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BIZARRE INTERACTIONS AND ENDGAMES: Entomopathogenic Fungi and Their Arthropod Hosts

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■ **Abstract** Invertebrate pathogens and their hosts are taxonomically diverse. Despite this, there is one unifying concept relevant to all such parasitic associations: Both pathogen and host adapt to maximize their own reproductive output and ultimate fitness. The strategies adopted by pathogens and hosts to achieve this goal are almost as diverse as the organisms themselves, but studies examining such relationships have traditionally concentrated only on aspects of host physiology. Here we review examples of host-altered behavior and consider these within a broad ecological and evolutionary context. Research on pathogen-induced and host-mediated behavioral changes demonstrates the range of altered behaviors exhibited by invertebrates including behaviorally induced fever, elevation seeking, reduced or increased activity, reduced response to semiochemicals, and changes in reproductive behavior. These interactions are sometimes quite bizarre, intricate, and of great scientific interest.

INTRODUCTION

> An epidemic in the open air, caused by *E. muscae*, I have, however, observed in one locality where a hairy black fly (*Anthomyia* sp.) about as large as *M. domestica*, was found killed by this fungus. This locality was in the

> region in the immediate vicinity of the snow arch at the head of Tuckerman's ravine, on Mt. Washington, where the affected flies occurred sticking in large numbers to the flowers of *Solidago* and *Heracleum*. These flowers were also visited by an abundance of other flies, among them many examples of the same species of *Syrphus* previously mentioned from Mt. Washington and Albany; but in no instance did I find one of these or the numerous other Syrphidae and Muscidae visiting the flowers, that showed any sign of infection from the black flies with which they could hardly have failed to come in contact.
>
> Thaxter (100)

Entomopathogenic fungi, such as the one described in 1888 by Thaxter (100), and their hosts are taxonomically diverse. However, there is one unifying concept relevant to all such parasitic associations: The pathogen and host adapt to maximize their reproductive output and ultimate fitness by adopting physiological, ecological, and behavioral adaptations that are as diverse as the organisms themselves. In this review we specifically explore pathogen-induced alterations to host behavior and focus on pathogenic fungi and their insect hosts. We consider whether specific changes in host behavior benefit the host or pathogen or are simply a nonadaptive side effect of pathogenesis.

THE COEVOLUTIONARY ARMS RACE BETWEEN ENTOMOPATHOGENIC FUNGI AND THEIR HOSTS

The term coevolution was first developed by Mode (63) to describe the influence interacting species have on one another through evolutionary time. Janzen (47), more specifically, defined coevolution as a process in which each interacting species changes its genetic structure in response to a genetic change in its partner. Pathogens and hosts provide excellent models for studies on coevolution. They are intrinsically linked but their evolutionary interests diverge: Selection on the pathogen is for greater exploitation of the host and selection on the host is for exclusion of the pathogen (12). Pathogens and hosts are constantly evolving in an aggressive manner toward one another.

It has traditionally been accepted that a coevolutionary arms race between a pathogen and its host would inevitably result in commensalism. More recent theory suggests that outcomes ranging from highly antagonistic to commensal are possible (58) and are dependent on many complex and interacting factors. An arms race manifests in a variety of adaptations, but host-pathogen relationships are often considered only in terms of the impact of host susceptibility and pathogen virulence on physiology. Behavioral alterations are often ignored, even though they affect parameters essential to pathogen and host evolution such as transmission and longevity (66).

BIOLOGY AND TAXONOMY OF ENTOMOPATHOGENIC FUNGI

Entomopathogenic fungi (Table 1) are common natural enemies of arthropods worldwide, attracting attention as potential biological control agents (38, 90). Fundamental research has focused on many theoretical and practical aspects of their biology, physiology, ecology, and epidemiology, but predominantly from the viewpoint of their potential in host population regulation. There are more than 700 species of entomopathogens from within the fungal kingdom. Most species are from the fungal divisions Ascomycota and Zygomycota. The ascomycete fungi were previously divided into two groups, the Ascomycota and the Deuteromycota, the latter of which were known as the Fungi Imperfecti, species for which no sexual stage was known. Cultural and molecular studies have demonstrated that some of these "imperfect fungi," (formally class Hyphomycetes in the Deuteromycota) were anamorphs (asexual forms) of the Ascomycota within the order Hypocreales, family Clavicipitaceae (30, 40, 53, 91). Within the Zygomycota most entomopathogenic species are in the order Entomophthorales.

Entomopathogenic fungi produce infective spores (conidia) that attach to, germinate, and penetrate the cuticle of their host without the requirement for ingestion. Once within the host they proliferate as a progression of single- or multicelled structures (protoplasts, blastospores, hyphal bodies) exploiting the

TABLE 1 Current classification of the genera of entomopathogenic fungi included in this review

Division	Class	Order	Family	Genus
Zygomycota	Zygomycetes	Entomophthorales	Entomophthoraceae	*Entomophaga*
				Entomophthora
				Erynia
				Eryniopsis
				Furia
				Massospora
				Strongwellsea
				Pandora
				Tarichium
				Zoophthora
			Neozygitaceae	*Neozygites*
Ascomycota	Sordariomycetes	Hypocreales	Clavicipitaceae	*Beauveria*[a]
				Cordyceps
				Cordycepioideus
				Lecanicillium[a]
				Metarhizium[a]
				Nomuraea

[a]*Beauveria* and *Metarhizium*: anamorphic Clavicipitaceae with teleomorphic connections to *Cordyceps*; *Lecanicillium*: anamorphic Clavicipitaceae with teleomorphic connections to *Torrubiella*.

nutritional resources of their hosts, ultimately killing them and producing more infective conidia for transmission or resting structures for persistence (Figure 1). Fungal activity is strongly influenced by the biotic and abiotic environment; humidity in excess of 95% is required for conidium germination, infection, and sporulation, and the speed of kill is modulated by temperature. Their ecology, physiology, and life cycles are highly variable, reflecting adaptation to overcome environmental limitations and the host's defenses (44, 76).

Species in the order Entomophthorales exhibit a continuum of adaptations, from dispersive actively discharged conidia produced postmortem on the host to the absence of this in species in which continued host activity ensures conidia dispersal prior to death. Species in the Entomophthorales do not produce toxins of importance for the progression of the infection and are obligate pathogens. They are characteristically biotrophic, keeping the host alive until all resources are utilized, and have narrow host ranges among foliar arthropods. This is in contrast to the strategy used by the hypocrealean fungi, which are hemibiotrophic, switching from a biotrophic phase (parasitism) in the hemocoel to a saprophytic phase, colonizing the body after death. Host death is, for the most aggressive strains, usually achieved by the production of secondary metabolites. Conidia are not actively discharged. Species belonging to the order Hypocreales have characteristically broad host ranges when associated with soil-inhabiting arthropods in temperate regions (24, 44, 76). Both the Entomophthorales and the Hypocreales produce resting structures, such as sexual or asexual resting spores, chlamydospores or mummified hosts, to survive in the absence of new hosts.

It is predicted that host-specific pathogens engage in a tight process of coevolution, whereas generalist pathogens interact more diffusely, although this may not always be the case. Changes in host behavior reflect these diverse relationships and enable us to begin to address whether these are pathogen-induced, host-mediated, or incidental. The tendency for evolutionary biologists to embrace the Panglossian paradigm can result in overstating the adaptive significance of a trait. Indeed Poulin (80) suggests that rigorous criteria are needed to decipher whether a behavioral modification is adaptive. For example, the behavior should be complex and increase the fitness of the pathogen or the host. In many cases the experimental evidence to support the hypothesized adaptive nature of many behavioral modifications is absent. This should be a priority for future work.

BEHAVIORAL FEVER

Behavioral fever is the elevation of body temperature in infected insects above that normally occurring in uninfected insects. Infected insects achieve this by seeking out locations in the environment that are at a higher temperature, and the outcome is death or suppression of the pathogen and a delay in the time until death. Fever is a common host response to many pathogens. It is an energetically costly process and is not inevitably beneficial to hosts, but there are many examples in which the

