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# Plant venereal diseases: insights from a messy metaphor

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## Summary

**Key words:** anther smut, pollinator transmission, sexually transmitted disease.

The concept of plant venereal disease is examined from definitional, operational and axiomatic viewpoints. The transmission of many plant pathogens occurs during the flowering phase and is effected either by pollinators or by wind dispersal of spores from inflorescences. Attraction of insects by pseudo-flowers or sugary secretions also serves to spread many diseases. Given the diversity of processes involved, a simple all-encompassing parallel with animal venereal diseases is not possible. Operationally establishing the routes of disease transmission, as well as quantifying the relative magnitudes of these different routes, remains critical for understanding disease dynamics and controlling spread in agricultural contexts. From an axiomatic viewpoint, sexually transmitted diseases are characterized by frequency-dependent transmission, transmission in the adult stage, and by virulence effects involving sterility rather than mortality. These characteristics serve to differentiate the dynamics and evolution of sexually transmitted diseases from that of other diseases and are features that are also shared by many pollinator-transmitted diseases. However, the majority of plant diseases that involve the reproductive structures show a rich biology that defies easy categorization. The experimental convenience of plants and their pathogens is likely to play an important role in understanding the evolution of disease traits, irrespective of what descriptive terms are applied to the natural history of the transmission process.

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## Introduction

Because disease biology has developed independently in the applied spheres of the medical and agricultural sciences, and because disease generates emotional responses that are transferred to technical issues, there are very many areas where the use of disease terminology differs or is transferred uncritically between fields. For example, the term 'virulence'

is used by the plant pathologist to mean the ability of a pathogen to infect its host, but for a zoologist and evolutionary biologist 'virulence' is a measure of the severity of disease symptoms caused by a pathogen. To an applied plant pathologist disease severity is often instead referred to as the 'aggressiveness' of the pathogen. Similarly, it has also been argued that the term 'epidemic' should be restricted to human epidemics, and that it is more appropriate to use 'epizootic' to

refer to a disease epidemic in animal populations. Fortunately, plant biologists, for once, have not been tempted by their own term (although 'epiphytotic' does come to mind).

The term 'venereal disease' has been used by myself and others to describe some pollinator-transmitted plant diseases, especially anther-smut disease which infects many members of the Caryophyllaceae (Alexander & Antonovics, 1995; Kaltz & Schmidt, 1995; Shykoff & Bucheli, 1995; Kaltz & Shykoff, 2001). We can, understandably, be accused of simply throwing out a cheap metaphor that serves to hook the attention of the large category of 'plants-don't-count' biologists. Indeed, when I first started to work on pollinator-transmitted diseases, this was precisely the aim of seminar titles such as 'Birds, Bees and STDs'. The concept of sexually transmitted diseases in plants has also been recently discussed by Wennstrom & Ericson (2003). They make the important point that 'reproductive diseases' in plants are very diverse, and that applying a singular definition to all of them fails to capture their rich biology. While I very much agree with their perspective, I take a somewhat different approach to the question of what are plant venereal diseases.

To answer this question, I turn for inspiration to Humphreys (1998), a philosopher who was faced with a similar problem when asked to write an encyclopedia entry on probability. He pointed out that there were three ways to approach the issue of terminology: definitional, operational and axiomatic. The nature of these different approaches will become clearer later because it is from these three standpoints that I want examine our use of the term venereal disease as it pertains to plants.

There are also other approaches to terminology. From an etymological standpoint, 'venereal' means 'of Venus' or of the Greek goddess of love. In so far as love is a human feeling correlated (albeit tenuously) with sexual contact, venereal diseases are defined as diseases that are transmitted during the act of sexual intercourse in humans. Clearly, plants neither have feelings nor do they have sexual intercourse, so this may appear to preclude them from having venereal diseases. However, plants do transmit disease via their sexual organs: pollinators fly from plant to plant spreading yeasts, mites, fungi, bacteria and viruses, and pollen grains carry viruses. So are these plant diseases also venereal diseases in the sense that we use the word in humans? This is the issue I wish to explore, not for the sake of semantics, but to show that the metaphor continues to lead to new perspectives on plant, animal and human disease.

