Epidemics

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June 2019

Demography helps us understand the history of epidemics and pandemics. Likewise 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.

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. In the strict sense of the term, an epidemic involves the upswing of infectious or contagious disease with a subsequent decline so that there is a peak in the number of infected people, before a fall to lower or even zero levels. With the move to primitive pastoralism came the regular close contact between animals and humans, providing pathogens with a fertile environment for reproduction, mutation and transmission. Hippocrates (460-377 BC) wrote about diseases that we can identify today as malaria, mumps, diphtheria, tuberculosis and possibly influenza. The advent of urban aggregation and the establishment of travel and trade networks provided the required number of susceptible individuals needed to sustain bacterial and viral infections, as a result common diseases began to appear at regular intervals. Many of these ‘common disease’ were of *zoonotic* origin, meaning they originated in animals.

The vocabulary employed is descriptive but not very analytical. Nearly all terms come from Greek and are built around ‘demos' the Greek word for people, from which demography is derived: examples are endemic (‘en’ means ‘in’ + ‘demos’ = ‘in the people’), epidemic (‘epi’ means ‘upon’ + ‘demos’ = ‘upon the people), and pandemic (‘pan’ means ‘all’ + ’demos’ = all the people).

Epidemic Theory

In order to fully understand the relationship between demography and epidemics it is important to become familiar with epidemic theory. For a major epidemic to occur there are a number of criteria that need to be met:

a) The pathogen should have a *reproductive ratio*, R_0, greater than 1. The R_0 of an infectious pathogen refers to the average number of cases generated by a single infected individual during their *infectious period*, given that the population is susceptible. For instance, if the R_0 for measles in a population is 15, then in a susceptible population we would expect that each new case of measles would infect another 15 people.
b) The R_0 is influenced by the rate of contact between the infected individual and susceptible individuals, the *pathogenicity* of the pathogen, *transmission route* and the duration of *infectivity (Nelson and Williams, 2014)*. Some epidemics can be 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. (Bartlett, 1960)
c) A population's *susceptibility to infection* is determined by the proportion of the population that are vulnerable to infection. Initially, a small number of infected people can be enough to trigger an epidemic. However, for a major epidemic to occur the population size needs to be large enough to sustain continued transmission to susceptible individuals. As an epidemic continues so the susceptible population decreases; infected individuals either die or recover. Recovery can result in temporary or life-long immunity reducing the pool of susceptible individuals, and thereby breaking the *chain of transmission*. The susceptibility of a population to infection can be influenced by many factors including age, baseline health, immune response and prior exposure, and vaccination status. Environmental characteristics such as population density, social structure, physical structures, human behavior and climate are also important predictors for major epidemics.

The presence of a disease at a fairly constant level in a population is described as an *endemic* disease. Endemic diseases are characterized by a particular population, environment or region. Endemic diseases may experience seasonal fluctuations in prevalence, however these increases in prevalence are not considered an 'outbreak' unless the number of new cases is above what is expected. Rises in the number of cases of an endemic disease are usually accompanied by a fall as the number of susceptible individuals falls (those most

likely to be infected have died or the survivors have built up resistance to the disease; among those infected, many may have developed a natural resistance). Vector-transmitted diseases such as malaria, dengue fever, and Japanese encephalitis are dependent on mosquito vectors for their transmission. Therefore, season peaks are expected in the wet season where these diseases are endemic, as the climatic and environmental conditions during this period are favorable for mosquito reproduction.

The size of the host population adequate to sustain new infections varies inversely with the transmissibility of pathogens but directly with its *virulence*. The more virulent a pathogen the larger the population that is required to sustain transmission. A less virulent pathogen is able to survive in a smaller population for a longer time frame. There are many infectious diseases which are endemic in populations and have potential to become an epidemic: measles, whooping cough, chicken pox, influenza and cholera.

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 mortality due to the bubonic plague 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. In 1851 the International Sanitary Conferences attempted to bring countries together to coordinate infectious disease control, with limited success. The Spanish influenza epidemic prompted the League of Nations (later to become the World Health Organization in 1948) to form in 1919, the world's first global political system.

