Epidemics, especially epidemic deaths, are important to demographers because:

- they constitute a major demographic event and can affect population growth,
- they are of central importance to demographic theory.

In the strict sense of the term, an epidemic involves an upswing of infectious or contagious disease with a subsequent decline so that there is a peak in the number of infected people, and usually in the number of deaths, before a fall to lower or even zero levels. Infectious diseases, and hence epidemics, have been largely a post-neolithic-revolution and post-urban revolution phenomenon because sparse hunter-gatherer populations could not sustain a continuing process of infection. Indeed, epidemics have tended to be worse in denser populations and in urban areas. Some epidemics can be long sustained only in dense populations beyond a minimum size. For instance, in cities with fewer than 300 thousand inhabitants, measles epidemics finally die out until a new epidemic is, after a number of years, introduced again from outside.

Infectious disease at a fairly constant level is described as being endemic. Endemic disease may rise to a higher level, but if it remains at this new plateau it is not described as an epidemic. Usually, however, rises are followed by a fall. This occurs for a number of reasons: those most likely to be infected have died or the survivors have built up a resistance to the disease; among those infected, many have a natural resistance; the densest populations have already been decimated. Some infectious diseases do not provoke strong immune reactions, and hence it is difficult to produce vaccines: examples are cholera and modern bubonic plague.

The vocabulary employed is descriptive but not very analytical. Nearly all terms come from Greek, through Latin and French into English. Several are built around ‘demos’ the Greek word for people, from which demography is derived: examples are endemic (among people), epidemic (through or over people), and pandemic (among all people).

There are many infectious diseases which may become epidemic: measles, whooping cough, chicken pox, scarlet fever, influenza, smallpox, cholera, influenza, and poliomyelitis. In the following section three examples are given which can be called pandemics because they infected many people over broad areas and were associated with high mortality. These are:

- the Plague in various forms;
- Spanish Influenza;
• HIV/AIDS, the Human Immunodeficiency Virus / Acquired Immune Deficiency Syndrome.

None were originally endemic. They all resulted from the mutation of pathogens from previously milder or less infectious diseases or from the transfer of diseases from other species, and most have or might become extinct.

THE PLAGUE

This section focuses on major pandemics such as the Plague of Justinian, the Black Death, the Great Plague of 1665, and modern or Oriental bubonic plague. These pandemics used to be thought to be identical, but this is now very doubtful (cf. Herlihy 1997; Cohn 1997, 2002, 2003). Oriental bubonic plague emerged from southern China in 1894, and attacked Hong Kong, then Bombay, and other places including Sydney. It is not yet extinct. In Hong Kong it was shown to be caused by a bacillus (Yersinia pestis) and in Bombay (Mumbai) it was proved that this was transmitted by a rat flea.

Because it was characterized by buboes, or pronounced swellings of the lymph glands in the groin, it has long been assumed that Oriental Bubonic Plague was identical with the Black Death and the Great Plague. So it was believed that by studying this pandemic modern science could unlock the secrets of these earlier pandemics. This error was compounded in what Cohn (2002: 7–24) calls scientists squaring the circle. Modern experts on topical diseases and medical historians such as Le Roy Ladurie (1981) drew attention to the buboes in the groin, forgetting that the Black Death and the Plague had been characterized also by pustules on the upper body, and they seized on rare references to dead rats in the earlier epidemics to identify all these epidemics. They ignored these facts:

• The Black Death and Plague moved rapidly, as the ill infected others, while the Oriental Bubonic Plague moved only as slowly as the rats.

• The mortality rate of the Black Death was very high compared with only 3 per cent in the modern bubonic plague.

• Modern disease had occasionally become pneumonic (capable of being transmitted from person to person by the breath) but only in very unusual ecological conditions in parts of Manchuria in 1911 and 1922 (Cohn 2002: 22–23).

• Survivors of the Black Death and Plague built up strong resistance against further infection from the disease while this was not true of Oriental Bubonic Plague.

• Oriental Bubonic Plague was also characterized by buboes, but many diseases cause swelling of the lymph glands, and such a symptom is too weak evidence to relate this epidemic to the earlier ones.
In contrast the 14th century Black Death in Europe is almost certainly the forerunner of the Great Plague which attacked London in 1665 and had continuing outbursts until at least the outbreak in Moscow which killed 100,000 people in the 1730s. What seems to distinguish the Black Death from the Plague over the next 400 years was its much higher case mortality rate but the explanation seems to have been a growing resistance in the population of Western Europe to the disease.

