Comparative epidemiology

A tool for better disease management

Proceedings of the session on
Comparative epidemiology,
3rd International Congress of Plant Pathology

Munich, 16–23 August 1978

Editors: J. Palti & J. Kranz

Centre for Agricultural Publishing and Documentation
Wageningen–1980

ISBN 90-220-0721-9

Cover design: Pudoc, Wageningen

Printed in the Netherlands

Preface

The aim of the session on comparative epidemiology was to discuss methods for and approaches to the fuller use of epidemiological observation and research for better disease management. More particularly, the value of comparative studies in epidemiology was to be brought out, and ways were to be shown for reducing the multitude of disparate phenomena relating to epidemics of numerous diseases to fewer concepts of wider application to the benefit of more rational crop protection.

Those who profited most immediately from these discussions seemed to be the contributors themselves: the majority of the papers presented here have been thoroughly revised after the congress, with a great deal of thought invested in these revisions. The editors have, in addition, felt that the six papers offered in the session of the congress could be usefully complemented by two papers read in other sessions; in these papers some of the more immediate applications of comparative epidemiology to farming are outlined.

The contributions to this volume are being presented in the following sequence: The first paper (Zadoks & Schein) defines the position of comparative aspects of epidemiology and its relation to ecology and plant disease management. The two papers of Kranz and Butt & Royle outline various concepts, approaches, and definitions to be applied to comparative epidemiology as a scientific tool. This is followed by three papers of Aust et al., Thresh and Jones that review interactions of biotic and environmental factors in the development of epidemics due to fungi, viruses and nematodes; these papers illustrate the application of the principles involved to various systems levels. In conclusion, the papers of Putter and Rotem & Palti present practice-oriented comparisons of the use of epidemiological concepts in chosing ways of reducing the economic impact of disease in tropical subsistence farming and in more advanced farming economies.

The contributions offered here do not pretend to cover the entire field of comparative epidemiology. Thus, no comparisons are made of various pathosystems within one crop, or of the components of epidemics in field crops and plantations etc. Brief reference only is made to the potential use in comparative epidemiology of data gained from research or observations made in other contexts.

Nevertheless, it is hoped that this volume will stimulate thought and provoke future research, so that practical solutions to urgent disease control problems may be derived from this approach to the science of plant pathology.

J. Palti
J. Kranz

Contents

Epidemiology and plant-disease management, the known and the needed

J. C. Zadoks and R. D. Schein

Abstract

Epidemiology as a science stands at the cross-road of problem-oriented phytopathology and principle-oriented ecology. Epidemiology operates at three integration levels: individual, population, community. Comparative epidemiology can use several 'tools' such as components analysis, life table statistics, the 'epidemiologic quintuplet', and dynamic simulation, with which life strategies of pathogens can be analyzed and pathosystems can be modelled. Plant disease management is the practical application of epidemiologic knowledge; it operates at entrepreneurial and collective strata, in the pre-planting and the post-planting mode. Epidemiologists are staff officers and not managers; they report alternatives from which managers can choose, taking into regard numerous non-epidemiological constraints. Epidemiology has extended its scope thanks to influxes of ideas and people from other sciences, among which are computer science, genetics, and ecology. Some possible future developments are indicated, as is the need for a 'comprehensive' approach.

Key words: Epidemiology, plant disease management, pathosystem, ecology, future development.

Introduction

'The known and the needed' in epidemiology is our theme. During the 'Third International Congress of Plant Pathology', the known has been extended and deepened so much, that it seems superfluous to dwell with the 'known'. The 'needed' has been indicated by so many distinguished speakers, that little – it seems – can be added. The known and the needed in epidemiology are well covered by recent publications among which Horsfall & Cowling's (1978) advanced treatise on plant disease Vol. II 'How Disease Develops in Populations' merits special mention. When, nevertheless, we want to discuss the known and the needed in epidemiology, our theme must be placed in a wider context. Our endeavour is not to discuss the content matter of epidemiology but rather to formulate its present position in the area where theory and practice meet, and merge. In doing so we may proceed from a retrospective to a prospective view.

Our discussion will consist of three parts. In the first part we shall indicate the position of epidemiology as a science. In the second part we shall discuss the uses of comparative epidemiology. In the third part we will touch upon the relation between epidemiology and plant disease management.

The ideas exposed here in brief will be explained in detail in a book entitled 'Epidemiology and Plant Disease Management' that has appeared recently (Zadoks & Schein, 1979).

The position of epidemiology

To determine the position of epidemiology in the field of biological sciences we must recall the hierarchy of integration levels in biology.

Levels of integration in biology

What is an integration level? To explain this concept let us make an excursion into the area of cybernetics. If a system is brought out of balance, it needs time to restore that balance. The span of time needed is characterized by a value called 'relaxation time'. A climax forest partly destroyed by fire has a relaxation time measured in years or decades. Algal cells suspended in a nutrient solution adjust to changes in the environment in minutes or, at most, in hours. Evidently, the relaxation times of the climax forest ecosystem and the cell suspension system differ by several orders of magnitude. Phenomena with relaxation times of about the same order of magnitude belong to the same integration level. In biology, a hierarchy of integration levels can be designed as in Table 1.

Table 1. An example of a hierarchy of integration levels in biology.

Community	ecology
Population	population dynamics, p. genetics
Individual	anatomy, physiology
Organ	
Cell	histology
Organel	molecular biology
Molecule	

What is the position of epidemiology within this hierarchy? There is no epidemic without a population of host plants and a population of infectious units (or 'individuals') of a pathogen. So epidemiology is a science operating at the population level. Vanderplank (1963) has made this point explicitly in his famous 1963 book (Page 2) 'Epidemiology is the science of disease in populations. Sometimes one needs to distinguish between the study of disease in populations and the study of populations of pathogens, ...'

To understand phenomena at the population level we have to descend one integration level and to study the behaviour of individuals. With respect to pathogens, studies at that level have been summarized by famous authors like Ingold (1971) and Gregory (1973). For plants we must refer to current text books.

Going one more level downward, we enter the domain of plant and fungus physiology. Most epidemiologists avoid entering this domain, but at times they feel forced to do so. Rijsdijk (in press) explains phenomena at the population level by changes in host plant and fungus physiology; he states that the physiological condition of the host plant has become an essential element in an experimental disease management system called EPIPRE (Rijsdijk & Zadoks, 1978). Studies at the integration level of physiology we do not regard as part of epidemiology, but to acknowledge the explanatory value of

findings at this level for epidemiology (and for ecology at large) we recall the term 'ecophysiology' (Van der Wal & Cowan, 1974).

Whereas Vanderplank laid emphasis on populations, Robinson directed our attention to the community level. He coined the term 'pathosystem' to indicate the host-pathogen community (Robinson, 1976). A pathosystem consists of at least two interacting populations, operating at different trophic levels, those of host and parasite.

Pathosystems

For natural pathosystems the classical disease triangle (Fig. 1) provides an adequate picture. However, plant pathologists occupy themselves – too exclusively – with agricultural or managed pathosystems. In managed pathosystems man changes the functioning of the pathosystem profoundly by his crop husbandry, resistance breeding, and chemical control, and therewith man makes himself an essential part of the interactive community studied in epidemiology. Man becomes part of the pathosystem, because the pathogen reacts to him as much as he reacts to the pathogen; the disease triangle becomes a disease tetrahedron (Fig. 2). Man creates the 'boom-and-bust' cycle, and man devaluates costly chemical compounds by soliciting tolerance in the fungi through overspraying. Gradually, however, epidemiologists gain an understanding of the managed pathosystem and its behaviour in the long run (Browning, 1974; Robinson, 1976).

The paragraph on relaxation times clearly indicated that there is a relation between the time span of a process and its integration level. Processes at the individual level are, as a rule, monocyclic processes; they are measured in hours, sometimes in minutes or days. Examples are the duration of time needed by a spore to germinate, the latent period, and so on. Exceptions are the shooting of ascospores, to be measured in milliseconds, but here we touch upon the integration level of the cell where relaxation times are short indeed. Processes at the population level are, usually, polycyclic processes to be measured in days, like Vanderplank's r value, the apparent infection rate, expressed in units per unit per day. At the community level, finally, time is conveniently

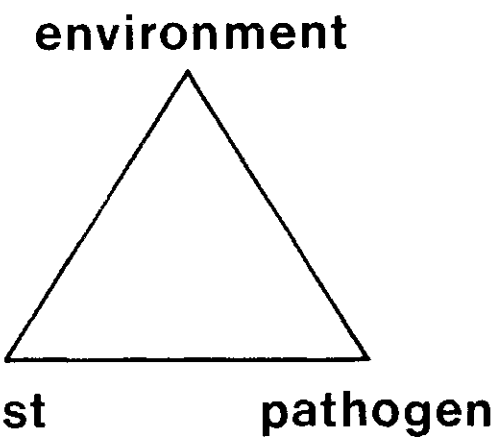

Fig. 1. The disease triangle. Host, pathogen, and environment mutually influence each other.

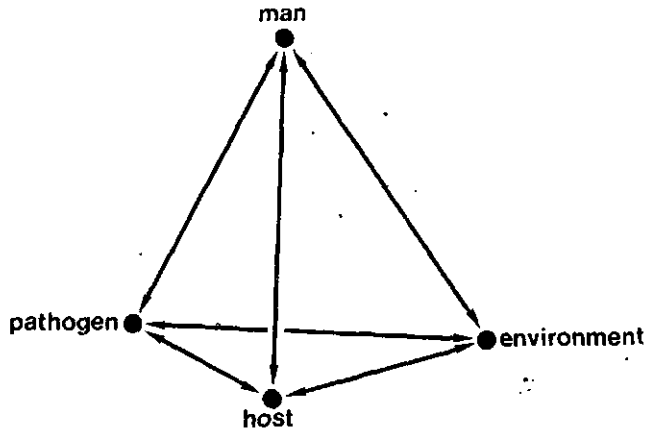

Fig. 2. The disease tetrahedron. Its base is formed by the disease triangle. Man can affect any point of the base plane, but the pathosystem will react and by its reaction it will affect man. So man becomes part of the managed pathosystem.

expressed in the number of generations of the host plant, that is – for annuals – in years; we speak of polyetic processes, that is, processes extending over many years (Zadoks, 1974).

We suggest that in the future epidemiologists spend part of their time studying community processes. New areas will be opened. Theoretical studies by Vanderplank (1963, 1968) and Robinson (1976) paved the way. Detailed analyses of phenomena observed in the field using population genetics as a tool have added to our knowledge (Wolf & Barrett, 1977). Browning's 1974 summary of work done in Israel deserves special mention; it points towards the usefulness of studying natural pathosystems in the wild.

The studies indicated fall under the heading of polyetic studies, studies of the development of an epidemic over the years. Entomologists, nematologists (Jones, this volume), and plant pathologists working with soil-borne diseases are familiar with the idea that parasitic populations build up and decline over the years, apart from any seasonal fluctuations. Epidemiologists working on foliar pathogens tend to overlook polyetic effects. Computer simulations of polyetic effects have been made by Kiyosawa (Kiyosawa & Shiyomi, 1976; Kiyosawa & Yabuki, 1976) and Rijsdijk (1975).

Positioning epidemiology

The epidemiologist is a plant pathologist who thinks in terms of populations, communities and environmental effects, be they of biotic or abiotic nature. His mind is pervaded by ecological thinking. In an opening address to the NATO Advanced Study Institute 'Epidemiology of plant diseases', held in Wageningen, 1971, A. J. P. Oort described epidemiology as a 'branch of ecology dealing with ecosystems in which a predatory, parasitic or pathogenic relationship exists between an organism and its host' (Butt, 1972).

We regard phytopathology as a multilevel but practical and problem-oriented

science. We see ecology in the first place as a principle-oriented science, as evidenced by the motto chosen for the First International Congress of Ecology, The Hague, 1974, 'Unifying concepts in ecology' (Van Dobben & Lowe-McConnell, 1975).

We position epidemiology at the cross-roads of phytopathology, primarily a problem-oriented science, and ecology, primarily a principle-oriented discipline. For too long a period, epidemiology has developed within phytopathology, apart from the main stream of ecological thinking. Now, epidemiologists can learn much from ecology.

Conclusions

1.1. Epidemiology stands at the cross-road of the problem-oriented but multileveled science of phytopathology and the principle-oriented discipline of ecology (Fig. 3).
1.2. Epidemiology, to remain a consistent and applicable body of knowledge, must limit itself to the study of pathosystems at three integration levels: those of the individual, the population, and the community, studying processes that are, respectively, monocyclic, polycyclic, and polyetic (Fig. 4).
1.3. In the next few years epidemiology must spend much effort in polyetic studies of natural and managed pathosystems, at the community level (Fig. 5).
1.4. Epidemiologists should divide their time more evenly between pathogen and host.

Comparative epidemiology

Long, too long, epidemiologists have lingered at details, details of fungus *a* on host *b* in environment *c*. Such honourable and indispensable studies often tend to lead from one special question to the next and more specialized question. If a researcher is caught and encapsulated in this process, his way toward alternative views may be blocked. Comparative epidemiology can serve as a counterpoise and offer an approach to alternatives. We will add our views on comparative epidemiology to those later explained by Kranz (this volume) and others.

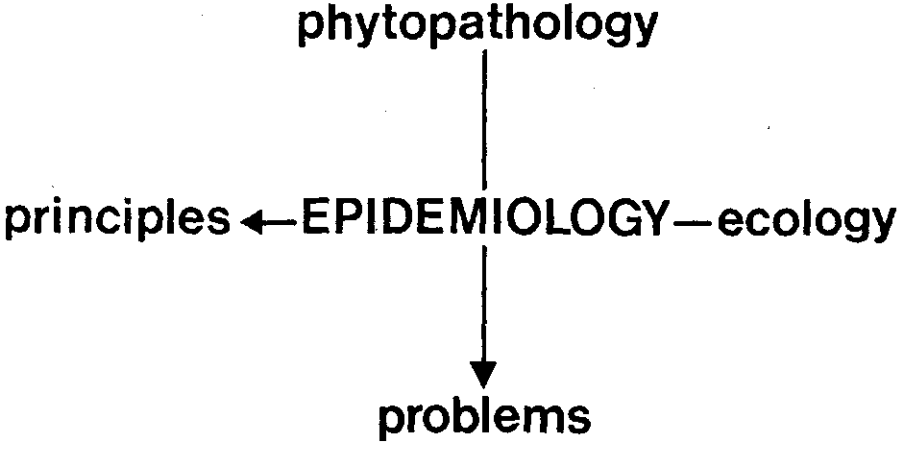

Fig. 3. The position of epidemiology in the field of sciences.

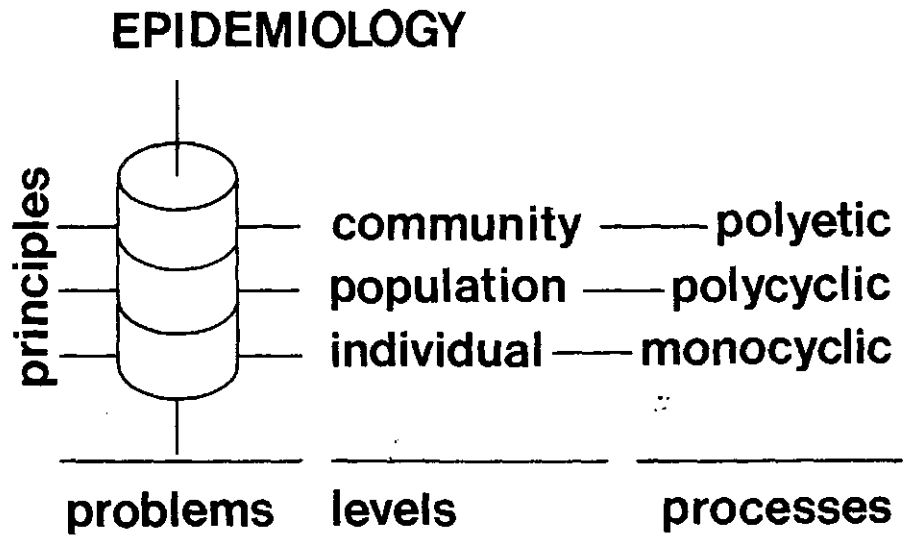

Fig. 4. Integration levels covered by epidemiology.

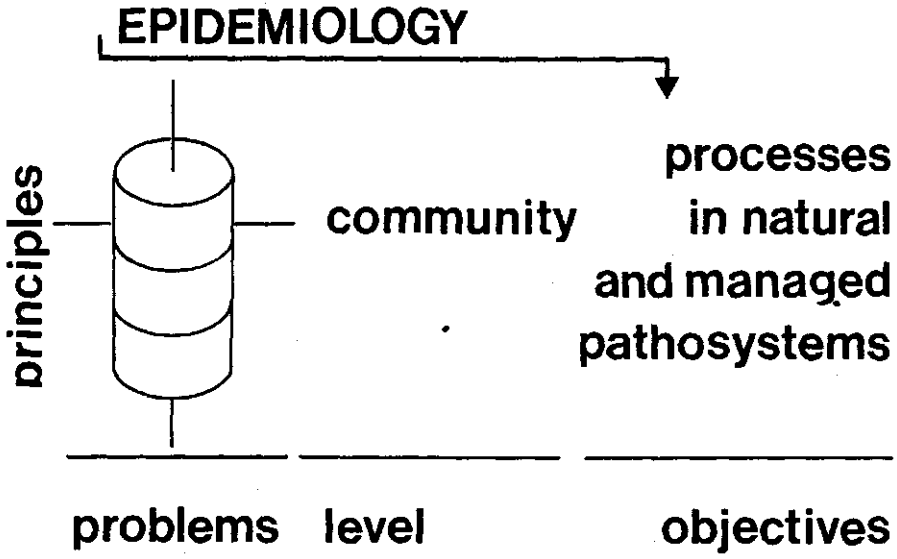

Fig. 5. Suggested future research.

The level of the individual

The behaviour of pathogens can be studied at the individual level by means of 'components analysis' (Zadoks, 1972). In components analysis measurable phases of the infection cycle are studied quantitatively. It is possible and, perhaps, sometimes necessary to combine two or more successive phases into one measurement. Ideally, the end result of the infection cycle is the algebraic function of all identifiable components. The smallest identifiable and measurable components of the infection cycle can be regarded as the basic elements of fungal behaviour.

Table 2. Components that can be measured in a components analysis.

Infection efficiency
Latent period
Lesion growth
Infectious period
Number of spores per lesion per day

Comparisons can be made in many ways. Two isolates can be compared on one host cultivar; any differences found are attributed to differences in components of virulence within the pathogen. The responses of two cultivars can be compared by testing them with the same isolate; the differences found between cultivars are ascribed to differences between hosts in components of resistance. When one cultivar-isolate combination is tested in two different environments, any differences found indicate differential effects of environmental factors on pathogen and/or host or their interaction.

By simplistic or refined components analysis varietal resistance, horizontal and vertical, is tested, physiologic and ecologic races are identified, and environmental effects, whether of weather, fertilizers, or fungicides, are determined.

Choosing the right components to be measured, the transition from the individual level to the population level can be made by applying a technique well-known in animal ecology, the life table technique (Zadoks, 1977). This technique permits to obtain generalized values useful in comparative epidemiology, such as:

r_{max}	= intrinsic growth rate of the population	
	= maximum relative growth rate of the population	$[T^{-1}]$
T_g	= mean length of a generation	$[T]$
R_0	= net reproduction rate per generation	[1]

The value r_{max}, the intrinsic growth rate as the ecologists say, is a relative rate of increase, as is Vanderplank's apparent infection rate r, and it is also expressed in units per unit per day, with dimension $[T^{-1}]$. It indicates the maximum possible value of r, a value never obtained because many spores are lost or otherwise non-functional.

Values of r measured in the field in polycyclic experiments are indeed lower than those calculated by means of life table statistics obtained from monocyclic experiments, but interestingly they are of the same order of magnitude. In brown leaf rust of wheat (*Puccinia recondita* Rob.) r_{max} values were up to about 0.6, whereas r values observed in the field rarely exceeded 0.2.

The level of the population

We have now entered the next higher integration level, that of the population, where epidemiologic processes are, often but not always, polycyclic processes. Again there is a basis for quantitative comparison. Comparison can begin with empirical r values, but such comparison will not satisfy in the long run because the information is inadequate for analytical purposes.

We fare better with Vanderplank's (1963) analysis of the rate of increase of an epidemic, in which

$$\frac{dx_t}{dt} = R_c \cdot (x_{t-p} - x_{t-i-p}) \cdot (1 - x_t)$$

where p is the latent period and i the infectious period. The value R_c, the corrected basic infection rate, is the product of two variables, the daily spore production per lesion N and the effectiveness of the spore E or the proportion of the spores that will effectively lead to new sporulating lesions. If we add a necessary piece of information, x_0, the proportion diseased at the beginning of the epidemic, we have five values that together completely describe the polycyclic epidemic: the 'epidemiologic quintuplet'.

Comparative epidemiology at the population level can characterize the life strategy of every pathogenic fungus by means of these five values. The concept 'strategy of life' has been developed in ecology.[1] Consider the logistic equation[2] as the ecologist use it:

$$\frac{dN}{dt} = r \cdot \frac{N}{K} \cdot (K - N)$$

in which N is the number of individuals of the population, K is the maximum possible number or the 'carrying capacity' of the environment, and r is the relative growth rate of the population. Ecologists state that there are two major life strategies called the r and the K strategies. The r strategy calls for a rapid reproduction, a high r value, whenever there is an opportunity; individuals are small, short-lived, and they give little care to the young. Under favourable conditions overcrowding occurs. K strategists, on the other hand, are relatively large and long-lived; they are slow breeders which care well for their young. They usually live in relatively low numbers and in fair equilibrium with their environment; there is no overcrowding. Lemmings are r strategists, and so are the cereal rusts; elephants are K strategists and so are various soil-borne diseases as, for example, *Sclerotium rolfsii* Sacc.

Evidently, different groups of pathogenic fungi follow different life strategies. We claim that life strategies of phytopathogenic fungi can be expressed in terms of our 'epidemiologic quintuplet' (Fig. 6). It seems to us that fungi offer an excellent opportunity for life strategy studies; there probably exist more strategies than the two extreme types mentioned, the r and the K strategies. Note that the distinction made by Jones (this volume) of two types of nematodes, the 'exploiters' and the 'persisters', is relevant here Thresh (this volume) is concerned with strategies of survival among viruses.

1. Thresh (page 66) and Jones (page 73) also discusses this concept.
2. An alternative form of the same logistic equation is used by Thresh on page 58 of this volume.

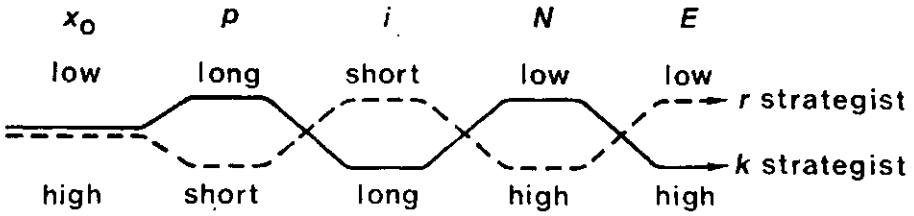

Fig. 6. The r and K life strategies expressed in terms of the epidemiologic quintuplet.

The level of the community

Can we continue our reconnaissance of comparative epidemiology and extend it to the integration level of the community? We can, but maintaining our standards of quantitative analysis, we must now enter the area of qualitative characteristics, the area of genetic variation, where we encounter specific genes for virulence in the pathogen toward resistance genes in the host and specific genes for tolerance in the pathogen toward active compounds in fungicides.

Powerful tools for comparative studies are provided by population genetics. Studies in this area are highly necessary; they promise a rich harvest.

Mackenzie (1978) and others (Leonard, 1969; Van Leur, unpublished) have demonstrated that competition between isolates of different fitness under the actual managerial status of the pathosystem leads to changes in genotype frequencies in the course of time according to the well known logistic function, with r_d instead of r:

$$r_d = \log_e (1 - s)$$

where r_d is the relative rate of disappearance of the weaker strain and $(1 - s)$ is the fitness of the weaker strain relative to the stronger one.

Comparative epidemiology at the community level has to take care of the problem of scale. A conclusion from an experiment may be valid for the experimental area at a certain instant, but is it also valid for the area of a state, for a span of time covering several years? Scaling effects are well known in industry; processing technology has developed methods to tackle problems of scale. Epidemiologists just began: whereas Zadoks & Kampmeijer (1976) assumed that in spore dispersal one principle is applicable to various scales of distance, Rijsdijk (1979) showed that one model of spore dispersal applied to different scales of distance leads to different conclusions.

It is the writers' opinion that during the following years available tools must be used to shape comparative epidemiology, which at present is a pertinent 'need' but certainly not a conspicuous 'known'. Epidemiologists should extend their efforts to pathosystems hitherto considered to be 'difficult', like those involving perennial crops and those involving bacteria and viruses.

Conclusions

2.1. At the level of the individual, comparative epidemiology can use two tools, components analysis and life table statistics; they lead to characteristics of general value: r_{max}, T_g and R_0 (Fig. 7).

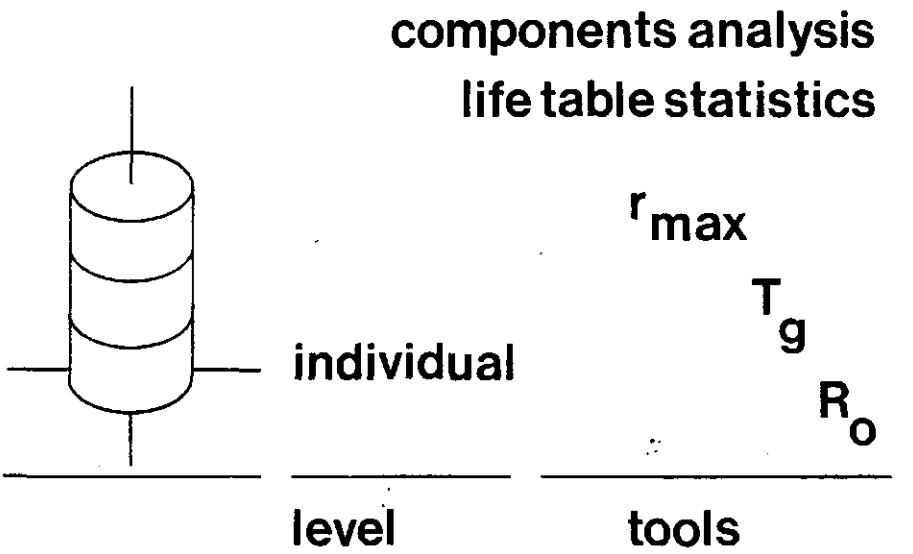

Fig. 7. Tools of comparative epidemiology at the individual level.

2.2. At the level of the population, comparative epidemiology can make use of the 'epidemiologic quintuplet': x_0, p, i, N and E (Fig. 8); existing data can be re-used when viewed from this new perspective.

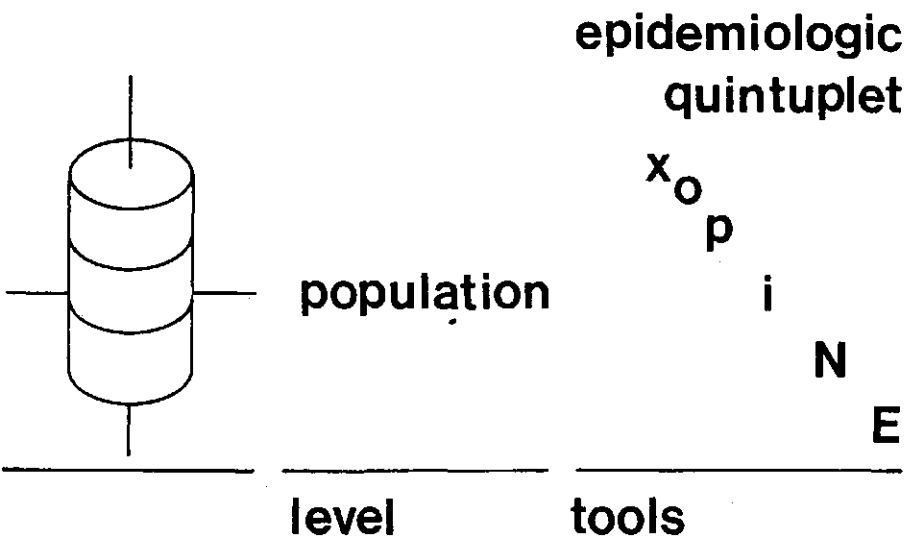

Fig. 8. Tools of comparative epidemiology at the population level.

2.3. At the level of the community, comparative epidemiology is *in statu nascendi*; dynamic simulation will undoubtedly be a major tool (De Wit & Goudriaan, 1978); problems of fitness and problems of scale must be tackled (Fig. 9).
2.4. During the next few years the available tools must be used to shape comparative epidemiology.

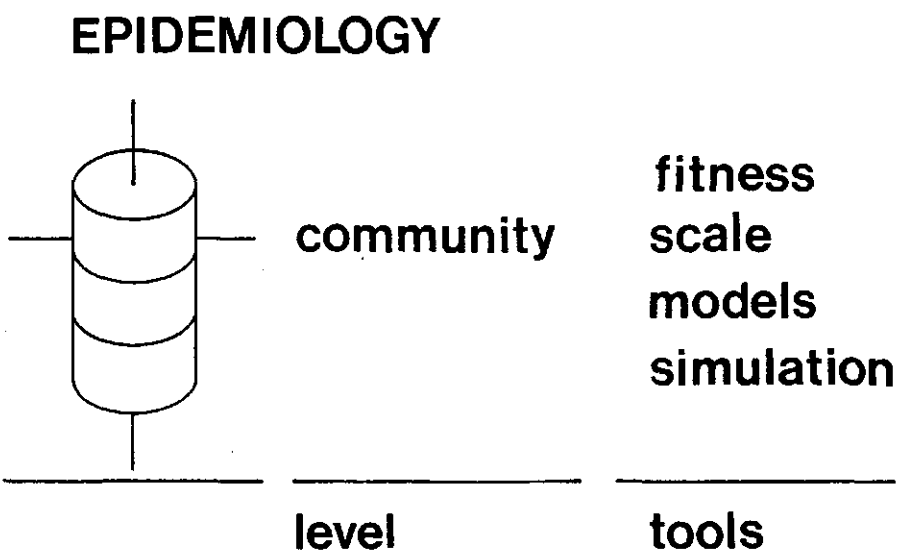

Fig. 9. Suggested tools of comparative epidemiology at the community level.

Plant disease management

Epidemiology as a science has acquired a body of knowledge which must be put into practice. Its practical application is, or is at least part of, plant disease management. We stick to the term plant disease management, though the term pathosystem management, with pathosystem used in the wide sense including man himself, is a more appropriate term. Epidemiology and plant disease management belong together as the two sides of a coin; the former has no social relevance without the latter; the second is no longer possible without the first.

Definitions

Talking about plant disease management is fashionable nowadays, but these talks do not solve problems. Let us see what is meant by plant disease management, taking Webster's dictionary as a guide. We will pass by the medical meaning of management 'the whole system of care and treatment of a disease or a sick individual' as being too narrow, and we choose: 'the conducting or supervising of something as a business, the executive function of planning, organizing, coordinating, directing, controlling and

supervising any industrial or business project or activity with responsibility for results'. That puts us right on the spot. Clearly, we, epidemiologists, are not managers, cannot be managers, as we have no executive function with responsibility for results.

The task of the epidemiologist is not 'directing, controlling or supervising', but a far more modest activity. This is not a line function but a staff function (Fig. 10), rendering assistance to the responsible manager in supervising, planning, organizing, and coordinating. The tools are description, analysis, synthesis, prognosis, and comparison. The epidemiologist should design alternative approaches, based on comparative epidemiology, from which the manager can choose.

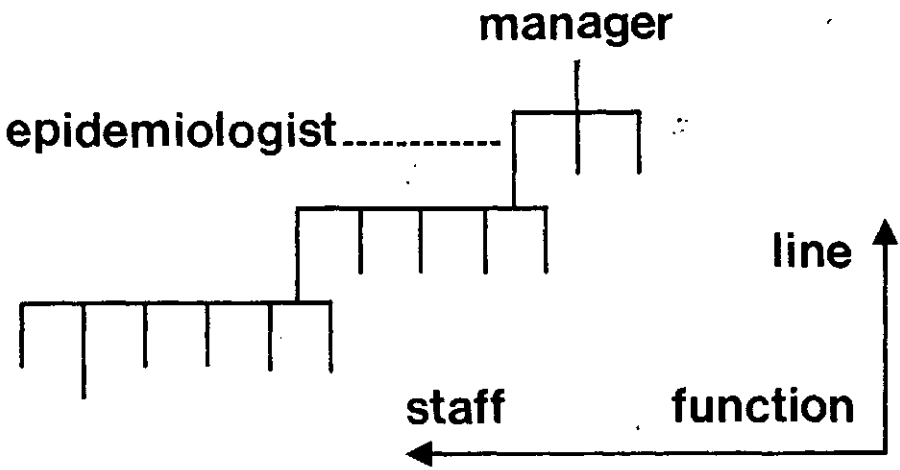

Fig. 10. The position of the epidemiologist.

Management seems to take place at two main strata, that of the individual entrepreneur and that of the collectivity. Individual entrepreneurs, single and as a group, may have views and interests different from those of the collectivity. The collectivity is usually represented by government. With respect to disease management there are two modes of operation, the pre-planting and the post-planting mode (Fig. 11).

PLANT DISEASE MANAGEMENT

modes		pre-	post- planting
strata	collective		
	entrepreneurial		

Fig. 11. Strata and modes of plant disease management.

The entrepreneurial stratum

The pre-planting mode deals with the choice of crop and cultivar (where rotation and therefore x_0 problems may appear), the choice of tillage including zero-tillage (again an x_0 problem), and the choice of genotype (single genotype or polygenotype crop, eventually a multiline crop, a problem in x_0 and r). In the Netherlands, we are attempting to advise the use of 'polygenotype crops' (mixed cultivar plantings). If we learn anything it is modesty. The matter is extremely complicated, in its legal, agronomical, technical and epidemiological aspects. So-called 'non-target' diseases especially may cause trouble (Groenewegen & Zadoks, 1979).

The post-planting mode deals with the damage threshold, a problem in x, and timing of treatment. We should stress the adage: 'treat as little as you dare'. To this purpose the system of negative forecasts should be extended. The German PHYTPROG potato late blight warning system contains a negative forecast (Burckhardt & Freitag, 1969), as does the American BLITECAST (Krause et al., 1975). The Netherlands program EPIPRE, an attempt to develop a disease warning system for cereals, also emphasizes negative forecast (Rijsdijk & Zadoks, 1978).

In positive forecasts, a clumsy term for the advice to treat, we recommend to strive for individual advice, tailored to each grower or even every separate field. The well-known Mills apple scab (*Venturia inaequalis (Cke)* Aderh.) warning system is a classical example of an epidemiologist helping growers to help themselves. The EPIPRE program tries to give advice for every field contained in its data bank, taking into regard geographic position, soil, cultivar, sowing date, fertilizer usage, and preceding fungicidal treatments. BLITECAST is a customer-designed, computerized warning system. Originally, it was a computer-operated system with the operator as a spider in her web of telephone cables, but recently BLITECAST has been condensed to a thousand dollar black box, complete with sensors and programmed calculator, which the farmer can place in his field. By pushing a button the farmer obtains advice based upon local within-field conditions. The system is completely decentralized, individualized (Mackenzie & Schimmelpfennig, 1978).

The difference between BLITECAST and EPIPRE is that the farmer must assess the initial disease level in his field. Involving the farmer again by making him responsible for his own disease rating seems to have a positive psychological effect, motivating him for optimal disease control (in contrast to maximal disease control). We strongly recommend individualization of disease warning systems, where the epidemiologist remains a background adviser, so that the grower can take his own decisions as befits a manager.

The collective stratum

At the collective stratum, where the same two modes of operation, the pre-planting and the post-planting, exist, epidemiologists should be far more careful because at present their experience is incommensurate with the scope of their theories. The theories are wonderful, but reliable facts are scanty and scarce.

Field experiments are always performed on a small scale. Because of scaling effects, indicated before, a result found to be good in experiments on a small scale is not necessarily good when applied in general practice on a large scale. A classical example is chemical treatment, perfect on the experimental scale but often yielding unexpected

side-effects, pollution of the environment, health hazards, and the appearance of tolerance in pathogens. Another classic is the 'boom-and-bust' cycle, where cultivars promising during the introductory phase succumb rapidly to new genotypes of the pathogen. Problems of scale have never been studied systematically in epidemiology. Advice at the collective stratum without appropriate knowledge of scaling effects is irresponsible.

Selected examples

A few plant disease management systems have already been proposed or even tested. We limit ourselves to some selected examples.

Gene management systems (Frey et al., 1977) should be introduced with great care. A major problem may arise from non-target diseases. One of the Netherlands experiments with a wheat multiline variety developed against yellow stripe rust (*Puccinia striiformis* West.) succumbed to brown leaf rust (*P. recondita*) (Zadoks & Groenewegen, unpublished). Those who see a specific goal far ahead are often short-sighted with respect to other and, in their eyes, secondary problems. Notwithstanding this reservation we feel that polygenotype cultures are a major epidemiologic issue for the next five years (Groenewegen & Zadoks, 1979); at the same time the gaining of approval from the seed regulation authorities and acceptance by the farmers are major managerial issues. We include in this statement systems of gene management combined with restricted fungicide application schedules (Wolfe & Barrett, 1977).

The economic life of a fungicidal compound is limited by, among other things, the phenomenon of tolerance in the fungus. This is a loss to the chemical company as well as to the farming community. In the future, chemical management may become another activity in which epidemiologists with an interest in population genetics could participate. Again, there is a problem of non-target diseases to be dealt with.

Disease warning systems at the collective level, a classical occupation of epidemiologists, should stress two points, 1. negative forecast, and 2. stand-by warnings. Other warnings and management decisions should be transfered to the individual level whenever possible.

The comprehensive approach

No staff epidemiologist may forget that the manager always deals with a great number of production constraints. During the Third International Congress of Plant Pathology the word 'comprehensive' has often been used to indicate that there is usually a basket-full of constraints. Epidemiologists have not been trained in 'comprehensive' thinking, on the contrary, current phytopathological training is geared to the one-disease-at-a-time approach. Statistically significant and economically important interaction between constraints is well known by now: an extra difficulty for the comprehensive approach. Epidemiologists with a managerial pursuit must learn to think comprehensively. How to learn this is a problem of the future that needs to be solved. In the experimental study EPIPRE, (see above) the problem will be tackled by filling the basket with solutions for one constraint after the other. Maybe, a revival of teaching the old-fashioned 'crop husbandry' provides a solution.

