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The use and abuse of pollinators by fungi

B.A. Roy

Fungi can use flower-visiting insects such as bees, flies and butterflies in at least three distinct ways. First, some rust fungi use pollinators the same way as flowers, that is, to ferry gametes between different fungal individuals for sexual reproduction (Fig. 1a). Second, some pathogenic fungi use their hosts' pollinators to transfer infectious spores to new hosts; these are the 'sexually transmitted' plant diseases such as the anther smuts (Fig. 1b). And third, a few non-pathogenic fungi, such as the stinkhorns (Phallales, Fig. 1c), use pollinators to disperse their spores.

The mechanics of pollinator attraction

Some fungi do not attract pollinators on their own, but instead take advantage of the insect-attracting ability of flowers^{1–8}. For example, the violet-spored smut fungi (some species of *Ustilago* and *Microbotryum*⁹; see Box 1) sporulate in the anthers of dicot flowers (Fig. 1b). While foraging for pollen and nectar, pollinators visit both infected and uninfected flowers and transmit smut spores to new hosts^{3–5,10}. Despite the apparent passiveness of this system, anther smuts partially or completely sterilize their hosts^{5,11,12} and may alter their hosts in ways that enhance spore production and transmission^{2,13}. For example, smut-infected plants often have more flowers than uninfected ones^{2,5}, infected plants flower earlier than uninfected ones⁵, and infected flowers can remain open longer than uninfected ones⁵.

Instead of using existing flowers, other fungi have evolved their own pollinator attraction systems^{14–19}, though these systems often share some of the characteristics of true flowers such as bright coloration and/or the production of some kind of food reward and scent. Sexually receptive rusts, for example, are typically red, orange or yellow (Fig. 1a)^{17,20}, and the stinkhorns (Fig. 1c) can be bright orange-red (e.g. *Mutinus*, *Aseroe*) or starkly contrast-

Some fungi use flower-visiting insects to facilitate sexual reproduction or to disperse spores. These fungi have evolved elaborate techniques, such as floral mimicry and the invasion of extant flower parts, for attracting 'pollinators'. Recent research shows that fungal exploitation of pollinators has the potential to affect floral evolution, pollination ecology, plant life history traits, as well as disease-transmission dynamics and fungal evolution.

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ing dark olive and white (e.g. *Dictyophora*). Rust fungus spermata (non-motile sperm) are borne in solutions containing sugars¹⁷, and sugars are also found in the spore mass (gleba) of stinkhorns²¹ (Fig. 1c). Many rust fungi emit an odor – some species smell pleasant and flower-like^{8,17,22}, whereas other species smell like carrion⁸. The stinkhorns stink: to our noses they are nasty and repellent – rather like rotting meat with an overlay of cloying sweetness.

Many rust fungi and flowers attract flying insects. Thus, it is not too surprising that they have evolved similar ways for doing so. However, some rust fungi are clearly parasitizing the relationship between flowers and their pollinators. An example of a rust fungus that takes advantage of its host's pollinators is *Uromyces cladii*¹⁹ (also see Patt, J.M., unpublished dissertation, Rutgers University, USA, 1992). Early in the spring, before the aroid *Peltandra* flowers, *U. cladii* erupts on its leaves. At this time, the rust exudes spermata in a sugary fluid which smells like the host's flowers. The host-specific pollinators of *Peltandra* – flies in the genus *Elachiptera* – visit and feed on the spermata and, in the process, aid in outcrossing the fungus. Later, as the flowers of its host begin to open, the fungus produces aeciospores, which are infectious. The flies then act as vectors, transporting the aeciospores to *Peltandra* flowers, where they germinate and infect a new host. Patt¹⁹ suggests that the relationships between *Uromyces*, *Peltandra* and *Elachiptera* may be mutualistic. The fungus provides a food source for the host-specific pollinators of *Peltandra* before they flower. This may facilitate population growth of the flies, leading ultimately to more *Peltandra* pollination later. The flies are critical for fertilization and for dispersal of the fungus, and infection causes little reduction in host survival.

