AN EPIDEMIOLOGICAL ANALYSIS OF THE *PHYTOPHTHORA* AND *ALTERNARIA* BLIGHT PATHOSYSTEM IN THE NATAL MIDLANDS

by

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- DECLARATION -

I hereby certify that this research is the result of my own investigation.

C.A.J. PUTTER

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GENERAL INTRODUCTION

Ever since the notorious Irish late blight epidemic of 1845/46, which launched the science of plant pathology (Large, 1940), *Phytophthora infestans* has continued to be one of the most prominent and infamous of plant pathogens. The occurrence of the epidemic in Ireland was blamed on unusual weather and the study of the association between weather and disease became formally entrenched as an important aspect of plant pathology. Recent retrospective studies (Bourke, 1964) have shown, however, that the 1845 epidemic was not associated with weather unduly favourable for blight. Nevertheless, the eponymous description of cool and wet or misty weather as "blight weather" has endured.

Although no universal exact definition of "blight weather" exists, the concept has become firmly established. In the minds of laymen and researchers there is an intuitive unanimity about the deleterious effects on potatoes of cool and damp weather. This has been a particularly valuable belief in that it has given rise to more potato late blight disease prediction systems than for any other disease (Krause & Massie, 1975).

The type of weather pattern associated with late blight outbreaks was first definitively described in the Dutch rules (Löhnis, 1924; Van Everdingen, 1926). Later various modifications of these rules were used in other late blight warning systems (e.g. Beaumont, 1947; Large, 1952, 1956;

Bourke, 1953, 1955; Hyre, 1954, 1955; Wallin, Eide and Thurston, 1955; Hirst & Stedman, 1960). Various retrospective studies of late blight warning systems have been published (e.g. McKay, 1957; Boyd, 1974; Croxall & Smith, 1976) which confirm that the monitoring of weather phenomena is a fundamental ingredient of all late blight warning systems (Young, Prescott & Saari, 1978).

As new methods for measuring and interpreting critical weather periods became available, the forecasting systems evolved accordingly. The application of statistical tools, notably multiple regression analysis (Schrödter & Ullrich, 1965; Butt & Royle, 1974) and computer technology (Kranz, 1974a; Waggoner, 1974) has brought late blight forecasting to the high level of sophistication and immense practical value achieved, for example, in the "Blitecast" system (Krause *et al.*, 1975). Thus the evolution of blight forecasting systems conveys the distinct impression of being a purely technological progression, rather than a conceptual advance punctuated by the development of new epidemiological theory.

Both earlier and more recent literature on disease forecasting reflect a general approach to the problem that is dominated by studies of weather patterns, while the host X pathogen interaction is frequently relegated to a position of secondary importance. This is not surprising as several of the classical publications on disease forecasting bear the names of climatologists (e.g. Bourke, 1955, 1957).

This branch of plant pathological research has thus been largely dominated by weather men and their concepts and by plant pathologists who have a major interest in climatology.

While disease forecasting remains so umbilically dependent on weather it is unlikely that disease prediction, as opposed to forecasting (*sensu*: De Weille, 1964), can become a reality unless weather can be accurately predicted. The Dutch late blight forecasting system based on synoptic weather information (Post & Richel, 1951; Cox & Large, 1960; De Weille, 1964; Zadoks & Schein, 1979), aims to achieve such advance prediction. Bourke (1957) also employed synoptic weather data but not with the express purpose of disease prediction (Cox & Large, 1960). In general, however, long term weather prediction has not yet been adequately perfected and predictions beyond two or three days become progressively more unreliable.

With the high degree of sophistication of modern electronic weather measuring devices and the computer, the paradigm of the disease X weather interaction must surely by now have been exploited to the full. On the other hand the problem of disease monitoring has been sadly neglected. Such value judgements as those which an Irish peasant of the last century may have used, e.g. "lots, not so much and very little blight", have merely been given a more formal and quantitative basis, as for example, in the BMS key for late blight (Anon., 1947) and for other diseases in the keys summarized by James (1971). Disease measurement, as such, has attracted little attention and achieved minimal instrumental sophistication.

Blight ratings when applying the BMS key (Anon., 1947) are not usually made at intervals of less than a week. In contrast, changes in weather determining variables, such as temperature, can theoretically be measured at nanosecond intervals. Moreover, measurements of weather-determining variables can be made accurately to several decimal places, be collected by remote sensing and stored conveniently and indefinitely with the aid of micro-chip technology.

Since disease monitoring plays such an important rôle in disease simulation its neglect could be the reason why the simulation of plant disease epidemics has had such a floundering start (Vanderplank, 1975). Simulation remains of largely theoretical value, perhaps because it is based on a lop-sided technoloy in which abiotic variables are measured with micrometer-like precision and biotic variables with an infrequent, sledge-hammer degree of accuracy. To fill the gap between these extremes, simulators and the new generation of "computer pathologists" resort to intuition, value judgements and imaginative iterative procedures - euphemistically labelled the "black box" approach (Waggoner, 1974) - to construct elegant computer simulations which attempt to mimic reality.

One of the most fundamental requirements of epidemiology is the need to plot the amount of disease as a function of time (Vanderplank, 1963). Indeed, this is the methodological first step on which all quantitative epidemiology is based. Once this information can be graphed the mathematical manipulations which constitute the bulk of epidemiological theory can be carried out. Such disease monitoring is also funda-

mentally necessary for the development and validation of forecasting methods (Zadoks, 1972; Vanderplank, 1975).

Rotem, Cohen and Bashi (1978) have pleaded for more accurate measurement of biotic variables, especially if these are intended for simulation. They have argued persuasively that the demands of simulation and ecological interpretation of disease necessitate a qualitatively different experimental approach. Their concern is justified because the literature reveals a definite paucity of attempts to place disease first and to interpret disease patterns and incidence in terms of fundamental epidemiological paradigms.

In a classic and detailed study of potato blight epidemics Van der Zaag (1956) warned of the dangers of interpreting epidemic patterns only in terms of weather criteria. He listed four factors which influence late blight epidemics viz:

(i) Number of primary foci;

(ii) Extensiveness of the potato cropping;

(iii) Host susceptibility;

(iv) Micro- and macro-climate.

Other authors of this period, e.g. Hirst (1955) and Cox and Large (1960), discussed additional factors, such as host growth stage and source of inoculum, as being important late blight determinants. However, these considerations have not yet been combined into a single epidemiological approach, nor has the experience and astute observations of these

pioneers been developed to carry as much weight as did subsequently the school of weather-based concepts. Encouragingly, a few modern, but limited, exceptions to this generalization are to be found. For example, Croxall and Smith (1976) in England presenting blight progress curves for the period 1923-1974, commented that their information was collected with a view to finding additional (they imply "new"), epidemiological knowledge of late blight disease patterns. Trend-analyses of these disease progress curves revealed the important modifying influence of the terminal amount of disease in one season on the accuracy of weather-based forecasts in the following season. However, although they discuss inoculum and amount of disease they fail to offer an epidemiological typology which would enable the reader to formulate more than mere visual evaluation of the many S-curves for disease which they present. A more sophisticated approach, based on a greater number of biological considerations, is that of Dirks and Romig (1970) in the U.S.A. who developed a forecast system to warn of *Puccinia graminis* f. sp. *tritici* and *P. recondita* f. sp. *tritici*, the incitants of stem and leaf wheat rust, respectively. They evaluated five "biological" variables including three which directly measured pathogen activity, and six "climatological" variables, as a basis for their disease forecasting system. Butt and Royle (1974) discussed this example and also list two others in which biological variables are considered as important factors in disease forecasting.

Schrödter and Ullrich (1965) have expressed concern about a number of shortcomings in the various rules which had been advanced in the past to warn of attacks of potato late blight. Instead they proposed a multiple regression equation in which an attempt was made to synthesize, within a regression model, functionally-based expressions of the component events of disease multiplication. They measured spore germination and infection, sporulation and lesion expansion in addition to a few key meteorological variables. Vanderplank (1975) has described this publication by Schrödter and Ullrich (1965) as one of the milestones of plant pathology and their pioneering work appears to be the first definitive use of disease and pathogen data in late blight forecasting.

Whereas Croxall and Smith (1976) studied late blight at the level of the pathosystem, Schrödter and Ullrich studied late blight at sub-pathosystems levels in order to develop their forecasting system. They subsequently validated the predictions of their model by comparing their simulated disease progress curves with the progress of late blight in a natural potato crop. In many ways their work represents a refined application of Crosier's (1934) approach modified for the purposes of forecasting. This refinement owes much to developments in statistics and computer technology and it is significant that Vanderplank's (*loc. cit.*) praise of their work was for the introduction of multiple regression analysis to plant pathology rather than for fresh epidemiological insights.

The epidemiological approach to plant disease problems synthesized by Vanderplank (1963) has had a profound influence on several aspects of plant pathology. However, with certain noted exceptions, it would appear that these principles have not had a similar impact on disease forecasting. In the case of epidemic spread, for example, host susceptibility is at least as important as weather and mechanisms of dispersal. Yet, in such an authoritative text on weather and disease spread as that by Gregory (1973) it receives only cursory mention. Furthermore, one might argue that since one can do nothing to change weather, it is host susceptibility rather than weather which should receive the most attention.

This discussion should not be interpreted as a case against the use of weather variables. Instead, it should be viewed as arguing for an approach in which other disease determining components at the population level are also integrated into a coherent strategy based on sound epidemiological theory.

This thesis explores such an approach in a specific blight pathosystem (*sensu*: Robinson, 1976) in which *P. infestans* and *Alternaria solani* interact to form a disease complex. In the process, disease at the population level is confronted on a geographical basis and interpreted in terms of ecological and mesoclimatological criteria. Late blight forecasting is studied in terms of the peculiarities of cropping patterns in Natal which have led to the suggestion that

late blight follows a definite annual migratory route across the province. The competitive exclusion principle (*sensu*: Gause, 1934) as it occurs in the interaction between *P. infestans* and *A. solani* is also studied. These findings and other socio-economic factors are then integrated to generate a new epidemiological concept, the pathotope, which is explored as the basis for a new management strategy for the Natal blight pathosystem.

SECTION 1 PATHOSYSTEM DESCRIPTION

1.0.0 BLIGHT* IN NATAL

1.1.0 Introduction

Potato blight caused by *Phytophthora infestans*, was first reported in South Africa in 1890 (Blersch, 1890; cited by Gorter, 1973). The disease has been in Natal on potatoes and tomatoes since 1913 and 1922 respectively (Doidge, 1931; cited by Wager, 1940). The high rainfall and frequent occurrence in the province of periods during which drizzle and fog occur simultaneously means that potatoes and tomatoes are vulnerable to late blight attack. Potato late blight in particular has been extensively studied in the region with respect to its economic significance, chemical control and interaction with South African bred cultivars. In addition, investigations into the association between late blight incidence in the region and specific weather patterns has led to the establishment of a late blight forecasting system.

Certain theories have also evolved in terms of which late blight epidemic patterns are seen as being integrated with

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* Throughout this thesis, "blight" refers to both late blight, caused by (*Phytophthora infestans*) and early blight caused by (*Alternaria solani*). When only one of these diseases is referred to the adjectives "late" or "early" will be used to refer to *Phytophthora* - and *Alternaria* - blight, respectively. "Blight period", "blight forecast", "blight warning" or "blight prediction" will refer to the late blight disease only.

the ecological diversity and topographical features of the province. In particular, interpretation of epidemic patterns in terms of such mesoclimatological features gave rise to the *Phytophthora*-pathway theory, according to which late blight inoculum follows an annual migratory route from east to west, across the province. In the international literature there is no report or even indication of the existence of any other late blight pathway: the nearest parallel is the pathway postulated for *Puccinia graminis* f. sp. *tritici* in western North America (Stakman & Fletcher, 1930; Chester, 1946: Stakman & Harrar, 1957). Very little of the accumulated hypotheses concerning the behaviour of the Natal potato late blight pathosystem has been published or authoritatively summarized. Nevertheless, such information is crucial to an understanding of the topic of this thesis. Therefore, instead of an introductory literature review, an ethograph (*sensu*: Putter, 1980) describing the current status of the blight pathosystem in Natal and its management, will be presented. Several local anecdotes and theories supported by many years of observation by experienced and observant researchers and farmers will be documented. Much of the research associated with the inception and early history of the late blight research programmes was performed by B.W. Young who was for twenty years the government plant pathologist at the Cedara Research Institute of the Department of Agriculture and Fisheries. Although other researchers were closely involved, it was he who collected and collated the data, and who guided the theory of late blight migration to its

present popularity.

A major factor which prompted the present author to re-evaluate blight management strategies and their ecological matrix was the introduction of a new systemic fungicide for the control specifically of late blight. Several of the properties of this fungicide, metalaxyl (Ridomil), could have far-reaching consequences for blight control. If these consequences are to be understood it is necessary for the traditional blight control measures in Natal first to be explained.

1.2.0 The status and management of blight at the time of the introduction of the systemic fungicide Ridomil

Natal comprises a sub-tropical coastal region and a temperate inland plateau. A mistbelt occurs along the escarpment separating these two regions. In most of the province and especially in the mistbelt area, late blight occurs with annual regularity and is potentially the most important threat to potato and tomato growers. Early blight is also endemic and potato and tomato farmers have come to accept that control of these two blights is the minimum routine crop protection measure which they are obliged to undertake. In recent years both these diseases have been controlled by the repeated application of a single, dual-purpose, contact fungicide, e.g. mancozeb (Dithane M 45).

However, potato and tomato crops in Natal differ epidemio-

logically with respect to blight. In tomatoes early blight may attack young seedlings and juvenile plants. Usually such attacks preceed those of late blight. However, this is not the case in potatoes where late blight generally preceeds early blight. For this reason the potato spray programme commences when the first warning is issued by the late blight forecasting service operated from the Cedara Research Institute. This forecasting of disease is the traditional way of reducing costs by eliminating unnecessary sprays, thus optimizing fungicide use. After the first application spraying is repeated routinely at ten-to-fourteen-day intervals. The duration of the spray programme in potatoes is determined by disease severity and the particular practice employed by the farmer in preparing for harvesting. Where weeds are a problem, the farmers generally spray the crop with herbicides, thus destroying both haulm and weeds. However, where weed growth is unimportant, cessation of the fungicide spray programme shortly before harvesting allows the blight to increase and destroy the haulm.

This practice of deliberately allowing blight to increase on the senescing haulm is not employed in major potato growing areas of the world where it could lead to very high incidences of tuber blight. In Natal, conditions during the tuber bulking period apparently do not favour tuber infection by the late blight pathogen and incidence of tuber infection is considered to be unimportant. Young (1967) reported that in Natal, blighted tubers are not the source of

inoculum for the new epidemics as is the case elsewhere in the world. The implications of this will be discussed later, in detail.

Potato early blight generally is best able in Natal to exploit the weather conditions that occur during the latter half of the season, building up extensively on senescing haulms. Thus the "blight" on which farmers rely to kill their potato haulm after spraying is stopped, is mainly early blight. A major disadvantage of this practice - and one which has not elicited official concern - is the potentially undesirable build-up of large amounts of early blight inoculum which could result. Where there is an overlapping of cropping periods of either potatoes, or tomatoes and potatoes, the practice is potentially harmful and warrants investigation.

In the U.S.A. *A. solani* can infect tubers, thereby causing losses of up to 30%. In that country early blight is relatively unimportant as a foliar disease. Apparently it increases in the foliage until the inoculum reaches a threshold level beyond which both the incidence and severity of early blight tuber infection increase exponentially (Waggoner & Horsfall, 1969). With the introduction of Ridomil this may have important implications for early blight control in Natal, where early blight tuber infection has not yet been reported a problem. In the subsequent discussion of early blight control in Section 2.2.4.0 the case will therefore be made for considering the late and early blight

interaction as a single complex which demands an integrated dual-purpose management strategy.

Because late blight is potentially much more destructive than early blight, Natal potato farmers are generally referring to late blight when they speak of "the blight". Early blight is not usually considered to be a major disease and most potato farmers consider it to have a largely nuisance value and to be adequately controlled by their late blight spray programme. Indeed, because of its timely exacerbation of haulm death (*loc.cit.*) early blight is often viewed as a blessing in disguise. Early blight has a much more important rôle in tomato production because it defaces fruit and can destroy young seedlings due to stem infections. As it requires only a single spot to render a tomato fruit unsaleable, the economic importance of early blight in tomatoes can be disproportional to the amount of disease present in the foliage.

Ridomil, which systemically controls late blight, but has no effect on early blight, was introduced into Natal in 1978. Because of their assumption that early blight is not very damaging, or due to neglect, or in ignorance of the fact that Ridomil has no effect on early blight, several farmers using only Ridomil have suffered heavy losses due to early blight. Suddenly early blight started earning new respect among farmers, some of whom until then could not even differentiate between the two blight diseases. Using Ridomil they were forced to consider early blight separately

and to devise a separate management strategy for it.

The balance between the two pathogens which had hitherto co-existed in virtually the same niche, was considerably and differentially altered. It became necessary to understand more fully the nature of the complex formed by the interaction of late blight and early blight, and to determine the economic significance of early blight both in the presence and absence of late blight.

The author's concern for the possible rapid development of resistance to Ridomil in the Natal *P. infestans* population, was aroused due to Ridomil's selective, systemic properties. Subsequently, *in vivo* tolerance of *P. infestans* to Ridomil has been demonstrated (Staub *et al.*, 1979). The only way of minimizing the risk of developing tolerance to Ridomil would be to use the excellent properties of Ridomil in a strategy based on sound epidemiological principles. These concerns were not initially shared by the local distributors of Ridomil who were commercially euphoric at being able to offer the panaceae for the dreaded "plant destroyer" (*sensu*: Hickman, 1958). Consequently the first two or three years of the use of Ridomil caused as much confusion as it did delight.

Much of this thesis was initially inspired by the conviction that if Ridomil is to replace the existing dual-purpose fungicides then it could best be used by applying it in accordance with an improved forecasting service. Since late

blight weather is not optimal for early blight it may be argued that Ridomil should be sprayed when favourable weather for late blight demands it, and another fungicide when weather conditions favour early blight. Thus by separating the two blight diseases Ridomil not only provided the stimulus to re-evaluate the existing late blight forecasting system but also to study early blight control in order to optimize its treatment with fungicides.

1.3.0 Late blight forecasting in Natal

The first experiments aimed at developing a late blight forecasting system were initiated by Prof. Susarah J. Truter and B.W. Young in 1957. In subsequent years other researchers, notably W.G. Nevill and Pearl Scotney, also made significant contributions. However it was Young who remained responsible for the project until 1978 and because of this, the system is commonly referred to as the Young forecasting system.

Early exploratory experiments in the late blight forecasting programme consisted of serial plantings of potatoes in which the dates of first occurrence and the spread of late blight were diligently and regularly recorded. Thermohygrographs and maximum/minimum thermometers were maintained in these plantings with the intention of finding correlations between disease incidence and the weather criteria of relative humidity and temperature. These experiments were repeated over a period of three years at several stations in Natal. However, on many stations trained observers were unavailable

and this approach had to be abandoned because of the inaccuracy of weather recordings and the uncertainty as to whether farmers were reporting correctly, the dates of initial infections. Then followed a period during which serial potato plantings and the concomitant weather measurements were undertaken at Cedara only. Disease observations were still collected from field stations, but the forecasting system for the entire province became based on weather data recorded at a single weather station at Cedara. This made the Young forecasting system unique amongst potato late blight forecasting systems.

By 1960 tentative rules for a blight forecasting system had been developed. They were based on the critical period concept according to which a blight warning, i.e. a public notice advising farmers to commence spraying against late blight, is issued when three critical late blight-favourable periods have been recorded, usually in early summer. Such a critical blight period comprises an unbroken forty-eight hours during which air temperatures are between 10,0°C and 23,9°C and the relative humidity at both 1400 hr readings is greater than 72%, all readings to be taken in an A-class Stevenson's screen. The warning does not apply where irrigation is carried out and is only aimed at the summer potato growing areas of the Natal Midlands. Three blight forecasts are given per season, one for each of the early (August), mid-summer (October) and late (after November) plantings, respectively.

Unfortunately, the derivation of these rules as logical deductions from observed weather patterns, was apparently not documented or published. The rationale behind accepting the temperature ceiteria comes from the data of Crosier (1934) but it is not clear from the progress reports and the original thermohygrograph records from this research programme whether Crosier-type periods were in fact recorded and, secondly, whether such periods were correlated with disease appearance or severity.

It is also unclear why the particular value of r.h. 72% for two consecutive 1400 hr readings was chosen. A very similar forty-eight hour relative humidity requirement is also being used in Argentina (Calderoni, 1965) where it was only introduced after the Young system had been on trial for several years.

It would seem, therefore, that the Young system evolved from acceptance of the critical temperature values based on Crosier's (1934) criteria modified in the light of locally gained intuition and field experience, rather than from empirical experimental evidence. This is no reason to doubt the validity of the rules. Indeed this intuitive approach has traditionally been the basis of many forecasting systems, as for example, that in the Netherlands, which is based on the "Dutch rules" (De Weille, 1964).

Between 1960 and 1963 the Young system was tested in what was described as 'ghost' forecasts of blight appearance. Apparently field visits followed the ghost forecast to assess

its accuracy. This testing of the system was obviously *in lieu* of an observed versus predicted analysis which Vanderplank (1975) and Zadoks (1972) describe as a fundamental methodological necessity in the development of forecasting systems. In mitigation one should mention that such an analysis would have been extremely difficult, given the agro-ecological diversity of Natal. However, it should be attempted, especially as conditions at Cedara cannot be assumed to be representative of the Natal Midlands.

The first official blight warnings were issued in the 1964/65 season. Although Young has not published a definitive evaluation of the forecasting system he nevertheless considers it to be successful (Young, p.c.).

To the best of the author's knowledge only one experiment has been conducted specifically to evaluate the potential benefits of the Young forecasting system. Putter (1968) conducted an experiment at Cedara in which potato crops were sprayed either strictly according to the forecasting system or according to a routine preventative programme, initiated in accordance with the advice printed on the fungicide labels. In early, mid-season and late plantings it was found that spraying according to the forecasting service saved four, four and three spray applications, respectively. Blight control throughout the course of the experiment was excellent. Thus the Young system accurately forecast the occurrence of potato blight epidemics at Cedara, thereby also giving some credence to the assumption by its initiators that the system

could serve in other areas with climates similar to that of Cedara.

The rules of the Young-system have not always been strictly adhered to and certain blight warnings have been issued on near-critical periods while other near-critical periods have been ignored. This feature emphasizes the subjective quality in the Young-system. Young (1973) explained that this flexibility is possible "if the responsible officer has had experience in such a (forecasting) programme".

The extent to which the method is subjective increases the difficulty for the outsider to improve or modify the forecast system. Furthermore, optimum use of an expensive and potentially fragile (in the sense that it might lose its efficacy) fungicide such as Ridomil, is unlikely if the spray programme for the whole area is based only on Cedara data. Since the Young rules have been shown to be valid on Cedara, and assuming that they can be objectively standardised, every farmer should ideally have his own weather station as in the Blitecast system (Krause *et al.*, 1975). However, such sophisticated technology is not easily or cheaply available in South Africa. Unfortunately, despite the difficulties inherent in applying the Young-system as it is to the whole Natal potato growing area - difficulties acknowledged by Young (1973) - the department of Agriculture and Fisheries, the C.S.I.R.* and fungicide companies that were approached

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* C.S.I.R.: Council for Scientific and Industrial Research.

were unable to contribute research funds for meteorological equipment that would make possible improved forecasting, based on decentralised weather measurements.

Weather measurement is essential to the Young forecasting system which is ideally suited to a contact spray technology based on preventative applications. However, as mentioned in the General Introduction, there are now strong reasons for seeking to advance the study of disease, particularly disease interactions such as late and early blight, in terms of modern epidemiological laws and theories. Also on the basis of the law of diminishing returns, a new approach based largely on disease rather than on weather, might now be the profitable way to success.

It could be argued that a Ridomil orientated late blight spray programme for the whole region could simply be based on the Young-system. However, this would be erroneous because, as already discussed the system has important limitations which would require prior investigation, namely:

(i) The rules on which the system is based have not been tested on a large scale in an observed versus predicted analysis;

(ii) The rules cannot be defined objectively because they are based on a degree of intuitive knowledge and personal experience;

(iii) It is based exclusively on weather criteria and does not consider the modifying influence

of host factors or disease levels;

(iv) It does not apply to irrigated crops.

On the other hand, the postulated *Phytophthora*-pathway, by the very possibility of its existence, becomes equally important. This point is appreciated by the Department of Agriculture and Fisheries which has contributed considerable financial support to the research programme reported in this thesis.

The Young forecast system, suitably modified and improved, and the *Phytophthora*-pathway hypothesis are two of the important fundamental components accommodated by the new pathotope concept proposed and developed in detail in a later section of this thesis (Section 3.1.0.0).

Consideration will first be given to the *Phytophthora*-pathway concept that is firmly entrenched in the folklore of potato growers in Natal.

1.4.0 The *Phytophthora*-pathway hypothesis

Although the origins of the *Phytophthora*-pathway hypothesis are not clear, it apparently became popular in the early 'sixties and gained wide acceptance in the decade which followed.

Vanderplank (cited by Cox and Large, 1960) apparently first suggested the possibility that late blight on potatoes and

tomatoes occurring in the coastal regions of Natal might provide the inoculum for the summer crops of the main potato producing areas of the Transvaal highveld. To the best of the author's knowledge this is the only published reference to the possible existence of a late blight inoculum pathway in South Africa. Young first drew attention to the possible existence of the late blight pathway in Natal in an unpublished project report of the Department of Agriculture and Fisheries in 1975.

In view of the dearth of published information the grounds on which the pathway hypothesis is based have to be gleaned from verbal reports and speculation concerning changes in the spectrum of cultivated potato varieties, geographical production patterns and irrigation methods.

Prior to about 1960, South Africa imported large amounts of Scots Up-to-Date seed potatoes annually. Many of these tubers were found to be infected with *P. infestans* and were implicitly assumed to be the source of inoculum for late blight epidemics in South Africa (Vanderplank, cited by Cox and Large, 1960). Local seed potatoes were assumed to be free of *P. infestans* inoculum because the relatively high winter temperatures during storage compared to the U.K. for example, meant that infected tubers rotted in storage before they could be planted (Vanderplank, p.c.).

In the decade after 1960, the locally bred BP_1 cultivar, which is much more resistant to tuber-infection by *P.infestans*

than is Up-to-Date (Vanderplank, p.c.), rapidly displaced the latter. Consequently South Africa's imports of seed potatoes from Scotland showed a rapid decline and tuber blight became scarce to the point where local research workers considered it to have a negligible epidemiological rôle in the overwintering of *P. infestans*.

Intermittently, freshly harvested BP_1 potatoes are found to be infected with *P. infestans* in Natal (Hall, p.c.) but there is at present no specific evidence indicating that such infected tubers survive winter storage under Natal conditions. Neither are there records of healthy:infected tuber ratios determined *after storage*. Thus the possibility that *P. infestans* does survive cannot be ruled out although it appears to be unlikely.

Concurrent with the decline in the popularity of Up-to-Date, the areas devoted to out-of-season potato production increased in the warm frost-free areas of the country, such as in the Natal coastal region. Simultaneously, over-head irrigation methods displaced furrow and other flood-irrigation methods. It appears possible therefore that the movement of inoculum along the postulated pathway could have first become epidemiologically significant at the time of these changes in potato production patterns and then gained further momentum as the area under sprinkler irrigation in the frost-free regions increased.

The Natal version of the *Phytophthora*-pathway developed from

the observations made by local farmers, collated by B.W. Young. According to the pathway theory late blight moves annually from east to west across the province apparently following the river-valleys which form the basic framework of Natal's ruggcd topography. The topographical, ecological and climatological features of Natal constitute the meso-climatological matrix of the blight pathosystem and need to be understood in preparation for the remainder of this thesis.

In Natal, the near-alpine Drakensberg ('Berg) mountains, averaging in height about 3 000 m above sea level, form an escarpment which runs roughly parallel to, and about one hundred and fifty kilometers from, the coast. The land be tween the mountains and the coast is dissected by deep, parallel river valleys running in a west-east direction. These major rivers of Natal have their source high in the 'Berg and flow through temperate climatic regions to the coastal belt. The climate of the 'Berg and its influence on the weather of Natal has been studied by Tyson *et al.*, (1976). They give detailed explanations of the anabatic and katabatic wind systems of which the major features are illustrated in Figure 1. The slope, direction and geometry of secondary and tertiary valley-systems each has a modifying influence on the wind patterns. However, what is clear is that a regular, alternating wind system operates, which may disperse the pathogen. Such a dissemination mechanism is an essential ingredient of the *Phytophthora*-pathway theory.

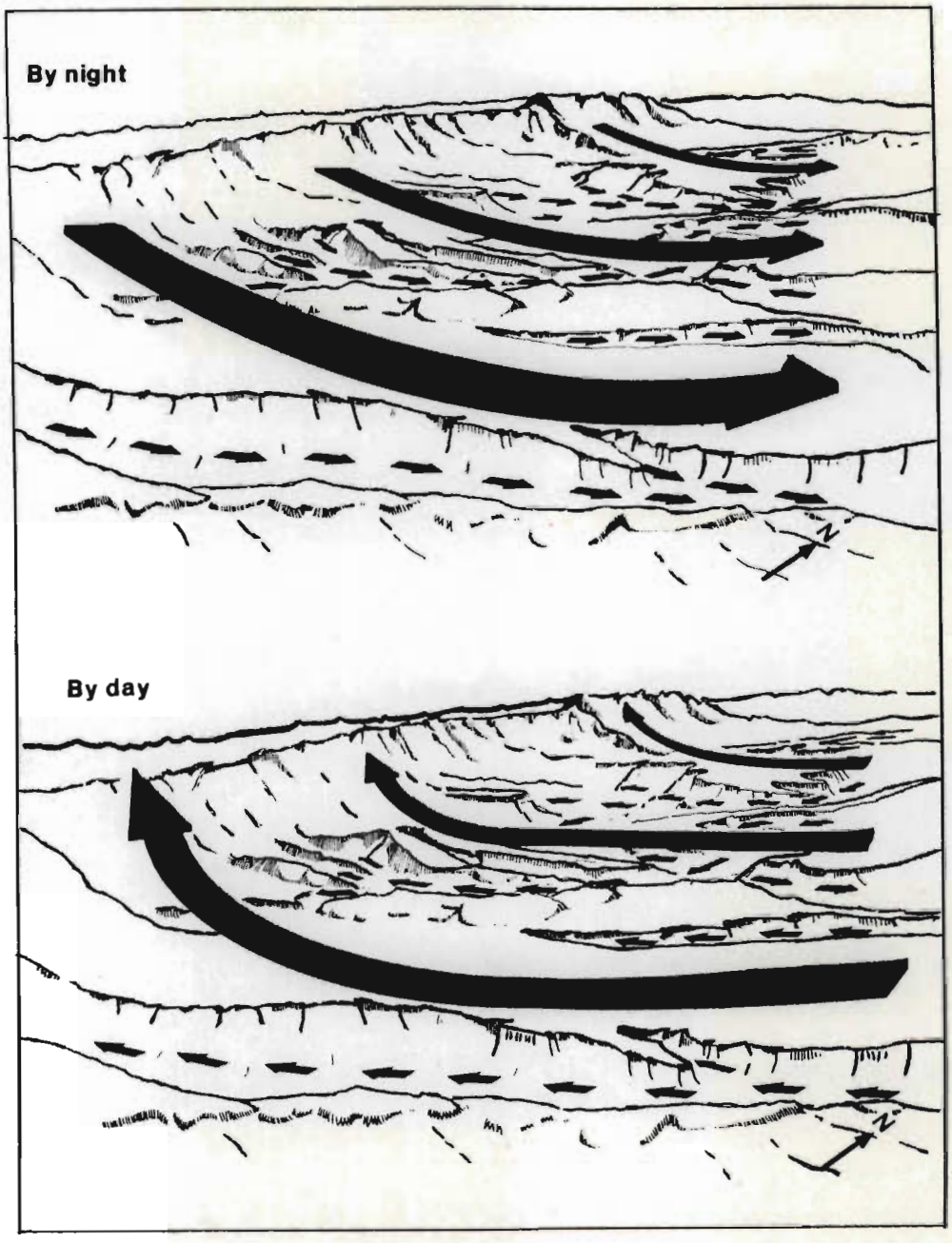

Fig. 1 Large-scale mountain-plain winds that constitute the "Minza" by night (katabatic wind), and the plain-mountain winds of the "Umzansi" by day (anabatic wind). Reproduced from Tyson *et al.*, 1976.

A further requirement of the pathway theory is a continuous supply of susceptible host tissue on a temporal gradient as in the *Puccinia*-pathway (*loc. cit.*). In Natal and also the neighbouring Eastern Cape and Eastern Transvaal, this requirement is met by the east-west climatic gradient which imposes an east-west temporal gradient on potato production. In addition, because of mild winter conditions in the coastal zone potatoes can be grown within this zone throughout the year. This latter feature does not apply in the major potato producing areas of the world, where summer production is terminated by a severe winter,too cold for potato growth. Continuous potato production is responsible for a very small percentage of the world potato crop and is found *inter alia* in Israel (Rotem and Cohen, 1974), New Zealand and parts of South America (Cox and Large, 1960). The potato cropping pattern in Natal, relative to certain altitudinal features, is illustrated in Figure 2.

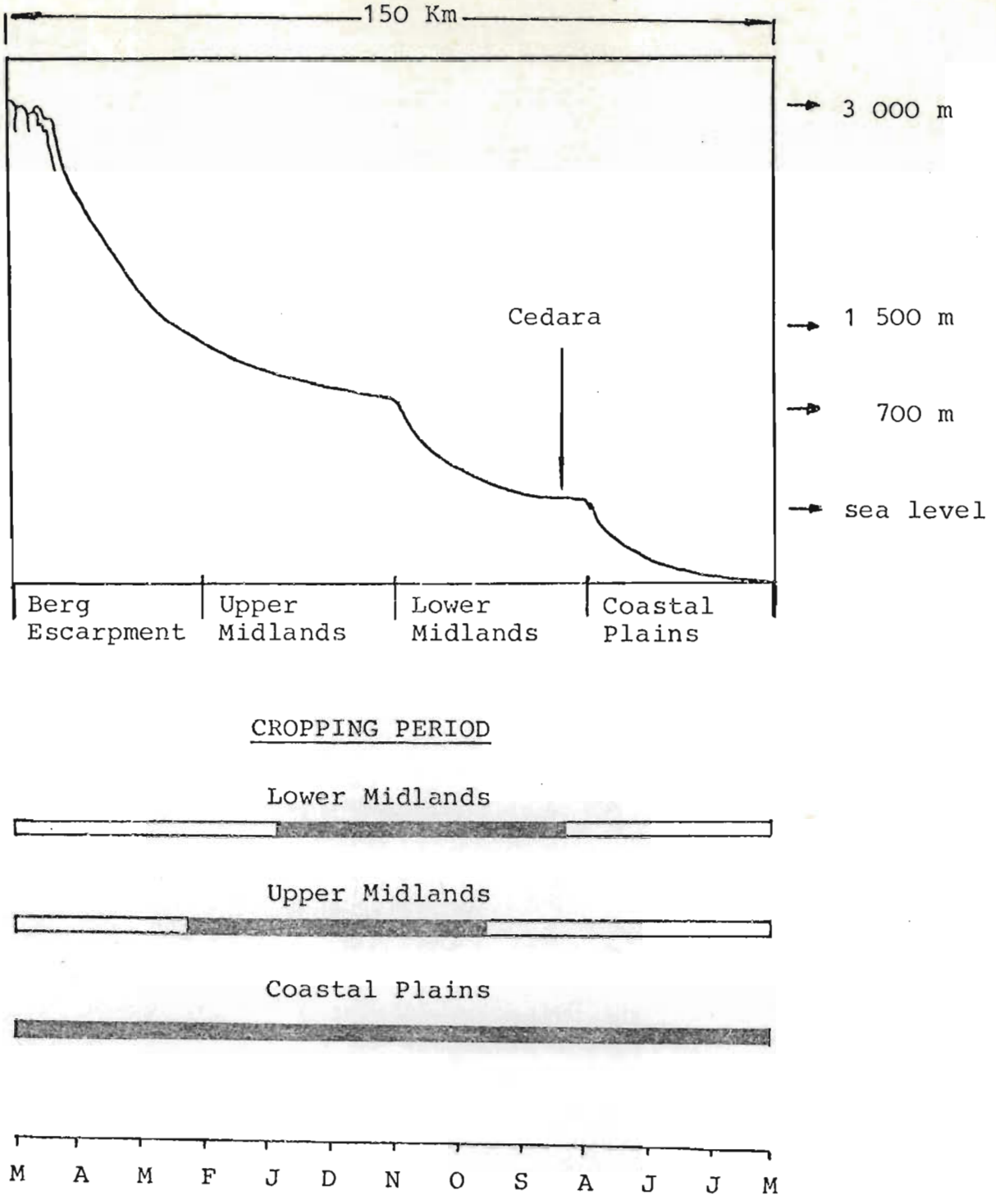

Fig. 2 Potato and tomato production patterns at different altitudes in Natal.