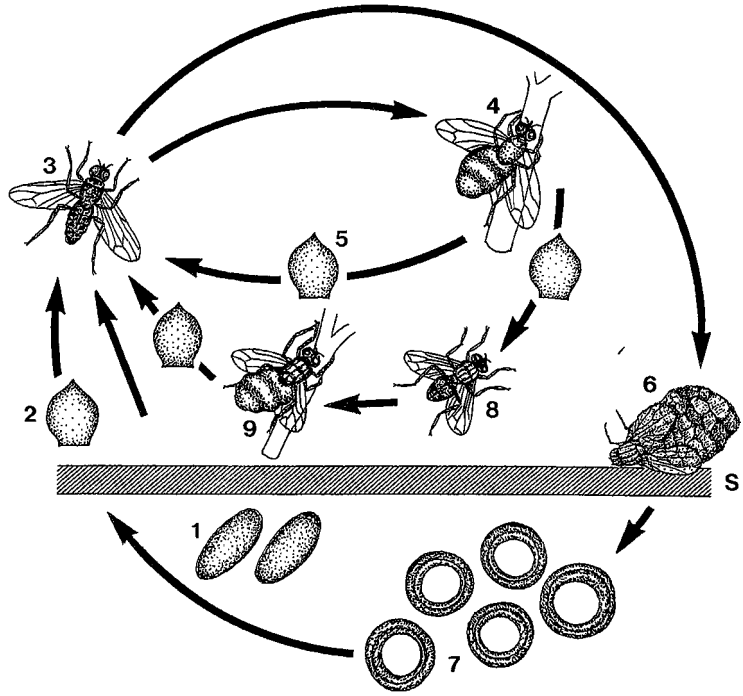

Figure 1 Life cycle of *Entomophthora muscae* in the host *Delia radicum*. Each host-pathogen system exhibits its own peculiarities. For example, some fungi have only one host species, whereas others have many. However, life cycles of entomopathogenic fungi exhibit many general similarities. S = soil surface. 1. Overwintering stage of the host, in this example, pupae in the soil. 2. Infective, asexual conidia are produced by the winter-surviving structures of the fungus and are ready to infect susceptible stages of the host by sticking to the cuticle and penetrating into the hemocoel. 3. Adult flies (in this case the susceptible stage of the host) emerge from the pupae and become infected. 4. After the incubation period the host dies. In the case of *E. muscae* the dead host sticks to the vegetation and conidia are produced from the dead host. 5. These conidia may infect susceptible hosts. These flies die from infection and produce more inoculum, and as a result an epizootic may occur over a period of time. 6. When the number of susceptible hosts decreases (for example, during autumn), some infected individuals do not produce infective conidia upon their death. Instead fungal structures, which can survive a prolonged time without the presence of the host, are produced. In this case flies drop to the soil surface and produce thick-walled resting spores (sexual or asexual). These structures are not themselves infective. 7. The resting spores survive the winter in the soil and produce infective conidia next year (2). 8. The fungus may have an alternative host, often taxonomically related to the primary host. In this case another fly species becomes infected. 9. The alternative host dies after infection and the conidia produced from this host may infect specimens of the primary host. From Reference 19 with permission from Springer Science and Business Media.

onset of fever does suppress pathogens and so reduces or delays host mortality (67). Observations on fungal-infected grasshoppers and caterpillars demonstrated the benefits of raising body temperature because basking at elevated positions reduced pathogen-induced mortality (13, 57). Such a response has been noted in many taxonomically diverse host-pathogen interactions but it is sometimes difficult to distinguish between active behavioral fever and a benefit that is a side effect of the natural thermoregulatory basking behavior of some insect hosts.

A particularly well-studied example of true behavioral fever is that of house flies, *Musca domestica*, infected with *Entomophthora schizophorae* or *E. muscae* (Entomophthorales). Olesen (73) first documented behavioral fever in fungal-infected house flies. In the first few days of infection, house flies seek temperatures in excess of 40°C (104) and benefit from this preference by suppressing the pathogen. Further studies demonstrated that the survival time of infected house flies increased if they were exposed to high temperatures shortly after exposure to fungus inoculum. Interestingly, it appears that the degree of induced behavioral fever was to some extent dependent on fungus isolate.

Field studies on the same host-pathogen system showed that in the first day after infection house flies seek high temperatures, whereas at later stages of infection flies preferred cooler places. Kalsbeek et al. (49) showed that most infected flies captured in cool positions within farmyard barns died within 2 days but infected flies sampled from sun-exposed places took between 6 and 8 days to die after capture, inferring that they were newly infected at the time of sampling. In the same field study, Kalsbeek et al. (49) released additional marked infected flies as well as a marked control group and found that significantly more newly infected flies were located around the heat lamps in the barn.

This phenomenon is associated not only with entomophthoralean fungi but also with species of fungi in the Hypocreales attacking grasshoppers and locusts. Insect species that naturally thermoregulate can maintain body temperatures that restrict pathogen growth without changing their normal behavior (6–8, 45, 46). This effect is enhanced significantly if they also exhibit behavioral fever whereby they change their behavior in direct response to disease challenge by elevating their temperature even higher (7, 10, 46, 75). A recent study (22) demonstrated a direct relationship between behavioral fever and host fitness. Infected locusts, which were allowed to reach body temperatures preferred by healthy hosts but not allowed to fever, took longer to die than infected insects at cooler temperatures. Furthermore, whereas infected locusts at cooler temperatures did not live long enough to reproduce, those achieving fever temperatures did molt, mature, and reproduce, demonstrating a clear fitness benefit.

FEEDING BEHAVIOR

Changes in Food Consumption

Many studies have investigated the effects of fungal infection on feeding by host insects. These studies show that although insects killed by entomopathogenic fungi

often take longer to die than if treated with chemical pesticides, damage to crops is decreased during the disease incubation period because infected insects eat less than healthy ones. Studies evaluating food consumption and infection have focused on phytophagous hosts, including lepidopteran larvae (33, 35, 37, 99), acridids (1, 15, 101), and coleopterans (20, 26). Most studies investigating insect species infected with the hypocrealean fungi, *Metarhizium* sp. or *Beauveria* sp., have demonstrated a significant reduction in feeding as early as 1 to 4 days after inoculation (99). Feeding then decreases through time until death (26). Several studies have shown that the reduction in feeding is associated with dose, e.g., at the highest dose of *Metarhizium anisopliae* var. *acridum* (=*M. flavoviride*), *Schistocerca gregaria* had eaten only as much wheat before death on day 5 after treatment as insects that received the lowest dose had eaten by day 3 (66). Nutritional indices were calculated for lepidopteran larvae infected with *Beauveria bassiana* and *Nomuraea rileyi* to show that weight gain and efficiency of conversion of ingested and digested food decreased at the same time as food consumption (64). Therefore, as infected insects start eating less, they are less able to digest food. It has been hypothesized that reduction in feeding may be due, at least in part, to toxic substances or mechanical disruption by these hypocrealean fungi (14, 88).

Feeding reductions documented for *Entomophaga maimaiga*, *Entomophaga aulicae*, and *Zoophthora radicans*, all of which are entomophthoralean fungi, seem to occur later in disease incubation than observed for hypocrealean infection. Gypsy moth, *Lymantria dispar*, larvae infected with *E. maimaiga* reduced feeding only 2 days before death (37). Spruce budworm larvae, *Choristoneura fumiferana*, infected with *E. aulicae* appeared to assimilate food similarly to healthy insects until 24 h before death and this was similar for diamondback moth, *Plutella xylostella*, larvae infected by *Z. radicans* (35, 103). The delayed reduction in feeding associated with these entomophthoralean pathogens suggests that these more obligate pathogens affect host behavior only near death, thus allowing hosts to feed and grow for as long as possible, ensuring maximum growth and reproductive potential for these pathogens. There is a direct relationship between cadaver size and the number of conidia produced (36). Transmission electron microscopy has shown that these fungi do not invade the hosts' vital tissues until late in the infection process (31) and therefore hosts behave normally until shortly before death. Maintaining normal rates of food consumption and digestion in fungal-infected insects for as long as possible clearly benefits the fungal pathogen because this maximizes the amount of food available for the entomopathogen. The same phenomenon can be observed for insect viruses. Some nucleopolyhedroviruses block ecdysis and pupation by larval lepidopteran hosts to prolong feeding by infected insects, which further increases the amount of host material for the entomopathogen to use (74).

Changes in Feeding Location

In contrast to the extensive literature on changes to food consumption after infection, few studies have been conducted on shifts in feeding location by infected hosts

during disease incubation. In general, the feeding and resting locations of fungal-infected insects do not appear to change throughout most of the disease incubation period and distinctive changes in location occur briefly before death when insects are no longer feeding (see Endgames, below). However, pea aphids, *A. pisum*, infected with *Pandora neoaphidis* were found on the undersides of leaves or had left alfalfa plants and were found in the surrounding habitat more frequently than were healthy aphids (48). Distribution of infected versus healthy pea aphids at lower densities on bean plants was not significantly different (85), although there was a trend toward more infected aphids at mid-height and lower positions on plants (30.0%) compared with healthy aphids (12.5%). The less mobile aphid *Sitobion avenae* on wheat did change feeding height when infected (85). Changes in location of infected *A. pisum* could be a fungal-induced modification for optimizing spore dispersal and transmission. It could, however, also be considered altruistic behavior (kin selection) moving the aphids away to protect progeny and sisters from infection. Aphids moving off plants could even be attempting "suicide" (59), which would decrease chances of infection spreading to other colony members. Thus far, it seems that this behavior is only characteristic of more mobile aphids. Infected aphids are alive for several days between infection and death, and it would be interesting to evaluate when during disease incubation these movements begin. These changes in feeding behavior may be further examples of summit disease (see Endgames, below).

