential organs like the brain and lungs are relatively protected from a reduction in growth at the expense of visceral organs such as the liver, pancreas, muscle and spleen.
(3) Programming of liver metabolism, as reflected by permanent changes in the activities of key hepatic enzymes of glycolysis and gluconeogenesis in a direction that would potentially bias the liver towards a starved setting.
(4) Deterioration in glucose tolerance with age.
(5) An increase in the lifespan of the offspring exposed to maternal protein restriction only during the lactation period, and a decrease in lifespan when exposed to maternal protein restriction only during gestation.

## 7.17 Stress hormones and pregnancy

Malnutrition triggers an increase in stress hormones in the mother and also disrupts the growth of the placenta so that its enzyme barrier, that normally stops these hormones crossing into the blood of the unborn baby, breaks down. As long as the woman is malnourished, the stress hormones may breach the placenta and upset the development of the baby's organs. Exposing sheep foetuses to a synthetic stress hormone for only 48 hours led to hypertension in the lambs that worsened as they grew older. Rats also are born smaller and have high blood pressure if they are exposed to stress hormones in the uterus.

Low birthweight babies and rats also have high levels of stress hormones as adults, suggesting that the brain may operate in a permanent 'flight or fight' mode, thereby triggering high blood pressure (Schmidt, 1999). The extra cortisol from the mother probably reduces the number of cortisol receptors grown by the foetus so that, after birth, they take much longer to settle down.

Thus, any stressful event, even a relatively mild one, during pregnancy can

cause a marked rise in the level of stress hormones in the mother (Dodic *et al.*, 1999), with serious repercussions for the foetus and neonate. Did the hand-to-mouth existence which the bulk of the population suffered in pre-industrial England cause stress in pregnant mothers in addition to their inadequate diet, so raising still further neonatal mortality and the incidence of low birthweight babies?

## 7.18 Intergenerational effects on foetal development

Barker (1998) has summarised the important studies that show that undernourishment of a female foetus can have long-term, knock-on, intergenerational effects. Women who were small at birth are at twice the risk of having, in turn, babies of low birthweight who are also thin; these infants are found to be more likely to die during the perinatal period (Hackman *et al.*, 1983; Klebanoff *et al.*, 1984, 1989; Carr-Hill *et al.*, 1987; Alberman *et al.*, 1992; Emanuel *et al.*, 1992; Skjaerven *et al.*, 1997). In this way, the original birthweights of mothers have persistent effects and influence the birthweights of their children and of their grandchildren. Barker (1998) concludes that mothers constrain foetal growth and that the degree of constraint they exert is set when they themselves are *in utero* (Ounsted *et al.*, 1986). Maternal constraint is thought to reflect the limited capacity of the mother to deliver nutrients to her foetus. Sisters, who experienced a common level of constraint *in utero*, exert a similar level of constraint on their own foetuses. The mechanisms by which a mother's poor foetal growth impairs the foetal growth of her offspring are not known, but one possibility is that a reduced uterine vasculature is laid down *in utero* and this impairs placentation in the next generation.

The conclusion that the diet of a mother may affect the offspring in several subsequent generations has been confirmed in an undernourished colony of rats with a protein-deficient diet over 12 generations. When they were refed with normal diet it took three generations before foetal growth and development were restored to normal (Stewart *et al.*, 1980).

Hence, the foetus adapts its rate of growth and the lifelong structure and function of its body, not only to its mother, but to the environment its grandmother provided for its mother. Sensitivity to more than one generation allows the foetus to adapt to the level of nutrition which has prevailed over many years rather than only to that at the time of its conception (Barker, 1998). The demographic consequences of this intergenerational effect for populations living under marginal conditions with a generally inadequate nutritional level are clear: these inevitable knock-on effects of low birthweight babies and high neonatal mortality would mean that, even with ameliorating conditions, it would be difficult to escape from the vicious circle of events which constrained population growth (see Chapter 9).

## 7.19 Recommended nutrient intake in pregnant women today

The importance of a balanced and adequate diet of the pregnant mother is evident from the foregoing and Canada, the USA and the UK have each produced recommended guidelines. Only the poorer sections of the population in industrialised nations will be at serious risk, but the diet of mothers in Third World countries will usually be substandard in several important respects (Greeley & King, 1999).

There is general agreement that a pregnant mother in modern Western society needs only an extra 200 calories a day above her usual daily requirement of about 2000 calories. Where women are subsisting regularly under marginal conditions with a lower daily energy intake, as in Third World countries or in pre-industrial England (when a considerable proportion of the energy intake in winter would have been consumed merely in maintaining body temperature), the additional demands of pregnancy may automatically ensure that the foetus is undernourished.

Recommended protein intakes are between 57 and 60 g per day (0.75 g per kg per day), although the Canadian panel advise a progressive rise, 55, 65 and 74 g per day in each trimester.

The essential fatty acids are (i) linoleic acid, with a recommended intake of 1–2% of the total energy intake and (ii) α-linolenic acid, with a recommended intake of 0.2–0.5% of the total energy intake.

Recommended daily intakes of vitamins and minerals are:

*Vitamin A*

700–800 μg retinol equivalents. However, retinol is teratogenic above 3.3 mg per day and pregnant women in industrialised countries are advised not normally to take supplements.

*Vitamin D*

Supplementation is probably not usually necessary with a good basic diet, but intake may be more critical in countries like Scotland, where exposure to sunshine is more limited.

*Vitamin C*

An additional 10 mg per day during pregnancy above the normal daily intake (30–60 mg per day).

*Thiamin*

The requirement increases during pregnancy and is related to energy metabolism, with need ranging from 0.4 to 0.5 g per 1000 kcal.

*Riboflavin*

The extra need for riboflavin placed on the pregnant mother by the foetus is approximately 0.3 mg per day.

*Vitamin $B_6$*

The requirement is related to protein intakes, with a conversion factor of about 15–16 μg per g protein. The national panels differ widely in the recommended additional daily intake, varying from 0 to 0.6 mg per day.

*Folate*

Requirements increase in late pregnancy to support cell division during this period of rapid growth. Bone marrow megaloblastosis was prevalent even in otherwise well-nourished populations prior to the widespread use of supplemental folate. An additional 100 or 200 μg of folate are recommended daily for the pregnant woman.

*Vitamin $B_{12}$*

The maternal body stores are normally sufficient to provide the additional demand of 0.1–0.2 μg per day, but a supplement of about 0.2 μg per day is recommended by the panels.

*Magnesium*

A healthy full-term infant contains about 1 g magnesium at birth; most of this is acquired during the last two trimesters at a rate of approximately 6 mg per day. The US panel estimates an incremental need of 20 mg per day to cover foetal and maternal needs and this value is doubled to 40 mg per day to allow for 50% absorption of dietary magnesium. A total daily intake of 320 mg was recommended.

*Iron*

All panels agree that, except for well-nourished women who begin pregnancy with adequate iron stores, the increased iron requirement for pregnancy cannot be met by habitual diet and supplemental iron is necessary. The recommendations for the additional iron intakes vary from 14 to 30 mg per day. The WHO committee suggested nearly an 8-fold increase in iron between the first and third trimesters.

*Zinc*

The national panels' recommendations vary from 7 to 15 mg per day and the US and Canadian panels recommend a total of 15 mg zinc per day throughout pregnancy, whereas the UK panel recommends only 7 mg per day. The WHO committee suggests that a pregnant woman in the third trimester consumes 13.3 mg zinc per day, which is about twice that recommended for a nonpregnant woman.

We can compare these recommendations for the diet of pregnant women today with the probable diet of the subsistence class in pre-industrial England, as suggested in section 1.9. The major inadequacies during the crucial period of pregnancy (as distinct from the malnutrition of the general population) were probably a suboptimal calorie intake with an unsatisfactory protein/carbohydrate balance (Langley-Evans, 2000) and low levels of vitamins of the B complex (particularly folate), vitamin C, iron and zinc.

## 7.20 Conclusions

The foetus and mother are effectively in conflict, but natural selection has acted on human populations existing under marginal conditions to ensure that as many infants as possible will survive and continue into adult life and reproduce. Establishing the disease patterns of later life (such as heart disease and diabetes) by malnutrition during pregnancy would have had negligible demographic effects on pre-industrial societies.

However, the work of Barker and his colleagues has shown clearly that malnutrition (operating in a complex way) before and during pregnancy results in a high incidence of low birthweight babies and high neonatal mortality. This was probably the most important factor that regulated the demography of pre-industrial societies. Since low birthweight girls grew up to carry, in turn, low birthweight children with a compromised immunity to infectious diseases, irrespective of their nutrition after birth, it is evident that these populations were locked into a vicious loop from which it was difficult to escape.

We have seen how brief periods of malnutrition in different months of the pregnancy can have different deleterious effects on birthweight, disproportionate infants and neonatal mortality. This aspect of seasonal malnutrition is considered further in Chapter 13. The bulk of the population in pre-industrial communities in England was probably existing on a suboptimal diet (section 1.9), but had probably partially acclimated or, indeed, adapted to it. For example, one of the most striking series of current experiments involves feeding rats with diets low in protein but with sufficient carbohydrates. This practice changes cells in the liver, so that those that make glucose are activated, whereas those that store it are shut down. This is a good acclimatory response because it prepares the animal for

living under conditions of food scarcity: the basic strategy is to protect the brain and not to use precious resources on building other organs. The basic evolutionary (and demographic) drive is to live long enough to reproduce. The major exacerbating effects of a qualitatively and quantitatively poor diet would have come during periods of increased nutritional deprivation. These would have come about in two ways: (i) a cycle with a period of 5–6 years, associated with high wheat prices (see section 3.3) and (ii) an annual cycle associated with the hungry season (see Chapter 13). The effect of these periods of deprivation will depend on which trimester in pregnancy suffered the most serious malnutrition.

A striking feature of these findings is that different risk factors are each related to different patterns of early growth. For example, blood pressure is related to birthweight but is independent of weight at one year, whereas plasma fibrinogen concentrations are strongly related to the weight at one year but are unrelated to birthweight. This suggests that the critical period when blood pressure is sensitive to programming is during foetal life not infancy.

Chapter 8

# Infancy

Infancy is defined for our purposes as the period from birth to 12 months of age. Death occurs in the first month of life in neonatal mortality; post-neonatal mortality occurs between the ages of 1 and 12 months.

As we have seen in Chapter 7, malnutrition in pregnancy can result in disproportionate, low birthweight infants who are at risk of neonatal mortality. Even those that survive the trauma of birth and the first week of life are ill equipped to face the vicissitudes of infant life. Neonatal mortality in the past was high in places where many babies were born with low birthweight; it is also known to have been associated with high maternal mortality. High rates of both neonatal and maternal mortalities have been reported in places where the physique and health of women were poor (Barker & Martyn, 1992). The nutrition of the mother during pregnancy is the major determinant of how the infant survives the first month of life and of its health, development and mortality during the post-neonatal period.

Nutrition is also of great importance during infancy. Somatic growth is rapid and will falter in nutritional deficiency; both somatic growth and brain development can be permanently impaired if malnutrition is prolonged. Malnutrition in infancy is relatively common because of the high nutritional requirements of growth and development (Poskitt, 1999). The mother's milk, produced in mammae or mammary glands, is, of course, the source of this infant nutrition. Macadam and Dettwyler (1995) consider that breast-feeding is a biocultural phenomenon: not only is it a biological process, but it is also a culturally determined behaviour and, as such, it has important implications for understanding the past, present and future condition of our species.

## 8.1 Lactation

Lactation is an evolutionary strategy of profound significance because, by this means, mammals can continue to nourish their offspring after birth. The mammary glands are believed to have arisen from the selective modification of apocrine sweat glands (Mepham, 1987). In addition to the wide variation in the

physical characteristics of mammary glands between mammalian species, there is also considerable divergence in the biological strategies adopted for lactation. These are revealed as differences in such variables as the relative duration of gestation and lactation (altricial vs precocial birth), the quantity and composition of milk produced and maternal feeding strategies during lactation. The mammary gland is a highly adaptable organ capable of producing milk over a wide range of composition in different mammalian species; for example, seals produce milk containing up to 500 g fat/kg and negligible milk sugar. It seems reasonable to assume that the composition of milk in any species has evolved to suit that species, and may represent a compromise between the nutritional stress which is sustainable by the mother and the optimal growth and developmental needs of the offspring (Prentice & Prentice, 1995).

Among mammals, humans stand out as producing milk with a very low nutrient density (with the exception of lactose), and as having a very low stress of lactation. It is usually assumed that this lactational strategy has been determined by the very slow growth rate of the human infant (Brody, 1945), which, in turn, has been naturally selected as providing the optimal time for the growth, development and training of a large brain (Martin, 1980). The low stress of lactation per unit time means that the incremental food intake required by a lactating woman is much lower than that for most non-primates. This allows women to adopt a number of different mechanisms to rebalance their energy and nutrient budgets in lactation, and liberates them from the obligate need, incumbent on most other species, to increase food intake markedly (Prentice & Whitehead, 1987).

Although the stress per unit time of lactation is low for humans, the total cost, at least in terms of energy, is high since the mother has to supply the maintenance needs, in addition to the growth needs, of the infant over a long period. A similar analysis can be performed for gestation and yields a similar picture of low stress, but high total costs in women (Prentice & Whitehead, 1987). This is important since the high level of maternal investment in a baby and the very slow human reproductive cycle commit a mother to sustaining each conception, even when nutritional conditions are severely suboptimal. This is particularly true in lactation since the mother has already invested heavily throughout gestation (Prentice & Prentice, 1995). In this chapter, we describe first the nutrients required by infants and what is provided by breast milk in Westernised countries today, before considering the effects of malnutrition on both the mother and her infant.

## 8.2 Nutritional requirements in infancy today

Feeding patterns in the first year alter more than at any other stage of life, reflecting the rapid changes in nutritional requirements and physiological development of an infant. In the first 10 days, the infant's diet is dominated by a

milk, colostrum, which is unique in its content of immunoglobulins and relatively higher in sodium but lower in fat than mature human milk. The transitional period is completed when mature human milk is fed. Inevitably, the milk becomes nutritionally and physiologically inadequate and the next major change occurs in feeding patterns. At this stage, which for the majority of infants is not before 4 months of age, foods other than milk are gradually introduced, initially to supplement milk intake but, eventually, to become the dominant source of nutrients (Morgan, 1999a).

In young infants, the nutrient requirement for growth is substantial, representing up to 30% of the energy requirement, but there exists a large variation within the normal range in the rate of growth and probably also in the composition of the tissue laid down. The average composition of human milk has been used as a basis for estimating requirements, although it varies from mother to mother, and from day to day and it also depends on the stage of lactation and on the mother's diet (particularly the content of polyunsaturated fatty acids). As well as providing nutritional substances, milk contains several growth factors, including epidermal growth factor, although the physiological importance of these substances has not yet been established (Morgan, 1999b).

The energy requirements are conventionally estimated from the observed intakes of healthy infants growing normally today, and are shown in Table 8.1. Table 8.2 shows the reported intake of protein in babies between birth and 4 months. There are few data on the growth and nutrient intake of exclusively breast-fed infants after 4 months of age.

Infants have a requirement for fats to provide energy, to provide essential fatty

**Table 8.1** Calculated energy requirements of infants from birth to 1 year.

| | Median body weight (kg) | | Total requirement (kJ per day) | |
|---|---|---|---|---|
| Age (months) | Boys | Girls | Boys | Girls |
| 0–5 | 3.8 | 3.6 | 1965 | 1860 |
| 1–2 | 4.75 | 4.35 | 2300 | 2115 |
| 2–3 | 5.6 | 5.05 | 2550 | 2280 |
| 3–4 | 6.35 | 5.7 | 2740 | 2470 |
| 4–5 | 7.0 | 6.35 | 2910 | 2635 |
| 5–6 | 7.55 | 6.95 | 3055 | 2800 |
| 6–7 | 8.05 | 7.55 | 3220 | 3010 |
| 7–8 | 8.55 | 7.95 | 3390 | 3140 |
| 8–9 | 9.0 | 8.4 | 3580 | 3350 |
| 9–10 | 9.35 | 8.75 | 3870 | 3620 |
| 10–11 | 9.7 | 9.05 | 4060 | 3790 |
| 11–12 | 10.05 | 9.35 | 4395 | 4080 |

*Source:* Morgan (1999a).

**Table 8.2** Average daily protein requirements for infants (both sexes).

| Age (months) | Average protein requirements (g $kg^{-1}$ body weight) | Intake from breast milk (g $kg^{-1}$ body weight) |
|---|---|---|
| 1–2 | 2.25 | 1.93 |
| 2–3 | 1.82 | 1.74 |
| 3–4 | 1.47 | 1.49 |
| 4–5 | 1.34 | — |
| 5–6 | 1.30 | — |
| 6–9 | 1.25 | — |
| 9–12 | 1.15 | — |

*Source:* Morgan (1999a).

acids, to facilitate the absorption of fat-soluble vitamins A, E and D and as a precursor of structural lipids and eicosanoids. Triacylglycerols in human milk contain more than 150 different fatty acids, but the origin and role of many of these is largely unknown. The average intake of fat is shown in Table 8.3. Infants have a specific requirement for long-chain polyunsaturated fatty acids and certain of them are suggested to have a specific role in visual development. Eicosapentaenoic and docosapentaenoic acids are found in high concentration in human milk.

**Table 8.3** Average daily intake of fat by breast-fed infants aged 0–4 months.

| Age | Breast milk consumed | Weight | Average fat intake | |
|---|---|---|---|---|
| (months) | (ml) | (kg) | (g) | (g $kg^{-1}$) |
| Boys | | | | |
| 0–1 | 719 | 3.8 | 30 | 8 |
| 1–2 | 795 | 4.75 | 33 | 7 |
| 2–3 | 848 | 5.6 | 36 | 6 |
| 3–4 | 822 | 6.35 | 35 | 5 |
| Girls | | | | |
| 0–1 | 661 | 3.6 | 28 | 8 |
| 1–2 | 731 | 4.35 | 30 | 7 |
| 2–3 | 780 | 5.05 | 32 | 6 |
| 3–4 | 756 | 5.7 | 32 | 6 |

Fat content of breast milk 4.2 g per 100 ml.
*Source:* Morgan (1999a).

The fat requirements of children in developing countries today are probably similar to those of children in affluent nations, except for the additional needs imposed by environmental stresses, particularly recurrent infections. The outstanding feature in Gambia is the great importance of breast milk as the source of fat and essential fatty acids until the end of the second year of life. Weaning (and

adult) foods contain low amounts of fat, so that when children are weaned from the breast there is a sharp transition from adequate to inadequate fat intakes (Prentice & Paul, 2000).

Eighty per cent of the total carbohydrate in human milk is lactose (approximately 7 g per 100 ml); in early infancy, when the diet consists wholly of milk, 37% of the energy intake is derived from this source. As the diet of the infant becomes more varied, the carbohydrate content also changes (Morgan, 1999b).

The requirements of the infant for vitamins and minerals can be briefly summarised as follows

- *Vitamin D* Only small amounts are secreted in human breast milk and it is assumed that the major source of this vitamin is the action of ultraviolet light on the skin. Infants in malnourished communities probably did not suffer from a deficiency of vitamin D.
- *Vitamin A* Breast milk today contains 40–70 μg of retinol and 20–40 μg of carotenoids per 100 ml milk. Assuming milk is consumed at 750 ml per day, the infant would receive about 300 μg retinol per day. In developing countries, vitamin A deficiency is uncommon in the first year of life. A drastic fall in the vitamin A content of milk has to be preceded by the exhaustion of the mother's stores of this vitamin.
- *Vitamin C* Human milk contains approximately 4 mg of vitamin C per 100 ml. An infant receiving 750 ml of human milk per day will receive in the order of 30 mg of vitamin C.
- *Iron* A normal healthy foetus stores iron in the last trimester of pregnancy. Because of this, a full-term infant can maintain satisfactory haemoglobin levels, without any other sources of iron, for the first 3 months of life. Breast milk is noticeably low in iron (76 μg per 100 ml). However, because the iron is well absorbed, and because an infant has hepatic haemoglobin stores, a dietary source is unnecessary. After 3 months of age, and in non-breast-fed infants from birth, an iron intake of 1 mg per kg of body weight per day is considered necessary.

## 8.3 Nutritional value of breast milk

Human milk resembles living tissue such as blood, whereas milk formula is an inert multinutrient medium. Human milk contains live cells, a wide range of biologically active factors, a large number of hormones and growth factors and at least 60 enzymes. Its composition is not uniform and changes during the course of lactation, and even throughout a single feed, showing a diurnal variation with differences from one woman to another.

The greatest change in composition, however, occurs during the first 10 days post-partum when colostrum changes in composition to mature milk. Colostrum

contains a much higher concentration of protein compared with mature breast milk; it has half the concentration of fat but higher amounts of sodium and vitamins A and $B_{12}$ compared with mature milk (Table 8.4). It aids the evacuation of the sticky, black contents (meconium) from the neonate's intestine, and contains several antibodies and other proteins which protect the newborn infant from bacterial infections, particularly in the intestine, and also against fungal disease, such as thrush. About the third or fourth day after delivery, colostrum is replaced by more normal-looking breast milk, but this retains certain properties until the infant is about two weeks of age. It has large numbers of cells which engulf infective organisms in the gut and also liberates several protective proteins. In addition, it has concentrated amounts of nutrients, notably zinc, which has a role in protecting against neonatal infections; lack of zinc may also eventually lead to stunted growth (Fildes, 1986).

**Table 8.4** Energy, macronutrient and selected micronutrient content of colostrum and mature human milk (per 100 g).

| Nutrient | Colostrum | Mature human milk |
|---|---|---|
| Energy (kJ) | 236 | 289 |
| Protein (g) (% PER) | 2.0 (14) | 1.3 (9) |
| Fat (g) (% FER) | 2.6 (42) | 4.1 (55) |
| Carbohydrate (g) (% CER) | 6.6 (47) | 7.2 (42) |
| Calcium (mg) | 28 | 34 |
| Sodium (mg) | 47 | 15 |
| Potassium (mg) | 70 | 58 |
| Iron (mg) | 0.07 | 0.07 |
| Retinol (μg) | 155 | 58 |
| Carotene (μg) | (135)[1] | (24)[1] |
| Vitamin $B_{12}$ (μg) | 0.1 | 0.01 |
| Vitamin C (mg) | 7 | 4 |
| Vitamin D (μg) | [2] | 0.04 |
| Thiamin (mg) | Trace | 0.02 |
| Riboflavin (mg) | 0.03 | 0.03 |
| Niacin (mg) | 0.1 | 0.2 |
| Folate (μg) | 2 | Trace |

PER, protein energy ratio; FER, fat energy ratio; CER, carbohydrate energy ratio.
[1] Estimated amount.
[2] Significant quantities but no reliable information.
After Macdonald and Shaw (1993) and Morgan (1999b).

The healthy neonate is immunologically naïve (i.e., has not seen antigen) and has not acquired immunological memory. Maternal colostrum and milk can significantly augment resistance to enteric infections (see section 15.6); the mechanisms are thought to be both passive, involving a direct supply of antimicrobial factors, and active, by promoting the development of specific immune function. A tolerance response to dietary and non-invasive antigens is generally

induced in the gut, and the education of the immune system in early life is thought to be critical in minimising the occurrence of immune-based disorders (Kelly & Coutts, 2000).

Between 1550 and the end of the seventeenth century, the medical authorities advised that the infant was not put to the mother's breast during the first few days after birth and even for up to a month after delivery. In pre-industrial Britain, feeding neonates with physick and/or food rather than colostrum was a cause of a high neonatal mortality, mainly from gastro-intestinal complaints, and milk fever and breast disorders in the mother. The change towards early maternal breast-feeding during the eighteenth century contributed to a notable decline in neonate mortality and a decrease in maternal morbidity and mortality from milk fever (Fildes, 1986).

Milk composition changes from 10 days post-partum, but less markedly than the earlier changes described above, and between 2 and 16 weeks the protein content and micronutrients such as calcium, phosphorus and sodium fall. Breast milk provides about 242 kJ (58 kcal) per 100 ml at 3 months post-partum.

The issue of whether a mother's diet affects the composition and the volume of her milk is complex. The fatty acid composition of breast milk reflects that of the maternal diet. It has been reported that failure to thrive in a breast-fed infant could be attributed to reduced milk production because the mother restricted her energy and protein intake. A review of evolutionary and environmental influences on human lactation concluded that there must be a threshold of nutritional status, probably at quite an extreme level of malnutrition, at which the maternal system can no longer sustain lactation and its own survival (Morgan, 1999a).

In summary, a breast-feeding mother needs at least 300 to 400 extra calories a day in the first few weeks, rising to nearly 600 a day as her baby gets older and hungrier. Breast milk from a woman who is in good health and nutritional status provides a complete food for healthy infants during the early months of life. Prolonged breast-feeding, practised by some women, may not necessarily result in growth faltering, although the infant's micronutrient status may be compromised.

## 8.4 Low birthweight infants

The problems of low birthweight infants are similar to those of prematurity, although the greater immaturity of the premature infant's gastrointestinal tract and of other organs makes its nutrition more difficult. Premature birth has a significant impact on the newborn's ability to metabolise nutrients. Foetal metabolism is optimised for energy accumulation and is adapted to a situation of steady fuel supply from maternal sources, primarily as glucose. Birth causes a dramatic shift in that metabolic steady state: the exogenous glucose supply ceases abruptly and the newborn must rapidly activate enzymatic pathways for gluco-

neogenesis and for the utilisation of fatty acids and ketone bodies as alternative sources of energy. Although many of the key enzymes for fuel utilisation increase their activity in response to substrate load, they may still fall short of the large fuel supply needed by the premature newborn (Caballero, 1999).

There is a need for more nutrients per unit of body weight to cope with the rapid growth in premature infants and with catch-up growth in small-for-dates infants. The smaller stomach volume in these infants means that there are restrictions on the amounts of food that can be administered and there is a conflict between increased nutrient needs, especially for protein, calcium, phosphate, fluid and energy, and the amounts that can be safely given. Table 8.5 lists the problems of low birthweight infants.

**Table 8.5** Nutritional problems of low birthweight infants.

| Problem | Precipitating factors |
|---|---|
| Immediate hypoglycaemia | Limited liver glycogen<br>Delay in mobilising fat stores<br>Problems with administering nutrients |
| Hypocalcaemia | Reaction to loss of calcium via high flux across placenta<br>Immaturity of calcium homeostatis<br>Occasional maternal vitamin D deficiency |
| Later poor growth | Nutrient density of breast milk probably not sufficient<br>Problems with administering high-energy density, low birthweight formula milk<br>Poor fat absorption because of immaturity<br>Increased needs of bronchopulmonary dysplasia |
| Bone disease of prematurity | Deficiency of substrate; especially phosphate<br>Renal calcium losses<br>Occasional vitamin D, protein or copper deficiency |
| Anaemia | Vitamin D deficiency<br>Folate deficiency<br>Iron deficiency |

*Source:* Poskitt (1999).

Low birthweight infants are also at increased risk of dying from infectious diseases. Infant mortality profiles from San Antonio, Texas, between 1935 and 1984 reveal that the population of infants who died from infectious underlying causes was increasingly composed of preterm and very low birthweight newborns, many of whom would not have survived without sophisticated medical intervention at birth. Between 1935 and 1944, 4% of infectious infant deaths had associated causes involving prematurity and related conditions; by 1980, 25% of infectious infant deaths involved prematurity and more than 40% of those infants weighed less than 2500 g. Sowards (1997) considers that, under conditions of advanced perinatal technology, infectious infant mortality should no longer be

viewed as wholly exogenous and that these findings further undermine the contemporary relevance of the exogenous–endogenous distinction.

## 8.5 The malnourished infant

Mothers in communities suffering from malnutrition, in addition to producing a greater proportion of low birthweight babies, may be unable to produce sufficient quantities of good quality milk to satisfy the requirements of the growing infant. The result may be a rise in post-neonatal mortality.

Iron deficiency may be a problem in these circumstances. Newborn infants have high haemoglobin levels because of the low oxygen tension *in utero*. After birth, in response to the high oxygen tension of the extra-uterine environment, the bone marrow undergoes a period of relative quiescence and haemoglobin levels fall. The bone marrow becomes more active again at 4–6 weeks of age, but by then infants have grown and their blood volumes have expanded. Haemoglobin levels fail to catch up with the increasing blood volumes because of growth, and remain below adult levels until late childhood.

Where infants are small and growing very rapidly, with low total blood volume and low total body iron, the need for this mineral for new blood formation may exceed either the stores or the iron absorbable from the diet, resulting in anaemia. Once the body weight has doubled, iron deficiency is likely in all infants since, without good external sources of iron, the stores of the body will be distributed over twice the blood volume – and haemoglobin levels will have halved.

Prentice and Prentice (1995) have provided a comprehensive review of their studies of marginally-nourished rural women in the Gambia. A recurring feature of these investigations has been that lactational performance appears to be exceptionally resilient to such influences as climatic conditions (mediated via effects on maternal water balance), maternal work patterns (mediated via effects on energy balance, fatigue and the time available for feeding the baby), maternal and infant infections and cultural attitudes to breast-feeding patterns (Prentice *et al.*, 1986). For example, milk synthesis was largely unaffected by maternal fasting during Ramadan, when women consumed no food or fluids between 04.00 and 19.00 hours, and lost an average of 7% of body weight (Prentice *et al.*, 1984). Similarly, it is particularly noteworthy that milk output declines by only 10% in the annual hungry season during which the mothers are working extremely hard in the fields, are in negative energy balance, are separated from their infants for up to 8 hours per day, thus interrupting the usual demand-feeding patterns, and have infants with raised levels of infections and consequent inanition which reduces the demand for milk (Prentice *et al.*, 1986).

In spite of some conflicting reports, most studies agree that there is no

apparent relationship between lactational performance (in terms of milk volume) and maternal BMI, even at levels normally interpreted as indicating moderately severe, chronic undernutrition of the mother. Prentice *et al.* (1986) and a range of current studies, including the World Health Organization Collaborative Study on Breast-feeding (World Health Organization, 1985), conclude that there is little evidence for associations between maternal poverty or undernutrition and the volume of milk produced. Indeed, milk outputs are often greater in the poor because they are more committed to breast-feeding.

The use of different milk-sampling procedures and analytical methods creates significant difficulties in comparing milk *quality* between different women. Notwithstanding these problems, there is little evidence to indicate important differences in the macronutrient content of milk between well-nourished and undernourished women (Prentice *et al.*, 1986). A possible exception is in the fat (and hence, energy) content of milk, for which there are conflicting results. Studies from the Gambia (Prentice *et al.*, 1981), USA (Nommsen *et al.*, 1991), Mexico (Villalpando *et al.*, 1991) and Bangladesh (Brown *et al.*, 1986) have observed significant positive relationships between milk fat content and various measures of maternal fatness. However, a number of other studies have found no relationships. Prentice and Prentice (1995) conclude that, in general, as in the case of milk volume, the macronutrient content of human breast milk appears to be surprisingly insensitive to differences in maternal nutrition.

The influence of maternal nutrition on the vitamin and mineral content of breast milk is highly variable, but is governed by a number of basic principles (Bates & Prentice, 1988). Water-soluble vitamin content is closely related to maternal plasma levels of the nutrients and there is a marked increase in riboflavin and vitamin C content of breast milk in response to programmes of maternal supplementation. Fat-soluble vitamins, such as vitamin A, are less responsive because of the buffering effects of maternal stores and carrier proteins (Villard & Bates, 1987). Bates and Prentice (1988) have also reviewed the influence of maternal nutrition on the mineral and trace element content of breast milk and they conclude that breast milk secretions of Fe, Zn, Cr, Cu, Na and Mg also do not appear to be related to dietary intake of the mother and are likely to be unresponsive to maternal dietary supplementation.

Prentice and Prentice (1995) conclude that, in general, milk nutrient levels are well protected and that lactational performance is remarkably unaffected by environmental factors, including maternal nutrition, although the protection of the quality and quantity of the milk is often achieved through the depletion of the body stores of the mother. It is evident that in pre-industrial communities, where the bulk of the population was malnourished, the diet of the mother before and during pregnancy must have had a much greater effect on the health and survival of the infant and, hence, on population demography, than did maternal nutrition during breast-feeding. Nevertheless, Fildes (1986) has suggested that women had insufficient milk to breast-feed a child during famines.

## 8.6 Weaning

Mature human milk meets the needs of babies, but they quickly outgrow its provision. At a certain time in the life of a young infant, the volume of milk theoretically required to cover the nutritional requirements becomes so great that most mothers cannot produce the necessary quantities and the infant cannot drink them. Gradual introduction of other foods is essential to meet the increasing nutritional requirements. The rate at which this weaning process can occur depends on the coincidental development of the gut and associated structures. Failure to take into account these various considerations can lead to growth faltering, adverse reactions to foods and, possibly, to diseases later in life. Thus, weaning represents one of the most crucial dietary events in the life of an infant because, either the incorrect timing of the introduction of a food, or the use of inadequate foods may impair the health and development of a child. In poorer countries today, or in pre-industrial England, the introduction of contaminated weaning foods, or foods with an inappropriate composition, may lead to diarrhoea, disease, growth failure and, ultimately, to the death of an infant.

There are ample studies indicating that, until recently, breast-fed infants experienced lower mortality risks than artificially-fed infants in developed countries today, and there is scattered evidence that this still holds true in the contemporary developing world (Knodel & Kintner, 1977; for a general review of such studies, see Wray, 1977). For example, in a study of infant mortality in Derby, England, shortly after 1900, Howarth (1905) found that breast-fed infants were three times as likely to survive as 'hand-fed' infants. The mortality due to diarrhoea and enteritis was particularly great for infants who were not breast-fed. Aykroyd (1971) stresses the similarity between the causes of infant mortality in Edwardian England and less developed countries today and their relationship to infant feeding practices. Most studies which differentiate fully breast-fed infants from partially breast-fed infants typically find that the mortality of the partially breast-fed is intermediate between the mortality rates of the fully breast-fed and those fed completely artificially (Wray, 1977).

In the full-term infant, by 6 months of age, the sources of iron and zinc accumulated *in utero* have become exhausted (but much earlier in the infant born prematurely). As breast milk is a poor source of these trace elements, a readily available dietary source of them, from foods other than breast milk, is vital. Iron deficiency anaemia is common in the preschool child throughout the world today (Morgan, 1999a). Thus, mothers today who continue to breast-feed beyond 6 months without supplementation may be depriving their babies of essential nutrients.

On the other hand, up to the early part of the twentieth century, the infants of mothers who were unable to provide adequate supplies of breast milk had a high probability of dying. Of those who were not breast-fed, there was a high probability that the milk used to feed these infants originated from animals and was

nutritionally inappropriate for human neonates. High infant mortality rates forced wealthy women to employ women as wet nurses to feed their infants, but this was not always satisfactory. These women often carried infectious diseases, some diluted their milk and there was a widespread belief that the milk of the nurse transmitted her personality to the baby (see section 9.9).

Diarrhoea is probably the single biggest threat to the survival of the infant after weaning; it is usually interpreted as frequent, poorly formed and watery stools. It is most commonly the result of infection in infancy, either gastro-intestinal, or a generalised viral infection. In such cases, onset is acute and associated with other signs of infection: pyrexia, vomiting, anorexia and general malaise. Diarrhoea caused by infection is usually short-lived, but may persist with certain infecting organisms in very young infants and in those who are already malnourished. Frequent episodes of even short-lived diarrhoea can lead to malnutrition, because of anorexia and reduced energy intakes. Prior malnutrition predisposes to increased morbidity and mortality from diarrhoeal illness, i.e. the malignant interaction of nutrition and diarrhoeal disease which affects so many children in less developed parts of the world (Poskitt, 1999). Weanling diarrhoea was originally investigated and named in 1963 in the Punjab, India, and several confirmatory studies have been done, particularly in Latin America, which have established it as an epidemiological entity. Such diarrhoea in infants is closely connected with the introduction of foods other than breast milk, and is thought to be the result of several interacting factors during the early weaning period including: (i) enteral infection, associated either with a sudden change in intestinal microflora, or with large doses of environmental bacterial contamination; (ii) the effects of malnutrition, such as diminished intestinal enzymes, and (iii) a diet of indigestible, ill-cooked foods which may be poorly absorbed or irritant. In the Punjab, the observed death rate was relatively low in the first 6 months of life (20.6 per 1000), more than doubled in the second 6 months of life (53.6 per 1000) and decreased thereafter until children were completely weaned from the breast by the beginning of the third year of life.

Since all three of these contributory factors would have been present among the poor in the seventeenth and eighteenth centuries, especially in times of poor harvests and famine, and at least two, (i) and (ii), among the better fed infants, it seems that there existed a similar, if not identical, condition to the weanling diarrhoea of the twentieth century (Fildes, 1986).

The association between breast-feeding and mortality in children hospitalised for diarrhoea has been investigated in a prospective manner in a hospital in New Delhi that predominantly catered for the poorer strata of the society. Annual paediatric admissions are about 7750–8000, of which about 17% are for diarrhoeal diseases. Breast-feeding had a strong protective effect against mortality, even after allowance was made for confounding variables. Further stratified analysis suggested a greater benefit in children with severe wasting, stunting, protracted illness or diarrhoea (Sachdev *et al.*, 1991).

There are also problems and dangers associated with the constitution of the weaning diet in poorer populations. It is clear from the foregoing that unsterilised cow's milk would not be adequate. The bulk of pre-industrial communities survived on a largely vegetarian diet based on grains (see section 1.9), and infants weaned onto vegetarian diets may become deficient in a variety of nutrients. Without substantial supplies of milk, riboflavin, calcium and total energy intakes may not meet requirements. If infants consume eggs and milk in addition to their vegetarian foods, it is possible to achieve basic dietary requirements; they should be fed frequently (at least four times a day) to enable them to consume large enough amounts of the low-energy, vegetarian diet. A variety of vegetable protein sources (e.g. beans, cereals, dark green leaves, homogenised nut products) should be provided at each meal, since essential amino acid requirements can be met only by mixing vegetable proteins (Poskitt, 1999).

Writers in the eighteenth century who gave advice about weaning diet were mainly directing their suggestions towards the wealthy or better educated. They tended to recommend a variety of meats and their products and, to a lesser extent, bread, because these were the main foods eaten by these groups in British society. Therefore, the few writers who thought the weaning diet worthy of mention in this period all recommended food that was familiar to their readers: chicken broth, minced or prechewed meat and meat gravies with breadcrumbs or rice. The description given by John Jones (1579) of the food given after weaning to the children of the French king was probably very similar to that in wealthy English households in the same period (Fildes, 1986):

> 'Bread of fine wheate floure, of fine starch, also of almonds, of barley, or bigge, of wheat, which we call furmentie, of rye, of pease and suchlike, or soft bread steeped in the broath of fleshe of kiddes, tuppes, calves, hennes, etc. And sometimes a capon's wing minced in small pieces, or the breast of a pheasant rosted, cut in pieces.'

Fildes (1986) believes that if such a diet were given to richer children regularly over a long period, diseases such as scurvy, rickets, bladder-stone and some degree of night blindness, accompanied by a lowered resistance to infection, would have been common after weaning because, in a mainly meat and cereal diet that excludes dairy foods, vitamins A, D and C are absent, and the amount of calcium may be insufficient for a growing child. With the exception of rickets, these diseases were all common among the adult population during the eighteenth century.

She states, however, that poorer families had a better diet than that suggested by Drummond and Wilbraham (1991), consisting mainly of bread, cheese, salt meat (predominantly pork) and pulses; thus the poorer child may well have fared better in nutritional terms (provided he received a sufficient quantity) than the richer one. White meats or dairy foods, including eggs, were eaten by the poor in

the sixteenth century and, as many cottagers kept a cow, milk was more likely to be drunk by these families than by the wealthy, although milk drinking was rare in towns because of poor supplies; a situation not remedied until the late eighteenth and early nineteenth centuries. Until at least the late eighteenth century there were regional differences in milk-drinking habits; the north of England used milk as a drink more than was usual in the southern counties. The child weaned on to this type of diet, which in summer and autumn probably included hedgerow fruits such as blackberries, may have suffered some degree of scurvy during the winter (as did the adult population) but, otherwise, would have received all the nutrients necessary for healthy growth.

The condemnation (by Pemell, 1653) of fish and fats (possible sources of vitamins A and D) was because they were thought to cause worms. This may explain the absence of these items from the recommended diets of physicians, although both rich and poor did eat fish, especially those living near the sea (Fildes, 1986).

We suspect that the poorer members of communities in pre-industrial populations did not enjoy such a varied diet and that it was in short supply at times, so that weaning nutrition was often suboptimal.

## 8.7 The age of weaning

Because of the different factors involved, the age of weaning varies between populations and individuals, but the UK Department of Health recommends that the majority of infants should not be given solid food before the age of 4 months and a mixed diet should be offered by the age of 6 months. Early weaning may be associated with infection and obesity, whereas late weaning may be associated with failure to thrive and iron deficiency anaemia (Morgan, 1999a).

Prentice (1991b) has reviewed the possible benefits and disadvantages to the older infant of breast-feeding being continued after the introduction of solid foods. The limited evidence from industrialised countries suggests that prolonged partial breast-feeding has little influence on child health and growth. In contrast, in poor areas of the developing world, the continuation of breast-feeding for 1–2 years after the introduction of other foods appears to have several major benefits. These include the supply of nutrients, the delivery of protective, digestive and trophic agents, and extending the period of infertility in the mother. Partial breast-feeding after 6 months is associated with a reduced severity of infectious diseases, particularly in severely malnourished individuals. There is no evidence that partial breast-feeding plays a causal role in poor growth performance. She recommends that in poor areas of the developing world, breast-feeding, together with the provision of adequate amounts of other foods, should be encouraged for the first two years of life.

In contrast to the recommendations of health authorities in Westernised

countries, there was in earlier centuries and in developing countries today a very wide variation in the age of weaning. Children were taken off breast-feeding at ages ranging from birth (because of the force of circumstances) to more than two years. As we shall see (section 9.11) there were clear differences in the practices of different sections of the same community, each trying to balance the local customs, the risks to their infant (and, to a lesser extent, to the mother) and the available supply of nutrients.

Fildes (1986) has presented a very careful study of weaning practices in earlier centuries, relying on contemporary published medical advice and written sources. These are, inevitably, applicable to the rich and well educated, and the upper strata of society, including royal families, who made considerable use of wet nurses. She has identified three types of weaning age:

(1) Recommended age – the age of weaning advised by physicians and surgeons; the median ranged from 21 to 24 months in the sixteenth and seventeenth centuries to 10 months in the eighteenth century.
(2) Common age at which most children were said to be weaned and was deduced by Fildes from a variety of generalised statements. The median value varied little: it ranged from 11 to 12 months in the sixteenth and seventeenth centuries to 10.5 months in the eighteenth century.
(3) Actual age of weaning obtained by examining letters, diaries and case histories given in medical textbooks.

Statistical analysis appears to indicate that the age at which children were commonly said to be weaned was similar to the age at which a sample of children were actually weaned, and that the age recommended by physicians in the sixteenth century was unrelated to actual practice; in the seventeenth and eighteenth centuries, when the recommendations changed, it became similar to common and actual practices.

However, Fildes (1986) warns that not all these findings can be taken as evidence of what was happening in Britain. All the recommendations before the second half of the seventeenth century were derived from non-English writers, as were four out of the 13 references to what was common practice. When the non-English references were excluded, there was no significant differences between the recommended and actual ages in the seventeenth and eighteenth centuries; the actual age of weaning in the late eighteenth century (7.3 months) was significantly earlier than in the sixteenth and seventeenth centuries (about 14 months).

Mixed feeding was frequently introduced before the cessation of breast-feeding among the upper strata of society. In the sixteenth century, the ideal age of introducing mixed feeding was 7–9 months, but during the late seventeenth and eighteenth centuries, the much earlier age of 2–4 months was favoured. The age at which foods other than breast milk were first given was probably earlier

than the time recommended by physicians and midwives, depending upon the custom of each woman. Age and the cutting of the first teeth were the two main considerations when starting infants on a mixed diet. Sixteenth-century authors did not relate infantile disorders to the introduction of mixed feeding, but those of the seventeenth and eighteenth centuries described several conditions which resulted from giving 'improper food', particularly pap. The level of concern of medical authors in this aspect of feeding reflected the increased interest of physicians in childhood diseases, especially in the eighteenth century. Disorders of the gastro-intestinal tract, probably weanling diarrhoea, were a major problem (Fildes, 1986).

The foregoing, which has been derived from letters and from the writings of physicians, tells us little about the poorer sections of the rural communities in pre-industrial England. The technique of Bourgeois-Pichat plots for the analysis of infant mortality and point of weaning is described in section 9.1.

It has been suggested that in pre-industrial societies the variation in the availability of suitable weaning foods influenced the frequency and duration of breastfeeding – the so-called 'weaning food availability' hypothesis. Sellen and Smay (2001) have examined the available data on weaning age variation in pre-industrial populations and report the results of a cross-cultural test of the predictions that weaning occurred earlier in agricultural and pastoral populations because dairy and cereal production increased the availability of easily digestible, nutrient-rich foods appropriate for weaning. They found that, contrary to prediction, supplementation with liquid foods other than breast milk was delayed in agricultural populations relative to less agriculturally dependent ones, and complementary feeding with solid foods was delayed in pastoral populations relative to those less dependent on herding. Although the duration of breastfeeding was longer in populations dependent on hunting, there was no qualitative evidence that such populations lacked foods appropriate for weaning. The patterns observed suggest that the relationships between demography and subsistence observed among pre-industrial societies cannot be explained by the 'weaning food availability' hypothesis.

These findings for the age of weaning in England in earlier centuries may be compared with weaning in rural and urban societies in different parts of Africa in the 1970s (see Table 8.6). Urban societies weaned their infants at a significantly earlier age (median 15.6 months) than the rural communities (median 24.1 months; $P < 0.001$). This phenomenon has also been observed in Central and South America, the Middle East and Asia.

## 8.8 Catch-up growth

The low birthweight neonate may catch up its peers by rapid post-natal growth as a result of an increased nutritive intake, but this catch-up growth carries addi-

**Table 8.6** The age of weaning in 12 rural and 10 urban societies from different parts of Africa.

| Rural communities | Age (months) | Urban communities | Age (months) |
|---|---|---|---|
| Rural Gambia | 21 (18–24) | Abidjan | 13.5 |
| Rural Guinea | 36 | Lagos | 12 |
| Rural Ivory Coast | 42 (36–48) | Ibadan | 14+ |
| Rural Nigeria | 21 (20–22) | Dakar | 18.7 |
| Inesis, rural | 23.2 | Brazzaville | 18 (12–24) |
| Sine, rural | 24.3 | Leopoldville | 18 (12–24) |
| Burundi (suburb) | 24 (18–30) | Kinshasa (I) | 16.8 |
| Bambara, rural | 30 (24–36) | Kinshasa (II) | 19 (11–27) |
| Highland tribes | 27 (18–36) | Baganda, Kampala | 14 (12–16) |
| Kenya Masai | 36 | Algeria, urban | 11.3 |
| Somalia (shepherds) | 24 | | |
| Algeria, rural | 14.4 | | |
| Median | 24.1 | | 15.6 |

Note difference in median values. Data from Fildes (1986).

tional risks. First, the long-term damage to the foetus has already been done by malnutrition *in utero*, and the risk of hypertension, diabetes and heart disease is further increased by rapid post-natal growth. Epidemiological studies in Finland showed that death from heart disease is greatest in men who were small at birth and who then gained weight heavily in childhood. In another study, Swedish men who were small at birth, but grew taller than average later, had high blood pressure. Another study in Finland has also shown that reduced intra-uterine growth and low birthweight are associated with raised rates of stroke in adult life, and this risk of stroke is increased by accelerated growth in height during childhood (Eriksson *et al.*, 2000).

Three possible explanations have been advanced for these counterintuitive findings. One is that small babies who grow quickly later may have high fat-to-muscle ratios – a body composition that predisposes adults to diabetes.

Alternatively, rapid growth may force a limited number of cells to divide too many times. Growth-retarded baby rats who caught up after birth died young; they had kidney cells with shorter than normal telomeres, which determine how many cell divisions are possible. If an animal is born with fewer kidney cells and these have to divide rapidly to catch up, each cell will quickly arrive at the position when further division is not possible.

The third explanation is that the hormones that drive catch-up growth in small babies may ultimately be damaging. Growth-retarded sheep and guinea pigs are very sensitive to insulin just after birth and this hormone raises the uptake of sugar and amino acids from the blood. But then the tissues become insulin-resistant and this process is reversed.

Chapter 9

# Infant Mortality

Jones (1980) analysed the changes in infant mortality in rural parishes of north Shropshire through the period 1561–1810, but concluded

> 'In essence, the difficulty is that we have no real information about the causes of infant mortality outside the pattern of the statistics themselves. Books containing child care advice give only what was considered desirable practice and then only, effectively, in middle and upper class households. There are no local descriptions of child care practices: at best we can obtain a small amount of information from the diaries or letters of, again, a middle and upper class minority. For this area and period, we have very little information about causes of death; at best some general notices of major epidemics and a few registers giving ill-defined causes of individual deaths. Even these few registers do not consistently extend the practice to very young children, in them infancy itself is considered a sufficient cause of death. We are, therefore, reduced to juggling with the figures derived from parish registers if we are to make any attempt to explain the trends in infant mortality between the seventeenth and the nineteenth century.'

We show in this and the succeeding chapters that by applying different analytical techniques to a detailed case study and relating the results to what we know of the importance of the mother's physiology during pregnancy (Chapter 7), it is possible to show the important, but not readily-detectable, effects of nutrition in determining the separate components of infant mortality.

We have seen (section 1.9) how the lowest social classes, who formed the bulk of the population in the communities of preindustrial rural England, suffered from a subadequate diet and had probably become partially acclimated, or even partially adapted to it. Malnutrition exercised its most damaging demographic effects during pregnancy (Chapter 7), modifying the programming of foetal development and causing low birthweight and disproportionate babies and raised infant mortality. Fluctuating levels of malnutrition could have had even more serious consequences and would have been correlated with the 5–6 year cycle in grain prices (section 3.3) or the annual effects of the hungry season (Chapter 13).

## 9.1 Bourgeois-Pichat plots

The biometric analysis of infant mortality, developed and described by Bourgeois-Pichat in a series of articles (1946, 1950, 1951a, b), has become a widely-accepted and utilised technique in studies of both historical and contemporary patterns of mortality. It provides a simple method for separating endogenous and exogenous infant deaths. Endogenous infant mortality is defined as all deaths attributable to factors preceding, or associated with, birth (congenital anomalies, short gestation, low birthweight, birth trauma, and certain other conditions originating in the perinatal period); exogenous infant mortality is defined as all deaths under the age of one year that are attributable to factors in the post-natal environment, infectious diseases (such as weanling diarrhoea), poor nutrition, poisonings and accidents. The rationale for making this distinction is that, as we have seen, endogenous and exogenous mortality predominantly result from distinct sets of causes, follow different trends over time, and respond to different kinds of preventive and ameliorative interventions.

The biometric model depends upon two features of infant mortality: first that virtually all endogenous mortality occurs within the first month of life and, second, that cumulative exogenous mortality is proportional to $[\log (n + 1)]^3$, where $n$ is the age in days. With cumulative infant mortality plotted on this basis, the intercept on the ordinate represents the calculated endogenous mortality rate for the community. An example of the biometric method for graphically determining the endogenous and exogenous components of mortality during the first year of life is shown in Fig. 9.1; for infants in rural England in 1905, the cumulative mortality from age one month falls on a straight line as predicted. Estimated endogenous mortality is 22 per 1000 live births, and total exogenous infant mortality is 85 per 1000 live births (Knodel & Kintner, 1977).

Many Bourgeois-Pichat plots show a significant deviation from linearity and Pressat (1972) states that the most common exceptions are those where the cumulative mortality rises more steeply after several months of life, i.e. there is a break in the plot so that the mortality during the later months of the first year of life fits a second straight line that has a steeper slope than the first. Such a significant deviation of the cumulative infant-mortality plots from linearity has been regarded as evidence for differences in breast-feeding practices. Where breast-feeding was common and of long duration, mortality was found to rise more rapidly in the later months of the first year of life than would be expected on the basis of the mortality in the earlier months. Where breast-feeding was uncommon or of very short duration, the reverse pattern was found: mortality increased more rapidly in the first few months of life than in the latter half of the first year of life. The rise in mortality associated with the start of weaning is attributed to digestive impairments and weaning diarrhoea (Bourgeois-Pichat, 1952; Pressat, 1972; Knodel & Kintner, 1977; see section 15.6). Consistent deviations from the expected linear relationship are evident in a study of infant mortality in eight

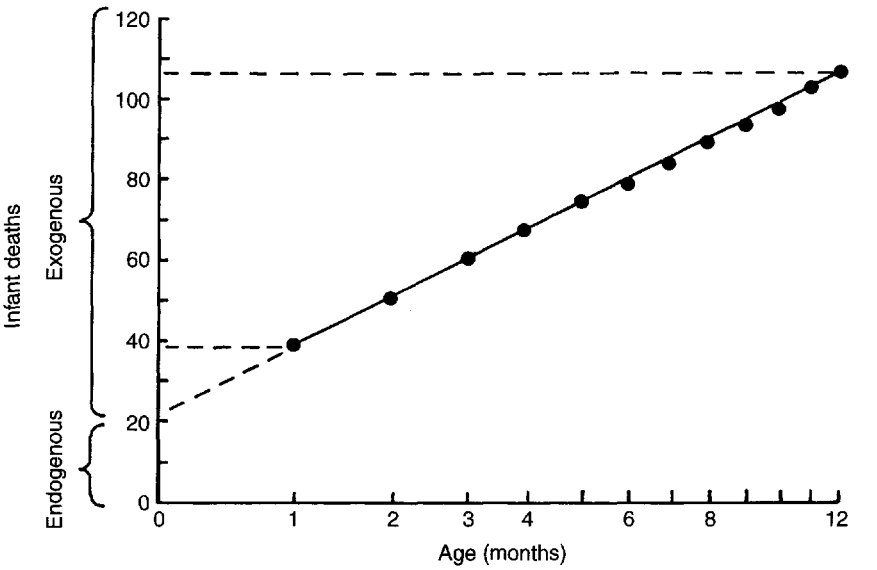


**Fig. 9.1** Diagram to illustrate a Bourgeois-Pichat biometric plot. Cumulative infant deaths in England and Wales per 1000 live births, 1905 (ordinate). Abscissa: age at death (months) plotted as $[\log (n+1)]^3$, where $n$ = age in days. The line is extrapolated to the ordinate to derive the estimated endogenous mortality. From Knodel and Kintner (1977).

cities in the USA from 1911 to 1916. Breast-fed infants showed an almost linear relationship for the Bourgeois-Pichat plot during the first 12 months of life, whereas other infants had a sharp upturn in mortality at the point of weaning (Knodel & Kintner, 1977; see Fig. 9.2).

Knodel and Kintner (1977) give other examples of data from the twentieth century of non-linear Bourgeois-Pichat plots which are shown in Fig. 9.3. The plot for Sardinia during 1948–50 shows the rise in mortality at 3 months and the ratio of the slopes of the lines after and before the break is 1.60. In contrast, the cumulative plot for Quebec during 1944–7 shows a *fall* in mortality at 5 months and the ratio of the slopes of the lines after and before the break is 0.62. Bourgeois-Pichat (1951a) attributes this, in part, to a prolongation of life of constitutionally weak infants through modern medicine, thus postponing some endogenous deaths until after the first month, and, in part, to certain sanitary or medical techniques which can be applied only after the infant reaches a certain age. The plot for Quebec (Fig. 9.3) emphasises how medicine in the twentieth century in developed countries could produce subtle changes in the patterns of infant mortality. In particular, problems associated with weaning could be greatly ameliorated by the use of sterilisation techniques and antibiotics.

The Bourgeois-Pichat biometric model has been used to analyse the differences in patterns of mortality in artificially-fed and breast-fed infants in the early twentieth century. The data sets were some 23 000 births in eight cities in the

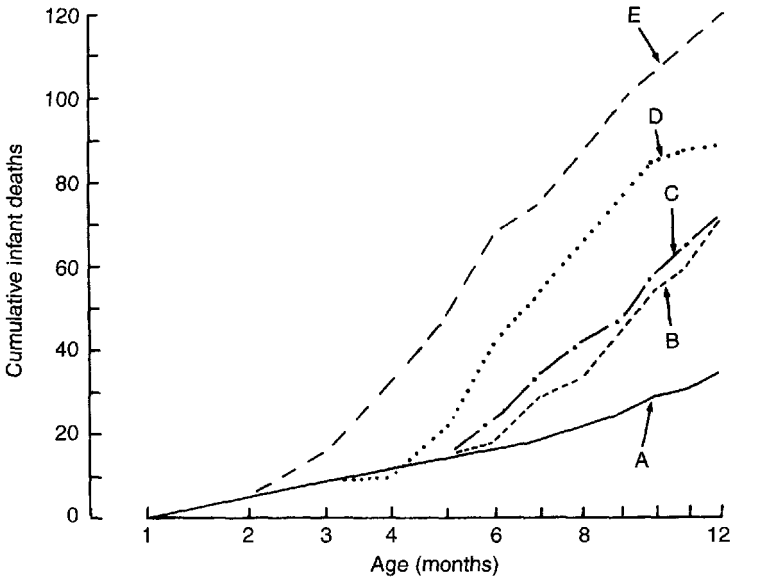


**Fig. 9.2** Bourgeois-Pichat plots of cumulative infant deaths from age one month per 1000 live births from eight US cities 1911–16 (ordinate). Abscissa: age (months). Separate plots for different ages at weaning. A: Breast-fed; B: weaned at 5 months; C: weaned at 4 months; D: weaned at 3 months; E: weaned at 2 months. From Knodel and Kintner (1977).

USA and four studies of German cities (see Fig. 9.4). Breast-fed infants showed a small rise in mortality (mean ratio of slopes = 1.34), whereas artificially-fed infants showed a slightly greater fall (mean ratio of slopes = 0.62). However, inspection of the plots shown in Fig. 9.4 reveals a much greater demographic difference between the two groups: overall cumulative infant mortality in the artificially-fed groups is some four times greater than in their counterparts in the breast-fed groups. This confirms the findings given in section 8.3 of the beneficial results of breast-feeding in the periods before the universal acceptance of a proper formula diet.

A number of studies have drawn attention to deficiencies and deviations from this rule of Bourgeois-Pichat for the distribution of deaths in the first year of life, particularly for the late nineteenth and the twentieth centuries in industrialised countries. This should not surprise us because the advances in medical sciences over this period, including sterilisation techniques and improved weaning diet, changed the basic conditions and tenets to which Bourgeois-Pichat's original thesis applied.

Examples of these studies are, first, Galley and Woods (1998) and Galley and Shelton (2001) who point out that the registration system for births and deaths needs to be accurate for the Bourgeois-Pichat model to be accepted. Furthermore, they believe that, even when it can be demonstrated that under-registration has not occurred (as after 1838 in England), considerable doubt

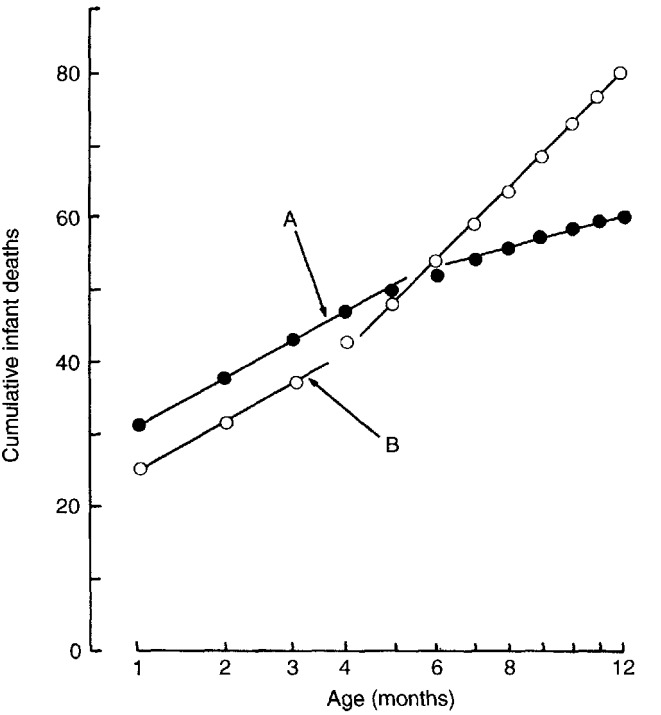


**Fig. 9.3** Bourgeois-Pichat plots of cumulative infant deaths per 1000 live births. A: Quebec, Canada, 1944–47; B: Sardinia, 1948–50. From Knodel and Kintner (1977).

persists as to the validity and applicability of the technique for the determinations of the true level of endogenous mortality. They cite 19 districts which had an apparent negative endogenous mortality rate during 1839–46 when calculated by linear regression. However, it is obviously impossible, with the data available, to determine a true rate of endogenous mortality, particularly for periods before civil registration. But this is relatively unimportant for our purposes. If endogenous mortality is estimated by a standard procedure and from the extrapolation of the line fitted to the points for the early months, before any inflection (so avoiding misleading negative values for endogenous mortality), it is possible to compare the rates in different populations and at different times. Furthermore, as we shall see below, Bourgeois-Pichat plots can supply a wealth of demographic and sociological information, in addition to an estimation of endogenous mortality rates.

