Dynamics of pathogen population

Changes over time-scales longer than either crop or pathogen lifetime

Possible dynamical patterns

It is helpful to distinguish internal and external forces acting on a population. This is largely an artificial distinction but makes it possible to think about patterns of change in a structured way. If mathematical models of how populations change over time are used, internal forces are those that change only because of processes described by the model. External forces are those that change in particular ways over time or at specific times, in ways for which the model offers no explanation.

The simplest type of model includes only the pathogen population. In a constant host population and a steady environment, if time lags are not considered, this kind of model will always show the pathogen population tending to return to an equilibrium level from arbitrary starting conditions. If environmental disturbances alter the position of the equilibrium, the population will tend to track these disturbances.

However, the state of the pathogen population at some past times may influence how it now grows: this influence may arise from the age structure, or from changes induced in the host. In this case, other, more complicated dynamical patterns can arise. Even in a steady environment, a pathogen population may tend to oscillate (fluctuate) regularly between two or more distinct levels, or to oscillate irregularly in a quasiperiodic (irregular periodicity) or chaotic pattern. A quasi-periodic pattern is one in which there are two fundamental frequencies of oscillation in population numbers present but the ratio of the two frequencies is an irrational number, so no matter how long a time is considered, no multiple of one frequency is ever equal to

a multiple of the other. A chaotic pattern is one in which very small changes in population numbers cause very large changes in the population at later times and in which, even in a steady environment, no regularly repeating sequence of population numbers ever appears.

In general, populations are more likely to show complicated behaviour if they both (a) change very fast (on their natural time-scale as discussed in section "time scales") and (b) are strongly influenced by their state or size a considerable time before. The main ways in which the past state of a population is likely to influence its present growing conditions are through interactions with its host, through interactions with other populations which compete with it or consume it by parasitism or predation, or through strong natural selection operating differently on different sizes or types of population.

A model including the host population must have at least two variables, one each to represent the host and the pathogen populations. If the pathogen has a free-living stage, or the host can become immune, more complex models still may be appropriate. Any model with more than two variables considered, or which includes time lags, *may* show complicated dynamics. Simple models of two interacting populations in continuous time, without time lags, are often simple to construct and may capture the essentials of host-pathogen interaction. Mathematically, however, they can *only* converge to an equilibrium or to steady repeating oscillations. This can be a trap, since apparently similar descriptions of a pathosystem may be capable of showing very different behaviour and it may be difficult to distinguish the models by comparison with the very short runs of data that are available.

Host-pathogen population linkages

In agricultural settings, the host population may be only indirectly linked to the pathogen population. In the most extreme case the same crop cultivar (or one with equivalent disease resistance) may be planted, regardless of the disease level. This may occur because there is no economic alternative, as with black Sigatoka and Sigatoka diseases of banana caused by *Mycosphaerella fijiensis* and *M. musicola*, where both marketing and breeding constraints restrict the available cultivars, or it may simply be that agronomic and market imperatives override consideration of disease resistance in choice of a cultivar, as with the growth of wheat susceptible to *M. graminicola* in the UK during the 1980s and 1990s. In other cases, planting areas of cultivars that suffer severe disease may decrease, as in the classic boom-bust cycles of gene deployment in cereals.

In some natural habitats, by contrast, the success of reproduction by the host is directly linked to that of the pathogen. For example, *Linum marginale* is a wild relative of flax native to Australia. Plants infected with *Melampsora lini* (flax rust) do not perennate (survive a winter or dry season and grow again in one or more seasons), whereas healthy plants may survive many years. Likewise, it has been shown that populations of *Silene alba* infected with *Microbotryum violacearum* smut were smaller than those which were uninfected. They also showed that this was at least in part because the populations were seed limited and seed output was reduced by infection with the smut.

