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Special Lecture

THE NATURE AND CONTROL OF EPIDEMICS

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An epidemic occurs when disease spreads in a population. Infection of a plant results from the activity of a single spore, rather than from the synergistic action of a group of spores. Infection is not the result of a threshold effect. The typical curve of progress of disease in a population with time is of sigmoid shape, and results from two effects. Early, when inoculum increases logarithmically and most plants are healthy, infection is proportional to inoculum. Later, when a majority of plants are infected, inoculum competes for infectable sites on healthy plants. The sigmoid relation can be converted to a straight line by suitable transformations. That appropriate for a multiple cycle disease differs from that for a single cycle disease; each constitutes a different type of epidemic.

Of disease forecasts, some are based on the amount of inoculum present at a given time. Others are based upon the rate of multiplication of inoculum as well, as this is affected by weather. Epidem simulates an epidemic when sufficient information is available or can be assumed. It has the added feature of evaluating the impact of a control measure upon the course of an epidemic.

Two types of epidemic are contrasted. Multiple cycle diseases that produce local lesions on their hosts develop rapidly and can affect a whole crop rapidly. Usually inoculum is rapidly air-borne. Systemic diseases are usually slow paced and inoculum is either produced infrequently or is distributed by agencies that operate slowly or seasonally. The systemic diseases tend to be more important as epidemics in perennial plants or in plants that are vegetatively propagated when inoculum is carried in the process.

Epidemics of systemic disease can be controlled if sufficient inoculum can be destroyed. In such diseases, however, inoculum is often not available so that its destruction is difficult. For multiple cycle diseases, the most effective treatments involve both destruction of inoculum with protectant fungicides and reduction of its rate of production with eradicant or antisporulant materials, or with systemic fungicides and chemotherapeutants. A combination of control measures offers additive advantage over these same measures applied singly when modes of action differ.

Man blamed the weather for plant diseases long before he knew their cause. Blaming the weather, like other kinds of blame, shifts responsibility without finding the underlying cause. Early accounts attributed blasts and blights, rusts and smuts to hot weather and to cold, to wet seasons and to dry.

Hundreds of years later came the demonstration that diseases are caused by germs, by fungi, bacteria or viruses, and that diseases caused by them are contagious. This was the beginning of modern epidemiology. Then L. R. Jones and his students at Wisconsin turned their attention to a study of the effects of environment on disease development. Plant pathologists soon learned which diseases are weather dependent and which are not; they learned also which factors in the environment affect disease development and why they do so.

Fear is often associated with the word epidemic. To the common man, it means deadly disease running out of control. Everyone is threatened. Not all epidemics are explosive, however. The most spectacular examples of plant pathology involve the epidemics of late blight of potato, stem rust of wheat, coffee rust, rice blast. But many other pathogens of crop plants cause epidemics where the increase of disease with time occurs slowly.

An epidemic occurs when disease spreads in a population of individuals. The simplest requirement is that disease will spread from one lesion to cause more than one additional lesion. If lesions are local, they must multiply. If the disease is systemic, the lesion comes to involve the entire plant and the number of diseased plants must increase.

The Relation of Amount of Inoculum to Number of Infections

The increase in disease with time, obviously, can occur only if inoculum increases, is spread to a court of infection, and there successfully invades healthy host tissue. The amount of inoculum is obviously an important factor in the development of an epidemic. Given a population of susceptible plants in an environment favouring infection, the greater the amount of inoculum, the greater the number of infections. This is the concept of inoculum potential, initially introduced by Horsfall (1932). As first used, the term referred strictly to the quantity of inoculum present. More recently, this definition has been modified to include qualitative factors as well (Dimond and Horsfall 1960, 1965; Garrett 1960), and the effect of the environment of the efficiency of inoculum in producing disease (Horsfall and Dimond 1963).

Usually a population of inoculum is associated with a single infection. The number of spores involved in the development of a lesion varies with circumstances. However, under optimal conditions, for each of three tomato foliar conditions, McCallan and Wellman (1943) determined that an average of 15·4 spores is associated with a late blight lesion, 59 spores with an early blight lesion, and 500 per square centimetre of leaf surface with a *Septoria* blight lesion.