Conclusions

3.1. Epidemiology and plant disease management relate to each other as two sides of a coin; acquisition of knowledge versus application of knowledge.
3.2. Epidemiologists are not managers; in plant disease management epidemiologists do not have a line function but a staff function.
3.3. In plant disease management there are two strata of operation, the entrepreneurial and the collective, and two modes of operation, the pre-planting and the post-planting.
3.4. Efforts at the entrepreneurial stratum in the post-planting mode should be directed towards decentralization, with individual advice to each grower separately, or – alternatively – toward helping the grower to help himself.
3.5. Efforts at the collective stratum should be in the pre-planting as well as in the post-planting mode, directed towards avoidance of chemical treatment where possible, and optimization of chemical treatment where it is unavoidable.
3.6. Epidemiologists will become effective staff officers only if they can quantify policy alternatives by means of the methods of quantitative comparative epidemiology, taking into regard eventual scaling effects.
3.7. The basket-approach to comprehensive disease management is a problem of the not-too-distant future.

Summary

In the absence of sufficiently sophisticated epidemiology, breeding was for reduced x_0. An euphoria of success permeated our field. What happened, the actual behaviour of disease, was studied by epidemiology. Explanations of behaviour were provided by genetics. Certain of those capable of genetic work became interested and proficient in epidemiology. They asked: How can we manage disease? They predicted how disease would behave with certain kinds of genetic manipulation, for instance partial resistance, gene deployment, polygenotype crops. This was epidemiology. It brought up questions of pathogen adaptation and opened the area of fitness inquiries. Controversy ensued and continues. The new breed of epidemiologists looks for ways to stabilize disease by genetic manipulation of the host in such a way as to stabilize the parasites' genetics. These are strategies. They are modelled and tested. If applied, they become tactics of disease managements. Their importance in future is enormous. The progress made would not have occurred without epidemiology. And without the other fields, ecology, genetics, computer science, and so on, epidemiology would not be where it is today. This interdependence between epidemiology and other areas extends itself. Fungicide specialists today use epidemiology and contribute to it.

We have explained that epidemiology is a science bridging three levels of integration: the individual, the population, and the community. Such is already more than the average scientist can handle; it has been stated that most people can only switch easily between two adjoining levels of integration in a cause-and-effect relation. The reason is obvious; every level of integration necessitates other auxiliary sciences. Epidemiologists usually solve the problem unconsciously by becoming problem-oriented.

At higher levels of integration new problems appear and the importance of knowledge at the lower levels seems to fade away. But that effect is only optical illusion, to which many students and not a few professors fall a victim. Detailed knowledge at the

lower and middle level is, and will remain, indispensable for appropriate action at the upper level, for which little factual knowledge exists. We stress the need to proceed gradually and patiently from one level to the next, and backwards again. Old experiments must be reconsidered, old data recalculated (Kranz, this volume). Desk research is respectable if its results are repeatable. It can save time and money, and it can help us avoid spurious strategies.

Comparative epidemiology provides the tool-kit with which speculations about disease management strategies can be transferred into quantified and testable hypotheses, which after due testing can become elements of disease management tactics. Such tactics must be compatible with other objectives within the array of total crop management. Managers, at both the entrepreneurial and collective strata, choose the tactics suitable to them. To facilitate such a choice anthropomorphic terms such as pathogen behaviour, life strategy, and pathosystem behaviour are acceptable. In the implementation and supervision of the tactics chosen, epidemiologists have a pertinent though modest role to play, thus serving science and society.

References

Browning, J. A., 1974. Relevance of knowledge about natural ecosystems to development of pest management programs for agro-ecosystems. Proc. Am. Phytopathol. Soc. 1: 191–199.

Burckhardt, H. & E. Freitag, 1969. Die Phytophthora-Negativ-Vorhersage. Der Kartoffelbau 20: 176–180.

Butt, D. J., 1972. Epidemiology of plant diseases. A commentary on the 1971 Advanced Study Institute. Rev. Plant Pathol. 51: 635–638.

Dobben, W. H. van & R. H. Lowe-McConnell, 1975. Unifying concepts in ecology. Junk, The Hague & Pudoc, Wageningen, 302 pp.

Frey, K. J., J. A. Browning & M. D. Simons, 1977. Management systems for host genes to control disease loss. Ann. New York Acad. Sci. 287: 255–274.

Gregory, P. H., 1973. The microbiology of the atmosphere. Blackey Int. Texts, Bishoprigg, Glasgow.

Groenewegen, L. J. M. & J. C. Zadoks, 1979. Exploiting within-field diversity as a defense against cereal diseases: a plea for 'polygenotype' varieties. Proc. Int. Wheat Genetics Conf., Delhi. Indian. J. Genet. Pl. Breed.

Horsfall, J. G. & E. B. Cowling, 1978. Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, 436 pp.

Ingold, C. T., 1971. Fungal spores, their liberation and dispersal. Clarendon Press, Oxford, 302 pp.

Kiyosawa, S. & M. Shiyomi, 1976. Simulation of the process of breakdown of disease-resistant varieties. Japan. J. Breed. 26: 339–352.

Kiyosawa, S. & S. Yabuki, 1976. Modeling on the race frequency change in a host-pathogen system with genes for resistance and avirulence. Japan. J. Breed. 26: 237–246.

Krause, R. A., L. B. Massie & R. A. Hyre, 1975. Blitecast: a computerized forecast of potato late blight. Pl. Dis. Reptr. 49: 95–98.

Leonard, K. J., 1969. Selection in heterogeneous populations of *Puccinia graminis* f. sp. *avenae*. Phytopathology 59: 1851–1857.

Mackenzie, D. R., 1978. Letter to the editor. Estimating parasitic fitness. Phytopathology 68: 9–13.

Mackenzie, D. R. & H. G. Schimmelpfennig, 1978. Development of a microcomputer unit for forecasting potato late blight using the Pennsylvania State University's Blitecast system (Abstract). Am. Potato J. 55: 384–385.

Rijsdijk, F. H., 1975. A simulator of yellow rust of wheat. Bull. Rech. Agron. Gembloux: 411–418.

Rijsdijk, F. H., 1978. A model evaluation of spore dispersal inside and above canopies. Proc. 1st Int. Conf. Aerobiology, Munich. In press.
Rijsdijk, F. H., 1979 Systems analysis at the cross-roads of plant pathology and crop physiology. Z. Pflansenkrankheiten. In press.
Rijsdijk, F. H. & J. C. Zadoks, 1978. EPIPRE, een poging tot geleide bestrijding van graanziekten met gebruik van een computer. Verslag Graanziektendag 1978, Wageningen, p. 51–59.
Robinson, R. A., 1976. *Plant pathosystems. Springer, Berlin, 184 pp.*
Vanderplank, J. E., 1963. Plant diseases: Epidemics and control. Acad. Press, New York, 349 pp.
Vanderplank, J. E., 1968. Disease resistance in plants. Acad. Press, New York, 206 pp.
Wal, A. F. van der & M. C. Cowan, 1974. An ecophysiological approach to crop losses, exemplified in the system wheat, rust and glume blotch. II. Development, growth, and transpiration of uninfected plants and plants infected with *P. recondita* f. sp. *triticina* and/or *Septoria nodorum* in a climate chamber experiment. Neth. J. Pl. Path. 80: 192–214.
Wit, C. T. de & J. Goudriaan, 1978. Simulation of ecological processes. Simulation Monographs, Pudoc, Wageningen, 183 pp.
Wolfe, M. S. & J. A. Barrett, 1977. Population genetics of powdery mildew epidemics. Ann. New York Acad. Sci. 287: 151–163.
Zadoks, J. C., 1972. Modern concepts of disease resistance in cereals. In: F. G. H. Lupton, G. Jenkins, R. Johnson (Eds): The way ahead in plant breeding. Proc. 6th Congr. Eucarpia, Cambridge, 1971. Cambridge, p. 89–98.
Zadoks, J. C., 1974. The role of epidemiology in modern phytopathology. Phytopathology 64: 918–923.
Zadoks, J. C., 1977. On the epidemiological evaluation of fungicide action. Neth. J. Pl. Path. 83 (Suppl. 1): 417–426.
Zadoks, J. C. & P. Kampmeijer, 1977. The role of crop populations and their deployment, illustrated by means of a simulation EPIMUL–76. Ann. New York Acad. Sci. 287: 164–190.
Zadoks, J. C. & R. D. Schein, 1979. Epidemiology and plant disease management. Oxford Univ. Press, New York, 427 pp.

Comparative epidemiology: an evaluation of scope, concepts and methods

J. Kranz

Abstract

The scope and objective are outlined of comparative epidemiology as a tool for research. An attempt is made to define concepts for the comparison of epidemics by application of approaches and methods commonly used in experimental science and based on a systems approach. For strict comparisons of various epidemics, and of phenomena within an epidemic, the choice of appropriate systems levels and criteria is deemed essential. The use of published evidence and of established experimental procedures for epidemiological comparison are discussed. Some results obtained by these means are reviewed.

Scope of comparative epidemiology

Cognition starts with comparison. But comparison is also a major technique of research. It helps to derive principles and models from discrete data – in our context – on diverse plant diseases or from a large number of different epidemic phenomena. In this way, comparative epidemiology attempts to enlarge our comprehension of complex pathosystems beyond simple description of similarities and differences within and amongst epidemics and their constituents, or comparison of experimental treatments.

Comparative epidemiology thus has both analytic and synoptic functions. It consequently can direct research by establishing or sustaining principles. As research generally elucidates phenomena against the background of accepted theories, comparative epidemiology has to gauge their validity, and abolish them if they fail the test. Such a 'Darwinism of hypotheses' (Popper, 1973) certainly advances science and professionalism in epidemiology.

Epidemiology develops tactical and strategic concepts for control of plant disease. For these practical ends, comparative epidemiology is expected to reduce the apparently unlimited diversity of epidemics and their components to a convenient number of basic systems or types to which individual epidemics may be assigned and from which future disease management can benefit (Kranz, 1978). Comparative epidemiology also provides guidelines for policy-making, and has to cater to teachers and textbook writers.

To achieve all this, and to develop a philosophy of epidemiology, comparative epidemiology has to assume a unifying and crystallizing role. Comparative epidemiology, in addition, requires some universally accepted approaches, as well as appropriate and greatly standardized methods. Also forms of presentation are needed that avoid remoteness and ambiguity. In this volume Butt & Royle deal with this particular problem in relation to terms and definitions.

Some relevant concepts for comparative epidemiology

Epidemiology has been conceived in various ways. For some plant pathologists, epidemiology is the textbook chapter that deals with the effect of ecological factors on disease; for others it is the dynamics of diseases in host populations, and for others again it is, more narrowly, disease dispersal and spread. Within all of these concepts, comparison is possible and justifiable. For instance, comparison of effects that weather factors have on severity of cereal rusts, may explain the sequence of their appearance in wheat fields. Disease progress of *Septoria nodorum* (Berk.) Berk. and *S. tritici* Rob. apud Desm. on wheat can be compared '. . . to determine the basic difference in the behaviour of the two *Septoria* pathogens as head parasites in terms of yield reduction' (Cooke & Jones, 1970). Different disease gradients amongst diseases, may be analysed by a study of their modes of spread and their relative efficiency of infection.

However conclusions thus derived may fall short of explaining the whole intricate biological interactions behind disease progress. We, therefore, conceive epidemics as open, coupled and dynamic systems of pathogen and host populations interacting under the influence of environment and human interference. Every biological system has levels and structures and behaviours resulting therefrom, and so have epidemics (Kranz, 1974a, 1974b, 1978; Robinson, 1976). In the hierarchy of biological systems epidemics belong to the population level; and they in turn have their own levels (e.g. the pathogen, the host, the disease, the field or agroecosystems). The behaviour of epidemics as systems does not result from linear causal relationships but from programs inherent in their structures. Programs should be understood here in the sense used by geneticists. The control of such systems is to a large extent possible by human interference. Thus epidemiology closely relates to disease control. This aspect is dealt with in this volume by Putter and by Rotem & Palti.

Before we refer to appropriate methods of comparative epidemiology within the framework of systems approach, let us refer briefly to another concept of comparative epidemiology, which we, tentatively, call the 'wholesale approach'.

By wholesale approach, we refer here to attempts to compare epidemics of diseases occurring in different, often unrelated, agroecosystems (e.g. forest and field; field and greenhouse), or epidemics having different ecological or historical backgrounds (e.g. root and leaf diseases; endemic and invading pathogens). There obviously is a need and a fruitful potential for exploiting such inferences, particularly for teaching and even for policy-making.

However, we have not so far found satisfactory ways of tackling this type of comparison. Wholesale comparison depends largely on published information. The literature, however, does not often permit us to draw valid conclusions. Furthermore, indiscriminate use of publications for this purpose may violate certain scientific principles. A serious shortcoming is that wholesale comparisons are hard to verify experimentally. Unless we can overcome this obstacle, a wholesale comparison can merely yield tentative deductions. On the other hand, we should certainly think more seriously about ways and means to implement adequate research which satisfies this demand. We shall return to this topic in a wider context. (Section 'Use of published evidence').

Methods for comparative epidemiology

The mechanistic comparison of variables, constants and parameters alone does not ensure successful comparative epidemiology. As a matter of fact, endeavours to compare epidemics and their components can be convincing only if they convey a grasp of the extent of phenomena and problems involved, and feeling for the diversity of possible views and interactions. A given criterion may have a different weight in another context or systems level. Hence, apart from a deep and sensitive comprehension of the epidemics under study, comparative epidemiology needs both appropriate experiments to test hypotheses and adequate descriptive inventories. In ethology, for instance, the existence of a certain inherent pattern of animal motion could have only been discovered by research workers who were familiar with the whole range of possible patterns (Aktionssysteme) in phylogenetically related animal taxa and who were also capable of comparing them by the same method (Lorenz, 1966).

Choice of systems level for comparison

Comparative epidemiology can be achieved at various system levels. Table 1 gives examples of levels that one could envisage in pathosystems and their epidemiology. The choice of level depends on the objective of the comparison. This volume attempts to demonstrate how this could be done at the pathosystem level or any subsystem level as well as with factors affecting (Aust et al., this volume). Finally, comparison is possible at the level of different etiological groups (Thresh and Jones, this volume). Other aspects, like the comparison of epidemics at different sites, climates, years and treatments, will always relate to one of the levels listed in Table 1.

Comparative epidemiology following the concept of systems approach obviously has its merits for the elucidation of programs referred to under Section 'Some relevant concepts'. This in turn requires an understanding of the organization of pathosystem components, their levels and elements, and the kind, strength, and flexibility of relations between elements and finally, their contributions to the system's behaviour.

Programs consist of interactions of structural elements, their quantitative contributions, as well as their interactions with external factors. In addition there are specific program elements. These are features inherent in populations like random processes, recurrence, limits or dimensions, discontinuities and thresholds. They regulate an otherwise latent structure, which then results in behaviour, e.g. growth and decline of a population of lesions, or their spatial and age distribution (Kranz, 1978). All of these facets lend themselves to comparative studies. Environmental factors and human interference in turn provide the stimuli to implement these open programs. Therefore, each system is effective for different reasons with a corresponding diversity in epidemiology and in control measures (Thresh, 1974a).

The *structures* of epidemics themselves are composed of important rate-determining elements (Kranz, 1978) which may be the objective of a comparison. The *behaviour* of these elements (i.e. their interaction) is expressed in *patterns* such as disease progress curve or gradients. For more practical purpose, however, it would suffice to compare only those components that characterize the 'core dynamics' (Patten, 1971) of epidemics, like the element 'infection' and relevant interactions between inoculum, leaf wetness and temperature. The 'core dynamics' prove the existence of a certain hierarchy of elements in every system, also in epidemics.

Table 1. Levels for comparisons in epidemiology (an example).

Levels	Examples
A. Agroecosystem	wheat crop (various constraints, incl. diseases, and their actions)
pathosystem A ↔ B ↔ . . . ↔ n	wheat stem rust (eye spot, . . . , powdery mildew)
pathogen / host	pathogen level: *Puccinia graminis* vs *Pseudocercosporella herpotrichoides*. host level: different wheat cultivars (e.g. its development, or type of resistance of wheat or barley as hosts of the same pathogens)
B. *disease:* epidemic	stem rust or eye spot disease, or barley or wheat powdery mildew, or stem rust under irrigation, different fertilizer regimes, control measures.
structure	elements of epidemics of stem rust and eye spot: or lifecycle of pathogens, and factors affecting it.
elements	comparison of infection process of *P. graminis* & *P. herpotrichoides* and factors affecting them.
behaviour, pattern	comparison of disease progress of stem rust and eye spot in time (space), under certain conditions.
partial	comparison of the logarithmic phases of stem rust and eyespot epidemics, within the same pathosystem.
behaviour, rate	comparison of rates in untransformed disease progress curves of both epidemics, within the same pathosystem. different treatments.
partial	variation of infection rates during various phases of the disease progress curves.

Within the systems concept, comparison of patterns can either be for entire epidemics, or their parts. Basic patterns of entire cumulative curves of disease progress have been described earlier (Kranz, 1968c; 1974a; 1978). Undisturbed progress curves of pathosystems are bilateral, bimodal, multimodal or oscillating. Bimodal, multimodal and oscillating (periodic) progress curves often reflect disease cycles, discontinuities in infection progress, variation in incubation periods, changes in host susceptibility due to new growth, or other factors. Periodic progress curves may also be recorded in multiple-wave epidemics (Kranz, 1978). Most progress curves, however, are incomplete, as diseases are usually recorded only until harvest.

Entire rate curves identify at least three major classes of epidemic patterns: symmetrical (bell-shaped) curves and asymmetric curves with either positive or negative skewness. For details see Kranz (1978).

Parts of progress curves lend themselves to a comparison as they may represent essential biological facets of the disease's progress and may, for instance, have a bearing on forecasting (Kranz, 1974a). Baker (1971) suggested four consecutive phases: (1) the true logarithmic, (2) the synergistic (exponential), (3) the transitional and (4) the plateau phase. When used for quantitative comparisons, the inherent variability of these phases should be adequately understood (Kranz, 1968c; Kranz & Lörincz, 1970).

Within the comparison of different etiological groups, one may, with suitable criteria, compare similarities or differences, for instance, in the spread of viruses and other types of plant pathogens: 'Mycoplasmas that multiply in plants and in leafhoppers resemble propagative viruses with aphid or leafhopper vectors in that they can be carried far to give shallow gradients of infection resembling those due to windborne spores. The much steeper gradients of infection caused by the splash dispersal of spores or bacteria by water droplets are analogous to local spread of viruses by leaf contact or by vectors of limited mobility . . . . Despite these apparent similarities quite different factors influence the spread of viruses and of fungi' (Thresh, 1976).

Criteria

The choice of criteria in comparative epidemiology again depends on the objective of comparison. In general, however, comparisons should be based on features which for a given disease, have a high degree of invariance or consistency. The similarity in such consistent characteristics is the major criterion for comparisons between pathosystems. Criteria of primary, secondary or tertiary importance may be distinguished, according to the degree or range of variability in the features selected for comparison. Normally, characteristics redundant in the taxonomic sense should be excluded. However, problems may arise to define what is redundant in epidemics under study. There may even be epidemics in which the number of identical redundant features may be used to determine similarity. Criteria chosen for comparison have to belong to systems of the same level. This ensures that only the really comparable is compared.

Similarity of structures (and of elements) and of behaviour (i.e. patterns and rates) can be measured by standard statistical tests. These include tests of significance between two or more regression coefficients or correlation coefficients. Similarity or matching coefficients (Kranz & Lörincz, 1970), factor and cluster analyses and their various test criteria (Kranz, 1974a) seem appropriate. They all help to avoid conclusion from superficial resemblance.

Structures can be compared by the following means:

1. degree of agreement in the organization of the structure,
2. similarity of existing structural elements
3. similarity of epidemiologically relevant functions of these elements, and the correlations among their output – input relationships.

For means 1, number and kind of elements in the structure, and their interrelationship are the criteria. When comparing the similarity of existing elements (means 2), homologue and analogue components may be distinguished. Homologue components are phyllogenetically alike, e.g., infection by conidia. If components of the epidemic structures are only similar in their 'operational mechanisms' (Kranz, 1978) or functions (e.g. infection by sclerotia and not by conidia), they should be regarded as analogues. Similarity of epidemiologically relevant functions (means 3) of structural elements may

be judged by their program elements (Section 'Choice of level') or their output – input relationships between preceding and following elements (i.e. state variables). The latter can be exemplified by the effect intensity of infection has on length of incubation periods and other phases. When studying aspects by means 3, emphasis should not be so much on the influence of a factor on, for instance, sporulation as such, but rather on the effect of more or less inoculum on the dynamics of the epidemics (Kranz, 1978).

Patterns and rates describe the behaviour of epidemics (Section 'Choice of level'). The shape of disease progress curves or gradients often carries useful biological information. Consequently, basic patterns can be expected, around which observed patterns may be grouped. These groups or clusters may serve for the reduction of the apparently unlimited diversity of epidemics to a convenient number of basic types that comparative epidemiology is expected to achieve. If linearization is required for statistical purposes, slope and position of the lines could be used in comparisons as well as the transformation equation used to obtain the best fit. For more details and technical aspects, see Kranz (1974a; 1978).

Rates express the response or behaviour of the system to external or internal factors in relation to previous state. They primarily change disease intensity but not necessarily the underlying pattern. But rates are variable. Comparison of rates, e.g. Vanderplank's (1963) apparent infection rate, may, therefore, be valid only when comparison of environmental effects and resistance is intended. Otherwise it may be preferable to assign rates to meaningful classes (Table 2), or to give rates inferior weight within the set of criteria chosen for comparison. For the use of classification in comparative epidemiology see Kranz (1974a). As different transformation equations are available they may also be used for classification of epidemics with different biological backgrounds (Hau & Kranz, 1977). The choice of an appropriate transformation equation

Table 2. Classes for apparent infection rates r defined by central values and limits[1].

Class No.	Median value[2] r	Limits[3] r
I	0.03	0.01 – 0.05
II	0.08	0.06 – 0.10
III	0.15	0.11 – 0.16
IV	0.20	0.17 – 0.25
V	0.30	0.26 – 0.30
VI	>0.30	0.31 – (1.00)

1. From experimental data of 40 pathosystems in 2 years (Kranz, 1968b, Table 6).
2. r values that have distinctly higher frequencies than adjacent ones.
3. Limits in this context should be regarded as flexible and applied with common sense; e.g. if r in one year is 0.08 and in another 0.12, both should be considered as belonging to the same class.

thus assigns an epidemic to those of similar pattern, for instance whether best fit of the lines to the observed values is obtained by ln y (no asymptotic progress curve) or by Bertalanffy function (with distinct asymptote).

Use of published evidence in comparative epidemiology

Comparison of epidemics should be attempted with well defined objectives, with adequate inventories and with full comprehension of the pathosystems under study and their epidemics, as well as with appropriate experimental methods.

What kind of inventories? Description of phenomena will be needed to develop working hypotheses, research programs and methods. Essentially four types of publications can serve as material for comparison:

(1) Papers that have been published outside the scope of comparative epidemiology. They comprise pure description of phenomena, reports, and reviews, as well as experimental results of any kind, including comparison of effects of experimental treatments. Though not always specifically comparable, such information frequently gives rise to hypotheses because of disagreement between accepted theories and new facts, or through intuition and speculation. Their use and interpretation for comparison should, however, be made with caution (Section 'Some relevant concepts' for 'wholesale' comparison, and below). This applies to information described under Items 2 and 3, respectively.

(2) Reviews specifically written to extract general conclusions from scattered evidence by comparison, e.g. methodology in comparison of epidemics (Kranz, 1974a), temporal patterns in virus spread (Thresh, 1974b), or gradients in disease spread (Gregory, 1968; Thresh, 1976).

(3) Papers in which authors have supplemented their own data with information from the literature. Such evaluations are usually made critically and competently. Examples of this approach are the studies by Populer (1972) of powdery mildew of rubber (*Oidium heveae* Steinm.) and by Zadoks (1961) of stripe rust of wheat (*Puccinia striiformis* West.).

(4) Reports of results from experiments specially designed for the comparison of some epidemiological facets. These are obviously the most reliable and valid material for a given objective. Only a few such data are presently available.

When published evidence is used in comparative epidemiology, it is essential that hypotheses and theories inferred should be amenable to experimental testing and verification. Therefore data should be critically scrutinized for experimental techniques, their evaluation and their interpretation by the authors, to assess their limits for inference. Obviously the evaluation of publications itself must be subject to the same standards that apply to the evaluation of laboratory experiments or field trials. Hence, the material utilized should have similar objectives and share some common factor. Finally, really good and valid data are based on the same, or at least, comparable criteria, and on units as well as techniques of measurement that apply to all the material used.

Experimental comparison

Comparative epidemiology should basically be both quantitative and experimental as hypotheses have to be tested in well designed experiments (Section 'Use of evidence').

Systems analysis often will be the best choice to cope with all the complexities in epidemics. Design of experiments should ensure strict comparative measurements of features of interest (e.g. structural elements, progress curves and interactions) and pertinent statistical evaluation. Simultaneous measurement under exactly the same conditions would be ideal for comparison. Measurements should be expressed in the same units for all pathosystems to be compared. All this follows from what has been said in the Sections 'Choice of level' and 'Criteria'.

We advocate specific comparative field trials. These are more easily performed when carried out on diseases of a particular crop (e.g. ear blotch, powdery mildew and stripe rust of wheat), or on some specific disease as it affects different crops that can be grown in the same field. Thresh (1976) and Kranz (1972) emphasize requirements for proper comparative field trials. Plots must be of adequate size to avoid border, positional and exposure effects, and background interference, which can easily blur gradients.

Entire pathosystems may be beyond the competence or capacity of a single research worker. Therefore, more often a partial study, i.e. of one or few elements in several diseases is preferred. However, the type of customary laboratory comparisons of, for instance, sporulation of fungi P_1 and P_2 under some defined condition usually bear little relation to epidemics caused by these pathogens, unless verified in 'mini-epidemics' (Cohen & Rotem, 1971), or other experimental arrangements. Partial comparisons are often the choice in field trials. One can, however, also compare whole graphs of epidemics (Kranz, 1968a, b, c; 1975a, b) in field trials, i.e. progress curves and gradients that consider the behaviour of the pathosystems as 'black boxes' (Kranz, 1974b).

Some remarks on the procedure of comparison

Results obtained from experiments conducted as suggested (Sections 'Choice of level', 'Criteria' and 'Experimental comparison') can be compared by means of simple plotting, mapping or tabulation of quantitative data to elaborate statistical, mathematical and computer methods (Kranz, 1974a; Thresh, 1976).

To cope with complexity in epidemics, comparison may either follow the stepping-down or stepping-up procedure. The former starts from the graphs of an epidemic as 'black boxes' and elucidates more details. The stepping-up procedure builds from well studied elements the essential structure of an epidemic. Both approaches are possible and justified. The choice will depend on the objective and often also on the facilities available.

Obviously, the stepping-up approach is more appealing to the research worker with good laboratory facilities, or to those who can use a simulator. Whereas the field pathologist is more likely to prefer the stepping-down approach. Either way, the scope of comparison (disease progress only or with study of incubation or other phases) will again largely depend on the objective. Perhaps the stepping-up approach is also in line with a certain tendency in science to go from the simple to the more complex. This is justified as long as it is not forgotten that the more complex problems of higher systems are only partially explicable in this way. There is too little experience yet to decide which is better, or when one is more appropriate.

Simulators may be used as synoptic instruments to test sensitivity and validity of assumptions, conclusions or models derived from comparisons. Simulators can thus partly replace field and laboratory experiments, and eventually find their place as

operational instruments in pest management. The uses of computers in comparative epidemiology has been reviewed by Kranz (1974a).

Some results obtained by comparative epidemiology

The usefulness of comparative epidemiology is obvious from the second sentence of this article. It is prerequisite for modelling, or at least for the generalization of models. Let us imagine that some years hence the objective of crop protection will no longer be control of one or two diseases or insects in one crop at a time. Plant protectionists may then be obliged to maintain fields reasonably free from the constraints of all relevant pests (Kranz, 1978). Modelling will enable us to describe and explain essentials in epidemics, and help us to meet such obligations.

Looking back, we notice that Gäumann (1951) and Vanderplank (1963, 1968, 1975) already have extracted quite a few concepts and hypotheses from literature by comparing epidemics and their components. Also some of Yarwood's 'Principles of plant pathology' (Yarwood, 1973) may be considered the out-come of comparative epidemiology. However, the experience, common sense and intuition for which these authors are noted have to be supplemented by systematic research. Kranz (1974) and Thresh (1974) have discussed temporal aspects and their utilization. Gradients have been compared by Gregory (1968) and Thresh (1976).

Very little systematic and experimental comparison has as yet been published. Some experiments along the lines proposed have been conducted (Kranz, 1968a, b, c; 1975a, b; 1976; 1977; Bashi & Rotem, 1974; Reuveni & Rotem, 1973). Some of the results obtained corroborated a wide application of postulates by Vanderplank (1963), or corroborated the concept underlying negative prognoses (Schrödter & Ullrich, 1965). They also showed that transformation equations for disease progress were not universal (Hau & Kranz, 1977).

The effect of available susceptible leaf mass (Kranz, 1975a), the mechanisms determining decline of disease progress curves (Kranz, 1975b) and the effect some diseases have on the loss of leaves (Kranz, 1976) have been studied concurrently in four to five pathosystems of foliar diseases. Studies of this kind reveal some of the features that operate in epidemics and indicate their variability. In the same comparative field trial with five pathosystems, the maximum severity of disease was compared per leaf. On the vast majority of leaves, maximum severity was below 13.8 percent of area fraction of leaves infected at most a sixth of the leaves affected by powdery mildews and a rust disease had a maximum severity ranging from 13.8 percent to 100 percent. This has obvious implications for disease assessment (Kranz, 1977).

More examples of comparative epidemiology applied to plant pathology and crop protection will be presented by my fellow contributors to this volume. We hope that still more, better and more relevant work on comparative epidemiology will be published in future.

References

Baker, R., 1971. Analysis involving inoculum density of soil-borne plant pathogens in epidemiology. Phytopathology 61: 1280–1292.

Bashi, Esther & J. Rotem, 1974. Adaption of four pathogens to semi-arid habitats as conditioned by penetration rate and germinating spore survival. Phytopathology 64: 1035–1039.

Cohen, Y. & J. Rotem, 1971. Field and growth chamber approach to epidemiology of *Pseudoperonospora cubensis* on cucumber. Phytopathology 61: 736–737.
Cooke, B. M. & D. G. Jones, 1970. The epidemiology of *Septoria tritici* and *S. nodorum* II. Comparative studies of head infection by *Septoria tritici* and *S. nodorum* on spring wheat. Trans. Br. mycol. Soc. 54: 395–404.
Gäumann, E., 1951. Pflanzliche Infektionslehre. Birkhäuser-Verlag, Basel, 2nd ed., 681 pp.
Gregory, P. H., 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathology 6: 189–212.
Hau B. & J. Kranz, 1977. Ein Vergleich verschiedener Transformationen von Befallskurven. Phytopath. Z. 88: 63–68.
Kranz, J., 1968a. Eine Analyse von annuellen Epidemien pilzlicher Parasiten. I. Die Befallskurven und ihre Abhängigkeit von einigen Umweltfaktoren. Phytopath. Z. 61: 59–86.
Kranz, J., 1968b. Eine Analyse von annuellen Epidemien pilzlicher Parasiten. II. Qualitative und quantitative Merkmale von Befallskurven. Phytopath. Z. 61: 170–190.
Kranz, J., 1968c. Eine Analyse von annuellen Epidemien pilzlicher Parasiten. III. Über Korrelationen zwischen quantitativen Merkmalen von Befallskurven und Ähnlichkeiten von Epidemien. Phytopath. Z. 61: 205–217.
Kranz, J., 1972. Einige Voraussetzungen für die Planung und Durchführung von Feldversuchen in der Epidemiologie. Z. Pflanzenkrankheiten. 79: 573–589.
Kranz, J., 1974a. Comparison of epidemics. Annu. Rev. Phytopathology 12: 355–374.
Kranz, J., 1974b. The role and scope of mathematical analysis and modeling in epidemiology. In: J. Kranz (Ed.): Epidemics of plant diseases. Mathematical analysis and modeling. Springer Berlin – Heidelberg – New York, p. 7–54.
Kranz, J., 1975a. Beziehungen zwischen Blattmasse und Befallsentwicklung. Z. Pflanzenkrankheiten 82: 621–654.
Kranz, J., 1975b. Das Abklingen von Befallskurven. Z. Pflanzenkrankheiten 82: 655–664.
Kranz, J., 1976. Einfluß einiger Blattkrankheiten auf den Abgang der Blätter. Z. Pflanzenkrankheiten 83: 234–237.
Kranz, J., 1977. A study on maximum severity in plant diseases. In: Traveaux dédiés à G. Viennot-Bourgin, Paris, p. 169–173.
Kranz, J., 1978. Comparative anatomy of epidemics. In: J. G. Horsfall & E. B. Cowling: Plant disease, Vol. II. p. 33–62.
Kranz, J. & D. Lörincz, 1970. Methoden zum automatischen Vergleich epidemischer Abläufe bei Pflanzenkrankheiten. Phytopath. Z. 67: 225–233.
Lorenz, K., 1966. Über tierisches und menschliches Verhalten. Bd. II, Piper paperback, Piper & Co. Verlag, München, 398 pp.
Patten, B. C., 1971. A primer for ecological modeling and simulation with analog and digital computers. In: B. C. Patten (Ed.): Systems analysis and simulation in biology. Vol. I. Academic Press, New York, p. 4–121.
Popper, K. R., 1973. Objektive Erkenntnis. Ein evolutionärer Entwurf. Hoffmann & Campe, Hamburg, 417 pp. (transl. from 'Objective Knowledge', 1972. Clarendon Press, Oxford).
Populer, C., 1972. Les épidémies de l'oidium de l'hévéa et la phénologie de son hôte dans le monde. INEAC Ser. Sci. No. 115, Brussels.
Reuveni, R & J. Rotem, 1973. Epidemics of *Leveillula taurica* on tomatoes and peppers as affected by the conditions of humidity. Phytopath. Z. 76: 153–157.
Robinson, R. A., 1976. Plant Pathosystems. Springer, Berlin – Heidelberg – New York, 184 pp.
Schrödter, H. & J. Ullrich, 1965. Untersuchungen zur Biometeorologie und Epidemiologie von *Phytophthora infestans* (Mont.) de By auf mathematisch-statistischer Grundlage. Phytopath. Z. 54: 87–103.
Thresh, J. M., 1974a. Vector relationships and the development of plant viruses. Phytopathology 64: 1050–1056.

Thresh, J. M., 1974b. Temporal patterns of virus spread. Annu. Rev. Phytopathology 12: 111–128.
Thresh, J. M., 1976. Gradients of plant virus diseases. Ann. appl. Biol. 82: 381–406.
Vanderplank, J. E., 1963. Plant diseases: Epidemics and control. Academic Press. New York, 349 pp.
Vanderplank, J. E., 1968. Disease resistance in plants. Academic Press, New York, 206 pp.
Vanderplank, J. E., 1975. Principles in plant infection. Academic Press, New York, 216 pp.
Yarwood, C. E., 1973. Some principles of plant pathology. II. Phytopathology 63: 1324–1325.
Zadoks, J. G. 1961. Yellow rust on wheat. Studies in epidemiology and physiological specialization. Tijdschr. Plantenziekten 67: 69–256.

The importance of terms and definitions for a conceptually unified epidemiology

D. J. Butt and D. J. Royle

'A living language must keep pace with improvements in knowledge and with the multiplication of ideas'

Noah Webster, 1817

Preface

The authors feel obliged to assure readers that it is not the purpose of this paper to present a pedantic treatise with a glossary of arguable definitions.

This assurance is needed because plant pathologists, the writers included, are inclined to withdraw from seemingly tiresome and mundane considerations of terminology. Will future epidemiologists blame the present generation, however, if they inherit a technical language that is clouded with ambiguity and vagueness? If epidemiology is to earn the status of a modern quantitative science we must face and solve problems of meaning, not dismiss them as 'mere semantics'.

Introduction

As befits all scientists, epidemiologists do more than observe, describe and classify; they also seek to understand epidemics in order to explain, predict and control disease. In the empirical sciences understanding is expressed in empirical laws that state consistencies between phenomena, e.g. the Law of the origin (Vanderplank, 1975, p. 20), and in general laws that summarize the theoretical 'orderliness' of nature, e.g. the differential difference equation of epidemic growth (Vanderplank, 1975, p. 104). Indeed, the provision of coherent, consistent and wide-ranging theories has been called an essential test of science (Toulmin, 1977). In his paper on methodology, Zadoks (1978) describes how the 'empirical cycle' of rational investigation leads to unifying concepts which broaden and deepen understanding. In biology generally, comparison has been rewarding in the search for general principles and theories that 'bridge' groups of organisms; Kranz (1978) has assigned to comparative epidemiology this role of reducing epidemic structures and behaviours to a few general patterns.

In plant disease epidemiology there is a rapidly growing tendency towards abstraction, modelling and concepts (Zadoks & Koster, 1976), and this trend will increasingly unite educators, researchers and practitioners with a common philosophy. A more pragmatic benefit will be more reliable explanations and predictions of disease phenomena because insight, general principles and fundamental understanding are better premises than are descriptions of particular past experiences; with the support of sound theory, strategies of crop protection and resource management will be improved as will decision-taking at farm level.

The pursuit of theoretical epidemiology is important for it is a widely held view that only a theory adequately unifies and explains phenomena (Hempel, 1966). In his autobiographical essay, Vanderplank (1976) remarks that synthesis and generalization must be as brisk as the churning out of data. Thanks to his own work and that of other imaginative synthesists like Gäumann, we share some powerful concepts like the disease progress curve, the infection chain and the disease cycle. Although it can be expected that the longest strides to greater unification will continue to be taken by only the most imaginative, epidemiologists as a whole can contribute by ensuring that their communication of data and ideas is adequate for the age of synthesis, modelling and integrated crop protection.

The diversity of plant pathogens imposes differences in experimental techniques which in turn reduce the comparability of ostensibly similar measurements. Progress towards a unified epidemiology would be faster if data were given a universality, an 'exchange-value', beyond the purpose and time of each investigation. Epidemiology is a quantitative science and measurement begins with a clear definition of the quality or quantity whose magnitude is to be determined. Unless a term is operationally defined (see below) there is no criterion of what to measure; furthermore, unless the definition is widely accepted there can be no unequivocal meaning of the information.

