PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

The Aerial Dispersal of the Pathogens of Plant Disease [and Discussion]

P. E. Waggoner, J. S. A. Green and F. B. Smith

Phil. Trans. R. Soc. Lond. B 1983 **302**, 451-462
doi: 10.1098/rstb.1983.0067

Phil. Trans. R. Soc. Lond. B **302**, 451–462 (1983)
Printed in Great Britain

The aerial dispersal of the pathogens of plant disease

By P. E. Waggoner

The Connecticut Agricultural Experiment Station, P.O. Box 1106, *New Haven, Connecticut, U.S.A.*

The classic model, the logistic, often fits the course of an epidemic of plant disease although the assumptions of propagules proportional to infected plants and random distribution of the propagules are violated. When a thorough simulation of the life cycle of a pathogen is composed, knowledge of its dissemination is the weak link. Although some plant diseases are caused by bacteria and viruses, some pathogens are not airborne and some plants grow indoors; plant pathogens are distinguished by the prevalence of fungi and the durability of their propagules; their hosts are usually exposed outdoors; the pathogens are unaided in their take-off by coughing or sneezing and their landing by breathing; and they are not carried by perambulating hosts. Thus investigations and models of dissemination of plant pathogens concentrate on ways in which spores can be detached and become airborne, on hardiness and relatively long flight and on settling or impacting on foliage outdoors.

1. Introduction

Within the classic, logistic model of the course of an epidemic of a plant disease are the complexities of the take-off, flight and landing of an airborne pathogen and all the complications of infection, incubation and growth of a new generation of propagules. From these complexities I have chosen to discuss the dispersal of plant pathogens and to explore the conception of dispersal in a highly abstract model.

(*a*) *The logistic law*

This law, written first by Verhulst in 1845, explored thoroughly by Lotka, and applied widely to plant diseases by van der Plank (1963), predicts the increase $\mathrm{d}N/\mathrm{d}t$ in diseased hosts in a population of K hosts as time t passes:

$$\mathrm{d}N/\mathrm{d}t = rN(K-N)/K = rN(1-x), \tag{1}$$

where r is the proportionality factor and x is the proportion N/K of the plants that are diseased. From (1) is derived the linear relation between time and logits, the logarithm of $x/(1-x)$. The linear increase of logits in accord with the logistic law as time passes encourages plant pathologists to plot logits of an epidemic and summarize the course of the epidemic in the slope or rate r. Van der Plank (1963) and many others since have shown the linear increase of logits and hence presumed fit of the logistic law to many real epidemics.

To untangle dispersal from the other portions of the disease cycle incorporated in the logistic law, I begin with the rate $\mathrm{d}n/\mathrm{d}t$ of production of the propagules n. This rate is made proportional to the number N of diseased plants, not to the number or size of lesions:

$$\mathrm{d}n/\mathrm{d}t = rN. \tag{2}$$

Second and less simply, the increase dN/dn in the number N of diseased hosts per addition of dispersed propagules n is set proportional to the fraction $(K-N)/K$ of the population that is healthy at the time of the dispersal.

$$dN/dn = (K-N)/K. \tag{3}$$

The acceptable assumptions that all dispersed and effective propagules infect and that multiple infections do not increase the number N of infected hosts are incorporated in (3). Less acceptable is the additional assumption in (3) that the propagules are randomly distributed.

(*b*) *Contagious distribution*

The spread of propagules over a field can be tracked by the visible lesions they cause. If n effective propagules were distributed at random, they would cause n visible lesions distributed at random; and the number of, say, maize leaves with no, one, two and so forth rust pustules should be fitted by the Poisson frequency distribution. In fact, 98 out of 112 frequency distributions of lesions of disparate diseases departed significantly from the Poisson. Too many hosts had no lesions and were healthy and too many had many lesions for the distribution to be fitted by the Poisson function and be considered evidence of random distribution.

Non-random distributions are often called 'contagious' and fitted by the negative binomial function (Anscombe 1950). Turning again to the 112 frequency distributions of lesions of disparate sorts, one finds only 5 out of the 112 distributions were not fitted by the negative binomial. The negative binomial function includes the mean, as does the Poisson, and also a positive exponent k, which is small when the distribution is far from random and very large when the distribution is random and fitted by the Poisson function. A value of unity for k is representative of the range of 0.01 to 4.58 found in the 112 distributions (Waggoner & Rich 1981).

Since the negative binomial generally fits observations of the number n of lesions, it can be used to express lesions n as a function of the number K of all hosts, the portion x diseased and the exponent k:

$$n = Kk\{(1-x)^{-1/k}-1\}. \tag{4}$$

So much for the weak strand of random dispersal of propagules over the field. The concept of a contagious distribution represented by the negative binomial and (4) is stronger.