Second, London (1993) used data from the second half of the twentieth century to analyse the assumption of the biometric model that no endogenous deaths occur after the first month of life. Direct examination of the age distribution of infant mortality shows that the proportion of deaths that occurred at this time declined from 75% in 1970 to 65% in 1985. A significant and increasing proportion of the mortality occurring after the first month of life was because of

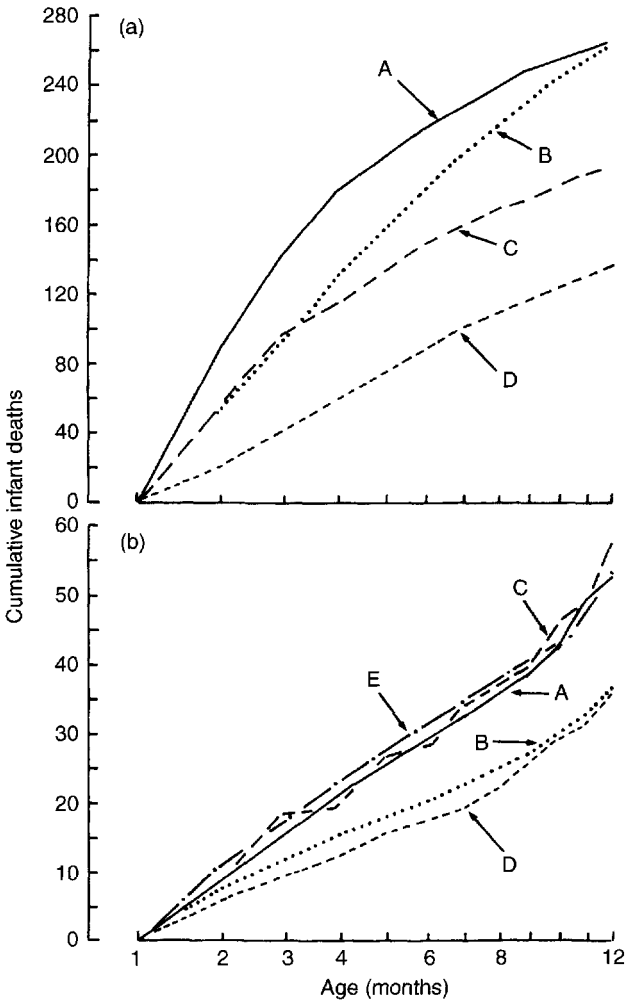


**Fig. 9.4** Bourgeois-Pichat plots of cumulative infant deaths after the age of one month per 1000 live births in American and German cities (ordinates). Abscissae: age (months). (a) artificially-fed infants (b) breast-fed infants. A: Barmen, Germany, 1905; B: Berlin, 1895–96; C: Hanover, Germany, 1912. D: eight US cities, 1911–16; E: Cologne, 1908–09. From Knodel and Kintner (1977).

endogenous causes. The biometric model was found to underestimate endogenous and to overestimate exogenous infant mortality rates and it is concluded that recent advances in medicine extend endogenous mortality past the first month of life.

Third, Galley and Woods (1998) have examined data from a number of countries for populations also from the late nineteenth and twentieth centuries. They find no single and universal distribution, such as that described by the Bourgeois-Pichat biometric model, but they suggest that the existence of a

number of distinct patterns subject to change over time is revealed. Figure 9.5 shows their biometric plots of the cumulative rates of infant mortality in three towns compared with the rural counties in England in 1889–91. Both plots show a very small upward inflexion, which was evident at age 3 months in the urban populations and after age 5 months in the rural counties. Although the endogenous mortalities in the two data sets are similar and close to the value for the rural counties of England and Wales in 1905 (see Fig. 9.1), the two lines have very different slopes, with urban exogenous mortality rates 2.2 times as high as that recorded in the rural counties. Galley and Woods (1998) also compare the biometric plots for towns and rural counties in England and Wales in 1889–91; the plots are very similar to those shown in Fig. 9.5 and we conclude that the Bourgeois-Pichat model, using large samples of aggregated data at the end of the nineteenth century, reveals little evidence of the effects of weaning on infant mortality.

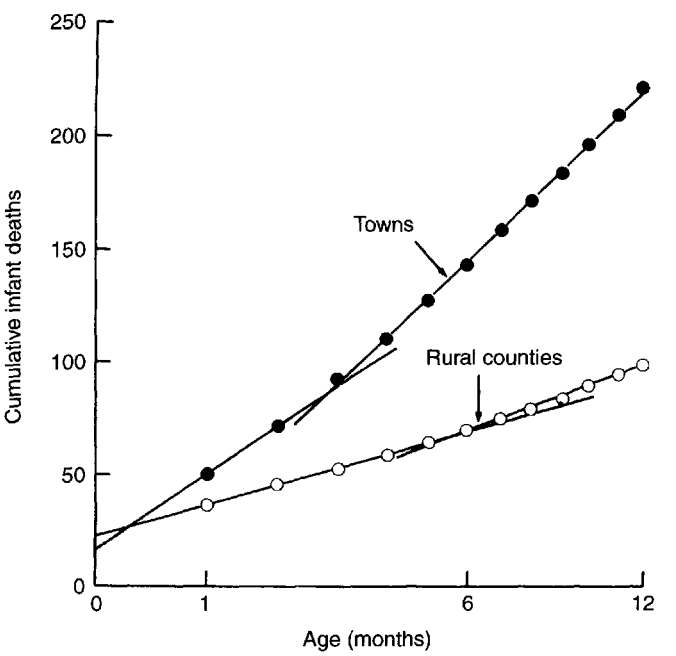


**Fig. 9.5** Bourgeois-Pichat plots of cumulative infant deaths per 1000 live births (ordinate) in three towns and rural counties of England, 1889–91. Abscissa: age (months). From Galley and Woods (1998).

## 9.2 Infant mortality in pre-industrial England

Jones (1976, 1980) and Wrigley (1977) have explored in detail the quality of the recording in the ecclesiastical registers for use in determining the levels of infant mortality and other demographic parameters in pre-industrial England. Wrigley

writes that there are many contributory elements to the shortfall between the events of which a record has survived and the births, deaths and marriages which occurred, for example complete breaks in registration because of the subsequent loss or decay of the register or due to some failing on the part of the minister of the day; periods when registration, although never collapsing completely, fell far short of its normal level; losses because of the presence of people who were not Anglican in religion and who ceased to use the Anglican rites. In general, the baptism shortfall appears to have been larger than the comparable shortfalls in the marriage and burial series. Moreover, there is a feature that is of particular importance in the construction of biometric plots that is peculiar to baptism series: between the sixteenth and nineteenth centuries, the average interval between birth and baptism increased substantially and with it the danger that a young child would die before baptism. Since approximately half of all infant deaths occur within the first month of life, mostly in the first few days, there is a strong probability that many children did not live to be baptised, although many parents secured the early baptism of a child who seemed likely to die.

Figures 9.6 and 9.7 show a number of Wrigley's biometric plots for two periods, 1550–1749 and 1750–1799, and all of them are either linear or reveal a *fall* in the infant mortality after about 6 months of age. The biometric plot for Ludlow, 1577–1619 (Schofield & Wrigley, 1979) is also shown in Fig. 9.7 and, again, there is a clear fall in infant mortality after 3 months of age. A further feature of Figs 9.6 and 9.7 is the wide range of endogenous mortalities shown in the different populations studied; these varied from 40 to 95 deaths per 1000 live births. Some of these differences are explicable as an amelioration of mortality with time (see Alcester, 1550–79 vs 1750–99; Fig. 9.7), but why is the endogenous mortality of Banbury twice that of Colyton for the same time period (see Fig. 9.6)?

## 9.3 Infant mortality at Penrith, Cumbria, England: a case study

A family reconstitution study has been carried out for the parish of Penrith, Cumbria, for the years 1600 to 1800 (Scott & Duncan, 1998), following the techniques described by Wrigley (1966). From this database with 30 000 entries, 1079 families were chosen as the basis for the analyses of infant mortality. The criteria for inclusion in the study were that the date of marriage and the end of marriage (the date of death of either partner) had to be known and that the marriage be the first and only for both spouses. Childless marriages were not included. Infant mortality for this pre-industrial period can be determined only by family reconstitution studies, and the parish of Penrith is close to the maximum size that can be handled conveniently. This constraint limits the number of infant deaths that are available for analysis.

Wrigley *et al.* (1997), in their studies of mortality, explored in great detail the method of handling the records of child burials when they had no link to

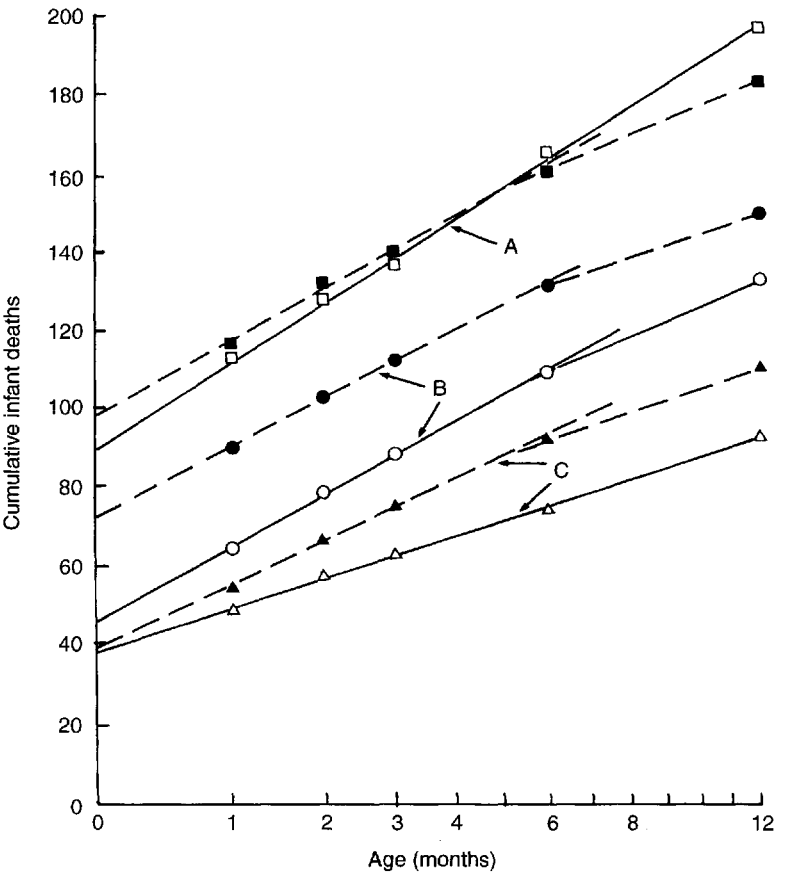


**Fig. 9.6** Bourgeois-Pichat plots of cumulative infant deaths per 1000 live births (ordinate) in A: Banbury, Oxfordshire; B: mean of Colyton, Devon; Alcester, Warwickshire; Banbury, Oxfordshire and Aldenham, Hertfordshire; C: Colyton, Devon. Closed symbols (dashed line): 1550–1749. Open symbols (continuous line): 1750–99. Abscissa: age (months). From Wrigley (1977).

baptisms, and we have followed their recommendations. The technique involves the creation of a dummy birth entry, but with an over-riding proviso that there be an appropriate space in the existing sequence of baptisms in the family, thereby showing the likelihood that the burial entry is evidence of an infant death. This strategy guards against the possibility of the child being born earlier in another parish. Only 'standard' dummy births (terminology of Wrigley and Schofield) were included in our analysis (Wrigley, 1966; Wrigley *et al.*, 1997).

Wrigley *et al.* (1997) have surveyed 26 parishes over a period of 250 years, concluding that, provided that the safeguards described above are maintained, the dummy-births technique provides the best estimate of endogenous mortality. Nevertheless, we also recalculated our data, omitting the dummy births. As

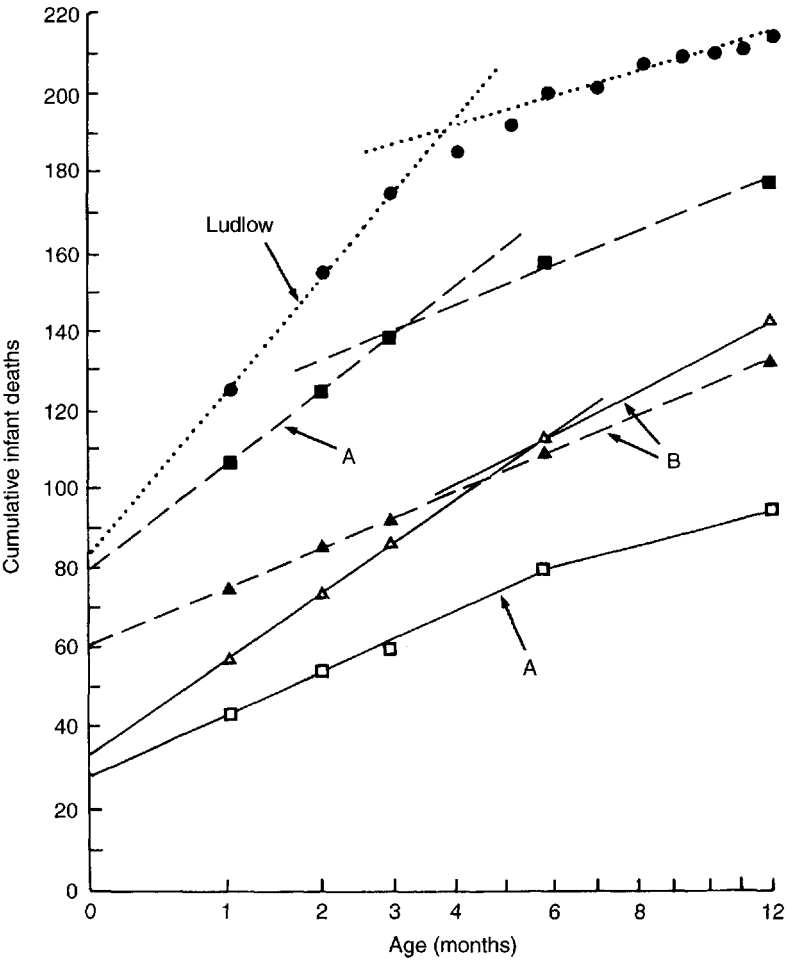


**Fig. 9.7** Bourgeois-Pichat plots of cumulative infants per 1000 live births (ordinate). Abscissa: age (months). Upper (dotted) line: Ludlow, Shropshire, 1577–1619 (from Schofield and Wrigley, 1979). Lower lines: squares (A): Alcester, Warwickshire; triangles (B): Aldenham, Hertfordshire. Closed symbols (dashed lines): 1550–1749. Open symbols (continuous lines): 1750–99. From Wrigley (1977).

expected, the level of endogenous mortality is thereby reduced in each cohort, but the patterns of overall infant mortality are broadly similar.

The cumulative infant death rates (per 1000 live births) at Penrith after the first month of life (that is, post-neonatal mortality) were plotted against age, expressed as $[\log (n + 1)]^3$, where $n$ is the age in days since baptism (the Bourgeois-Pichat biometric model). The calculated regression lines are shown in the figures, and the results are compared by an analysis of covariance program. Lines were also fitted in curvilinear plots for the points covering 1 to 3 months and extrapolated to age 0 for estimates of the endogenous mortality.

The Bourgeois-Pichat plots of cumulative infant mortality (1 to 12 months) for males and females in the different cohorts at Penrith are shown in Figs 9.8 and 9.9, respectively, and the data derived therefrom are summarised in Table 9.1. Some of the plots depart significantly from linearity. The calculated regression lines for the period of 1 to 12 months of life are shown on the figures, and the slopes are given in column 1 of Table 9.1. Lines that did not depart significantly from linearity are indicated.

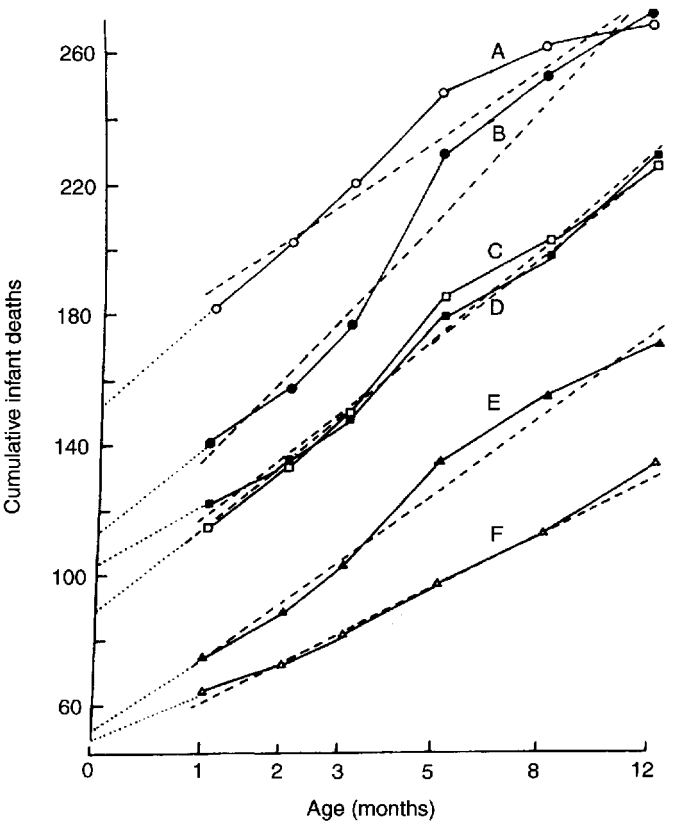


**Fig. 9.8** The Bourgeois-Pichat biometric model: changing male infant mortality at Penrith. A: 1557–99; B: 1600–49; C: 1650–99; D: 1700–49; E: 1750–74; F: 1775–1812. Dashed lines: extrapolated lines for 1 to 3 months for the calculation of endogenous mortality. Ordinate: cumulative infant mortality (deaths per 1000 baptisms). Abscissa: age (months) plotted as $[\log (n+1)]^3$, where $n$ is the age in days since baptism.

Because of the non-linearity of certain plots and the curvilinear shape evident by eye (see Figs 9.8 and 9.9), the endogenous mortality was estimated by the value of the intercept when the fitted line for 1 to 3 months was extrapolated to the ordinate. Endogenous mortality calculated in this way is shown in column 4, Table 9.1.

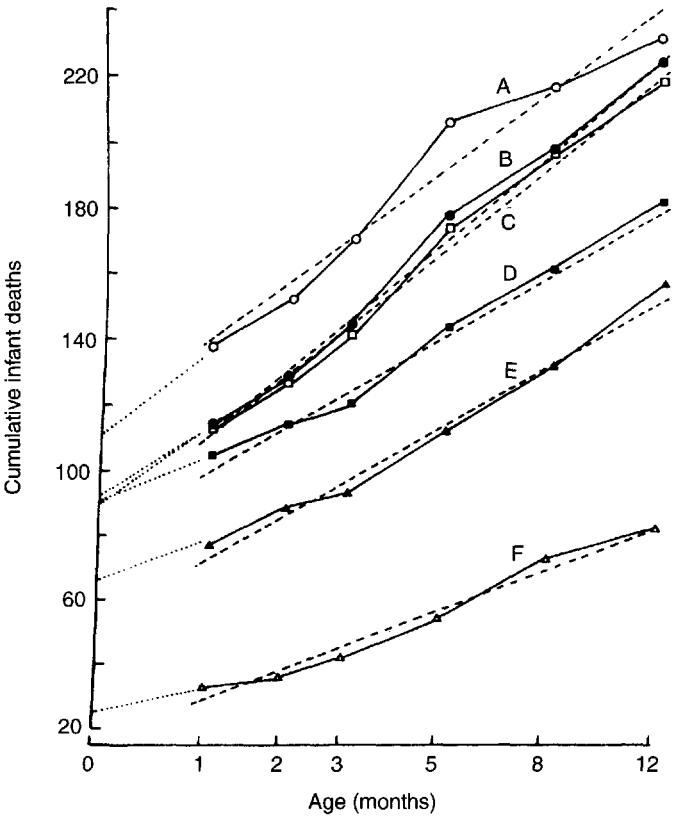


**Fig. 9.9** The Bourgeois-Pichat biometric model: changing female infant mortality at Penrith. A: 1557–99; B: 1600–49; C: 1650–99; D: 1700–49; E: 1750–74; F: 1775–1812. Dashed lines: extrapolated lines for 1 to 3 months for the calculation of endogenous mortality. Ordinate: cumulative infant mortality (deaths per 1000 baptisms). Abscissa: age (months) plotted as $[\log (n+1)]^3$, where $n$ is the age in days since baptism.

Two points are evident. First, infant mortality improved through the cohorts from 1557 to 1812, as shown in Figs 9.8 and 9.9, with a progressive fall in the cumulative totals at 12 months of age (Table 9.1, column 3). Second, in each cohort, the infant mortality for males is greater than the corresponding value for females (Table 9.1, column 3).

Although the slopes of the calculated regression lines of both male and female infant mortality, in general, fall slightly with time, few major changes occur until the last cohort, 1775–1812, as is evident in Figs 9.8 and 9.9 and in column 1, Table 9.1. The reason for the amelioration in overall infant mortality lies principally in the progressive fall in endogenous mortality, which is most striking in male children (see Fig. 9.10). The endogenous mortality rate, 1600 to 1800, for boys fell from 152 to 49 and from 110 to only 25 for girls. Boys and girls show the same broad pattern of improvement in endogenous infant mortality with time (Fig.

**Table 9.1** Infant mortality at Penrith, Cumbria, 1557–1812.

| Cohort | No of baptisms | Sex of infant | No of deaths | Slopes (1 to 12 months) (1) | Pearson *R* (2) | Mortality at 12 months (3) | Endogenous mortality (4) | Slopes (1 to 3 months) (5) | Exogenous mortality (6) |
|---|---|---|---|---|---|---|---|---|---|
| (1) 1557–99 | 2002 | Male | 289 | 6.49 | 0.969 | 266 | 152 | 8.9 | 113 |
| | | Female | 211 | 7.32* | 0.978 | 231 | 110 | 7.8 | 121 |
| (2) 1600–49 | 2454 | Male | 341 | 10.37* | 0.982 | 270 | 113 | 8.2 | 157 |
| | | Female | 248 | 8.42* | 0.996 | 224 | 89 | 7.2 | 135 |
| (3) 1650–99 | 2260 | Male | 266 | 8.31* | 0.992 | 224 | 89 | 8.0 | 135 |
| | | Female | 233 | 8.13* | 0.995 | 218 | 91 | 6.5 | 127 |
| (4) 1700–49 | 2502 | Male | 292 | 7.93* | 0.995 | 227 | 103 | 5.9 | 124 |
| | | Female | 221 | 5.95* | 0.995 | 182 | 91 | 4.0 | 89 |
| (5a) 1750–74 | 1485 | Male | 127 | 7.47* | 0.991 | 170 | 52 | 6.7 | 119 |
| | | Female | 115 | 5.87 | 0.991 | 156 | 66 | 3.6 | 88 |
| (5b) 1775–1812 | 2292 | Male | 158 | 5.16 | 0.997 | 133 | 49 | 4.3 | 84 |
| | | Female | 91 | 3.93 | 0.988 | 82 | 25 | 2.2 | 58 |

* Does not differ significantly from linearity.

*Notes:* Mortality expressed per 1000 live births. Endogenous mortality calculated by extrapolation of the points from 1 to 3 months. Exogenous mortality equals column (3) minus column (4).

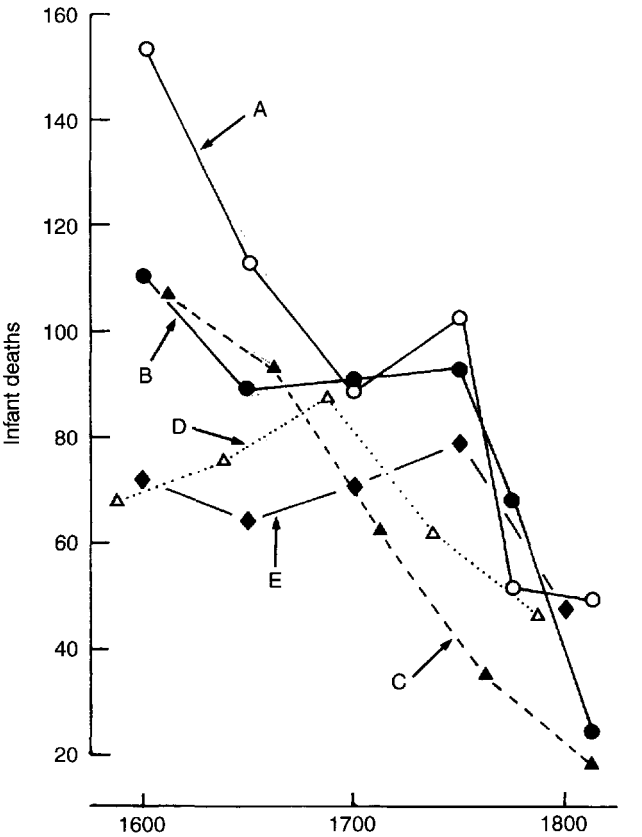


**Fig. 9.10** Endogenous infant mortality. A (open circles): Penrith, male infants; B (closed circles): Penrith, female infants; C (closed triangles): Shropshire parishes (sexes combined); D (open triangles): 16 parishes studied by Wrigley (sexes combined); E (closed diamonds): four parishes studied by Wrigley (see Table 11.7). Ordinate: cumulative endogenous infant deaths per 1000 baptisms. Abscissa: date at end of cohort under study.

9.10). The amelioration was particularly marked for females from 1775 to 1812, whereas male endogenous mortality showed the greatest reduction during the preceding period, from 1750 to 1774.

Exogenous mortality is determined as the total mortality at 12 months minus the endogenous mortality (column 6, Table 9.1; Fig. 9.11). It is also shown as the calculated regression lines of Figs 9.8 and 9.9. Exogenous mortality at Penrith (in contrast with endogenous mortality) did not change greatly for either males or females, except for the amelioration shown after 1775. The exception to this general conclusion is that the male exogenous infant mortality apparently rose during the period from 1600 to 1700. Figure 9.11 traces the changing patterns of exogenous mortality at Penrith. The total exogenous infant mortality falls clearly below the 1557–99 level only at the end of the eighteenth century.

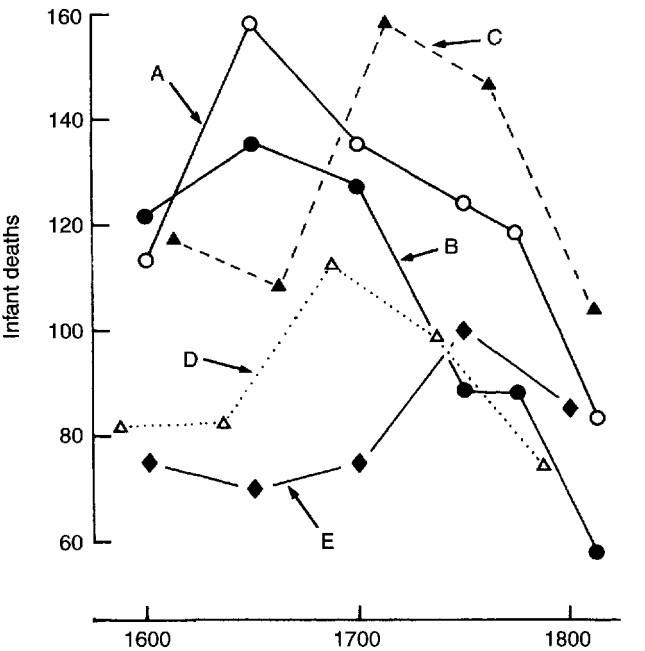


**Fig. 9.11** Exogenous infant mortality. A (empty circles): Penrith, male infants; B (filled circles): Penrith, female infants; C (filled triangles) Shropshire parishes (sexes combined); D (empty triangles): 16 parishes studied by Wrigley (sexes combined); E (closed diamonds): four parishes studied by Wrigley (see Table 11.7). Ordinate: cumulative exogenous infant mortality per 1000 baptisms. Abscissa: date at end of cohort under study.

Studies of the demography of the community at Penrith (Duncan *et al.*, 1992; Scott & Duncan, 1996, 1998, 1999a, b, 2000; Scott *et al.*, 1995, 1998) have shown how the conditions and nutritive levels there gradually improved. Short wavelength oscillations in infant and child mortality were strongly and significantly correlated with a comparable cycle in grain prices (see Chapter 10). However, at the end of the sixteenth and in the early seventeenth centuries, the community suffered a series of mortality crises when high grain prices synchronised with low wool prices, the local economy being dependent on both commodities (see section 4.10). As we have seen, exogenous infant mortality reached its peak at this time (Fig. 9.11).

Perinatal (endogenous) deaths, on the other hand, are dependent on the general health of the mother and on her nutrition during pregnancy. Endogenous mortality, except for a small rise in the period from 1700 to 1750, showed a marked and progressive fall in successive cohorts and, since it is the dominant component of infant mortality, we conclude that the steadily improving nutrition of the mother at Penrith (both before and during pregnancy) was of most importance in the amelioration of infant deaths at Penrith, rather than the quality and quantity of food available to post-neonates (Godfrey *et al.*, 1996).

Epidemics of lethal smallpox, largely confined to children, began at Penrith in the mid-seventeenth century (see section 15.8). Improving infant mortality resulted in a larger pool of children who could die of the disease and, hence, greater childhood mortality. Therefore, total annual child mortality (infants plus children under 15 years of age) remained almost constant until the mid-eighteenth century.

Since the community at Penrith was living under distressed conditions, maternal health and nutrition were probably sensitive to adverse circumstances. Boys almost always had the heavier endogenous and exogenous mortalities. When conditions at Penrith improved in the early eighteenth century, the population experienced a relatively prosperous period from 1700 to 1750, but, paradoxically, endogenous infant mortality rose slightly at this time.

## 9.4 The three social classes in the population at Penrith

The levels of infant and child mortality in a population depend on many social, economic, environmental, physiological and behavioural factors, so that it is often difficult to disentangle the diverse influences that may have the most important effects. In this section, we suggest that malnutrition before, during and after pregnancy was an important, subtle and not readily-detectable factor in determining the various levels of infant and child mortality in the pre-industrial community at Penrith. This study suggests how the different behavioural and physiological responses to a shortage of resources may have interacted in a saturated habitat.

The propensity of the population at Penrith to respond adversely to variations in the price of their staple food and the poor quality of the diet which we have described in section 9.5 suggest that this community is an ideal population to ascertain whether nutrition was an important determinant of the population dynamics. The results are assessed by social class, which is used as an indicator of the different economic constraints and hence of differences in nutrition. The division into elites, tradesmen and subsistence farmers is described in section 6.9.

## 9.5 Diets in the different social classes of a marginal community

The diet of the population in the saturated habitat at Penrith must have been far from well balanced nutritionally. In contrast with southern England (see section 1.9 and Tables 1.2 and 1.3), wheat was still little known among the poorer, subsistence classes in the northwest, who ate large amounts of oatmeal; although they had little meat (occasional bacon or fowl), milk was cheap and they added this to their daily ration of oats or barley. It is suggested that, although they had some peas and beans, their diet did not contain an adequate supply of esculent

vegetables (Gough, 1812) and it is possible that these people shared the popular superstition that fruit caused fevers (Drummond & Wilbraham, 1991). Cheese was rarely eaten.

The more affluent social classes (the elites) in Cumbria lived on very different diets in which meat, fish and cheese formed a much larger proportion of the meals than bread. The elite probably had adequate supplies of butter and there are frequent references to butteries in their wills and inventories. Honey was the main sweetening agent and beehives were found in many inventories of the elites and tradesmen. Ale, wine and brandy were drunk. However, these educated classes were influenced by the medical view that milk was appropriate only in infancy and should be discarded soon after weaning; very young children were therefore often put onto bread, soups and thin gruels. Esculent vegetables were rarely eaten, being regarded as the food of poverty (Drummond & Wilbraham, 1991).

The trades ate more meat than the subsistence class and their diet was probably better balanced with respect to proteins, fats and carbohydrates and, if supplemented by salted herrings (Marshall, 1973), would provide some intake of vitamins A and D. Eggs, however, were a delicacy (Drummond & Wilbraham, 1991).

The probable daily diets of the three social classes are given in Table 9.2; these are based on the information given by Eden (1797) and Drummond and Wilbraham (1991), but are modified to take account of the dietary habits of the northwest as shown by a study of the wills and inventories and as suggested by Thirsk (1967) and Marshall (1973). Eden (1797), in his book *The State of the Poor*, provides a remarkably detailed analysis, county by county, of the living conditions and diets that he observed in different classes, but particularly the labourers, throughout the country. He gives details of weekly consumption of the different foods available. Drummond and Wilbraham (1991) give an account of a labourer and his family of three children in Westmorland in 1797; they had an annual income of £30, of which £20 was spent on food. We present an estimate of their daily diet in Table 9.2, column (iv) which, at the end of the eighteenth century, was similar to the meagre rations of the subsistence farmer at Penrith 200 years before, except that potatoes now formed a substantial part of their daily fare. Probably the most important difference between the diets at Penrith in all three social classes in comparison with their counterparts in the south, but particularly in the subsistence group, was the lack of esculent vegetables, which are a valuable source of folic acid. Labourers in southern England grew vegetables and fruit in their cottage gardens (Drummond & Wilbraham, 1991). It must be emphasised that these values are only average estimates, but they provide an indication of the calorific value and the nutritive constituents in the daily diets in the seventeenth and eighteenth centuries in the northern territory. Obviously the constituents would vary with the season of the year and in times of hardship and plenty. It should be borne in mind that the 1790s, in particular, was a difficult decade because of wartime inflation and harvest failures.

**Table 9.2** Suggested average daily diets of the three social classes at Penrith, Cumbria, 1600–1800, compared with other diets in the seventeenth and eighteenth centuries.

| Elites | Trades | Subsistence | Westmorland diet in 1797 | Labourer's diet in southern England in the seventeenth century | Berkshire diet in 1797 |
|---|---|---|---|---|---|
| (i) | (ii) | (iii) | (iv) | (v) | (vi) |
| 4 oz cheese | 1 pt milk | 1.5 lb oats | 1 lb oats | 2 lb bread | 2.8 lb bread |
| 1.5 lb meat | 1 pt whey | 1 pt milk | 0.14 oz meat | 3.5 oz cheese | 1 oz cheese |
| 6 oz herring | 2 oz cheese | 8 oz peas | 1 pt milk | 9 oz peas | 1 oz butter |
| 1 oz butter | 1 oz bacon | | 0.5 oz butter | | 1 oz sugar |
| 1 lb bread | 2 lb bread* | | 1 lb potatoes | | 0.02 oz oatmeal |
| 1 pt wine | 2 oz peas | | (treacle) | | 0.3 oz bacon |
| 1 qt ale | 0.5 oz butter | | (sugar) | | 0.07 pt milk |
| (honey) | (honey) | | | | |

Diets are per adult per day. The subsistence diet of the seventeenth century (iii) improved slightly by the end of the eighteenth century, as shown by the diet of a labourer in Westmorland (iv). The subsistence diet (iii) can be compared with a labourer's diet in southern England in the seventeenth century (v) and in 1797 (vi). Data from Eden (1797) and Drummond and Wilbraham (1991).

* Or equivalent in oats or barley.

Table 9.3 gives an analysis of these diets with estimated energy values, protein and fat contents, and vitamin and trace element levels, together with the recommended daily dietary allowances in 1950 for adult men and women undertaking medium work and for pregnant and lactating women. Although the calorific value of the subsistence diet was suboptimal it was probably nearly adequate at good times of the year and in years of plenty. In the hungry season (see Chapter 13) and in years of high grain prices the energy supply would have been deficient in the subsistence diet, whereas the other two social classes exceeded the minimum calorific requirements. However, the most striking point about these analyses is the major deficiency in vitamins A, C and D in all three classes, except for vitamin D in the elites, and, as a consequence, scurvy, rickets and xerophthalmia were common ailments (Drummond & Wilbraham, 1991).

## 9.6 The maternal diet in pre-industrial Cumbria

We have seen (Chapter 7) how important was the mother's diet before and during pregnancy; malnutrition during these critical months could result in underweight babies and high infant mortality (Chen *et al.*, 1996). A mother's body size before pregnancy is an important determinant of the size of her baby; women of low body weight because of chronic undernourishment since childhood have small babies. Maternal deficiencies in vitamins A, C and D and folic acid influence foetal growth (Barker, 1998). Although the fat in the diet of the subsistence class at Penrith was boosted by eating oats instead of bread (see Table 9.1), the women would have had inadequate fat reserves. As we have seen, the mother stores fat in the first half of pregnancy and mobilises it in the second and so spares glucose for the foetus by switching to fat as her primary energy source (Barker, 1998). Low intakes of animal protein (as in the subsistence diet) in relation to carbohydrate were associated with low placental weight and lower birthweight (Campbell *et al.*, 1996). The monotonous, high-carbohydrate diet of the subsistence class would have contributed to the risk of infants of low birthweight.

The poorer sections of the community at Penrith would not have been able to rely on a consistent supply of even their meagre diet, which would have varied both seasonally and annually. Milk was scarce during the winter (Thirsk, 1967) and of poor quality (Drummond & Wilbraham, 1991). Oats, barley and wheat were in shortest supply, and consequently at their highest price, in the summer months before the harvest during the hungry season (see Chapter 13). Infant mortality at Penrith was also significantly associated with the regular oscillation in the annual price of grains (Duncan *et al.*, 1992; see section 10.8), and a sharply fluctuating food supply could exacerbate infant mortality, particularly for the poorer classes (Scott *et al.*, 1995; see section 10.4).

**Table 9.3** Analysis of the daily diets given in Table 9.2 together with modern recommended allowances.

| | | Recommended human daily dietary allowance | | | | | Population at Penrith (sexes combined) | | | Westmorland family in 1797 | Berkshire family in 1797 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Male | Female | | | | | | | | |
| | | Medium work | Medium work | Pregnancy | | Lactation | Elite | Trades | Subsistence | | |
| | | | | 1st half | 2nd half | | | | | | |
| Energy | (cal) | 3000 | 2500 | 2500 | 2750 | 3000 | 4750 | 3310 | 2960 | 2718 | 3575 |
| Protein | (g) | 87 | 73 | 93 | 102 | 111 | 200 | 100 | 110 | 84 | 120 |
| Fat | (g) | | | | | | 190 | 72 | 60 | 73 | 65 |
| Calcium | (g) | 0.8 | 0.8 | 0.8 | 1.5 | 2.0 | 1.3 | 1.6 | 1.0 | 0.98 | 1.6 |
| Iron | (mg) | 12 | 12 | 12 | 15 | 15 | 39 | 17 | 24 | 23 | 32 |
| Vitamin A | (IU) | 5000 | 5000 | 6000 | 6000 | 8000 | 2800 | 1950 | 700 | 1213 | 1268 |
| Vitamin B | (mg) | 1.2 | 1.0 | 1.0 | 1.1 | 1.4 | 1.5 | 1.9 | 2.3 | 3.0 | 2.8 |
| Vitamin C | (mg) | 20 | 20 | 40 | 40 | 50 | ? | ? | ? | 8.5 | 0.4 |
| Vitamin D | (IU) | — | — | 400 | 600 | 800 | 950 | 14 | 9 | 10.7 | 15.2 |
| Riboflavin | (mg) | 1.8 | 1.5 | 1.5 | 1.6 | 2.1 | 3.7 | 2.8 | 1.3 | — | — |

The analysis of the diets of the three social classes at Penrith (diets (i) to (iii) in Table 9.2) compared with the subsistence diet in 1797 (diet (iv)) and with the labourer's diet in southern England (diet (vi)). Recommended human daily dietary allowances taken from Long (1961).

## 9.7 Birth intervals following deaths in infancy at Penrith

The effect of the death of an infant should be apparent from the birth intervals following a child who survived infancy compared with those who died during the first 12 months of life (Hobcraft, 1992). The mean birth interval in elite families at Penrith was 27.6 months, which is significantly shorter than that recorded for the other social groups (tradespeople = 32.7 months; subsistence 33.5 months; $P < 0.001$; see Table 9.4).

**Table 9.4** Mean birth intervals at Penrith, Cumbria, 1600–1800, following offspring who either died or survived infancy.

| Social group | Sex | Birth intervals following: (1) Children who survived infancy | | (2) Children who died in infancy | | (3) Difference in the birth intervals (column 1 − column 2) |
|---|---|---|---|---|---|---|
| | | months | *n* | months | *n* | months |
| Elite | Male | 28.2 | 206 | 22.1 | 55 | 6.1 |
| | Female | 26.9 | 203 | 25.3 | 28 | 1.6 |
| | All | 27.6 | 409 | 23.2 | 83 | 4.4 |
| Tradesmen | Male | 32.3 | 643 | 22.3 | 158 | 10.0 |
| | Female | 33.1 | 623 | 24.8 | 115 | 8.3 |
| | All | 32.7 | 1266 | 23.4 | 273 | 9.3 |
| Subsistence | Male | 34.0 | 547 | 22.2 | 142 | 11.8 |
| | Female | 33.0 | 550 | 20.3 | 113 | 12.7 |
| | All | 33.5 | 1097 | 21.4 | 255 | 12.1 |

Analysis of variance: (i) Social classes significantly different ($P < 0.001$); Scheffe test shows that elites are significantly different from tradesmen and subsistence ($P < 0.05$). (ii) Columns (1) and (2) significantly different ($P < 0.001$).

However, if an infant died, the mean time to the next birth decreased to 23.2 months for the elite, 23.4 months for tradesmen and to 21.4 months for subsistence families, i.e. the next birth arrived, on average, 5 (for the elite) and 12 (for the subsistence class) months earlier than if the preceding child had survived. The small differences between the social groups were not significant and the shorter interval for the subsistence group may reflect the fact that the mean age at death of the subsistence infant was 2 months earlier than for the other social groups.

Furthermore, there were differences in the intervals following the deaths of female and male infants and between the social groups, as shown in Table 9.4. In the elite category, there was little difference in the birth interval following the death of a female infant, whereas the birth interval was shortened from 28 to 22 months following the death of a male infant. Amongst the tradesmen, the birth interval was substantially reduced for both sexes: by 10 months for male and by 8

months following the death of a female infant. Although there was little difference between the sexes, the interval in subsistence families was markedly reduced from 34 to 22 months following male and from 33 to 20 months following female infant deaths.

These results may be compared with other reports of historic populations where the largest differences in birth intervals were associated with extended breast-feeding, whereas the smallest differences between normal birth intervals and the intervals following an infant death were recorded when breast-feeding was uncommon (Knodel, 1978). The results of Table 9.4 suggest, therefore, that the elite mothers at Penrith followed different practices in the duration of breast-feeding and in the timing of the introduction of supplementary foods. Furthermore, the elite mothers seemed to have different practices dependent on the sex of their offspring. It is suggested that breast-feeding and weaning may provide the essential clue to the apparent differences in the reproductive success and investment strategies of the different social groups at Penrith. These differences are explored further in the next section by the use of Bourgeois-Pichat plots.

## 9.8 Infant mortality in the three social classes at Penrith

The Bourgeois-Pichat plots of cumulative infant mortality at Penrith for the three social classes for males are given in the upper part (lines A, B and C) and those for females in the lower part (lines D, E and F) of Fig. 9.12. The results are summarised in Table 9.5 which shows the exogenous mortality rates (i.e. the slopes of the lines) before and after the suggested points of the start of weaning.

The plots for male and female infants of tradesmen at Penrith (Fig. 9.12; lines B and E) are similar to one another, with a break at 5–6 months. This change in the slope of the line is not sharp and lines have been fitted for 1–3 months and for 8–12 months. These slopes above and below the break were significantly different from one another (Males: $P = 0.01$; ratio = 2.1. Females: $P = 0.001$; ratio = 2.4). There is no significant difference between the pattern of the plots for males and females. The cumulative mortality of male infants of the tradesmen lies above that for females and the extrapolations of lines B and E show that this is entirely because of the higher endogenous mortality of male infants in this class. We suggest that the marked change in the slopes (i.e. the breaks) of the infant mortality rates of both sexes of the tradesmen class was associated with the start of weaning. Since this change in the slope of both lines took place gradually between 4 and 7 months (Fig. 9.12), we conclude that the tradesmen mothers showed individual variation and first introduced non-breast-milk foods for both males and females over this period.

In contrast, the Bourgeois-Pichat plots for the elites at Penrith (Fig. 9.12; lines C and F) appear to be completely different for male and female infants. Both show a clear break, which is at 2 months for males, but at 8 months for females;

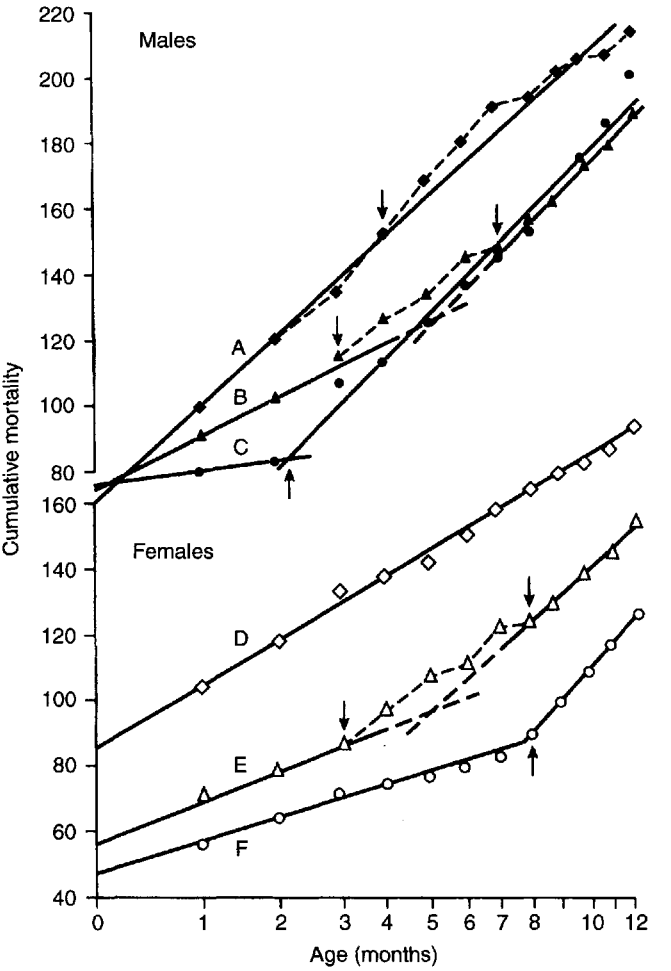


**Fig. 9.12** Bourgeois-Pichat plots for infant mortality at Penrith, 1600–1800. A–C: males; D–F, females. A and D: subsistence class; B and E: tradesmen; C and F: elites. Ordinate: cumulative infant deaths after the first month of life (deaths/1000 live births). Abscissa: Age (months) expressed as $[\log (n + 1)]^3$, where $n$ = age in days. The fitted lines for A and D are shown; other lines are fitted above and below the break point. Arrows show suggested start of weaning (A, C and F) or period over which weaning began (B and E).

the slopes of the lines of exogenous mortality for males and females after the break did not differ significantly. It is the timing of the break and not the slopes of the lines wherein the plots for male and female elite infants display their critical differences. Comparison of the slopes after the break (post-weaning; 8–12 months) shows that there was also no significant difference between the male infants of the elites and trades, although there was a small difference in the slopes of the female infants of the elites (b = 11.4) and trades (b = 9.4) which was significant ($P = 0.02$). Furthermore, there was no significant difference between the

elites and the trades in the rates of female infant mortality *before* the break (i.e. during breast-feeding; 1–4 months). We conclude that the markedly different timing of the break in the plots for male and female elite infants suggests that this class practised a form of differential parental investment, with a very early start of weaning for the boys in comparison with their sisters.

The subsistence infants show the highest total mortality and, in contrast with the elites and trades, the Bourgeois-Pichat plots for both sexes do not show a clear break (Fig. 9.12, lines A and D). The line for female infants does not depart significantly from linearity and their total exogenous mortality is the same as that of the female infants of tradesmen. We conclude that subsistence female infants were exclusively breast-fed for at least 6 months and probably longer. The line for subsistence male infants departs significantly from linearity, suggesting that supplementary feeding for some may have been introduced after 5 months, thereby accounting for the higher exogenous mortality than for subsistence females. The overall slopes of these lines for the subsistence infants are also different from the Bourgeois-Pichat plots for the other two social groups: for females ($b = 5.75$; does not depart significantly from linearity) the mortality rate was higher than the mortality rate during breast-feeding shown by the other social groups, whereas it was lower than the post-weaning mortality rate of the other two social groups, perhaps because of a long continuation of breast-feeding in the subsistence group.

To summarise, the wives of tradesmen apparently did not differentiate between their children and breast-fed both exclusively for at least 6 months. Female infants of the subsistence group were breast-fed for at least 6 months (and probably much longer), but some males may have had supplementary foods introduced at 4–5 months. Wives of elite men probably breast-fed their infant sons only until they were 2–3 months old, when supplementary feeding was introduced, whereas their infant daughters were probably exclusively breast-fed for the first 8 months of life. However, this suggested feeding practice for elite female infants does not agree with the effect of the death of a female elite child in infancy on birth intervals (see Table 9.5) and it is assumed that nursing by the mother of daughters was probably not common. It is suggested below that the answer to this apparent discrepancy is that the female infants of elite families were wet-nursed and that they enjoyed a longer period of breast-feeding without supplementary foods.

## 9.9 Breast-feeding and wet-nursing at Penrith

There is no direct, historical evidence concerning the practice of wet-nursing in the sixteenth and seventeenth centuries at Penrith, but inspection of the 1851 census shows the presence of a small number of nurse-children. Is there evidence of a differential investment both between the sexes of the offspring and between

**Table 9.5** Birth intervals and endogenous and exogenous infant mortality for each social group at Penrith, Cumbria, 1600–1800.

| Social Group | Mean birth interval | No of families | Sexes combined | | Sex | Total infant mortality | Endogenous infant mortality | Total exogenous infant mortality | Slope of exogenous mortality | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Total endogenous infant mortality | Total exogenous infant mortality | | | | | Preweaning | Post-weaning |
| Elite | 26.8 (492) | 120 | 65 | 102 | Male | 202 | 76 | 126 | (<2) | 9.94 |
| | | | | | Female | 127 | 46 | 82 | 2.93 | 11.4 |
| Tradesmen | 30.0 (1539) | 451 | 68 | 105 | Male | 190 | 75 | 115 | 4.8 | 10.0 |
| | | | | | Female | 154 | 57 | 97 | 3.9 | 9.4 |
| Subsistence | 31.2 (1352) | 508 | 77 | 122 | Male | 215 | 69 | 146 | 8.96 | 8.96 |
| | | | | | Female | 183 | 85 | 98 | 5.75 | 5.75 |

Mean birth interval expressed in months; *n* given in brackets. Elites have a significantly shorter birth interval than the other two social groups ($P < 0.001$). Mortalities expressed as per 1000 live births. Total exogenous mortality = total infant mortality minus endogenous mortality. Total mortality for male and female elites significantly different, $P < 0.05$. Slopes of cumulative exogenous mortality calculated from the regression lines fitted to the Bourgeois-Pichat plots.

the social groups in the historic community at Penrith with reference to the practices of breast-feeding? The interval until the birth of the next elite infant was 6 months shorter if a son died, than if he survived (Table 9.4). If a mother were breast-feeding, the death of an infant would reduce the interval before the next birth because of an early return to ovulation. However, if an elite daughter died, there was only a 2-month difference in the elapsed interval until the birth of the next infant, whereas Fig. 9.12 suggests, contrary to expectation, that these infants were breast-fed for 8 months, and certainly for longer than their brothers. No such discrepancy exists for tradesmen or subsistence farmers. We suggest that elite sons were breast-fed by their mothers who introduced supplementary feeding early, with the attendant risks of infection and poor diet, whereas the breast-feeding of daughters was delegated to wet-nurses. The consequence of this practice was that, rather than contributing to higher mortality risks, as has been suggested in previous studies, these female infants received better quality food in the breast milk and for a longer period, which significantly reduced their levels of mortality when compared with their infant brothers.

The effect of lactation on mortality is strongest during the first 6 months of life when breast milk is most beneficial to infant survival (see Chapter 8). Infants who are not breast-fed, or who are weaned early, generally have higher mortality and this appears to have been the fate of the male, elite-born infants at Penrith. Therefore, although the wet-nursing of the daughters in the elites might be regarded as a form of under-investment in females, the increased period of breast-feeding by wet-nurses apparently resulted in much improved survival prospects for these infants when compared with their male counterparts, simply because of their improved nutrition and the reduction in the chances of infection (see section 15.6) during a critical period of their lives.

A consequence of wet-nursing elite female infants and of a shorter period of breast-feeding for elite males at Penrith was a markedly reduced birth interval in these families which led to a higher fertility when compared with the other social groups. The effect of breast-feeding on suppressing menstruation is discussed in section 6.12. A woman having a greater number of pregnancies can incur costs in terms of a poorer physical condition and an impaired reproductive physiology, which eventually will not support further reproduction. This condition has been identified as a major factor in the empirical relationship between close birth intervals and high infant mortality risks, because there would be only a limited time for the mother to recuperate which, in turn, would affect the development and nourishment of the foetus and of the young infant during lactation because of the poorer quality of the milk (Bohler & Bergstrom, 1995; Ronsman, 1996). This suggested differential investment between the sexes may have contributed to the higher mortality experienced by the male infants of the elite; maternal efficiency was compromised by the short birth intervals (shown by Table 9.4), which probably resulted in children of below average birthweight and greater susceptibility to various infections. The girls of the elite were provided with greater

protection via satisfactory and prolonged breast-feeding, whereas the boys were inadequately breast-fed by their mothers for only short periods of time followed by early weaning, perhaps because of the lowered fat content of the milk which necessitated early supplementary feeding.

In contrast, there was no indication of differential parental investment in early infancy among the tradesmen; apparently males and females were breast-fed for a similar duration because the typical Bourgeois-Pichat plots of Fig. 9.12, lines B and E, both show a sharp up-turn in mortality between 4 to 7 months and the mean birth interval after male or female children was the same (Table 9.4). The birth intervals tended to be longer than in the elite group, as is usually found in societies where the mothers practise prolonged, on-demand feeding. These two important factors of prolonged breast-feeding and consequently the longer intervals between births would have allowed a recuperative period for the trades mother, leading to an effectively greater amount of parental investment for each child and to the relatively lower rates of infant mortality for this middle stratum at Penrith.

Child mortality for both sexes was high in the lowest social class, the subsistence farmers (Table 9.5), and surviving family size was only 2.5 – barely above replacement level, thereby effectively maintaining this community in a steady state over 200 years. The Bourgeois-Pichat plots for these poorer families are shown in Fig. 9.12, lines A and D; that for boys shows a small deviation after 5 months and we conclude that subsistence daughters were breast-fed for at least 6 to 8 months, whereas some of the boys may have been weaned at 5 months. The suggested nursing practices in the three social groups at Penrith are summarised in Table 9.6.

**Table 9.6** Summary of suggested nursing practices in the different social groups at Penrith, Cumbria, 1600–1800.

| Social group | Male | Female |
|---|---|---|
| Elite | Breast-fed for 2 months | Wet-nursed for 8 months |
| Trades | Breast-fed for about 6 months (range = 4 to 7 months) | Breast-fed for about 6 months (range = 4 to 7 months) |
| Subsistence | Breast-fed for at least 5 months | Breast-fed for at least 6 to 8 months |

One conclusion that can be drawn from sections 9.7 and 9.9 is that the interval between successive births is of great importance in determining the health and physiological efficiency of the mother and, hence, in regulating neonatal mortality. A woman needs to recover completely from one pregnancy before beginning the next.

The median birth-to-conception interval among women in less developed countries who breast-feed their infants is approximately 3 years, but the interval

might be shorter in women from better nourished populations, even if their practices of breast-feeding and sexual intercourse are similar to those of women in the less developed areas. Well-nourished women who do not breast-feed their infants may ovulate as soon as 27 days after delivery, whereas exclusive breast-feeding delays the return to ovulation by a variable length of time, with less than 50% of women ovulating by 6 months post-partum. The actions of a woman have a profound influence on the interval between pregnancies, and the 'optimal' interval – that is, the interval associated with the greatest probability of giving birth to a normal infant born at full term – may be more important to her than a 'natural' interval (Klebanoff, 1999).

This conclusion is supported by a recent study (Zhu *et al.*, 1999) which analysed data from the birth certificates of 173 000 singleton infants born alive to multiparous mothers in Utah from 1989 to 1996. The risk of low birthweight, preterm birth or small size for gestational age was 30 to 40% higher among infants conceived less than 6 months after a birth than among infants conceived 18 to 23 months after a birth.

Published reports are remarkably consistent in showing that short interpregnancy intervals are associated with an increased risk of a variety of adverse perinatal outcomes and less consistent in showing that long intervals are also associated with this risk. However, whether extreme intervals themselves carry a risk or are simply markers that a woman is already at risk is uncertain. Norwegian women with registered births who became pregnant after extremely short or long intervals had smaller children than those who became pregnant after intermediate intervals. However, the birthweights of Norwegian children who were born before short or long intervals were also lower than those of the children whose births preceded intermediate intervals. This finding suggests that much of the apparent risk of extreme interpregnancy intervals may actually be due to other factors (Klebanoff, 1999).

We discuss the importance of the duration of the interbirth interval on a woman's reproductive efficiency in the detailed case study given in section 9.11.

## 9.10 Indicators of nutritional deficiency in pre-industrial Penrith

(1) Sub-fertility in the early child-bearing years has been described in section 6.9 (see Fig. 6.1). In summary, the elites had a subfertility only up to age 25 years, being normal thereafter, suggestive of a delayed menarche. The trades remained subfertile when compared with other English parishes until age 40 years. Subfertility persisted in the subsistence group throughout the child-bearing years, suggesting that there were other factors operating in addition to a delayed menarche in this class.

(2) Endogenous infant mortality. Table 9.7 shows that, for the period 1557–1649, the overall endogenous mortality at Penrith was greater than that

**Table 9.7** Endogenous and exogenous infant mortality in different cohorts at Penrith, Cumbria, compared with other English parishes.

| | Endogenous infant mortality ($1000q_x$)[1] | | Exogenous infant mortality ($1000q_x$)[1] | |
|---|---|---|---|---|
| | Penrith | English | Penrith | English |
| 1557–99 | 143 | 77 | 107 | 93 |
| 1600–49 | 98 | 84 | 150 | 75 |
| 1650–99 | 88 | 88 | 133 | 88 |
| 1700–49 | 87 | 82 | 118 | 108 |
| 1750–74 | 53 | 61 | 110 | 102 |
| 1775–1812 | 31 | 46 | 78 | 100 |

Mortality (sexes combined) at Penrith taken from family reconstitution study. Mortality in 26 English parishes calculated from the data provided by Wrigley *et al.* (1997).
[1] Burial rates per 1000, expressed as $q_x$, the probability at age $x$ of dying before reaching age $x + n$.

found in 26 representative English parishes. Endogenous mortality at Penrith ameliorated after 1650 and was then comparable with southern England. Endogenous mortality rates of the elites and tradesmen at Penrith were 65 and 68 respectively, in contrast with a value of 77 for the subsistence class. We conclude that high endogenous mortality rates were associated with an inadequate maternal diet in pregnancy.

(3) Preweaning, exogenous infant mortality. Table 9.7 shows that the community at Penrith was characterised by a high level of exogenous mortality in comparison with other English parishes. There were also differences between the social classes and between the sexes (Table 9.5). The elites and tradesmen were comparable in that the Bourgeois-Pichat plots both showed clear breaks (associated, it is suggested, with the start of weaning) and both had broadly comparable preweaning exogenous mortality rates for boys and girls (Table 9.5). The elites differed (as discussed above) in their poor quality breast-feeding of short duration (because of maternal depletion) of their male children, wet-nursing of their female children and (critically) in the points of weaning. The Bourgeois-Pichat plots of the subsistence class, in contrast, showed no clear breaks (Fig. 9.12). This is suggestive of late weaning, but exogenous mortality was at a high level (Table 9.5), and was probably related to low birthweight infants and overall poor maternal nourishment.

(4) Post-weaning, exogenous infant mortality. Again, the elites and tradesmen showed similar high rates of infant mortality in both sexes after the start of weaning (Table 9.5). This was probably linked with the associated risk of infection. We conclude that the different nursing practices were the major determinants of exogenous mortality in these classes, whereas in the sub-

sistence class the poor diet of the mothers during pregnancy and lactation was of the most importance.

(5) Neonatal versus post-neonatal mortality. We have shown that oscillations in infant mortality at Penrith correlated with oscillations in wheat prices: high neonatal mortality correlated with high wheat prices in the preceding year (during pregnancy), whereas the peaks of post-neonatal mortality were found during the years of high prices, suggesting that they had a direct effect (Scott *et al.*, 1995; section 10.8). We conclude that, in this community living under marginal conditions, food shortages during pregnancy or during the first year of life could have had severe effects on endogenous or exogenous mortalities, respectively.

## 9.11 Overview of the effects of malnutrition and nursing practices in the different social classes at Penrith

We can summarise the conclusions from the preceding sections as follows. The tradesmen class (Fig. 9.13) probably represents the norm for the people at Penrith. They probably enjoyed adequate nutrition in most years and maternal health was probably above average for the community. Prolonged breast-feeding and later weaning had two important consequences: a relatively low exogenous male mortality and longer birth intervals than in the elites, thereby promoting improved maternal health during the next pregnancy. This relatively simple pattern of events is summarised in Fig. 9.13, which shows the essentially stable and favourable lifestyle of the tradesmen class at Penrith.

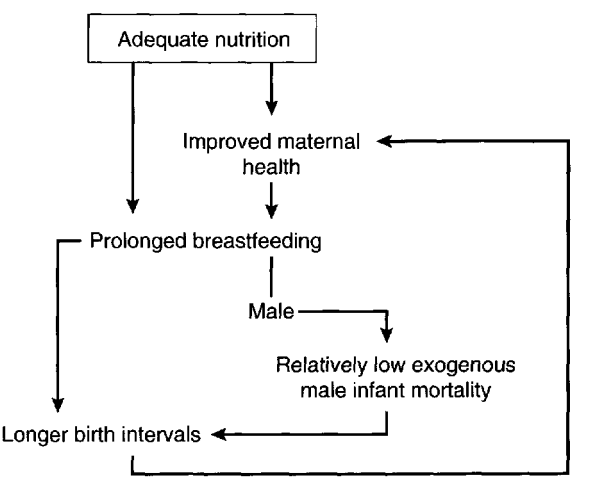


**Fig. 9.13** The suggested interacting effects of nutrition and breast-feeding practices on infant mortality in the tradesmen class at Penrith.

The subsistence class suffered from poor nutrition and would have been the section of the community that was most susceptible to fluctuations in grain prices, leading to a complex web of interacting events that is summarised in Fig. 9.14. Maternal health would have been impaired during both pregnancy and lactation because of a poor diet, resulting in infants of low birthweight, high endogenous mortality, a reduction in the quality of breast-feeding and higher exogenous mortality. This class apparently breast-fed their female infants for at least 6 months, but probably weaned their male infants at an earlier age and, consequently, exogenous mortality was higher in male than in female infants (Table 9.5; Fig. 9.14). High infant mortality contributed to the shorter birth intervals and impaired maternal health (Hobcraft, 1992), acting synergistically with the poor overall nutrition, so that a detrimental feedback loop was probably established, with the infants being consistently born with low birthweights (see section 7.18). Thus, infant mortality in the subsistence class was primarily governed by the sharply fluctuating and the poor quality of the food supply of the mothers. Figure 9.14 illustrates in detail a vicious circle from which the subsistence mother could not escape.