Vagueness and ambiguity also obscure the communication of ideas across the network of activities which engage epidemiologists. Studies range from the organism to the community, from the analysis of processes in the disease cycle to the synthesis of explanatory models, from field observations to the simulation of theoretical epidemics, from experiments in growth chambers to the measurement of microclimate, from producing mini-epidemics to tracking the intercontinental dispersal of inoculum. Is it important for basic terms like 'infection' and 'latent period' to mean the same throughout this network? The age of synthesis is at hand (Cowling & Horsfall, 1978) and the current fashion is for a systems approach which itself leads to broad, multidimensional theories (Kast & Rosenzweig, 1972). This calls for closer team-work of pathologists, meteorologists, crop physiologists, mathematicians and other specialists than ever before. The need for clarity and precision in communication is urgent as epidemiology increasingly accepts its social responsibility to protect the world's crops.

Elements of communication

Technical terms, symbols and expressions are the verbal fabric of scientific communication and before discussing them it is necessary to understand their relationships with concepts and definitions.

A 'concept' is an idea, a lasting mental image or a general notion invoked by a term, and includes all that is associated with or suggested by the term. Primary concepts spring directly from the perception of phenomena: examples are the mental images invoked by the terms 'spore', 'germination', 'local lesion', 'primary focus'. Secondary concepts are not of perceivable phenomena and are, therefore, of a higher degree of abstraction; examples are invoked by 'inoculum potential', 'basic infection rate'. The idea of degrees of abstraction is expressed when related concepts are placed in order of increasing abstraction, as with the sequence zoospore → spore → propagule → inoculum → inoculum dose → inoculum potential sensu Garrett (1956).

The word or symbol which stands for a definite concept and therefore has a limited

meaning is a 'term'; it is the word label or name for a concept. Primary terms, many of which are nouns from the vocabularies of mycology and other supporting disciplines are the names of primary concepts. The terms associated with secondary concepts are abstract (theoretical) terms which can only be defined with primary terms. Notwithstanding difficulties of measurement (see below), abstract terms are valuable in allowing complex phenomena to be described. Furthermore, Stamper (1973) suggests that abstract terms in the physical sciences have a good semantic standing because they can be used in laws which are testable.

A 'definition' bounds and characterizes a concept and so allows concepts to be related. Obviously, a term can invoke a universal concept only if the definition is explicit. The more abstract a concept the more difficult it is to define explicitly because it does not exist as an object in the real world. Consequently, abstract terms tend to be ambiguous and vague, as typified by 'inoculum potential' (Wood, 1967).

The need for operational definitions

Measurement determines quantity (e.g. count, mass, length) and quality (e.g. temperature, colour) by using scales or by counting, and in a scientific enquiry measurements are made at two recurring stages. First, in the observation and description of phenomena; in collecting facts. Second, it is a condition of empirical science that all hypotheses and theories shall be verifiable (Hempel, 1966; Stamper, 1973), and so experiments are done to compare predictions with experience. In this way hypotheses are accepted, rejected or modified and the empirical cycle progresses to higher levels of unified understanding.

Consider the measurement of inoculum density. In the mind there is the concept and on the plant surface or in the soil is the reality of scattered units of inoculum. Clearly, if the testing of a hypothesis requires the measurement of inoculum density then a vague, abstract definition of the concept is inadequate; before measurement can begin an explicit set of procedural rules is needed. This is known as the 'operational definition' (Hempel, 1966), described by Zadoks (1978) as having the character of a recipe written for a specific purpose. Such a definition allows a hypothesis to be operationalized for testing.

It should be noted that all terms, descriptive as well as measurement, can be operationally defined (Hempel, 1966).

In the physical sciences operationalism plays a central role in providing universal criteria for the use of terms. Epidemiology must develop a similar respect for definition if its data base is to have the consistency and precision that is demanded of a quantitative science.

Dimensionality

Physical quantities are measured in units (e.g. metre, gram) on various scales of measurement. Dimensions are names for units measured on similar scales and any quantity can be given a dimensional formula (Ellis, 1966). Dimensions are shown in brackets, like mass [M], length [L], time duration [T] and count [N], and indicate the fundamental units in which quantities are measured. Strict rules of dimensional analysis must be satisfied in statements of quantitative relations. Some concepts and their

associated terms have dimensionality, e.g. sporulation density [$N.L^{-2}$], deposition velocity [$L.T^{-1}$], apparent infection rate sensu Vanderplank (1963) [T^{-1}]; Zadoks (1977) and Kampmeijer & Zadoks (1977) list others. (Ratios and 'efficiency' terms are dimensionless, [1].) Alternatively, a *sense* of measurement may be implied by the words used e.g. infection pressure, inoculum potential sensu Garrett (1956), infection ratio. In this paper we call all such terms 'measurement terms' in contrast to 'descriptive terms' which have no connotation of measurement.

Descriptive terms

Table 1 presents some descriptive terms in two ways. First they are grouped according to levels of organization of the pathosystem: the non-parasitic, the parasitic (which includes inoculum, germination and ingress) and the epidemic. At the non-parasitic level the pathogen is nutritionally isolated from the living host so that the terms mainly relate to dissemination and survival. At the parasitic level there is a vital interaction of pathogen and host; most of the terms at this level are inherited from the descriptive era of plant pathology. The terms 'disease cycle' and 'infection cycle' (which embrace both parasitic and non-parasitic levels) lead into the concept of generations of the pathogen spreading disease among populations of hosts – at the epidemic level of the pathosystem. Whilst only terms at the epidemic level may be said to be truly epidemiological, the understanding of epidemics draws upon events at the two lower levels; hence the inclusion of terms of the parasitic and non-parasitic levels.

Secondly, the terms used at each level are listed according to their type: common names, terms of quality and terms of behaviour. Some terms are synonymous with others and some occur more than once in Table 1.

Table 1. Types of descriptive terms grouped according to levels of the pathosystem.

Common names of entities	Qualitative terms denoting properties	Behavioural terms denoting processes
Terms used at the non-parasitic level		
infective unit	survivability	release
dissemination unit	longevity	discharge
propagule	persistence (of virus)	liberation
saprophyte		take-off
spore cloud		dispersal
spore		dissemination
resting body		transport
vector[1]		transmission
		interception
		deposition

1. This list could be lengthened to include other nouns which are primary terms in supporting disciplines like mycology.

(Table 1 continued)

Common names of entities	Qualitative terms denoting properties	Behavioural terms denoting processes
Terms used at the parasitic level		
inoculum	infectiousness	germination
infection court	survivability	penetration
point of infection	viability	ingress
infection (state of)	*infectivity*	*entry*
disease	effective inoculum	infection (process)
symptom		invasion
sign		establishment
syndrome		colonization
latent infection		incubation
local lesion		sporulation
colony		
systemic disease		
disease cycle		
infection cycle		
monocycle		
Terms used at the epidemic level		
inoculum source	pandemic	disease progress
target crop	endemic	disease spread
initial inoculum	epidemic	removal
primary inoculum	epiphytotic	perennation
initial infection	polyetic	overwintering
primary cycle	recurrent epidemic	oversummering
primary focus	local epidemic	
secondary inoculum	compound interest disease	
polycycle	simple interest disease	
infection chain	esodemic	
crop disease	exodemic	
pathosystem		

Common names

These nouns name entities which are physical (e.g. vector) or conceptual (e.g. infection chain). The list in Table 1 could be lengthened to include many other primary terms in underlying disciplines like mycology. Concepts associated with these terms tend to increase in degree of abstraction as the complexity of organization increases from the non-parasitic to the epidemic level.

Terms of quality

At the non-parasitic level these terms reflect the hazards of dissemination and isolation from the living plant. At the parasitic level they denote mainly the quality of inoculum. At the epidemic level they describe the temporal and spatial structure and nature of disease outbreaks.

Terms of behaviour

Because they denote activities these terms can be represented as verbs, e.g. to release, to enter, to sporulate, to overwinter. In the context of systems analysis most of these terms describe dynamic processes which change the state of the system. Apart from perennation, overwintering and oversummering, each can be assigned a rate, e.g. rate of colonization, rate of disease spread. Therefore, although behavioural terms and their concepts have no sense of measurement they describe processes of profound quantitative significance. Some processes are quantified by measurement terms like 'trap dose' sensu Gregory (1973) for deposition, 'sporulation index' sensu Shaner (1973) for sporulation and 'apparent infection rate' sensu Vanderplank (1963) for disease progress (see below). Other processes, notably germination, are directly measureable once an operational definition has been specified. Zadoks (1978) uses an operational definition of germination in his discussion of stochasticity, a further complication in the measurement of time-dependent variables.

It is noteworthy that ingress (entry, penetration) is, like germination, a relatively tangible phase of the disease cycle. The tendency to quantify events in the disease cycle for use in explanatory models (Shrum, 1978), indicates a need for agreed operational definitions of this process, as well as of germination; Teng et al. (1977) measure the penetration ratio.

Ambiguity of terms

What is infection?

'Infection' is, par excellence, a basic term. It virtually epitomizes the science of disease. Yet surprisingly, plant pathologists are divided in their conceptions of infection! Webster's Third New International Dictionary (1961) gives two definitions: 1. An act or process of infecting; the establishment of a pathogen in its host. 2. The state produced by the establishment of an infective agent.

Gäumann (1946, p. 5) defines infection as a process extending from germination to the instant a stable parasitic relationship is established. Infection period he defines (p. 35) as the time required to achieve an established parasitic relationship (EPR). Other standard texts which regard the EPR as signalling the end of the infection process are by Walker (1969, p. 6), Agrios (1969), Strobel & Mathre (1970, p. 439) and the Federation of British Plant Pathologists (FBPP) (1973).

In contrast, some workers define infection as a stage extending from the EPR (Stackman & Harrar, 1957, p. 259) or from the first response of the host (Whetzel, 1929; Chester, 1942, p. 464). Wood (1967, p. 6) considers that infection begins with the alteration or damage to cells. The appearance of symptoms may even be

taken as the start of the infection stage (Wheeler, 1969, p. 5; Shurtleff & Kelman, 1977).

In the terminology presented by Hirst and Schein (1965) infection is allocated to all processes from germination to the appearance of symptoms and signs.

This semantic shift is intolerable. The writers believe the definition of infection within plant pathology as a whole must be stipulated to be the process which ends with EPR; in this sense there are post-infection conditions which determine the pathogen's growth and development and the infected plant's response (Day, 1974, p. 7). There are three arguments to justify this usage. Firstly, there is no other existing term for this process. In the alternative sense of its use, infection comes close to the concept of disease itself because a plant in which a pathogen has established a parasitic relationship (EPR) is usually disturbed and so, by most definitions, diseased (FBPP, 1973). Secondly, although the well-known term 'apparent infection rate' is usually measured by recording change in amount of disease, it and the related 'basic infection rate' quantify pathogen multiplication initiated by inoculum, i.e. the process of infection, not multiplication within the host (Vanderplank, 1963 p. 30; 1975 p. 47). Thirdly, as a process the concept of infection extends logically to terms associated with the initial invasion of the plant: 'infection court', 'point of infection', 'infection efficiency of inoculum' and 'infection period'. The latter term appears often in the vocabulary of practitioners because they firmly identify infection with the initiation of disease, with the notion of attack and with the establishment of parasitism; in the words of Gäumann (1946 p. 441), infection is the pre-condition of infectious disease.

Because germination is more easily observed and manipulated than is ingress and because plants sometimes respond to pre-penetration events, the writers follow Gäumann (1946) and Hirst & Schein (1965) in regarding germination as the first phase of the infection process. Others see ingress as the start (Strobel & Mathre, 1970; FBPP, 1973). The difference is trivial in the context of this Section which is intended to expose gross ambiguity, not fine distinctions which in any case are dictated by research objectives and technological constraints. Likewise criteria for the EPR would fill a book on the histopathology, histochemistry and cell physiology of host-parasite relations, but this difficulty will arise in attempting to specify most transitions in living organisms. The EPR is a logical reference point in both fundamental and applied work to mark the end of the infection process.

The frequent use of the word establishment in this Section is deliberate. Webster's Dictionary defines it as the act of setting up so that a certain continuance is assured; to establish is to make firm or stable, to fix or implant in gaining a firm hold. It seems appropriate to elevate 'establishment' to the status of a technical term meaning the phase of the infection process between ingress and the EPR.

What word should be used to describe the post-infection (post-EPR) growth and development of the pathogen in the host? Gäumann (1946 p. 57), Wood (1967 p. 6) and Krause & Massie (1975) use the term 'colonization'. 'Invasion' has also been applied to this stage of the disease cycle (Agrios, 1969) but is, we think, too general a word to be given a specific definition.

Confusion over incubation and latency

Webster's Dictionary defines incubation as the period between the infection of a plant

or animal by a pathogen and the manifestation of the disease it causes. Latent is defined as being present or capable of living or developing in a host without producing visible symptoms of disease. It follows that during incubation a pathogen is latent. Latency has some special meanings; latent infections remain dormant at the site of infection until reactivated, and cause several well-known post-harvest diseases (Wood, 1967 p. 115; Verhoeff, 1974). Also, latency describes cases of protracted incubation during which colonization continues without the appearance of symptoms (Gäumann, 1946 p. 441; Butler & Jones, 1949 p. 33).

Although 'latent-' and 'incubation-period' are measurement terms (next Section) aspects of ambiguity are best dealt with here. Most workers use 'incubation period' for the stage of the disease cycle which ends with the appearance of symptoms (Gäumann, 1946 p. 36; Hirst & Schein, 1965; Vanderplank, 1963, 1975). There can be no disputing the logic of also naming this stage the 'latent period' (Whetzel, 1929; FBPP, 1973; Jones, 1978). It is worth noting that in virology the latent period is the time to detect newly produced virus; it also refers to vector-virus relations (Wheeler, 1969).

The first confusion arises with Whetzel (1929) whose concept of incubation ended at EPR; Yarwood (1978) perpetuates this definition (and in error attributes it to Gäumann).

Next, the term 'latent period' has been borrowed from medical epidemiology to mean the period ending with infectiousness (Vanderplank, 1963 p. 41) and is widely used today in this sense by plant disease epidemiologists. This important parameter has also been called the 'pre-reproduction' or 'pre-sporulation period' (Gäumann, 1946, pp. 37, 247) and the 'generation time' (Hirst & Schein, 1965). Unfortunately, the confusion now turns a full circle when this same period is called the 'incubation period'! (Stakman & Harrar, 1957 p. 259; Evans, 1968 p. 74; Colhoun, 1973; Polley & Clarkson, 1978). It is not surprising that Vanderplank (1975 p. 107) appeals for this mess to be sorted out.

At a meeting of epidemiologists at Pau, France in 1963, there was disagreement over the terminology of epidemiological processes (Hirst & Schein, 1965). Fifteen years later, at the 3rd International Congress of Plant Pathology in Munich, epidemiologists disputed the meaning of the term 'incubation period'!

Measurement terms

For the purpose of this paper terms with physical dimension or just with a sense of measurement are called measurement terms and are listed in Tables 2–4.

It remains convenient to group measurement terms according to levels of the pathosystem already used for descriptive terms; terms used at the non-parasitic level (Table 2), at the parasitic level (Table 3) and at the epidemic level (Table 4). The terms at each level are classed according to the complexity and abstraction of their associated concepts. In Class I we have listed good terms whose concepts are clear and explicitly defined; their definitions are equivalent or almost equivalent to operational definitions which provide rules for measurement. Concepts associated with terms in Class III have a high degree of abstraction. These are the worst of the measurement terms because they are vague and difficult to make operational. Terms of intermediate value are in Class II. It must be stressed that the classification is partly subjective and the writers have been unsure about the precise positioning of a few of the terms. Where appropriate, dimensions are shown in Class I.

Measurement terms at the non-parasitic level

Terms which measure processes and attributes at the non-parasitic level are in Table 2. Most are used solely in the context of the dissemination and interception of airborne spores and similar passive entities. It is striking that nearly all are aerobiological terms. Also, these are in Class I, largely because from take-off to deposition the movement of particles obeys physical, not biological laws; hence the lack of Class III terms. Physical systems lend themselves to precise measurement and to the creation of 'crisp' measurement terms in which the manner of measurement is explicitly stipulated. Thus in Class I are terms with words like concentration and velocity; other terms, for example 'bulk deposition coefficient', represent mathematical formulae. The definitions are rules of measurement, e.g. to measure trap dose count the number of particles deposited on a unit area of surface. There is no ambiguity. What is more, all these Class I terms have a universal value because the processes of transport and deposition and the trapping of particles are common to all airborne pathogens. Despite many good measurement terms at this level of the pathosystem, some problems of dispersal remain difficult to resolve, especially the behaviour of spores in the vicinity of plants and crops (Aylor, 1978).

Table 2. Classes of measurement terms used at the non-parasitic level of the pathosystem.

Class I (Precise terms explicitly defined) ←	Class II	→ Class III (Abstract terms conceptually defined)
airborne spore concentration [$N.L^{-3}$]	deposition gradient	
area dose [$N.L^{-2}$] (Gregory, 1973)		
terminal velocity v_s [$L.T^{-1}$]		
deposition velocity v_g [$L.T^{-1}$] (Chamberlain, 1966)		
bulk deposition coefficient [1][1] (Chamberlain, 1975; Gregory, 1973)		
trap dose [$N.L^{-2}$] (Gregory, 1973)		
trapping efficiency [1] (Gregory, 1973; Chamberlain, 1975)		
detection threshold [$N.L^{-3}$] (Hirst, 1959)		
impaction efficiency E [1] (Sell, 1931)		

1. [1] Indicates dimensionless.

'Deposition gradient' is in Class II because there is no single measure which characterizes this pre-infection gradient. There are empirical laws for calculating spore numbers at positions downwind of a source. Also, gradients, observed or calculated, can be

represented graphically and described by regression coefficients (Gregory, 1968). But as a measurement term 'deposition gradient' is inadequately operational. (Post-infection gradients are discussed below.)

Measurement terms at the parasitic level

Inoculum terms

Terms which measure natural or applied inoculum and its effectiveness are shown in Table 3A. Of five terms which measure quantity none are in Class I. Both density and concentration are used to relate inoculum to both area and volume; often density expresses inoculum per area of foliage and per volume of soil. Load vaguely expresses number being carried. 'Inoculum dose' only implies quantity so that units must always be specified in the way Schein (1964) defined dose to mean number. 'Effective inoculum dose' is even more vague (FBPP, 1973). The studies of Petersen (1959) and Heald (1921) illustrate how terms like 'inoculum density' and 'spore load' must be precisely defined.

The other terms in Table 3A express the ability of inoculum to infect. The amount of disease is determined by factors which include the quantity and quality of inoculum, susceptibility of the host and the physical environment. The interactions of this complex and the difficulties of separating the components in studies of soil-borne pathogens has led to the concept of inoculum potential. This concept has evolved with time to become increasingly complicated (Dimond & Horsfall, 1960; Wood, 1967 p. 112; Baker, 1978). Garrett (1970 p. 11) notes that biological concepts differ from those of the physical sciences in that their complexity often imposes a limit on the precision of definition. This is true, and a good concept can be a powerful stimulus to thinking. It must be stressed, however, that although such terms offer illusions of quantification they lack operational specificity. This is why Vanderplank (1975 p. 87) states that inoculum potential cannot be quantified. The same is true for infection pressure (FBPP, 1973).

Finally, the efficiency of inoculum is expressed by 'penetration ratio' (Teng et al., 1977) and two synonyms, 'infection ratio' and 'infection efficiency of inoculum' (Gregory, 1973 p. 237; Schein, 1964). On the assumption that infection refers to the process leading to EPR, these are good terms for the ratio of number of infections to the number of inoculum units.

In view of the dubious nature of the concept of a numerical threshold of infection (Vanderplank, 1975 p. 6), this term is in Class II.

Temporal terms

Temporal terms are listed in Table 3B. The terms in Class I are the names of precise specifications of the physical conditions of the environment needed for infection to occur. These criteria or rules define the 'infection periods' which are central to the prediction of many diseases (Tarr, 1972; Krause & Massie, 1975; Van der Wal, 1978).

With most diseases the pathogen is hidden from view during the period from ingress until signs appear at the end of the latent period. In the exceptional case of the ectophytic powdery mildews, colonization remains visible, of course, so that this process can be directly observed and measured. In general, however, quantification is restricted

Table 3. Classes of measurement terms used at the parasitic level of the pathosystem.

Class I (Precise terms, explicitly defined) ←	Class II	→ Class III (Abstract terms, conceptually defined)
A. Inoculum terms		
infection efficiency of inoculum [1] (*s.* Gregory, 1973)	inoculum dose	effective inoculum dose
infection ratio [1]	inoculum density	inoculum potential (*s.* Garrett, 1956)
penetration ratio [1]	inoculum concentration	inoculum potential (*s.* Horsfall, 1932)
	spore load	inoculum potential (*s.* Dimond & Horsfall, 1960)
	numerical threshold of infection (Gäumann, 1946)	infection pressure
B. Temporal terms[1]		
Mills Period (apple scab) (Mills & Laplante, 1954)	germination period	infection period[2]
Beaumont Period (potato late blight) (Beaumont, 1947)	infection period (*s.* Gäumann, 1946)	
	incubation period	
	latent period	
	generation time	
	sporulation period	
	infectious period	
C. Inoculum production terms		
sporulation density [$N.L^{-2}$] (*s.* Gäumann, 1946)		sporulation intensity
sporulation index [1] (*s.* Shaner, 1973)		
source strength [$N.T^{-1}$] (*s.* Aylor, 1978)		
sporulation rate [$N.T^{-1}$] (*s.* Zadoks, 1972b)		
sporulation potential [$N.T^{-1}$] (*s.* Kato, 1974)		

1. This list includes other sets of specified environmental conditions e.g. Wallin's System (potato late blight) (Wallin & Waggoner, 1950).
2. I.e. conditions of the physical environment necessary for the infection process.

to measuring periods of time which elapse between recognizable and visible events during parasitism and pathogenesis. Time is easily measured so why are most temporal terms not in Class I? The reason is the problem of defining the terms. When, for example, does the latent period end? Comfortably vague concepts of the various temporal terms do not suffice in quantitative epidemiology. If testing a hypothesis

requires the measurement of an infection period, a definition is needed which is operationally explicit. Zadoks (1972a, 1978) mentions the importance and difficulty of operationalizing the measurement of temporal terms which are stochastic by nature. Teng & Close (1978) discuss the terminology of time periods and their operational definition. Vanderplank (1975 p. 107) considers the epidemiological implications of operationally defining latent period.

'Infection period' appears twice in Table 3B. Gäumann (1946) defined this period as the time from germination until EPR and in this sense it is listed with the other Class II terms. There is another concept of infection period (Class III), however, especially valuable to practitioners, which is the period during which specified environmental conditions must be satisfied if the process of infection is to be completed. In this sense 'infection period' has the meaning of a set of environmental criteria, examples of which are shown in Class I. For many foliar pathogens germination and ingress are dependent upon surface wetness, and 'infection period' sometimes becomes identified simply with the idea of a wet period.

Inoculum production terms

Table 3C lists terms used in the measurement of inoculum production. All relate to spore yield and reflect the return (in the case of most fungal pathogens) to a perceivable stage of the disease cycle when spores and their supporting structures appear on the host's surface and can be counted. A few workers have operationalized the concept of sporulation intensity in terms like 'sporulation index', listed in Class I.

Measurement terms at the epidemic level

In Table 4 are listed terms which measure the dynamics and patterns of disease progress or disease itself. In the first of these categories are two explicitly defined infection rates; they are parameters in mathematical models of epidemic growth and therefore rank as Class I terms, together with 'sanitation ratio' (Vanderplank, 1963).

Although gradients of infection and disease do not necessarily result from dispersal, the three gradient terms in Class II are all measured from observations of disease and are often taken to have the same meaning (Gregory, 1973; Vanderplank, 1975 p. 133). Dimond & Horsfall (1960) have defined 'disease gradient' as a regression coefficient but, in general, the gradient terms lack explicit definition and fail as good measurement terms for the reasons already given above for 'deposition gradient'.

With respect to disease appraisal the only technical terms which qualify for Class I are those which specify precise methods and scales of measurement, such as the McKinney Index and the Horsfall-Barratt Scale. Disease severity, prevalence, intensity and incidence are often used loosely as measures of disease. Incidence and prevalence have meanings similar to each other and the former has been defined by James (1974) to mean the number of plant units diseased, a definition also used by Horsfall & Cowling (1978). Chester (1959) and the FAO Crop Loss Manual (1971) use intensity in the general sense of amount of disease, but Horsfall & Cowling (1978) improve the value of this term by defining it as the area or volume of plant tissue that is diseased. James (1974) uses severity to mean area diseased, but severity is a bad word because it denotes only a severe state.

Table 4. Classes of measurement terms used at the epidemic level of the pathosystem.

Class I[1] (Precise terms, explicitly defined) ←	Class II	→ Class III (Abstract terms, conceptually defined)
apparent infection rate r [T^{-t}] (*s.* Vanderplank, 1963)	disease gradient	disease severity
basic infection rate R [T^{-1}] (*s.* Vanderplank, 1963)	infection gradient	disease prevalence
sanitation ratio [1] (*s.* Vanderplank, 1963)	dispersal gradient	disease incidence
disease gradient [$N.L^{-1}$][2] (*s.* Dimond & Horsfall, 1960)	disease incidence (*s.* James, 1974)	disease intensity
McKinney Index (McKinney, 1923)	disease intensity (*s.* Horsfall & Cowling, 1978)	disease rating
Horsfall & Barrett Scale (Horsfall & Barrett, 1945)	disease severity (*s.* James, 1974)	

1. Class I includes other named methods for measuring disease.
2. When disease is measured as number of infections.

Summary

Two reasons gave rise to this critique of terms and definitions in modern epidemiology. First, there is a feeling among some workers that although conceptual unification is inevitable in any scientific enterprise, it is timely to strive purposefully for this goal in epidemiology in order to accelerate the conversion of research findings into efficient crop protection. The quest for a theoretical epidemiology is to be encouraged for it serves to integrate and synthesize disease phenomena into a broad vision and unified understanding of epidemics. The question arises as to whether terms and their definitions might be impeding progress along the paths towards this goal, signposted by comparative epidemiology.

The second reason stems from the 'modernness' of epidemiology. This feature is largely due to the marriage of crop disease science with mathematics, systems analysis and modelling; oscillating from analysis to synthesis, epidemiologists are reaching upward to more comprehensive views of their discipline. Again, the question arises as to whether definitions are adequate for today's technology.

The writers have approached these questions tentatively, fearful of accusations of quibbling over each term and its meaning. We have, therefore, refrained from unduly stipulating definitions and from coining new terms. It has been necessary to omit terms used specifically in connection with the host (e.g. 'predisposition'), resistance (e.g. 'virulence', 'adult plant resistance') and crop protection (e.g. 'negative prognosis').

The main purpose of this paper has been to sharpen the awareness of epidemiologists to the importance of technical language so that they might become more fastidious; epidemiology might then more easily consolidate into a unified science. To this end there have been three main objectives. First, to consider the nature of concepts, terms and definitions and to produce a classification which is epidemiologically meaningful.

Hopefully, this classification will be a framework for future discussions and also serve to highlight the quantity and quality of measurement terms used for research and development at various levels of the pathosystem.

The second objective has been to expose some gross ambiguity in basic terms used for the communication of ideas and for the formulation of concepts. Consistent definition is of crucial value.

Finally, the writers have stressed the importance of 'sharpening' measurement terms by the use of operational definitions. 'Blunt' definitions do not lead to precision in quantitative epidemiology and are certainly incompatible with modern technology and data usage. Operational definitions are a pre-requisite for precise empirical data which provide a sound foundation for a conceptualized epidemiology. It seems unlikely that standard rules of measurement can be adopted which will be suitable for the full diversity of plant pathogens and diseases; much could be done, however, towards establishing standard operational definitions within groups (e.g. powdery mildews) which have biological and ecological affinities.

Toulmin (1977) describes terminology as one of the intellectual 'keys' of science. Each generation of epidemiologists has a professional duty to safeguard the keys and to re-shape them as appropriate to open doors to progress.

References

Agrios, G. N., 1969. Plant pathology. Academic Press, New York.

Aylor, D. E., 1978. Dispersal in time and space: aerial pathogens. In J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 159–180.

Baker, R., 1978. Inoculum potential. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 137–157.

Beaumont, A., 1947. The dependence on the weather of the dates of outbreak of potato blight epidemics. Trans. Br. Mycol. Soc. 31: 45–53.

Butler, E. J. & S. G. Jones, 1949. Plant pathology. MacMillan & Co. Ltd., London.

Chamberlain, A. C., 1966. Transport of *Lycopodium* spores and other small particles to rough surfaces. Proc. R. Soc., London, Ser. A296: 45–70.

Chamberlain, A. C., 1975. The movement of particles in plant communities. In: J. L. Monteith (Ed.): Vegetation and the atmosphere. I. Principles. Academic Press, London, p. 155–203.

Chester, K. S., 1942. The nature and prevention of plant diseases. Blakiston Company, Philadelphia.

Chester, K. S., 1959. How sick is the plant? In: J. G. Horsfall & A. E. Dimond (Ed.): Plant pathology: An advanced treatise. I. The diseased plant. Academic Press, New York, p. 99–142.

Colhoun, J., 1973. Effects of environmental factors on plant disease. Annu. Rev. Phytopathol. 11: 343–364.

Cowling, E. B., & J. G. Horsfall, 1978. Prologue: How disease develops in populations. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 1–15.

Day, P. R., 1974. Genetics of host-parasite interaction. W. H. Freeman & Co., San Francisco.

Dimond, A. E. & J. G. Horsfall, 1960. Inoculum and the diseased population. In: J. G. Horsfall & A. E. Dimond (Ed.): Plant pathology: An advanced treatise. III. Academic Press, New York, p. 1–22.

Ellis, B., 1966. Basic concepts of measurement. Cambridge University Press.

Evans, E., 1968. Plant diseases and their chemical control. Blackwell Scientific Publications, Oxford & Edinburgh.
Federation of British Plant Pathologists, 1973. A guide to the use of terms in plant pathology. Phytopath. Pap. No. 17.
Food and Agricultural Organization of the United Nations, 1971. Crop loss assessment methods: FAO manual on the evaluation and prevention of losses by pests, diseases and weeds. FAO, Rome.
Garrett, S. D., 1956. Biology of root-infecting fungi. Cambridge University Press.
Garrett, S. D., 1970. Pathogenic root-infecting fungi. Cambridge University Press.
Gäumann, E., 1946. Pflanzliche Infektionslehre. Birkhäuser, Basel. (English Edition by W. B. Brierley, 1950. Crosby Lockwood, London).
Gregory, P. H., 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6: 189–212.
Gregory, P. H., 1973. The microbiology of the atmosphere. 2nd ed. Leonard Hill, London.
Heald, F. D., 1921. The relation of spore load to the percent of smut appearing in the crop. Phytopathology 11: 269–287.
Hempel, C. G., 1966. Philosophy of natural science. Prentice-Hall, Inc., New Jersey.
Hirst, J. M., 1959. Spore liberation and dispersal. In: C. S. Holton (Ed.): Plant pathology – Problems and progress 1908–1958. University of Wisconsin Press, p. 529–538.
Hirst, J. M. & R. D. Schein, 1965. Terminology of infection processes. Phytopathology 55: 1157.
Horsfall, J. G., 1932. Dusting tomato seed with copper sulphate monohydrate for combating damping-off. N.Y. Agric. Exp. Stn. Geneva, Tech. Bull. 198: 1–34.
Horsfall, J. G. & R. W. Barrett, 1945. An improved grading system for measuring plant disease. Phytopathology 35: 655.
Horsfall, J. G. & E. B. Cowling, 1978. Pathometry: the measurement of plant disease. in: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 119–136.
James, W. C., 1974. Assessment of plant diseases and losses. Annu. Rev. Phytopathol. 12: 27–48.
Jones, I. T., 1978. Components of adult plant resistance to powdery mildew (*Erysiphe graminis* f.sp. *avenae*) in oats. Ann. appl. Biol. 90: 233–239.
Kampmeijer, P. & J. C. Zadoks, 1977. EPIMUL, a simulator of foci and epidemics in mixtures of resistant and susceptible plants, mosaics and multilines. Simulation Monographs, Pudoc, Wageningen, the Netherlands.
Kast, F. E. & J. E. Rosenzweig, 1972. The modern view: a systems approach. In: J. Beishon & G. Peters (Ed.): Systems behaviour. Harper & Row Ltd., London, p. 14–28.
Kato, H., 1974. Epidemiology of rice blast disease. Rev. Pl. Prot. Res. 7: 1–20.
Kranz, J., 1978. Comparative anatomy of epidemics. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 33–62.
Krause, R. A. & L. B. Massie, 1975. Predictive systems: modern approaches to disease control. Annu. Rev. Phytopathol. 13: 31–47.
McKinney, H. H., 1923. Influence of soil temperature and moisture on infection of wheat seedlings by *Helminthosporium sativum*. J. Agr. Research 26: 195–218.
Mills, W. D. & A. A. Laplante, 1954. Diseases and insects in the orchard: apple scab. Cornell Agr. Exp. Stn. Ext. Bull. 711: 20–28.
Petersen, L. J., 1959. Relations between inoculum density and infection of wheat by uredospores of *Puccinia graminis var. tritici*. Phytopathology 49: 607–614.
Polley, R. W. & J. D. S. Clarkson, 1978. Forecasting cereal disease epidemics. In: P. R. Scott & A. Bainbridge (Ed.): Plant disease epidemiology. Blackwell Scientific Publications, Oxford, p. 141–150.
Schein, R. D., 1964. Design, performance, and use of a quantitative inoculator. Phytopathology 54: 509–513.

Sell, W., 1931. Staubausscheidung an einfachen Körpern und in Luftfiltern. Forschungsver. Dtsch. Ing. 347: 1–22.
Shaner, G., 1973. Reduced infectability and inoculum production as factors of slow mildewing in Knox wheat. Phytopathology 63: 1307–1311.
Shrum, R. D., 1978. Forecasting of epidemics. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 223–238.
Shurtleff, M. C. & A. Kelman, 1977. Diseases of plants. In: The new encyclopaedia Britannica macropaedia Vol. 5, p. 880–896.
Stakman, E. C. & J. G. Harrar, 1957. Principles of plant pathology. Ronald Press Company, New York.
Stamper, R., 1973. Information in business and administrative systems. B. T. Batsford Ltd., London.
Strobel, G. A. & D. E. Mathre, 1970. Outlines of plant pathology. Van Nostrand Reinhold Company, New York.
Tarr, S. A. J., 1972. The principles of plant pathology. Macmillan, New York.
Teng, P. S., M. J. Blackie & R. C. Close, 1977. A simulation analysis of crop yield loss due to rust disease. Agric. Syst. 2: 189–198.
Teng, P. S. & R. C. Close, 1978. Effect of temperature and uredinium density on urediniospore production, latent period, and infectious period of *Puccinia hordei* Otth. N.Z. J. Agric. Res. 21: 287–296.
Toulmin, S. E., 1977. Philosophy of science. In: The new encyclopaedia Britannica macropaedia Vol. 16, p. 375–393.
Vanderplank, J. E., 1963. Plant diseases: Epidemics and control. Academic Press, New York.
Vanderplank, J. E., 1975. Principles of plant infection. Academic Press, New York.
Vanderplank, J. E., 1976. Four essays: autobiography and re-viewing. Annu. Rev. Phytopathol. 14: 8–10.
Van der Wal, A. F., 1978. Moisture as a factor in epidemiology and forecasting. In: T. T. Kozlowski (Ed.): Water deficits and plant growth. V. Water and plant disease. Academic Press, New York, p. 253–295.
Verhoeff, K., 1974. Latent infections by fungi. Annu. Rev. Phytopathol. 12: 99–110.
Walker, J. C., 1969. Plant pathology. 3rd ed. McGraw-Hill, New York.
Wallin, J. R. & P. E. Waggoner, 1950. The influence of climate on the development and spread of *Phytophthora infestans* in artificially inoculated potato plots. Plant Dis. Reptr. Suppl. 190: 19–33.
Wheeler, B. E. J., 1969. An introduction to plant diseases. John Wiley & Sons Ltd., London.
Whetzel, H. H., 1929. The terminology of phytopathology. Proc. 1926 Int. Congr. Plant Sci. 2: 1204–1215.
Wood, R. K. S., 1967. Physiological plant pathology. Blackwell Scientific Publications, Oxford and Edinburgh.
Yarwood, C. E., 1978. Water and the infection process. In: T. T. Kozlowski (Ed.): Water deficits and plant growth. V Water and plant disease. Academic Press, New York, p. 141–173.
Zadoks, J. C., 1972a. Methodology of epidemiological research. Annu. Rev. Phytopathol. 10: 253–276.
Zadoks, J. C., 1972b. Reflections on disease resistance in annual crops. In: R. T. Bingham, R. J. Hoff, and G. I. McDonald (Ed.): Biology of rust resistance in forest trees. Proceedings of a NATO-IUFRO advanced study institute, August 17–24, 1969. U.S.D.A. Forest Service, Misc. Publ. 1221, p. 43–63.
Zadoks, J. C., 1977. On the epidemiological evaluation of fungicide action. Neth. J. Pl. Path. 83 (Suppl. 1): 417–426.

Zadoks, J. C., 1978. Methodology of epidemiological research. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. II. How disease develops in populations. Academic Press, New York, p. 63–96.

Zadoks, J. C. & L. M. Koster, 1976. A historical survey of botanical epidemiology. A sketch of the development of ideas in ecological phytopathology. Meded. Landbouwhogesch. Wageningen, the Netherlands 76: 1–56.

Flexibility of plant pathogens in exploiting ecological and biotic conditions in the development of epidemics

Hans J. Aust, Esther Bashi and Joseph Rotem

Abstract

The ability of pathogens to cause epidemics under dissimilar environmental conditions is assisted by the phenomenon of tolerance or of compensation. Tolerance is expressed in pathogens which develop in a wide range of environmental conditions (e.g. powdery mildews) or require specific conditions for development but pass the periods of adverse conditions with highly resistant resting bodies. In the case of compensation, the disease develops in spite of inferior weather conditions or a biological weakness of the pathogen or the host. In three categories of compensation phenomena, (i) a limiting environmental factor, (ii) a specific weak feature of the pathogen, and (iii) an inhibited phase of its development, compensation is provided by another (i) more nearly optimal environmental factor, (ii) strong feature of the pathogen, or (iii) more successful phase of its life cycle, respectively. In the fourth category of compensation, the host participates in compensation phenomena when its relative resistance is overcome by improved weather conditions or by large amounts of inoculum. In all cases, compensation extends the scope of disease by affecting the minimum and maximum, rather than the optimum, range of development. Experimental and circumstantial evidence suggest that in the field the combined effects of more than one compensation category are involved in compensation phenomena. Some are illustrated schematically.