Such data have been interpreted in two ways. Gaumann (1950) referred to the numerical threshold of infection, which implicitly carries the assumption that inoculum in populations is required to overcome the barriers of the

host. Alternatively, inoculum can be assumed to act independently, rather than synergistically. As van der Plank (1967) has stressed, the answer to this question has important consequences for disease control. If spores can infect only when present in a population above the threshold level, then no infection can occur if inoculum is reduced by any means below this critical level. If inoculum behaves independently, then the probability of infection is steadily reduced as inoculum is decreased. In the first case, control can be absolute when inoculum is reduced below the critical threshold level: in the second case, infections steadily decrease with inoculum but absolute control is unlikely because eradication of all inoculum is unlikely.

Both Garrett (1960) and van der Plank (1967) have carefully considered existing evidence on the relation between the amount of inoculum and the amount of infection and particularly the question of whether a single spore can cause an infection or a population is required. Infectivity titrations have shown that whenever plants have been exposed to carefully controlled concentrations of inoculum the number of infections is proportional to the amount of inoculum present. No evidence exists for a threshold. This relation holds for such diverse organisms as stinking smut of wheat (Heald 1921), *Sclerotium rolfsii* on sugar beet (Fig. 1) (Leach and Davey 1938), potato wart (Glynn 1925), and Fusarium wilt of tomato (Haymaker 1928). In rust studies, when inoculum consists of a mixture of races, the pustule that develops after infection is of a single race. When infection occurs, it is the result of successful invasion of host tissue by a single spore, the one that invaded first or that dominated the population. The other spores failed.

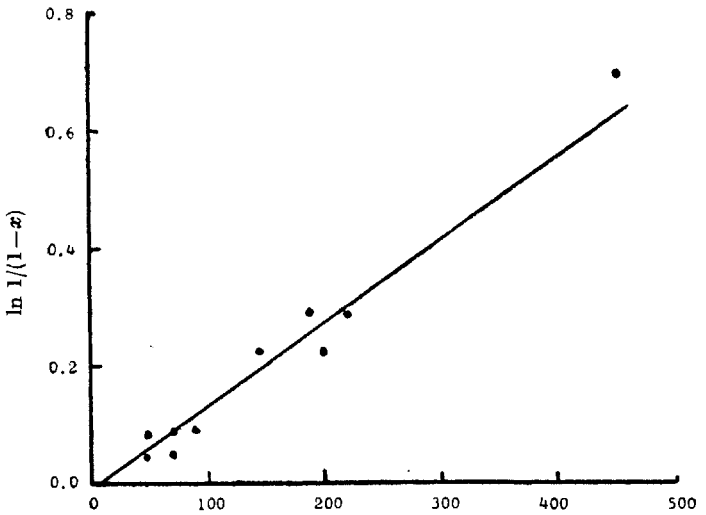

FIG. 1. Relation of inoculum density to frequency of infections of sugar beet by *Sclerotium rolfsii* (Data of Leach and Davey).

Some spores contain compounds that influence germination or infection. What of them? Spores of *Puccinia graminis* f. sp. *tritici* contain a stimulator of germination, but Allen and Dunkle's (1971) studies have indicated that this compound is present in low amounts in spores and is demonstrable only when spores occur in mass. Dispersed spores, about to infect the host, do not occur in such densities. On this basis and the experimental results of infectivity titrations of Rowell and Olien (1957), van der Plank (1967) considers that these stimulators have little function in infection, naturally. Spores of bean rust and stem rust of wheat contain germination inhibitors (Allen 1955), but these spores cause infections in proportion to their concentration when spore density is low, but depart from a linear relation when density is high (Rowell and Olien 1957; Davison and Vaughan 1964).