The elites (Fig. 9.15) enjoyed better nutrition than the other social classes (although it was probably still deficient in certain vitamins and minerals) but,

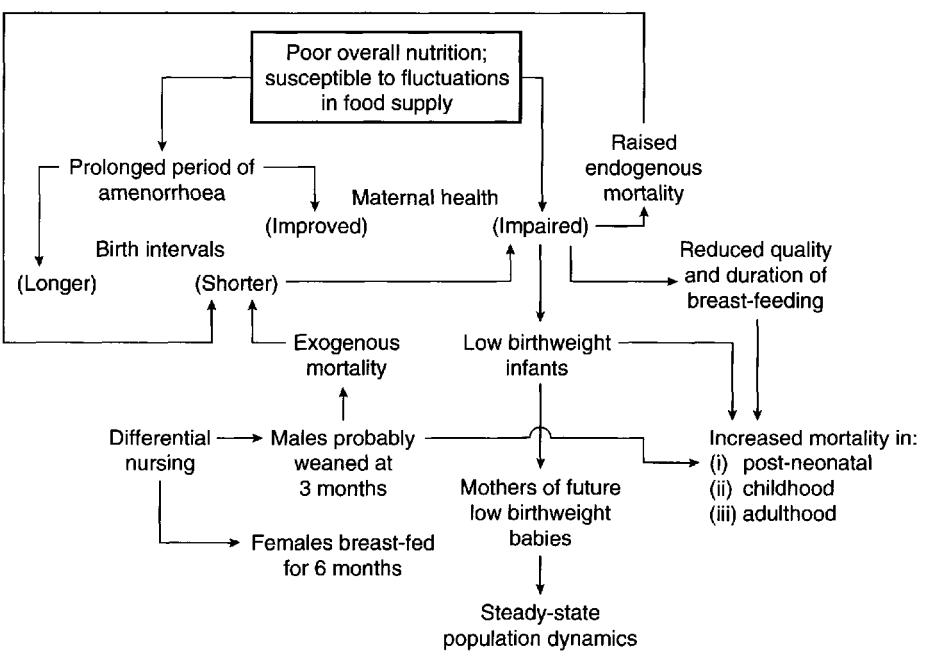


**Fig. 9.14** The suggested interacting effects of nutrition and differential nursing practices on infant mortality in the subsistence class at Penrith. A poor level of nutrition leads to the feedback loops causing impaired maternal health, maternal depletion and low birth-weight infants.

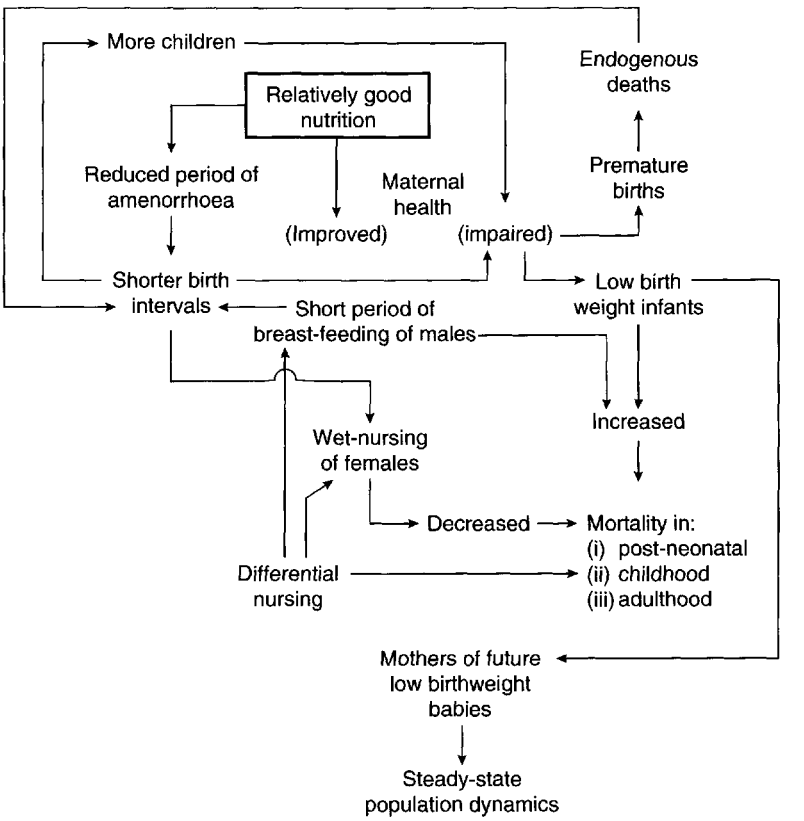


**Fig. 9.15** Suggested interacting effects of nutrition and differential nursing practices on infant mortality in the elite class at Penrith. The differential practices with respect to breast-feeding established the feedback loops that caused impaired maternal health and low birth-weight infants and largely abnegated the better nutritive standards enjoyed by this class.

paradoxically, this did not lead to an improved total infant mortality. Indeed, male endogenous mortality was greater in the elites than in the two other social groups, and exogenous mortality was higher than in the tradesmen class (Fig. 9.12). The practice of wet-nursing female infants and the short duration of breast-feeding of males (perhaps because the suckling infant did not thrive) resulted in shorter birth intervals which impaired maternal health and so led to maternal depletion, premature babies, increased endogenous deaths, lower birthweight infants and higher exogenous mortality, thereby establishing the adverse feed-back loops shown in Fig. 9.15. Early weaning of male elites also raised their exogenous mortality whereas prolonged wet-nursing of the female infants pro-vided them with considerable protection and, consequently, reduced their exo-genous mortality (Table 9.5).

## 9.12 Contribution of malnutrition and differential nursing practices to steady-state dynamics at Penrith

The nutrition of the majority of the mothers at Penrith would have been inadequate, probably even in years of plenty, as shown by the marked subfertility that persisted from 1600 to 1800 in all three social classes in the 17- to 30-year age groups (section 6.9). Women who themselves are born with a low birthweight have a tendency to produce lower birthweight infants (Godfrey *et al.*, 1996) and, consequently, girls born to malnourished mothers carried this handicap forward and grew up to be unhealthy mothers themselves and the effect would be perpetuated in successive generations (see section 7.18). An amelioration of infant mortality at Penrith, therefore, awaited improvements in maternal nutrition and, without being aware of the implications of their actions, an early and sustained investment of food for daughters would be important for their ultimate reproductive success. The strategy of investing in female infants (see section 9.8) may, in the long run, have been crucial to the population dynamics at Penrith, enabling them eventually to escape from the steady-state conditions by a reduction in infant mortality, which resulted in the population boom which eventually began in 1750 (Duncan *et al.*, 1992).

In conclusion, we have seen that Penrith was in steady state during the period 1557–1750 (see section 5.3); mean annual births equalled mean annual deaths and were constant throughout. Forty-four per cent of children died before reaching the age of 15 years, and this overall level of mortality was also constant for over 200 years (Scott *et al.*, 1998). The average family size was only four children which, with the prevailing high rates of infant and child mortality resulted in a level of reproduction that was only just sufficient for replacement (Fig. 9.16 and Chapter 6).

Since the majority of the population at Penrith were subsistence farmers, the health and nutrition of the mothers of this lowest social group had the most important consequences for the population dynamics. They were probably particularly sensitive to adverse environmental conditions, and a shorter and less efficient reproductive span for this major section of the population because of poor nutrition appears to be the most likely explanation for the relatively low levels of fertility during the sixteenth and seventeenth centuries. Chronic malnutrition of the mother coupled with maternal depletion would have led to poorly-developed fat stores and, hence, to lowered fertility (see section 6.1). If subfecundity contributed to low fertility, then the rise in fertility that occurred during the first half of the eighteenth century may be because of better nutrition, for it is only during this time that fertility rates approached the level found for other comparable historical populations.

There may have been a movement away from the subsistence level and an improvement in living standards during 1700–49: there was a fall in the mean birth intervals, an increase in the age of mother at the birth of her last child, a rise

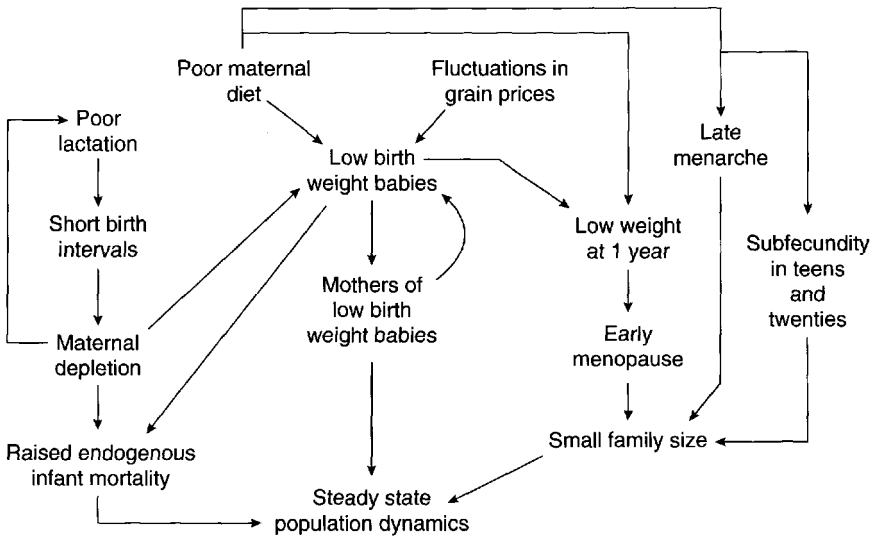


**Fig. 9.16** Diagram to show the suggested interacting effects of maternal nutrition on fertility, infant mortality and the population dynamics at Penrith.

in the average family size and a small reduction in teenage subfecundity (Scott & Duncan, 1999a). This possibly reflects an improvement in the standard of living for the subsistence group to the level enjoyed by the tradesmen. The reason for the improvement in conditions was because, paradoxically, this was the period of the agricultural depression (1730–50), when there was a changing balance between arable and pastoral areas. The fall in grain prices and increased demand for meat and dairy products favoured the livestock farmer of the northern territory; the cattle trade was buoyant and provided some protection against the adverse effects of the agricultural depression. This apparent halcyon period for Penrith was short-lived, however, because a cattle plague broke out in the south of England in 1745 which reached the north in 1749. The adverse effects of falling demand were immediately apparent, with a noticeable rise in the number of burials of paupers in 1748 and 1749. The second half of the eighteenth century was a period of rising grain prices that reached a zenith during the Napoleonic Wars. The result was the reappearance of the significant relationship between the oscillations in grain prices and the cycles in the burials of infants, children and adults that was much in evidence during the sixteenth and seventeenth centuries (see Chapter 10).

During the population boom of the second half of the eighteenth century, family size at Penrith fell again; there was a slight rise in the mean birth interval and a fall in fertility. The age of the mother at the birth of her last child increased by 2 years. The most marked improvement, during the period, occurred in infant mortality, which fell from 21% to 13%, mainly because of a reduction in mortality early in the neonatal period (Scott & Duncan, 1999a; see Chapter 11).

Penrith was a market town and the increase in its population after 1750 was linked to a rise in the tradesmen class who, as we have seen, probably had the most favourable nutrition and apparently did not employ differential breast-feeding practices.

Thus, the escape from steady-state conditions was probably related to an amelioration of infant mortality (see Chapter 11) rather than to an improvement in fertility. The adverse effects of female malnutrition would have been perpetuated in successive generations and a reduction in the rate of infant mortality and stillbirths would not be expected until there was a sustained improvement in nutritional status and in weight gain prior to and during the pregnancy of mothers of the preceding generation. This may have occurred in Penrith when, during the period 1700 to 1750, an amelioration in the effect of high wheat prices (see above) led to better nutrition for mothers who conceived during this period and, consequently, in the birthweight of their daughters, who then produced the next generation which experienced a marked reduction in the rate of infant mortality. In this way, the beneficial effects of the halcyon period, 1700–50, were not fully expressed until after 1750, when endogenous infant mortality fell sharply to very much lower levels (Table 9.7) and the population escaped from steady-state dynamics and the population boom began.

Chapter 10

# Exogenous Cycles: A Case Study

## 10.1 Interactions of exogenous cycles

An analysis of infant, child and adult mortalities can be derived only from a family reconstitution study which we have carried out for the parish of Penrith in the Eden Valley, Cumbria (see section 4.9), and in this chapter we present an intensive case study of the interactions of the population cycles, building on the matrix modelling described in section 5.2. The main objective of this study is to show how the methods of time-series analysis can be used to elucidate the population dynamics of a single community and to determine the economic and geographical factors on which they are based. The findings are derived from quantitative data and statistical confidence limits are given. These ideas are developed progressively through this chapter which reveals the complexity of the interacting factors that regulate the exogenous population cycles, which, in turn, are superimposed on the endogenous, longer wavelength oscillations that are described in Chapter 5. The contribution of smallpox epidemics to the childhood mortality cycles at Penrith will be described in section 15.8.

The thesis presented in this section is that the oscillations in grain prices, described in section 3.3, and the corresponding availability of food, had profound, but subtle effects on the population at Penrith and was one of the major factors in driving the short wavelength oscillations in deaths and, indirectly, in births. Since these exogenous cycles were found in boom as well as steady-state conditions, it is evident that they were probably not dependent on homeostatic dynamics, nor on density-dependent constraints. The national prices for wheat, oats and barley moved in synchrony and, as explained in Chapter 3, the wheat price series has been used as a general index of nutritive levels. The short wavelength cycle in wheat prices did not correlate with weather conditions but was apparently driven by economic factors and the short-term effects of a good or bad harvest (auto-regressive factors; see section 3.10); it was of the most direct importance in driving the exogenous mortality cycles at Penrith.

Spectral analysis of the total burial series at Penrith (see Fig. 5.1A) for the whole period, 1557–1812, reveals a short wavelength cycle in mortality (period ~5 years). However, this series is complex, being compounded of adult, child

and infant deaths and, when these series are analysed separately, it is apparent that the periodicity predominantly arises from a persistent 5-year oscillation in child burials (0 to 15 years of age) which is evident by eye in the unfiltered series and is revealed more clearly after filtering in Fig. 10.1. Spectral analysis of the unfiltered series shows that the wavelength of this oscillation overall was predominantly 5 years (Fig. 10.2), but it was non-stationary before 1700, changing from 6 to 5 years. During the first 100 years (between 1557 and 1650), when the parish experienced several major mortality crises (see section 4.8), the oscillations in adult and child burials are synchronous and this cross-correlation is highly significant ($P < 0.001$), with a cross-correlation factor (ccf) of +0.9.

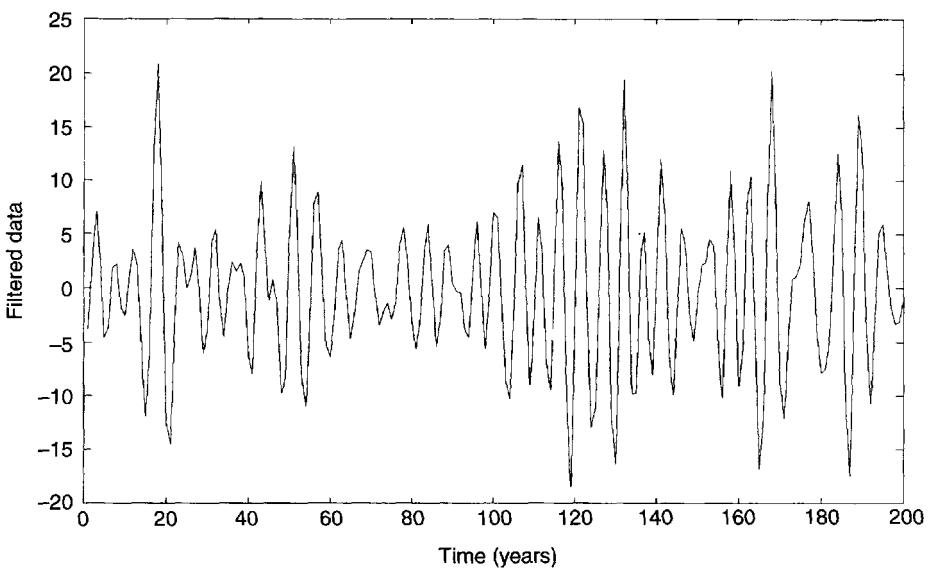


**Fig. 10.1** Burials of children plus infants at Penrith, 1607–1807, filtered to reveal the short wavelength oscillation (filter window = 4 to 10 years). Data derived from family reconstitution study.

Are these cycles in child and adult mortality at Penrith exogenous, being a direct response of the population to famine and hardship? As a first step to answering this question, the family reconstitution study of Penrith has been used to divide the total child burial series into infants (0–12 months) and children aged from 1 to 14 years, because infant mortality responded differently to fluctuations in the wheat price series. In brief, the short wavelength oscillations in child (aged from 1 to 14 years) and adult burials were both strongly correlated with wheat prices for the first 100 years, but after 1650 they responded differently.

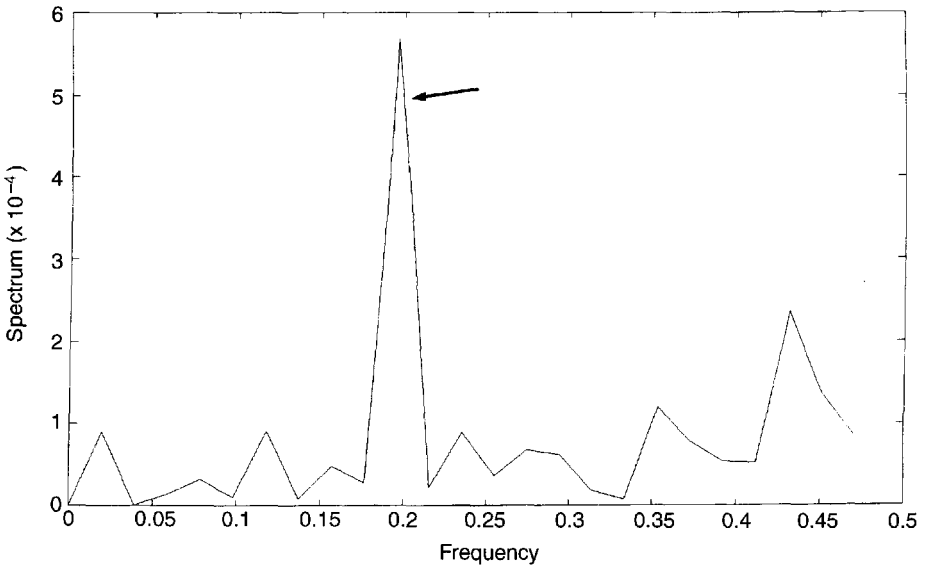


**Fig. 10.2** Spectral analysis of the child burial series (ages 1 to 14 years) at Penrith, 1700–50. Unfiltered data. Significant peak ($P < 0.05$) at a wavelength of 5.1 years (see arrow).

## 10.2 The short wavelength cycle in child burials

Spectral analysis of the burial series of children aged 1–14 years in different cohorts shows a 5–6 year oscillation from 1557 to 1700, which became strongly 5-yearly thereafter. Coherence analysis of the period 1557–1650 shows that child burials were strongly correlated with wheat prices in the short waveband ($P < 0.01$). The cross-correlations of child burials vs wheat prices can be summarised as follows:

| | | |
|---|---|---|
| 1557–1600: | ccf = +0.6 | Lag = 2 years, i.e. cycles not synchronous |
| 1600–1650: | ccf = +0.2 | Zero lag |
| 1650–1700: | ccf = +0.3 | Zero lag (see Fig. 10.3) |
| 1700–1750: | ccf = −0.4 | Lag at +1 years, i.e. child burials are no longer synchronous with the corresponding oscillation in wheat prices |
| 1750–1812: | ccf = +0.4 | Zero lag, i.e. the two series are again in synchrony. |

Thus, a short wavelength oscillation in the child burials series at Penrith emerged in the sixteenth century with a periodicity of 5–6 years, which developed into a clear 5-yearly oscillation after 1700, as can be seen in Fig. 10.1 and is confirmed by the spectral analysis in Fig. 10.2. This mortality cycle was closely associated with a corresponding oscillation in wheat prices which also appeared at the end of the sixteenth century (section 3.3), suggesting that a rise in child

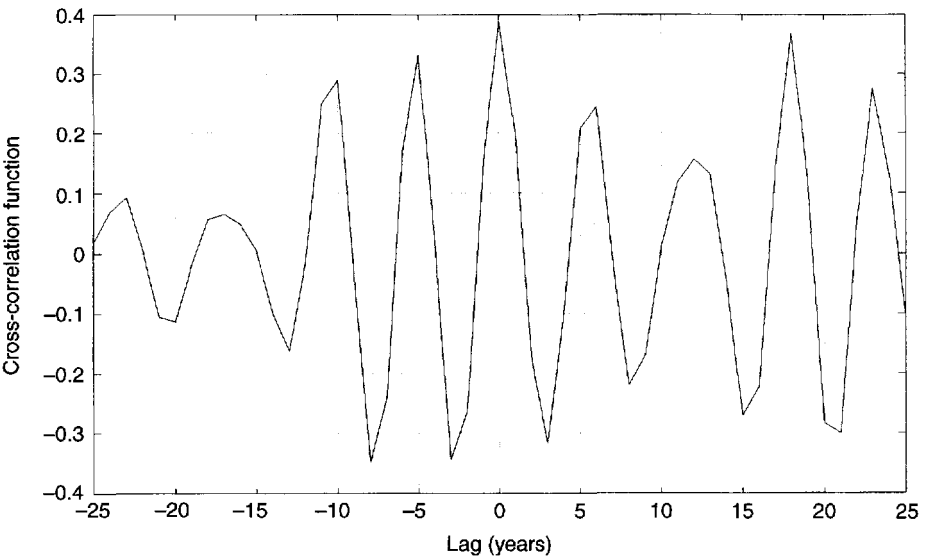


**Fig. 10.3** Cross-correlation function, burials of children aged 1 to 14 years versus wheat prices, 1650–1700. Filter window = 4 to 10 years. Maximum ccf (+0.4) at zero lag shows that the two series are in synchrony.

deaths may have been associated with years of high wheat prices and the consequent malnutrition.

The short wavelength oscillation in child burials became more regularised after 1650 and spectral analysis for the period 1700–75 (see Fig. 10.2) shows that the 5-year oscillation was now strongly established. Are these cycles associated with periodic outbreaks of a lethal epidemic disease? A fatal strain of smallpox appeared in England around 1630 (Razzell, 1977; Corfield, 1987) and, although smallpox is specifically mentioned only twice in the Penrith registers (in 1656 and 1661), it will be shown in section 15.8 that there is good evidence for the development of 5-yearly epidemics of smallpox (Scott & Duncan, 1993). These regular epidemics were superimposed on the pre-existing cycles of child mortality which, it is suggested, were driven by the short wavelength oscillation in wheat prices described above. It is suggested in section 15.8 that cycles of malnutrition caused by regular high grain prices produced a corresponding oscillation in susceptibility that was sufficient to drive the system and trigger the epidemics of smallpox. In this way, the oscillatory tendency of the dynamics of the smallpox epidemics at Penrith would become phase-locked to the driving effects of increased susceptibility associated with high wheat prices and malnutrition, and the pre-existing, short wavelength oscillation in child mortality that was established in the sixteenth century was sharpened and intensified. The 5-yearly periodic epidemics of childhood smallpox mortality made a dominant contribution after 1650 to the 5-year oscillations in overall child mortality.

## 10.3 Adult mortality cycles

Spectral analysis and filtering of the adult mortality series shows that the periodicity of the short wavelength oscillation in adult deaths was predominantly 5 years (Fig. 10.4); it was strongly coherent with wheat prices during the first 100 years (1557–1650) and the cross-correlations between adult burials and prices correspond closely with that described for child burials above:

| | | |
|---|---|---|
| 1557–1600: | ccf = −0.7 | Lag at +1 years, i.e. cycles not synchronous |
| 1600–1650: | ccf = +0.35 | Zero lag |
| 1650–1700: | ccf = +0.2 | Zero lag |
| 1700–1750: | ccf = −0.2 | Lag at +1 years, i.e. cycles not synchronous |
| 1750–1812: | ccf = +0.2 | Zero lag. |

Although the 5-year short wavelength oscillation in adult burials was still correlated with wheat prices after 1650, the coherence was less significant than that of child burials, and adult burials were found to be more significantly associated with climatic conditions in the medium waveband.

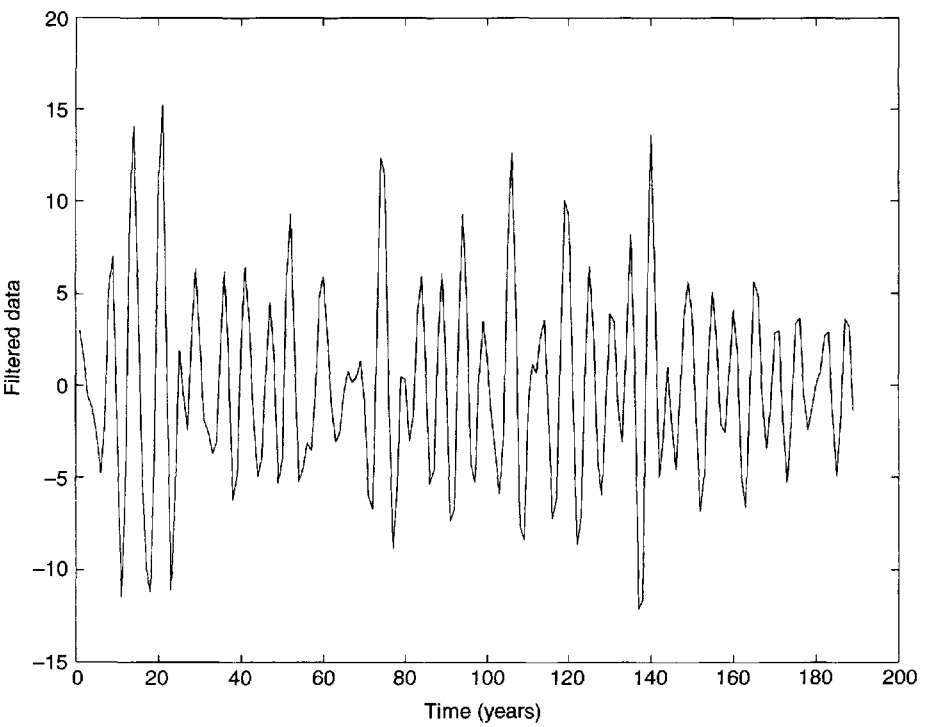


**Fig. 10.4** Annual adult burials at Penrith, 1625–1812, filtered to reveal a short wavelength oscillation (filter window = 4 to 10 years).

It is suggested, therefore, that the exogenous short wavelength cycles in both child and adult deaths were driven by periods of malnutrition associated with the peaks of the oscillations in grain prices that emerged and developed in the late sixteenth century in England. Adults and children responded differently after 1650: child mortality cycles were exacerbated by regular smallpox epidemics, whereas adult burials correlated more significantly with climatic conditions.

## 10.4 Infant mortality at Penrith

Infant mortality at Penrith was high and, until the middle of the seventeenth century, 25% of the children died during the first year of life; mortality fell progressively thereafter. Table 10.1 shows this steady fall in infant mortality at Penrith in successive cohorts and the corresponding rise in mortality in the 1–4 year age group, which is the result of the establishment of regular lethal smallpox epidemics. Thus, the improvement of infant mortality was counterbalanced by rising childhood mortality until the introduction of inoculation and, later, vaccination against smallpox became effective at the end of the eighteenth century. This amelioration of infant mortality is shown in the downward slope of the unfiltered infant burial series shown in Fig. 10.5. Spectral analysis of the annual infant mortality at Penrith (expressed as a percentage of births in that year) reveals a short wavelength oscillation, which overall has a 5–6 year periodicity, and this is shown for 1557–1806 after filtering in Fig. 10.6. Analysis of the characteristics of this oscillation in infant deaths in the different cohorts is summarised in Table 10.2.

Table 10.1 shows that the infant mortality rates at Penrith were high up to the mid-seventeenth century but, by the beginning of the eighteenth century, the rate had improved to one in five and, by the start of the next century, to one in nine. It

**Table 10.1** Life table burial rates ($1000q_x$)[1] for infants and children at Penrith, Cumbria (sexes combined), 1557–1812.

| Age groups (years) | Cohort | | | | | |
|---|---|---|---|---|---|---|
| | 1557–99 | 1600–49 | 1650–99 | 1700–49 | 1750–74 | 1775–1812 |
| 0–1 | 250 | 248 | 221 | 205 | 163 | 109 |
| 1–4 | 98 | 132 | 141 | 158 | 154 | 119 |
| 5–9 | 53 | 42 | 38 | 53 | 54 | 42 |
| 10–14 | 33 | 21 | 27 | 24 | 26 | 54 |
| % death rate (0–14 years) | 43 | 44 | 43 | 44 | 40 | 32 |

Burial rates derived from the family reconstitution study. Infant deaths calculated as the first 12 months after baptism. Children aged 1 to 14 years.

[1] Burial rates per 1000, expressed as $q_x$, the probability at age $x$ of dying before reaching age $x+n$.

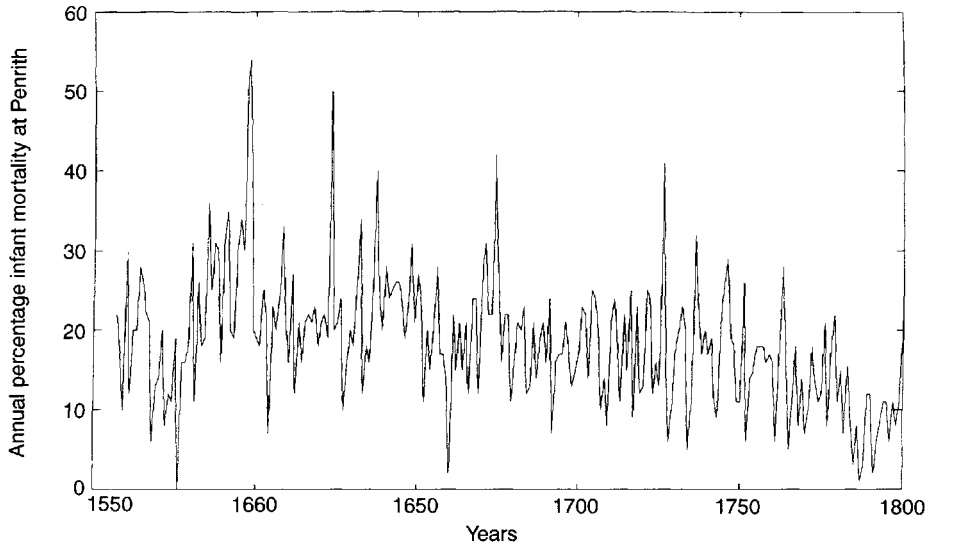


**Fig. 10.5** Annual infant burials at Penrith, 1557–1800, expressed as a percentage of the baptisms in the year of death. Note the falling trend after 1740. Data derived from the family reconstitution study.

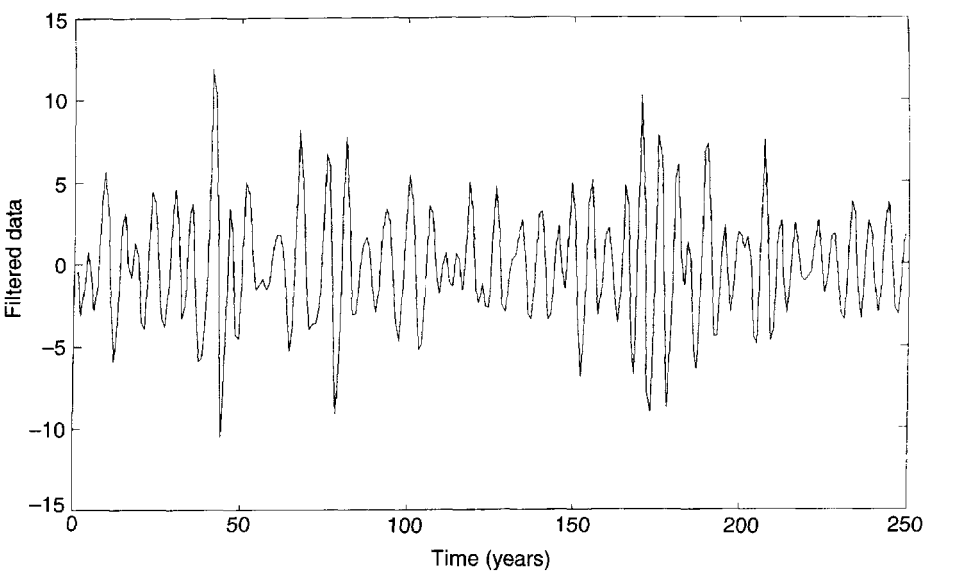


**Fig. 10.6** Annual infant burials at Penrith, 1557–1806, expressed as a percentage of the baptisms in the year of death, filtered to reveal the short wavelength oscillation (filter window = 4 to 10 years).

**Table 10.2** Characteristics of the short wavelength oscillation in infant deaths at Penrith and its correlation with wheat prices, 1557–1812.

| Cohort | Period determined by spectral analysis (years) | | Cross-correlation with wheat prices | |
|---|---|---|---|---|
| | Unfiltered | Filtered | Ccf | Lag (years) |
| 1557–1600 | 5–6 | 5–6 | 0.55 | −1 |
| 1600–1650 | 7 (secondary 5) | 7 and 5 | 0.6 | 0 |
| 1650–1700 | 6 | 6 | 0.1 | 0 |
| 1700–1750 | 5 | 5 | 90° out of phase | |
| 1750–1812 | 5 | 5 | 0.5 | 0 |

has been suggested that an infant mortality rate under 10% (see Table 10.1) for any period before the twentieth century is very low and so it might be reasonable to be suspicious of a rate close to this value at Penrith for the last period. This might be a consequence of the under-registration of children who died soon after birth, a phenomenon known to have become increasingly widespread by the eighteenth century (Schofield & Wrigley, 1979). Comparing the infant mortality rate with the death rates of children aged 1–4 is one way of assessing any discrepancy since serious under-registration would show low values for infants relative to those for the older group. However, at Penrith, the burial rates for this group also showed a significant fall during the period in question (Table 10.1).

The hypothesis that a rise in wheat prices caused an increase in infant mortality was tested by a coherence program and it was found that the two series were significantly correlated ($P < 0.001$) in the short waveband at zero lag or at a lag of +1 years. The cross-correlation between infant mortality and wheat prices in the different cohorts is summarised in Table 10.2. Again, this synchronous correlation is particularly evident during the first 100 years and the ccf for 1600–50 is shown in Fig. 10.7. This finding suggests that the complex fluctuation in infant mortality was driven, in part, by periods of hardship and malnutrition to which the population at Penrith was susceptible.

Using the family reconstitution study at Penrith, the annual infant burial series was then subdivided into neonatal mortality (28 days post-baptism; normally no data are available in parish registers for the exact day of birth) and post-neonatal mortality (1 to 12 months of life), expressed as a percentage of baptisms. Both mortality series are strongly coherent with wheat prices in the short wavelength bands ($P < 0.001$). The data series of neonatal and post-neonatal mortalities (expressed as a percentage of baptisms) were then divided into five cohorts and each was run on a coherence programme with wheat prices as input and mortality as output. The results are summarised in Table 10.3, which shows that both

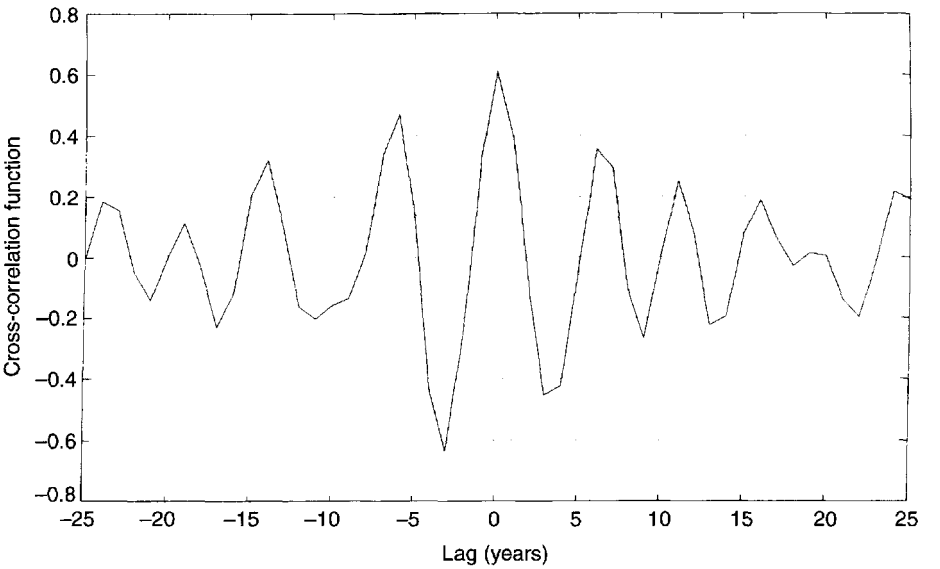


**Fig. 10.7** Cross-correlation function, wheat price series versus infant burials (expressed as a percentage of the baptisms in the year of death) at Penrith, 1600–50. Filter window = 4 to 10 years. Maximum ccf (+0.6) at zero lag shows that the two series are in synchrony.

**Table 10.3** Coherence between the wheat price index and neonatal or post-neonatal mortalities.

| | % Neonatal mortality | | | % Post-neonatal mortality | | |
|---|---|---|---|---|---|---|
| Cohort (dates) | Lag (years) | Waveband (years) | *P* | Lag (years) | Waveband (years) | *P* |
| 1557–1599 | +1 | 3–4.5 | <0.001 | 0 | 3.5–4 | <0.01 |
| | | | | | 5–8 | <0.001 |
| | | | | | 10–30 | <0.01 |
| 1600–1649 | +1 | 2.5–3 | <0.001 | +1 | 2 | <0.001 |
| | | 3–3.5 | <0.01 | | 3–3.7 | 0.001 |
| | | 5–11 | <0.01 | | 4 | <0.01 |
| | | | | | 7–11 | <0.001 |
| 1650–1699 | 0 | 2 | <0.001 | 0 | 2.5–4 | <0.001 |
| | | 3 | 0.001 | | | |
| | | 7–9 | 0.01 | | | |
| 1700–1749 | +1 | 3–4 | <0.001 | 0 | 3 | <0.01 |
| | | 7–11 | 0.001 | | | |
| 1750–1812 | +1/+2 | 2 | <0.01 | +1 | 3 | <0.001 |
| | | 3 | 0.01 | | 5–6 | 0.001 |
| | | 5 | 0.01 | | | |

Mortality expressed as a percentage of baptisms in the year of death.

mortality series are strongly coherent ($P < 0.001$) with wheat prices in the short waveband in every cohort. During the period 1750–1812, when conditions at Penrith ameliorated and the population boom began, the significance of the coherence between wheat prices and neonatal mortality fell, with $P = 0.01$. The lag between the data series (Table 10.3) was 0 or +1 years, with post-neonatal mortality tending towards synchronisation (zero lag) and neonatal mortality showing predominantly a lag of +1 years, i.e. the peaks of post-neonatal mortality tended to occur in years of high wheat prices, whereas high neonatal mortality tended to occur in the following year (i.e. when the pregnancy occurred during a period of high wheat prices). These findings suggest, therefore, that raised neonatal mortality was related to the malnutrition of the mother during pregnancy, whereas cycles of post-neonatal mortality were directly dependent on the food supply during the first year of life, which would have had a direct effect on both lactation and weaning. However, since conditions of malnutrition would frequently overlap pregnancy and infancy, a clear separation of the lag for the two components of infant mortality would not be expected.

In conclusion, time-series analysis shows that raised levels of infant deaths were significantly correlated with high wheat prices, suggesting that these short wavelength mortality cycles were exogenous, driven by a corresponding cycle of malnutrition. There is evidence that neonatal infant mortality at Penrith was related to the nutrition of the mother during pregnancy (as shown in Chapter 7), whereas post-neonatal mortality was directly related to the nutritive levels during the first year of life, i.e. during lactation and weaning. These findings concerning the differential effects of nutrition confirm the conclusions from the study of the famine at Penrith in 1623 (see section 4.7).

## 10.5 Short wavelength oscillation in baptisms at Penrith

In addition to the long wavelength oscillation (see section 5.1), spectral analysis reveals a short wavelength cycle in the baptism series at Penrith, which is shown after filtering in Fig. 10.8. It has a wavelength which varied between 5 and 8 years, with a dominant 5 year oscillation during 1557–1650, an 8-year oscillation from 1650 to 1750 and a 6–8 year oscillation thereafter.

The short wavelength oscillation in baptisms cross-correlates well with the oscillation in total burials, but is completely out of phase: i.e. *high* annual burials correspond with *low* annual baptisms. This negative correlation is particularly significant for the first 100 years (see Fig. 10.9; ccf = -0.8) and overall the two series correspond negatively at zero lag, with $P < 0.01$. However, analysis of the different cohorts shows that after 1700 this negative correlation was lost and the two cycles came into synchrony.

The short wavelength oscillation in baptisms revealed in the 404 parishes of rural England is also negatively correlated (i.e. 180° out of phase) with total

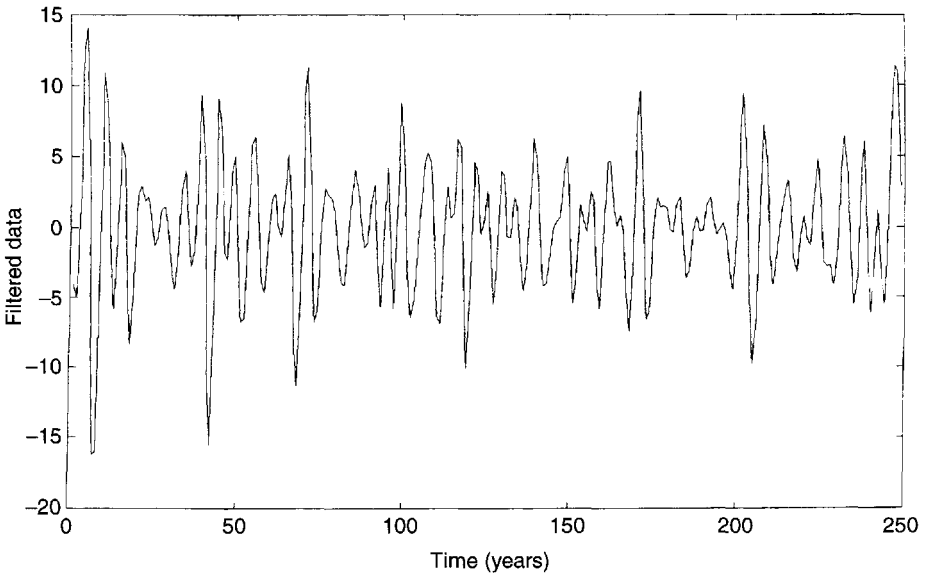


**Fig. 10.8** Baptisms at Penrith, 1557–1806, filtered to reveal the short wavelength oscillation (filter window = 4 to 10 years).

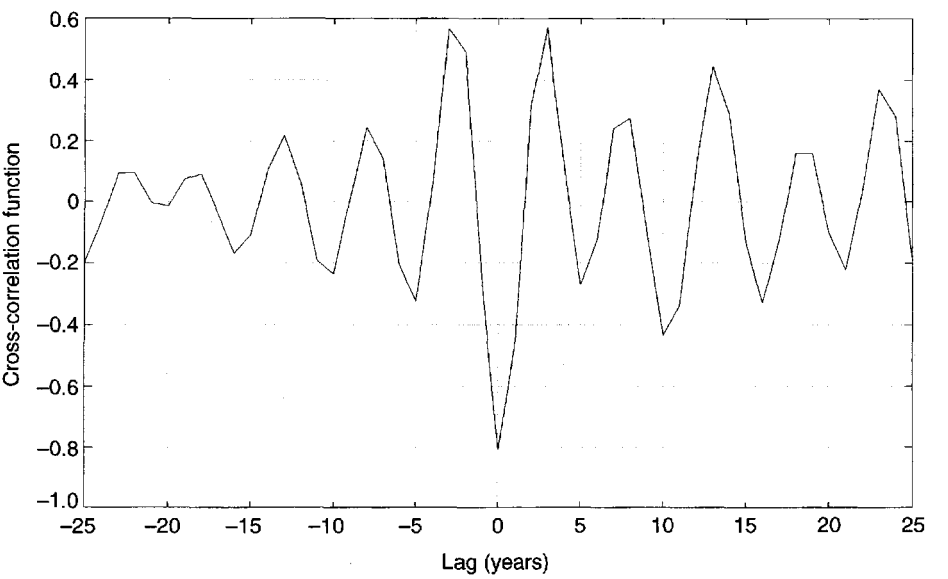


**Fig. 10.9** Cross-correlation function, baptisms versus total burials at Penrith, 1578–1657 (filter window = 4 to 10 years). Minimum value of ccf ($-0.8$) at zero lag shows that the two series are 180° out of phase.

burials, as we describe in section 2.4. The negative correlation between baptisms and total burials can be explored by using the simple matrix model without feedback developed in section 5.2. If (say) a 5-year oscillation in mortality is fed into the input, the matrix model responds with a 5-year oscillation of births in the output, where the input and output are 180° out of phase, i.e. deaths and births are *negatively* correlated at zero lag. These results suggest that the cycle in baptisms (see Fig. 10.8) was not an immediate response to variations in grain prices and malnutrition, but was driven directly by fluctuations in mortality.

## 10.6 Medium wavelength oscillation in adult burials at Penrith

The medium wavelength oscillation that is detectable in wheat prices had a less significant effect on the cycles in mortality: the population responded quickly and more significantly to the sharper changes in hardship and malnutrition that are reflected in the short wavelength oscillations in grain prices. However, a medium wavelength oscillation in *adult* mortality is detectable by time-series analysis and it is strongly developed in the years between 1660 and 1760; its wavelength during this time was 11 years. Coherence and cross-correlation studies show that this medium wavelength oscillation in mortality cross-correlates directly with low winter temperatures (ccf = $-0.65$; the cycles cross-correlate negatively; $P < 0.025$).

## 10.7 Oscillations in migration at Penrith

Migration played an important part in maintaining the steady-state conditions at Penrith, acting as an important component of the feedback control (Duncan *et al.*, 1994b). It is difficult to obtain quantitative evidence of emigration and immigration accurately by family reconstitution, particularly before 1730, when the registers provide little additional information, but an estimate of the cycles of movement of people into the parish can be derived from the reconstitution family forms. For example, the appearance of a family in the registers where there is no previous record of either partner at Penrith was scored as two immigrants; when one partner at marriage came from another parish, it was scored as one immigrant. The most important immigrations demographically would be the influx of married couples following a mortality crisis (such as a famine) to fill available 'spaces' in the community, i.e. when the density-dependent constraints were lifted. Sixty-five new families appeared in Penrith during the first few years of the seventeenth century after the plague, and the number of immigrants continued to rise until the middle of the century. Between 1650 and 1750 there was a slight decrease in immigration, but this rose again after 1750. Overall, the mean annual number of immigrants for 1600–1750 was 15.4.

An estimate of the scale of emigration is more difficult. Although it is obvious that families did leave the parish, it is not known when this occurred. However, it is assumed that a couple left the parish after marriage when there were no further events, and the native-born bride and/or groom were scored as emigrants. There is a rise in the number of people leaving the parish at marriage from 1600 to 1620 on this analysis, after which there is a slight decrease until 1750. The mean annual number of emigrants for 1600–1750 is 3.95. From 1735 to 1812, information on both partners is given in the marriage registers of Penrith. An annual index of the percentage of partners coming from outside the parish has been calculated as

$$\frac{\text{Number of partners from outside the parish} \times 100}{\text{Number of marriages} \times 2}$$

Spectral analysis of this percentage immigration index at marriage shows a 5-year oscillation that is synchronous (i.e. cross-correlates at zero lag) with a corresponding oscillation in adult deaths (ccf = +0.4). We have seen (section 10.3) that the peaks in the short wavelength oscillations in wheat prices were associated with rises in adult burials which then, in turn, promoted an influx of migrants and there was a concomitant rise in illegitimacy. The association between immigration and illegitimacy was particularly noticeable after the mortality crises of the late sixteenth and early seventeenth centuries and during the boom period of 1750–1812 (Scott & Duncan, 1997b).

Spectral analysis also reveals a medium wavelength oscillation in immigration at Penrith and this is significantly associated with elevated wheat prices ($P$ = 0.001) (Scott & Duncan, 1997b). These migratory movements also correspond with adult mortality: between 1650 and 1750 the medium wavelength cycle in immigration cross-correlates with adult mortality with a lag of 1or 2 years ($P <$ 0.05).

## 10.8 Interactions between the different oscillations: a demographic overview

The ways in which, it is suggested, the different cycles in the demographic data at Penrith interact and are driven by oscillations in wheat prices and malnutrition are shown in summary in Fig. 10.10. The short and medium wavelength cycles in wheat prices that have been identified by time-series analysis (see Chapter 3) are shown in the upper part of the figure and are separated by a vertical line. The different demographic oscillations that they drive are shown below. Adult mortality and migration cycles are shown spanning the vertical dividing line, because analysis shows that both types of oscillation can be detected therein. The arrows indicate significant relationships ($P < 0.01$).

The short wavelength oscillation in wheat prices had the dominant (and sta-

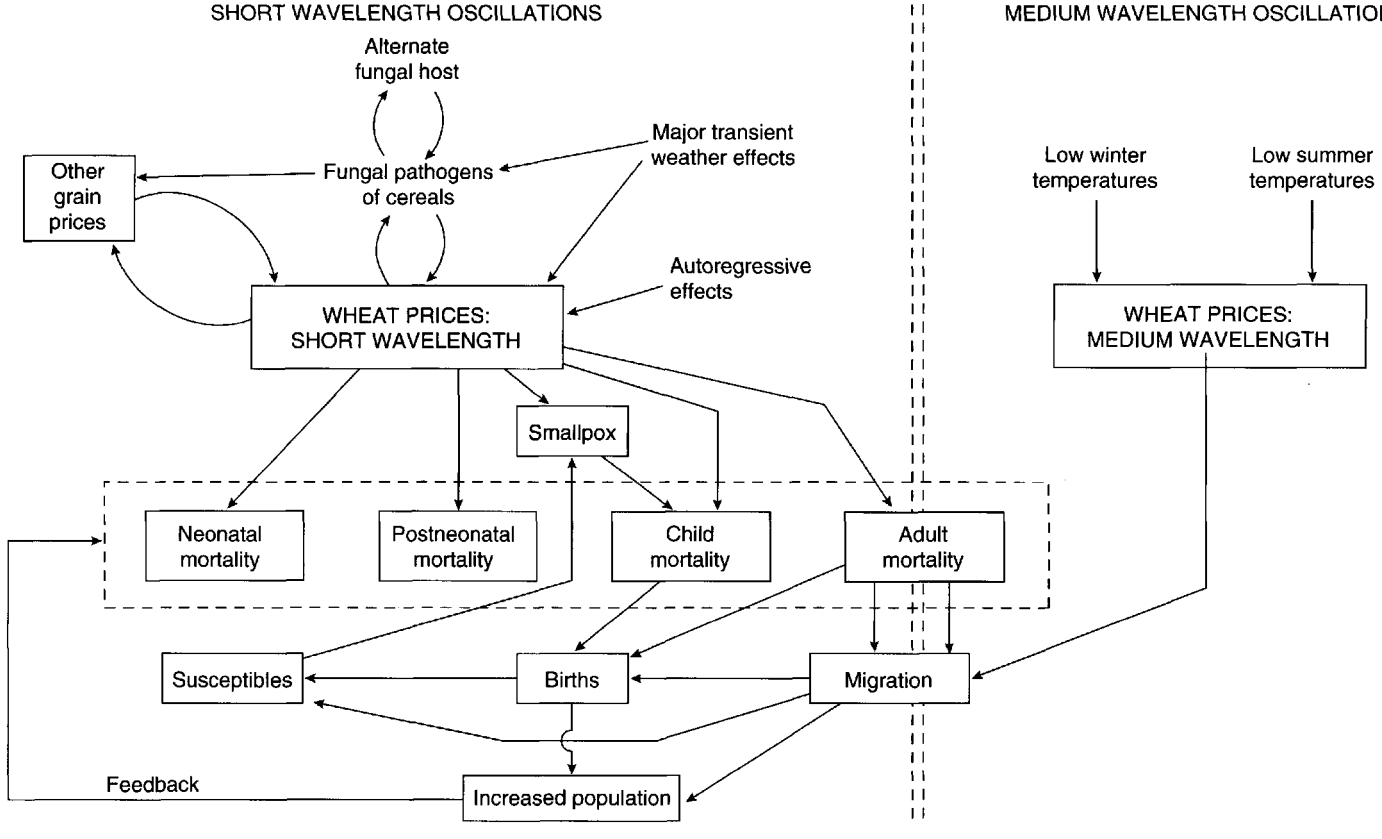


**Fig. 10.10** Summary diagram of the suggested interaction of exogenous cycles at Penrith. The short and medium wavelength oscillations that have been detected in the wheat price series by time-series analysis (see Chapter 3) are shown in the upper part of the figure, together with the various factors that may interact with, and serve to drive, them. The short wavelength oscillation in wheat prices is also subject to autoregressive factors (Chapter 3, section 3.9). These oscillations in wheat prices represent cycles of malnutrition. The distinction between the corresponding short and medium wavelength *mortality* oscillations is shown by the vertical dividing line; where a population response exhibits both types of oscillation (i.e. adult mortality), the appropriate box is shown spanning this line. The arrows indicate highly significant correlations and operate at zero lag except for neonatal mortality and migration (each at +1 years) and births which are negatively associated with total mortality. The feedback effect of increased population size on mortality is indicated.

tistically significant) effect throughout, but this is particularly noticeable during the first 100 years of study when all the mortality series were strongly associated with fluctuations in wheat prices. Thereafter, adult mortality showed a much less significant response and analysis suggests that low winter temperatures had the dominating effect (Fig. 10.10). In very cold winter months, the population would be more susceptible to respiratory infections, influenza, intestinal disorders and hypothermia. Studies of pre-industrial England, Sweden and Italy have shown that higher mortality correlated with low winter temperature (Eckstein *et al.*, 1984; Galloway, 1985), and mortality from heart disease in the twentieth century is sometimes 70% greater in winter than summer (Wilmshurst, 1994). In addition, rises in adult mortality at Penrith, both in the short and medium term, resulted in an influx of immigrants to fill the vacated niches in this saturated habitat.

The results presented in this chapter have suggested that young children and infants from the mid-seventeenth century had different responses to the effects of variations in the quality and quantity of food: the short wavelength cycles in grain prices appeared to drive the oscillations in neonatal (acting during pregnancy), post-neonatal (directly during the first year of life) and child (via susceptibility to infectious diseases) mortalities.

Smallpox has been shown to contribute substantially to the cycles in child mortality (see section 15.8 and Duncan *et al.*, 1993, 1994c) and it is suggested that the epidemics were triggered by the peaks in the short wavelength oscillation in wheat prices. Initiation of an epidemic is critically dependent on the build-up of a sufficiently large pool of susceptible individuals by new births (see section 15.2) and by the immigration of families that have not previously been exposed to the disease. Such families would have moved from smaller communities in the surrounding countryside where the density and size of the population was below a critical level for the development of a full smallpox epidemic (Duncan *et al.*, 1993, 1994c). Although it is generally inferred that nutritional deficiency had little or no direct responsibility for the effects of the disease (Livi-Bacci, 1991), it is shown in sections 15.2 and 15.8 how a small variation in susceptibility to smallpox caused by cycles of malnutrition could contribute to the initiation of an epidemic. Those on the edge of subsistence would be more directly affected by harvest failure, resulting in an ill-nourished, and occasionally starving, substratum who would be the first victims but, thereafter, the disease would spread rapidly throughout the population. This interaction between wheat prices, malnutrition, smallpox and child mortality, together with the effects of cycles of immigration and births which provided a build-up in the pool of those susceptible to the disease, is included in Fig. 10.10. The next epidemic of smallpox was critically dependent upon the build-up of a sufficient density of susceptibles (section 15.2), thereby completing the loop contained in this figure. In this way, the smallpox epidemics (and consequent child mortality) became locked into the short wavelength oscillation in wheat prices.

Infant mortality fluctuated throughout the period under study and it has been

suggested that this oscillation was driven by alternating periods of hardship and plenty; the coherence between infant burials and grain prices is highly significant ($P < 0.001$). The availability of food and the inadequacy of the mother's diet, particularly during pregnancy, had multiple effects on infant mortality; a high proportion of babies with low birthweights would be produced and there would be a rise in neonatal and post-neonatal mortalities, as explained in Chapter 9.

The short wavelength oscillation in baptisms at Penrith was significantly and *negatively* correlated with total burials (see Fig. 10.9), corresponding exactly with the matrix modelling. Thus, in these short wavelength oscillations, a rise in mortality is accompanied by a fall in births, which then rise to a peak 2–3 years later. In this way, positive feedback is established: oscillatory peaks in child deaths trigger peaks in births, which then augment the pool of young children susceptible to smallpox and thereby accelerate their rise to threshold density for the initiation of the next epidemic. Once established, the 5-year cycle of epidemics would be self-propagating, driven by the time required to raise the susceptibles to this critical density and requiring only occasional resetting by raised wheat prices. Although it is difficult to separate the role of primary and secondary influences on baptisms, the model suggests that it was the fluctuations in mortality that drove the oscillations in baptisms and that these were not an immediate response to high or low grain prices.

During the first 200 years of the period under study (1557–1750), the population at Penrith remained in steady state: mean annual births equalled mean annual deaths and were constant throughout. However, long wavelength oscillations were superimposed on the mean steady-state level by which this community maintained homeostasis (see section 5.4): the population dynamics were regulated by feedback in which mortality crises were counterbalanced by rising birth rates and immigration, until saturation of the habitat produced rising mortality and the emigration of individuals and families who were unsuccessful in securing a permanent livelihood in the parish. This complex effect of feedback on the population dynamics at Penrith is illustrated in Fig. 10.10, wherein the different types of mortality that have been identified, each with their individual sensitivity to external factors, are shown enclosed within a dashed line. These mortalities together drive migration and births which, in turn, not only augment the pool of susceptibles to smallpox, but also increase the population size. This rise in population size acts as a density-dependent control which increases the pressure on scarce resources in an area where malnutrition and famine were ever-present threats, and is followed by the inevitable increase in mortality (see Duncan *et al.*, 1992).

Adults would tend to respond to population pressure by emigration, whereas the effects of density-dependent control in this community were seen most strongly in the mortality of infants and children which, as shown above, were subtly regulated via the complex effects of nutritive levels. Malthus (1798) suggested that improvements in mortality would be short-lived since, under steady-

state conditions, if deaths from one disease were eliminated they would be replaced by mortality resulting from another malady. Table 10.1 shows the life table burial rates for infants and children at Penrith. In total, 44% of children died before reaching the age of 15 and this overall level of mortality was constant for over 200 years. The pattern, however, was not identical for all age groups of children: although mortality in the first year diminished steadily (see Chapter 11), this reduction was balanced by an upturn of mortality in the older age groups because surviving infants encountered new and greater threats in early childhood. The level of child mortality for ages 1–4 years rose steadily until the middle of the eighteenth century which was consequent upon the emergence of smallpox as a lethal disease, before improving considerably, perhaps because of the introduction of inoculation during the last quarter of the eighteenth century. In contrast, age groups 5–9 and 10–14 showed less dramatic changes, except for a sharp rise in mortality for the latter group during the period after 1750.

## 10.9 Variation in the interaction of exogenous cycles at Penrith in different cohorts

The interacting oscillations illustrated in Fig. 10.10 represent a generalised overview of events at Penrith throughout the 250-year period, 1557–1812. Correlating links (arrows) are shown only where good cross-correlation coefficients and statistical probabilities can be demonstrated, even though, as would be expected in any human population, the data series are inevitably noisy. The population was living under marginal conditions for the first 200 years, a homeostatic regime where malnutrition and epidemic disease acted to regulate the balance between resources and population size. However, conditions varied throughout this period and, consequently, the linkages differ in their strength and importance in different cohorts. For example, the major mortality crises occurred during the first hundred years studied, the population was in steady state until 1750 and boomed thereafter, and the lethal form of smallpox did not emerge until about 1630 and before that time the disease was of little demographic importance.

The period under study has, therefore, been divided into four sections (1557–1649, 1650–99, 1700–49 and 1750–1812) and, using the family reconstitution study, the significance of the cross-correlations in each cohort is analysed to highlight the subtle differences that reflect the detailed changing patterns of the demography of this community at Penrith.

### *Years 1557–1649*

The end of the sixteenth and beginning of the seventeenth centuries were dominated by the following features: an upward trend and the emergence of

clear, short wavelength oscillations in grain prices, major crises of plague and famine and progressively less spectacular mortality responses to fluctuating grain prices. The rising trend in grain prices would also add greatly to the density-dependent pressure so that, in the Malthusian sense, a positive check operated via increased mortality to moderate the rise in population and to restore the balance with resources. The major mortality crises operated as transient perturbations to the population and triggered long wavelength oscillations which were clearly established from 1600 and were because of the feedback control, of which infant and childhood mortality were a major factor. Smallpox was not evident and so the periodicity of the exogenous oscillation in childhood deaths was not geared to a threshold density of susceptibles and, therefore, the oscillation was non-stationary and directly driven by wheat prices. Infant mortality rates were particularly sensitive to the changes in wheat prices and were at their highest level with one in four dying before the age of 1 year. See summary in Fig. 10.11.

### *Years 1650–99*

Child burials at Penrith were closely related to barley prices, with respect to the short wavelength cycles during this period. Although mortality was still linked to grain prices, the population had become less sensitive to harvest failure and vital events fluctuated less significantly. When the worst of the mortality crises of the sixteenth and first half of the seventeenth centuries had passed, the cycles in mortality and baptisms were of a low amplitude, with a fundamental 5-year periodicity. Child and adult burials were no longer in synchrony because they were now driven by different factors: adults had escaped from the over-riding restriction of nutritive levels to be more affected by the medium wavelength cycles in low winter temperatures, implying that unfavourable weather conditions, more often than crop failures, initiated deaths. Neonatal and post-neonatal mortalities were still significantly affected by fluctuations in grain prices, with post-neonatal mortality displaying the strongest response.

Epidemic outbreaks of smallpox played an important role in influencing fluctuations in mortality and there was an associated rise in mortality for children in the 1–4-year age group. The small reduction in infant mortality increased the size of the group at risk to smallpox and hastened the time taken to accumulate a sufficiently-sized pool of susceptible children.