To analyze long-term dynamics, it is necessary to study population changes over the entire pathogen life-cycle, including if they exist, seasons where a new host is unavailable for infection: often in the dry season(s) in tropical areas, typically winter in temperate countries. The simplest example to examine is an annual crop plant, affected by a disease that infects entire plants annually from an over-seasoning

source, then grows within plants and produces new propagules which will carry the disease forward to the next season. A well-known example would be *Sclerotinia sclerotiorum* infecting oilseed rape (canola) and a range of other crops. Wind-blown ascospores initially colonise fallen petals, producing a mycelium which is then capable of penetrating a leaf on which the petal may be lying. These leaf infections become systemic and the pathogen eventually produces cankers in the stem containing sclerotia which will survive in the soil until the following spring when sclerotia near the soil surface produce apothecia from which ascospores are discharged to infect the new season's crop. Thus, to understand population changes it is only necessary to consider the host and pathogen population at the time of flowering in each year. If the same host area is sown each year and conditions are similar on average each year, the pathogen population will tend to change by a constant factor each year. If only one individual existed in the first year, producing R_0 individuals in the following year, we would expect exponential growth, with R_0^2 in the following year and so on. Clearly, for the population to persist, R_0 must be more than 1. As stated earlier, an aim of management is to reduce R_0 as much as possible.

Oilseed rape is generally grown as part of a rotation. Infection will come from old sclerotia within the same field (since some sclerotia survive for several years) and from spores from more distant sources. The distant sources affect R_0 in a rather simple way since the ascospores are wind-blown and may be regarded as distributed more or less at random over a wide area. If the crop area is doubled, twice as many of these spores will land on susceptible tissue. The number of locally generated spores depends on the time since the last susceptible crop was grown. Since this depends on the particular rotation used on a farm, it may depend rather little on the area of crop grown regionally.

This also provides a way to think about the long-term dynamics of diseases that have a phase of rapid population growth during the crop season. The population at the start of the season was simply assumed to be a known quantity. However, the start of active host growth is simply the low point of the pathogen population cycle, while the end of that season is usually the high point. A pathogen population at a single time in the year can therefore be thought of as being in balance between an increase during the host's growing season and a decrease during the off-season. This is usually combined with a change from one phase to another, for example from the conidial stage to the sexual stage, or from mycelium to sclerotia. The chances that the increase would balance the decrease exactly over millennia (periods) are very small but, if the factor of decrease were greater than the factor of increase, the pathogen would become extinct. In fact, this would happen if the decrease outweighed the increase for more than a few years.

There are two possible ways to explain why pathogens do not become extinct. In the first place, it is unlikely that conditions over large areas would be exactly synchronised. Extinction would require simultaneous coincidences of poor conditions over the whole of the pathogen range. The time-scale for this may be so long that it very rarely occurs even over millions of years. An alternative explanation is that the potential increase during the cropping season is on average greater than the decrease in the off-season but that the population size must be limited during the growing season by some factor or factors that are density-dependent.

Parasitism and predation

In very many cases, as already mentioned, it appears that the growth rate of an epidemic within an annual crop slows long before crowding for space is plausible, even if it is assumed that the area visibly damaged by a disease is much smaller than the area from which nutrients are withdrawn and direct competition with another

pathogen individual can occur. This suggests the operation of a density-dependent factor early on. Perhaps the most obvious factor is that attack by a pathogen will stimulate various types of acquired resistance: hence the success rate of fungal spores on a new, juvenile host may be much greater than on a host already attacked (successfully or unsuccessfully) by other spores.

Off-season survival may also be density-dependent because of natural enemies. For example, sclerotia of *Sclerotinia* spp. are attacked by various hyperparasites, including both fungi and viruses. As *Sclerotinia* becomes commoner, so its hyperparasites will become more common and overwinter survival of the sclerotia will decrease. This means that natural regulation by such agents will tend to occur only when the pathogen is common.

Such density-dependent processes can lead to equilibria in which both pathogen and hyperparasite have an annual cycle in abundance, the pathogen peak populations being lower than they would otherwise be. If population growth rates are very rapid, the result could be massive fluctuations between years without simple repeating patterns: chaotic fluctuations. Chance effects would be likely to lead to extinction for the hyperparasite or for both hyperparasite and pathogen, unless the pathogen is sufficiently widespread (or its dispersal sufficiently restricted) that cycles in different parts of its range do not coincide.

Competition

Pathogens may compete simply by depleting the space or nutrients available in a host (exploitative competition), by some more active interaction such as the excretion of antibiotics (interference competition) or, indirectly, by triggering changes in their hosts, or supporting larger populations of hyperparasites than would otherwise exist.