Introduction

Associations between plant diseases and specific weather have been observed since ancient times. Experimentally determined minima, optima and maxima of environmental factors for disease development contributed to the acceptance of a direct link between epidemic rates and specific weather conditions. However, in numerous cases, the association between disease and weather deviates from the accepted rules. For instance, introductions of crops from many native habitats to habitats with entirely different weather have often been followed by the appearance of diseases hitherto unknown and unexpected in the new habitat. This point is illustrated by the spread of *Phytophthora infestans* (Mont.) de Bary to habitats with weather greatly deviating from classical 'blight weather'.

Waggoner & Horsfall (1969) pointed out the ability of early blight of potatoes to develop epidemics under widely varying climatic conditions. This and other examples suggest that the link between disease and weather is not always simple. In studies by Rotem et al. (1971) and Bashi et al. (1973), the minimum, optimum and maximum for the life cycle phases of several diseases were found not to be absolute but to vary with other factors. Following these and other findings, Rotem (1978) postulated that interactions between numerous meteorological or biotic factors allow the disease to by-pass an environmental obstacle. Due to these interactions, the same disease is able to develop in different climates; and diseases with different requirements can thrive, side by side, in the same habitat.

One of the objectives of comparative epidemiology is to gain better insight into the behaviour of host/pathogen/weather systems and to define principles explaining diverse or similar processes in plant diseases or in individual phases of their development (Kranz 1978). The phenomenon of compensation, as derived from comparative analysis of a number of diseases or their phases, constitutes one of these principles. It helps us better to understand the ecological pathways through which a given pathogen achieves its goal in different environments, or in which different pathogens do so in the same environment. Establishment of similarities and differences in epidemic patterns is another goal of comparative epidemiology, and is achieved by classifying the mechanisms of compensation into four categories. Most of the examples given come from our own experiments, which fitted our purpose because they have been specially designed for comparison of epidemiological facets on a quantitative basis.

A discussion of the phenomena of tolerance is included to illustrate additional ways in which pathogens can overcome environmental hazards.

Tolerance

Tolerance can be expressed in two ways: The ability to complete all the phases of the pathogen's life cycle under a wide range of climatic conditions; and the ability to survive prolonged periods of unfavourable conditions and enter a favourable, often short, vegetative season with an amount of active inoculum.

The first kind of tolerance is illustrated by many powdery mildews, which cause epidemics in a variety of geographic and climatic zones (Hirata, 1966). A large number of powdery mildews prefer dry conditions, but also develop in humid habitats, and spread without being seriously inhibited by humidity. A good example of the wide range of conditions for development of this group is *Sphaerotheca fuliginea* Schlecht. on cucurbits, the development of which is hardly affected by humid conditions (Reuveni & Rotem, 1974).

The second kind of tolerance is illustrated by a variety of pathogens that can develop only in relatively narrow environmental conditions, but due to formation of highly resistant resting bodies can overwinter or oversummer, without being adversely affected by weather. To this category belong many soil-borne diseases that require specific conditions for infection but survive for years if conditions are too dry or too hot for infection, or if their host is absent. Let us say no more about the characteristics of tolerance, but consider the phenomenon of compensation, which allows us to compare epidemics on a factor level (Kranz, this volume).

The categories of compensation

The phenomenon of compensation derives from processes of interaction, mechanisms of adaptation, and phenomena of substitution or balancing of ecological or biotic factors. However, not all of these processes, mechanisms or phenomena result in compensation in our sense. We refer only to those cases in which a limiting factor (or factors) of a given ecological or biotic nature would prevent epidemic development unless compensated by another factor (or factors) of a different ecological or biotic nature.

Rotem (1978) classified compensation phenomena into three categories.

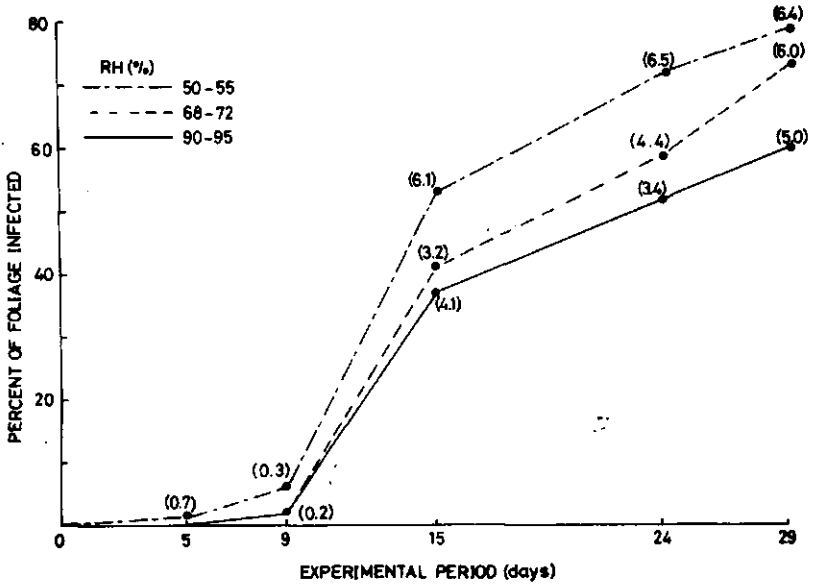

Fig. 1. The phenomenon of tolerance. The negligible effects of different levels of relative humidity on epidemic development of *Sphaerotheca fuliginea* on squash (figures in parentheses are standard errors). Growth chamber experiments after Reuveni & Rotem (1974).

1. The factor for factor category: 'A highly favorable state of one factor essential for development of a given phase in the life cycle of pathogen can compensate for the limitations imposed by the simultaneously unfavorable state of another factor.'
2. The strength for weakness category: 'A specific weakness in a pathogen can be compensated for by a specific strength.'
3. The phase-for-phase category: 'A high frequency of occurrence of one phase in the life cycle of a pathogen can compensate for a low frequency of occurrence of another phase in the life cycle of the same pathogen.'

We suggest a fourth category, in which relative resistance of the host, induced by horizontal resistance or age factors, is overcome by improved weather conditions, or by an increased amount or virulence of the inoculum. Compensations cannot, of course, occur in hypersensitive reactions.

The factor-for-factor category

Examples of the compensation phenomenon, when the temperature, the wetting period or the inoculum dose approaches marginal levels, have been provided by Rotem et al. (1971) for the infection process of potatoes by *P. infestans*. They showed (Fig. 2) that under conditions suitable for one or two out of the three factors, infection can succeed despite a marginal status of the third factor. This form of compensation is especially to be expected when one of the environmental factors limits a given phase of disease development. This is often so for *P. infestans* which, during the cool nights of temperate zones, is helped by prolonged wetting, but which overcomes short wettings in

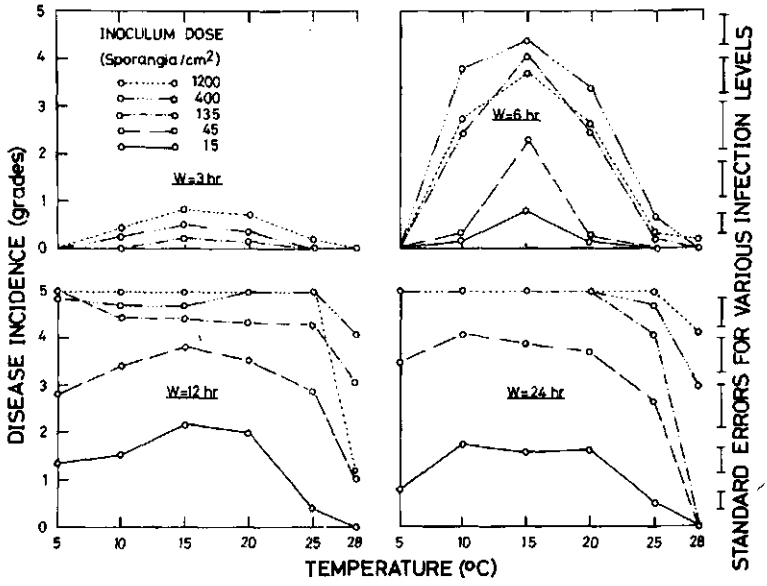

Fig. 2. Factor-for-factor compensation. The relativity of minimum, optimum and maximum in infection. Potato plants inoculated with various doses of sporangia of *Phytophthora infestans* and exposed to various temperatures during different durations of wetting became heavily infected also when one of the tested factors was nearly marginal for infection but its limiting effects were compensated for by another factor(s) present at a favourable level. After Rotem et al. (1971).

some Mediterranean countries like Israel, by conveniently warm night temperatures. Another example of the factor-for-factor category is the case of *Puccinia recondita* Rob. on wheat in which high light intensity compensates for the effect of low temperature on the rate of pustule maturation (Eyal & Peterson, 1967). Other cases are cited by Rotem (1978).

The strength-for-weakness category

The second form of compensation, that of a strong for a weak feature in the character of a given pathogen, was illustrated by Bashi & Rotem (1974; 1975) for *Stemphylium botryosum* (Wal.) f. sp. *lycopersici* R.C.W. in tomatoes. This pathogen requires for infection and sporulation wetting periods as long as 24 or 48 hours, which can be considered a 'weakness' in dry habitats. Disease caused by such a pathogen is expected to be restricted to very wet areas, but it also develops in Israel, where moist periods longer than 10 hours are rather rare. The ability of *Stemphylium* to develop under semi-arid conditions results from its 'strength', the resistance of slowly penetrating germ tubes or slowly emerging conidiophores to transient dryness. Due to this 'strong' feature, the pathogen can use, for either infection or sporulation, several short dew periods in several successive nights. Infection or sporulation is completed when the sum of several short wetting periods approaches the total required. In wet habitats *Stemphylium* may use continuous wetting without depending on compensation. In semi-arid

zones, it uses the interrupted wettings. The results in both regions are similar, although the ecological pathway to reach these results is different. Also pathogens that use prolonged wetting periods only for sporulation may face difficulties in some climatic regimes. The ability to sporulate with interrupted wetting periods was detected in *Alternaria solani* Sorauer on potatoes (Bashi & Rotem, 1975), *Rhynchosporium secalis* (Oud.) J. J. Davis on barley (Rotem et al., 1976), *Drechslera maydis* (Nisikado) Subram et Jain (Nelson & Tung, 1973), and *D. turcica* (J. Levi & Y. Cohen, unpublished data) on maize, and *Ascochyta pisi* Lib. on peas (R. Neubauer and J. Rotem, unpublished data). Rotem et al. (1978) speculated about the evolution of these phenomena. They suggested that the distribution of pathogens that need prolonged wetting (weakness in the sense of compensation) would be restricted to specific areas. However, their ability to survive transient conditions of dryness (strength in the sense of compensation) allows them to thrive equally well under wet and under alternating wet-dry conditions.

The ability to use interrupted wetting periods is apparently absent (and in fact not needed) where fungi require only a short wetting period to sporulate, e.g. *Peronospora tabacina* Adam on tobacco (Cruickshank, 1958) or to infect, e.g. *P. infestans* on potatoes (Bashi & Rotem, 1974). The relatively rapid conclusion of the sporulation or infection processes demonstrates a different kind of strength which compensates for a weakness in the inability of the sporangiophores or germ tubes to withstand dry conditions. This helps these pathogens to develop in a humid as well as in a drier habitat.

Rotem et al. (1978) point out that, in general, the proportion of a given pathogen species that survives contrasts with its productivity. In fertile but sensitive species, prolific sporulation may compensate for the low survival of the spores produced, while, in the robust but less fertile fungi, the high survival of spores produced may compensate for low productivity.

The strength-for-weakness compensation results from intrinsic features of the pathogen, which we tend to take for granted. Only a comparative analysis of the behavioural patterns of different pathogens can reveal the ways through which they succeed in different habitats. Studies of this kind are rare, although the processes involved can contribute a great deal to a better understanding of the phenomena of flexibility.

The phase-for-phase category

Environmental conditions may often highly favour a specific phase in the pathogen's life cycle, but much less favour its other phases. For instance, in a humid (but not rainy) habitat or period, nearly optimum conditions may exist for sporulation but not for air-dispersal of spores, which is encouraged by dry weather. Similarly in another habitat or with other weather, the short wetting periods may decrease the sporulation efficiency but not that of infection, which usually requires shorter wetting periods (Rotem et al., 1978). According to the classical concept of a harmonious sequence of phases in the pathogen's life cycle, a partial disturbance of one of its phases would limit disease development. However, situations rarely occur in which all phases of the life cycle proceed in an orderly succession under optimal conditions (Rotem, 1978). With assistance of compensating phenomena of the phase-for-phase category, the disease can overcome this obstacle.

The phase-for-phase compensation comprises cases in which massive sporulation

compensates for deficient infection and vice versa: for instance, efficient dispersal of inoculum compensates for deficiency in either sporulation or infection. An illustration of this kind of compensation was provided by Rotem & Cohen (1974) for patterns of potato blight epidemics induced in growth chambers with various climatic conditions. The most severe development of disease was favoured by a continuous drizzle, in which sporangia dispersed mainly by splash, as occurs in some typical blight habitats, and were not exposed to the survival hazards of wind dispersal and dryness. However, next in line were the severe epidemics which developed when only the nights were wet and the days were dry rather than humid. These are typical conditions of many semi-arid areas in which also the late blight disease succeeds well. The dominant factor for success of epidemics under dry rather than humid conditions was the most efficient dispersal of sporangia facilitated by the dry day-time conditions. Only when temperatures approached 30 °C did the adversely affected survival of dispersed sporangia counteract the dominant effect of dispersal.

This case again illustrates the different ecological pathways leading to epidemic development in different habitats.

The compensation for host resistance category

The factor mentioned, but not stressed enough in Rotem's (1978) description of compensation phenomena is the sometimes dominant role of the host. Indeed, some of the process of the pathogen's life cycle (germination, dispersal and survivability of spores) are mostly environment-dependent. However, the processes of penetration, colonization, and sporulation are also host-dependent. The relative resistance of the hosts (dependent on age or genetic factors) may also condition the obstacle faced by the pathogen, and the compensation phenomena needed to overcome it.

Compensation of large amounts of inoculum for low susceptibility of plant tissue was found by Aust (1979) in the barley powdery mildew system. The flag leaf and the next leaf are less susceptible to *Erysiphe graminis* DC. (adult resistance), because of their thicker epidermis and larger number of silified epidermis cells. Table 1 shows that increasing numbers of conidia were needed to induce infection of leaves which emerged

Table 1. The number of conidia of *Erysiphe graminis* f. sp. *hordei* needed in the field to induce one infection target in barley leaves that emerged on different dates and under different weather conditions (average of 10 replicates ± S.E.; after Aust, 1979)

Date in 1978	Mean temperature[1] °C	Leaf 1 (primary leaf)	Leaf 3	Leaf 5	Leaf 6	Leaf 7	Leaf 8 (flag leaf)
8–10 May	10.8	4.6±2.5	–	–	–	–	–
16–18 May	11.9	–	5.3±3.8	–	–	–	–
24–28 May	15.0	–	–	7.0±1.9	16.0±1.6	–	–
30 May–1 June	20.2	–	–	–	82.0±65	96±44	–
5–7 June	21.2	–	–	–	–	236±96	197±125
12–14 June	12.2	–	–	–	–	–	256± 96

1. Arithmetic mean of daily temperature.

later in the season. However, increased build-up of inoculum in the field compensates the need for larger amounts of inoculum to infect less susceptible leaves or, in other words, the amount of inoculum available compensates for the resistance of leaves. Bashi (unpubl. data) demonstrated that in the potato early blight system, resistance of the young leaves was overcome by increase in the wetting duration during the infection process. In pea plants, on the other hand, susceptibility to infection by *Peronospora pisi* Syd. is higher in young than in ageing plants. R. Neubauer and J. Rotem (unpubl. data), demonstrated that the relative resistance of the ageing plants can be overcome by more suitable temperatures or longer periods of wetting. A paradoxical but possibly quite common compensation by massive inoculum for age-induced resistance was described by Rotem & Bashi (1977) for *Stemphylium* blight epidemics in tomatoes. In this system, young rather than old plants are most susceptible to infection, but slow build-up of inoculum to a peak late in the season causes the epidemic to develop on old rather than young plants (Fig. 3).

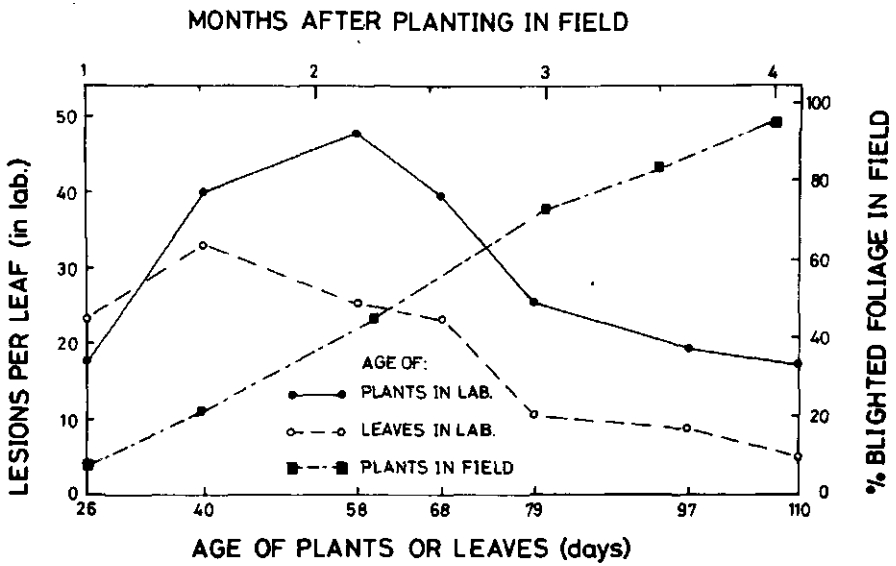

Fig. 3. Compensation for host resistance. The discrepancy between the climax in host susceptibility to *Stemphylium botryosum* f. sp. *lycopersici* in young tomato plants and the climax of epidemic development in more resistant, ageing plants in the field. Low disease incidence in the young plants in the field resulted from a slow build-up of inoculum characteristic of *S. botryosum* f. sp. *lycopersici*. Resistance to infection by ageing plants in the field was compensated by large amounts of inoculum present at this stage. After Rotem & Bashi (1977).

Finally, the compensating role of a long wetting period for the cultivar-dependent resistance was demonstrated by Hosford (1975, 1978) for wheat infected with *Phoma glomerata* (Corda) Wr. & Hochapf. and *Leptosphaeria microscopica* Karst. This suggests that resistance may be relative rather than absolute and that, in spite of its uniform genetic background, it may reach a different level in different environmental conditions.

Compensation phenomena

Compensation phenomena in the field

A precise establishment of the compensation category involved is possible only in carefully designed experiments under controlled conditions. Some of these experiments suggest that two, three or four of the compensation categories assist disease to develop under apparently unfavourable conditions. This is for potato late blight, in which the factor-for-factor (Rotem et al., 1971), strength-for-weakness (Bashi & Rotem, 1974) and phase-for-phase (Rotem & Cohen, 1974) categories act. Kröber (1967) demonstrated that the factors determining the wetting periods required for development of *Peronospora tabacina* on tobacco, are temperature, age of leaves, susceptibility of a given cultivar, and the amount of inoculum. We suggest that the almost world-wide distribution of tobacco downy mildew and potato late blight is made possible in part by the compensation phenomena.

Due to the variety of dynamic factors affecting epidemics in the field, establishment of compensation phenomena in general, and of the category of compensation in particular, is difficult. In the field, the final results of all the processes are to be seen. The climax of *Stemphylium* blight epidemics in less susceptible, ageing tomatoes, due to abundance of inoculum at that stage (Rotem & Bashi, 1977), would scarcely be possible without the factor-for-factor (Bashi et al., 1973) and strength-for-weakness (Bashi & Rotem, 1974, 1975) compensation. Analysing the role of inoculum build-up in epidemics, Rotem et al. (1978) pointed out the interaction of many factors affecting build-up. For instance, a rapid build-up of inoculum is characteristic of *Pseudoperonospora cubensis* Rost. in cucumbers, because of relatively high numbers of sporangia produced, their high infectivity, and relatively short latent period; this compensates for low survival of the sporangia. We suppose that the combined effects of more than one compensation category are usually responsible for most cases in which abundant inoculum compensates for some environmental or biotic deficiencies. This may possibly be true for epidemics of *Diplocarpon rosae* Wolf in roses in England (Saunders, 1966). This disease spreads when there is frequent rainfall, but once a critical level of inoculum is present, the epidemic continues to progress even though rainfall may become less frequent. The action of more than one compensation category is probably involved in the extension of epidemics of *Peronospora destructor* (Berk.) Casp. on onions to seasons not suitable for their development (J. Palti, personal communication): this downy mildew develops in Israel in the cool and humid winter, and continues to develop at the beginning of the hotter and drier season, because of large amounts of available inoculum. By a similar mechanism, cucumber downy mildew, chiefly a warm temperature (summer) disease, extends its action to the cool autumn and winter.

Compensation phenomena in soil-borne diseases

We can only speculate about how common compensation phenomena are in soil habitats. Although the environmental factors in the soil are relatively stable, they interact frequently with biotic factors. These interactions result in an extremely complex environment in which a change in one factor (such as temperature, moisture, oxygen, carbon dioxide or biotic factors) leads to a change in the others (Rotem, 1978).

These conditions may stress the flexibility required from a successful pathogen. Indeed, compensation phenomena for soil-borne diseases have been described in publications by Colhoun (1961), Colhoun et al. (1968) and by Peerally & Colhoun (1969). For instance, in clubroot of *Brassicae*, *Fusarium culmorum* (W. G. Smith) Sacc. in wheat, and *Phoma chrysanthemicola* in chrysanthemum, the large amount of inoculum, the soil acidity and some other factors compensate for unfavourable conditions of soil moisture or temperature.

With *Rhizoctonia solani* Kühn on potatoes, the effect of different temperatures on disease development was felt only when the inoculum level was low. An increased amount of inoculum on seed tubers compensated for unfavourable (high) temperatures and allowed the pathogen to succeed under conditions otherwise marginal (Bolkan et al., 1974).

A comprehensive view of compensation phenomena

An octagon, instead of the classical host/pathogen/environment triangle, illustrates schematically the interwoven action of compensation (Fig. 4). For instance, the limiting effect of a small amount of inoculum can be compensated by high susceptibility of the host or high infectivity of spores, or suitable weather. Similar compensations are presumed to act in other combinations of the factors. The limit for compensation starts when one of the major factors is submarginal. Thus, the most suitable temperatures or

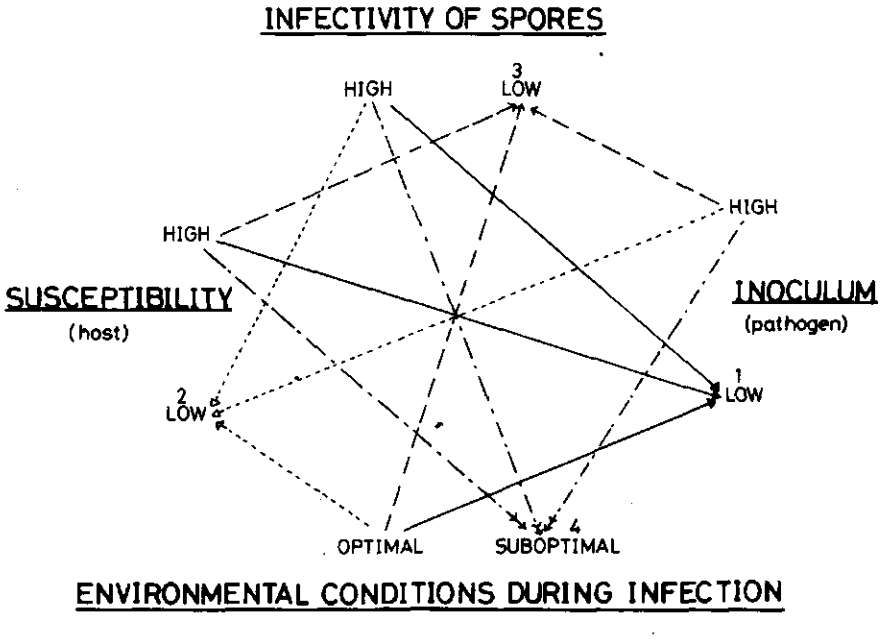

Fig. 4. Interaction between host, pathogen and environment, illustrating schematically the general concept of compensation. The classical host/pathogen/environmental triangle becomes here an octagon.

the greatest amounts of inoculum will not compensate for the absence of moisture or for the lack of compatibility in the host-pathogen system.

The literature suggests that compensation acts on the minimum and maximum rather than on the optimum range of disease development (for example, Fig. 2). A schematic presentation of this trend is given in Figure 5, in which the solid line illustrates development without compensation. The columns show increase of disease in the limiting ranges (dotted line) with compensation. Thus, compensation extends (arrows) the scope of disease development beyond its normal limits. The examples of *Stemphylium* blight in tomatoes (Rotem & Bashi, 1977), *Diplocarpon rosae* on roses (Saunders, 1966), *Rhizoctonia solani* on potatoes (Bolkan et al., 1974), and downy mildew on onions and cucumbers (J. Palti, personal communication), illustrate this point.

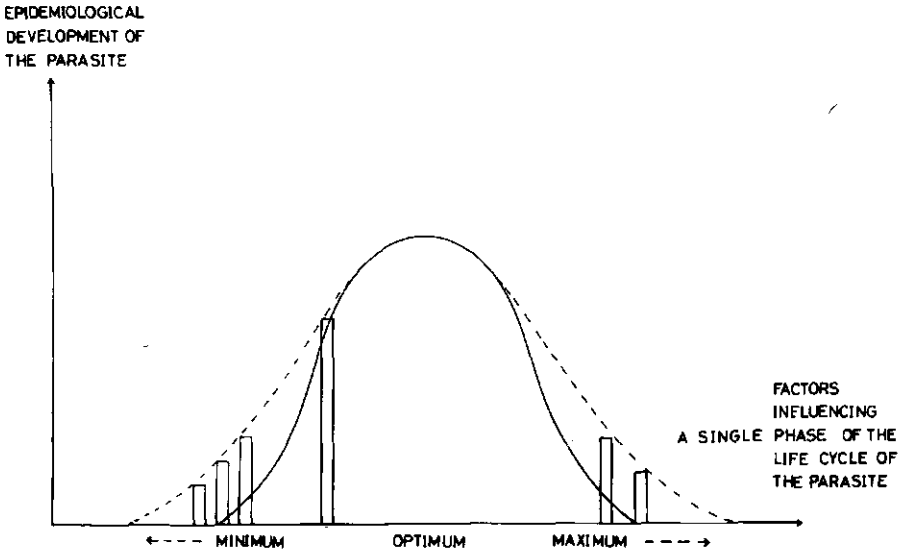

Fig. 5. Schematic illustration of the effect of compensation acting on the minimum and maximum rather than on the optimum range of disease development and extending its scope beyond its 'normal' limits.

So a proper understanding is required of pathogens' tolerance for various conditions and of their ability to compensate for inferior conditions or biological weakness, and to develop epidemics in spite of them. Such an understanding should be of help in deciding where to intervene in disease development, or indeed, whether such intervention has a reasonable chance of success.

References

Aust, J., 1979. Über den Verlauf von Mehltauepidemien innerhalb des Agro-Ökosystems Gerstenfeld. Habilitationsschrift, Universität Giessen.

Bashi, E. & J. Rotem, 1974. Adaptation of four pathogens to semi-arid habitats as conditioned by penetration rate and germinating spore survival. Phytopathology 64: 1035–1039.

Bashi, E. & J. Rotem, 1975. Sporulation of *Stemphylium botryosum* f. sp. *lycopersici* in tomatoes

and of *Alternaria porri* f. sp. *solani* in potatoes under alternating wet-dry regimes. Phytopathology 65: 532–535.

Bashi, E., J. Rotem & J. Putter, 1973. Effect of wetting duration, and of other environmental factors, on the development of *Stemphylium botryosum* f. sp. *lycopersici* in tomatoes. Phytoparasitica 1: 87–94.

Bolkan, H. A., H. T. Wenham & K. S. Milne, 1974. Effect of soil temperature on severity of *Rhizoctonia solani* infection on potato shoots. Plant Dis. Rep. 58: 646–649.

Colhoun, J., 1961. Spore load, light intensity and plant nutrition as factors influencing the incidence of club-root of Brassicae. Trans. Brit. mycol. Soc. 44: 593–600.

Colhoun, J., G. S. Taylor & R. Tomlinson, 1968. *Fusarium* diseases of cereals. II. Infection of seedlings by *F. culmorum* and *F. avenaceum* in relation to environmental factors. Trans. Brit. mycol. Soc. 51: 397–404.

Cruickshank, I. A. M., 1958. Environment and sporulation in phytopathogenic fungi. I. Moisture in relation to the production and discharge of conidia of *Peronospora tabacina* Adam. Aust. J. Biol. Sci. 11: 162–170.

Eyal, Z. & J. L. Peterson, 1967. Uredospore production of five races of *Puccinia recondita* Rob. ex Desm. as affected by light and temperature. Can. J. Bot. 45: 537–540.

Hirata, K., 1966. Host range and geographical distribution of the powdery mildews. Niigata University, Japan.

Hosford, R. M. Jr., 1975. *Phoma glomerata*, a new pathogen of wheat and triticales, cultivar resistance related to wet period. Phytopathology 65: 1236–1239.

Hosford, R. M. Jr., 1978. Effects of wetting period on resistance to leaf spotting of wheat by *Leptosphaeria microscopica* with conidial stage *Phaeoseptoria urvilleana*. Phytopathology 68: 908–912.

Kranz, J., 1978. Comparative anatomy of epidemics. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise. Vol. II. Academic Press, New York, p. 33–62.

Kröber, H., 1967. Der Einfluss der Benetzungsdauer auf die Entstehung der Blauschimmelkrankheit an Tabak. Phytopathol. Z. 58: 46–52.

Nelson, R. R. & G. Tung, 1973. Influence of some climatic factors on sporulation by an isolate of race T of *Helminthosporium maydis* on a susceptible male-sterile corn hybrid. Plant Dis. Rep. 57: 304–307.

Peerally, M. A. & J. Colhoun, 1969. The epidemiology of root rot of chrysanthemums caused by *Phoma* sp. Trans. Brit. mycol. Soc. 52: 115–123.

Reuveni, R. & J. Rotem, 1974. Effect of humidity on epidemiological patterns of the powdery mildew (*Sphaerotheca fuliginea*) on squash. Phytoparasitica 2: 25–33.

Rotem, J., 1978. Climatic and weather influences on epidemics. In: J. G. Horsfall & E. B. Cowling (Ed.): Plant disease: An advanced treatise Vol. II. Academic Press, New York, p. 317–337.

Rotem, J. & E. Bashi, 1977. A review of the present status of the *Stemphylium* complex in tomato foliage. Phytoparasitica 5: 45–58.

Rotem, J., B. G. Clare & M. V. Carter, 1976. Effects of temperature, leaf wetness, leaf bacteria and leaf and bacterial diffusates on production and lysis of *Rhyncosporium secalis* spores. Physiol. Plant Pathol. 8: 297–305.

Rotem, J. & Y. Cohen, 1974. Epidemiological patterns of *Phytophthora infestans* under semi-arid conditions. Phytopathology 74: 711–714.

Rotem, J., Y. Cohen & E. Bashi, 1978. Host and environmental influences on sporulation *in vivo*. Annu. Rev. Phytopathol. 16: 83–101.

Rotem, J., Y. Cohen & J. Putter, 1971. Relativity of limiting and optimum inoculum loads, wetting durations, and temperatures for infection by *Phytophthora infestans*. Phytopathology 61: 275–278.

Saunders, P. J. W., 1966. Epidemiological aspects of blackspot disease of roses caused by *Diplocarpon rosae* Wolf. Annu. appl. Biol. 58: 115–122.

Waggoner, P. E. & J. G. Horsfall, 1969. Epidem. Bull. Conn. Agric. Exp. Stn. 698. 80 pp.

An ecological approach to the epidemiology of plant virus diseases

J. M. Thresh

Abstract

The spread of pests and diseases into and within crops has many features in common with the colonization and subsequent exploitation of natural habitats by plants and animals. This justifies the ecological approach adopted in this paper, using concepts developed from studies of the reproductive strategies and dispersive abilities of plants and animals in relation to the type and stability of the habitats exploited.

Attention is drawn to the great difference in rates and patterns of spread of plant virus diseases. Those of short-lived annual crops spread quickly and behave like '*r*-selected' colonizing species that exploit ephemeral habitats. By contrast, diseases of woody perennials resemble '*K*-selected' equilibrium species of relatively stable situations. Intermediate types also occur and categorizing diseases in this way can be used to gain new insights into their behaviour.

An ecological approach along these lines is shown to be widely applicable in comparative epidemiology and can be extended to the whole array of pests, parasites and diseases of plants and animals.

Introduction

Higher plants and animals provide the habitat for a diverse array of pests, parasites and diseases, many of which have long been recognized as the cause of plague, pestilence and famine. Their importance in human affairs provided the initial impetus for an ever-expanding range of studies into the various diseases of man, livestock and crop plants. This research has inevitably become increasingly specialized and there is now only limited contact between workers on medical, veterinary and botanical topics or between bacteriologists, mycologists, virologists, parasitologists and entomologists. Such a restricted and inhibiting approach has serious limitations, whether considered pragmatically or on theoretical grounds. Progress is impeded by the limited interchange of information and yet many of the most intractable problems can only be solved by a multidisciplinary approach. Moreover, common biological and ecological features that characterize the evolution, growth, survival, perennation and dispersal of all types of plant and animal pathogens tend to be overlooked or to be considered as separate unrelated phenomena.

One of the main aims of comparative epidemiology is to facilitate liaison and to promote the development of unifying concepts of wide general validity by comparing and contrasting the behaviour of different types of disease. This paper contributes towards this objective by considering the epidemiology of plant virus diseases in relation to current ecological principles. These have been almost totally ignored by plant pathologists and yet they are widely applicable and can be used to gain new insights into the behaviour of all pests, parasites and diseases of plants and animals.

Ecological concepts and their application to epidemiology

Some relevant ecological concepts

Ecologists have recently been giving considerable attention to the reproductive strategies and dispersive abilities of plants and animals in relation to the type of habitat exploited. The work of MacArthur & Wilson (1967) as presented in their 'Theory of Island Biogeography' has been particularly influential in developing current attitudes. These authors consider the initial colonization and subsequent exploitation of islands and other types of discrete habitat in relation to their size, climate, diversity and isolation. A distinction is made between the main attributes of species prominent in the early stages of colonization and those predominating later, when there is increased crowding of the environment and competition within and between species. Initially when sites are largely unoccupied and provide an 'ecological vacuum' the optimum reproductive strategy is to maximize numbers by quickly producing numerous progeny of the smallest effective size. As density-dependent competition effects become increasingly important conditions favour quite different species. These channel all available resources into producing and maintaining a few outstandingly fit offspring able to compete successfully for the available substrates.

Opportunist, colonizing species are referred to as '*r*-selected' for comparison with equilibrium '*K*-selected' species. These terms are derived from the well-known logistic equation (for r and k concepts see p. 8 and p. 73) in which the rate of change in a population (dN/dt) is related to the existing population (N) and to the maximum intrinsic rate of reproduction (r_{max}) as modified by an additional factor ($1 - N/K$) dependent on the extent to which the population approaches the carrying capacity of the habitat (K) i.e.:

$$dN/dt = N.r_{max}\,(1 - N/K)$$

Actual rates of reproduction are greatest when N is small in relation to K and conditions favour highly productive '*r*-selected' species. Reproductive rates are least when N approaches K in situations where the predominant species are K-selected with highly-developed competitive abilities.

Pianka (1970) and Southwood (1977a) list various ancillary features of r and K-selected species (Table 1). They stress the longevity, large size, limited mobility and low, delayed fecundity of K strategists. These frequent durable environments and usually maintain extremely stable populations, although they are vulnerable to extinction if there is a sudden unusually drastic change in conditions. Strategists of the r type are mainly opportunist, mobile species with well-developed abilities for rapidly invading and exploiting ephemeral sites. They have the resilience to recover quickly from drastic decreases in number of the type encountered regularly in their 'boom and bust' cycles of population growth and decline in transient habitats.

Considering the animal kingdom as a whole, vertebrates are regarded as K strategists, whereas insects and other invertebrates tend to be mainly of the r type. However, within each taxon there is great diversity and some species are much nearer the extreme limits of the continuum than others. Physical factors are the main constraints on population growth in regions where the climate is markedly seasonal and favours the emergence of r strategists. The situation is completely different in the relatively uniform conditions

Table 1. Some contrasting features of extreme *r* and *K* selected species. Adapted from Pianka (1970) and Southwood (1977a).

	r species	*K* species
Body size	small	large
Generation time	short	long
Fecundity	high	low
Reproduction	early	late
Longevity	short	long
Development	rapid	slow
Populations	variable	fairly constant
	often 'overshoot'	seldom 'overshoot'
	usually below *K*	usually approach *K*
Mortality	density-independent	density-dependent
Competition	often lax	usually intense
Defence mechanisms	infrequent	well developed
Colonisation	frequent	infrequent
Mobility	high	low
Climate	variable	less variable
	often unpredictable	more predictable
Habitats	unstable	stable
Dominant characteristic	productivity	efficiency

of the humid tropics where competition effects are of paramount importance (Dobzhansky, 1950).

The concept of a continuous spectrum of reproductive strategies and behaviour ranging from extreme forms of *r* to *K* has already been used widely in assessing the overall features of diverse organisms of various taxonomic groups including mammals (Pianka, 1970), birds, insects (Southwood et al., 1974), corals (Loya, 1976), echinoderms (Moore, 1978), vascular plants (Gadgil & Solbrig, 1972; Harper, 1977), algae (Wyatt, 1974) and animal parasites (Esch et al., 1977). Comparisons have been made within and between groups to facilitate an understanding of their ecology and of the ways in which organisms have become adapted to the type of habitat exploited. Similar assessments are possible of plant diseases, which are considered in the following sections with particular reference to those due to viruses.

An ecological approach to epidemiology

There are obvious parallels between the invasion of crops by an influx of weeds, pests or diseases and the colonization of natural habitats by plants and animals (Harper, 1977). This means that the epidemiology of virus and other diseases can be assessed in ecological terms. Indeed, crops can be regarded as discrete island habitats fully comparable to those considered by MacArthur & Wilson (1967). Similar factors influence the invasion of crops and islands at a rate dependent on the catchment area and on the size and proximity of the nearest source of inoculum (Thresh, 1976). Extensive plantings close to major foci are particularly vulnerable and the invasion of remote sites is facilitated by the presence of intervening areas of susceptible hosts. These act like the islands of an archipelago in aiding colonization.