REPRODUCTIVE BEHAVIOR

The fitness of an individual is directly dependent on the number of viable offspring produced, and both the pathogen and the host adopt strategies to maximize reproductive output. Therefore, it is not surprising that a number of studies report modifications of host reproductive behavior ranging from direct effects on fecundity to changes in the production of and response to sex pheromone.

Direct Effect on Fecundity

Reduction in fecundity can increase pathogen fitness by diverting host resources such as energy to the pathogen. Studies on both hypocrealean fungi (4, 9) and entomophthoralean fungi (4) have shown that infected hosts produce fewer progeny. The total reproductive output of fledgling desert locusts, *S. gregaria*, infected with *M. anisopliae* var. *acridium* (Hypocreales) was lower than that of uninfected individuals, although in the first few days of infection the locusts produced more eggs (9). From an evolutionary perspective, early onset of reproduction is a sensible strategy because it ensures that individuals realize part of their reproductive potential. In the same study the fecundity of mature infected locusts was also assessed and interestingly the pathogen had no effect on reproductive behavior. The authors suggest that this is due to effects on the synthesis of juvenile hormone. Production

of juvenile hormone is linked to oocyte maturation and young fledgling locusts do not produce the hormone until 3 to 4 days after emergence. Infection is thought to induce rapid synthesis of juvenile hormone and this likely has a greater effect on newly emerged adults, in which concentrations of the hormone are nil or low, compared with older adults (10 to 12 days), which already have high hormone levels (9).

A reduction in reproductive output was observed for pea aphids, *A. pisum*, infected with *B. bassiana* (Hypocreales) and similarly for pea aphids infected with *P. neoaphidis* (Entomophthorales). In both cases fecundity was significantly decreased within 24 h of infection, and the total number of nymphs produced over the infection period was significantly lower than that of uninfected aphids (4). It seems that these taxonomically distinct fungi disrupt the reproductive output of the host aphid in a similar way, but consideration of the life histories of these fungi would suggest that the actual mechanisms employed are likely different. *P. neoaphidis* requires about 4 days from initial infection until host death, and like most obligate entomophthoralean fungi it is essential for the host to remain alive until just prior to sporulation. In contrast, *B. bassiana* takes approximately 6 days to kill the aphid, often by the production of secondary metabolites; it then enters a saprophytic phase, sporulating a few days post death. It is conceivable that *P. neoaphidis* is inducing the reduced reproductive rate and therefore diverting host resources for its benefit. However, the reduction in reproductive rate observed for *B. bassiana*-infected aphids may simply be a result of the fungus indiscriminately invading the host's tissues and producing secondary metabolites that interfere with nymph production. These hypotheses require further exploration.

Baverstock et al. (4) also assessed the reproductive rate of the progeny produced from infected and uninfected host aphids and found no effect of infection. These fungi directly affect production of nymphs by the host aphid but have no effect on the fitness of the host's progeny.

In the case of adult carrot flies, *Chamaepsila rosae*, infected with *E. schizophorae* the effect on fecundity is more indirect. Normally, female carrot flies deposit their eggs on the ground near the base of food plants such as carrots, and after hatching the larvae move into the soil, where they eat small roots. However, infected flies seek elevated positions such as the top of trees and shrubs in the hedgerows, and sporulating cadavers are found several meters aboveground (18) (Figure 2*d*). Infected female flies deposit their eggs at this height (Figure 2*e*). These eggs are fully fertile and develop normally, but the emerging larvae are unable to find appropriate food plants and are therefore unlikely to contribute to the carrot fly population. Further observations showed that the fungus also induced changes in egg-laying behavior particularly when female flies were infected shortly after their emergence from pupae, thus when eggs were not yet developed. These females consistently laid their fertile eggs at elevated positions. Whether this behavioral modification entails any adaptive significance to either the pathogen or the host is difficult to speculate and further investigation is required.

Increase in Sexual Attractiveness of Infected Hosts to Mates

The exposed abdomen, which is commonly seen among adults of some insect species (especially flies) killed by entomophthoralean fungi (Figures 2*a–c*), is apparently highly attractive to individuals in the host population seeking a mate for copulation. Male house flies, *M. domestica*, are thus significantly more attracted to fungus-killed females than to uninfected females (65). It is assumed that this increased attractiveness is a result of the increase in size of the swollen abdomen of the infected females. However, a further study has demonstrated that even significantly smaller infected females are more attractive to males than are larger infected female flies (108). Furthermore, sex pheromone production by young, infected female house flies is reduced compared with uninfected house flies (108). Therefore, the occurrence of increased attractiveness of infected female house flies to male flies cannot be explained by size or sex pheromone production alone. Other visual or chemical cues are likely to account for the observed phenomenon; perhaps the fungus is producing a semiochemical that attracts males. Elucidating the exact mechanisms involved requires further research.

The increased attractiveness is undoubtedly advantageous to the fungus for a number of reasons. First, males may themselves receive infection during physical contact with sporulating cadavers. Second, males trying to copulate with the cadavers may transmit conidia to uninfected females that they subsequently copulate with (105). For the fly, this behavior is disadvantageous. Not only do more individuals die from infection but females lay fewer eggs after mating with infected males compared with females mating with uninfected males (105).

Response to and Production of Sex Pheromones

Female *P. xylostella* moths produce sex pheromone to attract males, which respond by pre-mating wing fanning. When females were infected by *Z. radicans*, release of pheromone was significantly reduced compared with uninfected females, but only within 24 h of death (81). The response of infected male *P. xylostella* was also significantly reduced compared with uninfected moths 2 days after infection and by 3 days after infection no infected males responded at all and were dead on day 4 (81). These pre-mortality effects, which are thought to be a side effect of fungal tissue invasion, only manifest quite close to the time when the insect dies and reflect the lack of disruption of normal host behavior by entomophthoralean fungi prior to host death. As the normal life span of moths in the field is only about 5 days (34), it is to the benefit of the fungus to keep the insect alive as long as possible because it cannot develop saprophytically on a cadaver and needs 3 to 4 days to kill.

SOCIAL BEHAVIOR

Eusocial insects (all ants and termites; many bees and wasps) are similar to solitary insects in many aspects of life history, but fitness in eusocial insects is the result of cooperative effort (68). Therefore, any behavioral response to a pathogen will have

fitness implications beyond the individual and will be more complex to decipher in terms of costs and benefits to the host and pathogen.

Despite this complexity there have been many reports of host-mediated behavior reducing pathogen transmission within colonies. Host behaviors observed include increased grooming, increased nest cleaning, secretion of antibiotics, pathogen avoidance, dispersal of infected individuals, and relocation of the entire colony.

Increased grooming in response to fungal pathogens has been documented widely in both solitary and eusocial insects (72, 92). Removal of fungal conidia by allo-grooming and mutual grooming can be highly effective. The number of *B. bassiana* conidia on the integument of both larval and adult red imported fire ants, *Solenopsis invicta*, was significantly reduced by grooming (72). Similarly, *Cordyceps* spores were removed by another species of ant, *Cephalotes atratus*. Although an increase in grooming behavior would appear beneficial to the host in terms of reducing infection, inoculum could be incidentally disseminated to conspecifics within the nest (52). Infective propagules that are removed by grooming with mouthparts are not ingested but are stored in an infrabuccal cavity within the buccal chamber, where they are discharged as a pellet. Infection can occur if inocula germinate and penetrate through the buccal chamber, but it has been hypothesized that the labial gland, which produces secretions rich in chitinases, has a fungistatic function (25, 72).

Consideration of hygienic behaviors can be extended beyond grooming to encompass nest cleaning. Most colonies provide conditions that favor microbial growth: high humidity and stable temperatures. Furthermore, pathogen dissemination could be high within colonies because social insects often live at high densities and in close proximity to one another. However, social insects employ a number of hygienic behaviors to reduce transmission within the colony. Necrophoresis or cadaver removal is a common behavior of social insects in response to most pathogens and this could of course benefit intercolony transmission. Imported red fire ants, *S. invicta*, have also been observed to bury nestmates infected with *B. bassiana*, and this reduces transmission (78). Ants also use antimicrobial secretions that are sprayed from the gaster over the brood. The presence of *B. bassiana* within the nest correlates with an increase in the concentration of these antimicrobial substances, and so it can be inferred that release of these secretions is induced by the pathogen (72).

In some circumstances social insects avoid areas of high inoculum density within the nest or establish new nest sites (72). The benefits to the host are clear in terms of reducing contact with the pathogen but there are undoubtedly costs in moving. Leaf-cutting ants living in high densities in colonies had much greater resistance to *Metarhizium anisopliae* infections than would be predicted, and apparently at a low cost (42). Hygienic behaviors and pathogen avoidance are cooperative behaviors that reduce contact and transmission of pathogens, increasing the inclusive fitness of host insects, but the pathogen is not acting directly on the individual eliciting the behavior. However, a well-documented behavioral alteration of social insects infected with fungal pathogens is dispersal from the colony. This has

been termed "adaptive suicide" (60), but this phrase is contentious not just because of the anthropomorphic connotations it implies but also because the process does not necessarily increase the inclusive fitness of the host (79). Dispersal of infected individuals undoubtedly removes the pathogen from the colony but it is likely that this would happen through nest cleaning anyway. Therefore, unless transmission is so rapid that nest cleaning would be ineffective, the inclusive fitness would be unaffected (72). Indeed, increased dispersal behavior may ultimately benefit the pathogen through increased transmission. There have been many observations of fungal-infected ants dispersing to elevated positions, which is characteristic of summit disease and is discussed below.