The Black Death appears to have originated in Central Asia (more specifically in modern Turkmenistan) where its pathogen was usually restricted to small mammals. This situation was disturbed by growing trade and urbanization along the Silk Route between Europe and China, so that fleas carrying the pathogen infected rats and then people travelling the route. Once it reached the Mediterranean it spread rapidly because infected rats boarded ships travelling around the European coast. From 1347 to 1352 it invaded Europe, wiping out according to different accounts between one-quarter and two-thirds of the population (for convenience cautious most modern historians have provisionally agreed upon one-third). In the towns at least two-thirds died. Its passing owed even more to it thinning the ranks of the rats than of people. Europe’s population in the early 14th century was probably less than 100 million (compared with over 700 million now), and perhaps 30 million of them died when the Black Death struck. Every new visitation of plague over the next four centuries killed in Western Europe a smaller proportion of the population.

SPANISH 'FLU

Spanish influenza (usually just Spanish ‘flu) acquired its name because Spain was the first heavily infected European country, but it almost certainly originated elsewhere, possibly in Asia. Its pathogen could not at the time be identified, but efforts are still being made to secure DNA for identification from its long-dead victims. It was certainly a virus. The epidemic burst on Europe in 1918 and spread to the whole world, a spread partly facilitated by troops returning home.

Spanish influenza reflected the existence of modern transport by soon becoming a global epidemic. It probably killed about 30 million people (estimates vary from 20 to 50 million) of whom six million were in India. It is estimated that it infected around 950 million people (or half the population of the world at that time) but had a low case mortality rate: globally over 3% but varying from 2.5% in developed countries to 5% in India. Thus it killed around 2% cent of the world’s population. It came in three waves, with the second and third waves causing most deaths as if the pathogen was gaining greater virulence. Unlike other forms of influenza, its most common victims were not the aged or children but young adults, 20–40 years of age.

HIV / AIDS

Symptoms of a new disease began to be noticed in the United States in 1979 and official confirmation of its existence and of deaths arising from it came in 1981 and later the same year in Europe. It was not until 1983 that the pathogen (a retrovirus) was identified and 1985 before a test for its presence was available.

The disease has certain unusual characteristics. It kills by suppressing the immune system, thus permitting death from pneumonia, tuberculosis and other causes. Its average period from infection to death was ten years and to major symptoms almost
as long, compared with a few days in the case where the Black Death or Spanish Influenza led to death. Treatment with antiretrovirals can now lengthen that period although perhaps not indefinitely. Until the development of antiretrovirals, case mortality was 100% and that may still be the level of premature death. Unlike Spanish Influenza and probably the Black Death, HIV is not transmitted from person to person through the breath, but through blood by sexual intercourse, shared use of intravenous drug needles, during birth, or in maternal breastfeeding. Where heterosexual intercourse is an important means of infection, as in sub-Saharan Africa or the Caribbean more females than males may be infected, but where homosexual transmission predominates as in North America, Western Europe and Australasia, only 20–25% of those infected are females.

By the year 2000 HIV/AIDS had killed 20 million people (15 million of them in sub-Saharan Africa) and had infected 40 million others, condemning the great majority to premature death. In a strip of countries down East Africa from Ethiopia to South Africa, half the infected people in the world are to be found among only 4% of the world’s population. Here, those people that do not practice male circumcision exhibit much higher HIV rates than do societies where the practice is not followed. Everywhere commercial sex workers have unusually high levels of infection and are themselves a serious source of infection.

As yet no cure or effective method of immunization has been discovered. Some countries, especially in Africa, are attempting to control the epidemic by adopting an abc approach:

a = abstain from sexual relations;
b = be careful with whom you have sex (i.e. limit the number of your sexual partners, preferably to one who in turn has no other partners, and defer your sexual debut);
c = use condoms.

At first there was no sign that HIV levels in Africa would fall (compared with, for instance, the Black Death or the Plague where rates fell after a few weeks). Now declines have begun in some countries, most steeply in Uganda, where, in the capital, Kampala, HIV levels peaked among the adult population (defined as those 15+ years of age) at 30% and are now around 11%. In contrast, levels in Southern Africa are everywhere above 20% with no decline anywhere.

A search for HIV/AIDS on the Web will result in hundreds of thousands of hits. A list of useful Web addresses is given at Appendix 3A.1.

**EPIDEMICS AND DEMOGRAPHY**

Epidemics hastened the development of modern demography, largely because their impact was so great that the need to count deaths became obvious. Reasonably successful attempts were made in some of the early Renaissance cities of Italy to record Black Death mortality in the fourteenth century and these records have been increasingly used by historical demographers. London listed deaths in bills of mortality during the seventeenth century plague. These were employed by John Graunt to publish the first text of demographic analysis, including life tables, in 1662. The Spanish Influenza epidemic prompted the League of Nations to establish a
Global Surveillance System and influenza and other epidemics diseases led to the World Health Organization promoting National Virological Centres.