The analogy between crop and island habitats can be extended to include taxonomic and temporal aspects as well as spatial ones. Host species can be regarded as evolutionary islands and their pests and diseases are more likely to become adapted to attack closely related species than those that are remote (Janzen, 1968). Similarly, short-lived annual crops grown for brief periods of the year in regions of markedly seasonal climate are isolated in time from previous and later plantings. They provide a transient habitat for pests and diseases, which encounter formidable problems of survival during the sometimes prolonged periods of drought or extreme cold. The risk of extinction is much less where susceptible crops are grown in continuous sequence throughout the year, or as long-lived perennials. Many of these are propagated vegetatively and retained for many years at the same site or at a sequence of sites.

There are great differences between crops in the type, extent and stability of the habitat they provide for pests and diseases (Table 2). Thus it is hardly surprising that viruses resemble other pathogens in having evolved various strategies of survival. There are also big differences between viruses in their 'epidemiological competence', using this term in the sense of Crosse (1967) for the ability of a pathogen to sustain the necessary sequence of infection.

Table 2. Some features of the crop habitat influencing the spread of virus diseases.

	Spread	
	facilitated	impeded
Host susceptibility	high	low
Host longevity	long	short
Host size	large	small
Vulnerable plantings	many	few
	contiguous	scattered
Crop stands	pure	mixed
Crop spacing	close[1]	wide
Sources of infection	many	few
	local	distant
	potent	less potent
Growing seasons	long	short
	overlapping	distinct
Winter/dry season	mild	extreme
	short	prolonged

1. The spread of some aphid-borne viruses is decreased at particularly dense spacings (e.g. Hull, 1964).

The spread of plant viruses

Means of spread

Plant viruses exploit various means of spread by:

1. direct contact

2. pollen and/or seed
3. soil-inhabiting vectors
4. arthropod vectors
5. vegetative propagation

These methods have been discussed in detail elsewhere (Thresh, 1974a, 1978) and it suffices here to consider their overall effectiveness in achieving an adequate distribution of inoculum.

Local spread leads to the progressive enlargement of existing outbreaks with little loss of inoculum except to plants already infected. However, spread is circumscribed and mainly to plants growing under the same conditions and in a similar phase of development. Spread over greater distances is much more hazardous, as much of the inoculum is carried into unfavourable habitats or far beyond the range of susceptible hosts. Nevertheless, it is of crucial importance in starting new outbreaks in additional areas and in younger plantings. In ecological terms there are obvious advantages in a dual strategy of dispersal that permits the invasion of new habitats and the exploitation of existing ones. This is achieved by a single versatile mean of dispersal or by two or more distinct but complementary methods.

Each of the various means of spread is involved in the local distribution of inoculum, but only some contribute to long-distance dispersal (Table 3). For example, spread is obviously restricted when due to contact between adjacent plants or to soil-inhabiting or other vectors of limited mobility (Fig. 1). Viruses spread in these ways are unlikely to become prevalent without additional means of dispersal in seeds, crop debris or vegetative propagules. The extensive traffic in plant material is particularly important in facilitating the dissemination of viruses, and in some instances their vectors, at a speed and over distances unlikely to be achieved by natural means.

Table 3. Methods of virus spread locally and over greater distances.

Transmission	Spread	
	local	distant
Contact	+	−
Pollen	+	±
Seed	+	+
Vegetative propagules	+	+
Inactive vectors	+	−
Active vectors	+	+

Nematode and fungal vectors move slowly through the soil and over short distances, yet they are highly effective in transmitting viruses between the roots of adjacent plants. Spread is also facilitated by the ability of the vectors and the viruses they transmit to attack a wide range of hosts or to persist during the sometimes prolonged period between plantings of susceptible crops (Harrison, 1977). In several respects the role of nematodes and fungi is similar to that of the mite and tick vectors of animal viruses that

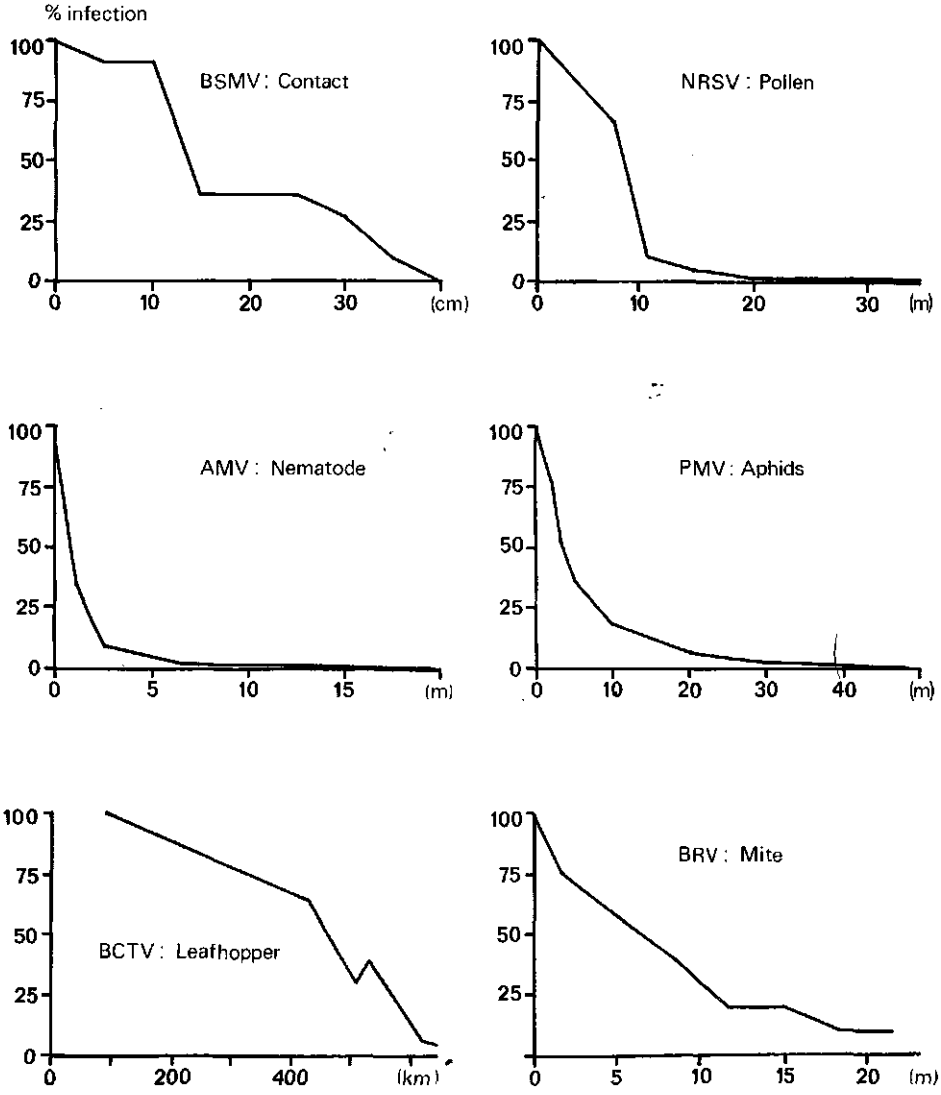

Fig. 1 Disease gradients due to the spread of: barley stripe mosaic virus (BSMV) by contact (Slack et al., 1975), prunus necrotic ringspot virus (NRSV) by pollen (Davidson & George, 1964), arabis mosaic virus:– hop strain (AMV-H) by nematodes (Thresh & Manwell, unpublished), peanut mottle virus (PMV) by aphids (Demski, 1975), beet curly top virus (BCTV) by leafhoppers (Romney, 1939), black currant reversion virus (BRV) by eriophyid mites (Thresh, 1966). Note the different scales expressed in centimetres (cm), metres (m), or kilometers (km).

persist for long periods without food whilst waiting to encounter suitable vertebrate hosts (Sonenshine, 1974).

Arthropod vectors have elaborate dispersal and host-finding mechanisms and they play a much more active role in the spread of plant viruses than soil-inhabiting nematodes or fungi. With aphids and some other specialized groups there is at least some degree of polymorphism, involving the production of different types of contrasting structure, behaviour and mobility. Variable proportions of active and less active individuals are produced according to circumstances, so providing a single versatile means of spread locally and periodically over much greater distances. The forms of limited mobility contribute to movement within crops and tend to predominate at times when conditions favour growth and reproduction. By contrast, the active migrants appear and disperse when seasonal or other conditions are unfavourable, when populations are crowded, or when the host plants are beginning to senesce and die.

The migratory habit is a particularly well-developed feature of species exploiting short-lived hosts or ephemeral habitats (Southwood, 1962). It is of crucial importance in survival, enabling the transfer of a portion of the breeding population to fresh sites before the original ones disappear or become untenable. Migration also ensures that viruses are carried frequently and in some instances over very great distances by vectors that are infective on take-off or that acquire virus whilst dispersing. The rapidity and efficiency with which crops are colonized by arthropod vectors account for the early appearance and subsequent spread of many viruses. There are many striking examples of virus spread by vectors migrating from ripening crops or otherwise deteriorating environments. Spread may be to nearby crops at an earlier and more vulnerable stage of development, or over greater distances into entirely new areas (Fig. 1).

Some of the most spectacular instances of long-distance spread have been recorded in North America, where curly top virus is carried far into sugar beet and other important crops by leafhoppers migrating from ephemeral weed hosts (Bennett, 1971). Similarly barley yellow dwarf virus is regularly introduced into the north-central states and sometimes into Canada by winged aphids originating from maturing grain crops hundreds of miles to the south in Texas and Oklahoma (Bruehl, 1961). Groundnut rosette in Central Africa and subterranean clover stunt in Australia are other persistent aphid-borne viruses that are regularly transported between distant localities (Gutierrez et al., 1974).

There are obvious analogies between the distant spread of plant viruses by migrating vectors and the long-distance dispersal of certain insect pests (Johnson, 1969), fungal spores (Gregory, 1974) and viruses of livestock (Sellers & Forman, 1973; Sellers et al., 1977). Similar back-tracking procedures have been used in each instance to analyse meteorological data so as to identify the most likely source and trajectory of the wind-borne inoculum. However, air-borne spores can be treated as inert particles, whereas complex behavioural factors influence the take-off, flight and landing of insect migrants. Unlike fungal spores these can reach and infect a sequence of host plants before eventually settling to feed and breed.

Rates of spread

When following the spread of diseases into and within crops virologists can seldom consider changes in the amount of pathogen present or in the quantity of infected tissue.

It is usually necessary to assess changes in the number or proportion of infected plants occurring in representative plots or transects. Data of this type showing the progress of diseases in commercial or experimental plantings of diverse crops have been obtained in various regions of the world (Thresh, 1974b).

In assessing the extensive literature it is important to appreciate the serious limitations of the available data. It is seldom apparent whether published observations relate to typical situations or to exceptional ones where there has been unusually rapid spread. Moreover, attention has been concentrated on particularly important crops, mainly in temperate regions of the world with highly-developed systems of agriculture. There is little information on the spread of many virus diseases of obvious economic significance and studies are only just beginning in many important agricultural areas of the world.

Despite these limitations, generalizations are possible and many curves of disease progress show a sigmoid trend with time. However, within the same overall pattern there are great differences between sites, seasons and diseases in the onset, rate, duration and total amounts of spread (Fig. 2). After the first appearance of disease there is usually a rapid increase in the cumulative total of infected plants. The rate of increase then declines as weather or other conditions become unfavourable or because progressively fewer healthy plants remain to be infected and multiple infection becomes increasingly important (Gregory, 1948). With many diseases the true rate of spread declines as an increasing proportion of the infected plants become remote from the remaining healthy ones and make little further contribution to spread. The effect is particularly important with 'crowd' diseases: those that do not usually spread far in any considerable amount (Vanderplank, 1948). This explains why virus diseases seldom have a truly logarithmic phase of increase during which the amount of new infection is proportional to that already present.

A striking feature of the published data is the rapid spread of virus diseases of herbaceous annuals compared with those of woody perennials. The time-scale of epidemics in plantation crops such as cacao, grapevine, citrus, cherry, plum and peach is measured in years. By contrast, plantings of herbaceous crops including tobacco, tomato, sugar beet, brassicas, grain legumes, rice and various other cereals may be almost totally infected within a single growing season of a few months duration (Fig. 2). There is much less information on the spread of diseases in herbaceous perennials and the main observations relate to potato, sugar cane, strawberry, carnation and various other ornamentals in which generally intermediate rates of spread have been reported.

It is rarely possible to compare the behaviour of different diseases caused by the same virus in dissimilar hosts. However, cucumber mosaic virus can spread rapidly in lettuce, cucurbits, sugar beet and other herbaceous hosts, whereas it spreads slowly or not at all at the few sites where infection has been encountered in woody perennials such as raspberry, black currant, red currant, cherry and plum. There are also big differences in the rates of spread of diseases caused by viruses of the same taxonomic group that are transmitted similarly by their vectors. For example, plum pox spreads slowly in orchards, yet other members of the potyvirus group that are also transmitted non-persistently by aphids soon become prevalent in plantings of cucurbits and capsicum peppers. There are also great differences in the rates of spread by aphids of the closteroviruses causing sugar beet yellows and citrus tristeza diseases.

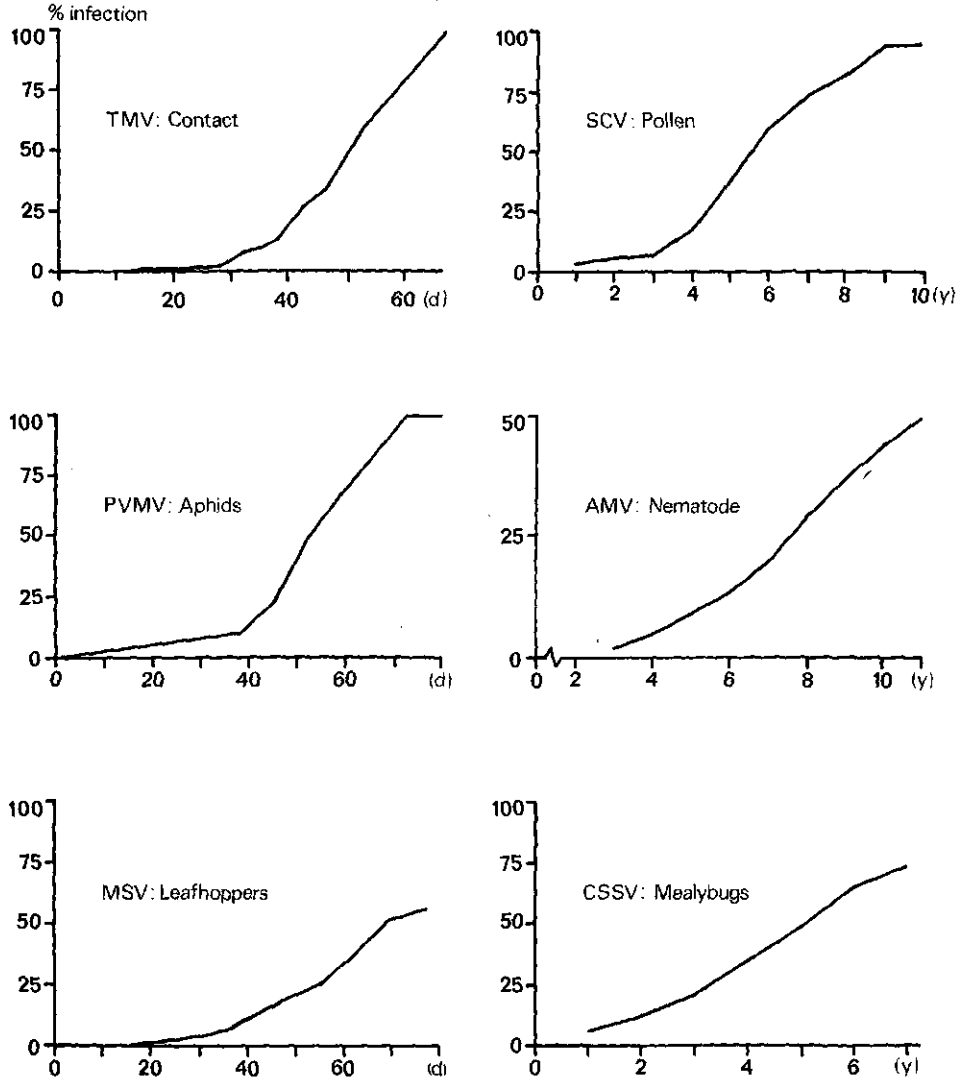

Fig. 2. Disease progress curves due to the spread of: tomato mosaic virus (TMV) by contact (Selman, 1943), viruses of sour cherry (SCV) by pollen (Davidson & George, 1964), arabis mosaic virus:– hop strain (AMV-H) by nematodes (Thresh & Manwell, unpublished), pepper vein-banding mosaic virus (PVMV) by aphids (Simons, 1957), maize streak virus (MSV) by leafhoppers (Rose, (1974), cacao swollen shoot virus (CSSV) by mealybugs (Thresh 1958, and unpublished). Note the different time scales expressed in days (d) or years (y).

The different behaviour of viruses in herbaceous annuals and woody perennials was first considered by Vanderplank (1949, 1959). He suggested that the large size and longevity of trees makes them so vulnerable to infection that they have evolved a general type of resistance as an important compensating feature. Woody plants certainly tend to be difficult to infect and there is a generally prolonged interval after infection before they are invaded systemically, develop symptoms, and become infectious foci from which spread can occur to other plants.

Spread in annuals is facilitated by the close spacings adopted and by the short time required for inoculated plants to be invaded systemically and become infectious. This permits numerous cycles of infection within a single growing season. However, diseases such as tomato spotted wilt and 'knomnek' disease of tobacco can become prevalent even though they are monocyclic and do not spread within crops. They are carried into plantings by an influx of active vectors dispersing from outside weed sources.

r and K strategies of virus disease spread

The various contrasting attributes of *r* and *K* species listed in Table 1 became apparent from studying free-living plants and animals (see also Zadoks & Schein, this volume). Nevertheless, they can be used with little modification as a basis for assessing the behaviour of virus diseases. An initial difficulty concerns the term '*r*' and it is necessary to consider rates of disease progress within host populations. Rates of virus multiplication within plant tissues are inappropriate because they are not closely related to pathogenicity or to spread between plants.

Many virus diseases of annual crops have the main features of *r* species. They have such mobile vectors that they soon reach new plantings and spread quickly to cause serious epidemics during the brief periods when susceptible hosts are available and climatic conditions are suitable. Groundnut rosette, barley yellow dwarf, beet curly top, subterranean clover stunt and beet yellowing diseases are particularly striking examples. Turnip mosaic, pepper vein-banding, lettuce mosaic, watermelon mosaic and many other diseases of market garden crops also behave as *r* strategists, albeit with somewhat different characteristics. They spread over relatively short distances and yet maintain a continuous sequence of infection in successive short-term plantings.

The ability to spread quickly from a few initial foci whenever conditions are favourable can be seen as an essential adaptation for long-term survival in ephemeral hosts. It confers great resilience and enables diseases to withstand determined attempts at control and to persist despite major fluctuation in weather or other conditions. These lead to great differences between sites and seasons in the prevalence of infection (Fig. 3).

The situation with perennial crops is completely different. A slow rate of spread will suffice to maintain infection and may even be advantageous for the long-term survival of very damaging viruses and their sensitive hosts. This accounts for the behaviour of virus diseases of woody perennials, which have many of the characteristics of *K*-selected species. Diseases such as cacao swollen shoot, plum pox and citrus tristeza are slow to invade new areas unless introduced in contaminated batches of plant material. Spread is slow at existing outbreaks and can be impeded by eradication measures that are seldom effective against virus diseases of annuals. However, there is no evidence that spread is influenced by the presence of other viruses in the way that populations of *K*-species of plants and animals are restricted by intra- or inter-specific competition effects.

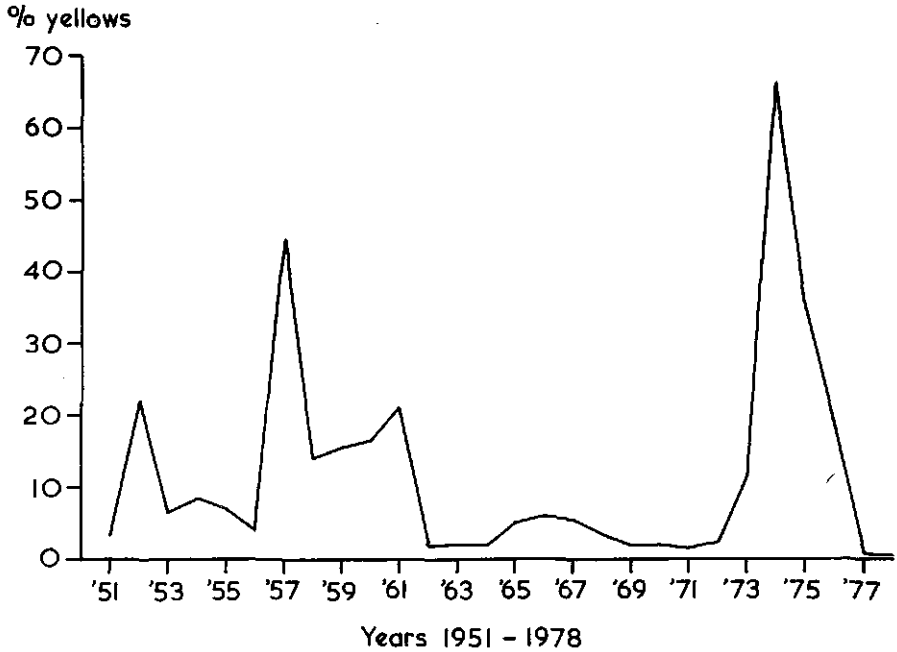

Fig. 3. The overall incidence of virus yellows in British sugar beet plantings 1951–1978. Data compiled at Broom's Barn Experimental Station from surveys of representative sites done annually at the end of August.

Comparative epidemiology

There are many possible approaches to comparative epidemiology, but the ecological one adopted in this paper is particularly rewarding because of its general applicability. Categorizing plant virus diseases in terms of the continuum of r and K provides new insights into their behaviour and control. Moreover, comparisons can be made with weeds and the whole array of pests, parasites and diseases of plants and animals.

Conway (1976) and Southwood (1977b) have already considered the population dynamics of a wide range of animal pests and related these to possible control measures. Such diverse organisms as elephants, bullfinches, tsetse flies, codling moth (*Cydia pomonella*) and certain protozoan parasites are regarded as 'K strategists', vulnerable to drastic changes in their specialized habitats. Extreme 'r pests' include house flies, fruit flies, locusts, the armyworm (*Spodoptera exempta*) and many aphids. These are seldom amenable to control by natural enemies and the widespread use of pesticides is often followed by the emergence of resistant strains.

Comparable assessments are possible of plant parasitic nematodes. For example, the free-living soil-inhabiting virus vector *Xiphinema diversicaudatum* is long-lived and behaves as a K strategist. In southern England it produces only one generation a year and maintains stable populations that are slow to recover after fumigation. They also respond slowly to a change of crop or even to bare fallow regimes (Flegg, 1968;

McNamara & Pitcher, 1977). By comparison, certain endoparasitic species referred to as 'exploiters' (Jones, this volume) are obvious *r* strategists, increasing rapidly in the aerial parts of plants during the limited period when host tissues are available and climatic conditions are suitable. Many other nematodes, including certain *Trichodorus* spp., display intermediate characteristics producing several generations a year and variable populations.

Weeds, plant pathogenic fungi and diseases of animals have yet to be assessed in detail and they have been considered only briefly in reviews devoted primarily to other topics (Bradley, 1977; Southwood, 1977b; Newman, 1978; Thresh, 1978). However, many of the rusts, mildews and other fungi attacking the leaves of annual crops behave as *r* strategists. Their mobility and extremely rapid rates of multiplication enable them to spread quickly from a few initial foci of infection. They are largely free from intra- and inter-specific competition and are well adapted to exploit rapidly-changing habitats of limited duration. Powdery mildews attacking the leaves of apple and other deciduous perennials exhibit similar features, whereas fungi colonizing the roots, trunk or main branches behave differently. They spread relatively slowly within and between trees and there is a long interval between infection and the production of inoculum. Another striking similarity with *K* strategists is that they encounter considerable competition from other species. Indeed, antagonists have been used successfully to control diseases such as silver leaf of plum (Corke, 1978) and those caused by certain soil-inhabiting fungi with similar *K* characteristics (Newman, 1978).

Diseases of man and livestock can also be considered in terms of the continuum of *r* and *K* (Bradley, 1977; Southwood, 1977b). For example, measles, influenza, the common cold, chicken pox, cholera, anthrax and foot-and-mouth cause spectacular epidemics after relatively quiescent periods. They have an opportunist *r* strategy of high transmission rates and rapid invasion of hosts that are transient either because they die or soon develop antibodies, become non-infectious and recover. Pathogens that are adapted for long-term survival in the host cause endemic diseases such as infectious hepatitis, tuberculosis, leprosy, schistosomiasis and sleeping sickness. These have the *K* characteristics of stability and low transmission rate that are associated with the exploitation of long-lived habitats.

These examples illustrate how diverse diseases of widely different hosts can be assessed in the same basic way to stress the underlying similarities between them. Moreover, wider analogies are possible with non-parasitic species in developing a general relationship between behaviour, mobility and habitat stability. The success of this approach confirms the value of considering pathosystems in the same way as other ecosystems. This has already been advocated on conceptual grounds (Robinson, 1976) and emphasizes the important integrating role of ecology in comparative epidemiology. There are obvious advantages in considering the population dynamics of host-parasite inter-relationships in the same terms as those relating to predators and their prey or to plants and herbivores.

References

Bennett, C. W., 1971. The curly top disease of sugar-beet and other plants. Monograph 7. American Phytopathological Society, 82 pp.

Bradley, D. J., 1977. Human pest and disease problems: contrasts between developing and

developed countries. In: J. M. Cherrett & G. R. Sagar (Ed.): Origins of pest, parasite, disease and weed problems. Blackwell Scientific Publications, Oxford, p. 329–345.
Bruehl, G. W., 1961. Barley yellow dwarf. Monograph 1. American Phytopathological Society, 52 pp.
Conway, G., 1976. Man versus pests. In: R. M. May (Ed.): Theoretical ecology. Blackwell Scientific Publications, Oxford, London, p. 257–281.
Corke, A. T. K., 1978. Microbial antagonisms affecting tree diseases. Ann. Appl. Biol. 89: 89–93.
Crosse, J. E., 1967. Plant pathogenic bacteria in the soil. In: T. R. G. Gray & D. Parkinson (Ed.): The ecology of soil bacteria. Liverpool University Press, p. 552–572.
Davidson, T. R. & J. A. George, 1964. Spread of necrotic ringspot and sour cherry yellows viruses in Niagara peninsula orchards. Can. J. Pl. Sci. 44: 471–484.
Demski, J. W., 1975. Source and spread of peanut mottle virus in soybean and peanut. Phytopathology 65: 917–920.
Dobzhansky, T., 1950. Evolution in the tropics. Am. Sci. 38: 209–221.
Esch, G. W., T. C. Hazen & J. M. Aho, 1977. Parasitism and r- and K-selection. In: G. W. Esch (Ed.): Regulation of parasite populations. Academic Press, New York, 253 pp.
Flegg, J. J. M., 1968. Life-cycle studies of some *Xiphinema* and *Longidorus* species in south-eastern England. Nematologica 14: 197–210.
Gadgil, M. & O. T. Solbrig, 1972. The concept of r- and K-selection: evidence from wild flowers and some theoretical considerations. Am. Nat. 106: 14–31.
Gregory, P. H., 1948. The multiple-infection transformation. Ann. Appl. Biol. 35: 412–417.
Gregory, P. H., 1974. The microbiology of the atmosphere. 2nd edn. Leonard Hill Books, London, 377 pp.
Gutierrez, A. P., H. A. Nix, D. E. Havenstein & P. A. Moore, 1974. The ecology of *Aphis craccivora* Koch and subterranean clover stunt virus in south-east Australia. III. A regional perspective of the phenology and migration of the cowpea aphid. J. Appl. Ecol. 11: 21–35.
Harper, J. L., 1977. Population biology of plants. Academic Press Ltd., London and New York, 920 pp.
Harrison, B. D., 1977. Ecology and control of viruses with soil-inhabiting vectors. Ann. Rev. Phytopathol. 15: 331–360.
Hull, R., 1964. Spread of groundnut rosette virus by *Aphis craccivora* Koch. Nature, London 202: 213.
Janzen, D. H., 1968. Host plants as islands in evolutionary and contemporary time. Am. Nat. 102: 592–595.
Johnson, C. G., 1969. Migration and dispersal of insects by flight. Methuen, London & New York, 763 pp.
Loya, Y., 1976. The Red Sea coral *Stylophora pistillata* is an r strategist. Nature, London 259: 478–480.
MacArthur, R. H. & E. O. Wilson, 1967. The theory of island biogeography. Princeton University Press, Princeton, N.J., 203 pp.
McNamara, D. G. & R. S. Pitcher, 1977. The long-term effects of four monocultural regimes on two field populations of the nematodes *Xiphinema diversicaudatum* and *Longidorus* spp. Ann. Appl. Biol. 86: 405–413.
Moore, R. J., 1978. Is *Acanthaster planci* an r-strategist? Nature, London 271: 56–57.
Newman, E. I., 1978. Root micro-organisms: their significance in the ecosystem. Biol. Rev. 53: 511–544.
Pianka, E. R., 1970. On r- and K-selection. Am. Nat. 104: 592–597.
Robinson, R. A., 1976. Plant pathosystems. Advanced series in agricultural science. 3. Springer-Verlag, Berlin, Heidelberg, New York, 184 pp.
Romney, V. E., 1939. Breeding areas and economic distribution of the beet leafhopper in New

Mexico, Southern Colorado and Western Texas. Circular 518, United States Department of Agriculture, Washington D.C., 14 pp.
Rose, D. J. W., 1974. The epidemiology of maize streak disease in relation to population densities of *Cicadulina* spp. Ann. Appl. Biol. 76: 199–207.
Sellers, R. F. & A. J. Forman, 1973. The Hampshire epidemic of foot-and-mouth disease. 1967. J. Hyg. 71: 15–34.
Sellers, R. F., D. E. Pedgley & M. R. Tucker, 1977. Possible spread of African horse sickness on the wind. J. Hyg. 79: 279–298.
Selman, I. W., 1943. The appearance and spread of mosaic infection in the tomato crop and the relation to seed transmission of the virus. Ann. Appl. Biol. 30: 331–338.
Simons, J. N., 1957. Effects of insecticides and physical barriers on field spread of pepper vein banding mosaic virus. Phytopathology 47: 139–145.
Slack, S. A., R. J. Shepherd & D. H. Hall, 1975. Spread of seed-borne barley stripe mosaic virus and effects of the virus on barley in California. Phytopathology 65: 1218–1223.
Sonenshine, D. E., 1974. Vector population dynamics in relation to tick-borne arboviruses: a review. Phytopathology 64: 1060–1071.
Southwood, T. R. E., 1962. Migration of terrestrial arthropods in relation to habitat. Biol. Rev. 37: 171–214.
Southwood, T. R. E., 1977a. Habitat, the templet for ecological strategies? J. Anim. Ecol. 46: 337–365.
Southwood, T. R. E., 1977b. The relevance of population dynamic theory to pest status. In: J. M. Cherrett and G. R. Sagar (Ed.): Origins of pest, parasite, disease and weed problems. Blackwell Scientific Publications, Oxford, p. 35–54.
Southwood, T. R. E., R. M. May, M. P. Hassell & G. R. Conway, 1974. Ecological strategies and population parameters. Am. Nat. 108: 791–804.
Thresh, J. M., 1958. The spread of virus disease in cacao. Technical Bulletin No. 5. West African Cocoa Research Institute, 36 pp.
Thresh, J. M., 1966. Field experiments on the spread of black currant reversion virus and its gall mite vector (*Phytoptus ribis* Nal.). Ann. Appl. Biol. 58: 219–230.
Thresh, J. M., 1974a. Vector relationships and the development of epidemics: the epidemiology of plant viruses. Phytopathology 64: 1050–1056.
Thresh, J. M., 1974b. Temporal patterns of virus spread. Ann. Rev. Phytopathol. 12: 111–128.
Thresh, J. M., 1976. Gradients of plant virus diseases. Ann. Appl. Biol. 82: 381–406.
Thresh, J. M., 1978. The epidemiology of plant virus diseases. In: P. R. Scott and A. Bainbridge (Ed.): Plant disease epidemiology. Blackwell Scientific Publications, Oxford, p. 79–91.
Vanderplank, J. E., 1948. The relation between the size of fields and the spread of plant-disease into them. Part I. Crowd diseases. Empire J. Exp. Agric. 16: 134–142.
Vanderplank, J. E., 1949. Vulnerability and resistance to the harmful plant viruses: a study of why the viruses are where they are. S. Afr. J. Sci. 46: 58–66.
Vanderplank, J. E., 1959. Some epidemiological consequences of systemic infection. In: C. S. Holton, G. S. Fischer, R. W. Fulton, H. Hart and S. E. A. McCallan (Ed.): Plant pathology: Problems and progress 1908–1958. The University of Wisconsin Press, p. 566–573.
Wyatt, T., 1974. Red tides and algal strategies. In: M. B. Usher and M. H. Williamson (Ed.): Ecological stability. Chapman & Hall Ltd., London, p. 35–40.

Some aspects of the epidemiology of plant parasitic nematodes

F. W. G. Jones

Abstract

Unlike fungi, all stages of nematodes are distinct and highly organized individuals. Usually all stages except those in the egg are self-propelled to distinct feeding sites where ingestion is via the needle-like mouth stylet. Some species are 'exploiters' (*r*-strategists) others are 'persisters' (*K*-strategists). Relationships with hosts are broadly similar to those of other plant pathogens and a gene-for-gene relationship is known in some species of cyst-nematode. Differences arise from the more advanced sensory equipment and behavioural patterns of nematodes and their more precise mode of feeding.

Once established, some nematodes disperse themselves below ground but for most, and especially for those with sedentary females, self-dispersal is extremely slow. All species, 'exploiters' in particular, require effective means of dispersal. The extraneous agencies that effect dispersal above ground are basically the same as those dispersing other plant pests and pathogens. However, nematodes lack the rapid means of long-range aerial dispersal of many spore-bearing organisms, aphids and certain other insects and mites. The cysts of cyst-nematodes resemble the resting spores of fungi and greatly aid dispersal.

The self-dispersal of *Radopholus similis* proceeds at the rate of 1 to 1.6 tree spaces/year. That of the virus-vector species *Xiphinema diversicaudatum* is about 0.3 m/year and of cyst-nematodes (*Heterodera*, *Globodera* spp.) in which only the second-stage juvenile migrates, is about 0.1 m/generation. Spread of the stem nematode (*Ditylenchus dipsaci*) on seeds or in plant debris, that of cysts and of many species within planting material or in soil attached to it, is far more rapid.

The spread of detectable infestations, whether measured as the number of fields or groves, or in hectares, appears to be exponential initially. That of infestations too small to detect by field sampling is even more rapid. Nevertheless the progress of nematode epidemics is slow, akin to that of soil-borne pathogens. Unlike epidemics of leaf-pathogens, the time scale is in decades rather than years or months. A modified exponential growth curve is probably common to all.

The principles governing the behaviour of cyst-nematode populations in established foci have been much studied and simulated by computer models. Some details of these are given. When host crops are grown continuously, population oscillations are determined by the amount of damage done to host roots and damped by that part of the population remaining as unhatched eggs within cysts. The annual rate increase (which is limited) has little effect. The persistent element in the population greatly slows the rate of selection of pathotypes able to break host resistance based on major genes. However, a sibling species able to break resistance is more rapidly selected and its take-over is delayed only by competition with the incumbent species, numbers of which decline fairly rapidly under the influence of egg-hatching factors produced by the roots of the resistant host.

Introduction

Nematodes have rarely been considered from an epidemiological standpoint and satisfactory data over an adequate time span are scarce. Being animals, they differ from plant pathogenic organisms in important respects e.g. organization, dispersal methods,

population strategy and feeding tactics. Nevertheless, as will be apparent, there are features common to plant pathogens and animal pests. The epidemiology of nematodes most closely resembles that of soil-borne pathogens in the relative slowness of dispersal and the length of time needed for infestations to reach their end-point.

Differences will first be discussed followed by dispersal and population dynamics, both essential ingredients in epidemics. No attempt will be made to review the literature. Examples are chosen to illustrate self-dispersal by the characteristic serpentine movement of nematodes (the burrowing nematode causing spreading decline of citrus in Florida, *Radopholus similis* (Cobb)), dispersal in plant material (the stem nematode, *Ditylenchus dipsaci* (Kühn)), and dispersal by agricultural operations (cyst-nematodes, *Heterodera* and *Globodera* spp.). Little mention will be made of virus-vector nematodes as these are considered by Thresh (this volume).

Some characteristics of nematodes and nematode populations

Nematodes differ in several respects from other lower organisms that attack plants (Table 1). All stages are distinct individuals possessing highly organized systems and usually all except those in the egg are self-propelled to discrete feeding sites where cell contents are extracted via needle-like mouth stylets. Dispersal of nematodes occurs in various ways (Table 2) which basically are no different from those of most other noxious

Table 1. Plant nematodes: some differences from viroids, viruses, bacteria and fungi.

All stages are distinct individuals	Digestive system
The effective unit is of larger size	Excretory system
Highly organised limiting membrane (cuticle)	Nervous system
Feeding via mouth only	Advanced sense organs
Locomotory system	Complex behaviour patterns

Table 2. Dispersal of nematodes.

Means		Distances	Speed
Self-dispersal	– juveniles and adults of most species	immediate	slow
	– 2nd–stage juveniles of species with sedentary females	vicinity only	very slow
Natural agencies	– rain splash	local	slow
	– water currents		
	– wind	mostly local	sometimes rapid
	– seeds	medium	mostly slow
	– animals	and long	
Human agencies	– agricultural operations	local	rapid
	– commerce related and unrelated to agriculture	medium and long	rapid
	– tourism	long	fortuitous

organisms. However, nematodes lack the rapid means of long-range aerial dispersal possessed by spore-forming organisms, by aphids and by certain other insects and mites.

The population strategies of plant nematodes vary. Broadly they fall into two groups (Table 3): 'exploiters' (*r*-strategists) which are able to multiply rapidly on suitable hosts within a limited time, and 'persisters' (*K*-strategists) which multiply less rapidly but maintain more stable population densities. To capitalize opportunities as they arise, whether in nature or in agriculture, 'exploiters' need efficient methods of dispersal and this is achieved by such methods as attachment to seeds (*D. dipsaci*), transference in plant debris (*Aphelenchoides ritzemabosi* Schwartz), in definitive galls (*Anguina* spp.) or carriage by insects (*Rhadinaphelenchus cocophilus* (Cobb)). All depend on a marked ability to endure desiccation. Dispersal of 'persisters' (species that 'wait' for their hosts to reappear) is also essential if less pressing and some of these have ability to withstand desiccation and well-adapted dispersal arrangements (e.g. the cysts of cyst-nematodes which resemble the resting spores of fungi).