### The Definitional Approach

A disease can be defined as venereal if pathogen transmission occurs largely as a result of sexual contacts between individuals. Currently, rather than labeling them venereal diseases, we are generally more comfortable calling them sexually transmitted diseases or STDs. In animals we usually think of STDs as

diseases that are transmitted between parents during mating. Because the process of mating in most animals is distinct and directly observable, this definition is fairly straightforward. As we will see, this definition is much less easy to apply to plants. It is also why in an earlier extensive review of sexually transmitted diseases, we confined the scope of the review to animals (Lockhart *et al.*, 1996).

We usually think of sexual transmission in humans as being a form of horizontal transmission, in that one of the partners gives the disease to the other. There is a strong case for arguing that pathogens that are transmitted from a parent to the zygote via the gametes are also STDs, but vertically transmitted ones. Nonsexual vertical transmission would occur by parent to offspring contact, for example, during lactation. In plants, such vertically transmitted pathogens include many viruses (Pathipanawat *et al.*, 1995), plasmids (Cornu & Dulieu, 1988), dsRNA molecules (Osaki *et al.*, 1998), and transposons (Wright & Schoen, 1999). This perspective of self-replicating genetic elements as vertically transmitted STDs (Hickey, 1982) has led to important insights into the evolution of uniparental inheritance (Hurst & Hamilton 1992; Law & Hutson 1992) and into the relationships between breeding systems, transposable elements, and genomic evolution (Wright & Schoen, 1999). However, in this review I will focus on horizontally transmitted STDs.

In flowering plants (and I will restrict discussion to this group), individual adult plants do not normally have direct sexual contact; direct contacts occur between the haploid generation of the male parent (the pollen grain) and the diploid generation of the maternal parent. Adult plants contact each other indirectly via a whole suite of pollination mechanisms including insect or bird visitation and wind dispersal of pollen. A disease that is transmitted from flower to flower by pollinators could therefore be defined as a venereal disease or STD because it is transmitted in the act of pollen transfer that in plants constitutes mating. From a definitional standpoint, there is immediate ambiguity because such a disease can also be classified as vector-transmitted. This ambiguity means that plant STDs might need to be modeled somewhat differently from STDs that are transmitted through direct physical contact, and this is discussed further later. Similarly, a pathogen that presents its infectious stages on the inflorescence but is carried from plant to plant by wind could be considered as being both sexually and aerially dispersed. Wennstrom & Ericson (2003) further point out that real definitional difficulties arise because pathogen presentation ('transmission from') may be on floral organs but that pathogen entry ('transmission to') may be via vegetative structures, to seedlings, or even mediated by overwintering in the soil. They favor restricting the term 'plant STD' to situations where there is disease transmission from flower to flower. Without trying to be comprehensive, I present in the following sections examples of several major routes of sexually related transmission in plants.

### The infectious stages of the disease are carried by pollinators

Our own studies have focused on anther-smut disease that is caused by *Microbotryum violaceum* (formerly *Ustilago violacea*), a basidiomycete fungus that invades the anthers, destroys the developing pollen mother cells, and replaces the anther sacs with fungal spores (Baker, 1947). The spores are then dispersed when pollinators visit the flowers (Altizer *et al.*, 1998). The disease is readily visible when the plant flowers because instead of the anthers producing yellow pollen, they are filled with dark-colored 'smutty' spores. The infection results in complete sterility because the ovary remains rudimentary and produces no seed. The pathogen itself, although often referred to by a single name, consists of many host-specific lineages (Zillig, 1921; Shykoff *et al.*, 1999). Anther smuts are most commonly found in the Caryophyllaceae where well over 100 species are infected (Thrall *et al.*, 1993a; Hood & Antonovics, 2003), but they are also present in the related Portulacaceae and other families such as the Lentibulariaceae, Dipsacaceae (Almaraz *et al.*, 2002) and Liliaceae (Fischer & Holton, 1957).

Many other types of pathogen are pollinator transmitted. By far the most important economically is fire blight of pears and apples, caused by the bacterium *Erwinia amylovora*. This can infect over 100 species within the Rosaceae and remains difficult to control in orchards (Bubán & Orosz-Kovács, 2003). The bacterium overwinters inside the plant and in the spring produces sugary lesions on the bark. The disease is carried to the growing tips of the shoots and developing flowers by insects where the bacteria infect the style, multiply in the flower, and penetrate the vegetative tissues through the nectary. Bees visiting the flowers then carry the disease from plant to plant.

