Chapter 1

Historical background

This chapter provides a brief history of epidemiology. We shall see how closely epidemiology and plant ecology are linked throughout history dating back to the old Greek. We shall also see relevant research questions were already put forward centuries ago. We are still dealing with those questions in modern science. Finally, we get grip on the term ‘epidemic’ and define it. We have a good starting point for the remaining of the book having some feeling and a definition of the term ‘epidemic’.

1.1 Observations of epidemics

Epidemiology deals with the abundance of diseases in populations. The Greek physician Hippocrates (460–380 BC) already observed disease among people. He introduced the term *επιδεμιοσ*. This means literally ‘what is among people’. This ‘what’ were diseases and we may read the word ‘people’ as population. Hippocrates wondered that some individuals of a population got diseased whereas others did not. He described, for example, epidemics of mumps among young males in a gymnasium. He asked why one young boy is ill and the other one not. He also asked why is the disease among the boys in one year abundant and the other year less so.

Theophrastus (372–287 BC) extended the observations from people to plants. He also observed some individuals of a plant population diseased whereas others were not. Still, we observe such variation in infection among individuals of a population, or among populations, and try to explain it. Even shoots of a clonal plant like *Cirsium arvense* L. Scop. (‘creeping thistle’) may show differences in infection at a scale of centimeters.

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The rust fungus *Puccinia punctiformis* (Str.) Röhl. pervades thistle shoots systemically in spring. A shoot becomes pale and slender. Whereas one shoot can be completely infected, the neighbor shoot does not show symptoms of infection at all (Fig. 1.1.1).

Variation of environment is one explanation for the variation of infection within and among populations, as Theophrastus observed already. He wrote about the abundance of plants infected with a rust fungus the following:

> '*…for lands which are exposed to the wind and elevated are not reliable to rust, or less so, while those that lie low and are not exposed to wind are more so*'.

Wind-exposed sites are drier inhibiting, in general, infection of plants by pathogens. Although Theophrastus did not know the positive effects of water on the process of infection, he recognized the importance of the abiotic environment just by observing the abundance of plant disease minutely.

Environment may indeed explain abundance of diseases in plant populations like it may determine abundance of plants. The parallels between

Fig. 1.1.1 Shoots of *Cirsium arvense* systemically infected by the rust fungus *Puccinia punctiformis*, which are the larger and paler ones, and uninfected ones. The infected shoots grow-up quickly in spring, but die back before flowering. Infected and non-infected shoots are present side by side.

epidemiology and plant ecology are obvious. Both sciences deal with interactions between organisms and their environment.

Environment only is one of the factors explaining abundance of disease. Transmission of disease is another factor. Although the Greek had already some notion of contagion, Gerolamo Fracastoro recognized formally the infectious character of diseases. He wrote in 1546:

> '*…the contagion of a putrefaction goes from one body to another whether adjacent or distant…the seeds have the faculty of multiplying and propagating rapidly*'.

He suggested transmission of disease by way of 'seeds'. The microscope was not developed at that time yet. Fracastoro, therefore, could not detect propagating particles like spores of fungi. Neither he was able to detect microorganisms like viruses transmitting from one individual to another. Seeds, however, were known as a way of transporting plants from one location to another. Ignoring the use of the term 'seeds', Fracastoro deduced correctly that transmission of disease is possible.

Nowadays, transmission of diseases is still an important research topic, especially in medicine. We humans face from time to time outbreaks of new diseases like the one of 'Severe Acute Respiratory Syndrome' (SARS) in 2002. We try to understand and predict the impact of such outbreaks on human society. Knowing the mode and rate of transmission of disease is indispensable for such understanding and prediction. Similarly, knowledge about the mode and rate of disease transmission is necessary to understand the abundance of disease in plant populations. We see here again the parallels between epidemiology and plant ecology, as we use information about the mode and rate of spread of plant propagules to understand abundance of plants.

Fracastoro did other interesting observations on infectiousness of diseases. He wrote in 1546:

> '*There are diseases of plants which do not contaminate animals, and vice versa animal diseases which do not attack plants; there are other diseases limited to man or certain animals as cattle, horses and so on. Certain diseases have a special affinity for certain individuals or certain organs*'.

