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Introduction

Anderson (1994) suggested that 'Most of the infections that have attracted the greatest attention in the historical literature on human demography and disease are epidemic in character where the infection sweeps through a population . . . inducing high mortality. To examine their potential impact on human populations a model that combines both epidemiological and demographic processes is ideally required'; we hope to rise to the challenge in this study of the demography of historic populations. A new approach to historical epidemiology is attempted in which we apply the statistical technique of time-series analysis to a range of different data series to elicit quantitative information concerning not only population cycles but also the occurrence and biology of the epidemics of lethal infectious diseases, comparing the findings with mathematical models. In this way, we try to bridge the gap between historical studies of diseases and the current interest in the mathematical modelling of epidemics that occurred in the 20th century.

The publication of *The Population History of England 1541–1871* (Wrigley & Schofield, 1981) represented a landmark for the study of historical demography. These authors showed that the parish registers of baptisms, marriages and burials in earlier centuries contain a wealth of valuable information and that, by using relatively simple (although time-consuming) techniques, it is possible to extract detailed demographic data and to reconstruct the population history. Much can be achieved by aggregative analysis but to determine many of the demographic characteristics it is necessary to employ family reconstitution.

Wrigley & Schofield worked with the aggregative monthly and annual totals of baptisms, marriages and burials of 404 sample parishes in England; when these data are pooled the resulting plots suggest a slowly rising trend in population numbers in England from 1640 to 1740 which

accelerated sharply thereafter. However, this pooling of aggregative data obscures the clear differences between individual communities; it is better to consider England in earlier centuries as a metapopulation, i.e. a population of populations, an ecological approach that is discussed in Chapter 15. When an individual community (usually a parish) is studied in detail, the rather static view is replaced by a dynamic picture of the population: cycles in births, deaths and migratory movements can be detected, the community (depending on its underlying dynamics) may have been stricken by regular epidemics of lethal infectious diseases; major mortality crises may have had an impact on the parish. All these factors would affect the overall population dynamics of a single community and, in turn, may be explicable in terms of its demography, geographical location and local economy. It is the oscillating biological systems that are the most interesting.

A study of the dynamics of a community, based on its burial and baptism records, therefore, begins with the elucidation of the underlying cycles in births and deaths. At first sight, most parish register series are very 'noisy' (i.e. contain much random variation) and appear as something of a jumble. We hope to show in this book how time-series analysis can be used to identify and characterise the different cycles (or oscillations) in a parish register series. It is a computer-based, statistical technique that permits a careful analysis of a data series, the separation of cycles of different wavelengths and the evaluation of confidence limits, so replacing anecdotal evidence and supposition with a firm quantitative approach. Time-series is used here to analyse a variety of data series: Bills of Mortality, temperature, rainfall and annual commodity prices as well as the baptisms, marriages and burials listed in parish registers.

It will be suggested that two completely different types of cycle can be detected by this means in single historical populations: exogenous and endogenous. The exogenous cycle is the more common and is the term that is given to fluctuations in the population that are driven by external factors. The second role for time-series analysis is the determination, with confidence limits, of the possible correlation between an exogenous cycle in baptisms or burials with cycles in environmental factors, such as seasonal weather conditions.

Endogenous cycles are less commonly detected in parish register series and are dependent on the inherent properties of the dynamics of the population. Once endogenous cycles in baptisms and burials in a community have been identified and characterised, it is possible, as we describe in Chapter 4, to construct matrix models of the population dynamics and to

compare the results of running the model with the historic events determined by time-series analysis.