The degree to which two species compete is determined by the similarity with which they use available resources. Two species that use resources identically will not be able to co-exist indefinitely in a stable environment. One will inevitably be very slightly more efficient and will grow more rapidly than the other, supplanting it slowly or rapidly. This is the competitive exclusion principle.

At first sight, this conclusion is incompatible with the regular occurrence, on the same plant organ, of many similar pathogens: for example, in the UK on wheat, it is easy to find leaves simultaneously infected with *Mycosphaerella graminicola*, *Didymella exitialis* and *Phaeosphaeria nodorum*. In fact, several of the assumptions underlying the competitive exclusion principle are violated: hosts vary dramatically in abundance and quality during the year, so that the environment for a plant pathogen is never stable; pathogens and hosts are spatially aggregated, so that the strength of competition between species is reduced relative to that between related individuals within a dense patch of disease and there may well be subtle differences in resource use by apparently similar pathogen individuals, related to temperature and wetness requirements for infection, preferred age of host tissue, etc. On the other hand, the apparent replacement on banana of *Mycosphaerella musicola* by the more pathogenic *Mycosphaerella fijiensis* (cause of sigatoka disease) in parts of the world where both have occurred is in accord with the theory.

It seems common for competition between plant pathogens to be very asymmetric. For example, *Phaeosphaeria nodorum* reduces the growth rate of *Blumeria graminis* populations on the same leaves but preferentially infects leaves infected with *B. graminis*.

An interesting case where competition has been revealed by agricultural change is the balance among the eyespot (straw-breaker foot rot) pathogens *Oculimacula*

yallundae and *O. acuformis* and the sharp eyespot pathogen *Rhizoctonia cerealis*. Treatment of north European wheat crops with demethylation-inhibiting fungicides (DMI) has had two effects. First, an increase in population density of *R. cerealis* correlates well in individual fields with a decrease in *Oculimacula* spp. and this is due to antagonism. Second, *O. acuformis* increases relative to *O. yallundae*. This is more likely to be because of differential sensitivity to fungicide treatment than competitive release because the absolute numbers of *O. acuformis* do not necessarily increase and *O. acuformis* appears to multiply later in the season than *O. yallundae*, so it may partly escape the effects of spring fungicides.

It also appears that populations of *Oculimacula* spp. are partly controlled by competition with true saprophytes. The evidence is that in fields in which the straw from preceding wheat crops was regularly incorporated, *Oculimacula* infection of subsequent crops was less than in fields in which the straw was burnt. The burning reduced the number of *Oculimacula* individuals surviving from the crop. However, it also reduced the amount of straw incorporated and so reduced the population of lignin- and cellulose-decomposing saprophytic fungi. In fields with straw incorporated, the straw on which the overseasoning *Oculimacula* survived was rapidly colonised by specialist decomposers and the *Oculimacula* was deprived of nutrients, so that fewer spores were produced than in the burnt fields.

Genetic Resistance or Susceptibility and co-evolution of host and pathogen

Obviously, host plants carrying race-specific (vertical) resistance do not allow a pathogen to become established in them, and thus no epidemic can develop. Host plants carrying partial (horizontal) resistance will probably become infected, but the rate at which the disease and the epidemic will develop depends on the level of resistance and the environmental conditions. Susceptible host plants lacking genes

for resistance against the pathogen provide the ideal substrate for establishment and development of new infections. Therefore, in the presence of a virulent pathogen and a favorable environment, susceptible hosts favor the development of disease epidemics.

The linkage between host and pathogen populations sets up a co-evolutionary race between pathogen and host, the host evolving towards resistance and the pathogen to virulence. In agriculture this can produce a boom (prosperous) and bust (broken) cycle of repeated release and failure of cultivars. In natural settings also, the consequences of this evolution may be far from a smooth progression. For example, in the *Linum* case, the rust can multiply dramatically within a season and therefore the genetic structure of the population may adapt very quickly to that of the host. The host, however, has a seed-bank. This will be well-stocked with the survivors of past years of severe disease, to which the pathogen may now be illadapted. This is likely to produce extremely complicated population dynamics but no models have yet been published.