While progress in infectious disease prevention, control, and treatment has improved our ability to respond to epidemics, globalization processes relating to human behaviour, demographics, and mobility have increased the threat of pandemic emergence and accelerated global disease transmission. In the following section we describe pandemics caused by three highly virulent pathogens which have had a significant impact on population demography; the plague, influenza and human immunodeficiency virus (HIV). These pandemics all resulted from the mutation of pathogens from previously milder or less infectious diseases or from the transmission of a pathogen from animals to humans.

The impact of a pandemic is dependent on its intensity, duration and the size of the affected population. With these factors in mind we must also be cautious in interpreting events by distinguishing between the local and global impact.

Mortality may be high in raw numbers but expressed as a percentage of the affected population may not be high enough to have a significant impact on economy or demography. The mortality estimates provided below should be interpreted with caution due to the limitations associated with historical demography data sources, which we do not expand on here as they have been addressed in Day (2018).

Epidemics and colonialism

This risk of transmission of infectious disease increases where large numbers of people live in close proximity, and is much higher in those who have not previously been exposed to the pathogen. As the European explorers travelled previously uncharted waters, discovering new lands and peoples, they bought with them European diseases such as small pox, measles, influenza, diphtheria, malaria and typhus. The introduction of these diseases into populations who had no prior exposure had a devastating outcome. Historical estimates of the Aztec population of Mexico are 25 million, at the time of the arrival of the Spanish conquistadors, within 100 years this had plummeted to under 1 million, the cause of the rapid decline has been attributed to multiple epidemics (Bianchine and Russo, 1992; Callaway, 2017). An epidemic of small pox was introduced to the island of Hispanioloa at Santa Domingo in 1548 and is believed to have reduced the population from approximately one million Indians to only 500 (Bianchine and Russo, 1992). Populations of the Pacific islands suffered a similar fate, with high mortality from infectious diseases introduced by European explorers (Shanks, 2016). Cliff and Hagget (1985) report that over a six month period in 1875 a measles outbreak led to 40 000 deaths in Fiji, which at the time had a population of 150 000. The infection was brought to the island by the Fijian Chief, who had recently returned from a state visit to Australia.

Plague

This section focuses on major plague pandemics; 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 doubtful (cf.Herlihy 1997; Cohn 1997, 2002, 2003). The host of the plague pathogen (*Yersinia pestis*) is the black rat, although the rock squirrel, prairie dog and gerbil can also act as hosts. Typically, a human is infected through a flea vector. A person becomes infected when the flea first bites a rat infected with the plague pathogen and then bites the person; transmission can also occur through direct contact with rats. In the case of pneumonic plague, transmission can spread from person-to-person through droplets in the air, and therefore is the most contagious and dangerous form of plague. The combination of increasing urbanisation with people living in close proximity, in over-crowded living quarters with poor sanitation and poor access to clean water contributed to higher

transmission and mortality in urban centres than in rural villages (Ziegler, 1997). Table 1 provides an overview of estimated mortality from past plague epidemics.

Table 1: Estimated mortality of historical plague pandemics

Disease	Area	Deaths reported	Estimated % of affected population that died
Plague of Justinian 6th Century CE	Middle East, Mediterranean (originated in Egypt)	50 million	15%
Black Death 1346-1352	Europe (originated in Central Asia)	20 million	33%
	World	24 million	8%
Oriental bubonic plague 1855-1959	World (originated in China)	15 million	5-15%

The Plague of Justinian is the earliest documented pandemic of plague and occurred in the sixth century CE during the reign of the Roman emperor Justinian. Modern historians have credited the social and economic consequences of the pandemic with the fall of the Roman Empire; although the scale of impact is refuted in more recent literature (Meier, 2016). The pandemic is believed to have commenced in Lower Egypt in the summer of 541 and spread east to Palestine reaching Constantinople in late 541 while simultaneously infecting the port cities of the Mediterranean. It is believed to have spread as far east as Persia and as far north as the British Isles. Historical recounts of this pandemic make it sound apocalyptic, with reports of mass graves and death rates in excess of 10 000/day, although these have been met with scepticism in recent times (McCormick, 2015a; Meier, 2016). Feldman et al. (2016) describe the excavation of a 6th century cemetery in Aschheim, Germany where bones were found to contain traces of the bacillus *Yersinia pestis* DNA. Using skeletal evidence archaeologists estimate that this rural village lost 35-53 percent of its population within a few months to plague (McCormick, 2015a, 2015b).