A more complicated example of fungal exploitation of pollinators is found in the mustard rusts, *Puccinia monoica*



Fig. 1. Three ways insects are used by fungi. (a) *Arabis holboellii* infected by *Puccinia monoica* and being visited by *Polygonia zephyrus*. Insects visiting infected plants transfer spermatia (gametes) between different mating types of the fungus^{17,18}. Insects thus aid the rust fungus' sexual reproduction in much the same way that they facilitate angiosperm reproduction by moving pollen between flowers. Photograph by B.A. Roy. (b) *Silene acaulis* infected by *Ustilago violacea*. The flower in the center with light-colored anthers ('H') is healthy and its anthers are filled with pollen, whereas those with dark anthers ('I') are infected by *Ustilago violacea* (*Microbotryum violaceum*; see Box 1) and their anthers are filled with teliospores. Insects act as vectors of this disease by transferring teliospores from one host to another^{1,5}. Photograph by B.A. Roy. (c) *Mutinus caninus*, a non-pathogenic fungus (the *Iris* plant just happens to be in the background; it is not being parasitized) being visited by a fly. Stinkhorn fungi (Phallales) disperse their spores by attracting insects that eat the spore and sugar-containing gleba ('G'). The spores pass through the insect's gut unharmed, then germinate if they are deposited on a suitable substrate³⁶. Photograph by W.J. Sundberg.

and *P. thlaspeos*. These fungi transform the vegetative morphology of their hosts (*Arabis* and other genera in the mustard family) into flower-like structures (pseudoflowers, Fig. 1a) on which the yellow fungus erupts, producing nectar, scent and spermatia¹⁷. Insects visiting pseudoflowers outcross the fungus. In an interesting twist, the pseudoflowers do not mimic the flowers that the host normally produces (which usually do not flower at the same time), but instead they mimic co-blooming yellow-flowered species such as buttercups^{17,18}. In contrast with the *Peltandra* rust described above, the relationship between the mustard rusts and their hosts is not mutualistic and represents a clear case of parasitism since they typically sterilize and kill their hosts.

Consequences of pollinator attraction by fungi

The consequences for the fungus of having spores transported by insects depends on the kind of spores being transferred. Some fungi require insect visitation for sexual reproduction and the continuation of their life cycles. These fungi may also benefit from the increased genetic variation that results from outcrossing. For other fungi, spore transmission by flower visitors (sexually transmitted diseases) allows exploitation of new hosts. And finally, spore dispersal by pollinators promotes dispersal of non-pathogenic fungi to new environments. So why is insect transportation favored over wind or water dispersal? The most obvious advantage of biotic as opposed to abiotic transport is that insects, particularly pollinators, move in a more directed fashion²³.

The differing biological functions of the spores (sex versus transmission or dispersal) may influence the course of selection on both the fungus and the insects. For example, when pollinators are being used to transfer gametes between fungal individuals, the fungus is better off if the insect visits another infected plant next, instead of a flower where the fungal gametes would be lost. In this case, selection on fungal features that are attractive to insects is strong. Unique fungal features that are attractive to insects may ultimately lead to the evolution of insects specialized to respond to them. Is there an association between insect-mediated fungal reproduction and special-

ist visitors? In two of the cases described here, *Uromyces cladii*¹⁹ and *Epichloe typhina*^{15,24}, fungal-specific visitors have evolved. However, the mustard rusts (*Puccinia monoica* and *P. thlaspeos*) depend on their ability to attract generalist pollinators¹⁸. Nonetheless, some visitors to the rust-induced pseudoflowers may show a preference for them¹⁸.

