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# FORUM

# Beyond biological control: non-pest insects and their pathogens in a changing world

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**Abstract.** 1. Over the last few decades, there have been considerable advances in the fields of insect pathology and insect conservation but the two disciplines rarely meet. The potential of entomopathogens as biological control agents of pest insects is widely recognised but information on the role of pathogens in insect population regulation, more generally, is limited. For example, the role of pathogens as natural enemies of non-pest insects, including those of conservation value, is seldom considered beyond their context as 'non-targets' of microbial control agents.

2. Entomopathogens are prevalent in natural systems and should receive greater attention in life-history studies. There is no doubt that viruses, bacteria and fungi are major mortality agents of insects but their significance tends to be overshadowed by the attention given to predators and parasitoids.

3. We highlight the critical function that entomopathogens could have in insect population dynamics with particular reference fragmented habitats as illustrated by the theoretical literature. However, we emphasise that there are few empirical studies to test theoretical predictions.

4. We suggest that since an increase in the incidence of disease is predicted in most environmental change scenarios, it is more important than ever to turn our attention to insect pathology when we consider insect population dynamics.

**Key words.** Covert virus, entomopathogen, environmental change, habitat fragmentation, non-pest insects, population dynamics.

The fields of insect pathology and insect conservation have made considerable advances over the last few decades, but the two disciplines rarely meet. The role of pathogens as natural enemies of insects (entomopathogens), including those of conservation value, is seldom considered (Roy & Cottrell, 2008). Naturally occurring entomopathogens are diverse and widespread (Lacey *et al.*, 2001); 50% of living organisms are microbial (Watson *et al.*, 1995) and insect pathogens are ubiquitous in both above- and below-ground ecosystems (Hajek, 2004). However, most studies on insect life history refer only

to predators and parasitoids when considering natural enemy guilds. It is possible that pathogens have been neglected in life-history studies because of the technical difficulties involved in assessing host–pathogen relationships within a natural community context. Hawkins, Cornell & Hochberg (1997) compiled life-history tables for 78 holometabolous insect species to quantify the impact of predators, parasitoids and pathogens as mortality factors and concluded that there was little or no mortality attributed to pathogens. It is accepted that pathogen abundance could be underestimated; the covert nature of some pathogen infections (Burden *et al.*, 2003; Cooper *et al.*, 2003) has certainly contributed to an underestimate of their importance (Boots *et al.*, 2003; Bonsall *et al.*, 2005). However, modern molecular techniques are now available to enable the balance to be addressed.

### Pathogen life cycles

Although entomopathogens are taxonomically diverse, it is possible to make some generalisations about their life cycles and these are important when considering their importance in host population dynamics (Roy *et al.*, 2006). Viruses, bacteria and protists usually enter the host via oral routes whereas fungi invade through the cuticle (Steinhaus, 1964). Once inside the living host, they proliferate and often then kill the insect when resources are depleted, although some pathogens may persist as covert infections inside the host. The final stage of pathogen life cycles results in the release of infectious propagules for subsequent transmission.