Baptisms were strongly negatively associated with adult burials in the 5-year bandwidth ($P < 0.001$) and, less significantly, with child burials ($P < 0.01$). The fall in baptisms occurred one year after the rise in mortality, but recovered by year two or three.

Although not so prone to famine conditions, there was still ample evidence of continuing poverty during this cohort; for example, all tax assessments for this period (1660, 1672 and 1693) placed Cumberland as the lowest in terms of tax

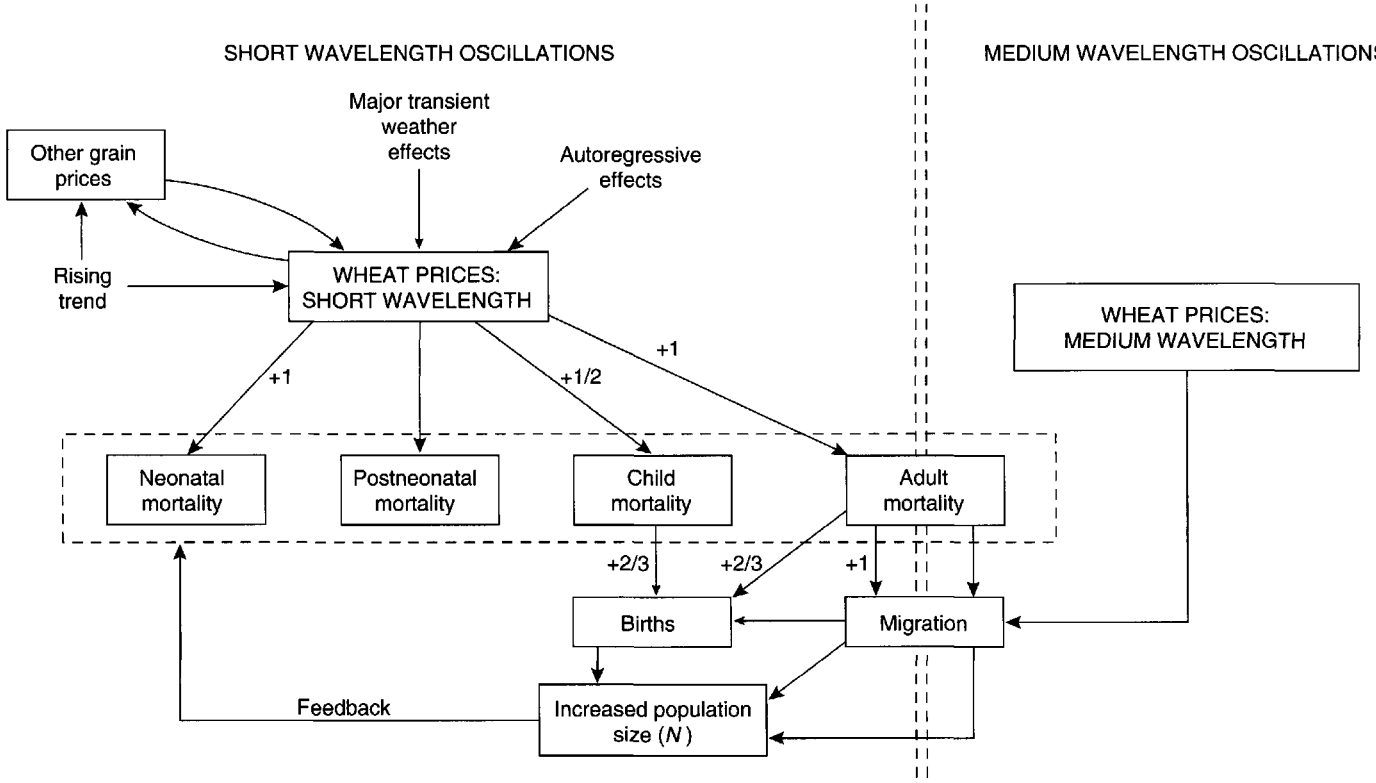


**Fig. 10.11** Summary diagram of the interaction of cycles at Penrith driven by exogenous factors during the period 1557–1649. The distinction between short and medium wavelength oscillations is shown by the vertical (doubled-dashed) dividing line; where a cycle exhibits both types of oscillation the appropriate box is shown spanning this dividing line. Large arrowheads indicate dominant effects with a $P$ value of <0.001; small arrowheads represent significant associations at $P < 0.01$. The time lags are zero unless indicated.

assessed per acre. Farm holdings were still small, but the rural population was steadily declining as towns offered more opportunities for betterment, and the economic differential acted as a magnet, attracting the rural poor with the lure of employment possibilities. (See summary in Fig. 10.12.)

### *Years 1700–49*

The demographic data derived from the family reconstitution study for the first part of the eighteenth century suggest a brief movement away from subsistence level and an improvement in living standards. Marital fertility was at its highest and approached the level seen for other English parishes (see section 6.9). There was an improvement in infant mortality rates, particularly for those aged 1–5 months. Post-neonatal mortality which was, as has been demonstrated, particularly sensitive to changes in wheat prices, was now less significantly correlated – a condition seen only during this period (see Table 10.3). More importantly, mortality overall had escaped from the domination of the joint effect of high wheat prices coupled with low real wages, and statistical analysis for this period shows that peaks in both child and adult mortality synchronised with low wheat prices and high wages. Smallpox was the most important disease of children and the epidemics were regular.

A major factor in the population dynamics of this period was determined by the migration of workers both in and out of the parish. When wheat prices were low and wages were high, there was a steady influx of migrants into the community, particularly up to the decade of the 1720s. Immigration was predominantly from outlying (upland) districts and a significant entry of people with their families would contribute on a cyclical basis to a corresponding cyclic rise in the pool of susceptibles to smallpox, probably both adults and children. Another example of cycles of malnutrition having a subtle demographic effect.

Thus, although feedback dominated the total number of people that could be accommodated within the parish, as witnessed by the clear persistence of the long wavelength oscillations, 1700–49 stands out as a period of superficial calm with mortality less closely related to wheat prices. Fertility rose and mortality was slightly improved, albeit the persistent domination of child mortality by smallpox; there was no evidence of vaccination or variolation having significant effects during this period. In fact, analysis of the data suggests that this unusual halcyon period was probably restricted to a mere 20 years, i.e. 1720–40, and it is interesting that deaths of the immigrants during the early part of this period contributed significantly to the large number of pauper burials when there was a downswing in economic conditions in the 1740s. In spite of this, however, there is no doubt that Cumbrians were prospering by comparison with their contemporaries elsewhere, and with their predecessors of the previous Century. (See summary in Fig. 10.13.)

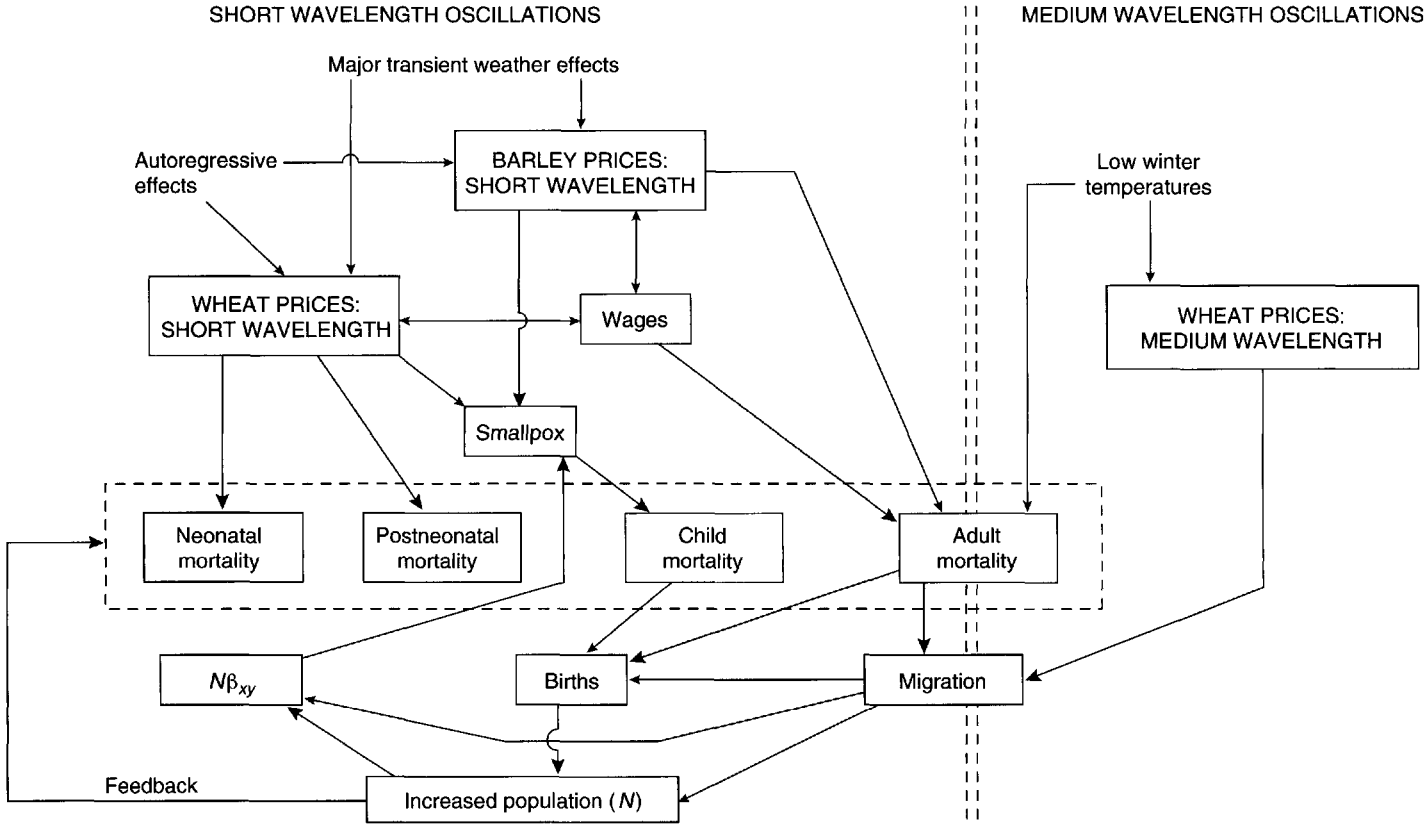


**Fig. 10.12** Summary diagram of the interaction of cycles at Penrith driven by exogenous factors during the period 1650–99. The distinction between short and medium wavelength oscillations is shown by the vertical (doubled-dashed) dividing line; where a cycle exhibits both types of oscillation the appropriate box is shown spanning this dividing line. Barley and wheat prices are shown separately. Smallpox emerges as a factor in child mortality. Adult mortality is influenced more by wages and low winter temperatures than by grain prices. Large arrowheads indicate dominant effects with a $P$ value of <0.001; small arrowheads represent significant associations at $P < 0.01$. The time lags are zero unless indicated.

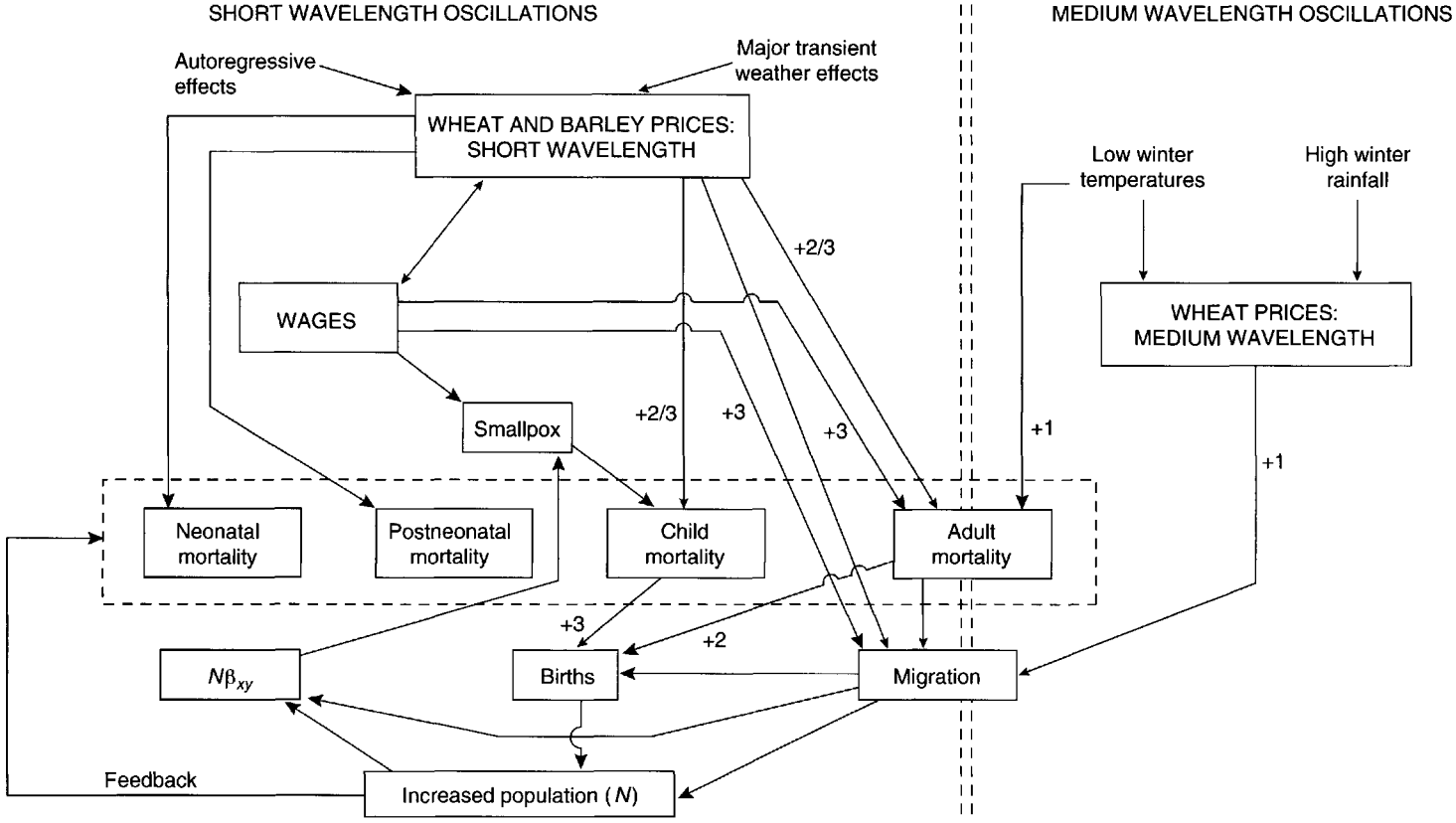


**Fig. 10.13** Summary diagram of the interaction of cycles at Penrith driven by exogenous factors during the period 1700–49. The distinction between short and medium wavelength oscillations is shown by the vertical (double-dashed) dividing line; where a cycle exhibits both types of oscillation the appropriate box is shown spanning this dividing line. Wages now have a more significant effect on both adult and childhood mortality, and on migration. Large arrowheads indicate dominant effects with a $P$ value of <0.001; small arrowheads represent significant associations at $P < 0.01$. The time lags are zero unless indicated.

### *Years 1750–1812*

The outstanding demographic feature of the population boom after 1750 is the disappearance of the link between population size and mortality. For 200 years (1550–1750) the population at Penrith had existed under steady-state conditions, when the most interesting oscillations were revealed, but the following period was characterised by a steady increase in the population. During the period 1750–85, the loop gain on the births changed from 1.0 (see Fig. 5.5) to 1.011, and to 1.019 during 1785–1812. These appear to be small rises on a gain of 1.0, but the increase in births over deaths is sufficient to generate a steadily-rising population boom which is amplified by positive feedback. The short wavelength oscillations in deaths continued during this period, and these generated corresponding oscillations in births, showing that these cycles still occurred even when the loop gain was greater than 1.0 and when conditions were no longer marginally stable. Annual baptisms rose sharply and almost doubled in 50 years. Annual adult

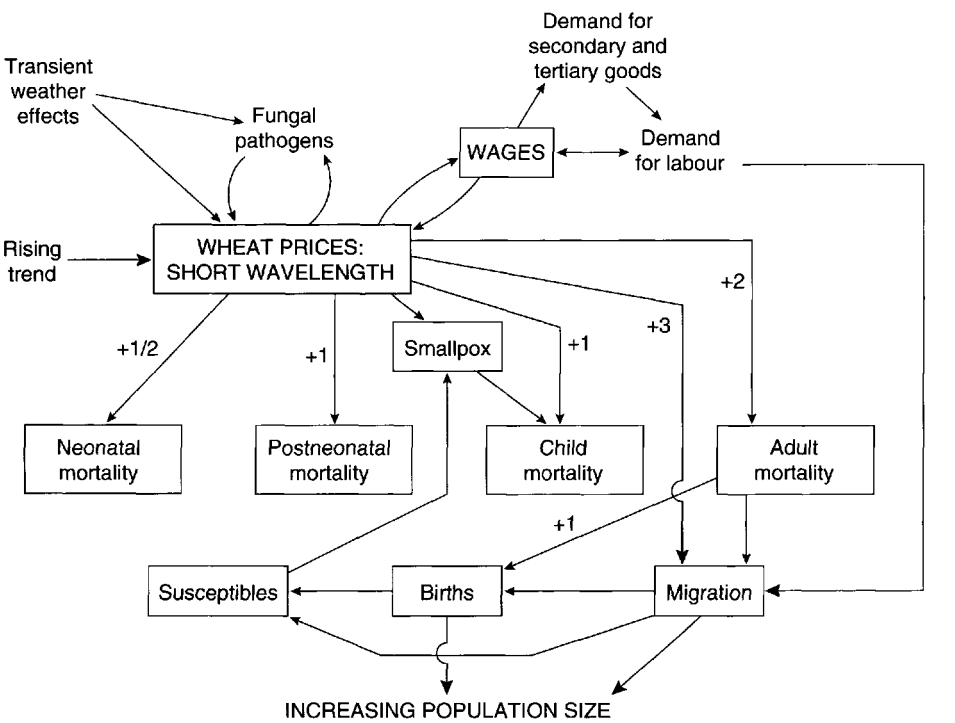


**Fig. 10.14** Summary diagram of the interaction of cycles at Penrith during the boom period, 1750–1812. Large arrowheads indicate the dominant effects with a $P$ value of <0.001; small arrowheads represent significant associations at $P < 0.01$. The time lags are zero unless indicated. There are no medium wavelength oscillations in wheat prices and feedback is no longer operating during the boom. The effects of economic factors, detectable by time-series analysis, are shown in the upper, right hand side of the figure.

deaths also rose during 1750 to 1800, but less dramatically and there was a progressive increase in longevity and an improvement in adult mortality, which was no longer adversely affected by medium wavelength oscillations in winter temperatures.

The effective resources of the community at Penrith were limited prior to 1750 and these determined the maximum size of population that could be sustained. The resources that constituted the most important part of the density-dependent control were food supply and employment niches. The optimal level of population that could be supported rose after 1750; the family reconstitution study shows that fertility did not increase (see section 6.9), whereas the total childhood mortality fell progressively. The benefits of improved maternal nutrition during pregnancy during the previous cohort meant that the adverse inter-generational effects of low birth weight daughters were gradually eliminated, thereby facilitating an amelioration of neonatal (endogenous) mortality. These ideas are developed in the next chapter. A sustained period of low mortality, which applied to adults and children, was the primary cause of the population boom after 1750. This marked change in the population dynamics and the removal of density-dependent controls probably resulted from improvements in agriculture and transport in Cumberland (McKeown, 1983). The interactions of the different cycles at Penrith during the boom are summarised in Fig. 10.14.

Since the community at Penrith was probably living very close to the margin, the effects of fluctuations in nutrition and variations in behaviour, including breast-feeding and other maternal practices (section 9.9), were magnified. In consequence, the parish is not necessarily typical of communities outside the northern territory. Nevertheless, there are important clues here that confirm the subtle and continuing effects of maternal malnutrition, and add to the on-going debate about the reasons for the accelerated population growth that took place in England in the 'long' eighteenth century.

Chapter 11

# The Amelioration of Infant Mortality in Rural England

We saw in section 10.9 that infant mortality at Penrith steadily and progressively ameliorated during the period 1600–1800. Conditions in the northern territory may have been more severe than in other parts of England. Is there evidence of a similar improvement elsewhere in England at this time? Wrigley (1977) presents results for 16 English rural and urban parishes and Jones (1980) for a group of rural parishes in north Shropshire. In spite of their caveats concerning the imperfections of the data series (see section 9.2), the results show clearly that infant mortality in England ameliorated progressively during the period 1600–1800.

## 11.1 Infant mortality in rural Shropshire, 1561–1810

The detailed infant mortality rates corrected for both age loss and birth–baptism age shift given by Jones (1980) for the Shropshire parishes are shown in Table 11.1. Civil registration infant mortality data for 1839–46 and 1889–91 for comparable rural areas are also shown in this table. The 1839–46 figures are those from the 63 health districts analysed by the Registrar-General in 1875. The level and structure of infant mortality in these districts was very close to that in Atcham, which was the registration district with the lowest infant mortality in north Shropshire and was a completely rural population during the same period. The 1889–91 data are for the three counties of Hertfordshire, Wiltshire and Dorset, in which infant mortality at that time was very low.

Jones (1980) summarises the complete change in the pattern of infant mortality with time as being much more complex than a simple decline in early infant deaths:

> 'On the basis of the rural North Shropshire data, this transformation had three major aspects – a very large decline in endogenous mortality, taking place between the mid-seventeenth and the mid-eighteenth century, a halving of

**Table 11.1** North Shropshire, rural parishes: corrected infant burials in various parts of the first year of life per 1000 baptisms.

| Period | Burials per 1000 baptisms | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Endogenous | Age in completed months | | | | | | | Total infant burial rate | Exogenous |
| | | 0 | 1 | 2 | 0–2 | 3–5 | 6–8 | 9–11 | | |
| 1561–1610 | 107 | 141 | 24 | 19 | 184 | 29 | 6 | 5 | 224 | 117 |
| 1611–1660 | 93 | 128 | 27 | 17 | 172 | 20 | 6 | 4 | 202 | 109 |
| 1661–1710 | 62 | 112 | 40 | 25 | 177 | 30 | 7 | 7 | 221 | 159 |
| 1711–1760 | 35 | 70 | 24 | 21 | 115 | 33 | 20 | 14 | 182 | 147 |
| 1761–1810 | 18 | 44 | 20 | 12 | 76 | 21 | 14 | 13 | 124 | 106 |
| 1839–1846 | 22 | 39 | 13 | 9 | 61 | 20 | 16 | 13 | 110 | 88 |
| 1889–1891 | 21 | 35 | 10 | 7 | 52 | 18 | 14 | 14 | 98 | 77 |

*Source:* Jones (1980).

exogenous mortality during the first three months of life, taking place mainly in the late eighteenth century, and a doubling of mortality during the second half of the first year of life, taking place around 1710'.

He concludes that there are two clearly defined periods in the history of mortality rates between 6 and 11 months of life. From a very low level before 1710, mortality at this age suddenly doubled during the period 1711–60 and remained at the new level until the end of the nineteenth century. The nineteenth century rates for this age group in rural areas are not exceptionally high. Rates in the towns at that period are noticeably higher, the difference being mainly attributed to the higher urban incidence of infectious diseases and, in particular, of epidemic infantile diarrhoea (see section 8.6). In this context, the 'normal' mortality rates at ages 6 to 11 months in rural north Shropshire during the eighteenth and nineteenth centuries need no particular explanation; however, there is the surprisingly low level of mortality in this age group found before 1710.

The corrected infant mortality, 1661–1710, in rural north Shropshire is compared in Table 11.2 with that for Oswestry, the parish containing the largest north Shropshire town, during the same period. When the Oswestry rates for the period are average, it can be seen that, apart from the effects of the remarkably high endogenous mortality of the 1660s, rates during the first half of infancy are very similar to those in rural parishes. On the other hand, the rates during the second half of infancy at Oswestry are at least double the rural rates, and close to the eighteenth and nineteenth century rates for this age group in the countryside.

Jones (1980) concludes that very low mortality rates in *later* infancy in the seventeenth century were a rural phenomenon: the difference is likely to be because of the relative absence of infectious disease in the countryside. By the eighteenth century, however, infectious diseases (e.g. smallpox, measles and whooping cough) were apparently affecting older infants more frequently in rural

**Table 11.2** Oswestry, 1662–1715, and north Shropshire, 1661–1710. Corrected infant burials (per 1000 baptisms).

| Period | No of baptisms | Burials per 1000 baptisms | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Endogenous | Age in completed months | | | | | | | Total infant burial rate | Exogenous |
| | | | 0 | 1 | 2 | 0–2 | 3–5 | 6–8 | 9–11 | | |
| Oswestry: | | | | | | | | | | | |
| 1662–1670 | 775 | 130 | 176 | 35 | 27 | 238 | 32 | 19 | 14 | 303 | 173 |
| 1686–1700 | 1682 | 50 | 116 | 50 | 35 | 201 | 37 | 20 | 13 | 271 | 221 |
| 1701–1715 | 1568 | 70 | 99 | 20 | 17 | 136 | 24 | 16 | 15 | 191 | 121 |
| 1662–1715 | 4025 | 73 | 121 | 35 | 27 | 183 | 31 | 18 | 14 | 246 | 173 |
| North Shropshire rural parishes: | | | | | | | | | | | |
| 1661–1710 | 2569 | 62 | 112 | 40 | 25 | 177 | 30 | 7 | 7 | 221 | 159 |

*Source:* Jones (1980).

north Shropshire. Jones suggests that the pattern of decadal variation in mortality between the ages of 6 and 11 months shown in Table 11.3 reflects the changes in the incidence of these diseases during the period around 1700 in rural north Shropshire.

**Table 11.3** Rural parishes in north Shropshire. Corrected infant burials at 6–11 months per 1000 baptisms, 1661–1760.

| Period | No of baptisms | Burials of infants aged 6–11 months per 1000 baptisms |
|---|---|---|
| 1661–1670 | 340 | 10 |
| 1671–1680 | 294 | 4 |
| 1681–1690 | 604 | 25 |
| 1691–1700 | 784 | 15 |
| 1701–1710 | 547 | 11 |
| 1711–1720 | 589 | 36 |
| 1721–1730 | 887 | 45 |
| 1731–1740 | 782 | 27 |
| 1741–1750 | 682 | 28 |
| 1751–1760 | 580 | 33 |

*Source:* Jones (1980).

Jones also considers early exogenous infant deaths during the first 3 months of life in north Shropshire. Table 11.1 shows that it was fairly steady between 1561 and 1660, rose to a peak in the later seventeenth century and then fell steadily, reaching lower levels than those between 1561 and 1660 by the end of the eighteenth century. The pattern of mortality during the last 6 months of infancy rules out the most obvious explanation of these changes in early exogenous

mortality: namely that rates were high in the sixteenth and seventeenth centuries because of the presence of artificial feeding on a considerable scale. The rapid rise of early exogenous mortality in north Shropshire during this period is similar to that found in communities practising early artificial feeding, but in those communities it is always associated with high mortality during the later months of infancy. Yet, as we have seen, later infant mortality was remarkably high in rural north Shropshire before 1710 (Jones, 1980).

It has been suggested (Blayo & Henry, 1967) that the similar pattern of excess mortality in early infancy found in eighteenth century western France was caused by the effect of respiratory diseases on young infants during the winter months. Such an impact produces a strongly seasonal pattern of infant mortality, with infant deaths peaking during the coldest months, clearly distinct from that of the late summer peak associated with intestinal diseases, particularly those caused by artificial feeding (see section 15.6). Table 11.4 shows the quarterly pattern of infant mortality in three rural parishes of northwest Shropshire in the sixteenth and seventeenth centuries, together with the quarterly rates in 1871–80 for the registration subdistricts covering the same parts of northwest Shropshire (Jones, 1980). The seasonal pattern of infant mortality is similar in the seventeenth and nineteenth centuries, with a generally marked peak during the first quarter, although the pattern of seasonality was more exaggerated in the seventeenth than in the nineteenth century. Table 11.5 shows the detailed breakdown of mortality during the first year of life at Selattyn, the parish with the strongest seasonal variation; the four individual months, from December to March inclusive, which showed very heavy infant mortality, have been grouped together, and the mortality during the first year of life for those months is compared with that for the remainder of the year. Mortality was heavier during the winter months at Selattyn at each period of infancy; this seasonal effect was particularly strong in exogenous mortality during the first 2 months of life and in late infancy.

The seasonal pattern of infant mortality common in rural England in the nineteenth century is, therefore, found in a more marked form in the seven-

**Table 11.4** Rural areas in northwest Shropshire. Infant burials per 1000 baptisms in each quarter of the year.

| Area | Period | Burials per 1000 baptisms | | | |
|---|---|---|---|---|---|
| | | Jan–Mar | Apr–Jun | July–Sept | Oct–Dec |
| Selattyn | 1586–1677 | 259 (533) | 188 (432) | 107 (358) | 218 (409) |
| St Martins | 1673–1688 | 247 (150) | 118 (128) | 158 (140) | 151 (131) |
| Kinnerley | 1686–1722 | 247 (300) | 210 (282) | 166 (238) | 224 (259) |
| Llansilin | 1871–1880 | 130 (292) | 65 (336) | 88 (306) | 102 (226) |
| St Martins | 1871–1880 | 149 (625) | 118 (695) | 84 (628) | 101 (536) |
| Knockin | 1871–1880 | 138 (326) | 105 (344) | 83 (338) | 125 (289) |

*Source:* Jones (1980). Number of baptisms given in brackets.

**Table 11.5** Selattyn, 1586–1677. Corrected infant burials per 1000 baptisms in the same months of the year.

| Months | No of baptisms | Burials per 1000 baptisms | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Endogenous | Age in completed months | | | | | | | Total infant burial rate | Exogenous |
| | | | 0 | 1 | 2 | 0–2 | 3–5 | 6–8 | 9–11 | | |
| Dec–Mar | 665 | 137 | 176 | 35 | 17 | 228 | 24 | 11 | 9 | 272 | 135 |
| Apr–Nov | 1067 | 80 | 101 | 15 | 14 | 130 | 21 | 5 | 4 | 160 | 80 |
| Total | 1732 | 102 | 130 | 23 | 15 | 168 | 22 | 7 | 6 | 203 | 101 |

*Source:* Jones (1980).

teenth century, and Jones suggests that the heavy early infant mortality of sixteenth and seventeenth century in rural north Shropshire can be attributed to the effect of winter respiratory diseases on young infants. The fall in mortality during the first 3 months of life between the seventeenth and nineteenth centuries is, therefore, most likely to be due to a reduction in the incidence of serious respiratory diseases among infants in that age group because the bulk of the fall between the two periods shown in Table 11.4 occurred during the colder quarters of the year.

Jones (1980) considers the endogenous infant mortality in the Shropshire parishes to be the most complex aspect of the subject. Endogenous mortality fell sharply from levels around 100 before 1660 to around 20 by 1800 (Table 11.1) and so accounted for most of the overall decline in infant mortality in rural north Shropshire between the seventeenth and nineteenth centuries. Jones analyses the mortality during the first month of life in more detail in data series from (i) the detailed registers of the market town of Oswestry and (ii) the rural parish of Selattyn which adjoins Oswestry parish, with figures from Kinnerley for the late seventeenth century inserted where the Selattyn register is defective (see Table 11.6). The results can be summarised as follows: mortality in weeks 2–3 shows similar trends to those of mortality during the next 2 months of life, with fluctuating rates up to the early eighteenth century and declining rates thereafter. Jones (1980) concludes from his careful analysis of infant mortality in Shropshire that:

(1) The fall in infant mortality was largely because of a fall in exogenous mortality during the first 3 months of life and, perhaps, to a smaller extent, to a decline in endogenous mortality.
(2) This substantial fall in early exogenous mortality began in the late seventeenth century as a fall within 2 weeks of birth, but spread, in the course of the eighteenth century, to exogenous mortality throughout the first 3 months of life.

**Table 11.6** Oswestry, Selattyn and Kinnerley. Corrected infant burials in the first 4 weeks of life per 1000 baptisms.

| Parish | Period | No of baptisms | Burials per 1000 baptisms | | | |
|---|---|---|---|---|---|---|
| | | | Age in completed weeks | | | Total (0–3 weeks) |
| | | | 0 | 1 | 2–3 | |
| Oswestry | 1581–1590 | 1029 | 49 | 51 | 22 | 122 |
| Oswestry | 1621–1630 | 1132 | 45 | 41 | 12 | 98 |
| Oswestry | 1662–1670 | 775 | 77 | 61 | 35 | 173 |
| Oswestry | 1686–1700 | 1682 | 56 | 26 | 28 | 110 |
| Oswestry | 1701–1715 | 1568 | 39 | 19 | 29 | 87 |
| Oswestry | 1761–1767 | 729 | 27 | 22 | 23 | 72 |
| Selattyn | 1586–1677 | 1732 | 60 | 43 | 19 | 122 |
| Kinnerley | 1686–1700 | 443 | 37 | 27 | 23 | 87 |
| Selattyn | 1721–1750 | 537 | 40 | 13 | 31 | 84 |
| Selattyn | 1786–1812 | 760 | 12 | 9 | 9 | 30 |

*Source:* Jones (1980).

(3) This fall in early exogenous mortality was largely the result of a reduction in the very heavy winter mortality of young infants, probably caused by the effects of cold and of infection on the respiratory systems of newly born infants.

## 11.2 Infant mortality in England 1550–1849

Table 11.7 shows the corrected infant mortality rates for four English parishes calculated by Wrigley (1977). The data are divided into two categories, 1550–1749 and 1750–99, and the means for each parish are shown in the Bourgeois-Pichat plots in Figs 9.6 and 9.7. The slopes of the Bourgeois-Pichat plots of the means of the four parishes for the two time periods are almost identical and the lines differ only in the fall in overall endogenous mortality from 71 to 48 deaths per 1000 live births after 1750 (see Table 11.7). This result indicates that neonatal mortality in these four parishes ameliorated markedly between 1550 and 1800, whereas post-neonatal mortality was unchanged. We conclude that in these four parishes the amelioration in infant mortality was solely because of an improvement in maternal nutrition in pregnancy (see section 7.9), which caused a reduction in endogenous mortality rates.

The plots for the individual parishes differ within the mean lines (see Figs 9.6 and 9.7 and Table 11.7) and may be summarised as follows:

- Banbury (Oxfordshire). Highest endogenous rate, but no change after 1750.
- Colyton (Devon). Low endogenous rate which did not change after 1750. Small amelioration in the exogenous rate after 1750.

**Table 11.7** Infant mortality rates (per 1000 live births) in four English parishes, 1550–1799.

| | 1550–99 | 1600–49 | 1650–99 | 1700–49 | 1750–99 |
|---|---|---|---|---|---|
| Colyton, Devon | | | | | |
| Total | 130 | 92 | 109 | 109 | 94 |
| Exogenous | 78 | 64 | 65 | 79 | 56 |
| Endogenous | 52 | 28 | 44 | 30 | 38 |
| | (1619) | (2459) | (1467) | (1133) | (1469) |
| Alcester, Warks | | | | | |
| Total | 167 | 140 | 188 | 220 | 97 |
| Exogenous | 93 | 70 | 103 | 115 | 68 |
| Endogenous | 74 | 70 | 85 | 105 | 29 |
| | (854) | (1687) | (1685) | (1207) | (1133) |
| Banbury, Oxon | | | | | |
| Total | 156 | 170 | 167 | 240 | 199 |
| Exogenous | 63 | 76 | 71 | 122 | 106 |
| Endogenous | 93 | 94 | 96 | 118 | 93 |
| | (2270) | (3427) | (2987) | (3365) | (4047) |
| Aldenham, Herts | | | | | |
| Total | 131 | 132 | 118 | 147 | 143 |
| Exogenous | 64 | 67 | 61 | 85 | 109 |
| Endogenous | 67 | 65 | 57 | 62 | 34 |
| | (1149) | (1340) | (1093) | (1204) | (1208) |
| Mean rates for four parishes | | | | | |
| Total | 146 | 133 | 145 | 179 | 133 |
| Exogenous | 75 | 69 | 75 | 100 | 85 |
| Endogenous | 71 | 64 | 70 | 79 | 48 |

*Source:* Wrigley (1977).
Numbers in brackets equal number of live births from which mortality rates were derived.

- Alcester (Warwickshire). Endogenous rate improved markedly after 1750, falling by 50%. Exogenous rate unchanged.
- Aldenham (Hertfordshire). Marked amelioration of endogenous mortality after 1750, but an accompanying *rise* in the exogenous rate.

Wrigley (1977) extended his analysis of infant mortality to a further 16 English parishes (listed in Table 11.8), although he used a simpler form of the nominal record linkage technique. The infant mortality rates and the distribution of deaths in the first year of life are shown in Tables 11.8 and 11.9. The entries in the first column of Table 11.9, headed 0* days, show the number of registered infant deaths where there is no linking baptism, but the burial entry indicates that the child died soon after birth. The Bourgeois-Pichat plots for the 50-year intervals for the 16 English parishes are shown in Fig. 11.1 and a similar story is found: changing endogenous mortality but broadly comparable slopes to the line and hence unchanging exogenous mortality. It is not until the 1780s that

**Table 11.8** Infant mortality rates (per 1000 baptisms) in 16 English parishes.

| | 1580s | 1630s | 1680s | 1730s | 1780s |
|---|---|---|---|---|---|
| Barton-under-Needwood (Staffs) | 100 | 82 | 109 | 143 | 119 |
| Bromfield (Shropshire) | 112 | 147 | 117 | 120 | 62 |
| Bruton (Somerset) | 194 | 119 | 140 | 214 | 81 |
| Dymock (Gloucs) | 90 | 64 | 61 | 47 | 64 |
| Eastham (Worcs) | 87 | 118 | 110 | 94 | 108 |
| Farnham (Yorks WR) | 135 | 175 | 232 | 115 | 114 |
| Ludlow (Shropshire) | 273 | 105 | 257 | 194 | 112 |
| St Martin, Coney St (York City) | 269 | 257 | 185 | 177 | 184 |
| St Michael Cornhill (London) | 53 | 124 | 333 | 333 | 170 |
| Middleton (Lancs) | 102 | 88 | 171 | 169 | 78 |
| Ottery St Mary (Devon) | 88 | 107 | 171 | 72 | 59 |
| Tatenhill (Staffs) | 138 | 237 | 283 | 95 | 56 |
| St Vedast (London) | 93 | 200 | 246 | 188 | 160 |
| Wedmore (Somerset) | 243 | 158 | 143 | 105 | 79 |
| Wem (Shropshire) | 155 | 83 | 114 | 76 | 92 |
| Widecombe-in-the-Moor (Devon) | 81 | 88 | 115 | 48 | 46 |
| Mean rate | 138 | 135 | 174 | 137 | 99 |
| Exogenous | 81 | 82 | 113 | 99 | 74 |
| Endogenous | 57 | 53 | 61 | 38 | 25 |

*Source:* Wrigley (1977)

one sees any marked improvement in exogenous mortality during the ages 1 to 4 months.

The changing levels of endogenous and exogenous infant mortality in the 16 English parishes, 1600 to 1800, are shown in Figs 9.10 and 9.11, respectively. Wrigley (1977) points out that the values for endogenous mortality are not absolute and should be accepted with caution. Nevertheless, they are invaluable as one set of comparative figures which can be used to show how infant mortality rates changed over a critical 250-year period. However, as we shall see below, the results for these 16 English parishes studied by Wrigley are atypical, in particular the low total infant mortality (probably attributable to a low endogenous mortality), which was constant from 1580 to 1730, save for a sharp rise in the 1680s.

Are the 16 parishes selected unusual? They are listed in Table 11.8. With the exception of Farnham in West Yorkshire and Middleton in Lancashire, they are confined to central and southern England; there are no representatives of populations from the northern territory. Inspection of the data in Table 11.8 reveals that the individual parishes differed markedly from each other and from the mean rate. For example, in the 1580s the mean infant mortality rate varied from 53 (St Michael Cornhill, London) to 273 (Shropshire), a 5-fold difference. There is no obvious geographical effect: infant mortality rates were low in Devon but high in the adjacent county of Somerset; they

**Table 11.9** Distribution of infant deaths within the first year of life in 16 parishes.

| | Days | | | | | | | | Weeks | | | Months | | | | | | | | | | | Total of related baptisms |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0* | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 1 | 2 | 3 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | |
| 1580s | | | | | | | | | | | | | | | | | | | | | | | |
| Cumulative total | 17 | 24 | 45 | 56 | 64 | 70 | 74 | 82 | 109 | 130 | 153 | 186 | 207 | 225 | 240 | 249 | 257 | 262 | 265 | 269 | 270 | 273 | 2156 |
| % | | 9 | 16 | 21 | 23 | 26 | 27 | 30 | 40 | 48 | 56 | 68 | 76 | 82 | 88 | 91 | 94 | 96 | 97 | 99 | 99 | 100 | |
| 1630s | | | | | | | | | | | | | | | | | | | | | | | |
| Cumulative total | 21 | 27 | 44 | 62 | 68 | 73 | 81 | 88 | 117 | 133 | 149 | 184 | 202 | 212 | 224 | 236 | 243 | 246 | 248 | 259 | 265 | 269 | 2295 |
| % | | 10 | 16 | 23 | 25 | 27 | 30 | 33 | 43 | 49 | 55 | 68 | 75 | 79 | 83 | 88 | 90 | 91 | 92 | 96 | 99 | 100 | |
| 1680s | | | | | | | | | | | | | | | | | | | | | | | |
| Cumulative total | 18 | 20 | 28 | 42 | 51 | 54 | 61 | 69 | 95 | 117 | 131 | 157 | 174 | 198 | 207 | 217 | 224 | 233 | 239 | 248 | 256 | 260 | 1747 |
| % | | 8 | 11 | 16 | 20 | 21 | 23 | 27 | 37 | 45 | 50 | 60 | 67 | 76 | 80 | 83 | 86 | 90 | 92 | 95 | 98 | 100 | |
| 1730s | | | | | | | | | | | | | | | | | | | | | | | |
| Cumulative total | 11 | 14 | 16 | 32 | 40 | 46 | 53 | 60 | 85 | 94 | 103 | 131 | 150 | 169 | 173 | 183 | 187 | 198 | 203 | 209 | 218 | 223 | 1981 |
| % | | 6 | 7 | 14 | 18 | 21 | 24 | 27 | 38 | 42 | 46 | 59 | 67 | 76 | 78 | 82 | 84 | 89 | 91 | 94 | 98 | 100 | |
| 1780s | | | | | | | | | | | | | | | | | | | | | | | |
| Cumulative total | 19 | 23 | 28 | 33 | 39 | 48 | 54 | 55 | 71 | 85 | 101 | 126 | 146 | 160 | 172 | 181 | 193 | 212 | 224 | 236 | 240 | 249 | 3004 |
| % | | 9 | 11 | 13 | 16 | 19 | 22 | 22 | 29 | 34 | 41 | 51 | 59 | 64 | 69 | 73 | 78 | 85 | 90 | 95 | 96 | 100 | |

Parishes listed in Table 11.8. The column headed 0* days shows the number of registered infant deaths where there is no linking baptism.
*Source:* Wrigley (1977).

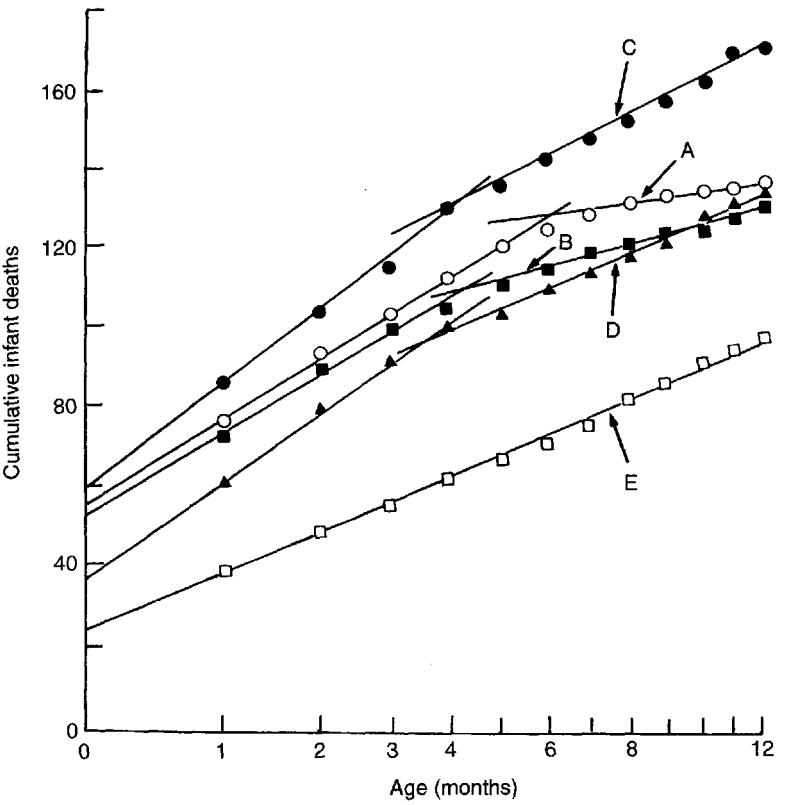


**Fig. 11.1** Bourgeois-Pichat biometric plots. Cumulative infant deaths per 1000 baptisms in 16 English parishes, 1580–1780. Note the marked amelioration in endogenous mortality after 1700 whereas exogenous mortality is little changed over a 200-year period. A: 1580s; B: 1630s; C: 1680s; D: 1730s; E: 1780s. From Wrigley (1977).

were low in Gloucestershire (adjacent to Somerset) and Worcestershire but high again in Shropshire. However, when the fluctuating rates for the two Shropshire parishes, Wem and Broomfield, are compared in Fig. 11.2 with the mean values for the four Shropshire parishes (Table 11.1) and with Oswestry (Table 11.2) the four plots show the same progressive but erratic fall in infant mortality. These populations, all within the county of Shropshire, differed dramatically in their calculated rates.

The parishes also differed in their patterns of change in infant mortality rates in successive cohorts (see Table 11.8). The overall steady mean rates rose sharply in the 1680s, but this was very much driven by St Michael Cornhill, London (where mortality rose 6-fold from the rate in the 1580s), and to a lesser extent by St Vedast, London, and Tatenhill, Staffordshire. Omitting the two London parishes (which were probably exceptional) brings the mean rate for the 1680s down to 158.

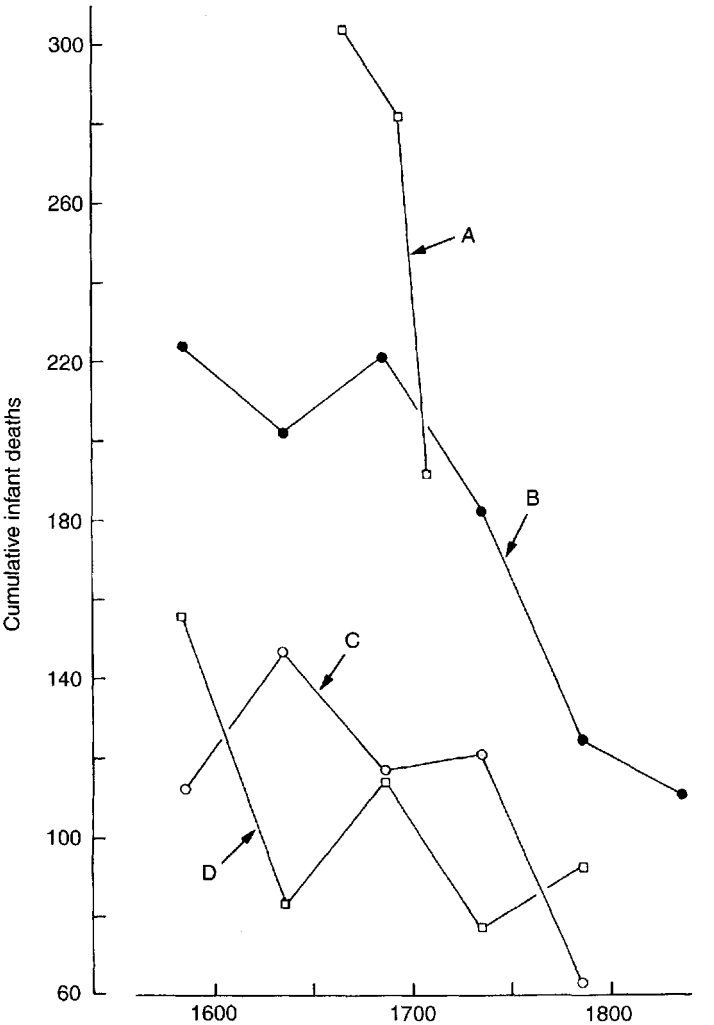


**Fig. 11.2** Variation in the total infant mortality rate per 1000 baptisms (ordinate) in the county of Shropshire (1580–1839). A: Oswestry; B: mean of four Shropshire parishes (see Table 11.1); C: Wem; D: Bromfield. Data from Jones (1976) and Wrigley (1977).

## 11.3 The 26 rural parishes studied by Wrigley *et al.*

Wrigley *et al.* (1997) have extended the studies described above in section 11.2 and have provided a comprehensive review of English population history from the family reconstitution studies of 26 parishes, which include the four parishes described in section 11.2. The parishes studied were:

| | |
|---|---|
| Northumberland | Earsdon |
| Yorkshire, West Riding | Birstall |
| | Methley |
| Lincolnshire | Gainsborough |
| Nottinghamshire | Gedling |
| Leicestershire | Bottesford |
| | Shepshed |
| Warwickshire | Austrey |
| | Alcester |
| Cambridgeshire | March |
| | Willingham |
| Suffolk | Lowestoft |
| Essex | Great Oakley |
| | Terling |
| Bedfordshire | Southill |
| Oxfordshire | Banbury |
| Hertfordshire | Aldenham |
| Kent | Ash |
| Surrey | Reigate |
| Hampshire | Odiham |
| Devon | Bridford |
| | Colyton |
| | Dawlish |
| | Hartland |
| | Ipplepen |
| | Morchard Bishop |

With the exception of the smaller parish of Earsdon in Northumberland, these communities are concentrated in central and southern England, with a northern limit of the latitude of the Humber. Six parishes are grouped in the southwest, in Devon. There appear to be no representatives of populations living under marginal conditions.

The results for the infant mortality in the different cohorts (usually 25-year periods) are shown in Table 11.10 and, since they cover 26 well-studied parishes, they can be regarded as an accurate description of the *aggregated* data of populations in central and southern England during the period 1600 to 1800. However, they almost certainly disguise wide variations between the parishes, both in the levels of endogenous and exogenous mortality and in the patterns of change in successive cohorts.

Total infant mortality overall in the 26 parishes was somewhat higher than that recorded in the study with 16 English parishes (see Fig. 11.3). Endogenous infant mortality was slightly erratic, but the trend remained steady until 1740, whereupon it fell progressively (Fig. 11.4). Exogenous infant mortality in the 26

**Table 11.10** Mortality within the first year of life ($1000q_x$) in the 26 English parishes studied by Wrigley *et al.* (1997).

| Cohort | Maternal mortality per 1000 birth events | Days within the first year of life | | | | | | | | | Endogenous | Exogenous |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0–1 | 1–6 | 7–29 | 0–29 | 30–59 | 60–89 | 90–179 | 180–273 | 274–365 | | |
| 1580–99 | 12.3 | 34.8 | 30.0 | 40.7 | 101.9 | 17.5 | 12.2 | 25.0 | 11.9 | 12.3 | 77.6 | 93.1 |
| 1600–24 | 12.8 | 44.9 | 31.6 | 35.8 | 108.2 | 15.9 | 10.1 | 18.6 | 11.5 | 9.5 | 88.5 | 76.7 |
| 1625–49 | 14.0 | 45.1 | 24.5 | 28.0 | 94.5 | 13.5 | 8.4 | 18.7 | 12.3 | 13.8 | 80.0 | 73.3 |
| 1650–74 | 17.0 | 52.5 | 25.2 | 30.2 | 104.2 | 16.2 | 9.3 | 19.1 | 14.1 | 13.0 | 87.3 | 79.4 |
| 1675–99 | 15.6 | 50.9 | 28.9 | 34.1 | 109.7 | 17.8 | 13.1 | 24.6 | 16.0 | 16.6 | 88.3 | 97.1 |
| 1700–24 | 13.4 | 31.6 | 33.9 | 44.7 | 106.3 | 19.3 | 14.1 | 26.1 | 21.2 | 17.5 | 84.0 | 106.7 |
| 1725–49 | 12.3 | 29.6 | 34.1 | 41.5 | 101.6 | 20.0 | 13.8 | 26.8 | 22.5 | 20.4 | 80.5 | 110.3 |
| 1750–74 | 9.5 | 22.6 | 28.3 | 29.8 | 78.5 | 16.0 | 11.6 | 25.7 | 22.6 | 19.1 | 61.3 | 101.5 |
| 1775–99 | 9.0 | 31.9 | 15.8 | 25.2 | 71.3 | 17.2 | 11.0 | 26.6 | 23.0 | 17.7 | 52.6 | 104.1 |
| 1800–24 | 6.3 | 22.4 | 11.5 | 24.4 | 57.3 | 14.1 | 10.9 | 23.6 | 21.0 | 16.8 | 41.0 | 95.0 |
| 1825–37 | 4.7 | 6.2 | 16.6 | 26.6 | 48.7 | 17.1 | 10.7 | 27.1 | 27.2 | 22.3 | 33.3 | 110.8 |

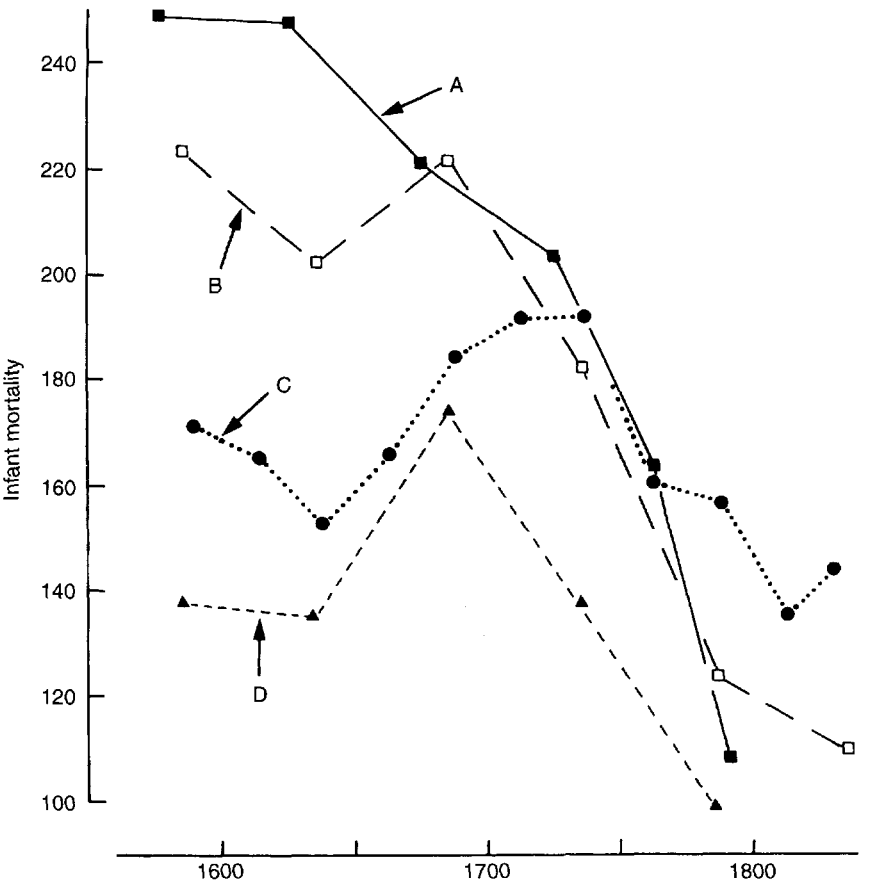


**Fig. 11.3** Falling total infant mortality in the eighteenth century in England. A: Penrith, Cumbria; B: Shropshire parishes studied by Jones (1976); C: 26 parishes studied by Wrigley *et al.* (1997); D: 16 parishes studied by Wrigley (1977). Ordinate: total infant mortality per 1000 baptisms.

parishes was remarkable and showed a completely different pattern from the other studies, including the 16 English parishes (Fig. 11.5): the rate fluctuated but the overall trend for 1590 to 1830 was slowly rising.

Finally, Woods (2000) has constructed mortality curves for infancy for England for two periods, 1725–49 and 1825–37, and calculations from his data show that the rate of exogenous infant deaths remained constant during this 100-year period, whereas, again, endogenous mortality rates fell by 50%.

## 11.4 Infant mortality rates in France in the seventeenth and eighteenth centuries

Wrigley (1977) has collated the results of a number of French studies of the endogenous and exogenous components of infant mortality and these are shown

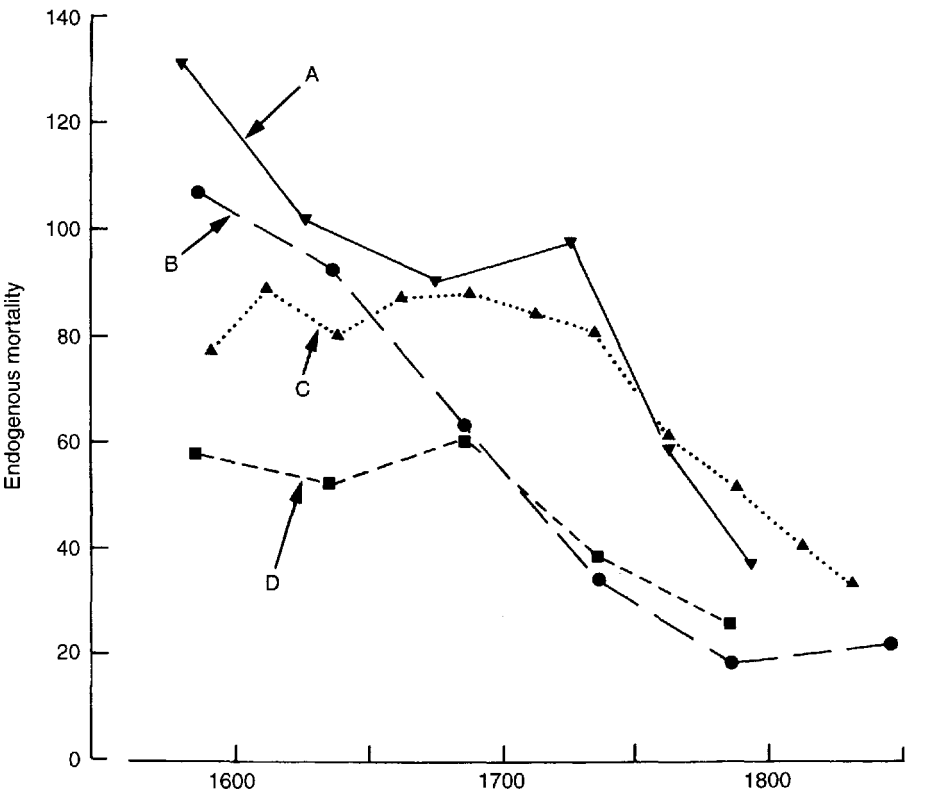


**Fig. 11.4** Changing rates of endogenous infant mortality in rural England, 1580–1840. A: Penrith, Cumbria; B: mean of Shropshire parishes studied by Jones (1976); C: 26 parishes studied by Wrigley *et al.* (1997); D: 16 parishes studied by Wrigley (1977). Ordinate: endogenous mortality per 1000 baptisms.

in Table 11.11. Most are based on reconstitution studies and some include infants who were baptised at home and died shortly thereafter, thereby inflating the endogenous rate. Endogenous rates vary from 58 to 152 (mean 88) and exogenous rates from 53 to 192 (mean 119) per 1000. The level of infant mortality not only varied very substantially from place to place, but also in the same place over time. In Sainghin-en-Mélantois during the 11 decades, 1740–1849, the total rate fluctuated between 132 and 259, the endogenous rate between 50 and 120 and the exogenous rate between 41 and 139 (Wrigley, 1977).

These data for French populations each cover a specific period but generally centre around 1730. Although they are not strictly comparable, Table 11.12 shows the results of the studies of the English parishes and the mean of the French populations for the 1730s, the time at which mortality rates were falling in many English parishes (see Fig. 11.3). The French populations are characterised by a high total mortality largely driven by a high exogenous infant mortality.

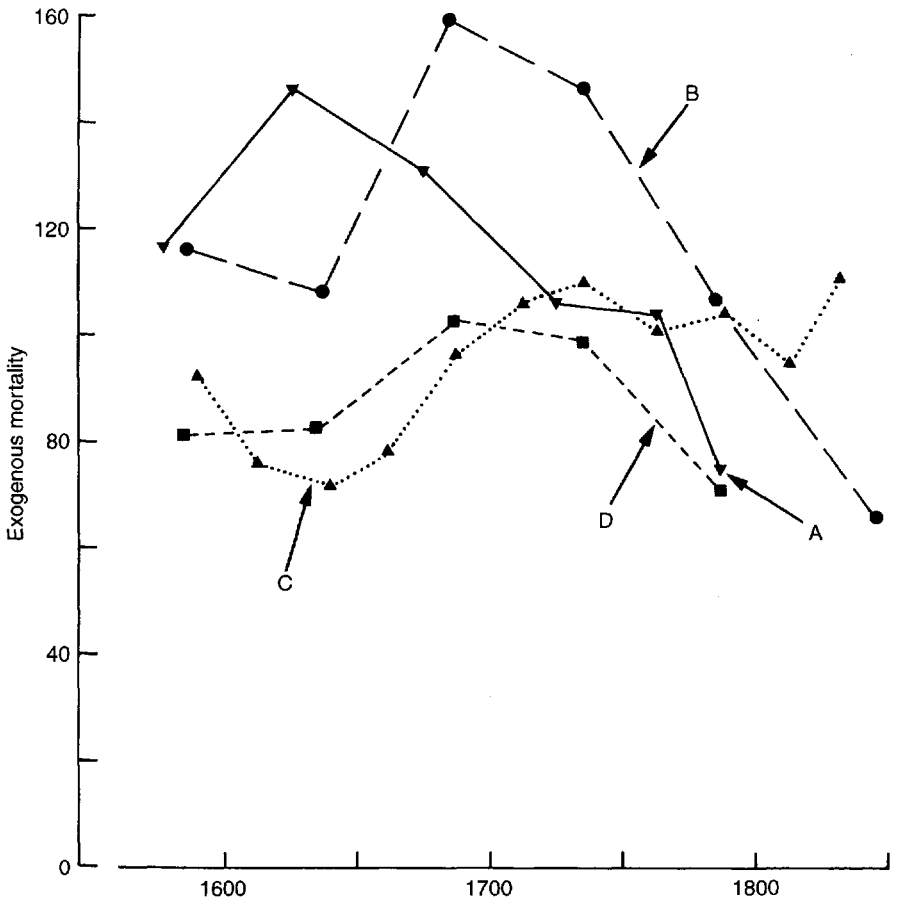


**Fig. 11.5** Exogenous infant mortality in rural England, 1580–1840. A: Penrith, Cumbria; B: mean of Shropshire parishes studied by Jones (1976); C: 26 parishes studied by Wrigley *et al.* (1997); D: 16 parishes studied by Wrigley (1977). Compare with endogenous infant mortality in Fig. 11.4. Ordinate: exogenous mortality per 1000 baptisms.