Concerning the production of propagules in proportion to the number N of diseased plants, I simply argue that although a host with one lesion and another with two are both diseased, the host with two lesions will produce twice as many propagules as the host with only one. Thus it is more reasonable to conceive of propagules as being produced in proportion to the number n of lesions rather than in proportion to the number N of diseased hosts, making the production of dn/dt of effective propagules r times the n lesions given by (4). Multiplying this new dn/dt by the dN/dt derived from (4) manufactures a replacement for the logistic law:

$$dN/dt = (dn/dt)(dN/dn) = rKk\{(1-x)^{-1/k}-1\}(1-x)^{1+1/k}. \tag{5}$$

The replacement incorporates the concepts of propagules produced in proportion to the number of lesions and then their contagious distribution.

To compare this new, plausible law with the former logistic law, I chose a value of unity for k, the parameter of the negative binomial distribution. Then (5) becomes

$$dN/dt = rN(K-N)/K, \tag{1}$$

which is the same equation as the original logistic law derived from the concepts of propagules in proportion to diseased plants and their random distribution.

After untangling the conceptions of dispersal from the logistic law, and replacing the terms for production and spread of propagules, we see that the two replacements counteract one another, returning us to the differential equation (1) that Verhulst wrote in the nineteenth century and the linear relation between logits and time that van der Plank (1963) found in many epidemics of contagious plant diseases. This is probably why many contagious diseases whose dispersal cannot be conceived as the random distribution of propagules produced in proportion to the number of diseased hosts nevertheless increase as if they could. Perhaps we should not be surprised that two misconceptions about dispersal have counteracted one another in the 'black box' of a highly abstract model. Simulators, which are less abstract, reveal the spores of actual pathogens and crops within the black box.

2. Simulation

Many of the mechanisms that make an epidemic grow can be incorporated into the computer program of a simulator. The consequent realism of simulators led Bourke (1970) to call them 'full-blooded models'.

A pecularity of the pathogens of plants is the predominance of fungi: the 1965 *Encyclopaedia Britannica* devotes fully half the pages for plant diseases to diseases caused by fungi, relegating all the others, those caused by viruses, bacteria, nematodes and even other flowering plants, to the remaining half. The distinct and visible stages of these fungal pathogens, the cycle of lesions, sporophores, spores, dispersal, invasion, incubation and then more lesions, look like the flow chart of a computer program and whet the appetite for composing a simulator with each stage a step in the master DO loop that is one cycle in the life of a fungus on its host.

Beginning in 1968 with a simulator of the steps in the life of the infamous fungus that caused the Irish famine during the nineteenth century, a host of simulators have been composed (Waggoner 1968; Waggoner & Horsfall 1969; Waggoner *et al.* 1972; Kranz *et al.* 1973; Shrum 1975; Zadoks 1971). The critical question here is 'How well has dispersal been treated in the simulators?'

Although the first simulators, like the logistic equation, ignored distance and direction, the stage of dispersal had to be completed in the life cycle and flow chart, and this evoked some conceptions of the mechanisms of dispersal. For example, the simulator of a fungal blight of corn calculated the take-off of spores as a function of windspeed and stress, flight appeared as a simple function of the windspeed that would blow some spores outside both the field and consideration, and landing was set proportional to the leaf area (Waggoner *et al.* 1972). Thus some rational effects of wind and foliage upon dispersal were incorporated into this early simulator, but the dimensions of the field remained hidden.

Unlike the early simulations that moved in time but not in space, an epidemic is played on the chessboard of a field with dimensions that the fungus must travel to succeed. Characteristically an infection in a field causes a few infections near itself, making a steep gradient from, say, a third of the leaves diseased at 1 m from the initial infection to 1% or less at a distance of 5 m. Later, new foci of infection appear, infection becomes general and the gradients of disease with distance across the field become indistinct (Waggoner 1952).

In an all-purpose simulator of fungal disease, Shrum (1975) recognized the expanse of a field by conceiving a chessboard divided into cells. Spores from one cell spread over other cells in

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

a widening Gaussian plume along the wind. No calculations of spread over the chessboard were, however, published.

In still another simulator of foci and epidemics on the chessboard of a field, Kampmeijer & Zadoks (1977) conceived the spores from each source cell to be distributed over the field in a two-dimensional Gaussian fashion, with the number at each receiving cell depending upon the distance between the source and receiver and also upon the specified variance of the Gaussian distribution. There was dispersal over the chessboard but no plumes following the wind and no increased scatter with changing turbulence. Although the simulator produced spreading stains of disease on plans of the imaginary field and provided a foundation for discussing different arrangements of resistant and susceptible hosts, it has the peculiarities of never producing new foci of infection nor making the gradients of disease across the chessboard less distinct as infection grows more general.

The power of computers invited more realism in calculating dispersal and even incorporating the link of dispersal into simulation of the entire life cycle of the pathogen. Unfortunately the removal of the limit on calculation mainly revealed that our knowledge of dispersal was a weak link. The answer to the question with which I began is 'So far, dispersal has not been well treated in simulators'.