Pollinators are also the major vectors for nectar-inhabiting organisms. There has been continuing discussion of whether these organisms are simply commensals or whether they have negative effects on the host. Microorganisms, especially yeasts, are abundant in flowers (Phaff *et al.*, 1978). Golonka (2002) isolated over 20 yeast species (including ascomycetes and basidiomycetes) from the nectar of *Silene latifolia*. The nectar yeast *Metschnikowia* forms colonies of four cells with a cruciform or 'airplane' morphology that appears well adapted to becoming attached to insect parts (Phaff *et al.*, 1978). *Metschnikowia reukaufii* has been shown to have a detrimental effect on seed-set in *Asclepias* largely because of inhibition of pollen tube growth (Kevan *et al.*, 1989). However, *Metschnikowia* had no impact on seed set in *Silene latifolia* (Golonka, 2002).

The anther smut, *Microbotryum*, also produces a yeast-like haploid 'sporidial' stage that multiplies in nectar and it has been isolated from nine other species flowering in the vicinity of diseased *S. latifolia* (Golonka, 2002). This stage deserves much further study, especially with regard to its role in the spread of anther-smut disease via the flowers of species other than the primary host.

Floral mites are another group of organisms that is transmitted by pollinators, including bees, butterflies and humming birds (Walter & Proctor, 1999). The humming bird-transmitted floral mites have been studied most extensively (Colwell & Naeem, 1994). It has been shown that they can be voracious eaters of pollen (Paciorek *et al.*, 1995) and nectar (Colwell, 1995; Lara & Ornelas, 2001), reducing nectar volume by as much as 50%.

It has long been speculated that, in addition to having a high sugar concentration, nectar may contain antimicrobial constituents. While nectar frequently contains toxic compounds (Adler, 2000) many of which have antimicrobial properties, there have been few direct studies of their efficacy in preventing floral infection. Golonka (2002) found that nectar isolated from *S. latifolia* inhibited the growth of yeast species significantly more than pseudo-nectar made from water and glucose and fructose sugars at concentrations found in real nectar. Recent molecular studies (Thornburg *et al.*, 2003) have shown that tobacco nectar contains several proteins, including one that is a superoxide dismutase, and results in high levels of hydrogen peroxide, which is toxic to microorganisms. The nectary tissue itself shows a greater expression of proteins involved in pathogen resistance than does the leaf tissue of the plant (Thornburg *et al.*, 2003), a situation that has close parallels with the presence of a large suite of antimicrobial proteins in the reproductive tract of mammals (Wira & Fahey, 2004).

### The infectious stages of the disease are presented in the flowers or on the inflorescences but they are wind dispersed

This transmission mode is characteristic of many smuts in the order Ustilaginales that infect the floral organs of plants, especially the grasses and other monocots (Fischer & Holton, 1957; Agrios, 1997; Ingram & Robertson, 1999; Wennstrom & Ericson, 2003). Very often the wind-dispersed spores infect through the leaves or seeds, the infections are systemic, and the various floral organs (the ovaries or the flowers themselves) are converted into dusty masses of spores. The main advantage to the pathogen of dispersal during the flowering stage appears to be that the spore-forming structures can be raised in the air and that therefore the spores can be carried longer distances. Moreover, the dispersal of the pathogen occurs at a time when the plants would normally be wind-pollinated, which are also presumably conditions that often favor the dispersal of spores. While spore dispersal is usually from the inflorescences, spore germination and infection can occur at the seedling stage, vegetatively or directly into the ovary of a flowering plant and then into the seed.

