The ecology of host-parasitoid-pathogen interactions in natural lepidopteran populations

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Submitted in accordance with the requirements for the degree of
Doctor of Philosophy

The University of Leeds
School of Biology

May 2015

The candidate confirms that the work submitted is his own, except where work which has formed part of jointly-authored publications has been included. The contribution of the candidate and the other authors to this work has been explicitly indicated below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to the work of others.

Chapter 4 is written only by the candidate, but is based on a jointly-authored manuscript (Hicks et al., 2015) for which the candidate designed the research, collected and analysed the data, and wrote and revised the manuscript for this publication. The other authors supervised the research and provided feedback on the manuscript.

Acknowledgements

I would first like to thank my supervisor Steve Sait, who was instrumental in encouraging me to pursue a PhD. I am extremely grateful for his guidance, advice and encouragement throughout the PhD process, and for his commitment and hard work as a supervisor. Similarly, I would also like to thank my co-supervisor Rosie Hails for her excellent advice and support.

Helen Hesketh and Bob Possee provided me with vital training in insect-pathogen techniques, and were extremely generous with their time when providing me with ongoing advice and assistance during my time at CEH. A big thank you must also go to Tim Carthy for providing the Orkney winter moth work with its vast supplies of artificial diet over the years.

A number of people provided crucial assistance with field work throughout the PhD including Steven White, Liz Elliott, Stewart Jennings and my father Paul Hicks. I thank them all for their efforts, and hope they enjoyed themselves and the Orkney experience. Mark Shaw kindly identified the parasitoids we collected, and Keith Johnson provided us with some extremely useful local Orkney weather data. Thanks must also go to the RSPB and other land-owners for access to their land. I would also like to thank David Brooks and Rothamsted Research for providing me with long-term British winter moth data.

Thank you to all my friends and colleagues at the University of Leeds who provided scientific advice and support, as well as the social side of academic life. In particular I would like to mention Mark Goddard, Richard German, Richard Gunton, Marianne Mugabo, Peter Steward, Gorm Shackelford and Charlie Marsh, and wish them all well with their lives and careers.

Finally, but most importantly, I would like to thank all of my family, who always cared how things were going. In particular I would like to thank Tiffany Aslam for all her help and support over the last few years. A special thank you must also go to my late maternal grandparents, whose generosity and kindness played a huge role in enabling me to pursue my education to this extent. Finally, I would like to express my deepest gratitude to my parents for being who they are, and I dedicate this thesis to them.

Thesis abstract

Understanding population dynamics and the biotic and abiotic processes that drive and influence them is one the most fundamental issues in ecology, and is vital for successful ecological management of populations in the face of global environmental change. Species interactions influence population processes, and natural enemies in particular can have important impacts on vital rates, and are thought to be capable of population regulation. This thesis investigated the host-natural enemy interactions and spatio-temporal dynamics of two Lepidoptera-parasitoid-pathogen communities, which were used as model systems in which to explore these issues.

Using multi-year field data, potentially regulatory delayed density-dependent pathogen mortality was demonstrated in both the cyclical *Operophtera brumata*, but also unexpectedly in the non-cyclical *Abraxas grossulariata*. In addition, there was evidence that increasing temperature-related climatic conditions negatively influenced the interactions of *O. brumata* and its pathogen. Immune functioning was investigated in wild populations of the non-cyclical *A. grossulariata*, and unexpectedly found to be influenced by population density. Evidence consistent with trans-generational immune costs from defence against parasitism were also found. Scale-dependent effects of habitat fragmentation were investigated in the *A. grossulariata*-natural enemy community, and were found to have direct negative effects on host density at both small and large spatial scales, indirect negative effects on virus mortality at the largest scale, and, unexpectedly, direct positive effects on parasitism at small and medium scales. Finally, it was found that spatial population synchrony in *O. brumata* at the scale of Britain may be due to spatially correlated environmental processes, but that unlike *O. brumata* populations within mainland Europe there was no evidence for travelling waves in abundance within British populations, either driven by the mainland European travelling waves or occurring separately. The significance of these findings is discussed in the context of current research, and potential areas for future research are also addressed.

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1. General introduction

1.1 Population regulation

Understanding the factors that govern population fluctuations is one of the most basic ecological questions, and has frequently been framed in terms of the importance of density-dependent and density-independent regulatory mechanisms (Begon et al., 2006). This long-standing debate is often highlighted as going back over 50 years to the assertion of Nicholson (1954) that populations are primarily regulated by density-related changes in birth and death rates, and the counter assertion by Andrewartha and Birch (1954) that populations are primarily regulated by stochastic environmental processes.

A variety of potential density-dependent regulatory processes have been identified, but may be primarily divided into those density-related changes in demographic rates driven by intra-specific competition for resources, which can either act directly on individuals (Sibly & Hone, 2002; Stewart et al., 2005), or on their offspring via trans-generational effects (Beckerman et al., 2002; Inchausti & Ginzburg, 2009); and those density-related changes in demographic rates, primarily the rate of mortality, driven by variation in consumer-resource trophic interactions between herbivores and plants and prey/hosts and their natural enemies (Hudson et al., 1998; Krebs et al., 2001; Klemola et al., 2010; Kessler et al., 2012). Density-independent processes that may influence populations primarily involve environmental stochasticity, such as climatic fluctuations (Boggs & Inouye, 2012), but may also include density-independent effects of trophic interactions (e.g. Ylioja et al., 1999; Hassell, 2000; Solbreck & Ives, 2007).

However, it is now generally recognised that whilst all populations are ultimately *regulated* by density-dependent processes at least to some extent (because unrestrained long-term population growth in natural systems is unknown), density-independent factors are often also highly important in *determining* the actual abundance and spatial/temporal variation in

populations found in nature (Ziebarth et al., 2010). Therefore, the populations of most species are influenced by a mixture of both density-dependent and density-independent processes (e.g. Solbreck & Ives, 2007; Nowicki et al., 2009; Boggs & Inouye, 2012), which often act together in complex and system-specific ways, usually resulting in complex population dynamics (Benton et al., 2006). However, a number of species across a wide range of taxa display strikingly regular cyclical oscillations, despite the extensive environmental stochasticity of nature, suggesting the existence of powerful, density-dependent, regulatory mechanisms underlying their dynamics (Kendall et al., 1998). Cyclical species therefore represent excellent model systems in which to develop and test hypotheses about possible mechanisms regulating populations, given their relatively simple and regular population dynamics (Inchausti & Ginzburg, 2009). Consequently, cyclical species have received extensive attention in this regard for many decades (e.g. Elton, 1927; Korpela et al., 2014), and although other hypotheses exist, such as intrinsic regulatory mechanisms driven by density-related changes in intra-specific competition (Inchausti & Ginzburg, 2009), consumer-resource trophic interactions, particularly those involving host-natural enemy interactions, are generally believed to be the primary drivers of cyclical population dynamics (Krebs, 2011; Myers & Cory, 2013; Sundell et al., 2013; Korpela et al., 2014).

1.2 Natural enemies and population processes

A wide ranging and detailed understanding of population dynamics, and in particular the mechanisms regulating them, is important for many, if not all, applied ecological issues. Top-down effects from natural enemies, such as predators, parasitoids and pathogens, are typically of primary concern in such considerations. For example, the successful management of pest populations in natural systems, semi-natural systems and agroecosystems through biocontrol efforts (e.g. Hudson et al., 1998; Wu, 2010) relies heavily on the accurate prediction of pest's population responses over the long-term to the impacts of natural enemies used as biocontrol agents, such as pathogens and parasitoids (Tang et al., 2008; Hesketh et al., 2010). Failure to understand long-term responses of pest populations to introduced natural enemies can potentially result in worse management outcomes than simply the failure of biocontrol, with greater pest population pressure resulting

(Reilly & Elderd, 2014). Population harvesting has parallels with the effects of predation, and also interacts with the impacts of natural enemies (Hatcher & Dunn, 2011), and sustainable harvesting must avoid population crashes, extirpations and extinctions. Therefore, an accurate understanding of the impacts of harvesting on exploited populations, in terms of their long-term dynamics, is also crucial for their successful management (Mora et al., 2007; Brown, 2011; Ohlberger et al., 2014).

Natural enemies also play very important roles in the invasion of introduced species, where their absence can release introduced populations from regulation, facilitating invasions (Liu & Stiling, 2006). As invading species themselves, natural enemies such as parasites can have positive or negative impacts on invading and native species; for example, they can directly impact the demographic rates of native hosts through density-mediated effects, and indirectly impact native host behaviour and life-history traits through trait-mediated effects (Dunn et al., 2012; Vilcinskas et al., 2013). Similarly, the success of ecological restoration programs also depends heavily on accurately understanding and predicting the population responses of species intentionally reintroduced into ecosystems, along with those of existing species within the ecosystem, in the context of their interactions with key natural enemies (White & Garrott, 2005). At present, long-term dynamical surprises appear to be common in such situations, and so pose a serious problem for successful ecological management (Doak et al., 2008).

It is also important to consider natural enemies in the conservation and management of species and biodiversity. Natural enemies are important components of most ecosystems, maintaining biodiversity and influencing ecosystem structure and functioning, both directly through their impacts on prey/host populations, and also indirectly through their effects on their prey/hosts, which then interact with other of species leading to trophic cascades of influence (Hawkins et al., 1997; Hudson et al., 2006; Ritchie & Johnson, 2009). For example, removal of predators from communities can release prey populations previously regulated by predation, allowing them to greatly suppress the populations of their food plants, changing the entire ecological structure and functioning of communities (Terborgh et al., 2001). However, removal of apex predators can also release of mesopredator populations, leading to suppression of their herbivorous prey, and thereby releasing their food plants from regulation (Ritchie & Johnson, 2009). Consequently, successfully management of populations in general requires an effective understanding of their population dynamics and the factors that

have important influences on them, with natural enemies being a primary influence.

1.3 Host-natural enemy interactions and population regulation

Given the important mortality impacts that natural enemies have on many wild populations (Hawkins et al., 1997), there has been much theoretical research attempting to understand whether they are capable of population regulation, and in particular whether they are able to generate the oscillatory dynamics seen in cyclical species. This work largely began with the continuous-time predator-prey model of Lotka and Volterra (Volterra, 1926; Lotka, 1932), and the discrete-time models of Nicholson and Bailey (Nicholson & Bailey, 1935), inspired by insect-parasitoid systems with discrete generations. Both modelling frameworks made very simple assumptions, most importantly that there was a density-dependent relationship between the rate of enemy attack and prey/host density, representing a numerical response by a specialist natural enemy. Although largely biologically unrealistic in most other ways, the models indicated that natural enemies could regulate host dynamics, and generate oscillatory dynamics. However, the dynamics they produced were unstable (highly sensitive to any perturbation) unlike cyclical dynamics in nature, which typically display striking regularity despite environmental stochasticity (Krebs et al., 2001; Hogstad, 2005). It was subsequently found that allowing for heterogeneity in the risk of attack from natural enemies generally promotes dynamical stability (May, 1978), with spatial heterogeneities, such as prey refuges and habitat structure, thought be very common and important in this regard (Briggs & Hoopes, 2004).

There is also a well-developed theoretical literature investigating the effects of pathogens on host dynamics. This is largely based on the key work of Anderson and May (e.g. Anderson & May, 1978, 1979), whose models assumed certain key processes such as a threshold host population size for pathogen persistence, multiple pathogen generations in the environment, host resistance and recovery. Most importantly though, their models showed that pathogens transmitted by density-dependent processes should also be capable of regulating host population dynamics (Anderson & May, 1981), and generating population cycles (Anderson & May, 1980).

Conversely, models of pathogens transmitted by density-independent mechanisms, such as many sexually transmitted diseases (McCallum et al., 2001), cannot typically regulate host dynamics (Getz & Pickering, 1983). Again, heterogeneity in the risk of attack from pathogens has also been found to be very important for stabilising the dynamics of host-pathogen models (Hagenaars et al., 2004; Elderd et al., 2008).

Subsequent developments in the modelling of host-natural enemy dynamics have explored the implications of adding many different aspects of real systems. For example, the inclusion of different functional responses has allowed for modelling of more biologically realistic changes in the rate of predation/parasitism with prey/host density, representing, for example, Allee effects mediated by predation (McLellan et al., 2010). Similarly, in host-pathogen models different functional forms of transmission have been explored, showing how systems characterised primarily by density-dependent transmission may become unstable when also subject to a small degree of density-independent transmission (Ryder et al., 2007). Competition effects have also been explored. For example, in host-parasitoid models stronger intra-specific competition from unparasitised hosts has been shown to cause parasitoid extinction, whilst stronger intra-specific competition from parastised hosts was found to be stabilising (White et al., 2007). In predator-prey models, varying the importance of competing specialist and generalist predators has been found to produce variations in oscillatory dynamics similar to those seen in natural geographical gradients, providing a possible explanation for their cause (Hanski et al., 2001). Age-structure effects, particularly important for insect systems, have also been explored. For example, in host-pathogen models of insects, including age structure has important effects on dynamical outcomes, with time delays in host developmental periods influencing the length and stability of the dynamics produced (Briggs & Godfray, 1995).

In general though, depending on the properties of the specific host-enemy system being represented, a wide range of dynamics, similar to those seen in nature, are produced by most host-enemy models (Begon et al., 2006). This includes stable-point equilibria, chaos and the multigeneration-length population oscillations characteristic of cyclical species (e.g. Hassell, 2000; Hanski et al., 2001; Dwyer et al., 2004; White et al., 2007; Elderd et al., 2008; Ammunet et al., 2014; Korpela et al., 2014), which suggests that predators, parasitoids and pathogens can be capable of regulating population dynamics, and generating population cycles. In terms of cyclical

dynamics specifically, theory also indicates that their generation is best explained by delayed density-dependent interactions ('second-order processes'), such as those occurring from a lagged numerical response in specialist natural enemy populations to changing prey/host population abundance (Berryman, 2002; Turchin, 2003; Korpela et al., 2014).

Although less well explored, some theoretical research has also investigated the effects of multiple, functionally different natural-enemies on host dynamics. For example Hochberg et al. (1990) considered a host-parasitoid-pathogen system, and found a wide range of dynamical outcomes were possible, including the exclusion of either natural enemy, or the coexistence of both, which could then lead to stable equilibria, periodic cycles or chaos depending on the chosen characteristics of the natural enemies, primarily their relative competitive abilities. Similarly, Sait et al. (2000) combined analysis of laboratory data and a theoretical model of a host-parasitoid-pathogen system to show that the order in which different enemies invaded the system could generate alternative cyclical dynamics with different generation lengths. More recently, Preedy et al. (2007) modelled a host-parasitoid-parasitoid-pathogen system and found that the presence of the pathogen acted to promote the diversity of the system, but that again a wide range of dynamics, including cycles, were possible depending on assumptions made about the competitive abilities of the natural enemies. Finally, in host-pathogen models based on a cyclical insect, Dwyer et al. (2004) and Allstadt et al. (2013) have both shown that the inclusion of a generalist predator produces periods of cyclical and noncyclical behaviour in the host's dynamics, matching the temporal variability of the system being modelled in nature.

Currently though, empirical evidence in support of theoretical modelling results indicating that specialist natural enemies may regulate the dynamics of their prey/hosts, and in particular generate cyclical dynamics through delayed density-dependent interactions, is inconsistent and often lacking. Some compelling experimental and observational evidence suggests that the 10-year cycle of the snowshoe hare (*Lepus americanus* Erxleben) in North America is driven primarily by predation, but with important indirect effects of predation-induced stress likely accounting for the delayed density-dependent nature of the interaction through maternal effects on reproduction (Peckarsky et al., 2008; Krebs, 2011). Similarly, some compelling experimental and observational evidence suggests that delayed density-dependent parasitism may drive population cycles in the autumnal

moth (*Epirrita autumnata* Borkhausen) (Klemola et al., 2010; Klemola et al., 2014). However, for other cyclical species the picture is less clear, with empirical research sometimes indicating support for alternative hypotheses, such as induced plant defences and maternal effects of crowding (e.g. Massey et al., 2008; Inchausti & Ginzburg, 2009), or failing to find significant (direct or lagged) effects of natural-enemy driven mortality on changes in host abundance (e.g. Hagen et al., 2010; Schott et al., 2010).

In many cases though there is simply a lack of adequate empirical data, specifically long-term population and natural enemy data, gathered at sufficiently large spatial-scales, necessary to adequately test hypotheses of natural enemy regulation in natural systems (Myers & Cory, 2013). Such is uncommon for predator-prey and host-parasitoid systems (e.g. Sundell et al., 2013; Klemola et al., 2014), and very rare for host-pathogen systems (e.g. Kukan & Myers, 1999). Additionally, research into the influence of natural enemies on their prey/host's dynamics has overwhelmingly focused on cyclical species (Solbreck & Ives, 2007; Myers & Cory, 2013), and there is consequently a substantial lack of long-term large-scale empirical data examining host-enemy dynamics in non-cyclical natural systems. Logically this problem is even more acute for research into the effects of multiple natural enemies on prey/host dynamics, particularly of different functional types (e.g. pathogens and predators/parasitoids), with empirical research having been largely previously restricted to laboratory systems (e.g. Begon et al., 1996; Sait et al., 2000). Therefore, there is a need for long-term and large-scale studies to adequately investigate the hypotheses suggested by models of natural host-enemy systems, in order to fully determine how important host-enemy interactions are for population regulation in real systems, and how their importance may vary, and why.

1.4 Spatial structure, spatial scale and spatio-temporal dynamics

Species exist within spatially structured environments (Tscharntke & Brandl, 2004), and it is clear that such spatial structure can have important influences on the interactions between species, with interacting species responding differently to the same spatial contexts (Cronin & Reeve, 2005;

Ostfeld et al., 2005; Gripenberg & Roslin, 2007; Martinson & Fagan, 2014). Given the ongoing global effects of habitat loss and habitat fragmentation (Sala et al., 2000), it is crucial to understand the influence that habitat structure, and in particular changes to habitat structure, have on populations and their natural enemies, because this is likely to impact on their dynamics leading to long-term changes. This is particularly important for many applied areas of ecology. For example, in terms of conservation and biodiversity management, habitat fragmentation can disrupt host-enemy interactions, leading to trophic cascades through direct and indirect impacts on interacting species, which can affect ecosystem structure and functioning (Terborgh et al., 2001; Fenoglio et al., 2012). Habitat structure also affects the economically hugely valuable pest control services provided by natural enemies (Losey & Vaughan, 2006). For example, the amount of complex habitat surrounding agricultural systems can have big effects on natural enemy populations, primarily benefitting them as it increases (With et al., 2002).

Closely related to habitat structure is the issue of spatial scale. The influence of habitat structure, and therefore habitat fragmentation, on host-natural enemy interactions will be dependent upon the scale at which natural enemies and their prey/hosts respond to spatial structure (Roland & Taylor, 1997; Elzinga et al., 2007). Species experience landscapes at different scales dependent on their dispersal abilities and foraging ranges, which also may be influenced by other traits such as their trophic position and specialisation (Tscharntke & Brandl, 2004). However, these relationships are often variable, making general predictions difficult (Martinson & Fagan, 2014; Hicks et al., 2015). The spatial scale at which population dynamics are investigated is also key, with certain spatial processes only apparent at much larger scales, from the landscape to the continent (see below). Consequently, it is important to take a multi-scale approach when trying to understand host-enemy dynamics in nature (Tscharntke et al., 2007; Chaplin-Kramer et al., 2011; Hicks et al., 2015).

One of the most well developed theories of spatial dynamics is the metapopulation framework (Hanski, 1998). Initially developed for single populations, metapopulation theory explains how restricted (non-global) migration between local populations with unstable dynamics (i.e. prone to extinction) can allow the regional persistence of a population of populations - a metapopulation (Hanski, 1998). Metapopulation models have also been extended to host-enemy systems, and have, for example, helped to explain

how heterogeneous spatial structure and asynchronous local dynamics can promote persistence of host-parasitoid systems (Hassell, 2000; Briggs & Hoopes, 2004). Similarly, metapopulation models applied to host-pathogen systems show important effects of spatial structure, highlighting how the connectivity and density of host populations are critical for determining whether pathogens can persist within host metapopulations (Thrall & Burdon, 1997), and for determining whether epidemics can occur in spatially structured, interconnected populations or not (Grenfell & Harwood, 1997), with increased connectivity typically enhancing the spread of diseases and promoting epizootics (Hess, 1996). More generally, other spatial epidemiological models have also shown how important spatial structure is for pathogen evolution, with increasing connectivity between populations tending to enhance the virulence of diseases, affecting host-pathogen dynamics and promoting epidemics (Boots & Sasaki, 1999; Boots et al., 2004).

Currently though, it appears difficult to predict the responses of host-natural enemy interactions to changes in habitat structure, such as those driven by habitat loss and fragmentation, because the observed responses show extensive variation and often little consistency (Tylianakis et al., 2008; Martinson & Fagan, 2014). Overall, host-parasitoid interactions appear to respond most consistently to increasing loss and fragmentation of habitat, typically declining in strength (Martinson & Fagan, 2014), although this is not always the case even when expected based on species' traits (Hicks et al., 2015). Predator-prey and host-pathogen interactions show a wider range of variation in their typical responses to increasing habitat change though (Martinson & Fagan, 2014), highlighting how important it is to understand the effects of species- and system-specific variation in interacting species responses to habitat structure. At present though, there has been a particular lack of attention given to the effects of habitat fragmentation on host-pathogen interactions, and apparently no attention to fragmentation's effects on species interactions between hosts, pathogens and other functionally different natural enemies. Therefore, empirical studies at large spatial scales, and considering the effects of habitat structure across a range of spatial scales, are needed to improve understanding of the responses of host-natural enemy systems to habitat fragmentation.

The spatial scale at which population dynamics are observed is also an important consideration when attempting to understand population dynamics. Some species show strikingly regular patterns in their large-scale

spatio-temporal dynamics, in particular those species displaying population cycles. For example, spatial synchrony in the population dynamics of cyclical species from a range of taxa, including mammals, birds and insects, can extend across landscapes, regions and even continents (Bjornstad et al., 1999; Klemola et al., 2006; Krebs et al., 2013; Tenow et al., 2013), indicating the occurrence of important processes not apparent from local scale dynamics alone. In addition, other more complex patterns in spatio-temporal dynamics are also known, in particular periodic travelling waves (Sherratt & Smith, 2008). These population processes have now been reported in large-scale population studies on a number of cyclical species from a similarly diverse range of taxa as large-scale spatially synchronous dynamics are known from, and across a similarly wide range of spatial scales from the landscape to the continent (Ranta & Kaitala, 1997; Moss et al., 2000; Bjornstad et al., 2002; Tenow et al., 2013; Berthier et al., 2014). It is therefore important to understand these large-scale processes and their drivers, in order to fully understand species' dynamics for ecological management of systems.

Spatial population synchrony is thought to be driven by either biotic mechanisms, which involve the dispersal of either individuals of the spatially synchronous populations themselves (Bjornstad et al., 2002), or other organisms that have important influences on the dynamics of the spatially synchronous populations, (e.g. through important trophic interactions such as predation) (Ims & Andreassen, 2000); or abiotic mechanisms, which involve the synchronising influence of spatially correlated environmental fluctuations known as Moran effects (Moran, 1953). In addition, more recent work indicates that Moran effects may also act through trophic interactions to generate spatial synchrony (Haynes et al., 2009; Haynes et al., 2013). However, although theoretically well described (Bjornstad et al., 2002), the relative importance of these processes in natural systems is still debated (Vasseur & Fox, 2009; Fox et al., 2011; Fox et al., 2013), and it is generally difficult to rule out the influence of the different possible mechanisms in most studies, and this is not always attempted (e.g. Tenow et al., 2007).

Understanding of travelling waves is also still very much developing, and is largely based on the results of theoretical models, which indicate a number of possible mechanisms including hostile boundaries, invasions, heterogeneous habitats and migration between subpopulations (reviewed by Sherratt, 2013). Models indicate that cyclical dynamic are generally needed for travelling wave phenomena to occur, and so host-enemy population

cycles are generally believed to be key to these processes (Sherratt & Smith, 2008). However, empirical exploration of the model-driven hypotheses about travelling waves are not common. Again, a major problem is the difficulty of obtaining long-term and large-scale data relevant to the predictions made by hypotheses, such as that travelling waves in cyclical species' abundance can be produced at the boundaries to hostile landscape features (Sherratt, 2013). Given the limited empirical exploration of travelling waves, there is also a lack of comparison between cyclical species' large-scale spatio-temporal dynamics in different regions. Exploring and understanding geographical variation in the large-scale dynamics of species may help to highlight driving mechanisms though, and factors influencing these processes.