The host specific toxin victorin is present in spores of *Helminthosporium victoriae*. It determines pathogenicity of virulent strains to susceptible lines of oats. Such spores cannot attack lines of oats resistant to Victoria blight. When spores lack the toxin, they cannot attack susceptible oats, but can do so if toxin is supplied artificially. In the case of *H. carbonum*, the picture is the same on corn. Because of its control over pathogenicity, such toxins have been called by Scheffer and Pringle (1967) primary determinants of disease.

The effect of such host specific toxins on the infectivity titration has not yet been formally studied, but available information permits one to draw tentative conclusions. Preliminary evidence suggests that the toxins are compatible with a components of cell membranes in the susceptible host but not in the resistant host (Scheffer and Pringle 1967). Their function is one of recognition. These toxins are potent to the extent that they can act on host tissue from an individual spore in all probability. If so, spores act independently in causing infections even in this case. We must conclude, therefore, that infection is rarely, if ever, a threshold phenomenon.

The Typical Epidemic Curve

In the course of a typical epidemic, the number of infections increases with time as the amount of inoculum increases. In a multiple cycle disease such as late blight of potato, the amount of inoculum increases logarithmically with time. While most plants are still healthy, the number of lesions also increases logarithmically with time. Soon multiple infections occur on the same plant. As inoculum continues to increase and more plants become infected, the proportion that is still healthy becomes limited. As healthy plants become rare, the chance that inoculum will fall on a healthy plant gets less and less. These are the reasons why the typical curve for an epidemic is sigmoid in shape when proportion of diseased individuals is plotted against time (Fig. 2). Van der Plank (1963) has shown how the sigmoid curve can be

converted to a straight line for a multiple cycle disease by plotting the function $\ln x/(1-x)$ against time. The slope of this line, r, defines the apparent rate of increase in proportion of diseased plants, x, with time, t. As van der Plank (1967) has emphasized, these relations describe the situation accurately only early in the development of an epidemic.

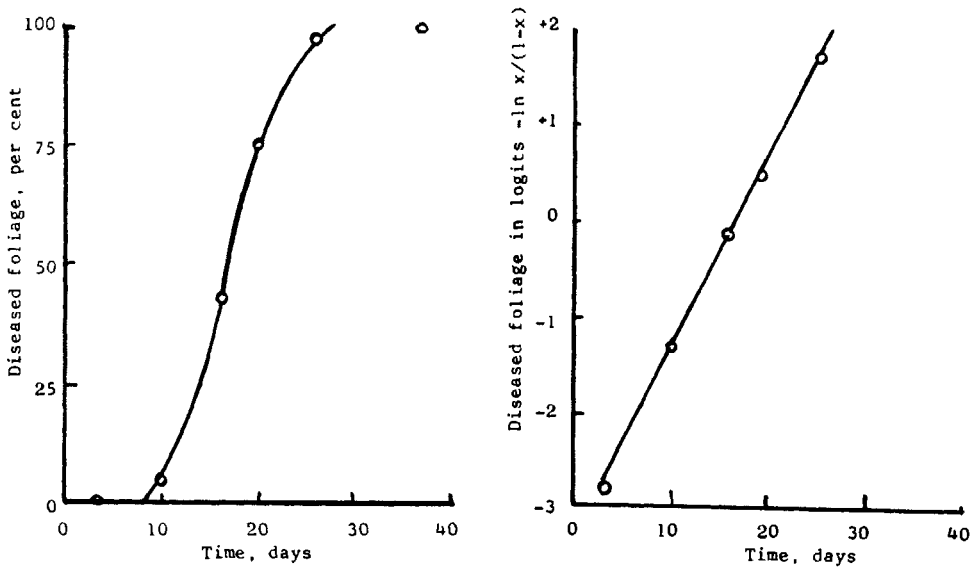

FIG. 2. Time course of a late blight epidemic (Data of E. C. Large).