## 11.5 The population boom after 1750

Figures 2.1 and 2.2 show that the overall demography of rural England changed in about 1750 when a national population boom began. What were the causes of this change in the population dynamics? A substantial and progressive increase in the population could have been achieved by an increase in fertility or by a reduction in infant or child mortality, thereby increasing the proportion of the population that reached reproductive age. Probably a combination of these factors interacted to produce the boom, with fertility or mortality having the predominant effect in different populations. In the long term, the overall economy of the metapopulation must have been able to adapt to accommodate the extra people, if necessary by migration.

**Table 11.11** Infant mortality rates (per 1000 live births) in French communities in the seventeenth to nineteenth centuries.

| | Endogenous | Exogenous | Total |
|---|---|---|---|
| Argenteuil, 1740–90 | 64 | 192 | 256 |
| Lézinnes and Vireaux, 1751–80 | 99 | 130 | 229 |
| Villedieu-Les-Poëles, 1711–90 | 72 | 82 | 154 |
| Tamerville, 1624–1792 | 64 | 91 | 155 |
| Bretagne and Anjou, 1740–89 | 97 | 157 | 254 |
| 18 parishes, south of Paris, 1774–94 | 58 | 119 | 177 |
| Crulai, 1672–1742 | 152 | 53 | 205 |
| Trois villages | 94 | 118 | 212 |
| Sotteville-les-Rouen, 1760–90 | 85 | 160 | 245 |
| Sainghin-en-Mélantois, 1740–1849 | 90 | 80 | 170 |

*Source:* Wrigley (1977).

**Table 11.12** Infant mortality rates (per 1000 baptisms) in England in the first half of the eighteenth century compared with mean values for the French communities given in Table 11.11.

| | Infant mortality rates | | |
|---|---|---|---|
| | Endogenous | Exogenous | Total |
| 16 parishes[1] | 38 | 99 | 137 |
| 4 parishes[1] | 79 | 100 | 179 |
| Penrith, Cumbria | 97 | 106 | 203 |
| Shropshire[2] | 35 | 147 | 182 |
| 26 parishes[3] | 81 | 110 | 191 |
| France (mean values) | 87 | 118 | 205 |

[1] Wrigley (1977); [2] Jones (1980); [3] Wrigley *et al.* (1997).

The fall in total infant mortality (Fig. 11.3) made a major contribution to the population boom which began in 1750. Even the 26 English parishes showed a fall in infant mortality after 1740, and the populations at Penrith and the four Shropshire parishes (where mortality rates were very much higher in the late sixteenth and throughout the seventeenth centuries) showed sharp falls by 1680.

Endogenous and exogenous rates exhibited different patterns of change during the seventeenth century. Endogenous mortality rates were high in 1580 at Penrith, probably reflecting the marginal conditions under which the population were living, but fell thereafter until they were comparable with the 26 rural English parishes by 1760 (Fig. 11.4). This progressive change in endogenous mortality with time is also illustrated in Fig. 9.10. Endogenous infant mortality was also high in the four parishes from north Shropshire in the 1580s, but fell sharply and consistently thereafter, reaching a very low level (34 per 1000 baptisms) by 1740 (Fig. 11.4). These dramatic improvements in endogenous, neonatal mortality suggest that, in the late sixteenth and the first half of the

seventeenth centuries, maternal nutrition before and particularly during pregnancy (see section 7.9) was inadequate and so played a dominant role in maintaining the high endogenous infant mortality rates. We conclude that maternal nutrition improved during the period 1650–1750, so that endogenous mortality progressively ameliorated, eventually coming into line with the parishes of central and southern England. As we have suggested (section 9.11), a population that suffered from maternal depletion gave birth to low birthweight girls who would, in turn, produce low birthweight babies, so locking the population into a vicious cycle of high infant mortality.

The studies of 16 and 26 English parishes reveal a different pattern of endogenous mortality. Mean rates were much lower at the end of the seventeenth century (57 and 78 per 1000 baptisms or legitimate births, respectively), but remained at this level for the next 100 years. Only after about 1750 did endogenous rates begin to fall, so contributing to the initiation of the population boom (Fig. 11.4). This low, but constant endogenous mortality in central and southern England is confirmed by the results shown in Tables 11.7 and 11.10. We conclude that maternal nutrition in the more affluent parishes of southern and central England was, on average, more satisfactory than in the marginal communities in the north (and probably also in rural France; Table 11.11), resulting in a 'standard' endogenous mortality rate until the mid-eighteenth century. Thereafter, improvements in economic conditions and agricultural techniques brought about a progressive improvement in maternal diet and, as a consequence, an amelioration of neonatal mortality.

The intergenerational effects of subadequate maternal nutrition, described in sections 9.11 and 9.12, probably also operated, although to a lesser extent, in the less harsh conditions experienced in central and southern England.

Figure 11.5 shows that the pattern of change of exogenous infant mortality was very different both from endogenous mortality in the same study and between the different populations:

(1) Exogenous rates at Penrith rose from 1590 to 1630 and, although falling thereafter, they remained high until 1760. This is in complete contrast to endogenous mortality, which fell throughout this time (Fig. 11.4).
(2) Exogenous mortality in the four parishes of north Shropshire was also high in the 1580s and at the same level as Penrith, but it rose even further after 1640, reaching a peak in about 1680. Thereafter, it slowly ameliorated but remained high, even in 1840 (Fig. 11.5). This pattern is similar to that at Penrith, except that the rise is delayed by about 50 years.
(3) Exogenous mortality in the 16 English parishes shows the same general pattern as that of the Shropshire parishes, although at a lower level. It rose after 1640, peaked in the 1680s and ameliorated slowly thereafter.
(4) The 26 English parishes showed a very different pattern of exogenous mortality. From a rate of 93 per 1000 births, exogenous mortality *fell* until

1640 and thereafter the trend was upwards, with the *highest* level being recorded in the period 1825–37. These results are in contrast to the other studies.

Inspection of Fig. 11.5 reveals a feature of the exogenous mortalities that is of demographic importance: in none of the samples had the rate in 1750 fallen much below the level 150 years earlier, at the end of the sixteenth century, and in most the mortality was higher. We may conclude from this that the fall in total infant mortality in all samples after 1740 shown in Fig. 11.3 is almost completely because of the progressive amelioration in endogenous rates (see Fig. 11.4). We suggest that, probably from about 1700, maternal nutrition during and before pregnancy was steadily improving and the proportion of low birthweight babies and neonatal mortality were falling, so that many populations succeeded in breaking out of the generational knock-on stranglehold. Of course, as endogenous rates fell, more neonates survived to swell the pool of post-neonates, who could then succumb to the vicissitudes of the first year of life.

Why did exogenous mortality remain high during the period 1600 to 1750 and in some cases actually rise, unlike the endogenous mortality rates? We suggest that the following factors may all have contributed to post-neonatal mortality:

(1) Lethal infectious diseases, particularly smallpox, were gaining momentum during this period and may have been responsible for some post-neonatal deaths.
(2) Weaning practices would not have improved: sterilisation was, of course, unknown and weanling diarrhoea would have accounted for many infant deaths, particularly among the sickly. The weaning diet was not completely satisfactory and probably lacked several essential nutrients (see section 8.6).
(3) Although the amelioration of endogenous infant mortality suggests an improvement in the diet during pregnancy, maternal nutrition was almost certainly still not satisfactory. Deaths in the immediate perinatal period may have been steadily reduced, but the majority of the surviving neonates would have had low birthweights. This would have had two effects. First, they would have been more likely to succumb to the vicissitudes of postnatal life. Second, the daughters would inevitably have tended to produce, in turn, low birthweight children, so inexorably promoting the inter-generation, knock-on effect.
(4) Lactation has a high energy cost and requires about 2.5 MJ a day (Frisch, 1999). The breast milk produced when times were hard may not have been adequate in either quality or quantity and so may have contributed directly or indirectly to the continuing high level of exogenous infant mortality.

## 11.6 Effects of nutrition on steady-state population dynamics

The quality and quantity of the nutrition was the dominant demographic factor controlling the steady-state level of discrete populations living under marginal conditions. Nutrition constituted the major part of the density-dependent constraints under which such populations lived. The influence of continued malnutrition was all-pervasive. And, judged by present-day standards, the diet of the bulk of the population during the seventeenth and eighteenth centuries in rural England was incomplete and unsatisfactory and it is remarkable that the metapopulation escaped from the stranglehold in 1750 and the boom began, even in communities which experienced particularly difficult economic and social conditions.

We have described case studies of the demography of Penrith, a particularly susceptible marginal community, and these suggest that poor underlying nutrition, exacerbated by periodic variations in the price of grains, led to a suboptimal reproductive performance and high infant mortality. These effects led to a web of interacting events that are summarised in Fig. 9.16, wherein it is shown how these effects may have contributed to the steady-state population dynamics in this compromised community.

## 11.7 Infant mortality in nineteenth and twentieth century England

We have seen (Table 9.7; Figs 11.1, 11.4 and 11.5) that, in broad terms, endogenous and neonatal mortalities improved steadily in England from the sixteenth to the nineteenth century, whereas exogenous and post-neonatal mortalities showed little sign of amelioration until the end of the eighteenth century. Of course, there was wide variation between different communities but we conclude that the *overall* improvement in infant mortality over a period of 200 years was, in the main, because of steadily improving standards of nutrition.

The amelioration in neonatal mortality in the seventeenth century resulted in a greater proportion of the infants reaching childhood. However, concomitantly, lethal infectious diseases (particularly smallpox; section 15.8) now increased in their ferocity and caused rising mortality in the children.

The savage effects of infectious diseases on childhood mortality continued throughout the nineteenth century (see Chapter 16), but were the effects of improved maternal nutrition reflected in an amelioration of infant mortality through this 100-year period? Several authors have addressed this problem (Woods *et al.*, 1988, 1989, 1993; Lee, 1991; Galley & Shelton, 2001) and we support the view that, *overall*, the infant mortality rate in England and Wales remained at a constant level (150–160 per 1000 births), albeit with fluctuations, until the end of the nineteenth century. Obviously, this disguises wide regional variations (Lee, 1991; Galley & Shelton, 2001). After 1900, it fell sharply to

about 40 in 1950 (Lee, 1991). Woods *et al.* (1989) have established the following points concerning this dramatic amelioration during the first part of the twentieth century. First, the timing of the infant mortality decline was remarkably consistent throughout the country, regardless of district or social class. Second, in urban places, where infant mortality was high, the decline was even more precipitous. Third, although neonatal mortality rates remained relatively constant, suggesting among other things that obstetric practice had not improved radically, post-neonatal mortality fluctuated from year to year and was largely responsible for at least the first stage of the secular decline in total infant mortality rates. Fourth, the increase in infant mortality during the 1890s was largely because of the combination of hot, dry summers creating favourable conditions for epidemic diarrhoea (see section 15.6), and when the effects of diarrhoeal diseases are discounted, infant mortality appears to have declined continuously from 1890. Fifth, the widespread practice of breast-feeding probably gave Britain a lower level of infant mortality than several other western European countries.

Did improved maternal nutrition contribute to the progressive amelioration in infant mortality in the early decades of the twentieth century? Woods *et al.* (1989) suggest that the change was multifactorial:

(1) The decline in fertility, both marital and illegitimate, from the 1870s served to reduce the level of infant mortality by affecting both the number of pregnancies a woman experienced and by increasing intervals between successive births.
(2) Long-term improvements in levels of women's education helped not only to increase the likelihood that family limitation would be attempted, but also served to improve the status of women, their access to information, the way in which they cared for their babies and the way in which they were themselves cared for.
(3) The 'health of towns' movement made significant advances possible.
(4) The improvement in milk supply and food quality; the availability of more highly qualified midwives; the institution of antenatal care and a post-natal health visitor service were all important, but usually they served to reinforce an existing trend by focusing attention on those children most at risk in areas with the highest infant mortality (see also Lee, 1991).

These authors are suggesting, therefore, that improving levels of nutrition had, at best, only a secondary effect on the rapidly-ameliorating infant mortality rate, the most important demographic factor in twentieth century England. However, as we have seen, infant mortality is compounded of a number of different factors and Galley and Woods (1998) have estimated the total endogenous mortality rates (deaths per 1000 births) for England and Wales, 1906–75 (see Fig. 11.6A). This held steady (at 22–24) from 1906 to 1945, but fell progressively thereafter,

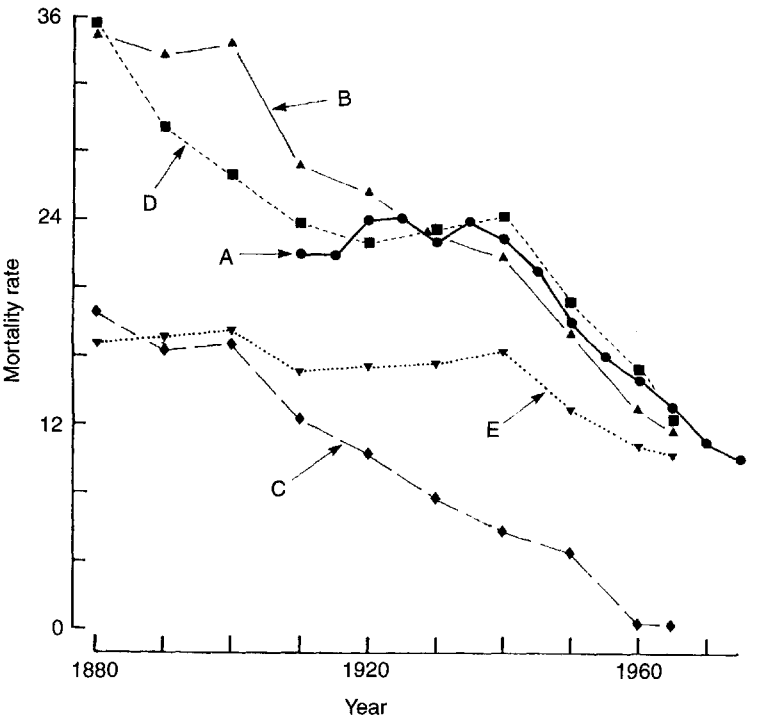


**Fig. 11.6** Infant mortality in the twentieth century. A: Estimated endogenous infant mortality in England and Wales, 1910–75 (data sources, Galley and Woods, 1998); B–E: stillbirth and infant mortality rates, Norway, 1876–1965 (data sources, Wrigley, 1998). B: 0–27 days; C: 7–27 days; D: stillbirths; E: 0–6 days. Ordinate: mortality rate per 1000 births.

with a rate of 10 in 1975. Stillbirths also remained at a steady level up to 1940 and fell steadily thereafter (Hart, 1998).

Since endogenous infant mortality reflects the nutrition *in utero*, it is evident that there was no improvement in the diet of pregnant women in England between 1900 and 1945. The amelioration of infant mortality in the first half of the twentieth century was wholly because of a clear improvement in exogenous infant deaths. A more detailed analysis of infant mortality in England in the 20th century (Hart, 1998) confirms this conclusion: post-neonatal mortality fell progressively but stillbirths and neonatal mortalities remained steady until 1940. We suggest in section 12.5 that the marked improvement in endogenous infant mortality in England after 1945 was because of the increase in the levels of iodine in the diet of women.

Lee (1991) has addressed the question of why the *exogenous* infant mortality ameliorated steadily through the first half of the twentieth century. He highlights that, for England and Wales in the decade 1901–11, the record indicates that 17% of infant deaths were caused by bronchitis, pneumonia and influenza, another 4% by whooping cough, 2% by measles and 12% by cholera, dysentery, diarrhoea

and gastro-enteritis. Less specific causes, like teething and convulsions, which were probably related to some of the complaints noted above, received frequent mention.

These findings may be compared with infant mortality in Norway in the twentieth century (Wrigley, 1998). Infant mortalities for 0–27 days (Fig. 11.6B) and 7–27 days (Fig. 11.6C) ameliorated progressively from 1900, whereas stillbirths (Fig. 11.6D) and deaths during 0–6 days of life (Fig. 11.6E) remained largely steady during 1900–40 but ameliorated thereafter. The very high level of stillbirths in Norway is noteworthy but, according to Hart (1998), reflects the situation in England and Wales at this time.

A decline in the perinatal mortality rate also began in the 1940s in Denmark and Sweden and the factors associated with this amelioration have been studied by Vallgarda (1995). It is suggested that the factors behind this change in the perinatal mortality rate are improvements in the health of the mother, together with the decline in the total fertility rate from the beginning of this century. The risk of infection was reduced with a smaller number of children and the amount of food available to each child and pregnant woman was increased. This better health in early life may then have been associated with improved reproductive health in the adult years.

Chapter 12

# Iodine Deficiency and Endogenous Mortality

Derbyshire neck was a familiar medical condition to earlier generations in England; it was characterised by a goitre, an overdevelopment of the thyroid gland, because of a deficiency of iodine in the environment in Derbyshire (Turton, 1933). Endemic goitre was at one time widespread in Britain (Phillips, 1997) and several studies have shown that there are areas of the world today with a high prevalence of endemic goitre, which is associated with raised mortality from thyrotoxicosis (Clements, 1954; Prendergast *et al.*, 1961). Indeed, iodine deficiency disorders constitute a global problem and are one of the major health issues today. Over 14% of the world's population are estimated to live in iodine-deficient areas and the World Health Organization estimates that at least 1500 million people are at risk today in 118 countries, with 43 million affected by some degree of mental impairment (Houston, 1999).

Unlike many nutritional deficiencies that are more directly related to socio-economic status, insufficient intake of iodine is a geographical disease, related to a lack of iodine in the environment. Iodine, originally present in the soil, was subjected to leaching by snow and rain and, although a portion of the iodine in the oceans evaporates and is returned to the soil in rainwater, this amount is small. Thus, many areas have insufficient iodine in the environment and this is reflected in the low iodine content of the food plants grown there (Houston, 1999). Many developed countries introduced national iodisation programmes during the twentieth century to eliminate iodine deficiency by adding this micronutrient to salt or bread, although this policy, according to Phillips (1997), was never carried out in Britain. Yet endemic goitre has disappeared and iodine intakes in the UK rose progressively during the last half of the twentieth century. He suggests that the most reasonable explanation for this is that changes in farming practice, with iodine supplementation of dairy herds, led to the iodine contamination of milk and dairy produce. The policy of successive governments in encouraging milk consumption eventually eradicated this age-old disease.

Under normal circumstances, about 90% of dietary iodine is derived from food, and the remaining 10% from drinking water. Poor iodine content of food and

water is caused by a low iodine content in the soil from which the food or water is derived. People living in areas of iodine-poor soils and relying on locally-produced foods would have had very low iodine intakes, sometimes below 2.5 μg per person per day, leading to severe iodine deficiency disease (Kavishe, 1999). Leaching of iodine from the soil by rain, glaciers and recurrent floods has concentrated iodine in the ocean waters. Thus, people living in mountainous areas or in areas subject to frequent flooding are at the highest risk of iodine deficiency disease, whereas those near the oceans are least at risk. Because of the high iodine concentration in the oceans, seafoods such as saltwater fish are particularly rich in iodine.

The behaviour of iodine in the soil is not clearly understood and disorders in livestock associated with the deficiency of this element have been described in an area that has none of the contributing factors that are normally characteristic of iodine deficiency. North Melton, Exmoor, southwest England is not characteristic of many other recognised areas of endemic iodine deficiency, in that it is proximal to the ocean, underlain by sandstones, not enriched in fluorine, calcium or arsenic and has not been glacially scoured. Lidiard (1995) asserts that the reclamation and improvement of these moorland soils dramatically reduces their ability to retain iodine derived from atmospheric sources. The element is thought to be lost from reclaimed soils, probably by leaching and possibly by volatilisation, which result from changes in soil pH after liming, the dispersal of organic carbon throughout the profile by ploughing and increased soil aeration consequent upon improvements to drainage.

## 12.1 Seasonal changes in iodine metabolism

Marine fish are the food that is richest in iodine but, except in coastal towns, these would have featured rarely in the diet of pre-industrial England. Cow's milk is another foodstuff that is relatively rich in iodine, except in areas with iodine-deficient soils. Twentieth century studies (Broadhead *et al.*, 1965; Lee *et al.*, 1994) of the iodine content of milk showed a clear seasonal fluctuation: winter milk contained 200 μg/kg, whereas this concentration fell to 90 μg/kg (Lee *et al.*, 1994) or 10 μg/kg (Broadhead *et al.*, 1965) in summer. This large seasonal difference was the result of winter feeding of cattle cake to which iodine had been added in large amounts, a practice that, clearly, was not followed in earlier times. If the iodine concentration of summer milk in the twentieth century reflected that throughout the year in pre-industrial England, dietary sources of this micronutrient would have been insufficient (Hetzel *et al.*, 1987).

## 12.2 Effects of iodine deficiency

Iodine is an essential micronutrient because it is a constituent of thyroid hormones; the thyroxine molecule ($T_4$) contains four atoms of iodine. It is usually

ingested as an iodine or iodate compound and is rapidly absorbed by the intestine. It is then actively trapped by the thyroid gland. Thyroxine released by the thyroid is converted into triiodothyronine ($T_3$) in the tissues. Thyroxine is required in the metabolism of virtually all tissues and, hence, iodine plays an essential role in human and mammalian development. If cows are unable to obtain sufficient iodine from the pastures, their health suffers and their milk does not provide a supply of iodine for humans. The quantitative physiological requirement for iodine is minute; only 100–150 μg per person per day is required for normal growth. Complete lack of iodine is probably incompatible with life, but a deficiency produces a wide spectrum of ill effects which depend on the severity of the deficiency and the stage of life at which the deficiency is experienced (Kavishe, 1999).

In the most simplistic physiological model, inadequate intake of iodine results in a reduction in thyroid hormone production, which stimulates increased production of thyroid stimulating hormone (TSH) by the pituitary gland. TSH acts directly on thyroid cells and, without the ability to increase hormone production because of iodine deficiency, the gland becomes hyperplastic and a goitre develops. A goitre is simply a thyroid gland that is bigger than normal; its size is classified clinically into three grades (Dunn *et al.*, 1986):

0 No goitre
1A Goitre detectable only by palpation and not visible even when the neck is fully extended
1B Goitre palpable and visible only when the neck is fully extended; this stage includes nodular glands even if not goitrous
II Goitre visible with the neck in normal position; palpation is not needed for diagnosis
III Very large goitre that can be recognised from a considerable distance.

However, this simplistic model is complicated by complex adaptive mechanisms which vary depending on the age of the individual affected. In adults with mild deficiency, reduced iodine intake causes a decrease in extrathyroidal iodine and reduced clearance, but iodine concentration in the gland may remain within normal limits. With a further reduction in intake, this adaptive mechanism is overwhelmed and the iodine content of the thyroid decreases with alterations in the iodination of thyroglobulin. The ability to adapt appears to decrease with decreasing age, and in children the iodine pool in the thyroid is smaller and the dynamics of iodine metabolism and peripheral use more rapid. In neonates, the effects of iodine deficiency are more directly reflected in increased TSH. Diminished thyroid iodine content and increased turnover make neonates the most vulnerable to the effects of iodine deficiency and decreased hormone production, even with mild deficiency (Houston, 1999).

In addition to causing a recognisable goitre, iodine deficiency is the most

common cause of mental retardation; the most severe effect is cretinism, which is rare in areas of mildly endemic deficiency, but may have reached 5–10% in areas with severe deficiency. All the effects of iodine deficiency are generally more severe and permanent when they occur early, rather than later, in life. The consequences in the adult are more serious in women, particularly when pregnant, than in men: endemic goitre, like thyrotoxicosis, is more prevalent among females than males (Phillips *et al.*, 1983; Wynn & Ma, 1998). The major disorders resulting from iodine deficiency are summarised in Table 12.1, and abortions and neonatal and infant mortality are of particular demographic importance. Equally important is the not readily-detectable effect of iodine deficiency on lowered fertility and reproductive failure in women. Compared with women living in iodine-sufficient areas, mothers in severely iodine-deficient areas experience more abortions, miscarriages, stillbirths and other problems of pregnancy. Most significantly, they also bear children with higher rates of congenital abnormalities, lower birthweights and lower survival rates, as indicated by a higher perinatal and infant mortality (Hetzel & Mano, 1989; Hetzel, 1989; Pharoah & Connolly, 1991). Women presenting with mild thyroid abnormalities also had a greater thyroidal risk during pregnancy, and thyroid autoimmunity was clearly associated with an increased risk of spontaneous abortion (Glinoer *et al.*, 1991). The reason that women suffer from thyroid disorders between 4–10 times as frequently as men is because iodine deficiency in pregnancy can damage the thyroid; this is not true of women who have never been pregnant (Wynn & Ma, 1998).

The evidence indicates that these varying manifestations of iodine deficiency in the foetus probably arise from lowered $T_4$ levels in the blood of the iodine-

**Table 12.1** Range of disorders resulting from iodine deficiency according to the life stage at which the deficiency occurs.

| Life stage | Major disorders |
|---|---|
| Foetus | Abortions, stillbirths, congenital anomalies, foetal hypothyroidism, increased perinatal and infant mortality, psychomotor defects, low birthweight, cretinism |
| Neonate and infancy | Neonatal hypothyroidism, neonatal goitre, increased neonatal and infant mortality, retarded mental and physical development |
| Childhood and adolescence | Goitre, juvenile hypothyroidism, impaired mental function, retarded physical development |
| Adult | Goitre with its complications – hypothyroidism, impaired mental and physical function, iodine-induced hyperthyroidism. Reduced sperm motility and sperm counts. Reduced fertility |

Data from Hetzel *et al.* (1987), Kavishe (1999), Geelhoed (1999), Del Rio *et al.* (2000).

deficient mother, and the lower the level of maternal $T_4$, the greater the threat to the integrity of the foetus (Hetzel, 1989). All these manifestations can be prevented today by iodine supplementation which, for example, reduced infant mortality in Subang, West Java and Indonesia (Cobra *et al.*, 1997). These studies indicate that iodine deficiency would have had a serious impact on child survival and development before the twentieth century.

However, to be wholly effective, iodine deficiency has to be corrected *before* ovulation and conception; if this is done, the mortality of the children is significantly reduced and their motor and cognitive functions are improved. Much research has been done on the contribution of thyroid hormones to the growth and development of the ovarian follicle and the ovum. High rates of gene transcription are essential during the maturation of the ovum and sperm before conception and this requires an adequate concentration of thyroid hormones around the germ cells (Samuels *et al.*, 1988; Chin & Yen, 1996; Helmer *et al.*, 1996). A slow-down in the rates of cell replication and growth is a first consequence of a reduction in the concentration and local availability of thyroid hormones, notably $T_4$. This reduction in growth rate is mediated to different degrees by hormones particularly concerned with growth. The family of growth hormones called insulin-like growth factors (IGFs) are found in all parts of the body and are partly regulated by thyroid hormones (Fagin *et al.*, 1989; Wolf *et al.*, 1989; Ceda *et al.*, 1992; Cacicedo *et al.*, 1993). The rate of growth of the ovarian follicle and subsequent size of the corpus luteum are partly controlled by IGFs and thyroid hormones.

Animal experiments have shown that if iodine deficiency begins during the period preceding mating, it causes a much more serious range of congenital malformations than if it begins only a few days after mating (Langman & van Faasen, 1955). Low $T_4$ levels resulting from iodine deficiency can cause mutations in both males and females that may be inherited in their $F_1$ offspring and in the following $F_2$ generation (Wauben-Penris & von Buul-Offers, 1982) and so may have important, but not readily-detectable, demographic effects.

## 12.3 Endemic goitre in England

Endemic goitre occurs when the prevalence of thyroid enlargement in the population of an area exceeds 10% (Lamberg, 1991). Iodine may have had subliminal effects on a woman's fertility by being deficient either in the drinking water or, more importantly, in the soil and, hence, in the vegetation. In the latter situation, both the cows (and hence their milk) and the women in the area may have suffered from malnutrition.

Phillips (1997) records that Inglis reported in his *Treatise on English bronchocoele, with a few remarks on the use of iodine and its compounds* that endemic goitre was as common in the Yorkshire dales in 1838 as in Geneva or any of the

Alpine valleys. Children could be seen at play with pieces of black velvet tied around their necks to charm away the goitre evil. Other early records reported that goitre and even cretinism, usually associated with severe iodine deficiency, could be found in several English counties, including Norfolk, Monmouthshire and Cornwall (Kelly & Snedden, 1960). Cretinism was said to be notorious in Chiselborough, Somerset. An English goitre belt was recognised (McEwan, 1938; Barker & Phillips, 1984; Phillips, 1997) which extended from the West Country through Somerset into the Cotswold and Chiltern Hills and northwards into Derbyshire and the Peak District. There were well-defined offshoots affecting both north and south Wales (Fig. 12.1). In 1924, a survey of 12-year old schoolchildren in England and Wales ($n = 375\,000$) confirmed the geographical pattern of the disease shown by previous studies and revealed a high prevalence of visible goitre (up to 30% in some communities), which led to calls for a national iodisation programme. Goitre appeared to be unusually common in young women drafted into factories during the 1939–45 war. Several surveys showed visible goitre to be present in 50% of the adult women in Hook Norton, Oxfordshire, in 43% of girls in Sherborne, Dorset, and in 26% of boys and girls in St Albans, Hertfordshire. In contrast, only 2% of children showed thyroid enlargement in Maldon, Essex, where the drinking water is rich in iodine (Phillips, 1997). Figure 12.1 shows the areas of England and Wales where endemic goitre has been prevalent in the past.

Barker and Phillips (1984) have conducted a study of the incidence of thyro-

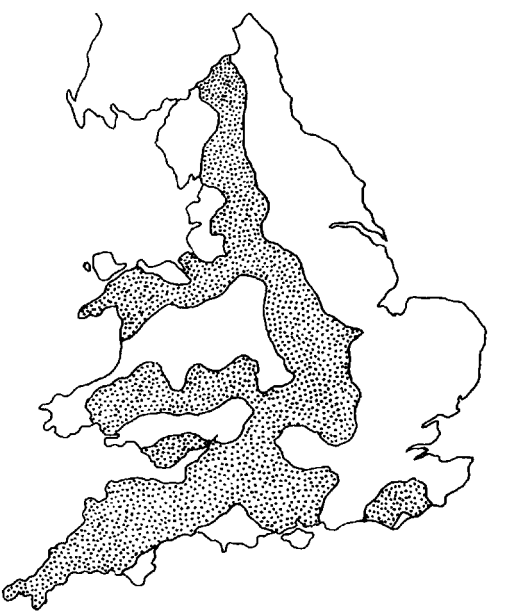

**Fig. 12.1** Areas of England and Wales (stippled) where endemic goitre has been prevalent in the past. After Phillips (1997).

toxicosis in 12 towns spread through England and Wales which was strongly correlated with the previous prevalence of endemic goitre. They conclude that current high dietary intakes of iodine (which are largely the result of milk contamination) may cause toxic modular goitre in people made susceptible by a lack of iodine in early life. See also Geelhoed (1999).

## 12.4 Case study of Chesterfield, Derbyshire

Goitre has been endemic in Derbyshire for many centuries and Turton (1933) has summarised the earlier references to the condition in 1602, 1684 and 1782. Chesterfield was described in 1867 as being one of four endemic centres of goitre and we have therefore examined the parish registers for evidence of a shortage of iodine having any demographic effects.

Chesterfield is a medium-sized market town, 24 miles north of Derby in the fertile vale of Scarsdale. The mean annual number of baptisms in the early part of the seventeenth century was 75 and the population of the parish in 1821 was 9000. Pigot's Directory of 1835 describes it as a prosperous town with a free grammar school founded in the sixteenth century which was formerly one of the largest in the north of England.

A superficial inspection of the parish registers, which date from 1558, does not suggest that a location in the goitre centre of Derbyshire caused any marked demographic abnormalities. We therefore carried out a full family reconstitution for the period 1588–1634 from which monthly infant mortality can be determined: total baptisms were 3533, total infant mortality was 769 (21.8%), total neonatal mortality was 547 (15.5%) and total post-neonatal mortality was 222 (6.3%). These results suggest that neonatal mortality during this period was high, whereas post-neonatal mortality was relatively low.

This conclusion is supported by the Bourgeios-Pichat plot shown in Fig. 12.2. Extrapolation of the line for 1 to 5 months gives an estimate for the endogenous mortality of 135 per 1000 baptisms, which contrasts with an exogenous mortality of 83 per 1000 baptisms. This endogenous rate is comparable to that of 131 per 1000 baptisms at Penrith in 1577–99, but the mortality there falls to 101 per 1000 baptisms during 1600–49 (see Table 9.1). The endogenous mortality rate of 135 at Chesterfield greatly exceeds those estimated by Wrigley *et al.* (1997) for the 26 parishes in rural England that they have studied: 77.6 (1580–99), 88.5 (1600–24) and 80.0 (1625–49).

We conclude that the most serious *demographic* effects of living in the iodine-deficient area of Chesterfield operated on the women in pregnancy and resulted in high endogenous and neonatal mortalities. Abortions and stillbirths were not recorded in the parish registers, but we suspect that these were also at a high frequency. Iodine deficiency, apparently, did not have a marked detrimental effect on the survival of post-neonatal infants. Indeed, the exogenous mortality

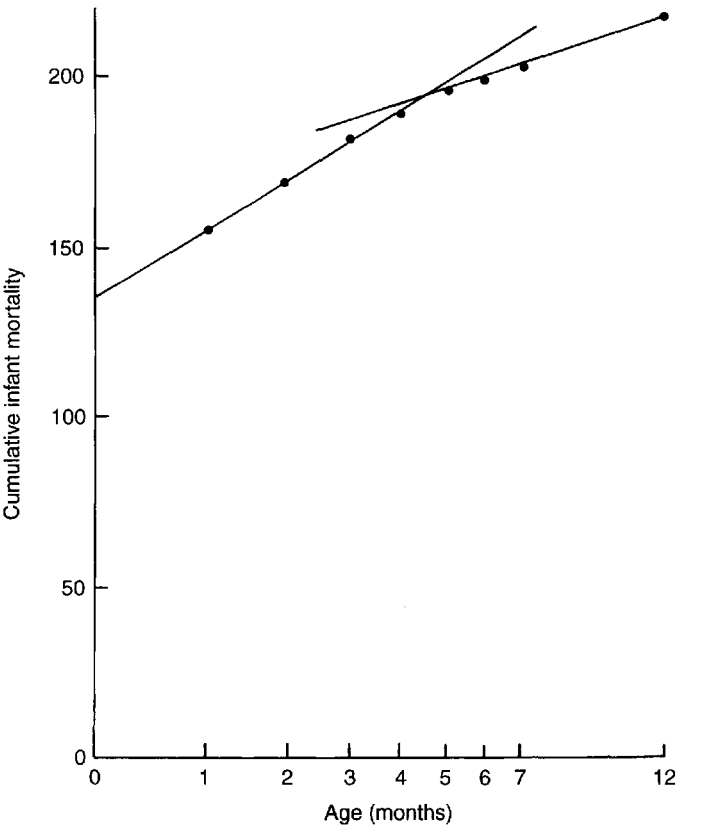


**Fig. 12.2** The Bourgeois-Pichat model: infant mortality at Chesterfield, 1588–1634. Ordinate: cumulative infant mortality (deaths per 1000 baptisms). Abscissa: age (months) plotted as $[\log (n+1)]^3$, where $n$ is the age in days since baptism. Data from family reconstitution study. Estimated endogenous mortality by extrapolation = 135 per 1000 baptisms.

rate at Chesterfield is low, but this may be because so many of the infants had already died as neonates.

We have also carried out an aggregative analysis of two further Derbyshire parishes, namely Dronfield and Wirksworth. These Derbyshire parishes were not operating under steady-state dynamics and so were not under density-dependent control. Instead, geographical iodine deficiency was exerting a density-independent regulation of population numbers via an exacerbation of endogenous mortality.

## 12.5 Endogenous mortality and iodine deficiency in England and Wales in the twentieth century

Phillips (1997) has summarised the national responses to iodine deficiency during the twentieth century as follows. Countries with endemic iodine deficiency have

tended to solve the problem by the compulsory iodisation of staple foodstuffs, such as bread or salt. However, research in Britain in the 1920s showed that iodine supplementation of livestock could improve their reproductive performance. This provided an economic incentive for the iodine supplementation of dairy herds, which was achieved by providing iodised salt licks for cattle (introduced by Boots in 1928) and subsequently by the use of iodine enriched cattle feed (marketed by Spillers in 1937). The result was a spectacular rise in the iodine content of milk, particularly during the winter months when pasture is in short supply and cattle are dependent on iodine-rich artificial feed. The use of iodinated casein, given to cows as a lactation promoter, and iodophor disinfectants, used for cleaning teats and bulk milk tankers, also contributed to the iodine contamination of milk.

In parallel with this increase in iodine content, successive governments put policies into effect to increase the consumption of milk and dairy produce. Before the 1930s milk had been a luxury affordable only by wealthier families. A survey of 1932 found a mean intake of only 3.1 pints per week. The subsequent establishment of the Milk Marketing Board and the Milk Act (1934) and the National Milk Scheme (1940), which provided milk free or at a subsidised price for all expectant mothers and young children, greatly boosted consumption.

By the early 1950s average liquid milk consumption had nearly doubled. Figure 12.3B and C illustrates how the amount of iodine provided by average milk consumption in the UK has increased since 1931. By the 1980s the iodine content of milk during the winter was such that milk alone could almost satisfy the recommended dairy requirement of 150 μg/day. Indeed, the iodine content in some milk samples was so high that this led to concerns about possible toxic effects. The increase in the iodine content of milk and related dairy products is likely to have been the major explanation of the 3-fold rise in dietary iodine intakes, from 80 to 255 μg/day, which occurred between 1952 and 1982. Iodine intakes were also boosted by the iodine enrichment of meat and eggs, probably because of the same processes of spillover from the use of iodine in animal feeds.

This steady mitigation of the problems of iodine deficiency in England and Wales in the twentieth century may be compared with the corresponding annual rates of endogenous infant mortality (Galley & Woods, 1998) shown in Fig. 12.3A. This remained constant (21–24 deaths per 1000 births) for 40 years from 1906 to 1946, whereupon the situation ameliorated steadily and progressively, falling to 10 endogenous deaths per 1000 births by 1975.

Thus, as can be seen from Fig. 12.3, the clear improvement in endogenous mortality rates correlated with the average daily intake of iodine from milk (Phillips, 1997). This finding suggests that the iodine intake of women in England and Wales may have been a major (but not readily detectable) demographic factor in the twentieth century.

This conclusion is supported by a study of stillbirths and infant deaths resulting from congenital abnormalities in Tasmania, Australia (Potter *et al.*, 1979). Tas-

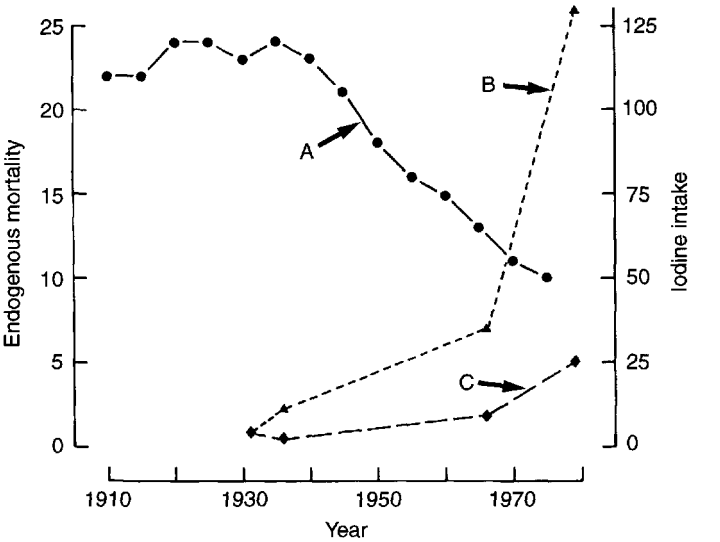


**Fig. 12.3** Amelioration of endogenous infant mortality compared with the average daily iodine intake from milk in England and Wales in the twentieth century. A: Estimated endogenous mortality per 1000 births (left hand ordinate); data from Galley and Woods (1998). B and C (right hand ordinate): average daily iodine intake from milk (g/day) in the UK based on surveys of milk intake and the iodine content of milk. B: winter samples; C: summer samples; data from Phillips (1997).

mania began iodine supplementation in 1950 and stillbirth rates declined more rapidly than in Australia as a whole. Congenital anomalies causing death showed an initial rise in the early 1950s and it is suggested that this was because of the persistence of iodine deficiency in the foetus after maternal iodine status had improved sufficiently to allow term delivery of a live infant. The later fall coincided with the first cohort of Tasmanian women, who had been iodine-sufficient since childhood, reaching reproductive age. These mothers would therefore not have deprived their own foetus of iodine. Examination of trends in New Zealand, Switzerland and Finland, which have each introduced iodisation programmes, confirms the largely beneficial effect of iodine supplementation on the rate of infant death associated with congenital anomalies.

Chapter 13

# Seasonality

When humans changed from a hunter-gatherer to an agricultural lifestyle, they automatically (certainly in Europe) became locked into the annual cycle of vegetative growth which determined the availability of food. The activity throughout the year was geared to the production of the main grain crops which were harvested in the summer. The food harvested had to last for the next 12 months and had to provide the necessary seed corn. There is also a seasonal pattern to food production in the tropics, usually geared to the rainy season. If the crop should fail because of climatic or other factors, the results could be literally catastrophic and the effects would be felt for two to three years.

From nutritional and demographic viewpoints, the critical period in the year was the hungry season. Its effects on demography were seen in pre-industrial England back to medieval times. Grass was cut and dried, and hay was harvested in July; it provided fodder for the animals. This was now the toughest time of the year, the spring crops had not yet matured and the grain stores were at their lowest point and could well have been empty. Paradoxically, people could be starving in the balmiest month on the eve of the August harvest. The period July–August was the time of the hungry gap, a phenomenon unknown in the modern Western world. The divide between the rich and poor (who formed the bulk of the population) became most apparent at this time. The rich could survive on the contents of their barns and could afford to pay the higher prices commanded by the dwindling stocks of food. The poor tried to survive through their annual midsummer purgatory, grinding up the coarsest of wheat bran and even shrivelled peas and beans to make some sort of bread (Lacey & Danziger, 2000).

Thus, the annual cycle imposed on an agriculturalist economy based on grains is the consequence of the regular progression through the seasons. There are a number of points in this annual cycle of events when a woman's nutritional status may have been compromised:

(1) The hungry season came in the summer when stocks of grain were running low but when heavy work in the fields demanded a higher energy input.
(2) During the autumn, grains became more freely available after the harvest but it would take some time for a woman's fat reserves (on which her fer-

tility depended; section 6.3) to be re-established after the depletion experienced during the hungry season.

(3) Probably, of almost equal importance, a woman's intake of key vitamins or minerals could be compromised by malnutrition in the hungry season. Current studies show that a woman's ability to conceive can be dramatically improved by taking a daily multivitamin pill (section 6.6).

(4) The supply of milk (particularly important in northern England) tended to fail during the winter because, in part, many cattle were slaughtered in the autumn because of the lack of grass in the forthcoming winter. Additional energy was required in the diet because of the high metabolic demands of maintaining homeothermy in the bitterly cold winters. A woman's diet was probably also deficient in vitamin C in winter.

We suggest, therefore, that there may be critical periods during the year when a woman's fertility or pregnancy may suffer risks in addition to the overall poor level of nutrition. These critical periods may fall during conception, the first, second or third trimesters of pregnancy, or during the early months of infant life and so, as we have seen, they may have markedly different seasonal effects on fertility and neonatal mortality.

## 13.1 Astrology

The health and future development of a neonate is strongly dependent on the nutrition of the mother during the different trimesters of pregnancy (see section 7.9). Since the quality of the maternal diet in pre-industrial Europe varied seasonally, it would be expected that children born in different months of the year would have different underlying health problems and long-term prospects.

Astrology is bunk, but there is a growing body of research that shows that, even today with a steady supply of nutrients during pregnancy throughout the year, there might be some trends in our mental and physical characteristics associated with the season in which we were born:

- English professional soccer players in the 1991–92 season were almost twice as likely to have been born between September and November as during the summer months.
- Fast bowlers at cricket were likely to have been born in the early part of the year.
- Babies born between January and March had a higher bone mineral content, lower serum osteocalcium and higher serum calcium than babies born between July and September, suggesting that the vitamin D status of the mother 6 months prior to parturition affects the calcium status of her offspring.
- Men born between March and May were, on average, 6 mm taller than those

born between September and November when conscripted into the Austrian army over a 10-year period.

- People born in winter tend to get fatter in later life.
- Winter babies tend to live longer.
- In a study lasting from 1929 to 2000, it was found that more people with schizophrenia and manic depression were born between December and April than would be expected by chance (Thomas, 2000).
- A study of children born in Denmark, 1973–94, shows that those born in December were, on average, 19 mm shorter than those born in April.
- A recent study of 6000 women in Scotland reveals that those who have the eating disorder, anorexia nervosa, were twice as likely to have been born in the month of June as October. The spring months of March to April are also a peak period. It is suggested that the reason may be that the mother suffered from a winter infection such as influenza during pregnancy. The foetal brain is at its most sensitive during the second trimester and, if it is damaged by an intra-uterine infection, anorexia may develop many years later.

## 13.2 Seasonality of births

Clearly, the foregoing seasonal effects could be the result of viral infections of the mother during pregnancy, or environmental factors such as weather and temperature, as well as a fluctuating nutritional level of the mother. These minute effects are of no demographic significance and so do not concern us here, but they do illustrate how events during pregnancy may have subtle effects on the fate of the offspring in later life.

Much more important is the seasonal fluctuation in births in developing countries today, suggesting an underlying seasonal cycle in fertility. We briefly summarise some of these studies below:

(1) Lese subsistence farmers and nomadic Efe pygmies who lived in the Ituri Forest in northeast Zaire, 1980–87: rainfall patterns determined the size of the Lese gardens which, in turn, determined the nutritional status for both the Lese and the Efe. Negative changes in female nutritional status diminished ovarian function. Conceptions were lowest in May and highest in September; there were fewer conceptions during periods of poor food availability. Bailey *et al.* (1992) conclude that the causal change of these demographic effects began with rainfall, which determined food production, which determined energy balance, which determined ovarian function, which determined fertility. They suggest that this model can be applied to any population in any geographical region; it integrates knowledge of behaviour and cultural practices with the biology of human reproduction.

(2) Crook and Dyson (1981) make two generalisations concerning demographic seasonality. First, the phenomenon can be found in data from all around the world and, second, for any one country, there is a considerable degree of consistency in seasonality from year to year. Some regional patterns of seasonality in births are as follows: (i) births along the North African coast peak in December–February and are low between May and September, (ii) births peak in the Caribbean around September and January, (iii) the countries with similar seasonal birth patterns are in regions of the world that are comparatively homogenous from socio-economic, cultural, and climatic points of view, (iv) many countries near the equator experience some seasonality in their births, and (v) in no developing country north of the equator do the estimated maximum number of conceptions occur in June–August, and the peaks for conceptions are usually December–May. For all countries in the Caribbean, Middle America and South America, the peak months for marriages are December–February.

(3) Igbo Ora, a rural area in southwestern Nigeria, 1965–75: the monthly number of registered births showed a consistent and significant pattern, with a peak in May and lowest values recorded in November, with a variation about the mean of $\pm$ 7%. Ayeni (1986) concludes that this seasonal pattern may be related to climatic factors as well as sociocultural patterns of life associated with cyclical farming activities characteristic of a predominantly agricultural community.

(4) In Kenya, monthly variations in the number of births display a measure of seasonal regularity with a major peak in September and a subsidiary one in April–May. The results suggest a positive correlation between conception rates and food availability (Ferguson, 1987).

(5) Mean birthweight peaked in the later part of the dry season in rural Tanzania and fell at the beginning of the rainy season, reaching its lowest level during or just after the rains. The births peak corresponds to a conception peak in December–January, coinciding with the height of the birthweight and seasonal birthweight variation. The most striking finding was the combination of high mean birthweight and a strong birthweight seasonality in the low rainfall areas, and of low birthweight and a weak birthweight seasonality in the high rainfall areas. It is suggested that seasonal birthweight fluctuations are influenced by broad shifts in the birthweight distribution related to variations in food supply and health-related factors. Both these components are related to climatic seasonality, mediated by the agricultural cycle. In the drier areas, birthweight seasonality appears to result primarily from fluctuations in food supply (Bantje, 1987).

(6) In a study of San bushmen of the Kalahari Desert, in northwestern Bots-

wana, 1973–80, Wilmsen (1982) noted seasonal fluctuations in diet and positive correlations between fertility and body composition.

(7) The frequency of monthly births in a fishing village in Taiwan, 1926–76, showed no significant correlation with time of year. This difference from other patterns observed in Taiwan is attributed by Mosher (1979) to the dependence of the population on year-round fishing rather than on one or two major harvests, where there is a birth seasonality based on nutritional fluctuations.

(8) A rural community in Gambia, 1949–85, experienced seasonality of food intake, energy expenditure and infectious disease. Seasonal troughs in birth rate were related to seasonally low intakes of dietary energy, and childhood mortality was lowest in children conceived at the time of year when fertility was lowest (Ulijaszek, 1993).

(9) Birth seasonality was examined in a non-contracepting population with low fertility and seasonally heavy workloads in rural Nepal, 1983–93. Both the frequency distribution of live births and a life-table analysis of women's exposure to the risk of conception show significant seasonality. Two troughs were observed, one in the winter and the other in mid-monsoon. The former is probably associated with behavioural variables affecting intercourse, namely the temporary separation of spouses. The latter coincides with suppressed ovulation and low progesterone profiles, and indicates a temporary loss of fecundity for women in negative energy balance during the monsoon agricultural season (Panter-Brick, 1996).

(10) In a study in Matlab, rural Bangladesh, it was found that weight gain was substantially lower in pregnancies that began in May or June. Indeed, women who conceive during these two months lose weight on average and the pregnancies are much more likely to result in an intra-uterine death (Pebley *et al.*, 1985). The following months, June to October (the first trimester) are the lean period in Matlab because they immediately precede the major rice harvests. Before the diet improves, the rice must be harvested and processed. While the men do most of the actual harvesting, women are responsible for the arduous tasks of threshing, parboiling, drying and husking the rice.

Rosetta (1993) concludes that those populations that are most exposed to external conditions are those with a 'natural' way of life, usually experiencing poor living conditions and directly dependent on the land and the climate for their food resources. Under such conditions the factors influencing fertility are more likely to be nutrition and physical workload, rather than rainfall and temperature. A combination of seasonal food shortages and high levels of physical activity, if acute, may well regulate reproductive function in women and she

believes that there is evidence to suggest that the quality and quantity of food intake may play a role in such regulation. Many rural populations living in tropical or equatorial areas today have heavy physical workloads during the rainy season which may act synergistically to impair reproductive function.

## 13.3 Seasonality of infant deaths in developing countries

In contrast to the foregoing studies that report a seasonality in the number of births (and hence in fertility) in developing countries with an agricultural lifestyle today, there appear to be fewer reports and little direct evidence of a seasonality in infant mortality.

Chao & Merritt (1991) have surveyed 11 subSaharan African countries and have studied the effect of seasonality on neonatal and post-neonatal mortality and on the nutritional status of children 3 to 36 months old. The low rainfall countries (Mali, Senegal, Botswana and Zimbabwe) had the highest percentages of neonatal deaths; neonatal deaths in Mali and Senegal were higher than post-neonatal deaths throughout the year, except during 2–3 months of the dry season. On the other hand, a high percentage of neonatal deaths occurred during the first few months of the rainy season in Botswana and Zimbabwe. Those countries with an average annual rainfall of 47–56 inches (Kenya, Uganda and Burundi) had higher percentages of post-neonatal deaths than neonatal deaths. Indeed, in Kenya, post-neonatal deaths were higher in all but 2 months of the year. The West African countries of Ghana and Togo had higher percentages of neonatal deaths than post-neonatal deaths; neonatal deaths always outpaced post-neonatal deaths in Ghana and the difference between the two grew during the dry season. Liberia, with the highest average rainfall (about 120 inches), had, on average, 4% higher post-neonatal deaths than neonatal deaths, although the latter exceeded post-neonatal deaths during the rainy seasons. Overall, infants born during the dry season tended to be stunted because conception occurred during the rainy season when women were at greater risk of nutritional depletion.

Becker (1981) has analysed the seasonal pattern of deaths in a rural area of Bangladesh, 1972–4, and concludes:

(1) The number of neonatal deaths had a peak in October but, after adjustment for the seasonal pattern of births, the actual risk of neonatal mortality was found to peak 2 months earlier.
(2) Post-neonatal deaths peak in April with an above average proportion of deaths in the peak month attributed to dysentery.
(3) Deaths of persons above the age of 45 years peak in the cool season.
(4) Dysentery and chronic diarrhoea deaths are highest in December.
(5) Accidental deaths are maximum in July because of increased drownings during the monsoon.

Moore *et al.* (1999) have studied three rural Gambian villages, 1949–99, which were affected by a marked annual seasonality in diet and disease. They conclude that early life exposures, correlated with the season of birth, strongly influence susceptibility to fatal infections in young adulthood. The evidence suggests that nutritionally-mediated intra-uterine growth retardation may permanently impair the development of immune function.

It is evident that any attempts to detect the effects of a seasonal variation in nutritive levels on neonatal mortality, even in subsistence agricultural communities today, are complicated by the direct effects of climatic variation (particularly temperature and rainfall) and by the incidence of lethal infectious diseases.

## 13.4 Seasonality of baptisms in England, 1613–30

The early decades of the seventeenth century were a time of great hardship in northwest England and events culminated in the famine of 1623 (see section 4.11). If the constraints of the annual cycle of the farming year had a seasonal effect on fertility, it might be expected that this would be most readily detected during the period 1613–30. We have, therefore, examined the monthly baptisms in 42 parishes during this period. About 50% of these were upland parishes of the northwest, with the other parishes acting as controls:

| | |
|---|---|
| Cumberland: | Bridekirk, Greystoke, Penrith, Crosthwaite |
| Westmorland: | Newbiggin, Cliburn, Morland, Lowther, Brough, Crosby Ravensworth, Kendal |
| Cheshire: | Bebington |
| Lancashire: | Thornton-in-Lonsdale, Urswick, Cartmel, Lancaster, Cockerham, Poulton-le-Fylde, Whalley, North Meols, Prestwich, Sefton, Ingleton, Walton-on-the-Hill |
| Lincolnshire: | Lincoln (St Marys), Grantham, Alford |
| Durham: | Whitburn, Coniscliffe, Bishop Middleham |
| Devonshire: | Parkham, Plymtree, Exeter (St Pauls, All Hallows, Cathedral) |
| Sussex: | Cowfold, Cuckfield, Horsham, Bolney, Ardingley, Angmering |

The results are shown in Fig. 13.1B; there were no significant differences between the parishes and the total monthly baptisms were plotted. Baptisms peak in February and it is concluded that the peak period for births is January–February. The results show a clear correlation with monthly baptisms, 1600–49, in the 404 rural parishes of England studied by Wrigley and Schofield (1981) (see Fig. 13.1A). Thus, Fig. 13.1 shows that, in both studies, baptisms rose sharply after December and remained at a high level until April, whereupon the birth rate fell to a trough in the summer, May to August. These monthly totals are significantly different ($P < 0.001$).

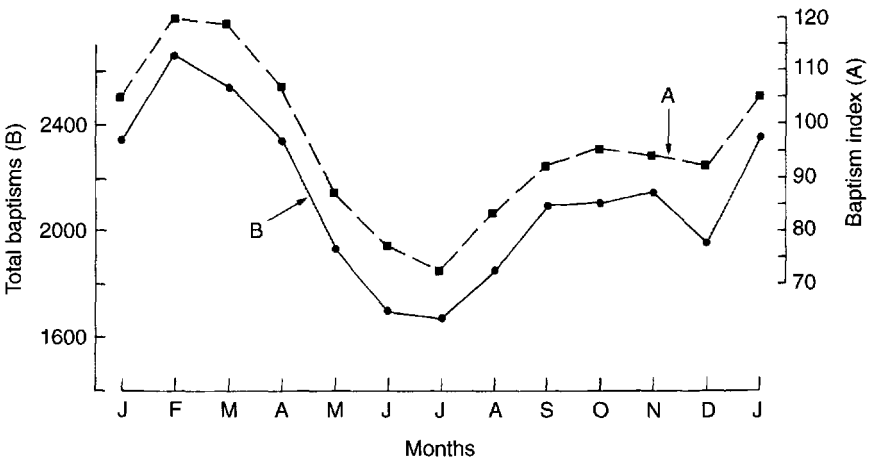


**Fig. 13.1** Monthly baptisms in England in the first half of the seventeenth century. A: Index of 404 rural parishes, 1600–49. Data from Wrigley and Schofield (1981) (right hand ordinate = monthly index). B: 42 rural parishes, 1613–30 (left hand ordinate). Abscissa: months of the year.

## 13.5 Seasonality of baptisms at Penrith, Cumbria, 1600–1800

The series of monthly baptisms at Penrith, 1600–1800, is shown in Fig. 13.2A. Again, there is a clear drop in baptisms in the summer (minimum in July), which was found to be most pronounced in the early cohorts (1600–99); the seasonal effect was reduced after 1700. In summary, over this 200-year period, baptisms at Penrith were at their highest level January to March and fell sharply thereafter to a trough in May to August, with a minimum in July. After August, the level of baptisms recovered erratically through the autumn, with a secondary peak in September. This seasonal pattern corresponds closely with the results given by Wrigley and Schofield (1981) for the 404 parishes for the period 1600–1800 (Fig. 13.2B).

Figure 13.3 shows the monthly totals of baptisms at Penrith, 1600–1800, subdivided into the three social classes (elites, trades and subsistence farmers) that have been identified there (see section 9.8). There are very much smaller numbers in the data series for the elites and no significant seasonality in their baptisms is detectable (Fig. 13.3E).

The trades (Fig. 13.3D) and subsistence (Fig. 13.3C) classes show the same annual pattern of baptisms, with a significant peak in February–March and a trough in June–July ($P < 0.01$). It is interesting that the maxima and minima for the subsistence precede those for the trades by one month. The two data series were pooled and are shown in Fig. 13.3B, in which the peak in February–March and the trough in May–August can be seen; baptisms rise erratically after August to the February peak. This monthly variation is statistically significant ($P < 0.001$).

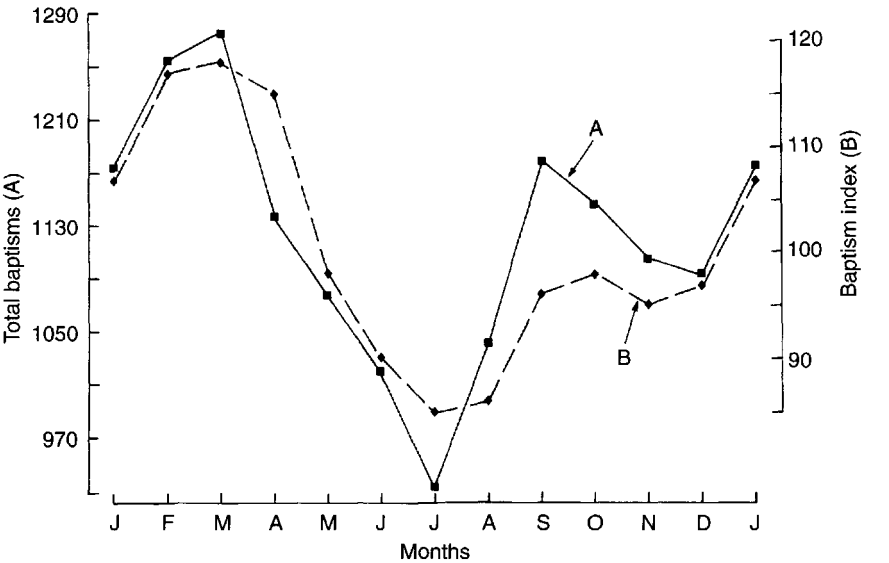


**Fig. 13.2** Seasonality of baptisms. A: Total. monthly baptisms at Penrith, 1600–1800 (left hand ordinate); B: monthly baptism index for 404 rural parishes in England, 1600–1800 (right hand ordinate). Data from Wrigley and Schofield (1981). Note close correspondence of the two plots.

## 13.6 Seasonality of baptisms in England, 1600-1800

We conclude from the foregoing sections that there was a significant seasonality in the monthly number of baptisms in England, 1600–1800, with a peak in February–March and a trough in May–August. These conclusions can be compared to the findings of Wrigley and Schofield (1981), who report similar findings for the 404 parishes that they studied. Their data for 1600–49 are shown in Fig. 13.1A and correspond almost exactly with the results for the 42 parishes, 1613–30 (Fig. 13.1B). Both show a maximum/minimum ratio of 1.6. The averaged data of Wrigley and Schofield (1981) for the cohorts between 1600 and 1800 are shown in Fig. 13.3A and can be compared to Fig. 13.3B; both plots show a maximum/minimum ratio of 1.4.

