1 Epidemiology is …

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So what is epidemiology, anyway? As shown in Box 1.1, the *Concise Oxford Dictionary* (1964) defined it accurately, but not very helpfully, as ‘the science of epidemics’. In 1970, MacMahon and Pugh came up with something a bit more concrete: ‘the study of the distribution and determinants of disease in humans’ (MacMahon and Pugh, 1970).

![Box 1.1 Epidemiology is ...](image)

The next definition, from the 2008 edition of the *Dictionary of Epidemiology* (Porta, 2008), takes things two steps further by broadening the scope to include health in general, not just disease, as well as highlighting the essential role of epidemiology in translating research findings into health policy and medical practice to control disease. The most recent definition (Porta, 2014) elaborates further still but, in doing so, loses some of the elegance of the earlier versions.

Epidemiology, therefore, is about measuring disease or other aspects of health, identifying the causes of ill-health and intervening to improve health. But what do we mean by ‘health’? Back in 1948, the World Health Organization (WHO, 1948) defined it as ‘... a state of physical, mental and social well-being’. In practice, what we usually measure at a population level is physical health, and this focus is reflected in the content of most routine reports of health data and in many of the health measures that we will consider here. However, methods that attempt to capture the more elusive components of mental and social well-being are now emerging. Instead of simply measuring ‘life expectancy’, WHO introduced the concepts of ‘health-adjusted life expectancy’ (HALE) and subsequently ‘disability-adjusted life years’ (DALYs) to allow better international comparisons of the effectiveness of health systems. In doing so, WHO recognised that it is not longevity per se that we seek, but a long and healthy life. We will discuss these and other measures in more detail in Chapter 2.

Perhaps epidemiology’s most fundamental role is to provide a logic and structure for the analysis of health problems, both large and small – or, as described by Wade Hampton Frost, epidemiology involves the ‘orderly arrangement of [established facts] into chains of inference which extend more or less beyond the bounds of direct observation’ (Frost, 1927). Epidemiology emphasises the sound use of numbers – we have to count and we have to think. We have to think about what is worth counting and how best to count it, about what is practical and, importantly, about how well we (or others) measured whatever it was we set out to measure, and what it all means. Accurate measurement of health is clearly the cornerstone of the discipline, but we believe the special value of epidemiology...
flows from a way of thought that is open, alert to the potential for error, willing to consider alternative explanations and, finally, constructively critical and pragmatic.

We offer this book as an aid to such thought. It does not aim to turn you into a practising epidemiologist overnight, but will give clear directions if that is where you decide to go. Its primary goal is to help you interpret the mass of epidemiological literature and the various types of health data that you may come across. We hope that you will see, by reading and by doing, that the fundamental concepts and tools of epidemiology are relatively simple, although the tasks of integrating, synthesising and interpreting health information are more challenging. But before we go any further, let us do some public health epidemiology.

**A case of food poisoning**

Epidemiology is a bit like detective work in that we try to find out why and how disease occurs. Our first example illustrates this. After an outbreak of food poisoning at a youth camp, the local public health unit was called in to identify the cause (Hook et al., 1996). They first asked everyone at the camp what they had eaten prior to the outbreak, and some results of this investigation are shown in Table 1.1.

| Table 1.1 Numbers of people who became ill after eating various foods at a youth camp |
|-----------------------------------|----------------------------------|---------------------------------|------------------------------|-------------------------------|
| **Food**                         | **People who ate the food**      | **People who did not eat the food** |
|                                  | **Total**                        | **Number ill**                  | **Total**                   | **Number ill**               |
| Friday dinner:                   |                                  |                                 |                             |                              |
| Hot chicken                      | 343                              | 156                             | 231                         | 74                           |
| Peas                             | 390                              | 175                             | 184                         | 55                           |
| Potato fries                     | 422                              | 184                             | 152                         | 46                           |
| Saturday lunch:                  |                                  |                                 |                             |                              |
| Cold chicken                     | 202                              | 155                             | 372                         | 75                           |
| Salad                            | 385                              | 171                             | 189                         | 59                           |
| Saturday dinner:                 |                                  |                                 |                             |                              |
| Fruit salad                      | 324                              | 146                             | 250                         | 84                           |

Source: Adapted from Hook et al., 1996, with permission from John Wiley & Sons, Inc. © 1996 The Public Health Association of Australia Inc.