In contrast to transportation of fungal gametes, when flower visitors are being used to transmit infectious spores, such as in anther smut infections, the fungus is better off if the insect visits an uninfected flower next. In this case, there may be little or no selection on the ability of the fungus to attract insects to itself. Instead the fungus relies on the attractiveness of the host flowers. For disease to be transmitted effectively, insects must visit both healthy and infected flowers. Do insects visit diseased and healthy flowers randomly, or do they show preferences? The limited empirical evidence gathered to date suggests that insect movement is nonrandom with respect to anther

Box 1. Evolution in smut taxonomy: *Ustilago* versus *Microbotryum*

The Caryophyllaceae is a dicot plant family that is prone to anther smut infection. A historic trend in both parasitology and pathology has been the tendency to assign a different species name to the pathogen for each genus, or sometimes even species, of host infected. Thus, there is a plethora of names for the anther smut infections of the Caryophyllaceae. Up until 1982, the genus of the Caryophyllaceae anther smuts was *Ustilago*, and the most commonly used species name was *Ustilago violacea*. In 1982 Deml and Oberwinkler³⁸ transferred the species of *Ustilago* that infect the family Caryophyllaceae to the genus *Microbotryum*. However, the taxonomy remains confused for three reasons. First, the species of *Microbotryum*, which are based primarily on host differences, intergrade morphologically⁹. Second, there are additional violet-spored anthericolous smuts that infect other families, such as the Lamiaceae, Lentibulariaceae, Portulacaceae and Primulaceae, and that may ultimately be transferred to *Microbotryum* but which are still being called *Ustilago*⁹. Finally, many people have chosen to continue to call the most common Caryophyllaceae smut *Ustilago violacea*, instead of *Microbotryum violaceum*, because so much work is published under the name *Ustilago* (H.M. Alexander, pers. commun.).

smut disease. Bees tend to visit infected plants less often than healthy ones^{6,25,26}, however, some individual bees prefer infected plants^{25,26}, and nocturnal insects such as moths also seem to prefer infected plants²⁵. What are the consequences of nonrandom insect visitation? Simulation models of vector movements²⁵ (and L. Real, pers. commun.) indicate that when the frequency of disease is high, preference for healthy flowers will increase the rate of disease spread because the probability of accidentally visiting a diseased flower increases. On the other hand, vector preference for infected hosts will cause more rapid disease spread when the frequency of disease is low because healthy flowers are more prevalent, thus it is more likely for vectors to accidentally visit healthy flowers.

One result of the tendency of pollinators to specialize on a single flowering species is that the density of infected individuals in a population may be less important for disease transmission than their proportional representation (frequency) since pollinators can adjust their flight distances to accommodate differences in density¹⁰. The dynamics of host-pathogen coexistence depend, in part, on how disease is transmitted (Fig. 2). Host-pathogen co-

existence is possible under density-dependent transmission models when host density is low enough to limit, but not eliminate, pathogen transmission^{27,28}. However, coexistence is not stable under simple frequency-dependent transmission models²⁹ because disease can continue to spread even when host densities are low. Coexistence under frequency-dependent transmission is thus restricted to the occasions when high infection rates are combined with high host recruitment rates. In reality, both density-dependent and frequency-dependent processes are likely to contribute to population regulation and host-pathogen coexistence^{29,30} (Fig. 2). Simulations²⁹ indicate that coexistence of the host and pathogen is likely when density-dependent processes other than disease act more strongly on healthy individuals than on infected ones. For example, in *Silene*, juveniles are almost always healthy because infection typically occurs when the adult plants flower. Recruitment into the healthy class may be regulated by density-dependent processes, such as competition, that limit the number of juveniles that survive to adulthood, whereas recruitment into the diseased class depends primarily on frequency-dependent transmission of infection.