3. The three parts of dispersal

The mechanism of dispersal can be illustrated by the classic pathogen of the downy mildew of potato, which causes late blight. Its dispersal begins with the take-off of lemon-like sporangia about 30 μm long from the branches of tree-like sporophores that stand about 150 μm tall in groves upon the lesions that are blackening and destroying the leaves. Then the airborne sporangia fly by simultaneously settling at 1–2 cm s^{-1}; being carried up, down or forward as fast as 1000 cm s^{-1}; and being separated from their fellows in the chaos of the air tumbling above the crop and swirling through it. Dispersal ends for one of the sporangia when it fails to follow the streamline of the air in which it is travelling and instead lands because it either strikes or settles upon some object. The landing can produce a new lesion. These are the three parts of dispersal: takeoff, flight, and landing.

(*a*) *Take-off*

Since plants neither cough nor sneeze, pathogens of plants must take off without help from their host. Some pathogens – notably viruses, mycoplasmas and bacteria – are carried aloft by insects, but they are called insect-borne and are not our subject. A virus of tobacco was carried aloft on a raft, a fragment of its host that was blown away (Smith 1937). However, exemplary plant pathogens, the fungi, can get their propagules into the air without insect or raft. Some pathogenic spores are shot or flung into the air, others are blown away.

The scabs on apples are caused by an ascomycete, a fungus whose sexual stage in dead leaves on the orchard floor produces spores in elongated sacs clustered within a microscopic cup. When the warmth of spring has arrived and rain falls, the spores are squirted from the sacs into the air, exemplifying the firing of the sexual spores of ascomycetes and basidiomycetes, which cause scabs, rusts and powdery mildews.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

(i) *Asexual spores*

These are the work horses that carry epidemics through crops. Sexual spores from the orchard floor renew apple scab, but asexual spores from scab on leaves cause most of the lesions seen on foliage and fruit by late summer. Whereas the sexual spores of *Peronospora tabacina* are hard to find, its abundant asexual spores carry epidemics of tobacco blue mould across whole continents. Asexual spores have varied ways of taking off.

If the little, tree-like sporophores of *Peronospora tabacina* growing 100–200 μm above a tobacco leaf are watched beneath a microscope, one sees them twist and turn as they dry, flinging into the air the asexual spores or sporangia that transmit blue mould (Pinckard 1942). When aerial spore concentrations observed above tobacco fields were plotted against relative humidity, the maximum concentration was reached when the humidity had fallen to about 60%, although only a tenth of that concentration was reached when the humidity had fallen to about 77% (Aylor & Taylor 1983).

A light wind alone carries other asexual spores aloft. The asexual spores or conidia that spread powdery mildew are grown in chains, and after 16 h the connection between the spores is too weak to survive a shake or even support the weight of spores (Bainbridge & Legg 1976; Aylor 1977). Thus a breeze of only about 1 m s^{-1} causes the main release of the conidia of this mildew of cereal (Hammett & Manners 1973).

(ii) *Strongly held spores*

Pathogenic spores that are strongly anchored but still take off are exemplified by the spores of *Helminthosporium maydis* that winged southern corn leaf blight from Florida across the American Corn Belt in its first season as a common disease. They seem nearly inaccessible to the wind because they are anchored to sporophores, the sporophores are within the boundary layer of air creeping over diseased leaves, and most of the diseased leaves are sheltered within the canopy. Paradoxically, many spores take off in an average wind of only 1 m s^{-1} (Aylor & Lukens 1974). The paradox was resolved by measuring how strongly the spores are attached, whether the boundary layer of air shields the spores from gusts, and whether gusts penetrate the windbreak of the canopy.

The strength of attachment of spores to sporophores was measured by first blowing them and then centrifuging them. When spore-laden leaves were held in front of a fan, no spores were removed until the windspeed rose to a steady 6 m s^{-1}. All the spores that could be removed were removed in a narrow range of windspeed around 10 m s^{-1}. In a centrifuge, no spores were removed at a force less than 700 times their weight, and all the spores that could be removed were removed in a narrow range of force around 2000. Calculation showed that the force required to break the attachment was much the same by either wind or centrifuge, that the attachments were uniform and strong, and that a fast wind directly on the spore was required to cause the leaf bound spore to take off (Aylor 1975*a*).

Near a leaf where the spore is held, viscosity changes the chaos of the wind above to a laminar layer of air creeping along, thin at the windward edge and thick at the leeward edge of the leaf. Although the boundary layer of air that the average wind produces explains the removal of most of the spores from the leading edge of a leaf in front of a fan steadily blowing at 5 m s^{-1} (Waggoner 1973), it fails to explain how an average wind of only 1 m s^{-1} can remove spores all over the leaves in a real field of maize.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

To make matters more absurd, centrifugation showed that the force of a 5 m s^{-1} wind directly on a spore held within the boundary layer is required to remove it, and an average gale of fully 25 m s^{-1} is required to propel the boundary layer at the requisite 5 m s^{-1} all over the leaf. The resolution of the paradox lies in 'average'. Like hats, spores are carried away by gusts, not averages.

