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DISEASE MODELING

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SUMMARY

To achieve efficient control of pests and diseases in crops a thorough understanding of the influence of weather on epidemics is needed. Different approaches used to forecast foliar pathogens are considered in four categories: simple forecasting rules; statistical associations; logistic equations; and computer simulation models. Relationships between weather and disease which are suitable for incorporation in simulation models are examined. These include the effect of weather on sporulation and dispersal, infection, latent period and infectious period.

INTRODUCTION

Weather has a major influence on pests and diseases of crops and is the main cause for season to season differences in severity of attack. These differences are an important source of uncertainty in producing crops. Poor farmers may have no other choice than to take the risk of growing crops without control and they will be entirely at the mercy of the weather. The other extreme, common in the developed world, is to use profilactic chemicals (that is to apply pesticides whether or not they are necessary) to guarantee control of pests and diseases. Both of these extremes have major disadvantages. On one hand the poor farmer may face bankrupcy if the crop is devastated, and on the other hand excessive use of pesticides leads to build up of resistance by pests to chemicals, undesirable consequences in the environment and, commonly, unnecessarily reduced profit margins. It has long been realized that decisions for applying expensive control measures can and should be based on the season's

weather and there are numerous examples of proposals for disease forecasting schemes in the scientific literature. However, only a small proportion of these schemes are utilised by farmers or agricultural extension services. For a forecasting scheme to be utilised, it must be robust and reliable and at the same time be practical and easy to use.

Alternative approaches to the use of chemicals for pests and disease control include agronomic methods and the use of resistant plant material. To design agronomic practices which reduce risk (such as manipulation of crop density or sowing date), a thorough understanding of the way that weather affects pests and diseases is necessary. The same is true for the correct interpretation of screening trials in breeding programs for resistance to pests and diseases.

There is a long history of research aimed at improving our understanding of how weather affects pests and diseases. Here we will consider some approaches of different complexity which have been used to forecast foliar pathogens of crops: 1) simple forecasting rules; 2) statistical associations; 3) logistic equations; and 4) computer simulation models. Most attention will be given to computer simulation models and the methods used to incorporate the effects of weather

1. Simple forecasting rules

Numerous attempts have been made to identify simple weather variables which can be used to forecast serious outbreaks of disease (epidemics). Schemes of this type were first proposed in 1912 to provide advice for the control of vine downy mildew in Europe (Popular, 1981). Similar schemes were proposed for potato late blight (eg. Beaumont, 1947) and for apple scab (eg. Mills and LaPlante, 1954) and there are numerous examples for these and other diseases in more recent literature. Rules of this type were normally based on careful observations of disease in the field and concurrent weather records. They relied to a large extent on intuition and, because of their simple nature (which is attractive), their use was usually limited to the region where they were originated. An example is the Beaumont period for potato late blight, applicable in the south west of England. This requires a minimum temperature of 10^{o} C or above and a relative humidity of 75% or above for at least two days.

2. Statistical associations

A variety of statistical techniques have been used to determine quantitative relationships between weather variables and disease. These include multiple regression analysis (eg. Royle, 1973), correlation and path analysis (eg. Mehta *et al.*, 1990) the value of these analyses varies in providing insight into the way that weather affects diseases. The techniques are often used to indicate which weather variables are most important to disease in the field, but for this the results must be interpreted very carefully. This is partly because there is a large degree of autocorrelation between the weather variables themselves, so it is usually not possible to deduce cause and effect from the statistical relationships. In addition the amount of disease in a crop changes with time and any weather variable which also changes with time is likely to be correlated with the amount of disease, whether variables and the rate of change of disease with time would be more valuable. Some researchers have used multiple regression to associate weather variables with particular processes in the life cycle of pathogens such as infection or sporulation (eg. Royle, 1973). These have lead to some success in arriving at simple expressions for forecasting disease but they are likely to break down if the weather variables fall outside the range encountered during their formulation. Their validity may also be limited geographically.

3. Logistic equations

Disease epidemics are commonly observed to follow a sigmoid pattern with time. Van der Plank (1963) formulated equations to explain this pattern and identified a parameter (the apparent infection rate (r) which determines the speed at which epidemics develop.

$$dx/dt = rx(1-x) \qquad (1)$$

where x is the proportion of host material affected by disease and dx/dt is the rate of change of x with time. The value of r depends on the latent period, the infectious period, the extent to which the weather is congenial to sporulation, dispersal and infection and the susceptibility of the host.

Equation 1 has often been shown to be a good descriptor of epidemics but is of little value in ascribing the reason for differences between seasons. The effect of changing the value of r is shown in Figure 1.