1.5 Host defences

Animals are not just passive actors in their interactions with natural enemies, but have evolved a range of mechanisms by which they can defend themselves and resist attack. These include defensive and escape behaviours, morphological adaptations and defensive chemicals (Nishida et al., 1994; Van Buskirk & McCollum, 2000; Smedley et al., 2002; Kortet et al., 2007; Parker et al., 2010), allowing resistance against predators, parasitoids and pathogens. Vertebrates and invertebrates also possess complex immune systems (Strand, 2008; Schulenburg et al., 2009), allowing them to resist attacks from pathogens. However, both the evolution and expression of defensive traits against natural enemies are costly, and trade-offs with other important life-history traits therefore also occur (Kraaijeveld & Godfray, 1997; Kraaijeveld et al., 2002; Stoehr, 2007; McNamara et al., 2013). Dependent on the balance between the costs of defence and the risks of attack, differential investment in defensive traits may therefore affect host-natural enemy interactions, which could influence host-enemy dynamics. Such processes may occur via both evolutionary processes and phenotypic plasticity, and therefore over a range of ecological and evolutionary timescales. For example, modelling has shown that evolutionary variation in the strength of anti-predator defences in response to variation in risk of attack can promote the coexistence of predator-prey systems, and may dampen population fluctuations when evolution is rapid (Yamamichi et al., 2011).

Similarly, given the relationship between host density and risk of pathogen infection (Anderson & May, 1981; McCallum et al., 2001), variation in population density itself may act as a cue by which phenotypically plastic defensive investment is varied during the lifetime of a host to ensure investment in resistance is made when most needed (Wilson & Reeson, 1998). Modelling indicates that this process can have important implications for host dynamics, both stabilising host-pathogen dynamics under certain conditions (e.g. when there is a cost to pathogen resistance), and destabilising them under other conditions (e.g. when the length of time between changes in density and hosts' responses is short or absent) (White & Wilson, 1999; Reynolds et al., 2011). Theory also indicates that high amplitude population cycles are possible in host-pathogen interactions when density-dependent resistance is present, but only when the mechanism carries no cost (White & Wilson, 1999). Linking density-driven changes in resistance at both ecological and evolutionary timescales, other modelling had shown that if there is a small cost associated with phenotypic plasticity in resistance, and specialised non-plastic resistant genotypes exist, then the plastic phenotype will not be able to persist (Yamamichi et al., 2011). Furthermore, where both rapid evolutionary change in resistance and phenotypic plasticity in resistance occur, modelling indicates that phenotypically plastic changes in resistance tend to stabilise population dynamics, and dampen oscillations, more strongly than rapid evolution of resistance (Cortez, 2011; Yamamichi et al., 2011).

Therefore, resistance can have important effects on host dynamics in the context of host-enemy interactions, and population processes such as changes in host density may also influence host resistance and the resulting host-enemy dynamics. However, the vast majority of work in this area has been carried out via laboratory studies. Laboratory studies are able to control the unwanted effects of many factors, but are not necessarily representative of how such processes work in natural systems, with all their messy complexity, abiotic influences, and complex biotic interactions. Consequently, there is a need to study resistance and its functioning in wild systems, to understand these issues, and the implications they may have for host-enemy dynamics in nature (Pedersen & Babayan, 2011).

1.6 Insects and their natural enemies as model systems

Insects are particularly useful organisms for studying population dynamics in natural systems because they have relatively short generation times, and abundant, well distributed species can usually be selected for study within most environments. Consequently, it is often possible to gather large amounts of useful data from insect systems, facilitating studies of population dynamics. Insects are also an intrinsically important group. In biodiversity and conservation terms they comprise the majority of all known species (Gaston, 1991), performing vital functions in almost all ecosystems (Begon et al., 2006). Economically, insects cause vast amounts of agricultural damage as crop pests, but as natural enemies of crop pests they also supply hugely valuable pest control services (Losey & Vaughan, 2006). They therefore provide model systems in which to explore general ecological processes, as well as being worthy of greater research and understanding on their own merit for their biodiversity and economic importance.

Insects are also known to suffer often substantial levels of mortality in all life stages from a huge range of predators, parasitoids and pathogens (Hawkins et al., 1997; Roy et al., 2009). Predators of insects include vertebrates such as rodents (Hansen et al., 2009) and birds (Ries & Fagan, 2003), and invertebrates such as predatory beetles (Raymond et al., 2002b) and spiders (Denno et al., 2002). Like predators, most parasitoids eventually kill their hosts (Hawkins et al., 1997), but in common with pathogens, parasitoids (particularly endoparasitoids) may also be resisted and killed by hosts through their immune defences (Kraaijeveld & Godfray, 1997). Insects also suffer from a broad range of pathogens including viruses (Graham et al., 2004; Graham et al., 2006), fungi (Liebhold et al., 2013), bacteria (Graham et al., 2011) and microsporidia (Vilcinskas et al., 2013). These organisms can cause a range of negative, nonlethal impacts on insect health (Sait et al., 1994; Hesketh et al., 2012), as well as causing the death of their hosts, leading to potentially high levels of host mortality within populations (Kukan & Myers, 1999; Graham et al., 2004; Liebhold et al., 2013). Like parasitoids, pathogens may also be resisted via host immune defence mechanisms (Strand, 2008).

Therefore, insects represent excellent model systems in which to investigate the full range of possible host-natural enemy trophic interactions, and their implications for population processes. The original research in this thesis is based on the study of herbivorous insects, and their interactions

with their parasitoid and pathogen natural enemies in natural systems. However, wherever possible wider links to general ecological principles and conclusions are also made.

1.7 Overview of the research

1.7.1 The study system, prior work and data sources

In this thesis all data comes from the study of one or both of two host Lepidoptera, the winter moth (*Operophtera brumata*, (L.) Lepidoptera: Geometridae) and the magpie moth (*Abraxas grossulariata*, (L.) Lepidoptera: Geometridae), as well as the dominant larval-stage parasitoid and viral pathogen of each host within Orkney. The research contained in this thesis has built on much previous research on the winter moth and magpie moth host-natural enemy communities on Orkney. This research initially began in the early 2000s in an attempt to understand what was causing widespread winter moth deaths in some abundant larval populations on heather plants in Orkney. The causal agent turned out to be a baculovirus, and more specifically the nucleopolyhedrovirus (NPV) OpbuNPV. Subsequently in 2003, systematic field collections of winter moth larvae at 14 sites were made using a standardised quadrat-transect design (as detailed in chapter 2). This allowed the quantification of larval density, and through individual rearing of larvae on artificial diet it was possible to record each larva's fate (i.e. whether it reached the imaginal stage, or died due to OpbuNPV infection, parasitoid attack or for unknown reasons). This process therefore yielded data on both the host population and the impact of its natural enemies.

These field collections have been repeated yearly ever since, and in combination with additional field work on Orkney have formed the basis for two previous PhDs, and four published papers. The first of these PhDs, 'The impact of viral pathogens upon host lepidopteran populations: the Winter moth and its natural enemies' (Graham, 2006), was undertaken by Dr Rob Graham, and focused largely on the molecular aspects of the pathogen community infecting the winter moth (the magpie moth did not appear in field samples until 2005). This work produced four papers, which detailed how high levels of spatial genetic variability in OpbuNPV exist across Orkney (Graham et al., 2004), as well as characterising and analysing the genetic

sequences of a number of additional pathogens attacking winter moth larvae, including three novel species of reovirus (one of which appeared to be vectored by the parasitoid *Phobocampe tempestiva* (Holmgren, Hymeoptera: Ichneumonidae)) (Graham et al., 2008), and two novel cypoviruses (Graham et al., 2006; Graham et al., 2007). Work presented in the thesis also analysed the level of vertical (trans-generational) transmission of both OpbuNPV and the novel cypoviruses, and found substantial levels of vertical transmission, leading to covert infections, in OpbuNPV, and to a lesser extent in the cypoviruses.

The second of the PhDs, 'Spatial ecology of an insect host-parasitoid-virus interaction: the winter moth (Operophtera brumata) and its natural enemies in Orkney' (Harold, 2009), was undertaken by Dr Simon Harold, and focused largely on the spatial aspects of the interactions between the winter moth and the magpie moth and their respective pathogen and parasitoid natural enemies. In particular, one study in the thesis looked at the relationship between winter moth instar stage and risk of natural enemy attack within a single field site over three years at a fine temporal scale, and found a greater risk of attack from all natural enemies for mid-instar larvae, which also displayed a more aggregated spatial distribution than other larval stages, potentially in response to this elevated risk of attack. However, no density-dependent parasitism or infections were detected in the data. Another study based on an outbreaking population of winter moth at a different single site found that winter moth larvae displayed a wave-like pattern of local dispersal away from the outbreak's epicentre over three years, potentially driven by the high rate of natural enemy attack. In data from this site some density-dependence in OpbuNPV transmission and parasitism was evident, but only during the outbreak. However, similar patterns were not evident for the magpie moth population at the site, which was did not display outbreaking behaviour. A further study sampled both species' larval populations at 36 sites across Orkney over two years, and indicated that winter moth populations may be synchronised at a regional scale, but that neither species responded significantly to changes in habitat availability. However, winter moth populations were positively associated with increasing elevation, and magpie moth populations with south-facing aspects and taller heather, suggesting the importance of microclimate for their distribution. There was also no evidence that local scale patterns in host-natural enemy interactions reflected regional-scale patterns, or that there was any density-dependence in the host-enemy relationships across any scale over the two years. Finally, spatial population genetic analysis

showed that magpie moth populations appear to have reached Orkney from the north east coast of Scotland, and that genetic variation in populations on Orkney may be related to AbgrNPV prevalence, potentially signalling significant evolutionary effects of differential mortality from AbgrNPV in magpie moth populations on Orkney.

Therefore, from 2003-2014 the 14 core sites were sampled yearly, alongside other sites sampled as part of shorter-term PhD studies. Previously, no more than 3 years' worth of this data has collectively been analysed, and this thesis makes use of this data in full for this first time, along with additional data collected from both the same and different sites on Orkney during the summers of 2012, 2013 and 2014. Chapter 2 uses data on both hosts and their natural enemies, whilst chapter 3 and 4 only use data on the magpie moth and its natural enemies, and chapter 5 only uses data on the winter moth. For chapters 2, 3 and 4 all data were gathered from field sites around Orkney within heather (*Calluna vulgaris* L.) habitats, where the larvae of both the winter moth and the magpie moth feed on heather plants, whilst data for chapter 5 were obtained from Rothamsted Research, and consisted of long-term light trap counts for the winter moth, gathered as part of the Rothamsted Insect Survey, a network of insect light traps operated in various habitats and locations around Great Britain ('Britain') between 1968 and 2012.

1.7.2 Chapter 2

Although theoretical models of predator-prey (Hanski et al., 2001; Korpela et al., 2014), host-parasitoid (Hassell, 2000; Ammunet et al., 2014) and host-pathogen interactions (Dwyer et al., 2004; Fuller et al., 2012) indicate that natural enemies should be capable of regulating host populations, it is still generally unclear how important a role they play in most species' dynamics. This is the case both for non-cyclical species, where little attention has generally been directed in understanding natural enemy regulation, but also for cyclical species, where most attention has been directed (Inchausti & Ginzburg, 2009; Myers & Cory, 2013; Barraquand et al., 2014). This lack of clarity is primarily due to a lack of adequate empirical data, particularly data collected over multiple years and at large spatial-scales, with which to test the hypothesis that natural enemies can regulate population dynamics. This issue is especially acute for understanding the regulation of host-pathogen

and host-multi-enemy systems, where previous empirical research is rare, and where present has been largely restricted to short-term small-scale field studies (e.g. Dwyer et al., 2000), or laboratory studies (e.g. Sait et al., 2000).

Therefore, in chapter 2, multi-year landscape-scale data on host-parasitoid-pathogen interactions in a cyclical and a non-cyclical insect host (the winter moth and the magpie moth respectively) were analysed, in order to answer the question are natural enemies (specifically in this case pathogens and parasitoids) capable of host regulation in natural systems, and what is the relative importance of functionally different natural enemies for regulation of shared hosts? More specifically, the study also aimed to answer the question are pathogens (in particular) and/or parasitoids capable of generating the delayed density-dependent mortality thought necessary to generate cyclical dynamics, something not previously demonstrated for pathogens in natural insect populations? Also, is there variation in the strength and importance of direct and delayed density-dependent mortality from functionally different natural enemies, in the two taxonomically similar cyclical and non-cyclical species, which share the same environment, and might this indicate possible causes for their different dynamics? Density-independent processes may also influence regulation of populations, but this is rarely investigated in terms of the impacts on host-enemy dynamics, and so the influence of climatic factors on the interactions between the cyclical and non-cyclical hosts and their natural enemies was also investigated to see how important these effects were. In addition, winter moth dynamics have previously been studied in different habitats in relation to the possible role of parasitoids in driving their cyclical dynamics. Therefore, the study also aimed to answer whether the winter moth displays similar or contrasting dynamics in different habitats, and whether the similarity or differences in the observed dynamics may be related to the different natural enemies regulating their dynamics in different habitats.

1.7.3 Chapter 3

Immunological functioning has important effects on the interactions between hosts and their parasitic natural enemies (Tompkins et al., 2011). Population dynamics primarily consist of changes in population abundance or density over time, and there can be important interactions between these changes

and plastic changes in host resistance through altered investment in defences, particularly immunity (Reeson et al., 1998). Density-dependent resistance is known to occur in cyclical insects (Wilson & Cotter, 2009), and may ultimately affect their population dynamics through its effects on regulatory host-pathogen interactions (White & Wilson, 1999; Reynolds et al., 2011). However, it is unclear whether the same processes also occur in non-cyclical species, and so whether the process may be an important consideration when trying to understand population dynamics more generally. The functioning of density-dependent resistance in natural systems has also been poorly investigated. This is because most previous research has been largely based on the study of a relatively small number of cyclical species, and almost exclusively in laboratory-based studies. However, natural populations are subject to extensive environmental and genetic variation, as well as being embedded within a web of species interactions, which may influence the functioning of immunity in nature (Kraaijeveld & Godfray, 1997, 1999). Therefore, it is also necessary to conduct 'wild immunology' studies using natural populations (Pedersen & Babayan, 2011), something which is only starting to occur. Studies of wild immunology will enable immune functioning to be investigated under more realistic conditions, and ultimately help improve understanding of its importance for population dynamics.

Therefore, in chapter 3 immune functioning in a non-cyclical insect (the magpie moth) was investigated in wild-caught populations, in order to answer the question of whether density-dependent resistance is important for non-cyclical species, and how important the phenomenon may be in such species' populations under natural conditions. By investigating the correlations between natural enemy mortality (from a pathogen and a parasitoid) in the current and previous year it was also possible to explore whether the risk and/or non-lethal impacts of natural enemy attack may have direct or delayed effects on host immunity, helping to improve understanding about the effects of natural enemies on immunological functioning in natural systems.

1.7.4 Chapter 4

All species exist within a spatial context which has important and pervasive influences on their population dynamics and interactions with other species

(Hanski, 1998; Gripenberg & Roslin, 2007). One of the most important ways that spatial processes affect populations and species interactions is through the structure of the habitat within which they exist, and the most important influence affecting habitat structure is human-driven land-use change and the resulting loss and fragmentation of habitat (Sala et al., 2000). However, although this topic has received a great deal of attention for its obvious conservation importance, the primary focus has been on the responses of species diversity and abundance (Fahrig, 2003). Less attention has been given to the effects of habitat fragmentation on species interactions, but in particular very few studies have addressed the responses of host-pathogen systems to habitat fragmentation, or the responses of host-enemy systems involving multiple functionally different natural enemies. Natural enemies are expected to respond to habitat structure in different ways and at different scales though, depending on their characteristics (Holt et al., 1999; Tscharntke & Brandl, 2004; Ostfeld et al., 2005), which could result in complicated and contrasting responses in host-enemy communities.

Therefore, in chapter 4 the effects of habitat fragmentation on a host-parasitoid-pathogen system (the magpie moth and its pathogen and parasitoid) were investigated in order to answer the question how do functionally different natural enemies, particularly pathogens, attacking a shared host respond to habitat fragmentation, and at what spatial scale do they respond? The host's responses were also investigated, and the question of whether the enemies' responses to fragmentation were likely due to direct effects or indirect, host-mediated effects, was also addressed.

1.7.5 Chapter 5

Many cyclical species display large-scale dynamical patterns, including extensive spatial synchrony and periodic travelling waves in abundance (Bjornstad et al., 1999; Sherratt & Smith, 2008). Climate, host dispersal and dispersal of regulatory natural enemies may all be important influences driving spatially synchronous dynamics (Bjornstad et al., 1999), while a number of processes may drive travelling waves, with most attention focusing on hostile habitat boundaries at present (Sherratt, 2013; Tenow et al., 2013). However, the possible causes of spatial population synchrony are difficult to test, are not always explicitly addressed in studies (e.g. Tenow et al., 2007), and spatially synchronous dynamics in species are rarely

compared in different regions, where there might be different driving mechanisms. In addition, empirical exploration of the influence of large-scale habitat boundaries on travelling waves has been very limited to date, with most research based on either demonstrating the existence of travelling waves (e.g. Tenow et al., 2013), or modelling them in theoretical systems. Long-term and large spatial-scale population datasets are difficult to obtain because of the logistical effort needed, and so empirical studies into these issues are rare.

In chapter 5 an analysis of a long-term large-scale dataset on the abundance of the cyclical winter moth within Britain is presented. The species is known to display extensive spatial synchrony in mainland Europe, believed to be caused by abiotic factors, but this has not been explicitly tested previously. Therefore, this question is addressed via a comparison of the extent of spatial synchrony in winter moth populations to the extent of spatial synchrony in potentially synchronising environmental variables within Britain. In addition, continental scale travelling waves in winter moth abundance are known to occur every 9-10 years across mainland Europe, but like all travelling waves at present, it is not known how they are generated, or whether British winter moth populations are a part of this phenomenon. Whether or not the travelling waves reach Britain has implications for the validity of the hostile boundary hypothesis though, and this question is therefore addressed. The question of whether travelling waves in winter moth abundance occur within Britain is also addressed for the first time, and the results related to the hostile boundary hypothesis. More generally chapter 5 also allows an investigation of the long-term (44 years) large-scale population dynamics of the winter moth within Britain, something that has previously only occurred in Fennoscandia.

1.7.6 The ecology of Orkney and heather moorlands

The Orkney Islands is an archipelago of approximately 70 islands, which begin approximately 10 km from the most north-easterly point of the Scottish mainland (Fig. 1.1). Field work was restricted to the Mainland (the largest island at 523 km^2), Hoy (the second largest island at 143 km^2) and Rousay (the fifth largest island at 49 km^2). All three islands possess large areas of heather-dominated habitat, covering 19.8%, 78% and 45.7% of their areas respectively (Fig. 1.1). The heather-dominated habitats are

primarily in the form of extensive heather moorlands, although there are also many hundreds of small, discrete patches of heather varying in size from a few hundred metres to a just a few metres across (Fig. 1.1) (Hicks et al., 2015). Outside of the heather-dominated habitats the overwhelming majority of the remaining land-area consists of improved grasslands and pasture, used for livestock grazing. All three islands are also without any forests, and there are only a few very small patches of woodland and plantation. The Orkney heather host-parasitoid-pathogen community of the winter moth and the magpie moth is therefore a relatively simple model tritrophic community in which to explore the ecological questions in this thesis.

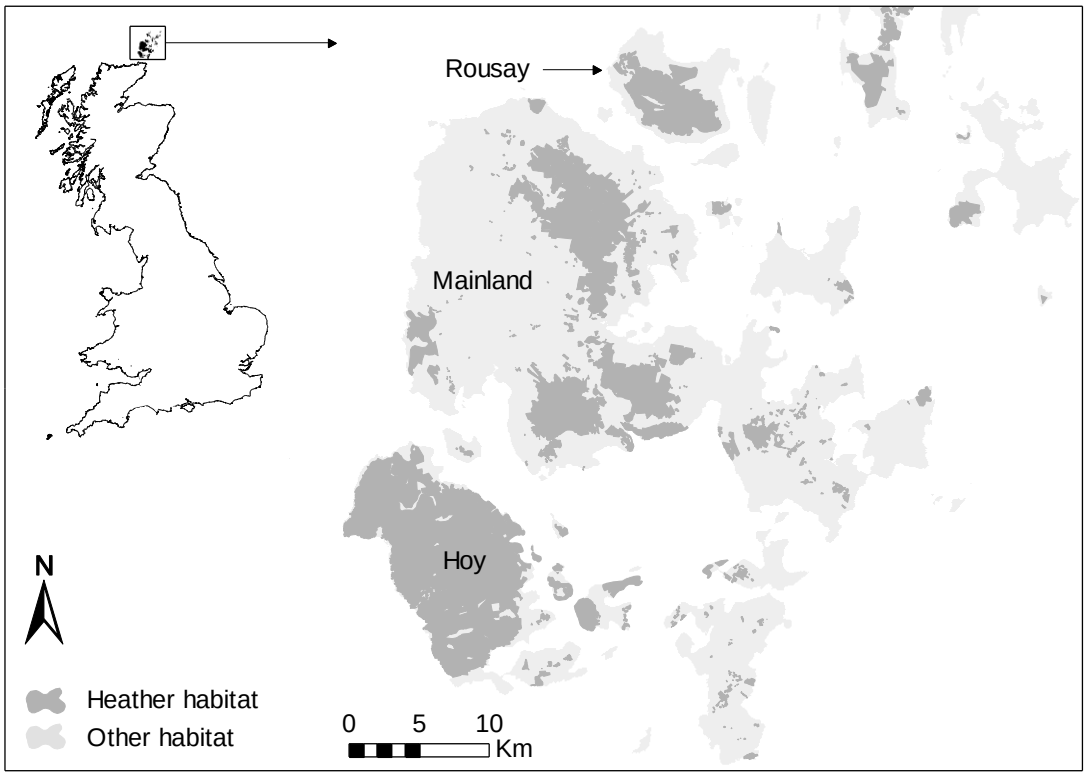

Figure 1.1 Distribution of long-term sampling sites and heather habitat on Mainland Orkney, Hoy and Rousay.

Moorland habitats are unique environments found throughout the world in the uplands of temperate regions including Scandinavia, Russia, New Zealand, Japan and Tazmania (Holden et al., 2007). They are characterised by being open areas with acidic or strongly base-deficient soils including peat, but the plant communities found on moorlands within and between regions vary substantially, with moorlands often known by their dominant vegetation, such as heather moorlands (Holden et al., 2007). In Great Britain, moorlands cover approximately 38% of Scotland and 5.5% of England and Wales. However, 75% of the world's heather moorlands are found within UK uplands (Holden et al., 2007), and are therefore of global conservation importance (Thompson et al., 1995). Heather moorlands are characterised by a blanket coverage of common heather (Gimingham, 1972), and form the dominant habitat within Orkney moorlands (Whitelaw & Kirkpatrick, 1997). Therefore, the research in this thesis contributes to the general understanding of the ecology of these globally important habitats.

Heather plants typically live for 30 or more years, passing through what can be described as four phases: a pioneer phase as newly established seedlings begin to dominate ground cover (3-10 years), a building phase as a heather canopy is formed excluding other plants (7-13 years), a mature phase dominated by woody growth and a reduction in canopy cover (12-28 years), and a final degenerate phase as the central branches of plants die and the canopy becomes patchy and very open (Gimingham, 1985). Heather plants represent a relatively nutrient-poor food resource for insect herbivores, with a relatively high carbon:nitrogen ratio and high levels of defensive compounds (Kerslake et al., 1996). However, they are utilised by a range of lepidopteran (and other herbivorous insect) species as a larval food plant (Fielding & Coulson, 1995), and a positive relationship is known to exist between heather plant-height and the abundance and diversity of lepidopteran species (Haysom & Coulson, 1998).