Fracastoro recognized a barrier between plants and animals with respect to disease transmission. Pathogens of plants cannot infect animals and *vice versa*. He also noticed some individuals were more prone to disease than other ones, as already remarked by the old Greek. In addition, he observed pathogens might specifically attack some organs instead of a whole individual. The smut fungus *Microbotryum violaceum* (Pers.: Pers.) Deml. Fuckel is a well-known example of such a pathogen in plant ecology. It predominantly infects the inflorescences of *Silene alba* (Miller) Krause resulting in sterilization of the host plants. The evolutionary consequences of such sterilizing effects were subject of a suite of studies (Alexander *et al*., 1996).

We make now a jump of two centuries from the observations of Fracastoro in the 16th century to the 18th century. We see the interest for epidemics increases. The impact of epidemics on economics may explain this increased interest, as the Danish Fabricius wrote in 1774:

> '*Knowledge of the diseases both of animals and plants forms an important part of our rural economy but is still too much neglected… With plants the condition is far worse; rural economy contains no complete description of their diseases*'.

The lack of knowledge of plant diseases, and more importantly the ability to control these, became manifest in the 1840s. Severe epidemics of the disease 'potato late blight' destroyed potatoes in northwestern Europe. Potato, *Solanum tuberosum* L., was the major food source of poor people at that time. The epidemics of potato late blight caused especially a disaster in Ireland. About two million Irish people died due to fame. Another million migrated to North America escaping the devastating effects of the disease. The disaster also triggered a quest for the causal agent. In 1861, the German De Bary demonstrated the fungus *Phytophthora infestans* (Mont.) de By. as causal agent. In the same era, the French Louis Pasteur demonstrated microorganisms as self-reproducing organisms rejecting the, at that time, common belief of spontaneous generation.

The progress in thinking about microorganisms as causal agents of disease resulted in the postulates of Koch. These were proposed in 1890 providing a basis for modern plant pathology. The postulates are listed here to pinpoint their importance (Stanier *et al*., 1976):

1. the microorganism must be present in every case of disease;
2. the microorganism must be isolated from the diseased host and grown in pure culture;

3. the specific disease must be reproduced when a pure culture of the microorganism is inoculated into a healthy susceptible host;
4. the microorganism must be recoverable once again from the experimentally infected host.

These postulates are still up to date. Plants bear many microorganisms. Not all of them have to cause disease. Detecting the pathogen of a specific disease is not always that obvious.

The middle of the 19th century was not only of crucial importance with respect to botanical epidemiology. The development of medical epidemiology also accelerated due to a disease epidemic occurring in London at the year 1848. The epidemic was investigated by the British physician John Snow. He detected a suspect water pump mapping precisely all cases of disease. Although the causal agent, *Vibrio cholerae*, was not known yet, the epidemic could be stopped closing the contaminated water pump. Thus, John Snow did not only observe an epidemic, as many did before, but he also tried to manage it. John Snow also was the first to quantify and map diseased cases carefully. With John Snow, we enter the era of quantitative epidemiology.

1.2 Quantification of epidemics

Already the ancient Greek wondered about the fact that some individuals get ill whereas others do not in a population. John Snow did not stop at that point of wondering, but he started to count. He observed a rapid increase of diseased people around a water pump in the Soho district of London. Moreover, he detected workers at a local beer brewery were not ill. They drank, working at the brewery, only beer. Combining these facts, Snow suggested closing the suspected water pump. The epidemic stopped subsequently, as the history tells us.

John Snow introduced quantification of disease as a tool to understand and, especially, to manage epidemics. He, therefore, is called the father of epidemiology. If so, we should call him the father of medical epidemiology, as epidemiology split into a botanical and medical branch by the end of the 19th century. One reason to explain the separation may be that plants and human beings do not share pathogens. Having the postulates of Koch at hand, the observation of Fracastoro in 1546 turned out to be an axiom.

The Swiss Gäumann put forward another reason (1951). He noted the accumulation of knowledge was faster in the medical than botanical epidemiology. The progress was so fast, following Gäumann, because medicine dealt only with one species, *Homo sapiens*, and its diseases. In contrast, botanical epidemiology had to cover an uncountable number of host species and all the diseases of these.

Whatever the reason was, we see a separation of botanical and medical epidemiology at the end of the 19th century. We, of course, shall follow the history of botanical epidemiology further. This history went, subsequently, along the quantification and understanding of crop diseases.