1.1 Mortality and demographic theory

Malthus (1798) suggested that population growth is regulated by the relative strengths of positive and preventive checks, and believed that the very slow rise in the population of European countries was the result mainly of a positive check that linked poverty and high mortality. He therefore placed greater emphasis on mortality, especially among the lower orders of society, but conceded that the process of delaying marriage (a preventive check) did operate to some extent. Landers (1993) pointed out that the progress that has been achieved in historical demography has been accomplished largely at the price of excluding mortality from the domain of structural analysis, concentrating instead almost exclusively on fertility and, in particular, nuptiality. Thus, Wrigley & Schofield (1981) have changed the emphasis from the Malthusian view, suggesting that the demographic pattern was determined more by fertility than mortality; from 1551–1751, England appears to have been controlled by an effective preventive check homeostatic equilibrium, with an intrinsic growth rate of approximately 0.5% per year. The changes in fertility were determined almost entirely by changes in nuptiality. Age at marriage was relatively stable and fertility responded closely to changes in the proportion marrying; there was an accelerated growth rate particularly during the late 18th century (Wrigley & Schofield, 1981). Landers (1993), working on the data from London for the period 1670 to 1830, presented the opposite viewpoint and analysed the available data within the framework of a structural model of mortality change which describes the specific demographic and epidemiological characteristics of early modern metropolitan centres. Both viewpoints may be correct, although we have found (see Chapters 7 and 8), following a family reconstitution study of a rural town in Cumberland, northwest England, that mortality was of overriding importance in determining the population dynamics there, illustrating the importance of studying individual populations and not pooling the data from communities where the underlying demography may be very different.

Historical mortality patterns are still little understood and the demographic parameters of mortality decline are unclear. A reduction in the intensity of mortality fluctuations in England spanned the latter part of the 17th century to the beginning of the 19th and was, apparently, characterised by a marked reduction in the crisis mortality that was caused

by infectious epidemic diseases with, firstly, the disappearance of plague and then, a century later, the substantial decline in smallpox and typhus. However, the level of non-crisis mortality, in fact, increased during this period (Wrigley & Schofield, 1981). Wrigley & Schofield (1981) suggested that the lulls between mortality crises allowed the population to recover; they found that mortality in England worsened between 1580–1680 such that life expectancy declined from nearly 40 years to just above 30 years. From 1740–1820, there was a sharp improvement and expectation of life at birth rose from 31.7 to 39.2 years. Between these two periods, however, there was little discernible long-term trend, although this was a time of worsening mortality levels for both infants and for children aged 1–4 years (Wrigley & Schofield, 1981).

Two themes that run through this book are the role of homeostasis in populations in steady-state and the importance of the interaction of the different mortality cycles in population dynamics. Human populations often appear to fluctuate in cycles. Oscillations with a periodicity of 15 years have been imposed, it is suggested, on demographic rates by the climate or by fluctuations in the quality of the harvest, and a 20-year Kuznets cycle has been found which may reflect economic–demographic interactions (Easterlin, 1968). Longer wavelength oscillations have been detected in different historical periods; a 30-year cycle is found in the plotted data of baptisms from preindustrial parishes (Lee, 1974); other cycles have a periodicity of one generation, but longer cycles of 40 to 60 years that are closer to two generations have also been described and the 50-year Kondratieff cycle runs through the demographic variables of 19th century Europe (Lee, 1974; Herlihy, 1977). These latter oscillations have attracted most attention and two types of cycle have been identified. Firstly, transient, or generation-long, oscillations which reflect the intrinsic dynamics of population renewal and can occur in populations growing without effective constraint, such as might occur temporarily in newly settled areas; these oscillations taper over time (Lee, 1974, 1987). Secondly, control (or limit) cycles which differ from generation-long cycles in that they have no tendency to damp or decay (Wachter & Lee, 1989) and demographers such as Malthus and Easterlin have suggested theories for the existence of a steady-state population size or growth rate in which oscillations are driven by feedback (Easterlin, 1980). Although the factors causing these cycles are unclear, changes in fertility and birth rate within a controlled system have been advanced as the most probable underlying control mechanism (Lee, 1974). These cycles can continue unabated as long as the feedback factor is dominant (Wachter, 1991). The baby boom and bust pattern in the fertility

of the USA in the 20th century is an example; the series displays a trough in the mid-thirties, a rise to a peak in the late fifties and a further decline thereafter (Lee, 1974). The cyclic pattern of these detrended US births 'is as clear a sinusoidal curve as any social scientist could dream of' (Wachter, 1991); the cycles have a periodicity of about 44 years, although the years covered, 1900–1984, allow for only two oscillations.