1.7.7 The winter moth and its natural enemies

The winter moth (Fig. 1.2) is a polyphagous, univoltine geometrid, which feeds predominantly on deciduous trees and dwarf shrubs, including a number of birch species (*Betula* spp.) pedunculate oak (*Quercus robur* L.), sitka spruce (*Picea sitchensis* Bong), various currant species (*Ribes* spp.) (Allan, 1979). However, whilst most research on winter moth ecology has

focused on populations in deciduous forests (e.g. Hogstad, 2005; Schott et al., 2010; Vindstad et al., 2010; Jepsen et al., 2013), winter moth are also known to feed on common heather (Kerslake et al., 1996; Kerslake & Hartley, 1997), and in chapter 2 this thesis presents the first long-term study of winter moth population dynamics and host-enemy interactions in heather moorland habitat. The winter moth occurs throughout Britain, and is widely distributed throughout the holarctic ecozone (Macphee, 1967) as a native species in Europe and Asia (Jepsen et al., 2008), but an invasive in North America (Embree, 1966). The winter moth is also extremely cold tolerant, with its northern distribution only climate limited by winter temperatures lower than -33°C (Macphee, 1967). Following budburst larvae emerge from eggs during spring, and typically feed on one food plant whilst developing (Roland, 1994) (Fig. 1.3). Larvae pass through 5 instars before pupating in the soil in late summer, with adults emerging to mate and lay eggs during the winter (Roland, 1994) (Fig. 1.3).

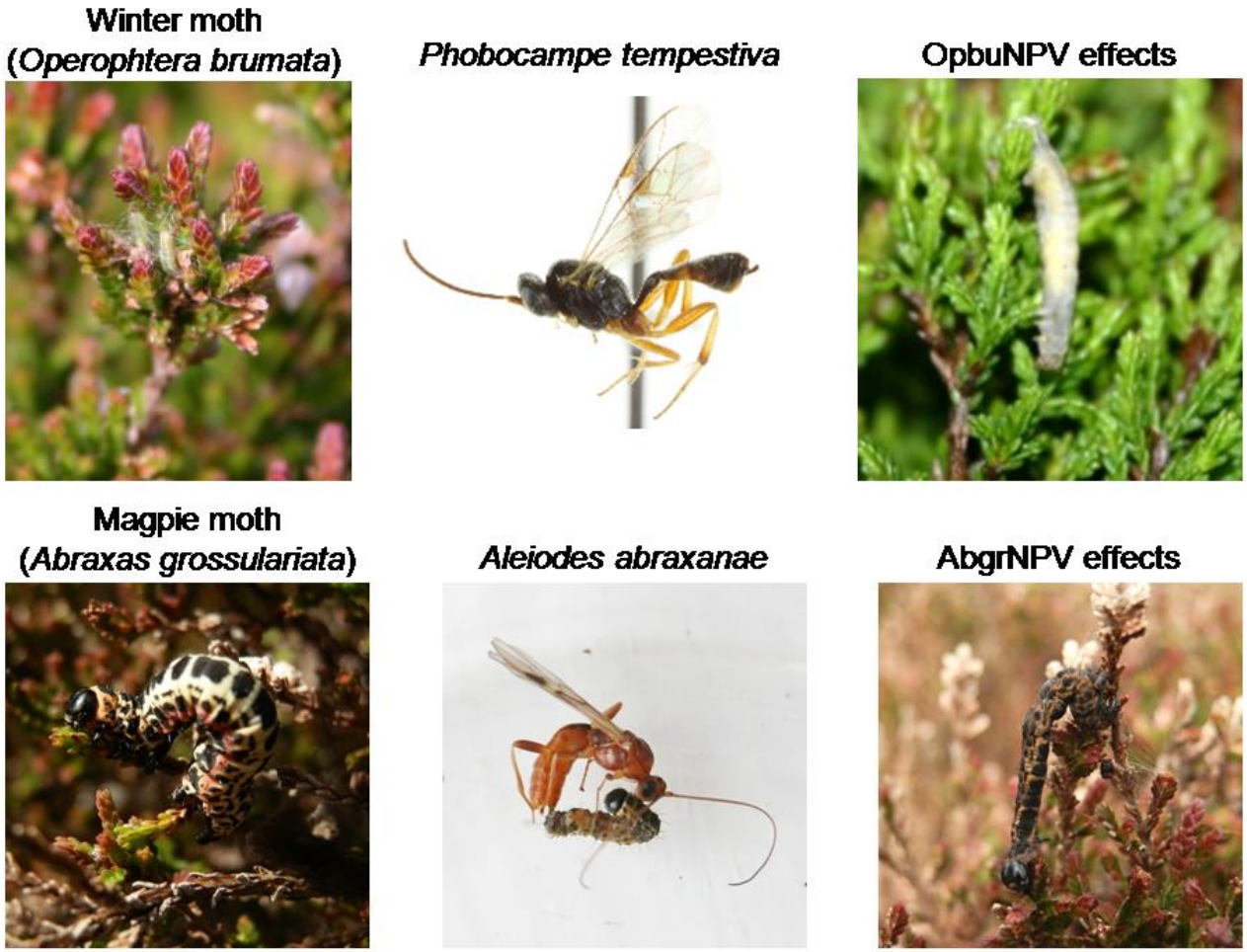

Figure 1.2 Study system species photos.

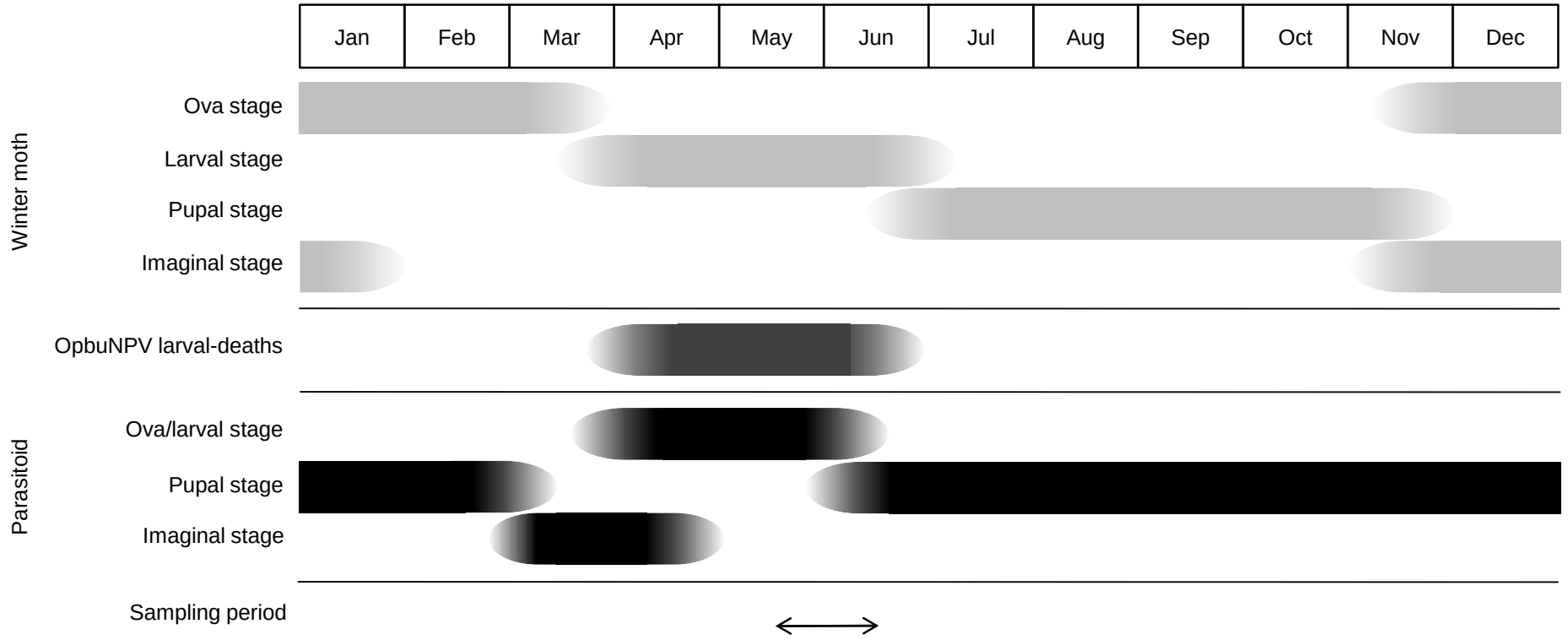

Figure 1.3 Life-cycle of the winter moth and its natural enemies on Orkney, and the sampling period.

Winter moth ecology has been well studied for over 50 years (e.g. Canning, 1960), in part because the larvae are frequent pests of economically important fruit crops (Gould, 1965; Embree, 1966) and agroforestry (Stoakley, 1985), but scientific interest has also focused on understanding the cyclical nature of the winter moth's population dynamics, but to date just within European deciduous forests (Hogstad, 2005; Hagen et al., 2010; Schott et al., 2010). In common with a small number of other well-studied cyclical forest Lepidoptera (Myers & Cory, 2013), the winter moth displays largely regular peaks in abundance, typically every 9-10 years, which can often result in spectacular outbreaks resulting in extensive defoliation of forests (Jepsen et al., 2008; Jepsen et al., 2013). However, as with all cyclical Lepidoptera, it is not clear what drives the dynamics of the winter moth (Schott et al., 2010; Myers & Cory, 2013). Most attention has focused on density-dependent mortality from parasitoids and predators though (Raymond et al., 2002b; Klemola et al., 2009; Schott et al., 2010; Klemola et al., 2014), and prior to this thesis there had been no field studies examining whether pathogens could drive winter moth cycles, something suspected in some other cyclical Lepidoptera (Kukan & Myers, 1999; Dwyer et al., 2004), and tested for the first time in chapter 2.

In common with most cyclical Lepidoptera winter moth dynamics within deciduous forests also display extensive spatial synchrony, often extending for hundreds of kilometres (Tenow et al., 2007). Spatial population synchrony can be related to inter-population dispersal or spatially correlated climatic fluctuations (Bjornstad et al., 1999), but female winter moths display brachyptery (winglessness), and males have limited dispersal abilities (Van Dongen et al., 1996). However, if early-instar larvae determine their environment to be unfavourable they are able to disperse at least a few hundred metres via ballooning (Bell et al., 2005), and analysis of winter moth population genetics within Orkney suggests that populations are well-mixed, suggesting that dispersal via ballooning may be more extensive than previously believed (Leggett et al., 2011). Large-scale spatio-temporal winter moth dynamics have not previously been explored within Britain, and the extent of winter moth population synchrony has also not been previously explicitly compared to the extent of spatial correlation in environmental factors in any region, but both issues are addressed in chapter 5. It has also recently become clear that locally and regionally synchronous winter moth dynamics within mainland Europe are actually part of a much larger phenomenon, whereby continental-scale travelling waves in winter moth abundance pass from eastern Europe to the western Atlantic coastline (c.

3000 km distance) every 9-10 years (Tenow, 2013; Tenow et al., 2013). Currently, it is not clear what causes these travelling waves (Tenow et al., 2013), and it is also not clear whether they reach Britain, as suggested by Tenow et al. (2013), or whether winter moth travelling waves occur within Britain at smaller scales. Therefore, these questions are also explored in chapter 5.

Within Orkney the winter moth is a resident species. In northern mainland Scotland and Orkney high density outbreaks of winter moth larvae have been reported on heather moorlands since the 1980s (Picozzi, 1981; Lorimer, 1983; Kerslake et al., 1996), and as reported for the first time in chapter 2, winter moth populations on heather moorlands also display cyclical synchronised dynamics, similar to those exhibited in deciduous forests (Hogstad, 2005; Klemola et al., 2014). On Orkney winter moth larvae are primarily parasitised by the solitary, koinobiont, endoparasitic wasp *P. tempestiva* (Fig. 1.2). *Phobocampe tempestiva* parasitises 1st and 2nd instar larvae during the spring, and eventually forms a cocoon in which the parasitoid larva overwinters, before emerging during the following spring to parasitise the next generation of winter moth larvae (Shaw et al., 2009) (Fig. 1.3). A number of other parasitoids (5 morphospecies) have been found during the rearing of all winter moth larvae collected for the long-term monitoring of populations providing the data for chapter 2, but as their numbers have been small relative to *P. tempestiva* (e.g. 8% in 2008 and 2009, S. Harold, unpub. data), and their recording inconsistent, they have not been formally identified, and are not considered further in this thesis. *Phobocampe tempestiva* is also known to attack a number of other Lepidoptera (Shaw et al., 2009), but not the magpie moth. In Orkney winter moth also suffer pupal predation from Carabid and Staphylinid beetles (Raymond et al., 2002b), and larval mortality from some bird species (Picozzi, 1981), and outside of Orkney winter moth larvae and pupae are also known to suffer mortality from a range of specialist and generalist invertebrate predators and parasitoids (Frank, 1967; Varley et al., 1973; Vindstad et al., 2011; Klemola et al., 2014). However, this thesis has only considered mortality from *P. tempestiva* within Orkney populations of winter moth.

Within Orkney the viral community has been particularly well characterised for an insect (Roy et al., 2009). OpbuNPV (Fig. 1.2) occurs widely, displaying high levels of spatial genetic variation and causing substantial levels of mortality in larvae (e.g. up to 62% in populations tested

by Graham et al., 2004). NPV infections are primarily transmitted horizontally when larvae consume vegetation contaminated with NPV occlusion bodies (OBs) (Cory & Myers, 2003). These proteinaceous structures protect the infectious virions in the external environment, but break down once in the midgut of insects, leading to their infection and eventual death, causing the release of millions more OBs into the environment (Cory & Myers, 2003) (Fig. 1.3). NPV infections can also be transmitted vertically leading to sublethal or covert infections in offspring (Vilaplana et al., 2008; Vilaplana et al., 2010), which may also subsequently emerge as overt lethal infections (Burden et al., 2002; Burden et al., 2003). Substantial levels of vertical transmission of OpbuNPV have previously been documented in winter moth collected from Orkney (Graham, 2006), but is not explicitly investigated in this thesis. The host specificity of NPVs can vary from one to many species of Lepidoptera (Cory & Myers, 2003), but it is not currently clear how host-specific OpbuNPV is, or whether it can infect the magpie moth.

In addition to OpbuNPV, three novel species of reovirus have also been found in winter moth populations on Orkney, including two cypoviruses (CPVs) and a reovirus that appears to be vectored by *P. tempestiva* (Graham et al., 2006; Graham et al., 2007; Graham et al., 2008). However, although CPVs are known to cause sub-lethal and lethal effects, particularly through vertical transmission of infections to offspring of infected insects (Rothman & Myers, 1996), it is not clear to what extent these viruses contribute to levels of larval mortality in Orkney populations of winter moth (Graham, 2006), and they are not considered in this thesis. Outside of Orkney winter moth pathogens have received little attention, aside from a few historical reports of viral infections (presumed to be NPVs) in populations in Nova Scotia (Embree, 1966) and northern Scotland (Stoakley, 1985), microsporidian infections in populations in Oxford (Varley et al., 1973), and a more recent report of OpbuNPV infections in populations in Massachusetts (Burand et al., 2011).

1.7.8 The magpie moth and its natural enemies

Like the winter moth, the magpie moth (Fig. 1.2) is a polyphagous, univoltine geometrid whose larvae can also feed on heather, as well as a range of shrubs and deciduous trees, primarily various *Ribes* spp., but also

blackthorn (*Prunus spinosa* L.) and hazel (*Corylus avellana* L.) (Allan, 1979). The magpie moth is found throughout Britain, and has a substantial geographical distribution throughout large parts of temperate Asia, and as far west as Japan (Alford, 2014). Magpie moth larvae hatch from eggs in late summer and feed before entering diapause in the autumn (Fig. 1.4). Early-instar larvae then overwinter in sheltered areas, before emerging from diapause following budburst in the spring to resume feeding. Pupation occurs in mid-late summer, with adults emerging to mate and lay eggs a few weeks later (Fig. 1.4). It is not certain how many instars larvae go through, but this author has raised larvae under ambient conditions through three instars until they entered their winter diapause, and larvae collected during the spring typically pass through a further three instars, implying a total of six instars.

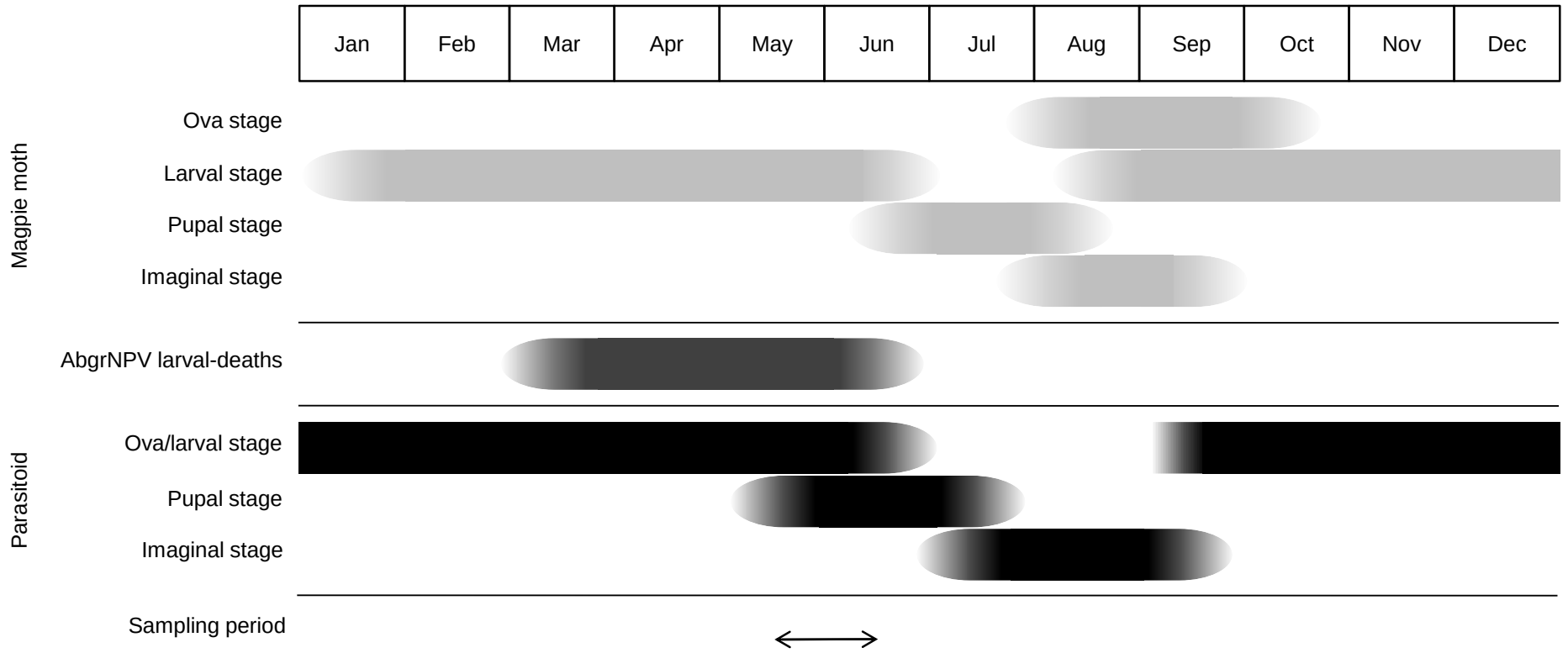

Figure 1.4 Life-cycle of the magpie moth and its natural enemies on Orkney, and the sampling period.

The pupal, larval and adult stages of the magpie moth are all substantially bigger than those of the winter moth, with final instar larvae measuring approximately 50 mm in length, compared to final instar winter moth larvae, which measure approximately 25 mm length. As a relatively large-bodied species the magpie is expected to be a relatively strong disperser, and relatively robust to the effects of habitat fragmentation as explored in chapter 4, although not specific information on its dispersal ability is available.

No prior studies of magpie moth population dynamics exist, but across Britain as a whole the population appears to have declined during the period 1968-2002 (Conrad et al., 2006). Within Orkney the presence of the magpie moth has not always been clear, but it appears to be a recent arrival or re-arrival to the islands. There was only a single reported sighting of an adult prior to 1981 (Lorimer, 1983), but populations appear to have increased dramatically during the early 1990s, with substantial numbers of larvae and adults being reported on Hoy in 2001, and larvae and adults then recorded as being widespread across the largest islands in subsequent years (Waring, 2006). Reports of some large-scale outbreaks of magpie moth larvae on heather moorlands along the northern coast of Scotland after 2003 (Horsfield & Macdonald, 2004) also suggest the possibility of population increases occurring during the 1990s-2000s in northern Scottish heather habitats, from which the Orkney populations may be derived.

Within Orkney an obligate, specialist parasitic wasp of magpie moth larvae, *Aleiodes* sp. (Hymenoptera: Braconidae) (Fig. 1.2), which is awaiting formal naming by C. van Achterberg & M.R. Shaw (unpub. data), is now widespread. However, although the parasitoid was previously reared from larvae collected on Hoy and Mainland Orkney in 2003 (M. Shaw 2011, pers. comm.), it was not found in magpie moth populations that were sampled yearly as part of the long-term data analysed in chapter 2 until 2010. Since then it has caused increasing levels of larval mortality as it has apparently undergone a rapid population expansion across the Mainland, Hoy and Rousay (see chapter 2). This solitary, koinobiont, endoparasitic wasp is known throughout Britain, and parasitises early-instar larvae in late summer (C. van Achterberg & M.R. Shaw, unpub. data) (Fig. 1.4). The parasitoid therefore overwinters within magpie moth larvae before resuming its development in the spring (C. van Achterberg & M.R. Shaw, unpub. data). Upon completion of development the parasitoid larva then creates a characteristic 'mummy' from the exoskeleton of magpie moth larvae, which

it attaches to the stem of a larval food plant, and from which it emerges in late summer after completing its development as a pupa (C. van Achterberg & M.R. Shaw, unpub. data) (Fig. 1.4). It is not known how effective a disperser this parasitoid is, but as an obligate specialist it is expected to be more strongly negatively affected by habitat fragmentation than its host, something that is tested in chapter 4. Magpie moth larvae have also produced unidentified parasitoids in previous years, but numbers have been very small (approximately 5 in total), and therefore this thesis only investigated parasitism from the *Aleiodes* sp. parasitoid.

In addition to the parasitoid, within Orkney a highly pathogenic NPV, AbgrNPV (Fig. 1.2), has been causing substantial levels of mortality in larvae since at least 2006 (Harold, 2009; Hicks et al., 2015) (Fig. 1.4). At present it is not clear how host-specific AbgrNPV is, but it does appear able to infect winter moth, although it does not appear to be common in winter moth larvae on Orkney (S. Harold, unpub. data). It is also not known whether AbgrNPV may be transmitted vertically, although the wider evidence of NPV transmission routes in insects suggests that this is a very likely possibility (Sorrell et al., 2009). This is therefore considered as a likely transmission route that could be affected by changes in host movement patterns, something investigated in chapter 4 where the impact of habitat fragmentation on the magpie moth host-parasitoid-pathogen system is investigated. Therefore, although not thought to be a classically cyclical or outbreaking pest species, the magpie moth appears to undergo moderately large population fluctuations in some areas of northern Scotland and Orkney, and suffers potentially high levels of mortality from a range of natural enemies (Harold, 2009), potentially influencing density-dependent prophylactic mechanisms, which is investigated in chapter 3.

It is not known what other pathogens affect the magpie moth on Orkney, and aside from a Cypovirus isolated from larvae from an unspecified location in the U.K. in the 1970s (Payne & Rivers, 1976), nothing is apparently known about pathogenic natural enemies of the magpie moth outside of Orkney. Similarly, outside of Orkney there appears to be very little published research on the invertebrate and vertebrate natural enemies of the magpie moth, although a record of magpie moth larval predation by the common cuckoo (*Cuculus canorus* L.) exists (Newman, 1851). Interestingly though, the magpie moth displays aposematic colouration in both larval, pupal and adult life stages, and is the only known British Lepidoptera to do so in its pupal stage (Nishida et al., 1994), suggesting an evolutionary

pressure from vertebrate natural enemy predation may have driven the evolution of this defensive mechanism (Guilford, 1988).