When a fluid over a surface is started suddenly, the velocity near the surface at the instant of starting is equal to the velocity of the air well above. Although the viscosity of the air against the leaf begins to form a boundary layer that grows outward, there is a moment when the air next to the leaf and around the spore gusts forward.

To visualize or observe the rapid change in wind velocity over a leaf, Aylor & Parlange (1975) built a flat-plate anemometer, which was nothing more than a film painted with liquid crystals that revealed their temperature and thus the movement of eddies over the plate. When the plate was held in an average wind of 1.5 m s^{-1} in the field, eddies moved over it, dappling its surface as the liquid crystals cooled and changed colour. Thus there were transition zones with brief times when the wind at the surface was as fast as in the air above swept over the plate, and as the eddies and their transition zones swept over the film, spores were blown from sporophores growing on paper attached to the plate.

Although *Helminthosporium* spores are anchored to their sporophores, no boundary layer around the leaf shelters them from eddies and gusts. To resolve the paradox of spores that require the force of a 5 m s^{-1} wind to separate them from their sporophores but nevertheless take off in a average wind of 1 or 2 m s^{-1} requires only one more step: observing a few gusts to 5 m s^{-1} in an average wind of only 1 m s^{-1}.

Gusts or eddies were measured in a field of maize with a three-dimensional anemometer that responded in only 0.02 s (Shaw *et al.* 1979). Although the average of 360000 observations of wind in 1 h was about 1 m s^{-1}, speeds of 5 m s^{-1} were as frequent as 1 in 10000 and even speeds of 7 m s^{-1} were found among the 360000 observations. Further, individual gusts penetrating the vegetation were not diminished as rapidly with depth as the mean wind itself. This penetration of gusts into the canopy of maize plus the frequency of gusts faster than 5 m s^{-1} explain how spores anchored to sporophores rising only 100–200 μm from leaves sheltered within a crop can take off in a fairly slow average wind and be ready for flight.

(*b*) *Flight*

Because plants neither walk nor run, their epidemics would spread slowly indeed if their propagules did not fly. They fly to nearby plants in the same field or neighbourhood and also to distant plants in another neighbourhood or even state.

(i) *Nearby*

The pattern of disease on a field is the footprint of the cloud of propagules that has flown over, and the sharp decrease in concentration with distance that is reflected in the footprint is striking. Around small sources of the late blight fungus, the disease and presumably the concentration of flying spores decreased by 2–3% with each 1% increase in distance. Consequently the concentration of spores at a distance of 10 plants from the source of inoculum will be only one hundredth of that at the plant next to the source (Gregory 1968).

Do the spores simply fall out of the air? Although the spores of plant pathogens are larger than the viruses and bacteria that infect animals, still their spores have dimensions and settling velocities near those of the cloud and mist droplets that remain suspended in the air. When

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Stepanov (1935) released three species of spores with settling velocities differing tenfold from 0.2 to 2 cm s^{-1}, the decrease in concentration with distance was no faster for the largest than for the smallest species of spore. The falling or settling of the spores of plant pathogens scarcely affect their concentration as they fly further from the source.

Only the usual processes of diffusion and depletion decrease the concentration of the propagules as they fly along, and we are surprised because the decrease is dramatized by its footprint of disease. In Gregory's (1945) adaptation of O. G. Sutton's equation for turbulent diffusion, he derived the mean number of spores at a distance X from a point source of propagules. The mean concentration near the ground decreases inversely as X raised to the power $(1+\frac{1}{2}m)$. Because m is Sutton's parameter that is bounded by 1.5 and 2, the power of X is 1.75–2, and diffusion alone decreases the concentration by about 2% for each 1% increase in distance.

In addition, spores flying among the leaves of a crop are depleted or scrubbed from the air by the plants. When a cloud of pollen grains moved from a sugar-beet field, making a model of a spore cloud, the concentration near the ground was much higher than the concentration at a height of only 2 m (Hirst & Stedman 1971). The cloud then moved into a crop of wheat, and within 20 m the concentration near the ground had been scrubbed to much less than the concentration above the crop. When Bainbridge & Stedman (1979) released spores at the top of a barley crop, they found that scrubbing and diffusion within the crop made the vertical gradient steeper within the crop than diffusion made the gradient above.

(ii) *Distant*

At this point, where the discussion passes from short to long flights, Gregory's (1982) paradox is pertinent: a precipitous decrease in spore concentration near the source should not be extrapolated to a prediction of less disease afar. Gregory's (1945) equations for the diffusion and settling of spores show that the same turbulence that decreases the concentration nearby can cause more spores to escape and thus increase the concentration of spores causing disease at a great distance.