### The infectious stages are presented on pseudo-flowers, or in other ways that attract pollinators

The transmission of a substantial number of diseases in plants involves 'pseudo-flowers' or 'floral mimicry', where true

reproductive structures are not formed, but the pathogen induces changes in the host morphology and physiology that attract 'pollinators' which then act as disease vectors. The best-documented cases in agriculture include ergot (*Claviceps* spp.) of grasses and cereals (Butler *et al.*, 2001), and mummyberry disease (*Molinitia* spp.) of blueberries (Batra & Batra, 1985). The fungus causing ergot overwinters as a sclerotium formed in the ovary of the host plant. In the following spring, it germinates to produce fruiting bodies that release ascospores. These ascospores infect grass plants vegetatively, and when they flower, the fungi produce conidia in sugary secretions in the floral bracts that attract insects that spread the disease to new flowers. The ovaries of these flowers are converted by the fungus into overwintering sclerotia, or ergots. The pathogen causing mummyberry has a similar lifecycle. The pathogen overwinters as a sclerotium formed by the infected, hardened fruit (the mummified berry). When it germinates in early spring, the ascospores are ejected and infect young shoots. These shoots take on an elongated form, produce a sugary secretion containing conidia and become ultraviolet (UV)-reflective. This serves to attract bees that carry the conidia to the flowers, where they infect the ovary to produce the mummified berry.

The biology of 'pseudo-flowers' has been extensively studied by Roy and colleagues (Pfundner & Roy, 2000; Pfundner *et al.*, 2001; Naef *et al.*, 2002). The production of pseudo-flowers by rust fungi is especially common in the Brassicaceae where the phenomenon is known in over 1000 host species in 11 genera (Roy, 2001). These rust fungi induce the production of raised vegetative rosettes that are often yellow, fragrant and secrete sugary 'nectar' that attracts insects. However, these insects disperse the sexual stages of the fungus (spermatia) to the receptive hyphae, and do not directly transmit the fungus to new hosts. Instead, this occurs via the subsequent asexual stages that are wind dispersed from the vegetative parts of the host plants. A similar process occurs in some endophytes (e.g. *Epichloë*), where spermatia are transmitted between fruiting bodies by flies that also use the fruiting bodies as a food source (Bultman & Leuchtman, 2003). However, these fruiting bodies do not appear 'flower-like' and the cues used by the flies are likely to be olfactory. Definitions, therefore, become very confusing: in these cases the disease itself is not primarily transmitted by pollinators or insects, but they play role in the pathogen life-cycle (i.e. fertilization) that is analogous to the role played by pollinators in flowering plants.

### The pollen grains themselves carry the infection from plant to plant

Mandahar (1981) listed 37 viral diseases for which there was evidence of pollen transmission, but later Mandahar & Gill (1984) considered that pollen transmission was only firmly established in 19 of these. Given the large number of viral diseases known in plants, Mandahar (1981) considered this

transmission mode to be relatively rare. Moreover, most of the viruses were vertically transmitted to the seed rather than to the receiving maternal plant. There are direct analogies here with viruses that are sperm-transmitted in animals (Lockhart *et al.*, 1996), especially in groups such as the fishes (Mulcahy & Pascho, 1984) and possibly in many marine invertebrates that predominantly have external fertilization with no direct adult-to-adult contact.

A more recent review by Mink (1993) pointed out that while exclusion experiments in many cases had confirmed that many pollen grains carry viruses, the actual entry of the viruses into the parent plant appeared in many cases to be facilitated by the pollinator itself, or by thrips, causing damage to the flowers. It was therefore debated whether these viruses should or should not be considered as 'pollen transmitted' even though they were 'pollinator transmitted' from plant to plant. Regardless of definitions, clearly knowing the involvement of floral damage by thrips in transmission opens up another opportunity for disease control by, for example, reducing thrip populations on flowers. Identifying routes of transmission, rather than defining or classifying these routes, is the essence of the operational approach.

### The Operational Approach

The goal of an operational approach should be to measure and identify the pathways of transmission without being too concerned about how they are named. This is clearly important if we wish to actually control a disease, and it is a major approach of applied human and plant epidemiology. In animals such as insects, the standard way in which to study the degree to which a disease is sexually or nonsexually transmitted is to confine diseased and healthy individuals either in same-sex or two-sex mixtures. Such experiments also identify close-contact diseases that may not involve the sexual organs directly, and clearly may produce different answers from the definitional one discussed above. For example, close-contact diseases such as influenza or the common cold can be considered operationally as being partly sexually transmitted. The dilemmas of definitions is illustrated by mononucleosis, in that it has been labeled a 'kissing disease' (Biddle, 1995) rather than an STD.