What has been said of potato production, applies also to tomatoes because in general, where potatoes can be grown, so can tomatoes. However, most of the tomatoes in Natal are produced in the midland and coastal regions with very little large scale production in the higher areas such as Underberg and the foothills of the 'Berg escarpment. Tomatoes are an alternative host of *P. infestans* (Vanderplank, cited by Cox and Large, 1960) and can therefore also act as a food source for the migrating population of *P. infestans*. There have been some reports in Natal that *P. infestans* from potatoes can infect tomatoes but that the pathogen cannot move back from tomatoes onto potatoes with equal ease. Cox and Large (1960) have pointed out that a similar belief was held in parts of the U.S.A. but it was later found to be due to a serial planting pattern which inhibited the movement of inoculum in one direction (tomatoes → potatoes) but not in the other (potatoes → tomatoes). Peterson (1947) and Driver (1957) have investigated the matter in terms of different races of *P. infestans*. Their cross-inoculation studies showed that tomatoes in no way inhibited or 'filtered' the virulence of *P. infestans* isolates. Thus if tomatoes are unable to alter the virulence (*sensu*: Vanderplank, 1963) of *P. infestans* isolates, they are even less likely to interfere with the aggressiveness of horizontal pathotypes such as might be found in Natal where the horizontal potato pathodeme BP_1 accounts for eighty-five per cent (85%) of the potatoes grown in the province.

The pattern in Natal whereby late blight is endemic and

causes sporadic epidemics (*sensu*: Putter, 1980) has important implications for the pathosystem's management. The definition of endemicity (*sensu*: Vanderplank, 1975) suggests that for endemic disease in general, overwintering inoculum might be expected to be less important than the continuous generation of inoculum from disease which is constantly or habitually present. Thus it is not surprising that in Natal late blighted tubers do not apparently contribute significantly to the perennation of *P. infestans* (Young, 1975). Indeed it is probably because Young (*loc. cit.*) could not demonstrate a rôle for tuber-borne inoculum in ensuring endemicity that he looked for an alternative source of blight. He regarded infected foliage to be the source of inoculum for other, more distant and as yet healthy foliage generated by the continuous cropping cycle.

Hirst (1958) described the problem of how the potato late blight fungus overwinters, as one of the classical problems in plant pathology. He pointed out that the most generally accepted explanation was that proposed by de Bary, viz. that the fungus perennates in diseased tubers which, when planted the next season, serve as the inoculum source for new epidemics. Blighted tubers, either in planted seed or in cull-piles near potato fields, are apparently the major source of inoculum in the other major potato growing areas of the world. The situation in Natal would therefore be unusual if Young's (*loc. cit.*) assertions are correct that tuber-borne inoculum plays no rôle.

Discussions of the *Phytophthora*-pathway have been dominated by considerations regarding the route and mechanism of dispersal. However, the absence of a rôle for tuber-borne inoculum raises the important question as to how continuity in the blight cycle is maintained. If, as is postulated, the blight migrates annually from east to west, how does it return? If, on the other hand, it does not return, what and where is the source of inoculum in the east which guarantees continuity of the pathogen?

In the absence of tuber-borne inoculum, there are two possible explanations which would both provide a mechanism for the required continuity.

Young (p.c.) suggested that the pathogen recycles in an ebb and flow pattern by hopping from one area to the next as the serial, sequential cropping pattern progresses in that direction. As the pathogen moves westward the host is harvested behind it so that ultimately when the westward journey is completed in April/May the eastward return journey of the pathogen must take place in the absence of commercial plantings of the host. According to Young (p.c.) an alternative source of host tissue which would enable the pathogen to survive the eastwards return journey could be provided by small potato gardens and the voluntary re-growth on old potato lands. Considering the isolated nature and small size of such host populations it could be argued that late blight on these plantings is unlikely to occur at levels significant enough to ensure a regular recycling of the

disease. It also seems unnecessary to invoke such an improbable event when the return journey could be completed in a single hop in the katabatic winds which regularly blow from west to east.

The night winds which blow from the 'Berg escarpment towards the coast (Fig. 1) do so until approximately 0800 hr (Tyson *et al.*, 1976). These winds could quite conceivably acquire and transport *P. infestans* sporangia which are produced more abundantly at the higher relative humidities and lower temperatures prevailing at night. A further factor which would mitigate against desiccation of the sporangia would be the fact that as much as 30% of total precipitation in certain sub-escarpment areas is due to fog interception (Tyson *et al.*, 1976). Nightly, fog-laden winds drawing from west to east across blighted potato fields would seem an ideal means of returning *P. infestans* to lower altitudes, especially if it is borne in mind that Bourke (1964) has shown in Europe that *P. infestans* is capable of 'jumping' distances of sixty kilometres.

Another version of the pathway theory was first proposed apparently, by Dr V.A. Wager, who was previously and for many years, plant pathologist of the Department of Agriculture and Fisheries, serving the coastal region of Natal. According to this theory, the eastern coastal plain acts as a permanent reservoir from which *P. infestans* sets out annually in spring on a unidirectional westward journey. The river valleys are seen therefore as dead-ends, and any

consideration of a return mechanism is considered to be irrelevant.

Both potatoes and tomatoes are grown all the year round in the coastal belt of Natal. For reasons already discussed it would seem safe to conclude that tomatoes can contribute late blight inoculum for the maintenance of a late blight epidemic pattern. This is consistent with Wager's hypothesis.

What has been said about a late blight pathway can conceivably apply *mutatis mutandis* to early blight. The mycelium of *A. solani* remains viable in dry, infected leaves for a year or more, and the conidia remain viable for as long as 17 months at room temperature. Additionally, overwintering may take place in infected plant debris and on tomato seed and in potato tubers (Walker, 1957). Therefore, if an *Alternaria*-pathway exists it would probably be obscured by within-field saprophytic perennation. The river-valley network and its concomitant, regular wind pattern may well prove to be the mesoclimatic framework also for many other pest and disease problems in Natal. On a global basis this climatic pattern, in which a warm ocean current causes sub-tropical conditions to occur only 150 km from a rugged escarpment with a near-alpine climate, appears to be fairly unique. The anabatic and katabatic wind system could daily provide a shuttle service for any pest or pathogen able to exploit either or both these climatic and ecological extremes and their intermediates.

The aphid vector of potato leaf roll virus, for example, could

conceivably accompany *P. infestans* on its west-bound journey to healthy potato fields. Similarly, the wind and valley network system which may form a potential pest and disease dissemination matrix could explain why the peanut rust pathogen *Puccinia arachidis* was seen in small peanut plantings in Pietermaritzburg within weeks of its first recorded presence in the country noted at Dundee some 200 kilometres away (Rijkenberg, p.c.).

1.5.0 Conclusions

The ethograph of the Natal blight pathosystems presented here seeks to present the fundamental ecological determinants to be considered in the search for an appropriate potato and tomato blight pathosystem management strategy which is the aim of this thesis. Any analysis of this pathosystem would be inappropriate without the Young forecasting system and the *Phytophthora*-pathway theory, superimposed on the mesoclimatic peculiarities of Natal, being perceived as the warp and weft of the blight pathosystem.

SECTION II PATHOSYSTEM ANALYSIS

2.1.0.0 DISEASE SURVEYS AND FIELD OBSERVATIONS

2.1.1.0 Introduction

The peculiar ecological complexity of blight in Natal may have important advantages for the management of this blight pathosystem. If the annual, apparent westward migration of potato late blight is indeed a manifestation annually of westward-moving inoculum, i.e. a *Phytophthora*-pathway, it should be feasible, knowing the position of the advancing late blight front, when the inoculum will move on, its destination and probable time of arrival, to apply fungicides to check its advance. In addition, if late blight warnings could be integrated with the late blight route, then warnings could be forecast in terms of the advancing disease front. The current system of three late blight warnings represents a modification of the forecast system to accommodate Natal's cropping pattern of three different potato planting dates. However, these warnings do not take into account the amount of disease in any specific area.

Thus, before the blight management strategy can be improved it must be established firstly, whether there is a *Phytophthora*-pathway based on inoculum movement and secondly, to what extent farmers can and do rely on the Young forecasting system in its present form. Also necessary is a thorough knowledge of the ecological framework of the pathosystem, the interaction of late blight and early blight,

and the socio-economic constraints imposed on the management of the system.

Young (*loc. cit.*) has pointed out that implementation of the forecasting rules for Natal requires a degree of intuitive knowledge in addition to empirical evidence. Young regarded the forecast system and the pathway hypothesis as inseparably integrated products of his accumulated experience of and familiarity with, late blight in the region.

In an attempt to acquire such an understanding of Natal potato and tomato production and the associated blights and their control, a programme of field visits, survey-questionaires and meetings with farmers was undertaken between January, 1978 and December, 1980. During 1978 the field trial reported in Section 2.2.0.0 was conducted and simultaneously, observations were made on the accuracy of the Young forecasting system. Farmers were requested via the press and radio to report late blight incidence. Responding farmers were visited and confirmed reports were then used to evaluate the accuracy of the Young forecasting system.

In May, 1979, a survey-questionaire was distributed to potato and tomato growers throughout Natal. Details of this survey are reported in Appendix I. During 1980, however, the study was focussed on the Cato Ridge-Tala Valley area east of Pietermaritzburg both because of financial constraints that precluded more extensive activity and also because of the crucial importance of establishing whether or not late

blight is endemic to this area, which could therefore constitute the "spring board" for a *Phytophthora*-pathway.

An extensive publicity campaign was maintained throughout this entire period. On numerous occasions the aim and importance of the research programme was explained to the press, in radio bulletins and interviews and at public meetings with farmers. In addition, ca. 12 000 km p.a. were travelled in visiting farms for the purpose of consultation and to inspect crops for blight*.

Unfortunately, due to limitations of funding, testing of the *Phytophthora*-pathway hypothesis and evaluation of the accuracy of the Young forecasting system were achieved to a limited extent only. However, a close working relationship was established with many farmers. The major achievement of the survey was the understanding gained of the potato and tomato production patterns and the associated blight problems. From this, in turn, emerged a new epidemiological concept the pathotope, described in Section 3.1.0.0 of the thesis.

2.1.2.0 Late blight movement and distribution

2.1.2.1 The origin of inoculum in relation to the *Phytophthora*-pathway

The postulated *Phytophthora*-pathway will be discussed with

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* This programme was partly funded by a research grant from

reference to the salient landmarks mapped in Fig. 2a.

The various versions of the pathway hypothesis agree that late blight migrates annually westward between September and March but disagree as to whether the initial inoculum arises from the frost-free irrigated winter potato growing areas of Cato Ridge, Umlaas Road, Eston and the Tala Valley or from the urban and peri-urban gardens of the coastal strip lying further to the east. Small scale intensive vegetable farms occur throughout this coastal strip which is about thirty (30) kilometres wide. Thus the initial step in evaluating the pathway theory was to try to establish whether late blight is present in either or both these areas throughout the year.

During much of the period of the survey severe drought conditions prevailed over large areas of Natal. Over an extensive area, little or no rain had fallen for several months at a time, rivers had dried up and many farming operations were checked by lack of water for irrigation. Therefore the reports reported here do not accurately reflect the late blight situation as it would prevail in more normal years. Neither was the survey distributed among the farmers in Kwa-Zulu because of the difficulty of establishing contact with the isolated peasant farmers in this region. As potato production in Kwa-Zulu increases, blighted crops in this area might be expected to become important components of the postulated pathway theory.

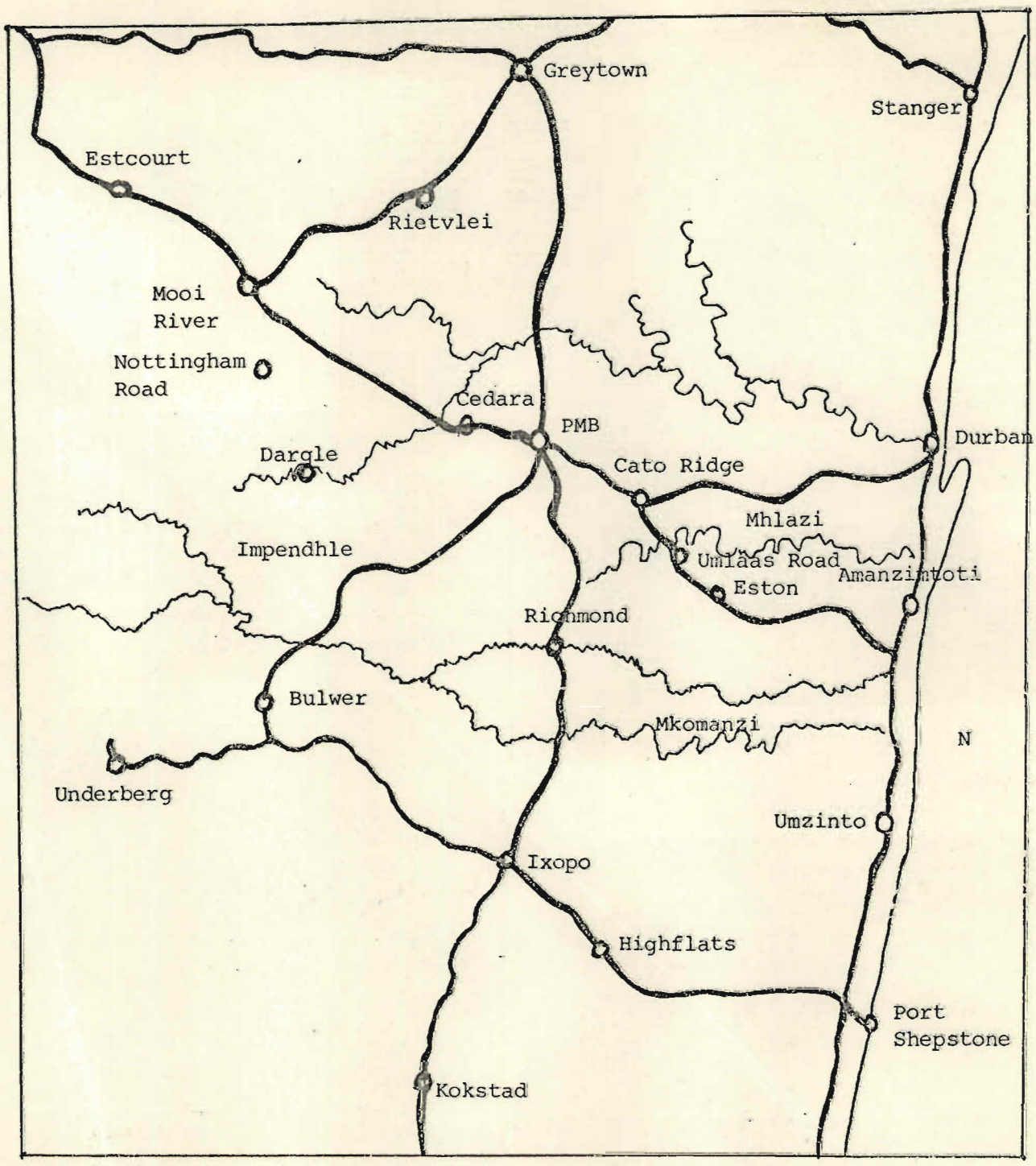


Fig. 2a Map of the potato and tomato growing areas discussed in this thesis.

2.1.2.2 Late blight in the coastal regions

The survey response from potato and tomato growers in the coastal strip was poor, notwithstanding the saturation publicity campaign, including an interview on an Indian radio magazine programme. Here late blight information was sought concerning the many gardens and smallholdings scattered throughout the area. Six reports of late blight in suburban vegetable gardens were received, of which only four could be confirmed. These reports indicated the presence of late blight in the greater Durban metropolitan area only in October and November of 1979 and April and May of 1980.

From slightly further inland the Indian market gardeners between Durban and Cato Ridge reported blight present sporadically from November, 1979 to April, 1980, on furrow-irrigated plots. Another similar source reported late blight in May and June, 1980, but this information could not be confirmed because contact could not be re-established with the farmer concerned.

The survey for continuity of late blight in the coastal region therefore cannot be considered as conclusive. If it is that late blight remained unreported, small-scale growers might either have regarded their planting as too small to be of importance, or they could not see any benefit for themselves in making the report. Alternatively, late blight was not recognised or was absent due to drought, or because it is not endemic to the area. However, Dr V.A. Wager's

(p.c.) observations during many years as extension plant pathologist in this region have led him to conclude that late blight is present in this coastal strip throughout the year.

2.1.2.3 Late blight in the Cato Ridge-Eston area

The area between Cato Ridge in the north, Baynesfield in the west, Tala Valley and Eston in the east and Mid-Illovo in the south, will be referred to as the Cato Ridge-Eston area. Situated on the fringe of the coastal zone it experiences only occasional light frosts. Here potatoes and tomatoes are grown fairly extensively during winter under irrigation, thereby producing these crops at a time when few other Natal farmers can do so. Because these crops are irrigated they are not included in the Young forecasting system which is issued only for non-irrigated crops west of, and at, Cedara. Falling as it does outside the area traditionally included in the forecasts, late blight in the Cato Ridge-Eston area has not been as well studied as at Cedara and areas further west.

The drought of 1979 continued into the autumn and winter of 1980 and many farmers believe this drought to have been the worst ever experienced in the Cato Ridge-Eston area. Apparently it was the first time in living memory that the Tala river had dried up completely. If late blight could be shown to survive during such a drought, then it could reasonably be assumed to do so in more favourable years.

To determine if this was the case an extensive publicity and survey campaign aimed at the farmers in the Cato Ridge-Eston area was launched in February,1980.

Between July, 1979 and February, 1980, i.e. from early spring to late autumn, regular reports of late blight were received and confirmed. Therefore, to ascertain whether or not late blight could survive throughout the year in this area,data on the presence of late blight was sought particularly for the autumn-winter period of February, 1980 to July, 1980.

The additional steps taken to obtain this information yielded unequivocal results. Ten confirmed late blight reports were received spanning the February-July period. These indicated that the disease, albeit at very low incidences, was present throughout the period. Two instances related to domestic vegetable gardens, whilst, significantly, in all cases the hosts (eight tomato crops and two potato crops) had received some overhead irrigation.

It may be deduced, therefore, that late blight survives throughout the year in this area, even if only at times at progeny to parent lesion ratios (Pr:Pa LR) of less than 1:1 (*sensu*: Vanderplank, 1975). Such low values of the Pr:Pa LR confer an ostensible effect of population immunity (*sensu*: Vanderplank, 1975) which in the absence of meticulous searching could lead to the erroneous conclusion that late blight disappears in the Cato Ridge-Eston area.

Amongst potato farmers in these four neighbouring potato growing areas there is talk of two "waves" of late blight each year; in June and in September. In confirmation of this D.V. Horton of the farm Cosmoore, Umlaas Road, has records collected on his farm over a period of 20 years, showing that the first major late blight attack in his irrigated winter potatoes consistently occurs between 15th and 30th June. In early autumn, i.e. prior to June, late blight is occasionally present at levels too low to warrant protective spraying. The rows of immature potato plants have at this stage not yet canopied and the microclimate in the foliage is probably therefore not conducive to epidemic development of late blight.

Mr. Horton's records further show that during July and August each year late blight incidence decreases markedly, increasing sharply again during September. J. Allsopp of Cato Ridge has also observed this trend of late blight reappearance in September. Thus there is longstanding evidence that the pathogen does not die out completely during February to August. Instead the Pr: Pa LR probably fluctuates between 2:2 and 2:1 thereby conferring the apparent population immunity mentioned earlier.

Strong evidence supporting this interpretation was noted on several farms in the Tala Valley. Mr. Steenkamp, who had an overlapping, irrigated tomato cropping pattern running from January, 1980 to August, 1980, found it necessary to use fungicides throughout this period to keep late blight in

check. In this instance, despite the extreme drought, irrigation was possible from a private dam. On each of three occasions when the author visited this farm in May, June and July,1980, the presence of late blight was readily confirmed. In July, 1979 the author found late blight on volunteer potatoes in an irrigated, newly planted maize field at Umlaas Road in July, 1980. In August, 1980 late blight was reported from Baynesfield Estates on their irrigated tomatoes and on T.L.B. Hilliar's irrigated potato crop in the Tala Valley.

Thus, in less severe years, when overhead irrigation is routinely practised on a large scale,late blight could develop and persist on many more farms. This may elevate the *P. infestans* spore load in the atmosphere to levels which could be considerably higher than during dry seasons such as 1980.

On all visits to farms in the Cato Ridge-Eston area since 1978, some early blight was found. In many instances the reason for the visit was to detect late blight and although frequently late blight was absent, early blight was always present. Volunteer potato plants - a serious weed problem in maize crops following potatoes - were a reliable, continuous source of early blight. Abandoned, senescing tomato crops also showed very high levels of early blight.

There is thus good evidence that in the Cato Ridge-Eston area, both late blight and early blight are endemic. Because of

its proximity, this area is also most likely to be able to contribute inoculum to Cedara and possibly the inland potato producing areas. Thus in terms of the reservoir analogy of Wager's version of the pathway (*loc. cit.*) there is no apparent reason to doubt that if there is indeed a *Phytophthora*-pathway, the Cato Ridge-Eston area could fulfil this function, serving as the annual springboard of west-bound *P. infestans* inoculum. It is possible that the coastal strip provided the springboard before irrigation and the concomitant winter potato and tomato production became extensive in the Cato Ridge area but under present cropping patterns, the coastal region does not appear to contribute significantly to blight in the inland areas.

2.1.2.4 First *P. infestans* migratory hop : Cedara

The nearest potato growing area to Cato Ridge along the route of the postulated late blight pathway is that around Cedara. This area experiences severe frosts between May and August making it impossible to grow potatoes. Here the first late blight critical weather period is generally recorded in late September or early October. Based on the average of annual observations over twenty years, the third late blight critical weather period occurs, and thus the first late blight warning is issued, between 13th October and 2nd December, with maximum probability occurring in the period 23rd October to 2nd November. Traditionally crops in this area are not irrigated although increasingly, some supplementary irrigation is practised.

A tenable scenario would consist of the following steps. Late blight inoculum builds up initially to high levels during September in the Cato Ridge-Eston area. Such a late blight build-up is feasible because at this time the potato crops in this area are senescing and approaching harvest-time, which is usually between late September and October. Irrigation during this critical tuber-bulking period (Rutherford, 1977) is strictly maintained and both host and environmental conditions are ideally favourable to *P. infestans*. As inoculum concentration increases in this area, despite spraying, so too do the chances of the pathogen successfully completing a wind-mediated hop to the Cedara area. This hop could be accomplished during any of the first three blight periods recorded at Cedara. However, it is more likely to occur during either the first or second, because on an average, the Cato Ridge-Eston crops are harvested before the third late blight favourable weather period is recorded at Cedara.

Thus it is possible that a late blight focus, or several isolated foci, could be established in the Cedara region during either the first or second blight period. By the time the third blight period is measured at Cedara, inoculum levels in the foci have built up sufficiently for the transition from focal epidemic to general epidemic, thereby necessitating the issue of a general blight warning for the early summer plantings.

When a fourth blight period is recorded at Cedara, a second

warning is issued,on this occasion for spraying of mid-summer plantings to commence in regions to the west of Cedara. The third and final blight warning is issued to initiate spraying of the late potato plantings in the western areas of Bulwer and Underberg, immediately the fifth blight period occurs at Cedara. Thus there is a definite temporal gradient to the west of Cedara along which late blight is observed as summer progresses.

2.1.2.5 Second migratory hop: Mooi River

At meetings and in the returned survey forms,many farmers in the Mooi River area, which lies immediately to the west of Cedara, reported that late blight is first seen in potato crops on average 18 days after the first blight warning is issued from Cedara.

Inspection of the late blight weather records at Cedara for the period 1957-1979 reveals that the fourth blight period is recorded at Cedara on average,18 days after the third blight period. The fact that the Mooi River farmers observe late blight to be generally present at the time when the fourth Cedara blight period is recorded, suggests that if the inoculum for Mooi River blight is indeed provided by blighted foliage in the Cedara area, then the inoculum probably moved from Cedara to Mooi River during the second or third Cedara blight period which may only be the first and second blight periods,respectively, in Mooi River. This suggests that the meteorological readings at Cedara, are also representative

of conditions at Mooi River although specific meteorological data is at present unavailable to confirm this. These data also incidentally support the assumption that a warning issued from Cedara by the Young forecasting system is accurate for the Mooi River area, provided it is a warning issued after the fourth blight period is recorded at Cedara.

Some of the Mooi River potato crops are only ca. 35 km from Cedara and *P. infestans* inoculum produced abundantly during the third Cedara blight period could conceivably migrate to Mooi River at this time and increase there in foci prior to a general epidemic outbreak which coincides with the recording of the fourth Cedara blight period. Therefore, although these data for Mooi River blight outbreaks relative to Cedara-based weather measurements do not constitute proof for the existence of the pathway, neither do they contradict the assumption of its validity.

2.1.2.6 Additional possible *P. infestans* migrations

In the Impendhle-Bulwer area, late blight apparently also appears later than it does at Cedara. However, no long term trends regarding this delay could be concluded from the survey reports. In 1978 and 1979 late blight was observed in this area at Ivanhoe Farming Co., 22 days and 32 days, respectively, after it had been observed at Cedara. In general, Ivanhoe potatoes are planted four weeks later than the early Cedara plantings although there are some notable exceptions to this. It is therefore not possible on the

basis of survey data only, to decide whether Cedara contributes inoculum to the Impendhle-Bulwer area as it may do to the Mooi River area.

Underberg plantings represent the western extreme of the postulated *Phytophthora*-pathway. Here potatoes are planted in mid- to late summer and the crops become vulnerable to late blight attack much later than the early planted potatoes at Cedara. One might speculate therefore that the *Phytophthora*-pathway follows the route of the solid red arrow in Fig. 3. However, the possibility of late blight reaching the Impendhle-Bulwer area via Richmond cannot be excluded. Indeed, in many respects this is an equally feasible alternative in terms of the postulated valley-wind dissemination mechanism because of the river-valleys which connect these two areas.

The Richmond area is fairly variable with respect to planting times and late blight appearance, judging by the survey returns. Very early to late plantings occur and some crops are irrigated. Data from the remaining areas, viz. Greytown, Ixopo, and Kokstad could not be collected with the same detail and degree of accuracy due to financial limitations. Their possible relationship to the pathway was therefore not studied.

2.1.2.7 Other explanations for the *Phytophthora*-pathway

The central problem which the survey failed to resolve is whether the pathway effect is a real consequence of physical

migration of *P. infestans* inoculum or merely reflects the triggering of pre-existing inoculum sources along the environmental gradient.

For example, the appearance of late blight in the Mooi River area could be due to events not necessarily related to the physical arrival of inoculum from Cedara. The Mooi River area could possibly experience blight periods independently of, or qualitatively different from, those recorded at Cedara. This has not yet been tested although it is presently assumed that Cedara blight weather is similar to and occurs at the same time as Mooi River blight weather. Thus there might exist local sources of inoculum, for example in planted tubers of isolated cull piles, which could initiate focal late blight epidemics in the Mooi River area. Both the source of inoculum and the weather favouring late blight in the Mooi River area may be independent of events in Cedara, it being merely fortuitous that the fourth blight period recorded at Cedara coincides with the transition from the focal to the general epidemic phase in the Mooi River area. It must be borne in mind, that the requirement of three blight periods has not been experimentally validated for any region other than Cedara.

Since Mooi River planting dates are usually two to four weeks later than the early planting dates in the Cedara area, the fourth Cedara blight period probably coincides fairly closely with the attainment at Mooi River of full-canopy and the concommitant onset of late blight favourable micro-

climatic environment in the crops. Hirst (1953), Cox and Large (1960) and Vanderplank (1963) have shown that in potato late blight the transition from focal to general epidemic usually coincides with full-canopy stage and its related changes in micro-climate can be expected to be significantly buffered against unfavourable changes in macroclimate

Whatever the explanation for the coincidence between the fourth Cedara late blight period and the appearance of late blight in the Mooi River area, the twenty year data confirming this correlation must clearly be seriously considered when developing a late blight management strategy for the area.

However, given the existence of a potentially suitable dissemination mechanism linking areas along the proposed pathway route, also a total absence of evidence of infected tubers as sources of initial inoculum and, finally, the serial nature of late blight occurrence between Cato Ridge and Mooi River, the pathway hypothesis is at least as acceptable as the alternatives offered above.

2.1.2.8 Circumstantial evidence favouring *P. infestans* inoculum migration rather than inoculum arising from blighted seed tubers

Evidence obtained at two late blight outbreaks in the Cedara area are seen as providing circumstantial evidence against accepting an epidemiologically significant rôle for

tuber-borne inoculum. Furthermore these observations also support the pathway hypothesis which postulates that *P. infestans* inoculum migrates in a east-west direction in Natal. These will therefore be discussed in detail because Cedara experiences severe frosts so that living host material is absent throughout the winter and inoculum must be regenerated each spring. The first outbreak to be discussed occurred in the late blight spray trial laid down as part of this thesis study (see Section 2.2.0.0). This experiment was planted in September and did not receive irrigation.

Immediately after the first blight warning had been issued from Cedara on 18/10/78, the blight spray trial was inspected for late blight. On a single plant in an unsprayed plot ten late blight leaf lesions were counted between ground level and 15 centimetres up the stem, and five lesions between 15 and 30 centimetres. No lesions occurred in that part of the canopy above 30 centimetres from the ground. Judged by the size and number of the sporulation bands, the lesions were of at least three different ages, the older lesions being lower in the canopy.

There are three possible explanations for the distribution of symptoms as described above on the first infected plant.

Firstly, the disease might have been slowly increasing on the plant, possibly from a single lesion produced by inoculum derived from a distant source not on Cedara but

from a more easterly source consistent with the pathway hypothesis. Progeny lesion development would have increased during the favourable weather of the second and third late blight periods bringing the disease to an observable level. Such a pattern is consistent with the initial "cryptic" development of late blight epidemics reported by Hirst (1955). When the first infected plant was found, late blight could not be found in the rest of the experimental area. The second blight locus was observed six days after the first observation in another of the unsprayed control plots and the infected plant discussed here was undoubtedly the point of origin for disease spread in the experimental area.

Alternatively, the first infected plant might have resulted from inoculum minimally present in its own tuber. This was contra-indicated two weeks later when the tuber was examined and found to be intact without symptoms of late blight infection.

A second possible inoculum source within the area could conceivably have been late blight infected cull-piles. However, in South Africa, and especially at Cedara where potatoes are carefully harvested, sorted and weighed, potato cull-piles are exceedingly uncommon. Most of the rejected potatoes are removed by the labourers for planting or consumption after damaged or rotten portions have been removed. The author deliberately maintained a cull-pile for three seasons on his own potato farm. All rotten or rejected

potatoes from a three hectare seed potato crop on which late blight had occurred, were placed in a shallow hole, one metre deep and six metres square. Each spring a dense mat of potato foliage developed and quickly created an ideal situation for late blight to develop from any infected tubers present. However, the cull-pile (more correctly the "cull-pit") did not give rise to diseased plants originating from tubers in the pit. In all three seasons the plants only showed late blight symptoms after these had been found in the adjacent fields.

Whilst this finding accords with the accumulated experience that late blight inoculum apparently does not readily survive in the form of blighted tubers it does not contradict the possibility of a very low level of tuber transmission under field conditions where rotting and decay by secondary organisms would not destroy an infected mother tuber as rapidly as in the pit situation reported here.

In Maine, U.S.A. where cull-piles have been eliminated (Bonde and Shultz, 1943; Cox and Large, 1960) late blight epidemics occur nevertheless. Vanderplank (1963) has explained how difficult absolute sanitation would be in the case of late blight. He has shown that minute amounts of *P. infestans* inoculum could initiate severe epidemics. Thus although cull-piles are not a feature of potato harvesting practices in Natal, inoculum arising from diseased tubers cannot be absolutely discounted.

Late blighted seed tubers are unlikely to escape detection in the rigorous sorting process undertaken by growers of Government certified seed potatoes. The repeated manual sorting,to which seed potatoes are subjected would sooner or later reveal the rare presence of blighted tubers. Furthermore, winter seed storage in Natal takes place without artificial cooling and therefore at temperatures higher than, for example, in Europe. In early spring, the increasingly warm temperatures would be expected to cause the majority of late blight infected tubers to rot rapidly due to secondary infections. Consequently in Natal at the time of planting, individual blighted tubers would, because of secondary rotting, tend to be more conspicuous and therefore more likely to be discarded than would be the case in Europe. The invariable manual use of sorting and planting methods also means that the tubers are individually handled so that at the time of planting, fewer diseased tubers would be planted than might be the case with bulk mechanical planting used in Europe and America.

Van der Zaag (1956) estimated that in Holland about 100 planted,diseased tubers are required to produce a single focus and that these could be expected to be distributed in approximately 1 600 kilograms of seed potatoes. Assuming an average of 75 grams per tuber the required hundred tubers would weigh 7,5 kg. If these diseased tubers were randomly distributed, the ratio of the number of diseased to healthy tubers would be 7,5:1 600 i.e. 1:213. Low ratios such as these could easily lead to infected tubers going undetected,

especially those showing mild symptoms only. Thus the evidence presented here cannot be construed as absolute proof that planted blighted tubers do not contribute to late blight epidemics in Natal.

The second outbreak in the Cedara area meriting discussion occurred in September, 1978. On 10/9/78, 38 days before the official late blight warning had been issued, a heavy infestation of late blight was reported on the farm "Ingalankulu", belonging to J. Teichman. This 10 hectare crop of irrigated potatoes, approximately 20 kilometres north of Cedara, was infected with late blight 10 days before the *first* of the compulsory three blight periods required for a blight warning, was observed on Cedara on 20/9/78. Thus it appears that irrigation elicits a late blight attack in spite of the fact that weather in general and in the Cedara area in particular, was unfavourable for late blight.