Pathogens and hosts are engaged in a co-evolutionary arms race, adapting to maximise their own reproductive output and ultimate fitness. Pathogen fitness is defined as the basic reproductive rate ($R_0$) – the number of new infections generated by one original infection when invading a clean, susceptible population. Both transmission efficiency and survival within and outside the host are components of $R_0$. The host will be avoiding or resisting the pathogen or optimising survival after infection. The strategies adopted to achieve this are almost as diverse as the organisms themselves and are modulated by the biotic and abiotic environment in which these organisms co-exist and/or compete for resources (Roy *et al.*, 2006). Some pathogens have wide host ranges with the potential to exploit many different hosts others alternate between biotrophy and saprotrophy and some are host specialists. Entomophthoralean fungi are often extremely host specific, infecting insects within a family, while hypocrealean fungi are more generalist in their host range, infecting taxonomically distinct orders of insects and functioning as facultative saprotrophs (Hajek *et al.*, 2008). Baculoviruses are a group of DNA viruses that occur widely, but not exclusively, in Lepidoptera, and have been developed as biopesticides in some instances (Szewczyk *et al.*, 2006). A few baculovirus species appear to be very host specific (e.g. Barber *et al.*, 1993), but some such as *Mamestra brassicae* nucleopolyhedrovirus (MbNPV) and the closely related *Panolis flammea* nucleopolyhedrovirus (PaflNPV) have a relatively wide host range (Doyle *et al.*, 1990). Indeed, it is thought that infection of different insect species plays an important role in the maintenance of genetic diversity within virus populations (Hitchman *et al.*, 2007). The bacterium *Bacillus thuringiensis* as a species has a wide host range, but the diversity of crystal proteins (cry toxins) expressed by the bacterium ensures that each strain, which carries a different cry toxin, is specific often to an insect order (De Maagd *et al.*, 2001). Pathogen host specificity is a contentious issue and while the taxonomic status of many pathogens remains unresolved (Hibbett *et al.*, 2007), it is difficult to interpret the available literature.

Much of the evidence for host range is laboratory based and such studies do not consider the complexities of a heterogeneous environment (Samways, 2005; Solter, 2006). Potential hosts may be physiologically susceptible under laboratory conditions but this may not translate to ecological susceptibility in the field for a number of reasons (Roy & Pell, 2000; Solter, 2006). For example, under field conditions, hosts may rarely become exposed to concentrations of the pathogen sufficient to cause infection, or there may be a lack of spatial or temporal synchrony between host and pathogen populations. Recent studies have contributed to our understanding of the intricate and often complex nature of insect–pathogen interactions, but still much of the emphasis in this research area focuses on entomopathogens as biological control agents. There is a need for fundamental research to further our understanding of the evolution of life-history strategies and the ecology of insect–pathogen interactions in the natural environment.

### Economic uses of insect pathogens

The significance of insect pathogens in regulating economically important insect populations has been recognised and exploited for centuries (Steinhaus, 1956). The successful development of the bacterium *B. thuringiensis* as a control agent of insects marked the advent of modern microbial control (Lacey *et al.*, 2001; Lord, 2005) and the discipline of insect pathology. The link between insect pathology and pest control has continued and a wealth of literature exists on entomopathogens as natural pest–insect population regulators that can be manipulated for enhanced impact in microbial control strategies (Evans, 1986; Lacey *et al.*, 2001; Hajek *et al.*, 2007). Landmark studies that exemplify this include those on gypsy moths, *Lymantria dispar*, and the fungus *Entomophaga maimaiga* (Reardon & Hajek, 1993); pine beauty moth, *Panolis flammea*, and nucleopolyhedrosis viruses (Entwistle & Evans, 1987); cotton aphids, *Aphis gossypii*, and the fungus *Neozygites fresenii* (Steinkraus *et al.*, 1995); palm rhinoceros beetle, *Oryctes rhinoceros*, and a non-occluded virus (Bedford, 1980); the bacterium *B. thuringiensis* and European corn borer, *Ostrinia nubilalis* (Beegle & Yamamoto, 1992), *L. dispar* (Burges & Daoust, 1986), vector blackflies and mosquitoes (Lacey & Undeen, 1986) and stored-product pest, *Plodia interpunctella* (McGaughey, 1986) and the development of genetically modified baculoviruses (Cory, 2000; Hails, 2001; Hails *et al.*, 2002). These studies on pest insects highlight the potential of entomopathogens to regulate populations, but pathogens are rarely included as natural enemies in studies on non-pest insect species. By definition, non-pest insects are often at lower densities than insects designated with pest status and this could impede horizontal transmission making highly visible virulent epizootics a rare event.