In agricultural settings, the host population is linked to the pathogen population through farmers' and breeders' responses to disease. Much of the detail in the evolutionary and dynamical response of a pathogen population to the host composition depends on chance events. For example, in the mid 1980s barley cv. Triumph was widely grown in the UK, carrying effective resistance alleles to *Blumeria graminis*. After virulence against these alleles became common, almost half the population of *B. graminis* was represented by a single phenotype carrying at least three virulence alleles not needed on any variety then grown widely. This is almost certainly because the necessary mutations for virulence on cv. Triumph occurred in a single individual already carrying the extra virulences. The result was

an abundant pathogen population, composed of a few very common clones and many much rarer ones.

In populations of aerially dispersed pathogens, once a virulence allele is present at a moderate frequency its fate depends on how common the matching resistance is, and on the strength of selection against the virulence over the whole life-cycle of the pathogen. Many virulence alleles appear to carry moderately small selective disadvantages in the absence of the corresponding resistance, and will persist at moderate frequencies for many years. If the matching host resistance is reintroduced, or introduced into a new area, the population of the pathogen carrying the matching virulence allele is likely to become common faster than when the cultivar was first introduced, because the allele is already present in the population.

Under exceptional circumstances, the long-distance movement characteristic of aerially dispersed pathogens may make selection for virulence ineffective. For example, in the Canadian great plains wheat cv. Selkirk remained resistant to *Puccinia graminis* (cause of black stem rust) for many years. It contained a suite of at least six resistance alleles, to all of which individually virulence was present within North America. However, the rust did not overwinter in the area where Selkirk was grown, but migrated in each season from the south. The pathogen population probably did not evolve resistance because the precise combination of resistance alleles in Selkirk was never used in the hosts of the overwintering population.

With soil-borne pathogens of restricted mobility, the genetic structure of a population is likely to be much more fragmented, and rapid replacement of the whole genetic structure of the population is less likely. Use of single resistance genes against some soil-borne pathogens has been successful in some cases, and even

where the pathogen has evolved to overcome the resistance in one place this is followed by sporadic rather than rapid and universal spatial spread.

There is increasing evidence that populations of many trees are limited by the build-up of root pathogens in the soil around a mature tree. Most seedlings fall close to the parent tree and are killed by root damage from soil pathogens which have multiplied on the parent roots, without killing it because of its size and reserves. Only rare individual seedlings germinating far from the parent survive. This seems likely to produce a particularly fine-grained mosaic of host and pathogen co-adaptation, since selection on the pathogen around one parent tree will be long-continued, but surviving seedlings nearby are likely to select for different strains.

Spatial population structure

The spatial structure of populations can have a profound influence on population dynamics. It can also make the study of dynamics much harder, because the population studied may, in fact, be composed of several independent populations which are being unknowingly averaged, or the population may be part of a much larger system and be controlled by factors which cannot be observed within the area studied.

In some pathosystems, vectors or propagules can move long distances. Examples include rusts and powdery mildews of cereals or many whitefly- and aphid-borne viruses. In such cases it is not sensible to consider a crop area isolated unless it is a long distance from other such crops; often, this may mean many kilometres. The dynamics of the pathogen within a defined crop area will, therefore, be determined both by processes within the crop and by immigration and emigration of propagules from the surrounding crops. This can make experiments on control very difficult, because the small crop areas desirable to make it feasible to include many treatments

may not only not be independent but will have population dynamics very different from a larger, effectively closed, system treated in the same way. For example, spore transport by wind may be best modelled by an inverse power-law. One way to understand this is to visualise spores travelling in wind, dying or settling out only slowly, so that their trajectories spread out in proportion to distance. In this case the chance of a spore passing through a point would decrease inversely to distance. In practice, of course, many other factors reduce long-distance transport, but more realistic models of turbulent atmospheric dispersal do predict a power-law relationship between distance and probability of a spore reaching a given place.

The consequences of such a relationship can be quite surprising. If spores disperse approximately according to an inverse square law at intermediate scales (for example, falling to 1/41 m from the source, 1/92 m from the source and so on) and an experiment were conducted in a large uniform crop area, then a disc-shaped plot 10 m across would receive the overwhelming majority of its spores from outside. Even with dispersal according to an inverse 2.5 power, such a plot would receive almost equal contributions from inside and outside. In practice, most spores do not escape a canopy to take part in long-distance air movements, so the position is slightly less extreme than this; but plots of the order of 100 m^2 should not be assumed to be independent units.