The mortality levels of the great epidemics are important demographic phenomena and their magnitude is shown in Table 1. The table shows that, as measured by deaths, HIV/AIDS which would account for 60 million deaths even if no one else was ever infected has killed or will kill at least twice as many people as the previous great epidemics. Unless there are major biomedical advances, the AIDS epidemic may well ultimately be responsible for 100–150 million deaths or even more. Nevertheless, in terms of the proportion dying, even in East and Southern Africa it will not equal the Black Death in Europe. That is because the world’s population is now twenty times as great as it was in the fourteenth century. Spanish Influenza and the Black Death were similar in the proportion of the population infected, but in contrast to the Black Death and HIV/AIDS it had a much lower case mortality rate.

Table 3A.1 Estimated mortality from three Plagues

<table>
<thead>
<tr>
<th>Plague/Period</th>
<th>Area</th>
<th>Infected population (millions)</th>
<th>Case mortality rate</th>
<th>Deaths or expected deaths (millions)</th>
<th>% of area's population died / or deaths anticipated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Death</td>
<td>Europe</td>
<td>25</td>
<td>80%c</td>
<td>20 (12–45)a</td>
<td>33.0%</td>
</tr>
<tr>
<td>1457–62</td>
<td>World</td>
<td>30b</td>
<td>80%c</td>
<td>24m</td>
<td>8.0%</td>
</tr>
<tr>
<td>Spanish 'Flu</td>
<td>World</td>
<td>950</td>
<td>3%</td>
<td>30 (20–50)a</td>
<td>1.6%</td>
</tr>
<tr>
<td>1918–19</td>
<td>World</td>
<td>60</td>
<td>100%</td>
<td>60</td>
<td>1.0%</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>East and South Africa</td>
<td>30</td>
<td>100%</td>
<td>30</td>
<td>13.3%</td>
</tr>
</tbody>
</table>

Notes:  
*a* Range of estimates.  
*b* Author’s estimate, based on evidence that no great Asian epidemic occurred that time.  
*c* Authors’ estimate based on postulates in Cohn, Jr. 2003.

Epidemics are so important that they have led the statistical study of morbidity and mortality to be called ‘epidemiology’. They are also important for demographic theory and raise three important questions:

(a) To what extent can the partial conquest of epidemics (or, more broadly, of the great mortality crises) explain the modern decline in mortality?  
(b) Are the great epidemics explained by Malthusian theory?  
(c) Do they have a permanent impact on population numbers or do various demographic compensating mechanisms come into play?
(a) The IUSSP turned the interests of one of its scientific committees to the issue of 'The great mortalities: demographic crises in the past', and produced a book on the needed methodologies for studying their magnitudes (Charbonneau and Larose 1979). Yet none of the authors ventured to address the three epidemics described above. In his introduction to that book, Hollingsworth (1979: 17–18) argued that population crises in the past must have kept populations in equilibrium. He noted that a single famine can kill as many people as the worst epidemic, as shown by the Great Bengal Famine of 1869–70 causing the death of one-third of Bengal's population (a figure comparable with the Black Death in Europe). The longest series of vital rates available to us, those of Wrigley and Schofield (1981) span the period 1541–1871 and so do not include any of our major three pandemics. Their analysis of those 330 years is mostly in terms of mortality crises with only passing attempts to distinguish epidemics from famines. However it is clear that epidemics — the Plague, small pox and cholera — played an important role in the exceptionally severe mortality crises in England in 1650–1680 and 1725–1750 and did raise the general death rate for these periods. Johansson (2003) assesses the evidence to show that epidemics caused more deaths than famines. Life expectancy actually fell slightly for the half century after 1680 but rose modestly after 1750. There is no theoretical reason why the passing of those mortality peaks caused by epidemics should lower the long-term death rate. It may merely be that, with continuing population growth especially in urban areas, epidemic diseases become endemic ones. Admittedly that change may cause general resistance to the disease to remain higher. In contrast, the reduction of famine mortality peaks almost lowers long-term mortality, although even here there is a possibility that long-term nutrition is not improved. But persistent mortality decline, when it arrived in the 19th century had little to do with the declining incidence of the great mortality and everything to do with a reduction in the base level of mortality.

(b) Malthus (1959:49) believed that much of the control of population numbers was achieved by 'sickly seasons, epidemics, pestilence and plague', so that famine was unnecessary and forestalled. But the epidemics were less likely if the population was not forced to subsist on 'unwholesome food [probably including insufficient food]' (Malthus 1959:40). That is malnutrition triggered both epidemics and starvation but rising disease mortality usually came first or alone.