The fact that the Young-system specifically precludes irrigated crops, is an acknowledgement that irrigation may render crops vulnerable to late blight attack contrary to what might be expected in terms of general weather. Also relevant here are the data already reported for the Cato Ridge area during the severe drought in 1980, i.e. that late blight soon became apparent where irrigation could be practised in spite of the fact that the weather generally did not favour late blight development.

In Mr. Teichman's case, there are three possible explanations

for this irrigation elicited blight outbreak, i.e.

(i) Inoculum was provided by nearby blighted crops;

(ii) It derived from a within-field source, or;

(iii) It arrived from a more distant source.

The first possibility could be discounted with reasonable certainty because there were only eight other potato farms within a radius of 25 km, none of them infected with late blight. On three of the farms supplementary irrigation had been applied viz. the farms of Messrs. R. De La Hey, K. Todd and P. Train. The crops of the former two were planted in the same week whilst Mr. Train's crop was planted three weeks after that of Mr. Teichman. The other five were dryland crops on the farms of Messrs. I. Shaw, R. Williams, A. Putter, M. Hunt and L.Lund. None of these farmers found it necessary to spray against late blight before the first late blight warning was issued from Cedara on 18/10/78.

Within-field sources of inoculum arising from alternative weed hosts and saprophytic survival of *P. infestans* in the soil, could also be discounted because Teichman's farm had been under climax redgrass pasture (*Themeda triandra*) for at least 80 years and moreover, contained no solanaceaous weeds. By a process of elimination, inoculum arising from a diseased tuber was the only possible within-field source of inoculum the presence of which, however, cannot either be discounted or proved.

Inoculum arriving from a more distant source could also conceivably have been responsible for this isolated blight outbreak. The distance separating Teichman's farm from the Cato Ridge-Umlaas Road-Eston winter potato-growing area is ca. 35 km. The wind system depicted in Fig. 1 consisting of daily winds as illustrated for example in Fig. 3 below, could have provided the means to disperse inoculum from Umlaas Road to the Karkloof valley where Teichman's crop was situated.

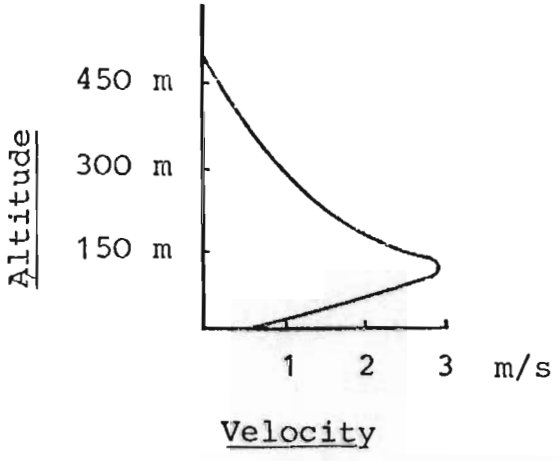


Fig. 3 Anabatic wind profile showing wind speed at different altitudes above the valley floor. (Reproduced from Tyson *et al.*, 1976).

Although the wind profile in Fig. 3 is for May, the example, according to Tyson *et al.*, (1976), is typical of the situation prevailing throughout the year. The maximum velocity of these anabatic winds is reached soon after midday. This is one to two hours after the time when the maximum number of sporangia can be expected to be released into the atmosphere above a late blight infected field (Hirst, 1953). At a wind velocity of three metres per second this inoculum would take approximately three hours to move from Umlaas Road to the Cedara area where Teichman's crop was situated.

If, as postulated for the first migratory hop of *P. infestans*, inoculum arising at Cato Ridge-Eston is carried to the Cedara area where successful infection occurs during favourable weather, the destination in the Cedara region of the migrating inoculum may have been Teichman's crop rather than the unirrigated crops at the Cedara research station. The other irrigated crops of Messrs. De La Hey, Todd and Train could just as easily have served as the site for the establishment of the first late blight focus in the Cedara region. Implicit in the definition of the focal epidemic stage, is the assumption that there are few diseased farms against a background of healthy farms (*sensu*: Vanderplank, 1963). Thus it is not necessary for the focus to be established on several farms simultaneously nor is it assumed that inoculum will be dispersed to the same site every year.

Although the two weeks preceeding the late blight outbreak had been dry and sunny,Teichman's irrigation may have coincided with high levels of late blight in the irrigated, mature potato crops in the Cato Ridge-Eston area. It should be noted that irrigation at Umlaas Road is not a prerequisite for sporangial release. Hirst's (1953) data reflects release unaided by wind or splash. Indeed, Gregory (1973) has shown that only at velocities above 3,9 m/s does wind enhance the natural release of *P. infestans* sporangia. Although sporulation of *P. infestans* is more rapid on leaves atomized with fine droplets than on leaves kept at 98% relative humidity or on leaves covered excessively with big drops of water (Rotem, Cohen and Bashi, 1978). Henderson,

(1952) showed that water droplets 90 µm in diameter have a life expectancy of only one second in air at 50% relative humidity. Thus it is unlikely that sporangia will be dispersed in small water droplets, indeed, Gregory (1973) concluded that long distance *P. infestans* dispersal is not critically dependent on wind-blown rain.

However, germination and penetration of *P. infestans* have long been known to depend on free water or dew on the leaves (Crosier, 1934). Therefore, the irrigation practised by Teichman during the day could well have provided the outstanding component to complete a cycle commencing with dry-liberation of sporangia, peaking at ca. 1100hrs, at Umlaas Road, followed by dispersal by anabatic winds and completed by deposition three to four hours later in Teichman's irrigated crop in the late afternoon, where successful germination and infection could be initiated and completed during the night when temperature and relative humidity were favourable.

Neither of the two mechanisms proposed here in explanation of Teichman's late blight outbreak, viz. tuber-borne inoculum or inoculum migration, can be proved by the application of the *ceterus parabus* approach. However, the liklihood of blighted tubers rotting under the temperature conditions in Natal before they can be planted (*loc. cit.*) mitigates against a rôle by tuber-borne inoculum. Clearly, inoculum for the late blight outbreak in the spray trial described above, did not come from an infected tuber. In

contrast the circumstantial evidence that the inoculum arose from a distant, easterly source, as postulated in the pathway theory is strong.

2.1.3.0 Note on the accuracy of the Young forecasting system

During both the 1978/79 and 1979/80 seasons unusually severe droughts in Natal resulted in less late blight development than is the case in normal years. Because of the drought, many more potato farmers than usual applied supplementary irrigation and therefore could not base the timing of their first late blight spray on the Young forecasting system. This combination of drought and unusual irrigation practices meant also that these years were not suitable for a large scale assessment of the validity of the forecasting system. Where farmers indicated that they had applied their first spray following the warning, it could not be established whether late blight had indeed been present. Thus although several farmers did rely on the late blight warning service, none actually checked to see whether it was accurate or not. However, there were no specific complaints of inaccurate warnings, which perhaps can be construed as limited evidence confirming the validity of the Young forecasting system.

2.1.4.0 Conclusions

(i) Late blight appears to be endemic in the Cato Ridge x Umlaas Road x Tala Valley area.

(ii) There is limited evidence to support the *Phytophthora*-pathway theory inasmuch as it represents a spatial and temporal gradient of observations of late blight presence in the areas around Cato Ridge, Cedara and Mooi River.

(iii) In the Cedara region, the Young forecasting system accurately predicted the transition from the focal to the general epidemic stage, during the period of this study.

(iv) Although late blight on seed potato tubers could not be ruled out as a source of inoculum for new epidemics it is unlikely to be a major factor in the postulated *Phytophthora*-pathway.

(v) There is strong circumstantial evidence that the *Phytophthora*-pathway represents the migration of air-borne inoculum, rather than the erruption of pre-existing inoculum along an environmental gradient. However, since tuber-borne inoculum cannot specifically be ruled out or inoculum migration absolutely be proved, it may mean that inoculum migration along a definite route is

superimposed on a gradient of errupting, pre-existing inoculum, with the latter having the lesser epidemiological significance.

2.2.0.0 THE PHENOLOGY AND EPIDEMIOLOGY OF THE COMPETITIVE INTERACTION BETWEEN *PHYTOPHTHORA INFESTANS* AND *ALTERNARIA SOLANI* ON BP_1 POTATOES

2.2.1.0 Introduction

The advent of metalaxyl (Ridomil) for the control of late blight in potatoes and tomatoes brought about a change in the disease management strategy for these crops. The efficacy of the systemic properties of Ridomil makes it the most desirable fungicide for the control of late blight. However, because it does not control early blight at all it cannot entirely replace the contact fungicides in current use on potatoes and tomatoes but must be used in conjunction with a fungicide affording early blight control. The manufacturers now encourage its use in this manner.

Many farmers who previously were unaware of the importance of early blight suffered heavy losses due to this disease when they adopted Ridomil exclusively for blight control. This was largely because early blight had until then also been controlled by the normal spray programme for late blight. During 1978 and 1979 it became obvious that an accurate assessment of the economic importance of early blight was essential for cost-efficient spray decisions to be made. Such an assessment would be facilitated by the use of Ridomil because of its potentially total control of late blight and consequent exposure of early blight effects for easier measurement.

The introduction of Ridomil also made feasible the study of the influence of differential fungicide control and weather patterns on the competitive interaction between late and early blight. Furthermore, since these two diseases have weather requirements that are in many respects opposite, a knowledge of their competitive interaction may make it possible to devise spray programmes in which the control of late blight and early blight is consecutive rather than simultaneous.

Competitive displacement in plant pathosystems has only recently received attention and this only in the limited field of the contribution of differential genetic fitness and fungicide tolerance to competitive interactions. These studies, furthermore, have been based on theoretical deductions rather than empirical experimentation (Leonard, 1977; MacKenzie, 1978; Zadoks and Schein, 1979). Thus the interaction between *P. infestans* and *A. solani* in the experiment reported here, may well be the first experimental, quantitative evaluation of competitive displacement in a plant pathosystem.

2.2.2.0 Materials and methods

2.2.2.1 Spray treatments

A randomized blocks experiment with four treatments and four replications, using the potato cultivar BP_1, was planted at the Cedara Experiment Station on 10/9/1978. Spraying commenced on 20/10/1978 after the first blight warning was

issued on 18/10/1978. The treatments were as follows:

Treatment Symbol	Description
R	Sprayed only with metalaxyl (Ridomil) at 14-day intervals until full flower on 20/11/78; thereafter at 10-day intervals. Thus spraying occurred on 20/10/78, 8/11/78, 17/11/78 and 27/11/78 at a rate of 1,5 Kg Ridomil/ha.
A	Sprayed only with propineb (Antracol) at 10-14 day intervals at a rate of 2 Kg Antracol/ha. Spraying took place on the same dates as in the R-treatment with one additional application on 7/12/78.
RA	Sprayed with a mixture of Ridomil and Antracol, each applied at full strength i.e. Ridomil at a rate of 1,5 Kg/ha and Antracol at a rate of 2 Kg/ha, at 10-14 day intervals. Application dates were the same as for the A-treatment.
C	Unsprayed control.

Each of the treatment plots contained 240 potato plants in five rows of 48 plants each. Every plot was surrounded by a border row. In addition a one metre wide path was left between replicates. Routine weed-control was undertaken

uniformly over the whole experimental area. Cutworms (*Agrotis* sp.) were controlled by bait treatment of endosulfan applied at a rate of 10 Kg/ha when the potatoes were in the "green-row" stage, i.e. 7 days after the first plants had emerged.

The potatoes were ridged once, at the flower-budding stage on 10/11/78. The field was overhead-irrigated once, before planting. The crop itself was not irrigated. Although rainfall throughout the growing period was below average, the potato drought stress trials conducted concurrently elsewhere on Cedara showed that drought stress was negligible and therefore supplementary irrigation was not necessary (Rutherford and Mould, p.c.).

2.2.2.2 Disease ratings

For reasons given below a more elaborate method of disease ratings had to be devised for the sprayed treatments than for the control plots. Also, because of the earlier occurrence of disease on the unsprayed plots, these were rated almost a month in advance of the sprayed plots.

Apparent infection rates were calculated according to equation 3.5 of Vanderplank (1963).

Late blight favourable weather periods occurring during the trial were also recorded to facilitate interpretation of the disease progress curves.

Foliar rating of control plots

Late blight assessments were made on 20/10/78 and 28/11/78 in the unsprayed control plots. Visual assessments were carried out according to the BMS key (Anon, 1947) once the first late blight focus had been noticed on 20/10/78. Thereafter all control plots were regularly inspected at four-to-six day intervals to ascertain whether early blight was present. However in the unsprayed control, severe necrosis and shrivelling of the leaves due to late blight made the search for early blight increasingly difficult.

Foliar rating of sprayed plots

Ratings were undertaken to determine separately the proportions of late and early blight. After the first early blight was seen in a Ridomil treated plot on 17/11/78, regular inspections of all plots were made and when early blight could be found in all of the treatment plots, the first rating was made, viz. on 28/11/78, followed by a second, final rating on 18/12/78.

Ratings were made as follows: Four unskilled farm labourers were instructed to remove the sixth compound leaf from the apex of the shortest stem of each plant. All four treatment plots making up one replicate were picked simultaneously, each labourer being allocated a different treatment in each replicate such that on both sampling dates, each sampler collected the entire sample of one plot in each of the four

different treatments, one from each replication.

All the leaflets were removed from the rachides of the compound leaves and pooled according to treatment, i.e. the replicates were not measured separately since the object was to estimate the apparent infection rate, r, for each treatment. A minimum of 1 000 leaflets per treatment was collected. Total leaf area per treatment was rapidly determined by covering A4 sheets of paper as completely as possible with the leaflets of each treatment fitted side by side and measuring the area of the paper underneath the leaves.

Having determined the total leaf area, late and early blight lesion areas were then measured separately. With a number three cork borer, circles were marked on the respective lesions and the number of circles required to cover each lesion was recorded. In this way, the total number of cork-borer circles per lesion type could be counted for each treatment. Since the cutting area of the cork borer was known, the total leaf area destroyed by each of the two blight pathogens per treatment, could be calculated. The proportions of leaf necrosis due to late and early blight could thus be calculated separately and disease progress plotted as a function of time.

This method of rating, achieving accuracy of detail at the expense of leaf sample distribution, was decided upon principally because of the overriding need to measure each

disease separately. Visual ratings of disease severity made according to the Weber-Fechner law depend on the ability of the human eye to measure the proportions of different colour patterns (Kranz, 1974a). In the case of late and early blights the lesions have similar colours. Thus when viewed from a distance the 'measure-at-a-glance' type of ratings employed in the BMS key (Anon, 1947) would not distinguish between them. On closer examination, the concentric circles so characteristic of lesions of early blight, otherwise known as target-spot disease, could be used diagnostically. However the frequent necessity of making two different visual assessments on the same leaf would have required switching back and forth continuously from one disease symptom to the other, deliberately concentrating on each in turn. This is contrary to the very large degree of spontaneity implicit in visual assessments based on the Weber-Fechner law.

Accordingly, and because of the destructive sampling necessary to determine the incidence of each disease separately, it was decided to sample only at a fixed leaf position on a particular stem. This, rather than random sampling was necessary because with the random process the sampler finds his attention drawn to diseased leaflets which stand out against the background of healthy tissue. Thus a pre-determined sampling point is more likely to yield an unbiased estimate. However, the system used here, of sampling the sixth leaf from the apex, underestimates the total amount of disease in the plot because disease is more

severe in the lower canopy than in the upper. This leaf position was chosen, however, rather than one further down the stem, because leaves in that vicinity were more likely to survive a blight attack and thereby provide material at approximately the same station on the two disease-rating dates. The disease ratings of the sprayed plots therefore underestimate the total amount of late blight and early blight present but are probably unbiased.

2.2.2.3 Yield assessments

All treatments were harvested manually on 16/1/1979. Yields were assessed by weight and submitted to statistical analysis.

2.2.3.0 Results

2.2.3.1 Disease ratings and apparent infection rates

The recorded foliar disease levels of late blight and early blight are shown in Tables 1 and 2 respectively, which summarize the individual plot responses shown in Table 3.

The low late blight ratings on 28/11/78 of the sixth-leaf samples from all three sprayed treatment plots are in contrast to the very high level of late blight recorded four days later in the control plots. These results are indicative of highly effective late blight control by both Ridomil and Antracol.

Table 1 Effect of three fungicide treatments on foliar late blight on BP_1 potatoes

Treatment	Late blight rating[*1]					
	20/10/78	28/11/78	3/12/78	18/12/78	r[*2]	S.E.
Ridomil	-[*4]	0,0003	-	0,0	N/N[*3]	-
Antracol	-	0,008	-	0,002	N/N	-
Ridomil/ Antracol	-	0,004	-	0,0	N/N	-
Control	0,005	-	0,73	-	1,154	±0,020

*1 Amount of disease measured as proportion of total leaf area. Values for control estimated according to the BMS key, for late blight (Anon., 1947); values for Ridomil, Antracol and Ridomil/Antracol measured on sixth leaf from apex on 28/11/78 and 18/12/78. In contrast to ratings based on the BMS key, those based on only the sixth compound leaf underestimate the actual amount of disease present.

*2 r = Apparent infection rate calculated by equation 3.5 Vanderplank (1963).

*3 N/N = Infection rate negligible but negative, i.e. the proportion of late blight decreased.

*4 - = No ratings made.

Early blight was first noticed on 17/11/78 when a few isolated lesions were seen in one of the Ridomil plots. Between 17/11/78 and 28/11/78 isolated early blight lesions were seen in the unsprayed control plots. On 28/11/78 early blight was present in all treated plots and ratings

commenced. In the unsprayed control plots, early blight levels did not increase beyond a few isolated lesions, although, as already mentioned, the severity of late blight hampered the search for early blight.

Table 2 Effect of three fungicide treatments on foliar early blight on BP_1 potatoes

Treatment	Early blight rating*1			
	28/11/78	18/12/78	r*2	S.E.
Ridomil	0,004	0,265	0,238	±0,030
Antracol	0,0005	0,126	0,282	±0,023
Ridomil/ Antracol	0,001	0,138	0,279	±0,043
Control	-*3	-	-	-

*1 Amount of disease measured as a proportion of total leaf area. Measurements taken on sixth leaf from apex. In contrast to ratings according to the BMS key for late blight, ratings based on only the sixth compound leaf, underestimate the actual amount of disease present.

*2 r = Apparent infection rate as in Table 1.

*3 - = Negligible amounts of early blight observed in control plots because by 28/11/78, necrosis due to late blight in these plots was 0,73.

Table 3 Early blight and late blight ratings on three dates

Treatment	Date	Replicate							
		1		2		3		4	
		eb*1	lb*2	eb	lb	eb	lb	eb	lb
Ridomil*3	20/10/78	-*4	-	-	-	-	-	-	-
	28/11/78	0,00775		0,00131	0,0002	0,00403	0,00081	0,00209	0,00353
	18/12/78	0,28975	0,0	0,19711	0,0	0,26546	0,0	0,30674	0,0
Antracol*3	20/10/78	-	-	-	-	-	-	-	-
	28/11/78	0,0076	0,00263	0,00036	0,02882	0,00026	0,02282	0,00086	0,0
	18/12/78	0,16919	0,00008	0,09725	0,0037	0,10334	0,00214	0,13295	0,00292
Ridomil/*3 Antracol	20/10/78	-	-	-	-	-	-	-	-
	28/11/78	0,00054	0,0	0,00063	0,00019	0,00277	0,00022	0,00014	0,01692
	18/12/78	0,13089	0,00015	0,12826	0,0	0,1400	0,0	0,15609	0,0
Control*5	20/10/78	-	0,004	-	0,004	-	0,002	-	0,006
	3/12/78	-	0,75	-	0,70	-	0,68	-	0,80
	18/12/78	-	-	-	-	-	-	-	-

*1 eb = Early blight

*2 lb = Late blight

*3 Diseased proportion of sixth leaf from apex of shortest stem

*4 - = No rating

*5 Estimated according to BMS key for the assessment of late blight (Anon., 1947)

Tables 1 and 2 show that the two pathogens were active at different times during the growing season, late blight developing initially and early blight later. Evidence of the initial insignificance of early blight while late blight was actively spreading, is seen in that:

(i) On 28/10/78 early blight was absent in the control plots whereas a late blight rating of 0,005 (BMS scale) was recorded.

(ii) By the end of November the BMS rating of late blight in the control plots had reached 0,73 indicating that much of the tissue of the sixth leaf from the apex had been killed. In contrast, the mean early blight rating in the Ridomil plots, that were in effect unsprayed with respect to early blight because of Ridomil's specificity, showed only 0,004 tissue necrosis.

During December, late blight was inactive while early blight activity increased. Prior to 28/11/78 early blight increased to only 0,004 in the Ridomil plots, while in the 20 days between 28/11/78 and 18/12/78, early blight levels in the same plot showed a 66 fold increase (Table 2). Furthermore after 28/11/78 late blight levels decreased in all sprayed plots (Table 1). Especially significant was the decrease in late blight activity after 28/11/78 in the Antracol treatment compared to the earlier increase in these plots between

20/11/78 and 28/11/78. Assuming Antracol was equally effective against late blight in both November and December, the decrease in late blight activity during December can be attributed to a decrease in late blight favourable weather.

The amount of early blight was finally highest in the Ridomil plots although the infection rate was lower for this treatment than it was for the Antracol treatment. The lower r-value in the Ridomil plots is due to the higher levels of early blight on 28/11/78 compared to the levels in the Antracol plots on the same day. Apparent infection rate values are significantly affected by initial recorded amounts of disease (Vanderplank, 1963) and the Ridomil treatment should therefore not be judged by its apparent anomalous infection rate but by the final level of disease in these plots.

2.2.3.2 Disease progress curves and weather pattern

The severity of the late blight epidemic in the control plots is due to the 18 days in November which had weather conditions favouring late blight development. This is the maximum number of late blight favourable days recorded at Cedara for November, since 1958 when the monitoring of late blight weather commenced. In contrast, between 28/11/78 and 18/12/78 late blight favourable weather was recorded on only the first two days of the period. The disease progress curves of late and early blight during November and December, respectively, are depicted relative to weather

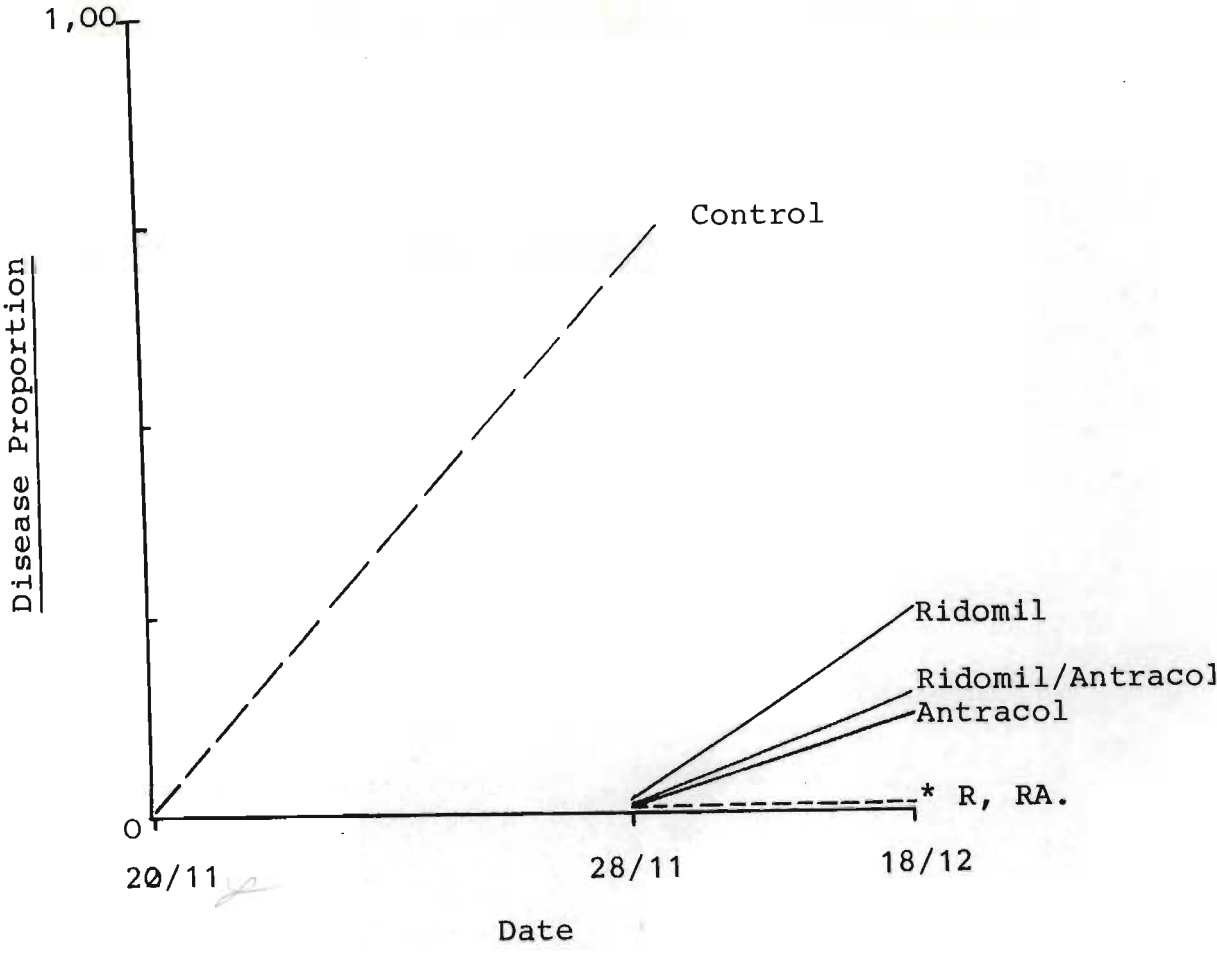
patterns, in Figure 4.

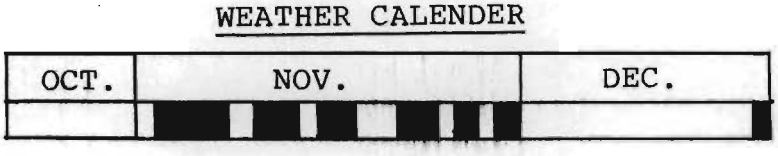
The increased early blight activity during December may be due to several interacting factors determined by weather, host growth stage and susceptibility as well as changes in the microclimate.

Waggoner and Horsfall (1969) discussed the confusion in the literature about the weather requirements of *A. solani*. They conclude that the pathogen is favoured by intermittent wet and dry periods and wind activity during the dry spells. Persistentwetness, as was recorded for much of November, severely limits early blight spread (Waggoner and Horsfall, 1969). Thus one may conclude that the late blight favourable weather recorded during November was unfavourable for early blight development.

During December, warmer, less humid days and cool nights with regular dew incidence are typically recorded at Cedara (Reynolds, p.c.). This is similar to the weather requirements for early blight development reported by Rotem and Reichert (1964) and Rotem (1968) thereby supporting the conclusion that the increased early blight activity recorded in December is at least in part, due to favourable weather.

The growth pattern of the host may influence early blight development in many ways. Ageing plants become increasingly susceptible to early blight. As the disease increases and senescence proceeds the crop canopy opens up and favours





Weather favourable for late blight

Weather not favourable for late blight

Late blight

Early blight

* R, RA : Late blight in R and RA plots was negligible and decreased between 28/11 and 18/12

Fig. 4 Proportions of late blight and early blight between 20/11/78 and 18/12/78 relative to the occurrence of late blight favourable weather patterns.

rapid early blight increase and spread (Waggoner and Horsfall, 1969). As the canopy becomes less dense, fluctuations in the microclimate become more frequent thereby further contributing to rapid early blight development (Waggoner, 1965).

2.2.3.3 Yield assessments

The mean potato yields associated with the three fungicide treatments and with the unsprayed control are recorded in Table 4.

Table 4 Effect of three fungicide treatments on yield of BP_1 potatoes

Treatment	Mean yield of marketable tubers*1 in Kg/plot
R	168,150
A	167,850
RA	186,375
C	126,275

*1 S.E. of means: $\pm$ 6,144 Kg

L.S.D.'s : P < 0,05 = 19,656

P < 0,01 = 28,238

For the purpose of computing the L.S.D.'s, statistical comparisons were made between the unsprayed control and the fungicide treatments as a group. In this way the contribution of the diverging mean of the control plots to the statistical variance of the experiment as a whole was

accounted for. In spite of this, the difference between the Ridomil/Antracol treatment on the one hand and the Ridomil and Antracol treatments on the other, was not significant for $P < 0,05$.

2.2.4.0 Discussion

2.2.4.1 Implications of weather effect on the blight complex for the blight management strategy

The interaction between certain weather patterns and the blight complex has important implications for the management of the blight pathosystem. In November, weather exceedingly favourable for late blight prevailed, whereas in December, very few days were recorded as having conditions favouring late blight (Fig. 4). Assuming by extrapolation of the data of Rotem and Reichert (1964), Rotem (1968) and Waggoner and Horsfall (1969), that when mid-summer weather at Cedara is not favourable for late blight it is potentially favourable for early blight it may be deduced that as much as the November, 1978 weather favoured late blight, that in December, 1978 favoured early blight. It appears, therefore, that there was a clear-cut change in weather phases, late blight being favoured initially whereas later, both weather and condition of the crop favoured early blight. This suggests that the optimal blight complex control strategy might be to aim primarily at late blight control until the end of November after which early blight control should be emphasized.

Such an integrated control strategy would have several important implications for the management of the Natal blight pathosystem, e.g. it would eliminate unnecessary fungicide applications when only one member of the blight complex rather than both, are active. However, before these are examined it is necessary to consider whether the November-December, 1978, weather pattern was exceptional. Historical weather data from 1958 to 1979, the period during which weather records have been kept at Cedara for the purposes of the Young forecasting system, were therefore examined.

Graphs of various weather statistics are presented in Figure 5.

These revealed no evidence that, in the long term, December weather is markedly different to that of November, or that the weather does not follow a smooth trend consistent with the summer weather pattern.

When, however, the daily weather records for the months of November and December in the period 1958 to 1979 were investigated in terms of the number of days per month when conditions were favourable for late blight, a very different relationship emerged. As shown in Figure 6 the number drops from an average of 10 in November to five in December, increasing again to 8,8 in January. Thus there is a clear long-term trend for December to be less favourable for late blight than either November or January. Considering that potatoes at Cedara are only planted in September, it is

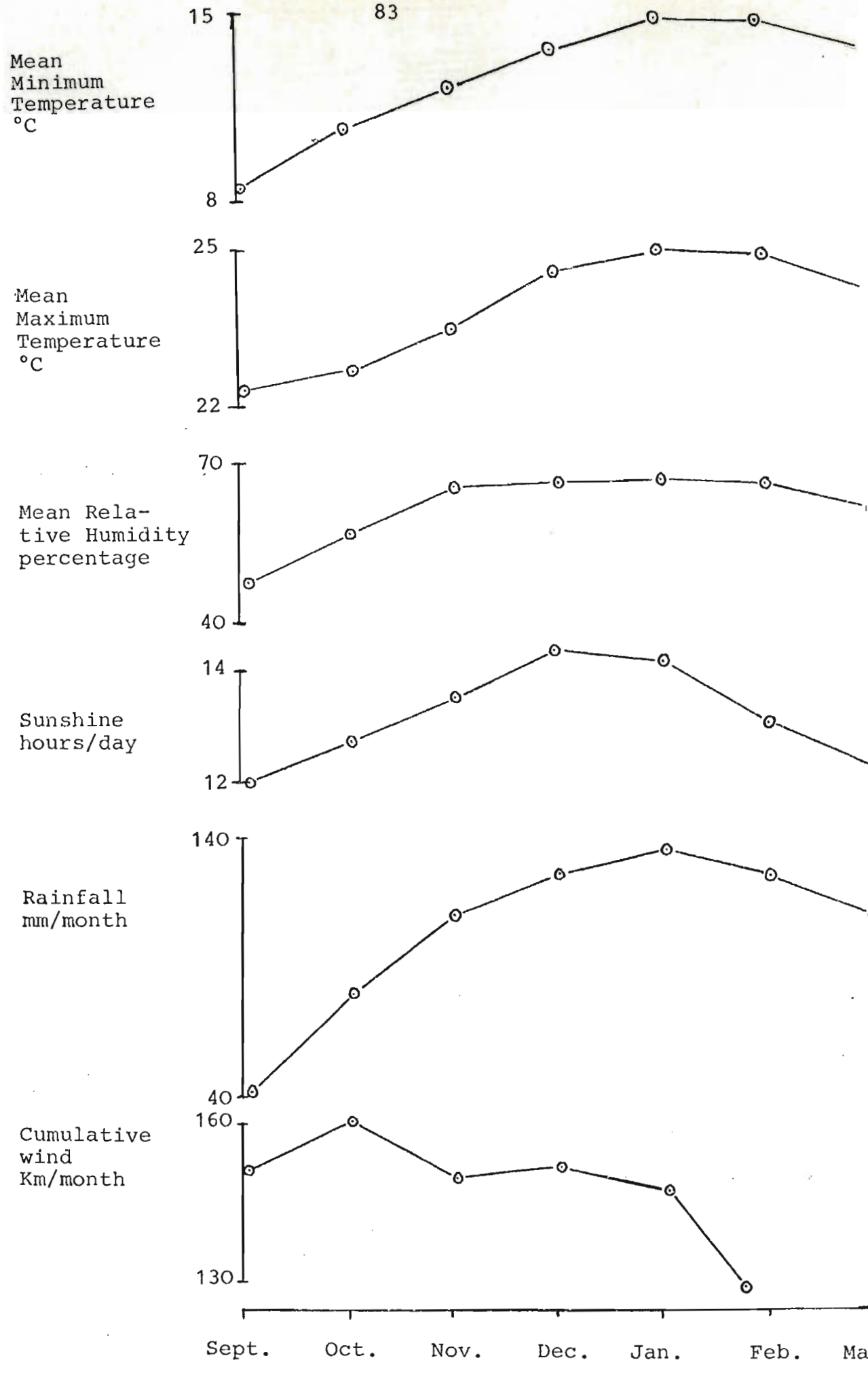


Fig. 5 Mean weather data recorded at Cedara between 1958 and 1978

apparent that December is the least favourable month for late blight in early (September) and mid-summer (October) potato plantings. The only anomalous factors in Figure 4 which might explain this December phenomenon is the peak for December in the graph which measures mean number of sunshine hours per day and the slight increase in December in the graph depicting the cumulative wind index in km/month. Compared to December, the mean maximum temperature and the number of sunshine hours per day in November are also both decreased. However these differences must be interpreted cautiously because they are based on means calculated for a 20 year period only.

It became quite evident, therefore, that the daily weather pattern for November-December, 1978, depicted diagrammatically in Figure 4, was consistent with the long-term daily weather pattern. Consequently there is good reason to accept that the potato blight management strategy for the Cedara region of Natal should consist of two separate phases in which initially, late blight and, subsequently, early blight control tactics should predominate.

The manufacturers of Ridomil have apparently not yet attempted to determine the epidemiological principles which would govern the optimum use of Ridomil in Natal. Instead the standard recommendation is that Ridomil be mixed with an acceptable fungicide for early blight control, in a routine preventative spray programme.