### Effects of pathogens on non-target organisms

Historically, insect pathogens have been noted as potentially important in the regulation of non-pest insects (Perrin, 1976; Dwyer, 1991; Myers, 2000; Lacey *et al.*, 2001), but even the first experimental studies in 1835 focused exclusively on economically important insects namely honey bees, silk moths and pest insects (Steinhaus, 1975; Howarth, 2001). This legacy has continued and prevalence of pathogens in non-pest insects is generally considered only when the insects are designated 'non-target' hosts of a generalist entomopathogen that is under development as a microbial control agent. These non-target insects are restricted to particular taxonomic groups such as honey bees, predators, parasitoids or insects closely related to the target pest insect

often within ecosystems already perturbed through agriculture or forestry (Thomas & Blanford, 2003; Hajek *et al*., 2007).

## Importance of pathogens in natural systems

Our understanding of host–pathogen interactions from a pest management perspective, although often limited by scale, does have great relevance when we consider the potential importance of pathogens in populations of non-pest insects of conservation importance in natural and semi-natural ecosystems, for which there is currently very little information. Bierne (1955) comprehensively reviewed the causes of natural fluctuations in abundance of British Lepidoptera and recognised the importance of fungal diseases particularly in wet summers. There are further examples of insect populations cycling from low to high density where pathogens are implicated in declines from high density. For example, epizootics of the fungal pathogen *Pandora neoaphidis* in nettle aphids, *Microlophium carnosum* (Perrin, 1976); transmission dynamics of the nucleopolyhedrosis virus of douglas-fir tussock moth, *Orgyia pseudotsugata* (Dwyer, 1991); population fluctuations of western tent caterpillars, *Malacosoma californicum pluviale*, attributed to nucleopolyhedrosis virus (Myers, 2000). In a more recent study, Graham *et al*. (2004) assessed the prevalence of a nucleopolyhedrovirus in spatially separated populations of the winter moth *Operophtera brumata* in Orkney. Virus was found in 11 of the 13 winter moth populations sampled. In two of these winter moth populations, more than 50% mortality was attributed to viral infection. Furthermore, three previously undescribed reoviruses were found, with prevalence ranging from 0–50% and presence correlating with reduced host fecundity (Graham *et al*., 2006). The authors concluded that the high prevalence of disease, and particularly covert disease, in these natural lepidopteran host populations should encourage further studies into the role of pathogens in the regulation of host insect populations. We agree with this view but further suggest careful consideration should be given to rare species and common species that, with environmental change (such as habitat fragmentation and climate change), are suffering continuing population declines.

## Effects of pathogens on rare species

Most organisms are host to specialist and generalist microbial natural enemies, but it is important to ascertain whether these diseases are of conservation concern for our rare species (Lafferty & Gerber, 2002). The transmission of an infectious disease agent through any population will increase with the density of susceptible and infectious hosts (Anderson & May, 1986). However, some species are nationally scarce but locally abundant and transmission between hosts could be facilitated by local aggregations. Simple epidemiological models indicate that there will be densities at which a host population is too low for invasion by a pathogen which relies solely on horizontal transmission, signifying that rare or significantly depleted populations should be at minimal risk from their microbial natural enemies. Horizontal transmission is usually strongly density dependent and so pathogens are unlikely to result in host extinction, particularly if hosts are rare. Indeed, highly pathogenic organisms that rapidly kill their hosts will tend to extinction themselves (burn out) if transmission to new hosts at low densities is insufficient to maintain $R_o$ above 1 (Anderson, 1979). Epidemiological theory would therefore suggest that infectious diseases which rely wholly on horizontal transmission are irrelevant to conservation biology when species are rare and subsequently at low population densities for much of the time. This conclusion relies on a number of assumptions which may be contravened. Three examples are given below.