Table 3. Examples of population strategies of nematodes.

Exploiters – unstable populations	
Stem nematode races	*Ditylenchus dipsaci*
Bud and leaf nematodes	*Aphelenchoides* spp.
Flower-gall nematodes	*Anguina* spp.
Red-ring nematode	*Rhadinaphelenchus cocophilus*
Persisters – relatively stable populations	
Cyst-nematodes	*Heterodera, Globodera* spp.
Needle and dagger nematodes	*Longidorus, Xiphinema* spp.
Spiral nematodes	*Helicotylenchus, Rotylenchus* spp.
Root-lesion nematodes	*Pratylenchus* spp.
Pin nematodes	*Paratylenchus* spp.
Stubby-root nematodes	*Trichodorus, Paratrichodorus* spp.

In the long-distance dispersal of small, imperfectly water-proofed organisms the implications of desiccation endurance are not always appreciated. Within the soil, where dispersal is constrained by the pore system and cannot be other than local, the relative humidity of the soil atmosphere is usually greater than 99 percent which is the value for apparently dry soil in which plants wilt permanently (pF 4.15, Schofield & DaCosta, 1935). Rapid and distant spread can only occur above ground in an essentially aerial environment where the relative humidity often falls below 70 percent (pF 5.7) which corresponds to a water extracting force of 500 atmospheres. Here the rate of water loss is also speeded by air movements and insolation. Although not all stages or all species withstand desiccation it is remarkable that organisms so highly organized have adapted to endure so great a degree of drying.

Apart from population strategy, nematodes have evolved various tactics which play a role in attacks on crops. If climate does not synchronize the life-cycle of host and parasite, mechanisms must be developed to ensure synchrony e.g. the various forms of diapause. As animals, with ability to move and complicated internal systems, they use energy at a rate faster than non-animal pathogens. Since nematodes cannot utilize fibre,

lignin or cellulose and vacuoles in parenchyma contain too much water, the tendency is to feed on young tissue, growing points, developing seeds, or fruits and storage organs as do insect pests (Southwood, 1973). Mobile stages can move to feed elsewhere when incompatible components in saliva destroy the feeding site but sedentary females must produce saliva compatible with the contents of cells on which they feed. Usually such nematodes induce transfer-cell systems which ensure nutrient flow to the feeding site (Jones & Northcote, 1972).

Besides providing food, the host must also supply sensory triggers that enable the nematode to find its host, to find its feeding sites and to initiate feeding and sustain ingestion. These triggers probably include secondary plant substances peculiar to a particular host or group of hosts which are susceptible to attack. Although a single gene change might conceivably cut the sensory chain and so prevent host-finding or feeding, changes in host status are more probably dependent on polygenes which affect the palatibility and quality of food supplied. In those species with sedentary females that induce long-lasting transfer cells, a single major gene change may lead to products incompatible with the nematode's saliva and so block development and egg production. A similar relationship may exist in other obligate endoparasites that induce specific host reactions (e.g. the races of *D. dipsaci*).

The relationships of nematodes with their hosts are therefore broadly similar to those of viral, bacterial and fungal pathogens and for cyst-nematodes a gene-for-gene relationship is known to occur (Jones, 1974). Differences arise mainly from the more advanced sensory equipment and behavioural patterns of the nematode and its more precise mode of feeding.

Self-dispersal

Movement of juvenile and adult nematodes occurs in soil and about plants only when water is available at small suctions up to pF 0.25 (Wallace, 1963; Jones, 1975) and temperatures lie between the lower and upper thresholds for activity, 5–10°C and 35–40°C respectively. Within-soil movement is also influenced by the amount of usable pore space. In most tilled soils oxygenation of the macropores is usually adequate except when the soil is flooded or over-irrigated. In temperate regions, moisture is usually adequate in winter but temperatures may be too low. In summer temperatures are no longer limiting but moisture may be inadequate. Spring and autumn are more nearly ideal. Similar influences undoubtedly govern the development of soil-inhabiting bacteria and fungi, especially of fungi that produce motile zoospores (Griffin, 1970).

The two dimensional rate of spread of any organism colonizing territory by unlimited outward spread at a steady rate is given by:

$$\pi(pt_1)^2,\ \pi(pt_2)^2,\ \pi(pt_3)^2\ \ldots\ldots\ldots\pi(pt_n)^2$$

where t is the time in generations (or years) and p the distance colonized in one generation (or year).

The amount of self-dispersal possible within an area of land during one season is probably characteristic of species, host and habitat. Species with several generations are doubtless able to move further than those with only one (*Heterodera schachtii* Schm. in N. Europe, 2½ generations/year; Imperial Valley California, 5 generations/year). Self-dispersal of endoparasitic species with sedentary females (*Heterodera*, *Globodera*,

Meloidogyne, *Rotylenchulus*, *Tylenchulus*) is by juveniles which move a distance of the order of 10 cm from the hatched egg to nearby roots. The domain of such species is limited to a column of soil with radius 10 cm and height 25 cm (plough depth less 5 cm) per generation and has a superficial area of 0.03 m^2 and a volume of 0.008 m^3. Vermiform endo- and ectoparasitic nematodes with several generations a year are able to occupy far larger domains within a single year. In the citrus groves of Florida, USA, *R. similis* extends its territory apparently unaided by about 1.6 tree spaces a year in all directions in the highly favourable deep coral sands characteristic of Florida citrus groves (Suit & DuCharme, 1957). Rainfall is frequent and soil temperatures high, so movement is rarely limited. Spread above ground and by implements is virtually absent because cultivations are few and the nematode is scarce in the top 15 cm of the soil. The nematodes occupy the soil below this to a depth of about 2 m. Hence, if one tree space has a radius of 5 m, the superficial area of the domain occupied in one year is $\pi 1.6^2 \times 5^2$ or 201 m^2 and the domain volume 402 m^3 which is about 50 000 times the domain occupied by a species of *Heterodera* or *Globodera* with one generation a year.

From counts of infested trees (Suite & DuCharme, 1957) in six plantations in 1945 and again in 1950, six years later, the achieved rate of spread of *R. similis* appears to be about one tree space a year (Fig. 1) which is less than the expected 1.6 tree spaces a year possibly because some trees are missing and because the edge of the advancing infestation encounters roadways, plantation boundaries or those of other extending foci. Evidently the local spread of this species is almost wholly due to self-dispersal.

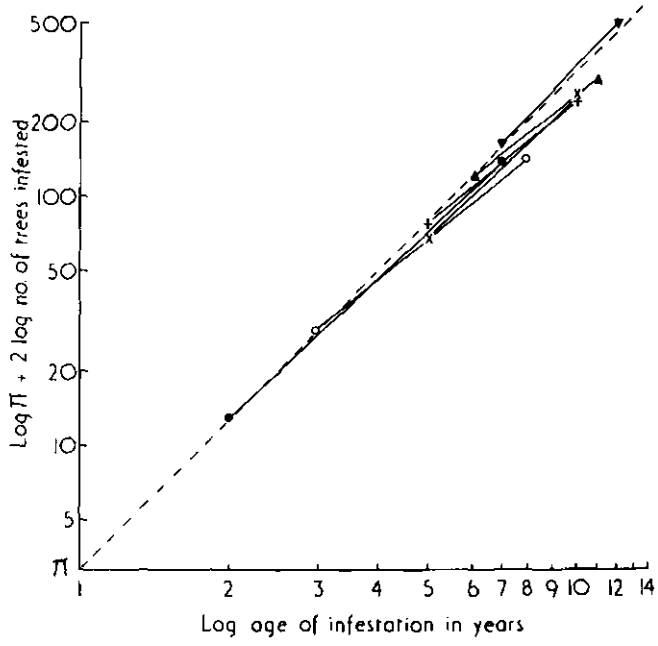

Fig. 1. Extension of spreading-decline in Florida citrus groves. – – – – expected if extension is by one tree space/year ●, ○, +, ×, ▲, ▼, observed rate in six groves. Data from Suite & DuCharme (1957).

If the notion that the domain of a cyst-nematode has radius 10 cm is correct, then for a species with one generation a year and starting from a single focus, self-dispersal would take > 5 years to colonize 1 m^2, >500 years to colonize 1 ha and >1500 to colonize 10 ha. Even if the domain radius were ten times greater, i.e. 1 m, colonization of 1 ha would take >50 years and of 10 ha >150 years. The observed spread of cyst-nematodes within fields is far faster than this, hence in agricultural situations local spread from agricultural operations must play a dominant role, a situation representing the opposite extreme from the self-dispersal of *R. similis*.

The migratory root ectoparasitic dagger nematode, *Xiphinema diversicaudatum* (Micol.), extends into uninfested land a distance of approximately 0.3 m a year (Harrison & Winslow, 1961), hence the superficial area of its domain is $\pi 0.3^2$ or 0.29 m^2 and its volume, assuming occurrence from 5 to 30 cm deep is 0.07 m^3, ten times the domain volume of a cyst-nematode. Very active root ectoparasites such as *Trichodorus viruliferus* Hooper, which aggregate strongly around the rapidly extending cord roots of apple, have a domain related to the root zone of their host as does *R. similis*, an active migratory endoparasite.

Dispersal by natural means

Origins of dispersal and its consequences

Nematodes, in common with other pest and disease organisms, have co-evolved with their hosts in natural situations. Their association with field and plantation crops is very recent and, although new adaptations to local climatic and other conditions and new races able to attack particular cultivars may have arisen as a result, most nematode populations seem to have changed little. Their basic characteristics are ancient and races able to multiply on resistant cultivars probably antedate plant breeding. This is certainly true of the pathotypes of *Heterodera avenae*, *Globodera rostochiensis* (Woll.) and *G. pallida* (Stone), and also probably of the races of *D. dipsaci*.

In nature, hosts were sparsely and discontinuously distributed. This must have greatly favoured the evolution of efficient additional dispersal mechanisms (e.g. attachment to seeds, to insects and other mobile animals, in seed galls, in cysts, etc.) or ability to persist (e.g. development of diapause arrangements, response to hatching factors, endurance of desiccation etc.) especially in those nematodes that became increasingly host specific.

Dispersal by wind and flood

Wind and flood are potent means of local dispersal. The nematode fauna of deserts and coastal sand dunes is doubtless effectively spread in wind-blown sand, although no estimates of the rate of spread are available. Wind erosion of the friable surface of cultivated agricultural land in areas of peat or light sand soils is also commonplace. Apart from local floods, river systems and irrigation systems may transport great numbers of nematodes locally or distantly (Faulkner & Bolander, 1970). Whether transport by wind and water is natural or artificially assisted, the usual effect is to disseminate widely populations too small to be detected and whether these survive and multiply in their new surroundings depends on the presence and frequency of hosts and on the abundance of enemies and competitors.

Thompson, Roebuck & Cooper (1949) sampled silt deposited on grass fields and flotsam, straw and other debris over an extensive area subject to flooding in 1947, following the thaw after an exceptional winter. Few cysts were found in silt but many were found attached to flotsam etc. They concluded rightly that there was no widespread heavy infestation of land previously 'free'. What they failed to make clear is the limitations of their sampling procedures (Jones, 1969). There can be little doubt that a great many trivial infestations of all species of cyst- and other nematodes were widely disseminated. Results of cyst counts from the routine surveys of many fields for potato cyst-nematodes revealed that in the basins of four rivers 62 to 80 percent of the fields were infested before the floods occurred. Allowing for the difficulties of detecting small infestations, it can be assumed that almost all potato-growing land in these areas was already infested and that the floods helped to complete its colonization.

Dispersal within fields by agricultural operations

Infested seed

Nematodes are spread by all agricultural operations. When seed-borne, foci are established randomly over the whole of the seeded area, their number depending on the frequency of infested seeds and the viability of the nematodes. Usually foci are small in the first year, extend in subsequent host crops and involve most of the planted area by the third or fourth year provided the crop interval is not unduly long. This is the pattern of infestation by races of stem nematode in onions, lucerne and field beans (*Vicia faba* L.).

Two races infest field bean, the oat race and the giant bean race (Hooper, 1975). The previously uninfested classical experiments at Rothamsted (Barnfield, roots etc. 1953–1966; Broadbalk, wheat or fallow 1844–1966; Hoosfield, barley 1852–1966) became infested in 1967 when field beans were first planted. In the first year, individual plants, small groups of a few stems or short lengths of row were attacked. Part of Barnfield grew field beans continuously from 1967 to 1976. In the second and subsequent 9 years more than 80 percent of plants were infested and yields suffered. Evidently the seed sown in 1967 was heavily contaminated. On Broadbalk and Hoosfield where beans were grown on a 3-year cycle, the first crop in each cycle had a few scattered infestations. In the second, infestations were more frequent and the patches larger. In the third and fourth year the crop was generally and heavily infested but not as heavily as in Barnfield where beans were grown continuously. In other fields, rotations longer than every 3 years usually maintained light infestations but shorter rotations resulted in heavy ones and yield loss. Both nematode races persisted when immune crops were grown and some survived even when land was fallow for 7 years.

Infested planting material

Dispersal and establishment of infestations within fields in soil adhering to seed-tubers, sets, root-stocks or other transplanted material, or of endoparasitic species within plant tissue, is similar to seed transmission. Staniland (1946) emphasized the importance of transplants with adhering soil in spreading potato cyst-nematodes. Individual broccoli transplants carried as many as 66 cysts. However, in citing broccoli fields

which had never grown potatoes being infested with 7 to 10 cysts of the potato cyst nematode in 20 g soil there was evidently some confusion with cysts of *H. cruciferae* (Franklin, 1959). As there are 2.5×10^9g of topsoil per hectare, this rate of infestation approximates to 10^9 cyst per hectare. Thus, broccoli transplanted at 25 000 plants per hectare would each have had to carry 0.4×10^6 cysts, which is absurd. Staniland was correct in emphasizing the importance of transplants as means of spreading nematodes but had overlooked the fact that some multiplication is necessary before populations reach a density readily detectable by soil sampling, and more before plants are visibly stunted. This conclusion appears generally true but the time scale varies with the amount of inoculum transferred and the reproductive potential of the nematode.

Spread by implements

The rate of spread of nematodes is greatly accelerated by agricultural operations. Figure 2 illustrates the hypothetical spread of potato cyst-nematodes as a result of harvesting by spinner in Scottish fields. The spinner moves up and down the rows to be harvested, throwing ridge soil sideways about 1 m and enlarging foci by approximately 2 m in every potato crop harvested, a process far outweighing other means of spread. The spinner is now rarely used; modern harvesting machinery also displaces much soil but mainly along the row.

Figure 2 also illustrates other characteristics of nematode infestations developing in new territory: (i) the establishment of a few well-defined primary foci with steep gradients of population density at their edges and (ii) a larger number of smaller and

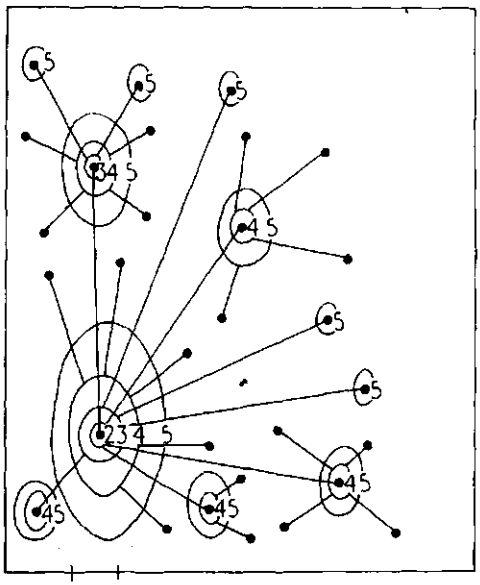

Fig. 2. Notional scheme for the spread of potato cyst-nematode in Scottish potato fields. Enlargement of successive foci by potato spinner during harvesting. From data in Bedi (1968).

more diffuse foci derived from them. The few larger foci resemble those of air-borne fungal pathogens spreading within a crop and the many smaller foci that of spread above the crop. Hence, nematode spread within soil is analogous to fungal spread within a crop, and nematode dispersal above the soil to that of air-borne spores above the crop and the models derived for fungal spread might conceivably be applied to nematode spread within fields but with a greatly attenuated time scale.

Most agricultural operations are in the direction of the longest axis of the field so most spread, whether by implements or by furrow irrigation, is along that axis. However, some operations are across the rows. The effects of spread along both axes result in the so-called kite-shaped patches visible in aerial photographs e.g. of Californian beet fields where the beet cyst-nematode is in the process of establishing itself. Harvesting of root crops (e.g. sugar beet, potatoes, carrots and other vegetables) which distributes much soil probably contributes most to the rapid spread of nematodes within fields and not only those nematodes of which these crops are immediate hosts. Soil planing for irrigation is also a potent means of spreading nematodes within fields as is the return to fields of waste soil accumulated at beet loading sites and factories.

Information on the quantitative aspects of within-field spread is hard to find but some is available from the ley-arable experiment at Woburn Experimental Farm which started in 1968 to test the effects on soil fertility of arable and ley rotations. (Rothamsted Experimental Station, 1970; Evans, 1979). Interest centres on Block 5 planted wholly with potatoes in 1935. By 1955 plots had been planted with potatoes, four, six and eight times in 18 years, some being in the arable rotation and some being test crops between rotations. Until 1950 and 1953, the mean yields of potatoes from these plots followed the seasonal trend (Fig. 3A). In 1955, plots that had grown potatoes eight times were a total failure from attacks of *Globodera rostochiensis* Ro1. Plots that had grown potatoes seven times under 'ley-arable-ley' (LAL) were also severely infested and yielded less than half the expected crop. In 1953, after six crops, these plots yielded normally as did those in the 'arable-ley-arable' (ALA) and in the 'continuous ley' (LLL) which had grown six and four potato crops respectively. Post-harvest numbers of *G. rostochiensis* were in line with yields.

Figure 3B shows the distribution of *G. rostochiensis* in Block 5. Cysts, which indicate the history rather than the current intensity of infestations, were most numerous in the 'continuous arable' plots (AAA) and the plots with largest numbers, 151 and 115, are probably those where the first foci developed. Egg numbers were also greatest on the AAA plots presumably because they carried most potato crops. The distribution pattern reflects the frequency of cropping with the host and spread along the plots by harvesting and cultivations; few operations were made across the plots. The infestation appears to have taken almost 18 years to reach the ALA plots in the top left hand corner which grew six potato crops during that period. The LAL plots, which also grew six crops, became more heavily infested because they were both in the same row as AAA plots.

Other evidence is available confirming that it takes five to six host crops or more to establish heavy infestations of potato or beet cyst-nematodes. For example, a field at East Harling, Norfolk, (Petherbridge & Jones, 1944) was planted with beet continuously for 6 years. In the first five years crops were excellent but in the sixth large beet-sick patches appeared. Similarly at Brooms Barn Experimental Station, plots that grew beet continuously were found to be heavily infested in the thirteenth year although

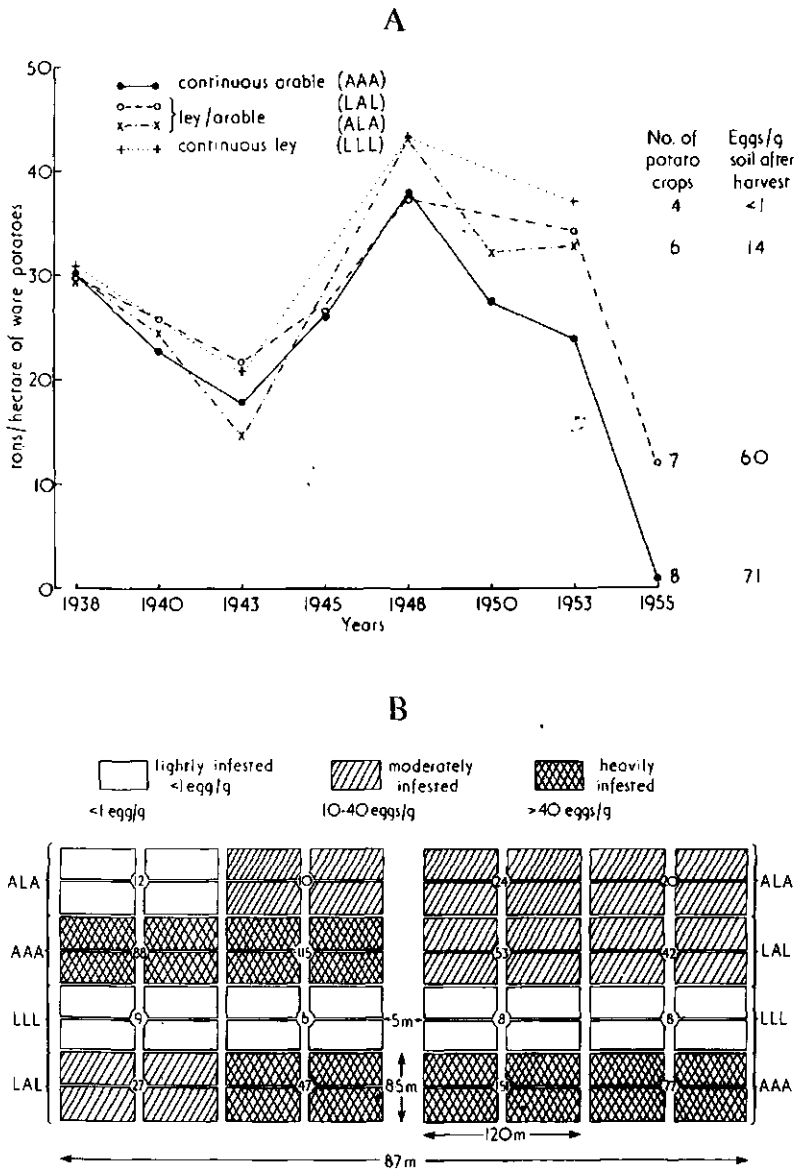

Fig. 3. A. Yields of potatoes on block 5 of the Woburn ley-arable experiment 1938–1955. B. Spread of potato cyst-nematode during the same period. Numbers in centre of quarter plots are of cyst/100 g. soil. Shading indicates degree of infestation, eggs/g soil, in 1955–1956. Data from Rothamsted records.

they had been examined periodically in previous years (Cooke, 1978). At Woburn Experimental Farm, cysts of *G. rostochiensis* Ro1 were applied to alternate plants in alternate rows to establish sites for experiments with nematicides, but it required four potato crops before the soil was sufficiently infested.

In observing the development of cyst-nematode infestations within fields, the effects of spread by farming operations and of multiplication are confounded and cannot be easily unravelled. Dissemination probably follows an exponential rule whereas multiplication is greatest at small population densities. The two processes together result in insidious spread followed by rapid colonization and the sudden appearance of stunted patches in the crop. The patches may appear in as few as 5 or 6 years when a host crop is grown continuously or as many as 20 to 30 years or more when the host crop is grown in rotation.

Dispersal between fields

The field perimeter, especially if marked by a hedge, ditch, fence or some other semi-permanent boundary, provides the first obstacle to dispersal. Similarly the boundary of the farm is another, but both are inadequate to impose more than a temporary delay on the movement of nematode populations once foci have become established.

The seed-borne infestations of stem nematodes on Rothamsted and Woburn Farms led to the colonization of some 150 ha of bean growing land within 5 years, and most of the spread occurred in one disastrous year (1967) when batches of heavily infested seed were sown inadvertently. More useful data are available for the burrowing nematode, the beet cyst-nematode and the potato cyst-nematode.

Suit & DuCharme (1957) give data for rate of spread of burrowing nematode in Florida which suggest the exponential coefficient is approximately 0.24 (Fig. 4A). The beet cyst-nematode was first found in the British peat fenlands in 1934 (Petherbridge & Jones, 1944; Jones, 1951) and subsequently spread throughout much of the area (Fig. 5). By 1936 a survey was instituted which gave the numbers of heavily infested fields found, the number of infested fields detected by root examination and the total area of known infestations between 1936 and 1943 (Fig. 4B). When plotted on a log scale, increase in the numbers of heavily infested and of detectable fields were linear i.e. exponential, with the latter increasing more rapidly. It can be assumed that many more fields contained infestations below the detection level and that the numbers of these were increasing even more rapidly. This underlines the insidious nature of the early stages of cyst-nematode infestations (and also that of most other soil-borne nematode infestations). Chitwood (1951) gives similar information from a well-conducted survey of potato cyst-nematode infestations in Long Island, New York, from 1942 to 1949. Although, for a different crop, a different nematode species and in a different place (potatoes, *G. rostochiensis*, 1 generation per year; sugar beet, *H. schachtii*, 2½ generations per year), the exponential coefficients (b) for the rate of extension of the infested area are similar (Fig. 4C).

In the early stages of spread, the occupation of new territory appears to be exponential. However territory is not unlimited and the rate of infestation must fall off because nematodes begin to re-infest already infested ground at an increasing rate. Gregory (1948) deduced the relationship between percentage infestation (territory occupied) and the rate of infection (spread) of spores falling at random from a distant source.

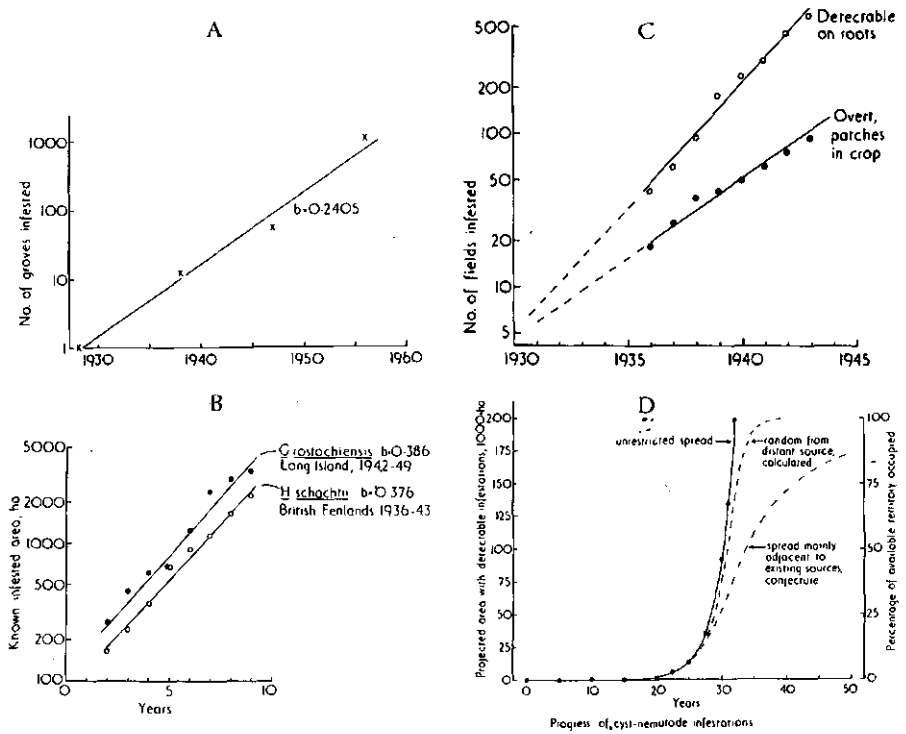

Fig. 4. A. Known increase in the number of Florida citrus groves infested with *R. similis*, 1930–1960. Data from Suite & DuCharme (1957).
B. Known increase in the number of fields infested with beet cyst-nematode in the British Fenlands, 1936–1945. Detectable on roots, b = 0.371: overt patches, b = 0.214. Data from Petherbridge & Jones (1944), Jones (1951).
C. Known increase in the area infested by potato cyst-nematode (Long Island) and the beet cyst-nematode (British Fenlands) over eight-year periods. Data from Chitwood (1951).
D. Colonization of a large territory (200 000 ha). Unrestricted spread calculated using b = 0.38 (mean of beet and potato cyst-nematode coefficients). The shape of the curves would be similar for the colonization of smaller areas. For explanation see text.

Using the equation he gives, it is possible to convert the expected exponential spread of nematodes into the percentage territory occupied (Fig. 4D). However, the spread of nematodes is largely by extension from local foci which is as yet impossible to characterize. In practice it would give a sigmoid curve of the general form shown which resembles that known for other animals e.g. the grey squirrel in the UK, the Colorado beetle in Europe (Jones & Jones, 1964). A model of the type shown in Fig. 4D can probably be applied to the spread of many organisms into new territory.

Spread within and between countries

Once foci are established within a country where conditions are suitable, spread is slow and insidious at first. By the time crops are stunted, the infestation is well

established and the possibility of eradication usually negligible. Spread accelerates and the later stages of colonization of all but the last remnants of occupiable territory are rapid. This picture fits a model similar to that for individual fields but with a longer time scale and is more akin to the establishment of soil-fungi than to outbreaks of leaf and stem pathogens above ground. Whereas many of the latter (including the aphid vectors of viruses) must recolonize their host plants anew each year and multiply rapidly if they are to exploit them before they are harvested, nematodes like many soil fungi are residues from previous host crops.

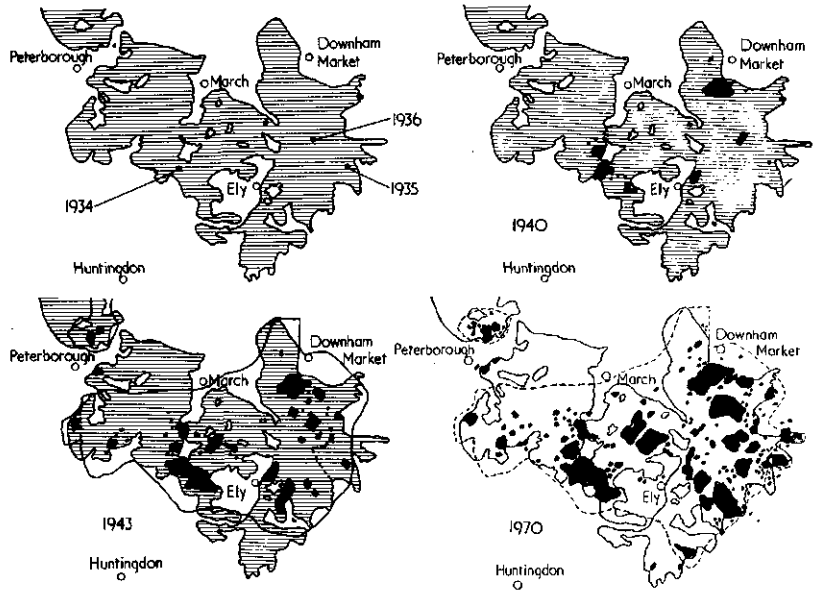

Fig. 5. Spread of the beet cyst-nematode in the British Fenlands 1934–1970. Area of peat fen soil shaded. Boundary of scheduled area shown as a continuous line (1943) and a broken line (1970). Data from Petherbridge & Jones (1944), Jones (1951) and other sources.

The end point in the dissemination of nematodes is a distribution coincident with those parts of the host territory suited climatically and edaphically to the species. In the UK, before the sugar beet crop became established around 1925, the beet cyst-nematode seems to have existed in isolated 'root fields' on farms and on sewage works where effluent was treated by land drainage. Here mangolds and brassicas were grown repeatedly to feed livestock (Jones, 1951). Over a period of some 50 years, almost the whole of the peat Fens, originally marshes, have become infested (Fig. 5). Elsewhere, on mineral soils, the number of known infestations has also increased and the distribution of the nematode resembles increasingly that of the sugar beet crop.

The colonization of potato-growing areas in the UK is also well documented. For example, potatoes were first grown on a field scale in the fen silt soils of south Lincolnshire in the 1880s and by the 1940s more than 80 percent of fields carried detectable infestations. Probably every tilled field was infested to some extent and many

so heavily that the planted area could not be maintained (Jones, 1970). Again the period required for colonization was of the order of 50 years. For some years now the distribution and intensity of potato cyst-nematodes in the UK has been virtually coincident with the distribution and intensity of the cultivation of potatoes for consumption. Seed potato-producing areas have been safeguarded and remain relatively free.

Much is known of the world distribution of the potato cyst-nematode complex (*G. rostochiensis*, *G. pallida* and their pathotypes) (Evans & Trudgill, 1978). Figure 6 depicts the distribution and presumed routes of spread of both species which seems to have begun about 1850 or possibly earlier and to have taken about 125 years.

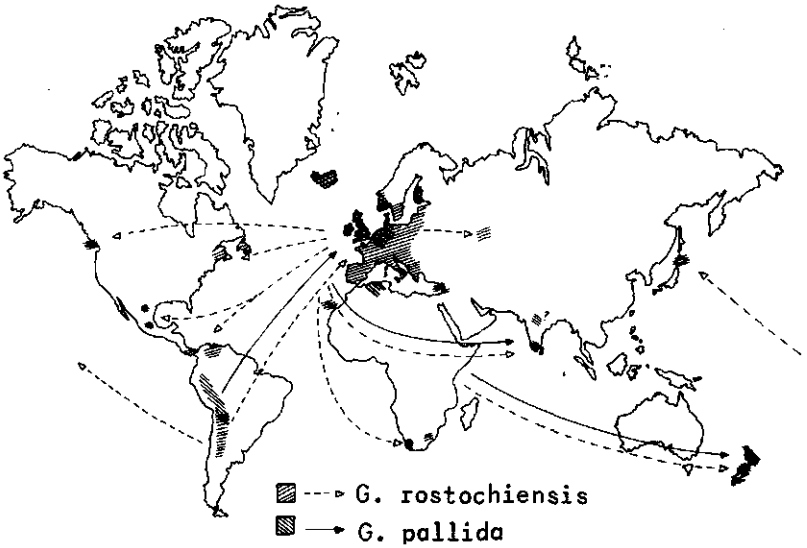

Fig. 6. Known world distribution of the potato cyst-nematodes and supposed routes of spread in commerce from the Andean epicentre (Evans & Trudgill, 1978).

In transportation to distant parts of the same country, or to other countries across the geographical barriers limiting natural spread, often only a part of the gene pool is exported. This is apparent in the spread of the two sibling species of potato cyst-nematodes and their pathotypes or sub-races. The United Kingdom, Scotland and East Anglia have been colonized predominantly by *G. rostochiensis* Ro1 and the basin of the river Humber and South Lincolnshire by what seems to be *G. pallida* Pa3. Elsewhere fields are colonized by one or other in different proportions. *G. pallida* Pa1 is sparsely scattered and relatively unimportant. This distribution disregards climatic dissimilarities and appears to be the result of early introductions and subsequent dispersal by seed tubers and other means. Pathotypes unknown or scarce in Britain occur in the Andes and in Continental Europe. Probably there are similar phenomena in other dispersed and numerous nematode populations which have not yet come to light.

Establishment and progress of foci

Events following the introduction of an organism into new territory are summarized in Figure 7. Should the numbers of individuals (or propagules) introduced be too few, the appearance of a host too long delayed, enemies too many or the environment too harsh, none may survive. If the organism is an obligate bisexual animal, too few may survive to ensure an adequate number of matings.

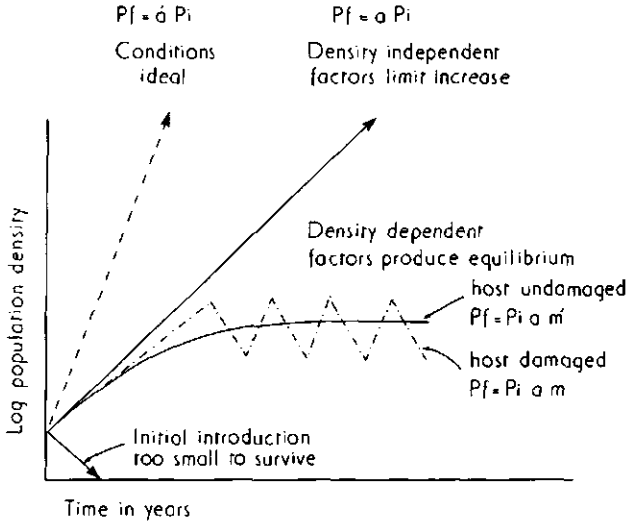

Fig. 7. Analysis of events following the introduction of an organism into new territory. Partly after Jones & Jones (1964).

If conditions were ideal, once established the organism would increase exponentially at the fastest possible rate. This may be represented by $P_t = a'P_i$ where P_i is the population density initially, P_t that at the end of one generation (or of unit time) and a' the ideal increase rate. Rarely are conditions ideal and a number of factors (Table 4) that are independent or dependent on the population density decrease the ideal rate. Density independent factors do no more than slow the rate from a' to a whereas density dependent factors slow the rate increasingly and tend to produce an equilibrium

Table 4. Factors regulating populations

Density-independent	Density-dependent
weather	quantity of food
quality of food	space
some non-specific enemies	enemies
pesticides	competitors

population density. A factor m', that modifies the rate of increase as P_i increases, can be derived from the logistic law which relates food or space available to the number of individuals competing for it (Jones & Kempton, 1978; Jones et al., 1978). However, pest or disease organisms damage their hosts and diminish the food supply. Hence m' is modified to m to account for this. Further modifying factors are required when enemies are numerous and effective (e.g. Perry, 1978). For nematode species with one principal generation a year and no effective enemies

$$m = 1/\{[1 + (a-1)P_i]/c(E/c)P_i/E\} \text{ (Jones \& Perry, 1978)}$$

where population densities are expressed as a proportion of E_1 the logistic equilibrium density, E is the observed equilibrium density and c is a constant >1 representing the tendency of the host to compensate for injury caused. For cyst-nematodes and other species that tend to persist we may write

$$P_t = P_i(1 - C)\,.a.m + CP_i$$

where C is the fraction of the population that persists. Because immigration and emigration are slight, they can be ignored: inbreeding is favoured.

To investigate the nature of population oscillations about the equilibrium, we differentiate the full equation:

$$\Delta = [d(P_t - P_i)/dP_i]\,P_i = E = (1 - C)\,(a - 1)/a\,[\log_e (E/c) - 1]$$

As the value of a is usually >20, (a $-$ 1)/a approaches 1 and so oscillations are governed by the two other terms on the right hand side of the equation. If $C = 0$, $(1 - C)$ becomes 1 and only the remaining term, which relates to host damage, influences population behaviour. May (1973) and others have shown that for values of Δ between 0 and -1, equilibrium was approached gradually, for values between -1 and -2 oscillations about the equilibrium were damped, and for values < -2 oscillations were large. By plotting the fraction of the population carried over (i.e. persisting) against the percentage damage to the plant, the boundaries of these three states can be defined (Jones & Kempton, 1978).

Sufficient is known about the population dynamics of certain cyst-nematode species to enable many aspects of their population dynamics to be simulated on a computer (Fig. 8).