Demographic feedback models have been used to reproduce the regularity of these cycles (Lee, 1974; Wachter & Lee, 1989), but the models were all deficient in one way or another and the strength of feedback response either failed to generate control cycles or produced a periodicity of 90 years (Frauenthal & Swick, 1983). It has been concluded: 'A few models, in their pure form, do generate cycles of appropriate period and amplitude when fertility response takes on the strength observed. These are rather special models. Moderate changes in specification spoil the success' (Wachter, 1991). Questions still arise as to how far the intrinsic tendencies of the population may be affected by temporary conditions, by declining mortality or by waves of immigrants, and whether in human populations there are feedback mechanisms of sufficient sensitivity to generate self-sustaining cycles (Wachter & Lee, 1989). The identification and characterisation of population cycles, their interactions with one another, the reasons for their genesis and the validation of the conclusions advanced by the construction of mathematical models are the subject of this book.

1.2 Malnutrition and famine

Malthus (1798) suggested that the children of the poor suffered from malnutrition, which undermined their health and stunted their growth and the result was a high mortality. From this time, many demographers have considered the insufficient supply of food resources to be the main constraint of population growth and the main cause of the high mortality prevailing in pre-industrial times. McKeown (1976) believed that the effect was indirect and that population growth was mainly the consequence of the decline in mortality caused by the reduction in infectious diseases following an improvement in nutrition. Progress in transport and agriculture in the 18th century, particularly the creation of national grain markets, produced a more even distribution of grain and the enhanced nutritional status led to an increased resistance to infectious organisms. In support of this theory, research from developing countries affirms a clear relationship between malnutrition and susceptibility to infectious diseases and mortality (see section 11.12). The synergism between malnutrition and infection

is particularly apparent in young children; not only are they more predisposed to infectious diseases, but the illnesses are likely to be more severe and with a higher risk of death. Babies with low birthweights in developing countries are eight times more likely to die as neonates and four times more likely to die during the post-neonatal period (Ashworth & Feacham, 1985). The relationship extends beyond infancy, and low body weight increases a child's susceptibility to illnesses such as diarrhoea (Scrimshaw, Taylor & Gordon, 1968). Nutritional levels have also been indirectly linked to mortality in adult populations and there are positive associations between short-term fluctuations in grain prices and adult mortality both at the time and in subsequent years (Lee, 1981; Galloway, 1988). More recently, this theme has been taken up by the work of Barker and his colleagues who have found ecological correlations in the 20th century between mortality rates by geographical regions in a given modern cohort and mortality rates in the same region at the time that the cohort was born. Death rates from diseases such as bronchitis, heart disease and strokes were linked to the body weight at birth and it is suggested that nutritional deprivation at different stages during pregnancy could affect developing organs and the consequences were then expressed in adult life (Barker & Osmond, 1986a,b; Barker, 1992a).

Livi-Bacci (1991) presented a different viewpoint. He analysed the available evidence over the period from the Black Death to the industrial revolution, interpreting the scanty quantitative information concerning calorific budgets and food supply, prices and wages, changes in body weight and epidemiological history and the contrasting demographic behaviours of the rich and poor. Livi-Bacci cast doubt upon the existence of any long-term interrelationships between nutritional levels and mortality, showing that the level of the latter was determined more by epidemiological cycles than by the nutritional level of the population. He suggested that the permanent potential conflict between food supply and population growth was also mediated by the biological adaptability of the human species to nutritional stress.

Because malnutrition and famine could potentially have had important effects on the population dynamics of a community, the area chosen for special study and reported in this book was northwestern England in the 17th and 18th centuries. Following the pioneering work of Appleby (1978), the northwest of England has been suspected to be subject to famine and sensitive to malnutrition (Howson, 1961; Rogers, 1975; Millward, 1983). The Eden Valley (Cumbria, England) was backward, the farming conditions were marginal (Searle, 1983, 1986) and mortality crises occurred at