Until recently, it was assumed that the transmission of baculoviruses between hosts was primarily horizontal, resulting in overt and lethal infection. However, there are now several studies which have revealed a degree of vertical transmission of these viruses, with either imperceptible or sublethal effects on host fitness (Hughes *et al*., 1993, 1997; Burden *et al*., 2002, 2003; Graham *et al*., 2004). Furthermore, the evidence suggests that these persistent infections do retain the ability to revert to the overt lethal state (Cooper *et al*., 2003; Burden *et al*., 2006). Theoretical models illustrate that for a wide range of conditions, covert infections may become endemic in the host population, to the exclusion of clean, uninfected hosts (Bonsall *et al*., 2005). Vertical transmission does not rely on host densities being above a critical threshold and therefore persistent, covert infections represent a mechanism by which potentially lethal disease may persist in a declining population. Vertical transmission is also widely reported for bacteria and microsporidia and is potentially an important mechanism for persistence within the life cycle of these groups (Solter, 2006). However, the emphasis for experimental work has been from the perspective of biological control or medical importance and this has limited the range of host orders and pathogens evaluated. This mode of transmission is almost certainly important for far more pathogen species than is currently known.

Our second example challenges our knowledge of the host range of many pathogens. We suggest that many pathogens are thought to be host specific because they have not been looked for beyond the host from which they were originally isolated. In particular, covert infections in 'unexpected' hosts are likely to go undetected. In *O. brumata* populations in which three novel reoviruses were detected, it was subsequently discovered that two other members of the heather feeding community, the grey mountain carpet, *Entephria caesiata*, and the July highflyer, *Hydriomena furcata*, were also carriers (Graham, 2005; Graham *et al*., 2006). Pathogens with a wider host range can exploit alternative (reservoir) hosts, and once again this decouples the reliance of the pathogen on the density of any one particular host species.

Third, persistent infective stages may allow pathogens to ride out periods of low host density, theoretically even to the point of causing host extinction. The soil represents a very stable environment which allows some pathogens to persist in an infectious state for many years. Organisms that spend periods of their life cycle in those environments can be at risk even when they are at low population densities. For example, baculoviruses and fungi (both hypocrealean and entomophthoralean) can persist in the soil where they may infect late stage larvae or pupae of Lepidoptera

which descend to the soil to pupate (Raymond *et al.*, 2005; Meyling & Pell, 2006; Roy *et al.*, 2006).

We conclude, for all the reasons discussed above, that the impact of pathogens, even those thought to be primarily horizontally transmitted, may be greater than has been formally recognised to date.

## Effects of environmental change on epidemiology

Having considered the potential role of pathogens in the natural regulation or even extinction of rare species, we now consider how this may be affected directly and indirectly by anthropogenically induced global environmental change, including climate change and habitat fragmentation, which is becoming increasingly implicated in the decline of populations (Balmford *et al.*, 2005). It is anticipated that infectious diseases in general will increase in prevalence in temperate zones where the climate is predicted to become warmer and wetter (Intergovernmental Panel on Climate Change, 1996; Millenium Ecosystem Assessment, 2005). Infectious diseases and pathogens that have previously had negligible impact on insect populations could have dramatic consequences in the future because of climate change and also habitat destruction.

### Effects of climate change

Even modest changes to the climate are expected to have a rapid impact on the distribution and abundance of pest insects and their associated pathogens because they are physiologically sensitive to temperature and have short life cycles, high mobility and high reproductive potential (Ayres & Lombardero, 2000). Many non-pest insects are already responding rapidly to climate change (Parmesan & Yohe, 2003) and expanding northwards (Asher *et al.*, 2001; Hickling *et al.*, 2006). There is currently no information on comparable broad-scale changes in the dynamics of pathogen populations.