Only in unusual circumstances can the amount of inoculum or the increase in inoculum be measured directly in an epidemic. When inoculum can be caught in a spore trap and its concentration determined by counting the numbers of spores in a unit volume of air, inoculum is already abundant and the epidemic is already well in progress. The stage of an epidemic that is of greatest interest occurs when methods of quantitative measurement are least reliable. At the stage of an epidemic where changes in inoculum can be measured quantitatively, the relation between amount of inoculum and number of infections may no longer be proportional. Our interest in epidemics is to forecast them and, hopefully, to control them. This can be done only when lesions and inoculum are rare. The purpose of a protective fungicide is to destroy inoculum. When inoculum is rare and distributed it is difficult to cover the crop with a protectant fungicide so as to destroy a high percentage of inoculum, but destruction of inoculum at this time can lead to control. Later, when inoculum is abundant, much of it survives a fungicide and its rate of multiplication can keep the epidemic going. It is common experience that in a bad year for late blight, fungicides delay the epidemic, but eventually

tually all fungicides fail as more inoculum survives and multiplies rapidly. To delay an epidemic is easier and more meaningful than to attempt to curb the epidemic after it has well begun.

We have noted how, in the early stages of an epidemic, the increase in number of infections with time is logarithimic, when other factors remain unaltered. At this time, the number of infections is more or less directly related to the diseased individuals. During this period, the same amount of time is required for the percentage of diseased plants to change from 0·01 per cent to 0·1 per cent as the time for the percentage to change from 1 per cent to 10 per cent, as van der Plank (1960) has noted. During the first interval inoculum is too low to be measured and lesions on plants may escape detection when only one plant in 10,000 bears a lesion. When from 1 to 10 per cent of the plants are infected in the field, a forecast is of doubtful value. An epidemic is already under way and control measures should already have been imposed. This is our dilemma.

Disease Forecasts

Some methods of forecasting disease are based on estimates of the amount of inoculum at the beginning of the season. When spread is slow, initial inoculum provides a useful index of potential damage to the crop. When a disease is not weather dependent, once it is introduced into the crop, or when weather seldom limits disease development, once the season starts, initial inoculum is a useful index for a forecast.

This is the basis of Leach and Davey's (1938) forecast of damage to sugarbeets, based on the number of sclerotia of *Sclerotium rolfsii* in the soil at the beginning of the growing season.

Stevens (1934) developed a comparable method of forecasting the abundance of Stewart's wilt of sweetcorn in the northeastern U.S. The causal bacterium overwinters in the corn flea beetle and is carried by it to spring-planted corn. Steven's forecast is based on the mean temperatures of the winter months, which is an index of survival of the corn flea beetle. This in turn measures the amount of inoculum at the start of the growing season.

Chester's (1942) forecast for wheat leaf rust in Oklahoma provides an indirect measure of the amount of inoculum at the beginning of the growing season. It is based upon his observation that temperature and rainfall between December and March are well correlated with the amount of leaf rust infection in wheat in early April.

A disease forecast based on a sensitive indirect measure of primary inoculum is that for the bacterial blight of rice, caused by *Xanthomonas oryzae*. The bacteriophage for this pathogen can be detected and counted before the pathogen itself becomes abundant. Forecasts for outbreaks are made and

control measures are instituted on the basis of the time of appearance and abundance of the phage in irrigation water of rice fields or in water from the paddy itself (Mizukami and Wakimoto 1969).

Forecasts for a number of diseases are based, not upon the amount of primary inoculum, which is presumed to be present, but upon a sequence of meteorological conditions that favour multiplication inoculum. A forecast of disease outbreak is made when abundance of inoculum and weather conditions combine to make an outbreak of disease probable. Present methods of forecasting late blight of potato are examples of this kind of forecast.

EPIDEM (Waggoner and Horsfall 1969), which Horsfall has discussed, instead of being a method of forecasting in the sense that we have been discussing, is a basis for simulating a disease outbreak, or mimicking what will happen under a sequence of weather conditions. It is based, not upon averages of weather events involving long periods of time, but upon events in three-hour intervals, because the time clock of *Alternaria solani* is programmed to respond in these short intervals. To use EPIDEM for forecasting purposes, one must make assumptions about the sequence of weather events in three-hour intervals for as long as one desires to extend the forecast. If EPIDEM can simulate with high accuracy what will happen, the accuracy is based on detailed assumptions. Accuracy of the forecast depends upon accuracy of the assumptions.