2. Host and multiple enemy interactions in cyclical and non-cyclical insect population dynamics

2.1 Abstract

The drivers of high-amplitude cyclical population dynamics are still unclear for almost all species, including well-studied outbreaking lepidopteran forest pests. Cyclical species are not common, but represent strongly regulated model systems. In insects, the most favoured causal hypothesis for population cycles involves delayed density-dependent mortality from specialist natural enemies, primarily parasitoids and pathogens. However, although strongly supported by theoretical models, the hypothesis has rarely been tested with long-term data from field systems, particularly those involving pathogens, and never involving both parasitoids and pathogens. This study investigated the evidence for direct and delayed density-dependent mortality from the parasitoid and pathogen of two lepidopteran hosts, the cyclical winter moth and the non-cyclical magpie moth, using multi-year data from larval field populations on Orkney. The direct and delayed effects of the temperature (experienced during larval development) on host-enemy interactions were also analysed. These analyses revealed evidence for delayed (but not direct) density-dependent mortality from the specialist pathogen of both the winter moth and, unexpectedly, the non-outbreaking magpie moth, but there was no density-related mortality from either species' parasitoid. Therefore, the study provided the first evidence from multi-year field data consistent with specialist pathogen regulation of insect cycles (in the winter moth), but it appeared likely that other mechanisms may also play a role. In addition, there was a negative effect of temperature in the previous year on winter moth pathogen mortality, indicating a possible delayed negative effect of temperature on the pathogen's environmental persistence. Unexpectedly, the dynamics of the non-cyclical magpie moth appeared to be even more strongly regulated by

its specialist pathogen than those of the winter moth, highlighting the need to consider a wider range of species when attempting to understand population regulation. Therefore, the study also highlighted the need to consider pathogens as potentially important factors in population dynamics generally.

2.2 Introduction

A fundamental goal of ecology is the understanding of population dynamics (Begon et al., 2006), and regular, multiannual population cycles have been studied by ecologists for over 90 years (Elton, 1927), with the phenomenon known from species across a range of taxa including mammals, birds, fish and insects (Kendall et al., 1998). Population cycles occur despite the stochasticity of natural systems, suggesting powerful regulatory mechanisms (Inchausti & Ginzburg, 2009). Therefore, although only a minority of species actually exhibit population cycles (Kendall et al., 1998), understanding the mechanisms generating them may further the understanding of population dynamics more generally, as the same mechanisms are likely to occur in most species, even if they are less important. Prominent examples include the 10-year snowshoe hare cycle in North America (Krebs et al., 2001), the cycles of varying length displayed by a number of rodents in northern Europe (Gilg et al., 2009; Berthier et al., 2014), the 4-8 year cycles in British red grouse populations (*Lagopus lagopus scoticus*) (Hudson et al., 1998), and the 8-10 year cycles seen in European larch budmoth (*Zeiraphera diniana*) populations (Johnson et al., 2010). Most hypotheses of causal mechanisms have focused on trophic interactions (i.e. biotic factors), and specifically the role of predatory or parasitic natural enemies (Hudson et al., 1998; Hanski et al., 2001) and induced defences of food plants (Nykanen & Koricheva, 2004; Haukioja, 2005), but endogenous mechanisms driven by maternal effects have also been considered (Inchausti & Ginzburg, 2009). However, there is still substantial uncertainty regarding the precise underlying causes of most cyclical species' dynamics (Myers & Cory, 2013), and their relative importance, primarily because of limited conclusive empirical data.

In insects, studies have largely focused on a relatively small number of forest Lepidoptera whose populations exhibit regular cycles, typically resulting in outbreaks that cause substantial defoliation of trees and even entire regions of forest (Jepsen et al., 2013). In trying to understand these cycles a number of exogenous and endogenous causal mechanisms have been proposed, including the impacts of delayed induced plant-defences (Kessler et al., 2012) and maternal effects (Inchausti & Ginzburg, 2009). However, most attention has focused on the role of natural enemies, specifically delayed density-dependent mortality from parasitoids and pathogens, and although there is still much debate (e.g. Inchausti &

Ginzburg, 2009) the natural enemy hypothesis remains the most favoured (Myers & Cory, 2013; Klemola et al., 2014). A substantial body of theory has also been developed clearly demonstrating that delayed density-dependent mortality, such as that caused by specialist parasitoids and pathogens, can generate cyclical dynamics in insects (e.g. Anderson & May, 1980; Anderson & May, 1981; Reynolds et al., 2013; Ammunet et al., 2014). Both parasitoid- and pathogen-focused models have been made increasingly realistic through the inclusion of additional biology seen in natural systems; for example, the incorporation of generalist predators into models has provided potential explanations for geographical gradients in the length of cycles (Bjornstad et al., 2010) and the variability of cycle periodicity over time (Dwyer et al., 2004), as seen in real cyclical populations.

Parasitoids and pathogens are clearly capable of causing substantial levels of mortality in insects, consistent with an ability to regulate host populations. For example, during the early increase phase of the cyclic autumnal moth (*Epirrita autumnata*), when host abundance is low, specialist parasitoids are usually extremely scarce, but at the peak and post-peak phases levels of parasitism can jump to >90% (Ruohomaki et al., 2000; Klemola et al., 2007; Klemola et al., 2008). The vast majority of work on pathogens in lepidopteran population cycles has focused on NPVs, which are commonly found in many lepidopteran populations (Cory & Myers, 2003), and like parasitoids mortality from NPV infections can exceed 90% during the peak and post-peak phases of lepidopteran population cycles (Myers, 1988; Cory & Myers, 2009; Liebhold et al., 2013; Myers & Cory, 2013). NPVs also have long-lived infectious stages, enabling persistence between generations (Cory & Myers, 2003), which can provide a mechanism for delayed density-dependent mortality (Anderson & May, 1980).

However, empirical evidence from multi-year host-enemy field systems supporting the role of specialist natural enemies in lepidopteran population cycles is inconsistent (Myers & Cory, 2013). A recent 10-year observational study of parasitism in the egg and pupal stages of the autumnal moth found significant correlations between host density and delayed levels of parasitism, indicative of delayed density-dependent parasitoid regulation of autumnal moth cycles (Klemola et al., 2014). However, two comparable studies covering the increase, peak and decline phases of the winter moth's cycle (over a 5-year period) found no association between host density and parasitism at all (Hagen et al., 2010; Schott et al., 2010). Stronger empirical evidence for the ability of parasitoids

to generate cyclical dynamics comes from the study of Klemola et al. (2010), who demonstrated that autumnal moth populations in parasitoid exclosures increased over a four-year period, whilst populations in control plots declined due to high levels of parasitism. Long-term field data on host-pathogen dynamics is even rarer though, and although Liebhold et al. (2013) found a relationship between host density in the gypsy moth (*Lymantria dispar*) and mortality from the NPV LdNPV across two three-year periods, Kukan and Myers (1999) found contradictory evidence for density-dependent mortality from the NPV MpNPV in western tent caterpillar (*Malacosoma californicum pluviale*) populations over an incomplete 14-year period. Additionally, apparently no cyclical insect study has explicitly tested for delayed density-dependent mortality from pathogens to date, although models indicate that stable periodic cycles are best explained by this form of density dependence (Anderson & May, 1980; Dwyer et al., 2004; Reynolds et al., 2013; Barraquand et al., 2014), and delayed density-dependent mortality has been detected in cyclical mammals (Cavanagh et al., 2004; Burthe et al., 2006).

Unsurprisingly, given the limited field data for host-parasitoid and host-pathogen dynamics in cyclical insects, there are apparently no published field studies on the multi-year dynamics of host-parasitoid-pathogen systems. However, laboratory-based host-parasitoid-pathogen systems indicate that both types of enemy can have important effects on host dynamics, with longer cycle periods occurring in host-parasitoid-pathogen systems than in simpler host-parasitoid systems, and with alterative cyclical dynamical patterns occurring depending on the order in which enemies invade existing systems (Begon et al., 1996; Sait et al., 2000; Bonsall et al., 2005). Theoretical work also indicates important effects from both enemies in host-parasitoid-pathogen systems, with different host-dynamics possible, including periodic cycles and chaos, depending on the order in which the enemies invade the system and other key parameters (Hochberg et al., 1990; Preedy et al., 2007). Consequently, although a substantial body of compelling theory indicates that specialist natural enemies are capable of generating cyclical dynamics in insects through delayed density-dependent mortality, the hypothesis has very rarely been tested with multi-year field data, and never in a field system involving both parasitoids and pathogens.

Therefore, this study primarily aimed to test whether specialist parasitoids and pathogens can cause direct and delayed density-dependent

mortality in lepidopteran hosts, consistent with the regulation of cyclical dynamics. To investigate this question multi-year data were gathered on the density and levels of mortality from parasitism and NPV infections in natural populations of winter moth and magpie moth larvae from heather moorlands across Orkney. The winter moth exhibits 9-10 year population cycles in European deciduous forests that often result in outbreaks and defoliation (Hogstad, 2005; Tenow et al., 2013), and its cycles are generally believed to be driven by mortality from parasitoids (Myers & Cory, 2013; Klemola et al., 2014), but the effects of pathogens have not previously been investigated. In common with all other cyclical Lepidoptera winter moth dynamics also display extensive local- and regional-scale synchrony in European forests (Tenow et al., 2007; Hagen et al., 2008). Despite some amateur reports of unquantified localised magpie moth outbreaks on heather moorlands in northern Scotland and Orkney (Horsfield & Macdonald, 2004), the magpie moth is not previously known to exhibit cyclical dynamics, and is assumed to be non-cyclical. The study could therefore compare host-parasitoid-pathogen dynamics in both a cyclical and a non-cyclical system, within the same habitat and area. Therefore, in line with the natural enemy hypothesis for cyclical species it is expected that the winter moth will suffer strong direct and delayed density-dependent mortality from its parasitoid and pathogen. However, as a non-cyclical species the magpie moth is only expected to suffer from weak direct density-dependent mortality from its parasitoid and pathogen. Orkney-wide regional synchrony in winter moth and magpie moth dynamics were also investigated to allow comparisons between winter moth dynamics in European deciduous forests and Orkney heather moorlands, and between the spatio-temporal dynamics of winter moth and magpie moth within Orkney in the context of their potential regulation by natural enemies.

Although stochastic (density-independent) climatic factors are not generally believed to be capable of driving regular population cycles, there is good evidence that they can modulate these dynamics (Esper et al., 2007; Ims et al., 2008; Haynes et al., 2014). In addition, density-independent climatic factors are known to influence the population dynamics of many non-cyclical species (Nowicki et al., 2009; Ziebarth et al., 2010; Boggs & Inouye, 2012). Ongoing climate change appears to be impacting the dynamics of some cyclical insects already, with evidence suggesting both increasing severity and frequency of outbreaks in some species (Bentz et al., 2010; Paritsis & Veblen, 2011), but decreasing severity of outbreaks in others (Esper et al., 2007; Haynes et al., 2014). However, the precise mechanisms by which climate influences the dynamics of cyclical insects so

far remains elusive, and is likely to vary between systems. To properly investigate the influence of climate on cyclical populations requires lengthy time-series data covering many population cycles, such as those gathered from dendrochronological records spanning hundreds or even 1000+ years (Esper et al., 2007; Johnson et al., 2010; Haynes et al., 2014). Such data do not exist for host-enemy interactions, but shorter-term studies can provide indications about the ways in which climatic stochasticity can indirectly affect host dynamics via its influence on host-enemy interactions (Stireman et al., 2005; Tylianakis et al., 2008; Martinez & Merino, 2011; Korpela et al., 2014). Therefore, as temperature is known to influence host-parasitoid and host-pathogen interactions (Martinez & Merino, 2011; Meisner et al., 2014), the study also aimed to investigate how mean temperature levels during larval development affected the host-enemy dynamics of each species.

2.3 Methods

2.3.1 Study species

On Orkney whilst the winter moth appears to be a long-term resident, the magpie moth appears to be a recent arrival or re-arrival, with larvae only found in the sampling sites used in this study from 2005 onwards. On Orkney both the winter moth and the magpie moth have a primary parasitoid and pathogen natural enemy of their larval stages. The primary parasitoid of winter moth larvae is *P. tempestiva*, a solitary, koinobiont, endoparasitic wasp. *P. tempestiva* parasitises early-instar larvae during the spring (Shaw et al., 2009), and is known to attack a range of lepidopteran species (Shaw et al., 2009), but is not a parasitoid of the magpie moth. The primary larval pathogen of the winter moth is the NPV OpbuNPV. The primary parasitoid of magpie moth larvae is an *Aleiodes* sp. parasitoid, which only appeared in sampled populations in 2010. Unlike *P. tempestiva*, the *Aleiodes* sp. parasitoid parasitises early-instar larvae in late summer, overwintering in hosts before resuming development the following spring (C. van Achterberg & M.R. Shaw, unpub. data). The primary larval pathogen of the magpie moth is the NPV AbgrNPV, which has been known in sampled populations since 2006.

2.3.2 Study sites and data collection

The study was carried out between 2003 and 2014 across three of the Orkney Islands: Mainland Orkney, Hoy and Rousay (Fig. 2.1). In 2003 11 sites on Mainland Orkney and one site on each of Hoy and Rousay were chosen within heather moorlands to sample obvious winter moth larval populations (Fig. 2.1), with magpie moth appearing in some of these sites in 2005. Sites were sampled once per-year between mid-May and mid-June of each year, from 2003-2014, with the author of this thesis participating in data collection at these sites between 2012-2014. In 2007 however two sites were not sampled (*li* and *sya* in Fig. 2.1), and in 2008 one of the same sites was also not sampled in 2007 (*li* in Fig. 2.1). Finally, an additional site was added in 2006 on Mainland Orkney (*swb* in Fig. 2.1), and sampled every year subsequently. Therefore, there were a total of 14 sites where winter moth were sampled, and 12 where magpie moth were sampled.

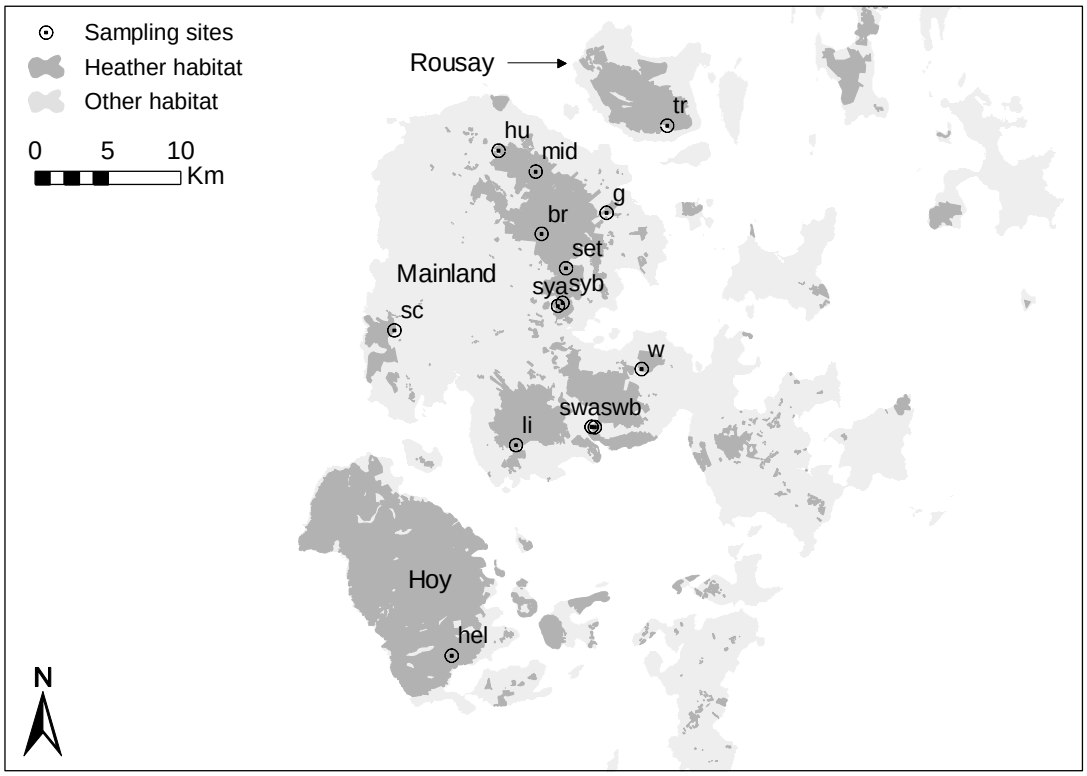

Figure 2.1 Distribution of long-term sampling sites and heather habitat on Mainland Orkney, Hoy and Rousay.

From 2003-2005 sampling was conducted by placing a 10 m transect within each site, parallel to and approximately 1m away from the previous year's transect. The initial transects in 2003 were purposively placed in areas of obvious winter moth larval populations. Then at 1 m intervals along the sampling transect a 0.25 cm^2 heather sample was removed and placed into a bag for later sampling of larvae (resulting in 10 such samples per transect and site). The ground under each quadrat was also carefully searched, and any living larvae found were also placed in the sampling bag. Any dead larvae were placed in a 2 ml Eppendorf tube using a disposable toothpick, and stored at -20 °C for subsequent analysis for NPV infection. In 2007 to increase sample sizes a majority of sites were sampled with three 10 m transects (resulting in 30 quadrat samples), with transects placed in parallel and separated by 5 m from one another, and from 2008 onwards all sites were sampled in this way. Following the sampling of a site all bagged heather vegetation was carefully sorted through by hand to locate any living and dead winter moth and magpie moth larvae, as well as any *Aleiodes* sp. parasitoid mummies, which can be found attached to heather stems towards the end of the sampling period (*P. tempestiva* do not kill their hosts until well after the sampling period). Again, any dead larvae were stored at -20 °C for subsequent analysis. Additionally, in order to increase the sample size of larvae available for subsequent calculation of natural enemy mortality rates approximately 38% of winter moth collections and 12% of magpie moth collections were supplemented with additional haphazard collections of larvae, taken from the immediate vicinity of the sampling transects.

All living winter moth and magpie moth larvae were then reared individually in 25ml pots, in order to record levels of parasitism and mortality from NPV infection. Winter moth larvae were provided with sterile, artificial diet (Hunter et al., 1984) to feed on *ad libitum*, and magpie moth larvae were provided with non-sterile green heather shoots to feed on *ad libitum* as they refuse artificial diet. Shoots were taken from the relevant heather samples from which larvae were collected, in order to avoid altering their risk of AbgrNPV infection. Pots were stored at room temperature, and larvae were checked every 1-3 days until pupation in order to replace food, remove faeces to prevent mould and identify any dead larvae (which were again stored at -20 °C for subsequent analysis) or emerged parasitoids. Larval cadavers were then tested for NPV infections. Data on larval mortality from NPV infection was collected for the winter moth from 2003-2009, and for the magpie moth from 2006-2014. OpbuNPV infections were confirmed in larval cadavers using PCR reactions based on primers listed in Graham et al.

(2004). AbgrNPV infections were confirmed in larval cadavers using PCR reactions based on primers listed in Harold (2009), apart from in 2014 when Giemsa staining and microscopy methods were instead used to detect AbgrNPV occlusion bodies (Cory et al., 2005; Lacey, 2012). Where NPV DNA or occlusion bodies were found in larval cadavers they were assumed to have died from the relevant NPV infection of each species, and where not found larval mortality was assumed to be due to unknown causes.

2.3.3 Statistical analyses and variables

For each site a standardised measure of larval density per m^2 was calculated based on the total number of larvae in the quadrat sampled heather collections. Despite the additional collections of larvae, particularly during the later years of the study, many sites yielded relatively few larvae with which to assess levels of natural enemy mortality (e.g. approximately 60% of winter moth and 54% of magpie moth collections from sites yielded <15 individuals per site). Due to their bounded nature prevalence estimates can change dramatically at low sample sizes with even very small increases/decreases in numbers of dead larvae. Therefore, instead of analysing the effect of many relatively-poorly estimated mortality rates from each natural enemy on the population growth rate of each moth, it was decided to analyse the evidence for direct and delayed density-dependent mortality from each species' parasitoid and pathogen. This was achieved by using logistic generalised linear mixed-effects models (GLMMs) to analyse the effects of larval density in the current and previous year, and climatic variation during the current and previous larval generation's developmental period, on the likelihood of larval mortality from parasitism and NPV infection for each host species. Logistic GLMMs are able to explicitly take into account sample sizes when estimating the likelihood of parasitism and NPV mortality in relation to the explanatory variables, resulting in reliable inference (Paterson & Lello, 2003). The logistic GLMMs used Bernoulli errors and logit-links, with the response variable being the binary outcome for each larva of either being killed by parasitism/not being killed by parasitism (i.e. surviving or dying from other causes), or being killed by NPV infection/not being killed by NPV infection (i.e. surviving or dying from other causes). Larvae dying as a result of handling deaths were excluded from these analyses. To control for the spatial and temporal structure in the data due to the study design (clustering within years, and clustering within sites within

years), models also included year and site nested within year as random factors (Zuur et al., 2009).

When analysing the effects of climate on ecological processes in observational settings there are so many possible associations (i.e. different climatic variables) that the probability of finding spurious correlations can be high (Myers, 1988). Therefore, as temperature is known to influence both host-parasitoid and host-pathogen interactions and dynamics (Martinez & Merino, 2011; Triggs & Knell, 2012; Stoepler & Lill, 2013; Meisner et al., 2014) this was the only variable considered. More specifically, in this study to allow direct or lagged effects of climate to be investigated only the effects of the mean temperature during the period in which either the current or the previous generation of winter moth and magpie moth larvae were developing as early-mid instars, and therefore most subject to parasitism and NPV infection (Cory & Myers, 2003), were considered. For the winter moth this meant the mean of the monthly-mean-temperatures during March, April and May in the current or previous year. For the magpie moth this meant the mean of the mean-monthly-temperatures during the previous late-summer months (July, August and September) and the current spring months (March, April and May). Temperate data was acquired from a Meteorological Office weather station located by the Loch of Hundland in Birsay, Mainland Orkney (59.115°, -3.21°).

A multimodel inference (MMI) approach to the analysis was followed using information-theoretic (IT) methods, based on the Akaike Information Criteria (AIC) (Burnham & Anderson, 2002), as used throughout this thesis. MMI is based on a different paradigm to that of null-hypothesis significance testing, and rejects the use of null-hypothesis tests entirely (Burnham & Anderson, 2002, 2014). Instead, inference is based on quantifying the strength of evidence supporting different hypotheses, represented as competing models, using IT methods applied to data (Burnham & Anderson, 2002; Burnham et al., 2011). Point estimates of parameters and measures of their uncertainty (such as 95% confidence intervals) can then be made, conditioned on the model(s) best supported by the evidence and with appropriate weighting, to allow judgements to be made about the direction and size of any effects (Burnham & Anderson, 2002; Burnham et al., 2011).

Consequently, for each natural enemy mortality dataset two sets of three models were first created to represent different hypotheses about the causes of the observed data. The first set analysed direct effects of larval density and direct effects of temperature during early-mid larval instars, and

the second set analysed delayed (lagged by 1 year) effects of larval density and delayed effects of temperature during early-mid larval instars. In each of the direct and delayed model sets one also model tested for a non-linear relationship between larval density and larval mortality through the inclusion of a second order polynomial term (larval density + larval density^2), whilst also testing for a linear relationship between larval density and temperature. A second model tested for a linear relationship between larval density and larval mortality, and a linear relationship between temperature and larval mortality. Finally, a null (intercept only) model was also included in all model sets for comparison. Datasets covered different temporal periods depending on the natural enemy, and whether direct or delayed effects were being assessed. For the magpie moth parasitoid data was available from 2010-2014 at both t and t-1; for AbgrNPV data was available from 2006-2014 at both t and t-1; for the winter moth parasitoid data was available from 2003-2014 at t, and from 2004-2014 at t-1; and for OpbuNPV data was available from 2003-2009 at t, and from 2004-2009 at t-1. Detection of OpbuNPV infections in frozen winter moth larval samples from the 2010-2013 field seasons failed due to the poor quality of stored larval cadavers, meaning the OpbuNPV data set ended in 2009. Each natural enemy dataset was analysed separately in terms of direct and delayed effects.