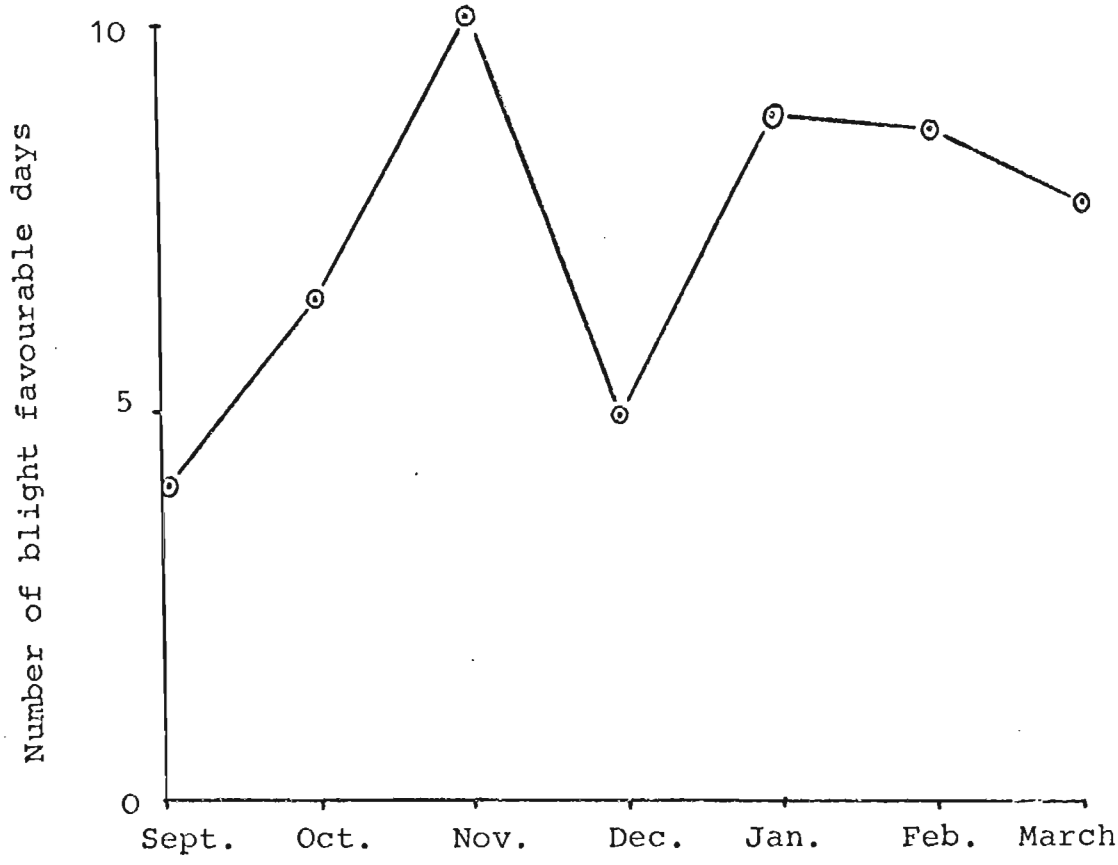


Fig. 6 Number of blight favourable days per month. Means calculated from weather data recorded at Cedara between 1958 and 1978.

It is useful to consider the implications of this recommendation in a season such as 1978, which has been shown above to be within the weather variation expected of 20 year trends. Clearly money spent on early blight control in November would have been totally wasted and a similar argument holds, *mutatis mutandis* for late blight in December.

Ridomil is a systemic fungicide which can be applied at low volumes. In contrast, fungicides currently available for early blight control are contact surface protectants which must necessarily be applied in high volumes to ensure that both upper and lower leaf surfaces are treated. Thus if an early blight fungicide is added to a Ridomil programme, the early blight control will not be adequate unless high volume application procedures are adopted. When this is done, the benefits of low volume spray technology fall away.

In addition, one of the advantages claimed for Ridomil is that it is not easily washed off by rain. However, the early blight component of the mixture needs to be re-applied after irrigation or if more than 13 mm rain has fallen (Bot, Vermeulen and Hollings, 1980). Therefore the farmer is still as susceptible to the vagaries of the weather as he had been hitherto.

During 1981, Ridomil will be sold only in a mixture with Mancozeb. The product will be sold under the trade name Ridomil MZ, and will contain 109,4g metalaxyl a.i. per kilogram and 450g mancozeb a.i. per kilogram in a 55,0%

active ingredient formulation (Jooste, Ciba-Geigy, p.c.). Farmers will be recommended to apply the Ridomil MZ at a rate of 2,2 Kg per hectare at 14-day intervals. This formulation at the above rate, means an application of 1,23 Kg mancozeb a.i. per hectare as opposed to the previously recommended rate for both late blight and early blight of 1,60-2,40 Kg mancozeb a.i. per hectare. It is as yet unclear why the level of mancozeb was not increased to the previously recommended level to obviate the need to add any mancozeb to the Ridomil MZ formulation when both blight diseases have to be controlled simultaneously.

Apparently the reason for the inclusion of mancozeb is not specifically to control early blight but to widen the fungicidal spectrum of the mixture, thereby minimizing the risk that tolerance to Ridomil will develop in *P. infestans*. For early blight control, it is recommended that the amount of mancozeb be increased or another appropriate fungicide added.

It is clear that the possibility of the development of resistance in *P. infestans* to Ridomil is considered more seriously now than it was in 1978 when Ridomil was first introduced. It is difficult to envisage what contribution a reduced dosage of mancozeb, a *surface protectant*, could make in preventing tolerance in *P. infestans* to Ridomil which is *systemically* active. Unless there is synergism between mancozeb and metalaxyl, or their respective inert ingredients, reduced mancozeb dosage is contra-indicated by the principles

advanced by Dekker (1976). The author has been unable to trace the data, published or unpublished which underlie the rationale for the Ridomil MZ formulation.

2.2.4.2 Effects of late blight and early blight on yield

Because Ridomil controls late blight only and Antracol controls early blight primarily but has some effect on late blight, the results in Table 3 can be used to obtain an estimate of the respective economic significance of late blight and early blight.

The difference between the Ridomil/Antracol treatment and the unsprayed Control, measures approximately the negative effect of the blight complex on yield. This is an underestimate because Antracol did not preclude all early blight infection. In the absence of spraying, yields were thus decreased by at least 32,2% due to the blight complex.

The difference of 41,875 Kg/plot between the Ridomil and Control treatment yields measures the yield increase achieved in the presence of uncontrolled early blight, by completely controlling late blight. Thus the late blight disease component reduced yields by approximately 24,9% in the presence of early blight.

If it is accepted on the evidence of the negligible foliar late blight levels in the Ridomil treatments in Table 1, that Ridomil controls late blight almost completely, then

the addition of Antracol might reasonably be expected, in effect, to contribute almost exclusively to early blight control rather than further improve the efficacy of Ridomil against late blight. Thus the yield increase of 18,225 Kg/plot when the Ridomil/Antracol and Ridomil treatments are compared is indicative of the early blight controlling properties of Antracol. Thus in the absence of late blight, due to the Ridomil, early blight decreases yields by 9,7%. However this is an underestimate because the disease is not totally controlled by Antracol. Because the individual effects of this fungicide on each of the two diseases is not known, no comparison of treatment yields is able to reveal the negative rôle of early blight in the presence of late blight.

It is noteworthy that the complete control of late blight only, by Ridomil, increased yields to approximately the same level as did the incomplete control of the blight complex by Antracol.

The Ridomil/Antracol and Antracol treatments were used to control both diseases of the blight complex and can thus be compared on this basis. The difference in yield of 18,525 Kg/plot between these treatments while just not significant for values of $P < 0,05$ is significant for $P < 0,1$.

2.2.4.3 Economic evaluation of fungicide options

The recorded foliar disease levels shown in Tables 1 and 2 are consistent with the subsequently obtained yield data in

Table 4, indicating that under the circumstances of the experiment there was no advantage in using Ridomil rather than Antracol. Both the Ridomil and Antracol treatments gave yields which were significantly better ($P < 0,05$) than the yields recorded in the unsprayed control.

Furthermore the yield increases of 18,225 Kg/plot and 18,525 Kg/plot respectively, between the Ridomil/Antracol treatment on the one hand and the separate Ridomil and Antracol treatments on the other, were almost significant for $P < 0.05$.

The economic implications of these yield figures can conveniently be appreciated if the yields in Kg/plot are converted to 15 Kg pockets/ha and evaluated in terms of potato prices prevailing on the municipal market when the experiment was harvested.

Table 5 Cost-benefit evaluation of the effects of three fungicide treatments on the yield of BP_1 potatoes

Treatment	Yield in 15 Kg pockets/ha	Income at R1,50/pocket	Number of spray applications	Chemical Cost/ha	Total fungicide cost/ha*1
Ridomil	2 067,19	R3 100,78	4 (R200)	R90,00	R190,00 (R290,00)
Antracol	2 063,50	R3 095,25	5 (R250)	R25,70	R150,00 (R275,70)
Ridomil/Antracol	2 291,24	R3 436,86	5 (R250)	R115,70	R240,70 (R365,70)
Control	1 552,69	R2 329,03	-	-	-

*1 Application costs are assumed to be R50,00/ha/application

The yield increase in the Ridomil/Antracol treatment over the untreated control was thus equivalent to a nett increased financial return of R867,13/ha after allowance is made for fungicide and spraying costs. Similarly the difference between the Ridomil/Antracol treatment on the one hand and the separate Ridomil and Antracol treatments on the other, shows financial returns in favour of the Ridomil/Antracol treatment of R386,78/ha and R430,75/ha respectively. Thus if statistical comparison between the Ridomil/Antracol and Antracol treatments is made for values of $P < 0,1$ the yield of the Ridomil/Antracol treatment will be significantly better than the yield of the Antracol only treatment to the extent of an increased financial return of R430,75/ha.

Another important observation is that if the cost of application increases to R65,00/ha the price advantage which Antracol has over Ridomil, when considering fungicide costs only, would disappear because Antracol requires five applications and Ridomil only four. Although the yield differences between the Ridomil and Antracol treatments are almost identical and statistically non-significant, these calculations nevertheless illustrate the important financial rôle of application costs.

2.2.5.0 Conclusions

(i) When late blight and early blight interact to form a disease complex, the relative importance of each of the pathogens is determined by the relative

occurrence of weather patterns favouring each disease, the differentiating rôle of selective fungicides, host susceptibility and host growth, factors which may alter the microclimate in favour of either of the pathogens.

(ii) The fungicide strategy for the management of the blight complex should be based on those epidemiological factors which determine the importance of each of the diseases. Specifically, the control strategy should be matched with the dominant disease rather than depend on *ad. hoc.* dual purpose spray programmes in which the rôle of each of the competitors is not quantitatively known.

2.3.0.0 GAUSE : A SIMULATOR OF THE COMPETITIVE INTERACTION BETWEEN *PHYTOPHTHORA INFESTANS* AND *ALTERNARIA SOLANI*

2.3.1.0 Introduction

The particular case of Ridomil and its selective narrow spectrum and therefore differential control of fungal pathogens, presented in Section 2.2.3.3 is an example of an ecological problem which such selective chemicals will introduce into pathosystem management. The consequences of selectively controlling only one member of a two-specie blight complex are far-reaching and sufficiently important in principle, to raise serious concern and doubts about selective systemic fungicides as a future panaceae for many plant diseases.

Potato or tomato foliage is a major niche requirement for the establishment of the blight complex. Any discreet unit of potato or tomato foliage becomes an arena when both *P. infestans* and *A. solani* occur together. A competitive interaction is established because both pathogens compete for the same food base. As in any ecological niche, simultaneous habitation by two organisms will result in one becoming dominant, to the extent that it is more capable of exploiting the niche than is the weaker competitor.

This ascendancy of one organism is due to the competitive displacement principle which follows from the competition equations epynomously attributed to Gause (1934), although

apparently not originally due to him (Hardin, 1960; Pielou, 1976). The genesis of the competitive displacement principle is placed with Volterra (1926) but it clearly dates back to at least Grinnel (1917) and possibly Steere (1894); and as Crombie (1947) and Whittaker and Levin (1975) point out, its roots are surely with Darwin. Indeed, competitive displacement is implicit in the struggle for the survival of the fittest and the principle is thus fundamental to evolutionary biology.

In spite of its evolutionary significance, competitive displacement in general has been studied more theoretically than empirically. The most definitive and oft-quoted experimental results in the literature are still the early yeast population studies by Gause (1934). Thus the competitive displacement principle is invoked as a heuristic model to explain events rather than as the basis for a deterministic strategy.

Competitive displacement in plant pathology has received attention in studies comparing the relative genetic fitness of two pathogenic races (Leonard, 1977; MacKenzie, 1978). Dekker (1976) and Zadoks and Schein (1979) discuss applications of the principle that selection pressure, exerted by a selective systemic fungicide, alters the relative fitness of two strains of cucumber powdery mildew caused by *Sphaerotheca fuliginea*. However, these approaches and the equations which these authors present are not in terms of basic epidemiological parameters and cannot therefore be

applied directly to plant pathosystems.

One of the most useful and versatile parameters in epidemiology is the growth rate of disease or more specifically the apparent infection rate, r (*sensu*: Vanderplank, 1963) which is a statistical estimate of this parameter. The logistic growth equation (Vanderplank, 1963, eq. 3.2) which calculates the apparent infection rate is probably the most fundamental equation in population ecology, dating, in its definitive form, to Volterra (1926). Although competition equations have been based on Volterra's equations (Pielou, 1976; Jeffers, 1978) these have been rather esoteric mathematical approaches not directly applicable to the logistic equation of the apparent infection rate, r, which characterises the behaviour of plant pathosystems.

Therefore, to evaluate the epidemiological significance of disrupted competition between two plant pathogens due to selective fungicide control for example, a dynamic simulator based on Vanderplank's (1963) logistic equations is developed in this section of the thesis. Specifically, it is intended as a strategic aid to deterministic pathosystem management so that specific competitive interactions may be evaluated and manipulated to achieve a stable interaction which may be different from the natural equilibrium in an uncontrolled system.

2.3.2.0 Materials and methods

The procedure for the development of the simulator follows three steps, i.e. a relational diagram (*sensu*: Jeffers, 1978) is presented as the conceptual model for the computer algorithm, the logistic equations which symbolize the behaviour of the components of this diagram are developed and linked mathematically, and finally, the computer programme for the simulator is written and its output evaluated in terms of the experimental results obtained in Section 2.2.3.0.

2.3.2.1 Relational diagram of the competitive interaction between *P. infestans* and *A. solani*

When *P. infestans* and *A. solani* compete in the same ecological niche, e.g. on potato or tomato foliage, each pathogen achieves a characteristic and intrinsic logistic growth rate, and these determine the nature of their competitive equilibrium. Furthermore, since they are competing for host tissue, the logistic growth rate of the host will be equally important in determining the competitive equilibrium. The interrelationship of these three interacting logistic growth processes can be depicted conveniently with the aid of conventions introduced by Forrester (1961) as in Figure 7.

The relational diagram in Figure 7 shows that the link between the three interacting logistic growth components is provided by the carrying capacity or amount of host tissue

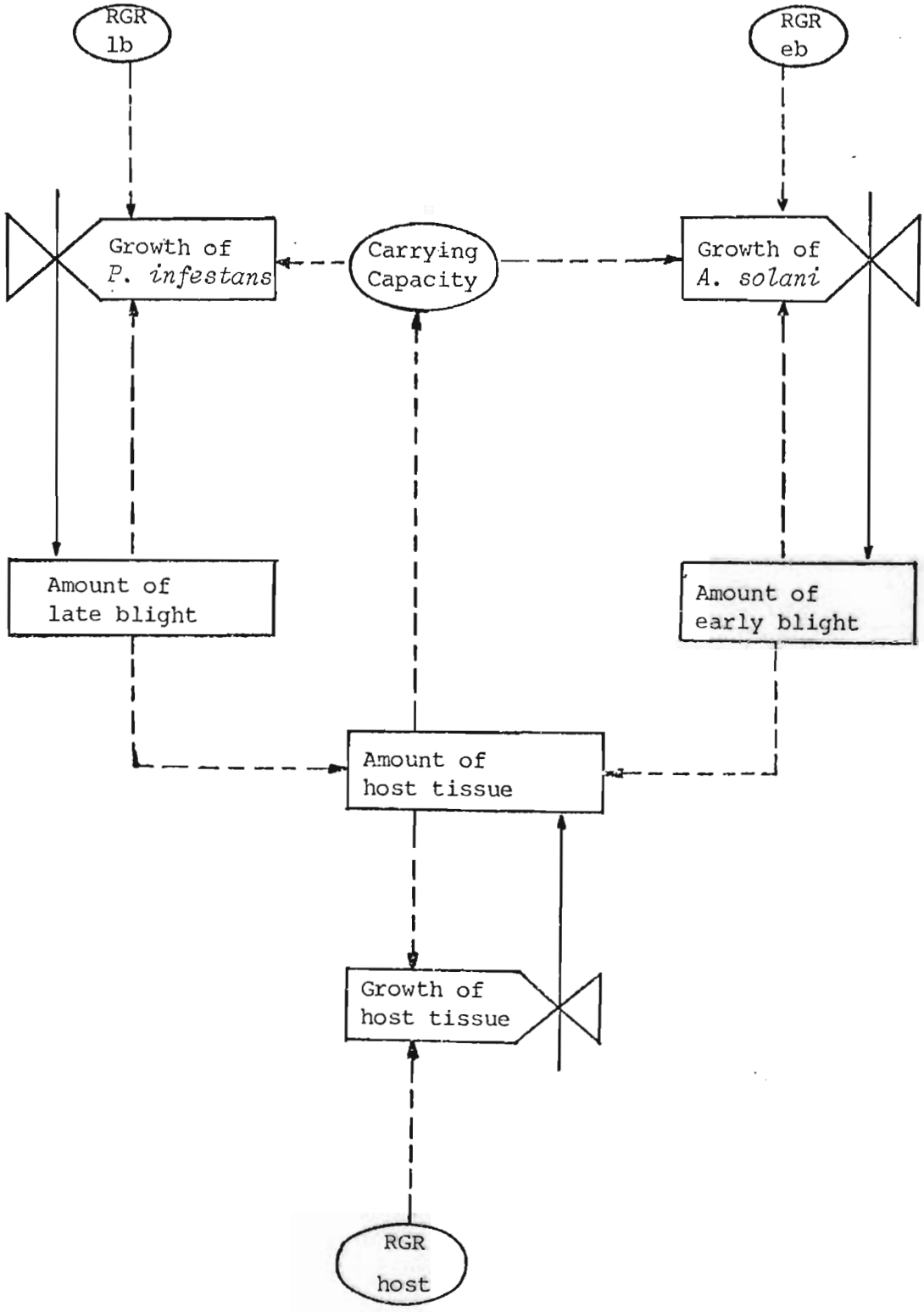


Fig. 7 Relational diagram depicting interacting logistic growth interactions between *P. infestans*, *A. solani* and their common host.

available for pathogenesis.

In the case of late blight and early blight of potatoes, these diseases are characteristically established only when the potato host has attained maximum, or near-maximum, growth. Therefore, the simulator developed here does not require specific correction for host growth, i.e. an unchanging (non-increasing) maximum amount of host tissue is assumed, for which *P. infestans* and *A. solani* compete. In other crops such as tomatoes this may not be the case, and although the problem of increasing host growth is ignored here, it remains to be solved before the simulator can be applied to such situations.

2.2.3.2 Logistic growth equations for *P. infestans* and *A. solani* competing for non-increasing amounts of potato tissue

If each of the two blight components were infecting alone, Vanderplank's (1963) equation 3.2 would apply. Therefore, for late blight:

$$\frac{dL}{dt} = r_L \cdot L(1-L) \quad \text{..... eq. 1}$$

Where L = proportion of host tissue infected with late blight;

r_L = the apparent late blight infection rate;

and 1 = the upper limit of host tissue available for colonization, which, in the calculations that

follow, will be fixed at its maximum level.

Similarly, for early blight

$$\frac{dE}{dt} = r_E \cdot E(1-E) \quad \text{..... eq. 2}$$

Where E = proportion of host tissue infected with early blight;

r_E = the apparent early blight infection rate;

and 1 has the same meaning as in eq. 1 above.

However, when the two pathogens are competing on the same host, as represented in Figure 7, the carrying capacity which they share requires modification to include the erosion caused by the other pathogen, viz:

$$\frac{dL}{dt} = r_L \cdot L(1-E-L) \quad \text{..... eq. 3}$$

and $$\frac{dE}{dt} = r_E \cdot E(1-L-E) \quad \text{..... eq. 4}$$

Thus the mathematical model which would define the competitive interaction depicted in Figure 7, with the proviso that the carrying capacity does not increase, would be provided by an explicit and simultaneous solution of equations 3 and 4. In general, such explicit solutions are not possible (Pielou, 1976) and therefore it was decided to resort to iterative numerical methods to find the solution; specifically to the fourth order Runga-Kutta methods for first order differentials as described by Carnahan, Luther and

Wilkes (1969).

2.3.2.3 The computer programme for GAUSE, a dynamic simulator of the competitive interaction between *P. infestans* and *A. solani*

The numerical solution of differential equations by means of the Runga-Kutta method (*loc. cit.*) is only feasible with the aid of high-speed computers. Therefore, the programme in Table 6 was written for use with an Apple II Plus, graphics microcomputer. The programme, named GAUSE, dynamically simulates the increase in late blight and early blight as determined by the competitive interaction between them.

In step 210 of the programme a logistic growth function for host foliage growth is also included, viz:

$$\frac{dF}{dt} = r_F \cdot F(1-F) \quad \text{..... eq. 5}$$

Where F = amount of host foliage as a proportion;

r_F = growth rate of the foliage;

and 1 = upper limit of foliage growth.

This equation is included here so that each run of the simulator will also plot the total amount of foliage available for the two pathogens. However, because equations 3 and 4 are not linked to equation 5, GAUSE merely plots the host growth pattern which leads to the assumed maximum

Table 6 Basic programme of the simulator GAUSE for the competitive interaction between *P. infestans* and *A. solani*

```
100  PRINT  "WHAT ARE THE CONSTANTS;  RL, RE, RF"
110  INPUT  RL, RE, RF
120  PRINT  "WHAT ARE INFECTION DAYS;  DL, DE"
130  INPUT  DL, DE
140  PRINT  "WHAT ARE INITIAL VALUES;  F, LI, EI,"
150  INPUT  F, LI, EI
160  HGR:HPLOT 0,0 TO 240,0 TO 240,159 TO 0,159 TO 0,0
165  T = 1
170  FOR D = 0 TO 120
180  IF (D = DL) THEN L = LI
190  IF (D = DE) THEN E = EI
200  LO = L:EO = E:FO = F
210  FI = T * (RF * FO * (1-FO))
220  LI = T * (RL * LO * (FO-LO-EO))
230  EI = T * (RE * EO * (FO-LO-EO))
240  F2 = T * (RF * (FO + 0.5 * F1) * (1-(FO + 0.5 * F1)))
250  L2 = T * (RL * (LO + 0.5 * L1) * ((FO + 0.5 * F1)-(LO + 0.5
       * L1)-(EO + 0.5 * E1)))
260  E2 = T * (RE * (EO + 0.5 * E1) * ((FO + 0.5 * F1)-(LO + 0.5
       * L1)-(EO + 0.5 * E1)))
270  F3 = T * (RF * (FO + 0.5 * F2) * (1-(FO + 0.5 * F2)))
280  L3 = T * (RL * (LO + 0.5 * L2) * ((FO + 0.5 * F2)-(LO + 0.5
       * L2)-(EO + 0.5 * E2)))
290  E3 = T * (RE * (EO + 0.5 * E3) * ((FO + 0.5 * F2)-(LO + 0.5
       * L2)-(EO + 0.5 * E2)))
300  F4 = T * (RF * (FO + F3) * (1-(FO + F3)))
310  L4 = T * (RL * (LO + L3) * ((FO + F3)-(LO + L3)-(EO + E3)))
320  E4 = T * (RE * (EO + E3) * ((FO + F3)-(LO + L3)-(EO + E3)))
330  F  = FO + (F1 + 2 * F2 + 2 * F3 + F4) / 6
340  L  = LO + (L1 + 2 * L2 + 2 * L3 + L4) / 6
350  E  = EO + (E1 + 2 * E2 + 2 * E3 + E4) / 6
360  X  = 2 * D
370  Y  = 159 - 159 * F: HPLOT X,Y
380  Y  = 159 - 159 * L: HPLOT X,Y
390  Y  = 159 - 159 * E: HPLOT X,Y
400  NEXT D
410  END
```

amount of available host tissue which is a fundamental constraint imposed on the model.

At the start of each simulation run, three variables, specified by steps 100-150 in the programme, have to be entered, namely:

(i) apparent infection rate (r/unit/day) for each of the two diseases;

(ii) dates on which the two diseases were first seen in the crop;

(iii) the amount of disease for each of the dates in (ii) above.

Thereafter GAUSE proceeds with the simulation of disease progress as determined by the constraint parameters entered.

2.3.3.0 Results

Because GAUSE was designed to mimic the competitive interaction between late and early blight it can be used to generate graphs depicting any particular interaction simply by varying the constraint variables. Many variations of these input variables are possible and GAUSE simply and rapidly simulates their consequences.

2.3.3.1 The effect of different levels of late blight control on the final amount of early blight

The simulated consequences of two arbitrary degrees of late blight control, due, for example, to selective fungicide efficiency or horizontal resistance (*sensu*: Vanderplank, 1978) to late blight, on the competitive interaction between *P. infestans* and *A. solani* are presented. By keeping both the other variables and the early blight apparent infection rate constant and only changing the apparent infection rate of late blight, the concomitant changes in the ultimate status of these two diseases can be determined as shown in Table 7.

Table 7 Changes in the competitive interaction between late blight and early blight as simulated by GAUSE

Three examples of degrees of late blight control	Apparent Infection rates r/unit/day		Observation of first disease *1		Ultimate % of diseased tissue	
	lb	eb	lb	eb	lb	eb
Late blight uncontrolled	1,145	0,3	50	60	99,999	,001
Moderate control of late blight	0,35	0,3	50	60	60	40
Nearly complete control of late blight	0,001	0,3	50	60	,001	99,999

*1 Number of days after crop emergence

NOTE ADDED IN PROOF

One week before this thesis was bound, the August, 1980 issue of Phytopathology appeared in the library of this centre containing a letter to the editor by G. Skylakakis in which the author of the letter discusses competition between two pathogen races for the host's susceptible sites. Two equations are presented which are identical to equations 3 and 4 of this thesis and it would appear that these were independently developed by the present author and the author of the letter to the editor of Phytopathology.*

Skylakakis arrived at the model from purely theoretical arguments and makes a request for field data which would verify the model. The data and experiments reported in this thesis provide the required validation and the appearance of Skylakakis's model is thus viewed as fortuitous, independent corroboration of the theme of Section 2.3.0.0 of this thesis.

As it stands, Skylakakis's treatment of competition ignores the possibility of changing amounts of host tissue. Neither are the implications of competition for the application and interpretation of Vanderplank's basic epidemiology equations considered.

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* Skylakakis, G. (1980).
Estimating Parasitic Fitness of Plant Pathogenic Fungi: A Theoretical Contribution.
Phytopath. 70 (8), 696-698.

The concept of the competition quotient, introduced in this thesis, is probably the most valuable contribution to the evaluation of competitive exclusion. Without such a quantitative statistic, the evaluation of competitive interactions are likely to remain of theoretical value as in Skylakakis's contribution.

A manuscript is in preparation for a mathematical model based on differential equations which can cope with competition between any number of competitors. It will cope with changing carrying capacity and the relative growth rates of the competitors, which rates may not only be different but also vary in time.

-oOo-

2.3.4.0 Discussion

Caswell (1976) in discussing the problem associated with the validation of models and simulators, points out that the observed versus predicted method of analysis is essential where the purpose is to make accurate predictions. In plant pathosystems, the need for this kind of validation of disease forecasting methods has been emphasised by many authors (Zadoks, 1972; Vanderplank, 1975; Teng, Blackie and Close, 1977; Dent and Blackie, 1979). However, in other models in which the objective is to advance understanding, a more appropriate test of the model is whether it is useful for learning more about the pathosystem (Mankin, O'Neil, Shugart and Rust, 1975). Because GAUSE is based on the fundamental logistic growth equation which serves as the foundation of epidemiology theory, it may be used either as a quantitative evaluator of specific events or heuristically towards an increased understanding of the possible epidemiological consequences of competitive displacement. Therefore the value and possible applications of GAUSE will be discussed initially with reference to the blight complex discussed in this thesis and in terms of its wider epidemiological implications.

2.3.4.1 Competitive displacement in the potato and tomato blight complex in Natal

GAUSE was developed in response to the need to evaluate quantitatively the implication of competition between *P.*

infestans and *A. solani* in the potato blight complex. On tomatoes the nature of the competitive interaction between *P. infestans* and *A. solani* differs significantly from that on potatoes and consequently imposes different constraints on the management strategy for the blight complex.

Implications of increased early blight activity in the potato blight complex due to decreased competition from late blight

In potato crops in Natal, early blight generally appears some weeks later than does late blight and consequently builds up only towards the end of the growing season. Thus although potato early blight epidemics have a shorter time span potential than do those of late blight, they occur during the critical terminal growth stage when rapid tuber bulking is expected that may account for as much as 30% of the final tuber yield (Rutherford, 1977). Reduction in leaf area during this stage due to early blight can severely limit yield (see Section 2.2.4.2). If, therefore, selectively reducing the incidence of late blight should have the consequence of increasing early blight disease levels in the maturing crop, the deleterious economic effect of early blight would be enhanced.

Table 7, row 1, based on the variables measured in the spray trial reported in Section 2.2.3.1, shows that uncontrolled late blight established 20 days before early blight and increasing at late blight apparent infection rate of r = 1,145

has the consequence of excluding *A. solani* as a competitor in the blight complex. Although not shown in Table 7 the 73% late blight defoliation reached on 3/12/78 in the spray trial was accurately simulated by GAUSE.

Because of this extremely rapid increase in late blight in the unsprayed control plots it was not possible to determine the rate of increase of early blight in these plots. However when the relevant data concerning the date of first late blight appearance and late blight apparent infection rate observed in the control plots are entered into the GAUSE programme, the simulator corroborates the observation made during the experiment that only a few early blight lesions could be found in the unsprayed control plots.

Similarly, when GAUSE is provided with the infection rates and first infection dates of both blights as recorded in the Ridomil, Antracol and Ridomil/Antracol treatments, it accurately simulates the amounts of blight recorded in these plots on 18/12/78. This provides strong evidence of GAUSE as an authentic simulator. Although this is to be expected, because of its reliance on the logistic differential equations (*loc. cit.*), such validation is proof also of the accurate application of the Runga-Kutta method (*loc. cit.*) and the programming procedure followed in generating GAUSE.

In Section 2.2.4.2 it was explained that the increased levels of early blight in Ridomil-treated plots, where late blight is nearly totally controlled, cannot be compared with the

early blight levels in either the Ridomil/Antracol or Antracol plots because in these treatments the effects of fungicide control of early blight by Antracol is confounded with the competition effect as a result of changed levels of late blight.

GAUSE resolves the problem and reveals (Table 7) the effects of moderate or severe reductions in the late blight apparent infection rate on the final levels of early blight. It can be seen that early blight can increase almost a hundred-fold in the absence of late blight without altering the early blight apparent infection rate or the time periods during which the diseases are increasing. Thus the valuable expedient of being able to substitute a simulator for experimentation (Kranz, 1974a;b;Waggoner, 1974; Shugart and O'Neil, 1979; Zadoks and Schein, 1979) is also met by GAUSE.

The epidemiological competence (*sensu*: Crosse, 1967) of early blight, due to the ability of *A. solani* to survive in soil-debris and on dried leaves and to remain infective for long periods (Walker, *loc. cit.*), coupled to increased terminal levels of early blight due to exclusion of late blight from the blight complex, could lead to increased year-to-year levels of early blight. Thus the advent of Ridomil may solve the late blight problem only to replace it with an early blight problem which, in terms of current fungicide technology, is not as easily controlled as late blight. This and other examples of fungicide-mediated competitive exclusion will be discussed below when the wider epi-

demiological implications of competitive exclusion are considered.

Competitive exclusion in the tomato blight complex

Vanderplank (1963) has pointed out that estimates of the apparent infection rate of disease may require correction because of the growth of the host. This was not considered in GAUSE because both late blight and early blight became established in the experimental potato crop after full flower when the potato haulm were fully grown.

In contrast to the situation on potatoes, both late blight and early blight commonly attack young tomato plants (Walker, 1952) when the amount of host growth is only a fraction of its potential. Moreover, tomato early blight usually preceeds tomato late blight (*loc. cit.*) and the competitive interaction is thus both serially and quantitatively altered. Without alteration, GAUSE cannot therefore be used to simulate the competitive interaction between *P. infestans* and *A. solani* on tomatoes.

The need for this correction, as well as a useful guide as to how it might be made, may be gained by discussing a specific numerical example in terms of Vanderplank's (1963) equation 8.2.

Consider an expanding tomato leaf with area y_1 at time t_1 and y_2 at t_2, on which there are two expanding lesions, one each

of late blight, with proportions XL_1 and XL_2 at t_1 and t_2 respectively, and early blight with proportions XE_1 and XE_2 at t_1 and t_2, respectively. Then the apparent infection rate for early blight ρE, corrected for both host growth increase and competition by late blight is given by:

$$\rho E = \frac{1}{t_2-t_1} \log_e \frac{m' \quad XE_2 \quad (1-XE_1)}{XE_1 \quad (1-XE_2)} \quad \text{..... eq. 6}$$

Where $m' = \frac{y_2\text{-absolute late blight lesion area}(al_2)\text{at } t_2}{y_1\text{-absolute late blight lesion area } (al_1)\text{at } t_1}$

Equations 3 and 4, on which GAUSE is based, are linked by corrective subtractions of the proportions of late blight L, and early blight E, respectively. In equation 6, these corrections are made to m' so that if differentiated, equation 6 could be used directly as a third equation linked to equation 3 and 4 thereby modifying GAUSE to take account of increased host growth as well as competition. Unfortunately differentiation of equation 6 is not simple or explicit because m' needs to be calculated in terms of an equation for the relative growth rate of the amount of host tissue, y, as follows:

$$\frac{dy}{ydt} = \frac{d}{dt} \log_e y \quad \text{..... eq. 7}$$

Where y = Absolute host leaf area.

Equation 7 introduces the problem of y measured in absolute areas whereas diseased area, in equation 6 and elsewhere in

epidemiology, is measured as a proportion. Therefore, since most disease ratings are done as relative estimates of the proportion of disease to quantity of host, an explicit growth function for y, and also for the terminal potential value of y, i.e. the hosts maximum growth potential, would first have to be determined. Having this information, equation 5 could be incorporated into GAUSE to simulate specific competitive interactions for that host under the defined growth conditions.

Mathematically, the problem of inter-pathogen competition concurrent with increasing carrying capacity, is formidable and has not been attempted, or apparently even been considered, in other mathematical investigations of the competitive displacement principle (Gause, 1934; Pielou, 1976; Jeffers, 1978). Nevertheless it is an approach of vital importance to pathosystem management and will be pursued by the author subsequent to this thesis.