The potato late blight outbreak of the 1840s had put plant diseases high on the agenda of agronomists. All the progress in understanding crop diseases cumulated in an outstanding book of Ernst Gäumann (1951). Unfortunately, he published this book only in German. The isolated position of Switzerland during and around World War II did not allow the publication of an English version. Reading the German text, we see the whole breath and fascination of epidemiology. Gäumann did not only describe precisely many disease epidemics on crops. He also put forward a lot of information about biotic and abiotic factors governing epidemics. He followed a bottom-up approach starting at the individual level. At this level, the basic compatibility between host and pathogen had to be explained. Why does one pathogen species enter a plant and another one not? How do the pathogens enter a plant? How do these establish within a plant? How do pathogens take up resources from the host? The book of Gäumann provided a lot of information to answer all these basic questions.

Nowadays, explaining the basic compatibility is a topic of science still. For example, why does the rust fungus *P. punctiformis* only attack plants of the one and only host *C. arvense*? Moreover, the fungus can only grow and reproduce on living host tissue. Culturing it on artificial media is nearly impossible like it is for all rust fungi. We, therefore, call these biothrophs. We wonder about the adaptations of biothrophs to specific host tissue. Still, we are far away from understanding such adaptations.

Even basic compatibility between a plant and pathogen does not mean that a pathogen infects all host phenotypes. Some phenotypes may resist infection. We enter the topic of resistance and pathogenicity. As at the time of Gäumann's 'Pflanzliche Infektionslehre', resistance and pathogenicity is a hot topic in science nowadays. The research even intensified having molecular tools at hand to discover underlying genes of resistance and pathogenicity.

Epidemics are phenomena at the population level. Gäumann (1951)went from the individual to the population level to understand epidemics. For example, he used information about the latent period p estimated at the individual level. The latent period is the period between a pathogen's penetration of the host and the start of its reproduction. The shorter the latent period, the faster an epidemic spreads, as we shall see later in this textbook. Gäumann (1951) quantified the effects of various biotic and abiotic factors on the latent period underpinning the ecological characteristics of it. We may read these parts of the book as an ecology textbook ignoring the plant pathology terminology.

Gäumann also introduced the term 'chain of infection' (German: 'Infektketten'). This term pinpoints an epidemic is a manifold repetition of the infection process on individuals of a host. He made a distinction between homogeneous and heterogeneous chains of infection. In case of a homogeneous one, the life cycle of a pathogen runs completely on plants of a single host species. In contrast, completion of the life cycle requires at least two host species in case of a heterogeneous chain of infection. The life cycle of the rust fungus *P. punctiformis* is an example of a homogeneous chain of infection. It completely runs on individuals of the host *C. arvense*. The life cycle of another rust fungus, *Coleosporium tussilagines* (Pers.) Lév. Is an example of an heterogeneous chain of infection. It completes a part of the life cycle on needles of *Pinus* species, and a part on other hosts like the annual plant *Senecio vulgaris* L.

Gäumann (1951) also, of course, treated the mechanisms of pathogen transmission. Propagating particles may be dispersed and transported by various mechanisms. Wind, water, and animals are the most common vectors of transmission. We see again the importance of considering environment as a factor influencing epidemiological processes. Ecology and epidemiology are closely linked sciences.

The approach followed in 'Pflanzliche Infektionslehre' was picked-up by Zadoks & Schein (1979). Their 'Epidemiology and Plant Disease Management' was for a long time 'the' textbook of botanical epidemiology. Zadoks & Schein introduced the terms 'monocyclic process' and 'polycyclic process' distinguishing infection processes at the individual and population level, respectively. We follow the spread of a pathogen. It starts at an individual plant. The pathogen enters the plant, grows, and reproduces on it. Subsequently, the pathogen passes to another plant. The infection process subsequently starts again on this plant. We observe the manifold repetition of the monocyclic process at the population level. We, however, cannot explain the spread of a pathogen, the polycyclic process, solely from the underlying monocyclic process. We need to take into account phenomena like pathogen dispersal and variation in plant resistance within a population.