Looking at the numbers in Table 1.1, it is difficult to see which of the foods might have been responsible for the outbreak. (Note that everyone is recorded as either having eaten or not eaten each food, and most people will have eaten more than one of the foods.) More people became ill after eating potato fries than after eating cold chicken (184 versus 155) – but then more people ate the fries (422 versus 202). How then might we best compare the two foods? One simple way to do this is to calculate the percentage of people who became ill among those who ate (or did not eat) each type of food. For example, 156 out of 343 people who ate hot chicken became ill:

\[
\frac{156}{343} = 0.45 = 45\%
\]
So 45% of people who ate hot chicken became sick. This is known as the attack rate for hot chicken, i.e. 45% of hot chicken eaters were ‘attacked’ by food poisoning.

Calculate the attack rates for the other foods. Which food has the highest attack rate?

Although cold chicken has the highest attack rate (77%), not everyone who ate it (or, more precisely, who reported eating it) became ill and 20%, or one in five people who did not eat cold chicken still became ill. This is to be expected; no matter what the cause of concern, it is rare that everyone who is exposed to it will show the effects (in this case, become ill). What can help here is to work out how much more likely people who ate a particular food were to become ill than those who did not eat it. For example, 45% of people who ate hot chicken became ill, compared with 32% of people who did not eat hot chicken. Hot-chicken eaters were therefore 1.4 times (45% ÷ 32% = 1.4) as likely to become ill as people who did not eat hot chicken. This measure gives us the risk of sickness in hot-chicken eaters relative to non-eaters, hence its name: relative risk.

Calculate the relative risk of developing food poisoning associated with each of the other food items. Which food is associated with the highest relative risk of sickness?

We can now conclude that the food item most likely to have been responsible for the outbreak was the cold chicken – people who ate this were almost four times as likely to become ill as those who did not. This is quite a strong relative risk; in comparison, eating any of the other foods was associated with no more than 1.5 times the risk of disease. The relevant data, including the attack rates and relative risks, are summarised in Table 1.2, which is much more informative than the raw numbers of Table 1.1.

In identifying the cause of the outbreak you have just solved an epidemiological problem. The ‘attack rates’ and ‘relative risks’ that you used are simple to calculate and are two very useful epidemiological measures. We will discuss them further in Chapters 2 and 5, and they will appear throughout the book.

Subdisciplines of epidemiology

The outbreak investigation above is an example of what might be called public health epidemiology, or infectious disease epidemiology, with the first name reflecting the broad field of application and the second the nature of both the aetiological (causal) agent and the disease. It is quite common to specify such subfields of epidemiology, which range, on the one hand, from nutritional epidemiology to social, environmental and eco-epidemiology, and on the other hand, from cancer epidemiology to injury or perinatal epidemiology; the former grouping being exposure-oriented and the latter focused on the particular disease or outcome. Nonetheless, the core methods and techniques of epidemiology remain common to all subdisciplines, so the contents of this book are relevant to all. Setting sub-speciality boundaries largely reflects the explosion of knowledge in these areas, although some areas do present special challenges. For example, capturing a person’s usual diet is remarkably challenging, and the subsequent data analysis equally so; epidemiologists coming fresh to the field of nutritional epidemiology will need to develop experience...
occupational epidemiology has the longest history of all, with influential early observations of diseases linked to occupations such as mining appearing in the sixteenth century, and a systematic treatise on occupational diseases published by Ramazzini back in 1700 (Rosen, 1958). Occupational health research in general, and epidemiology in particular, continue to contribute to the enhancement of workplace health today. Seminal contributions in the field include identification of the pulmonary (lung) hazards of asbestos for miners and construction workers (Selikoff et al., 1965) and the work practices that led to an epidemic of a rare fatal cancer in workers in the polyvinyl chloride industry (Makk et al., 1974). Company records of job tasks can provide measures of past exposure among employees, allowing researchers to look back in time and link, for example, past asbestos exposure to subsequent deaths in the workforce. (This type of study is an historical cohort design – see Chapter 4. It is only possible when there are good records of both exposure and outcome, usually death, and for this reason it has proved particularly useful in occupational studies where such records often do exist.)

Far more contemporary are the subdisciplines of molecular epidemiology and clinical epidemiology. The former aims to weld the population perspective of epidemiology with our rapidly increasing understanding of how variations in genes and their products affect the growth, form and function of cells and tissues. It thus has the potential to define genetic contributions to disease risk, and can also provide biological markers of some exposures (e.g. changes to DNA following exposure to tobacco smoke). In contrast, clinical epidemiology differs from other branches of epidemiology because it focuses on enhancing clinical decisions to benefit individual patients, rather than improving the
health of populations. For this reason it is sometimes regarded as a separate discipline; a view encouraged by the fact that clinical epidemiology has developed its own names for many standard epidemiological measures, as you will see later in the book. The foundations are, however, identical to those of public health epidemiology and, when appropriate, we will discuss the two in parallel, highlighting any differences in language or approach along the way.