It has been recognised for many years that temperature affects biochemical, physiological and behavioural processes in insects and pathogens (Thomas & Blanford, 2003). There are many studies demonstrating temperature optima of insects and pathogens (Kobayashi *et al.*, 1981; Carruthers *et al.*, 1992; Watson *et al.*, 1993). Recent research has shown that temperature can also have a crucial role in mediating the outcome of host–pathogen interactions (Thomas & Blanford, 2003). For example, thermal effects have been demonstrated for insect pathogenic fungi (Carruthers *et al.*, 1992; Watson *et al.*, 1993), viruses (Kobayashi *et al.*, 1981) and microsporidia (Campbell *et al.*, 2007). Temperature can affect host–pathogen dynamics in a variety of ways such as alterations to the latent period of infection (Blanford & Thomas, 1999), host recovery and pathogen mortality (Roy *et al.*, 2006), pathogen and host replication (Kobayashi *et al.*, 1981), pathogen virulence and host resistance (Blanford & Thomas, 1999; Arthurs & Thomas, 2000; Stacey *et al.*, 2003). Insects (and their pathogens) are likely to track ambient temperature (thermoconforming) and are likely to be more vulnerable to changes in temperature for example as a con-

sequence of global warming. However, effects of environmental temperature change on host–pathogen interactions will be varied because pathogen and host traits will have different thermal optima (Thomas & Blanford, 2003).

It is difficult to predict the ultimate outcome of host–pathogen interactions in response to climate change because of the complexity of the interactions and lack of available data. It is common for the performance of insect pathogens used as biological control agents in the field to be highly erratic (Blanford & Thomas, 1999; Thomas & Blandford, 2003). This variability is also evident in natural systems whereby in some years, a pathogen can reach epizootic proportions and in other years hosts remain uninfected. Temperature can affect both host susceptibility and (or) pathogen virulence (where virulence is defined as the net effect of the host–pathogen interaction on host fitness) and consequently influence disease dynamics; however, responses can vary from positive or negative linear responses to more complex relationships (Blanford & Thomas, 1999; Blanford & Thomas, 2000; Thomas & Blanford, 2003). For example, the entomophthoralean fungus *Entomophaga grylli* can be a major mortality factor of the variegated grasshopper *Zonocerus variegatus* (Chapman & Page, 1979). However, an increase in environmental temperature of 2°C enables infected individuals to recover from the pathogen by raising body temperature through behaviour such as basking (Blanford *et al.*, 2003). In contrast, insect viruses generally exhibit low virulence at cool temperatures and increase in virulence with increasing in temperature eventually reaching an asymptote as temperature rises further (Johnson *et al.*, 1982). Not all insects are behavioural thermoregulators, but it should not be assumed that thermoconforming insects lack thermal sensitivity; environmentally driven variation in host body temperature will also influence host defence mechanisms such as the innate immune system, host density and melanin production (Stacey *et al.*, 2003; Thomas & Blanford, 2003).

Several entomophthoralean fungi exhibit an adaptive diurnal pattern of host death (Nielson & Hajek, 2006; Roy *et al.*, 2006); host death occurs in the late afternoon or early evening and the fungus releases infective conidia during the night when relative humidity is high. This diurnal pattern benefits transmission of the fungus between hosts. The precision of synchrony, of host death with environmental conditions favouring fungal transmission, is associated with the climatic region in which the pathogen evolved (Milner *et al.*, 1984). So fungal species with a precise timing of death and a short sporulation time appear to be adapted to continental weather conditions with cycles of dry and moist conditions during the day and night (Milner *et al.*, 1984). In contrast, fungal species displaying weak diurnal rhythm in mortality with prolonged periods of sporulation seem adapted to cool, moist climates with less diurnal fluctuation in temperature and humidity (Milner *et al.*, 1984). The entomophthoralean fungus *E. maimaiga* infects *L. dispar* (gypsy moth) larvae and sporulation is synchronised with a diurnal rhythm (Nielson & Hajek, 2006). An empirical study on this host–pathogen system demonstrated that by raising the temperature from 15 to 20°C, the peak death time narrowed and sporulation was initiated earlier at night. Furthermore, alterations to abiotic conditions will differentially influence pathogen (and host) species of different genotypes. For example, Bidochka *et al.* (2002) demonstrated that the

habitat from which the isolate was derived drives the population structure of the fungus *Beauveria bassiana*. Isolates (genotypes) of the fungal species *B. bassiana* from arctic and forested habitats grew at lower temperatures than those isolates from agricultural habitats which grew at temperatures in excess of 37°C (Bidochka *et al*., 2002).