Behavioral alterations of social insects can have implications for the fitness of the entire social group. Therefore, the task of unraveling the adaptive nature of host-altered behavior in social insects is challenging. This group of insects presents a unique situation in which to study host-pathogen relationships from a behavioral perspective. The applied aspects of controlling or conserving such insects should promote further research in this area.

DEFENSIVE REACTIONS

Insects employ various defensive strategies in response to attack from predators and parasites ranging from crypsis to dramatic escape behaviors. Studies on insect defensive reactions have concentrated on the benefits to the prey or host in response to predators or parasitoids. A few studies describe defensive behaviors induced in response to pathogens. In general, defensive reactions of hosts to pathogens are considered from an immunological perspective. However, recent studies indicate that termites (*Reticulitermes flavipes* and *Zootermopsis angusticollis*) detect the presence of conidia of the fungal pathogen *M. anisopliae* and exhibit a striking vibratory display (82). Nestmates detecting the signal through the substrate increase the distance between themselves and spore-exposed termites, thus escaping infection. It is conceivable that the selection pressures to develop such alarm behaviors in response to pathogens are higher in subterranean environments where pathogen density is high and particularly in colony-forming insects where high host densities would be optimal for rapid pathogen transmission.

Host behavioral changes can be detrimental to the pathogen if the host is less able to escape predation or if greater movement makes them more apparent to predators prior to death. Arthurs & Thomas (2) found that *S. gregaria* locusts infected with *M. anisopliae* increased locomotion within 3 days of infection (11 days to kill), potentially making them easy targets for predators. Later in infection they became sluggish and less able to escape predation. Whether this is altruistic behavior on the part of the locust or a side effect of infection is unknown but could result in reduced pathogen fitness.

Pathogens can benefit from the defensive behaviors of infected individuals but there is also the potential for the pathogen to manipulate defensive behavior

to increase the advantage. Pea aphids infected with *P. neoaphidis* become less responsive to alarm pheromone produced by conspecifics (84). As a result, infected aphids could be more susceptible to attack by predators and this would reduce the pathogen density. However, aphid-specific predators do not generally consume fungal-infected aphids (77, 86). The escape response of pea aphids commonly includes dislodging from the plant, and infected aphids are less able to recolonize plants. Therefore, it is advantageous to the pathogen to prevent infected aphids from responding, ensuring a greater number of infected cadavers remain on the plant and thus benefiting pathogen transmission to other foliar-feeding aphids. Aphids infected with *P. neoaphidis* continue to produce alarm pheromone and elicit a response in neighboring aphids. Again, this is advantageous to pathogen transmission because host movement enhances transmission (29, 32, 77, 86).

Interestingly, aphids infected with the generalist pathogen *B. bassiana* continue to respond to alarm pheromone but do not produce it (83). It is hypothesized that host-specific fungi such as *P. neoaphidis* modify the behavior of their hosts to increase their transmission and subsequent fitness. The intimate association of such fungi with their host would seem to drive such adaptations. However, the selection pressure on more generalist fungi, such as *B. bassiana*, to manipulate individual host species is likely to be minimal because such fungi have diffuse relationships with many hosts and can also persist saprotrophically.

In contrast, because of the broad host range of *B. bassiana*, it is in the interest of potential hosts to avoid contact with the pathogen. This behavior is seen in termites that recognize and avoid conspecifics contaminated with virulent isolates of *M. anisopliae*, although they show no response to contamination with less virulent isolates of *B. bassiana* (69, 93). It is possible that termites can discriminate fungal species or even isolates that are pathogenic from those that are not. Alternatively, the interactions may represent new associations in evolutionary time and so avoidance behavior has not yet been selected for. This would explain the inability of the parasitoid *Cephalonomia tarsalis*, which attacks grain beetles, to identify and avoid *B. bassiana* conidia or infected beetles. Although the beetle and parasitoid are coevolved, the fungus (*B. bassiana*) is a new addition to the system (introduced as an augmentative biological control agent). *B. bassiana* not only competes with the parasitoid for host resources but can also directly infect the parasitoid (54). Intraguild interactions between insect and pathogen natural enemies are highly complex. When the insect natural enemy is susceptible to the same pathogen as its host, then it is at risk of direct infection as well as competition for a resource and may alter its behavior in response to this. For example, the predatory bug *Anthocoris nemorum* did not forage or oviposit on leaves contaminated with conidia of a virulent isolate of *B. bassiana* (originally isolated from *A. nemorum*), which could represent a selective fitness advantage to the bug (N. Meyling, personal communication). Interestingly, the bug was unable to detect and avoid inoculum on the soil, which is not a natural foraging site for the bug. Regardless of direct susceptibility, some parasitoids detect and avoid infected hosts (27, 61) and some decrease the susceptibility of their host to subsequent fungal attack,

an example of the parasitoid hijacking its host's physiology to avoid competition (23, 28). In contrast, there are also examples in which parasitoids do not alter their attack behavior until the host is close to death (11). *Aphidius ervi*, although not directly susceptible, competes for aphid hosts with the fungal pathogen *P. neoaphidis*. It is unable to detect *P. neoaphidis*-infected aphids and enters and forages normally in infected aphid populations (3). The interactions within this guild are highly coevolved and one would hypothesize that the parasitoid would avoid competition with the pathogen. The reason that it does not could be because the selection pressure in nature is not strong enough at the population level, given that the parasitoid is not directly susceptible, or because the fungus (characteristically for entomophthoraleans) is "cryptic" within the aphid population. Entomopathogenic fungi that kill the host faster than the parasitoid always win a competitive intrahost interaction and can benefit from such an interaction because parasitoid (and predator) foraging enhances fungal transmission.

Predator avoidance is another classic defensive behavior and could explain why termite (*Macrotermes* sp.) alates infected with the ascomycete fungus *Cordycepioideus bisporus* have rarely been observed in the field (70). Infected cadavers can only be found at the time of alate swarming (the rainy season), and the pathogen appears to induce behavioral modification in the host, resulting in alates moving under stones, where they die alone or in small groups (Figure 2*f*). Fungal structures such as ascospores are visible on the termite cadavers. Obviously, the fungus benefits from the moist microclimate in this semi-arid environment, but the hidden place also provides some protection against scavengers. The transmission mechanisms of the fungal pathogen from these cryptic sites to uninfected individuals are unknown.

PHOTOTROPIC AND GEOTROPIC RESPONSES

Few studies have specifically addressed relations between light and behavior during disease incubation. Results are somewhat equivocal because effects of light (positive phototrophism) are not easily separated from potential effects of gravity (negative geotrophism) and, to some extent, temperature. Hence, we deal with phototropic and geotropic responses together (the response to temperature is considered in Behavioral Fever, see above).

Common armyworm larvae, *Pseudaletia separata*, normally spend the day in the soil, where it is dark, and they leave the soil to feed at night. However, when infected with the entomophthoralean fungus *E. aulicae*, many larvae spend the day in the light instead and even move vertically upward on plants (71). Therefore, their normal behavior of avoiding light is completely changed when infected. Such a change in behavior that exposes larvae to visual predators could be risky, but it could also benefit the fungus because larvae die in exposed locations. Similarly, larvae of sciarid flies normally live in the upper soil layers. However, when a disease caused by *Erynia sciariae* is progressing, the larvae move to the top of the

soil or growth medium to die after which the fungus sporulates (Figure 2*g*). This behavior appears to benefit the fungus because infective conidia are dispersed on the soil surface, where they infect small larvae moving into the soil. More in-depth studies of these systems are necessary before researchers can determine whether these behavioral changes benefit the host or the pathogen and whether they are mediated by a phototrophic, geotrophic, or thermal response.

Numerous studies have documented that entomophthoralean fungi kill insect hosts during the late afternoon or evening (62, 71). Elegant experiments have been conducted to document associations between light cycles and timing of death of fungal-infected insects. Periodicity of death was found for six combinations of entomophthoralean fungi and aphid species maintained at 16:8 (light:dark). Most deaths were associated with photoperiod and occurred during periods of light (62). *Entomophthora planchoniana* and *P. neoaphidis* each caused death within 4-h intervals, with peaks at 8 to 9 and 14 h after dawn, respectively. *Zoophthora radicans* caused some deaths throughout the 24-h light cycle but with a peak at 12 hours after dawn, and for the remaining three species peak mortality was less distinct. Studies on house flies, *M. domestica*, infected with *E. muscae* have documented death 0 to 5 h before the onset of darkness, but deaths of flies held in constant dark after infection did not follow this rhythm (53). However, flies exposed to 12:12 (light:dark) for 3 days after infection and then maintained in the dark displayed periodicity in death. This suggests that this circadian rhythm is governed by a gated biological clock that is most probably controlled by the fungal pathogen. These examples demonstrate that fungi can kill hosts at specific times in synchrony with environmental conditions that are thought to favor pathogen transmission. Death in the late afternoon or evening ensures that cadavers sporulate under the humid conditions at night to infect new hosts. Rapid transmission is required because the longer a cadaver is present in the field, the greater the chance that it will fall, be overgrown with saprophytic fungi, or be eaten by a scavenger.

