1 Introduction

Why comparative epidemiology of plant diseases? Zadoks and Schein (1980) rightly stated that results from individual experiments and observations tend to catch and encapsulate scientists who then might generalize beyond their results; this often leads to far-reaching, but premature hypotheses on epidemiological aspects. More consolidated hypotheses, however, can be obtained when diseases and their epidemics are compared with others. Comparative epidemiology (CE) is called upon to test such hypotheses by falsification in appropriate experiments or posterior analyses. Also, hypotheses that arise from observation, professional experience and reviews may be tested in such a way.

Falsification, sensu Popper (1973), searches for errors to disprove and negate existing concepts, e.g. theories. The assumption "all swans are white" is falsified by a single black one. Kuhn (1978), however, warns of any naive use of Popper's approach (e.g. the swan example) because a theory must not necessarily be valid for all possible applications. The theory simply may not fit specific conditions. It may suffice to redefine a theory or make it more specific. If, however, comparative research reveals discrepancies between theory and new facts which can no longer be reconciled, then a change of paradigm will occur and previously held views and theories will be abolished. These changes may be slow and gradual, or abrupt, becoming something entirely new. Comparative research implies reduction, usually favoured by statistical methods. Popper (cited by Lorenz 1978) regards this as a scientific success, which, even if it fails, still leaves behind challenging questions as intellectual properties of research.

Comparative epidemiology essentially does across-studies of pathosystems (p. 7) and the temporal as well as spatial aspects of their epidemics and structural elements. The same pathosystem may also be compared across different characteristic environmental conditions (e.g. at pathotopes sensu Putter, pers. comm.; see Sect 6.4) and among distinct methods of system control or design (see Chap. 7). The latter comparisons are often made in the course of validating experimental results, though not necessarily seen in the context of comparative epidemiology.

A pathosystem results from interactions in the disease square (Zadoks and Schein 1979, based on Van der Plank's fungicide square of 1963) comprising the pathogen (or causative agents such as deficiencies, etc.), the host, the environment and human interference. A pathogen may be assisted by other organisms to cause disease, for instance, vectors, predisposing fungi or helper viruses, which become part of the pathosystem. Weather factors and human actions, e.g. agricultural practices, can substantially influence epidemics. Therefore, their effects may also be studied across climatic conditions or agricultural practices, even within the same pathogen-host combination, e.g. across pathotypes, different sites or agri-

cultural practices. Data obtained from the validation of an experimental comparison, usually done under varying climatic and agricultural conditions, may be used for posterior comparisons.

With comparative epidemiology, differences or similarities across studies of pathosystems or epidemics are examined with specifically designed experiments (Sect. 3.2.1) or posterior analyses (Sect. 3.2.2). By means of these two approaches, generalizations are attempted from the great diversity of epidemics of hundreds of diseases that occur under a variety of environmental conditions and agricultural practices. New concepts, hypotheses, theories, principles and laws may be established. Existing ones may be tested to determine if they are still valid against new facts and developments. All this is instrumental in placing epidemiological aspects into a meaningful context.

The comparison of epidemics is a method for both analytical and synoptic research. Comparative epidemiology compares underlying principles of epidemics *across* studies of pathosystems, their epidemics and factors affecting them. Comparative epidemiology thus plays a "...unifying and crystallizing role...," (Butt and Royle 1980) as it distils commonalities or differences in behaviour and structures which help to explain why they exist. From the apparently unlimited diversity of epidemics, a convenient number of basic types of epidemics may emerge eventually to which new epidemics could then be assigned (Kranz 1978, 1988b). This process would help to consolidate epidemiology and to pave the way for research to reach conclusions more speedily and with fewer detours. The plant pathologists then would see just the forest, rather than a multitude of trees.