Within each model set all models were ranked by their AIC scores, and Akaike weights calculated (Burnham & Anderson, 2002). Akaike weights represent each model's probability, given the data, relative to all other models in the set (Burnham & Anderson, 2002). If either the polynomial or the linear model in a model set had an Akaike weight greater than or equal to 0.95 then this was taken as strong evidence that this model was substantially better than the other models in the set, and inference was based on the model's parameter estimates and their 95% confidence intervals. However, if the polynomial model did not have an Akaike weight greater than or equal to 0.95 then the parsimonious decision was taken that there was not strong evidence for a non-linear effect of larval density. Consequently, the polynomial model was then removed from the model set and Akaike weights recalculated. Then if the remaining linear model had an Akaike weight greater than or equal to 0.95 inference was based on its parameter estimates and their 95% confidence intervals. Otherwise the parsimonious conclusion taken was that, as neither the polynomial or linear models were strongly supported relative to the null model, there was no evidence for any important effects of larval density or climate.

To investigate regional synchrony in the dynamics of both Lepidoptera the mean Pearson product-moment correlation between all populations (sites) over time was calculated. This represents a measure of temporal synchrony across all populations being considered (Gouhier & Guichard, 2014), with spatial synchrony known to be a key characteristic of the dynamics of cyclical insects (Myers & Cory, 2013). The significance of the value relative to a null hypothesis of no correlation was assessed by a two-tailed p-value calculated from the distribution of correlations calculated from 999 Monte Carlo randomisations of the data (Gouhier & Guichard, 2014). To ensure adequate site-specific data for the synchrony analyses only sites where 50% or more years produced at least one larvae were utilised, and for the magpie moth data from 2005 was excluded. Utilising the full dataset (i.e. including sites with many zero abundances) resulted in larger correlations, and so restricting it in this way was conservative. This resulted in the winter moth analysis being based on data from 9 sites, and the magpie moth analysis on data from 11 sites (out of a total of 14 possible sites).

To aid comparison of model coefficients all explanatory variables were first standardised to have a mean of zero and a standard deviation of one. To validate the larval density models the best AIC scoring model in each set was assessed for adherence to model assumptions using quantile-quantile and partial residual plots, based on a simulation approach outlined in Zuur et al. (2009). The same models were assessed for multicollinearity using variance inflation factor analysis, but all VIFs were <1.5 indicating no issues (O'Brien, 2007). All analyses were conducted in the statistical software R (R Core Development Team, 2014), with the package *lme4* (Bates et al., 2014) used for all GLMMs. Logistic and negative binomial GLMM models were fitted by the Laplace maximum likelihood approximation, (Zuur et al., 2009). Pearson product-moment correlation values were calculated using the package *synchrony* (Gouhier & Guichard, 2014).

2.4 Results

2.4.1 Winter moth population dynamics and natural enemy interactions

Within local populations (sites) there were some apparent cyclical patterns (Fig. 2.2 (a)), and when averaged across all local populations there was a clear cyclical trend across the 12 years of the study, with one potentially complete cycle observed, which appeared to have a period of six years (Fig. 2.3 (a)). In terms of total average-density across the study area the 2003 peak/post-peak was considerably greater than that in 2010 (Fig. 2.3 (a)). In line with the observation of regional cyclicity the mean Pearson product-moment correlation between the nine sites where at least one larva was found in at least 50% of the years during the study was 0.183 ($p = 0.016$). Therefore, there were moderate, positively-correlated fluctuations in population density across the study area.

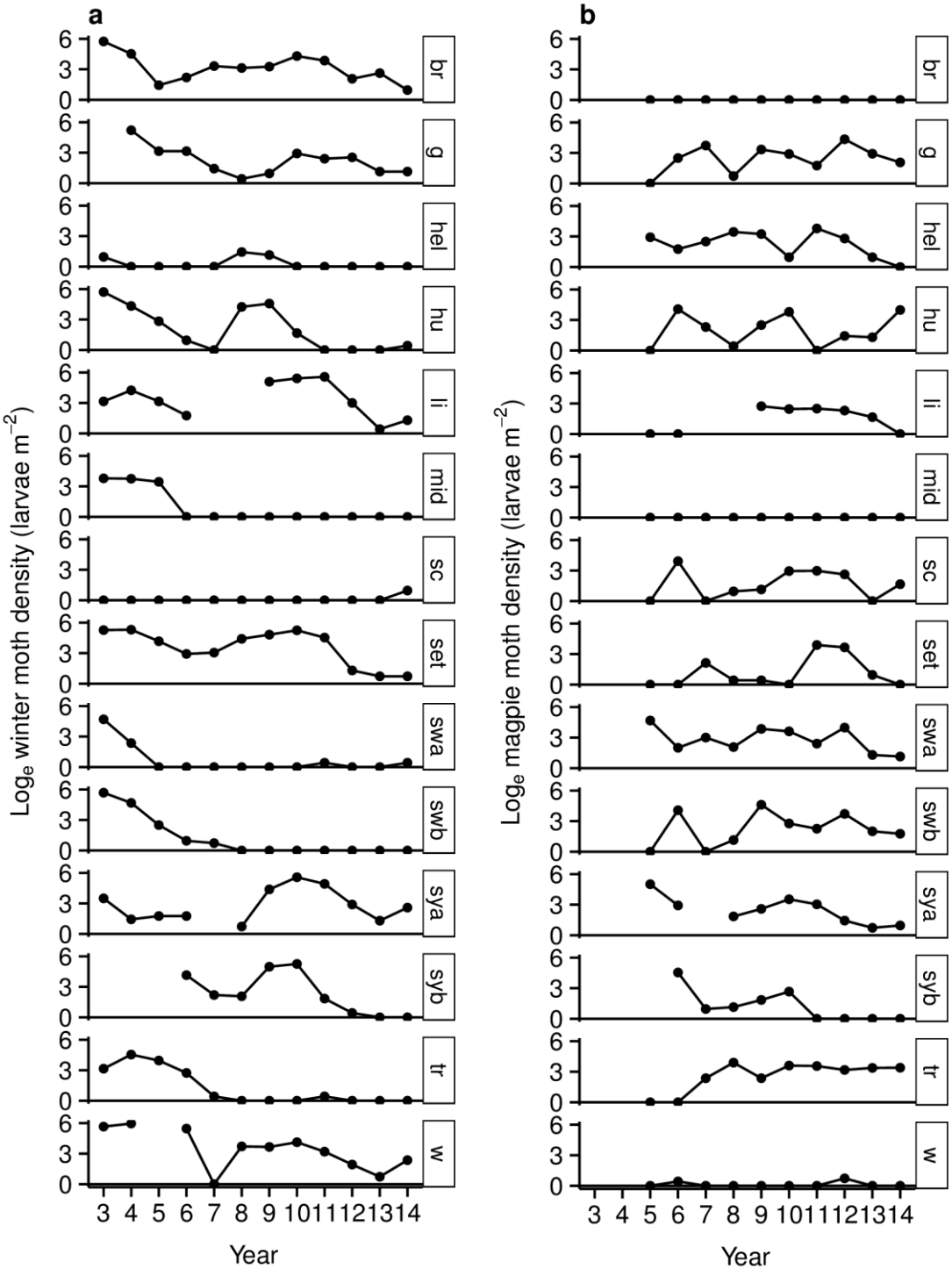

Figure 2.2 Yearly larval densities ($\log_e$ scale, constant of 1 added to all values before $\log_e$ transformation) within each site for both the winter moth (a) and magpie moth (b). See Fig. 2.1 for site locations.

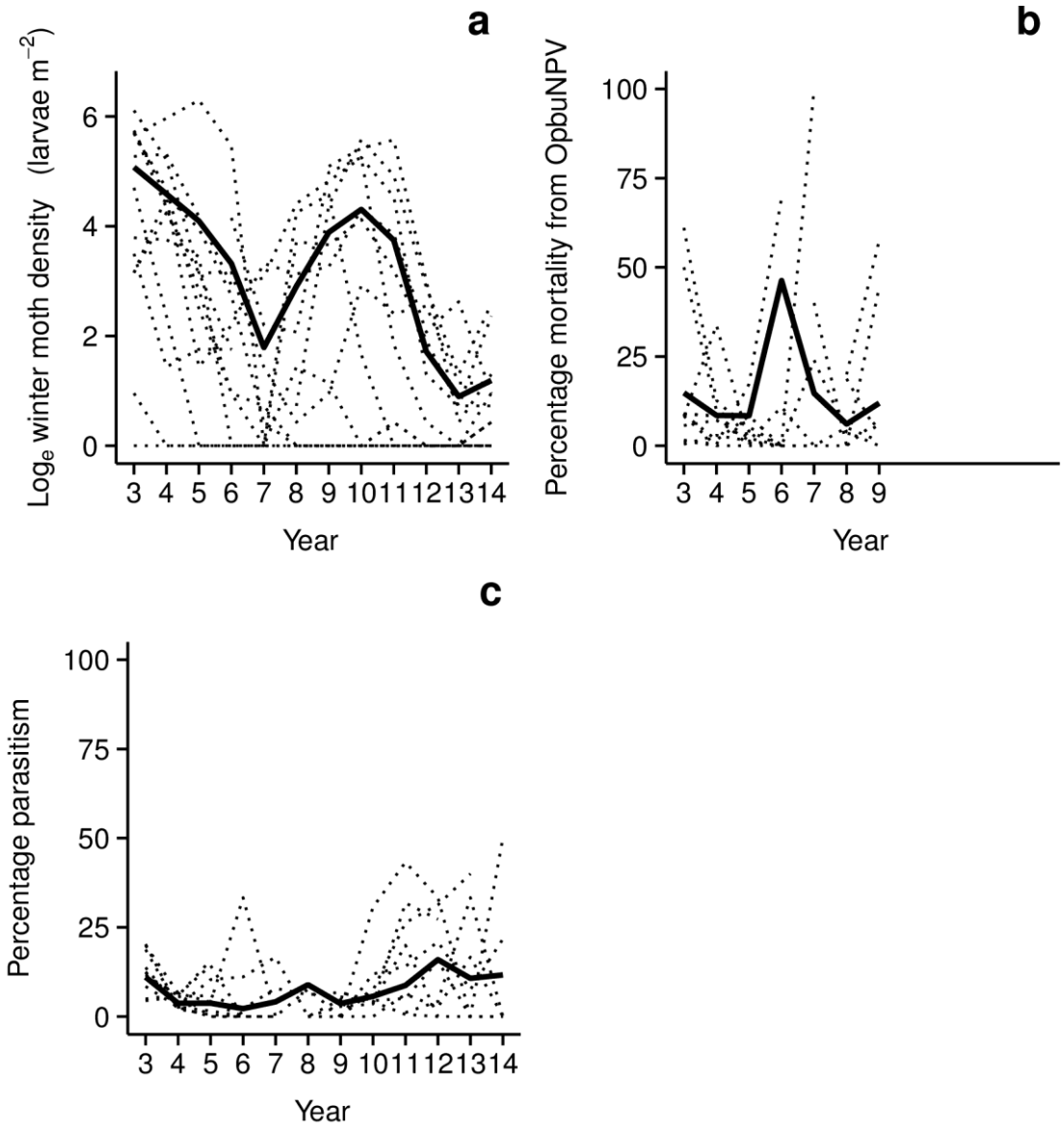

Figure 2.3 Yearly winter moth larval density on a $\log_e$ transformed scale (a), and percentage mortality from OpbuNPV (b) and parasitism (c). Dotted lines represent site-specific values, whilst the solid line represents either the mean $\log_e$ transformed density across all sites (a), or the total percentage of natural enemy mortality across all sites (b & c).

There was substantial between-population variation in larval mortality from parasitism and OpbuNPV over time, but no clear trends (Fig. 2.3 (b) & 2.3 (c)). OpbuNPV mortality data did not cover a full potential population cycle, but were available for the peak, post-peak, decline and most of the subsequent increase phase of one complete cycle across Orkney (i.e. the key stages of an entire cycle). In total from 2003-2014 7.1% of larvae were parasitised, and from 2003-2009 15.8% of larvae were killed by OpbuNPV infection (Fig. 2.3 (b) & 2.3 (c)), and from 2003-2009 a total of 19.1% of larvae were killed by both natural enemies together (Fig. 2.3 (b) & 2.3 (c)). The MMI analysis provided no strong evidence for the models explaining variation in the likelihood of winter moth larval parasitism in terms of either polynomial or linear effects of larval density and temperature during larval development in either year t or year t-1, compared to the null models (Table A1.1 & A1.2). Similarly, the MMI analysis provided no strong evidence for the models explaining variation in the likelihood of winter moth larval mortality from OpbuNPV in terms of either polynomial or linear effects of larval density and temperature during larval development in year t, compared to the null models (Table A1.3). However, there was strong evidence that the model explaining variation in the likelihood of winter moth larval mortality from OpbuNPV in terms of the linear effect of larval density and temperature during larval development in year t-1 was the best model (Table 2.1).

Table 2.1 AIC-based model selection table for models analysing the effect of larval density in year t-1 and temperature during larval development in year t-1 on the likelihood of OpbuNPV mortality in winter moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD_{-1} + T_{-1}	5	3252.124	0	0.987	0.987
Null	3	3260.733	8.609	0.013	1

LD_{-1} = larval density (year t-1), T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores.

This model indicated a medium-sized positive effect of larval density in year t-1, and a medium-sized negative effect of temperature during larval development in year t-1, on the likelihood of OpbuNPV mortality (Fig. 2.4 (a) and Fig. 2.4 (b); Table 2.2). Specifically, holding the effect of temperature at its mean level, the model predicted that the probability of larval mortality from OpbuNPV increased from 0.02-0.29 as winter moth larval density in year t-1 increased from its lowest to its highest observed value (Fig. 2.4 (a)). Similarly, holding the effect of larval density in year t-1 at its mean level, the model predicted that the probability of larval mortality from OpbuNPV decreased from 0.26-0.03 as temperature during winter moth larval development in year t-1 increased from its lowest to its highest observed value (Fig. 2.4 (b)). Therefore, there was only evidence for linear, delayed density-dependent mortality from OpbuNPV, and a delayed negative effect of temperature during larval development on mortality from OpbuNPV.

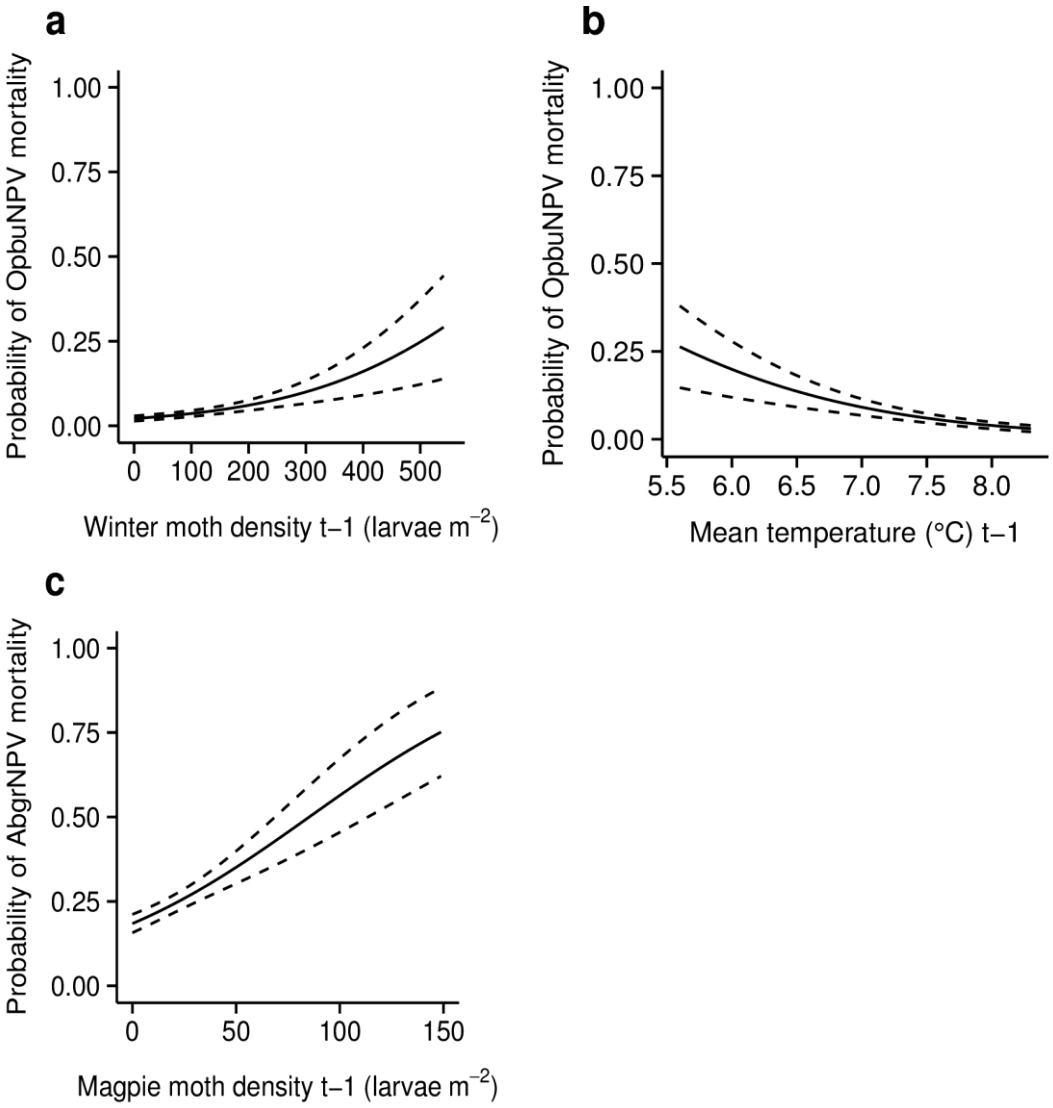

Figure 2.4 Logistic GLMM predictions (dashed lines are predicted mean probability ± 1 SE) for the relationship between winter moth larval density in year t-1 and the probability of larval mortality from OpbuNPV (a); and for the relationship between mean temperature during the early-mid instar developmental period of winter moth and the probability of mortality from OpbuNPV (b); and for the relationship between magpie moth larval density in year t-1 and the probability of larval mortality from AbgrNPV (c). All predicted values are based on fixed effects only, with the effects of temperature in year t-1 held at their mean level for (a) and (c), and the effects of larval density in year t-1 held at their mean level for (b). The predicted values are also back-transformed from the linear log-odds scale of the GLMMs to probabilities for ease of interpretation (this also leads to non-linearity on the probability scale).

Table 2.2 Parameter estimates and their 95% confidence intervals from the model analysing the effects of larval density in year t-1 and larval developmental temperature in year t-1 on the likelihood of OpbuNPV mortality.

Parameter	Estimate	UCI	LCI
LD_{-1}	0.861	1.383	0.4
T_{-1}	-0.493	-0.200	-0.785

LD_{-1} = larval density (year t-1) and T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development). Parameter estimates are untransformed from logistic GLMMs, and therefore represent the multiplicative change in the log-odds of OpbuNPV mortality given a 1 SD increase in the relevant explanatory variable.

2.4.2 Magpie moth population dynamics and natural enemy interactions

There were no apparent cyclical trends in the local population (within site) dynamics (Fig. 2.2 (b)), and when averaged across all local populations there was no regular pattern evident (Fig. 2.5 (a)), and no evidence of synchrony between populations across the study area (Pearson product-moment correlation -0.001, $p = 0.975$). There was substantial variation in the yearly levels of larval mortality from AbgrNPV between populations, and no clear overall trend (Fig. 2.5 (b)). The *Aleiodes* sp. parasitoid was not found in larval populations until 2010, but from 2010 onwards has undergone a clear, sustained population increase, with the overall level of parasitism broadly increasing over time (albeit with substantial variation) to a peak of approximately 25% in the final year of the study (Fig. 2.5 (c)). From 2006-2014 33.5% of larvae were killed by AbgrNPV infections, whilst from 2010-2014 14.8% of larvae were killed by parasitism, and from 2006-2014 40.9% of larvae were killed by both natural enemies together.

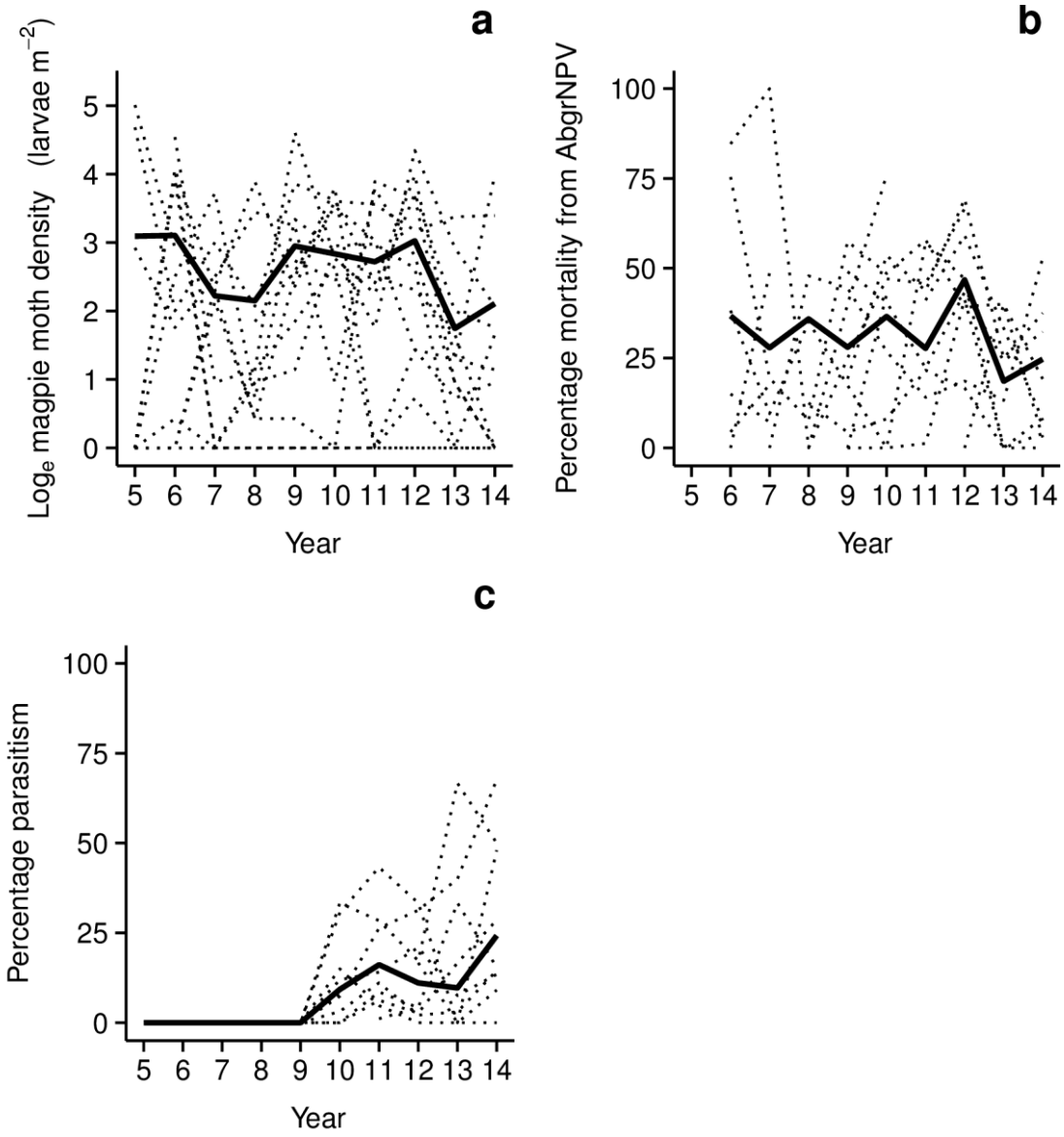

Figure 2.5 Yearly magpie moth larval density on a log$_e$ transformed scale (a), and percentage mortality from AbgrNPV (b) and parasitism (c). Dotted lines represent site-specific values, whilst the solid line represents either the mean log$_e$ transformed density across all sites (a), or the total percentage of natural enemy mortality across all sites (b & c).