In summary, the life-history traits of insect hosts and their pathogens are strongly influenced by temperature (and other abiotic conditions), and the precise outcome of an interaction will depend on the relevant environmental range of the genotypes of host and pathogen and the prevailing conditions. Spatial and temporal variation in host–pathogen dynamics is commonly observed and a minimal change in temperature could alter the outcome of the interaction dramatically. There is considerable need for more studies examining insect host–pathogen systems under a range of constant and fluctuating temperatures.

*Effects of habitat destruction*

Theories on the dynamics of pathogens in fragmented habitats have been based on the metapopulation paradigm whereby, as patches become smaller and more separated, rates of extinction increase and rates of recolonisation decrease (Hanski, 1997). It is also assumed that the remaining habitat is likely to be degraded as a consequence of high demand on the reduced resources and, therefore, individuals residing in these habitats could be abnormally stressed resulting in increased susceptibility to disease (Lafferty & Gerber, 2002). Reduced population size would increase the incidence of inbreeding which could promote a long-term loss in genetic diversity and hence reduced potential to adapt to infectious diseases (Lyles & Dobson, 1993). The risk of microbial control agents spreading from the site of application into environmentally sensitive areas will be increased in fragmented habitats because of greater edge effects (Lafferty & Gerber, 2002). It appears that environmental change has the potential to affect epidemiology, but these predictions need to be tested experimentally on insect–pathogen systems to gain an accurate measure of the overall impact. Currently such studies are lacking.

The spatial dynamics of pathogens have been extensively studied (Ostfeld *et al*., 2005) but not at the landscape level. Mechanisms of pathogen dispersal from an infected to an uninfected host are extremely varied ranging from direct contact (sexual diseases) and near-direct contact (pathogens released and then inhaled or ingested) to indirect contact via a vector (Ostfeld *et al*., 2005). In all cases, the probability of pathogen transmission decreases with increasing distance between susceptible hosts (except in the case of vertical transmission, when hosts may carry the seeds of their own destruction). Therefore, factors influencing the spatial distribution of pathogens, host and vectors are fundamental to disease dynamics. Work on viruses has highlighted the importance of spatial structure on pathogen survival and transmission. Habitat can influence the persistence of pathogens in the field; baculovirus in forested (sitka spruce and oak) areas retained infectivity longer than in an unshaded (heather) habitat (Raymond *et al*., 2005). The spatial scale of most host–pathogen studies is often at the field level or less. Few

empirical studies incorporate a landscape approach (Barta & Cagán, 2003). Consideration of the heterogeneous nature of the environment, embracing the challenges of the landscape scale, in which pathogens and hosts co-exist will be important in understanding how these systems will respond as the environment changes.

Corridors connecting remnant habitat patches are regarded as a conservation strategy (McCallum & Dobson, 2002). However, Hess (1996) developed a model, based on the classical Levins (1969) metapopulation structure, for pathogens in fragmented habitats which led to the conclusion that movement of individuals among populations is risky. However, McCallum and Dobson (2002) challenged this conclusion, recognising that not all pathogens are host specific and that a second host species may act as a pathogen reservoir. They extended the simplified model by Hess (1996) to examine cases where the pathogen infects both a rare host and a common reservoir species (McCallum & Dobson, 2002). From this revised model, it was concluded that habitat loss will cause extinction of the pathogen before loss of the host and, furthermore, while the pathogen may spread through corridors between habitat patches, the benefit of permitting recolonisation of vacant patches by the host counterbalances the cost of disease spread (McCallum & Dobson, 2002). In both the Hess and the McCallum and Dobson models, the pathogens were directly transmitted between hosts. Many entomopathogens are indirectly transmitted, but do form persistent reservoirs external to the host as well as in alternative host species. Consequently, the latter model is the most applicable, and increasing connectance between habitat fragments is likely to be beneficial for rare species.