Inspection of the detailed results provided by Wrigley and Schofield (1981) shows that, for the period 1540 to 1749, this pattern of a February–March peak and a May–August trough (with a July minimum) remained constant, although with a small fall in the maximum/minimum ratio after 1650. After 1750, the difference between the peak and the trough (maximum/minimum ratio) became progressively attenuated and the peak moved to April–May, with the minimum in August. We conclude that as conditions and nutrition steadily improved, particularly after 1750, the marked monthly variation was reduced and seasonal events were delayed by 1 to 2 months. Wrigley and Schofield (1981) report that

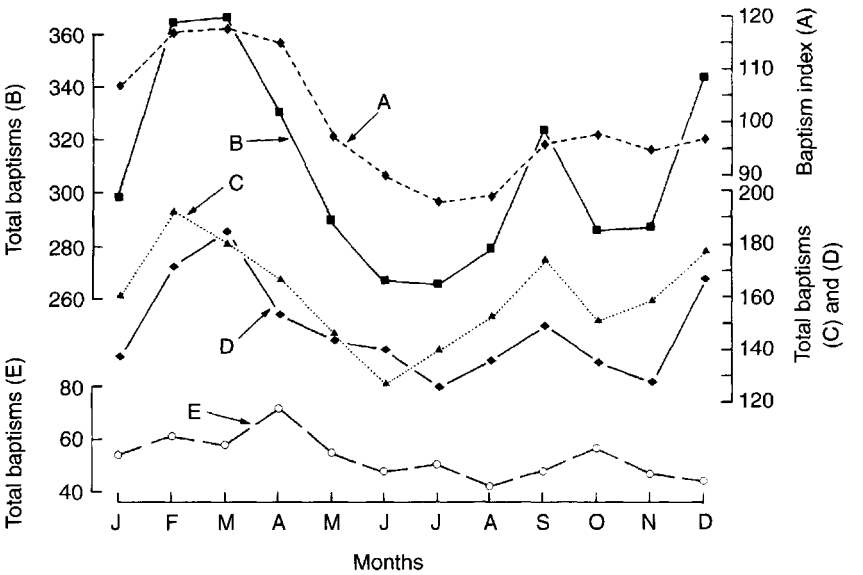


**Fig. 13.3** Seasonality of baptisms at Penrith, Cumbria, 1600–1800. A: Monthly baptism index for 404 rural parishes of England, 1600–1800, for comparison (upper right hand ordinate). Data from Wrigley and Schofield (1981). B: Total baptisms for trades and subsistence classes combined (upper left hand ordinate). C: Total baptisms for subsistence class. D: Total baptisms for trades class (lower right hand ordinate). E: Total baptisms for elite class (lower left hand ordinate).

not only was this seasonal pattern of baptisms persistent over a long time in England, but it can also be found over much of northwest Europe.

Figures 13.1 and 13.3 show a spring maximum and a summer minimum in baptisms, although the recovery after August is erratic (Figs 13.1B and 13.3B). A different pattern is seen in different cohorts but, in general, the baptisms tended to show less monthly variation after 1700, with a plateau from September to December.

The peaks in baptisms show that fertility and conceptions in pre-industrial England, particularly 1550–1750, were highest during the months April to July (peaking May–June), but thereafter fell dramatically in August and remained at a low level until the end of November (minimum in October). Since these conclusions are derived from baptism series and not from births, the mean dates for maximum and minimum fertility are probably on average some 2 weeks earlier.

We suggest that women, particularly in the period before 1750, had built up their fat stores and were enjoying their best (although still suboptimal) diet in the months immediately before the hungry season when the weather was warm (and the energy demands of homeothermy were reduced) and dairy produce was available and so they had their highest fertility. The fat reserves were rapidly depleted during the hungry season when food was scarce and there were

increased energy demands caused by the manual labour of the harvest and, consequently, a woman's fertility fell dramatically, reaching a minimum in October. Once the harvest was gathered in, the women slowly rebuilt their fat reserves. The elite women at Penrith may possibly have escaped from this marked seasonality of fertility (see Fig. 13.3E).

## 13.7 Seasonality of neonatal mortality in pre-industrial England

Since neonatal mortality (particularly endogenous mortality) is determined in part by maternal nutrition at critical periods during pregnancy, it might be expected that it would show a strong seasonality. However, although neonatal mortality in pre-industrial England fluctuated seasonally, it was difficult to detect a persistent and significant monthly pattern.

Wrigley *et al.* (1997) present a figure for the endogenous mortality (presumably the most sensitive indicator) in their reconstitutions of 26 English parishes, 1580–1837 (see Fig. 13.4C). There are winter peaks in December–January followed by a trough in which the monthly totals are erratic, but with a minimum in October. Calculations suggest that this monthly variation was not significant, but any underlying trends may be masked, as we shall see, by variations over the 250-year

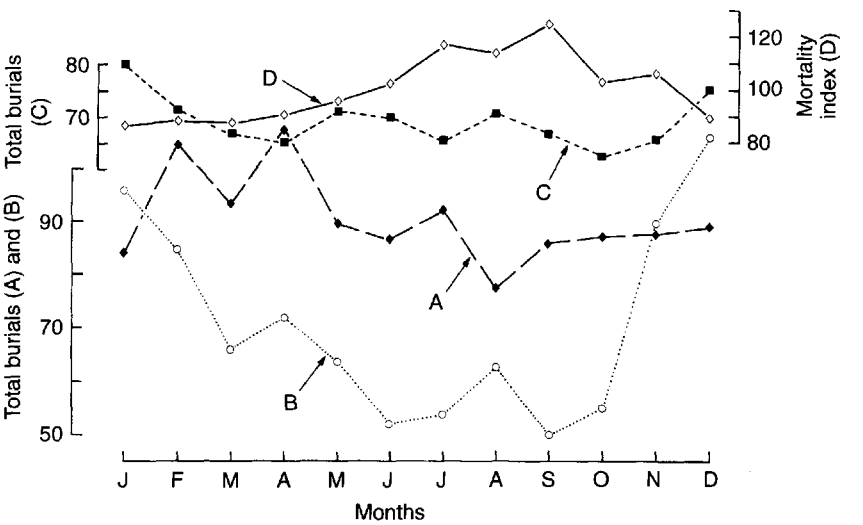


**Fig. 13.4** Seasonality of neonatal mortality. A: Monthly total neonatal burials, 1613–30, in 22 parishes in which no seasonal pattern could be discerned. B: Monthly total neonatal burials, 1613–30, in 20 parishes in which a clear seasonal pattern can be seen (lower left hand ordinate). C: Endogenous infant mortality in 26 rural English parishes per 1000 baptisms, 1580–1837 (upper hand ordinate). Data from Wrigley *et al.* (1997). D: Neonatal mortality index in the parishes within the city walls of York 1561–1700 (upper right hand ordinate). Data from Galley (1998). Note very small peak in deaths in September.

period of study and by the inclusion in the aggregation of parishes where there was no seasonality in infant mortality.

Inspection of the monthly neonatal mortalities, 1613–30, for the aggregated 42 parishes listed in section 13.4 shows that in 22 of these there was no seasonal pattern (Fig. 13.4A). The other 20 parishes showed (Fig. 13.4B) a clear and significant ($P < 0.001$) monthly variation, with a peak in November to January and with minima in June–July and September.

In summary, seasonality of neonatal mortality is not readily detectable in pre-industrial England; many communities apparently were not sensitive to seasonal events; in others, the pattern of the response changed over the centuries; in others, the seasonal effect was probably too small to be statistically significant. Nevertheless, Fig. 13.4B suggests that neonatal mortality in some communities was high over the winter, with a December–January peak and at a minimum in June–July. Of particular interest is the dramatic change between October and November in Fig. 13.4B: neonatal mortality is at a low level in October but rises sharply in November.

We present an analysis of the key features of Fig. 13.4B in Table 13.1; the critical feature appears to be the occurrence of the hungry month of July during the second trimester of pregnancy. Infants born in the months of June and July suffered the lowest neonatal mortality (rows 1 and 2, Table 13.1); the mothers in this group, conceived September to October, did not suffer from the adverse effects of the hungry season during their pregnancy. Neonatal mortality was still low in October (row 3, Table 13.1); these infants were conceived in January, and July fell in the third trimester. Neonatal mortality was highest in babies conceived February to April (rows 4–6, Table 13.1), when July fell in the second trimester.

**Table 13.1** Summary of the key features of the neonatal mortality shown in Fig. 13.4B.

| Conceived | Trimester | | | Baptised | Mortality level |
|---|---|---|---|---|---|
| | 1st | 2nd | 3rd | | |
| 1 Sept | Sept–Nov | Dec–Feb | Mar–May | Jun | Minimum |
| 2 Oct | Oct–Dec | Jan–Mar | Apr–Jun | July | Minimum |
| 3 Jan | Jan–Mar | Apr–Jun | [Jul]–Sept | Oct | Low |
| 4 Feb | Feb–Apr | May–[Jul] | Aug–Oct | Nov | High |
| 5 Mar | Mar–May | Jun–[Jul]–Aug | Sept–Nov | Dec | Maximum |
| 6 Apr | Apr–May | [Jul]–Sept | Oct–Dec | Jan | High |

*Note:* The occurrence of the hungry month of July is highlighted in square brackets.

The main feature of foetal growth is cell division, on which undernutrition has a direct effect. The tissues of the foetus grow during periods of rapid cell division at different times during pregnancy, and growth depends on the availability of nutrients and oxygen. Even brief periods of undernutrition may permanently reduce the number of cells in specific organs (Barker, 1999b). Post-mortem

examination of aborted foetuses or of babies who died perinatally reveals a high frequency of lung hypoplasia, suggesting that lung growth *in utero* is often impaired. Airway division down to the level of the terminal bronchioles is completed by week 16 of gestation. This is followed by a period of rapid lung growth, so that the lung cell population doubles between weeks 17 and 20, in the middle of the second trimester. Thus adverse influences may impair airway development or enhance alveolar growth, depending on their nature and timing, and so lead to airflow obstruction. Barker (1998) concludes that undernutrition in mid–late gestation is likely to impair airway growth.

We conclude that a significant seasonality of neonatal mortality is not detectable in many parish registers, but in several communities neonatal mortality was clearly highest in those foetuses conceived between February and April and who experienced the hungry season (centred on July) during their second trimester. Rapid growth and development of the lungs occurs during the second trimester and many of these neonatal deaths were probably linked with a failing respiratory function.

Galley (1998) has provided a comprehensive review of the demography of the parishes within the city walls of York and it is interesting to compare the seasonality of the neonatal mortality there with that experienced in rural communities, described above. The estimated index of seasonal neonatal mortality at York, 1561–1700, calculated as a proportion of monthly baptisms is shown in Fig. 13.4D. The pattern is strikingly different from that of the 20 rural parishes that showed a marked seasonality. Neonatal mortality at York remained at a steady rate from December to April, but began to rise progressively in May and peaked in September. Thereafter, it fell sharply in October and was again at its steady level by December. In the city of York in the seventeenth century, therefore, the neonates at greatest risk were conceived around the month of December, when the second trimester of pregnancy fell in the period March to May. These were probably the critical months for a foetus at York. Neonatal mortality was also high (see Fig. 13.4D) in August (March and April in the second and May in the third trimester) and July (March in the second, and April and May in the third trimester). We conclude that the city of York was less at risk during the hungry season than were agriculture-based, rural communities.

We have shown (Scott & Duncan, 1998) that the infant mortality cycles at York were most significantly associated with high wool prices and not with wheat prices. The situation in the seventeenth century was evidently different in York from that in northwestern England, perhaps explicable by different economic conditions. Galley (1995) has shown that York was a trading centre serving a rural hinterland; it was experiencing a population increase and was also an important administrative centre. The King's Council in the north, which acted as the northern parliament, was based in York from 1561 to 1641 and this administrative role stimulated the economy of the city, and the inns and victualling trades were important in catering for numerous visitors. It was a large

market centre, with butchers, cordwainers, bakers and tailors dominating the occupational structure. International trade was also important, and wealthy merchants exported cloth and other merchandise down the Ouse and on to the Continent. We suggest that, although infant and child mortalities were sensitive to the synergistic effects of wheat and wool prices, the prosperous community at York was largely protected during the three famines of 1586, 1597 and 1623 (see section 4.10) because of its diversified trading interests. Mortality was exacerbated by cycles of high (rather than low) wool prices, presumably because York was a buying and exporting market rather than a selling and producing market that was characteristic of the farms and communities in the fells of Cumbria.

## 13.8 Seasonality of abortions and stillbirths

The vicar or clerk recorded abortions or stillbirths in the burials register in a small number of parishes in England. We have analysed the following records for abortives and stillborns:

- St Michael-le-Belfry, York, 1571–86: abortives; 1633–1749: stillbirths
- Hawkshead, Lancashire, 1586–1704: abortives
- Ashton-under-Lyne, Lancashire, 1608–39: abortives; 1653–1700: stillborns
- Greystoke, Cumberland, 1579–1629: stillborns and abortives

The monthly totals for the number of abortions per 1000 live births for these four parishes are shown in Fig. 13.5A. The total number of abortions fall sharply from the February peak and remain at a low level only during March to June, rising sharply thereafter to a summer–autumn–winter plateau.

The monthly totals for the burials of non-baptised infants plus baptised-and-buried infants has been determined for the following parishes:

- Greystoke, Cumberland, 1579–1629: not baptised
- Ashton-under-Lyne, Lancashire, 1599–1639; 1653–1700: not baptised
- Horsham, Sussex, 1579–1635: unbaptised, baptised-and-buried
- Kendal, Westmorland, 1606–31: unbaptised

The monthly aggregated totals are shown in Fig. 13.5B. The totals fall sharply from a peak in February to a minimum in April and stay low through spring and summer, but rise again in November for a winter and early spring peak.

We have analysed the seasonality of the following three different types of perinatal deaths.

(1) Abortives were deaths of foetuses before birth which may have occurred at the seventh or eighth month of pregnancy, for example. They died in the womb because of adverse effects during pregnancy (see Fig. 13.5A).

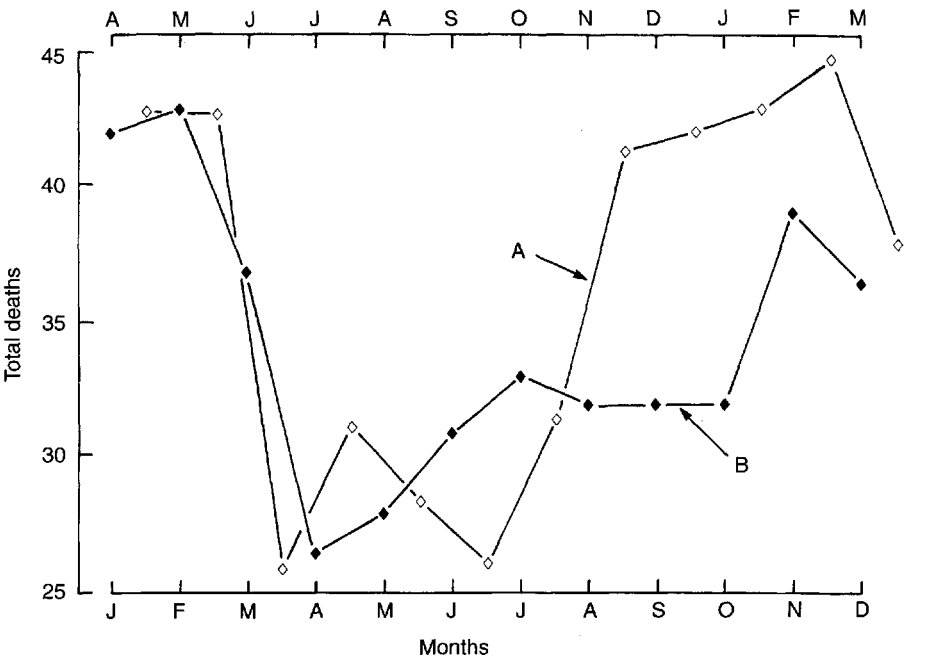


**Fig. 13.5** Seasonality of abortions and deaths in the first few hours of life. A: Total monthly abortions per 1000 baptisms in St Michael-le-Belfry, York, 1571–86; Hawkshead, 1586–1704; Ashton-under-Lyne, 1608–39; Greystoke, 1579–1629. Line for abortives moved 6 weeks to the right to allow for an average early termination of pregnancy. B: Total monthly infant deaths in the first few hours of life per 1000 baptisms (see text for further details) in St Michael-le-Belfry, York, 1633–1749; Ashton-under-Lyne, 1599–1639, 1653–1700; Greystoke, 1579–1629; Horsham, 1579–1635; Kendal, 1606–31. Lower abscissa: month of recorded death. Upper abscissa: month of conception.

(2) Unbaptised plus baptised-and-buried infants went to term (although they may have been premature) and were born alive, but died within a few hours of birth. These deaths may have been because of unfortunate circumstances at parturition, but most will have been the result of adverse effects in the womb (see Fig. 13.5B).

(3) Neonatal mortality, i.e. infants who were born alive but died in the first month of life. This series will include infants listed in (2) above and analysis is further complicated by the variable time between birth and baptism. Most will have failed to flourish during the first month because of compromised development (Fig. 13.4B). We have suggested (see Table 13.1) that the highest levels of neonatal mortality were recorded when the hungry season (particularly centred around the month of July) fell during the second trimester, with births November–January.

Both the monthly plots for the abortives (Fig. 13.5A) and infants dying in the first hours of life (Fig. 13.5B) fall sharply from winter peaks to minima in Feb-

ruary (abortives) or April (non-baptised). We suggest that the aborted foetuses were dying after 7 or 8 months of pregnancy and the plot for the abortives has therefore been moved 6 weeks to the right in Fig. 13.5A. The mortalities of the abortives and unbaptised infants can now be directly compared and are summarised in Table 13.2.

**Table 13.2** Summary of the monthly mortality levels of abortives and unbaptised infants based on Fig. 13.5.

| Conceived | Trimester | | | Mortality | |
|---|---|---|---|---|---|
| | 1st | 2nd | 3rd | Abortives | Unbaptised |
| Jul | [Jul]–Sept | Oct–Dec | Jan–Mar | Minimum | Minimum |
| Aug | Aug–Oct | Nov–Jan | Feb–Apr | Low | Low |
| Sept | Sept–Nov | Dec–Feb | Mar–May | Low | Rising |
| Oct | Oct–Dec | Jan–Mar | Apr–Jun | Low | Medium |
| Nov | Nov–Jan | Feb–Apr | May–[Jul] | High | Medium |
| Dec | Dec–Feb | Mar–May | Jun–[Jul]–Aug | High | Medium |
| Jan | Jan–Mar | Apr–Jun | [Jul]–Sept | High | Medium |
| Feb | Feb–Apr | May–[Jul] | Aug–Oct | High | High |
| Mar | Mar–May | Jun–[Jul]–Aug | Sept–Nov | High | High |
| Apr | Apr–Jun | [Jul]–Sept | Oct–Nov | High | High |
| May | May–[Jul] | Aug–Oct | Nov–Jan | High | High |
| Jun | Jun–[Jul]–Aug | Sept–Nov | Dec–Feb | Probably falling | Falling |

*Note:* The occurrence of the hungry month of July is highlighted in square brackets.

Conceptions during the period July to September suffer the lowest levels of mortality in either series; if the hungry season does not fall within the 9 months of a pregnancy, the chances of successful development of the foetus are greatest.

Conceptions around October are of interest; the incidence of stillbirths remains low (Table 13.2), but these probably aborted around the seventh month (April) and so did not experience the hungry season. In contrast, the mortality of infants who were conceived in October and who died at birth rose to a medium level. Conceptions during November to January also resulted in a medium level of mortality of newly-born infants, whereas abortions rose to their highest levels. In summary, the hungry season falling in the third trimester resulted in a high level of abortives, but only a medium level of mortality of newly-born infants. With conceptions in February to May, the foetus was exposed to the hungry season during the critical second trimester and also at the end of the first trimester, resulting in high levels of both abortives and newly-born infants.

To conclude: many communities in pre-industrial rural England were affected by the seasonality imposed by the constraints of the agricultural way of life. The

hungry season, centred on the month of July, could reduce fertility in the lowest social groups and, particularly if it fell in the second trimester, could affect the development of the foetus, leading to abortions, stillbirths and raised endogenous mortality.

Chapter 14

# Sex Ratios

The demographic importance of the imbalance in the numbers of the two sexes in different populations has been comprehensively reviewed by Clarke (2000). There are rarely equal numbers of males and females in human populations, either today or in earlier centuries. Countries with a marked shortage of males may have suffered recent wars, or have unusually high levels of male mortality, or have experienced major emigration of men. Countries with a clear shortage of females may have experienced an immigration of men, an emigration of women or an unusually high female mortality.

Famine or severe malnutrition may lead to migratory movements, so having a direct demographic effect (see section 10.7), but in this chapter we consider whether the sex ratio at birth can be modified by the nutrition of the mother during pregnancy. In general, there is a preponderance of male over female births and the sex ratio at birth is defined as

$$\frac{\text{number of live male births}}{\text{number of live female births}} \times 100$$

This is termed the secondary sex ratio to distinguish it from the primary sex ratio at conception.

It is generally accepted that the primary sex ratio is very much higher than the secondary sex ratio because of higher male prenatal mortality. It has been variously estimated to be up to 160, but may be 123 to 130 (Bittles *et al.*, 1993), although accurate assessments are very difficult. Excess mortality of male foetuses during gestation reduces the ratio to about 110 at term. There is a preponderance of male stillborn babies so that the sex ratio of live births is about 105 (Clarke, 2000).

The secondary sex ratio lies in the range 104–108 in the majority of national populations of the more developed countries. The ratios are more variable in less developed countries: some Central and South American countries record ratios of 100–104 whereas a few Asian countries report ratios of over 110. The sex ratio is higher for Asian than for white births (James, 1985; Ruder, 1986) and higher for white than for black births (Beiles, 1974; James, 1984). The secondary sex

ratio in England and Wales rose from 104 in 1911 to 106 during 1951–71, but then fell to its current level of about 105. Slight declines in the ratio have also been recorded in other industrialised countries.

## 14.1 Why are there more live male births?

Since it is generally agreed that the primary sex ratio is substantially more than 100, there appears to be an obvious answer to this question. More males are conceived, with a primary sex ratio of perhaps about 120, because, it is generally assumed that sperm carrying the small Y chromosome will have a smaller load and will therefore, on average, travel faster than sperm with an X chromosome and so have a better chance of achieving fertilisation. Differential mortality in the womb then reduces the sex ratio at birth to about 105. Environmental effects could potentially modify the relative success of the X and Y sperm, or the differential mortality during gestation.

Teitelbaum (1972) has reviewed 29 (mainly behavioural) factors that have been claimed to be associated with the secondary sex ratio, some of which can be regarded as serious, but does not mention the nutrition of the mother (or of the father) before and during pregnancy. Clarke (2000) believes that the topic is complex and multidimensional because it is difficult to distinguish between the relative contributions of various factors. Among the many suggestions we may briefly list the following:

- Teitelbaum (1972) considered that birth order was the most important factor, the ratio diminishing irregularly from the first birth.
- The number of male births is believed to rise during post-war baby booms, associated, it is said, with the rise in the number of first marriages (Teitelbaum, 1972).
- People of high social status and greater prosperity are said to have more sons than normal.
- Male occupations with significantly increased incidences of defective sperm are associated with reduced numbers of male children.
- High coital rates are said to lead to high sex ratios.
- James (1987) hypothesises that the hormone levels of both parents at the time of conception affect the probability of a male birth; high levels of oestrogen and testosterone increase this probability, whereas high levels of gonadotropin decrease it.
- More boys are said to be born after a heatwave, the most important effect being a rise in temperature about a month before conception. Presumably this effect of temperature would act during the maturation of the sperm (Lerchl, 1999).

## 14.2 The importance of sample size

Some of the hypotheses summarised in the preceding and following sections appear to be merely speculations. We believe that others may be the result of too small sample sizes. We have found that sex ratios in populations can fluctuate sharply on an annual basis and, if small samples are used, misleading results can be obtained.

Stein and Susser (1975) have analysed monthly variations in sex ratios during the Dutch hunger winter (see section 4.5). They present data over 36 months (1944–46) from the famine area and from two control areas. In the north control area in 1945, where *monthly* births totalled from 600 to 800, the monthly sex ratio ranged from a very low value of 82.6 to a high of 121.8. Total births for the year for this area were 8590, an apparently sufficiently large sample for statistical purposes, yet the sharply fluctuating monthly totals could be interpreted to support a variety of hypotheses. When the data series for the 3 years for the two control areas are pooled (total births = 65 000), the monthly sex ratios varied from 103.1 (November) to 109.8 (October), i.e. the ratio never fell below 100. The mean sex ratio overall is 107. Similarly, when the data for the famine area are pooled (total births = 154 000) the monthly sex ratios range from 103.2 (June) to 108.7 (January), with an overall mean of 106.3. There is no significant difference between the monthly sex ratios.

In summary, hypotheses concerning changes in sex ratios based on small samples should be accepted with caution. Nevertheless, genuine oscillations over long periods of time may be lost if the data are aggregated to produce large samples.

## 14.3 The preference for sons

The desire for sons and the belief that boys matter more than girls is an attitude that has long been present in most countries and is particularly prevalent in those societies where sons are the main source of economic security for parents in their old age. It is most deeply entrenched in many more traditional cultures with patriarchal social structures. This preference for sons is expressed to a variable degree as male preference and female neglect (including infanticide, Smith & Smith, 1994) throughout childhood and adulthood (Clarke, 2000). For example, the number of girls in India is still declining relative to boys. The sex ratio rose from 102.9 to 107.5 between 1921 and 1971 (Griffiths *et al.*, 2000). From 1991 to 2001, the number of girls for every 1000 boys under 6 has fallen from 945 to 927, and is as low as 793 in the Punjab. Only the highly educated southern state of Kerala shows a balance favouring women. Several authors believe that this abnormal sex ratio can be explained in terms of missing females and estimates range from 22 to 40 million. Many believe the

ability of modern medicine to detect prenatal gender allows couples to abort female foetuses; sex-determination clinics are very popular, especially in the northern farming states. However, Griffiths *et al.* (2000) have simulated the population dynamics of India and suggest that small differences in the mortality of children persisting over a long period, coupled with a sex ratio of 106, results in a highly male-biased population.

These studies have contributed to the evolutionary theory of sex allocation, i.e. that parents may invest more heavily in their children of one particular sex, particularly if resources are scarce (Maynard Smith, 1978; Charnov, 1982). Sociobiological theory predicts that the greater variance in reproductive success of males will lead parents with more resources to prefer to raise sons, whereas those with fewer resources would prefer daughters (Brittain *et al.*, 1988). We discuss sex-biased investment in more detail in section 9.11. This hypothesis has been developed to include adaptive modifications of the sex ratio, although the mechanisms suggested are not convincing.

Finally, the theory of sex allocation has apparently been extended to the point where, it is suggested, a woman can control whether her ovum is fertilised by an X or Y sperm. The mechanisms by which this might be achieved are not explained. We give two examples: Grant (1995) states

> 'My own biological-dominance hypothesis rests on the possibility of *differential spermatozoan access under the control of the female* [our italics]. I have shown that women who score high on self-report paper-and-pencil measures of dominance taken in the time period from eight weeks before conception to eight weeks after do conceive significantly more male infants ($P < 0.001$) and, conversely, that women whose scores on dominance are below the average for women are significantly more likely to conceive female infants. At present I believe that the male's role in the predetermination of the sex of the infant ceases at the point of mate selection. I would interpret Martin's data as suggesting that in difficult times women had to be (temporarily) more biologically dominant and this was reflected at the hormonal level. Most if not all of the known facts fit this biological-dominance hypothesis....'

Manning *et al.* (1997) claim that the age difference between parents (age of husband minus age of wife, $D_a$) predicts the sex of the *first* child, with an excess of sons in marriages with a high $D_a$. However, $D_a$ (mysteriously) has *no effect on the sex of subsequent children* of the marriage. These workers conclude

> 'In humans, the elite often form partnerships with high $D_a$ and have more sons than daughters. It may be that during wartime women prefer to marry older men with high resources and this leads to an increase in $D_a$. We do not know how the sex of first-borns is adjusted in relation to $D_a$. Women could *influence the motility of sperm* bearing either X or Y chromosomes or they *may invest*

*differentially in males and females in utero* [our italics] leading to higher miscarriage rates of one or the other sex...'

This is obvious nonsense.

## 14.4 Does maternal nutrition affect the primary or secondary sex ratios?

If sex ratios at birth do genuinely fluctuate, the most likely agencies to produce this effect are the nutrition and stress of the mother before and during pregnancy. They could operate differentially either (i) before and during conception or (ii) on the mortality of the foetus.

The generally accepted view is that changes in cervical mucus (consistency, viscosity, pH) could differentially modify the swimming performance of the sperm and possibly augment or nullify the advantage of those carrying the Y chromosome. The endocervical canal is filled with a mucus gel, the properties of which vary during the ovulatory cycle. The amount of mucus increases at mid-cycle, mainly because of an oestrogen-induced increased hydration of the gel; the mucus becomes less visco-elastic and the penetration of the spermatozoa is facilitated. In contrast, under the influence of progesterone during the luteal phase, mucus turns into a less hydrated, highly visco-elastic structure which acts as a barrier to sperm. The mucus gel is formed by very large and structurally complex glycoproteins which behave as random coils and occupy large spheroidal domains in dilute solution. The glycopeptides correspond to long stretches of protein covered with 100–300 oligosaccharides which protect the core from proteolysis. These regions of the macromolecule are referred to as oligosaccharide 'clusters' and subunits of cervical mucins contain, on average, two to five of these 'clusters'. Each 'cluster' is flanked by stretches of protein which are less substituted with carbohydrate and, consequently, more sensitive to proteolysis (Carlstedt & Sheehan, 1989).

X- and Y-carrying sperm are believed to prefer different conditions and to swim at different speeds. Stress would cause hormonal changes in the mother which could, in turn, modify the cervical mucus. Marked changes in nutrition might have a similar effect.

It is generally accepted that the mortality of the male foetuses is higher than that of the females. The growth trajectory of boys is more rapid than that of girls from early in embryonic life (see section 7.5) and Barker (1998) suggests that boys may therefore be more vulnerable to undernutrition. In summary, changes in the nutrition of the mother during pregnancy could augment or reduce this differential mortality between the sexes of the foetuses.

There is a range of scattered observations and studies that suggest that malnutrition and stress may interact to modify the secondary sex ratio and we summarise them briefly below:

- A study of 3282 children born to 684 women in a rural area of the Central African Republic suggests an association between maternal malnutrition and low sex ratio (Andersson & Bergstrom, 1998).
- Significant positive correlations were obtained between the amount of food that a country had available and the percentage of male births. Increases or decreases in the calories available were correlated with corresponding changes in the sex ratio of the country. Williams and Gloster (1992) suggest that nutritional deficiencies cause higher mortality rates for male embryos and foetuses.
- A statistically significant larger number of girls were born in poor harvest years in a Portuguese rural parish, 1671–1720, whereas in good years boys were born in excess of the expected sex ratio (Cowgill & Johnson, 1971).

Chapter 15

# Childhood Mortality and Infectious Diseases

We have seen in Chapter 11 how infant mortality ameliorated progressively in pre-industrial England, largely because of an improvement in endogenous infant mortality. The effect was more clearly seen in a population in the northern territory living under marginal conditions. Exogenous infant mortality was less responsive to improved nutrition but, nevertheless, because of the reduced neonatal mortality, a greater number of 1-year-olds were reaching the childhood years when they encountered the next major threat – lethal infectious diseases. Plague disappeared in Europe after 1670 (Scott & Duncan, 2001), but by that time a more deadly strain of smallpox had emerged (Scott & Duncan, 1998) which was killing some 20% of the children. Other potentially lethal infectious diseases were also becoming established – measles, whooping cough, scarlet fever and diphtheria, and so the improving levels of infant mortality were demographically cancelled out by rising mortality in childhood.

The traditional association between food shortage and mortality from epidemic disease is well documented. It is widely believed that malnourished individuals are more likely to die from infectious diseases than those who are well fed (Schellekens, 1996). However, it is proposed that 'the evidence linking malnutrition and mortality is surprisingly sparse' and research suggests that not all infections are affected in the same way by malnutrition. It is believed that many of the major diseases of the past, such as smallpox, typhoid and malaria, were influenced by nutritional status only to a minimal extent (Watkins & Van de Walle, 1985). The evidence concerning the susceptibility to malaria infection in relation to malnutrition is inconclusive (Bruce-Chwatt, 1985; Dyson, 1991). However, an improvement in nutritional levels may have played a role in ameliorating the health status of malarial populations allowing victims a better chance to combat attacks of the disease (Dobson, 1997).

## 15.1 The biology of infectious diseases

In a discrete population, say a large town in pre-industrial England, an infectious disease can exist in either an epidemic or an endemic state. In the epidemic

condition, the infectious agent is brought into the population by an infective and, if there is a sufficient density of susceptibles, an epidemic will explode. The disease spreads rapidly until most susceptibles have been infected, whereupon the epidemic fizzles out completely. There are no explosions in the inter-epidemic years and for the disease to be re-initiated the number of susceptibles must rise by new births to a critical level and an infective must come into the population (Scott & Duncan, 1998).

The endemic situation can be found in pre-industrial England in the larger cities and in the metropolis. The disease is persistent and there are always cases of the disease. Endemic disease is found only in populations of sufficient size and density for there to be a constant supply of susceptibles (new babies) to maintain the endemic level. In the majority of such populations, regular epidemics were superimposed on this endemic level.

As we have seen (section 2.5), England, Scotland and Wales can be considered as an island metapopulation. If an infectious disease died out *everywhere* in the metapopulation, it could be restarted again only by an infective coming from overseas. An example is haemorrhagic plague that brought devastating epidemics to England at irregular intervals between the middle of the fourteenth century and 1670. The plague was not endemic before about 1600 and there were no cases in the inter-epidemic intervals (Scott & Duncan, 2001).

## 15.2 Epidemiology and modelling of infectious diseases

Bailey (1975) has provided a comprehensive overview of the mathematical theory of infectious diseases, which Anderson and May (1991) developed into a study of the theory of epidemics that follow when a virus is introduced into a susceptible population. Thereafter events follow very rapidly: most of the susceptibles become infected before the epidemic dies out and the survivors become immune. These are termed SEIR (suspectibles-exposed-infectives-recovered) models and can be summarised as follows.

The population, $N$, is assumed to remain constant where the net input of susceptibles (births) equals the net mortality $\mu N$ (where $\mu$ = death rate; life expectancy = $1/\mu$). The population is divided into susceptibles ($X$), latents (infected, not yet infectious, $H$), infectious ($Y$) and recovered and hence immune ($Z$). Thus, $N = X + H + Y + Z$.

It is assumed that the net rate at which infections occur is proportional to the number of encounters between susceptibles and infectives, $\beta XY$ (where $\beta$ is a transmission coefficient). Individuals move from latent to infectious at a *per capita* rate, $\sigma$. They recover, thus becoming immune, at rate $\gamma$. The dynamics of the infection are then described by the following equations:

$$dX/dt = \mu N - \mu X - \beta XY \quad (15.1)$$

$$dH/dt = \beta XY - (\mu + \sigma)H \quad (15.2)$$

$$dY/dt = \sigma H - (\mu + \gamma)Y \quad (15.3)$$

$$dZ/dt = -\gamma Y \mu Z \quad (15.4)$$

The transmission coefficient, $\beta$, masks the complex factors that can modify transmission, including social (behaviour patterns, crowding, household and community sources of infection) and biological causes (virulence of the infectious agent and, most importantly, susceptibility to the disease).

The fundamental requirement for the establishment and maintenance of the disease is that the population of *susceptible* individuals exceeds a threshold density. This density (of course) depends on the birth rate, which in turn is dependent on the total size of the population ($N$). Consequently, the inter-epidemic interval is determined by the time taken to build up an adequately-sized pool of susceptibles by new births.

A model determining the fraction of suceptibles as a function of time, based on equations (15.1) to (15.4), exhibits a damped oscillation (i.e., the cycles of the epidemics dies out rapidly). The oscillation has a period ($T$) that is given by

$$T = 2\pi\ (AD)^{0.5} \quad (15.5)$$

where $A$ = average age of infection once the infection is endemic and $D$ = the sum of the latent and infectious periods.

The important point is that the epidemics are predicted by these equations to die out completely, whereas, as we shall see, epidemics usually persisted and, in many cases, increased in amplitude. We conclude that equations (15.1) to (15.4) are not an adequate description of the dynamics of infectious diseases and that the system is *driven* in some way, so maintaining the epidemics.

We have developed a linearised model in which the epidemics are *driven* by periodic variations in the transmission coefficient (or susceptibility), $\delta\beta$. This modelling and its application to various lethal infectious diseases have been described in detail elsewhere (Duncan *et al.*, 1996a, b; Scott & Duncan, 1998, and in the Appendix). In brief, the system can be characterised by its natural (resonant), undamped frequency, $\omega_r$, (i.e. the frequency of the epidemics) and by its damping factor, $\xi$, which is a dimensionless ratio in the range 0 to 1 and is a measure of the degree of damping within the system, i.e. the attenuation of the amplitude of the oscillation at its resonant frequency. The damping factor is given by

$$\xi = \frac{N\beta}{2(\mu + \nu)} \frac{\mu}{\sqrt{N\beta - (\mu + \nu)}} \quad (15.6)$$

where $\beta$ = transmission coefficient where the major component is susceptibility to the disease, and

$\nu$ = rate of recovery from disease = 1/infectious period.

For the values of $\mu$, $\nu$ and $N\beta$ that are used to describe the epidemics, $\xi$ is small (much less than 1), indicating that the system is lightly damped. Since $\xi$ is small, the frequency response of the system is sharp and a driver that has a frequency *that is the same* as the resonant frequency, $\omega_r$, will be amplified. The period of the resonant frequency of the system, $T$, is given by

$$T = \frac{2\pi}{\sqrt{\mu[N\beta - (\mu + \nu)]}} \tag{15.7}$$

Since $\mu \ll \nu$ this expression is equivalent to equation (15.5).

Thus, the important conclusion from this non-linear modelling of a driven system is that the inter-epidemic interval, $T$, for a particular population is determined by $N\beta$, effectively the product of population size and susceptibility to the disease (equation (15.7)). Although $N$ is most conveniently measured by the absolute numbers in the population, in reality the density of the susceptibles will be an important component of $N$ and would have had a major effect on the dynamics of infectious diseases.

The second important conclusion from the modelling is that a system that is oscillating (i.e. a population that is showing regular, non-decaying epidemics) is most readily pumped up or driven by a regular fluctuation in susceptibility (effectively $\beta$) that is *oscillating at the resonant frequency of the system* (see equations (15.5) and (15.7)).

## 15.3 Interaction of nutrition and infection

Scrimshaw *et al.* (1968) have provided us with an invaluable review of the complex interactions between nutrition and infection using examples drawn from the twentieth century and often from Third World populations. Usually the interaction of malnutrition and infection is detrimental to the host; in some examples there is no effect and in a few examples there is evidence of clinical antagonism (i.e. malnutrition inhibited the usual progression of the infection and attenuated the host response). The interaction where the host is adversely affected is frequently synergistic. Reduced resistance to infection is directly associated with nutrient deficiency. The frequency and severity of infections are increased for individuals whose nutritional status is poor, although this increase depends in part on the social and environmental circumstances frequently associated with malnutrition. During recovery from an infectious disease, the quantity and quality of food available is usually the limiting factor (Scrimshaw, 2000).

Thus, nutritional and infectious diseases are commonly shown to go hand in hand; there is a bidirectional influence in which malnutrition may predispose to infection or increase its severity, while infection itself results in nutritional abnormalities. To tease out these complex relationships, it is necessary to characterise the burden of infection and the cause and nature of the infections present. This specificity is needed because the impact of infection differs with the agent; its particular biology determines the effects on host nutrition and the interaction with the immune system (Keusch, 1998).

We shall not consider the interesting topic of the ways in which infectious diseases influence the nutritional status of affected individuals, but are here primarily concerned with the effects of malnutrition on the demography of populations acting via the agency of infectious diseases. Hence, it is only those infectious diseases (usually occurring in epidemics) that are potentially lethal that we consider in this and the succeeding chapter. The percentage mortality varies with each disease and, as we shall see, with the quality of the nutrition. Deaths in childhood have always been of particular demographic importance. Haemorrhagic plague represented a special case, mortality was probably over 95% and adults and children were infected indiscriminately (Scott & Duncan, 2001). In other diseases, such as smallpox, measles, whooping cough and diphtheria, mortality is lower and usually only children are infected in each epidemic because older people are immune, having been infected in a previous outbreak. These diseases remain a serious threat to malnourished children in the Third World today.

From a demographic point of view, therefore, the most significant effects of malnutrition are first on the resistance to infection (i.e. susceptibility; see section 15.2) and second on disease morbidity and mortality.

## 15.4 The impact of malnutrition on resistance to infection

Lunn (1991) avers that whatever the cause of the deterioration in nutritional status, it has become generally accepted that malnutrition predisposes an individual to infectious diseases. Moreover, when illness does strike, it is likely to be more severe and prolonged, and carries an increased risk of death or permanent damage. However, not all infections are affected in the same way; some respond far more than others. Table 15.1 shows the extent to which various illnesses are reported to be influenced by a reduced nutritional status. In essence, the incidence depends on whether the host's immune response can overcome the rate of replication and spread of the invading organism.

Scrimshaw (2000) concludes that every known nutrient deficiency can affect disease resistance if it is sufficiently severe; the same common mechanisms are affected by many different nutrients. These mechanisms include physical barriers that depend on the integrity of epithelial and other tissues, phagocytosis, cell-

**Table 15.1** Infectious diseases believed to be influenced by nutritional status.

| Disease category | Influence | | |
|---|---|---|---|
| | Definite | Variable | Slight |
| Bacterial | Tuberculosis<br>Bacterial diarrhoea<br>Cholera<br>Leprosy<br>Pertussis<br>Respiratory infections | Diphtheria<br>Staphylococcus<br>Streptococcus | Typhoid<br>Bubonic plague<br>Tetanus<br>Bacterial toxins |
| Viral | Measles<br>Rotavirus diarrhoea<br>Respiratory infections<br>Herpes | Influenza | Smallpox<br>Yellow fever<br>Encephalitis |
| Parasitic | Pneumocystis carinii<br>Intestinal parasites<br>Trypanosomiasis<br>Leishmaniasis<br>Schistosomiasis | Giardia<br>Filariasis | Malaria |
| Fungal | Candida<br>Aspergillus | Mould toxins | |

*Source:* Lunn (1991).

mediated immunity, some forms of non-specific resistance and antibody formation.

It is well established that nutrition is critical to the development of an effective immune system and to the enhancement of the natural immunosurveillance and its effector mechanisms. This enhancement could be mediated either by increasing the frequency and absolute numbers of effector cells, or by the upregulation of the cellular mechanisms by which these effector cells carry out their functions. Even in the Western world, large sectors of society often remain undernourished and show suboptimal immune responses, but the relationship between nutrition and immunity is best seen in developing and underdeveloped countries (Gershwin *et al.*, 2000).

Although an association between malnutrition and susceptibility of the host to infection has long been recognised, it has become clear only recently that tissues associated with the immune system, such as the thymus gland and lymphoid tissues, are more sensitive to nutritional deficits than many other organs of the body. In protein-energy malnutrition, histomorphological abnormalities occur in the thymus, including a reduction in size and weight, depletion of lymphocyte numbers and loss of cortico-medullary corpuscles. Similar degenerative changes are seen in the lymphoid areas of the spleen and lymph nodes; the tonsils are also much reduced in size (Lunn, 1991). The B lymphocytes develop immunoglobulins (or antibodies) on their surface and have been divided into five classes

which vary in their susceptibility to nutritional deficiencies. Some lymphocytes function as memory cells for the production of large numbers of specific antibody-containing cells when the host is exposed to a subsequent challenge or infection of the same kind (Scrimshaw, 2000). There is evidence that large numbers of these memory cells are attracted by the mammary gland when the mother develops an infection and contribute appropriate antibodies to breast milk (Hansen 1992; see section 8.3).

There are a number of different stages in cell-mediated immunity that can be affected by nutrient deficiencies. For example, the proliferation of both T and B cells in response to mitogens is impaired with various deficiencies that may include that of vitamin $B_6$. A reduced phagocytic response with severe protein malnutrition has been documented by Scrimshaw *et al.* (1968) and most of the antimicrobial systems found in phagocytes have been found to be susceptible to deficiency of one or more specific nutrients. The complement-3 system is susceptible to protein-energy deficiency (Scrimshaw, 2000).

Vitamin E is the most effective chain-breaking, lipid-soluble antioxidant in the membranes of all cells, but it is found in especially high concentrations in the membranes of immune cells because their high polyunsaturated fatty acid content puts them at high risk of oxidative damage. Free radical damage to immune cell membrane lipids may ultimately damage the ability of immune cells to respond normally to challenge. Meydani and Beharka (1996) believe that it is indisputable that adequate vitamin E is essential for normal immune function. Vitamin E deficiency impairs several aspects of the immune response, including B and T cell-mediated immunity as well as the function of phagocytic cells.

Similarly, it is well known that zinc plays an important role in the normal development and function of the immune system. Zinc deficiency results in increased susceptibility to infectious diseases (see review, Salgueiro *et al.*, 2000).

Protein-energy malnutrition manifests in both acute (wasting) and chronic (stunting) forms. Wasting protein energy, regardless of degree, exerts a negative impact on the cell-mediated adaptive immunocompetence of children (Rivera *et al.*, 1986); it increases the risk of childhood mortality from infection in a potent multiplicative manner and with no threshold effect (Pelletier *et al.*, 1995). Woodward (1998) believes that a substantial catalogue of clinical and experimental information now documents the influence of dietary protein and energy on the immune defences.

The action of leptin has been described in section 6.3; this hormone plays a crucial role during times of starvation when a person's energy economy becomes progressively more compromised. About 15% of the energy budget is normally devoted to the immune system but, during periods of severe undernourishment, the adipose tissue responds by reducing the amount of leptin in the circulation, so shutting down the activity of helper T cells and the release of complement proteins. Consequently, the immune system is compromised, but recovers quickly once the fat reserves have been built up again.

Groups of mice were fed either a normal diet or a diet deficient in selenium in a recent, interesting study. Both groups were then exposed to a mild strain of human influenza virus (influenza A Bangkok) and those on a selenium-deficient diet contracted a more severe form of influenza than did the controls. However, remarkably, the virus in the selenium-deficient animals had undergone many genetic changes so that it was now in a more virulent form, even for mice that had consumed a normal diet. It is suggested (Nelson *et al.*, 2001) that poor nutritional status may contribute to the emergence of new viral strains and, so, might promote epidemics, thereby having far-reaching and hitherto unforeseen demographic effects.

Finally, we have seen in section 7.9 how malnutrition of the mother during critical windows of her pregnancy can have far-reaching effects on the health of her children in later life, and deficiencies in maternal nutrition can affect the immune function in her offspring (see section 7.13). A study in rural Gambia confirms that the general principle of foetal programming of immune function is likely to be of wide significance, particularly in populations with marginal nutrition and high levels of exposure to disease (Moore *et al.*, 1997). It is interesting in this respect that animals who are born to zinc-deficient mothers have a compromised immune system (McArdle & Ashworth, 1999).

## 15.5 The effects of malnutrition on the mortality from infectious diseases

In children whose nutritional status is poor, episodes of any of the common communicable diseases of childhood tend to be more severe and to have more secondary complications. In Guatemala, 50% of children with whooping cough require more than 12 weeks, and 25% more than 25 weeks, to recover the weight lost because of the disease (Scrimshaw, 2000). Mortality rates among the serious infectious diseases are inevitably higher.

Vitamin A is the micronutrient deficiency that is most commonly associated with infectious, immune dysfunction and increased mortality in children. Improvement of vitamin A status results in the enhancement of growth and haematopoiesis, a decrease in infectious morbidity, an improvement in immune function and a reduction in childhood mortality. It is estimated that today over 100 million children, predominantly in developing countries, are affected.

The link between clinical vitamin A deficiency and infectious disease morbidity and mortality has been known for a long time; experimental observations and clinical trials in the 1920s and 1930s led to the reputation of vitamin A as the 'anti-infective' vitamin. By the 1960s, it was noted that among the micronutrients, 'no nutritional deficiency is more consistently synergistic with infectious disease than that of vitamin A' (Scrimshaw *et al.*, 1968). More recent community- and hospital-based clinical trials show that vitamin A supplementation reduces child mortality

by 20–30%. Vitamin A capsule distribution is recognised as one of the most cost-effective interventions to improve health, and ranks among vaccination and oral rehydration therapy in importance as a public health measure (Semba, 1998).

The widespread immunologic effects of vitamin A are mediated primarily via all-*trans* or 9-*cis* retinoic acid or other metabolites, and nuclear retinoic acid receptors may be utilised to regulate gene transcription or a pathway involving 14-dihydroxy-4, 14-*retro* retinal. Retinoids influence many aspects of immunity, including mucin and keratin expression, haematopoiesis, apoptosis, the growth, differentiation and function of neutrophils, natural killer cells, monocytes/macrophages, Langerhans cells, T and B lymphocytes, the balance between T helper type 1-like and T helper type 2-like immune responses, immunoglobulin production, and expression of cytokines and adhesion molecules (see Table 15.2). The impact of vitamin A deficiency on immunity, and hence on resistance and mortality, is better established in the impairment of mucosal immunity by alterations in keratins and mucins, compromised function of accessory cells such as neutrophils, macrophages and NK cells, alterations in cytokine networks which influence immune responses, and altered antibody responses to T-cell dependent and T-cell independent type 2 antigens (Semba, 1998).

It follows from the foregoing that vitamin A is important in maintaining the integrity of epithelial surfaces and immune function. Experimental studies indicate that vitamin A deficiency results in decreased cellular turnover, strati-

**Table 15.2** Immune response elements that may be regulated by retinoids.

| |
|---|
| Keratinisation |
| Mucin production |
| Haematopoiesis |
| Apoptosis |
| Neutrophil function |
| Natural killer cell function |
| Monocyte/macrophage function |
| Langerhans cell function |
| T lymphocyte function |
| Cytotoxic T lymphocyte function |
| B lymphocyte function |
| Immunoglobulin production |
| ICAM-1 expression |
| TNF-α production |
| TGF-β production |
| Phospholipase $A_2$ production |
| IFN-γ production |
| IL-1 production |
| IL-2 production |
| IL-3 production |
| IL-4 production |

*Source:* Semba (1998).

fication of epithelial cells and, ultimately, squamous metaplasia, keratinisation and desquamation of cells. The net effect of these changes is the loss of the first line defence barriers, predisposing the host to infections. Vitamin A supplementation in experimental animals has been associated with the restoration of the normal epithelium and the enhancement of cell-mediated cytotoxicity, natural killer cell activity, macrophage function and antibody production (Hussey & Eley, 1999). Children in Indonesia with clinically mild vitamin A deficiency have a 4-fold increase in mortality from all causes and a 3-fold increase in the incidence of respiratory and diarrhoeal diseases. Vitamin A supplements reduced preschool age childhood mortality by over 30% (Barclay *et al.*, 1987).

Measles is a viral disease that infects and damages epithelial tissues throughout the body. It induces a transient biochemical vitamin A deficiency and frequently precipitates xerophthalmia in malnourished children. There is recent evidence of a striking reduction in mortality after vitamin A supplementation was given to populations of underprivileged children in Indonesia and India and, in general, to poorly nourished children who acquire measles (Scrimshaw, 2000). Measles is also a particularly virulent disease among African children, accounting for most cases of childhood blindness and for considerable mortality. Recent data suggest that vitamin A deficiency may be prevalent in areas of Africa, including Tanzania (Barclay *et al.*, 1987). The following study carried out in 1982–3 in a rural general hospital in central Tanzania is illustrative of many investigations on the effect of vitamin A on the mortality related to measles. The local population were subsistence farmers living in a fairly arid environment; the staple diet was millet eaten with a green vegetable relish. Malnutrition was a great problem in children in 1982 because of the near famine conditions. About 25% of all children admitted were classified as severely malnourished and only 30% had a weight for age above 80% of the standard. Anaemia was common. One hundred and eighty children admitted with measles were randomly allocated to receive routine treatment alone or with additional large doses of vitamin A (200 000 IU orally immediately and again the next day). Baseline characteristics of the two groups were virtually identical for age, severity of measles and vitamin A and general nutritional states. Of the 88 subjects given vitamin A supplements, six (7%) died; of the 92 controls, 12 (13%) died. This difference in mortality was most obvious for children aged under 2 years (one death out of 46 children receiving supplements versus seven deaths out of 42 controls; $P < 0.05$) and for cases complicated by croup or laryngotracheobronchitis. Mortality was several times higher in marasmic than in better nourished children, regardless of study allocation ($P < 0.01$) (Barclay *et al.*, 1987).

## 15.6 Diarrhoeal diseases

Diarrhoea is one of the major hazards faced by an infant when supplementary feeding, in addition to breast milk, is introduced, not only in pre-industrial

England, but in Third World countries today. As malnutrition develops because of the poor weaning diet, acute diarrhoeal disease becomes increasingly likely to lead to death (Scrimshaw *et al.*, 1968). The primary effect of diarrhoeal diseases is dehydration because of the loss of fluid and electrolytes, which if not treated promptly can lead to death. Many diarrhoea-related deaths in young children are because of persistent rather than acute diarrhoea. The duration of each attack depends on the causative organism, and the longer the duration, the worse is the nutritional impact. Once malnutrition develops, the diarrhoea becomes more severe and a diarrhoea–malnutrition cycle develops (Molla & Molla, 1999).

Many of the patients with diarrhoea in developing countries are malnourished and an estimated 500 000 child deaths annually are attributed to an infection with *Shigella*, which causes a major loss of protein. These children are also likely to be deficient in important micronutrients such as zinc, because diarrhoea leads to the loss of this mineral in the stool. Maternal zinc supplementation (30 mg daily) of pregnant Bangladeshi women from 3 months to gestation significantly reduced the risks for infants at age 6 months to acute diarrhoea and dysentery. These reduced risks were seen in low birthweight infants but not in those with normal birthweight (Osendarp *et al.*, 2001). The relationship of vitamin A, diarrhoea and malnutrition is also a close one, especially in countries where vitamin A deficiency is common (Molla & Molla, 1999).

Scrimshaw *et al.* (1968) describe weanling diarrhoea as a synergism of infection and nutrition and have provided a comprehensive overview of this serious disease; in large parts of the world, deaths from diarrhoeal disease in the general population outnumber those from any other single cause and it continues to rank among the ten leading causes of death in early childhood. In a study in the Punjab, the fate of newborn infants seems to depend largely on whether or not they were breast-fed – either they are nursed or they die (see Table 15.3). Of the 775 infants in this series,

**Table 15.3** Deaths and death rates by feeding regimen in seven Punjab villages, 1955–1959.

| Feeding regimen | Newborn infants | | Neonatal deaths (0–28 days inclusive) | | Post-neonatal deaths (29 days to 11 months inclusive) | | Total infant mortality (0–11 months inclusive) | |
|---|---|---|---|---|---|---|---|---|
| | No | % of total | No | Deaths per 1000 | No | Deaths per 1000 | No | Deaths per 1000 |
| No food given | 16 | 2.1 | 16 | 1000.0 | — | — | 16 | 1000.0 |
| Artificial feeding from birth | 20 | 2.6 | 15 | 750.0 | 4 | 200.0 | 19 | 950.0 |
| Breast-fed from birth | 739 | 95.3 | 34 | 46.0 | 55 | 74.4 | 89 | 120.4 |
| Total | 775 | 100.0 | 65 | 83.9 | 59 | 76.1 | 124 | 160.0 |

*Source:* Scrimshaw *et al.* (1968).

95% were breast-fed and had a mortality rate of 12%, in contrast to a rate of 95% for the artificially fed. Table 15.4 records the fate of the children who were wholly breast-fed for varying periods. At all ages at which numbers were sufficient to permit reasonable comparison, not only was acute diarrhoeal disease less frequent among the wholly breast-fed than among those with subsequent dietary regimens, but it was often less fatal. The case fatality rate was 2.1%.

**Table 15.4** Cases and deaths from acute diarrhoeal disease among wholly breast-fed children, by quarter years, in seven Punjab villages, 1955–1959.

| Age in months | No of children breast-fed throughout period | Received breast milk | | Cases of acute diarrhoeal disease among breast-fed | Deaths from diarrhoeal disease among wholly breast-fed children |
|---|---|---|---|---|---|
| | | No | % | | |
| 0–2 | 736 | 524 | 71.2 | 178 | 4 |
| 3–5 | 685 | 380 | 55.5 | 118 | 0 |
| 6–8 | 669 | 196 | 29.3 | 90 | 3 |
| 9–11 | 638 | 91 | 14.3 | 29 | 0 |
| 12–14 | 421 | 31 | 7.4 | 7 | 0 |
| 15–17 | 378 | 17 | 4.5 | 5 | 2 |
| 18–20 | 296 | 3 | 1.0 | 1 | 0 |
| 21–23 | 252 | 1 | 0.4 | 0 | 0 |
| 24–26 | 100 | 1 | 1.0 | 0 | 0 |
| 27–29 | 77 | 0 | — | — | 0 |

*Source:* Scrimshaw (1968).

At all ages, children given solid foods had more acute diarrhoeal disease than children on a milk diet. The progression was: lowest rates for the wholly breast-fed, intermediate for those given additional milk and highest for children on the complex diet.

The maximum impact of the weaning process on diarrhoeal disease occurred at the end of breast-feeding, and the rates continued high for several months thereafter. An increase in diarrhoeal disease began with the first supplementation of breast milk; it ended with an adjustment to the new diet. The after-effects of that experience continued to influence the general health and the growth and development of the child for a long time.

Scrimshaw *et al.* (1968) also report field observations of acute diarrhoeal disease among rural Mayan Indian populations of the Guatemalan highlands during 1958–64. The outstanding feature of the attack rates is the extent to which diarrhoeal disease was concentrated in children aged 6 months to 2 years, the period of weaning. Acute diarrhoeal disease was relatively infrequent during the first 6 months, when the children were almost wholly breast-fed. After weaning was completed, usually early in the third year, the incidence declined sharply, so that at age 6 years, the attack rate was only 21.2% of children of that age per year.

Scrimshaw *et al.* (1968) point out that

'The true significance of the situation is often difficult to appreciate from a mere comparison of death rates from different causes. The importance of acute infections of the intestinal tract in countries like Guatemala is perhaps better understood by comparing the relative death rates from this cause with those prevailing in more favoured societies. For infants under one year, the death rate attributed to gastro-intestinal infections in the Guatemalan villages was 25 times that for infants in the USA. For the pre-school group, it was 519 times greater; and, for the general population, the excess in Guatemala was 115 times.'

Deaths from acute diarrhoeal diseases were examined over a 10-year period for some 20 village communities in Guatemala. Analysis showed that there was a succession of epidemics of fairly regular periodicity, usually three outbreaks every 10 years, each of relatively long duration, with an excess of deaths through a year or more and, occasionally, over a 2- or even 3-year period. In no epidemic examined did the outbreak develop sharply, last a brief time and end with much the same abruptness as it began, as is characteristic of common-source outbreaks related to water or another vehicle of spread. Rather, the epidemic evolved slowly and continued actively through many consecutive months. The broad behaviour is better characterised as fluctuating epidemicity rather than fluctuating endemicity (see Fig. 15.1).

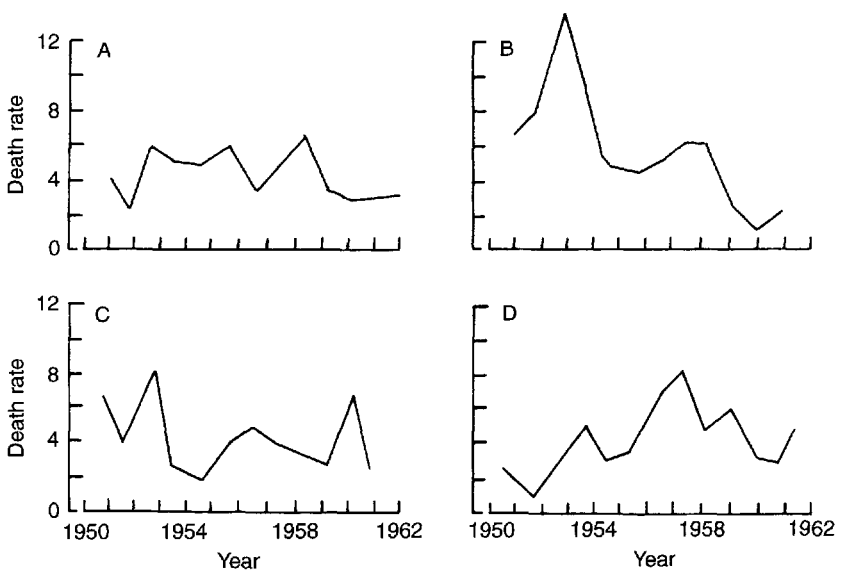


**Fig. 15.1** Annual mortality rates per 1000 from acute diarrhoeal disease in four Guatemalan villages, 1950–62. A: Chimaltenango; B: San Andres Itzapa; C: El Tejar; D: Santa Maria Cauque.

## 15.7 Conclusions: nutrition and infectious diseases

Any form and level of malnutrition may reduce the resistance to serious infectious diseases and increase the probability of dying. The most serious effects on the immune system are mediated via the depletion of the fat reserves, protein-energy malnutrition or vitamin A deficiency. In this way, malnutrition may have subtle, but profound, effects on both susceptibility (and hence on the transmission coefficient, $\beta$) and mortality. The demographic consequences of this are that in addition to raising the mortality from the disease, fluctuations in susceptibility can act as a trigger for epidemics and can markedly modify the endemic level of the disease. It is the children who mostly die in epidemics of infectious diseases and, since these have not reproduced, the demographic consequences are severe. We illustrate how malnutrition can have far-reaching demographic effects in the following case studies.

## 15.8 Smallpox in rural towns

Haemorrhagic plague disappeared completely by 1670 (Scott & Duncan, 2001) but, in England, a more virulent form of smallpox had already appeared (probably as the consequence of a mutation) and it rapidly became the most feared lethal infection with major demographic effects. When an infective arrived in a community the fundamental requirement for the establishment of an epidemic was that the population of *susceptible* individuals exceeded a threshold density.

The requirement that the 'reproductive rate' of the infection ($R$) is greater than 1 for the establishment of an epidemic can be formally expressed as the requirement that the population of susceptibles ($X$) exceeds a threshold density, $X > N_T$, where

$$N_T = (\gamma + \mu)\ (\sigma + \mu)\ \beta\sigma \qquad (15.8)$$

Thus, small and scattered communities never experienced a major outbreak (Scott & Duncan, 1998). Larger parishes recorded sporadic outbreaks after which the survivors were immune and a subsequent epidemic was again dependent on the slow re-establishment of an adequate pool of susceptibles by new births. Consequently, the majority of the cases (and hence deaths) of smallpox were confined to children. The disease became a major source of childhood mortality in the seventeenth and eighteenth centuries in populations of sufficient size and density.