There is also increasing interest in life-course epidemiology, which attempts to integrate events across the lifetime, often going right back to conception and sometimes to previous generations, to understand disease risk.

On epidemics

If we take the word ‘epidemiology’ itself, its origins from ‘epidemic’ are clear. If we talk about an epidemic we immediately conjure images of acute outbreaks of infectious diseases but, both for practical and for etymological reasons, it seems reasonable to use the term to describe a notable excess of any disease over time. Many developed countries could, for example, be described as undergoing ‘an epidemic of lung cancer’ over the past few decades (Figure 1.1). Notably, the pattern of lung cancer over time differs for men and women; rates in men rose sharply between 1950 and 1980 but have been falling for some years now, while those in women rose later and started to fall more recently – a consequence of the fact that, as a group, women took up smoking more recently than men. To describe this excessive occurrence of disease (or death) as an ‘epidemic’ captures some of the urgency the numbers demand.

![Figure 1.1](image_url)

**Figure 1.1** Time trends in lung cancer mortality rates in the United States (age-standardised to the 1970 US population) for White men and women, from 1950 to 2013

Source: Data from CDC WONDER Database, accessed 27 February 2015.

The derivation of the word ‘epidemiology’ itself is from the Greek *epi* (upon), *demos* (the people) and *logia* (study). Literally, therefore, it means ‘the study (of what is) upon the people’. Such study suggests a simple set of questions that have long lain at the heart of epidemiology:
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• What disease/condition is present in excess?
• Who is ill?
• Where do they live?
• When did they become ill?
• Why did they become ill?

The first question reflects the need for a sound, common definition of a disease so that like is compared with like. Epidemiology is all about comparison – without some reference to what is usual, how can we identify excess? The next three questions form the mantra of descriptive epidemiology: ‘person, place and time’.

Figure 1.2 Time trends in all-cause mortality at ages 0–64 years in the Russian Federation from 1980 to 2013

Source: Data from WHO, 2016–.

While in the Russian example the ‘epidemic’ provides the drama, the massive difference in mortality rates between the sexes is another important public health challenge in its own right.
health progress, but there is clearly no simple solution. In this case, a high proportion of the deaths were linked to excess consumption of alcohol during the 1990s: increases in mortality coincided with periods of economic and societal crisis, and rates fell when the economic situation improved (Zaridze et al., 2009). The earlier decline during the 1980s coincided with an anti-alcohol campaign involving higher taxes on alcohol and reduced production, which led to sharp decreases in alcohol consumption in the short term and lower rates of alcohol-related mortality and suicide (Pridemore and Spivak, 2003). This example highlights the importance of paying close attention to descriptive data that provide a ‘community diagnosis’ or take the public health ‘pulse’ of a nation. Much can be gleaned from apparently simple data to give a precise description of the overall health of a population or a more specific health event, as the following exercise shows.

An historical epidemic

Table 1.3 shows some data that relate to an actual human experience. It tells you how many people there were in various age, sex and socioeconomic groups and what percentage of these people died during the ‘epidemic’. The challenge is to use these data to describe the event systematically, in terms of whom this happened to (we have no data on place or time) and then to think about the sort of event that might have induced such a pattern.

Table 1.3 An historical event

<table>
<thead>
<tr>
<th>SES*</th>
<th>Adult males</th>
<th>Adult females</th>
<th>Children (both sexes)</th>
<th>Total population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>% dead</td>
<td>Total</td>
<td>% dead</td>
</tr>
<tr>
<td>High</td>
<td>175</td>
<td>67.4</td>
<td>144</td>
<td>2.8</td>
</tr>
<tr>
<td>Medium</td>
<td>168</td>
<td>91.7</td>
<td>93</td>
<td>14.0</td>
</tr>
<tr>
<td>Low</td>
<td>462</td>
<td>83.8</td>
<td>165</td>
<td>53.9</td>
</tr>
<tr>
<td>Other</td>
<td>862</td>
<td>77.7</td>
<td>23</td>
<td>13.0</td>
</tr>
<tr>
<td>Total</td>
<td>1667</td>
<td>79.7</td>
<td>425</td>
<td>25.6</td>
</tr>
</tbody>
</table>

* SES, socioeconomic status

Source: See Box 1.2.

The following questions are designed to help you identify key features of the data.

1. What is distinctive about this isolated population with regard to:
   - the numbers of men and women (sex distribution),
   - the numbers of adults and children (age distribution), and
   - the numbers in each socioeconomic group (socioeconomic distribution)?