The Black Death appears to have originated in Central Asia (more specifically in modern Turkmenistan) where its pathogen was usually restricted to small mammals. Transmission to humans was propagated by growing trade and urbanization along the Silk Route between Europe and China, so that fleas carrying the pathogen infected rats and then susceptible people travelling the route. Once it reached the Mediterranean it spread rapidly as infected rats' boarded ships travelling around the European coast (DeWitte, 2010). The

pathogen was highly virulent, from 1343 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 Italy the epidemic became known as *mortalega grande*, 'the great mortality' when more than half of the population of Florence succumbed to the disease (Frith, 2012). The Black Death is estimated to have killed 60% of Asia's urban population and 30% of the population in the Middle East (Frith, 2012). During this pandemic villages would often be abandoned, whole families were wiped out, crops left unharvested and travel and trade reduced. The labour shortages that followed are reported to have been the driver behind the formation of the 'middle classes' (Herlihy, 1997). This pandemic was followed by another European plague epidemic in 1361, *pestis secunda,* which is reported to have killed a further 10-20% of Europe's population. During this time Europe also succumbed to plagues of smallpox, infantile diarrhoea and dysentery which resulted in high mortality. These repeat epidemics caused Europe's population to be lower in 1430 than it had been in 1290, it would not be until the 16th century that the population would once again reach pre-pandemic levels (Frith, 2012).

Plague epidemics were spaced throughout history with large pandemics occurring when the population of susceptible individuals had increased enough to sustain ongoing transmission. A review of mortality data in London during the plague epidemics of 1563, 1603, 1625 and 1665 showed that each of the epidemics was of roughly the same magnitude with an estimated one fifth of the city's population dying within the space of a few months (Cummins, Morgan, and Ó Gráda, 2016; Herlihy, 1997).

The third great plague pandemic, the Oriental bubonic plague, emerged from southern China in 1894, spread to Hong Kong, India and by 1900 had reached ports on every continent, by way of infected rats travelling on the steamships following the international trade routes. It was during this time that Y. *pestis* was first isolated by a French Pastoien bacteriologist, Alexandre Yerson. The plague arrived in Sydney in 1900 with the first outbreak in Sydney resulting in 100 deaths. Twelve more outbreaks occurred in Sydney between 1900 and1925, resulting in a further 1215 cases and 467 deaths (Frith, 2012). The low case numbers are credited with advances in scientific knowledge that led to strict isolation and quarantine of cases and contacts limiting further transmission. It is estimated that by this time it had caused 15 million deaths, with India bearing the largest burden. Plague remained endemic in many countries with repeated epidemics persisting until 1959, effective treatment with antiserum in the late 1800's, sulphonamides in the 1930s and streptomoycin in 1947 resulted in decreased mortality and transmission (Butler, 2014). Today epidemics are localized in areas of Africa, Asia and South America (Frith, 2012).

Influenza

Throughout history influenza pandemics have caused significant mortality, morbidity and disruption to society. The nature of the influenza viruses is that they continually mutate, creating novel viruses and thus ensuring the population remains susceptible to infection. The viruses are also highly transmissible via person-to-person contact and therefore spreads very quickly in a susceptible population. Epidemics and pandemics have been occurring for centuries, however the virus was not isolated until the twentieth century, when in 1931 Richard Shope isolated the virus in poultry.

In recent history we have seen four major pandemics from novel influenza viruses for which humans had little or no immunity, The 1918 Spanish Flu, the 1957 Asian Flu, the 1968 Hong Kong Flu and the 2009 Swine Flu. Table 2 provides an overview of estimated mortality from pandemics.