Plague deaths were recorded by law in the parish registers, but only rarely in the seventeenth century did the clerks indicate deaths from smallpox. However, epidemics in the rural market town of Penrith were specifically recorded for the years 1656 and 1661. Spectral analysis of the child burials at Penrith for the period

1600–1812 has a major peak at a frequency of 0.2, showing that this 5-yearly oscillation is the dominant cyclical feature of child mortality (see section 10.2); it is shown after filtering in Fig. 10.1 and the years of recorded smallpox epidemics at Penrith synchronise with major peaks in the filtered series. It is suggested that this oscillation in child mortality after 1630 may represent periodic epidemics of this lethal disease. Since smallpox epidemics exploded suddenly but burnt out quickly, evidence for outbreaks of the disease at Penrith have been studied by time-series analysis using two different procedures:

(1) Child burials were plotted on a monthly basis and linear filtering revealed a clear oscillation with a period of approximately 60 months. The years of recorded smallpox epidemics synchronised with peaks in this filtered series.
(2) The child burial series at Penrith was analysed by determining the largest total of burials for three consecutive months in each year. Spectral analysis shows the major peak at a period of 5 years; the filtered series (1600–1800) is shown in Fig. 15.2 and can be compared with Fig. 10.1. The oscillations become more pronounced and of greater amplitude after 1700.

Again, the known smallpox epidemics at Penrith in 1656 and 1661 coincide with the peaks of these 5-year oscillations in both analyses.

We conclude that this analysis of the child burial series at Penrith reveals a 5-year oscillation of an infectious, lethal disease which is confined largely to

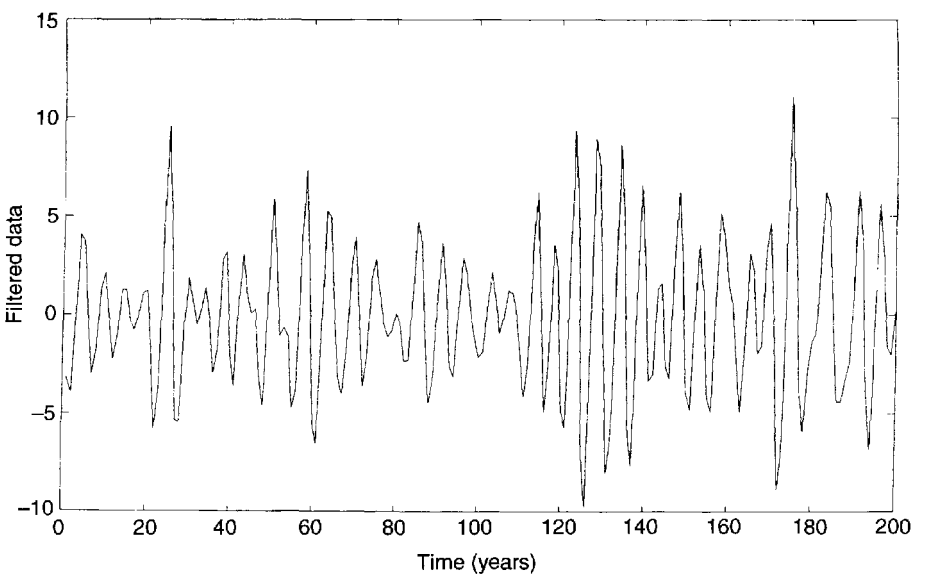


**Fig. 15.2** Child burials at Penrith, 1600–1800, plotted as the highest total for 3 consecutive months in each year. Filtered to show the short wavelength oscillation; filter window = 4 to 10 years.

children and has a strong seasonality in which the epidemic rapidly burnt out; we suggest that it provides indirect evidence of periodic epidemics of smallpox. Comparable studies of the adult burials series at Penrith showed no oscillation with these characteristics. Finally, there is no evidence to indicate that there were outbreaks of the disease in the inter-epidemic years, suggesting that, in this rural market town, with an estimated population of 1500–2000, smallpox was not endemic and its dynamics were completely different from those in large cities described in section 15.9.

The 5-year oscillation in child burials synchronises with the corresponding oscillation in wheat prices (see sections 3.3 and 10.8): the two series are significantly coherent in the 5 to 6 year wavebands. We have suggested that regular cycles in wheat prices drove oscillations in child deaths at Penrith (section 10.8), and that these deaths were associated both directly and indirectly with malnutrition. The 5-year oscillation in wheat prices became clearly established only after 1575 (see Fig. 3.1) and the 5-year cycle in child burials at Penrith developed at this time and high mortality was cross-correlated with high wheat prices.

We conclude that the 5-year oscillation in wheat prices and consequent malnutrition generated the 5-year oscillation in child deaths during the period after 1575, and 100 years later the smallpox epidemics were superimposed on this pre-existing mortality cycle. Epidemics were probably initiated by travellers because Penrith lay on the main road to Scotland and the spread of the disease during epidemic years would be facilitated if outbreaks of smallpox in nearby towns with similar dynamics were locked to the same cycles of hardship and famine. We suggest that regular oscillations of famine and malnutrition produced periodic fluctuations in susceptibility and so drove the epidemics. In summary, regular fluctuations in the degree of hardship and malnutrition generated corresponding driving oscillations in susceptibility to the disease ($\delta\beta$) and thereby greatly enhanced the likelihood of an epidemic explosion when an infective entered the population. Once an epidemic was initiated, most susceptible members of the community would be exposed and equally likely to become infected. Market towns would be particularly vulnerable to smallpox epidemics, being of sufficient size and density, with the regular movement and crowding resulting from incomers from the surrounding farms and villages.

Bengtsson (1999) also reports that in pre-industrial Sweden (1757–1850) smallpox mortality among children was determined by economic fluctuations.

## 15.9 Smallpox in London

Annual smallpox deaths in London, 1647–1893, are shown in Fig. 15.3 and the pattern is completely different from that at Penrith. The disease was endemic throughout the 250-year period, although the level fluctuated. Regular epidemics were superimposed on the basic endemic level and these are revealed by filtering

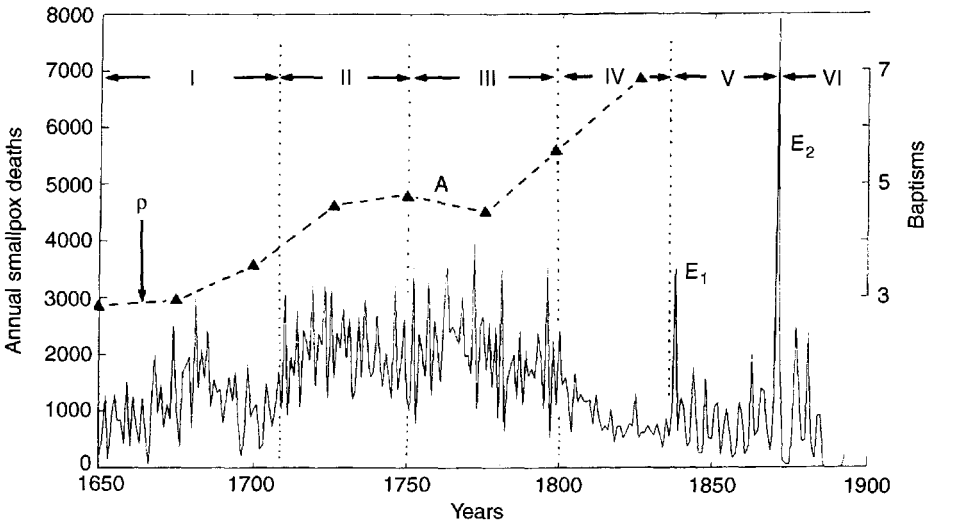


**Fig. 15.3** Annual smallpox deaths (left hand ordinate) in London over some 250 years, 1647–1893, divided into cohorts: I = 1647–1707; II = 1708–50; III = 1751–1800; IV = 1801–35; V = 1836–70; VI = 1871–93. The plague in 1665 (p) and the major smallpox epidemics of 1838 ($E_1$) and 1871 ($E_2$) are indicated. The dashed line (solid triangles; A) gives the cumulative number of baptisms in the preceding 25 years (thousands), right hand ordinate. Data sources: Bills of Mortality (Creighton, 1894) and Wrigley and Schofield (1981).

(see Fig. 15.4). Figure 15.3 can be divided into separate periods, each having a characteristic inter-epidemic interval ($T$):

(I) 1647–1707, $T$ changing from 4 years to predominantly 3 years.
(II) 1708–50; endemic level rising, $T$ changing from 3 to 2 years.
(III) 1751–1800; $T$ firmly established at 2 years.
(IV) 1801–35; vaccination becoming effective; endemic level falling steadily; epidemics greatly reduced in amplitude; $T$ = 2–3 years.
(V) 1836–70; epidemics re-initiated following a major outbreak in 1838; endemic level continuing to fall; $T$ = 4 years.
(VI) 1871–93; a major epidemic in 1871 (the biggest during the 250-year period; $E_2$ on Fig. 15.3) triggered three further epidemics before smallpox ceased to be a serious disease.

Can the epidemiology of smallpox in London be modelled and so explain this changing pattern in the biology of the disease and its interaction with the demography of the metropolis?

Equations (15.1) to (15.4) describe an SEIR model in which if a disease is introduced into the population it responds with epidemics at regular intervals ($T$), but these would decay and the annual number of those infected would settle at a steady, endemic level. Clearly, this was not the case with smallpox in London,

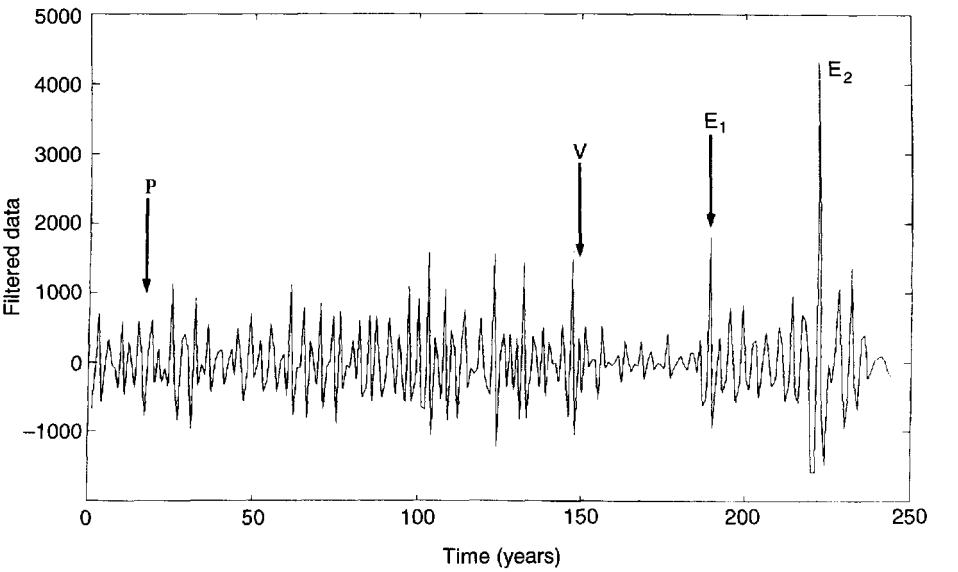


**Fig. 15.4** Filtered smallpox deaths in London, 1647–1893; filter window = 2 to 5.3 years. There is a clear change in the pattern of epidemics after 1800 when vaccination (V) became effective. The major epidemics of 1838 ($E_1$) and 1871 ($E_2$) triggered decaying oscillations indicative of SEIR dynamics. P = plague of 1665.

where major epidemics persisted for 150 years (periods I to III, Fig. 15.3) and extensive studies have sought to explain how such a system could be driven (or 'pumped up', Anderson & May, 1991) by stochastic effects or by seasonal forcing so that the epidemics were maintained. We present a linearised model of the epidemics in the Appendix which shows that, theoretically, the system could be driven and the epidemics maintained by a periodic variation in susceptibility ($\beta$) to the disease. Strictly, $\beta$ is defined as the transmission coefficient. We use time-series analysis to examine whether cyclical external factors could act to generate an oscillation in susceptibility ($\delta\beta$) and so maintain the smallpox epidemics in London shown in Figs 15.3 and 15.4

It is evident (see above) that the inter-epidemic interval of smallpox in London did not remain constant over the 250 years, but varied from 2 to 4 years in the different cohorts. Mathematical modelling shows that the system would oscillate readily (i.e. regular epidemics would be generated) at a 'preferred or natural frequency' in the same way that a tuning fork oscillates at its natural frequency when struck. Epidemics occur at this natural frequency which is inversely related to the product $N\beta$, where $N$ is most conveniently measured by the size of the population, although density probably has an additional effect; a greater density will exacerbate the spread of the disease (equation 15.8). Thus the modelling predicts that changes either in the population size or in overall susceptibility to the disease will be reflected in changes in the periodicity of smallpox epidemics.

An increase in $N$ (i.e. the size and/or density of the population) will also amplify the driving effect of an oscillation in susceptibility, i.e. the more crowded the population, the greater the effect of a standard oscillation in $\delta\beta$. The cumulative total of births in London for each preceding 25-year period is plotted in Fig. 15.3 and this provides a relative measure of population size ($N$) in terms of susceptibles. During 1647–1750 (cohorts I and II), $N$ rose steadily and, concomitantly, the inter-epidemic interval, $T$, fell from varying between 4 and 3 years (1647–1707) to varying between 3 and 2 years.

However, after 1750, when $T$ clearly changed to 2 years (suggesting a rise in $N\beta$), the number of baptisms was stationary and did not begin to rise again until the end of the eighteenth century, suggesting that the population was not expanding markedly. We have seen in Chapter 3 that the annual wheat prices provide a good measure of the fluctuating levels of nutrition in England in earlier centuries. Wheat prices show a falling trend from 1650 to 1750, but with a rising trend after 1750 to 1815. The latter was a period of high food prices and hardship in England, particularly among the poorer classes (Walter & Schofield, 1989) and we suggest that *malnutrition* caused an overall increase in susceptibility ($\beta$) to smallpox and, consequently, a rise in $N\beta$ and a fall in $T$ to 2 years after 1750. In summary, the inter-epidemic interval is reduced in 1647–1750, mainly because of rising $N$, and in 1750–1800, mainly because of rising $\beta$.

It is suggested that, during the period 1647–1800, the epidemics were driven by an oscillation in seasonal dry conditions (Scott & Duncan, 1998). The introduction of variolation and, more particularly, vaccination in 1796 had a dramatic effect on the dynamics of smallpox: there was a marked reduction in the size of the pool of susceptibles in London as progressively more young children were vaccinated, which was accompanied by a fall in the endemic level, a marked reduction in the amplitude of the epidemics and an extension of the inter-epidemic interval.

## 15.10 Measles epidemics in London, 1630–1837

Annual deaths from measles in London from 1630 to 1837 are plotted in Fig. 15.5. The disease became endemic after 1700 and the trend rose steadily until 1785 but, thereafter, the endemic level rose sharply (suggesting an increased susceptibility) until 1812, when it clearly fell again. The annual wheat price series for this period is also plotted in Fig. 15.5 and it can be seen that its trend after 1700 corresponds closely to that of the measles deaths series, with the same marked rise after 1785 and a fall after 1812. The trends in the two series cross-correlate ($P < 0.001$). This correspondence suggests that the rising trend in measles mortality may have been associated with an increased susceptibility which resulted from poorer nutritional standards. About 50% of food expenditure in England during the first half of the eighteenth century went on grain in the form of bread flour and ale, but this

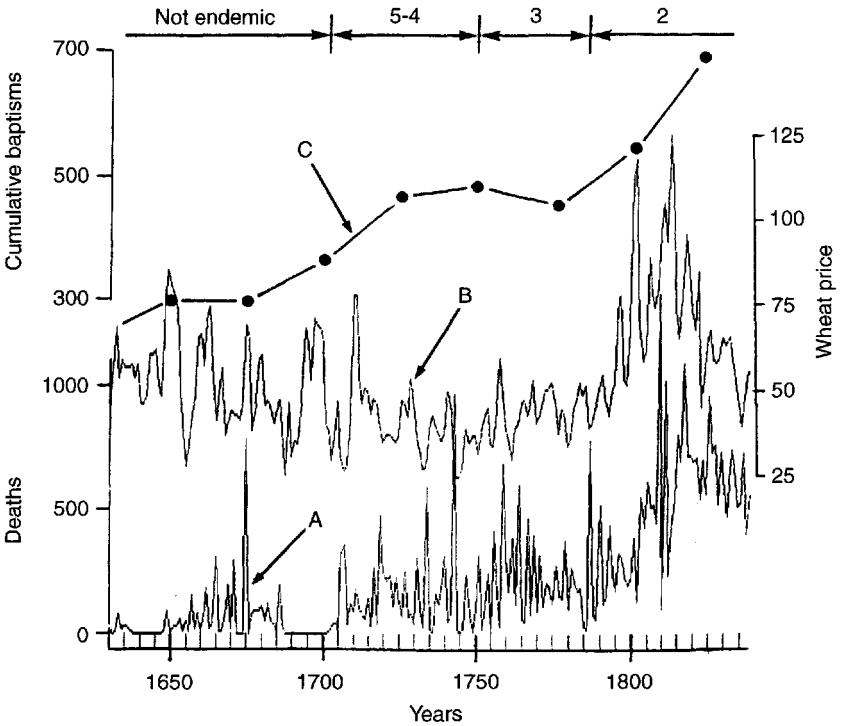


**Fig. 15.5** Measles in London, 1630–1837. Line A: annual measles deaths (lower left hand ordinate). Line B: annual wheat prices (shillings; right hand ordinate). Line C: cumulative number of baptisms in the preceding 25 years (thousands; upper left hand ordinate). Above: division into periods with different interepidemic years. Data sources: Creighton (1894), Stratton (1970) and Wrigley and Schofield (1981).

proportion rose after 1785 during the period of high food prices and population pressure, particularly among the poorer classes (see section 15.9) and we conclude that the marked rise in the trend in measles mortality (and probably in susceptibility also) after 1785 was probably directly linked to the malnutrition of the poorer classes which stemmed from the rising grain prices (Duncan *et al.*, 1997).

Spectral analysis of the measles deaths series in the different periods shows that the wavelength of the oscillations (i.e. the inter-epidemic interval, $T$) changed progressively through the eighteenth century from 5 to 4 years, when the disease first became endemic, to 3 years and then to 2 years after 1785 (see Table 15.5 and Fig. 15.5). Thus, the 2-yearly measles epidemics that were characteristic of developed countries in the early twentieth century were already firmly established in London by the end of the eighteenth century.

We have shown theoretically (see Appendix) how the periodicity of measles epidemics is dependent on the relative value of $N\beta$, i.e. the product of population size/density ($N$) and susceptibility to the disease ($\beta$). The predicted relative

**Table 15.5** Measles epidemics in London: inter-epidemic period, predicted mean age of contracting the disease and predicted $N\beta$ in different cohorts. Coherence between measles epidemics and annual wheat prices or mean annual autumn temperatures are shown for the different short wavebands.

| Cohort | $T$ (years) | Predicted age (years) | Predicted $N\beta$ | High wheat prices | | | Low autumn temperatures | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Wavebands (years) | Coherence | Lag (years) | Wavebands (years) | Coherence | Lag (years) |
| 1700–1720 | 5→4** | 10–15 | 64 | 4–6*** | 0.98 | 2 | 4–6***<br>3*** | 0.98<br>0.78 | 0 |
| 1720–1750 | 4→3* | 10 | 85 | 5*** | 0.75 | 2 | 5**<br>2–3*** | 0.51<br>0.98 | 0 |
| 1750–1785 | 3** | 5 | 133 | 5*** | 0.74 | 2 | 3*** | 0.75 | 0 |
| 1785–1837 | 3–2* | 2 | 270 | 5*** | 0.65 | 2→3 | 5***<br>3***<br>2*** | 0.75<br>0.72<br>0.91 | 0 |

*Note:* $T$ is the interepidemic period (determined by spectral analysis). Predicted mean age of contracting the disease is derived from equation 15.5, assuming $D = 15$ days. Predicted $N\beta$ derived from equation 15.7.
* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

values of $N\beta$ in the four cohorts are shown in Table 15.5 and the dynamics of the disease can be interpreted on this basis as follows. The cumulative total births in London for each preceding 25-year period is plotted in Fig. 15.5, line C, and this provides a relative measure of population size ($N$) in terms of susceptibles. Measles was not truly endemic during the period 1630–1700; $N$ and $\beta$ were both low. The epidemics were irregular, but spectral analysis and filtering suggest that they were basically 3-yearly, and cross-correlation tests suggest that they were probably directly triggered by fluctuating wheat prices. After 1700, as the population size ($N$) increased (indicated by the rising trend in Fig. 15.5, line C), measles became established as an endemic disease, initially with 5-yearly epidemics during cohort I (1700–20) but changing to a pattern of stable 4-year epidemic intervals during cohort II (1725–50), when relative $N\beta$ is calculated to be 85 (Table 15.5).

In cohort III (1750–85), $T$ shortened to 3 years, associated with a rising population size in London (Fig. 15.5, line C). Relative $N\beta$ for the 3-yearly epidemics in this cohort is calculated as 133 (Table 15.5); i.e. compared with the preceding period the calculated value for $N\beta$ rose 1.5 times, while $N$ rose only 1.1 times. We conclude that the rising population size at this time was not sufficient to account for the change in $T$ and that the small rising trend in wheat prices at this time (Fig. 15.5, line B) produced an increased susceptibility to the disease which contributed to the rise in $N\beta$ and the establishment of the 3-year period of the epidemics.

After 1785, 2-yearly measles epidemics became firmly established in London (Table 15.5), superimposed on the sharply-rising endemic death rate from the disease (Fig. 15.5, line A). The relative value of $N\beta$ for 2-yearly epidemics is calculated as 270, representing a doubling from the calculated $N\beta$ during the 3-yearly epidemics of the preceding period. Although the population was rising sharply after 1785 (Fig. 15.5, line C), the increase was only some 1.2-fold during the transition from 3- to 2-yearly epidemics. However, this was the period during which there was a marked rise in the trend of wheat prices (Fig. 15.5, line B) with associated malnutrition and we conclude that this caused an increase in general susceptibility ($\beta$), which now made a major contribution to the rise in $N\beta$. Thus, a progressive change in $N\beta$ in London during the eighteenth century would account for the observed change in the inter-epidemic period.

The cross-correlation between the measles and wheat prices series has been tested by coherence, cross-correlation function and by multiple regression in the different periods, and the results are summarised in Table 15.5 in which the years 1700–1837 are divided into four cohorts with different inter-epidemic periods ($T$). The two series are strongly and significantly correlated in the short wavebands ($P < 0.001$), but it is noteworthy that there was a 2-year lag in the coherence which changed to a 3-year lag after 1800, i.e. an oscillation in high wheat prices was followed by a measles epidemic with high mortality 2 or 3 years later. Sharp, oscillatory rises in wheat prices in England during the seventeenth and

eighteenth centuries led to a rise in neonatal mortality and subsequent impaired resistance to disease in very young children (Scott *et al.*, 1995; see also Chapter 10). It is suggested that the measles epidemics were also *indirectly* geared to oscillations in wheat prices: malnutrition in pregnancy produced a greater susceptibility to measles in the young progeny and hence a rise in the number of children dying from the disease in the next epidemic.

The possible cross-correlation between the epidemics of measles mortality and mean annual seasonal temperatures was similarly tested, but only low mean autumn temperatures were consistently and significantly ($P < 0.001$) correlated with the epidemics of the disease (Table 15.5).

Consequently, it is suggested that oscillations of malnutrition during pregnancy combined synergistically with lowered autumn temperatures to provide an oscillation in susceptibility ($\delta\beta$) of low amplitude, which was sufficient to act as an input to drive the system at its resonant frequency of epidemics which was determined by $N\beta$. Thus, the progressive rise in $N\beta$ determined the evolution of the measles epidemics in London during the eighteenth century from initially at 5-year intervals to finally at 2-year intervals, which persisted into the twentieth century until an extensive vaccination programme was promoted.

## 15.11 Whooping cough epidemics in London, 1701–1812

Annual deaths from whooping cough in London 1701–1812 are plotted in Fig. 15.6A and it can be seen that the disease was not endemic until 1720; thereafter the endemic trend rose progressively, with regular epidemics superimposed, and more steeply after 1785. The English annual wheat price series over the same time scale is plotted in Fig. 15.6B and the trend in prices corresponds with the trend of the whooping cough series; the two series cross-correlate ($P < 0.001$). Although correlation does not prove causality, it is suggested that the rise in whooping cough mortality (and probably in susceptibility also) after 1785 (Fig. 15.6A) was directly linked to malnutrition (as with other infectious diseases in the eighteenth century), which stemmed from the rising grain prices at that time.

Spectral analysis of the whooping cough deaths series shows that the eighteenth century can be divided into different periods wherein the period of the oscillations (i.e. the inter-epidemic interval, $T$) changed progressively. Between 1720 and 1750, as the epidemics emerged and became established, this interval fluctuated around 3 and 5 years but, during 1750–85, the inter-epidemic interval changed clearly to 3 years and the amplitude of the epidemics increased (see Fig. 15.6A). After 1785, associated with a rising endemic trend, the whooping cough epidemics again became less regular, but now with a basic periodicity of about 5 years.

The relative predicted values of $N\beta$ (the product of population size/density and susceptibility) for whooping cough in London are: 123 (for $T = 3$ years); 75 (for $T = 4$ years) and 53 (for $T = 5$ years) and the changing periodicity of the epidemics

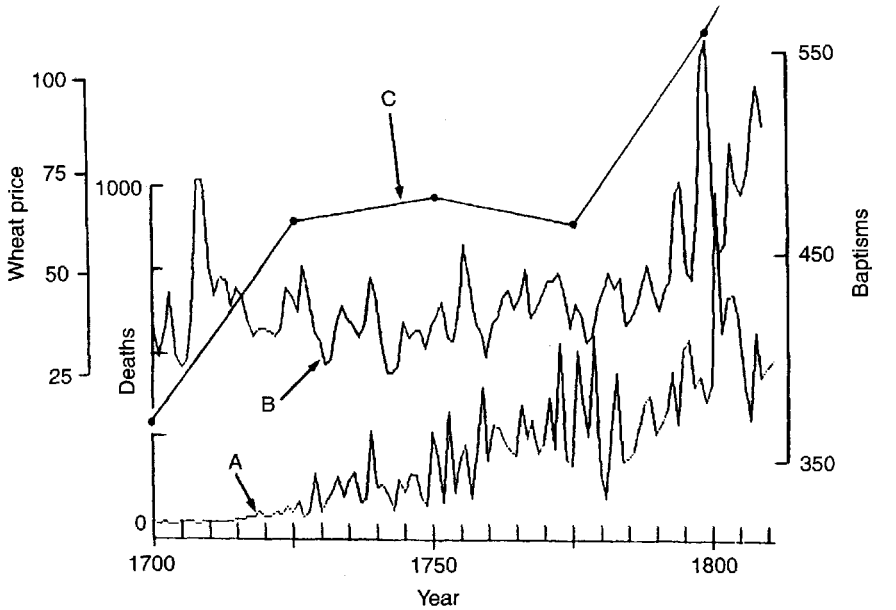


**Fig. 15.6** Whooping cough epidemics in London, 1701–1812. A: Annual whooping cough deaths from Bills of Mortality (lower left hand ordinate). B: Annual wheat price (shillings; upper left hand ordinate). C: Cumulative number of baptisms in London for the preceding 25 years (thousands; right hand ordinate). Data sources: Bills of Mortality (Creighton, 1984) and Wrigley and Schofield (1981).

in London (see Table 15.6) can be interpreted on this basis as follows. The cumulative total baptisms in London for each preceding 25-year period (see Fig. 15.6C) provides a relative measure of population size ($N$) in terms of susceptibles. During the early years of the eighteenth century, 1700–20, whooping cough was not endemic in London and $N$ and $\beta$ were low, but after 1720, following the population increase, whooping cough became established as an endemic disease, with variable 5-yearly epidemics (1720–50).

In cohort III, during 1750–85, $T$ shortened to 3 years and theoretical considerations suggest that $N\beta$ would have risen correspondingly from relative values of about 53 to 123. However, $N$ was not rising markedly at this time and it is suggested that the rising trend in wheat prices caused an increase in susceptibility ($\beta$) and was a component of the increased $N\beta$, which caused a corresponding change in the resonant frequency of the system and in the inter-epidemic interval from a variable 5 to 3 years (see Table 15.6). This suggested increased sensitivity to whooping cough associated with rising wheat prices after 1785 corresponds with the findings for measles mortality at that time (see section 15.10) and, again, there is evidence for malnutrition having profound but subliminal effects on the population dynamics that are not readily detectable.

Figure 15.6A shows clearly that the whooping cough epidemics did not decay

**Table 15.6** Characteristics of the whooping cough epidemics in London in the different cohorts.

| Cohort | Observed $T$ (years) | Predicted mean age (years) | Predicted $N\beta$ | Estimated relative size of pool of susceptibles (thousands) ($N$) | Seasonal driver | Correlation with driver at 0 lag | | | Whooping cough mortality vs wheat prices | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Coherence waveband (years) | $P$ | ccf | Coherence waveband (years) | $P$ | ccf | Lag (years) |
| I 1700–1720 | Not endemic | | | 405 | | | | | | | | |
| II 1720–1750 | 5(3) | 6–9 | 53 | 468 | (i) Low autumn temp | 5 | <0.001 | −0.22 | 5 | <0.001 | 0.38 | 0 |
| | | | | | (ii) Low winter temp | 5 | <0.001 | −0.33 | | | | |
| III 1750–1785 | 3 | 3 | 123 | 466 | (i) High autumn temp | 3 | <0.001 | 0.38 | 3 | <0.001 | 0.2 | 0 |
| | | | | | (ii) Low winter temp | 3 | <0.001 | −0.39 | | | | |
| IV 1785–1812 | 5 | 8–9 | 53 | 550 | | | NS | | 4–5 | <0.001 | 0.41 | 1 |

NS, not significant. $T$ was determined by spectral analysis. Predicted mean age of contracting whooping cough is determined from equation 15.5, assuming D = 27 days (Anderson and May, 1991). Predicted relative $N\beta$ for each value of $T$ is derived from equation 15.7. Relative values of $N$ derived from cumulative number of baptisms in London for the preceding 25 years (from Wrigley and Schofield, 1981).

during the eighteenth century in London; rather, they increased markedly in amplitude and the inter-epidemic interval was reduced and so it may be concluded that the system must be driven.

The whooping cough epidemics and the short wavelength oscillation in annual wheat prices are strongly coherent in the 5-year waveband ($P < 0.001$) at zero lag in the period 1720–50, and we conclude that the strong short wavelength oscillation in wheat prices caused a corresponding oscillation in susceptibility and so directly drove the whooping cough epidemics.

The correlation between the whooping cough epidemics and seasonal temperatures was also tested and strong coherence ($P < 0.001$) was found with both mean low winter and mean low autumn temperatures at zero lag in the 5-year and other short wavebands in cohort II, 1720–50.

We conclude that oscillations in both seasonal temperatures (in autumn and winter) and in wheat prices acted synergistically to produce the oscillation in susceptibility that drove the system, maintaining the epidemics at the resonant frequency of the system which was determined by $N\beta$ (see Table 15.6). Thus, during the period 1750–85 the whooping cough epidemics in London became established in the pattern that was shown later in the twentieth century in England (Anderson & May, 1991).

In cohort IV after 1785 (when $T$ lengthened again to $\approx$ 5 years and the amplitude of the epidemics was reduced), seasonal temperatures were found to have no significant driving effects, whereas the whooping cough series was again strongly coherent in the 4 to 5 year waveband with wheat prices ($P < 0.001$), but now at a lag of 1 year, i.e. high wheat prices were significantly associated with a peak of whooping cough epidemics in the following year. The rising trends in wheat prices (Fig. 15.6B) and in the endemic level of whooping cough (Fig. 15.6A) suggest that malnutrition was causing a marked increase in susceptibility to the disease and was now the dominant factor in generating a 5-year oscillation in $\beta$ and so driving the epidemics, but not at the resonant frequency of the system (see Scott & Duncan, 1998).

Once again, therefore, we have an example where malnutrition has a profound, but hidden, effect on population cycles. With its dependence on population and crowding, its sensitivity to inadequate nutrition and variations in susceptibility, whooping cough in the eighteenth century in London would have had many features in common with the dynamics of the disease in underdeveloped countries today, where severe malnutrition may be seasonal as a result of crop cycles (Keusch, 1991).

## 15.12 Direct effects of malnutrition on child mortality

Childhood mortality in pre-industrial England was probably less sensitive to cycles of malnutrition than was infant mortality, which could be affected on a

seasonal basis or by the short wavelength oscillation in grain prices, both directly and indirectly, via the nutrition of the mother during pregnancy (see Chapters 9 and 10). Infectious diseases, as we have seen, probably had the greatest demographic influence on mortality in childhood.

Nevertheless, there is no doubt that nutritional status is an important predictor of child survival in Third World countries today and that it continues to exert an overt demographic impact.

Schroeder and Brown (1994) have reviewed ten previously published prospective studies and have selected five from India, Bangladesh, Papua New Guinea and the United Republic of Tanzania. The results were pooled and estimates were made of the relative risks of mortality among young children months after they had been identified as having mild-to-moderate or severe malnutrition. These risk estimates, along with global malnutrition prevalence data, were then used to calculate the total number of young childhood deaths 'attributable' to malnutrition in developing countries. Young children (6–60 months of age) had 2.2 times the risk of dying during the follow-up period than their better nourished counterparts. Severely malnourished young children had 6.8 times the risk of dying during the follow-up period than better nourished children. Each year approximately 2.3 million deaths of young children in developing countries (41% of the total for this age group) are associated with malnutrition. These authors suggest that child survival programmes should assign greater priority to the control of childhood nutrition.

Panter-Brick (1997) reports on the prevalence of growth retardation, the impact of seasonality on height and weight gains, and significant relationships between growth velocity, nutritional status and morbidity, for a population living at subsistence level in rural Nepal. Monthly variation in growth pattern was examined for 71 boys and girls of 0 to 49 months of age. At the height of the monsoon season, 71% of children were moderately stunted, but none was wasted. Stunting deteriorated from moderate to severe after 1 year of age. No differences by sex or ethnicity were detected. Environmental changes from the winter to the monsoon seasons were reflected in significant losses of weight and lower weight-for-height scores, especially for 0 to 35-month-olds, although height for 12 to 35-month-olds continued to be gained over this period. Growth velocity was significantly related to previous growth status (thinner and shorter children did not show catch-up in height or weight) and to morbidity reported over the period of observation. The prevalence of illnesses rose 6-fold from the winter to the monsoon, and children with a high frequency of illnesses experienced a significant shortfall in weight and height increments. A poor diet and recurrent illnesses explain the slow and uneven growth of these children. Despite an increase in women's agricultural workloads in the monsoon season, childcare patterns *per se* did not seem to affect small children adversely. Small stature continuing through later childhood and in adults is one consequence of the growth pattern seen at these young ages.

Mortality among children aged over one year in pre-industrial Sweden, 1757–1859, was directly dependent upon economic fluctuations. The impact was stronger among the lower classes and was particularly strong in years following an extremely poor harvest (Bengtsson, 1999).

To conclude: malnutrition acting either directly or indirectly via the exacerbation of the consequences of infectious diseases need have only small effects on childhood mortality to produce major, long-term demographic changes. If the mean number of children produced per family that survives to reproduce successfully falls below two, the population will progressively decline.

Chapter 16

# Population Dynamics, Disease and Malnutrition in the Nineteenth Century in England

The birth and death rates in England and Wales are known with greater certainty after 1837, and the continued growth of the population in the nineteenth century was, in part, because of the excess of births over deaths and in part because of a continuing decline in mortality (McKeown & Record, 1962). By the mid-nineteenth century, over half of the population of Britain could be classified as urban and there is clear evidence of a rising standard of living during the second half of the century, with rising real wages, a falling cost of living (Woods & Woodward, 1984) and a fall in food prices (Woodward, 1984). Imports of meat trebled between 1870 and 1890 and the imports of butter and margarine more than doubled between 1880 and 1890 (Cronje, 1984). However, there is no positive evidence to show that there was any increase in the heights or weights of school children; working-class diets were still composed principally of carbohydrates and any movement towards proteins was limited to small increases in the consumption of dairy products rather than meat (Oddy, 1982).

## 16.1 Smallpox in England and Wales, 1847–93

The annual smallpox deaths for the second half of the nineteenth century are shown in Fig. 16.1A and the pattern is comparable with London at this time (see Fig. 15.3): the picture is dominated by the enormous epidemic of 1871 (which was associated with the Franco-Prussian War) when 23 000 died in England and Wales. This was superimposed on an endemic level which fell steadily from some 5000 deaths per annum in 1845 to disappear by the end of the century as vaccination became progressively widespread. The amplitude of the epidemics also fell.

Spectral analysis of this series shows a strong peak at 6.7 years which corresponds with a 6.7-year oscillation in national wheat prices at this period. However, the cross-correlation function, wheat prices versus smallpox deaths (Fig. 16.1B), shows that the two series are completely out of phase and that wheat

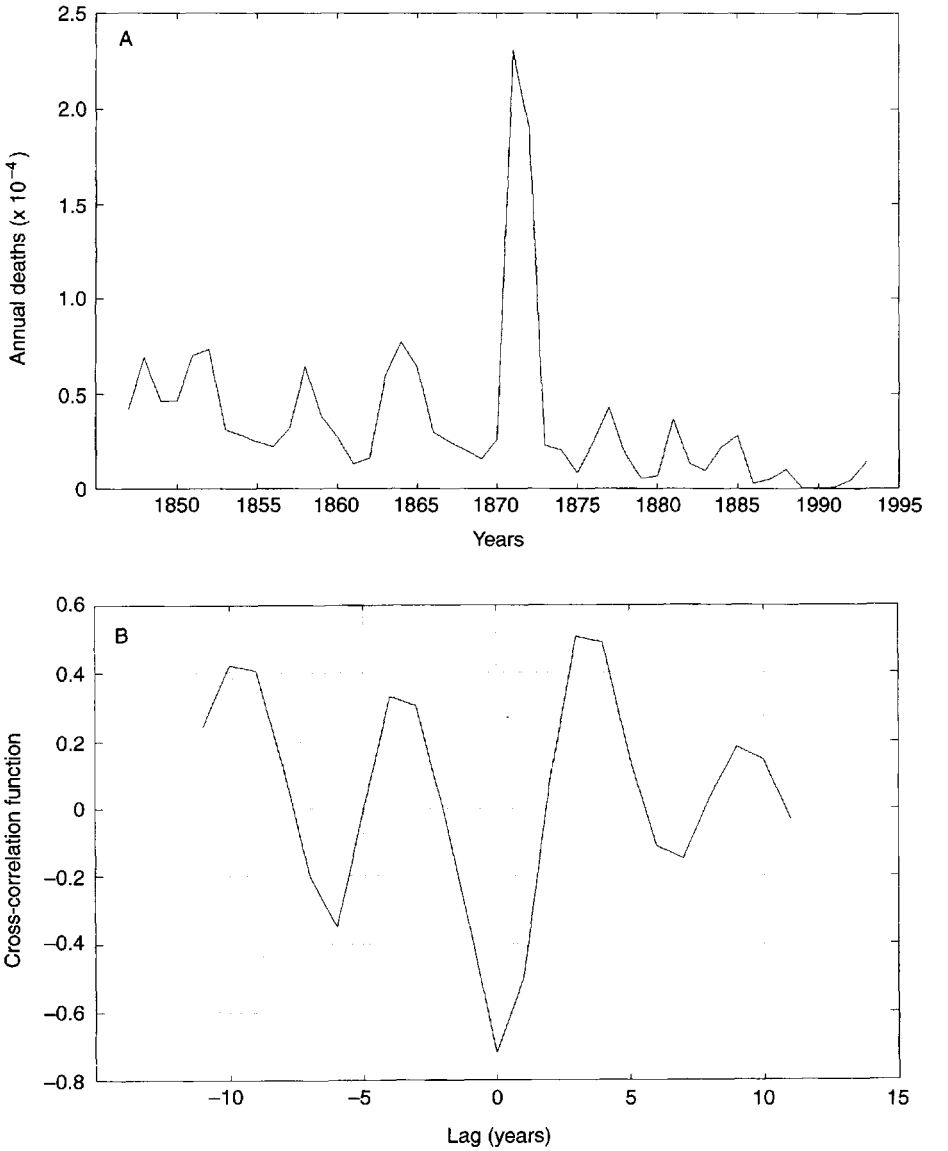


**Fig. 16.1** A: Annual deaths from smallpox in England and Wales, 1847–93 (ordinate). B: Cross-correlation function (ordinate), wheat prices versus smallpox deaths in England and Wales, unfiltered data, 1847–69. Minimum ccf (−0.7) at zero lag. Abscissa: lag (years).

prices do not directly drive the epidemics. The epidemics did not correlate with any of the seasonal weather conditions. We conclude that high wheat prices may produce susceptible children (who die in the next smallpox epidemic), via the effects of malnutrition during pregnancy or during the first year of life, and so contribute to maintaining the epidemics.

## 16.2 Scarlet fever in England and Wales, 1847–93

Scarlet fever is a bacterial disease caused by a haemolytic streptococcus (*Streptococcus pyogenes*) which also causes streptococcal tonsillitis and erysipelas in man. It has been regarded as becoming relatively benign over the last 150 years (Quinn, 1989), but the disease is not mild everywhere and no one can forecast what its epidemic future may be (Christie, 1980). A recent, unexplained increase in severe streptococcal diseases in the USA and UK has been compared to the 1825–85 pandemic of fatal scarlet fever (Katz & Morens, 1992).

There were an enormous number of deaths from scarlet fever in England and Wales during 40 years in the middle of the nineteenth century, whereas this period was preceded and followed by decades with very much lower levels of annual mortality from the disease. This major outbreak of scarlet fever with a high mortality during 1825–85 is regarded as remarkable by historical epidemiologists (Creighton, 1894).

Annual scarlet fever deaths in England and Wales are shown in Fig. 16.2A; there are clear epidemics from 1847 to 1880, but thereafter the endemic level of deaths falls rapidly and the fatal epidemics virtually disappear. Spectral analysis of the series for the period 1847–80 shows a peak at wavelength 5.3–5.6 years, showing that the interepidemic interval ($T$) was 5 to 6 years. $N\beta$ for this value of $T$ is 60.5 (see section 15.2). This system is lightly damped and so it will oscillate, and large amplitude epidemics will be maintained in response to an oscillation in susceptibility ($\delta\beta$) of very small amplitude, provided that its frequency is at the resonant frequency of the system (see Appendix).

The annual wheat price series for 1847–93, is shown in Fig. 16.2B; it closely mirrors the scarlet fever deaths series, with clear oscillations up to 1880 and a sharp fall in the index thereafter; spectral analysis of the wheat price series for this period shows a dominant peak at 6.7 years. The cross-correlation between scarlet fever deaths and the wheat price series during the period of the epidemics, 1847–80, was studied by the following techniques: (i) Cross-correlation function; there is a strong correlation between the two series (ccf = +0.87), but with a clear lag of 2 to 3 years. (ii) The coherence programme shows that wheat prices and scarlet fever deaths are significantly correlated in the 5 to 6 year waveband ($P < 0.001$), again with a lag of 3 years.

It is clear that after 1880 both the scarlet fever mortality (Fig. 16.2A) and the wheat price series (Fig. 16.2B) fall sharply and progressively. Thus, the trends in the two data series correlate closely ($P < 0.001$) during the period when scarlet fever eventually ceased to be a lethal disease. Dietary standards in the UK improved in the second half of the nineteenth century, with real wages rising particularly after 1870. Imported food supplemented and improved the diets of many sections of the population (Cronje, 1984). The findings in the present study suggest that the greatly improved general nutrition associated with low wheat

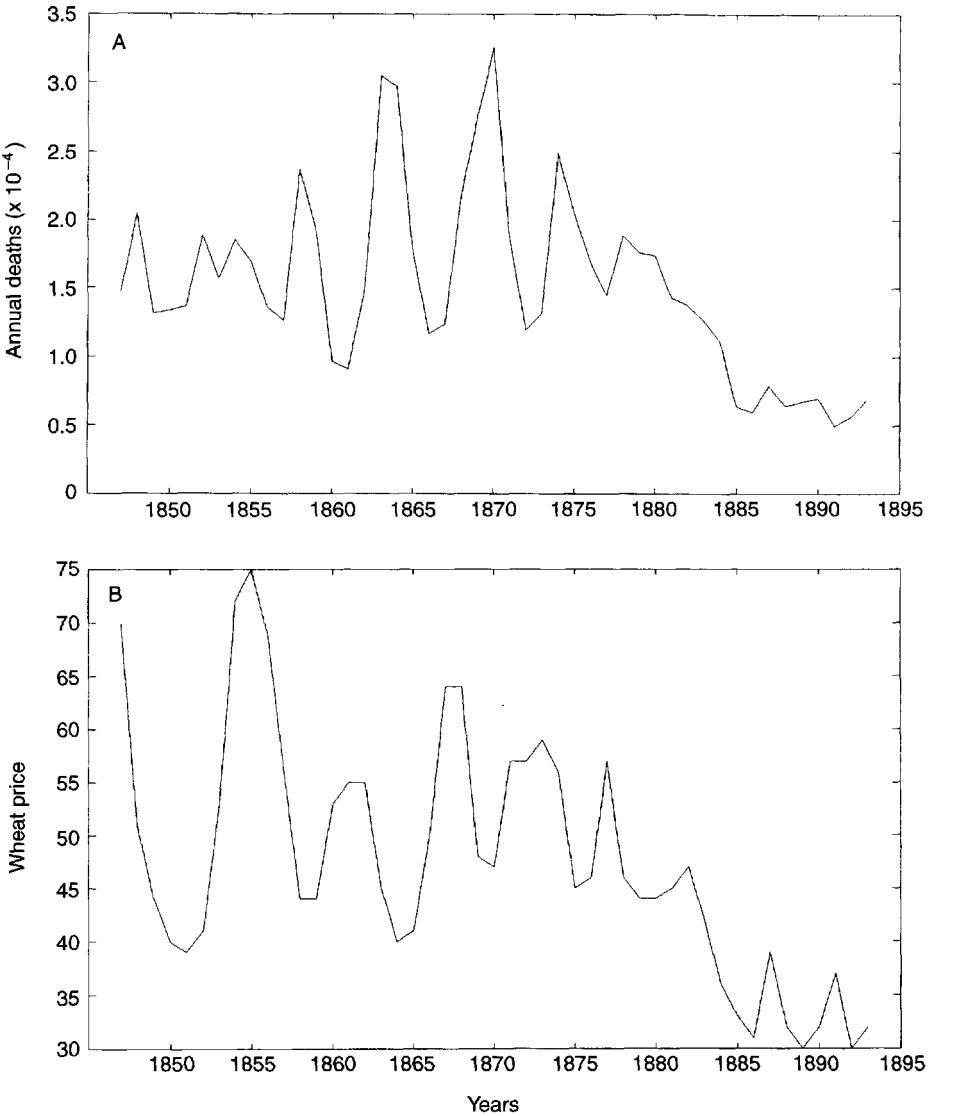


**Fig. 16.2** A: Annual scarlet fever deaths in England and Wales, 1847–93 (ordinate). B: National wheat prices (shillings; ordinate). Marked falling trend in both series after 1875. Data sources: Creighton (1894) and Stratton (1970).

prices in the last quarter of the nineteenth century contributed to the steady fall in the lethality of the disease.

Records for scarlet fever deaths are incomplete before 1847, but the scattered records show that mortality doubled in 1839 from the preceding year and was particularly severe in 1840, when there were 20 000 deaths from the disease. The endemic level of annual deaths then continued at the high rate shown in Fig.

16.2A until 1880. Inspection of the annual wheat price series shows that the index rose in 1838 and the mean level also remained high until 1880 (see Fig. 16.2B), suggesting that the remarkable 40 years of high scarlet fever mortality (Creighton, 1894) was initiated and was dependent on the poor nutritive levels at that time.

In addition to the effect on the overall endemic level and the final falling trend, the scarlet fever epidemics during 1847–80 were also significantly correlated with the oscillation in wheat prices, although with a lag of 2 to 3 years between the two series. This lagged effect suggests that high wheat prices and inadequate nutrition of the mother during pregnancy caused greater susceptibility to the disease in the subsequent children, who then contracted scarlet fever and died in the next epidemic. Although correlation does not prove causality, it is suggested that an oscillation of malnutrition, in this way, produced an oscillation of susceptibility to scarlet fever in young children which interacted synergistically (as we have shown, Duncan *et al.*, 1996b; Scott & Duncan, 1998) with periods of low rainfall in spring and/or summer to produce an oscillation in $\delta\beta$ which drove the system and maintained the epidemics at the resonant frequency of the system (see section 15.2). Waddy (1952) has shown that the infectivity of certain respiratory infections is increased in air of low absolute humidity in both Ghana and England and suggests that such air dries the nasopharyngeal mucosa more rapidly and may thereby lower its resistance.

## 16.3 Diphtheria in England and Wales, 1855–93

Diphtheria is a bacterial infection of the throat, nose or larynx and, although the causative organism, *Corynebacterium diphtheriae*, remains localised, it produces a powerful exotoxin which becomes widely distributed and may cause serious or fatal effects on other parts of the body. One reason for the observed variation in clinical severity is the state of the patient's immunity at the time of infection. The bacterium withstands drying and may be isolated from the dust round the beds in diphtheria wards and remains virulent for 5 weeks. It may also be isolated from the floor dust of classrooms, even after all known carriers have been removed, although spread is normally direct by droplets from an infective. The number of cases of diphtheria per year in England and Wales between 1915 and 1942 was about 50 000 and the number of deaths was around 4000 per year at the beginning and 2500 per year at the end of this period. During this period in the twentieth century, the disease ranked first as a cause of death in children aged 4 to 10 years and second in the 3 to 4 year age group. Mass immunisation then began to show its effect and by 1950 the number of cases had fallen to 962, with 49 deaths.

The annual deaths from diphtheria in England and Wales during the period 1855–93 are shown in Fig. 16.3. There was an impressive rise in mortality during

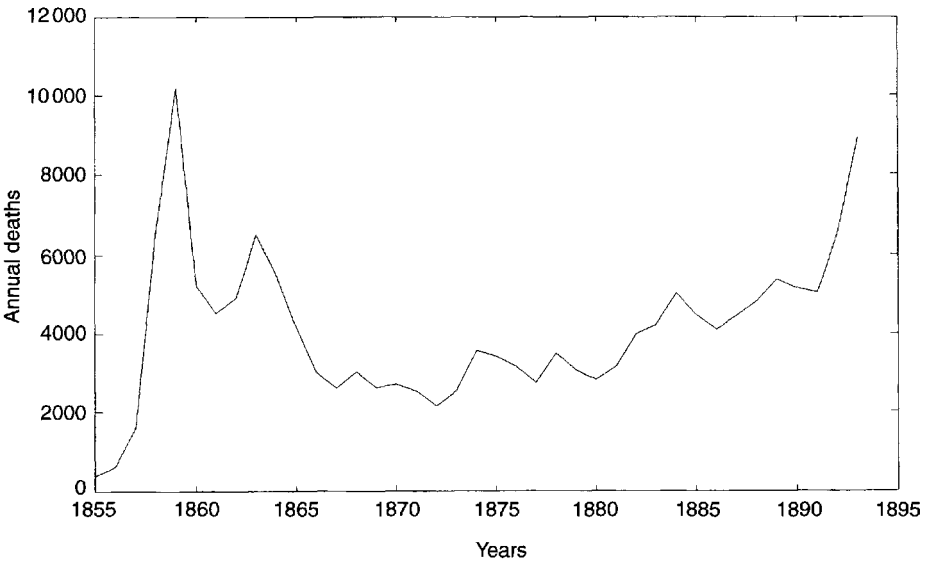


**Fig. 16.3** Annual deaths from diphtheria in England and Wales, 1855–93. Data source: Creighton (1894).

the pandemic of 1858–9 when it first appeared worldwide; it was only in some parts of France, Norway and Denmark that diphtheria had been epidemic in the generation before. Creighton (1894) stated that diphtheria came as a novelty to the medical profession in England in 1858 (see Fig. 16.3).

This epidemic was followed by a second, smaller outbreak in 1863, but thereafter the endemic level fell to some 2500 deaths per year, with very low amplitude epidemics superimposed and spectral analysis reveals that their period was 5 years. The trend in the endemic level of deaths then rose progressively after 1880 (Fig. 16.3).

A possible driver for these low-level, 5-yearly epidemics of diphtheria has been sought in the seasonal weather conditions and this analysis suggests that the epidemics were associated with dry seasons (i.e. low rainfall), although the correlation is not good. It is noteworthy that the first epidemic of 1858 coincided with a severe drought. The effect of warm winters is more significant and spectral analysis of the winter temperature series reveals a strong 5-year oscillation which cross-correlates with the diphtheria epidemics.

The 5-yearly diphtheria epidemics also cross-correlate strongly with high wheat prices, but with a 3-year lag. This finding corresponds with the effects of wheat prices on the epidemiology of scarlet fever (section 16.2) and it is concluded that malnutrition is having a similar effect in the two diseases by acting in pregnancy to produce susceptible children who die in the next epidemic.

## 16.4 Scarlet fever in Liverpool, 1848–80

Liverpool is a sea port on the River Mersey in northwest England which suffered from major public health problems and heavy mortality during the second half of the nineteenth century as the population increased rapidly in size. Lethal, infectious diseases were rife as we show in this and the following sections. Woods and Woodward (1984) stated that a baby born in Liverpool in 1861 had a life expectancy of 26 years, a value that can be compared with 57 years at Okehampton in rural Devon. Many of the large cities had life expectancies of less than 35 years at that time, but in none were the conditions as acute as those in Liverpool, where W.H. Duncan was appointed as the country's first Medical Officer of Health in 1848. He showed that typically 25% of his patients were living in cellar dwellings, with between 15 and 30 people in an airless room (Bickerton, 1936; Morris & Ashton, 1997). The problems had been exacerbated by the stream of Irish migrants in the 1840s fleeing from the potato famine and in 1847 some 300 000 had landed in Liverpool. Of these, 8000 died and many moved inland or migrated to the USA, but the numbers staying in Liverpool are estimated as between 80 000 and 160 000, who located themselves in every available niche (Bickerton, 1936; Morris & Ashton, 1997). The effects of these miserable living conditions were exacerbated by poverty and inadequate nutrition.

Annual scarlet fever deaths in Liverpool, 1848–1900, were taken from the Reports of the Liverpool Medical Officer of Health and are shown plotted in Fig. 16.4. Clear, regular epidemics are evident during the period 1848 to 1880, with a sharply falling trend in the endemic level thereafter and with the amplitude of the epidemics markedly depressed. Spectral analysis of the data series for 1848–80 shows the wavelength of the oscillations to be 3.7 years ($P < 0.05$).

The corresponding series for the annual wheat prices is shown in Fig. 16.2B. Again, oscillations are evident during the period 1848 to 1880, with a markedly falling trend thereafter. The data series for scarlet fever at Liverpool (Fig. 16.4) can be divided into two phases, each, it is suggested, with different underlying dynamics:

(1) During 1848–80, the system is oscillatory with clear epidemics superimposed on a basic 'endemic level'. Spectral analysis shows the wavelength of the epidemics to be 3.7 years (i.e. approximately 4-yearly) and the results are consistent with the modelling in which the epidemics are driven by an oscillation in the transmission coefficient, $\delta\beta$. The scarlet fever epidemics at Liverpool were also significantly correlated with dry conditions (cf England and Wales, section 16.2; see also Waddy, 1952; Duncan *et al.*, 2000), suggesting that these periodic seasonal effects were sufficient to produce a low amplitude oscillation in susceptibility which drove the system and maintained the epidemics.

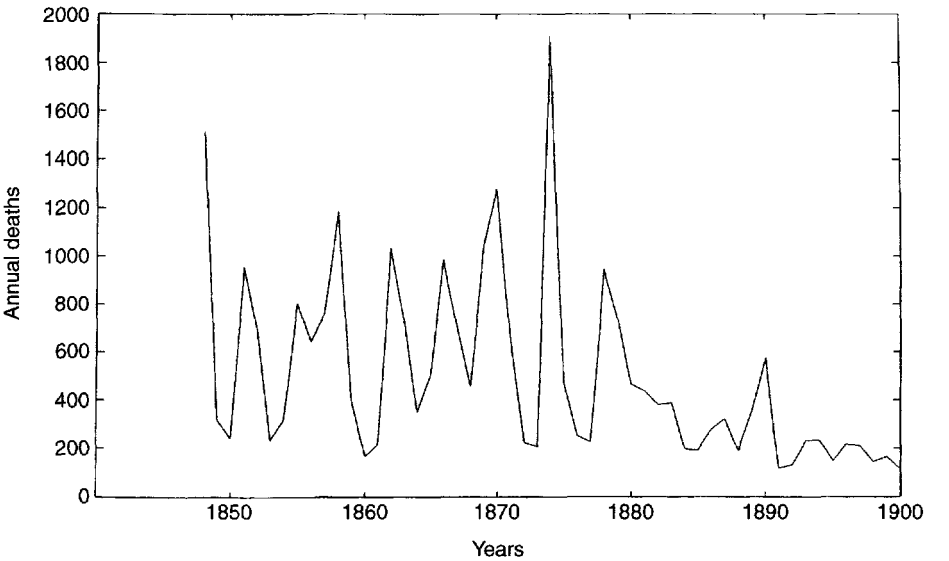


**Fig. 16.4** Annual scarlet fever deaths in Liverpool, 1848–1900. Note the clear epidemics during 1848–80, followed by a falling endemic level and decaying oscillations characteristic of SEIR dynamics, 1880–1900. Data from the Annual Reports of the Medical Officer of Health, Liverpool.

(2) After 1880, the number of scarlet fever fatalities fall sharply and progressively and the epidemics decay, i.e. the dynamics were consistent with an undriven SEIR system (see section 15.2).

It is clear that after 1880 both the scarlet fever fatalities (Fig. 16.4) and the wheat price series (Fig. 16.2B) fall sharply and progressively. The two data series correlate closely ($P < 0.001$) during this period and scarlet fever eventually ceased to be a lethal disease. Dietary standards in the UK improved in the second half of the nineteenth century (section 16.2) and it is suggested that the greatly improved general nutrition associated with low wheat prices in the last quarter of the nineteenth century contributed to the steady fall in the susceptibility and lethality of scarlet fever in Liverpool after 1880 and was the cause of the fundamental change in the dynamics of the disease, which became an undriven system with decaying oscillations during this period.

The scarlet fever epidemics during the earlier period, 1848–80, were also significantly and positively correlated ($P < 0.001$) with the oscillation in wheat prices. However, it is noteworthy that there was a lag of 2 years between the two series, showing that cycles of malnutrition were not directly driving the epidemics. Again, we suggest that high wheat prices and inadequate nutrition of the mother during pregnancy caused greater susceptibility to the disease in the subsequent children, who contracted it in the next epidemic and died. The results

in Liverpool correspond closely with the behaviour of scarlet fever for the whole country (see section 16.2).

## 16.5 Measles in Liverpool, 1863–1900

Measles was a lethal disease causing heavy mortality among children in the compromised community at Liverpool. Annual measles deaths in Liverpool, 1863–1900, are plotted in Fig. 16.5 and two periods are again evident: (i) 1863–85, major epidemics of irregular amplitude superimposed on an endemic level, and (ii) 1885–1900, a falling trend with regular epidemics superimposed, but these are of decreasing amplitude, and measles ceased to be a serious lethal disease by 1900. Spectral analysis shows that the wavelength of the oscillation is 2.4 years throughout the 37 years, but they are most significant ($P < 0.05$) during the first period. It will be suggested below that these results are consistent with a driven system during 1863–85 but, thereafter, there was a marked reduction in the lethality of the disease, so that the system was not driven after 1885 and changed to SEIR dynamics with decaying epidemics (see section 15.2).

Inspection of the results shows that the deaths in the measles epidemics peaked in the autumn quarter, and coherence analysis of seasonal temperatures and rainfall shows that the oscillations during 1863–85 (shown in Fig. 16.5) were significantly ($P < 0.001$) correlated with high autumn temperatures in the 2.1- to

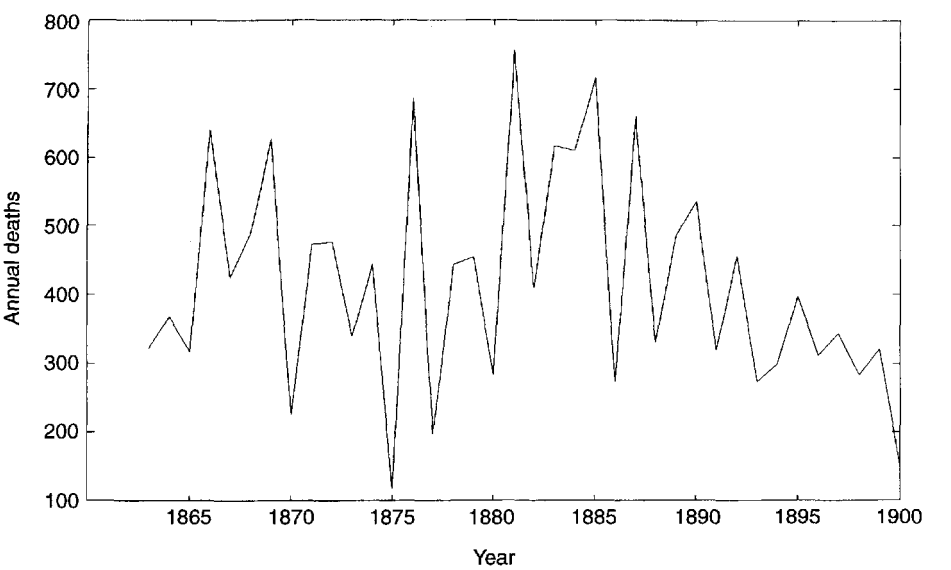


**Fig. 16.5** Annual measles deaths in Liverpool, 1863–1900. Note large, irregular outbreaks superimposed on an endemic level before 1885 and a falling trend with decaying outbreaks thereafter.

2.5-year waveband at zero lag. The correlation continued in the second period, 1885–1900, but now with low autumn temperatures. Measles deaths were also significantly coherent with wheat prices ($P < 0.001$) in the 5.3 year waveband at zero lag. It is suggested, therefore, that the results for the first period, 1863–85, are consistent with a system that is driven, i.e. the epidemics are maintained primarily by an oscillation in autumnal temperatures (the frequency of which is at the natural frequency of the system), and secondarily by a sharp oscillation in malnutrition (reflected in raised wheat prices) which has a wavelength (5.3 years) that is twice that of the epidemics.