The MMI analysis provided no strong evidence for the models explaining variation in the likelihood of magpie moth larval parasitism in terms of polynomial or linear effects of larval density and temperature during larval development in either year t or t-1, compared to the null models (Table A1.4 & A1.5). Similarly, the MMI analysis provided no strong evidence for the models explaining variation in the likelihood of magpie moth larval mortality from AbgrNPV in terms of either polynomial or linear effects of larval density and temperature during larval development in year t, compared to the null models (Table A1.6). However, there was strong evidence that the model explaining variation in the likelihood of magpie moth larval mortality from AbgrNPV in terms of the linear effect of larval density and temperature during larval development in year t-1 was the best model (Table 2.3).

Table 2.3 AIC-based model selection table for models analysing the effect of larval density in year t-1 and temperature during larval development in year t-1 on the likelihood of AbgrNPV mortality in magpie moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD_{-1} + T_{-1}	5	3968.291	0	0.997	0.997
Null	3	3979.858	11.567	0.003	1

LD_{-1} = larval density (year t-1), T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores.

This model indicated a large, positive effect of larval density in year t-1, but no clear effect of temperature during larval development in year t-1, on the likelihood of AbgrNPV mortality (Fig. 2.4 (c); Table 2.4). Specifically, holding the effect of temperature at its mean level, the model predicted that the probability of larval mortality from AbgrNPV increased from 0.18-0.75 as magpie moth larval density in year t-1 increased from its lowest to its highest observed value (Fig. 2.4 (c)). Therefore, there was only evidence for linear delayed density-dependent mortality from AbgrNPV.

Table 2.4 Parameter estimates and their 95% confidence intervals from the model analysing the effects of larval density in year t-1 and larval developmental temperature in year t-1 on the likelihood of OpbuNPV mortality.

Parameter	Estimate	UCI	LCI
LD_{-1}	0.486	0.771	0.201
T_{-1}	0.249	0.511	- 0.014

LD_{-1} = larval density (year t-1) and T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development). Parameter estimates are untransformed from logistic GLMMs, and therefore represent the multiplicative change in the log-odds of AbgrNPV mortality given a 1 SD increase in the relevant explanatory variable.

2.5 Discussion

As expected, the winter moth appeared to display cyclical dynamics, with local populations displaying synchrony across the study area. The winter moth population analyses also indicated medium-sized, delayed (but not direct) density-dependent mortality from OpbuNPV infection, but there was no evidence for any direct or delayed density-dependent mortality from parasitism. Additionally, there was also a medium-sized, delayed (but not direct) negative effect of temperature on mortality from OpbuNPV infection, but there was no evidence effect of temperature on parasitism. Also as expected, the magpie moth did not display clearly cyclical dynamics, either locally or across the study area, and there was also no population synchrony apparent across the study area. However, contrary to expectations there was strong, delayed (but not direct) density-dependent mortality from AbgrNPV infection, but no evidence for direct or delayed density-dependent mortality from parasitism, or for any effects of temperature on larval mortality from either natural enemy.

Theoretical models indicate that cyclical insect dynamics may be driven by delayed density-dependent mortality from parasitoids and pathogens (e.g. Anderson & May, 1980; Dwyer et al., 2004; Fuller et al., 2012; Ammunet et al., 2014), but the hypothesis has rarely been tested with host-enemy (particularly host-pathogen) time-series data from natural insect systems. The data that does exist provides mixed evidence for direct density-dependent natural enemy mortality from parasitism and disease, but the ability of natural enemies to generate delayed density-dependent mortality has apparently only been assessed for parasitoids (Kukan & Myers, 1999; Turchin et al., 2003; Hagen et al., 2010; Klemola et al., 2010; Schott et al., 2010; Liebhold et al., 2013; Klemola et al., 2014). However, an important condition for the validity of the natural enemy hypothesis is the observation of delayed density-dependent mortality from parasitoids and/or pathogens in real cyclical insect populations, which are subject to all the complex biotic and abiotic influences present in natural communities (Schott et al., 2010).

Therefore, the results from the winter moth mortality analyses presented here represent the first evidence of delayed density-dependent mortality from a specialist pathogen in a cyclical insect. Although detection of delayed density-dependent mortality by itself cannot prove population regulation through this mechanism (Liebhold et al., 2000; Ruohomaki et al.,

2000), it is at least consistent with the hypothesis that specialist natural enemies may drive cycles in insects (Anderson & May, 1980, 1981; Dwyer et al., 2004; Fuller et al., 2012). However, the delayed effect of winter moth larval density on mortality from OpbuNPV was only medium in size, with the model predicting that only around 30% of larvae succumb to OpbuNPV mortality when larval density in year t-1 was at its highest observed level. In contrast, in Fennoscandia where the cycles are often attributed to parasitism, mortality from parasitoids at peak and post-peak cycle phases can be two or three times higher; for example, up to 66% and 90% respectively (Hagen et al., 2010; Schott et al., 2010). Therefore, although consistent with OpbuNPV regulation, the relatively modest amount of mortality attributable to OpbuNPV at the highest population densities suggests that other factors may also play a role in winter moth population regulation on Orkney.

In contrast to OpbuNPV, there was apparently no influence from parasitism on winter moth dynamics. Some laboratory and theoretical studies have addressed host-parasitoid-pathogen dynamics, and shown that important changes can occur to host dynamics from the invasion of a new natural enemy into an existing host-enemy system (Hochberg et al., 1990; Begon et al. 1996; Sait et al., 2000). Therefore, in contrast to these studies, the results found here suggest limited or no important effects from multiple natural enemies on host dynamics. One issue with the winter moth's dynamics that was not addressed because of the limitations of the data was whether there were important interactions between the natural enemies, given the likelihood of within-host competition between the enemies, and the possible vectoring of the virus by the parasitoid (Cossentine, 2009). However, given the relatively stable rate of mortality from the parasitoid over the twelve years of the study for which data was available, and the apparent lack of influence of host density on levels of parasitism, *P. tempestiva*-OpbuNPV interactions appear unlikely to be very important for the regulation of cyclical winter moth dynamics on Orkney.

Unexpectedly, the magpie moth also displayed similar patterns of natural enemy mortality as the winter moth, with even stronger delayed density-dependent mortality from its NPV. However, although consistent with pathogen-driven population regulation, unlike the winter moth this apparently results in irregular dynamics, with no obvious multi-year cyclical patterns in either its local or regional dynamics. Some amateur reports of magpie moth outbreaks on heather moorlands in northern Scotland exist

(Horsfield & Macdonald, 2004), and as a probable recent arrival or re-arrival to Orkney magpie moth dynamics could still be in a transient stage before exhibiting greater cyclicity, but the species does not appear to be a typical outbreaking Lepidoptera and it seems more probable that this is not the case. Therefore, it is unclear why the magpie moth should not display cyclical dynamics if experiencing strong delayed density-dependent mortality. One possible explanation is that the magpie moth lacks other key traits typical of cyclical species, particularly the ability to increase its population size by a number of orders of magnitude during four or five generations of an increase phase of a population cycle (Myers & Cory, 2013). Certainly compared to the winter moth the magpie moth exhibited much smaller maximum population densities, potentially constrained by its greater body size.

Although the winter moth analyses were consistent with specialist pathogen regulation, a number of other possible mechanisms have been proposed as candidates for driving insect cycles. They may therefore help to explain winter moth dynamics on Orkney more fully, given the relatively modest increase in OpbuNPV mortality observed when lagged larval density increased. For example, defoliation induced changes in plant chemistry have direct impacts on larval growth rates, survival and fecundity, and may also influence host-parasitoid (Haukioja, 2005) and host-pathogen interactions (Raymond et al., 2002a; Raymond et al., 2005; Raymond & Hails, 2007), but at present these effects do not appear to be linked with population cycles in insects (Haukioja, 2005; Cory & Hoover, 2006; Kessler et al., 2012). Similarly, density-dependent prophylaxis (DDP), whereby insects increase their investment in immunity as the risk of infection increases (Wilson & Reeson, 1998), has been suggested as another possible cause of cyclical dynamics in insects, but again there is little empirical evidence for this at present (Hagen et al., 2006; Klemola et al., 2007; Reilly & Hajek, 2008). Density-linked maternal effects have also been proposed as providing a delayed density-dependent mechanism capable of generating population cycles (Ginzburg & Taneyhill, 1994), and multi-generational cyclical dynamics are known to occur in laboratory lepidopteran systems in the apparent absence of any influence from natural enemies (Bjornstad et al., 2001). However, the necessary changes in individual quality have not always been detected in field experiments (Inchausti & Ginzburg, 2009), and so far support for the hypothesis has been limited (Beckerman et al., 2002; Myers & Cory, 2013). Clearly though, there is still much that is not understood about the drivers of cyclical insect dynamics (including the Orkney winter

moth system), and whilst models can suggest plausible mechanisms, further progress will require hypotheses to be tested with more observational and, ideally, experimental data from natural systems, gathered at adequate spatial and temporal scales.

The results from the winter moth analyses also provided some interesting contrasts with previous work on natural enemy regulation of population cycles in the winter moth and similar Lepidoptera in other habitats. In Fennoscandian deciduous birch forests winter moth and autumnal populations peak every 9-10 years, often resulting in outbreaks and defoliation (Hogstad, 2005; Jepsen et al., 2013; Klemola et al., 2014). However, research on natural enemy regulation of these cycles has only focused on specialist parasitoids, because of the very high levels of parasitism seen during the peak and decline phases of both species cycles in Fennoscandian forests (Hagen et al., 2010; Schott et al., 2010). Therefore, although NPV epizootics in populations of both species may be associated with some outbreaks in Fennoscandian forests (T. Klemola, pers. comm.), their cycles are believed to be driven primarily by parasitoids (Klemola et al., 2010; Klemola et al., 2014). Consequently, the results presented here suggest that cyclical dynamics in the same species may be driven by qualitatively different specialist natural enemies in different habitats and/or geographical regions, something that does not appear to have been shown previously.

As there appears to be a much higher diversity and abundance of winter moth parasitoids in Fennoscandian forests (Hagen et al., 2010; Schott et al., 2010; Vindstad et al., 2010) compared to Orcadian moorlands, it may that in Fennoscandian winter moth populations the much richer and more abundant parasitoid community outcompetes the pathogen community for host resources. However, it was recently highlighted by Klemola et al. (2014) in a long-running observational study that specialist parasitism in the egg and pupal stages of the autumnal moth, and possibly also the winter moth, may play an important role in their population cycles. Therefore, it cannot be ruled out that parasitism of other life-history stages plays an important role in the dynamics of the winter moth on Orkney, and this clearly merits future exploration. Alternatively, differences between forest and moorland plant structure and/or chemistry may influence winter moth host-pathogen interactions (Cory & Hoover, 2006). However, the available evidence suggests that OpbuNPV actually persists for longer on pedunculate Oak leaves than heather plants, and that winter moth larvae infected by

OpbuNPV on Oak die sooner and yield more NPV than those infected on heather (Raymond et al., 2002a; Raymond et al., 2005), contrary to what might be expected if heather moorlands were more favourable to the virus than deciduous forests. Consequently, an important unanswered question is what determines which natural enemies play regulatory roles in cyclical insects' dynamics in different habitats and regions, and what determines their relative importance?

The length of the single observed, apparently complete, winter moth population cycle was just six years, compared to winter moth cycles in European deciduous forests, which are typically 9-10 years (Hogstad, 2005; Tenow et al., 2013). Therefore, although only an extremely tentative comparison can be made on the basis of a single cycle, Orkney moorland winter moth cycles may be shorter than those in European deciduous forests. Such a difference could be due to the regulation of winter moth populations on Orkney by OpbuNPV, rather than by specialist parasitoids (Klemola et al., 2010; Klemola et al., 2014). Alternatively, the periodicity of population cycles in insects and mammals often varies between different geographical regions and habitats, and modelling has indicated that this variation may be explained by variation in the presence, diversity and carrying capacity of generalist predator communities (Dwyer et al., 2004; Bjornstad et al., 2010; Allstadt et al., 2013). Consistent with this hypothesis is the fact that carabid communities, which are well-known predators of winter moth pupae (Raymond et al., 2002b), vary substantially between forest and moorland habitats (Ings & Hartley, 1999). Assessing these possibilities will first require much more time-series data on moorland winter moth population dynamics though.

All well-known cyclical Lepidoptera also exhibit spatially correlated population dynamics, often over substantial distances (Myers & Cory, 2013), which in some cases are the result of periodic travelling waves in abundance (Sherratt & Smith, 2008), such as the pan-European travelling waves now known to occur in forest-dwelling winter moth populations (Tenow et al., 2013), although it is not clear if such waves also occur within Britain (chapter 5). However, unlike the winter moth, the dynamics of magpie moth local populations were completely asynchronous across Orkney. Spatial population synchrony is believed to be driven by two main mechanisms: spatially correlated climatic stochasticity (i.e. Moran effects) (Grenfell et al., 1998; Post & Forchhammer, 2002), and dispersal of individuals and/or their natural enemies between local populations (Fox et al., 2011; Fox et al.,

2013). Clearly, both the magpie moth and the winter moth are subject to the same climate on Orkney though. In terms of dispersal, winter moth females are flightless and male dispersal is limited in range (Varley et al., 1973; Van Dongen et al., 1996). More recent genetic evidence suggests that winter moth populations on Orkney are actually quite well mixed though, and so subject to greater inter-population dispersal than previously assumed (Leggett et al., 2011). Little is known of magpie moth dispersal, but as a much larger-bodied species than then winter moth it is likely to be a comparatively better disperser (Sekar, 2012; Slade et al., 2013), and there have been reports of mass-migrations of magpie moth seen at least a few miles out to sea (R. Leverton, pers. comm.). Although inter-population dispersal of parasitoids does not appear to be a viable mechanism for winter moth synchrony on Orkney, OpbuNPV is known to be vertically transmitted to offspring (Graham, 2006), and so adult dispersal of OpbuNPV between local populations is very likely. However, vertical transmission of NPVs appears to be extremely common and expected evolutionarily (Burden et al., 2003; Burden et al., 2006; Vilaplana et al., 2008; Sorrell et al., 2009; Vilaplana et al., 2010), and so is likely to be occurring with AbgrNPV as well. Therefore, dispersal also seems an unlikely explanation for winter moth synchrony on Orkney, as the available evidence suggests it should be at least as important for magpie moth populations. Understanding of spatial synchrony in populations is clearly an ongoing endeavour, and as highlighted here probably the most important outstanding issue is the unknown role of local dispersal between populations (Myers & Cory, 2013).

Although stochastic (density-independent) fluctuations in climate have been shown to modulate cyclical dynamics, they are not generally believed to be capable of generating regular population cycles (Esper et al., 2007; Johnson et al., 2010). The results presented here indicated that the mean temperature during early-mid larval developmental stages had a moderately-strong, negative delayed effect on the likelihood of OpbuNPV mortality in the winter moth. Ascribing mechanisms to associations between climatic variables and ecological processes is very difficult to do with any certainty, because of the large number of possible correlations. However, NPV virions (within occlusion bodies) become inactivated by UV-radiation in sunlight (Cory & Myers, 2003). Therefore, one possible explanation for the negative, delayed effect of temperature on OpbuNPV mortality is that it reflected increasing inactivation of NPV in the environment in the prior year, due to elevated levels of solar radiation, reducing subsequent NPV infection prevalence. The size of the effect of temperature on OpbuNPV mortality was

comparable to that of larval density. Consequently, whatever the precise mechanism this highlights that climatic stochasticity, and therefore potentially climate change, has the potential to influence biotic interactions that may drive dynamical systems (Tylianakis et al., 2008; Martinez & Merino, 2011; Meisner et al., 2014). Therefore, although not generally believed capable of regulating cyclical species' dynamics, climatic effects may still have important implications for the nature of the resulting dynamics.

Therefore, this study provides the first evidence from natural populations of a cyclical insect of delayed density-dependent mortality from a pathogen, a necessary condition for pathogen-driven cyclical dynamics. The study also provides an interesting comparison between a cyclical moth and a non-cyclical moth, which appear to share very similar mechanisms of population regulation (and possibly population-outbreaking potential) within the same area and habitat, whilst exhibiting very different dynamics over similar time scales. This raises the question as to what makes some species display regular cycles, and not others, even when they appear to have the potential to do so. Further interesting contrasts were also apparent between winter moth dynamics on heather moorlands, and those in deciduous forests, where populations appear to be regulated by parasitoids rather than pathogens, and potentially exhibit longer cycles. Unfortunately datasets such as this are extremely rare, but are clearly very important to further the understanding of community dynamics.

3. Density-dependent prophylaxis and insect immunity in the wild

3.1 Abstract

Insects possess innate immune systems, but they are costly to maintain. Investment in insect immunity is therefore traded-off with other fitness-related traits. Insects are therefore expected to vary investment in their immune systems in relation to need, increasing investment when population density, and therefore the risk of attack from parasites, increases. This phenomenon is termed density-dependent prophylaxis (DDP), and has been reported in a range of insect taxa, but almost all studies on DDP have been laboratory based, with very few field tests. Additionally, DDP is primarily expected in species experiencing substantial fluctuations in density, particularly those displaying density-dependent phase polyphenism, such as many outbreaking pests. Again though, very few studies have investigated DDP in species not displaying these traits. Therefore, in this study the immunological functioning of the magpie moth, a species not known for outbreaking dynamics or density-dependent phase polyphenism, was assessed in wild populations, subject to attack from a specialist parasitoid and pathogen. Magpie moth immunity was measured using an assay of pupal encapsulation responses to nylon monofilaments, and related to population density and the prevalence of natural enemy mortality (as a proxy for the rate of attack by natural enemies) in both the current and previous year. Unexpectedly there was evidence for DDP in the magpie moth, and there was also an indication that defence against high levels of parasitoid attack in the previous year may have trans-generational costs for the immunity of offspring. This study showed that the functioning of density-related plastic changes in immunity in natural systems may be more variable than predicted by laboratory studies, and that DDP may be displayed by insects other than outbreaking, density-dependent phase polyphenic species.

3.2 Introduction

All animals experience attacks from parasitic natural enemies, with microparasites (e.g. bacteria, protozoa, fungi and viruses) and macroparasites (e.g. nematodes, mites and parasitoids) impacting all scales of biological organisation (Tompkins et al., 2011). At the individual scale, parasites can reduce host fitness through their impacts on key life-history traits such as growth, fecundity and ultimately survival (Cabodevilla et al., 2011; Hesketh et al., 2012). At the population scale, parasites may regulate host dynamics through sub-lethal (i.e. reduced fecundity) and lethal impacts on population abundance and population growth rates (Hudson et al., 1998; Sait et al., 2000; Albon et al., 2002; Klemola et al., 2010). Consequently, strong selection pressure from parasites has resulted in the evolution of many different methods of defence in animals, including complex immune systems (Schmid-Hempel, 2003, 2005). Although insects appear to lack adaptive immune responses, they possess well-developed innate immune systems that can provide defence against attacks by parasites such as parasitoids (Fors et al., 2014), mites (Yourth et al., 2002), bacteria (Schmitz et al., 2012) and viruses (Washburn et al., 1996). However, these systems are costly to maintain and deploy, and with finite resources available there is an inevitable trade-off between investment in immune functioning and other key determinants of individual fitness, such as growth, competition, longevity and fecundity (Kraaijeveld et al., 2002; Rantala & Roff, 2005; Cotter et al., 2011; Dmitriew, 2011).

Insect immune functioning is phenotypically plastic though, and investment in immunity is therefore predicted to be varied in relation to need (Schmid-Hempel, 2005). Microparasite transmission and risk of attack by macroparasites is usually expected to increase with rising host density (Anderson & May, 1981; Hassell, 2000; Ryder et al., 2007; Klemola et al., 2014). Therefore, animals experiencing strong variations in population density are expected to increase their investment in immunity as population density rises, in anticipation of an increased probability of parasite attack, a process that has been termed density-dependent prophylaxis (DDP) (Wilson & Reeson, 1998; Wilson & Cotter, 2009). DDP has been demonstrated in many insect taxa (Reeson et al., 1998; Barnes & Siva-Jothy, 2000; Wilson et al., 2002; Cotter et al., 2004a; Ruiz-Gonzalez et al., 2009). Alternatively though, invertebrate immune functioning may be negatively affected by high population density because of greater effects of stress from intra-specific

competition, leading to a reallocation of limited resources away from immunity and into other life-history traits (Reilly & Hajek, 2008). Immune functioning may also increase up to a threshold of population density, but beyond this decline due to increasingly negative impacts from crowding (Goulson & Cory, 1995; Piesk et al., 2013).

The evidence for DDP is broadly supportive (Wilson & Cotter, 2009), but comes almost entirely from laboratory studies of organisms reared at different densities under controlled conditions (e.g. Barnes & Siva-Jothy, 2000; Wilson et al., 2002; Bindu et al., 2012; Srygley, 2012; Kong et al., 2013), but the significance of these results for real populations in nature has rarely been tested with field studies. Specifically, very few studies have examined DDP using immune assays in organisms collected from wild populations, and therefore exposed to realistic natural conditions including multiple natural enemies, environmental variation and intra- and inter-specific competition. Indeed, there only appear to be three such studies that provide limited support for DDP compared to most laboratory-based studies: Miller and Simpson (2010) found a negative effect of population density on immune function in Australian plague locusts (*Chortoicetes terminifera*, Walker); Bailey et al. (2008) found increased immune functioning in high-, but also some low-, density populations of wild-collected Mormon crickets (*Anabrus simplex*, Haldeman); and Klemola et al. (2007) failed to find any evidence of DDP in wild-collected individuals of the autumnal moth.

Additionally, almost all research on DDP has focused on species that display outbreaking dynamics (high amplitude population cycles) and density-dependent phase polyphenism (e.g. Reeson et al., 1998; Wilson et al., 2002; Bindu et al., 2012; Wang et al., 2013). This is understandable since DDP is predicted to be most evident in species that experience the greatest variability in their risk of attack from natural enemies (such as outbreaking density-dependent phase-polyphenic species), and therefore can gain the most, in terms of fitness, by adjusting their investment in immunity according to when the risk is highest (Wilson & Reeson, 1998; Wilson & Cotter, 2009). However, insects may be usefully considered as existing along a continuum between constitutively solitary species (showing no phase polyphenic changes and rarely experience sizeable fluctuations in population density) and constitutively gregarious species (i.e. social insects that are constantly exposed to high population densities), with phase polyphenic species (that also typically experience substantial, regular fluctuations in density during outbreaks) existing as facultative intermediates

between these two extremes (Sword, 2002; Silva et al., 2013). Clearly though, most non-social species do not exhibit outbreaking dynamics or density-dependent phase polyphenism (Sword, 2002; Silva et al., 2013), and the relationship between population density and immune functioning has rarely been explored in such species. However, Piesk et al. (2013) found reduced immune functioning in Green-veined white (*Pieris napi*, L.) butterfly larvae reared at high densities, a species not known for outbreaking dynamics or density-dependent phase polyphenism. Therefore, it may be that DDP does not commonly occur in non-outbreaking species that do not display density-dependent phase polyphenism. If such species are rarely exposed to severe epizootics or aggregation of macro parasites such as parasitoids, then their fitness might instead be optimised at high population densities by reallocating resources away from immune functioning and into other key life-history traits (Piesk et al., 2013).

Therefore, this study primarily aimed to investigate the relationship between population density and immune functioning in the magpie moth, using individuals collected from field populations on Orkney heather moorlands. Winter moth could not be included in the study as originally planned, because of a population crash leading to unfeasibly low numbers in the field for collection (Fig. 2.3 (a)). The magpie moth does not display density-dependent phase polyphenism, and despite an amateur entomological report of unquantified 'large-scale outbreaks' of magpie moth larvae on northern Scottish heather moorlands (Horsfield & Macdonald, 2004), ten years' worth of population data from the area where the present study was conducted only indicates relatively modest, asynchronous local population fluctuations (chapter 2). It therefore does not appear to display the classical, high-amplitude, cyclical dynamics known in other outbreaking Lepidoptera (Esper et al., 2007; Myers & Cory, 2013). Therefore, it was hypothesised that there would a negative relationship between population density and immunity. In addition, although relationships between population density in previous years and immune functioning do not seem to have been empirically investigated (but for a relevant theoretical study see Reynolds et al., 2013), it was also investigated whether there was a relationship between lagged (by one year) population density and immunity, which was also hypothesised to be negative.