Comparative studies differ in branches of science in their philosophy, objectives and procedures. They have, when appropriately adjusted, some general features. For instance, in phylogenetics and systematics, comparative studies can be stripped down to the following five elements (Gittleman and Luh 1992): (1) The main hypothesis for the objective with or without causal explanations, e.g. ecological or evolutionary factors affecting phenotype, or co-variation between two criteria (traits); (2) the range of variation in the criteria or traits (in terms of standard deviation); (3) the presence, location and form of any correlation relating to the objective of the study; (4) the range of variation once the trait(s) under study have been transformed through some comparative statistical procedure, e.g. to remove correlations; and (5) the knowledge of the difference in rate of change (among the criteria used) that will impinge on the divergence of criteria. Properly adopted, these points are valid also for comparative epidemiology.

As a general development, however, comparative studies now may be "unavoidably statistical" (Gittleman and Luh 1992). This is because of the accumulation of basic data on many traits in many areas of science (e.g. behavioural science and ecology) and the availability of computers with ample capacity and suitable software. Also, the knowledge basis and framework for comparative research have become more solid than ever. All this leads to a better understanding of the intricacies and the background of the object under study. Statistical tests, where applicable, will reveal more objectively the importance of variants under comparison, i.e. those variants that are significantly similar or different from others, in particular, from standards or hitherto accepted concepts. Statistical methods also have reductional properties. A brief guideline on the use of statistical methods in com-

parative epidemiology can be found, together with other methods for comparison, in Section 3.3.

As biologists are usually interested in various aspects of living systems per se, their reductionism and abstraction may not go so far as, for instance, in physics. Comparison in biology may require the perception of entirety (Gestalt) which is more than the sum of its parts. For Koehler and v. Bertalanffy (cited by Lorenz 1978) "Gestalt" is the "...harmonic and – effective in both directions – interlinked causal chains, the harmonic interaction of which causes the entirety ". This sounds rather similar to the definition of a system (see p. 5) by Watt (1966). As a research guideline it helped ethologists, who were familiar with the range of possible patterns, to discover certain inherent patterns of animal motion in related animal taxa. Such a comprehensive perception can capture relations and configurations which, together with rational thinking and detailed studies, will help to discover unexpected principles by comparative research. For comparisons of behaviour and phylogeny, Wenzel (1992) likes to understand: "The very breath of life itself and the living world in all its richness demonstrates that the "whole" can be much more than the sum of its parts". He continues, "Although we must have a certain combination of genes to permit us to speak, there is likely no gene for speech." In epidemiology, the graphs of the temporal and spatial dynamics of epidemics (i.e. disease progress curve and gradient) may be taken as entireties. They will be dealt with in Chapters 5 and 6 as the result of many factors and reactions that interact. Robinson (1976) had already referred to the term "Gestalt" in the context of pathosystems. With the system and the entire context in mind, results from rational comparisons, even on detailed aspects, can then be successful as long as they convey a grasp of the extent of phenomena, the problems involved, and a feeling for the diversity of possible views and interactions.

In plant pathology, Gäumann (1951) and Van der Plank (1963) first used comparison of epidemics and relevant factors to arrive at generalities about epidemics. In his *Principles of plant pathology* Yarwood (1973) lists 17 of his principles as related to epidemiology. For instance, he considered the poor relation which exists between the biotic potential of the pathogen and the resulting severity of the disease it causes, as a principle. He also gives this rank to the always differential response of host and pathogen to environment and to the number of diseases that increase with the increasing production of a crop. An experimental comparison of epidemics for more generally applicable information was described by Kranz (1968a–c, 1974a, 1978). A range of possible applications of comparative epidemiology is presented in the volume edited by Palti and Kranz (1980). In this volume, Zadoks and Schein (1980) distinguish the individual, the population and the community level in epidemiology and choose a comparison of processes, objectives and tools. Aust et al. (1980) compared the ability of factors in the disease triangle that act on the system "epidemic" to compensate for each other. Thresh (1980) reviewed the effect of factors to compare virus diseases and Jones (1980) did so for nematodes. Rotem and Palti (1980) compared the efficacy of cultural versus chemical disease control and Putter (1980) discussed factors relevant to the management of outbreaks of endemic diseases under tropical subsistence farming.