The competition quotient : a new parameter of competitive interactions

Arising from the foregoing considerations, the concept of a competition quotient, CQ_x, is proposed to provide a measure of the status of the competitive interaction when the amount of host tissue is static. It is given by the ratio of the amount of disease in the absence of competition to the amount of disease when competition is operating. Thus in Figure 8 $CQ_x = xa_t \div xb_t$ where xa_t and xb_t are hypothetical amounts of early blight in the absence and presence

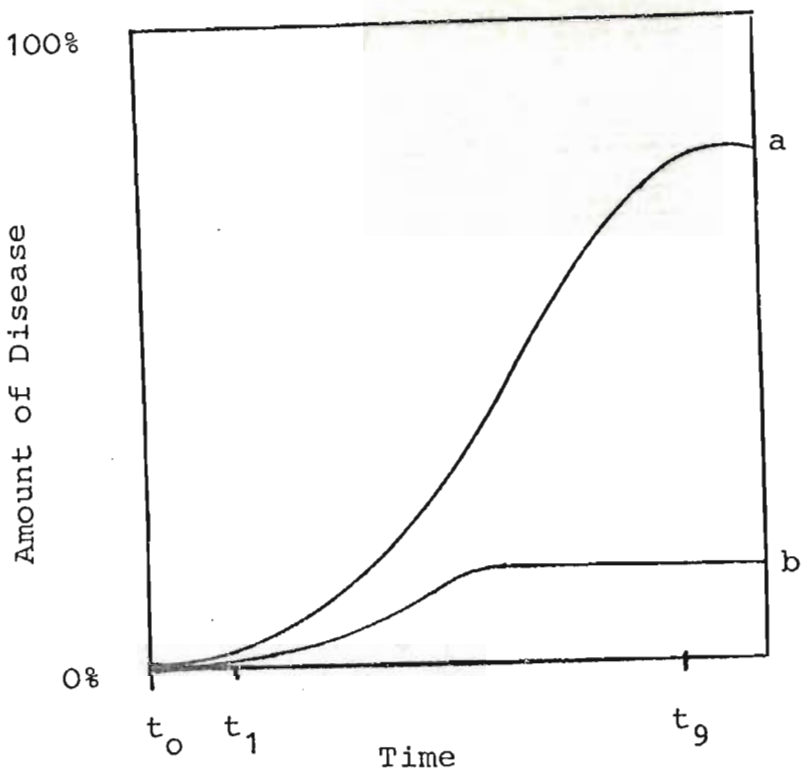

Fig. 8 Hypothetical early blight progress curves simulated by GAUSE; a - without competition; b - disadvantaged by competition from late blight. Progress curve for late blight not shown.

Alternatively and more simply, graph a in this Figure may be plotted to represent the late blight curve, i.e. the aggressor and graph b the early blight curve, i.e. the pathogen disadvantaged as a result of the competitive interaction.

CQ plotted as a function of time will depict the nature and extent of the competitive interaction. The graph of CQ = f(t) for the example in Figure 8 is as follows:

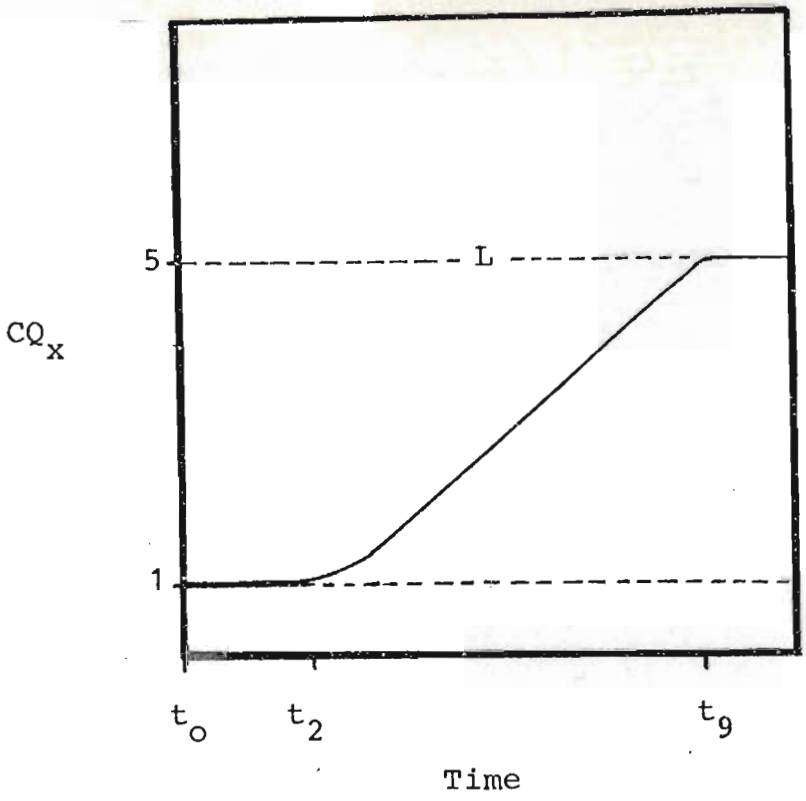

Fig. 9 CQ_x as a function of time. Values of CQ_x are calculated from Figure 8.

In the period between t_o and t_1 the sum of the proportions of xa and xb is less than 0,05; i.e. the apparent infection rate is logarithmic (Vanderplank, 1963). During this period host tissue is abundant relative to disease and competition from late blight will not limit the growth of early blight. After t_1, CQ_x increases slowly initially and then at a constant rate until it levels off at T_9, approaching an asymptote L. The value of L directly measures the maximum intensity of the suppressive effect of late blight on early blight under the prevailing circumstances. Thus, associated with each particular growth rate of late blight and its concomitant suppressive effect on early blight, there is a unique value of L. When there is no competition $CQ_x = 1$ and the graph of CQ_x will coincide with the asymptote L = 1; values of $CQ_x > 1$ directly measure the competition effect; i.e. CQ_x increases proportionally to the intensity of competition

In practice graphs such as those in Figure 8 will not be smooth. Consider a hypothetical case where late blight (the aggressor) is competing with early blight in a potato crop (Fig. 10a). The disease progress curves of each disease will tend to be irregular due to fluctuations in environmental and other factors, such as host resistance. Assuming both pathogens are established on the same day, their respective mean apparent infection rates may be calculated from the slope of the regression line when $\log_e x/(1-x)$ is plotted as a function of time.

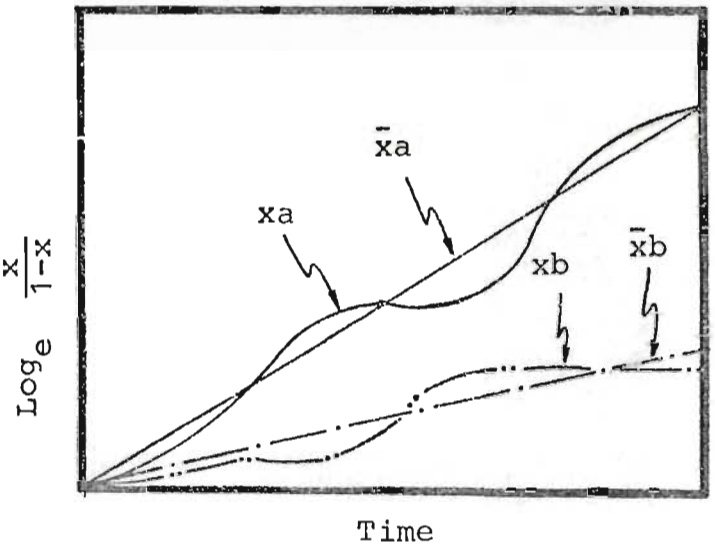

Fig. 10a Disease progress curves of two diseases a and b. Actual amounts of disease xa and xb as well as their respective regression lines $\bar{x}a$ and $\bar{x}b$ are plotted.

If CQ_x is calculated from these mean regression lines the graph of $CQ_{\bar{x}}$ will be a straight line parallel to the x-axis. It will be the asymptote of mean competition L and will have a value greater than one. However, if CQ_x is calculated from actual values of the proportions of disease at each instant of competition the graph of CQ_x will fluctuate about

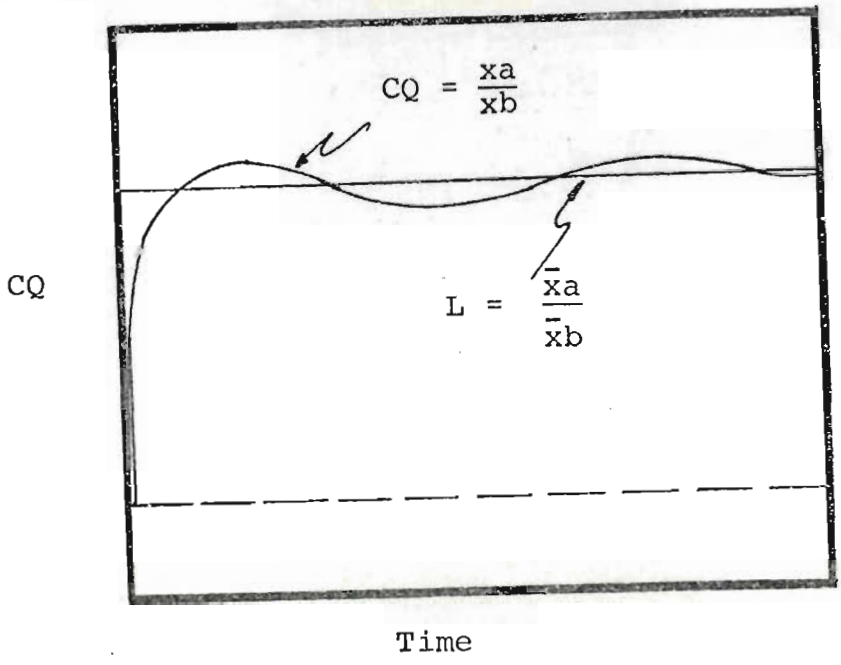

Fig. 10b Competition quotient calculated for actual amount of disease caused by pathogens a and b fluctuating about an asymptote L; the mean competition asymptote calculated from $\bar{x}a \div \bar{x}b$ for each instant of competition.

When the derivative of $CQ_x = f(t)$ is calculated and plotted as a function of time it, together with the graph of $CQ_x = f(t)$ will facilitate interpretation of the competitive interaction. For instance, when the graph of CQ_x is above the asymptote of mean competition L the aggressor pathogen is increasing its domination of the competitive interaction; when $CQ_x < L$ the disadvantaged pathogen increases its growth due to reduced competition from the aggressor. These changes in the level of CQ_x could be measured by the derivative of CQ_x.

These mathematical manipulations will have important applications in deterministic manipulation of competitive interactions for the management of plant pathosystems. However, at this stage these will only be discussed in general, rather than

with particular reference to the Natal blight pathosystem. Once the epidemiological significance of competitive displacement has been explored the application of the CQ to pathosystems management will be discussed.

2.3.4.2 Epidemiological significance of competitive displacement

Plant pathology and the training of plant pathologists have been dominated by a one-disease-at-a-time approach (Zadoks & Schein, 1980) and it is therefore not surprising that competitive displacement has not received much attention in plant pathology. This simplistic one-disease-one-cause approach is both unexpected and unrealistic because complexity is the rule rather than the exception in plant pathosystems. For example, it is reported that potatoes are affected by at least 18 virus diseases, 46 fungal diseases, 6 bacterial diseases, 5 nematode diseases and some 40 non-parasitic maladies (Zadoks & Schein, 1979). To this must be added several insect pests and weed problems which could detrimentally affect potatoes. Not all of these occur in a single region or crop, but it is improbable to the point of impossibility, that any stand of field grown potatoes will experience only one such adverse biotic or abiotic factor at a time.

Chester in 1950 drew attention to the problem of complex etiology. To accommodate this complexity, Wallace (1978) proposes that complex etiology should be considered as much an ecological as it is a plant pathological problem. He

also questions the validity of the dogma of specific etiology and the usefulness of Koch's postulates. Barker, Schoemaker and Nelson (1976) summarized the situation by emphasizing the need to examine many factors simultaneously when elucidating the etiology of certain diseases.

This realistic ecological appraisal of plant disease suggests that the malady be viewed as being the consequence of a spectrum of interacting constraints of which the limiting value will be uniquely determined for each ecological matrix. When a group of pathogens interact to form a disease complex the nature of the dynamic competitive equilibrium amongst members of the group will determine their relative contribution to disease severity. The implications of this rather obvious ecological truism will first be discussed in relation to specific epidemiology procedures and methods. In the next section a wider, speculative evaluation of the interface between ecology and epidemiology will be made.

Modifications to fundamental epidemiological equations necessitated by competitive displacement

The data in Table 7 clearly illustrate the dangers where only one disease of what is really a disease complex is studied. If the basic logistic growth equation for early blight (equation 2) had not been modified as in equation 4 to take account of the competition introduced by late blight, incorrect early blight progress graphs would result.

In equation 6, the parameter m' is introduced for the competitive situation as a modification of Vanderplank's (1963) equation 8.2, reproduced below as equation 8, where m is introduced because of host growth, to correct the apparent infection rate of a pathogen occurring alone. The biological significance of this correction factor needs to be clearly understood because it may be modified to produce a competition quotient CQ, a new parameter which measures competitive displacement.

$$\rho_a = \frac{1}{t_2 - t_1} \log_e \frac{m_a x_2 (1-x_1)}{x_1 (1-x_2)} \quad \text{..... eq. 8}$$

Where ρ_a = Corrected infection rate for pathogen a;

$m = y_2 \div y_1$, where y_1 and y_2 are the amounts of susceptible host tissue at times t_1 and t_2, respectively;

and x_1 and x_2 are the *proportions* of disease at times t_1 and t_2, respectively.

Here m is given for a specific time period. A more general statement, which suggests that $m_a = f(t)$ is given by:

$$m_a(t) = \frac{y(t)}{y(t_o)} \quad \text{..... eq. 9}$$

Thus, when a second pathogen, b, is concurrently competing with pathogen a for the same increasing amount of host tissue the competitive effect of pathogen b on m_a in equation 8, may be represented by m'_a such that:

$$m'_a(t) = \frac{y(t) - b(t)}{y(t_o) - b(t_o)} \quad \text{..... eq. 10}$$

Where $b(t_o)$ and $b(t)$ are the amounts of disease caused by pathogen b at times t_o and t, respectively. This removal effect of the second pathogen reduces the magnitude of the correction needed to the apparent infection rate of the first pathogen on account of host growth.

Graphically, the effect of competition between pathogens a and b when host growth is also considered, may be depicted as in Figure 11.

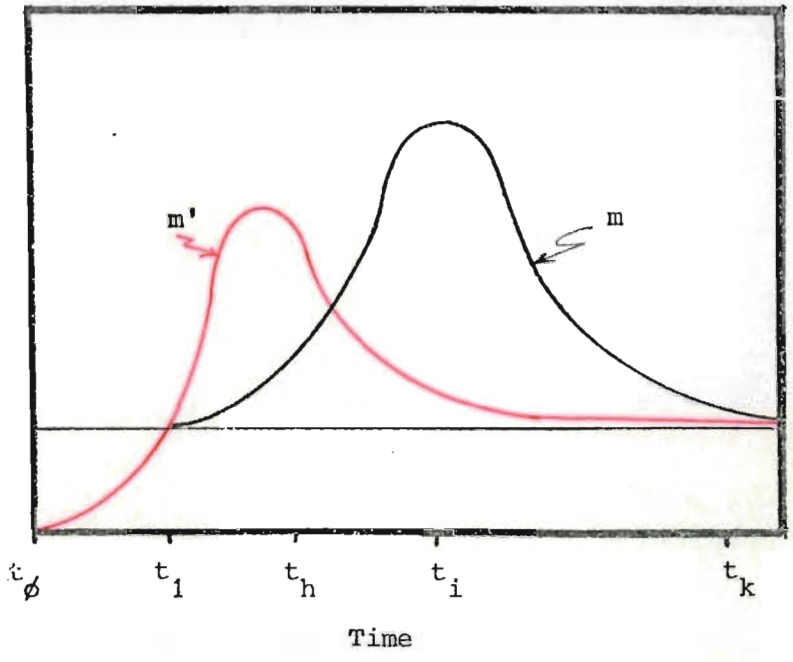

Fig. 11 Changes in the relative increase in the amount of susceptible host tissue, m, not corrected for removals by a competitor and m'a corrections to the ratio of pathogen a in terms of competition from a second pathogen.

The value of m_a is necessarily initially 1, from which it increases to a maximum value at time t_2, thereafter it decreases to approach a limit or asymptote value 2. As the value of m_a tends finally to 1, e.g. at t_k, host growth very nearly stops and estimates of the apparent infection rate r, no longer require correction for this factor.

When on the other hand, a competitive interaction occurs between pathogensa and b with the consequence that b, by virtue of a faster rate of increase, limits the growth of a, the initial value of m'_a will tend to a limit of zero. It will initially increase to intercept the asymptote, then increase to a turning point, e.g. at t_i and finally decrease to approach a limit value of 1, e.g. at t_j. At time t_j the more aggressive pathogen, b, is multiplying at a rate that in effect, precludes any of the amount of host tissue produced by host growth,from being available to pathogen a. Thus the need to correct the apparent infection rate of pathogen a in terms of host growth falls away sooner in the presence of competition than in its absence, the time difference being measured by $t_k - t_j$.

There will be a corresponding effect of pathogen a on m_b. However, since b is the more aggressive competitor the curve of m'_b will deviate less from m_b. In general, the more aggressive the one pathogen, the sooner will the need to correct the apparent infection rate of the other pathogen disappear. Thus, unless competitive interaction is specifically considered and corrected for, application of Vanderplank's (1963) equation 8.2 could lead to inappropriate correction of the apparent infection rate in question.

Dividing equation 8 by equation 6 yields two ratios, one for the left-hand-side (l.h.s.) and one for the right-hand-side (r.h.s.). The former, $r \div \rho$ is the ratio of the apparent infection rate to the rate corrected for host growth and

competition. This ratio can be used as a competition quotient but its equivalent, resulting from division on the r.h.s., i.e. $m \div m'$, is likely to be more useful because it can be readily calculated from the quantities routinely measured in epidemiological studies, i.e. the amounts of new host growth and increased diseased areas. The ratios $r:\rho$ and $m:m'$ are interchangeable and when each is plotted as a function of time, their graphs will differ only in matters of scale. Thus, when existing progress curves are evaluated for competitive interactions, $CQ_r = r:\rho$ may be used when infection rates are compared; $CQ_x = x_a(t) : x_b(t)$ when the proportions of disease are compared and $CQ_m = m:m'$ when the effect of competition on new host growth is evaluated. This notation will be followed for the remainder of this thesis with the exception that when CQ is used without a subscript, it will refer to the competition quotient in general.

The function of CQ_m is similar to that of CQ_x, represented in Figure 8, and can be calculated using the corresponding graphs of $m_a = f(t)$ and $m'_a = f(t)$ in Figure 9. It likewise measures the intensity of competition, i.e. when CQ_m is at a maximum the more aggressive pathogen b, achieves its maximum removal effect on the increased level of susceptible tissue which would otherwise have been available to pathogen a. The effect for example, of increasing still further the aggressiveness of pathogen b would be to alter the shape of the curve of $m'_a = f(t)$ in three possible ways, viz:

(i) Advance the time of the turning point, t_h,

towards t_o and reduce its altitude;

(ii) Change the symmetry of the curve, e.g. the derivative of $CQ_m = f(t)$ may change differently in the intervals $t_o - t_h$ and $t_j - t_h$;

(iii) Advance the time when the terminal value of 1 is reached.

There are many possible conceptual variations of the curves presented in Figures 8 and 9 and the consequential ratios of CQ_x and CQ_m. The first and second order differentials of the competition quotients might yield important new insights to epidemiology theory. However, in the absence of specific experimental data, further speculation would become too theoretical. It is clear, however, that competitive displacement, which is likely to be found to be as commonplace as it has so far been overlooked, could markedly alter the application and interpretation of the most fundamental epidemiology equations.

Changes in fungicide control strategy occasioned by competitive displacement

The one-disease-at-a-time approach which has dominated plant pathology (Zadoks, *loc. cit.*) has a corollary in the fungicide industry. Here chemicals are tested against specific pathogens one at a time so that their spectrum of activity can be determined. However the pharmacological principle of a known dose for a known cause has the consequence that the wider ecological implications of the introduction of a

new fungicide are not considered on an *a priori* basis.

The results obtained in this thesis whereby the differential control of late blight by Ridomil allowed early blight to increase (Tables 2 and 3) suggest that narrow spectrum vertical fungicides (*sensu*: Robinson, 1976) could initiate a boom-and-bust cycle which could conceivably reverberate throughout the entire agro-ecosystem. Epidemiological principles dictate that in Natal early blight will increase proportional to the widespread use of Ridomil. The greater epidemiological competence of early blight (*loc. cit.*) and deficiencies in current fungicides for the control of this disease have already been discussed. However, it must be borne in mind that the efficiency of a fungicide is tied *inter alia* to the infectiousness and apparent infection rate of the target pathogen. The value of a fungicide decreases geometrically as the apparent infection rate of the target pathogen increases (Vanderplank, 1963 - paraphrased). Thus the present value of early blight fungicides could be eroded as early blight intensity increases, necessitating *inter alia*, earlier commencement dates for spraying, development of new fungicides and the increase of host resistance. In terms of host resistance, late blight has been studied extensively (Vanderplank, 1963, 1968) whereas in comparison, early blight has not. Thus the introduction of Ridomil could have the effect of putting the clock back by necessitating approaches to early blight resistance studies which were taken many years ago for late blight.

On the basis of the data presented in this thesis and the growing awareness of the possible deleterious consequences of haphazard *ad. hoc.* fungicide introductions (Dekker, 1976; Zadoks and Schein, 1979), a good case can be made for a re-evaluation of the criteria used by government agencies to decide on the introduction and registration of new fungicides. For example, in addition to the Government agencies requiring residue and toxicity data which facilitate an evaluation of the potential repercussions that the chemical might have in the food industry, information should be gained concerning other harmful side-effects that could leave the farming community more vulnerable tc a "new" pathogen or a "super" race of the same pathogen. Thus instead of registering a fungicide for use against one or more specific organisms it could be registered as being safe to use in a particular agro-ecological context. A policy which registers fungicides for use in certain pathosystems and not in others is bound to be unpopular and difficult to implement. However, these obstacles cannot deny the basic epidemiological principles which validate these arguments.

Zadoks and Schein (1979) describe "inversion of dominance" as the process which takes place when fungicide application leads to a decrease of populations of microorganisms so that these no longer antagonize other microorganisms which can then develop freely and eventually cause disease. Whilst this is a rather narrow and specific application of the competitive interaction principle and is too limited to meet the wider ecological implications of complex etiology, it nevertheless

specifically describes the consequence of Ridomil spraying in Natal on the late blight-early blight interaction. It suggests a two-rôle situation, in which alternatives of dominance or subjugation may be reversed under certain fungicide regimes. In contrast, competitive displacement has no connotation of being limited to a certain number or specific inversion of rôles. Instead, many permutations of rôles are implied, each particular permutation having its own consequence and configuration. Therefore, competitive displacement is favoured in this thesis as being more versatile and consistent with the concept of complex etiology described by Wallace (1978).

Zadoks and Schein (1979) present data which show that field applications of benomyl on rye led to a decrease of symptoms caused by *Fusarium* spp. and *Cercosporella herpotrichoides*. However, sharp eyespot, caused by *Rhizoctonia* species, increased from 2% incidence to 35% in the sprayed crop. The authors quote this as an example of "inversion of dominance" although it would seem difficult to define precisely how this term applies. In a similar example ethirimol used in barley to control powdery mildew, causes *Helminthosporium sativum* to increase significantly. This is apparently due to the fact that ethirimol promotes germination of *H. sativum* conidia whilst increasing host susceptibility to *H. sativum* (Saur, 1976; Schuette & Diercks, 1975).

Selective, systemic fungicides may also interfere with biological control mechanisms. Bollen (1975, cited by

Zadoks and Schein, 1979) found that a benomyl resistant strain of *Botrytis cinerea* could be controlled by strains of *Penicillium brevicompactum* and *P. stoloniferum* more tolerant to the fungicide than the strain of *Botrytis*. *In vitro* the *Penicillium* strains showed antibiosis toward *Botrytis* but could effect control only in the presence of benomyl.

Fungicides may apparently also detrimentally affect biological control by insects,as reported on cucumbers where red spider mites are controlled by the predator mite *Phytoseiulus persimilis*. When benomyl is used to control some of the cucumber diseases it sterilizes the predator mite, thus annihilating the biological control (Woets, 1972).

In these examples differential control process are obviously involved and the data serve to reinforce concern expressed in this thesis for the possible consequences of such fungicides on ecological equilibria. Conceivably, these differential activities might be manipulated to alter complex ecological interactions to the benefit of the particular cropping system.

Deterministic manipulation of competitive displacement in an integrated pest control strategy

When a group of parasitic organisms interact to cause disease their total negative effect on the host will be governed in part by their mutual competition, antibiosis, antagonism and pathogenesis. This suggests that biological control may be

defined, in part, as the exploitation of these negative interactions among members of the parasitic group so as to minimize their collective negative influence on the host. Integrated control strategies, on the other hand, include selected aspects of biological control as components along with the many other cultural, chemical and crop factors such as resistance and tolerance. Thus, in an integrated disease control programme the differential control properties of fungicides may be used deliberately to alter the degree and nature of the interaction which determines the pest status of the group.

When these challenging crop protection concepts are more widely accepted as feasible the pathosystem manager will become aware that he is in fact attempting to manipulate competitive relationships. In this situation the competition quotient would serve to identify which manipulations of competitive interactions will produce the optimum integrated control strategy.

Consider the example of late blight and early blight for which the results in this thesis indicate that management of the blight complex should be sought rather than the control of its individual components. In this pathosystem *there is an optimum level of control of late blight, which, although less than the maximum desirable when late blight only is considered, will interact most favourably with the other constraints on early blight development so that this interaction in turn, yields the optimum degree of control of the*

blight complex.

Infection rates of late blight and early blight are differentially affected by many determining factors, such as fungicide type, weather pattern and host growth factors. The many permutations of combinations of these factors will each have a specific consequence for the infection rates of the blight pathogens. Assuming that the component effects can be quantified - and many have been, e.g. the effect of different fungicides on the apparent infection rate, r, of late blight (Hooker, 1956, cited by Vanderplank, 1963) - the difficult choice remains of having to decide which *combination* of apparent infection rates of late blight and early blight would give an optimum competitive interaction between these organisms such that the deleterious consequences of the complex can be minimized. This is precisely the function of the competition quotient. GAUSE can simulate the specific condequence of particular control strategies on the apparent infection rates and *CQ can then be maximized over the period of the interaction between the pathogens at a value giving the desired degree of control of the disease complex.* Implicit in this concept of CQ maximization is the fact that CQ has an optimum value which may be determined for any instant on the disease time scale, but also more realistically and typically, for discreet time periods. For example, a certain value CQ_x, resulting from disease ratios determined by a particular combination of r_a and r_b, where a and b are two different pathogens, may be desirable at one host growth stage but not at another.

Consider the possible application of CQ as an evaluator of the optimal use of *Peniophora gigantea* in the biological control of *Fomes annosus*, causing root, heartwood and sapwood rots of certain pines. Rishbeth (1963) developed a technique in which laboratory-produced conidia of *P. gigantea* are mixed with talc and thenmoulded and dried to form tablets. Each tablet, dissolved in 100 ml of water, yields a suspension of 10^6 viable conidia per ml, enough to inoculate 100 stumps 40 cm in diameter, in which *P. gigantea* then becomes the dominant pioneer, competitively excluding the less aggressive *F. annosus*.

There are at least two possible areas in this control programme where applications of the CQ-concept would be valuable. Firstly, if different strains of *P. gigantea* are discovered they may be indexed in terms of the CQ to determine which strain gives the best control of *F. annosus*. Secondly, CQ can be used to determine the optimum concentration of *P. gigantea* inoculum. Conceivably there may exist a combination of *P. gigantea* strains, each with its own desirable inoculum concentration, that in combination, will best control *F. annosus*. The CQ can assay these permutations of possible combinations and identify the best.

Cultural practises such as soil preparation methods may also differentially alter the competitive interaction among a group of plant pathogens. For example, *Septoria passerinii* on barley and *Gibberella zeae* on maize and wheat are favoured by the change from conventional soil preparation methods to

those involving minimum tillage (Yarham & Hirst, 1975). Minimum soil tillage has many agronomic advantages but its impact on soil plant pathosystems could fairly simply be evaluated by means of the CQ.

Consider another theoretical example in which competitive interactions may be manipulated deliberately, to exploit the phenomenon of tolerance to plant disease. According to Schafer (1971) the yield of a diseased, tolerant variety is not seriously affected in spite of the pathogen having a high apparent infection rate. Suppose now that such a host is attacked by several different pathogens, each with a different apparent infection rate. If the host is tolerant to the pathogen with the highest infection rate and susceptible to all the others where yield loss is proportional to disease intensity, then the aggressive, tolerated pathogen has the potential of competitively displacing those pathogens with a lesser apparent infection rate should they occur together on the tolerant host. One of the various possible interactions between the tolerated pathogen on the one hand and the remaining pathogens on the other, will result in a particular disease complex which will cause the least amount of disease. This interaction could be identified by the CQ and is an example of biological control in which competitive displacement is deliberately manipulated to achieve a particular goal.

These simple examples of possible applications demonstrate the value of CQ. They also do not reveal any obstacle to

the extrapolative assumption that it may prove to be an extremely versatile and valuable strategic aid to pathosystems management strategy. It may also be employed in analytical studies of compensation phenomena in the categories listed by Rotem (1978) and Aust, *et al.*, (1980). It is a parameter ideally suited to studies at many integration levels and can be adopted without modification in computer simulation studies aimed at evaluating the consequences of certain permutations of integrated control methods.

2.3.5.0 Conclusions

Competitive displacement in plant pathosystems has been undeservedly ignored because it has the following significant implications for pathosystems analysis and management:

(i) It may alter interpretation and application of basic epidemiology equations when these involve either a constant or changing amount of host tissue;

(ii) It reflects the nature and level of interactions in pathogen complexes and determines that these complexes should be evaluated as entities rather than in terms of the individual effects of the constituent members.

(iii) It can alter the value of control methods should these have to be applied in combination. In the potato blight complex this means that Ridomil cannot necessarily be used to control the late

blight in the complex merely because it is efficient when late blight only is present. The use of Ridomil must be dictated by its effect on the blight complex and its interaction with other factors which determine the amount of early blight.

(iv) It can be measured simply by a new versatile parameter the Competition Quotient CQ. This quotient can practically be applied in experiments which evaluate competitive interactions and theoretically in simulation studies where the aim is to develop an optimum integrated control strategy.

SECTION III PATHOSYSTEMS MANAGEMENT

3.1.0.0 THE PATHOTOPE : A PROPOSED NEW PATHOSYSTEM MANAGEMENT CONCEPT

3.1.1.0 Introduction

The paradigms introduced into epidemiology by Vanderplank (1963) resulted in a typology of disease at the population level which led to the description of epidemiology as an applied aspect of ecology (Oort, 1972). This trend towards an increasing ecological perspective of plant disease was further consolidated when Robinson (1976) introduced the concept of the pathosystem.

The holistic ecological approach implicit in the pathosystem concept introduced a major problem into plant pathology. Whereas plant disease fieldwork hitherto had customarily involved equal sized plots in an uniform experimental area, plant pathologists now had to adjust conceptually to a situation where populations of diverse diseased farms constituted the proper domain of epidemiological analysis. Most of the other aspects of plant pathology demanded similar conceptual re-alignment or quantum conceptual shifts in which ideas previously held, assume a new dimension (Chiarappa, 1976).

Many of the familiar methods and approaches provided valuable transitional concepts for those plant pathologists inclined to explore population phenomena. However, where these con-

cepts were unaccompanied by the required mental shift, this amounted to a simple re-labeling rather than a re-evaluation in terms of the Vanderplankian insights.

An epidemic can be defined as being a highly compatible interaction between a pathogen, its host and a suitable environment. Disease forecasting methods have hitherto relied only on the interaction between two of the disease triangle components, i.e. the interaction between pathogen and environment. In contrast, an epidemiological analysis at a higher systems level of integration would require the disease forecasting system to be based on wider interactions. Such a perspective is given in this section.

The pathotope concept is also introduced here. It is a new concept for the management of plant pathosystems and develops from a need to classify the elements of epidemic patterns in a generic sense. Once these elements are identified their contribution to the epidemic can be determined quantitatively.

The pathotope concept also provides a practical basis for comparative epidemiology which hitherto has been largely theoretical (Kranz, 1974a, b, 1978, 1980; Zadoks & Schein 1979, 1980, Palti & Kranz, 1980). As a relatively new subdicipline of epidemiology, comparative epidemiology needs to develop its own methods, approaches and concepts. Since comparison presupposes the existence of units of comparison the development and typology of appropriate units amenable to

quantitative analysis are thus a high priority. The pathotope concept introduced in this thesis could find a place in a hierarchy of units of comparison. It could also provide a means whereby comparative studies of epidemic patterns at higher systems levels could become quantitative and less descriptive. The reliance on description is a shortcoming which the pathosystem concept generally has in common with the ecosystem concept from which it was derived. Notable exceptions to this are the quantitative,population genetic models presented by Robinson (1976).

In the case of ecosystems one could, for example, compare a Savannah ecosystem with that of a primary rain-forest but each of these could be redefined if a different perspective is taken. For example, instead of these phenomenological descriptions, these ecosystems could be described in terms of their respective dominant components. The inter-relationship between specific components of ecosystems can be quantified; for instance, one might calculate that every ten units of prey support one predator. However, there is no one universal cardinal index system for ecosystems.

Pathosystems management pre-supposes that analysis and, specifically, quantitative analysis is required to identify and measure the contribution of each sub-system component to the state of the system. For deterministic control of disease it is essential to know how much each sub-component contributes to the epidemic,in order to identify those intervention points which would lead to the greatest reduction of disease for the minimum input.

A disease forecast applies to a pathosystem. Its validity and accuracy will vary from one pathosystem to another. To improve its value, the elements in each pathosystem which influence the accuracy of the warning system must be identified and submitted to quantitative analysis in order not only to improve the regional accuracy of the warning system, but also to anticipate, by means of extrapolation, the validity of the forecasting paradigm in other areas.

The term "ecotope" was introduced by Whittaker, Levin and Root (1973) and is an operational procedure for the quantitative analysis of the abstract ecosystem concept. Just as the pathosystem is based on the ecosystem, so too with modifications, is the pathotope derived from the ecotope.

3.1.2.0 The ecotope as antecedent of the pathotope

Whittaker, Levin and Root (1973) addressed the confusion in the ecology literature surrounding the use of the terms 'niche' and 'habitat'. They aimed to assign clear meanings to these terms and also introduced a powerful new concept on which they conferred the label 'ecotope'.

According to Whittaker *et al.*, (1973) the ecotope may be envisaged as an abstract representation of a conceptual hyperspace as in Figure 12 with habitat, niche and population variables as axes.