Immune functioning was measured via a pupal assay of the encapsulation response, which is a widely-used integrated measure of the strength of an individual's cellular and humoral immune system responses

(e.g. Siva-Jothy et al., 2005; Nagel et al., 2011; Srygley, 2012; Moreno-Garcia et al., 2013; Piesk et al., 2013). It is well established that the encapsulation response plays a key role in defence against macroparasites and microparasites including parasitoid eggs and/or larvae (Fors et al., 2014), nematodes (Sheykhnejad et al., 2014), mites (Yourth et al., 2002), bacteria (Schmitz et al., 2012) and protozoa (Volz et al., 2006). Additionally, some research has indicated that the insect encapsulation response can defend against NPV infections, through the isolation of infected tracheal cells within cellular capsules, preventing further spread of an infection (Washburn et al., 1996; Washburn et al., 2000; Trudeau et al., 2001). However, this has only been demonstrated in two species of Lepidoptera (Washburn et al., 1996; Washburn et al., 2000; Trudeau et al., 2001), and the occurrence of this process appears species and virus specific (Trudeau et al., 2001), and is therefore only seen as a tentative possibility here.

Prior exposure to parasite attack can also influence subsequent immune response in invertebrates, both within and between generations. Within generations attack from parasites may lead to immune suppression of hosts (Mahmoud et al., 2012; Ikeda et al., 2013), or alternatively to immune priming, whereby hosts exhibit up-regulated immune responses and resistance to subsequent challenges (Little & Kraaijeveld, 2004; Tidbury et al., 2011). The offspring of immune challenged individuals may also show reduced immune functioning indicative of trans-generational costs of immunity (Moreau et al., 2012), or they may show trans-generational immune priming, with up-regulated immune responses and increased resistance to subsequent immunological challenges (Moret, 2006; Tidbury et al., 2011). Therefore, as the magpie moth is known to suffer high levels of mortality from a specialist parasitoid and pathogen on Orkney (chapter 2; Hicks et al., 2015), it was also investigated whether the level of mortality from these two natural enemies in either the current or previous year (relative to the immunology assays) could also explain any differences in the encapsulation responses of individuals.

Individual body weight may also influence immune functioning, with variation in weight influencing the amount of resources available for investment in immunity (Zuk & Stoehr, 2002; Rantala & Roff, 2007; Piesk et al., 2013). Therefore, pupal weight was also recorded so that its effects on immune functioning could be controlled for in the analysis. Additionally, to independently assess stress factors influencing the magpie moth within the study, the same explanatory variables used to explain variation in the

encapsulation response (population density and natural enemy mortality in both the current and previous year) were also used to explain variation in pupal weight.

3.3 Methods

3.3.1 Study species, sampling protocol and sampling sites

Magpie moth larvae were sampled from 11 sites within heather habitats on Mainland Orkney, and one site within heather habitat on Rousay, between 24.05.2013 and 10.06.2013, and again between the 03.05.2014 and the 03.06.2014 (Fig. 3.1). All sites were separated from each other by a minimum distance of 2.68 km (and a maximum of 31.25 km), with magpie moth local populations separated by similar distances known to exhibit independent dynamics (chapter 2). The primary sampling protocol used in both years was identical, and followed a quadrat-transect method. This consisted of placing three 10 m transects parallel to each other, separated by 5 m, and then at 1m intervals along each transect using a 25 cm^2 quadrat to delineate an area from which all heather plants and any overhanging stems were carefully cut and placed in a plastic bag. The ground underneath this area was then searched for any living or dead magpie moth larvae, which were added to the heather collection. This resulted in 30 heather samples (10 per transect) per site, which were then carefully sorted through by hand to find any living or dead magpie moth larvae and any *Aleiodes* sp. parasitoid mummies (which attach themselves to stems). In 2014 additional collections were also made at each site to provide increased numbers of individuals for the immune assays. Additional collections were carried out around the immediate vicinity of the sampling transects, and were made by haphazardly selecting and shaking heather plants over a 50 x 50 cm tub for 30 seconds, and collecting all living magpie moth larvae found. This process was repeated until approximately 50 additional larvae were collected per site.

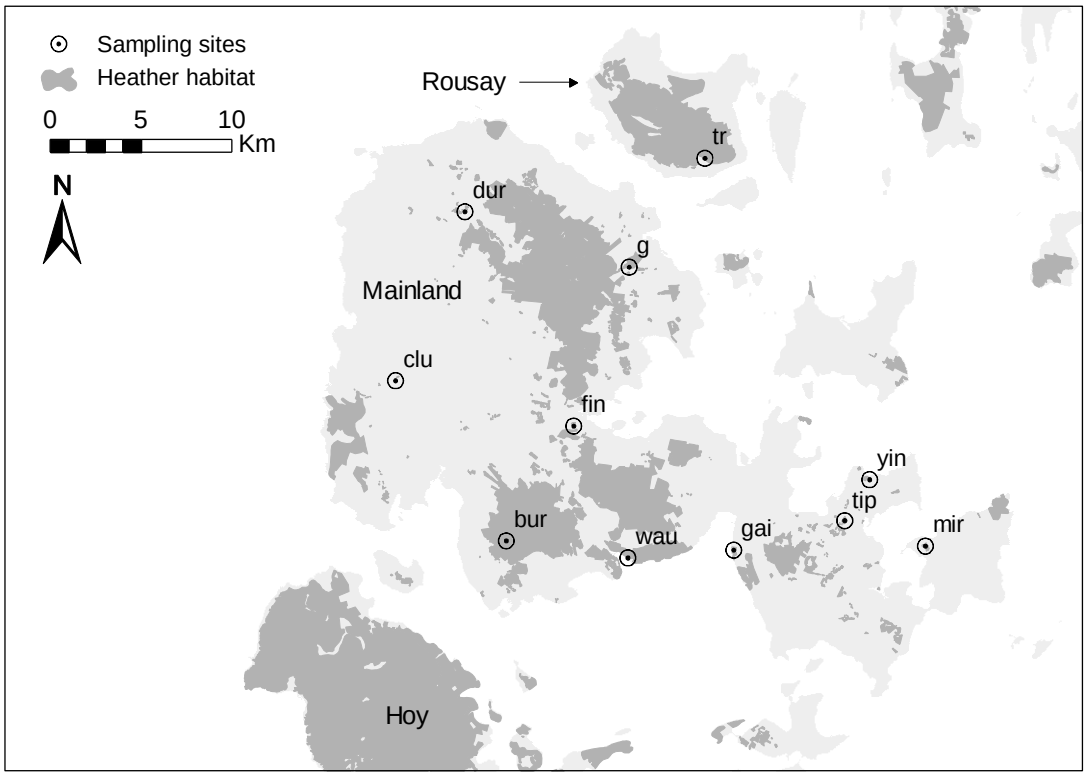

Figure 3.1 Distribution of sampling sites and heather habitat on Mainland Orkney and Rousay used in chapter 3.

All living magpie moth larvae were then reared individually in 25ml pots to determine whether they reached pupation, or died from parasitism or AbgrNPV infection. Larvae were provided with non-sterile green heather shoots to feed on *ad libitum* until pupation. To minimise altering the risk of AbgrNPV infection, shoots were initially taken from the collected heather samples of each relevant site, and once exhausted additional shoots were taken from isolated heather plants near a site known to have been free of magpie moth larvae for the last two years. Pots were stored at ambient conditions outdoors in perforated boxes, and every 1-3 days larvae were provided with fresh green heather shoots and any faeces removed to reduce mould contamination.

3.3.2 Measured variables

A measure of larval density, adjusted to units of larvae per m^2, was calculated for each site in 2013 and 2014 based on the total number of individuals present in the 30 quadrat heather samples from each site, including any living or dead larvae and any parasitised individuals. Any larvae in these samples found to be dead upon collection or during subsequent rearing checks were frozen and later tested for AbgrNPV infection using Giemsa staining methods (Lacey, 2012), and any larvae in these samples collected as, or subsequently developing into, *Aleiodes* sp. parasitoid mummies were classed as parasitised. Consequently, a measure of percentage AbgrNPV mortality and percentage parasitism was calculated for each site in 2013 and 2014 based on the larval collections from the quadrat heather sampling, with the percentage based on the total number of larvae killed by either AbgrNPV infection or parasitism per site and the total number of individuals per site. These measures represented proxy measures for the risk of attack by AbgrNPV and the *Aleiodes* sp. parasitoid in each year.

In 2014 all larvae (from both the main and additional collections) reaching pupation were also subjected to an immune response assay, which was carried out on pupae 5 days after pupation to standardise the developmental stage of the pupae (pupation lasts 2-3 weeks). Immune responses also vary with life-history traits, particularly weight, sex and age (Zuk & Stoehr, 2002; Rantala & Roff, 2007; Stoehr, 2007). Therefore, immediately prior to the assays pupae were also weighed (to the nearest mg) and sexed, and the assay date recorded as a proxy for individual age,

so that the influence of these factors on the immune assay could be controlled for. As an assay of immune defence strength, the insect encapsulation response was induced in pupae using an 'artificial parasite' (Rantala & Roff, 2007). The 'artificial parasite' consisted of a 2mm-long piece of nylon monofilament (0.2mm diameter) with a knot at one end, that had been rubbed with ultra-fine sandpaper to produce a roughened surface to facilitate encapsulation. Implants were first washed in 70% ethanol to kill and remove any microorganisms that might influence the encapsulation response (Stoehr, 2007). A sterilised (using 70% ethanol) insect mounting pin (size 0) was then used to make a small hole in the second segment of the pupal cuticle, centrally on the dorsal side, and an implant inserted into the haemocoel up to its knot. Pupae were then left for two hours to allow the development of an encapsulation response on the surface of the implant, which was then carefully removed and stored at -20°C. Preliminary trials showed that a time of two hours allowed most pupae to produce a sizable level of melanotic encapsulation on implants, whilst still allowing variation between individuals to be detected. As the immune assays had to be carried out at accommodation near the field sites they were conducted at room temperature, which varied from 17.5-20.5°C. Room temperature was therefore recorded for each assay, so that it could be controlled for in the analysis.

Subsequently, each implant was photographed in a random order at two different angles (capturing each side) on a white background, using a dissecting light microscope at 3x magnification and a digital microscope camera, with a standardised lighting setup. An encapsulation response measure was then created from each image based on standard methods (e.g. Stoehr, 2007). This involved using the image software ImageJ (Rasband, 1997-2014) to convert all images to greyscale, and within each image the average grey-value (where 0 is black and 255 is white) of the area covered by each implant below its knot was then recorded (i.e. the area of implant inserted into the pupae). To account for unavoidable slight differences in lighting between photographs the average grey-value of the area outside of each implant in each image (i.e. the background of the image) was subtracted from the each implant's average grey-value. Then an overall, background-corrected average grey-value was created for each implant from the two average background-corrected grey-values taken of the implant at each angle. The resulting encapsulation score ran from 0 to 255, and provided a combined proxy measure of the thickness of the cellular layer resulting from the encapsulation response and the degree of

melanisation, representing a measure of the strength of the encapsulation response, with higher scores representing a stronger encapsulation response (Rantala & Roff, 2007).

3.3.3 Statistical analyses

The strength of invertebrate immune responses, including melanotic encapsulation, typically covaries with body size/weight (e.g. Zuk & Stoehr, 2002; Stoehr, 2007; Piesk et al., 2013), and is often different for males and females (Stoehr, 2007). Therefore, the effects of pupal weight and sex were accounted for in the analysis of the encapsulation score data. In addition, to gain independent information on the factors causing stress to magpie moth larvae, the pupal weight data itself was also directly analysed using the same explanatory variables as those used to analyse the encapsulation score data (population density and population-level natural enemy mortality in the current and previous year).

Preliminary analysis indicated that the encapsulation score and pupal weight data varied non-linearly and irregularly with the date of assay. Therefore, additive mixed-effects models (AMMs), fitted by maximum likelihood methods, were used to analyse the two data sets, allowing the analyses to control for the irregular, non-linear effect of assay date via a smoothing effect, whilst analysing the effects of other explanatory variables in a standard linear mixed-effects modelling framework (Wood, 2006; Zuur et al., 2009). The analyses followed a MMI process (Burnham & Anderson, 2002), which involved creating a set of AMMs to represent different hypotheses about the importance of the explanatory variables 'larval density in 2013 and 2014', 'percentage parasitism in 2013 and 2014' and 'percentage AbgrNPV mortality in 2013 and 2014', in terms of their explanatory power relating to the encapsulation score and pupal weight data. For the encapsulation score data, aside from a null model all other AMMs always also contained explanatory variables for the effects of pupal weight, pupal sex and a smoothing effect of assay date, so that their influence could always be controlled for. Similarly, for the pupal weight data, aside from a null model all other AMMs always also contained explanatory variables for the effect of sex and a smoothing effect of assay date, so that their influence could always be controlled for. Additionally, for both the encapsulation score and pupal weight data analyses, site was included in all

models as a random factor to account for the clustered nature of the data, given the spatial clustering of larvae within sites (Zuur et al., 2009).

Preliminary analysis indicated no effect of assay temperature on encapsulation score, and so this explanatory variable was not considered further. Preliminary analysis also did not indicate the presence of interactions between the remaining explanatory variables, and so only main effects were considered. Therefore, a set of 17 models was then created for the encapsulation score and pupal weight data to represent a practical range of plausible hypotheses about which potential predictor variables had important effects on the observed encapsulation scores and pupal weights of sampled magpie moth (Table A2.1 & Table A2.2). For each dataset the resulting set of models were then ranked based on their AIC scores, and the Akaike weight calculated for each model (Burnham & Anderson, 2002).

All Akaike weights in each model set were <0.95, indicating that no single model was clearly the best explanation of the data (Burnham & Anderson, 2002). Therefore, a 95% confidence set of models was created from each full model set (Burnham & Anderson, 2002). Any models containing uninformative parameters were then removed from these model sets (Arnold, 2010), resulting in a final set of models (Table 3.2 & Table 3.4). Inference was then based on parameter estimates and their 95% confidence intervals when variables were only present in a single model in the set, or on model-averaged parameter estimates and their 95% confidence intervals when variables were present in multiple models (Burnham & Anderson, 2002). Model-averaged parameter estimates were calculated using the natural-average method (Arnold, 2010), with their 95% confidence intervals based on unconditional standard errors (Burnham & Anderson, 2002). Prior to model fitting all continuous explanatory variables were standardised by dividing by 2 standard deviations to place them on the same scale, which allows for careful interpretation of the comparative magnitudes of parameter estimates, both continuous and binary (Gelman, 2008).

Based on the residuals from the best AIC scoring model, spatial autocorrelation in model residuals was assessed using spline correlograms with 95% confidence envelopes from the R package *ncf* (Bjornstad, 2009), but was not evident. Similarly, AMM statistical assumptions were checked using plots of model residuals against fitted values and explanatory variables, and QQ normality plots, again based on the residuals from the best AIC scoring model (Wood, 2006; Zuur et al., 2009).

3.4 Results

3.4.1 Magpie moth population dynamics and natural enemy mortality

Between 2013 and 2014 overall larval density dropped by 31.1%, and the maximum larval density decreased from 88.8 to 50.1 larvae m^{-2}, although the 95% confidence intervals for the mean larval density in 2013 and 2014 overlapped substantially indicating substantial variation between sites in each year (Table 3.1). There was an 8.5 and a 7.1 fold difference between the minimum and maximum densities in 2013 and 2014 respectively, indicating that broadly similar levels of variation in population density, which may influence DDP, occurred in both years (Table 3.1). Mean site-level parasitism increased minimally between 2013 and 2014, with substantial overlap in the 95% confidence intervals of each mean value, and there was also little change in the range of values recorded indicating largely similar average levels of parasitism across the study area in each year (Table 3.1). Despite the decrease in overall larval density, most sites displayed increases in AbgrNPV mortality in 2014 compared to 2013 with minimal overlap in the 95% confidence intervals, resulting in a nearly 2.2 fold increase in mean site-level AbgrNPV mortality, along with a substantial upward shift in the range of values found (Table 3.1).

Table 3.1 Mean (± 95% confidence interval) and range in site-level larval density and natural enemy mortality in 2013 and 2014.

Year	Larval density (larvae m^{-2})	Parasitism (%)	AbgrNPV mortality (%)
2013	34.7 (± 21.4-47.9); 10.4-88.8	11.6 (± 7.1-19.2); 0-33.3	17.2 (± 5.7-28.7); 0-60.3
2014	23.9 (± 10.7-37.1); 6.9-50.1	13.1 (± 5.5-17.7); 3.2-36.9	37.7 (± 26.2-49.3); 7.7-72.6

3.4.2 Immune responses

There was only minimal variation in the encapsulation across all pupae (overall mean encapsulation score = 111.75, SD = 12.13, CV = 0.11, Fig. 3.2), and most variation in scores occurred between sites rather than within, indicating that most variation was at an individual host level (Fig. 3.3). There was a lack of support for any single model, with the MMI analysis of the encapsulation data resulting in a final set of eight competitive models (Burnham & Anderson, 2002) (Table 2.2) out of the original 16 (Table A1.1), and overall a large amount of the variation in encapsulation scores was not explained by the analysis, as indicated by the low R^2 values of all competitive models (Table 2.2). As well as the fixed variables in all models, the top ranked model contained an effect of 2014 larval density, whilst explanatory variables for each year's population density and natural enemy mortality were all present in at least one other model (Table 2.2). There was a clear, positive effect of 2014 larval density; a marginal, negative effect of 2013 percentage parasitism; and a clear, positive effect of pupal weight on the encapsulation scores (Table 2.3). Interpreting standardised coefficients should be done with care (Gelman, 2008), but the sizes of the effects of 2014 larval density, 2013 percentage parasitism and pupal weight were all weak, and broadly comparable in their absolute magnitude (Table 2.3). Based on their 95% confidence intervals there was little support for any effects of sex, 2013 larval density, 2013 and 2014 percentage AbgrNPV mortality and 2014 percentage parasitism on the encapsulation scores (Table 2.3).

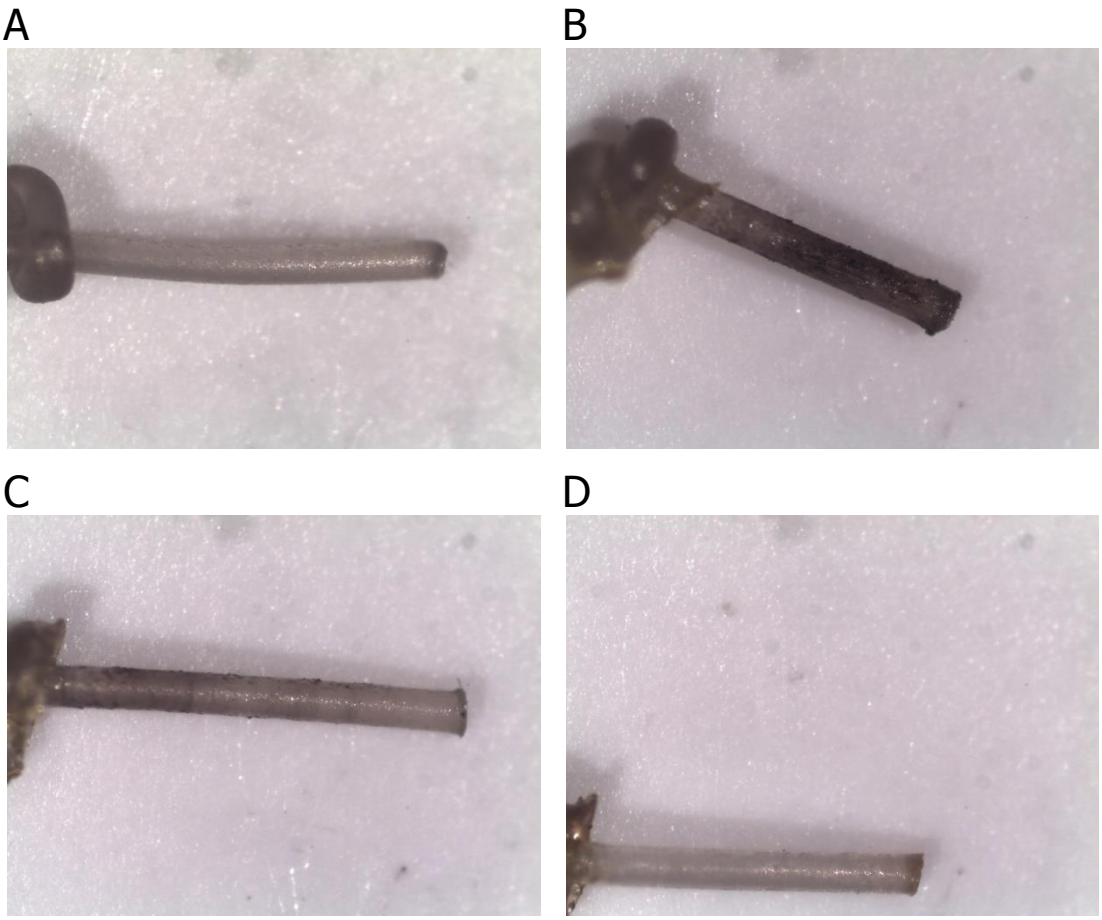

Figure 3.2 Representative range in degree of melanotic encapsulation of nylon implants used as artificial 'parasites' to assay the strength of the immunological encapsulation response of magpie moth pupae. A = control implant, never inserted into pupal haemocoel; B = approximately maximum level of melanotic encapsulation; C = approximately mean level of melanotic encapsulation; D = approximately minimum level of melanotic encapsulation.

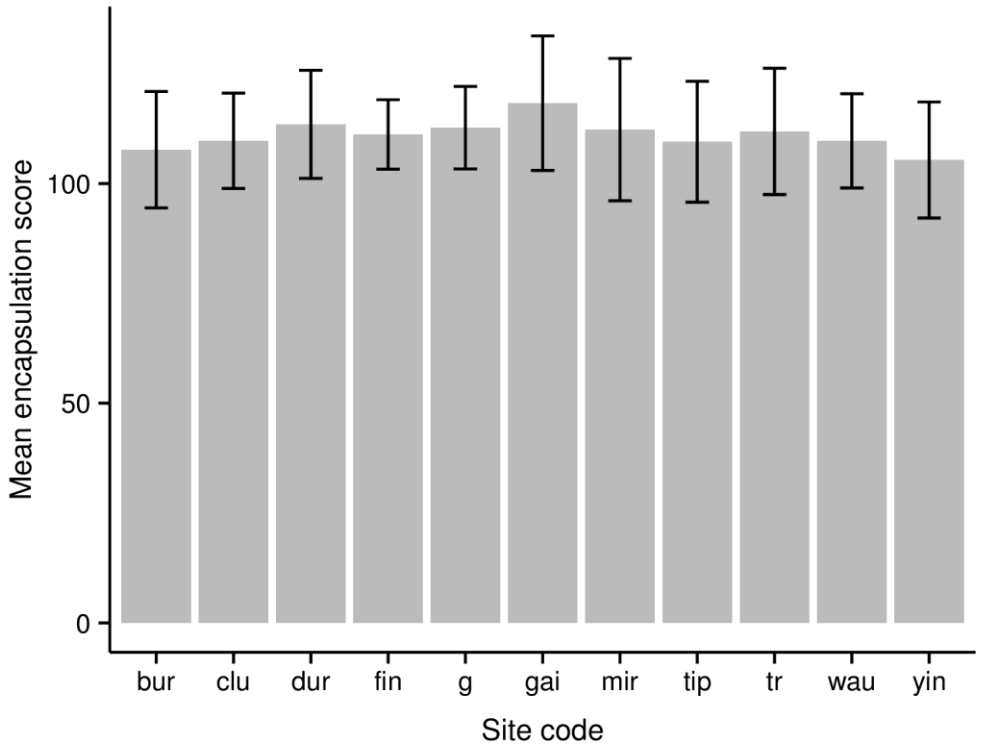

Figure 3.3 Mean encapsulation score values (± 1 SE) within sites. Site codes refer to the map codes used in Fig. 3.1. Encapsulation scores represent the degree of melanotic encapsulation of nylon implants, used as artificial 'parasites' to assay the strength of the immunological encapsulation response of magpie moth pupae.

Table 3.2 Model selection table for the final encapsulation data models remaining after the model selection process, with models ranked based on the difference in their AIC value from that of the lowest AIC scoring model. K = number of parameters in model; R^2 = adjusted R^2 for the number of predictors in models.