In Equation 1, it is assumed that the rate of increase in disease is directly proportional to the amount of disease. In reality only those lesions which are actively sporulating will affect the rate of increase of disease. There is a delay (the latent period) between the time of infection and the development of sporulating lesions and a limited period (the infectious period) during which lesions produce spores. Lesions which have stopped producing spores are included in disease severity assessments, but do not affect the rate of disease increase. A logistic equation can be written to account for the latent period (p) and the infectious period (i) (Van der Plank, 1963).

$$dx_t/d_t = R_c \left[x_{t-p} - x_{t-p-i}\right] \left[1-x_t\right] \qquad (2)$$

where x_t is the proportion of diseased material at time t. The corrected basic infection rate, R_c, will be affected by the host suceptibility and the extent to which weather is congenial to infection, sporulation and dispersal. Latent and infectious periods are also affected by the weather (temperature in particular).

4. Computer simulation models

Computer models to simulate disease epidemics were first developed by Waggoner (1968) and Zadoks (1968). Since then descriptions of simulation models have been published for more than 20 different diseases (Teng, 1985). The fundamental concept central to all these models is similar to that used to formulate Equation 2. The host is considered to consist of numerous disease sites, each of which has one of four possible states (Zadoks, 1971):

1) vacant (healthy)
2) latent (infected but not yet sporulating)
3) infectious (releasing spores)
4) removed (post-infectious).

Sites change from one state to another sequentially at rates which are dependant on the pathogen, host, environment and the state of the system. For example the rate of change from state (1) to state (2) will depend on the number of infectious sites (the value of (3)), the rate of spore production, and the effectiveness of both spore dispersal and infection. The rate of change from state (2) to state (3) will depend on the latent period and the rate of change from state (3) to state (4) will be depending on the infectious period. Weather affects sporulation, dispersal, infection and the latent and infectious periods. We will now consider examples of relationships used to link weather to these various parts of the disease life cycle.

Sporulation and dispersal

These two processes are sometimes considered together because experiments to determine relationships do not always differentiate them. For example, the number of airborne spores of *Cercospora arachidicola* over a groundnut crop during an epidemic of early leaf spot was found to be related to temperature (T, ^{o}C), rainfall (R, mm) and the period of relative humidity >90% (RHP, h) during the previous three days (Alderman *et al.*, 1987).

$$CD = (\exp(0.2968T + 0.1123RHP - 0.942R + 0.55717))/10000$$

where CD is the conidial density (conidia m^{-3}). Long periods of RH>90% probably coincide with long periods of leaf wetness which favour sporulation. The negative effect of rain is explained by spores being washed out of the air.

The effect of leaf wetness on sporulation was examined by Bashi and Rotem (1975). They demonstrated that for *Alternaria porri* on potatoes, alternate wet and dry periods stimulated spore production compared with continuous wetness. With *Stemphylium botryosum* on tomatoes similar spore numbers were produced with intermittent and continuous wetness.

In a model of rust on groundnut, spore production, liberation and deposition are considered separately (Savary *et al.*, 1990). The total number of spores produced per lesion depends strongly on temperature (Savary, 1985) and the response curve obtained from Savary's data (Fig.2) shows a sharp peak at the optimum temperature (27°C). Savary *et al.*, (1990) assumed that assimilates required for spore production were derived directly from net photosynthesis. As a result their model predicted a strong correlation between daily solar radiation and spore number in the canopy.

The number of spores per lesion can be reduced through leaching by rain (Savary and Janeau, 1986). This effect is represented in the model by considering three conditions: no rain, less than 5 mm and greater than 5 mm of rain , resulting in losses of 0, 0.25 and 0.5 of the canopy spore content respectively.

Spore liberation is assumed to depend on the minimum relative humidity and rainfall (Savary *et al.*, 1990). With no rain, if the minimum relative humidity is less than 70%, the rate of liberation is set to the maximum value for dry conditions. If the minimum relative humidity is greater than or equal to 70%, half the maximum rate of liberation is used. With rain, if the daily rainfall total is less than 5 mm, the maximum rate of liberation is increased by 10% of that in dry conditions. If the daily rainfall total is greater or equal to 5 mm, spore liberation is set to zero.

Spore deposition is taken to be three times greater with a wet than a dry canopy (Chamberlain and Chadwick, 1972). It is assumed that the canopy is wet if rain occurs.