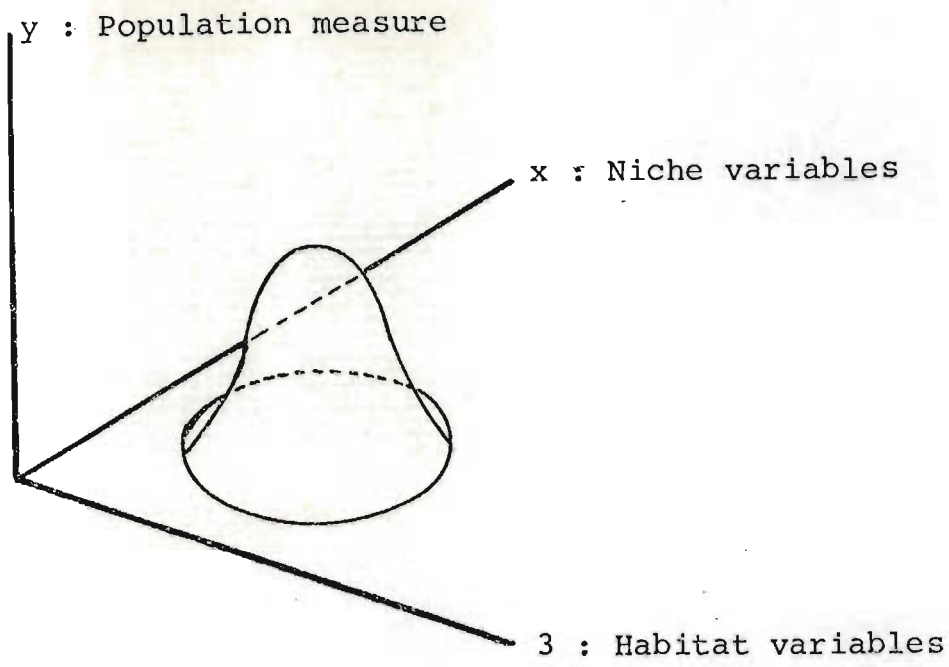

Fig. 12 A diagrammatic representation of the relations between Niche-and Habitat-variables as they determine the Ecotope. (Reproduced from Whittaker *et al.*, 1973).

The habitat variables are described along a third dimensional, Z-axis. This is a simplification of the habitat variable because rather than a single axis, it should be thought of as an m-dimensional habitat hyperspace. Similarly, the niche variables described along the x-axis are more correctly n-dimensional niche hyperspaces. One can then visualize these n-dimensional niche hyperspaces built upon the m-dimensional hyperspace as a base space to form an (m + n)-dimensional ecotope hyperspace which in Figure 12 is reduced for illustration, to a plane of habitat by niche variables. Finally the population measure superimposed on the (m + n)-dimensional hyperspace creates the (m + n + k)-dimensional surface pictured - the ecotope. Of the possible number of population variables, only one is plotted as the y-axis as an example.

Whittaker *et al.*, (1973) present examples of hyperspace diagrams. However, these are difficult to visualize especially when constructed as three dimensional volume diagrams in which some 'hypervolume' is subtended by two other volumes. Modern mathematical and theoretical ecology often has this esoteric property of complex models requiring a large degree of imaginative abstraction (e.g. Pielou, 1976).

Epidemiology can make an important contribution to this area of quantitative population ecology. In many ways plant pathosystems are among the simplest sub-systems of ecosystems; i.e. when considering systems which are not sub-divided beyond the point at which their systems-level complexity is lost. In a simple pathosystem such as a disease triangle involving one host population, one pathogen population and a few key environmental factors, the systems components are encountered near their lowest level of interaction, consistent with their being recognizable as within the ambit of the systems concept. Plant pathosystems are therefore ideal candidates for experimental exploration of complex ecological theories.

The ecotope concept (*loc. cit.*) has been defined at a high level of abstraction. It may, therefore, prove difficult for it to become the basis of practical ecology investigations. However, if its essential properties are distilled and applied to plant pathology, the synthesis leads to the pathotope concept which can have important practical

advantages for pathosystems management. These properties are the quantitative contribution of niche and habitat variables and their interactions as determinants of the quantitative behaviour of the population to which these niche and habitat factors apply.

3.1.3.0 Definition and epidemiological basis of the pathotope

By definition an epidemic occurs in a particular place, e.g. a community, and at a particular time. It is this *bounded* property of an epidemic which underlies the assumption that epidemics can be compared. It is assumed that some discrete epidemic pattern with its own identity exists which can be compared with another uniquely defined epidemic.

Consider the growth of an epidemic as disease spreads from a focus. The disease will spread, sometimes along a clearly defined gradient until it occupies all the available space which, in this case, will be the host community. There are many constraints which will determine the boundaries of an epidemic and consistent with ecological terminology and concepts, it is valid to say that the pathogen causing the epidemic has a *habitat* to which it is confined. Similarly, and consistent with the Eltonian functional niche concept (Elton, 1927) the pathogen *niche* is the status of the pathogen in its habitat. When the habitat x niche interaction is conducive to rapid pathogen development i.e. high values of the apparent infection rate, r, the epithet epidemic is used as a population measure of pathogen activity. The inter-

action between habitat, niche and apparent infection rate delimits the epidemic pathotope. The bounded property of an epidemic, i.e. each epidemic has "its place", and the suffering induced by epidemic disease are the etymological roots of the pathotope concept (*pathos* = suffering; *topos* = place; hence: pathotope).

It is important to note the distinction between pathotope and pathosystem. In a pathotope a population parameter, for example the apparent infection rate, *quantitatively measures* the events and interactions between the components of the pathosystem.

At a higher systems level, the pathotope emphasizes common denominator components which quantitatively determine communal vulnerability. All those populations or members of the group equally vulnerable to the same kind and amount of threat constitute a pathotope. Thus, pathotope analysis attempts to sort and classify populations into groups according to measured epidemiological criteria so that a common defence strategy can be implemented. Because sorting and classification involve comparison it is clear why the pathotope may be regarded as the unit of comparative epidemiology at the community level.

In contrast, the pathosystem is abstractly defined in terms of parasitism as the central characteristic which determines the behaviour of the system (Robinson, 1976). Thus one might typically think of a pathotope as a sub-pathosystem

although it can be defined at higher systems levels, e.g. when the wheat pathosystems of Australia are at risk to *Puccinia graminis* f. sp. *tritici* inoculum from southern Africa (McIntyre, p.c.).

Consider further the implications of an epidemic confined to a specific community. If this community comprises several host sub-populations,e.g. a number of potato farms, it emerges that this community of farms may be uniformly at risk and open to epidemic disease attack without a uniform infection rate throughout the pathotope being necessary. Thus for practical pathosystems management purposes a pathotope is defined as an area or community in which the individuals are equally at risk to the disease with a certain probability at a given time. Expressed differently, a pathotope is an area in which certain crop protection tactics are adopted as common denominators by all the farmers whose crops constitute the pathotope community.

Life-boat theory also provides a useful analogy for the understanding of a pathotope i.e. each pathotope may be regarded as a lifeboat into which individuals are placed by force of circumstance and in which they find it imperative to define and submit to a common survival strategy. Similarly a particular farmer is vulnerable because he belongs to a group. He also contributes to the vulnerability of other members of the group and it thus follows that there is some common denominator strategy which applies to the group and is uniquely determined by the nature of the group.

The pathotope concept explores and exposes key epidemiological parameters which may form the basis of the group strategy. The responsibilities of individual group members are identified so that they may be manipulated in order to decrease the vulnerability of the group as a whole.

A comparison with medical epidemiology could also be useful at this point. When human lives are threatened the responsible authorities feel no compunction about limiting individuals because of the danger they pose to the group. The extent to which an epidemic, as a group-bounded event, is appreciated in medical epidemiology may be gained from the emphasis on quarantine and uniform vaccination.

In contrast, individual farmers may choose whether or not to control their crop diseases. In general there exists no authority which enforces uniform control measures. Quarantine measures which are preventative, are the notable exceptions. On the other hand a gene deployment strategy to limit susceptibility and the breakdown of resistance is usually left entirely on a voluntary basis.

This must not be construed as an argument for authoritarianism or enforced, legally controlled crop protection. Instead the pathotope concept must be viewed as a rational explanation to a group in order to convince them of their fundamental interdependence. In this way it can serve as a powerful motivating argument for each group to identify the crop protection tactics which all members should practise in common.

The author has found that farmers are readily convinced about the validity of a common group strategy. One only needs to ask if any of them would consider deliberately leaving a portion of a field unsprayed while diligently protecting the remainder. Their emphatic denials of the wisdom of such a decision can then serve to establish the principle that untreated disease in crops,places other healthy crops at risk. By extrapolation of the example from within fields to between farms and further to within districts, one readily establishes the concept of the bounded interdependent group, characterised by a communal vulnerability.

3.1.4.0 The pathotope in relation to other examples of geographic disease studies

Weltzien (1972) distinguishes between "patho-geography" (*sensu*: Reichert & Palti, 1967) and "geophytopathology" (*sensu*: Weltzien 1967). The former refers to the geographic distribution of plant diseases,their places of origin and migration routes,while the latter incorporates additionally, causal explanations in terms of weather-effects, for example, in the study of the distribution phenomenon. According to Weltzien (1972) mapping of plant disease occurrence is still the most commonly used geographic approach.

The pathotope approach described in this thesis may thus most appropriately be viewed as a particular example of a patho-geographical approach with the emphasis on epidemiological criteria which determine group (≡ area) vulnerability

rather than *a posteriori* mapping of disease intensity, spread or economic significance measured in terms of damage caused.

Examples of geographical approaches which have elements in common with the pathotope concept are those of Zogg (1959), Drandarevski (1969) and Steudel & Heiling (1949). The first author mapped zones of disease intensity at different times. His maps show the epidemiological situation of each area at different times but no attempt was made to use this data for purposes other than the analysis of spread patterns. Zogg's (1959) work differs from most studies of disease spread, which largely concentrate on the elucidation of spread patterns as in the classic studies of Stakman & Harrar (1957), in that it combines studies of both incidence and severity.

Drandarevski (1969) made a detailed epidemiologic analysis of the environmental requirements of sugar beet mildew caused by *Erysiphe betae*. From this data he designed maps indicating *where* the fungus could be expected and *where* the disease might become severe. He indicated California as a danger area and five years later his prediction was proved to be correct (Kontaxis, Meister & Sharma, 1974). Drandraverski's (1969) studies are noteworthy because he looked at the situation on an international basis and identified areas in Eurasia, South America and Australia where disease might become severe.

Steudel and Heiling (1949) studied and mapped the intensity

of sugar beet yellows virus in Germany but made no attempt to group their observations.

One of the most vital questions in plant diesease management concerns the time of first disease appearance or the most probable first-attack date. None of the studies reviewed by Weltzien (1972) combine mapping procedures and epidemiological principles in an *a priori* strategy, specifically to control plant disease, as is the case with the application of the pathotope concept presented in this thesis.

It must be emphasized that the pathotope may be delimited in terms of any quantitative criterion provided it characterizes a community. However, in this thesis, pathosystems management is viewed as the primary objective of plant pathology and hence the pathotope is defined in terms of criteria which could lead to improved management strategies and tactics.

3.1.5.0 Identification of pathotope criteria as components of a pathosystems management strategy

To place the pathotope in perspective in terms of its intrinsic determinants, its relation to the ecotope concept and its relation to the well known disease triangle, it may be illustrated as in Figure 13.

As an example of the relationship between pathotope and ecotope and to explain the hyperspace concept, consider the

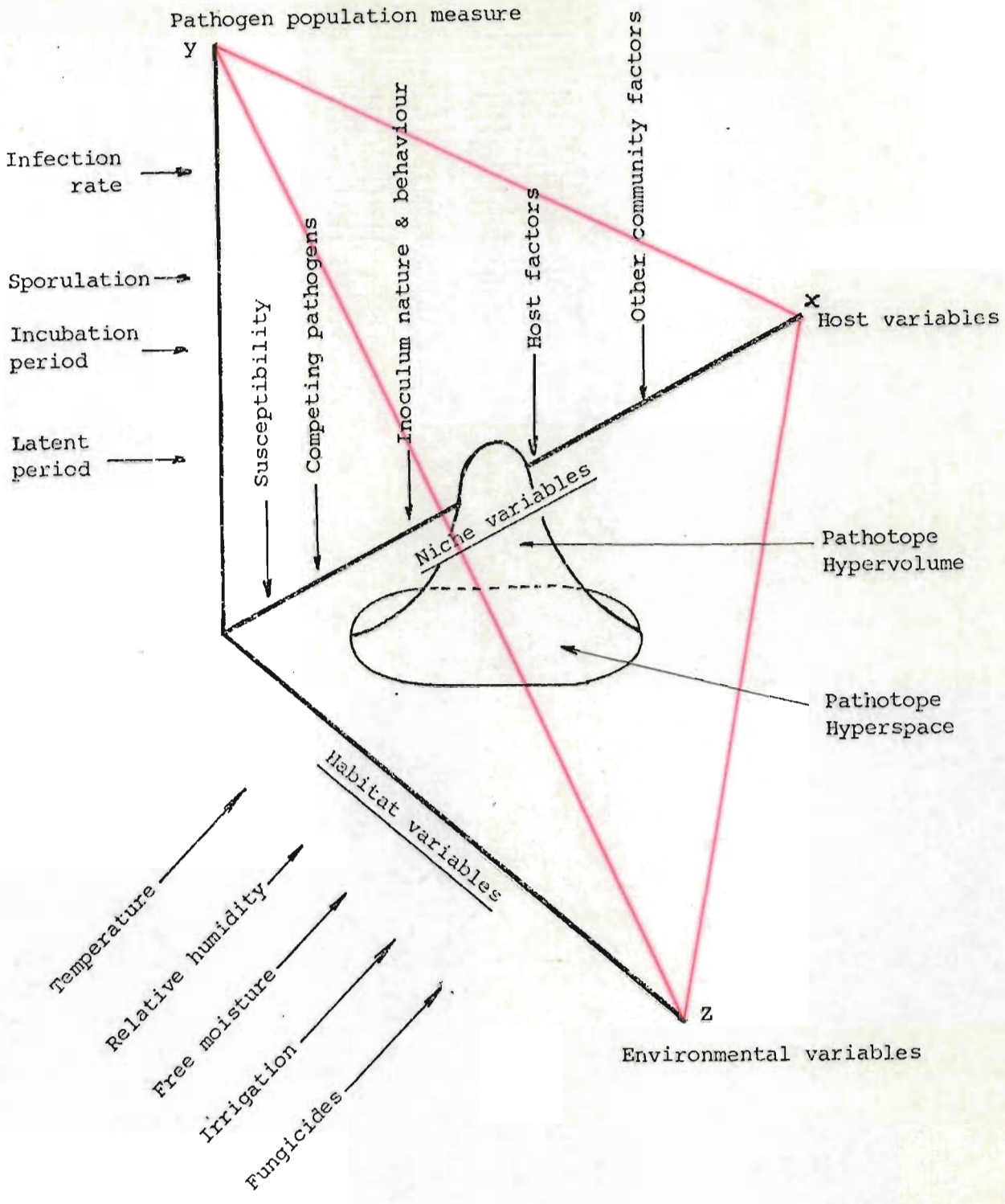

Fig. 13 A heuristic model of the pathotope with the disease triangle superimposed. Various population attributes may be measured on the y-axis, environmental factors external to the community are measured as habitat variables or the Z-axis and intra-community behavioural patterns are measured as niche-variables on the x-axis.

sporulation pathotope*1 in which sporulation (y-axis), host susceptibility (x-axis, niche factor) and temperature (z-axis, habitat factor), interact. According to Schrödter's (1965) temperature equivalence theorum, sporulation and temperature interact to form a sine-shaped graph. When variable host susceptibility is added as a third component, a three-way interaction ensues which is diagrammatically equivalent to the simplified ecotope concept presented in Figure 12.

For pathosystems management purposes it is more convenient to abandon attempts to represent the pathotope in abstract diagrams in terms of its hypervolume and instead to explain it in terms of a mathematical model. For the apparent infection rate pathotope the model would be as follows, viz:

$$Y_r = N_{i\text{-}ni} \times H_{j\text{-}nj} \quad \text{..... eq. 12}$$

Where Y_r = Pathogen population measured in terms of the apparent infection rate;

$N_{i\text{-}ni}$ = Niche-variables, $i = 1$

$H_{j\text{-}nj}$ = Habitat-variables, $j = 1$

Hereafter referred to as base variables

To simplify the discussion, we may consider a particular example of the more general form given in equation 12, involving two niche and two habitat variables, viz:

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*1 Note: Many of Robinson's (1976) pathosystem concepts apply to pathotopes, e.g. there are sub-pathotopes, wild pathotopes and crop pathotopes which all derive quite logically from the pathotope concept.

$$Y_r = N_1 x N_2 x H_1 x H_2 \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad \text{eq. 13}$$

Next, constants may be added which would weight each such independent variable in equation 13. For example, changes in temperature may be relatively more important to the pathogen than changes in relative humidity. In correcting for this consideration, equation 13 would now take the form:

$$Y_r = N_1^{a_1} x N_2^{a_2} x \ldots\ldots N_n^{a_n} x H_1^{b_1} x H_2^{b_2} \ldots\ldots x H_n^{b_n} \ldots\ldots\ldots \quad \text{eq. 14}$$

Where Y, N and H have the same meanings as in eq. 12.

and a_n and b_n are weighting constants which determine the relative contribution of each independent variable.

The model in equation 14 is somewhat different from the usual multiple regression analysis (MRA) approach of Butt & Royle (1974) to such a problem. This is deliberate, in anticipation of later applications to the pathotope theory. However, equation 14 can readily be altered to make it additive rather than multiplicative in order to submit it to MRA, e.g.:

$$\log y = a_1 \log N_1 + a_2 \log N_2 \ldots\ldots a_n \log N_n + b_1 \log N_1 + b_2 \log N_2 + \ldots\ldots b_n \log N_n \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad \text{eq. 15}$$

Finally, the model needs to be expanded to accommodate compensation phenomena (Rotem, Cohen & Putter, 1971; Bashi & Rotem, 1974; Rotem, 1978; Aust, Bashi & Rotem, 1980), i.e.

for the possibility that the negative influence of low values of one key variable can be compensated for by optimum or near optimum values of other variables. Thus the general model of the pathotope in equation 14 requires the addition, either of compensation constants, where the compensation effects are known, or of compensation variables where the compensatory effect is yet to be calculated:

$$Y = N_1^{c_1 a_1} \times N_2^{c_2 a_2} \times \ldots\ldots N_n^{c_n a_n} H_1^{c_1 a_1} \times H_2^{c_2 a_2} \times \ldots\ldots H_n^{c_n b_n} \quad \ldots \text{eq. } 16$$

Where Y, N, H, a and b have the same meaning as in the previous equations;

c_1 = compensation factor.

There is a distinct difference between weighting and compensation, although in certain cases they may have the same effect. By weighting a variable its contribution to the independent variable Y is altered directly. In contrast, a compensation factor alters Y only indirectly via its effect on other independent variables.

A particular pathogen might be sensitive to changes in relative humidity but relatively insensitive to changes in temperature within a certain range. Alternatively, the pathogen may occur in a habitat in which temperature fluctuations are so mild that they are beyond the pathogen's ability to discriminate. In other words, every one per cent change in relative humidity is more important than a one degree change in temperature.

When compensation is operative, temperature in the above example alters the rôle of relative humidity. The spores of a pathogen might for instance be less sensitive to a reduction in relative humidity at one temperature than at another. If relative humidity is fixed and temperature changed, the difference in spore survival values measures the compensation effect of temperature on relative humidity.

The effects of compensation and weighting are usually pooled in traditional applications of MRA. This combination obscures the distinctly separate biological rôles of each phenomenon. The biological actuality can more accurately be expressed in a mathematical model where the compensation effect is viewed as a factor which alters the rôle of a base variable. Thus the most convenient rôle for the compensation factor is to alter the weighting constant of the base variable in question. When the base variable does not require weighting, it is replaced by the compensation factor, i.e. the a's and b's in equation 14 will be replaced by c's.

The compensation factors and their derivations can most conveniently be explained with reference to a simple example in which:

$$Y = N_1^{a_1} x N_2^{a_2} \quad \text{..... eq. 17}$$

According to the theory of compensation (*loc cit.*), the second term on the right hand side (r.h.s.) of equation 17 will influence the contribution of the first term on the

r.h.s. to the independent variable, Y. Similarly if the compensation between base variables is reciprocal, then equation 17 becomes:

$$Y = N_1^{c_1 a_1} x N_2^{c_2 a_2} \quad \text{..... eq. 18}$$

Where $Y_1 N_1$ and a, have the same meanings as before;

$c_1 = f(N_2^{a_2})$

$c_2 = f(N_1^{a_2})$

If equation 18 is linearised by taking logs, then:

$$\log y = c_1 a_1 x \log N_1 + c_2 a_2 x \log N_2 \quad \text{..... eq. 19}$$

The terms $c_1 a_1$ and $c_2 a_2$ reveal how in traditional MRA a single statistic may be measured which pools the effects of compensation phenomena and weighting.

To apply the model in equation 16 to a pathotope management strategy, let us recall the definition of the pathotope, as stated for management purposes, viz. the pathotope represents a community within which individual populations are equally at risk to one another's disease, with a given probability on a given date (*loc. cit.*). In order to manage such a pathotope the following questions require answers:

(i) What are the niche and habitat factors which determine this communal vulnerability?

(ii) How may these be quantified conveniently and simply?

The current crop management strategy for blight in Natal, which is the theme of this thesis, involves the use of chemicals deployed according to a disease warning system. Therefore the above questions, as well as the definitive application of the pathotope theory, will be explored with reference to disease forecasting. However, the pathotope model can just as easily accommodate gene deployment strategies or any other factor which will alter the patho-system's behaviour.

3.1.5.1 Disease forecasting in terms of pathotope criteria

Disease forecasting systems issue warnings which apply to discrete cropping areas. Such a recommendation to farmers to spray on a specific day is, in fact, a warning to the group that the risk or probability of a disease outbreak in that area, is unacceptably high. It is clear that the probability of a successful outbreak, increases with time. Therefore at a particular time a threshold probability value is reached, beyond which disease could rapidly assume epi-demic proportions. Herein lie all the elements of the pathotope concept and the need to identify these factors which determine the probability of widespread disease out-break.

Disease forecasts coincide with the transition from the focal-to the general-epidemic phase (Vanderplank, 1963). During the focal epidemic phase there are a few diseased farms, against a background of many healthy farms. In the

general epidemic phase the situation is reversed. A disease forecast warning therefore may be quantitatively described as a threshold value of the ratio of focal epidemic stage : general epidemic stage (F:G ratio). When the ratio is low, the epidemic is in its focal stage and when high, in the general epidemic stage.

For the purpose of discussion let the critical threshold value of the F:G ratio be arbitrarily assigned the value of unity, then when F:G = 1 a disease warning is issued to all members of the relevant community that a widespread outbreak of the disease is imminent. Further let Y = F:G (where Y has the same value as in equation 16). In practice any F:G ratio may be chosen such that it represents the critical threshold value which corresponds to the maximum level of probability that a widespread epidemic will occur. Thus it is necessary to identify and quantify the dependent variables in equation 16 that will give a solution such that Y = 1. Mathematically it would be more rigorous to think of Y tending to a limit of unity, but for present purposes this complication need not concern us.

A convenient way of rating the dependent variables would similarly be to quantify them on a per unit basis. Thus, in an example where there is only one niche determinant and one habitat determinant, both contributing equally, i.e. without compensation phenomena playing a rôle, then:

$Y = N \times H = 1$ if and only if, $N = 1$ and $H = 1$. Thus

there is a unique solution which satisfies Y = 1. This occurs when the maximum value that N and H may assume is unity.

In addition, the per unit nature of the variables means that they can be measured arbitrarily. Consider for example, the case in which an attribute of the pathogen population, say sporulation, changes with changes in relative humidity (r.h.). Suppose further that r.h. < 80% is lethal and 100% is optimum. Then the 20% range of r.h. could be so apportioned that every 2% change in r.h. is represented by 0.1 on the unit scale, i.e. 80% r.h. = 0 and 100% = 1. This approach readily and yet simply, accommodates more complex approaches. Consider for the purposes of a consistent theme, Schrödter's (1965) temperature equivalence theorem for the rate of growth of a pathogen, Y:

$$Y = \sin^2(a_1x+a_2x^2+a_3x^3) \quad \text{eq. 20}$$

with the determining equation:

$$Y = \sin^2(1.28x+0.00746x^2+0.000127x^3) \quad \text{eq. 21}$$

Where x, the special temperature equivalent is

$$x = \frac{t - t_n}{t_x - t_n} \cdot 100 \quad \text{eq. 22}$$

and t = actual temperature

t_n = minimum temperature

t_x = maximum temperature

The sinoidal nature of equation 20 in which $0 \leq Y \leq 1$ makes it directly applicable to the pathotope model proposed in this thesis.

These theoretical examples show how the pathotope concept may be adopted for a wide variety of purposes when all its components and sub-components are measured on a per unit basis. However, in practise the individual quantitative contribution of a variate as part of a multivariate array cannot easily be determined. For pragmatic pathosystems management decisions therefore, it would be necessary to employ many different extrapolative or iterative procedures which would set the value of Y in equation 16 at fiducial limits consistent with either past experience or the degree of risk the group is *a priori* prepared to tolerate.

Before a particular, practical example, based on the potato late blight data in this thesis, can be presented, the relationship between the distribution of potato late blight pathotopes in Natal and the *Phytophthora*-pathway theory, must be explored.

3.1.5.2 Potato blight pathotopes in Natal

When Natal potato farms are grouped according to planting time, nine discreet potato producting areas emerge, as shown in Figure 14. Potato farms are clustered in this pattern for a variety of reasons.

From east to west across Natal, there is an altitude increase (see Section 1.4.0, Fig. 2). The step-like nature of the plateau determines planting dates naturally, due to a temperature gradient. The lower lying potato-growing areas nearer the coast experience milder winters and warmer temperatures earlier in spring than do those of the higher areas. Only the former do not run the risk, which decreases in an easterly direction, of late frost occurrences. The broken topography caused by deep river valleys also tends to consolidate farming areas in fertile valleys or arable plateaux. In some cases these potato areas, are separated by large tracts of afforestation or the tribal trust lands of Kwa-Zulu.

Several factors within these potato-growing areas suggest that they might be viewed as blight pathotopes, i.e. as areas within which individual farmers are at risk to late blight and early blight attack by virtue of belonging to the group. Within each of these pathotopes, planting dates are determined by frost patterns and other agronomic considerations concerning crop rotation. Since planting date determines full-canopy date which in turn is likely to coincide with the F:G transition, late blight-favourable microclimate conditions are likely to develop synchronously. In the case of early blight, ageing which favours the disease, may also largely be synchronized. Proximity of potato farms within a pathotope also suggests that within-group sources of inoculum are more threatening than distant sources of inoculum in other pathotopes. Common climatic features which establish an area as a "potato-growing area", such as

temperature regimes and rainfall distribution,creates the need for practices such as irrigation to be necessary for all members of the group. Finally, the Young forecasting system explicitly acknowledges that early-summer, mid-summer and late-summer potato plantings are differently at risk to late blight attack.

The pathotope concept does not require absolute accurate delimitation of pathotopes. Indeed, its very pragmatic nature allows for the formulation of a pathotope strategy based on approximate assumptions of equal vulnerability in the first instance. Then the outcome of this strategy could be used in the following year to re-evaluate and improve assumptions. In this way, the control strategy based on the pathotope concept will improve while simultaneously resulting in better understanding of factors determining disease vulnerability in discrete geographic areas.

If a *Phytophthora* inoculum pathway exists then it can be viewed as a route passing east-west through pathotopes, the initial inoculum for pathotope Number 2 in Figure 13, for example, coming from pathotope Number 1. When the "classical" problem of the origin of late blight inoculum (Hirst, *loc. cit.*) is viewed from this higher systems level of integration, an important epidemiological principle viz., the relationship between disease and initial inoculum may be invoked which would improve the current late blight control strategy both within and between pathotopes.

3.1.5.3 Control strategy within a pathotope

The general strategy of disease control consists of reducing the amount of inoculum from which disease starts, or reducing the rate at which it increases after it has started, or both (Vanderplank, 1972). Sanitation is an example of reducing inoculum, whilst horizontal resistance (*sensu*: Vanderplank, 1963) reduces the rate of disease increase.

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 for potato late blight in Figure 15.

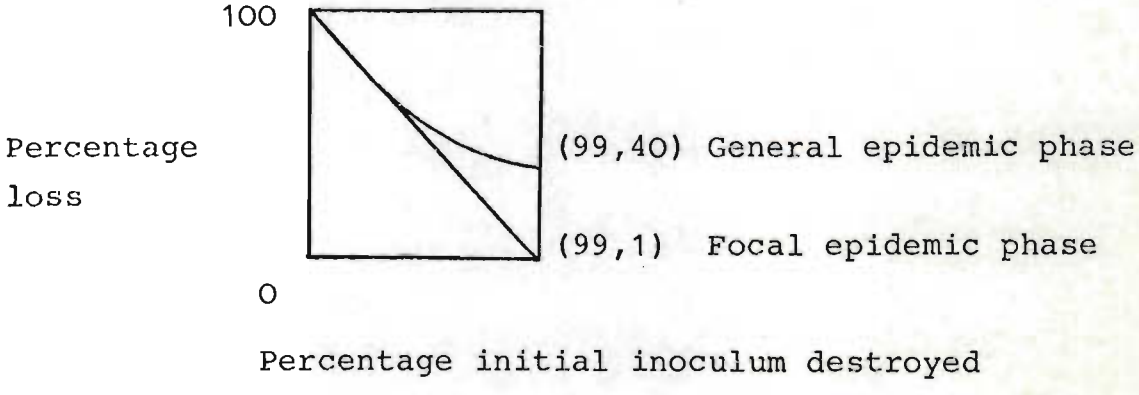


Fig. 15 The effect of destroying initial inoculum, on reducing disease loss, expressed as a percentage of what the loss would have been if no inoculum had been destroyed. (Adapted from Vanderplank, 1963).

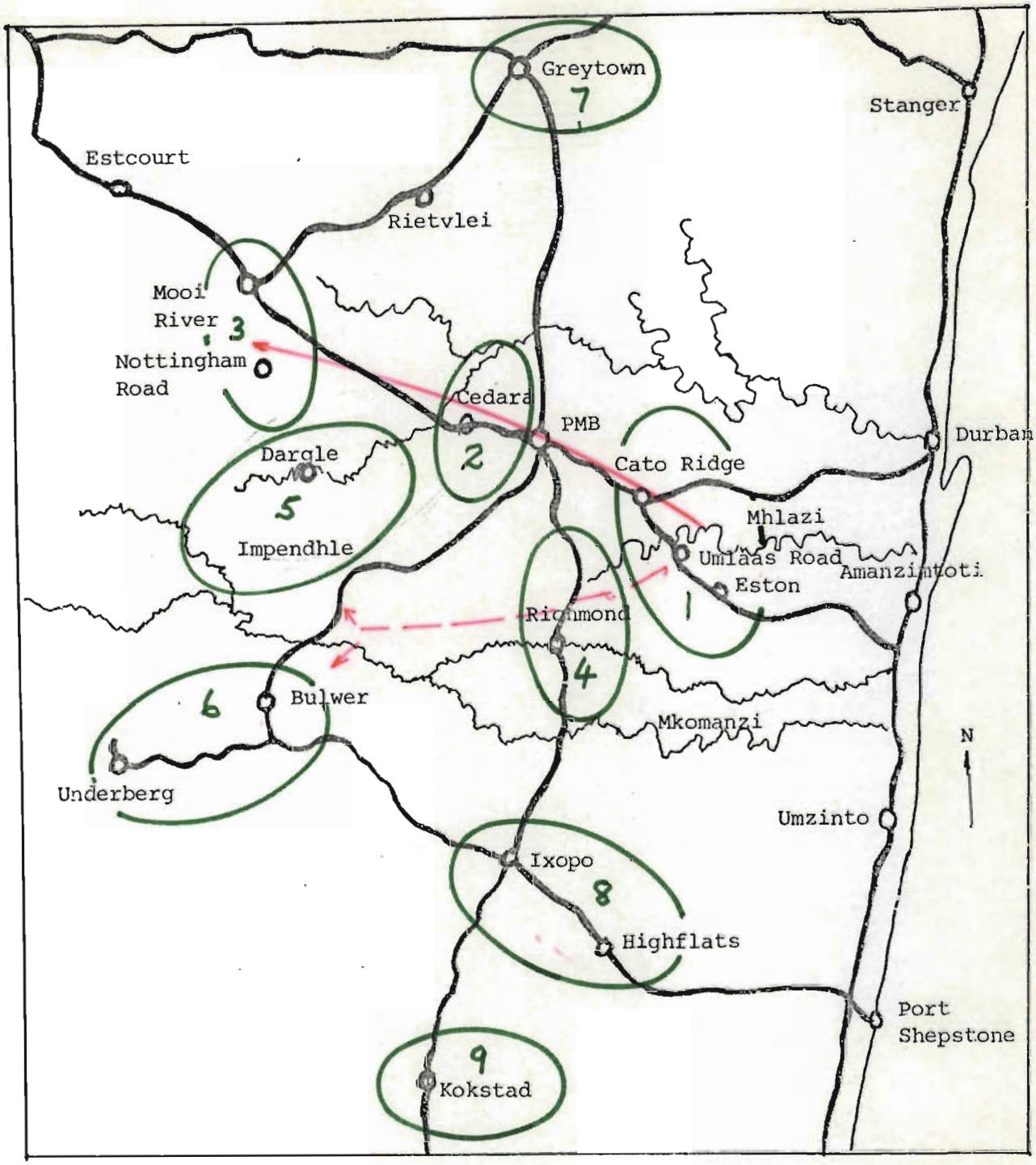


Fig. 14 The potato late blight pathotopes of the Natal Midlands.

——→: Postulated *Phytophthora*-route: Cato Ridge to Mooi River.

— — →: Alternative *Phytophthora*-route: Cato Ridge-Richmond-Impendhle.

In the proposed pathotope context sanitation as a disease control measure, may be evaluated at two levels, i.e. within and between pathotopes.

Within a pathotope, sanitation is the preferred strategy when the pathotope is in the focal epidemic stage, i.e. the F:G ratio is low. During this time, optimum strategy in the case of late blight would be to use the curative properties of Ridomil on the blight foci, thereby reducing the number of foci from which the disease can spread.

Ridomil has both curative and protective properties. Consequently where it is used it will simultaneously have a sanitation effect and an apparent infection rate-reducing result. Thus the benefits of sanitation, i.e. the delaying effect it has on an epidemic (Vanderplank, 1963) will be confounded with the beneficial consequences due to its protective properties, i.e. its ability to reduce the apparent infection rate, r. However, the other contact surface protectant fungicides available for late blight control do not have a sanitation effect. Therefore, it may be feasible for those farmers who currently do not use Ridomil, to consider its inclusion early in their spray programmes thereby adding a sanitary component to their disease control programmes at a time when it is likely to give the best results, i.e. when r is low.

For the control of early blight,only protective fungicides are available. Furthermore, there is as yet no definite

evidence pointing to an *Alternaria* inoculum pathway so that the disease-proportional-to-inoculum rule is not likely to help much in the case of early blight because the source of inoculum and its means and route of dissemination are not known. Thus although uniform control measures against early blight *within* a pathotope will be as valuable as it is against late blight, benefits from the pathotope approach cannot accrue *between* pathotopes unless there is an interdependence of pathotopes.

Where cultural practices such as destruction of crop residues are practical they will have a sanitation effect on early blight. Since sanitation efforts follow the law of diminishing returns (Vanderplank, 1963) the advantages to be gained from destroying crop residues and removing volunteer potato plants are likely to be disproportionately large compared to the effort required. Thus, for early blight control a strategy integrating cultural methods with a sanitation effect and protectant fungicide sprays is indicated.