Table 2: Estimated mortality of historical influenza pandemics

Disease	Area	Deaths reported	Estimated % of affected population that died
Spanish Flu 1918-1920	World (originated in China)	40-50 million	3-5%
Asian Flu 1957-1958	World (originated in China)	1-2 million	0.1-0.28%
Hong Kong Flu 1968-1970	World (originated in China)	500 000 – 2 million	0.5%
Swine Flu 2009-2010	World (originated in Mexico)	Up to 575 000	0.02%

While there is much rhetoric around the ‘Spanish Flu’ originating in Spain, this has been defunct in recent history. It is likely that due to the freedom of the press in Spain at the time, and Spain’s neutrality in World War 1, that the Spanish press were not restricted from printing reports on the devastation the country was facing. While simultaneous outbreaks were occurring in Britain and the America’s the reluctance to report the high mortality among soldiers led to misinterpretation of the source of the epidemic and it is still known today as the ‘Spanish Flu Pandemic’(Trilla, Trilla, and Daer, 2008). This pandemic was believed to have been driven by the transport of soldiers and laborers in support of the allied war effort and sustained due to the vulnerability of the population it infected. Poor sanitation, overcrowding and limited capacity in public health and medicine all drove the pandemic.

Spanish influenza killed between 40-50 million people 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 the time) but had a low case fatality rate. It came in three waves, with the second and third waves causing most deaths as if the pathogen had gained 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 (Zimmer and Burke, 2009). Poor sanitation, overcrowding and the limited capacity of public health and medicine to prevent transmission drove this pandemic to become a catastrophic event in the history of epidemics.

It would be 40 years until the next global flu pandemic, the 1957-1958 Asian Flu pandemic, believed to have originated in China. In the years since the 'Spanish Flu' the world had changed. The formation of the World Bank, International Monetary Fund, and the General Agreement on Tariffs and Trade had resulted in unprecedented international trade and liberalization. The international movement of goods and people was accompanied by population growth, increased urbanization and advances in public health and medicine. Laws against overcrowding were introduced which coincided with increased investment in sanitation and improved access to clean water. While the pandemic spread rapidly throughout the world, case fatality rate was much lower than that of 'Spanish Flu'. The decrease in mortality no doubt influenced by improvements in population health has been credited with a less virulent strain of the influenza virus than the strain responsible for the 1918 Spanish Flu pandemic. However, this has been debated in more recent years with the mortality during the 1918 pandemic attributed to secondary infection of bacterial pneumonia (Morens, Taubenberger, and Fauci, 2008).

The Hong Kong Flu pandemic, was next, hitting the world between 1968 and 1970. The expansion of air-travel propagated the spread of this highly transmissible strain of the influenza virus, however virulence was low with mortality estimated at between 500 000 and 2 million deaths globally.

These ssuccessive pandemics have decreased in severity over time. Estimates indicate that the Spanish Flu resulted in a global increase of all-cause mortality by 598 deaths per 100 000 people per year, the Asian Flu 40.6 excess mortality and Hong Kong Flu 16.9 (Morens, Taubenberger, and Fauci, 2009), is this due to a decrease in virulence of the causative pathogen or more a reflection of improved public health measures, the discovery of penicillin and subsequent treatment of bacterial pneumonia with antibiotics.

The 2009-2010 Swine Flu pandemic highlighted how quickly a highly transmissible virus can spread throughout the world. The world was better prepared for a pandemic of influenza, with stronger health systems, global surveillance systems, development of vaccines and knowledge of effective

prevention and control mechanisms. Even so the virus, which was believed to have emerged from Mexico in April of 2009, had spread to 30 countries within weeks. By July 2009 infection with the virus was reported in 122 countries; and by August 2010 cases had been reported in 214 countries and territories had confirmed cases (World Health Organization, 2010). The low virulence prevented this pandemic from having the devastating impact that the 1918 flu pandemic had on the world's population, the case fatality rate was 0.02% (Dawood, 2012; Saunders-Hastings and Krewski, 2016). The Swine Flu pandemic demonstrated how quickly a novel virus can spread across the globe. A pathogen with high virulence, transmissibility and infectivity could today have a rapid devastating impact on the world's population. This pandemic demonstrated the potential for a pathogen to travel the world in days rather than the months and years reported in past pandemics.