2. What strikes you about the percentage of people who died (the ‘death rate’)? Does this differ for (a) adults and children, (b) men and women, (c) high and low socioeconomic status (SES) and (d) any particular combinations of the above?
Table 1.3 displays more complicated data than Table 1.2 because you had to consider the joint effects of three factors (sex, SES and age) on mortality. The sequence of questions above underlines a general principle in describing such tables – i.e. look at overall patterns first, then move on to more detail. We all see things in different ways, but until you develop your own style the approach shown in Box 1.2 may help you avoid getting lost in the array of possible relationships. You need first to grasp the size of the whole group under study and how many died; then check the overall patterns (the numbers and death rates) across each ‘exposure’ separately (sex, SES, age). For example, first look at the death rates for all adult males, ignoring their SES, or for all people of high SES, ignoring their age and sex. Only then consider the more complex joint effects such as the influence of SES on mortality among women.

**BOX 1.2  An historical event**

Things to note about the population include:

- the predominance of adult males (1667 ÷ 2201 = 76%), the much smaller proportion of adult females (19%), and the very few children
- the substantial excess of persons of low SES (men and children in particular)
- the total population (2201) – a village, small town, an army barracks … ?

Things to note about the death rates include that:

- the overall death rate is very high – more than two-thirds died
- overall, death rates increased with decreasing SES
- the death rate in men (79.7%) was much higher than that in women (25.6%), while the death rate in children was between these two
- in men, the death rate was high in all socioeconomic classes, although those of high SES fared better than the rest; in women, the death rate was always lower than that for males of equivalent SES, but it increased strikingly from high to medium to low SES
- the only children to die were of low SES.

Overall, the relative risk (RR) for men versus women is 79.7 ÷ 25.6 = 3.1

The RR for low versus high SES is 74.8 ÷ 37.5 = 2.0
The RR for low versus high SES men is 83.8 ÷ 67.4 = 1.2
The RR for low versus high SES women is 53.9 ÷ 2.8 = 19.3
The RR for low SES men versus high SES women is 83.8 ÷ 2.8 = 29.9

A disaster has occurred, causing a high death rate that predominantly affected men (of all social classes) and, to a lesser extent, women and children of low SES. Overall, there is a modest benefit of belonging to a higher socioeconomic stratum, and among women this protection was exceptionally strong (a 19-fold higher risk of dying for low versus high SES).
In tackling this and the previous problem you have already done some serious epidemiology: you have described data, interpreted the patterns you observed and used epidemiological measures to help do this. We will build on this throughout the book, but first let’s step back a little and see what other lessons we can learn from the past.

The beginnings

The ‘great man’ theory has fallen out of favour in contemporary historical practice; however, linking historical events to individuals adds interest, so we will focus on some of the main players in this brief overview of the development of population health and epidemiology.

Good epidemiological practice and reasoning started long ago. Perhaps the first proto-epidemiologist (‘proto’ because he did not actually count anything) was Hippocrates of Cos (460–375 BC), who recognised that both environmental and behavioural factors could affect health (see Box 1.3).

Such substantial differences in risk reflect powerful preventive effects and, in this instance, it was a mix of social custom and the physical consequences of socioeconomic stratification. The event was the sinking of the Titanic, where those of higher SES (the first-class passengers) were situated on the upper decks and were therefore closer to the lifeboats than those of medium and low SES (those travelling second and third class, respectively). The males gallantly helped the females and children into the lifeboats first. Those of ‘other’ SES were the crew.

Source: Wreck Commissioner of the United Kingdom, 1912.

Box 1.3  On airs, waters and places

Whoever wishes to investigate medicine properly, should proceed thus: in the first place to consider the seasons of the year, and what effects each of them produces ... Then the winds, the hot and the cold, especially such as are common to all countries, and then such as are peculiar to each locality. We must also consider the qualities of the waters ... In the same manner, when one comes into a city to which he is a stranger, he ought to consider its situation, how it lies as to the winds and the rising of the sun; for its influence is not the same whether it lies to the north or the south, to the rising or to the setting sun. These things one ought to consider most attentively, and concerning the waters which the inhabitants use, whether they be marshy and soft, or hard, and running from elevated and rocky situations, and then if saltish and unfit for cooking; and the ground, whether it be naked and deficient in water, or wooded and well watered, and whether it lies in a hollow, confined situation, or is elevated and cold; and the mode in which the inhabitants live, and what are their pursuits, whether they are fond of drinking and eating to excess, and given to indolence, or are fond of exercise and labour ...

Source: Extracted from Hippocrates of Cos, 400 BC.