Contents lists available at ScienceDirect

# Infectious Disease Modelling

journal homepage: www.keaipublishing.com/idm

# Mathematical epidemiology: Past, present, and future

# Fred Brauer

University of British Columbia, Vancouver, BC, Canada

### ARTICLE INFO

Article history: Received 13 November 2016 Received in revised form 1 February 2017 Accepted 2 February 2017 Available online 4 February 2017

## ABSTRACT

We give a brief outline of some of the important aspects of the development of mathematical epidemiology.

© 2017 KeAi Communications Co., Ltd. Production and hosting by Elsevier B.V. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

## 1. Introduction

Communicable diseases have always been an important part of human history. Since the beginning of recorded history there have been epidemics that have invaded populations, often causing many deaths before disappearing, possibly recurring years later, possibly diminishing in severity as populations develop some immunity. For example, the "Spanish" flu epidemic of 1918–19 caused more than 50,000,000 deaths worldwide, and there are annual influenza seasonal epidemics that cause up to 35,000 deaths worldwide.

The Black Deaths (probably bubonic plague) spread from Asia throughout Europe in several waves during the fourteenth century, beginning in 1346, and is estimated to have caused the death of as much as one-third of the population of Europe between 1346 and 1350. The disease recurred regularly in various parts of Europe for more than 300 years, notably as the Great Plague of London of 1665–1666. It then gradually withdrew from Europe.

There are also diseases that have become endemic (always present) in some populations and cause many deaths. This is especially common in developing countries with poor health care systems. Every year millions of people die of measles, respiratory infections, diarrhea, and other diseases that are easily treated and not considered dangerous in the Western world. Diseases such as malaria, typhus, cholera, schistosomiasis, and sleeping sickness are endemic in many parts of the world. The effects of high disease mortality on mean life span and of disease debilitation and mortality on the economy in afflicted countries are considerable. The World Health Organization has estimated that in 2011 there were 1,400,000 deaths due to tuberculosis, 1,200,000 deaths due to HIV/AIDS, and 627,000 deaths due to malaria (but other sources estimate the number of malaria deaths to have been more than 1,000,000). In 1980 there were 2,600,000 deaths due to measles, but the number of measles deaths in 2011 was reduced to 160,000, primarily because of the development of a measles vaccine.

The goal of epidemiologists is first to understand the causes of a disease, then to predict its course, and finally to develop ways of controlling it, including comparisons of different possible approaches. The first step is obtaining and analyzing observed data.

E-mail address: brauer@math.ubc.ca.

http://dx.doi.org/10.1016/j.idm.2017.02.001





CrossMark

Peer review under responsibility of KeAi Communications Co., Ltd.

<sup>2468-0427/© 2017</sup> KeAi Communications Co., Ltd. Production and hosting by Elsevier B.V. This is an open access article under the CC BY license (http:// creativecommons.org/licenses/by/4.0/).

### 2. Some history

The study of infectious disease data began with the work of John Graunt (1620–1674) in his 1662 book "Natural and Political Observations made upon the Bills of Mortality". The Bills of Mortality were weekly records of numbers and causes of death in London parishes. The records, beginning in 1592 and kept continuously from 1603 on, provided the data that Graunt used. He analyzed the various causes of death and gave a method of estimating the comparative risks of dying from various diseases, giving the first approach to a theory of competing risks.

What is usually described as the first model in mathematical epidemiology is the work of Daniel Bernoulli (1700–1782) on inoculation against smallpox. In the eighteenth century smallpox was endemic. Variolation, essentially inoculation with a mild strain, was introduced as a way to produce lifelong immunity against smallpox, but with a small risk of infection and death. There was heated debate about variolation, and Bernoulli was led to study the question of whether variolation was beneficial. His approach was to calculate the increase in life expectancy if smallpox could be eliminated as a cause of death. His approach to the question of competing risks led to publication of a brief outline in 1760 (Bernoulli, 1760) followed in 1766 by a more complete exposition (Bernoulli, 1766, pp. 1–45). His work received a mainly favorable reception but has become better known in the actuarial literature than in the epidemiological literature. However, more recently his approach has been generalized (Dietz & Heesterbeek, 2002).

Another valuable contribution to the understanding of infectious diseases even before there was knowledge about the disease transmission process was the knowledge obtained by study of the temporal and spatial pattern of cholera cases in the 1855 epidemic in London by John Snow, who was able to pinpoint the Broad Street water pump as the source of the infection (Johnson, 2006; Snow, 1855). In 1873, William Budd was able to achieve a similar understanding of the spread of typhoid (Budd, 1873). In 1840, William Farr studied statistical returns with the goal of discovering the laws that underlie the rise and fall of epidemics (Farr, 1840, pp. 91–98).

#### 3. The beginnings of compartmental models

In order to describe a mathematical model for the spread of a communicable disease, it is necessary to make some assumption about the means of spreading infection. The modern view is that diseases are spread by contact through a virus or bacterium. The idea of invisible living creatures as agents of disease goes back at least to the writings of Aristotle (384 BCE–322 BCE). The existence of microorganisms was demonstrated by van Leeuwenhoek (1632–1723) with the aid of the first microscopes. The first expression of the germ theory of disease by Jacob Henle (1809–1885) came in 1840 and was developed by Robert Koch (1843–1910), Joseph Lister (1827–1912), and Louis Pasteur (1822–1875) in the late nineteenth and early twentieth centuries.

In 1906 W.H. Hamer proposed that the spread of infection should depend on the number of susceptible individuals and the number of infective individuals (Hamer, 1906). He suggested a mass action law for the rate of new infections, and this idea has been basic in compartmental models since that time. It is worth noting that the foundations of the entire approach to epidemiology based on compartmental models were laid, not by mathematicians, but by public health physicians such as Sir R.A. Ross, W.H. Hamer, A.G. McKendrick, and W.O. Kermack between 1900 and 1935.

A particularly instructive example is the work of Ross on malaria. Dr. Ross was awarded the second Nobel Prize in Medicine in 1902 for his demonstration of the dynamics of the transmission of malaria between mosquitoes and humans.

It was generally believed that so long as mosquitoes were present in a population malaria could not be eliminated. However, Ross gave a simple compartmental model (Ross, 1911) including mosquitoes and humans which showed that reduction of the mosquito population below a critical level would be sufficient. This was the first introduction of the concept of the basic reproduction number, which has been a central idea in mathematical epidemiology since that time. Field trials supported this conclusion and led to sometimes brilliant successes in malaria control.

The basic compartmental models to describe the transmission of communicable diseases are contained in a sequence of three papers by Kermack and McKendrick (1927, 1932, 1933). The first of these papers described epidemic models.

The original formulation of the Kermack-McKendrick epidemic model given in Kermack and McKendrick (1927) was

$$v(t) = -x'(t) x'(t) = -x(t) \left[ \int_{0}^{t} A(s)v(t-s)ds + A(t)y_{0} \right] z'(t) = \int_{0}^{t} C(s)v(t-s)ds + C(t)y_{0} y(t) = \int_{0}^{t} B(s)v(t-s)ds + B(t)y_{0}.$$
(1)

here, x(t) is the number of susceptibles, y(t) is the number of infectious individuals, and z(t) is the number of recovered individuals. Also  $\varphi(s)$  is the recovery rate when the age of infection is s,  $\psi(s)$  is the recovery rate at infection age s, and

Download English Version:

# https://daneshyari.com/en/article/5662873

Download Persian Version:

https://daneshyari.com/article/5662873

Daneshyari.com