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, known as Human Immunodeficiency Virus (HIV) has certain unusual characteristics. It kills by suppressing the immune system and the average period from infection to death was ten years; and to major symptoms almost as long, compared with a few days in the case of the Black Death or Spanish Influenza. Until the development of antiretrovirals the case mortality rate was 100%. HIV is transmitted through blood during sexual intercourse, shared use of syringes, during birth and through breastfeeding. Where heterosexual intercourse drove the pandemic in Sub-Saharan Africa (the worst affected region of the world), homosexual transmission and the sharing of syringes during intravenous drug use propagated the epidemic in North America, Western Europe and Australasia.

The HIV/AIDS pandemic is one of the worst demographic shocks to affect the world in recent history. Since the first cases of HIV were reported over 78 million people have become infected with HIV and at the end of 2016 HIV/AIDs had killed 35 million people, 76% of which were in Sub-Saharan Africa (UNAIDS, 2017). The African continent had experienced significant reduction in infant mortality and increased life expectancy from the 1950s to the 1990s, however, life expectancy in Sub-Saharan Africa declined by 6 years in the period between 1990-1995 and 2005-2010 (Defo, 2014).

At the end of 2003 prevalence rates in adult populations were as high as 37% in Botswana, 24.6% in Zimbabwe and 21% in Namibia and South Africa

(UNAIDS, 2004). Life expectancy at birth had decreased between 1992 and 2002 from 61 years to 49 years in Botswana, from 61 years to 54 years in Namibia and from 61 years to 53 years in South Africa. In Asia, by contrast, the prevalence of HIV/AIDS was reported above 1% in only 3 countries; Cambodia, Myanmar and Thailand (UNAIDS, 2004). In the late 1990s and early 2000s there was an influx of studies modelling and forecasting the demographic impacts of the HIV/AIDS pandemic (Clark, 2006; Gregson et al., 2007; Whiteside, 2001). Gregson and his colleagues (1994) predicted that the HIV/AIDS epidemic would significantly alter the age-specific sex ratio, population age structure and overall population growth rates. With the HIV/AIDS pandemic disproportionately affecting young adults it was forecast that the pandemic will demonstrate a positive selective mortality, similar to that of the 1918 influenza pandemic. The age-specific impact of the HIV/AIDS pandemic was predicted to reshape the population structure of countries most severely affected. The increased mortality among young men and women was thought to have lasting impact by reducing the number of adults of reproductive age and thereby having an added impact of reducing fertility; depopulating tiers of the age pyramid (Ngom and Clark, 2003). Young (2005, 2007) argued that the pandemic had a negative impact on women's desire to have children thereby reducing fertility, whereas Kalemli-Ozcan (2012) and Fink and Linnemayr (2008), among others, report the net effect of the pandemic on fertility to be either positive or negligible. A study by Karlsson and Pichler (2015) demonstrated that the HIV/AIDS pandemic has had little overall effect on fertility.

The HIV associated increase in mortality burden of 1521/100 000 population additional deaths in Zimbabwe, 600/100 000 additional deaths in South Africa and 244/100 000 additional deaths in Mozambique demonstrates the increased mortality burden in these nations, however the long-term impact on population size is less clearly defined (Karlsson and Pichler, 2015). The period of the HIV/AIDS pandemic has coincided with improved access to health services, improvements in infant and maternal mortality, reduction in mortality from infectious disease and improvements in other social determinants such as female education. By mid-2017, 20.9 million people living with HIV were receiving treatment with antiretroviral therapy (UNAIDS, 2017), decreasing mortality caused by opportunistic infections. So while the forecasting on the impact of the pandemic on the population structure of African nations was pessimistic, it has proven to be overdramatic. While life expectancy in South Africa (a nation with one of the highest prevalence rates of HIV/AIDS) dropped by nearly 9 years between 1995 and 2005, in 2015 it had returned to pre-pandemic estimates and in 2018 continues to increase (Statistics South Africa, 2018; Wang, Mohsen, Allen, Barber, and Bhutta, 2016).