After a brief introduction to plant disease epidemiology and a more detailed definition of comparative epidemiology with its aims and scope in Chapter 2,

Chapter 3 deals with its methodology. Posterior analyses (Sect. 3.2.2) should make use of the extensive amount of information that has accumulated from many experiments, which would otherwise just pile up or be buried in "data cemeteries" and collections. This unused and unrelated information is a treasure with which objectives of comparative epidemiology can be achieved. Chapter 4 is devoted to the systems level of host and pathogens and, thus, to the elements as criteria for comparisons within the monocycle of epidemics. Chapter 5 deals with the temporal and Chapter 6 with the spatial aspects of epidemics. Finally, Chapter 7 describes how the effects of epidemics on crops can be compared. This obviously relates to disease management through systems control and system design. Comparative epidemiology then aims for a more rational use of resources to identify real research needs worth studying in epidemiology.

Examples of across-studies will be presented to demonstrate feasible applications of comparative epidemiology. Also, a few published "within-study" comparisons are cited to exemplify the range of possible comparisons, useful criteria and methods that might be adopted for comparative epidemiology. The emphasis of this text will be on criteria, methods and procedures. No exhaustive account of any across-study is given, but ample references to suitable publications are provided which can be used as guidelines for intended projects.

2 Plant Disease Epidemiology and the Scope of Across-Comparison

In the epidemiology of plant diseases, the dynamics of populations of pathogens in populations of hosts are studied along with the resulting disease under the influence of environmental factors and human interference. Epidemiology is the ecological branch of plant pathology and it is dealt with exhaustively in a number of textbooks: Zadoks and Schein (1979), Campbell and Madden (1990), Rapilly (1991), Nagarajan and Muralidharan (1995), Bergamin Filho and Amorim (1996), Kranz (1996). In all of these volumes, there are chapters on comparative epidemiology. The epidemiology of plant virus diseases is covered comprehensively in the volume edited by McLean et al. (1986) and papers by Thresh (1974a, 1976, 1983). For particular aspects of soilborne and root diseases, the volume by Campbell and Benson (1994a) should be consulted. The proceedings of a symposium edited by Palti and Kranz (1980) are devoted entirely to comparative epidemiology.

Epidemiology is obviously concerned with epidemics, that is any increase or decrease in disease intensity y in the range $0<y\leq1$ (or 100%) in time and space (Kranz 1974b). This comprises Gäumann's (1951) classical definition of epidemics adopted from human medicine, i.e. a steep temporal or spatial increase in disease followed by a decline within a limited period of time. Within the context of crop protection, epidemiology is the research interface between laboratory research and actual disease management in the field. Comparative epidemiology can provide information, amongst others, for the design of integrated pest management (IPM) schemes, from the behaviour of epidemics and the reactions of their components to weather factors and control measures. Finally, comparative epidemiology provides generally valid information for teaching and textbooks. For this more communicational application, an unequivocal language and terminology is particularly required (Sect. 3.1).

Through systems analysis, comparative epidemiology can be used to develop tactics and strategies for a more efficient, economic and sustainable management of disease. Plant diseases are open, coupled dynamic systems regulated by external factors (Kranz 1974b; Robinson 1976; Kranz and Hau 1980). The systems are interlocking processes determined by many reciprocal cause-and-effect relationships that characterise biological systems (Watt 1966), as is shown schematically in a simple relational diagram (Fig. 2.1). Stimuli affect the structural elements of disease progress curves (see also Fig. 5.2) and disease gradients directly, or through other (often preceding) elements, either positively or negatively. Depending on the type of stimuli and the weight they have on the various elements of the structure, different behaviour outputs are produced by the system. Flow charts