The spatial complexity of population structure is an inherent component of source-sink metapopulation and metacommunity models (Hanski & Gilpin, 1991; Hanski & Simberloff, 1997; Namba *et al*., 1999; Rodriguez & Torres-Sorando, 2001). These models have demonstrated that habitat partitioning can promote co-existence of prey by relaxing apparent competition mediated by a shared predator (Holt, 1984) but could also result in extinction of the predator species (if prey productivity in a source population is insufficient to 'rescue' a deficient sink population) or the prey species (if the source is too rich giving rise to shared predation) (Namba *et al*., 1999). The number of sinks and spatial arrangement and connectivity between patches differentially influence persistence of populations in different trophic levels (Namba *et al*., 1999). The probability of establishment of a pathogen within a host patch is predicted to decrease with increasing spatial partitioning (fragmentation) and with decreasing host movement between patches (Rodriguez & Torres-Sorando, 2001). These models examine the role of trophic and spatial complexity on population persistence but there is little empirical evidence to support the model predictions. We believe that pathogens and their hosts could be exemplary systems for empirically testing theoretical models based on metacommunity structure.

In a recent empirical study, Tscharntke *et al*. (2002) examined the interactions between insects (grassland butterflies, legume and rape-feeding herbivores and their parasitoids) in a fragmented landscape. Habitat fragmentation differentially affected the interactions between species with varying community structure. Habitat fragmentation was more detrimental to specialised

butterflies than less specialised oligophagous and polyphagous herbivores (Tscharntke *et al.*, 2002). Species richness and abundance of polyphagous butterfly species was higher in small rather than large calcareous fragments and illustrates the importance of the landscape matrix within which the patches are embedded for less specialised species. Therefore, Tscharntke *et al.* (2002) conclude that an intermediate fragmentation conservation strategy (combining the advantages of both several small and single large fragment structures) could be beneficial in a human-dominated landscape. Further empirical studies are urgently needed to underpin the theoretical developments in this area. Briggs *et al.* (1995) stated that the extent to which pathogen dynamics will drive, rather than be driven by, the dynamics of their host is an important and open question in many natural systems. This is still a fundamental question today and understanding the role of pathogens in regulating host populations in fragmented habitats appears particularly challenging.

## Future perspectives

Conservation biology and the development of practical approaches to the prevention of species declines and extinctions are dependent on the integration of theories and disciplines. There is no doubt that in most studies on insect conservation, the roles of insect pathogens largely go unnoticed. Pathogens may not drive a host to extinction but they may result in population depletion to such a low density that other factors become important such as Alee effects or random stochastic events (Lafferty & Gerber, 2002). As discussed, some species are nationally scarce but locally abundant and transmission between hosts could be facilitated by local aggregations. Further research on spatial epidemiology of insect pathogens is required if we are to make realistic predictions of the impact of environmental change on host–pathogen dynamics.

There is currently much debate regarding the influence that a single mortality agent such as a pathogen can have on host regulation (Graham *et al.*, 2004). Host–pathogen relationships are complex and can be extremely subtle often varying both spatially and temporally; pathogen density within a host population is often underestimated because the pathogen remains undetected in symptomless individuals (Graham *et al.*, 2004) or studies are not of sufficient spatial or temporal scale to capture the role of pathogens on life-history traits.

We urgently need to apply, more widely, the wealth of molecular techniques available to document the diversity and prevalence of pathogens, in all their guises, within non-pest insect populations. Only then can we confidently comment on the current role of insect pathogens in population regulation and on the impact of future environmental regimes on host–pathogen interactions. Insect pathology has never been more relevant than in the current period of rapid environmental change.

## Acknowledgements

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