The turbulence that steepens the decrease in concentration near the source was seen helping the blue mould pathogen to escape from a tobacco field. Aylor & Taylor (1983) estimated the vertical diffusivity of the air from the wind profile, i.e. the increase in windspeed with height, and multiplied the diffusivity by the vertical gradient in spore concentrations. The product was the vertical flux or escape of spores from the field.

When turbulence increased as the wind at the top of the crop rose from 1 to 2 m s^{-1} and then to 3 m s^{-1}, the escape of spores from the crop rose from 3 to 5% and then to 7% of those that took off from lesions within the crop. Although more than 90% never left the field, the rate of escape from a severely diseased hectare during a 2 h take-off would be about 10^9 spores, and turbulence helps more escape to cause trouble downwind.

Distant flight has a different quality from the flight within a field. When after a few tens of kilometres the top of an expanding puff of spores reaches the cap of an inversion or the tropopause, the puff no longer expands upward. Then three-dimensional dilution is replaced by two-dimensional dilution, or if the source is large, dilution may practically stop. In and near the crop producing the propagules, the highest concentration is in the crop, and the spores diffuse upwards; but as the flight continues, the highest concentration is aloft, and spores diffuse downwards (Gregory 1973).

Dramatically, the converging air that makes convective storms can draw in large insects

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

that a pilot can tract (Rainey 1958). Although microscopic propagules of plant pathogens are neither visible nor able to maintain a compact swarm, they too are collected and carried in convective clouds (Heise & Heise 1948). The first gusts of a thunderstorm free some spores to be swept up (Rich & Waggoner 1962). In India, tropical storms form over a persistent source of rust spores and then carry the spores over central India, where they are deposited by rain (Nagarajan *et al.* 1976). Rain, of course, washes spores from a deep column of air and deposits more than would settle without rain (Gregory 1973). Thus a distant flight in a convective storm is more like a shipment by air freight than like the diffusion along a gradient of steadily declining concentrations.

The features of distant flight are exemplified by a cross section of the atmosphere from England to the Skagerrak (Hirst *et al.* 1967). The high concentrations, their modest dilution with distance from the large source of Britain, and the maximum concentration at fully 1000 m over the sea are all evident. Even increases in concentration with distance are seen where the sampler flew into air that had been over the land during the diurnal maximum of take-off before moving out to sea. Despite the small proportion of the spores that escape from a field and their millionfold dilution when distance only increases a thousandfold, a few dangerous fugitives take very long flights.

(iii) *Survival*

All the words about the aerial dispersal of plant pathogens are wasted if the propagules are dead on arrival. Because take-off and landing are quick, and flight can be a pilgrimage through a medium that dries and heats the naked microbe without shielding it from ultraviolet rays, survival is discussed as part of flight. An exemplary propagule is one of the fragile and hyaline downy mildews whose epidemics depend upon moist and temperate weather. Thus during the inhospitable day, fully 85–99 % of the spores of the late blight of potatoes fly, but only 60–77 % of the infections occur because fewer survive the desert of the day than the oasis of the night (Bashi *et al.* 1982).

When Bashi & Aylor (1983) exposed the spores of the downy mildew of tobacco in the laboratory to heat and cold and low humidities, they were surprised to find that the presumedly fragile spores survived for days at most humidities if they were cooler than 30 °C. Exposed in the sun outdoors, however, the spores survived for only 3 or 4 h. The hazards for a spore on a distant flight to another region are far greater than one on a sheltered flight to a nearby plant.

This echoes Gislén's (1948) summing up: 'While the lower cloudy air strata . . . form a suitable medium for the transport of micro-organisms, the higher layers are very inhospitable to them, not so much because of the low temperature, drought and barometric pressure as because of destructive radiation'.

Some fugitives, nevertheless, do survive ultraviolet. In sunny Kansas, spores of plant pathogens collected from the air were alive (Kramer & Pady 1968). The black fungus *Aspergillus niger* survives 300 times as long as the golden bacterium *Staphylococcus aureus* in ultraviolet radiation (Buttolph 1955). The ability of spores of plant pathogens to survive the ravages of ultraviolet, an ability that I suggest is modest but greater than that of typical propagules of animal disease, may be the reason that plant pathologists dwell so much on aerial dispersal.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

(*c*) *Landing*

Because plants neither eat nor drink, their pathogens must land upon them. An airborne pathogen may settle gently upon widespread foliage or be blown against some part of the host. Whether the propagule lands by settling or lands by impaction depends upon factors incorporated in the Stokes number, which increases with the settling velocity of the organisms and the speed of the wind, and decreases with the diameter of the host. The bigger the spore, the faster the wind, and the slimmer the host, the more likely the landing will be impaction. Although a very small propagule would have to reach its host by Brownian movement and a very large one would quickly fall, the typical spore of a pathogenic fungus with diameter about 10 μm can land by either settling or impaction, depending on the windspeed. Up to 2% of such spores approaching a common leaf at 1–5 m s^{-1} would strike the leaf and land (Chamberlain 1975).