ENDGAMES

Entomopathogens usually cause host death at the end of an infection cycle. At this stage, the pathogen depends on efficient transmission to maximize its fitness or, in the absence of potential hosts, on effective persistence until host populations increase again. Entomopathogenic fungi are often reliant on their hosts for spore dispersal and employ diverse strategies, including host manipulation, to maximize transmission efficiency. However, infection by pathogens may simply result in the host dying in situ, where it normally exists. This is true, for example, of beetle pupae dying in soil after infection by hypocrealean fungi such as *M. anisopliae* or *B. bassiana* (Figure 2*h*) but is relatively uncommon in the Entomophthorales, although it can occur in some cases when the resting-spore stage is produced or when soil-inhabiting hosts such as mites are infected. In these cases, the host apparently does not alter its behavior other than to become sluggish immediately before death.

Cabbage flies infected by *E. muscae* become sluggish and move to the soil surface, where they produce resting spores for winter survival. Resting spores are only produced after midsummer in the field, suggesting a strong influence of declining day length (102). Such a death may thus indicate a high degree of adaptation or coevolution of the pathogen to its host, because the host may be in the optimal site and at the optimal time for the pathogen to survive and infect future hosts.

In many cases the final interactions, or endgames, between a host and pathogen involve complex behavioral modifications such as the infected insect seeking an elevated position where wind currents can effectively disseminate conidia. Elevation seeking by insects at late stages of infection is a common phenomenon that was recognized by early insect pathologists who noted that diseased lepidopteran larvae, such as *Lymantria monacha* (the nun moth), infected with baculoviruses migrated to the tops of trees where they died (94). This host-altered behavior was named "Wipfelkrankheit" or "Wipfelsucht" (meaning tree top disease in German) for viral diseases (41) and "summit disease" for fungal diseases (24, 57, 106).

Classic Summit Disease

In their final hours many hosts infected with Entomophthorales climb to an elevated position to die (Figure 3*a–d*). This is, for example, true of grasshoppers infected with *Entomophaga grylli* but also occurs in other systems, such as infection of true armyworm larvae, *Pseudaletia unipuncta*, with *Furia virescens* (98). A particularly striking example of summit disease is sugarbeet root aphids, *Pemphigus betae*, infected with *P. neoaphidis*. These root-feeding aphids move up to the foliage of plants just prior to death and die and sporulate in a position that ensures horizontal transmission (39).

It is generally assumed that summit disease benefits the fungal pathogen by placing the host in sites conducive to widespread dispersal of the infective conidia, usually by wind. However, in some cases summit disease results in an increase in the host's inclusive fitness because the host moves away from clonal or closely related individuals. Furthermore, summit behavior can result in greater predation or scavenging on infected hosts, thereby reducing the dissemination of the pathogen. So it is sometimes difficult to ascribe the benefits of summit disease to either the host or the pathogen.

The exact mechanisms leading to elevation seeking are unknown and experimental studies demonstrating the results of these behaviors on survival of host species and pathogen species are few. It has been postulated that the fungus affects the host's nervous system (5) and releases compounds that induce the behavior or impair the respiratory system (50), but at this point few experimental studies have been done on these behaviors (24). However, observations of elevation seeking by fungal-infected insects are well documented and the single term summit disease does not do justice to the diversity of behaviors that result in this phenomenon. In this section we review these host-pathogen interactions and in some cases suggest alternative, more precise terminology.

Summit Disease of Aquatic Hosts: Flotation

Flotation is a rare type of interaction in which an aquatic host infected with the conidial stage floats on the water surface. Mosquito larvae and pupae killed by infections with *Erynia aquatica* (Figure 3*f*) are buoyant and the conidiophores grow out into the air from the cadaver and discharge conidia onto nearby mosquito pupae and emerging adults (97).

Host Attachment to Substrate by Fungal Holdfasts

Behavioral alterations leading to summit disease are almost always accompanied by other adaptations such as attachment of the host to the substrate by production of specialized fungal structures formed from hyphae, rhizoids, or glue-like materials secreted by hyphae. However, the formation of fungal holdfasts may exist in conjunction with modifications in host behavior such as elevation seeking. In either case, hosts attached by fungal structures may persist on substrates such as leaves, twigs, or even window glass for days or even weeks. This appears to benefit the pathogen because the host remains firmly fixed in a position that increases the chances of new hosts encountering the pathogen through feeding, resting, or mating. The fungal holdfasts also ensure the pathogen persists in the infected host, often surviving adverse environmental conditions (such as sunlight) and resulting in wide distribution of conidia. Fungal holdfasts are employed by many groups of fungi and take various forms depending on the particular fungal species. We consider from a functional approach a number of subcategories of the fungal holdfast type.

The first category is the most basic (basic fungal holdfasts) in which the fungal holdfasts are not highly differentiated from conidiophores. An example of this type is shown by the interaction between the true armyworm, *P. unipuncta*, and the conidial form of *F. virescens*. The larvae are weakly held to grass stems by the growth of apparently undifferentiated conidiophores and the larval prolegs (Figure 3*d*). *P. unipuncta* larvae usually rest on the ground beneath grasses during the day but larvae infected with *F. virescens* that produce conidia exhibit summit behavior, apparently always dying toward the tops of grass stems. Interestingly, larvae that produce *F. virescens* resting spores do the exact opposite and are attached to the soil by rhizoids (98) (Figure 3*e*).

The next category involves the production of structurally more complex fungal structures called rhizoids (rhizoidal holdfasts). Fungal rhizoids are differentiated from conidiophores. There are many examples of this subcategory, for example, aphids infected with *P. neoaphidis* or *E. planchoniana* are strongly attached by rhizoids to leaves. Infected aphids die in situ at their feeding sites, still in their aphid colonies, benefiting transmission of the fungus when conidia are explosively discharged around each host cadaver (21) (Figure 3*g*). Fungal rhizoids also firmly attach various rhagionid flies infected with *Erynia ithacensis* to the undersides of leaves of woody plants (51) (Figure 3*h*). Other examples of insects firmly attached by fungal rhizoids are adult Trichoptera held firmly beneath logs

and stones around rivers by rhizoids of *Erynia rhizospora* (Figure 4*a*), adult Culicidae infected with the resting-spore stage of *E. aquatica* held to moist logs near snowpools (Figure 4*b*), and Homoptera such as *Spissistilus festinus* held firmly to soybean plants by rhizoids of *Erynia delphacis* (Figure 4*c*).

A modification of the rhizoidal holdfast appears restricted to dipterans infected with *E. muscae* or *E. schizophorae* whereby rhizoids are restricted to the host's proboscis, growing out of the labellae and attaching the host to smooth surfaces such as leaves, wood, and even window glass (Figure 4*d*). Along with the rhizoids, glue-like material strongly fixes the host to the substrate (Figure 4*e*). Many questions remain concerning the mechanisms involved in these interactions, such as how such hyphal differentiation occurs and whether it is driven by the other hyphal bodies or by cues from the proboscis.

Host Attachment to Substrate by Host Structures

Some fungi do not produce rhizoids, but the host is held in situ by host structures alone, namely the legs or mouthparts (mandibles or stylets). The pose of the dead insect is generally characteristic of the pathogen species involved. For instance, *E. grylli*-infected grasshoppers (Figure 3*a*) and *E. scatophagae*–infected yellow dung flies (Figure 3*b*) seek elevated positions on grasses or other vegetation and cling tightly (death grip) with their legs (96). Tipulids infected with fungal species from the genera *Eryniopsis* and *Entomophaga* also attach to grasses with their long legs, often overhanging water (Figures 5*b*,*c*).

Attachment by mandibles is limited to Coleoptera (Cantharidae) infected with Entomophthorales. Adult beetles infected with *Eryniopsis lampyridarum* or *Entomophthora* sp. grip plants, particularly the flowers, by biting the plant with their mandibles (Figures 4*f–h* and 5*a*). Again no fungal holdfasts are involved. This attachment method is long lasting and this benefits the fungus because many living hosts are present on the flowers, feeding and mating (Figure 4*g*,*h*), and so readily contact conidia. It is noteworthy that the same attachment method is employed by two different fungal species in distinct genera and seems to reflect the efficiency of fungal transmission between cantharids.

Attachment of the host via the stylet is typified by aphids infected with the conidial stage of *Neozygites fresenii*. Infected aphids generally die attached to the undersides of leaves or at the base of pine needles held only by their stylets embedded within the leaf tissue (Figure 5*d*). The aphid's body becomes a source of explosively discharged conidia, which disperse rapidly over a wide area including the leaf surface, infecting other aphids in the colony (95). The fungus benefits from the higher humidity of the lower leaf surface or pine needle base.