Consider the implications of such successful inhibition of late blight foci. Assume that the threshold transition value of the F:G ratio for the pathotope in question is 0.08. Then, when eight out of a hundred farms are infected, the transition from focal to general epidemic stage can be expected and infection rate-reducing strategies would be the preferred choice. If the blight foci can be eradicated before contributing inoculum to other healthy farms the transition ratio will not reach its critical value. These

arguments apply *mutatis mutandis* to early blight regardless of the type of fungicide or other control method used. In practice however the achievement of such a situation is highly improbable, if not impossible because of late blight increase during the cryptic period (Hirst, 1953) when the late blight pathogen is active without causing easily observable symptoms.

However, sanitation practised against late blight in foci will reduce the spread to nearby healthy farms. The greater the number of spores produced in the foci, the greater the probability of more distant farms being infected. Gregory (1973) discusses several models for dispersal gradients which show conclusively that the greater the amount of inoculum at the source the greater the infection horizon.

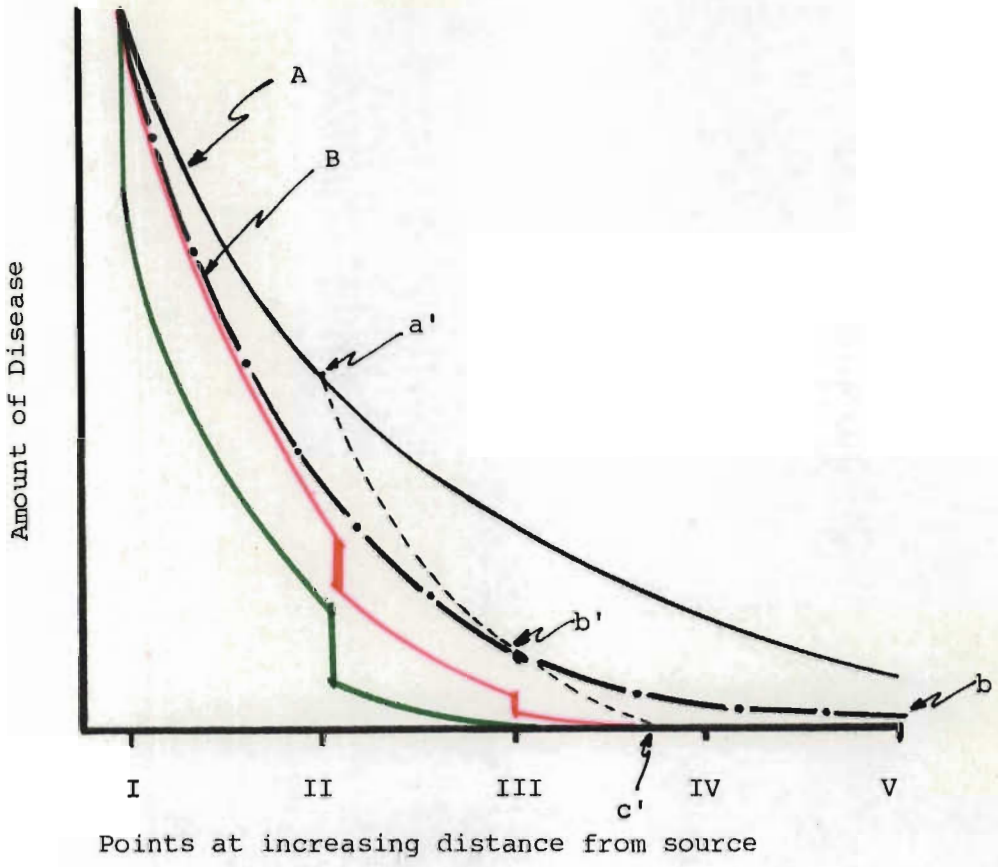
For the individual, sanitation, i.e. Ridomil spraying at the earliest possible detection of late blight, holds benefits because he is then applying control before the apparent infection rate increases to its full potential when environmental conditions are most favourable. In the single pathotope the F:G transition point is delayed by such early control of foci and the extra disease free time so gained reduces the severity of the final amount of disease in the pathotope as a whole, other things being equal.

3.1.5.4 Control strategy for interrelated pathotopes

Where pathotopes are interrelated by virtue of being vulnerable to each other's disease under certain circumstances

the same principles which apply to farms within pathotopes apply *mutatis mutandis* to pathotopes within a pathosystem or pathosystems within a geographical region. At such a higher level of integration, any diseased pathotope serves as a potential focus of infection for other healthy pathotopes. Early disease detection and appropriate sanitation within the source pathotope will clearly also benefit the other pathotopes. Should these pathotopes lie along an annual inoculum migration route, as in the postulated *Phytophthora* inoculum pathway, then the beneficial repercussions of greatly improved disease control in the focus pathotope, will radiate through the entire pathosystem as illustrated in Figure 16.

On page 8, the comment was made that host susceptibility could be altered more easily than weather. The effect of increased host susceptibility on disease spread can be explained with the aid of Figure 16. Here the curves a and b are the ideal dispersal gradients for inoculum dispersing from a point source in a susceptible variety A and a more resistant variety B respectively. If the susceptible variety was cultivated at I and II in the migration route of the pathogen and the more resistant variety B at III, IV and V in the migration route, the dispersal gradient would be the same between I and II as in the topmost graph in Figure 16. However, then it would decrease along the dotted line between a' and b' and thereafter remain the same as it had been on the more resistant variety between IV and V on the second graph from the top in Figure 16.



0	5	10	15	20
Distance in Km between points within a Pathotope				

0	50	100	150	200
Distance in Km between Pathotopes				

LEGEND

I,II....V: 'Stations' or sampling points along the pathogen's migratory route

a',b',c' : See text, p. 165-166

A: Dispersal gradient on resistant variety

B: Dispersal gradient on susceptible variety

Dispersal gradient where low level sanitation is practised at each point

Dispersal gradient with high level sanitation practised at each point

Fig. 16 Amounts of disease at various distances from a point source of inoculum, **assuming an** ideal dispersal gradient

In the absence of disease control the dispersal gradient between b' and b would then remain unchanged. If a mixture of strong vertical genes (*sensu*: Vanderplank, 1968, 1978) entirely different from those at I and II is interjected at III, IV and V, then the reduction in disease severity at III could be long lasting i.e. long enough for crops at IV and V to 'escape' disease attack. This is illustrated by the gradient c' between points b' and IV in Figure 16. However, the different rôles of horizontal and vertical resistance in these situations are outside the ambit of this thesis. They are mentioned here only as a further illustration of their possible application in pathotope theory.

Consider next the effect of different degrees of sanitation at each of the stop-over points in the disease migration route. If the variety B is cultivated at all five stop-over points, the dispersal gradient of the amount of disease as a function of the distance of the stop-over point from the initial point-source of inoculum, will be as in the second solid-line graph from the top of Figure 16. If one level of sanitation is practised at each of the stop-over points the graph b will take on a step-like character as in the red line in Figure 16 and if a higher level of sanitation is practised at each stop-over point, it will show a more extreme step-wise reduction, as in the green line in Figure 16.

Note that at point III in Figure 16 it would take only a

small improvement in disease control to eliminate disease. However, the exponential *decrease* in the benefits of sanitation as a function of the *amount* of sanitation (Vanderplank, 1963; Putter, 1980; Figure 5) should be taken into account before the decision is made to attempt the required improvement in disease control. Many other factors could alter the situation. Figure 16 should be regarded purely as a heuristic model.

Also significant in Figure 16 is the observation that a disease-free zone has been created as a result of the improved disease control at each station. If all the stations are arranged in concentric circles around the point source of inoculum the area of this disease-free zone increases exponentially as a function of the degree of reduction in disease at each station or point along the migration route. This deduction of exponential increase of the disease-free area follows from the relationship between the radius of a circle and its area. Obviously such an idealized dispersal pattern does not occur in nature but the concept is presented here to show that considerable areas of the pathotope might escape a significant degree of risk of disease outbreak. These comments are as valid between pathotopes as they are within pathotopes.

The considerations presented so far have indicated that the epidemiological validity of a disease control strategy which integrates methods of sanitation, improved preventative disease control and increased levels of host resistance,

both in kind and quantity, is as valid in a pathotope disease management strategy as it is to an individual farm.

Implicit in the inoculum pathway theory is the assumption that late blight arrives in an area from which it had hitherto been absent. Thus, in the latter area there is an inoculum arrival *date* and a date on which late blight in the pathotope changes from the focal to the general epidemic stage. A possible control strategy for both of these events, therefore, would be for all the farmers in a pathotope to synchronize their fungicide spray programmes in order to present a uniform defence barrier against pathogen activity. However, in practice this is inadvisable since the date of inoculum arrival is not known and also because it would not be economical - neither is it indicated epidemiologically - for all farmers to spray when the late blight is only present in a few foci.

The pathotope strategy developed here, requires that individual foci be treated with curative applications of Ridomil as soon as they are noticed. For neighbouring farmers, the advice is to synchronize their spray programmes, especially for the first two applications, on a date decided according to the pathotope disease risk formula, an example of which is presented below for the Mooi River pathotope.

3.1.5.5 A management strategy for the Mooi River blight pathotope

The fundamental decision to be made is when should the

farmers of a particular pathotope commence their synchronized late blight spray programme. Consistent with pathotope theory, this decision is based on the increasing risk of the epidemic errupting on a wide front on a particular date. Thus when the value of the dependent variable y in equation 14 is zero, there is a 0% probability of large scale epidemic activity; when y = 1 the pathotope is certain (100%) to be in the general epidemic phase. On this scale from 0-1, the farmers in the Mooi River pathotope can choose their own level of risk and democratically decide when to apply their first synchronized spray. For example, a value of y = 0.75 may be chosen, i.e. when there is a 75% chance that the F:G ratio will reach its crucial transitional value, spraying will commence.

Unfortunately there are no records of late blight severity or dates of first incidence for Natal. A limited amount of data is available for Cedara but unlike the United Kingdom for example, where disease progress curves have been recorded at many stations for more than fifty years (Croxall & Smith, 1976), disease records are not available in Natal for analysis in order to determine when the F:G transition occurs.

For the Mooi River pathotope, the factors which determine high risk to late blight attack are:

(i) The date of the 18th day after the first blight warning is issued from Cedara.

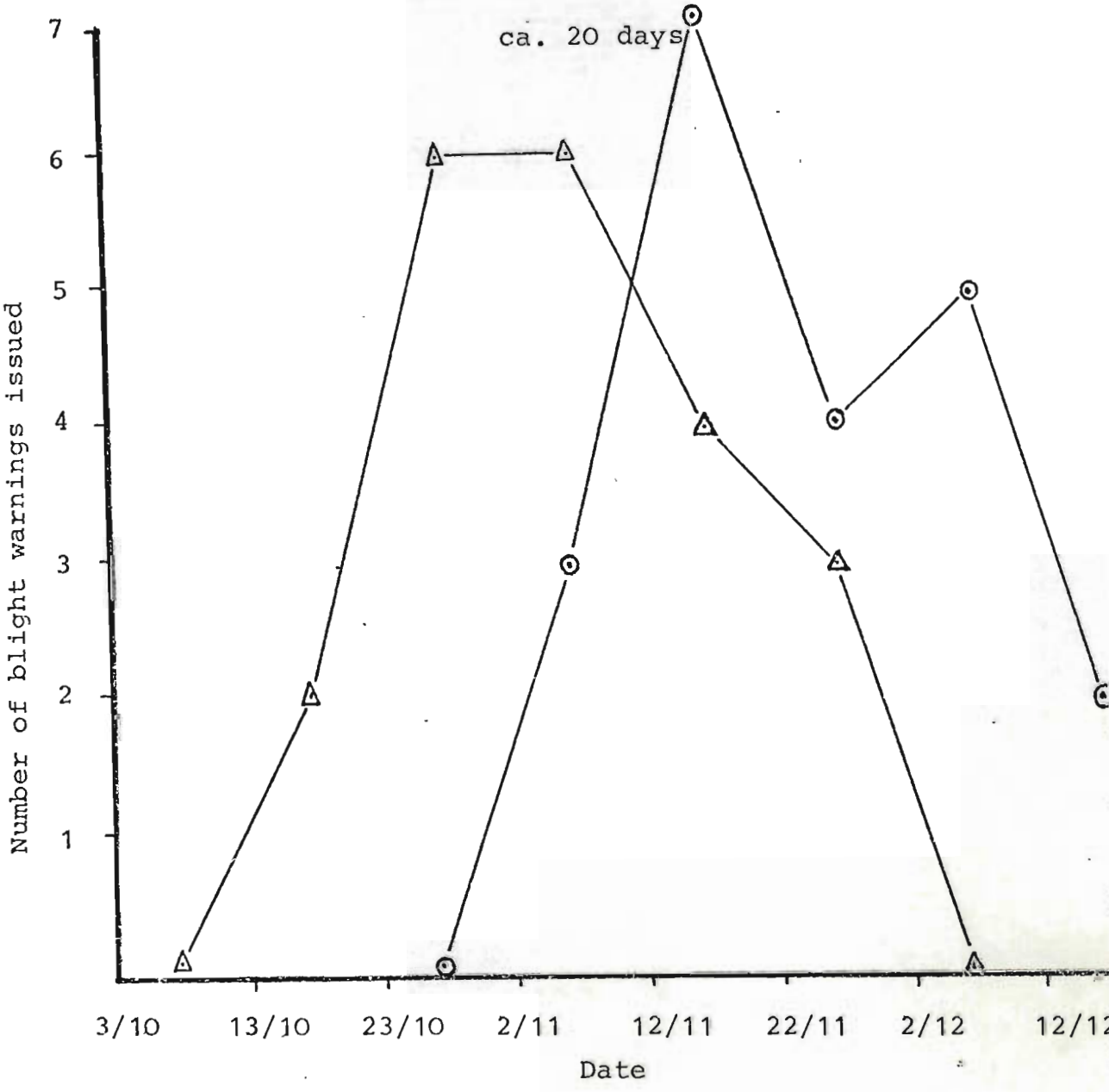
(ii) The growth stage of the crop at the time in (i) above.

(iii) The extent to which overhead irrigation is practised.

In Figure 17, the frequency of first and second blight warnings issued from Cedara are plotted as a function of time. The difference between the peaks of the two curves is 20 days and, in general, the second warning against late blight is about 18 days after the first. The observations of Mooi River farmers that late blight "arrives" about 18 days after the first Cedara warning, is thus further substantiated.

If the assumption is made that the first of the three factors listed above, i.e. number of days after the first Cedara late blight warning, reaches maximum probability (1), 15 days after the first Cedara late blight warning, then every increase of 1,5 days after the first warning represents a 0,1 (10%) increase in the probability of imminent attack.

Microclimate in the crop canopy becomes highly favourable for late blight when the rows meet and this stage of growth has been shown in England to coincide with the transition of focal to general epidemic phases (Hirst, 1953). The local potato cultivar BP_1, reaches full canopy about 55-65 days after emergence, i.e. after the 'green row' stage when the rows of potatoes can first clearly be seen (Mould p.c.). Thus if the 40th day and 60th day after emergence are respectively assigned zero and unity ratings then every in-



Distribution of first blight warning from Cedara, i.e. after 3 blight periods have been recorded.

Distribution of second blight warning from Cedara, i.e. after 4 blight periods have been measured.

Fig. 17 Distributions of first and second blight warnings issued from Cedara. Averages calculated from meteorological records kept at Cedara between 1958 and 1978.

crease of 2 days after the 40th day, represents 0,1 (10%) increase in the probability of the microclimate in the foliage becoming favourable for late blight.

These two factors viz. probable first blight date (PFBD), and host growth stage (HGS), will be considered here as the major factors determining the probability of the occurrence of the F:G transition. To decide which level of probability should determine the date for the first synchronized spray application in the Mooi River pathotope, the data from Figures 3 and 5 will be employed. In these figures it was shown that the transition date for a change from late blight to early blight control strategies should occur on about the 2nd December. In the *absence* of early blight, the use of Ridomil will give the best late blight control. Thus the Ridomil spray programme should be so timed that its protection expires on 2nd December. By that time, the Mooi River potatoes would have been in full canopy for about thirty days. The manufacturers recommend that Ridomil should be applied at ten day intervals. Therefore three applications of Ridomil should be applied, with the first being applied at the beginning of the full canopy stage. On 2nd November the probabilities of the PFBD and HGS factors (the latter being based on average planting times submitted in survey returns by Mooi River farmers) would be PFBD = 1 and HGS = 0,65 with an average of 0,825. An application of Ridomil on 19th October would expire on 2nd November but on the 19th October the combined probability of first blight being noticed is only 0,15,again based on

probability values for PFBD from Figure 17 and HGS from data submitted by the Mooi River farmers. However Figure 17 also reveals that late blight is unlikely to be seen in the Mooi River pathotope before 28th October *if* Cedara late blight weather and inoculum from the Cedara pathotope are the only factors to be considered. Where irrigation is practised blight foci in the Mooi River pathotope may appear at any time before the 28th October, and these farmers should rely on regular crop weather measurements to determine the degree of risk that their irrigation has caused.

From these considerations it would appear that the F:G transition for the Mooi River pathotope is most likely to occur after the 28th October. Therefore, to fit in with a ten day fungicide schedule (both Ridomil and Dithane M45 can be sprayed at ten day intervals) the first synchronized spray date FSSD for the pathotope as a whole, should not be later than 2nd November. After this date the PFBD increases rapidly to its maximum on about 17th November and any delay beyond 2nd November could, at most, save only on fungicide application.

If early blight is not a problem, three Ridomil sprays on 2/11, 12/11, and 22/11, would control the late blight because it is during November that late blight is present according to the 20 year weather data from Cedara. However, early blight development is favoured by the maturing process in the host and therefore early potato plantings are likely to become susceptible to early blight sooner than the 2nd

December at the *latest*. The actual starting date of spraying for early blight, will depend on the host susceptibility, as determined by HGS. However, there is no local data on these aspects of early blight, so the first early blight spray date, should be determined by inspection, i.e. when the first early blight lesion is noticed, a fungicide for early blight control, should either be included in the Ridomil programme or the Ridomil may be excluded and the spray programme continued with a dual purpose fungicide such as Dithane M45.

Hartill (1980) has found Dithane M45 and Ridomil to be equally effective against late blight, when used preventatively. Therefore, since it was concluded in Section 2.2.4.0 that the presence or absence of early blight determines whether Ridomil should be used or not, there seems little evidence for the continued use of Ridomil rather than Dithane M45 once early blight is present. In the Mooi River pathotope, this is likely to mean that only two Ridomil applications may be required before farmers change to Dithane M45. Depending on the early blight incidence, farmers in the Mooi River pathotope could include Dithane M45 with their second and last Ridomil spray and thereafter continue with Dithane M45 only, at seven to ten day intervals. Farmers who suffered focal late blight attacks, may have to apply three Ridomil applications and include Dithane M45 with the third Ridomil application.

The strategy for the Mooi River pathotope discussed, is not intended to be a fixed approach to which farmers must adhere permanently. Instead, it is a first approximation of the ideal strategy developed to explain the pathotope approach and to synthesise available knowledge concerning weather patterns and disease incidence dates. It is a framework for discussion and will be used for this purpose in meetings with farmers in the Mooi River area. During these meetings the emphasis will be on convincing them of the need for a common strategy, to motivate them to develope democratically an approach which would be compatible with their peculiar managerial, technological and financial resources. Every effort will be made to bring the farmers to where they will regard it as *their own* strategy; a hypothesis which *they* will test by collecting observations and measurements towards developing a new, improved approach for the next season.

3.1.6.0 Conclusions

(i) The pathotope concept accommodates the fact that epidemics have spatial as well as temporal attributes. It is founded on basic epidemiology principles and can be a valuable consideration in disease management and comparative epidemiology.

(ii) Disease forecasts, in predicting the transition from the focal to general epidemic phase are based on the collocation of weather data in geographical areas which are similar in terms of disease risk. Thus the pathotope concept can be integrated with disease

forecasting approaches thereby adding an ecological dimension to disease warnings.

(iii) The systems concept implies an interrelated linking of events and components. Thus the movement of inoculum postulated in the *Phytophthora*-pathway theory links the pathotopes of Natal into a single blight pathosystem.

(iv) Pathotopes, by delimiting areas in which common management strategies should prevail, are the quantitative units of comparative epidemiology studies and pathosystems management strategy. As new knowledge of the behaviour of the pathosystem becomes available, the management strategies can be modified accordingly.

(v) Late blight apparently arrives in the Mooi River pathotope of Natal approximately 18 days after it is first recorded at Cedara. This observation, along with other modifying factors which determine late blight severity, e.g. full canopy date and irrigation practices, should be incorporated into a management strategy in order to eliminate unnecessary fungicide applications.

3.2.0.0 A COMPUTER-BASED DISEASE MONITORING AND MAPPING PROGRAMME

3.2.1.0 Introduction

Disease monitoring and the concomitant drawing of disease progress curves is the *sine qua non* of epidemiology (Vanderplank, 1963). Without it, the quantitative, definitive practice of epidemiology dicipline - as opposed to mere qualitative description - is impossible. It is ironical that Vanderplank's theories should have inspired so many research workers outside South Africa while here at home the significance of the new dicipline was apparently not appreciated enough even to inspire the collection of disease progress data towards the creation of a meaningful data-base.

For example, in Natal the potential uniqueness of the postulated *Phytophthora*-pathway provides sufficient reason, by virtue merely of being possible, for the establishment of a disease monitoring system. If only a few strategic late blight observation points had been established when the inoculum pathway idea was first mooted in the early 'sixties, data from some 15 years would by now have been available for interpretation of *P. infestans* migration and the serial development of late blight epidemics.

Thus the major obstacle to the research programme reported in this thesis was the absence of long-term disease records and the absence of an adequate information network for the gathering of disease data. To correct this deficiency, the contact established with the potato and tomato farming community

during the disease survey described in Appendix I, was also used to develop a practical and versatile information gathering method. However, farming communities are the target of so many socio-political and economic surveys that they are often loathe to contribute to surveys which might only reap benefits in ten to twenty years time. Sensitive to this possibility, the blight monitoring system reported below, was developed specifically to provide immediate, convenient feed-back to participating farmers so that their future participation can be highly motivated in terms of immediately perceivable benefits.

Although survey questionaires as described below were distributed to all nine pathotopes, the mapping procedure and re-distribution of collected information to participating farmers are initially only being developed for the Cato Ridge-Eston pathotope. Here overlapping planting dates and widespread irrigation present peculiarly difficult obstacles in the way of a uniform blight management strategy, thereby presenting an ideal opportunity to test the pathotope approach.

3.2.2.0 Materials and methods

3.2.2.1 Data capture for all pathotopes

The following questions were identified as being the basic set applicable to all pathotopes:

(i) Farm grid reference on topo-cadastral map.

(ii) Details of crop, i.e. kind of crop, growth stage, cultivar, planting date and area cultivated.

(iii) Disease information, i.e. kind of disease, date first observed, severity and details of spray programme.

In certain pathotopes where irrigation is practised and to which the Young forecasting system applies, the following additional questions were also asked:

(iv) Irrigation details, i.e. furrow or sprinkler, rotation schedule and precipitation rates and comments regarding weather prevailing at time of irrigation.

(v) Whether or not the crop is sprayed according to the forecast system and if so, whether the forecast was accurate.

A simple code was developed to represent the answers to these questions. Two rubber stamps were then made in order to prepare index cards 10 cms x 13 cms having the requested information stamped on one side of the card and the code for the answers stamped on the other. Together with self-addressed envelopes these cards were then distributed amongst participating farmers. An example is given in Appendix I.

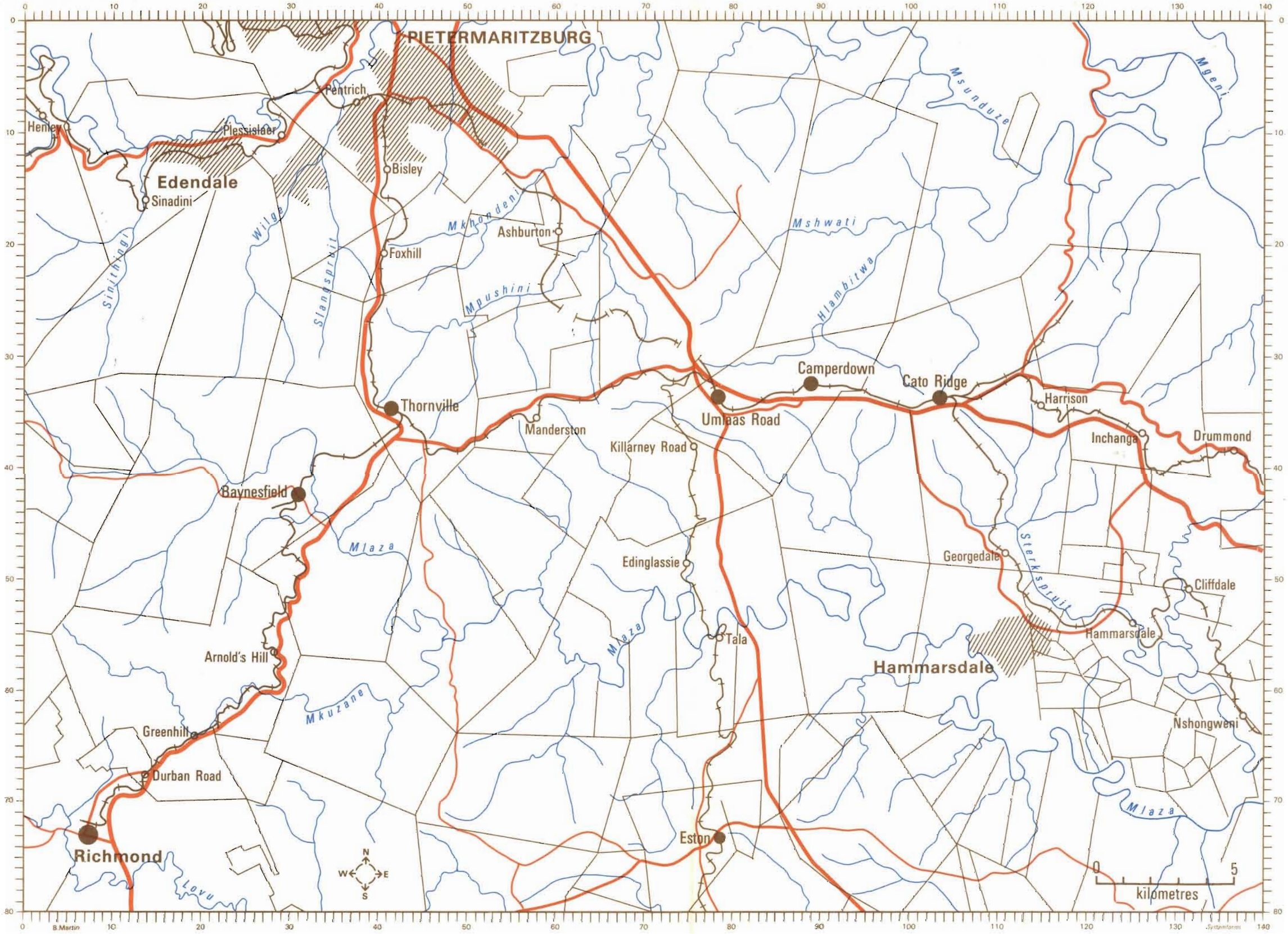
Whenever blight was reported for the first time, the farmer

was asked to include in the envelope with his data card, an example of the disease. Provided potato and tomato leaves bearing late blight or early blight symptoms were wrapped in newspaper, these samples travelled very well. The essential characteristics required to differentiate between late blight and early blight were preserved for at least four weeks. Where there was doubt, a telephone call soon established the identity of the disease and very few field visits had to be undertaken specifically to validate a report.

3.2.2.2 Information dissemination in the Cato Ridge-Eston pathotope

A pre-printed pathotope map (Fig. 18 overleaf) was prepared with a convenient grid reference index on all four margins so that each participating farmer could rule in his own farm position and be assigned a permanent grid reference on the map in Figure 18. At the start of the Cato Ridge-Eston pathotope survey, a meeting was called and all participaring farmers were given the base map displaying at the appropriate grid references the names of all farmers but not any disease detail. By this means provision was made for the encoded information supplied by participating farmers to be collated and printed on successive maps for distribution at regular intervals to all participating farmers.

With the aid of an Apple II Plus microcomputer all the collected information can be sorted rapidly and assigned to appropriate grid references. Disease codes properly


PIETERMARITZBURG
Pentrich
Henley
Plessislaer
Edendale
Sinadini
Bisley
Foxhill
Ashburton
Mkhondeni
Mpushini
Mshwati
Hlambitwa
Msunduze
Mgeni
Wilge
Slangspruit
Sinithingi
Thornville
Manderston
Killarney Road
Umlaas Road
Camperdown
Cato Ridge
Harrison
Inchanga
Drummond
Baynesfield
Mlaza
Edinglassie
Tala
Georgedale
Sterks spruit
Cliffdale
Hammarsdale
Nshongweni
Arnold's Hill
Mkuzane
Greenhill
Durban Road
Richmond
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allocated to grid references are then printed on the pre-printed pathotope maps by an Anadex DP 9500 high resolution graphics printer on line to the Apple microcomputer.

The computer is also programmed to provide a mailing list with addresses printed on gummed labels. Maps containing the coded disease information are simply folded, an addressed label affixed and the whole posted.

The mapping procedure was initiated in 1980 on a pilot basis for the Cato Ridge-Eston area. Consequently, specific disease trends cannot yet be presented. However, the acceptance of the method by farmers and their enthusiastic participation as well as the convenience and versatility of the method deserves comment.

3.2.3.0 Discussion

All participating farmers appreciated the value of knowing the whereabouts and severity of disease in the immediate vicinity of their farms. They immediately realized how important it is to base their crop protection decisions on information coming from further afield than their own farm boundaries. They also found it more easy to interpret the distribution pattern on a map, than would have been the case had the information been produced in Tabular form, which would fail to convey the spatial relationship between the tabulated events. In contrast, the colourful map immediately places the events in geographical perspective

and open to interpretation,according to personal knowledge and experience of the terrain and local weather patterns.

The method is also extremely convenient, both for the farmer and the research worker. It requires little time to complete the card, include a diseased leaf and post it. Furthermore, because the pathotope group decides democratically how frequently the information cards should be completed and returned, apathetic and irregular returns also dissappoint farm *neighbours* and not merely some distant *research worker*.

When a data card is not received from a particular farmer this may be indicated in bold print at his grid reference point. His ommission is then immediately noticeable and the remainder of the group may, if they wish, either telephone him for the information and/or bring group pressure to bear on him to supply the information. This is left entirely to the farmers, who are told that the programme will only be initiated if they wish to use the service. Once they have decided as a group to participate, they assume responsibility for maintaining efficiency from within the group. There is no pressure or cajoling from an outsider. As an extension operation the approach proved extremely valuable and particularly gratifying when farmers spoke of "our disease" rather than "my disease" or "the disease".

Probably the strongest merit of this mapping method is its versatility. Inclusion of information about other diseases

and crops requireslittle modification, only the choice and implementation of a new code, but the pathotope boundary could be quite different. Many other types of data may be collected with equal ease and then mapped for distribution. For example, cropping areas,yield estimates and harvesting dates can be used to rationalize the flow of produce onto the market, thereby possibly dampening severe price fluctuations due to an oscillating supply and demand cycle. When information of this nature is collated for all the pathotopes farmers may then be kept informed of events in other pathotopes.

Also, if one considers the interaction between a specific host and its environment as the basis of a disease triangle, it is clear that disease triangles for many other pathogens of the same host could be erected on this common base of host x environment. Thus, if at some future time a research worker wishes to study another pathogen on the same host, he could have at his disposal valuable,earlier data pertaining to the host x environmental interaction. Similarly, the environment corner of the disease triangle for one host x pathogen combination, is held in common with another host x pathogen combination of an entirely different crop. Then,when a historical data base for different diseases does come into existence,it is highly likely that valuable correlations between diseases, events and crops could lead to valuable comparative studies.

The first logical extension of this monitoring programme

is to select a representative farm for each pathotope where a weather station could be installed. Ideally, the following should be measured:

(i) Air temperature.
(ii) Leaf temperature.
(iii) Relative humidity.
(iv) Leaf wetness : degree.
(v) Leaf wetness : duration.
(vi) Soil temperature.
(vii) Soil moisture.
(viii) Soil pH.

With current electronic technology such a "weatherbox" with these minimum eight channels is entirely feasible. With microchip technology and electronic memory capacity, these variables can be measured almost continuously and then "dumped" directly onto a computer for storage and manipulation.

Current image-analysis technology is equally sophisticated. Thus it is possible to install a closed circuit colour video camera to record aspects of the host. These video tapes may then be fed directly into the Apple microcomputer whose colour graphics capabilities allow it to do colour separation and area analysis. Technically it is feasible to travel from farm to farm with a video camera recording predetermined leaf images and later to analyse these with the aid of the Apple microcomputer to determine the proportion

of disease. With this method data can be kept indefinitely should some future research worker wish to re-examine the records either for the same disease or some other attribute only accidentally recorded by the first research worker.

The obstacle to the implementation of these methods is conceptual rather than technological in nature. One such weatherbox and video camera would cost not much more than a research microscope in a plant pathology laboratory. However, only when plant pathology becomes less of a laboratory orientated science and more directed to investigating the dynamic ecological realities of disease in populations, will this exciting, dynamic technology come into its own right. Indeed, it is not fantasy to anticipate the day when a real epidemic will be monitored as accurately and with the same degree of serial continuity, as is currently only possible in computer simulation studies.

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SUMMARY

The history of the development in Natal of a forecasting service to warn of outbreaks of late blight disease caused by *Phytophthora infestans* is presented. The late blight pathogen and *Alternaria solani*, the causal organism of early blight disease, interact on potatoes and tomatoes to form a blight disease complex. Evidence is presented to show that it is expedient to manage this blight complex as a whole rather than to direct control at only one of the components in ignorance of the consequential enhancement of the potential of the other.

In a search for an improved blight complex management strategy, factors concerning the possible existence of an annual migration of *Phytophthora infestans* inoculum, first postulated in the 1960's, along an east-west route across Natal, are collected and collated. Corroboration of the existence of the *Phytophthora*-pathway is given, inasmuch as it represents a serial outbreak of late blight along a temporal gradient. The possibility that the pathway is a manifestation of disease resulting from the erruption of pre-existing inoculum along an environmental gradient, can not specifically be excluded. However, the peculiar pattern of anabatic and katabatic winds along a river-valley network, superimposed on a continuous cropping pattern and its concomitant opportunity for blight to be endemic in the province, supports the postulated *Phytophthora*-inoculum pathway.

A fungicide spray trial was conducted in order to investigate the possibility of using the pathway phenomenon as the framework for an improved blight control strategy and to explore the nature and level of the competitive interaction between *Phytophthora infestans* and *Alternaria solani*. This trial revealed that the interaction between the components of the blight complex was differentially altered by weather patterns and fungicide combinations. Treatments in which metalaxyl (Ridomil) alone was used for the control of late blight, gave a yield similar to those with propineb (Antracol), which inhibits *A. solani* primarily but also has some negative effect on *P. infestans*. The yields from both these treatments were significantly ($P < 0,05$) better than the yields recorded in the unsprayed control plots. A treatment in which Ridomil and Antracol were combined such that each was applied according to its recommended concentration, gave yield increases of 32,3% over the unsprayed control, although the yield from the Ridomil/Antracol treatment was not significantly greater ($P < 0,05$) than the yields recorded where either Ridomil or Antracol were used.