Selection of races and sibling species

Information on the selection of populations by cultivars with genes for resistance is available only for potato cyst-nematodes. Here the gene-for-gene relationship seems to apply as in certain rust-fungi (Person, 1965) and in the Hessian fly (Gallun, 1974). The basis of the relationship is illustrated in Table 5 which hinges on the compatibility of nematode saliva injected into the feeding site with components in the host cells. In the two examples investigated (Parrott & Berry, 1974; Jones, 1974), the resistance gene in the host is dominant and the matching gene in the parasite is recessive : other relationships are possible and the two simple alternatives (matching gene dominant, matching gene recessive) are included in the computer simulation outlined in Figure 8. When run with suitable parameters, the computer model suggests that selection of a race or

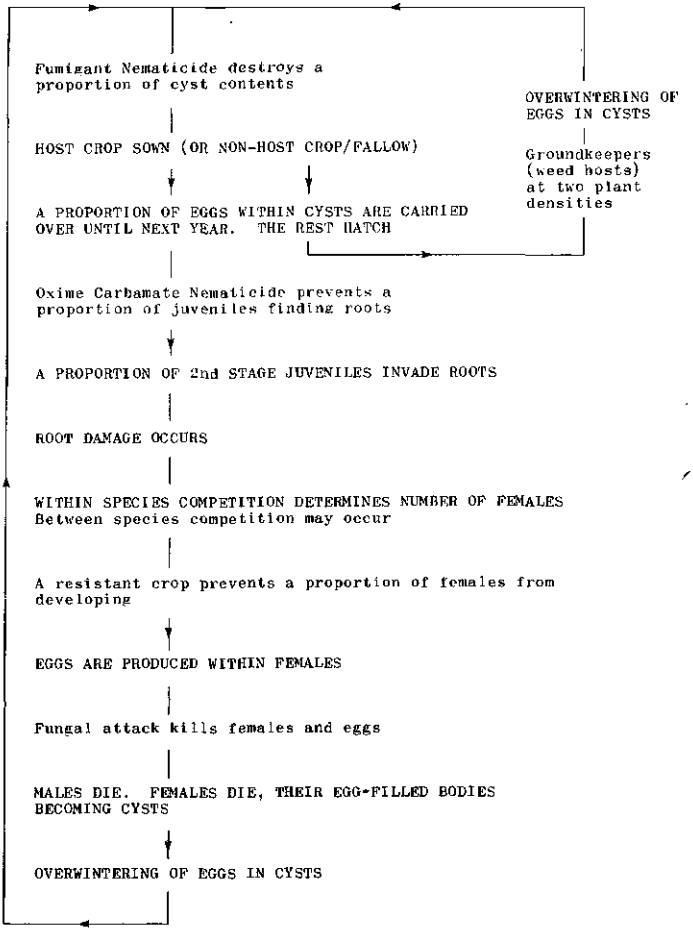

Fig. 8. Events in the life-cycle of cyst-nematodes modelled by Jones & Perry (1978). Capitals, included in the basic model; smalls, optional.

Table 5. Presumed basis of the gene-for-gene relationship.

Constitution of host			Gene products in transfer cell (feeding site)			Constitution of female† parasite	Effect
††h	→	nil		N factors	←	NN or Nn	Female reproduces
H	→	H factors	****	N factors	←	NN or Nn	Female dies
H	→	H factors		nil	←	nn	Female reproduces

† Males feed little and can have the constitution NN, Nn or nn.
†† H, *h* host genes for resistance; N, n matching genes in nematode.
**** genes products incompatible, feeding site spoiled.

pathotype of one or other of the species of potato cyst-nematode would take many years (Table 6) and this seems to be confirmed by field experience, especially with *G. rostochiensis* Ro1 which is the only known race occurring in Long Island, N.Y., USA, and where resistant potato cultivars with gene H_1 have been grown for many years.

Table 6. Years for immigrant or mutant gene to reach a frequency of 0.99 (0.50). Initial frequency 0.000001.

Rotation	Dominant	Recessive
RRR	322 (7)	70 (50)
RORO	574 (13)	161 (135)
ROOROO	829 (19)	295 (160)
ROOOROOO	1145 (29)	521 (237)
ROOOOROOOO	1656 (36)	986 (246)

Note: In the dominant condition the gene would reach a frequency of 0.50 rapidly but elimination of its recessive partner would be slow. In the recessive condition, a frequency of 0.05 takes many years to achieve but elimination of the dominant partner is faster. R, host cultivar with resistance gene; 0, non-host crop.

The slowness with which a mutant gene, whether a simple dominant or recessive, spreads through the population is largely because unhatched eggs from previous generations subsequently hatch and continue to produce males with the old genetic constitution. Because gene flow does not occur when reproduction is parthenogenetic or between sibling species that fail to interbreed successfully, their selection is relatively rapid. This is confirmed by experiences in the UK and the Netherlands where growing resistant potato cultivars selects *G. pallida* rather than a new pathotype of *G. rostochiensis*. The rate of change is determined largely by competition, i.e. *G. pallida* does not take over from *G. rostochiensis* until the density of the latter has declined (Fig. 9) and there is reason to believe that inefficient nematicides (e.g. the soil fumigant DD) actually speed the process by decreasing competition.

With species that persist as do cyst-nematodes, evolutionary selection evidently favours shutting off the gene flow and the development of sibling species. In other groups of species, e.g. *Meloidogyne*, it appears to have favoured parthenogenesis.

Discussion

The factors controlling the relationship between nematode pests and their host crops are of two types: those specific to the nematode-crop situation and those which are variable. Constant factors include the growth pattern of the crop, the nature of the injury inflicted and its characteristic distribution pattern on and between plants. Variable factors include time of attack in relation to growth, the intensity of damage caused, the duration of attack and environmental conditions affecting plant growth (Bardner &

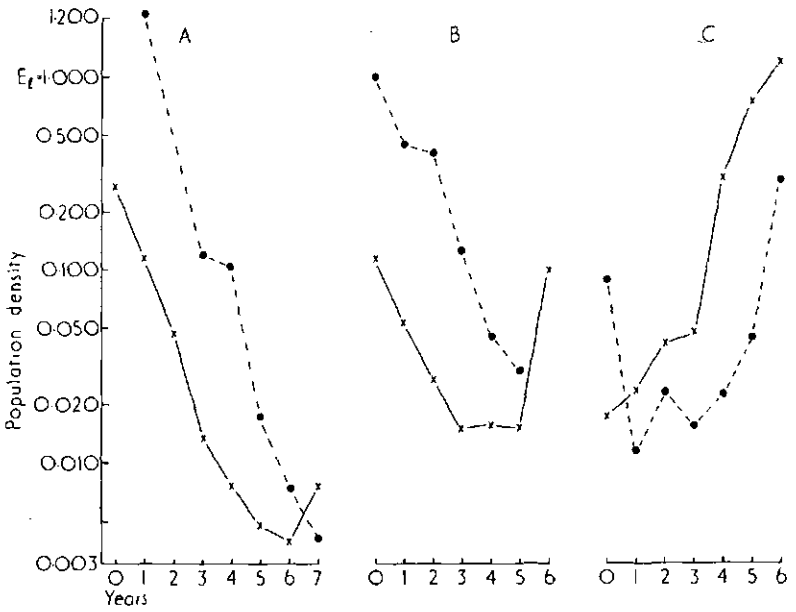

Fig. 9. Changes in the population density of potato cyst-nematodes at three sites A, B and C where potatoes with resistance gene H_1 were grown continuously for 6 or 7 years. – – – – heavily infested plots, —— lightly infested plots. Densities are eggs/g soil in spring expressed as a fraction of E_t the logistic equilibrium density. Descending curves represent diminishing numbers of *G. rostochiensis* and ascending curves are mainly eggs of *G. pallida* which was being selected. Data partly from Jones & Perry (1978).

Fletcher, 1974). To these must be added the population density of the pest and the rate at which it feeds and multiplies. The farmer has under his control previous cropping which determines the number of non-migratory pests in situ before the crop is planted, an especially important consideration for most nematode pests. In addition he controls the planting date, seed rate, fertilizer application and certain agricultural practices. Pesticides available to quell attacks by insects should they get out of hand are many but there are few that are outstandingly successful against nematodes and almost all of these have to be applied in advance (i.e. to prevent rather than to cure an attack). Factors the farmer cannot control are the weather and the inherent characteristics of the soil (Jones, 1976). However, for soil-inhabiting species, the soil climate is far more stable than the aerial climate is for species that live above ground.

In outbreaks of pest and disease organisms, all these factors apply whatever the group to which the organism belongs. The outstanding characteristic of nematodes, and especially of species with sedentary females, is their relative immobility, a characteristic they share with some soil-borne plant pathogens. Where the opportunity of dispersal on seeds or on or within planting material exists, the commercial and agricultural movements of these breach geographic barriers and secure widespread and rapid dispersal to new places where the host grows. Successful dissemination and the establishment of foci requires stages suitable for dispersal and preferably with a measure of desiccation

resistance. Movements of egg masses (e.g. of *Meloidogyne* spp.), possibly coupled with parthenogenetic reproduction, avoid some of the perils of under-population and tend to ensure survival. The cyst stage, which resembles the resting spores of fungi, and persistance related to hatching factors, greatly facilitate the dispersion of cyst-nematodes and this doubtless accounts for their success in regions climatically suited to them wherever their host crops are introduced.

The false root-knot nematode, *Nacobbus batatiformis* Thorne & Schuster occurs together with both species of potato cyst-nematode in S. Peru and N. Bolivia where it is perhaps more important as a potato pest. Although known in N. America and once recorded in Europe (Franklin, 1959), this species has not spread to temperate potato-growing areas. It appears to lack a dispersive stage equivalent to the cyst and may not be sufficiently resistant to desiccation or sufficiently persistent.

Although some of the nematodes dubbed 'exploiters' are able to increase more than 1000-fold in a season, this is a far smaller potential than that of many viral, bacterial or fungal pathogens. Many nematodes have seasonal increase rates less than 1000-fold and cyst-nematodes usually increase less than 100-fold. These slower increase rates inevitably mean that the time scale for nematode 'epidemics' is far longer than that of many fungal leaf and stem pathogens. The soil in which most nematodes live imposes severe limitations, and after foci of infestations are established, several years may be required for numbers to reach proportions that seriously diminish yields. Even when nematodes are carried on transplants, a delay of some years may ensue. For example, Blake (1961) reported that when banana sets infested with a race of the burrowing nematode were planted into virgin soil in Queensland, Australia, root-rot became apparent within 3 months but plantations usually persisted for 4 years before becoming uneconomic.

Much information has been gathered on the population dynamics of cyst-nematodes. The current model simulating their populations applies mainly to small plots (i.e. foci). The model needs to be made stochastic so that it may be applied to fields with patchy populations under circumstances that lead to the variation in model parameters (e.g. a, C, c, E_1, E). One piece of information lacking is quantitative data on the rate of dispersion within fields above and below ground, an essential ingredient if 'epidemics' are to be modelled successfully.

References

Bardner, R. & K. E. Fletcher, 1974. Insect infestations and their effects on the growth and yield of field crops. Bull. Ent. Res. 64: 141–160.

Bedi, A. S., 1968. A study of the variability in field populations of the potato cyst-nematode (*Heterodera rostochiensis* Woll.) in relation to resistant potatoes. Ph.D. thesis, Edinburgh University.

Blake, C. D., 1961. Root-rot of bananas caused by *Radopholus similis* (Cobb) and its control. Nematologica 6: 295–310.

Chitwood, B. G., 1951. The golden nematode of potatoes. U.S. Dept. Agric. Circular No. 875, 48 pp.

Cooke, D. A., 1978. In: R. K. Scott: Broom's Barn Experimental Station. Rep. Rothamsted Expl. Stn. for 1977 Pt 1, p. 52–53.

Evans, K., 1979. Nematode problems in the ley-arable experiment and changes in *Longidorus leptocephalus* population density with time, depth, cropping and soil type. Rep. Rothamsted Expl. Stn. for 1978 Pt 2, p. 27–45.

Evans, K. & D. L. Trudgill, 1978. Nematode pests of potatoes. In: P. M. Harris (Ed.): The potato crop: the scientific basis for improvement. London, Chapman & Hall, p. 440–469.

Faulkner, L. R. & W. J. Bolander, 1970. Acquisition and distribution of nematodes in irrigation waterways of the Columbia Basin in eastern Washington. J. Nematol. 2: 362–367.

Franklin, M. T., 1959. *Nacobbus serendipiticus* n.sp., a root-galling nematode from tomatoes in England. Nematologica 4: 256–293.

Gallun, R. L., 1974. Genetic interrelationships between host and organism and influence on resistance. In: F. G. Maxwell & F. A. Harris (Ed.): Biological control of plant insects and disease. Proceedings of the Summer Institute on Biological Control, Jackson, Mississippi University Press, p. 395–401.

Gregory, P. H., 1948. The multiple-infection transformation. Ann. Appl. Biol. 35: 412–417.

Griffin, D. M., 1970. Effects of soil moisture and aeration on fungal activity: an introduction. In: T. A. Toussoun, R. A. Bega & P. E. Nelson (Ed.): Root diseases and soil-borne pathogens. 2nd Symposium on factors determining behaviour of plant pathogens in soil, London, p. 77–91.

Harrison, B. D. & R. D. Winslow, 1961. Laboratory and field studies on the relation between arabis mosaic virus to its nematode vector, *Xiphinema diversicaudatum* (Micoletzky). Ann. Appl. Biol. 49: 621–633.

Hooper, D. J., 1975. Stem nematode, *Ditylenchus dipsaci* on field beans. Rep. Rothamsted Expl. Stn. for 1974 Pt 1, p. 183–184.

Jones, F. G. W., 1951. Further observations on the distribution of the beet eelworm. J. Helminth. 25: 223–230.

Jones, F. G. W., 1969. Some reflections on quarantine, distribution and control. In: J. E. Peachey (Ed.): Nematodes of tropical crops. Tech. Commun. Commonw. Bur. Helminth. No. 40, p. 67–80.

Jones, F. G. W., 1970. The control of potato cyst-nematode. John Curtis 'Woodstock' Lecture. J. R. Soc. Arts 118: 179–199.

Jones, F. G. W., 1974. Host parasite relationships of potato cyst-nematodes: a speculation arising from the gene-for-gene hypothesis. Nematologica 20: 437–444.

Jones, F. G. W., 1975. The soil as an environment for plant parasitic nematodes. Ann. Appl. Biol. 79: 113–139.

Jones, F. G. W., 1976. Pests, resistance and fertilisers. Proc. 12th Coll. Int. Potash Inst. Izmir, Turkey, p. 233–257.

Jones, F. G. W. & M. G. Jones, 1964. Pests of field crops. Edward Arnold, London, 406 pp.

Jones, F. G. W. & R. A. Kempton, 1978. Population dynamics, population models and integrated control. In: J. F. Southey (Ed.): Plant nematology. M.A.F.F., A.D.A.S. Booklet GD/1, p. 333–361.

Jones, M. G. K. & D. H. Northcote, 1972. Nematode induced syncytium – a multinucleate transfer cell. J. Cell. Sci. 10: 789–809.

Jones, F. G. W. & J. N. Perry, 1978. Modelling populations of cyst-nematodes (Nematoda: Heteroderidae). J. Appl. Ecol. 15: 349–371.

Jones, F. G. W., R. A. Kempton & J. N. Perry, 1978. Computer simulation and population models for cyst-nematodes (Heteroderidae: Nematoda). Nematropica 8: 36–56.

May, R. M., 1973. Stability in randomly fluctuating versus deterministic environments. Am. Nat. 107: 621–650.

Parrott, D. M. & M. M. Berry, 1974. Pathotypes and populations. Rep. Rothamsted Expl. Stn. for 1973 Pt 1, p. 152–155.

Perry, J. N., 1978. A population model for the effect of parasitic fungi on numbers of the cereal cyst-nematode, *Heterodera avenae*. J. Appl. Ecol. 15: 911–917.

Person, C., 1965. Genetical adjustment of fungi to their environment. In: G. C. Ainsworth & A. J. Sussman (Ed.): The fungi. New York, Academic Press Vol. 3: 393–415.

Petherbridge, F. R. & F. G. W. Jones, 1944. Beet eelworm (*Heterodera schachtii* Schm.) in East Anglia, 1934–1943. Ann. Appl. Biol. 31: 320–332.
Rothamsted Experimental Station, 1970. Details of the classical and long-term experiments up to 1967. Harpenden, 128 pp.
Schofield, R. K. & J. V. B. DaCosta, 1935. The determination of the pF at permanent wilting and at the moisture equivalent by the freezing point method. Trans. 3rd Int. Congr. Soil Sci. 1: 6–10.
Southwood, T. R. F., 1973. The insect/plant relationship – an evolutionary perspective. In: H. F. van Emden (Ed.): Insect/plant relationships. 6th Symp. Roy. Ent. Soc. Lond., 3–30.
Staniland, L. N., 1946. Potato root eelworm in the south-west. Agriculture 52: 538–541.
Suit, R. F. & E. P. DuCharme, 1957. Spreading decline of citrus. Bull. State Plant Board Fla. (II) 2: 24.
Thompson, H. W., A. Roebuck & B. A. Cooper, 1949. Floods and the spread of potato root eelworm. J. Minist. Agric. 56: 109–114.
Wallace, H. R., 1963. The biology of plant parasitic nematodes. Edward Arnold, London, 280 pp.

The management of epidemic levels of endemic diseases under tropical subsistence farming conditions

C. A. J. Putter

'In botanical ecology there are few fields likely to reap greater rewards than the study of endemic disease, with the pathogen as the focus of study.'

Vanderplank, 1975

Abstract

The epidemic patterns of temperate and tropical plant pathogens are contrasted in a comparison of diseases caused by two different species of *Phytophthora*. Disease control strategies for each of these two epidemic patterns are compared. Endemicity is proposed as a paradigm of tropical epidemiology and its implications for disease control are discussed and compared with the situation in temperate plant pathology.

Tropical plant diseases occur primarily in the developing countries. The socio-economic and ecological constraints which this imposes on pathosystems management are evaluated and an attempt is made to formulate a control strategy for tropical diseases.

Introduction

Subsistence farming is the major industry in the developing countries. It has been defined as an assemblage of techniques for living off nature while yet remaining in harmony with it. It cannot possibly be considered a single system, a single device or a single trait that can be changed by a single simple stimulus (Spencer, 1966). Instead it is more akin to a production philosophy which is inseparable from the larger socio-economic and cultural framework which gives each social group its unique identity.

Defined in this manner, these subsistence economies impose both socio-economic and ecological constraints on pathosystem management. Due to the close association between peasant culture and agriculture, a plant disease problem may be a social and cultural problem as much as it is a scientific and technical one. The economic constraints stem from the low level of technological development and poorly developed economic infrastructures. Consequently, the introduction of sophisticated crop protection technologies is usually unsuccessful. They often require the peasant to make a conceptual leap from a pre-wheel technology to that of a motorized knapsack sprayer. Furthermore, sophisticated crop protection strategies usually do not take into account either the ecological diversity on which subsistence agriculture is dependent, or the fund of ecological knowledge attributable to peasant cultivators (Anderson, 1952; Conklin, 1957; Barrau, 1958, 1961; Phillips, 1961a, b).

These considerations suggest that an appropriate crop protection strategy for subsistence economies should emphasize the manipulation of existing interactions while

rigorously limiting inputs incompatible with the ecosystem in question. This may be achieved if a pathogen ethograph[1] is formulated. Intervention points in the behaviour pattern may then be identified and employed to reduce disease severity while yet remaining compatible with the ecological – and socio-economic – matrices of subsistence farming communities.

A large portion of the developing countries is situated in the tropics and humid tropics. An important feature of tropical pathosystems is the endemic nature of plant diseases. Temperate pathosystems on the other hand are typically characterized by a seasonal disease-pattern, i.e. diseases which are absent for a period of each year and which subsequently pass through a focal and general epidemic phase (sensu: Vanderplank, 1963). A comparison of these two disease patterns facilitates, in endemic disease cycles, the identification of intervention points which may be exploited to formulate control strategies suitable for subsistence farming conditions.

Tropical endemic pathosystems compared with temperate seasonal pathosystems

Most plant pathologists are probably more familiar with temperate diseases and, therefore, the principles of botanical epidemiology are most frequently discussed using temperate examples. In Figure 1, the temperate *Phytophthora infestans* (Mont.) de By. – induced late blight of potatoes is compared with the *Phytophthora colocasiae* Racib. – incited leaf blight of taro, *Colocasia esculenta* (L.) Schott., as an example of a tropical endemic disease. The comparison would be valid for many important diseases of tropical perennial cash crops such as cacao, rubber, coconut palm and oilpalm. The choice made here is convenient rather than axiomatic and complies with the author's interest in subsistence farming. It is also thought that a comparison of two root crops affected by pathogens from the same genus is more meaningful.

In Figure 1 (d) the pathogen's progress, or the amount of disease, is diagrammatically represented as a function of time. This means that we can measure the pathogen's rate of increase by measuring the slope of the line which represents its progress.

During the endemic phase the line 'a' in Figure 1 has zero slope, i.e. the rate of increase is zero and disease is more or less pegged at a certain level. In contrast, the lines b1 and b2 illustrate rates of increase greater than zero. These lines, b1 and b2 as they are illustrated in Figure 1, show the similarity between epidemics of tropical and temperate pathosystems. This similarity is misleading and should not elicit the same strategic approach as is employed in temperate plant pathology. Specifically, in tropical pathosystems, the significance of an endemic phase when it acts as a background for explosive sporadic epidemics has been underrated.

An evaluation and comparison under two different disease patterns of strategies which reduce either the initial inoculum or the infection rate

The alternation between endemic and epidemic phases (Vanderplank, 1975), has important implications for disease control strategies. The general strategy of disease control consists of reducing the amount of inoculum from which disease starts, or

1. Ethograph: A description and quantification of the behaviour of an organism's interaction with its total environment.

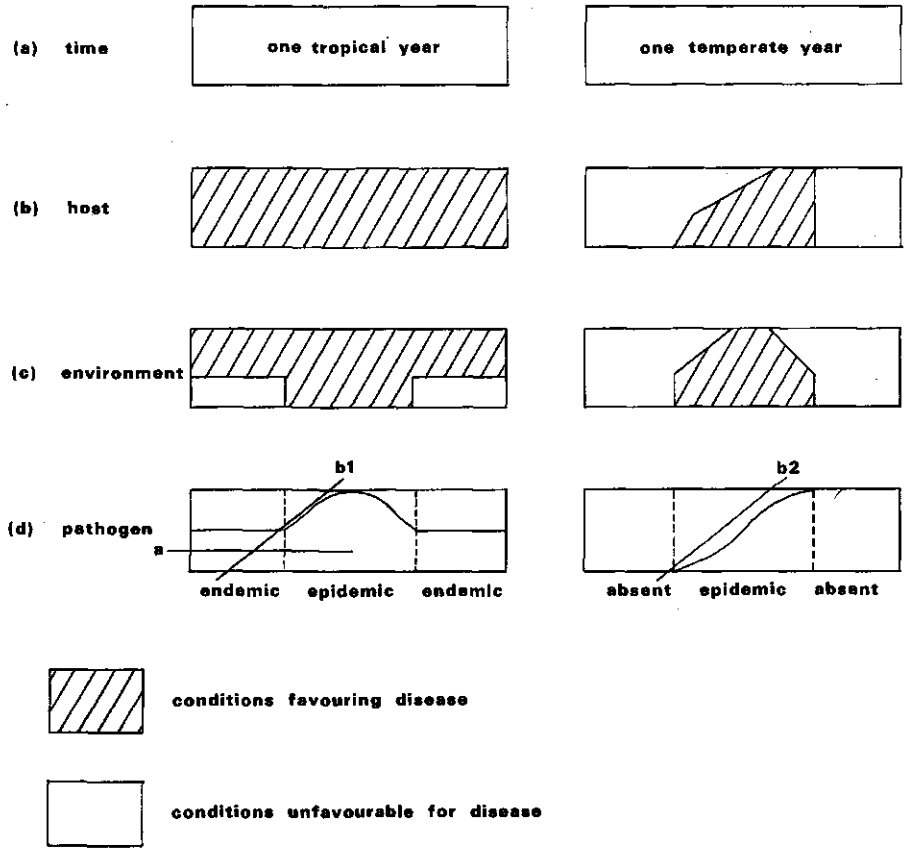

Fig. 1. A diagrammatic comparison of potato blight (right) and taro leaf blight (left) to illustrate the difference between tropical and temperate pathosystems.

reducing the rate at which it increases after it has started, or both (Vanderplank, 1972). Sanitation is an example of reducing inoculum, and horizontal resistance (sensu: Vanderplank, 1963) reduces the rate of disease increase.

Vanderplank (1963) explained how each of these broad categories of disease strategy is more efficient at some levels of disease development than others.

As a control measure, sanitation becomes less appropriate as the infection rate increases, i.e. the benefit from reducing the initial inoculum decreases as the rate of infection increases. For example, Vanderplank (1963) calculated that during the focal stage of potato blight epidemics, when the infection rate is low, disease losses are proportional to the initial inoculum. During the general epidemic phase, when the infection rate is high, disease losses are no longer proportional to the initial inoculum. This is illustrated in Figure 2.

If we consider the focal stage discussed by Vanderplank (1963) to be similar to the endemic phase inasmuch as they both have low infection rates, it follows that sanitation as a control measure will give better results during the endemic phase. On the other

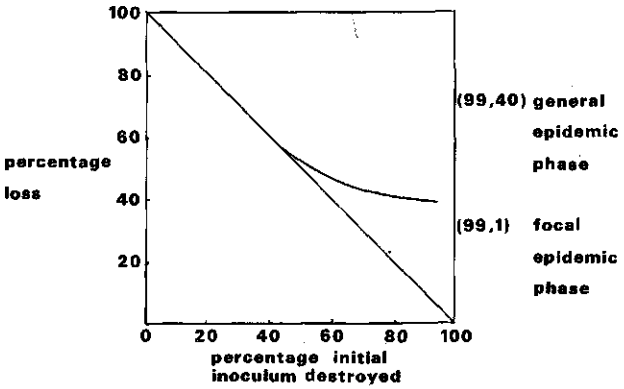

Fig. 2. The effect of destroying initial inoculum on reducing disease loss, expressed as a percentage of what the loss would have been if no inoculum had been destroyed. (Adapted from Vanderplank, 1963).

hand, control measures which reduce the infection rate are more effective when used to control explosive epidemics than they are with diseases which have a low infection rate. We can now consolidate this information and the concepts presented in relating the potential benefit of these two general control strategies to the level of disease as in Figure 3.

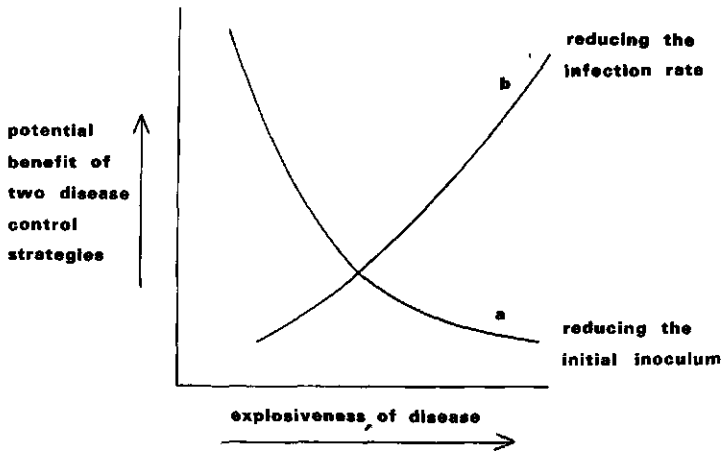

Fig. 3. A diagrammatic representation of the influence of disease explosiveness on the benefit obtained from reducing the initial inoculum and the infection rate.

When faced with a disease that is either purely endemic, i.e. its rate of increase is zero, or purely epidemic, i.e. with explosive infection rates, we would choose to reduce the initial inoculum or the infection rate, respectively. When sporadic epidemics occur against a background of endemicity (Vanderplank, 1975), the control strategy would have to be based on disease progress curves measured in such a way as to determine:

1. The proportion of time during which the disease is either endemic or epidemic.
2. The relative explosiveness of the disease during the endemic and epidemic phases, respectively.

This may be achieved by using Figure 3 as an heuristic model. Curve A diagrammatically represents the family of curves presented by Vanderplank (1963, Fig. 11.2, p. 126) and curve B is a diagrammatic representation of the fundamental paradigms developed by Vanderplank (1963, 1968, 1975). Accurate curves can be determined for any particular disease if enough data are available. The position of the intersection of these two curves will be unique for each disease and will determine the change in emphasis from one control strategy to the other, i.e. when to change from sanitation to infection rate reducing strategies.

The extreme sensitivity of endemic disease to sanitation may be discussed with reference to an equation proposed by Vanderplank (1975, eq..5.1 p. 114) which can be used to measure the degree of systems imbalance in endemic pathosystems. He explained that disease will increase and then level off at an asymptote L, where:

$$L = 1 - e^{i \cdot R \cdot L} \qquad (1)$$

Here $100L$ is the maximum percentage of disease reached in a focus as it levels off and $i \cdot R$ (from Threshold Theorum, Vanderplank, 1963), is a measure of the progeny per parent lesion ratio, *Pr/PaLR*. With selected values of $i \cdot R$ substituted in Equation 1, a graph can be drawn as in Figure 4.

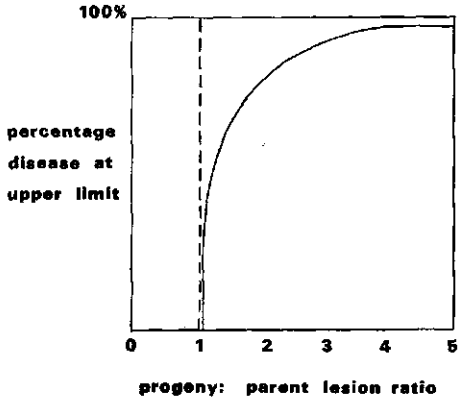

Fig. 4. The upper limit of disease with unlimited time to develop, as a function of the Progeny: Parent lesion ratio (based on Vanderplank, 1975).

When the *Pr/PaLR* lies between 1 and 2, the disease can be considered as being endemic rather than epidemic (Vanderplank, 1963). Within this range, small reductions in the *Pr/PaLR* have disproportionately large consequences in reducing the amount of disease. As the *Pr/PaLR* increases above 2:1, sanitation rapidly loses its spectacular effects on disease level.

The benefits of sanitation obey the law of diminishing returns for both tropical endemic and temperate seasonal diseases, as illustrated in Figure 5.

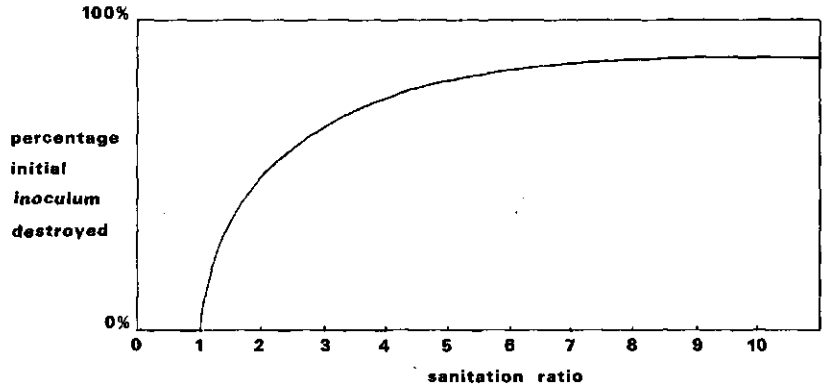

Fig. 5. The percentage initial inoculum destroyed as a function of the sanitation ratio (sensu Vanderplank, 1963).

In the case of the taro blight pathosystem, the ratio of absent:endemic:epidemic disease phases would be of the order of 0:8:4 months. In the temperate potato late blight pathosystem, the ratio of absent:focal epidemic phase:general phase would be nearer 8:1:3 months. Thus the benefits of sanitation accrue during a very short period in the temperate pathosystem whereas in the endemic pathosystem a much more significant period is spent at disease levels where sanitation would be the preferred strategy.

The rôle of environmental factors as determinants in the development of forecasting methods for endemic and seasonal diseases

From Figure 1, it can be deduced that in temperate disease the aim of forecasting is to predict the transition from focal to general epidemic phase. For instance, in the case of late blight of potatoes it is the date of this transition which determines when the first protective sprays will be applied (Vanderplank, 1963).

The equivalent rôle for forecasting in endemic diseases would be to predict when the transition from the endemic to the epidemic phase occurs, i.e. to predict the point in Figure 3 at which the two graphs intersect.

Putter (1976) analysed the taro blight pathosystem under subsistence farming conditions in Papua New Guinea (PNG). The taro (*C. esculenta*) is an annual root crop and in PNG it is cultivated in a continuous re-planting and harvesting-cycle. Often the central corm is removed and the side-shoots allowed to develop for future harvesting or to provide planting material for new gardens. In any one garden, taro foliage can be found throughout the year. In such a cropping pattern, taro blight is endemic and causes destructive epidemics only at certain times of the year. The change from endemicity to sporadic epidemics is related to weather patterns as indicated in Figure 6.

Figure 6 illustrates that taro blight epidemics occur during the 'wet' season. Yet in spite of this, the disease is present during the 'dry' season. Since the disease is polycyclic with a short generation time, it therefore follows that the host × pathogen × environment interaction is favourable throughout the year for successful sporulation, dissemination, infection and symptom development.

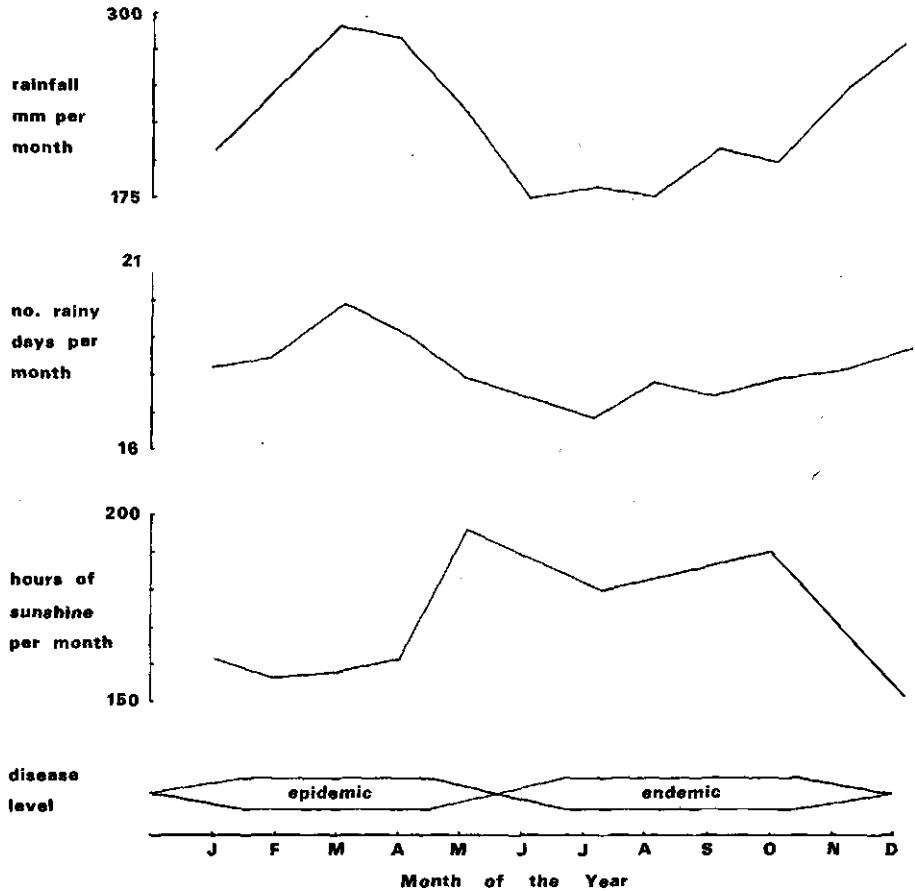

Fig. 6. Meterological data for Kerevat, PNG. 20 year averages of rainfall, sunshine and number of rainy days/month and the occurrence of taro blight epidemics.

Thus endemicity implies that at least the minimum threshold environmental requirements of the pathogen are present throughout the year. When these minimum requirements change and become more favourable for disease development, the pathogen increases to epidemic proportions. This pattern of environment × (host:pathogen) interaction in endemic pathosystems allows an evaluation of the minimum requirements of the pathogen by providing an opportunity for studying the pathogen when it is in ecological equilibrium with its host and environment. Such a situation also gives an ideal opportunity for studying compensation phenomena (sensu Aust et al., this volume) and the specific nature of weather changes required to increase endemic disease to epidemic proportions.

Environmental compensation phenomena may limit the disease to endemic status in several ways. They may suppress all the processes in the epidemic chain (sensu Gäumann, 1951) or act as limiting constraints on one or a few of the links in the epidemic chain.

With these considerations in mind, Putter (1976) was able to analyse the taro blight pathosystem depicted in Figure 6, which allowed him to:

1. Identify the environmental factors which ensure endemicity for the taro blight pathogen, i.e. endemicity is ensured by the non-seasonal pattern of nightly weather between 18:00 h and 06:00 h.
2. Determine that the increased incidence of favourable daily weather patterns during the wet season, by interacting with the preceding nightly weather, is conducive to epidemic development of the pathogen.
3. Determine that the pathogen is dispersed by wind-blown rain and run-off water which drains from infected to uninfected areas.
4. Determine that spread and dispersal are two distinctly separate epidemiological aspects of pathogen dissemination. Conditions which favour spread within a population occur throughout the year. Conditions which favour dispersal between discreet host populations are affected by wind-blown rain which occurs more frequently during the wet season than during the dry season.

It seems reasonable to postulate that compensation phenomena operate in most endemic pathosystems in which sporadic epidemics occur. Weather conducive to epidemics would be seasonal and would act in a compensatory manner when it augments an environmental factor or factors which keep the disease endemic and at low levels. Thus by simply comparing key meteorological variables measured during the endemic phase with those during the epidemic phase the likely compensatory factors may be identified.

The implications of endemicity for foci of infection and sources of inoculum

A comparison of the distribution pattern of infection foci in populations of fields in temperate non-endemic and tropical endemic diseases, reveals further implications for the choice of an appropriate disease control strategy.

In the case of potato late blight for example, the ratio of uninfected to infected fields is high during the focal epidemic stage and decreases as the general epidemic phase progresses. In contrast, the very definition of endemicity suggests that in tropical ecosystems, this ratio remains low throughout the year. This suggests that in tropical pathosystems within-field sources of inoculum could be more important than distant sources. From this hypothesis one may logically attribute inoculum in endemic situations to one of three sources:

1. It can be endemic in the soil or can survive on alternative or feral hosts within the area.
2. It can be introduced with the planting material or during cultural operations.
3. It can arrive at some later date when some agency of dispersal becomes available.