Hypocrealean fungi do not produce specialized holdfasts but some species have evolved alternative mechanisms of ensuring the cadaver is held in position for optimal transmission. For example, grasshoppers dying from infection by *Sorosporella* sp. become attached to foliage at both ends of their bodies by fungus-induced vomiting and defecating (89). *Cordyceps* species usually kill their hosts belowground

but then produce a large and highly visible fruiting structure aboveground to aid dispersal (Figure 5*i*). Some hypocrealean species that infect scarabaeid larvae produce exudates from their fruiting bodies that attract surrounding insect fauna that vector the fungus to other hosts (24).

Alternative Endgame: Host Liquefaction

Transmission of fungal conidia produced by entomopathogenic fungi often relies on attachment of the host to the substrate, sometimes at an elevated position within the environment. However, in contrast to this is a poorly understood type of endgame in which insects infected with the resting-spore stage of the fungus liquefy. The resting spores are thus released from the host into the environment. This is seen in aphids infected with the resting-spore stage of several *Neozygites* species (Figure 5*e*) and lepidopteran larvae infected with the resting-spore stage of *Erynia gammae*. Liquefaction ensures resting structures enter the soil from which they can germinate and infect hosts in subsequent seasons. *Tarichium rhagonycharum*–infected cantharid beetles are attached to the vegetation by rhizoids before the cadaver dissolves and resting spores are washed to the ground. It is difficult to envisage the benefit to the fungus of employing this dual strategy of both liquefaction and rhizoid production. Certainly this is an aspect requiring further research.

There are examples in which infected individuals from the same population die in different positions depending on whether they produce actively discharged conidia or resting spores. For example, aphids on plum trees are attacked by a number of entomophthoralean fungi and some individuals die on leaves producing actively discharged conidia and some die hidden in crevices in the bark and produce resting spores. It could be argued that this is the ideal site for infection of the next generation of nymphs from eggs laid around the leaf buds and is an adaptation benefiting the fungus. However, resting-spore production is also associated with older insects and it may be that their normal behavior is to move to the bark (24).

Ultimate Endgames: Utilizing an Active Host for Transmission

One of the most interesting interactions and possibly the most highly evolved is seen in the entomophthoralean fungal genera *Strongwellsea* and *Massospora*, and in certain species of *Entomophthora*, *Erynia*, *Entomophaga*, and the hypocrealean fungus *Lecanicillium longisporum*. In these cases the formation of conidia and sometimes resting spores occurs in or on the still-living host, which serves to disperse the conidia or resting spores to new hosts and in the environment.

In the case of *Strongwellsea*, the hosts are adult Diptera and the fungus growth is largely confined to the abdomen. One or two (in one case three) open wounds develop on the abdomen of infected flies, and conidia are discharged through the holes (Figure 5*f*,*g*). Infected flies remain active and seem to continue behaving normally.

Massospora species are limited to various species of cicadas. When *M. cicadina* infects periodical cicadas (*Magicicada* spp.), the hosts become living disseminators

of both the conidia and resting spores, though both spore forms do not occur in the same host (16). In both the conidial and resting-spore states living cicadas continue to fly and interact with other cicadas while their abdomens are filled with fungal material. The caudal segments of the abdomen are sloughed off and conidia or resting spores are dispersed (Figure 5*h*).

In the case of calyptrate flies infected with an *Entomophaga* sp., exudates from the disintegrating abdomens of infected flies adhere the still-living fly to the soil of river beds. As the flies struggle, more body contents are released that anchor the fly, release infective propagules, and also attract further flies, which subsequently become infected (43).

In some cases mirid bugs such as *Lygus communis* var. *novascotiensis* infected with *Entomophthora erupta* remain alive while conidia are being discharged (17). MacLeod et al. (56) considered this important in the spread of the pathogen. *Erynia kansana*–infected dipteran adults were observed walking and flying with missing abdominal parts similar to the case with cicadas infected with *Massospora* (55). *Entomophthora thripidum* (87) also produces actively discharged conidia from still-living hosts to aid dispersal. In one example from the Clavicipitales, aphids infected by *L. longisporum* showed outgrowth of fungal hyphae and associated sporulation around the tarsi of the host prior to death (107).

SUMMARY: ALTERATIONS TO HOST BEHAVIOR—WHO BENEFITS?

The diversity of pathogens and hosts is undoubtedly reflected in the variety of behavioral interactions observed. In many cases it is difficult to predict whether host-altered behavior is beneficial to the host or to the pathogen. Indeed, a behavioral change may enhance pathogen transmission or host defense in one relationship but not in another. Host-specific obligate pathogens, such as the Entomophthorales and a few species of Hypocreales, seem more likely to alter host behavior than generalist facultative pathogens, as typified by most Hypocreales. The host-specific fungi appear to modify the behavior of their hosts to increase their fitness and this has been exemplified by the studies described. The intimate association between these fungi and their hosts would drive such adaptations. However, the selection pressure on more generalist fungi to manipulate individual host species is minimal likely because such fungi have diffuse relationships with many hosts and can persist saprotrophically.

The situation is complex and dynamic, changing in response to the evolutionary arms race between pathogen and host. It is undoubtedly the case that host behavior affects fundamental aspects (virulence, propagation, transmission) of the biology of pathogens and ultimately their evolution. However, it is important to avoid the temptations of assigning an adaptive function to all host-altered behavior without thorough consideration and understanding of the underlying proximate mechanisms. Most studies on pathogen-host interactions focus on either host physiology

or behavior in isolation. A priority for future research is a combined approach to elucidate the underlying proximate causes (physiological, immunological, and biochemical) that result in specific behavioral modifications. Recent studies, particularly on insect semiochemistry and behavior, have begun to address these aspects (4, 83, 84, 108). Further understanding of these behavioral interactions will progress evolutionary understanding of host-pathogen relationships and also contribute to novel methods of pest control.

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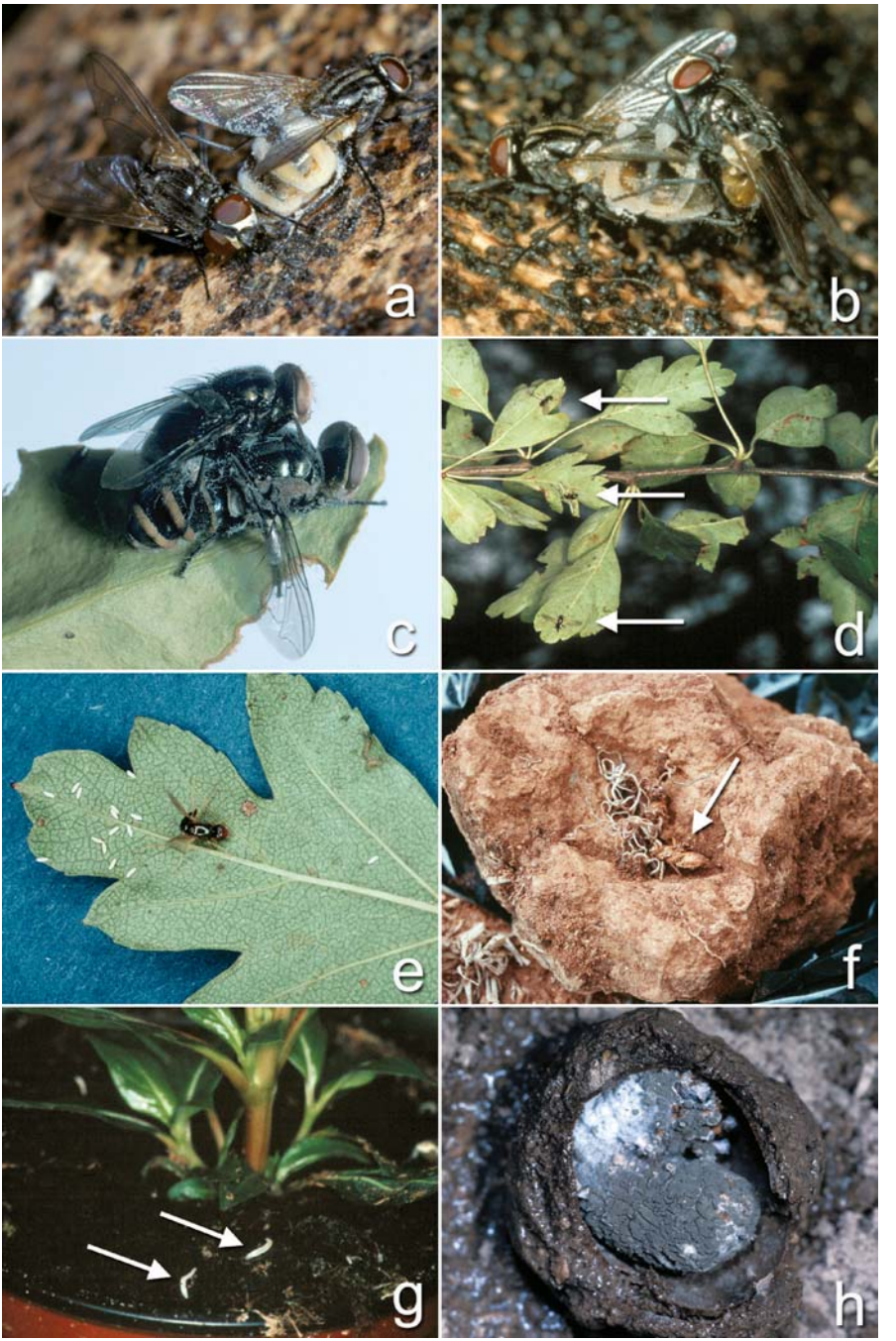