the end of the 16th and the start of the 17th centuries. Penrith is a market town that lies at the centre of the region and the derivation by a family reconstitution study of the demographic parameters of the community reveals a deprived society with low marital fertility where the dynamics were governed first by infant and then by child mortality. However, as we show in Chapter 8, the mortality crises in the northwest were not triggered solely by high grain prices, but occurred only in years where these coincided with low wool prices. Malnutrition is shown in Chapter 7 to have had a profound, but subtle, effect on mortality cycles that is not readily detectable by conventional demographic analysis; it is suggested that nutritive levels were particularly important during pregnancy and the first year of life (Scott, Duncan & Duncan, 1995; section 7.7), in agreement with current studies of historical epidemiology of the 20th century (Barker, 1992a). It is clear from Chapter 7 that an understanding of infant and child mortality is the key to population dynamics in the 16th and 17th centuries and it must be remembered that deaths in infancy can normally be determined only after a family reconstitution study.

Fogel (1994) has surveyed the extensive literature concerning the secular decline in mortality in western Europe during the late 19th and early 20th centuries and concluded that the elimination of crisis mortality accounted for less than 10% of the reduction in mortality rates. He suggested that by demonstrating that famine mortality was a secondary issue in the escape from the high mortality rates of the early modern era, these studies have shifted attention to the neglected issue of the principal contribution of *chronic* malnutrition to the high mortality rates of the past. Malnutrition and famine are contributors to a complex syndrome of multiple interconnecting effects involving three major demographic variables – mortality, fertility and migration (Chen & Chowdhury, 1977) and there has been a call for a conceptual framework to examine the complicated interrelationships between demographic processes and famine (Hugo, 1984). As Fogel (1994) has pointed out, we have not yet completed the escape from hunger and premature death that began nearly three centuries ago and chronic diseases and early death are still occurring, even in the rich countries. He suggested that economists need to take account of long-run dynamic processes through a study of history, although uncovering what actually happened in the past requires an enormous investment in time and effort. As Walter & Schofield (1989) have emphasized

> the incidence of crises only provides information on the more extreme consequences of famine in the form of exceptionally high mortality. There may also have been a less obvious, yet systematic, relationship between the availability of food and death

operating across the whole range of fluctuations in prices and mortality. Such a relationship would be difficult to detect by scanning long series of data with the naked eye, especially if the effects of fluctuations in food prices on mortality were spread over several years. Its investigation, therefore, requires a careful statistical analysis of the covariation of food prices and series of vital events.

An attempt will be made to meet these challenges and to provide a description and a matrix model of the population dynamics of a community living in Cumbria, northwestern England, under conditions of hardship and deprivation over a long period of time (Appleby, 1978; Walter & Schofield, 1989) and determining the statistical correlation between mortality and grain prices. We believe that this is the first time that the dynamics of a homeostatic population have been explored in this way.

1.3 Lethal infectious diseases

Livi-Bacci (1991) has drawn attention to a possible synergy in the short term between famine and epidemic infections in determining mortality crises, and Chapters 9 to 13 are based on the mathematical modelling of the epidemics of lethal infectious diseases and the exogenous factors by which they were maintained. Historical demographers have suggested correlations between food intake and disease and the mechanisms that push or pull populations (Rotberg, 1983) and recent research indicates that in many European nations before the middle of the 19th century, the national production of food was at such low levels that the poorer classes must have been malnourished and this led to the high incidence of disease (Fogel, 1994; Duncan, Scott & Duncan, 1994a). Malnutrition *in utero* may predispose young children to certain epidemic diseases such as whooping cough (Duncan, Duncan & Scott, 1996a), measles (Duncan, Duncan & Scott, 1997) and scarlet fever (Duncan, Duncan & Scott, 1996b).

Diseases that are mostly associated with crisis mortality, with the exception of typhus, have been suggested to be relatively insensitive to nutritional levels (Rotberg & Rabb, 1985) and other factors have been advanced. For instance, it has been suggested that a colder climate may have helped to mute the effect of disease (Perrenoud, 1991), although this is contrary to the idea that colder winters are associated with increased mortality (Lee, 1981; Wilmshurst, 1994; Duncan *et al.*, 1996a). Government intervention with improvements in public health may have reduced the effects of epidemics and lessened the consequences by the introduction of quarantine measures, improved sanitation and more efficient methods of burial. However, although it has been shown that government intervention was

decisive in reducing the consequences of the crisis in the early 1740s (Post, 1985), this argument is still speculative.