These operational approaches have also been used to establish pollinators as mediating disease transmission in plants. Roche *et al.* (1995) studied the relative importance of floral vs vegetative transmission of anther smut, by setting out healthy flowering and nonflowering plants in arrays at different distances from flowering diseased sources. Their results showed that there was substantial vegetative infection, especially close to the disease source. Further from the source only flowering plants became diseased, indicating that pollinators were largely responsible for longer-distance transport. Many other field experiments with this system have also shown infection of nonflowering plants (Alexander, 1989; Alexander *et al.*,

1993); indeed, our routine inoculation technique is to simply place a drop of spore suspension on young seedlings, just as might happen naturally when spores fall on plants under moist conditions. This further highlights the difficulty of applying purely definitional approaches to transmission; thus, anther smut dispersal could be considered as sexual, whereas pathogen infection might be deemed nonsexual!

Manipulative approaches have also been used to establish routes of transmission in agricultural systems. For example, Bristow & Martin (1999) showed that transmission of blueberry shock ilarvirus (BlShV) was greatest in experimental cages where small hives of honeybees had been placed, that plants only rarely became diseased when their flowers were removed, and that transmission only occurred during the blooming period. This was consistent with the detection of virus particles on the surface and within pollen grains. There were also extremely low levels of seed transmission, suggesting that honeybee transmission is the main pathway for disease spread of this virus. The demonstration of pollen transmission of necrotic or prunus ring spot virus was achieved by long-term experiments involving the removal of flowers from trees in orchards (Cameron *et al.*, 1973; Davidson, 1976).

## The Axiomatic Approach

*'The abstract marriage theorem has been proven ... perhaps the main benefit of this theorem is the possibilities it suggests.'*  
Mauldin (1982)

The principles of disease dynamics can be considered in abstract terms, divorced from any particular instantiation. Three major aspects distinguish animal STDs from their non-sexually transmitted counterparts (Lockhart *et al.*, 1996), namely the nature of the transmission dynamics, their age-specificity, and the fitness consequences of the disease on the host. In STDs transmission depends on frequency rather than density of infectives; STDs are transmitted and expressed in the adult rather than juvenile stage; and STDs generally result in sterility rather than in high mortality. These ideas will be developed in the following sections.

## Transmission

In applied plant pathology, where the major concern has been with the spatial spread in crops planted at relatively uniform spacing, the predominant axiomatic approach has been to consider dispersal from a point source. This contrasts with studies of disease in natural systems where the interest has been on understanding population regulation. Here, the main approach has been to consider how individual behavior influences contacts, and how these contacts vary with density. I will take this perspective here.

To gain a better axiomatic perspective on venereal diseases, I will focus on the relationship between contacts and density.

The intuitive and natural assumption is that the number of contacts will increase with increasing density, and this is the classical mass-action assumption that has been made in most epidemiological models. However, because there has been some semantic confusion about the term 'mass-action' as applied to epidemiological models (De Jong *et al.*, 1995), I simply call this 'density-dependent transmission'. Density-dependent transmission also follows from passive dispersal of propagules: the closer individuals are, the more likely they are to become diseased.

However, in sexually transmitted diseases in humans and many animals, the number of sexual contacts per individual in a given interval may be relatively insensitive to density. People generally do not acquire more partners at more crowded parties and animals are limited in their mating opportunities by many factors unrelated to population density (e.g. territoriality, social structure, mate guarding, sperm plugs, etc.). It follows that if contact number is independent of density, then transmission depends only on the fraction of individuals that have the disease: this is 'frequency-dependent transmission' (Getz & Pickering, 1983; Antonovics *et al.*, 1995; Lloyd-Smith *et al.*, 2004). This type of transmission is expected for human venereal diseases; it was used in the first models of gonorrhoea (Hethcote & Yorke, 1984) and is now used in general models of acquired immune-deficiency syndrome (AIDS) (Anderson & May, 1991). Therefore, when a disease biologist speaks of sexual transmission in animals, the transmission dynamics is now canonically assumed to be frequency dependent.

We can now ask if this kind of frequency-dependent transmission also occurs in plants: Do plants have 'axiomatic STDs'? The answer is that they do, at least approximately so, but for a very different reason than in animals. Plants do not limit their number of partners and do not have territories, but pollinators are able to adjust their flight distances to compensate for changes in plant density (Levin & Kerster, 1969; Handel, 1983; Schmitt, 1983). Indeed, much of the stimulus for the early work on pollinator flight distances was to show that genetic neighborhoods in plants might be relatively independent of plant density. Indeed, it was this expectation that led us to make a direct conceptual, rather than definitional, connection between pollinator transmission in plants and sexual transmission in animals (Antonovics & Alexander, 1993; Thrall *et al.*, 1993b).