Foliar pathogens and (non-waterborne) root pathogens have very different mobility. Splash-borne or airborne pathogens move on scales of metres or much more. Although infections are most likely on the immediate neighbours of an infected host, pathogen propagules may reach some distance and the approximation of random contact between healthy and infected individuals is useful, although it will overestimate the rate of disease progress.

In a soilborne disease, however, propagule movement may be very limited and infection rates will be very rapidly limited because most inoculum will reinfect an already infected host (this is an extreme form of the phenomenon discussed above). Thus, within a season, soil-borne disease typically has lower rates of increase than aerially dispersed disease. Furthermore, the movement is usually more by the host than the pathogen, putting a premium on the ability of the pathogen propagules to survive until a host root comes sufficiently close. The total population size of a pathogen like this will evidently have much smaller annual fluctuations than that of a typical foliar pathogen.

Because of the limited movement of soilborne pathogens, it is quite common for an area of study to include many effectively separate populations. In this case, it may be possible to model some effects of the averaging over populations by specifying that infection rates in the model should scale as some power of the population densities of host and pathogen. For example, if this power is more than 1 for the host population, then the infection rate of hosts by the pathogen is more efficient at high density than would be predicted from the performance at low density. An example of the application of this model is an analysis of data on the lettuce pathogen *Sclerotinia minor,* attacked by the hyperparasite *Sporodesmium sclerotivorum*. Experimets showed that it was necessary to include an assumption of heterogeneity, in this implicit form, in order to adequately fit these data. Without the assumption, the hyperparasite had to be assumed to be so efficient in order to fit the overall data, that sclerotia of the pathogen would have been eliminated from the soil; in fact, it survived for a long time at a low density.

Soil-inhabiting pathogens are notoriously patchy on very small scales. This is in part a function of the limited opportunity for smoothing out fluctuations in population density because of the difficulty of moving through soil and partly as a result of

heterogeneity in the soil itself. However, it is also possible that variability may be generated by population processes within the soil itself. It has been shown, in experiments with damping-off of radish (*Raphanus)* seedlings by *Rhizoctonia solani* in the presence of the antagonistic and non-pathogenic fungus *Trichoderma viride,* showed how the development of host resistance amplified very small initial variations in rates of attack and parasitism into very large final variations in pathogen population density. In the field, such patchiness could then persist for long periods, influencing both the vegetation and the location of pathogen populations.

If hosts exist in patches, it may be sensible to regard the patch as an individual in a population on a larger scale, the metapopulation. Then the host patches can be regarded as reproducing, becoming infected, dying and so forth. The metapopulation can be studied as an entity in its own right. Although the time-scales involved are long, processes at the metapopulation level are probably as important in determining the genetic structure and size of pathogen populations as process at the population level.

Dispersal and inverse power-law equation

It is assumed that dispersal obeys an inverse power-law with an exponent *n*, on intermediate scales. On very long scales, typical of the distance travelled by wind in a day or so, there is an exponential decline, with a scale parameter *l*. To simplify the calculations, it is also supposed that the wind is equally likely to blow from any direction; this assumption can be relaxed without changing the important conclusions from the argument. The density of spores moving a distance *r,* from a source with density ρ per unit area is then described by:

$$k(r,\theta)\alpha\, e^{-\frac{r}{l}}\frac{\rho}{(1+\frac{r}{a})}$$

Here a sets the short-distance scale of dispersal. It has units of distance and the larger it is, the further spores travel. Now the density of spores arriving from within a distance r_0 can be compared with the density of spores arriving from outside that distance. Each overall density can be obtained by the mathematical technique of integrating over all possible directions and all possible distances less than or greater than r_0. The result of this can be expressed analytically, but is very complex. The numbers in the text are obtained by substituting $r_0 = 10$; $a = 1$; $l = 100$ and $n = 2$ or 2.5; the qualitative results are very little affected by the value of l chosen, provided it is much larger than r_0.