But EPIDEM has a use that is not obvious at the outset. It can be used to predict the impact of a control measure, applied at different times, upon the course of an epidemic. One can modify the programme slightly and then evaluate the comparative effect of a protectant fungicide that only kills existing inoculum, an eradicant fungicide that kills out some existing lesions and so reduces multiplication of inoculum for a short time, or an antisporulant that reduces multiplication rate of inoculum by decreasing the number of spores produced in each lesion for a longer period of time. One can evaluate the impact upon the epidemic of a chemotherapeutic agent which, by virtue of changing resistance of a host, may significantly reduce the multiplication rate of inoculum over a period of several weeks. In my opinion, this kind of forecasting offers a powerful advantage of a kind that we have never had before.

Learning to exploit EPIDEM for a variety of different diseases will be a necessary next step. Learning to exploit our ability to evaluate different control measures in these situations will add tremendously to our understanding of epidemics and particularly how to control them.

Types of Epidemics

The familiar types of epidemics such as those caused by late blight or stem rust of wheat can cause extensive damage to an entire field in a few

days. Both the pathogens and the diseases they cause spread rapidly and multiply rapidly. Usually, though not invariably, these pathogens have air-borne spores, produce local lesions, and cause multiple cycle diseases.

Systemic diseases also cause devastating epidemics. Both the pathogens that cause them and the epidemics they produce have characteristics that stand in contrast to the pathogens that cause multiple cycle diseases and form local lesions.

A few pathogens causing systemic diseases produce air-borne spores, e.g., bunt or stinking smut of wheat. Inoculum of most systemic pathogens however, is transmitted in some other way. Virus diseases are transmitted by insects, other animals, through seeds, or mechanically. The vascular wilt diseases are caused by pathogens that may be soil-borne (*Fusarium oxysporum*), or insect-borne (*Ceratocystis ulmi* or *C. fagacearum*) and systemic bacterial pathogens may be borne in soil, by water, by insects, or transmitted mechanically.

When a pathogen undergoes a single reproductive cycle in a growing season, its reproduction rate is much less than for a pathogen that undergoes many cycles in the same period of time. In some systemic diseases, much of the inoculum is not available to cause infection of healthy plants. The pathogen that causes Dutch elm disease invades the tree and produces inoculum at the source. Only that which is borne by the insect vector or carried through root grafts to healthy trees arrives at the infection court where it can be effective. Most of the virus produced in a diseased plant is unavailable for infecting other plants. For insect-borne viruses the amount of inoculum transmitted and effective is but a fraction at the total produced. What limits the increase of virus disease is not the amount of inoculum produced, but the number of insects that bear inoculum to the infection court. The population of inoculum must coexist with the vector population and the population of susceptible hosts at the right times if inoculum is to be effective.

Whatever the cause, systemic diseases multiply more slowly in a population of plants than multiple cycle, local lesion diseases do (van der Plank 1960, 1963). Partly, this is a matter of quantities of effective inoculum in the two cases. Partly, this results from the method of transmission of inoculum. Partly, it results from the size of the lesion. Inasmuch as a single infection in a systemic disease eventually involves the entire plant, a second, or a third infection on the plant does not affect the pace of the epidemic. It is wasted. When only a few plants in the population are infected, and when inoculum is well distributed, additional infections are likely to occur on previously uninfected plants. As a higher proportion of individuals becomes infected, the number of multiple infections increases. The multiple infections contribute to the epidemic in a local lesion disease but are wasted in a systemic disease.

Relative to a local lesion disease, the latent period for a lesion in a systemic disease is long. Systemic diseases not only multiply slowly, they also spread slowly. We have noted how each lesion must cause more than one other lesion if an epidemic is to sustain itself. When the host plant is an annual, the rate of spread must be faster than when the host is perennial. Although many annuals suffer from systemic diseases, some of the most devastating systemic diseases occur on long-lived plants. The X disease of peach and the virus diseases of stone fruits and citrus, Dutch elm disease, and oak wilt are examples.