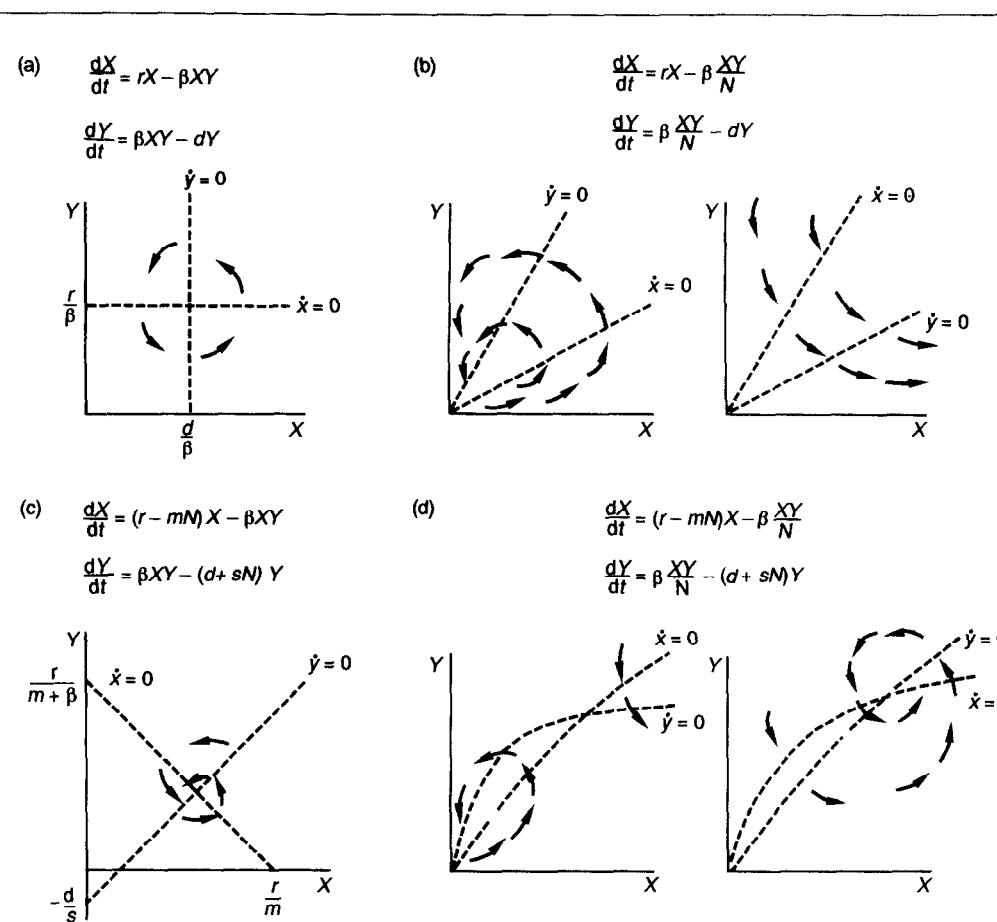


Fig. 2. The potential for stable coexistence of the host and pathogen under different conditions of frequency-dependent or density-dependent disease transmission. The phase diagrams show changes in numerical abundance of the host (X) and pathogen (Y). (a) Density-dependent transmission; (b) frequency-dependent transmission by pollinators; (c) density-dependent transmission with density-dependent host survival and reproduction; (d) frequency-dependent transmission by pollinators with density-dependent host survival and reproduction. In (a), disease transmission is density-dependent and coexistence of the host and pathogen is probable. In (b), disease transmission is frequency-dependent and coexistence is not stable. Disease is purged from the host population if birth rates are high, otherwise both the host and pathogen go extinct. In (c), disease transmission is density dependent, and survival and reproduction depend linearly on total plant density. Under these conditions, the host and pathogen can coexist. In (d), disease transmission is frequency dependent but survival and reproduction depend linearly on total plant density. Under these conditions coexistence depends on the values of the parameters used, and is most likely when density-dependent regulation acts differently on healthy and infected individuals. r = birth rate – death rate of host, β = disease transmission rate, d = death rate of the host, $N = X + Y$, s and m are constants. Zero growth isoclines are shown as dotted lines. The zero growth isoclines for (c) are implicit functions in X and Y , and have been drawn only approximately. Modified, with permission, from Ref. 30.

Under these conditions, where density-dependent processes act differently on the healthy and infected classes, frequency-dependent disease transmission can lead to stable coexistence of the host and the pathogen^{29,30} (see Fig. 2).