Where the HIV/AIDS pandemic has left its mark on populations is the large number of children who have been affected. The United Nations Children's Fund (UNICEF) report a 70% reduction in the number of newly infected children under the age of 15 years of age between 2000 and 2015, yet AIDS remains the leading cause of death in adolescents in Africa and the 2nd leading cause of death for adolescents globally (UNICEF, 2016). In 2018 an estimated 1.8 million children are reported to be living with HIV globally (UNAIDS, 2018). In addition it is estimated that 17.7 million children have been orphaned as a direct result of the HIV/AIDS pandemic(UNICEF, 2015). UNICEF defines an orphan as 'a child under the age of 18 years who has lost one or both parents to any cause of death' (UNICEF, 2018). In sub-Saharan Africa kinship networks, by way of the extended family, have acted as a social safety net for orphans. The increase in the number of children orphaned by AIDS in the context of extreme poverty lead to concerns and predictions that the number of child-headed households (CHHs) would increase as the pandemic spread. A CHH is defined as a household where the day-to-day management of the household is the responsibility of an individual less than 18 years of agev(UNAIDS, 2006). Meintjes et al. (2010) used the results of national household survey data in South Africa to show that between 2001 and 2007 there was no significant difference in the proportion of children living in CHH, even though the number of orphaned children had increased by 760 000 during the same period. In 2006 the proportion of CHH in South Africa was 0.47% with the 92% of children residing in a CHH having a living parent. This indicates that in the context of extreme poverty factors other than AIDS-related mortality may have played a role in forming such households.

Box 1: 2014-2016 Ebola Virus Disease West African pandemic

In 2014 West Africa was affected by the largest and deadliest outbreak of Ebola Virus Disease (EVD). The initial case was reported in December 2013 and following five additional cases a medical alert was issued on January 24th 2014 to local health officials. On March 23th 2014 the World Health Organization declared an outbreak with 49 confirmed cases and 29 deaths. By July 2014 the outbreak had spread to the capital city of Guinea, and had crossed the border to Liberia and Sierra Leone. During the following two and a half years the pandemic lead to 28 616 cases and 11 310 deaths in these three West African Countries and an additional 36 cases and 15 deaths in the seven additional affected countries (Centers for Disease Control and Prevention, 2018).

The spread of the pandemic has been attributed to the combination of the circulation of the virus in densely populated urban centers, increased mobilization of populations across international borders and the lack of engagement with local communities with respect to traditional cultural practices and beliefs that facilitated transmission (Centers for Disease Control and Prevention, 2018).

An important secondary impact of the EVD outbreak/response was the increase in all-cause mortality (3.4 times higher than expected). This was caused by the diversion of health workforce and facilities to focus on responding to the pandemic in addition to the loss of health workforce as they fell victim to the pandemic (Elston et al., 2015).

Due to the severity of symptoms and high case fatality rate (in excess of 50%), the West African EVD pandemic cause panic across the globe and has subsequently led to the outpouring of millions of dollars by governments to strengthen global health security. Yet in reality it is the risk of EVD becoming a global pandemic is extremely low. The R_0 of EVD is 1.2 which means it is not as highly transmissible as infectious diseases like measles (with an R_0 of 15). Transmission of EVD is via close contact with body fluids, and in the West African pandemic transmission was propagated through traditional practices involving close contact with the diseased. In combination with understaffed and undersupplied health systems, the disease spread through these three countries. Yet Neighboring countries, such as Nigeria, were able to quickly circumvent further transmission when imported cases were identified (Althaus, Low, Musa, Shuaib, and Gsteiger, 2015).

What the West African pandemic did highlight was the cost of a slow response to a novel pathogen surfacing in a context with poor health systems and cultural practices that facilitate transmission. The World Health Organizations delayed recognition of seriousness of the outbreak and the delayed and chaotic international response led to the loss of over 11 000 lives with direct costs in excess of 6 billion US dollars and global losses estimated at over 15 billion dollars (Ross, Crowe, and Tyndall, 2015).