A computer simulator, named GAUSE, was developed to simulate the consequences of the competition between various combinations of *P. infestans* and *A. solani*. Results simulated by GAUSE corroborated those obtained from the field trial and support the conclusion that diseases of complex etiology require more than simplistic, univariate analysis of single cause-and-effect pathways. The competition quotient CQ is developed as a new parameter of competitive interactions.

It is calculated as the ratio of the amount of disease in the absence of competition, to the amount of disease when the causal pathogen is competing with another pathogen in the same niche. The CQ may be calculated from various standard epidemiology statistics and it is used to demonstrate that the competitive displacement phenomenon places constraints on the interpretation and application of Vanderplank's basic epidemiology equations.

A new pathosystems management concept namely the pathotope (*pathos* = suffering; *topos* = place) concept, is introduced, having developed from the notion that epidemics have spatial as well as temporal attributes. Accordingly, an area in which individual farms are at the same level of probability at risk to disease, delimits the pathotope. The concept can be described at many integration levels and is presented as an important quantitative unit of comparative epidemiology.

The pathotope concept accomodates such notions as are contained in the postulated *Phytophthora*-pathway and is especially suited to integration with disease forecasting methods. An example of the application of the pathotope approach is presented and a strategy is proposed by which fungicide spraying is initiated and applied synchronously as determined by the degree of communal risk to attack and epidemic increase of disease.

Within a pathotope, several common factors collectively determine the vulnerability of the group to disease. If a

coherent, uniform strategy is to be developed and implemented by pathotope members, it is necessary that all members have access to the relevant information and that it be collected and disseminated conveniently and rapidly. A computer-based disease monitoring and mapping system which achieves these objectives is presented.

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APPENDIX I

Introduction

During the course of this thesis study, contact had to be established with farmers on numerous occasions. Initially, farmer participation had to be solicited, which required a comprehensive explanation of the objectives and potential benefits of the research programme. These aims were achieved by means of the attached letter, explanation of the project and the initial green questionaire. All these were despatched in the green folders so that farmers could readily file information about the research project.

Subsequent circular letters were too numerous to append here and were mostly of a routine nature. Therefore only the initial survey is appended as being adequately representative of the approach adopted by the author during this research programme.

Compilation of mailing list

No central register exists of potato and tomato growers in Natal, whilst many farmers grow these crops only intermittently. Therefore to compile an address list for the despatch of the survey questionaires, the following sources were approached for information about potato and tomato growers.

1. Market agents at the Pietermaritzburg and Durban fresh

produce markets, where farm produce is sold on behalf of Natal farmers. They have comprehensive records of past and present potato and tomato farmers.

2. The Potato Board to whom potato growers pay a levy on every pocket of potatoes sold. From this source a list of commercial potato producers was obtained.

3. The Department of Plant and Seed Control and the local Seed Potato Growers Association who have a register of seed potato producers.

4. Companies specializing in the supply of crop protection chemicals and several seed companies provided names of potato and tomato growers who had dealt with them.

5. An extensive publicity campaign was launched on the radio and in the press and a 24-hour telephone answering service was installed to receive calls and messages from farmers, smallholders and gardeners prepared to participate in the survey.

From these sources an address list containing 674 names was compiled. Many of these were farmers who had only grown one of these crops at some time during the last five years. Eventually, 254 farmers and gardeners indicated their willingness to cooperate. However, financial limitations dictated that those phases of the programme arising after initial confirmation of the

boundaries of the pathotopes, be limited to studies in the Coastal, Cato Ridge-Eston and Cedara regions as discussed in the body of this thesis.

Comment on longevity of disease samples

Blighted potato or tomato leaves submitted in envelopes as requested in the survey, survived the postal journey very well. Symptoms of late and early blight remained characteristic of these diseases for up to two weeks. Thus validation of disease reports could be made accurately and cheaply without necessitating a visit specially for this purpose.

The major advantage of the survey did not come directly from the information supplied by farmers. Instead it came from the opportunity to visit and meet farmers who were frequently reluctant to put ideas and observations in writing. The effort of compiling and distributing the survey questionaire was certainly disproportionately large to the amount of data acquired directly from completed questionaires. However, without these questionaires the invaluable personal contact alluded to, would not have been made.

UNIVERSITY OF NATAL

Tel. Add. "UNIVERSITY"

Dept. of Microbiology &
Plant Pathology

Pathosystems Research Project

Faculty of Agriculture
P.O. Box 375
PIETERMARITZBURG
3200

Date: .....

AFRIKAANS OP KEERSY

Mr .....

.....

.....

.....

Dear .....,

Attached please find a description of a research project which I am undertaking with the University of Natal. It is of a practical nature and with your cooperation, I am confident that some real advance can be made in the control of the two blight diseases of potatoes and tomatoes in Natal.

This may be achieved if we can map the distribution and incidence of these two diseases as they occur on farms in the Natal midlands. To compile such a map, I would appreciate your participation in a survey which would require you to :

(1) Complete and return a questionnaire once you have read the attached project outline.

(2) To make observations which would allow me to :

  (i) Record the date on which you first see late blight in your potatoes and/or tomatoes.

  (ii) To record the severity of late blight on three different dates by means of a simple visual check which will be explained in a later circular.

(3) To allow me the opportunity of visiting your farm should I be required to make additional observations and measurements myself.

I am particularly interested in your personal comments and any other observations you may have made. The idea for this project originally came from farmers observations and for this reason I set so much store by your practical ideas.

In talking to farmers, one often hears one or a few ideas repeated as common knowledge amongst farmers. If such ideas could be collected and analysed in search for a pattern, then it may be possible to identify feasible solutions to common problems. Natal potato- and tomato-farmers have lived with the two blight diseases for many years and I am sure that by sharing their knowledge and experience, we may, together, develop better ways of controlling these diseases.

2. / Should you ...

Should you wish to discuss any aspect of the project with me, or wish me to address a group of farmers in your area, please'phone me at Pmb. 63320 ext. 526 or 523. Alternatively, you may 'phone me at home at Howick (033212) 3931. Please call anytime and rest assured that I will respond to any messages which may be left should I be out.

Looking forward to an association of mutual benefit, I am,

Yours sincerely,

C.A.J. Putter

DRAAI IN GEHEEL OM VIR AFRIKAANS

AN INVESTIGATION OF A POSSIBLE LATE BLIGHT PATHWAY IN NATAL

A brief report to farmers on the occasion of soliciting their participation in a survey of disease incidence, May 1979.

C.A.J. Putter

Department of Microbiology & Plant Pathology
Faculty of Agriculture
University of Natal
Pietermaritzburg

CONTENTS :

An investigation of a possible late blight pathway in Natal.

1.0 Introduction: Description and history of the project.

Natal is the traditional home of late blight of potatoes and tomatoes in South Africa. The disease occurs with annual regularity and necessitates costly and time consuming control measures.

During his term as regional plant pathologist at Cedara, Mr Brian Young formulated a theory, based on farmers' observations, which would explain the movement of late blight in Natal. According to this theory, late blight migrates from infected to healthy farms from East to West between Cato Ridge and Underberg. Because the winter frost-free irrigation areas are adjacent to the summer planting areas, potato crops are available throughout the year and thus potato late blight is endemic in Natal. This is an unusual situation which does not occur in any other major potato growing area in the world and therefore presents a unique opportunity to study late blight epidemics. Specifically, it may be possible to determine WHERE, WHEN and under WHICH CONDITIONS the disease spreads. These questions are as crucial in a disease control programme as they are in a military campaign when asked about the movement of the enemy.

2.0 Potential benefits to farmers: Improved blight control strategies which may develop from this research programme.

In the long term it will probably be possible to predict not only the original date of the disease in a particular area, but also to relate disease increase to specific weather conditions and disease levels elsewhere in the Province. The first and subsequent applications of fungicides can then be applied as disease severity demands rather than on either a hit-and-miss basis or in a regular preventative programme. Thus we will spray our specific fungicide against late blight when late blight threatens. When it doesn't, we will switch to an alternative fungicide with more general properties for the control of early blight. Thus we will approach a solution to the dilemma of having to decide when to emphasize either late blight or early blight; or when to emphasize both.

For such an approach to disease control, long-term regional records of disease progress are essential. In England, for instance, records of late blight epidemic patterns for 50 years are on record and these data are being used to great advantag in blight control programmes. Because of the endemic nature and survival pattern of late blight in Natal, I am confident that significant progress can be made here

2 / within a

within a couple of years.

In South Africa disease monitoring on a geographical scale is practically unknown and in this regard, the data you submit will be amongst the pioneering contributions in this field.

2.0.1 Regional rationalization of spray programmes.

It may be feasible to divide the potato growing areas, as in Figure 1, into regions which may be equally open to attack by late blight on a given date. Indeed, which ever way the sub-divisions are made, they can only be refined as new knowledge becomes available and the usefulness of this idea does not depend on an immediate, permanent and highly accurate first attempt.

Disease severity and occurrence, i.e. presence or absence, in any one region may ther be related to disease in another region. For example, if late blight arrives in Mooi River on an average 20 ($\pm$ 5) days after it had arrived at Cedara, then not only do we know in advance when to spray at Mooi River, but also that everybody in the Mooi River equal risk region (the Mooi River late blight pathotope) should synchonize their spraying to spray on the same date.

The advantages of this approach will accrue both within and between pathotopes (= equal risk regions).

Within pathotopes (regions) this approach will evaluate the possibility of one or more farms not sprayed on the same high risk date, providing for the disease within that particular region. The value of this synchronization may be appreciated by considering the analogy of a farmer who sprays 90% of his potato crop and not the remaining 10%. Within a short time, the increase of disease in the unsprayed 10% would negate the effort spent on the remaining 90%.

Between pathotopes (regions) significant benefit will accrue if blight in one region influences or directly determines the amount of blight in another. Consider two adjacent regions, A and B in which blight in B has its origin in region A. If blight in region A is controlled more efficiently, then all the farmers in region B would benefit. If we should add regions C, D, E, etc., to the chain, with the sam assumptions, then improved control in one area could mean disproportionately larger benefits to other equal risk areas further along the pathway.

The project will aim to map the distribution of healthy and infected farms, from which maps it will be possible to :

(1) Group farms into areas of equal risk to late blight attacks on a given date.

Study Area : Potato and tomato late blight and early blight diseases.

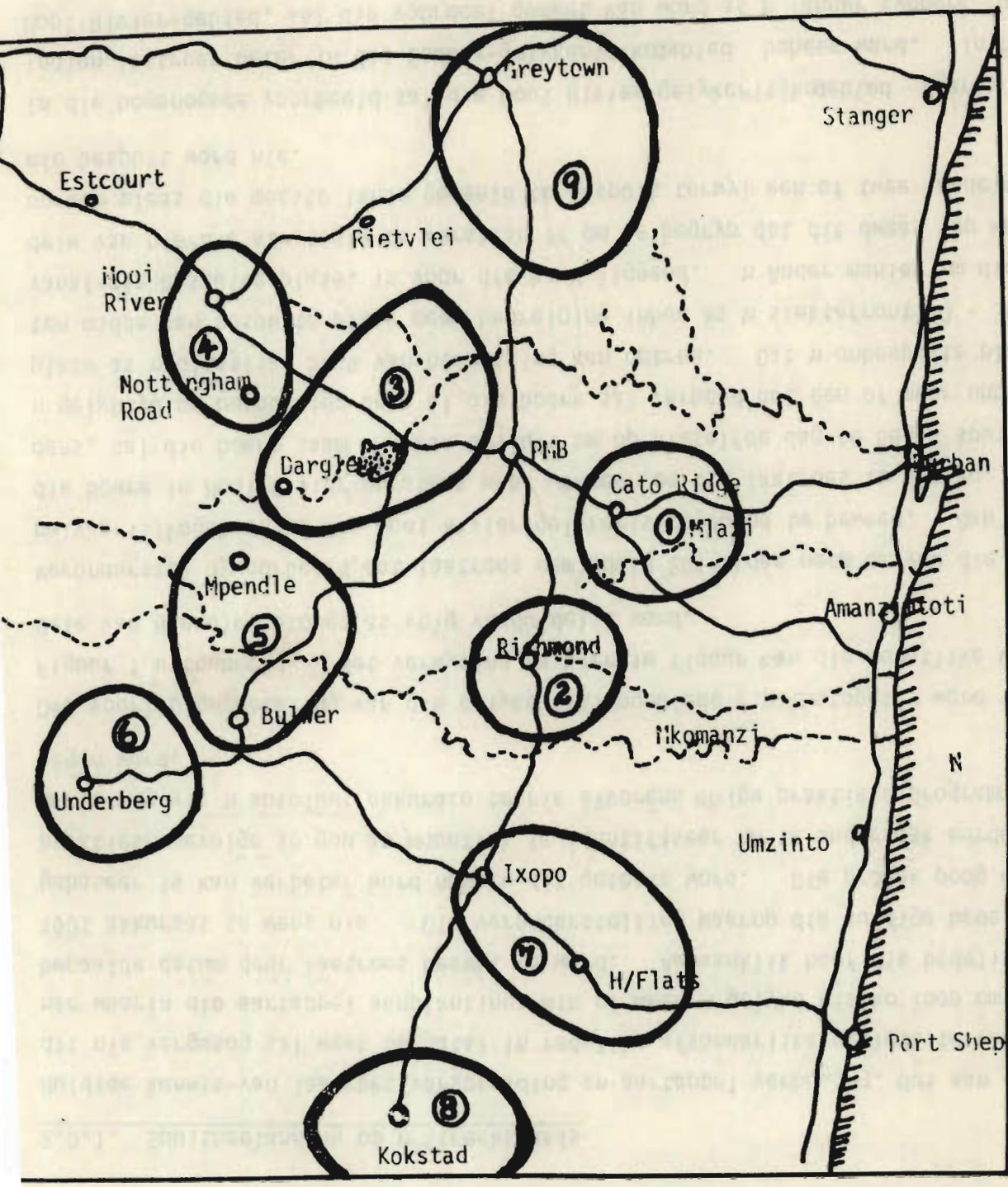

Fig. 1. Provisional distribution of areas equally at risk to late blight attack on a given date, i.e. the distribution of 9 late blight "pathotopes".

(2) Attach to each region specific high risk dates on which the first blight attacks may be expected.

(3) Correlate high risk dates of the different regions in a search for a formula whereby the movement of disease from one region to another may be predicted days or perhaps weeks in advance.

(4) Develop localized disease forecasting methods based on the climate of each region rather than relying on Cedara measurements to be representative of the Natal midlands.

3.0.0 How the farmers' assistance is required.

Before you can complete the questionnaire and cards explained below, you will need to know the difference between late blight and early blight.

3.1.0 How to distinguish between the two diseases.

3.1.1 The problem with the names:

Unfortunately, the names "late blight" and "early blight" are very misleading.

Late blight occurs neither late nor early in the season and its incidence is determined by wet-weather and perhaps we should think of it as "wet blight".

Early blight on the other hand, usually does not occur early in the cropping season (volunteer potatoes are excluded from this statement). Instead, it is more common late in the season when the plants are ageing or when the plant is under stress for some or other reason such as drought or lack of fertilizer. A better name for this disease would be "target spot" for reasons explained below.

3.1.2 Description of the disease.

The most distinguishing feature of late blight, i.e. "wet blight" or Phytophthora-blight, is the appearance of a whitish-grey band, 2 - 5 mm wide, of fuzz-like mouldy growth around the margins of disease spots. This "fuzz" is most apparent in the early morning and during cool, wet, overcast days.

The "fuzz" can easily be made to appear as follows :

(1) Place a diseased leaflet on a convenient piece of wood or cork and float it in a saucer of water.

(2) Place a drinking glass or honey jar upside down in the saucer so as to cover the floating leaf and at the same time trap some water.

This will create a high humidity in the glassed-in atmosphere surrounding the leaf.

(3) Leave for a day or two in a cool place and the fuzz will be seen on the edge of the lesion.

Early blight's most distinguising feature has earned it the name "target spot" amongst American farmers. A close inspection of the brown lesions, reveals concentric rings, or very slightly raised concentric contours, much like the pattern on a rifle- or archery-target.

Late blight lesions are irregular in shape and enlarge rapidly; target spot lesions are roughly circular and expand slowly.

3.2.0 Schedule for completion and return of questionnaire.

Your participation will be required on 2 separate occasions. The first is to establish whether you are growing potatoes and/or tomatoes, and if so, whether you are prepared to participate. The second request will be for you to record late blight severity on three different occasions.

3.2.1 First survey of crop distribution and first infection dates.

(a) Complete the attached green questionnaire, add more sheets if required and mail in the addressed, stamped envelope.

(b) Keep the card and its envelope until you first see late blight in your crop. Then, complete the card and post it off with a diseased leaf in the same envelope.

ALL INFORMATION SUBMITTED WILL BE REGARDED AS CONFIDENTIAL.

One problem concerns the correct identification of your farm. In order to pinpoint it on a map, I require accurate information. This is best supplied as the sub-division number and description of your farm as it appears on the title deed. If this is not at hand then perhaps you could supply the name of the original farm? Failing all else, and if you will oblige, a sketch showing where you are and the names of one or two neighbouring farms should solve the problem.

3.2.2 The second survey of disease severity :

Only those farmers who return the green questionnaire will receive the second survey which will consist of 3 cards and an explanation of how to use them.

If the response to this project warrants it, disease distribution maps may be pr

ted in the Natal Witness on a regular basis. Alternatively, you will be kept i
formed in a newsletter as the information becomes available.

"THANK YOU FOR READING THIS FAR!"

TUMBLETURN FOR ENGLISH

'n ONDERSOEK NA DIE MOONTLIKE BESTAAN VAN 'n LAATROES MIGRASIEROETE IN NATAL

- 'n Beskrywing van 'n navorsingsprojek ter geleentheid van 'n versoek vir samewerking deur aartappel- en tamatieboere.

C.A.J. Putter
Dep. Microbiologie en Plantsiektekunde
Natal Universiteit
Posbus 375
Pietermaritzburg, 3200

INHOUD

'n Ondersoek na die moontlike bestaan van 'n laatroes migrasieroete in Natal

1.0.0. Inleiding : Beskrywing en geskiedenis van die projek.

Natal is die tradisionele tuiste van aartappellaatroes in Suid Afrika. Die siekte vereis gereelde en duur beheermaatreëls indien die gewas suksesvol verbou wil word.

Op grond van sekere opmerkings van boere en op grond van sy eie ondervinding, het Mnr Brian Young tydens sy dienstermyn te Cedara, die teorie ontwikkel dat laatroes van Oos na Wes in Natal versprei. Volgens hierdie teorie word die siekte weswaarts deur die wind versprei en nie deur óf besmette saadmoere, óf oorlewende swammateriaal nie. Omdat die rypvrye produksiegebiede naby die someraanplantings voorkom, gebeur dit dat aartappelaanplantings dwarsdeur die jaar plaasvind in die 160 km. tussen Cato Ridge en Underberg. Die gevolg is dat 'n gerieflike kosvoorraad in die vorm van aartappel- en/of tamatie-lowwe deurentyd beskikbaar is. Die swam word dus nie genoodsaak om van moeilike oorlewingsmetodes gebruik te maak nie.

Hierdie ononderbroke aartappelverbouingspatroon in 'n redelike beperkte gebied, is buitengewoon en word nie in enige van die belangrike oorseese aartappelgebiede aangetref nie. Ons kennis van laatroes epidemies is tot 'n mate gebaseer op oorseese navorsing waar die swam die winter moet oorleef. Die siektepatroon in Natal bied dus 'n unieke geleentheid om vas te stel WAAR, WANEER en HOE die laatroesswam versprei. Hierdie inligting sal van dieselfde waarde wees as soortgelyke inligting oor die bewegings van 'n vyand tydens 'n militêre veldtog.

2.0.0. Voordele vir die boer wat mag spruit uit die navorsing.

Oor die langtermyn mag dit moontlik wees om die verspreiding van die siekte te voorspel. Die ergheid van laatroes in een gebied kan heel moontlik vertolk word in terme van ergheidsgraad in 'n ander gebied sowel as heersende en historiese weersomstandighede. Die hele spuitprogram teen roes kan dus op 'n rasionele grondslag beplan word in plaas van om in die duister te beplan en te bespuit in die hoop dat bespuiting en swambesmetting saam sal val.

Hierdie ondersoek sal die boer in staat stel om die beste beskikbare swamdoder teen laatroes te gebruik wanneer laatroes dreig. Wanneer laatroes nie 'n bedreiging is nie, kan 'n swamdoder gebruik word wat die aksent plaas op die beheer van vroëroes. Mettertyd mag dit moontlik wees om vroêroesbeheer op dieselfde grondslag te plaas.

Langtermyn opnames van laatroes verspreiding sal die grondslag van hierdie projek

vorm. In Suid Afrika is daar bykans geen langertermyn inligting oor enige plantsiekte beskikbaar nie. Hierteenoor is opnames van laatroes vir die afgelope 50-60 jaar reeds in Engeland gemaak en hierdie inligting word tans ontleed om te bepaal of beter voorspellings van laatroes-aanvalle moontlik is. Die endemiese patroon van laatroes in Natal is van so 'n besondere aard dat verbeterde metodes vir laatroes beheer binne 2 of 3 jaar ontwikkel mag word. Inderdaad, 'n begin wat dadelik gemaak kan word, word hieronder beskryf.

2.0.1. Spuitbeplanning op 'n Streeksbasis

Huidige kennis van laatroes verspreiding en aartappel verbouing, dui aan dat dit nie vergesog sal wees om Natal in redelike afsonderlike gebiede te verdeel nie waarin die aartappel aanplantings min of meer 'n gelyke risiko loop om op 'n bepaalde datum deur laatroes besmet te word. Aanvanklik hoef die bedeling nie 100% akkuraat te wees nie. Die veronderstelling waarop die huidige bedeling gebaseer is kan verbeter word namate dit getoets word. Die projek poog om praktiese gevolge so gou as moontlik te identifiseer en te ondersoek eerder as om te wag vir 'n absoluut akkurate teorie alvorens enige praktiese programme begin word.

Die voorlopige bedeling van die gelyke risikogebiede („pathotopes") word in Figuur 1 uitgebeeld. Met verwysing na hierdie figuur kan die moontlike voordele van hierdie metodes as volg verduidelik word.

Veronderstel byvoorbeeld, dat laatroes gemiddeld 20(±5) dae neem om van die Cedara-gelykerisikogebied na die Mooi Rivier-gelykerisikogebied te beweeg. Dan sal die boere in Mooi Rivier eerstens weet wanneer om die laatroes te verwag. Tweedens, sal die boere saam kan besluit om op dieselfde dag te begin spuit. So 'n gelyktydige bespuiting deur al die boere sal verhoed dat een of meer onbespuite plase as 'n plaaslike bron van besmetting kan optree. Dat 'n onbespuite plaas ten midde van bespuite plase meer bedreiging inhou as 'n siektefront 20 - 30 km vanaf die bespuite plase, is voor die hand liggend. 'n Ander manier om die voordele van hierdie strategie te verstaan is om te begryp dat dit dwaas sou wees om op een plaas die meeste lande gereeld te bespuit terwyl een of twee lande glad nie bespuit word nie.

In die bogenoemde voorbeeld sal die Mooi Rivier-gelykerisikogebied daarby baat indien laatroes beter in die Cedara-gelykerisikogebied beheer word. In die Mooi Rivier-gebied, sal die voordeel gemeet kan word as 'n langer tydperk tussen laatroes-vertrek by Cedara en aankoms by Mooi Rivier. Indien die gelykerisikogebiede 'n ketting vorm - en die Young-teorie is op so 'n veronderstelling gebaseer - dan is dit redelik om te bespiegel dat die siekte jaar-na-jaar verminder sal kan word namate die beheermetodes verbeter word.

3. / U samewerking ...

Opnamegebied : Laatroes en Vroëroes van Aartappels en Tamaties

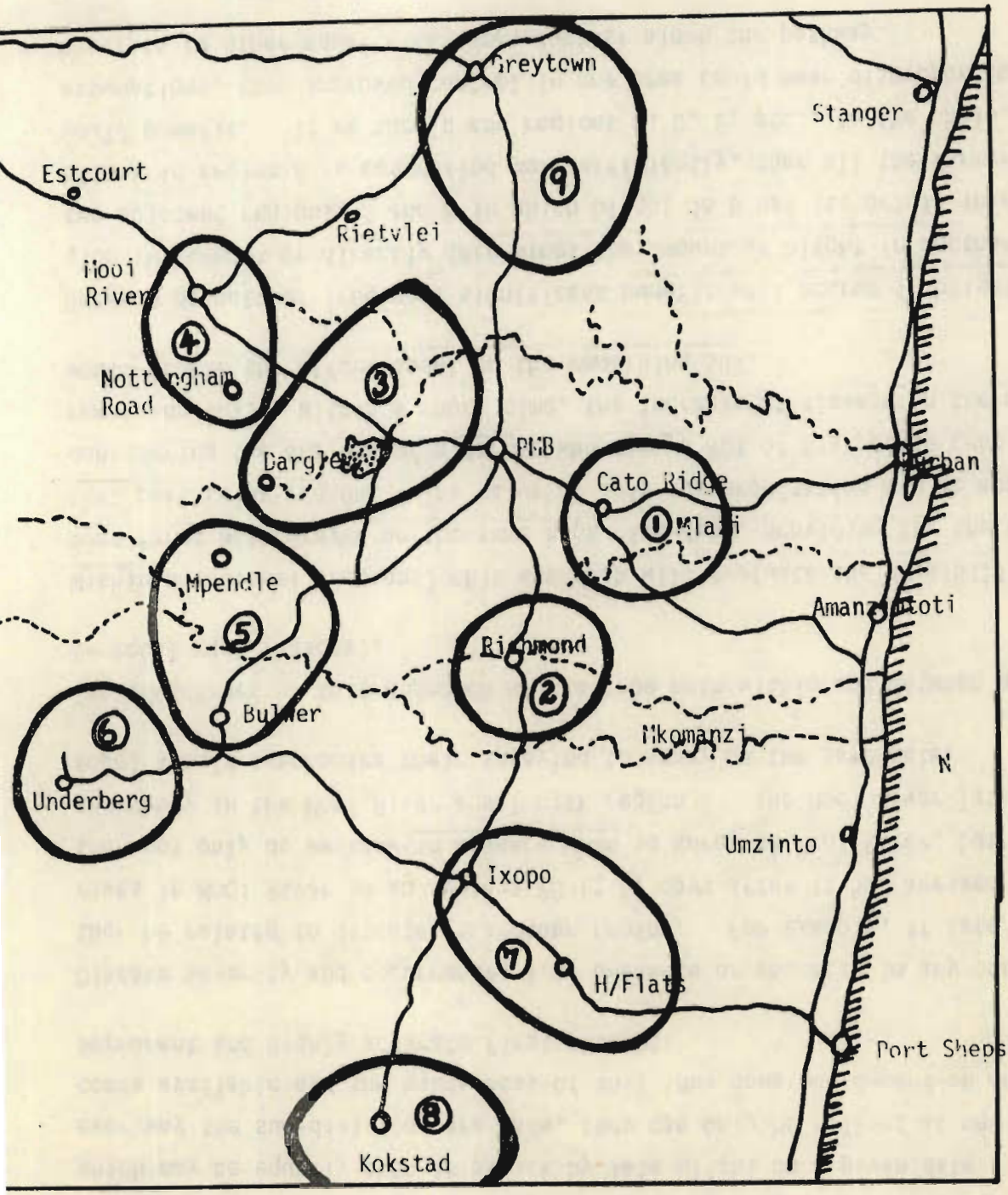

Fig. 1. Voorlopige verspreiding van gelykerisikogebiede („Pathotopes") in terme van laatroes besmitting op 'n gegewe datum.

U samewerking in die opname sal my dus in staat stel om :

(i) Plase te groepeer in gelyke risikogebiede;

(ii) Vir elke gelyke risikogebied, die waarskynlikste datum van eerste besmetting te bepaal;

(iii) Die eerste besmettingsdatums van die verskillende gebiede te vergelyk in die soektog na 'n patroon wat kan dien as basis vir siektevoorspelli

(iv) Plaaslike siektevoorspellingskemas te ontwikkel wat gebaseer is op plaaslike weersomstandighede eerder as op die veronderstelling dat Cedara as verteenwoordigend van die Natalse middellande beskou kan word.

3.0.0 Samewerking wat van die boer gevra word :

Alvorens u die vraelys kan voltooi sal u die verskil moet ken tussen laatroes en vroëroes.

3.1.0 Hoe om tussen laatroes en vroëroes te onderskei :

3.1.1 Tekortkominge van die name : „Laatroes" en „Vroëroes"

Laatroes kom nie noodwendig laat in die seison voor nie. Die siekte kan enige tyd van die seison uitbreek soos bepaal deur die weersomstandighede en nie deur die tyd van die jaar nie. Dit sal meer akkuraat wees om te dink in terme van „NATROËS" want nat-of vogtige-weersomstandighede vererger die siekte.

Vroëroes, daarenteen, kom as 'n reël eerder laat as vroeg in die seisoen voor. Vroëroes word bevoordeel deur omstandighede wat die plant verswak, bv. a.g.v. kunsmisgebrek, droogte en die normale veroudering van die plant.

3.1.2 Beskrywing van die twee siektes:

Die mees kenmerkende eienskap van laatroes is die voorkoms van 'n grys-wit, donsige skimmel wat die siektekolle gedurende nat - of vogtige-weer, omring. Die donsige skimmel is duideliker in die vroë môre as in die laat middag. Die skimmel kan as volg gekweek word :

(i) Plaas 'n besmette blaartjie op 'n grieflike grootte kurkprop of 'n stukkie hout en laat dit dan op 'n piering water drywe.

(ii) Plaas 'n drinkglas onderstebo bo-oor die blaar sodat 'n atmosfeer met hoë humiditeit in die glas om die blaar kan ontwikkel.

(iii) Laat staan vir 'n dag of twee in 'n koel plek en u sal dan die donsige skimmel kan sien.

Die kenmerkendste eienskappe van vroëroes is die volgende :

(i) Vroëroes siektekolle word gekenmerk deur konsentriese ringe -

soos die sirkels op 'n teiken - wat duidelik in die dooie weefsel gesien kan word. Om hierdie rede is die Amerikaanse naam van die siekte „targetspot". Die naam sal vertaal kan word as „konsentriese-blaarvlek" en sal meer paslik wees as „vroëroes"

(ii) Die siektekolle is gewoonlik klein (± 5 - 12 mm in deursneë) en as groter dele dood is, sal dit waarskynlik wees a.g.v. die samesmelting van twee of meer siektekolle.

(iii) Geen donsige skimmel kom normaalweg om vroëroes siektekolle voor nie.

(iv) Die vroëroes siektekolle is ook meestal sirkelvorming terwyl laatroes-vlekke geen vaste patroon volg nie.

3.2.0 Program vir die voltooiing van die opnames :

U samewerking word op verskille tye gevra. Ten eerste wil ek vasstel of u wel aartappels en/of tamaties gaan plant, en indien wel of u bereid is om aan die opname deel te neem. Tweedens moet u op drie verskillende datums die hoeveelheid laatroes in u aanplantings skat.

3.2.1 Eerste opname : Aanplantings en eerste laatroesbesmettingsdatums

(i) Voltooi en pos asseblief die groen vraelys so gou as moontlik. Heg ekstra blaaie aan, indien nodig.

(ii) Hou die klein kaartjie en koevert en voltooi en pos dit aan my waneer u laatroes vir die eerste keer opmerk. Draai 'n besmette blaartjie in papier toe - nie plastiek nie - en sluit dit ook by die koevert in.

ALLE INLIGTING SAL AS VERTROULIK BESKOU WORD

Dit mag moeilik wees om u plaas so te identifiseer dat ek dit akkuraat op 'n landkaart kan aandui. Kan u dus asseblief die korrekte titelaktenommer van u plaas ook voorsien. Indien u die oorspronklike plaasnaam ken, voorsien dié ook asseblief. 'n Skets wat verduidelik hoe om by u plaas uit te kom sal ook baie help.

3.2.2 Tweede opname : Die ergheid van laatroes :

Hierdie opname sal aan u verduidelik word in 'n tweede omsendbrief. M.a.w. indien u kies om nie deel te neem nie, sal u nie die tweede omsendbrief ontvang nie.

Al wat van u verwag sal word tydens die tweede opname, is om u aanplantings op 3 verskillende datums te inspekteur waartydens u 'n beraming moet maak van die hoeveelheid laatroes.

6. / Gereelde verslae ...

Gereelde verslae, van die laatroesverspreidingspatroon in kaartvorm, behoort in die „Natal Witness" te verskyn. As hierdie plan nie tot uitvoer kom nie, sal u in elk geval d.m.v. 'n nuusbrief op hoogte van sake gehou word.

DANKIE DAT U TOT HIER GELEES HET

Indien u enige aspek van die projek met my wil bespreek, of wil hê dat ek u en ander boere in u omgewing moet toespreek, skakel my asseblief by telefoonnommer 63320 (Pmb), uitbreiding 526 of 523. U kan my ook tuis skakel by telefoonnommer 3931 Howick (033212). Indien u genoodsaak is om 'n boodskap te laat, kan u seker wees dat ek met u in verbinding sal tree.

In afwagting van ons samewerking tot gemeenskaplike voordeel, is ek,

Die uwe,

C.A.J. Putter

UNIVERSITEIT VAN NATAL

Tel. Add. "UNIVERSITY"

Dept. van Mikrobiologie en
Plantsiektekunde

Falkulteit van Landbou

Posbus 375
PIETERMARITZBURG
3200

„Pathosystems" Navorsingsprojek

Datum:.....

ENGLISH ON REVERSE SIDE

Mnr. .....

.....

.....

.....

Geagte .....,

Aangeheg vind asseblief 'n beskrywing van 'n navorsingsprojek wat ek onderneem in samewerking met die Universiteit van Natal. Die projek is prakties van aard en m.b.v. u ondersteuning is ek oortuig dat verbeterde metodes vir die beheer van vroëroes en laatroes van aartappels en tamaties ontwikkel sal kan word.

Indien gereelde opnames van besmette en onbesmette plase gemaak kan word, mag dit moontlik wees om die verspreidingspatroon van die siektes in kaartvorm uit te beeld. Vir hierdie doel vra ek u hulp en samewerking in die volgende aangeleenthede:

(1) Lees asseblief die aangehegde beskrywing van die projek en voltooi dan die groen vraelys en stuur dit terug aan die Universiteit in die koevert wat ingesluit is.

(2) Om gereelde inspeksies van u aartappel- en/of tamatie-aanplantings te maak en om my asseblief te laat weet wanneer :

(i) U vir die eerste maal laatroes oplet. Stuur dan die aangehegde kaartjie en 'n voorbeeld van 'n besmette blaar aan my terug.

(ii) Laatroes erger word. 'n Eenvoudige sleutel om die ergheidsgraad van laatroes te meet sal voorsien word sodra ek u eerste groen vraelys ontvang.

(3) Indien nodig, sal ek u plaas wil besoek om verdere inligting in te samel en sal dit waardeer as u so 'n besoek sal toelaat.

Van besondere belang is enige aanmerkings wat mag spruit uit u persoonlike praktiese ondervinding van hierdie siektes. Hierdie projek het juis sy oorsprong in ondervinding wat deur boere opgedoen is en om hierdie rede beskou ek u ondervinding as baie waardevol. As hierdie opname idees van 'n gemeenskaplike aard aan die lig bring, mag dit die weg wees waarlangs na 'n oplossing van die gemeenskaplike probleem gesoek kan word.