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Figure 2 (*a*) Male house fly (*Musca domestica*) probing a dead female house fly killed by *Entomophthora schizophorae*. (*b*) Live male house fly copulating with dead female house fly killed by *E. schizophorae* infection. (*c*) Male secondary screwworm fly (*Cochliomyia macellaria*) attempting to copulate with dead female infected with *Erynia bullata*. (*d*) Carrot flies (*Chamaepsila rosae*) killed by *E. schizophorae* attached to leaves in a hedge at a height of four meters. (*e*) *E. schizophorae*-infected female carrot fly that has abnormally deposited eggs at a height of four meters. (*f*) Adult alate termites (*Macrotermes* sp.) killed by *Cordycepioideus bisporus* found under stones in a semi-arid area, Kadjiado, Kenya. (*g*) Sciarid fly larvae dying from infection with *Erynia sciariae* at the top of the growth medium. (*h*) Green june beetle larva (*Cotinus nitida*) killed by *Metarhizium anisopliae* in its pupal cell.

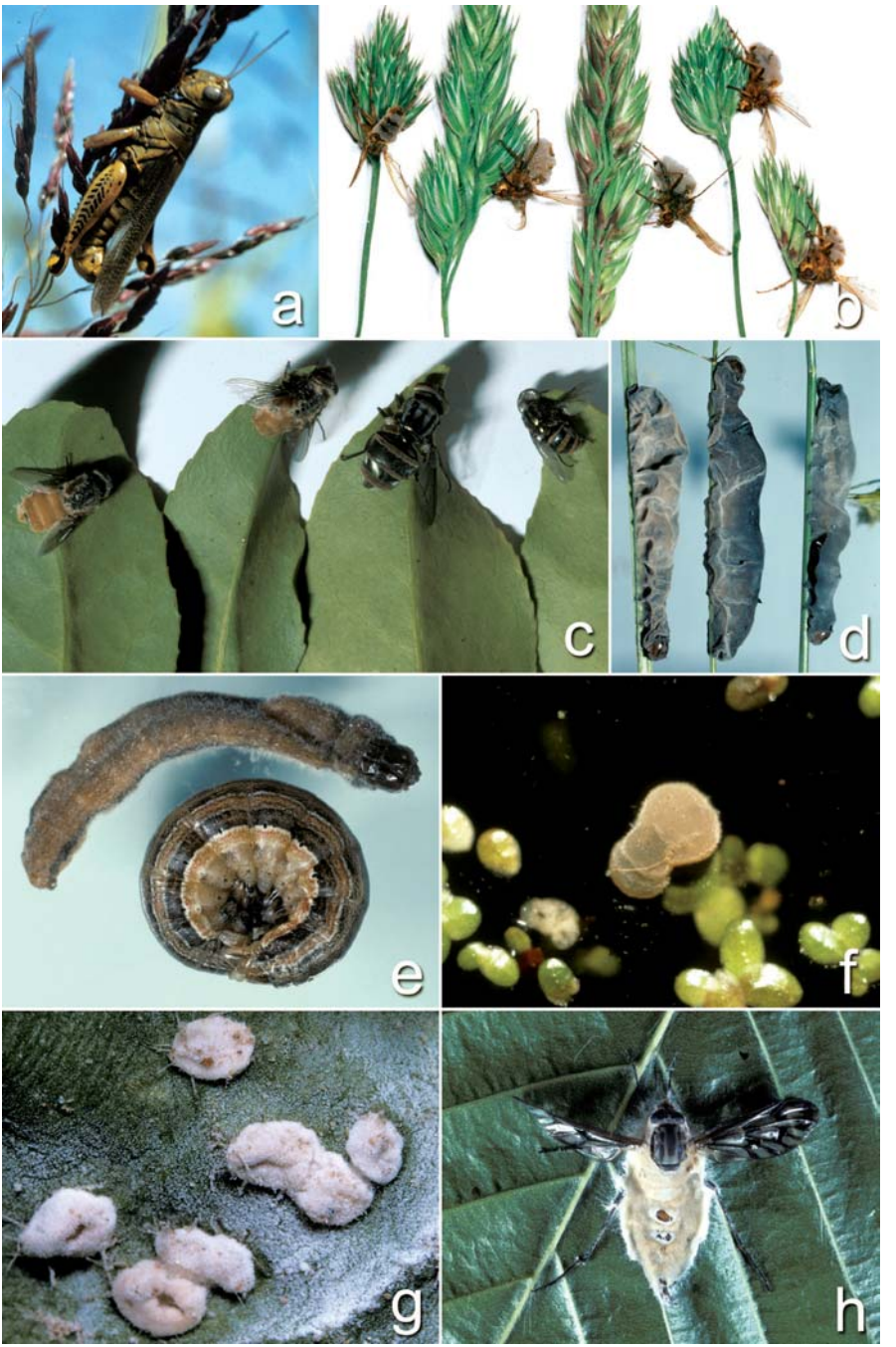

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Figure 3 (*a*) Differential grasshopper (*Melanoplus differentialis*) infected with resting spore stage of *Entomophaga grylli* showing summit behavior. The dead host is held to the grass only by its prothoracic and mesothoracic legs. (*b*) Dead yellow dung flies (*Scatophaga stercoraria*) infected with *Entomophthora scatophagae* clinging by their legs to orchard grass seed heads. (*c*) Secondary screwworm flies (*C. macellaria*) infected with *E. bullata* attached by rhizoids to the tips of *Euonymous* leaves. (*d*) True armyworm larvae (*Pseulaletia unipuncta*) killed by conidial stage *Furia virescens* infections on grass stems. (*e*) Larva of *P. unipuncta* killed by resting-spore stage of *F. virescens* (*top*) and healthy larva (*lower*). (*f*) Pupa of snowpool mosquito, *Aedes fitchii*, killed by conidial stage *Erynia aquatica* infection, floating on surface of snow pool. (*g*) Green peach aphids (*Myzus persicae*) killed by conidial stage *Pandora neoaphidis* attached by rhizoids to spinach leaves. (*h*) Rhagionid fly (*Rhagio mystaceus*) killed by *Erynia ithacensis* infection attached by rhizoids to underside of beech (*Fagus grandifolia*) leaf.