Merrill (1968) has calculated an average rate of increase of Dutch elm disease as of the order of 0·5 to 0·9 per unit per year. A disease with such a multiplication rate, obviously, could have little consequence in an annual crop, but in the case of a tree with a life span of a century, the survival of the American elm is threatened. In epidemics involving systemic diseases, rate of spread is significant in relation to the life span of the host.

When plants are vegetatively propagated and viruses are transmitted in the process, the course of the epidemic is similar to a systemic disease in a perennial plant with an infinite life span, even if the crop is grown as an annual. The common virus diseases of potato are examples, and the situation confronting the potato farmer of 50 years ago bears testimony to how a systemic disease that multiplies and spreads slowly can invade almost the whole population if given enough time.

When inoculum multiplies slowly or new infections appear slowly, destruction of inoculum is an adequate control measure. In the case of the tuber-borne potato viruses, inspection, roguing, and certification eliminated inoculum faster than it spread to new plants and eventually restored the health of potatoes in the principal countries where they are grown. When inoculum multiplies and new infections increase rapidly, the simple destruction of inoculum will not suffice to control an epidemic.

The Control of Epidemics

Types of epidemics have been contrasted in the foregoing presentation. In the multiple cycle disease, the pace is fast. Spores are rapidly air-borne. Successive lesions bear successive crops of spores in rapid sequence. When large numbers of spores are produced by each lesion, inoculum multiplies rapidly. When weather favours the process and healthy susceptible host tissue is available, the number of infections multiplies rapidly and the disease spreads rapidly in the population. Both initial inoculum and its multiplication contribute to the pace of the epidemic.

In the systemic disease, the transfer of inoculum is usually slow and the multiplication of inoculum is relatively slow. The systemic diseases tend not to depend so much on weather because inoculum is often introduced by

the vector into the host or the host, by its growth encounters inoculum in soil. Often inoculum is not readily available or exposed where it can be subjected to the action of fungicides. Because the distribution of inoculum occurs slowly or because the multiplication rate of inoculum is slow, control measures can be aimed at elimination of inoculum or of the vector.

As van der Plank (1960) pointed out, control measures can be classed according to whether they eliminate existing inoculum or affect its rate of multiplication. The inoculum potential in the quantitative sense that Horsfall (1932) originally defined it, is reduced when the number of infective propagules is reduced. The pace of an epidemic is reduced when multiplication of inoculum is reduced.

The function of a protectant fungicide is reduction of existing inoculum. This is the function of the soil fumigant and the fixed copper spray or dust. Fungitoxicity persists on the sprayed leaf surface for a considerable period of time so that the benefit from protectant fungicide may extend longer than the time required for one crop of spores. When fungicides are applied regularly, their effect is to delay the development of an epidemic. Often, this is enough. When primary inoculum constitutes the principal threat to a crop, a protectant fungicide is all that is required.

Some fungicides have eradicant action. Not only can initial inoculum be inactivated but also the incubating lesions that, if untreated, would soon produce an additional crop of spores. An eradicant fungicide reduces the multiplication rate of inoculum. Eradicant fungicides coupled with protectant fungicides have become a part of the established programme to control apple scab in regions where secondary inoculum is important.

The dithiocarbamate fungicides have an added effect upon the course of an epidemic. Not only are they fungitoxic directly, killing inoculum, but they also reduce appreciably the number of spores produced in lesions that develop subsequently, according to Hodgson (1963). Their effect is both upon initial inoculum and multiplication rate.

The antisporulant compounds that Horsfall and his colleagues have investigated recently function primarily in reducing the multiplication of inoculum. When compounds affect the multiplication rate, they can do so in a variety of ways. They can reduce the number of spores produced by a lesion. This is the obvious and direct course. They can increase the time required for the latent period before spores are formed. If one method of controlling epidemics consists of delaying its development, prolonging the time required for spores to be formed causes a delay with each generation that is produced. Third, a compound that shortens the time that a lesion is able to produce spores reduces the multiplication rate.