Another similarity between vector and sexual transmission arises if we assume that a vector such as a mosquito or tick takes a limited number of blood meals over its lifetime, or in some period of time. Transmission depends on the probability that the vector bites an infected host (i.e. on disease frequency within the host population), and measures of 'vectorial capacity' or the effectiveness of vectors at transmitting a disease include frequency-dependent terms (Anderson, 1981). There is also an increasing interest in applying frequency-dependent transmission to explain the prevalence of pathogens in vectors in natural communities that act as reservoirs for human

diseases (Schmidt & Ostfeld, 2001; LoGiudice *et al.*, 2003). Frequency-dependent transmission is therefore often posited as an approximation for both sexually transmitted and vector-transmitted diseases.

If we use this axiomatic approach and identify human venereal diseases as being canonically characterized by frequency-dependent transmission, it becomes clear that many plant diseases that I included in the previous sections as being venereally transmitted on 'definitional' grounds do not have frequency-dependent transmission. For example, most grass smuts that infect inflorescences are dispersed by wind and would be expected to show density-dependent transmission.

These distinctions are very important because whether a disease is transmitted in a frequency-dependent or density-dependent manner has large consequences for host–pathogen dynamics (Getz & Pickering, 1983; Antonovics, 1994). Single-species thresholds (Thrall *et al.*, 1993b; Lockhart *et al.*, 1996) and two-species thresholds are quite different, and in studies of disease prevalence in natural populations the expected positive relationship between density and disease prevalence may be reversed (Antonovics *et al.*, 1997). Moreover, while density-dependent transmission readily promotes population regulation, it is initially hard to see how frequency-dependent transmission would do so given that pathogen spread does not increase as host density increases. However, diseases with frequency-dependent transmission can regulate populations if there is also disease-independent regulation that only acts on the healthy class (Thrall *et al.*, 1993b, 1995). It is interesting that these conditions happen to be easiest to meet if the disease is also sterilizing and resource limitation acts on juveniles that, in the absence of vertical transmission, are not expected to acquire STDs. These features are typically found in many STDs, including the pollinator-transmitted anther smuts (Lockhart *et al.*, 1996).

The mechanisms of vector transmission are very rich biologically, and the simple scenario of pollinators flying further when host individuals are spaced further apart is a rather blatant oversimplification. Indeed, studies showing that seed set may be reduced at low densities suggests that such 'compensatory behavior' by pollinators may be restricted to a limited range of densities (Kunin, 1993, 1997; Wilcock & Neiland, 2002). Vectors may respond to host density because of 'functional' responses that result from changes in vector behavior, or 'numerical' responses that result from changes in the size of the vector population. Pathogens may also have life-history stages within the vector (e.g. many aphid and leaf-hopper transmitted plant viruses). Where the vector uses the host as a resource, it is likely that vector dynamics is coupled at some level with host dynamics; this is often assumed in many malaria models (Bailey, 1982). However, if the number of vectors is limited by factors other than host abundance, and if they cannot rapidly increase their visitation rate as host density increases, then the number of vector contacts per host individual is expected to decline as the density of hosts

increases. Indeed, at very high densities, one expects disease transmission by vectors to decline.

There have been essentially no experimental studies determining how disease transmission varies with density and frequency, either in plants or animals. The exceptions are studies of anther-smut transmission over a range of host and pathogen densities. Antonovics & Alexander (1993) showed that spore deposition, a surrogate measure of pollinator visitation, increased with an increasing frequency of diseased individuals, but declined somewhat with increasing density of diseased individuals. Antonovics *et al.* (1995), in a reanalysis of this data, also showed that spore deposition declined with an increase in total population density, indicating pollinator limitation. Bucheli & Shykoff (1999) also examined spore deposition over a range of disease densities and frequencies, as well as overall plant spacings. Their results showed a more complicated pattern, with treatment effects being significant only at the first sampling date. At the first sampling, spore deposition increased with diseased plant frequency, and was uninfluenced by plant spacings of 0.5 m and 1.5 m, but then declined at very large plant spacings (3.0 m). Biere & Honders (1998), studying anther-smut transmission in a natural population of mapped individuals of *Silene alba* and *Silene dioica* showed that disease spread was best explained by frequency-dependent transmission in patches where plants were closely spaced, but by density-dependent transmission in patches where plants were further apart. Because of their convenience and immobility, plants provide much better opportunities than animals to investigate the dynamics of the transmission process both experimentally and observationally. Extending such experiments to other disease systems is important for establishing empirical generalizations about the transmission process.