Demographic Theory

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 epidemics have a permanent impact on population numbers or do various demographic compensating mechanisms come into play?

a) Hollingsworth (1979) 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), or the catastrophic famine in China in the 1950s that saw the loss of 30 million people. 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 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, simultaneously lowering life expectancy. 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 as continual exposure increases resistance in the population and decreases the number of susceptible individuals. An alternate explanation is that the lowering of long-term death rates coincided with increased understanding of disease causation and control measures, improvements in public health practices, the introduction of quarantine and isolation measures, improved sanitation and access to clean water, vermin control and improvements in medical treatment. 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. A recent analysis of global

mortality data from 1980-2015 by Wang et al. (2016) demonstrated that global gains in life expectancy for this period were gradual, and attributed the impact of the Rwandan genocide, North Korean famines and increased mortality due to HIV/AIDS on global longevity. Hollingsworth's argument that epidemics are a form of population control, has not been demonstrated in the long-term.

b) Malthus (1959) 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). He believed the constant effort by human beings to increase the population was met by exceeding the resources to sustain the population increase, leading to distress in the population where the poor were required to live on less. That is malnutrition triggered both epidemics and starvation but rising disease mortality usually came first or alone. The 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.

c) 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 more 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 by 7.5 by 1963.

This situation did not, however, prevail during pre-modern times because of near stationary population or 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) 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, Ruzicka, and Pyne, 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 HIV/AIDS pandemic 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 pandemic. The post 1995 reversal of mortality trends and declines in life expectancy at birth in Sub Saharan Africa challenge demographic transition theory and inferred linkages between mortality, fertility and population growth (Defo, 2014; Omran, 1998).

Future Projections

The emergence of novel infections in the past 50 years such as HIV/AIDS, Severe Acute Respiratory Syndrome (SARS) in 2003, Bird flu in 2007, Swine flu in 2009, Middle Eastern Respiratory Syndrome (MERS) in 2012 and Zika virus in 2015 demonstrate that even with an exponential growth in medical advances, improvements in population health and life expectancy and a mortality transition away from infectious diseases the world's population remain vulnerable to the emergence of novel pandemic agents which have the potential to cause levels of devastation matching that of the 14th Century plague or 1918 influenza pandemic (Ross et al., 2015). Since 1940, over 400 of the emerging infectious diseases that have been identified have are zoonotic, that is have originated in animals.

Global travel and trade networks have created very porous international borders. In addition population growth, changes in population density have driven anthropogenic changes which have led to changes in land use and encroachment into wildlife habitats (Morse et al., 2012). The West African Ebola Virus Disease pandemic is an example of where all these factors acted together and combined with weak health systems and traditional practices that drove transmission led to a devastating outcome for Guinea, Liberia and Sierra Leone.

Morse (Morse et al., 2012) and colleagues describe three stages in disease emergence. The first stage is pre-emergence where the pathogen is in its natural reservoir but a change in land use or encroachment into wildlife habitats alters pathogen transmission. The second stage, Localised emergence, occurs when there is 'spillover' of the pathogen from animals into humans (as was the case with HIV/AIDS and Ebola Virus Disease). The third stage is that of the full pandemic emergence, this requires sustained person-to-person transmission and large scale spread. Governments are dedicating resources toward pandemic emergency preparedness. The international community is instilling pressure on countries to meet the International Health Regulations (2005) of which an important component is the requirement to report incidents of international concern. However, we still have a long way to go to ensure global health systems are able to cope with the next pandemic.

Summation

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. Currently the global

population is estimated to be 7.2 billion and predicted to grow to 9.6 billion by 2050 and 10.9 billion by 2100 (Gerland et al., 2014). In the organized world future catastrophic mortality events are unlikely to be famines, but could well be epidemics. As human beings change the way they interact with their environment the emergence of new diseases is inevitable. HIV/AIDS, the 2009-10 Swine Flu pandemic and the 2014 Ebola West Africa epidemic have shown us that in a world even more populous than our present one, with even greater movement, new infectious disease pathogens can evolve and rapidly spread across countries and around the world. It is ironic, but perhaps a related phenomenon, that, as biomedicine becomes more sophisticated, so do the pathogens.

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