Mathematics also got a prominent place in Zadoks & Schein (1979) referring to the seminal work of the South-African Van der Plank (1963). Van der Plank introduced an analytical model to describe epidemics. This model is known as the 'differential-difference equation'. Van der Plank combined three key parameters in the equation: the latent period p, the infectious period i, and the daily multiplication factor R_c. We saw p already in Gäumann's textbook. Van der Plank added the parameters i and R_c. The infectious period i indicates the period that a pathogen reproduces on an individual plant. After this period, the infected tissue of a plant is no more relevant for the progress of an epidemic. The daily multiplication factor R_c indicates the rate of reproduction of a pathogen, e.g. the average number of lesions produced by one lesion per day in a host population.

Combining the parameters p, i, and R_c in the differential-difference equation provides a wealth of insight in the progress of epidemics (cf. Chapter 3). It is remarkable that thorny phenomena like epidemics can be abstracted in just three parameters. We, however, shall see in the subsequent chapters that many biotic and abiotic factors influence these parameters. The parameters theseselves turn out to be complex phenomena. Nevertheless, Van der Plank provided a relatively simple framework around all this complexity. His work, therefore, was outstanding. It also provoked research even decennia after the publication of his textbook (Campbell & Madden, 1990).

The differential-difference equation provides an excellent tool to describe the progress of epidemics. This, however, means we look at the dimension time holding the area of spread constant. Describing adequately epidemics in time and space was the next step in the development of botanical epidemiology. One of the famous and perhaps the most famous, botanical epidemiologists of the 20th century set this step: Jan Carl Zadoks. He described the spatial dynamics first using a numerical approach, i.e. the simulation model EPIMUL (Kampmeijer & Zadoks, 1977). Then he was able to adapt an analytical, diffusion, model to epidemics (Van den Bosch *et al.*, 1988a–c). He went even outside mathematics to describe and understand epidemics. A physical model was proposed (Zawolek & Zadoks, 1992). The physical model has, however, not been generally accepted yet.

Nevertheless, Zadoks was able to give a mathematical description for a phenomenon that was observed since the ancient Greek: the spread of disease in time and space!

1.3 Defining epidemic

The term ‘epidemic’ has been associated with disasters throughout the centuries. We see the ghost of the past in the definition of epidemic in the Oxford Dictionary (Anonymous, 1989) still:

‘*Disease spreading quickly among many people in the same place for a time*’.

The word ‘quickly’ reflects the fear of a disease that cannot be stopped causing much harm. Such diseases exist, but these are rather the exception than the role. In general, the incidence and severity of plant diseases show patterns of increase and decrease in time. All kind of biotic and abiotic factors determine the rate of increase, as we shall see in the subsequent chapters. In addition, the impact of a disease on the host varies depending on biotic and abiotic factors. The rate of increase, therefore, is not necessarily associated with the extent of the damage caused to the host. For example, *P. punctiformis* may infect *C. arvense* systemically (Fig. 1.1.1). The increase of the incidence of systemic infection is relatively slow compared to that of the other type of infection caused by this pathogen, the local lesion infection. Nevertheless, the negative impact of systemic infection on *C. arvense* is larger than that of local lesion infection. The epidemic of the Acquired Immunodeficiency Syndrome (AIDS) developed also relatively slow, but the impact on human beings was, and is, tremendously. Comparatively, the rate of increase of the yearly influenza epidemic is very fast whereas the impact is relatively small.

Zadoks & Schein (1979) came up with a more neutral definition of epidemics following Gäumann (1951). Their definition will be adopted for the remaining of the book. It reads as follow:

‘*An increase of disease, limited in time and space, in a plant population*’.

This definition implies any increase of disease should be seen in a well-defined space and period. Alternatively, we can measure the spread of a disease in terms of velocity using the traditional unit of distance per time, e.g. m. sec^{-1}. If so, we do not need to limit epidemics in time and space.

Epidemiology is the science explaining increases of diseases in time and space. It is a science focusing on population dynamics as expressed in the definition of Campbell & Madden (1990):

'*The study of the temporal and spatial changes that occur during epidemics of plant diseases that are caused by populations of pathogens in populations of plants*'.

Epidemiology, however, goes a step further than studying spatial dynamics of pathogens only. Epidemiology is the ecology of diseases.

1.4 Recommended reading

The historical phrases cited in this chapter are taken from Zadoks & Koster (1976). They provide an overview of the history of botanical epidemiology up to the 70s of the 20th century. Campbell & Madden (1990) provide a shorter, but more up to date, overview of the history.