Plants that require insects to effect pollination, but that are also susceptible to pollinator-transmitted diseases such as anther smuts, face an awkward trade-off: the more pollinators they attract, the more likely it is that they will become infected³¹. One way plants can compensate for the problem of increasing infection with higher flower numbers is to produce numerous seeds per successful pollination event³¹. Another way plants may compensate is to reproduce before the infection has an opportunity to become systemic, thus avoiding the worst sterilizing effects of infection. The flowers that receive spores do not themselves become infected and can produce healthy seeds³². After inoculation it takes several weeks for anther smut fungus to infect flower buds and it can take up to two years for infection to become completely systemic¹. The best way of all to escape from vectored systemic infections may be the adoption of an annual life cycle. A recent survey of the

life history characteristics of a family prone to anther smut infection (the Caryophyllaceae) showed that anther smut is predominantly a disease of perennial species; annuals are rarely infected³³.

Is the attraction of flying insects an advantage to stinkhorns, which are not pathogenic? Or, are the bright colors, strong scents, sugar production and spores in a sticky mass the result of other historical or accidental processes? Numerous other fungi are dispersed by insects³⁴, but many of these are not so obviously attractive to flying insects. It remains to be determined whether or not the stinkhorns have a requirement for insect dispersal, or how often the insect associations with stinkhorns are obligate. The dispersal ecology of stinkhorn spores is likely to have parallels with animal seed dispersal. For example, flying insects may defecate spores in areas that are conducive for the growth of stinkhorns, spore germination may be enhanced after passage through insect digestive tracts, and the spores may grow better in the more nitrogenous environment that fly feces provide.

The spores of pollinator-visited fungi may serve as an important food source for insects. For example, the stinkhorns (Fig. 1c) are visited by two suites of insects: those that lay eggs in them (Diptera), and those that visit to feed on the gleba (Diptera, Lepidoptera, Coleoptera)³⁵. Experiments have established that blowfly oocytes can complete development when flies are fed a diet consisting only of the gleba of the stinkhorn fungus, *Mutinus caninus*³⁶ (Fig. 1c). They do develop but their development is slower than on meat because the fungus spores contain less protein. Spores are also known to be an important food source for the anthomyiid fly, *Phobia phrenione*, which both 'pollinates' and lays its eggs in the fungus *Epichloe typhina*^{15,24}. Other evidence that spores act as food sources is more circumstantial. For example, pollinators spend considerably more time feeding on spore-covered fungal pseudo-flowers than on true flowers^{17,18}, and some bees^{8,26}, moths²⁶ and butterflies⁴ preferentially collect spores instead of pollen. The presence of these fungal food sources may increase the number of pollinators available for pollination if they add to a resource base, or they may extend the normal season of a pollinator by occurring earlier or later than normal food sources. More studies are needed to assess the ability of insects that collect fungal spores to survive on diets containing them, particularly in systems that suggest a mutualistic interaction between the fungi and pollinators.

Conclusions and future research

The existence of fungal flower mimics, floral odor mimics, and pathogens that are transmitted by pollinators all suggest that some fungi are taking advantage of the relationship between flowering plants and their pollinators. Further research is required to solidify this assertion and to address the numerous questions raised by the few observations that have been made. For example, we have very little empirical data on the behavior of insects. When do they show preferences for the fungi? How often and under what circumstances are the visitors specialists or generalists? What are the consequences of different kinds of behavior?

The rust fungi are as old or older than angiosperms³⁷. They might well have evolved insect attraction on their own, and only secondarily have come to parasitize flower pollination systems by becoming more similar to flowers. On the basis of host association, Savile⁸ suggests that the rust fungi that smell like carrion may be more primitive

than the species that smell sweet like flowers. It would be interesting to know whether the bad-smelling rusts are visited primarily by non-pollinators and primitive insects such as beetles, and whether molecular characters also suggest that the sweet-smelling taxa are more derived. Ecological studies combined with phylogenetic analyses could provide answers to these and related questions.

Another set of unanswered questions concerns the ability of pathogenic fungi to use their hosts. Are the changes in host architecture all hormonally mediated, and if so, by whose hormones, the fungus' or the host's? For example, do the rust fungi derive energy from their hosts to manufacture the compounds for bright colors, sweet nectar and scents on their own, or are the rusts somehow using host chemical products directly? In anther smut infections, there are differences in morphology between infected and uninfected plants, but few studies have established experimentally that the fungus alters the plant and is not simply infecting plants with a particular morphology. It would be interesting to learn whether other anther smut fungi of dicots, besides *Ustilago violacea*², alter their hosts in ways that benefit the fungus.