When spores dispersed among the narrow leaves of barley, settling was the prevalent means of landing (Bainbridge & Stedman 1979), and 20 μm pollen mostly landed on maize leaves by settling (Aylor 1975*b*).

Although impaction was less that settling, it did occur. On a stalk of maize in the open and on the top of leaves of barley and maize in fields, impaction was as expected from the Stokes number. In the slow-moving air within a crop, however, the efficiency of collection was surprisingly great. Aylor *et al.* (1981) offered an explanation for these surprisingly great collections: the spores were not collected from the slow average wind within the crop with its low Stokes number but instead were driven by the occasional gusts that pass downward into the crop, carrying spores from the air above.

If viruses or bacteria somehow became airborne, most would be carried around the leaves and stems of plants, leaving them safely behind. The typical spore of about 10 μm diameter, however, is dangerous, sometimes settling on a horizontal leaf, sometimes drifting deeply within a crop on the streamlines around the leaves, and sometimes striking the susceptible plant in a quick gust.

4. Simulation, again

At the end I return to the goal of a better simulator of aerial dispersal of plant pathogens. The improvement requires the representation, within the bounds of practical calculation, of the mechanisms of take-off, flight and landing. Abstractly and simply, they can be represented in a single equation that makes the CATCH of spores equal to the TAKEOFF multiplied by a dilution FLIGHT and a LANDING velocity:

$$\underset{\text{spores s}^{-1}\text{ m}^{-2}}{\text{CATCH}} = \underset{\text{spores s}^{-1}}{\text{TAKEOFF}} \times \underset{\text{s m}^{-3}}{\text{FLIGHT}} \times \underset{\text{m s}^{-1}}{\text{LANDING}}. \qquad (6)$$

Equation (6) provides a rate that must be integrated over, say, a week to calculate the CATCH of spores during the week on a square metre of crop.

The easiest integration is made by estimating the number of spores that TAKEOFF during a week and then multiplying them by an average dilution or FLIGHT rate and by an average LANDING rate during the week. The necessity of taking the easy way is removed by modern computing machinery. This is fortunate because during the week take-off starts and stops, direction and dilution of flight change, and the nature of landing alters as the weather and

hours pass; and the integration of their product can rarely be the product of their means. In practice (6) becomes a flow chart for a computer program that integrates CATCH numerically as the parameters on the right-hand side of the equation vary independently with changes in the unsteady environment and the number of diseased sources.

The differences in the relations of the three parameters to the weather is so great among species of fungi that Gregory (1973) differentiated 'fine-weather' from 'damp-weather' spora. The diurnal cycles of different species of spores in the air are so disparate that they are classified into distinct types. The parameter for TAKEOFF will increase in the morning if drying causes spores to be flung from sporophores. FLIGHT will dilute the spores more and kill more if they fly during the day rather than the night. LANDING will increase impaction if the spores take off in gusts rather than being shot aloft during drizzles. Integration must be species by species as well as second by second.

This complexity does not prevent calculation. Aylor (1978) simulated the advection of Gaussian puffs and plumes that were depleted by deposition. The simulator incorporated the effects of gusts upon take-off and landing as well as upon flight. The simulations showed that when spores took off only in gusts, the number landing at, say, 20 m was about the same whether the wind was 1 or 3 m s^{-1}, but if the spores took off steadily, fewer would reach 20 m in slow winds because they would settle near the source. In another simulation, Aylor (1982) essentially integrated (6) for only those times when the wind was fast enough to carry the spores of powdery mildew into the air and thereby explained the surprisingly rapid depletion of airborne spores within a barley crop.

Although the first simulations of the life cycles of plant pathogens that Bourke (1970) would have styled full-blooded may have inspired investigations of dispersal, they incorporated little of the mechanisms that are now known. The stage seems set for an advance in simulation.

5. The uses

Forecasting the progress of an epidemic from week to week makes good use of the knowledge of the success of the pathogen during its most vulnerable stage, dispersal. Forecasts have been made for many decades, and this use of knowledge of dispersal can reasonably be expected to grow as the cost of fungicides or the avoiding of chemical residues makes the timing or even omission of sprays worthwhile.

Knowing the quantity of dispersal can determine the importance of local sanitation. The steep gradients of disease around cull piles of potatoes, or neglected seedbeds for tobacco, long ago put farmers to work cleaning their own farms. On the other hand, farmers of a region have no reason for sparing an effective fungicide that selects resistant races of the blue mould of tobacco if resistant races can fly in from other regions (Aylor *et al.* 1983).