Varieties of plants that are tolerant of a pathogen because they have a generalized resistance to it react in two ways to inoculum. Less of the avail-

able inoculum is effective in producing infections (Fig. 3). Infections result in lesions that produce fewer spores. The mechanisms of disease resistance, being different from those for chemical control, permit of additive benefits when the two methods are used in combination as van der Plank (1967) has stressed.

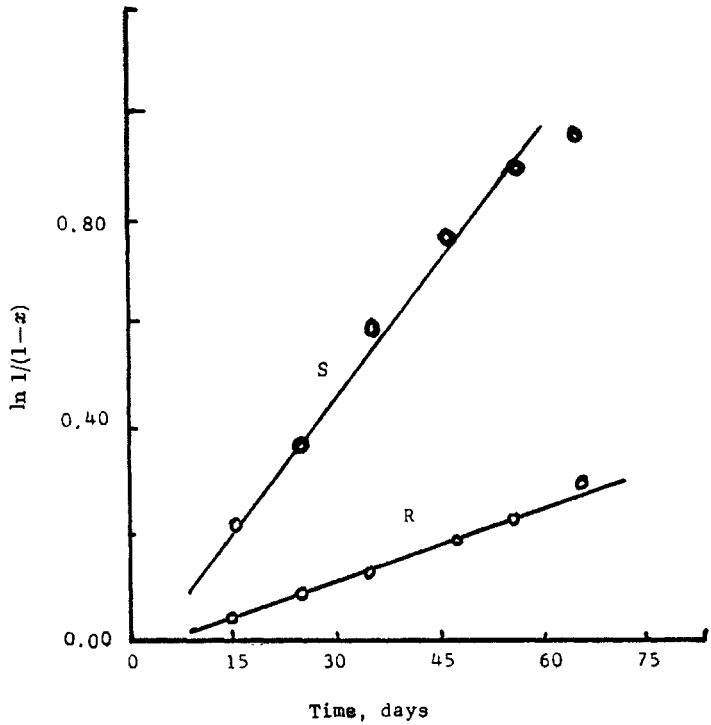

FIG. 3. Time course of *Fusarium* wilt of cotton; S = susceptible variety; R = resistant (Data of Ware and Young).

We cannot close this discussion without a brief mention of the relation of chemotherapeutants to the control of epidemics. These compounds fall into three groups at present. The systemic fungicides of which benomyl and the oxathiins are examples, seem to operate as some other organic fungicides do, in so far as inoculum is concerned. They reduce inoculum and its rate of production simultaneously. More than this, because they are systemic coverage of the host is not important as it is with conventional fungicides. One soil treatment has a much longer effective life than foliar fungicides do. These properties offer opportunity for disease control in situations where we have had no adequate method of control. If the long life of a treatment does not cause the problems that persistent insecticides have, and if residues are nontoxic to man and animals, these materials offer real promise in control of epidemics.

A second group of chemotherapeutants modifies disease resistance of the host through modifying metabolism. Potentially such compounds may

be useful against local lesion diseases, especially when used in conjunction with protectant fungicides in much the same manner as a combination of generalized genetic resistance with fungicides offers advantages over either method of control alone. A variety of growth regulants confer a temporary resistance to vascular wilt diseases, but the resistance is of a type that prolongs the life of an infected plant. Treatment does not affect the rate of infection and does not affect the pace of the epidemic.

A third type of chemotherapeutant modifies pathogenicity. The ability of rufianic acid to effect a cure of tomato seedlings infected with Fusarium wilt is apparently based on its ability to inactivate the pectolytic enzymes of the pathogen (Grossmann 1962). Cure of disease, if and only if effected broadly enough in the population, can reduce an epidemic to minor proportions. Compounds that inactivate primary determinants of disease, such as the known host specific toxins, can control epidemics through removing pathogenic capacity of spores. Such compounds can be sought readily, now that host specific toxins can be produced and assayed. These are possibilities for the future.

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