Infection

After spore deposition, the majority of pathogens require a period of leaf wetness for infection (powdery mildews are an exception to this). For a given amount of inoculum, the extent of infection and number of lesions which subsequently develop is strongly influenced by the duration of leaf wetness and temperature. A minimum period of leaf wetness

(W_{min}) must be provided for lesions to develop, and when wetness periods are increased from W_{min} the number of lesions increases to an asymptote (see Fig.3 for groundnut rust). Both W_{min} and the asymptote are temperature dependent. W_{min} increases as the temperature decreases from the optimum and the value of the asymptote can be defined in terms of three cardinal temperatures (T_{min}, T_{opt} and T_{max}, see Fig.4)

Pathogens which require long periods of leaf wetness for spore germination, germ tube growth and host penetration are usually adapted to intermittent wetness (Bashi and Rotem, 1974). In these cases (eg. late leaf spot in groundnut) infection depends on the total period of wetness, often spread over several days. Other pathogens such as groundnut rust invade the host more rapidly, but require continuous wetness for successful infection.

Latent period

The latent period is the time between inoculation and sporulation of resulting lesions. It is common to take the time of sporulation as the time when 50% of all the lesions are sporulating. The latent period depends on the pathogen, its host and the environment. Variation in the latent period with different host cultivars is an important component of resistance (Zadoks, 1972; Subrahmanyam *et al.*, 1983). Temperature is the main weather variable which influences the latent period. The relationship between the two variables is non-linear and has often been fitted with a polynomial equation (eg. Teng *et al.*, 1980). By looking at a wide range of temperatures, Beresford and Royle (1988) found that the relationship is hyperbolic (Fig. 5), and that the reciprocal of the latent period is linearly related to temperature (Fig.6). This relationship defines the latent period in thermal time, and for leaf rust on barley cv. Midas, the value is 136° C days. A similar relationship holds with data given by Savary (1985) for groundnut rust in West Africa, where the latent period is 192° C days and the base temperature is about 9° C.

Infectious period

Few studies have examined the effect of weather on the infectious period. It is likely however that, for certain pathogens in nature, there is no simple relationship because the infectious period is strongly dependant on the lesion density. Yarwood (1961) demonstrated that for *Uromyces phaseoli*, the infectious period was halved when the lesion density changed from 1 to 15 lesions cm^{-2}.

For groundnut rust the dynamics of spore production was examined by Savary (1985). His results show that the infectious period is influenced by temperature. The longest period which coincided with maximum total spore production was at the optimum temperature (27°C) and the period reduced at smaller and greater temperatures. The relationship probably follows a classical biological response curve and I have tentatively fitted a line of this form though points extracted from his data (Fig. 7). Experimental evidence over a wider temperature range is needed to confirm the relationship.

Rotem (1977) suggested that low temperature is likely to preserve the sporulating potential of the pathogen, and this would result in vigorous sporulation when the temperature rises. He also suggested that when night temperatures are optimum for sporulation, high daytime temperatures may shorten the sporulation period.

Progress

Computer simulation models have led to valuable insights into mechanisms affecting disease epidemics. For polycyclic diseases which have been considered here, much emphasis is normally placed on spore production and dispersal. This is because the multiplication rate (equivalent to R in equation 2) depends on the number of effective spores produced in each integration period. An effective spore is one which causes a new lesion (Zadoks, 1971). Validation of spore production and dispersal in the field is difficult, especially when dispersal by both splash and airborne spores are possible. This is an area where more experimental work is required.

Simulation models have demonstrated the importance of the latent period to disease epidemics. This is an important component of resistance which can be successfully incorporated in models. Methods are available to include delay functions which simulate the variability in latency within a pathogen population (Berger and Jones, 1985). The form of relationship between temperature and latent period are now well understood, and systematic experiments are needed to determine the latent period in thermal time for different diseases and different crop cultivars.

Quantitative information about the effect of weather on the infectious period is generally lacking and would a fruitful area of research.

Recently models of crop growth and disease have been combined (eg. Bourgeois, 1989; Savary *et al.*, 1990). This provides the opportunity to classify leaves of the host according to age and to incorporate the effect of leaf age on susceptibility. With some crops the latent period varies with the crop growth stage (eg. leaf rust of barley; Boresford and Royle, 1988) and this aspect of the system can be incorporated. The effect of pathogens on leaf death or defoliation can also be included. This is of particular importance when combined crop growth and disease models are used to examine the loss of yield caused by disease.

As disease models become more sophisticated, more detail is required about the crop microclimate. This is particularly true when considering the effect of intermittant wetness periods on infection and sporulation. These aspects of disease simulation may prove to be important to our understanding of epidemics, but wherever possible efforts to simplify the system should be explored. Over simplification will tend to produce models which are only applicable in a narrow range of conditions. Over complicated models usually contain redundant parameters which are virtually impossible to verify experimentally.