Because many exposed plant pathogens are susceptible to drying and radiation, their control by managing the microclimate sometimes seems logical. Overlooking the fact that microclimate affects dispersal can then bring disappointment. For decades the sides of shade tents have been raised to dry the tobacco growing inside and thus slow an epidemic of blue mould. A study of dispersal revealed that, unfortunately, raising the sides of a tent scarcely speeded drying in the shade of the tent – but the increased ventilation dispersed more spores (Aylor & Taylor 1982).

Understanding dispersal can aid science as well as practical affairs. The mixtures of resistant with susceptible varieties called multilines reduce an epidemic more than the proportion of

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

resistant hosts, and an explanation may lie in the landing of propagules on the resistant varieties and the consequent sparing of susceptible ones (Kampmeijer & Zadoks 1977). Since a barrier of only 2 m of a resistant wheat can halve the disease in a small plot, dispersal of pathogens into a typical test plot for either fungicides or resistant varieties can affect the outcome of the test, making an understanding of dispersal critical in interpreting experiments (Jenkyn & Bainbridge 1974). Finally, satisfying a curiosity about the flight of the fungi and gratifying a sense of order by a flow chart are respectable uses of a study of the dispersal of plant pathogens.

References

Anscombe, F. J. 1950 *Biometrika* **37**, 358–382.
Aylor, D. E. 1975*a* *Pl. Physiol.* **55**, 99–101.
Aylor, D. E. 1975*b* *J. appl. Met.* **14**, 52–57.
Aylor, D. E. 1977 *Trans. Br. mycol. Soc.* **69**, 160–161.
Aylor, D. E. 1978 In *Plant disease* (Ed. J. G. Horsfall & E. C. Cowling), vol. 2, pp. 159–180. New York: Academic Press.
Aylor, D. E. 1982 *Agric. Met.* **26**, 215–219.
Aylor, D. E. & Lukens, R. J. 1974 *Phytopathology* **64**, 1136–1138.
Aylor, D. E., McCartney, H. A. & Bainbridge, A. 1981 *J. appl. Met.* **20**, 1212–1221.
Aylor, D. E. & Parlange, J.-Y. 1975 *Pl. Physiol.* **56**, 97–99.
Aylor, D. E. & Taylor, G. S. 1982 *Proc. natn. Acad. Sci. U.S.A.* **79**, 697–700.
Aylor, D. E. & Taylor, G. S. 1983 *Phytopathology* **73**, 525–529.
Aylor, D. E., Taylor, G. S. & Raynor, G. S. 1983 *Agric. Met.* **27**, 217–232.
Bainbridge, A. & Legg, B. J. 1976 *Trans. Br. mycol. Soc.* **66**, 495–498.
Bainbridge, A. & Stedman, O. J. 1979 *Ann. appl. Biol.* **91**, 187–198.
Bashi, E. & Aylor, D. E. 1983 Submitted to *Phytopathology*.
Bashi, E., Ben-Joseph, Y. & Rotem, J. 1982 *Phytopathology* **72**, 1043–1047.
Bourke, P. M. A. 1970 *A. Rev. Phytopath.* **8**, 345–370.
Buttolph, L. J. 1955 In *Radiation biology* (ed. A. Hollaender), vol. 2, pp. 41–93. New York: McGraw-Hill.
Chamberlain, A. C. 1975 In *Vegetation and the atmosphere* (ed. J. L. Monteith), vol. 1, pp. 155–204. London: Academic Press.
Gislén, T. 1948 *Biol. Rev.* **23**, 109–126.
Gregory, P. H. 1945 *Trans. Br. mycol. Soc.* **28**, 26–72.
Gregory, P. H. 1968 *A. Rev. Phytopath.* **6**, 189–212.
Gregory, P. H. 1973 *The microbiology of the atmosphere*, 2nd edn. (377 pages.) New York; John Wiley.
Gregory, P. H. 1982 In *Advancing frontiers of mycology and plant pathology* (ed. K. S. Bilgrami, R. S. Misra & P. C. Misra), pp. 107–117. Delhi: Today & Tomorrow Printers.
Hammett, K. R. W. & Manners, J. G. 1973 *Trans. Br. mycol. Soc.* **61**, 121–133.
Heise, H. A. & Heise, E. R. 1948 *J. Allergy* **19**, 403–407.
Hirst, J. M. & Stedman, O. J. 1971 In *Ecology of leaf surface microorganisms* (ed. T. F. Preece & C. H. Dickinson), pp. 229–237. London: Academic Press.
Hirst, J. M., Stedman, O. J. & Hurst, G. W. 1967 *J. gen. microbiol.* **48**, 357–377.
Jenkyn, J. F. & Bainbridge, A. 1974 *Ann. appl. Biol.* **76**, 269–279.
Kampmeijer, P. & Zadoks, J. C. 1977 *EPIMUL, a simulator of foci and epidemics in mixtures of resistant and susceptible plants, mosaics and multilines.* (50 pages.) Wageningen: Pudoc.
Kramer, C. L. & Pady, S. M. 1968 *Mycologia* **60**, 448–449.
Kranz, J., Mogk, M. & Stumpf, A. 1973 *Z. pflKrankh.* **80**, 181–187.
Nagarajan, S., Singh, H., Joshi, L. M. & Saari, E. E. 1976 *Phytopathology* **66**, 198–203.
Pinckard, J. A. 1942 *Phytopathology* **32**, 505–511.
Rainey, R. C. 1958 *Q. Jl. met. Soc.* **84**, 334–354.
Rich, S. & Waggoner, P. E. 1962 *Science, Wash.* **137**, 962–965.
Shaw, R. H., Ward, D. P. & Aylor, D. E. 1979 *J. appl. Met.* **18**, 167–171.
Shrum, R. 1975 *Pa. State Univ. Prog. Rep.* no. 347. (81 pages.)
Smith, K. M. 1937 *Nature, Lond.* **225**, 199.
Stepanov, K. M. 1935 *Bull. Plant. Prot. Leningrad. Ser.* 2 (*Phytopathology*), no. 8, pp. 1–68.
van der Plank, J. E. 1963 *Plant diseases: epidemics and control.* (349 pages.) New York: Academic Press.
Waggoner, P. E. 1952 *Phytopathology* **42**, 323–328.
Waggoner, P. E. 1968 In *Biometeorology* (ed. W. P. Lowry), pp. 45–66. Corvallis: Oregon State University Press.
Waggoner, P. E. 1973 *Phytopathology* **63**, 1252–1255.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Waggoner, P. E. & Horsfall, J. G. 1969 Bulletin no. 698, Conn. Agr. Exp. Station, New Haven. (80 pages.)
Waggoner, P. E., Horsfall, J. G. & Lukens, R. J. 1972 Bulletin no. 729, Conn. Agr. Exp. Station, New Haven. (84 pages.)
Waggoner, P. E. & Rich, S. 1981 *Proc. natn. Acad. Sci. U.S.A.* **78**, 3292–3295.
Zadoks, J. C. 1971 *Phytopathology* **61**, 600–610.