The national wheat price series shows a steady, progressive fall in its trend after 1885 to about one-quarter by 1900 (Fig. 16.2B) and this was accompanied, as we have seen, by a marked improvement in nutritive levels, general health and living standards. This progressive change in the nutrition of the population at Liverpool was paralleled by the marked decline in the lethality of measles at this time (Fig. 16.5) and we suggest that this was because of an accompanying reduction in susceptibility (transmission parameter, $\beta$) and in the proportion of infectives that died from the disease. Not only was there a steady fall in the endemic level of deaths during 1885–1900, but the epidemics decayed progressively, suggesting that following the fall in $\beta$, the system was less forcefully driven and changed towards standard undriven SEIR dynamics with *decaying* epidemics (see section 15.2 and Appendix).

## 16.6 Whooping cough in Liverpool, 1863–1900

We have previously shown (Duncan *et al.*, 1996a and section 15.11) that the epidemiology of whooping cough in London during the eighteenth century can be described by the mathematics of linearised systems: the inter-epidemic interval is determined by the population size/density and by susceptibility to the disease. Susceptibility was governed by fluctuating levels of malnutrition which were directly associated with the oscillations in the wheat prices. In this section, we use time-series analysis to elucidate the dynamics of whooping cough in Liverpool in the second half of the nineteenth century and to determine whether annual or seasonal external factors acted to drive the epidemics.

Annual whooping cough deaths in Liverpool, 1863–1900, are shown plotted in Fig. 16.6 and major epidemics superimposed on a basal, endemic level are evident. There is a progressively falling trend through this period, during which the amplitude of the epidemics decays. Spectral analysis suggests that, again, the series may be subdivided as follows: (i) 1863–85, oscillations of wavelength 2.9 years ($P = 0.05$) and 5 years ($P < 0.05$); (ii) 1885–1900, an oscillation of wavelength 3.4 years (not significant). We conclude that the inter-epidemic interval over the period 1863–1900 was approximately 3 years and the epidemics are shown after filtering in Fig. 16.7.

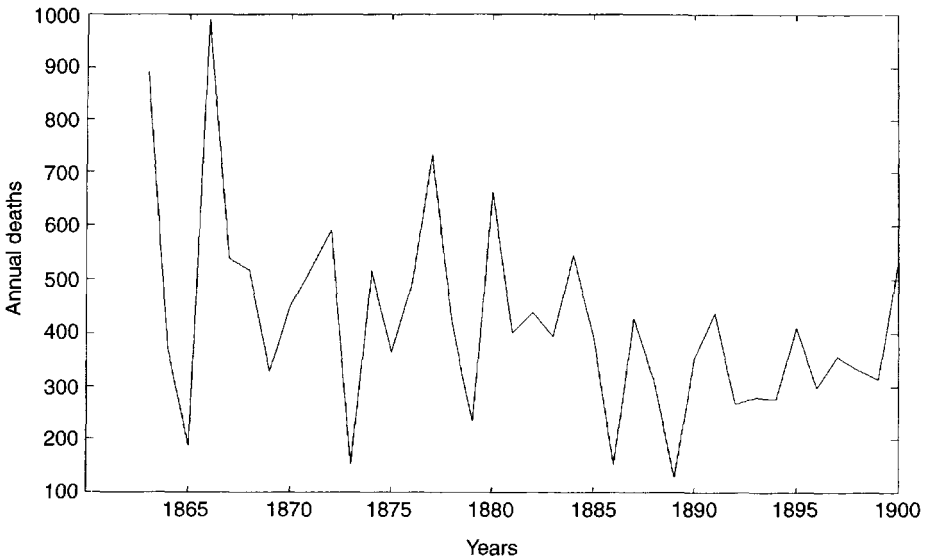


**Fig. 16.6** Annual whooping cough deaths in Liverpool, 1863–1900.

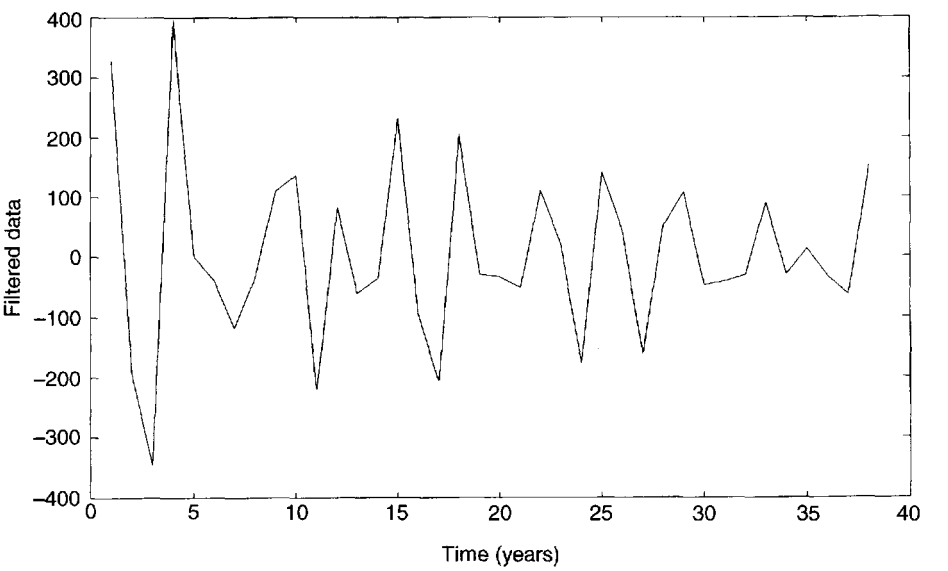


**Fig. 16.7** Whooping cough deaths series in Liverpool, 1863–1900, filtered to reveal short wavelength oscillation. Filter window = 2 to 6 years. Abscissa: time (years) after 1863.

There is a 5.3-year oscillation detectable in the series of national wheat prices and also a clear, falling trend after 1883 (Fig. 16.2B). This 5.3-year oscillation correlated with the corresponding 5-year oscillation in whooping cough deaths, as shown by the cross-correlation function at zero lag (Fig. 16.8; years 1863–93).

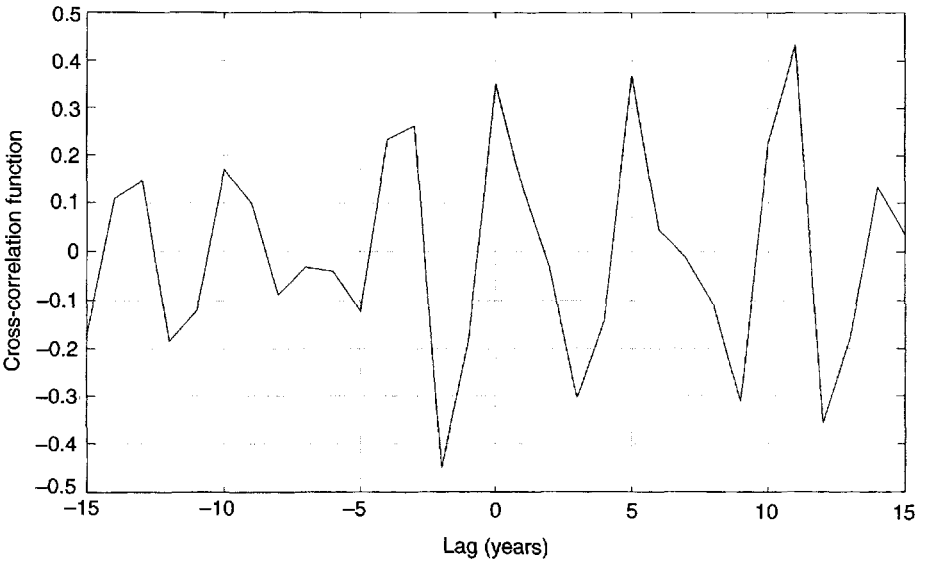


**Fig. 16.8** Cross-correlation function, wheat prices versus whooping cough deaths in Liverpool, 1863–93. Filter window = 2 to 6 years. Ccf = +0.35 at zero lag.

Whooping cough deaths were also strongly and significantly coherent with wheat prices at zero lag, $P < 0.001$.

The epidemics also correlated with seasonal conditions: during 1863–85 with mild winters and with low autumn temperatures, whereas, after 1885, the mortality peaks correlated strongly with cold dry weather in spring.

It is suggested, therefore, that the results are consistent with a system that is driven, i.e. the epidemics do not decay because they are primarily maintained in the compromised population at Liverpool by an oscillation in malnutrition (reflected in raised wheat prices) that has a wavelength that is approximately twice that of the epidemics, and secondarily by an oscillation in seasonal climatic conditions, the frequency of which is at the resonant frequency of the system.

In summary, the densely-crowded conditions (causing a high effective $N$) and inadequate nutrition (causing raised susceptibility) in Liverpool, particularly among the poorer classes, meant that the dynamics of whooping cough were dominated by a relatively high value for $N\beta$, which resulted in a characteristic resonant frequency and a mean interepidemic interval, $T$, of about 3 years. Consequently, it is probable that the epidemics were also directly driven at their resonant frequency by cycles in seasonal conditions that oscillated at this frequency.

However, the national wheat price series shows a steady fall in its trend after 1880 to about one-quarter by 1900 and this, as we have seen, was accompanied by a marked improvement in nutritive levels, general health and living standards. Thus, the progressive change in the nutrition of the population at Liverpool was

paralleled by the decline in the lethality of whooping cough and we suggest that this was because of an accompanying reduction in susceptibility ($\beta$) and, more particularly, in the proportion of infectives that died from the disease.

In conclusion, nutrition and living conditions in the UK in the nineteenth century (particularly well exemplified in Liverpool) improved progressively after about 1880–85, causing a marked change in the epidemiology of lethal infectious diseases which had been responsible for so many childhood deaths. National wheat prices had been at a high level before 1885 but they now began to fall steadily, bringing a progressive improvement in nutritive standards and a concomitant fall in the endemic levels of infectious diseases. This major reduction in childhood mortality in the UK by 1900, particularly apparent in the big conurbations, had important demographic repercussions by augmenting the population boom.

During the period before 1885, regular epidemics were superimposed on the high endemic level of the diseases. Their frequency was determined by the particular epidemiological characteristics of the disease and by the demographic factors that governed the population dynamics (see Appendix). The epidemics were driven (i.e. maintained) by a variety of environmental factors, including, in some cases, the oscillating wheat prices. But these cycles of malnutrition associated with high grain prices also had other, indirect effects on the mortality of young children from infectious diseases. Time-series analysis suggests that if a woman was exposed to a brief but severe period of malnutrition during pregnancy, she bore children whose health was compromised and who were more likely to die in the next epidemic.

Chapter 17

# Ageing

There has been a progressive improvement in human life expectancy from the Stone Age to the twenty-first century, particularly in the developed world (Fig. 17.1) and this has had profound effects on the demography of historical populations. Human longevity may be affected favourably, either by suppressing the causes of premature death or by postponing the process of ageing that causes our liability to disease and death to increase logarithmically with the passage of time. Of these, the elimination of premature death is much the most important factor governing the demography of populations before the twentieth century and this has been the main topic of many of the preceding chapters.

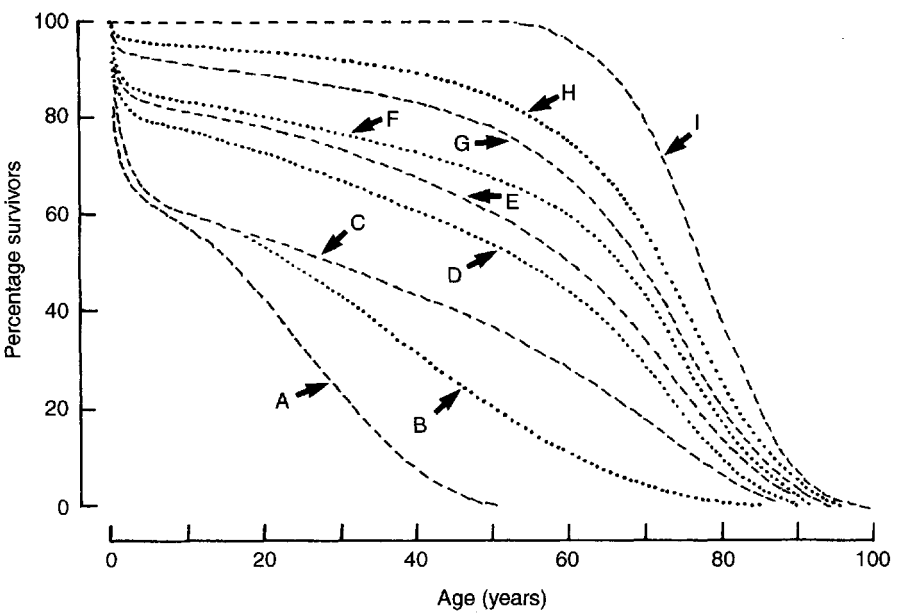


**Fig. 17.1** Historical changes in the human survival curve. A: Stone Age man; B: British India, 1921–30; C: Mexico, 1930; D: Japan, 1926–30; E: USA (whites), 1900–02; F: Italy, 1930–32; G: England and Wales, 1930–32; H: USA (whites), 1939–41; I: theoretical limit of unmodified human survival. After Comfort (1979).

## 17.1 Human survival curves

The human survival curve in the Stone Age resembled that of birds and mammals in the wild: after severe infant mortality, survival decayed almost exponentially with a constant annual probability of dying or being killed (Fig. 17.1). By the Middle Ages, and in the Third World today, the human survival curves had changed markedly; the high infant and child mortality persisted but, once individuals had passed puberty, mortality was sharply reduced and the survival curve 'levelled off' before falling sharply again in old age. In this way the human survival curve changed progressively from exponential decay to a sigmoidal function.

This progressive change in survival is illustrated at Penrith, Cumbria in Fig. 17.2, where the male and female curves in different cohorts are compared with standard demographic tables of populations with a low life expectancy. Figure 17.3 shows that male and female survival was broadly comparable and that the curves did not improve markedly between the first (1557–99) and last (1700–49) cohorts because they continued to be dominated by infant and, to a lesser extent, by child mortality (see Chapter 11). We have shown in Chapter 7 how the nutrition of the mother before and during pregnancy and during lactation had profound effects on the health and survival of infants; the progressive improvement of infant mortality in pre-industrial populations was largely because of better nutrition and, hence, this was the major factor in extending life expectancy, $e_0$, so contributing to the changes in human demography. Figure 17.1 shows how, by the twentieth century, selected human populations in the developed world had reduced infant and child mortality and the causes of premature death to very low levels, so that they are now approaching the normal theoretical limit of survival. This change from the sigmoidal shape of pre-industrial England to the survival curves of developed countries in the twentieth century is called rectangularisation or squaring.

The change from the exponential decay curve of the Stone Age (Fig. 17.1) to the sigmoidal curve of the sixteenth century in rural England (Fig. 17.3) meant that humans were reaching their biblical life span of three score years and ten if they escaped the vicissitudes of earlier life. This natural mean life expectancy has improved little since then, and by the end of the twentieth century was probably around 74 years, but the important demographic feature, illustrated in Fig. 17.1, is that a much greater proportion of the population reached old age (Fig. 17.4). Thus, it is a major achievement of civilisation that life expectancy has increased throughout history, but it was in the twentieth century that this increase, which was previously quite slow, underwent a dramatic acceleration. Average life expectancy in the USA increased from about 50 years in 1900 to about 76 years in 1990, with women living about 5 to 7 more years than men (Timiras, 1994). Not only are the elderly living longer, but they also represent the most rapidly growing segment of the population in developed countries, and so contribute to a

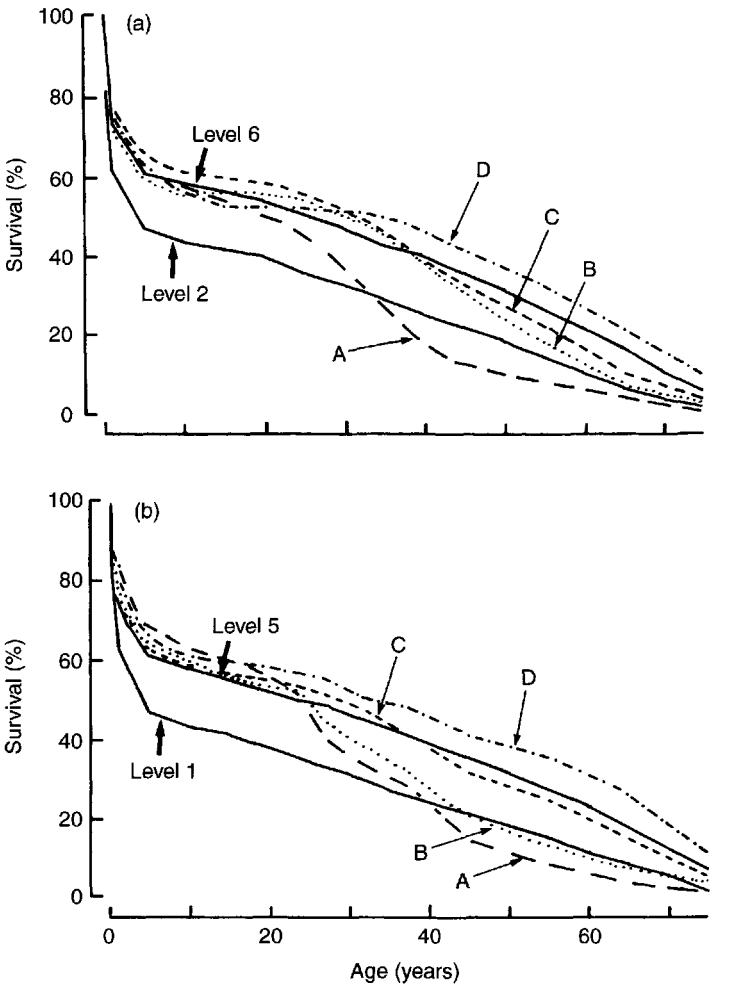


**Fig. 17.2** Calculated percentage male and female survival at Penrith, determined from family reconstitution. A: 1600–49; B: 1650–99; C: 1700–49; D: 1750–1812. (a) Males, survival curves compared with level 2 ($e_0$ = 20 years) and level 6 ($e_0$ = 30 years) (model West; Coale & Demeny, 1966). (b) Females, survival curves compared with level 1 ($e_0$ = 20 years) and level 5 ($e_0$ = 30 years) (model West).

major demographic change in recent years. This ageing of the population may be because of longer survival, but declining birth rates and consequently a higher proportion of individuals in the older age groups contribute to this effect. Demographic transition is the term applied to the shift from high fertility/high mortality to low fertility/low mortality, and this rise in the elderly population is expected to continue throughout the world in the twenty-first century. This fundamental change in the survival curve was largely dependent on the eradication of infectious diseases as major causes of death in the developed world

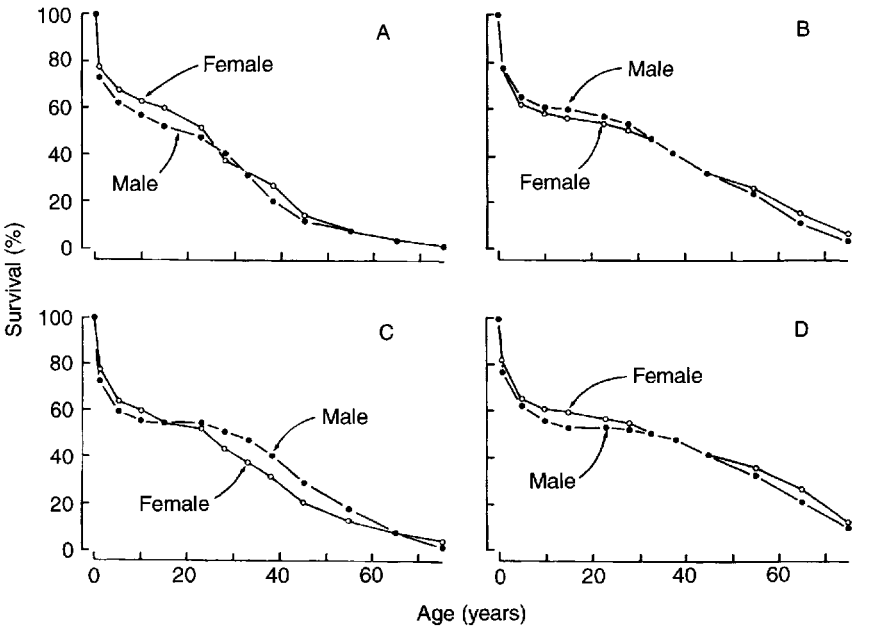


**Fig. 17.3** Calculated percentage male and female survival at Penrith by cohort. A: 1557–99; B: 1600–49; C: 1650–99; D: 1700–49. Ordinate: survival in each year (%). Abscissa: age (years).


**Fig. 17.4** Frequency distribution of ages at death in a cohort of 100 000 live births, based on the mortality of white males in the USA, 1939–41. Ordinate: number of persons dying in each year (thousands). Abscissa: age at death (years).

since 1900, in which, as we have seen in Chapter 16, the general level of nutrition of the poorer sections of the community played an important part.

The generally accepted view is that the theoretical limit of *mean, unmodified* human survival is about 85 years in the fully rectangularised curve (Harman, 1994) (Fig. 17.1).

## 17.2 Life expectancy

> 'Ageing is both multifaceted and hierarchic in its expression, with subtle changes occurring simultaneously at the molecular, cellular, tissue, and organ levels. Further complexity arises at each of these levels as metabolic and homeostatic compensations are made in an attempt to accommodate these age-induced changes in the normal functioning of the animal' (Merry & Holehan, 1994b).

There are many theories, some of them complementary, concerning the causes of inevitable ageing, but two of the most persuasive are:

(1) Chromosomal damage, as measured by the frequency of translocations, acentric fragments, telomere shortening, non-disjunction, chromosome loss, aneuploidy and micronucleus formation, has been shown to increase progressively with age (Fenech, 1998). Telomere depletion is suggested to be the most important of these changes. Telomeric regions, extra lengths of DNA at the ends of chromosomes, were found to be substantially shortened in older somatic tissue and it has been speculated that a 100 base-pair of the telomere cap is lost during each cycle of DNA replication. Eventually, after some 50 replication cycles, the telomeric cap to the chromosome would be used up and the cell line would enter senescence, being no longer able to proliferate.

(2) Oxygen radicals are capable of causing cellular damage and have been suggested as being responsible for the initiation and propagation of ageing. Mitochondria are probably the most important source of oxygen radicals which contribute to the mitochondrial malfunctioning and, in turn, to the genesis of cellular ageing.

It is the hope of experimental gerontologists that the ageing process may be delayed, so that the natural limit to human survival may be artificially prolonged. Manipulation of the diet in various ways is central to these studies. It has been recognised for many years that restricted feeding has a beneficial effect on survival in animal populations (Weindruch & Walford, 1988). Figure 17.5 shows the results of an experiment in which the life expectancy of both male and female rats was extended by some 30%; the animals were fed a restricted diet so that the

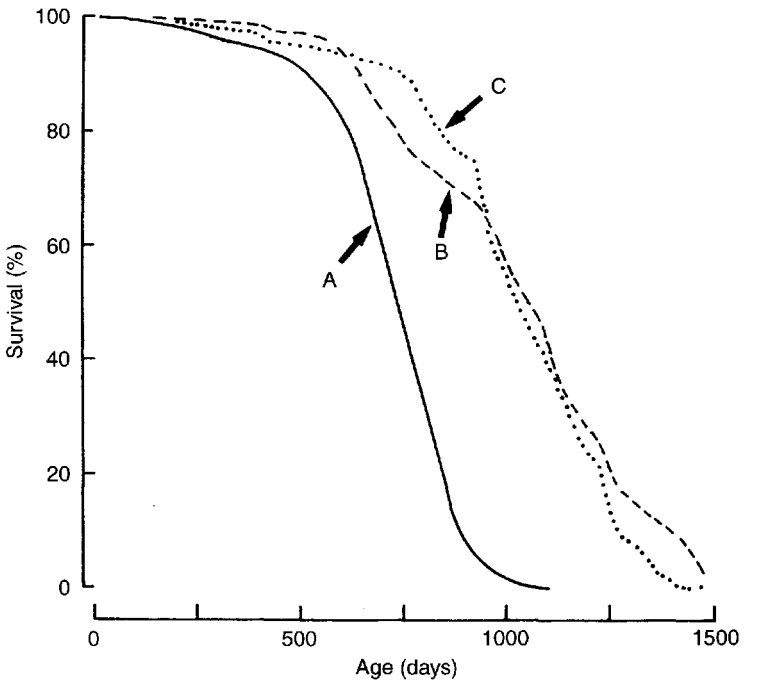


**Fig. 17.5** Survival profiles of CFY Sprague–Dawley rats. A: Fed *ad libitum*; 1094 males, 200 females, data combined. B and C: rats fed a restricted diet such that body weight was maintained at 50% that of the fully-fed animals. B: 450 males; C: 100 females. After Merry and Holehan (1994b).

body weight was maintained at 50% of that of fully fed rats (Merry & Holehan, 1994b). In brief,

> 'restricted regimes which successfully extend survival are designed to avoid malnutrition and to provide essential nutrients and vitamins, while restricting calorie intake by 30 to 70% of the *ad libitum* level. One of the simplest but most effective methods of extending lifespan and therefore, by implication, slowing the rate of aging, has been to limit access to the normal diet in rodents so that the body weight of experimental animals is maintained at 50% that of age-matched, fully fed control animals. This is approximately equivalent to maintaining age-matched animals on half-rations throughout postweaning life. This allows for a continuous slow rate of growth to occur throughout life and results in a 36 to 42% extension in mean and maximum lifespan' (Merry & Holehan, 1994b).

It is now accepted that the greatest effect of dietary restriction regimes on subsequent survival is recorded for animals underfed throughout the majority of the post-weaning period of life. Concomitantly, these animals showed a bene-

ficial effect in maintaining tissue integrity and in reducing a variety of diseased conditions, including the frequency of tumours.

## 17.3 How does a restricted diet decrease the rate of ageing?

Numerous studies with ageing rats have attempted to elucidate the component of the diet (proteins, carbohydrates or calorific value) that is responsible for the rate of ageing, but the results have been inconclusive, although they suggest that the effect on survival in mammalian species depends on restricting the total energy intake (Merry & Holehan, 1994b).

The major causes of death in an ageing human population are pathologies associated with the cardiovascular system in which hypertension is recognised as a major risk factor and, consequently, animal models have been developed to facilitate a detailed study of the genetic and environmental components of this pathology. The spontaneously hypertensive (SH) rat has been claimed to be a good model for the study of essential hypertension in humans, with death resulting at about 18 months compared with 24 months in the normotensive control rats. Restriction of the normal diet to 40% of the *ad libitum* intake extended the mean and maximum lifespan in both normotensive and hypertensive animals: mean lifespan in normotensive animals was increased by 8 months while mean lifespan in underfed SH rats was increased by over 12 months (Merry & Holehan, 1994b).

The KdKd mouse strain exhibits a renal pathology that is similar to that found in man and these animals die prematurely at between 7 and 9 months of age. Restricting dietary energy intake to 50% from the age of 60 days significantly reduced the incidence of renal pathology and survival was increased to over 16 months. When 8-month-old, calorie-restricted KdKd mice were returned to a higher energy intake, a rapid onset of renal pathology was observed and death occurred within 8 weeks (Merry & Holehan, 1994b).

Early studies employed such a severe degree of restricted feeding that rats were maintained for prolonged periods in a prepubertal condition, suggesting that arrested development and enhanced survival were inextricably linked. Refeeding such animals resulted in rapid sexual maturation which was followed by a much foreshortened adult phase of the lifespan, although total survival was increased. It has subsequently been shown that increased longevity in the rat can be achieved with less severe dietary restriction, which does not inhibit sexual maturation or result in the loss of fertility. In male rats maintained at 50% body weight, puberty was delayed by approximately 30 days; the decline in fertility with age was similar in both fully fed and diet-restricted animals, decreasing to 30–50%. In contrast, puberty in similarly restricted female rats was delayed until 63 to 189 days (compared with a range of 34 to 39 days in fully fed rats) and was associated with decreased circulating levels of follicle-stimulating hormone.

These studies of increased longevity by means of calorie-restricted diets appear to be counter-intuitive to what we have found in the preceding chapters, where a fully-balanced diet was essential to healthy living. It must first be emphasised that a satisfactory level of vitamins and minerals was maintained in these experiments with rodents. There are a number of theories concerning the possible mechanisms by which restricted feeding retards ageing. Some involve the reduction of oxygen radicals and oxidative stress generated in the mitochondria, so reducing the damage of these organelles (Armeni *et al.*, 1997; Feuers, 1998; Ames, 1998; Doubal & Klemera, 1999). Some success has been reported in retarding ageing by long-term dietary supplementation with antioxidants (Harman 1994; Meydani *et al.*, 1998). It is suggested that a low-calorie diet makes mice live longer by slowing age-related changes in several key genes, and Merry and Holehan (1994b) conclude that diet restriction induces a reduction in the cellular load of damaged proteins, lipids and DNA. This is achieved, in part, by a modification of age-related gene expression to increase the activity of free radical scavenger enzymes, to induce changes in the composition of membranes which makes them more resistant to free radical peroxidation and to induce a metabolic adjustment whereby lower rates of free radicals are produced. In addition, there is evidence that protein turnover is enhanced in certain tissues from diet-restricted rodents; the effect would be to increase the efficiency of the removal of damaged proteins and to enhance metabolic flexibility.

We have seen (Chapter 7) how malnutrition of women during pregnancy can lead to low birthweight babies and increased neonatal mortality. However, calorie-restricted pregnant rats respond to this physiological challenge by reducing their fecundity and having smaller litters, but the newly-born pups were of the same weight as control animals (B.J. Merry, personal communication). Recent studies on the effects of diet restriction during pregnancy and weaning in the rat reveal that the situation in rodents is more complex than hitherto supposed. Jennings *et al.* (1999) found that ageing in control male rats was associated with significantly longer telomeres in the liver and kidney. Pregnant and/or lactating rat dams were then fed on an isocaloric diet in which the protein constituent was reduced from 20% to 8%, thereby inducing foetal and/or neonatal growth restriction. All experimental animals were then weaned onto a normal diet fed *ad libitum*. Male rats that were growth retarded in this way during foetal life but allowed to 'catch up' during lactation showed a permanent alteration of liver metabolism and died young whereas, conversely, male rats that had grown normally during foetal life but were growth-retarded post-natally showed increased longevity and had significantly longer kidney telomeres (Hales, 1997). In contrast, the changes in female rats (which lived longer than the males) were in the same direction, but too small to reach significance. These workers suggest that these findings provide a mechanistic basis for linking early growth retardation to adult degenerative disease in rats and raise the possibility that the rate

of telomere shortening influences the rate of senescence of renal tubular cells and hence renal failure with ageing.

Recent studies by Lee *et al.* (2000) at the molecular level of brain ageing in the mouse are of particular interest. They looked at more than 6000 genes implicated in the ageing of the brain. These included (i) genes that cause inflammation around brain cells which can increase the risk of stroke, (ii) genes activated when brain cells acquire wear and tear, a likelihood that increases with time, and (iii) genes that appear to be involved in the formation of plaques seen in the brains of Alzheimer's patients. Any gene must be switched on to have an effect, and the experiments were extended to see whether a restricted diet would influence whether the switch was thrown. In the control mice there are 114 genes that are switched on as the animal ages, many being activated when brain cells are damaged or under assault. One example is the folding of proteins into a precise shape, a process that is policed by several genes. In damaged cells, however, the folding becomes unruly and more genes are recruited to maintain order.

In summary, ageing results in a gene-expression profile indicative of an inflammatory response, oxidative stress and reduced neutrophilic support in the neocortex and cerebellum. At the transcriptional level, brain ageing in mice displays parallels with human neurodegenerative disorders. In calorie-restricted mice, however, a third of the 114 genes remained completely dormant, while the remaining two-thirds were activated more subtly. The genes were not recruited as aggressively into supervising damage limitation and it is concluded that the brain cells in the less well-fed mice must have been healthier. The low-calorie diet also switched on a handful of genes that remained inactive in the control mice, but these tended to be ones with a beneficial effect, such as easing inflammation around brain cells.

Lee *et al.* (2000) conclude that the effects of caloric restriction on inflammatory and stress-related transcripts are in agreement with studies that have shown reduced oxidative damage in the brain of calorie-restricted mice (Sohal & Weindruch, 1996): caloric restriction attenuated both increases and decreases in gene expression associated with ageing, providing biological support for the validity of the age-associated transcriptional alterations as determined by microarray analysis. Their suggestions are summarised in Table 17.1.

## 17.4 Human longevity and diet

Is there reason to suppose that these studies of greater longevity with diet-restricted rodents are applicable to humans and other primates? The island of Okinawa has an incidence of centenarians which is 2 to 40 times that of other Japanese islands. A detailed study of this population has shown that the dietary energy intake for adults is about 20% less than the national average and, for school children, it is 62% of the recommended intake for Japan. Death rates from

**Table 17.1** Global view of transcriptional changes in the brain induced by ageing or caloric restriction.

| Normal ageing | Effects of caloric restriction |
|---|---|
| *Increased inflammatory response*<br>Induction of:<br>complement cascade<br>MHC molecules | *Increased growth and trophic factors*<br>Induction of:<br>developmentally regulated genes<br>neurotrophic support |
| *Increased stress response*<br>Induction of:<br>heat shock factors<br>oxidative-stress inducible genes<br>lysosomal proteases | *Increased DNA synthesis*<br>nucleotide precursors<br>DNA replication factors |
| *Decreased protein turnover*<br>suppression of ubiquitin–proteasome pathway | *Decreased protein synthesis*<br>tRNA synthesis<br>elongation factors |
| *Decreased growth and trophic factors*<br>developmentally-regulated genes<br>neural plasticity | *Decreased stress response*<br>Suppression of:<br>oxidative-stress response<br>NF-k-B signalling<br>heat shock factors |
| | *Immune modulation*<br>induction of interferons<br>suppression of inflammatory genes |

Ageing changes apply to both the neocortex and cerebellum. The study of the effect of caloric restriction was limited to the neocortex.
*Source:* Lee *et al.* (2000).

cerebral vascular disease, malignancy and heart disease are 59, 69, and 59%, respectively, of the average for the remainder of Japan. The total death rate for people 60 to 64 years of age was 1280 in Okinawa but 2181 per 100 000 elsewhere in Japan (Takata *et al.*, 1987).

Biosphere 2 is a closed ecological space of 7 million cubic feet near Tucson, Arizona, containing 7 biomes: rain forest, savannah, ocean, marsh, desert, agricultural station and habitat for humans and domestic animals. Sealed inside, four men and four women maintained themselves and the various systems for 2 years. All organic material, all water and nearly all air was recycled, and virtually all food was grown inside. On the low calorie but nutrient-dense diet available, the men sustained an 18% and the women a 10% weight loss, mostly within the first 6 to 9 months. The nature of the diet duplicated rodent diets that had been shown to enhance health, lower disease incidence and retard ageing. Using blood specimens frozen at different points during and after the 2-year period of the study, determinations were made of a number of biochemical parameters judged to be pertinent, based on past studies of rodents and monkeys on similar diets, including blood lipids, glucose, insulin, glycosylated haemoglobin and renin. The results clearly suggest that humans react to such a nutritional regime in a similar way to other vertebrates (Walford *et al.*, 1999).

Leaf (1988) describes how he visited three remote areas where people were alleged to live to extraordinary ages: (i) Hunza in the Hindu Kush mountains, (ii) the low Caucasus Mountains of Russian Georgia and (iii) the Andean village of Vilcabamba in Southern Ecuador. The stated ages in Vilcabamba were greatly exaggerated – the oldest citizen that he met later died at the age of 93, much less than the age of 134 years claimed. No centenarians existed in this community contrary to the claims of the Ecuador Bureau of the Census for nine such long-lived individuals. All three sites have similar lifestyles: agrarian cultures where the old people had laboured hard, eking a bare living out of the thin soil on the mountainous slopes of their small farms. Vigorous physical activity began at an early age and never stopped. Diets were low in calories at Vilcamba but not markedly restricted otherwise (see Table 17.2) and were largely vegetarian by necessity rather than by choice. In Hunza, where the land was too precious to permit animal husbandry, meat was essentially restricted to festive occasions during the long winters. Fat intake was very low by present-day standards, even in the more affluent Caucasus. No one retired; chores changed, but the elderly continued to do tasks that, although less vigorous, maintained a useful role for them in the community and supported their self-esteem.

**Table 17.2** Daily diets of the elderly in three sites renowned for their longevity.

| Reference | Energy (kcal) | Protein (g) | Fat (g) | Carbohydrate (g) |
|---|---|---|---|---|
| Vilcabamba | 1200 to 1360 | 35 to 38[1] | 12 to 19 | 200 to 360 |
| Caucasus | 1700 to 1900 | 70 to 90 | 40 to 60 | 250 to 300 |
| Hunza | 1923 | 50[2] | 35 | 354 |

Data from Leaf (1988).
[1] 12 g of protein from animal sources.
[2] Meat and dairy products provide <1.0% of total.

The conclusions from these studies seem to be that a vegetarian diet with a relatively low calorific intake accompanied by an active lifestyle into old age is conducive to a modest improvement in longevity. However, it is dangerous to extrapolate the studies of life expectancy of captive rodents with standards in seventeenth century England; these animals are specifically pathogen-free and maintained under germ-free conditions, their diet contains a full supply of micronutrients and the environmental conditions are strictly controlled. In contrast, nearly 50% of the population in pre-industrial England did not survive to maturity. Their diet was probably suboptimal in a number of micronutrients, they had low birthweights, which predisposed them to a variety of ailments, and they suffered grievously from infectious diseases.

## 17.5 Dietary fats and ageing

Eaton and Konner (1985) suggest that the diet of our hunger-gatherer forebears during the paleolithic period, 4 million years ago, was high in protein and cholesterol, but low in total fat, and many of the polyunsaturated fatty acids were the *n*-3 α-linolenic, eicosapentaenoic and docosahexaenoic acids resulting from the ingestion of meat of range-fed animals.

Bang and Dyerberg (1985) report that, despite a diet as high in fat as that consumed in Denmark and the USA, the age-adjusted atherosclerotic coronary heart disease of Greenland Eskimos was only one-eighth that of Danes and Americans. Comparison of the lipid composition of Danish and Eskimo diets revealed striking differences in the fatty acid composition, the Danes consuming twice as many saturated fatty acids as the Eskimos. Even more striking was the large proportion of *n*-3 compared with *n*-6 fatty acids in the Eskimo diet. The highly unsaturated, long-chain eicosapentaenoic and docosahexaenoic *n*-3 acids are of marine origin and appear to affect many important cellular and biological functions in a manner different from the polyunsaturated fatty acid, arachidonic acid of the *n*-6 series (Leaf, 1988). A comparison of atherosclerotic coronary heart disease among Japanese living in coastal fishing villages (see above) with that of their countrymen in inland farming communities confirmed the apparent role of a fish diet in reducing mortality (Hirai *et al.*, 1980).

However, about 10 000 years ago, agriculture was introduced (see section 1.3) and with it came human dependence on grains, which led to an increase in total saturated fatty acids and in the *n*-6 polyunsaturated fatty acids, linoleic and arachidonic acids (Leaf, 1988; see section 1.4). The effects of diet on longevity are probably more complex than the laboratory studies with rats suggest. Rodents are certainly fully adapted to a vegetarian diet, but it is not clear whether human physiology has adapted completely from a hunter-gatherer diet to that of an agricultural lifestyle (see section 1.4).

## 17.6 Are the rates of ageing determined *in utero*?

We saw in Chapter 7 how the nutrition of the mother during pregnancy can have profound effects on the mortality and long-term health of her offspring. This has been explained by programming, whereby undernutrition and other influences which restrict early growth permanently change the structure and physiology of the body. Do these effects extend to a modification of the rates of ageing? The long-term effects of poor early nutrition on ageing have been demonstrated in animals but, until recently, they have not been studied in man. Records of early weight were available for 1428 men and women who were born in Hertfordshire between 1920 and 1930 and who were traced. Lower weight at age one year was associated in later life with increased lens opacity score, higher hearing threshold,

reduced grip strength and thinner skin. Visual acuity, macular degeneration and intra-ocular pressure were not related to early growth. It is concluded that the associations between early growth and markers of ageing suggest that, in some systems, ageing may be programmed by events in early life. One potential underlying mechanism is the impaired development of repair systems (Sayer *et al.*, 1998). This is a further example of the insidious but far-reaching effects of malnutrition *in utero*.

Chapter 18

# Conclusions

The change, which began about 10 000 years ago, from a hunter-gatherer lifestyle to one based on plant domestication and, subsequently, on animal husbandry had, arguably, the most profound impact on the demography of mankind. Its effects were far-reaching and they still have an impact on the planet. First, the semi-nomadic existence which had sufficed for over half a million years was gradually exchanged for a more permanent, agriculture-based economy. Teleologically speaking, this change of trophic levels, from secondary consumer to primary consumer, feeding directly on the autotrophs of trophic level 1, makes sense. A rising population of *Homo sapiens* probably began eventually to have difficulty in finding and killing sufficient prey species and so, inevitably, changed to the lifestyle of a primary consumer. This led ultimately to the emergence of larger and more complex communities and, eventually, to civilisations.

The agricultural lifestyle brought with it a much greater workload. The energy costs of getting food from the soil greatly exceeded those of the hunter-gatherer way of life. Nor was the new dietary regime as satisfactory nutritionally. Heights (which are regarded as a proxy measure of health) fell from the levels of the hunter-gatherers and only recovered completely during the twentieth century. The grain-based diet was probably never completely adequate in calories, proteins and fats, and was almost certainly deficient at certain times of the year in many key vitamins, minerals and micronutrients. It is surprising that women ever survived and succeeded in reproducing to an extent that populations very slowly increased. Presumably, the human physiology can adapt to long-term exposure to deficient nutrient levels.

However, this revolutionary change of lifestyle and diet brought demographic consequences. Agriculturalists were now tied to the farming year and everything was focused on the time of the harvest. In this way, a strong seasonal component was imposed on their lives; food was plentiful after the harvest but may have been in short supply in the hungry season (see Chapter 13). Farmers were also very dependent on the weather; adverse conditions could result in poor crop yields and severe malnutrition, with serious and manifold consequences.

Usually, of course, food supplies were adequate and, in some years, there may have been a surplus and the technology of food storage gradually developed. But

this absolute dependence on seasonal climatic and other environmental factors exposed these early communities to the hazards of famine, a danger that has persisted and intensified in developing countries today. Famines had severe demographic consequences but, as we show in Chapter 4, these were immediate and often limited spatially so that the population recovered quite quickly, usually assisted by immigration, with few overt after-effects. However, as we have seen in Chapter 5, even a small mortality crisis can have subtle, and not readily-detectable, long-term demographic effects on a population. While sporadic famines have persisted in various parts of the world for thousands of years, they had only a marginal influence on the demography of pre-industrial England. Chronic and fluctuating malnutrition had more serious and much more important insidious demographic effects.

These problems of fluctuating malnutrition associated with a grain-based diet persisted into the nineteenth century in England (and continue in Third World countries today) and we show in Chapter 3 that regular oscillations in grain prices (that stood proxy for nutritive levels in the emerging economy) appeared in England in the sixteenth century. The causes of these oscillations were probably multifactorial, but we suggest that autoregressive factors were of the greatest importance.

## 18.1 To which diet are we adapted?

Since 1900, the eradication of infectious diseases as major causes of death has exposed another group of diseases of affluence in our Western industrialised societies. These diseases disproportionately afflict older individuals. Nutrition in either excess or improper balance bears considerable responsibility for the prevalence of atherosclerotic heart disease in our societies (Leaf, 1988).

Eaton and Konner (1985) conclude that the diet of our Paleolithic forebears, consumed during a period of some 4 million years when our genetic patterns were established, was that of gatherers and hunters. This diet was high in protein and cholesterol, but low in total fat (approximately 20% of the calories), with a high polyunsaturated/saturated ratio, and many of the polyunsaturated fatty acids were the *n*-3 α-linolenic, eicosapentaenoic and docosahexaenoic acids, which resulted from the ingestion of meat of range-fed animals. Human dependence on grains came with the change to an agricultural lifestyle and this led to an increase in the diet in total saturated fatty acids and in the *n*-6 polyunsaturated fatty acids, linoleic and arachidonic acids. The Industrial Revolution and finally the emergence of agribusiness with processed foods, grain-fattened livestock and hydrogenation of vegetable and fish oils have further reduced the content of *n*-3 fatty acids and increased *n*-6 fatty acids and saturated fats in Western diets. These changes in diet, together with smoking, arterial hypertension, indolence, obesity, diabetes and some forms of stress, have conspired to create the present epidemic

of cardiovascular disease that accounts for approximately one half of all deaths and huge health costs in affluent Western countries (Leaf, 1988).

Garrod (1989) postulated that individual variability in response to pathogens and other environmental hazards reflects biochemical diversity that must be related to evolutionary adaptation. Following the observation that there was a dramatic increase in the prevalence of diabetes in contemporary human populations, it was suggested that this might be because of dietary plenty imposed on what was called a thrifty genotype, the human biochemical constitution that had been selected to take advantage of sporadic food availability.

This concept was later extended to explain what became known as the New World syndrome: the extraordinarily high incidence of diabetes and obesity in certain Amerindian populations. It was suggested that it would have been advantageous for the early settlers of North America, as they crossed the Bering Strait and made their way through the ice-bound American continent, to have a thrifty metabolic genotype. Their present ailments may result from exposure to excessive diets. There has been a rapid change in our environments over a very short period and, at least in our most advanced countries, the opportunities for natural selection to act are limited. Added to this, the rapidly increasing longevity of our populations, with their inability to protect their DNA from the noxious agents that emanate from the byproducts of their body's chemistry and the environment, may render them prone to diseases that result from damage to their genes (Weatherall, 2001).

## 18.2 Nutrition in pregnancy

The thesis developed in this book is that the change to an agricultural lifestyle imposed on mankind a diet that was not completely satisfactory and, for the great bulk of the population, superimposed on this were regular and seasonal periods of malnutrition. This persistent, subadequate nutrition was the major factor that determined the levels of fertility and infant mortality and so had a profound, but not obvious, effect on human demography which is seen most clearly in pre-industrial England where the best contemporary data were compiled and maintained.

It is by analysing the wonderful parish data series of England, particularly by family reconstitution techniques, and comparing the results with the seminal work of the group at Southampton (Barker, 1998; see section 7.2) that the key to the demographic control of human population emerges. Fertility and infant mortality are primarily controlled by the nutrition of the mother before, during and after pregnancy. These demographic effects are most readily detected in a saturated habitat where the population is living under density-dependent constraints (see Chapter 10).

It is now recognised that the build-up of fat reserves in the adipose tissue is a

major determinant of a woman's fertility (see section 6.2). This would have posed no problems in a hunter-gatherer lifestyle, but it must have always been difficult for the bulk of the population with a meagre grain-based diet to maintain adequate reserves, particularly at certain seasons of the annual agricultural cycle. All reserves in marginal communities would be depleted after the hungry season (Chapter 13). We know from current studies (see section 6.6) that a woman's ability to conceive can be dramatically improved by taking a daily multivitamin pill and we conclude that chronic malnutrition could have had multifactorial adverse effects on fertility.

Malnutrition of the mother during pregnancy had severe consequences for the unborn child. At best, it was born underweight and of disproportionate small size and this led to later mortality or health problems (section 7.5). At worst, malnutrition led to stillbirths and perinatal deaths – arguably the most important factor in determining human demography. Malnutrition (which may cover widespread deficiencies or may be restricted to a few key elements of the diet) in just one of the trimesters of pregnancy produced specific effects in the development and programming of the foetus (section 7.4). The cyclical nature of the agricultural lifestyle producing a grain-based diet (section 1.9) caused two additional problems for the health of pregnant women in pre-industrial England: the regular oscillation in grain prices (section 3.3) and the annual hungry season (Chapter 13) could cause periods of acute malnutrition in the working classes which could fall in one or more of the trimesters of pregnancy, with serious consequences for the unborn child.

We show in Fig. 18.1 an outline of how nutrition could control the demography of a population living under marginal conditions via the diet of the mothers. At the centre is the malnutrition of the pregnant woman, upsetting the programming of the developing foetus and causing stillbirths and endogenous and neonatal mortality. Malnourishment of the foetus also caused small-sized disproportionate babies who could suffer from a number of problems:

- Deaths in post-neonatal infancy.
- Susceptible infants who died from weaning diarrhoea (see section 15.6).
- Susceptible children with an increased risk of dying from a range of infectious diseases (see section 15.4).
- Predisposition to a range of diseases in later life (Chapter 7), although this had marginal demographic effects in populations with a low life expectancy.
- If the small-sized neonate was a daughter, she would, irrespective of her nutrition in childhood, tend to produce, in turn, low birthweight daughters, so establishing a vicious circle from which there was little chance of escape (see section 9.11).

The conditions that a foetus experiences in the womb are determined largely by a mother's body composition going into pregnancy, because her unborn child

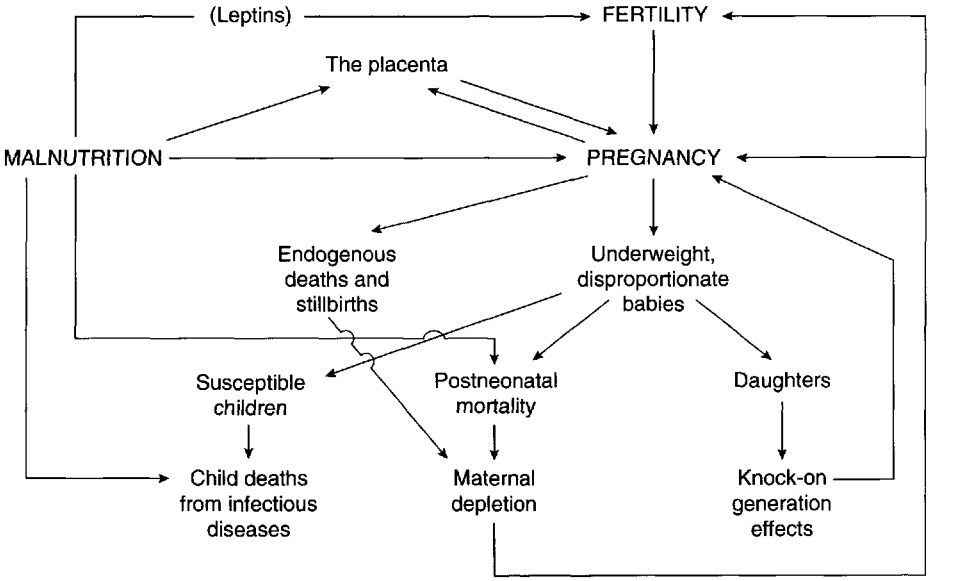


**Fig. 18.1** Summary diagram to illustrate how malnutrition could regulate population dynamics via its subtle effects on fertility and pregnancy. Note the pivotal role of the pregnant woman.

relies on protein derived from her muscles. A woman who has been seriously undereating cannot supply her baby with enough nutrients to sustain normal growth. It is now known that women who diet excessively in the years *before they become pregnant* may be condemning their children to poor health in later life. Even if such women change their eating habits once they are pregnant, their children are more likely than average to suffer heart disease, hypertension, diabetes and strokes.

These low birthweight babies have undergone irreversible changes to their metabolism and to their hormonal and circulatory systems that predispose them to disease. Improvements in diet during pregnancy are not enough to compensate for the physical condition of the mother when she conceives.

The dangers are magnified if a small baby grows slowly during the first year of life and then becomes overweight in later childhood. The result is a high proportion of body fat in relation to muscle, a known risk factor for heart disease and high blood pressure.

Finally, infectious diseases were the scourge of post-neonatal infants and children, and so they had a major demographic impact (see Chapters 15 and 16). Malnutrition of infants and children can have two direct and obvious effects. It can reduce resistance to the disease and can increase the chances of dying from it.

## 18.3 Overview of the interaction of human demography and nutrition

From the foregoing chapters, it is possible to construct an overview of how nutrition has shaped human evolution. Popkin (1999) recognises five trends which are necessarily restricted to particular periods of human history and some stages still characterise geographic and socio-economic subpopulations today. Popkin (1999) proposes that major shifts in population growth, age structure and spatial distribution are closely associated with nutritional trends and dietary change.

*The hunter-gatherers*

The varied diet was composed of plants and wild animals; they had primitive stone tools and discovered fire. They were nutritionally robust and lean, and probably had few deficiencies. They lived in low density settlements. Fertility and life expectancy were low; mortality was high. Consequently, the population was young. They suffered from infectious diseases, but there were no epidemics because of the low density. Small populations of hunter-gatherers continue to exist on this diet today (section 1.2).

*The change to an agricultural lifestyle (the Neolithic revolution)*

The diet was now less varied with cereals predominating and there was a gradual shift to monoculture. Animal husbandry began and the lifestyle was labour-intensive. Clay cooking vessels were invented. The children and women suffered most from a low fat intake; nutritional deficiency diseases emerged; stature declined. Natural fertility is believed to have been high but infant and maternal mortality were high leading to a low life expectancy. The population remained young.

*The second agricultural revolution*

The introduction of crop rotation, fertilisers, agricultural tools and technologies led to a diet with fewer starchy staples and more fruits, vegetables and animal protein, but the low variety continued. Maternal and child nutritional problems continued and, although many deficiencies disappeared, weaning diseases emerged. Stature began to grow. Fertility was initially static but declined later. Overall mortality declined so that there was cumulative population growth and a gradual shift to an older population began. Large cities developed with ever-increasing immigration.

*Degenerative diseases*

Greatly improved living standards led to a marked rise in life expectancy and an increase in the proportion of the elderly in the population. Fertility fell. The composition of the diet changed substantially. It was more varied but included more animal fat, sugar and processed foods and less fibre. This led to obesity, particularly since fewer jobs now involved heavy physical activity. This unsatisfactory diet and lifestyle, which was accompanied by pollution, led to a marked rise in chronic disease in late middle age.

*Nutrition today and in the future*

With the increasing realisation of the importance of a satisfactory and complete diet throughout life (particularly among women) disability-free life expectancy should increase, with people living into their 80s. The proportion of elderly will increase and there will be a complete change in the demographic profile of advanced populations. Preventive and therapeutic health promotion will increase, smoking and other pollution factors will decrease, and there will be a concomitant decline in coronary heart disease and an improvement in the age-specific cancer profile. Diets will include more carbohydrates, fruits and vegetables but less animal fat, sugar and processed foods, resulting in reduced body fat levels and obesity, and improved bone health – almost a return to an artificial lifestyle of the hunter-gatherers.

## 18.4 Malthusian demographic theory

> 'Malthus forcefully expressed the view that the rate of population growth was positively related to the general level of material welfare, which was in turn negatively related to population size, due to diminishing returns to labour inputs on scarce agricultural land. At some level of welfare, population would neither grow nor decline; this level was known as the long-run equilibrium wage' (Lee, 1986).

Malthus assumed that the *per capita* supply of food decreases as the population grows (Wood, 1998). We have shown in the previous chapters that this is an oversimplified view of the control of the population of pre-industrial England. There would have been a lower level of the daily food supply of the labouring classes (simply measured in calories) that would have been sufficient for the maintenance of life, but fertility would be low and infant mortality would be high. Such a population would not replace itself and it would automatically correct the situation by negative feedback, according to Malthusian theory. Usually, of course, the food supply for much of the year was adequate to ensure reproduction and a supply of female children who survived to child-bearing age.

The key to the long-term control of the population dynamics lay much more specifically in the nutrition of the mother before, during and after pregnancy. The vital factor was not just a satisfactory calorific intake, but also an adequate supply of specific vitamins and minerals at critical periods of pregnancy. In this way, a single vitamin or mineral could effectively and subtly exert demographic control in spite of a steadily improving maternal nutrition. For example, endogenous infant mortality in England ameliorated erratically through the eighteenth and the early part of the nineteenth centuries because of the improvement in overall nutrition levels, whereas exogenous mortality, although fluctuating, was largely unchanged, largely because of mortality from infectious diseases. This situation was reversed after about 1870 and the level of endogenous mortality was maintained until the widespread iodination of milk began in about 1940.

When humans changed from a hunter-gatherer to an agricultural lifestyle, another subtle, complicating factor was introduced. A seasonal component was imposed on the food availability of the labouring classes who, in some years, would have no reserves to see them through the hungry season. Most would survive by tightening their belts, but the foetuses of pregnant mothers could have been at risk at this time.

# Appendix

Equations 15.1 to 15.4 describe the dynamics of infectious diseases and represent a non-linear system, but a linearised model is described below. The dynamics are described by a standard SEIR model (Bolker & Grenfell, 1995) that is driven by periodic variations in susceptibility (Rand & Wilson, 1991; Duncan *et al.*, 1994a; Bolker & Grenfell, 1995). This has the form

$$\frac{dx}{dt} = \mu - \mu x - N\beta xy\ (1 + \delta\beta \sin \omega t) \qquad \textbf{(A.1)}$$

$$\frac{dy}{dt} = N\beta xy\ (1 + \delta\beta \sin \omega t) - (\mu + v)y \qquad \textbf{(A.2)}$$

where $N$ = number in the population
$x$ = number of suceptibles expressed as a fraction of $N$
$y$ = number of infectives expressed as a fraction of $N$
$\mu$ = death rate = 1/life expectancy
$\beta$ = transmission coefficient (susceptibility to disease)
$\delta\beta$ = fractional variation in susceptibility
$v$ = rate of recovery from disease = 1/infectious period
$\omega$ = angular frequency of oscillation in susceptibility = $2\pi$/period of oscillation

This model ignores both the latent period of the infection and the mortality resulting from the disease. These factors can be included in the model, but they do not substantially alter the results (see Duncan *et al.*, 1999).

In the absence of variations in susceptibility ($\delta\beta = 0$) the steady-state values for the proportion of susceptibles and infectives are (Anderson & May 1991):

$$x_0 = \frac{\mu + v}{N\beta} \qquad \textbf{(A.3)}$$

$$y_0 = \frac{\mu}{\mu + v}(1 - x_0) \qquad \textbf{(A.4)}$$

When $\delta\beta > 0$ is small, equations A.1 and A.2 can be approsximated by a linearised model, by defining

$$x = x_0 + x_1 \tag{A.5}$$

$$y = y_0 + y_1 \tag{A.6}$$

where $x_1$ and $y_1$ represent the variations in $x$ and $y$ from their steady-state values. Substituting equations A.5 and A.6 into equations A.1 and A.2 (using equations A.3 and A.4) and ignoring higher order terms gives

$$\frac{dx_1}{dt} \approx -(N\beta y_0 + \mu)x_1 - (\mu + \nu)y_1 - (\mu + \nu)y_0\ \delta\beta \sin \omega t \tag{A.7}$$

$$\frac{dy_1}{dt} \approx N\beta y_0 x_1 + (\mu + \nu)y_0\ \delta\beta \sin \omega t \tag{A.8}$$

These equations describe a forced second order linear system, where the forcing function is the periodic driving term $(\mu + \nu)y_0\ \delta\beta \sin \omega t$, i.e. oscillations in susceptibility ($\delta\beta$) can act as a driver for the system (Olsen & Schaffer, 1990; Rand & Wilson, 1991; Tidd *et al.*, 1993). The system can be characterised by its natural, undamped frequency, $\omega_n$, and by its damping factor, $\zeta$, which is a dimensionless ratio in the range 0 to 1 and is a measure of the degree of damping within the system, i.e. the attenuation of the amplitude of the oscillation at its resonant frequency. The damping factor is given by

$$\zeta = \frac{N\beta}{2(\mu + \nu)} \sqrt{\frac{\mu}{N\beta - (\mu + \nu)}} \tag{A.9}$$

For the values of $\mu$, $\nu$ and $N\beta$ used to describe the smallpox epidemics in London during 1847–80, $\zeta$ is small (much less than 1), indicating that the system is lightly damped. Since $\zeta$ is small, the system will amplify a driving term that has a frequency at the resonant frequency $\omega_r$, where

$$\omega_r = \sqrt{1 - \zeta^2}\ \omega_n \approx \omega_n \tag{A.10}$$

and

$$\omega_n = \sqrt{\mu[N\beta - (\mu + \nu)]} \tag{A.11}$$

If the driving term for the system is $\delta\beta \sin \omega t$, the linear response of the fraction of the population is simply a scaled and phase-shifted version of the input signal, i.e. a sinusoid at the same frequency, but with a different amplitude and phase. Thus the output becomes

$$\delta\beta\ A(\omega) \sin [\omega t + \varphi(\omega)]$$

where

$$A(\omega) = \frac{1}{N\beta}\sqrt{\frac{\omega^2 + \mu^2}{\left(1 - \frac{\omega^2}{\omega_n^2}\right)^2 + \frac{4\zeta^2\omega^2}{\omega_n^2}}} \tag{A.12}$$

and

$$\varphi(\omega) = \tan^{-1}\left(\frac{\omega}{\mu}\right) - \tan^{-1}\left(\frac{2\zeta\omega\omega_n}{\omega_n^2 - \omega^2}\right) \tag{A.13}$$

Because the frequency response of the system is very sharp, the amplification will be limited to driving terms that have a frequency close to this resonant frequency (Franklin *et al.*, 1994). As a result, the oscillations in $x_1$ and $y_1$ will be dominated by frequency components close to $\omega_r$. The period of the resonant frequency is

$$T = \frac{2\pi}{\sqrt{\mu[N\beta - (\mu + \nu)]}} \tag{A.14}$$

Thus, the inter-epidemic interval is determined by $N\beta$. Since $\mu \ll \nu$, this expression coincides with that given by Anderson and May (1991):

$$T = 2\pi\sqrt{(AD)} \tag{A.15}$$

where $A$ = average age of contracting the disease and $D$ = length of infectious period.

In addition to its dominant effect on $\omega_r$, $N\beta$ also affects the amplitude of the periodic driving term. Substituting equations A.3 and A.4 into

$$(\mu + \nu)y_0\ \delta\beta \sin \omega t$$

gives the periodic driving term

$$\mu\left[1 - \left(\frac{\mu + \nu}{N\beta}\right)\right]\delta\beta \sin \omega t,$$

where the amplitude is defined by

$$\mu\left[1 - \left(\frac{\mu + \nu}{N\beta}\right)\right]\delta\beta$$

Thus, an increase in $N$ (e.g. population size/density) will amplify the effect of the oscillations in susceptibility ($\delta\beta$), i.e. the more crowded the population, the bigger the effect of a standard oscillation in $\delta\beta$.

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# Index