The three pandemics studied here give only limited support to the Malthusian thesis. They give no support to the idea that periods of unusual malnutrition give rise to epidemics. Both Herlihy (1997) and Cohn (2002) stress that mid-fourteenth-century Europe, when the Black Death struck, was more prosperous and better fed than it had been for centuries. Spanish influenza did not emerge in a world that was unusually underfed, but its progress may have been catalysed by the living conditions of the armies on the Western Front and troop movements both before and after the end of the First World War. AIDS appeared in sub-Saharan Africa when economic growth had begun to slow but there is no evidence that malnutrition was rising or that the regions or the social classes most affected were the least nourished.
Finally, there is the question of how much mortality crises, and particularly epidemics, actually limit population growth. The answer may be different in contemporary society (19th – 21st centuries) with rapid population growth than in pre-modern society or the anticipated post-modern society with zero or negative population growth. Watkins and Menken (1985) examined famines of the 19th and 20th centuries and concluded that their demographic impact was relatively unimportant, partly because of the underlying positive rate of population growth but largely because of re-stabilising mechanisms such as subsequent lower death rates, higher marriage rates and higher birth rates. It is reasonable to argue that mortality crises lead to the premature deaths of weak or ailing persons who would soon have died anyway. Certainly mortality graphs show post-crisis death rates below long-term death rates, but this is partly a statistical artefact in that the long-term death rate is merely an average of the highs and lows. Certainly, in the best measured recent mortality crisis, the Chinese famine of 1959–61 that ended the Great Leap Forward, their description is apt. The subsequent age structure appeared to show about 50 million people missing, 20 million surplus deaths and 30 million fewer births. Half the missing births were explained by deferred marriage, and half by fewer conceptions arising either from sexual relations foregone or practised more carefully. There are good financial reasons why marriages should be postponed during famines (on India, see Caldwell et al. 1986). Marriage behaviour in China was not as tightly related to the timing of the famine as it was to the Great Leap Forward which must itself have disrupted weddings. Marriage rates fell by 20% between 1956 and 1959 and then rose 61% 1959 to 1962. The total fertility rate almost halved between 1957 and 1961 from 6.4 to 3.3 and then rose to 7.5 by 1963 (Caldwell and Srinivasan 1984).

This situation did not, however, prevail during pre-modern times because of near stationary population because of high mortality approximately equalling high fertility. Nor will it be the case in the world that we are just entering when fertility is as low or lower than mortality. According to Herlihy (1997: 33) the huge Black Death population losses were not made up at all before 1360 or 1370 and were not wholly made up until 1550, two centuries after the epidemic. Part of the explanation lies outside demography. One explanation is that the loss of life caused the farming structure to change so that it was less labour-intensive, and could not cope with rapid population growth. Spanish influenza occurred right in the middle of the period studied by Watkins and Menken (1985) and globally fitted in with their thesis. This was in the West partly because there was a post-war marriage and baby boom at the same time. Nevertheless, in Australia the annual number of deaths rose by 37% between 1917 and 1919, reaching a number not attained again for 20 years (when the base population was much higher) and natural increase fell by 28% (McDonald et al., 1987). In India, where around six million died, the intercensal population growth, which had been 14 million in 1901 to 1911, exhibited a decline of one million in 1911–21, and a rise of 28 million in 1921–31. In those three decades the life expectancy at birth in India was 22.6, 18.9 and 26.9 years. The impact of the influenza epidemic was horrendous, but the population deficit was probably made up in four or five years.

The AIDS situation in most of the developing world fits the Watkins-Menken thesis, but this is not the case in Southern Africa, where fertility transition was underway before the greatest intensity of the epidemic. According to the United
Nations (2003) 2002 statistics and medium projection South Africa’s population in 2005 (with a total fertility rate of 2.5) will peak and will never recover, with its 2050 population being 11% lower and falling. Estimates for the rest of Southern Africa are similar.

PERSPECTIVE

Epidemics should be a matter of central interest for demographers. For most of the past and probably for the indefinite future they can be regarded as a major moulder of population numbers. For a brief period of unusual population growth, perhaps after 1850 in the developed world and after 1950 in the developing world population growth was so rapid that even the great mortality crises eroded human numbers by only a few years’ growth. These times are passing. By 2075 global population growth will have come to a halt (United Nations 2004) and by 2100 this could be true for most countries. In these circumstances mortality crises could have a marked impact, lowering population numbers permanently. In the organized world of the future these crises are unlikely to be famines, but could well be epidemics. HIV/AIDS has shown us that in a world even more populous than our present one, and probably with even greater movement, new infectious disease pathogens can evolve and spread around the world. Some of them may take decades or generations to control. It is ironic, but perhaps a related phenomenon, that, as biomedicine becomes more sophisticated, so do the pathogens.

REFERENCES AND FURTHER READING


## APPENDIX 3A.1 WWW addresses of major HIV/AIDS sites

<table>
<thead>
<tr>
<th>Website</th>
<th>Maintained by</th>
<th>Types of issues covered</th>
</tr>
</thead>
</table>