Discussion

J. S. A. Green (*Atmospheric Physics, Imperial College, London, U.K.*). Dr Smith has reviewed the classical theory of turbulent diffusion, which was developed to describe the transfer of bulk properties, particularly heat and momentum. I suspect that the problem of the spread of disease is different. The most significant aspect of disease is the ability of a microbe to increase in number rapidly by breeding, once established on the host. Thus the continued reinfection described tolerably well by the most probable events reviewed by Dr Smith are not so important as the freak event that establishes a new colony at some hitherto disease-free place.

That the statistics of rare events is not the same as the statistics of probable events is well known, and the biologist is already aware of the existence of such statistics from the evidence of moths detecting pheromone plumes very far downwind of a source.

The crucial parameter that the meteorologist needs to know is the degree of rarity of the disease-propagating event. This might be a biological assessment of the number of microbes needed to initiate a disease in an organism, and the number of diseased organisms needed to initiate an epidemic. It was gratifying that some of these aspects, for instance the rare shock needed to launch spores, were touched upon in Professor Waggoner's contribution.

P. E. Waggoner. Our familiarity with the diffusion and the dilution of concentrations of gases may prevent our remembering that in epidemiology the simple success or failure of a single take-off, flight and landing may initiate an epidemic – or not.

Put in other terms, 10 h of a 1 m s^{-1} wind might carry away as much vapour as 1 h of 10 m s^{-1}, but 1 m s^{-1} might fail to release any spores, no matter how long it blew. In the same vein, a great dilution could merely lower the concentration of a gas while making it probable that none of the discrete spores in an initial puff would pass over a small field.

Finally, the landing of many propagules increases the probability of infection just as having many sons increases the chance that one's surname will persist. And a half a propagule won't succeed. Nevertheless, a single propagule may establish a beachhead, and then, multiplying in a hospitable environment, produce an epidemic.

F. B. Smith. If my limited understanding of how secondary airborne infections are established is correct, then in the great majority of occasions (*a*) the infection *does* occur where one might reasonably expect it to in terms of the overall windfield, and (*b*) potential hosts can usually withstand small 'dosages' and are overcome only when their natural body defences are swamped.

Both these factors suggest that the 'freak event' transport of viruses referred to by Dr Green may not be too important as long as they are sufficiently short-lived. In the emergency of an outbreak it is prudent to concentrate most of one's attention on the areas at maximum risk while, of course, not being entirely blind to the possibilities of infection in other surrounding areas. Deciding where these areas of maximum risk are is difficult enough, especially in regions of marked topography or near coastlines when sea-breezes are possible. These are the aspects covered in my paper.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES