Can plants use entomopathogens as bodyguards?


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Abstract
For 20 years, ecologists have been gathering evidence in support of the hypothesis that plants can use insect natural enemies such as predators and parasitoids as bodyguards to protect themselves from herbivory, but entomopathogens have escaped this consideration. We extend the bodyguard hypothesis to ask whether plants can use entomopathogens as bodyguards. We first discuss the evolutionary context of such tri-trophic interactions and then categorize possible mechanisms as: (1) maintaining a population of bodyguards on the plant surface, (2) increasing contact rates between insect host and pathogen and (3) increasing the susceptibility of the host. We explore these mechanisms further, examining published studies for evidence for the hypothesis. We then discuss potential costs to the plant of promoting pathogens as bodyguards which may include a reduction in the efficiency of other “bodyguard” species, the incidental promotion of plant pathogens and the risk of entomopathogens developing phytopathogenicity. Aside from our intention to stimulate the testing of the bodyguard hypothesis with entomopathogens and to provide a conceptual framework for this, we hope to bring evolutionary ecology and insect pathology closer together.

Keywords
Biological control, bodyguards, epizootiology, insect pathology, mutualisms, plant defence, tri–trophic interactions.

INTRODUCTION
There is something of a gulf between insect pathology and evolutionary ecology. Insect pathologists have historically been concerned with description of pathogens and their application to the resolution of pest problems. Reviews of the field have mainly been mechanistic syntheses. Meanwhile, many evolutionary ecologists developing theory have tended to skirt around insect pathogens.

In the area of tri-trophic interactions between plant, insect herbivore and pathogen of the herbivore, there have been many experimental studies of an applied bent, and one review (Duffey et al. 1995), the subject also receiving brief treatments in other reviews (e.g. Cory et al. 1997; Hajek 1997). Throughout this literature, there is a modest assumption that observed effects of the plant on the pathogen are the result of evolved defences to herbivores or phytopathogens. In the meantime, entomologists working on predators and parasitoids have spent nearly 20 years following up a suggestion by Price et al (1980) that natural enemies of herbivores could be considered part of the battery of defences available to a plant. This is increasingly yielding evidence that plants may use natural enemies as bodyguards (a term ascribed to the interaction more than a century ago; see Bentley 1977), by providing food or shelter for the enemies (e.g. Jolivet 1996; Walter 1996) or by producing herbivore-induced volatiles to betray the presence of herbivores (see Sabelis et al. 1999a, b). Curiously, the possibility that pathogens may be involved in similar mutualistic interactions with plants, whilst implicit throughout the literature, has largely been overlooked by both sets of scientists.

In this review, we ask whether plants can use entomopathogens as bodyguards. We argue that to understand the evolution of bodyguard interactions requires a connection to be made from the plant trait which affects the pathogen, through direct and indirect effects on the dynamics of the herbivore on the plant, to fitness consequences for the plant. We use this approach to recast some empirical studies in the light of the bodyguard hypothesis. In this, our aim is not to convince the reader that our explanation is universally applicable. It is rather to provide an alternative hypothesis, which at times is inevitably speculative in nature, to those normally explored. We have avoided a taxonomic treatment of the pathogens, or any related predictions according to life history, as the absence of literature specifically testing this hypothesis would render any such generalities at best premature and at worst limiting.
The flip-side of bodyguard interactions is then examined: costs for the plant to promote the pathogens such as potentially diminished efficiency of other “bodyguard” species, the incidental promotion of plant pathogens and the risk of entomopathogens developing phytopathogenicity.

It is intended that our arguments will at once stimulate the critical testing of the bodyguard hypothesis with pathogens whilst serving to bring food web ecology and evolutionary theory closer together with insect pathology.

WHAT IS THE BASIS FOR THE EVOLUTION OF PLANT-ENTOMOPATHGEN MUTUALISMS?

For mutualisms to have evolved between a plant and pathogen of its insect herbivores requires that the plant be able to affect the pathogen so as to counter the loss of plant fitness arising from herbivory. If there is genetic variation in plant traits that can affect the herbivore–entomopathogen interaction and there are resulting fitness consequences to the plant, then there is a basis for natural selection to act, i.e. plants may evolve to use entomopathogens as bodyguards. Twin assumptions arising from this are that herbivory in the absence of natural enemies reduces plant fitness and that pathogens are capable of limiting this loss of fitness. That the former is commonly (although by no means universally) the case is clear from the existence of evolved plant defences to herbivory. The latter is perhaps less clear so we shall now briefly address this.

An explanation for the anomaly that the world is green when there are so many extant herbivores has been found in the control of these herbivores by natural enemies on the third trophic level (Hairston et al. 1960). This is commonly taken to mean predators and parasitoids, but of course there is no reason to exclude pathogens from this explanation. There is an abundance of evidence that natural epizootics of pathogenic diseases can cause very high mortalities in populations of herbivorous insects (see Fuxa & Tanada 1987). This has led to a body of work attributing population cycles of forest Lepidoptera to baculoviruses (see Anderson & May 1980) but this view has been challenged (Berryman 1996). A review of life table data from 78 insect herbivore species from a range of habitats concluded that pathogens do not on average represent a potent mortality source, but there remain instances where they do and their impact may often be underestimated (Hawkins et al. 1997). The impact of this source of herbivore mortality on plant fitness has not specifically been assessed and in fact any negative effects on herbivore population dynamics (through reduced feeding, birth rates, etc.) should benefit the plant. Successful use of microbial control in agricultural crops (see Lacey & Goettel 1995) suggests that there may be (or have been in a plant’s evolutionary history) beneficial fitness effects of naturally occurring insect diseases to the plant.

As regards the pathogen’s fitness, it is worth clarifying that a bodyguard interaction, in the strictest sense, need not be mutualistic – the effect on the pathogen could be neutral or even detrimental so long as there is a positive effect on the plant. A conflict of interests may arise when the optimal strategy for an entomopathogen is slowly to exploit a patch of hosts, or to allow its hosts to continue to feed, while the optimal strategy for the plant is to promote the pathogen’s virulence and quickly be free of herbivory. In this case, a bodyguard trait on the part of the plant may not be beneficial to the pathogen and one might expect to see some coevolutionary interplay between plant and entomopathogen.

HOW MIGHT PLANTS MANIPULATE ENTOMOPATHGENS?

Most work on the ecology of entomopathogens has focused on the direct effects of pathogens on herbivore numbers. A bodyguard interaction is, by definition, an indirect one, i.e. between the plant and the herbivore via the pathogen. A plant may have an indirect effect on herbivores by affecting pathogen numbers (a numerical interaction), in essence maintaining a population of pathogens as bodyguards, or it may enhance the effectiveness of the pathogen (a functional interaction) by increasing contact rates with the host or the host’s susceptibility to the pathogen (see Janssen et al. (1998) for a discussion of direct versus indirect and numerical versus functional interactions in food webs).

While proposing possible interactions, it is important to bear in mind the constraints imposed by the biology of the organisms concerned. The three groups of entomopathogens discussed are fungi, bacteria and viruses. Some generalities are that fungi and bacteria may combine a saprophytic lifestyle with insect pathogenesis, whilst viruses are exclusively pathogenic. There are a variety of routes by which entomopathogens disperse, but dispersal in the air or in water is most important for fungi, whilst dispersal via movement of infected hosts and release from disintegrating host cadavers to drip down to lower leaves is most important for viruses and bacteria. To infect new hosts, fungi actively penetrate the cuticle. Bacteria and viruses are ingested together with the plant substrate, whereupon they must pass the hosts’ gut lining to infect it. These characteristics set the stage upon which the plant may act to increase its fitness by promoting the effectiveness of the pathogen.

Maintenance of a pathogen population

Where insect natural enemies of herbivores are employed by plants as bodyguards, the provision of food and shelter
seems to be one of the best strategies available to a plant as it allows for maintenance of a population of bodyguards that can act early against herbivore populations before they become established. One may therefore expect a similar strategy to be employed with respect to pathogens of insect herbivores. In fact, the relative immobility of pathogens may make this a very feasible form of bodyguard interaction as the bodyguard is unlikely to relocate to another plant. Much evidence exists of fungal endophytes reducing insect and mammal herbivory via toxins (Carroll 1988; Clay 1988; Saikkonen et al. 1998) and of microbial populations on the phylloplane and rhizosphere protecting the plant from phytopathogens (Blakeman & Fokkema 1982; Cook 1993; Elad et al. 1996). It is not then a great intuitive leap to suppose that a plant may have evolved to exploit pathogens of herbivores in a similar manner. Indeed, the fungal pathogen Beauveria bassiana sprayed on to US corn was found to grow into the plant and provide season-long control of corn borer larvae (Bing & Lewis 1991).

This type of interaction was in fact proposed by Smith & Couche (1991) to explain the high population densities of Bacillus thuringiensis on the phylloplanes of trees and other plants, and this in fact represents one of the few formulations of the bodyguard hypothesis we were able to find in the pathology literature. It has long been wondered why B. thuringiensis is commonly found in insects and soil, yet rarely causes epizootics in nature (e.g. Dulmage & Aizawa 1982). If one casts the bacterium as a plant mutualist then this begins to make a good deal of sense: the bacterium may be maintained by the plant on the phylloplane and in the soil while the plant benefits from the bacterium killing insects before they reach large numbers. Furthermore, microbial populations on leaves can be spatially very heterogeneous (Kinkel 1997); so, there may be a basis for herbivores to recognise and avoid plants or critical plant parts that are protected in this fashion, rendering the defence even more effective. Seeds may tolerate a certain amount of growth of such organisms and thereby gain protection, much as they do with fungal endophytes (Carroll 1988; Clay 1988; Saikkonen et al. 1998). The fact that fungi that combine a saprophytic lifestyle with an entomopathogenic one are able to grow on grains of rice or wheat (albeit boiled) implies that plant seeds are at least nutritionally adequate.

Many plant traits will influence directly or indirectly the survival of entomopathogens. Although certainly not the only selection pressure on these traits, selection may mould them to improve pathogen persistence. Examples are canopy architecture, leaf form and leaf colour, which can greatly diminish the harmful effects of UV on these pathogen propagules. Other examples include the density of hairs, waxiness, the veins, size and shape of leaf, angle of the leaf to the stem, number of stomata and density of the canopy, which will influence the microclimate of the leaf, the thickness of the boundary layer and the effects of wind and rain (Burrage 1971; Ferro & Southwick 1984). Finally, the phylloplane pH can affect infectivity of polyhedra of baculoviruses (Young et al. 1977) and plants may provide nutrition or other chemical needs. Conidia of the fungal pathogen Achemsonia aleyrodic were found to retain infectivity to whitefly much better on some plants (e.g. cucumber) than others (e.g. poinsettia). Whatever the reasons, the implication is that there is genetic variation upon which selection may act. It certainly seems that further investigation of this area could bear fruit.

Contact rates

The first means by which a plant may have a functional effect on entomopathogens is by increasing the contact rate of infective propagules with potential hosts, and this may be via an effect on the pathogen or alternatively on the host.

Plant growth and form may determine contact not only with phytopathogenic propagules but also with entomopathogenic ones. Evidence for the latter comes from a laboratory study by Ignoffo et al. (1977), who demonstrated that soybean seedlings can pick up spores of the fungus Nomuraea rileyi from the soil and that these spores can subsequently infect larvae of Trichoplusia ni (soybean looper). Inyang et al. (1998) showed that brassica leaf expansion may reduce the density of spores of the fungus Metarhizium anisopliae already present on the leaf and so reduce contact with larvae of Phaedon cothliarise (the mustard beetle). Plants may also influence the physical, chemical and biological properties of soil, and in turn the growth and movement of entomopathogens (Fuxa & Geaghan 1982; Storey & Gardner 1987; Storey et al. 1989; Studdert & Kaya 1990).

Furthermore, one can imagine an influence of leaf shape, glabrousness and waxiness on the ability of entomopathogen propagules to penetrate the leaf boundary layer and then to adhere. All of these factors influence the contact rate and may therefore be modified by selection.

When attacked, many plants release herbivore-induced volatiles which may attract predators or parasitoids. Few attempts have been made to investigate the effects of these volatiles on entomopathogens but it is conceivable that a plant may influence pathogen behaviour by providing signals, such as volatile chemicals, in order to stimulate germination of dormant spores, affect arrestment in the patch or even attract infected insects if the pathogen can alter its hosts’ responsiveness to plant volatiles. Indeed, the study that comes closest to testing the bodyguard hypothesis in entomopathogens is by Brown et al. (1995). The authors found that the germination of conidia of the entomopathoralean pathogen Pandora (= Erynia) neo-
phidis was inhibited by volatiles released by tobacco plants in response to herbivory by Myzus nicotianae (tobacco aphids). Germination would in this instance lead to the production of secondary conidia, which would re-disperse away from the leaf. Thus, the authors hypothesized that the conidia “sit and wait” until they are picked up, whereupon they germinate to infect the aphid hosts. This hypothesis was unfortunately not tested but such arrestment in response to herbivore-induced plant volatiles has very strong parallels with other systems where plant volatiles can cause predators or parasitoids to remain in a patch, thereby acting as bodyguards for the plant (Sabelis et al. 1999a, b).

A plant may also increase contact rates by manipulating the insect herbivore distribution rather than that of the pathogen. Heterogeneity in the concentration of resources or toxins within a plant is known to affect herbivore distribution, concentrating them in some areas of the plant (Hoy et al. 1998). An alternative means to this end is to limit new growth and expansion of leaves. These would almost certainly increase transmission rates, which is an important factor for many microorganisms. Plant form and defences can also influence the mobility of herbivores (Kartohardjono & Heinrichs 1984; Hoy et al. 1998) and so their pick-up of pathogen propagules. With respect to baculoviruses, the higher an infected herbivore feeds, the more drip-down of virions will protect leaves from further damage, so there may be a benefit to both the plant and the pathogen to concentrate these herbivores higher up (even at the cost of a higher risk of damage to the more vulnerable parts of the plant). Alternatively, it may benefit a plant for a mycosed herbivore to remain lower down or away from the extremities to provide a more suitable climate and to gain monopoly of dispersing spores.

Finally, a plant may increase the contact rate by employing a fourth party. Although pathogens, predators and parasitoids may compete for a common host or prey species, there may be instances where the presence of one enhances the effect of the other. The potential for other natural enemies and scavengers to effect the dissemination or horizontal transmission of pathogens has been demonstrated in the laboratory (Brooks 1993; Fransen & van Lenteren 1993; Pell et al. 1997), and there is some evidence that this may lead to increased infection rates in the field (Hochberg 1991). The presence of parasitoids may increase transmission due to the increased movement of the host (Furlong & Pell 1996) and predation pressure in the field can increase the aggregation of herbivores and thus make them more susceptible to pathogens (Cappuccino 1988). So, any mechanism whereby a plant increases the number or foraging activity of these fourth parties would conceivably have an extra benefit by increasing the contact rate and therefore disease.

Susceptibility

If a herbivore is diseased or liable to be so, then a plant defence which somehow increases its susceptibility can be just as effective as a more direct defence, or more so. One component of a herbivore’s susceptibility may be its development time, which will in turn affect the length of time for which it is vulnerable. The attractive hypothesis that sublethal plant defences (or poor food quality) can induce slow herbivore growth (perhaps especially of more vulnerable life stages) and so high natural enemy-induced mortality, has received mixed support from empirical studies when tested with insect natural enemies (Clancy & Price 1987; Benrey & Denno 1997). There is even evidence that high food quality and herbivore growth rates can be positively correlated with increased mortality due to baculoviruses (Hoover et al. 1998). It may be illuminating to explore this with more entomopathogens.

Different food plant types or species can alter differentially the susceptibility of herbivores to pathogens (Hajek et al. 1995; Peng et al. 1997). This leads into the most obvious and the most studied area of tri-trophic interactions within insect pathology, which is the effect of chemical plant products on pathogens. This has been the subject of a major review in the case of baculoviruses (Duffey et al. 1995) and there are many examples from other areas of insect pathology. Throughout the literature, effects on entomopathogens are seen as a side-effect of chemical defences against phytopathogens and herbivores. These effects may be positive or negative; where positive, fitness benefits to the plant may accrue, so the pathogen effectively functions as a bodyguard. Where negative, any fitness losses due to reduced herbivore disease are presumably outweighed by the advantages accruing from the direct protection from plant disease or herbivory. There is variation in the effects of toxins on pathogens: the toxin tomatine was found to be more toxic in vitro to B. bassiana than was solanine (Costa & Gaugler 1989). This may explain a lower natural prevalence of B. bassiana in Leptinotarsa decemlineata (Colorado beetles) in fields of tomato than fields of Solanum spp. (Hare & Andreidis 1983). We will probably never be able to test experimentally whether greater fitness costs from this herbivore have led to a greater selective pressure on Solanum spp. for its alkaloid to be less toxic to its potential ally, B. bassiana. However, we do see here a basis for such selection to occur. Costa & Gaugler (1989) noted that B. bassiana is relatively tolerant to alkaloids compared with phytopathogenic fungi, but questioned how selection pressure could be sufficiently strong on the pathogen to have led to this. But there is of course every reason to expect the pathogen to adapt to its chemical environment, without corresponding coevolution of the plant to
counter this (as might be the case with a plant pathogen). If indeed it does develop tolerance to plant alkaloids then the production by a plant of a toxin which does not harm *B. bassiana* may be selected for.

A particular case may be made for the production of pathogenesis-related (PR-) proteins as a bodyguard trait. These are synthesized by a plant in response to attack by herbivores or phytopathogens and the chitinase activity of many of these proteins may be able to degrade the insect peritrophic membrane (Gopalakrishnan *et al.* 1995; Mayer *et al.* 1995). This membrane is the principal barrier to infection by baculoviruses and the entry of *B. thuringiensis* toxins into the host body (see Lehane 1997). Laboratory and field studies have shown that the activity of baculoviruses and *B. thuringiensis* can be enhanced by the addition of chitinases to the diet of lepidopterous insects or to aerial spray applications in forestry (Smirnoff 1971, 1973, 1974; Morris 1976; Shapiro *et al.* 1987). The mechanism that has been proposed is that the chitinase attacks the insect’s peritrophic membrane, making it more permeable to virions or *B. thuringiensis* toxins. It may be that this indirect effect on the herbivore has been to some degree responsible for the evolution of this trait in plants. Such a strategy would combine very well with the maintenance of a population of bodyguard pathogens on the plant surface.

A parallel with the PR-proteins can be seen in the piercing of the insect gut by plant trichomes (Wellso 1973). Aside from potentially severe direct effects, this (or perhaps abrasion by other plant structures) would probably aid subsequent invasion by pathogens and so could be a bodyguard mechanism. Another possibility is that the nutritional quality of the food plant, such as amino acid concentrations, may diminish the herbivore’s ability to form a cuticle strong enough to resist penetration by fungi (Mollemma & Cole 1996).

**SO IS IT WORTH IT?**

For an entomopathogen to be employed as a bodyguard by a plant, it must represent a good return on the investment (the benefits of the trait must outweigh the costs), it must complement the plant’s other defences, and the investment should be to some degree secure (i.e. not be overly exploitable by herbivores or by the pathogen itself).

**Costs and benefits of the interaction**

For there to be a plant–bodyguard interaction it is the gain in plant fitness which matters. Any active defence mechanism on the part of the plant would probably entail direct energetic costs which would impact on its ability to invest in other activities, such as growth or reproduction. In some cases, these costs will be so small as to be difficult to detect experimentally (Dicie & Sabelis 1992), in others this will not be the case. They could also be so tied up in the costs and benefits of another trait that they become hard to disentangle. Furthermore, the costs and benefits may vary in space and time (Bronstein 1994; Saikkonen *et al.* 1998) and may not even add up to an overall benefit for a given individual or local population in a particular environment. Rather, defence should lead to an overall benefit over a sufficiently large temporal and spatial scale. It is clearly too early to discuss the direct costs in detail, but some indirect costs may be envisaged and are now discussed.

**Entomopathogenic bodyguards versus other plant defences**

The fostering of an entomopathogen by a plant may lead to disease in other potential bodyguard species and there may also be interference between natural enemies reducing the overall effectiveness. Investment in pathogen bodyguards may limit the investment in other defence mechanisms or may directly diminish their efficacy; alternatively, it may act in synergy with other defences.

Whether it is beneficial to promote pathogens as bodyguards over other defences, including other natural enemies, will to some degree depend upon the relative hierarchy of these defences. Modelling by Abrahamson & Weis (1997) suggests that defences occurring earlier in the sequence of the plant–herbivore interaction will generally be subject to greater positive selection because those acting later will have proportionally less herbivory to prevent, and so a proportionally diminished fitness benefit. Plant traits which promote bodyguards will be among the first in this sequence as they lead to reductions in herbivory. Where pathogen activity occurs earlier than that of other natural enemies, the pathogen is more likely to be utilized by the plant as a bodyguard.

Given that there are also predators and parasitoids around, does it help the plant to invest in a pathogen as a bodyguard? That mechanisms for the selective favouring of different natural enemies may exist is suggested by work on the effect of wheat plant resistance on the outcome of within-host competition between the fungus *Erynia (= Pandoro) neoaphidis* and the parasitoid wasp *Aphidius rhopalosiphi*. On a resistant wheat variety the pathogen was favoured, apparently via an increased parasitoid development time during competition within their host *Sitobion avenae* (grain aphid) (Fuentes-Contreras *et al.* 1998). An advantage of pathogens to the investor is their low mobility: predators and parasitoids can walk or fly away. Pathogens often have the means to survive periods of host scarcity in certain environments, such as within host cadavers or the soil, or as resistant spores, as occlusion bodies in the case of viruses, or as saprophytes. They may take a while to kill their hosts but can rapidly reduce feeding rates,
in contrast to some parasitoids (see van der Meijden & Klinkhamer 2000). Although pathogens may require a high host density to cause an epizootic, once they do the large number of propagules can lead to horizontal transmission to a sizeable proportion of the host population, and where vertical transmission also occurs, this can affect future generations. Compared with the insect herbivores and natural enemies, pathogens evolve much more rapidly, allowing for adaptations to the local situation over an ecological time scale. Where a plant supports a population of predators or parasitoids, their growth rates are comparatively low, whereas it may be possible to obtain a substantial population of a fungus or bacterium quite rapidly if it is saprophytic. Furthermore, a plant may be able to transmit a microbial bodyguard to its offspring (Clay 1988).

**Risks from pathogens or herbivores**

Fostering pathogenic microorganisms could be a risky business for a plant which may itself be subject to attack by pathogens. However, if they are able to use insect predators and parasitoids as bodyguards without falling foul of insect herbivores then the same may be true of microorganisms. One means by which a plant may selectively defend itself against phytopathogens is by employing defences targeted at infection and subsequent stages of the interaction, rather than earlier stages where defences may adversely affect both plant and insect pathogen. Where a reduction in generalized defences favours an entomopathogen (e.g. leaf allelochemicals), one can expect this also to favour herbivores or phytopathogens, much as the production of herbivore-induced plant volatiles may be exploited by another herbivore to locate a vulnerable plant (Sabelis et al. 1999a). Whether the bodyguard trait is selected for will then depend upon the biotic environment, principally which species are present and at what levels.

A pathogen of an insect herbivore may well be able also to exploit other bodyguard species as hosts, although many pathogens have a host spectrum sufficiently limited for this not to occur. Where a broad host spectrum is not maintainable over evolutionary time, switching to third trophic level hosts should be rare as these generally have a lower density and so a higher epidemic threshold (Anderson & May 1998). Of course, if predator or parasitoid levels (or biomass) are higher or more constant than herbivore levels because the plant is providing food to keep a population of bodyguards (Bakker & Klein 1992; van Rijn & Tanigoshi 1999), then these may present a better potential host.

There is the potential risk of an entomopathogen switching to phytopathogenicity. The fungal genera *Colletotrichum* and *Verticillium* contain both plant and insect pathogens, implying some flexibility over evolutionary time, but we know of no species which has a host range spanning both groups – this may, however, be because any which are found are rapidly discarded as biological control agents. If the plant represents to the pathogen a large resource whilst the herbivore represents an ideal means of dispersal, why should it not exploit both, much as plant pathogens may exploit herbivores via phoresy? Conversely, if a long-living plant is subject to risk from a plant pathogen which is also capable of attacking herbivores, it might benefit the plant to employ one of the traits described above to increase the herbivore’s susceptibility and so drive specialization of the pathogen towards an insect-pathogenic lifestyle. The degree to which these processes can occur will depend upon how much the specialization necessary to exploit adequately one type of host renders the other type unsuitable.

**PERSPECTIVE**

One cannot help but be speculative in proposing an alternative framework to interpret published results than that employed by the authors: the hypothesis we present has simply not been tested for entomopathogens within tritrophic systems. As we have stated, it is hard to disentangle the costs and benefits of a trait, and therefore to ascribe a coevolutionary function. However, this is not unique to the type of system we discuss.

The assumption throughout the literature is that plants, herbivores, phytopathogens, predators and parasitoids share an evolutionary history which may affect interactions with pathogens coincidentally to be found on the third trophic level. Only once this important group of natural enemies of insect herbivores is seen as having been a player in the coevolutionary game will adequate hypotheses be formed to explore and explain the ecological interactions being considered in a given system. Once it is realized that pathogens are very much part of this game, perhaps evolving faster than any of the other players and so allowing the plant to keep up with the more rapidly evolving herbivores (Carroll 1988), will their true potential be exploited in pest management.

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**REFERENCES**


Hare, J.D. & Andreidis, T.G. (1983). Variation in the susceptibility of Lepanotaarsa deceninata (Coleoptera: Chrysolinae) when reared on different host plants to the fungal pathogen, Beauveria bassiana, in the field and laboratory. Environ. Entomol., 12, 1892-1897.


**BIOSKETCH**

Sam Elliot’s research interest is applying concepts of evolutionary ecology to invertebrate pathology, particularly in the fields of food web interactions and the evolution of virulence.

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