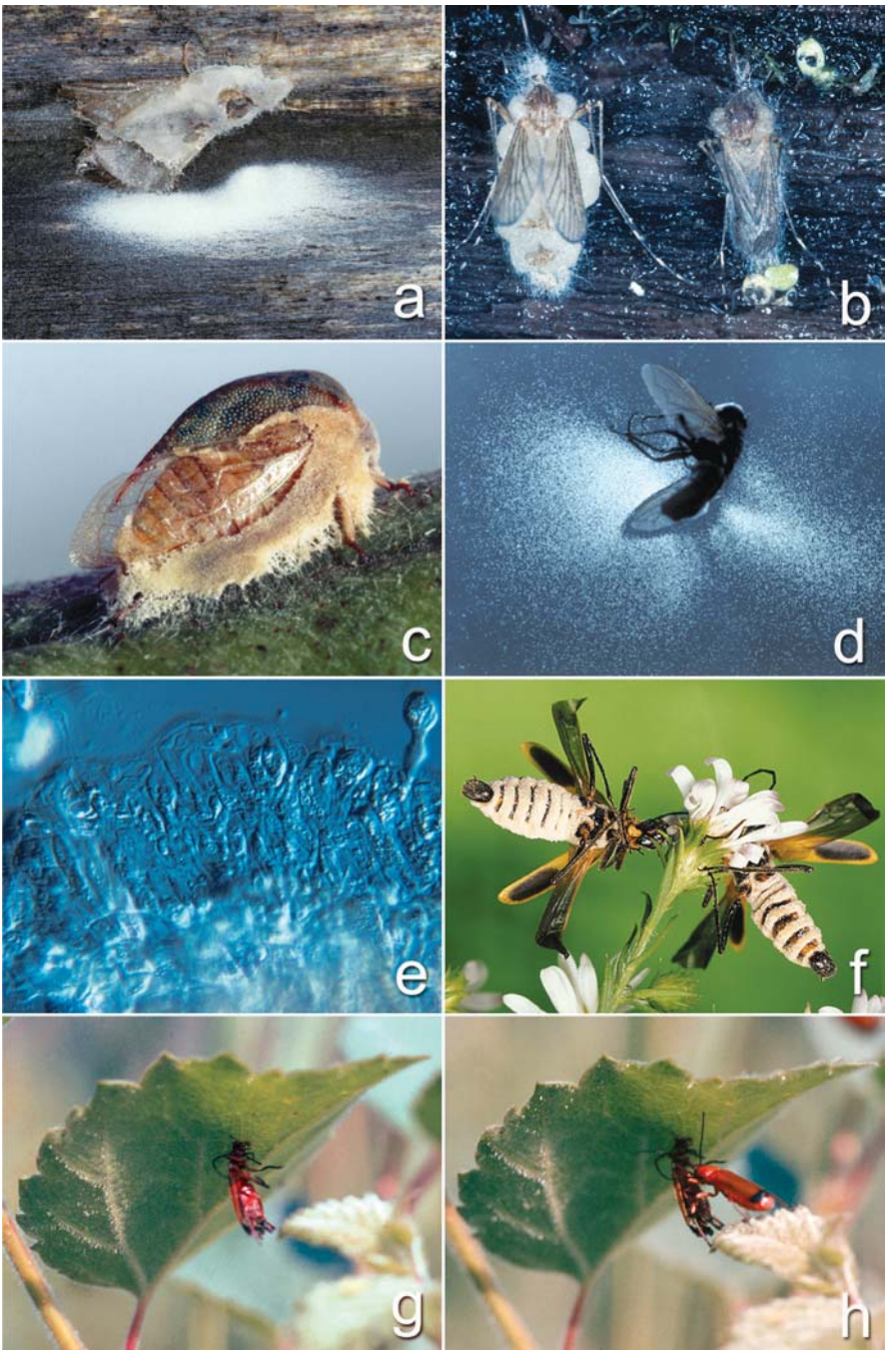

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Figure 4 (*a*) Adult caddisfly (Trichoptera) killed by *Erynia rhizospora* held underneath a damp log by rhizoids (note the primary conidia discharged beneath the caddisfly). (*b*) Snowpool mosquitoes (*Aedes fitchii*) killed by resting-spore stage of *Erynia aquatica* and held to damp wood by rhizoids. (*c*) Three-cornered alfalfa hopper (*Spissistilus festinus*) infected by *Pandora delphacis* and held to plant by rhizoids. (*d*) House fly (*M. domestica*) killed by *Entomophthora schizophorae* held to window pane by specialized rhizoids and "glue" that emerged from the labellae of proboscis (note primary conidia discharged onto the glass around the host). (*e*) Photomicrograph of the specialized rhizoids of *E. schizophorae* that emerged from the labellae of a house fly killed by *E. schizophorae*. (*f*) Two soldier beetles, *Chauliognathus pennsylvanicus*, killed by the fungus *Eryniopsis lampyridarum*. Beetles infected with the conidial stage of this fungus die with their mandibles tightly gripping flowering plants, and then after death their elytra and wings become raised as the conidiophores emerge through the integument. (*g*) Adult cantharid beetle (*Rhagonycha fulva*) killed by *Entomophthora* sp. The beetle is attached by its mandibles to the underside of a leaf. (*h*) *R. fulva* attracted to the *Entomophthora* sp.-infected *R. fulva* cadaver in photo *g*.

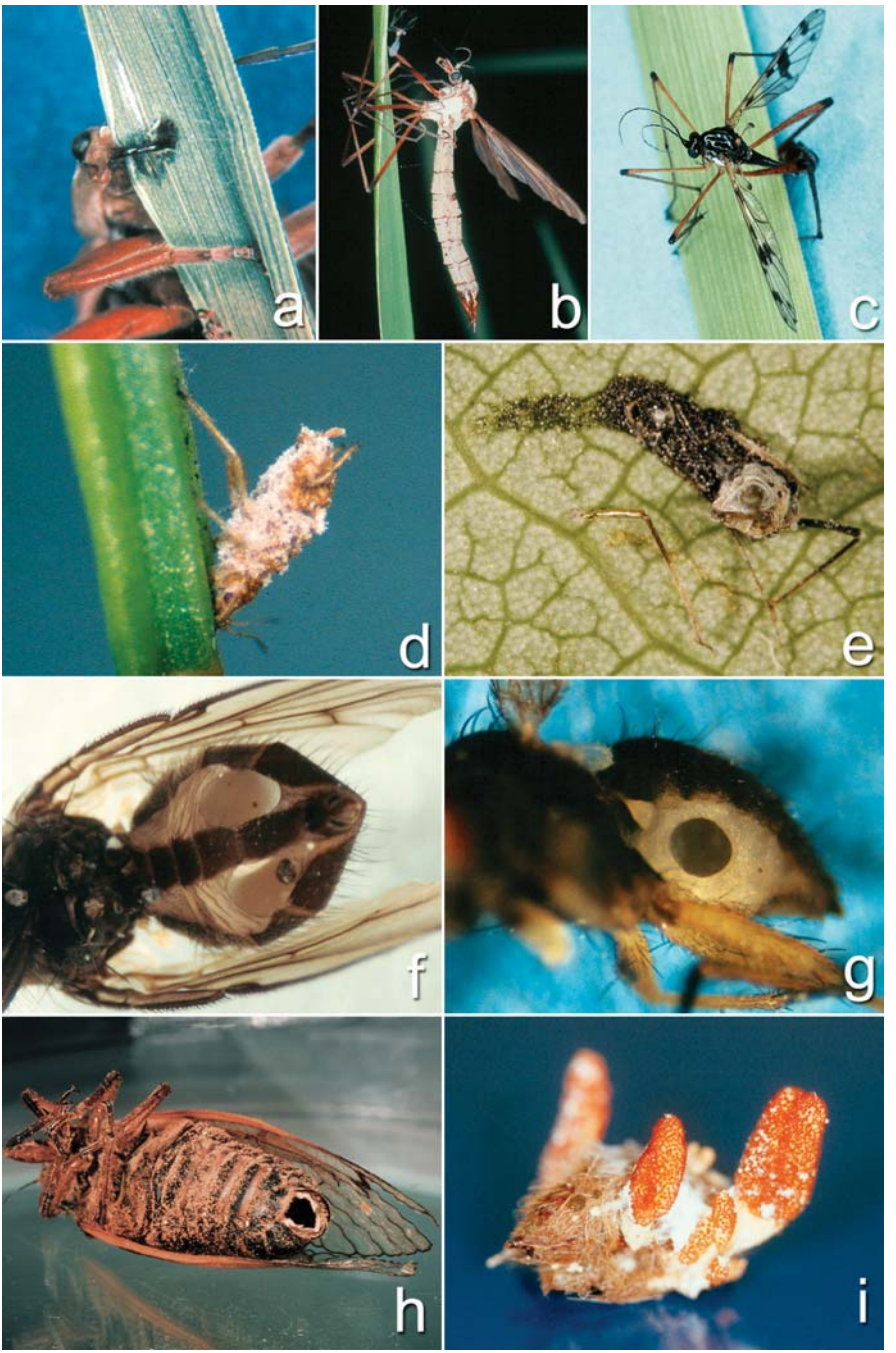

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Figure 5 (*a*) Close-up view of the mandibles of an *Entomophthora* sp.-infected cantharid beetle (*Cantharis livida*) fixed to the vegetation. (*b*) Adult tipulid fly attached to the vegetation by its legs. No fungal outgrowth is seen. This syndrome is typically caused by infections from *Eryniopsis caroliniana* and *Entomophaga tipulae*. Photo by Holger Philipsen. (*c*) Adult *Ptychoptera contaminata* killed by *Entomophaga ptychopterae* fixed to the vegetation by its legs. (*d*) Green spruce aphid (*Elatobium abietum*) infected by *Neozygites* sp. attached by its stylet to the base of a needle from sitka spruce. (*e*) Tuliptree aphid, *Illinoia liriodendri*, killed by an infection of the resting-spore stage of an undescribed species of *Neozygites*. The host body becomes filled with resting spores and then liquefies, spreading the resting spores. Photo: Jon Zawislak. (*f*) Adult Arctic fly, *Spilogona dorsata*, infected with *Strongwellsea* sp. nov. The infective conidia are discharged from the holes in the abdomen of the still-living host. Photo: Leif Stausholm Jensen. (*g*) Adult small muscoid fly *Coenosia testacea* infected by another undescribed species of *Strongwellsea*. The fly is only a few millimeters long, so the conidia must be discharged from this small cavity opening in the living fly to transmit the pathogen. (*h*) Periodical cicada, *Magicicada septendecim*, infected with resting-spore stage of *Massospora cicadina.* Powdery brown resting spores are dispersed through the hole in the abdomen while the cicada is still alive and able to fly. Resting spores are seen covering the body of the cicada. (*i*) Pupa of the lepidopteran species *Calliteara pudibunda*, killed by *Cordyceps militaris*. Ascospores were produced on the stroma that grew out of the pupa.

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