In humans, where experiments are impossible but individuals are more easily identified, individual behaviors and mating structures are used to infer rates of STD spread (Anderson *et al.*, 1989). There is therefore a rather large and somewhat overwhelming body of theory on effects of number of partners, copulations per partner and concurrence of multiple partnerships and sexual activity classes on STD spread in humans (Castillo-Chavez *et al.*, 2002). However, it is difficult to see how such a theory could be easily applied to natural populations of plants or animals. Even in mammals, where there is extensive data on mating systems, it is often unclear how to translate this data into measures of potential disease transmission (Thrall *et al.*, 2000; Altizer *et al.*, 2003).

### Age-specificity

Another axiomatic property of STDs in animals is that they are transmitted at the adult stage only during the mating or flowering season. Several expected features of STDs follow from this.

First, in the absence of alternative transmission modes or alternate hosts, the persistence of such diseases is dependent

on overlapping generations. If reproduction is continuous, as in humans, these conditions are easy to meet, but in seasonally reproducing organisms it requires that the hosts be perennial. We showed (Thrall *et al.*, 1993a) that records of anther-smut disease were much more frequent in perennial than in annual members of the Caryophyllaceae. Viruses that are transmitted to another adult plant via the pollen also appear to be frequent in small fruits and orchard crops that are perennial (Mandahar, 1981; Mink, 1993). Conversely, we can predict that if a disease is transmitted only in the adult phase, then there should be alternative adult transmission modes or methods whereby the disease can persist over winter; this appears to often be the case in many of the inflorescence smuts in the Ustilaginales that infect annual grasses (Fischer & Holton, 1957). It would be interesting to review the life-histories of adult-transmitted diseases in annual and perennial hosts.

Second, because of this requirement for host persistence, we would also predict that diseases transmitted in the adult phase would have a much smaller effect on host mortality than diseases which are transmitted in the juvenile stages. I discuss this further in the following section.

Third, if the disease is transmitted only to adults, as in the case of frequency-dependent sexual transmission, then the diseased class will include no juveniles (e.g. no seedlings or prereproductives), whereas the healthy class will include both juveniles and adults. It is well known that resource limitation on growth and survival in perennial plants are much more likely to act at the seeding stage than at the adult stage (Shaw & Antonovics, 1985). Therefore, resource factors limiting population size are more likely to act on the healthy class that includes juveniles than on the diseased class that includes only adults. This inequality of resource limitation on the diseased and healthy classes facilitates the coexistence of hosts and their pathogens when there is frequency-dependent transmission. The reason is that while the healthy class may increase at a greater rate than the diseased class when the population is small, and may therefore wash out the disease, this is reversed when the population is large. Therefore, the disease has an advantage in large populations not because it is transmitted more, but because diseased individuals are less affected by resource limitation (Thrall *et al.*, 1993b).

## Virulence

The negative effect of a pathogen on the fitness of the host can be expressed either as increased mortality or as decreased fecundity. Much of the focus in classical disease biology has been on the mortality component of virulence, but there are a number of reasons to expect STDs to primarily affect fertility. Diseases that affect fertility are likely to have a more severe effect on population size than diseases that affect mortality because increased mortality also reduces the duration of the infection. Consequently, the forces that affect the evolution of virulence are different depending on whether

the pathogen increases mortality or decreases fecundity (O'Keefe & Antonovics, 2002).