We show in Chapters 9 to 14, by using time-series analysis, that seasonal weather conditions were significantly correlated with the maintenance of the epidemics of certain lethal infectious diseases, but nutritional levels could also have had important effects on the dynamics of these diseases in different ways. Again, these effects are subtle and are not readily detectable by conventional historical analysis. Malnutrition has its effects on infection particularly in young children and these have been ignored previously because analyses of burial series have not separated children and adults (Chapter 13) and because the mathematics of the dynamics of epidemics (Chapter 9) have not been properly understood. We show that malnutrition can potentially (a) increase general susceptibility to the disease, (b) increase the chance of an infective dying, (c) act as a driver for the epidemics, (d) increase susceptibility in children indirectly via an effect in pregnancy, and (e) promote migration in search of jobs and food, so increasing the spread of infectives and susceptibles (Landers, 1987).

An understanding of the dynamics of lethal infections in earlier centuries is relevant to Third World countries today where conditions approximate to those of England in the 17th century. Malnutrition and overcrowding, both important determinants of the dynamics of epidemics in earlier centuries, are rife today. Smallpox has now been conquered but the World Health Organization reports that we face a global crisis with more than 17 million people dying from infectious diseases each year. New diseases, such as hepatitis C, ebola, haemorrhagic fever and the new strain of Creutzfeldt–Jakob disease are emerging; acquired human immunodeficiency syndrome (AIDS) has transformed into a global pandemic in just a few years. Some diseases have developed serious drug resistance. A vaccination programme for the world-wide elimination of measles is under consideration and there is also the real possibility of eliminating poliomyelitis and leprosy within a few years. Any such control measures depend crucially on an integrated understanding of the dynamics of the disease and of the population dynamics, an analysis that we have attempted in this book for populations living under broadly comparable conditions of hardship and deprivation in England in the 17th and 18th centuries. Modelling of the demographic impact of AIDS today, including its rate of spread in different subcontinents, the role of female prostitutes, age distribution of the population and the rates of change of sexual partner, is now being presented (Anderson, 1994; Garnett & Anderson, 1993a,b; Anderson *et al.*, 1991).

Even more exciting is the combination of molecular biology, ecology and epidemiology in studies in which mosquitoes are genetically engineered to secrete chosen proteins in their saliva. These proteins could be vaccines which would then be transmitted in the saliva of the mosquito which had thus been designed to act as a 'flying hypodermic syringe'. The scheme could be used to immunise humans or other mammals against a wide range of diseases, and any biting insect, not only mosquitoes, could be used to carry the vaccine. The appropriate gene has already been introduced into a mosquito and it produces antigen in sufficient amounts (Crampton, 1994; Crampton *et al.*, 1994). Again, the success of such schemes is dependent on an understanding of the underlying demography and epidemiology in Third World countries.

1.4 Readership

This book attempts to provide a new and integrated approach to human demography and infectious diseases in England in earlier centuries. It begins with detailed studies of single populations in the northwest and is set against the marginal economy of the region where mortality was of central importance in demographic control. Extensive use has been made of conventional time-series analysis to elucidate the nature of the interactions of the different oscillations that can be detected in the baptisms and burials series. The study investigates the nature of famine, food shortage and malnutrition, the prevalence and characteristics of infectious diseases and their impact on the population and on the mechanisms of homeostatic regulation. It is believed that this is the first description of a human population in steady-state, and computer simulation models are presented (Chapter 4) to describe the underlying population dynamics and the nature of the feedback mechanisms involved.

Smallpox was a major cause of childhood death in earlier centuries and the dynamics of this and other lethal, infectious diseases and their integration with the demography of populations ranging in size from small communities through rural towns and cities to London is discussed in the second part of the book. We hope that this quantitative, interdisciplinary approach will be of interest to a spectrum of readers:

Theoretical population biology

Ecologists have long been interested in population cycles, and the interrelationships of predator–prey cycles is used as an introductory example of the use of time-series analysis in section 2.2.1. However, one difficulty in such