The interaction of insect pollinators and fungi can generate selection on the reproductive systems of the fungi, the reproductive and vegetative systems of host plants, and on the insects themselves. These fungus–pollinator systems are amenable to the empirical study of both mutualistic and parasitic interactions and are probably much more common than the limited literature to date indicates.

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Small-step invasion research

Rob Hengeveld

There are six main areas of interest in the study of biological invasions: (1) the possibility of predicting which species will become an invader and which will not; (2) the way the invasion progresses through space; (3) the possibility of predicting the rate of invasion; (4) the local build-up of the newly settled population; (5) the pheno-genetic differentiation of the new range; and (6) the effect the invader has on native species.

These six areas can be grouped roughly into three categories, each with its own methodology. First, predicting the possibility of becoming an invader (i.e. area 1) will often involve physiological or genetic work when either habitat conditions are altered, or when the species itself is changing. At present, it can also involve historical ecological studies of broad-scale landscape restructuring. The second category (2,3,4) involves analysing the invasion process proper, using mathematical analysis. The third category concerns studies on either the adaptation of the invader itself to its new living conditions (5), or that of the native species to the potential or new invader (6); this involves genetic, physiological and morphological investigations, and work at the level of the community, respectively. This review is concerned mainly with the second category.

The mathematical approach assumes that a species has become an invader and that it is adapted to its new abiotic and biotic conditions. Although this simplifying assumption can be a drawback, it can also provide a null model for evaluating the impact of changes in abiotic conditions (1,5), or the impact of biotic conditions. The latter conception can prove to be very productive both for the study of a species' geographical dynamics and for community ecology. Thus, by making small steps, one can progress steadily. This approach is presently being followed mainly by a group of Dutch workers. Apart from applying it to invasions proper, it can also be applied to epidemiology, Quaternary ecology, to effects of genetic manipulation, and to gap dynamics inside a species range.

Recent Invasion research can be categorized broadly into two types: studies of the spatial spread, and those of the biological impact the invader has on the native biota. The first type is the most factual, and the second the most theoretical. So far, however, it is difficult to connect the two, implying that neither the spatial spread nor the species interactions can be explained in terms of each other. Recent models, analysing spatial spread, progress steadily by making small steps.

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The model

Most simply, invasions can be conceived to consist of two-component processes; the process of spatial spread and that of local population build-up after arrival and settlement. When the organisms are assumed to disperse and reproduce independently and, moreover, when that movement is Brownian and the trapping rate by the environment uniform, then the process (in its simplest form) can be described as a reaction-diffusion process in two dimensions¹:

$$\frac{\partial n}{\partial t} = rn + \frac{1}{2}s \left(\frac{\partial^2 n}{\partial x_1^2} + \frac{\partial^2 n}{\partial x_2^2} \right) \quad (1)$$

where $n(t, x_1, x_2)$ is the population density at time t at location $x = (x_1, x_2)$ in a two-dimensional plane; r is the species' intrinsic rate of increase; and s is the diffusion constant. The reaction component is represented by rn and the diffusion component by the remainder of the equation. Yet, although this equation is widely quoted in the context of invasions, it assumes that the rates of reproduction, mortality and dispersal are independent of age, which need not be true. This has been cured in the new approach described here.

Although the basic model of the new approach, and the assumptions made, are the simplest possible, both the model and its translation into parameters (whose values can be estimated with field data) are complex^{2–7}. The parameters can be grouped into those that describe the aspects of the species' life history relevant to population growth and those that concern dispersal. The life-history parameters are the specific survivorship $L(a)$, that is, the probability that an individual is still alive at age a , and the specific fertility $m(a)$, that is, the rate of offspring production of an individual at age a . These parameters can be estimated from a Leslie matrix⁸. Dispersal is assumed to be the same in all directions – rotationally symmetric – and is defined as the probability that an individual of age a born at location ξ is found still alive at location x . Dispersal parameters can be estimated from the distribution