First, as mentioned above, the persistence of many STDs without alternative transmission modes is dependent on perenniality; increasing host mortality would greatly decrease disease persistence. However, simple adaptive reasoning would suggest that all pathogens should evolve in the direction of increasing sterility rather than decreasing mortality, as sterility components of virulence do not affect the duration of the infectious period. Indeed, in many examples of 'parasitic castration' the growth rate and longevity of the host are increased (Clay, 1991). Second, because transmission is during the reproductive process, lesions that are associated with disease transmission will be likely to negatively influence reproductive success. Third, theoretical studies on conditions for coexistence of frequency-dependent pathogens with their hosts show that such coexistence is more likely when the disease causes sterility rather than mortality (Thrall *et al.*, 1993b). In an analysis of causal relationships between sterility and transmission mode, we failed to distinguish whether sexual transmission led to the evolution of sterility, or whether sterility favored the establishment of STDs (Lockhart *et al.*, 1996). However, this study was broadly comparative and there is a need for more rigorous phylogenetic studies of virulence evolution, and this may be especially productive in plants given the diversity of transmission modes that are associated with reproduction.

## Evolution of sexual transmission

Another axiomatic aspect of sexual transmission in animals is that we would expect this transmission mode to be favored at low population densities, when sexual contacts might more common than nonsexual contacts (Smith & Dobson, 1992). We used generalized contact functions to investigate the evolution of transmission mode (Thrall *et al.*, 1998) by considering the relative success of pathogen strains that allocated differently to sexual and non-sexual transmission. We showed that the relative success of pathogens with different degrees of the two types of transmission was indeed related to the equilibrium population sizes of the diseased populations, but that these sizes were not just determined by transmission mode but by the sterility and mortality effects of the disease. Another particularly interesting result was that even if two strains excluded each other from an individual host, they could still coexist if they had different transmission modes (Thrall & Antonovics 1997). It is interesting that several human sexually transmitted diseases have a nonsexually transmitted counterpart (e.g. herpes 1 and herpes 2, pubic lice and head lice, genital and ocular chlamydia, syphilis and yaws). There is little information in plant pathogens on factors that might determine the evolution of different transmission modes, or the pathways of such evolution as determined by phylogenetic relationships. A system that shows promise for

untangling the evolution of transmission modes is the *Epichloë* system, which has both vertical and horizontal transmission (Meijer & Leuchtman, 2000, 2001).

## Conclusion

Pursuing the 'messy metaphor' of plant venereal diseases can clarify our thinking about the component processes involved in disease dynamics. It especially focuses our attention on the importance of transmission mode in disease dynamics, rather than just on transmission mode as a target against which to direct disease control efforts. Perhaps, most importantly, it shows us that there are huge gaps in our knowledge of many aspects of disease biology in both plants and animals. For example, when we search for data on the functional form of the relationship between contacts (or probability of an individual becoming diseased) and host density there are very few data either in humans, animals or plants (but see Biere & Honders, 1998; Begon *et al.*, 1999), even though it is very fashionable to look for a positive relationship between density and disease incidence. Experimental studies are almost nonexistent, especially those that go beyond changing density over more than just a very limited range.

The study of disease in natural populations of both plants and animals has, until recently, been a neglected topic, and we are only now starting to show that disease can have important consequences for population dynamics, community succession and maintenance of species diversity. These studies have been further stimulated by the increasing realization that human and crop diseases emerge from, or are sustained by, natural reservoirs. Studying how to cure a plant disease will not help us directly to cure AIDS or influenza or any other human disease. However, there are important generalities that cross all the taxonomic boundaries and that inform our understanding of all diseases. Just as the phenomena of transposable elements and post-transcriptional gene silencing (which were first characterized in plants) stimulated extensive studies of those phenomena in animals and humans, so, for example, experimental studies with plants on the functional form of the transmission process has already focused our attention on transmission dynamics in animal and human populations. For example, including frequency-dependent transmission improves the explanatory power of models of measles epidemics (Bjørnstad *et al.*, 2002); measles is not a venereal disease, but it is primarily transmitted in schools where the number of students per classroom and hence number of contacts is relatively constant. Conversely, thinking about animal and human STDs leads us to enquire whether there are also general and as yet unexplored features of diseases such as the grass smuts which have elements of their biology that combine both sexually and nonsexually transmitted diseases.

By far the most useful aspect of the 'messy metaphor' is that it helps us to think about aspects of disease biology that cross organismal boundaries, rather than only the details and

particularities of those diseases that are the specific targets of research in medicine and agriculture.

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