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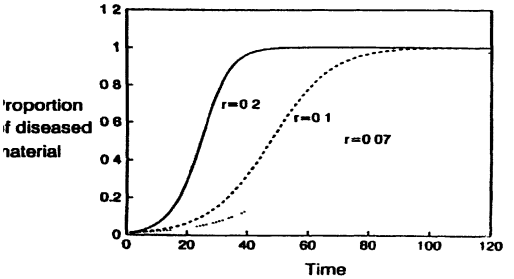

Fig. 1 : The effect of changing the apparent infection rate (r) in Equation 1

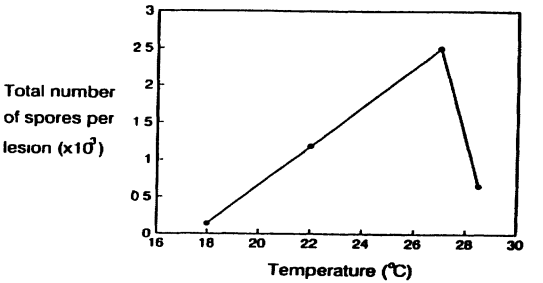

Fig 2 : The relationship between spore production and temperature for groundnut rust Data were obtained from Savary (1985)

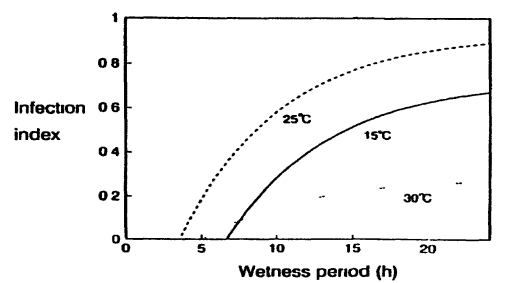

Fig. 3 : Relationships between leaf wetness period and lesion density for groundnut rust at different temperatures

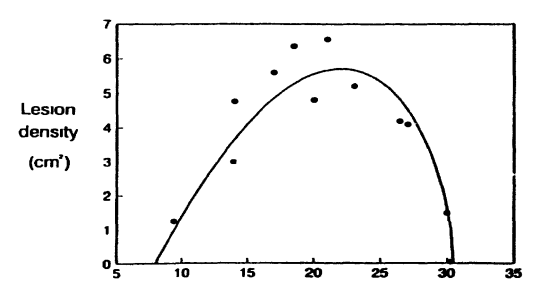

Fig 4 : The relationship between lesion density and temperature at the time of infection with non-limiting leaf wetness

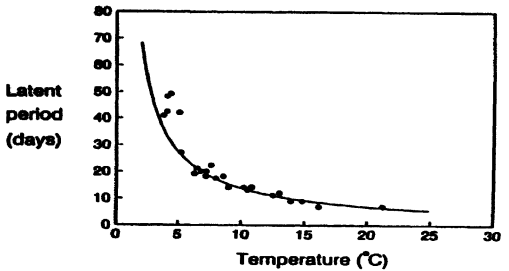

Fig. 5 : The relationship between latent period and temperature for leaf rust of barley. Redrawn from Beresford and Royle (1988)

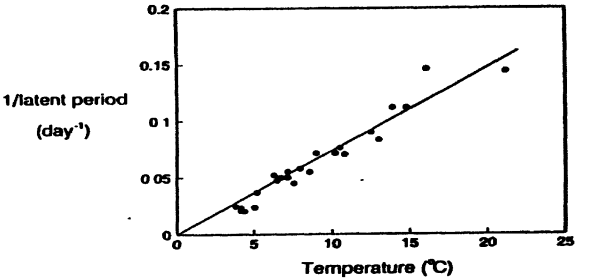

Fig. 6 : The reciprocal of the latent period plotted against temperature for leaft rust of barley. Redrawn from Beresford and Royle (1988)

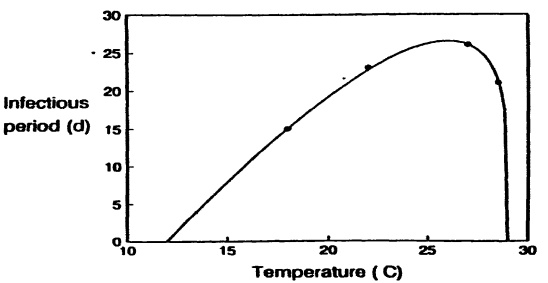

Fig. 7 : The relationship between infectious period and temperature for groundnut rust. Data were obtained from Savory (1985)