In the case of taro blight, Putter (1976) eliminated survival of *P. colocasiae* on alternative hosts and feral taro as sources of initial inoculum. Survival of inoculum in the soil was avoided because the long rotation of the bush-fallow cropping pattern means that new gardens are established in virgin soil. Inoculum on infected planting material was easily eliminated by a simple sun-drying process prior to planting. Finally, as the conditions which govern dispersal had been established, Putter (1976) was able to reliably forecast infections which would follow interpopulational dispersal.

The establishment of a focus or foci of infection follows upon the arrival of the

pathogen in a hitherto uninfected field. In the period immediately after this act of dispersal and successful invasion, the comparatively low infection rate in the foci and the clear delineation of the foci suggest that the removal of lesions, infected leaves or even plants is epidemiologically indicated as the appropriate strategy. To test this hypothesis Putter (1976) removed infected taro leaves daily for a couple of days following a first or new establishment of the disease. These tactics were successful and the disease could be turned 'on' and 'off' at will. This was done in a taro garden which the author maintained for student demonstration purposes.

The management of subsistence crop pathosystems

Certain paradigms and patterns of endemic disease have been compared here in order to develop disease control methods acceptable to subsistence farmers. The emphasis has been on sanitation because in the author's experience of subsistence farming conditions, few other disease control strategies are likely to be accepted by peasant cultivators. If excessive attention appears to have been devoted to providing epidemiological proof of the value of sanitation, it is in reply to the scepticism frequently elicited by the statement that sanitation alone may entirely control epidemics occurring against a background of endemicity. Part of this scepticism is based on the extrapolation of temperate disease control concepts to the tropics. In the process the ratios referred to on page 97 are ignored; the disease is noted only when it is spectacular and the value of sanitation is lost in the hurried search for a suitable fungicide which would reduce the infection rate.

Further components of this scepticism are founded on ignorance of the intimate involvement of the subsistence farmer with his crop, the scale of his operation and the diverse nature of the objective of subsistence farming.

Subsistence farmers are umbilically dependent on their crops to provide their daily food. They harvest daily or at very short intervals and often tend their plants individually. They need little or no encouragement to remove sick leaves or ugly spots. The subsistence farmer is submerged in his ecosystem, interacting as a component. He cannot stand outside the system, weighing his actions to determine whether or not they are economical or amenable to cost-benefit-analyses. The currency of a subsistence economy is food and its success (or failure) can most accurately be measured by the nutritional status of its participants.

The agricultural methods of developed countries are geared to achieve a single goal of yield optimization whereas subsistence farmers emphasize continuity and diversity of food supply. An additional complication is that the subsistence farmer's social status is determined by the quality and variety of his staple crops.

To fulfil such a spectrum of dietary and social needs, a genetically diverse cropping pattern has evolved. For generations the subsistence farmer has been schooled in an approach which would make him loathe to place all his eggs in the monoculture basket. This is tantamount to a production philosophy which is the very antithesis of the single 'miracle' variety concept on which the green revolution is based. The fact that these 'miracle' varieties require fertilizer to achieve their potential (i.e. optimum yield per unit area) further mitigates against their introduction and survival in subsistence ecosystems.

The same selection process, which provides the subsistence farmer with a spectrum of varieties, will have endowed him with a homeostatic genetic system which is in near-

ecological equilibrium. The mean quantity of population resistance will be high and the vertical- and horizontal-pathosystems (sensu Robinson, 1976) will be in a more natural equilibrium than controlled plant breeding could bring about.

This raises the question as to which disease resistance strategy would be most appropriate to tropical endemic disease situations.

Robinson (1976) recommended the deployment of horizontal resistance as the ideal strategy for the developing countries. Certainly his concept of a spectrum of horizontally resistant landrace-like cultivars is much more appropriate to subsistence farming conditions than is the green revolution concept of the monoculture of miracle varieties. However, in certain endemic tropical pathosystems, the judicious use of vertical resistance mechanisms is indicated. Consider an endemic pathosystem in which the *Pr/PaLR* is in the region of 1 and where environmental factors rather than genetic variation are responsible for sporadic epidemics. Here the selection pressure exerted by the host population to develop virulence in the pathogen population will be weak and use of vertical resistance is indicated (Vanderplank, 1978). Vertical resistance also reduces the initial inoculum (Vanderplank, 1978, equation p. 118) and its use is therefore appropriate to endemic diseases in which the *Pr/PaLR* is low for relatively long periods.

The obstacles in the way of manipulating host resistance to reduce disease severity under subsistence farming conditions are socioeconomic in nature rather than technical or epidemiological. The logistic and economic obstacles in the way of the distribution of new varieties are extremely large and it is questionable whether the results of plant breeding efforts can reach the majority of subsistence farmers.

It is fashionable to think of the traditional agriculture of subsistence farmers as a sinking ship which should be abandoned and from which, moreover, very little can be salvaged. The salvation of the developing countries is seen to be in the massive infusion of foreign capital and expertise. This approach ignores the positive aspects of the subsistence ecosystem which are the result of a selection process which incorporates many buffers and safeguards to prevent catastrophic system failure. If these positive aspects are ignored instead of being used as a foundation for further development, we may soon need to coin a term analogous to 'Vertifolia effect' (Vanderplank, 1963) to describe the resultant ecological disaster.

References

Allen, W., 1976. The African husbandman. Oliver and Boyd, London.

Anderson, E., 1952. Plants man and life. Little Brown & Co, Boston, 245 pp.

Barrau, J., 1958. Subsistence agriculture in Melanesia. Bernice P. Bishop Museum, Bull. 219.

Barrau, J., 1961. Subsistence agriculture in Polynesia and Micronesia. Bernice P. Bishop Museum, Honolulu.

Conklin, H. C., 1957. Hanunoo agriculture. A report on an integral system of shifting cultivation in the Philippines. FAO, Rome 1957.

Gäumann, E., 1951. Pflanzliche Infektionslehre. 2nd ed. Birkhäuser, Basel.

Phillips, J. F. V., 1961a. The development of agriculture and forestry in the tropics. Patterns, problems and promise. Faber & Faber, London.

Phillips, J. F. V., 1961b. Agriculture and ecology in Africa. A study of actual and potential development south of the Sahara. Faber & Faber, London.

Putter, C. A. J., 1976. The phenology and epidemiology of *Phytophthora colocasiae* Racib. in Papua New Guinea. M.Sc. thesis, University of Papua New Guinea.

Robinson, R. A., 1976. Plant pathosystems. Springer Verlag, Heidelberg.
Spencer, J. E., 1966. Shifting cultivation in Southeastern Asia. Univ. of California press.
Vanderplank, J. E., 1963. Plant diseases epidemics and control. Academic Press, N.Y., 349 pp.
Vanderplank, J. E., 1968. Disease resistance in plants. Academic Press, N.Y., 206 pp.
Vanderplank, J. E., 1972. The basic principles of ecosystems analysis. In: Crop protection strategies for the future. N.Y. Academy of Sciences.
Vanderplank, J. E., 1975. The principles of plant infection. Academic Press, N.Y., 216 pp.
Vanderplank, J. E., 1978. The genetic and molecular basis of plant pathogenesis. Springer-Verlag.

Epidemiological factors as related to plant disease control by cultural practices

J. Rotem and J. Palti

Abstract

Most cultural practices exert their effect on disease control through manipulation of the crop climate, which in turn depends largely on the prevailing macroclimate. The extent to which such practices can affect the crop climate is generally limited; their effect is therefore of greatest practical importance when they can tip the balance of conditions which are less than optimal for the pathogen, to be even less favourable to the latter, but not under conditions approaching the optimum for pathogen development.

An attempt is made to assess the prospects of controlling plant disease by cultural practices through a comparative evaluation of their interaction with host and pathogen characteristics – chiefly rate of inoculum build-up, survival potential of the inoculum, its mode of dispersal, and the rate of pathogen penetration into the host.

Even where their prospects for disease control are good, the use of cultural practices is often conditioned by economic, psychological and professional factors, which are discussed briefly.

Introduction

Epidemiology, in its practical sense, is the study of disease mounting to a level of economic thresholds. Cultural control aims at preventing or intercepting epidemics by means other than resistance breeding and chemicals.

Development of cultural practices for disease control largely reflects the way farmers, in their particular agro-ecosystems, act to interfere with disease surging to an epidemic climax. Comparisons of the practices adopted for this purpose are largely empirical and their validity is limited to the specific frameworks of climate and soil. In fact, conclusions drawn from comparisons of a given cultural factor (e.g. irrigation, or date, site and density of sowing) operating under widely divergent conditions, risk being quite misleading (Palti & Rotem, 1980).

In the present discussion we attempt to analyse how similar factors exert different effects on disease development under different conditions. The factors to be compared include meteorological variables as well as specific characteristics of hosts and especially of pathogens. For reasons to be detailed later, we shall focus in particular on measures designed to limit inoculum build-up.

Since cultural control of a crop disease is a decidedly practical subject, our analysis of the biotic and environmental factors determining prospects of success is preceded by discussion of some psychological, economic and professional factors that affect the farmer's desire to use cultural practices for disease control.

This presentation is not an enumeration of techniques used or recommended for disease control by cultural practices. For a more general appreciation of such techniques and of some theoretical aspects not treated here, the reader is referred to Berger (1977),

Collins (1978), Palti & Rotem (1980) and, especially, to the comprehensive review by Stevens (1960).

Essentials of disease control by cultural practices

Cultural control consists basically of the manipulation of pre-growing and growing conditions to the detriment of the pathogen. In more technical terms, the primary aim of cultural control is to minimize contact between a susceptible host and viable inoculum, and to interfere with the effects of such contact, i.e. reduce rates of infection and of subsequent disease development.

There are three principal approaches to this end:

1. Suppression of inoculum development or destruction of existing inoculum in the soil, e.g. by cultivation, crop rotation or heat disinfection, and prevention of the build-up of air-borne inoculum, chiefly by regional planning and modification of the crop climate.
2. Escape of crops from potential attacks by pre-sowing choices of soil, season, and topography, and by post-sowing choices such as irrigation techniques that do not wet leaves, etc.
3. Regulation of plant growth for minimum susceptibility, especially avoidance of weak or over-rich growth by choosing proper rates of sowing, fertilization and irrigation.

Obviously, these three approaches are often combined, and a single example may suffice here: Under specific weather conditions, attacks of *Phytophthora infestans* (Mont.) deBy on irrigated potatoes may be minimized by sowing them on heavy soil, where irrigation may be applied at longer intervals, irrigating by furrows or trickling instead of sprinkling (Rotem & Palti, 1969), sowing not too densely, and applying nitrogen at rates low enough to prevent excessive foliage growth. However, under different conditions, none of these practices may be of much use, and this is where epidemiological comparisons may help to predict success or failure.

The potential for cultural control is directly related to the opportunity of manipulating growing conditions, the expense involved therein, and the relative cost, reliability and speed of alternate control measures. Although applicable at all levels of farming, cultural practices seem particularly relevant to the lowest and highest levels of farming. In primitive or subsistence farming, introduction of new, resistant varieties is slow, chemicals may be unavailable or too expensive, and the farming community arrives by trial and error at practices that tend to minimize epidemics. The relevance of cultural control under such conditions is described by Putter in this volume.

At the other end of the scale, in sophisticated farming, cultural control assumes special importance where crops are grown out of season, by protection from the cold, or gifts of water in dry seasons; here opportunities for manipulation are greatly increased. Although such high-value crops are in general protected by chemicals, since the grower has to play safe, cultural practices can make decisive contributions to crop health, freedom from chemical residues, etc.

Comparison of economic, psychological and professional factors that influence the use of cultural versus chemical control measures

Economic factors

Modern farming is an industry based on large investments and striving for maximum profit at minimum risk. Farmers therefore incline towards the use of pesticides wherever this can be justified economically, either as producing direct benefit or as minimizing risks. The greater the benefit to be expected from chemical control, and the less acceptable the risk of losing yields (partly or entirely), the less the chance of reliance on cultural control.

For instance, with potatoes, the danger of explosively developing air-borne diseases such as late blight is much greater than that of slow spreading Verticillium wilt or nematodes; the farmer will therefore rely on cultural practices against the latter pathogen rather than against the blight. In high-value crops such as flowers, it is the speed and reliability of control rather than cost that matters most, and chemical control is then the obvious choice. As regards soil-borne diseases, farmers will further consider the high cost of chemical soil disinfection, as compared with rotation or other cultural measures. In every case the farmer's decision will depend on the destruction potential of the disease and the relative efficacy of the treatments, that is the returns to be expected from each treatment.

Psychological factors

The psychological limitation to the use of cultural control practices lies in their relatively slow, gradual and unspectacular achievements. In many cases, the effects of chemical control are rapidly apparent, while those of cultural practices are discernible only after a longer time. This often prejudices farmers in favour of chemical control.

Professional and educational factors

By their very nature, cultural control operations often have to be specifically adapted to particular field conditions in a given region or even on a given farm. Where there is a wide choice of such operations as especially in advanced, but not in primitive farming, the proper use of cultural techniques may therefore require a higher educational and professional level of the farmer than is demanded by most chemical control operations. With some elementary care, almost anybody can spray, but to manage soil, water and the many other elements of cultural control for maximum benefit, requires know-how and flexibility. The following discussion focuses chiefly on cultural control measures under multiple-choice conditions.

Another handicap for cultural, as compared with chemical, control lies in the respective research facilities and costs, and in the source of research funds. Fungicide research is financed largely by the chemical industry, requires only relatively small experimental plots, and is easily applicable to different climates and weather regimes. By contrast, research in cultural control has little industrial backing, is often drawn out over many years and/or variable weather conditions, and is much less widely applicable. Studies of crop cultivation and rotation may turn into life-time propositions, and are not overly

attractive to the research-paper-oriented scientist. In addition, cultural control research requires a farming know-how possessed by relatively few scientists, and this is one of the basic reasons for the paucity of such research.

Summary of factors that influence choice between cultural and chemical control measures

A comparison of the characteristics of cultural and chemical control, showing the limitations of the former, appears in Table 1.

Table 1. Characteristics of cultural and chemical control.

	Cultural control	Chemical control
Results:	Somewhat variable, gradual and unspectacular	More constant and predictable, rapid, often spectacular
Applicability:	Local and specific	Much more general
Know-how:	Considerable know-how required	Almost anybody can spray
Investment:	Mostly low or one-time investment	High and recurrent expenses
Field Experimentation:	Complex, long-term, funds hard to obtain	Simpler, more rapid, backed by industry

Comparison of meteorological and climatic factors that influence success of cultural control practices

The fact that cultural practices aim chiefly at adjusting field conditions to the detriment of the pathogen, has three corollaries:

1. If weather and field conditions are entirely unfavourable to disease development there is no room for their improvement by cultural means.
2. If these conditions are extremely favourable for disease development, adjustment of field conditions – always limited in scope – is unlikely to be of decisive influence in reducing disease.
3. If pathogens are capable of reaching epidemic development under a very wide range of field conditions, then there is little hope of checking the epidemic by cultural means.

This means, in effect, that in crop climates extremely beneficial or extremely detrimental for the pathogen, and in relation to highly adaptable pathogens, chances for the success of cultural disease control are, to say the least, very doubtful.

As mentioned repeatedly in this paper, cultural control measures have best prospects of success where conditions are only partly favourable to the pathogen. Under these conditions, the efficacy of cultural practices in controlling disease depends on their interference in the interplay of crop, pathogen and weather factors. Figure 1 illustrates schematically most of the potential interactions. It shows that disease control is achieved chiefly through the effect of cultural operations on the microclimate of soil and air, i.e. the crop climate. In addition, there are some important direct effects on the host, especially through the supply of nutrients and water; these in turn affect foliage density, and hence the crop climate, and sometimes also the susceptibility of tissues. Direct

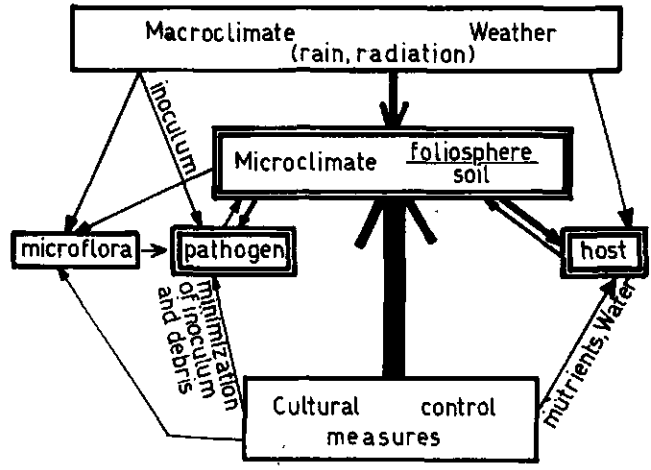

Fig. 1. Schematic representation of the interactions of crop, pathogen and environmental factors.

effects on the pathogen relate chiefly to the build-up, dispersal and viability of inoculum. It should be duly stressed that all cultural factors bearing on the crop climate are conditioned, to a greater or lesser extent, by the prevailing macroclimate (Rotem, 1978). Cultural practices may therefore modify, but not drastically change, the crop climate. The exceptions to this rule are irrigation under very dry conditions, and cropping under cover under cold conditions – both practices with fundamental effects on the microclimate.

Modifications of the microclimate may be most far-reaching under extreme conditions, e.g. in a rainless season with high radiation, high temperature and low relative humidity. However, under such conditions pathogen development is restricted anyway, and the control effect will be minimal (see below).

Effects of cultural practices on the crop climate, though more limited in scope, are more significant for disease control under conditions less extreme or even marginal, e.g. where some reduction in the number of hours of wetness per day, or somewhat higher or lower temperatures, can influence disease development markedly. It is under such conditions, only partly favourable to the pathogen, that manipulation of the crop climate can be used to greatest effect.

It follows from the above (a point made again and again in this discussion) that the efficiency of similar cultural control measures is conditioned by the specific local and actual weather conditions, and the effects of the same measure may differ widely under different environmental conditions. If, for instance, we try to control some soil borne pathogen by exposure to unfavourable environmental conditions, by means of soil cultivation followed by fallow, this may have a positive effect in a hot, dry and sunny region but may fail in a mild, rainly and cloudy region. Similarly, if we try to limit spread of an air-borne and humidity-dependent leaf pathogen by minimizing crop humidity, the choices are increased spacing, decreased supply of nitrogen, use of less compact cultivars, shift from overhead to surface irrigation, etc. Under the extreme conditions of a hot and dry site (e.g. a desert oasis) these measures are not needed because the macrometeorological conditions are, in most cases, not conducive to disease develop-

ment. In hot and dry, but not extreme conditions, they may have a profound effect on crop microclimate and hence on disease development. They are not expected to influence the crop microclimate and disease development in constantly humid, rainy and mild areas or seasons.

Comparison of pathogen characteristics that affect the efficiency of cultural control

The pathogen characteristics that most decisively affect epidemic development are (i) the rate at which the pathogen is capable of building up inoculum in the field; (ii) the survival potential of the inoculum from season to season and, once a field is infected, from sporulation to host penetration; (iii) the mode of dispersal; and (iv) the speed of the penetration process. The nature and size of effects of cultural practices on these characteristics are crucial for the success of these practices. It follows that the same cultural practice, applied under identical environmental conditions, may have totally different effects on diseases caused by pathogens differing markedly in the above characteristics.

Rate of inoculum build-up

Inoculum build-up is an integrative process related to many factors, including rate of infection, duration of incubation period and rate of necrotization (Rotem, 1978). Rate of inoculum build-up is therefore a convenient aspect to focus on, where effects of cultural practices on disease control are to be examined.

Few cultural measures, except perhaps solar disinfestation of soil, can interfere with inoculum build-up as incisively as fungicides. Compared with the latter, cultural methods are gradual investments that pay off only if maintained over extended periods. They are therefore more effective in the control of diseases that spread slowly, or, in the terminology of Vanderplank(1963), 'simple interest' diseases. These are much more often soil-borne than air-borne diseases. With some soil-borne pathogens it often takes years for the inoculum to reach levels that threaten the crop as a whole (Rotem, 1978); this process can be counteracted by properly conceived crop sequences (rotations), cultivation, debris management, and the above mentioned solar soil treatment (Katan et al., 1976).

The build-up of inoculum by airborne pathogens is much harder to prevent, since the diseases they cause, in Vanderplank's terminology 'compound interest' diseases, can take advantage of even short spells of favourable conditions to reproduce abundantly from a small amount of initial inoculum (Rotem, 1978).

The sources of initial inoculum will be more closely examined in the section on inoculum survival, but we must mention here the crucial role of even partial reduction of inoculum potential at the beginning of the growing season. Such reduction obviously has a more far-reaching effect with pathogens that multiply slowly; however, it also helps in delaying the onset of diseases caused by rapidly multiplying air-borne diseases. Effects of sanitary measures, such as destruction of potato tubers carrying *P. infestans* or of apple leaves carrying *Venturia inaequalis* (Cke.) Aderh., illustrate our point.

It should again be stressed, how much the success of cultural practices in retarding build-up of inoculum depends on specific local conditions as well as on all the factors affecting sporulation and infection. Thus, in Israel, multiplication of inoculum and

subsequent attack of *Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni on vine foliage could be restricted by proper irrigation management (Avizohar-Hershenzon & Hochberg, 1969), but this is true only when the following conditions are met: (i) little or no rain; (ii) short periods of dew; and (iii) most of the leaf tissue has passed the stage of extreme susceptibility.

The success of cultural practices under conditions partly favourable to the pathogen thus depends on a combination of environmental and biotic (susceptibility) factors. Under highly unfavourable conditions, even the availability of inoculum will not necessitate any control measures, since further build-up is unlikely. A case in point is that of late blight on tomatoes in late summer in the desert regions of Israel: although inoculum developed in earlier summer months is present, its build-up is arrested in later months and no control measures are needed.

One important way in which different climatic regimes affect the build-up of inoculum, is through their effect on the duration of the vegetative season. Where either cold winters or hot and dry summers (without irrigation) result in relatively short and discontinuous seasons of cropping, the amount of inoculum present at the beginning of the vegetative season is relatively low. It is always lower than the amount of the same pathogen species present in a climatic regime in which mild winters and moderately warm and rainy summers (or irrigated crops) make continuous, year-round cultivation of the host plant possible. In the latter case, persistence of the pathogen throughout the year provides a basis for rapid multiplication of inoculum in every newly sown field. Cultural control through elimination of overseasoning inoculum is then obviously difficult. This is one of the reasons for almost year-round epidemics of late blight in Israel (Palti & Rotem, 1973), and the absence of so-called zero dates (Grainger, 1950) before which epidemics are not expected for a number of diseases. In tropical areas this may be even more of a problem, since many crops can be grown continuously and productively, but unfortunately also more susceptible cultivars are being introduced continuously. Consequently, general cropping usages, developed under various climatic regimes, by their effects on rates of inoculum build-up, may affect local effectiveness of control measures. This topic is discussed further in the next section.

Survival of inoculum from season to season

Discussing crop rotation, one of the classical practices to reduce survival of soil pathogens, Walker (1957) has pointed out that this is probably the control measure most widely misused in farming; it is often recommended as a routine measure, without due consideration of the chances of actually reducing inoculum of the pathogen in question. These chances obviously depend on characteristics such as host range, saprophytic survival, presence of resting bodies, resistance to extreme conditions, etc., but these have not always been taken into account.

It is further obvious that the same control measure may have different consequences in different types of soil and in different climatic regimes. For instance, the already mentioned solar disinfestation of soil covered with plastic sheets destroys certain soil-borne pathogens in the hot regions of Israel (Katan et. al., 1976) and California (Pullman et al., 1978), but the same treatment would probably have little effect in areas with weaker insolation.

The rate of inoculum survival in seasons unfavourable to the pathogen and/or in the

absence of hosts is obviously a primary consideration in planning any measure of control. Pathogens, of course, differ vastly in their power of survival from one season to the next. Such survival may assume many forms, some amenable to direct control, others far less so. Where a fungus survives in dormant parts of the host, as *Podosphaera leucotricha* (E. & E.) Salm. does in apple buds, pruning the affected buds is of direct help. However, where survival is by soil-borne bodies, more indirect means have to be employed, such as cultivation, soil amendments, and stimulation of competitive or antagonistic soil flora (Baker & Cook, 1970).

With regard to many pathogens we are ignorant of the factors affecting their survival from season to season. This leads to 'blind' planning of cultural (as well as chemical) control measures, without the required knowledge of viable inoculum present. In fact, some of the commonly held beliefs on conditions favouring inoculum survival in the soil are quite erroneous. Thus it has long been held that pathogens in general survive better in wet than in dry soil, but this is true only for certain species. At least in the case of *Alternaria* blight of potatoes, the reverse has been found true: one of the reasons for epidemics of this blight in a desert zone has been found to be the successful over-summering of the pathogen under extremely dry conditions. Simple wetting of the soil, or growing irrigated crops in plots containing debris of *A. solani*, was found effective in reducing the amount of inoculum (Rotem, 1968). A similar procedure cannot be expected to reduce inoculum of those soil-borne pathogens which, in the absence of their specific hosts, may survive as saprophytes.

As regards overseasoning of pathogens spread by air-borne inoculum, a distinction must be made between climates in which either pronounced winter cold or summer heat and drought result in an effective incision in the annual cycle of crop and pathogen development, and those climates in which this is not so. Where the incision is pronounced, inoculum is normally reduced to low levels. If crops are then grown in spite of the unfavourable weather, this is done by application of cultural means (e.g. growth under cover, irrigation), which can often be manipulated to the detriment of the disease.

In climates without a comparable incision, where winters are mild and summers moderately warm and rainy, year-round persistence of inoculum on host shoots provides a basis for rapid multiplication when the main growing season arrives. Such survival may only take place in favourable ecological niches, and the overall amount of inoculum will be much lower than in the season, and yet this type of survival is apt to give the pathogen a rapid start at the beginning of the cropping season, or when new growth begins to form on evergreens such as olives and citrus.

Short-term survival of inoculum

An accurate understanding of the factors affecting survival of inoculum on unfavourable days, or parts of days, is required to guide cultural control measures to success against air-borne pathogens. One must have a good idea of the levels of temperature and humidity which the pathogen's spores can withstand, and for how long. In general, the more sensitive the dispersed spores, and the more adverse the environmental conditions, the better is the chance of interfering with disease development by cultural means. If the spores are drought resistant or, as in the case of *Alternaria solani*, can survive drought even when they have begun to germinate and simply continue germination and penetration with the advent of the next spell of wetness (Bashi & Rotem, 1974), then

there is little one can do by manipulating moisture in the field. On the other hand, if inoculum is as drought-susceptible as the sporangia of *P. infestans*, a great deal can be achieved by moisture management, e.g. sprinkler irrigation applied not early in the morning but later in the day, when some of the sporangia have died (Rotem & Palti, 1969).

The possibility of minimizing potato late blight by irrigation management brings out the interplay between spore susceptibility and environmental conditions which make such control possible. However, this method is efficient only in a hot and dry area or season, in which environmental conditions affect the dispersed sporangia adversely; it is not efficient in a cooler and more humid area.

Under specific weather regimes, which limit the efficiency of infection, cultural methods can also be employed to control pathogens with robust spores. This applies to the case of Stemphylium blight, discussed in the section on rate of penetration. It also applies to *A. solani* on potatoes, which spreads by extremely-resistant spores. In the climate of Israel, where dewfall is common, irrigation does not affect the development of this pathogen (Rotem & Palti, 1969; Rotem & Reichert, 1964), but in areas in which dew is sparse, overhead irrigation may favour the early blight disease, as it does in the summer season in the desert of Idaho (Guthrie, after Waggoner & Horsfall, 1969).

Mode of dispersal

The principal modes of dissemination above the ground are by wind and by splashing. Crucial for the efficacy of cultural practices aimed at minimizing dispersal is the distance over which spores are expected to travel without losing viability under the prevalent weather regime.

There is no effective way of interfering with the wind transport of spores that remain viable under wide ranges of moisture and temperature conditions. Thus certain rusts are capable of causing epidemics after long-range dispersal (Rowell & Romig, 1966; Nagarajan et al., 1977). The chances for interfering with dispersal are better with spores that tend to diminish in viability or lose it altogether if conditions are unfavourable. Handicapped by this susceptibility, fungi such as species of *Phytophthora* and some powdery mildews sometimes spread only step by step, within a given region. This gives a well-organized farming community the opportunity for control by regional regulation of factors affecting the onset of disease, e.g. dates of sowing, mode of irrigation, as well as chemical control on a regional basis. However, some pathogens, prominent among them *P. infestans*, compensate for the susceptibility of their spores by being able to establish 'bridge-heads' of infection by means of a few spores finding suitable ecological niches in a field, and then spreading explosively due to their high multiplication rate. Where conditions permit this, cultural control has little chance of success and chemical control is imperative.

Splash dispersal is difficult to restrict if rain is the splashing agent. However, where overhead sprinkling causes the splashing, the shift from sprinkling to surface irrigation, or in orchards from sprinkling over the tree tops to sprinkling below them, reduces splash dispersal decisively. This has been demonstrated by Pappo (1965) for *Glomerella cingulata* (Stonem.) Spauld. & Schenk., the cause of bitter rot of apples, under the conditions of Israel's coastal plain. However, eliminating dispersal by splashing can somewhat reduce, but not eliminate, spread of pathogens which are also wind-borne.

Rate of penetration

We use the term 'rate of penetration' to mean the duration of free leaf moisture required by spores of an air-borne pathogen to complete penetration into the host tissue, at favourable temperatures.

The wet period needed for infection is specific to different species and may last from about 15 min in *Phytophthora palmivora* (Butl.) Butl. on papaya to 24 h in *Stemphylium botryosum* Wallr. f. sp *lycopersici* R. C. W. on tomato (for pertinent literature, see Rotem, 1978).

The cultural practices available for shortening wet periods are limited, and this is one of the reasons why air-borne diseases are hard to control by cultural means. The few available means include ventilation in protected crops, wider spacing in the field, escape from wet sites or seasons, and choice of irrigation techniques. The manipulation of irrigation for disease control under various meteorological conditions has been discussed by Rotem & Palti (1969). No matter which method is employed, prospects of control are always better against diseases caused by pathogens which require long rather than short periods of wetness, and that have sensitive spores which die if moisture close to their period of dispersal does not ensure infection.

Prospects of disease control by cultural practices

In the following we shall examine the question, how far economic and epidemiological considerations permit an a priori assessment of the prospects of controlling a certain crop disease by cultural practices.

The economic and human factors that have a bearing on the prospects of using cultural control measures successfully, are listed in Table 2, the pathogen and crop factors in Table 3, and the climatic factors in Table 4.

Table 2. Economic and human factors affecting prospects for cultural control of crop diseases.

	Prospects for cultural control	
	improve when	diminish when
Crop value and level of potential crop loss is	low	high
Cost of chemical control, relative to overall growing expenses, is	high (*e.g.* cereals)	low
Chances for regional planning of crops to minimize inoculum build-up are	good	bad
Choices of pre-sowing practices (soil, season, topography) are	numerous	few
Chances for manipulation of field conditions are	many (*e.g.* irrigated crops)	limited (*e.g.* dry farming)
Educational level of the farmer is	high	low

Table 2 includes some economic factors, such as a crop value and consequently level of potential loss due to disease, which are so over-riding in their importance that they may make any further consideration superfluous. An obvious case is that of flower

crops, the high value of which demands the quickest and safest measures available, and that generally means chemical control. Table 3 reiterates the direct effect of pathogen characteristics, especially those relating to inoculum build-up, dispersal and survival, on the results to be expected, and Table 4 defines those climatic factors which constitute a framework for control by cultural means.

The above tables show the great variety of factors to be considered and the complexity of cultural control problems. They also show that establishment of suitable epidemiological know-how is a prerequisite for assessing chances of succeeding with cultural control. As we have stressed, cultural measures – far more than chemical control measures – have to be adapted to the specific crop-pathogen-environment interactions to be expected in a given field.

The comparison of factors listed in Table 3 conforms to what Kranz (this volume) has tentatively termed the 'comprehensive approach' applied to a variety of pathogens and crops under a variety of cultural conditions, but under the same macroclimate. In Table

Table 3. Pathogen and crop factors affecting prospects for cultural control of crop diseases.

	Prospects for cultural control	
	improve when	diminish when
Pathogen		
Dispersal of inoculum is by	splashing	wind
The wetting period needed for infection is	long	short
The rate of inoculum build-up is	rapid	slow
The temperature range for development is	narrow	wide
Susceptibility of overseasoning or dispersal of inoculum to heat and drought is	high	low
Crop host		
Amount of susceptible tissue available at any one time is	limited	plentiful
Range of adaptibility to various growing conditions is	wide	narrow

Table 4. Environmental factors affecting prospects for cultural control of crop diseases.

	Prospects for cultural control	
	improve when	diminish when
Climatic conditions in general, in relation to optimal growth conditions, are	sub-optimal, at least in some seasons	approach the optimum
Changes in air masses are	frequent	infrequent
Annual distribution of rain is	lacking in uniformity	uniform
Occurrence of dry periods is	frequent	infrequent
Differences between minima and maxima of seasonal or daily temperatures are	wide	limited
Radiation is	high	low

4, on the other hand, the comparison is between given pathogen/host combinations developing in different climates. However, as also pointed out by Kranz in the above paper, such comparisons are often hard to verify experimentally: pathogen and crop factors can be simulated up to a point in growth chambers, but the simulation of overall climatic factors and of the overall effect of some cultural practices is so complicated that the results of relevant experimentation are sometimes of doubtful value. There is, nevertheless, reason to hope that further refinements of experimental procedures will make it possible to predict the success of cultural control measures in major crops much more accurately than at present.

In the foregoing we have attempted to show where disease control by cultural practices, which in the past has often been based mainly on empirical findings and cumulative experience, could profit from a systematic epidemiological approach.

We have focused mainly on characteristics of the pathogen and how these may interact with changes in environmental conditions induced by cultural practices. No less important are the characteristics of the host, such as nutritional and water stresses which have been mentioned only in passing in this paper. They merit closer study in the context of comparative epidemiology.

References

Avizohar-Hershenzon, Zehara & N. Hochberg, 1969. Effet de l'irrigation sur le développement du Plasmopara viticola en présence ou absence de fongicides. Ann. Phytopathologie 1: 55–56.

Baker, K. F. & R. J. Cook, 1970. Biological Control of Plant Pathogens. W. H. Freeman & Co., San Francisco, Calif., 433 pp.

Bashi, E. & J. Rotem, 1974. Adaptation of four pathogens to semi-arid habitats as conditioned by penetration rate and germinating spore survival. Phytopathology 64: 1035–1039.

Berger, R. D., 1977. Application of epidemiological principles to achieve plant disease control. A. Rev. Phytopathol. 15: 165–183.

Collins, E. B., 1978. Agricultural and forest practices that favor epidemics. In: J. C. Horsfall and E. B. Cowling (Ed.): Plant disease, Vol. 2. Academic Press, New York, p. 361–381.

Grainger, J., 1950. Forecasting outbreaks of potato blight in West Scotland. Trans. Br. mycol. Soc. 31: 45–53.

Katan, J., A. Greenberger, H. Alon & A. Grinstein, 1976. Solar heating by polyethylene mulching for the control of diseases caused by soil-borne pathogens. Phytopathology 66: 683–688.

Nagarajan, S., H. Singh, L. M. Joshi & E. E. Saari, 1977. Prediction of *Puccinia graminis* f. sp. *tritici* on wheat in India by trapping the uredospores in rain samples. Phytoparasitica 5: 104–108.

Palti, J. & J. Rotem, 1973. Epidemiological limitations to the forecasting of downy mildews and late blight in Israel. Phytoparasitica 2: 119–127.

Palti, J. & J. Rotem, 1980. Control of crop diseases by cultural practices. C.M.I. Pocketbook.

Pappo, S., 1965. [Apple bitter rot caused by Gloeosporium fructigenum (Berk.).] Ph.D. thesis, Hebrew University of Jerusalem, Israel, 134 pp. (Hebrew).

Pullman, G. S., J. E. DeVay, A. R. Weinhold & R. H. Garber, 1978. Control of soil-borne fungal pathogens by plastic trapping of soil. Abstracts of papers from the 3rd Int. Congr. of Pl. Pathol., Munchen, 16–23 August 1978, p. 373.

Rotem, J., 1968. Thermoxerophytic properties of *Alternaria porri* f. sp. solani. Phytopathology 58: 1284–1287.

Rotem, J., 1978. Climatic and weather influences on epidemics. In: J. G. Horsfall and E. B. Cowling (Ed.): Plant disease. Vol. 2. Academic Press, New York, p. 317–337.

Rotem, J & J. Palti., 1969. Irrigation and plant diseases. A. Rev. Phytopathol. 7: 267–288.
Rotem, J. & I. Reichert, 1964. Dew – a principal moisture factor enabling early blight epidemics in a semi-arid region of Israel. Pl. Dis. Reptr 48: 211–215.
Rowell, J. B. & R. W. Romig, 1966. Detection of uredospores of wheat rust in spring rains. Phytopathology 56: 807–811.
Stevens, R. B., 1960. Cultural practices in disease control. In: J. G. Horsfall & A. E. Dimond (Ed.): Plant pathology. Vol. 3, p. 357–429.
Vanderplank, J. P., 1963. Plant diseases: Epidemics and controls. Academic Press, New York.
Waggoner, P. E. & J. G. Horsfall, 1969. Epidem. Bull. Conn. Agric. Exp. Stn. 698.
Walker, J. C., 1957. Plant pathology. McGraw-Hill, New York, 819 pp.

Addresses of contributors

H. J. Aust	Tropeninstitut, Justus Liebig-Universität, Schottstrasse 2, 6300 Giessen, W. Germany.
E. Bashi	Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel.
D. J. Butt	East Malling Research Station, East Malling, Maidstone, Kent, ME19 6BJ, UK.
F. G. W. Jones	Rothamsted Experimental Station, Harpenden, Herts, AL5 2JQ, UK.
J. Kranz	Tropeninstitut, Justus Liebig-Universität, Schottstrasse 2, 6300 Giessen, W. Germany.
J. Palti	Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel.
C. A. J. Putter	Box 270, Howick, Republic of South Africa, 3290.
J. Rotem	Agricultural Research Organization, The Volcani Center, P.O. Box 6, Bet Dagan, Israel.
D. J. Royle	Wye College, Wye, Ashford, Kent TN25 5AH, UK.
R. D. Schein	Department of Plant Pathology, Pennsylvania, State University, University Park (Pa), USA.
J. M. Thresh	East Malling Research Station, East Malling, Maidstone, Kent, ME19 6BJ, UK.
J. C. Zadoks	Laboratory of Phytopathology, Agricultural University, P.O. Box 85, Wageningen, the Netherlands.

Index