Model	K	R^2	ΔAIC	Akaike weight	Cumulative Akaike weight
S + W + AD + L14	8	0.068	0	0.455	0.455
S + W + AD + P13 + V13	9	0.063	2.2	0.153	0.608
S + W + AD + P13	8	0.059	2.2	0.15	0.758
S + W + AD + V13	8	0.052	3.8	0.068	0.826
S + W + AD	7	0.045	4.3	0.053	0.879
S + W + AD + L13	8	0.05	4.5	0.049	0.928
S + W + AD + V14	8	0.043	5	0.037	0.965
S + W + AD + P14	8	0.045	5.2	0.035	1

Variable codes represent the following: S = sex; W = Pupal weight; AD = Assay date (smoothed effect); L13 = 2013 larval density; L14 = 2014 larval density; P13 = 2013 % parasitism; P14 = 2014 % parasitism; V13 = 2013 % AbgrNPV mortality; V14 = 2014 % AbgrNPV mortality. All models also contained site as a random factor.

Table 3.3 Fixed effects estimates for non-smoothed terms, their standard errors and 95% confidence intervals based on the final set of AMMs remaining following the MMI analysis of the encapsulation data.

Variable	Parameter estimate	Standard error	LCI	UCI
Sex (male)*	-1.05	1.94	-4.88	2.78
Pupal weight	5.07	1.94	1.24	8.9
2014 larval density	4.31	1.67	1.03	7.59
2013 % parasitism	-3.27	1.62	-6.47	-0.08
2013 % AbgrNPV mortality	-2.48	1.7	-5.8	0.84
2013 larval density	-2.73	1.87	-6.41	0.96
2014 % AbgrNPV mortality	2.27	1.99	-1.65	6.19
2014 % parasitism	-1.99	1.86	-5.66	1.69

* Treatment contrast parameter, which represents the model-averaged difference in mean encapsulation scores in male pupae compared to female pupae.

Parameter estimates are based on standardised data, and therefore represent the additive change in encapsulation score that is predicted to occur following an increase in the given explanatory variable by 2 standard deviations (i.e. from broadly low to high values). Standard errors and 95% confidence intervals for larval density in 2013 and 2014, 2014 % AbgrNPV mortality and 2014 % parasitism are conditional on the single model these parameters were present in, but all other standard errors and 95% confidence intervals are based on unconditional (model-averaged) estimates.

3.4.3 Pupal weight

Variation in body weight can have important effects on an individual's immune functioning by determining the amount of resources available for investment, and so factors that affect body weight may have indirect influences on immunity (Vogelweith et al., 2013b). Compared to the encapsulation score data, there was more variation between sites, but most variation was still occurred at an individual host level (Fig. 3.4). The MMI analysis of the pupal weight data resulted in a final set of eight competitive models (Table 3.4) out of the original set of 16 (Table A1.2). All remaining models were broadly comparable in their ability to explain the data (Table 3.4). Relative to the encapsulation data models, there was also uniformly much less unexplained variation (Table 3.4). There was a clear, negative effect of 2014 larval density; a clear, negative effect of 2014 percentage AbgrNPV mortality; and a clear, negative effect of being male, with males being on average 23.48mg (18.4%) lighter than females (Table 3.5). The standardised coefficients indicated that the effects of 2014 larval density and 2014 percentage AbgrNPV mortality were quite weak and comparable in magnitude (Table 3.5).

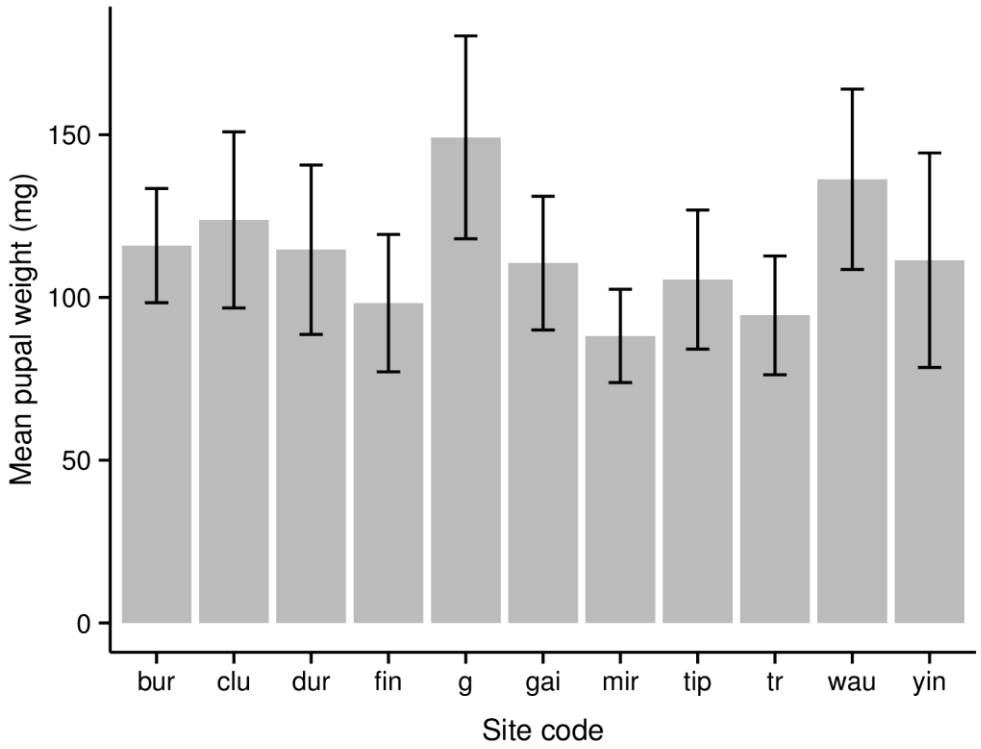

Figure 3.4 Mean pupal weight (mg) values and their 95% confidence intervals between sites.

Table 3.4 Model selection table for the final pupal weight data models remaining after the model selection process, with models ranked based on the difference in their AIC value from that of the lowest AIC scoring model. K = number of parameters in model; R^2 = adjusted R^2 for the number of predictors in models.

Model	K	R^2	ΔAIC	Akaike weight	Cumulative Akaike weight
S + AD + L14 + L13	8	0.375	0	0.309	0.309
S + AD + V14	7	0.328	0.4	0.255	0.564
S + AD + L14	7	0.352	1.4	0.15	0.714
S + AD	6	0.301	2.7	0.079	0.793
S + AD + P14	7	0.303	3	0.068	0.861
S + AD + L13	7	0.314	3.3	0.061	0.922
S + AD + P13	7	0.306	3.7	0.048	0.97
S + AD + V13	7	0.298	4.7	0.03	1

Variable codes represent the following: S = sex; W = Pupal weight; AD = Assay date (smoothed effect); L13 = 2013 larval density; L14 = 2014 larval density; P13 = 2013 % parasitism; P14 = 2014 % parasitism; V13 = 2013 % AbgrNPV mortality; V14 = 2014 % AbgrNPV mortality. All models also contained site as a random factor.

Table 3.5 Fixed effects estimates for non-smoothed terms, their standard errors and 95% confidence intervals based on the final set of AMMs remaining following the MMI analysis of the pupal weight data.

Variable	Parameter estimate	Unconditional standard error	LCI	UCI
Sex (male)*	-23.48	3.14	-29.67	-17.29
2014 % AbgrNPV mortality	-15.6	6.9	-29.28	-1.95
2014 larval density	-14.5	6.2	-26.74	-2.26
2013 larval density	-10.6	5.93	-22.28	1.08
2013 % parasitism	8.2	8.12	-7.8	24.2
2014 % parasitism	10.01	7.49	-4.74	24.77
2013 % AbgrNPV mortality	-0.18	6.76	-13.5	13.13

* Treatment contrast parameter, which represents the model-averaged difference in mean encapsulation scores in male pupae compared to female pupae.

Parameter estimates are based on standardised data, and therefore represent the additive change in encapsulation score that is predicted to occur following an increase in the given explanatory variable by 2 standard deviations (i.e. from broadly low to high values). Standard errors and 95% confidence intervals for 2013 and 2014 % AbgrNPV mortality and 2013 and 2014 % parasitism are conditional on the single model these parameters were present in, but all other standard errors and 95% confidence intervals are based on unconditional (model-averaged) estimates.

3.5 Discussion

Analysis of the immune assay data provided evidence for small increases in the observed encapsulation responses with increasing population density in the current (but not the previous) year, and with increasing pupal weight. In addition, there was evidence for small decreases in the observed encapsulation responses with increasing population-level parasitism in the previous (but not the current) year. The analysis of the pupal weight data provided evidence that males were approximately 18% lighter than females, and that there were small decreases in pupal weight with increasing population density in the current (but not the previous) year, and with increasing population-level mortality from the virus AbgrNPV in the current (but not previous) year.

Consequently, the analysis of pupal weight suggested that larvae from higher density populations suffered greater stresses than those in lower density populations, with reduced body weight likely resulting from increased intra-specific competition leading to reduced available resources and/or reallocation of more limited resources away from growth and into other life-history traits (Agnew et al., 2002; Walsh et al., 2011; Piesk et al., 2013). Pupal weight is often positively related to immune functioning (Zuk & Stoehr, 2002; Rantala & Roff, 2007; Piesk et al., 2013), but despite this individuals in higher density populations actually showed elevated encapsulation responses. Therefore, contrary to the primary hypothesis of a negative relationship between population density and immune functioning, there was evidence consistent with DDP occurring in the magpie moth. However, there was no evidence for any lagged effect of population density (in the year prior to the immune assays) on immune functioning, implying that the mechanism is likely based on within-generation cues about population density (Gunn, 1998). Although there was no relationship between population density in the previous year and immune functioning, it would seem that the possibility of DDP or negative immune responses to lagged population density increases merits further attention, given the delayed density-dependent natural enemy attack many outbreaking, and some non-outbreaking, insects suffer (chapter 2).

Given that the magpie moth does not exhibit outbreaking dynamics (i.e. regular population peaks across large areas leading to extensive defoliation) or density-dependent phase polyphenism (unlike the vast majority of species in which DDP has been investigated), the evidence for

DDP in the magpie moth provides an interesting comparison with the few studies in which DDP has been investigated in species also sharing neither, or just one, of these characteristics. Only three such studies appear to exist, and in the Green-veined white, a butterfly not known for outbreaking dynamics or density-dependent phase polyphenism, Piesk et al. (2013) found that all four measured immune parameters decreased with rearing density, indicating that the high costs of immunity resulted in traded-offs with other life-history traits more beneficial to fitness under stressful high density conditions. However, in the autumnal moth, Klemola et al. (2007) found neither evidence for DDP or for a negative effect of population density on the encapsulation response of assayed larvae, and in the gypsy moth Reilly and Hajek (2008) found reduced resistance to the NPV LdMNPV in larvae reared at higher densities. Although both of these species do not display density-dependent phase polyphenism, they do display regular outbreaking dynamics, usually followed by high levels of mortality from parasitoids and pathogens (Dwyer et al., 2004; Klemola et al., 2014).

As the Green-veined white is unlikely to be regularly exposed to very high population densities, which are known to be associated with epizootics and high levels of parasitism (chapter 2; Klemola et al., 2007; Myers & Cory, 2013), and potentially as a consequence has not evolved density-dependent phase polyphenism, its reduced immune functioning at high population densities (contrary to the DDP hypothesis) may result from a reallocation of resources away from immunity and into other life-history traits more optimal for fitness under conditions of high population density. However, although the autumnal moth and the gypsy moth do not display density-dependent phase polyphenism, they both display strongly cyclical outbreaking dynamics (Dwyer et al., 2004; Klemola et al., 2007), and should therefore be expected to strongly benefit from DDP (Wilson & Reeson, 1998). On Orkney, although magpie moth local populations do fluctuate sometimes substantially from year to year (chapter 2), and experience high levels of mortality from a pathogen and increasingly a parasitoid (chapter 2), the magpie moth does not exhibit classical outbreaking dynamics like those in the autumnal moth and gypsy moth. Therefore, it is unclear why the magpie moth would display DDP, but not the autumnal moth or gypsy moth. However, this does suggest that DDP may be more widely present in insects not typically viewed as benefiting strongly from the mechanism (Wilson & Reeson, 1998), and that the conditions under which it is favoured may be more species specific than previously assumed. Clearly, further studies are needed in species not displaying the typical traits predicted to lead to DDP in order to understand

the circumstances in which DDP may evolve and function. For example, it is not understood how important specific key natural enemies (and for example their functional responses) might be in influencing the evolution and functioning of DDP in hosts in different contexts.

Very few studies have explored relationships between population density and immune functioning in natural systems using wild collected individuals, and those that exist have either failed to find evidence for DDP Klemola et al. (2007), found mixed evidence Bailey et al. (2008), or found evidence for negative effects of population density on immunity Miller and Simpson (2010). All three of these studies involved species known to display outbreaking dynamics, whilst the species used by Miller and Simpson (2010) and Bailey et al. (2008) (Australian plague locusts and Mormon crickets respectively) also display density-dependent phase polyphenism. Therefore, again there is an interesting contrast between the results of these studies, involving species strongly predicted to display DDP (Wilson & Reeson, 1998), and the present results supportive of DDP in wild populations of magpie moth, a species not clearly expected to display DDP.

One possible explanation for these differences may be related to trade-offs between immunity and other life-history traits in natural settings. It is well known that the investment of resources into immune systems must be balanced against the costs imposed on other fitness-related traits, leading to trade-offs between immune functioning and other key life-history traits (Kraaijeveld & Godfray, 1997; Kraaijeveld et al., 2001). Immunological functioning is therefore predicted to be condition-dependent (Lazzaro & Little, 2009), and in insects factors such as temperature and diet quality are known to influence observed immune responses (Frid & Myers, 2002; McVean et al., 2002). Recently, Triggs and Knell (2012) demonstrated that whilst the Indian mealmoth (*Plodia interpunctella*) exhibits DDP when food quality is high, but when food quality is poor, and temperature low, larvae reared at higher densities actually exhibit reduced immune functioning. Therefore, it may be that when conditions become highly stressful in natural populations, particularly during outbreaks, species displaying DDP at lower population density increases can no longer maintain the increased investment in immunity due to the need for limited resources to be reallocated to other key life-history traits, resulting in increasingly reduced immune functioning as density increases above a threshold (Goulson & Cory, 1995; Piesk et al., 2013). Conversely, because the magpie moth only experienced moderate fluctuations in population density, its populations are

unlikely to have reached such a threshold, and may therefore have been able to increase immune investment at higher population densities. It is not clear whether highly sub-optimal conditions at the highest densities were present in the studies discussed, but in both Miller and Simpson (2010) and Bailey et al. (2008) primarily outbreaking populations were sampled, where high levels of stress might reasonably be expected. Therefore, across the range of population densities they surveyed, the species may already have been above such a threshold. Again, further studies are necessary, focusing on the relationships between population density, immune function and other influential factors in both controlled, and particularly natural settings, in order to better understand how population density-immunity relationships function in natural systems.

A limitation of the present study, enforced through the difficulties of measuring immunological parameters outside of laboratory conditions, is the use of a single measure of immunity. Care should be taken when using single measures of immunity to infer differences in overall resistance against natural enemies (Klemola et al., 2007), because different types of immune response are directed at different attacking organisms (Adamo, 2004), and there also appear to be resource-based and genetic trade-offs between different components of invertebrate immune systems (Cotter et al., 2004a; Cotter et al., 2004b). Natural enemies may also possess methods for evading certain immune defences, such as the polydnaviruses, virus-like particles and venoms possessed by Hymenopteran parasitoids, which act to nullify elements of their host's immune systems (Beckage, 1998). Therefore, although there is direct support for viewing encapsulation assays as realistic measures of resistance against parasitic attack (Paskewitz & Riehle, 1994; Gorman et al., 1996; Rantala & Roff, 2007), it cannot be ruled out that other magpie moth immune parameters display neutral or negative responses to population density. Future studies should attempt to address both this issue, and the relevance of immune parameters to actual resistance against a range of parasites, although doing this under field conditions will present many difficulties.

Assuming that higher levels of successful parasitism (i.e. mortality from parasitoids) correlated with higher rates of parasitoid attack, the negative relationship between the level of parasitism within populations in the year prior to the immune assays (2013) and the strength of assayed encapsulation responses in 2014 could reflect a trans-generational immune cost following challenge and successful defence against parasitoid

oviposition. Trans-generational costs to offspring immunity in insects have been demonstrated in the laboratory multiple times and in many taxa. For example, Sadd and Schmid-Hempel (2009) found that *Bombus terrestris* (L.) worker bumblebees became more heavily infected by a protozoan parasite if their mothers had been challenged by heat-killed bacteria, and Moreau et al. (2012) found a negative trade-off between levels of maternal and offspring (egg) antibacterial activity for small mothers in the mealworm (*Tenebrio molitor*, L.). However, trans-generational costs in immune functioning following successful defence against attack by parasitoids in particular do not appear to have been investigated previously. In contrast to trans-generational costs in immune functioning following immunological challenge, trans-generational immune priming of offspring immune systems has also been observed in a number of insects following parental immunological challenge (Freitak et al., 2009; Tidbury et al., 2011). Therefore, it will be important for future research to determine the conditions under which parental immunological challenge either leads to a cost or a priming of offspring immunity, and as with DDP studies an important part of this will be using wild-immunology studies to determine how these processes and trade-offs work under natural conditions to understand their importance for real populations.

Finally, assuming higher levels of AbgrNPV mortality correlated with higher levels of AbgrNPV infections, the negative response of magpie moth pupal weight to increasing AbgrNPV mortality within the current year may reflect resource trade-offs between immune functioning and investment in development following successful larval defence against AbgrNPV infections. Although it is not known how resistant the magpie moth is to AbgrNPV, it is known that Lepidoptera frequently exhibit resistance to NPVs, particularly in later instars (Cory & Myers, 2003). It is also not clear how important the encapsulation response is for lepidopteran defence against NPVs, having only apparently been investigated in two species (Washburn et al., 1996; Washburn et al., 2000; Trudeau et al., 2001), therefore this conclusion must remain tentative. Within-generation costs of immunity have been well-demonstrated in invertebrates in the laboratory though, with investment being moved away from growth and other key life-history traits and into immune defence following parasite challenge (Zuk & Stoehr, 2002). For example, gypsy moth larvae surviving infection from the NPV LdNPV were smaller as pupae (Myers et al., 2000). Additionally, the terminal investment hypothesis predicts that individuals suffering parasite attacks likely to result in death should make a final attempt to increase fitness by reallocating

resources into reproductive output (Cluttonbrock, 1984), which results in inevitable trade-offs with investment in growth (Bascunan-Garcia et al., 2010; Gonzalez-Tokman et al., 2013). Demonstrations of these processes under field conditions appear to be extremely rare though.

Unexpectedly, this study represents one of the first documented DDP responses in an insect when using wild-collected individuals exposed to natural field conditions during their development. Consequently, the supportive evidence for DDP found here in the magpie moth, which does not display outbreaking dynamics or density-dependent phase polyphenism, appears in contrast to the lack of evidence for DDP found in the few similar studies of species that whilst not displaying density-dependent phase polyphenism do display classical outbreaking dynamics, and are therefore expected to exhibit DDP. Consequently, this highlights the need for future research to also consider species outside of those most expected to exhibit DDP, and for further wild-immunological studies of the relationship between population density and immunity. These processes are likely to influence population dynamics (White & Wilson, 1999; Reynolds et al., 2011), and it is therefore important to better understand the functioning and importance of DDP in a wide range of species, and under natural conditions.

4. Scale-dependent, contrasting effects of habitat fragmentation on host-natural enemy trophic interactions

4.1 Abstract

Habitat fragmentation can have contrasting effects on species and their interactions within communities, changing community structure and function. Parasitoids and pathogens are key natural enemies in invertebrate communities, but their responses to fragmentation have not been explored within the same community. This study aimed to explore the scale-dependent effects of habitat fragmentation on the population density of a Lepidopteran host and particularly its trophic interactions with a specialist parasitoid and virus. Host density and host larval-mortality from the parasitoid and the virus were measured in twelve isolated sites and thirteen connected sites. An index of habitat isolation was created based on the amount of suitable habitat surrounding sites at a range of spatial scales (0.1-5 km radii), and the direct and indirect effects of habitat isolation were analysed using generalised linear mixed effects models. Consistent with predictions, habitat isolation had direct negative effects on host density at the smallest and largest spatial scales, and indirect negative effects on host mortality from the virus at the largest scale, but in contrast to predictions it had direct positive effects on parasitism at small and medium scales. Higher trophic level species may still display responses to habitat fragmentation contrary to predictions based on well supported theory and empirical evidence. The mechanisms underlying these responses may be elucidated by studying responses, contrary to expectations, shared by related species. Developing general predictions about the responses of host-pathogen interactions to fragmentation will require greater understanding of the system-specific mechanisms by which fragmentation can influence pathogen transmission.

4.2 Introduction

Globally, habitat fragmentation has led to declines in biodiversity at all scales, largely because of changes in the amount, but also the connectivity, of suitable habitat (Fahrig, 2003; Ewers & Didham, 2006). Although the responses of individual species have been well studied (e.g. Didham et al., 1998), the effects of habitat fragmentation can also cascade through communities via trophic interactions between species, which can lead to complex changes in community structure and function (Laurance et al., 2002). Some broad patterns in the responses of trophic interactions to habitat fragmentation are now evident. In general, habitat fragmentation has typically negative effects on trophic interactions, which are generally more severe for trophic specialists and higher trophic levels than trophic generalists and lower trophic levels (Martinson & Fagan, 2014). However, these overall patterns often hide substantial variability. For example, although habitat fragmentation generally has negative effects on host-parasitoid interactions, reducing levels of parasitism (Martinson & Fagan, 2014), contrasting results are also often found (van Nouhuys, 2005). Additionally, for other widespread trophic relations, particularly host-pathogen interactions, typical responses to habitat fragmentation have yet to be found (McCallum, 2008; Martinson & Fagan, 2014). Studies examining the responses of multiple types of trophic interaction within the same community can help to reduce these gaps in knowledge.

In terrestrial ecosystems parasitoids and pathogens are key drivers of mortality in insect populations (Hawkins et al., 1997; Graham et al., 2004). They can have important regulatory influences on their host's population dynamics (Hassell, 2000; Bonsall, 2004), and are thought to play a key role in the cyclical outbreaking dynamics of many insect pests (Myers & Cory, 2013). However, the effects of habitat fragmentation on host-parasitoid and host-pathogen interactions within the same community have yet to be examined. Higher trophic levels (e.g. predators and parasitoids) are predicted to be more severely impacted by habitat fragmentation than lower trophic levels. This is because their populations tend to be smaller, more variable and subject to both direct effects of fragmentation, and also indirect effects due to their dependence on lower trophic levels, which may themselves be negatively affected by fragmentation (Holt et al., 1999; Valladares et al., 2006). However, although there is good support for this 'trophic-level hypothesis' (Kruess & Tscharntke, 1994; Martinson & Fagan, 2014), studies also reveal both positive and neutral responses to habitat

fragmentation by higher trophic levels (e.g. Doak, 2000; Brückmann et al., 2011; Schnitzler et al., 2011). The reasons for this variation are not entirely understood, but may be explained by the influence of other key traits, particularly trophic specialisation (Holt et al., 1999; van Nouhuys & Hanski, 2005; Cagnolo et al., 2009). Trophic specialists are predicted to be especially vulnerable to habitat fragmentation, because it can separate them from their prey (Davis et al., 1998), whilst generalists can utilise alternative resources, which may result in neutral and even positive responses to habitat fragmentation (Brückmann et al., 2011; Schnitzler et al., 2011).

Host-pathogen dynamics are usually assumed to be regulated by density-dependent processes (McCallum et al., 2001), where transmission rates increase with host density (Anderson & May, 1981). Spatially explicit aspects of disease transmission have also been well studied, providing important insights into the effects that spatial structure, particularly the size and connectivity of host populations, can have on the likelihood of invasion and persistence of pathogens in host populations (Park et al., 2001, 2002), and the evolution of pathogen virulence (Boots et al., 2004; Boots & Mealor, 2007). Consequently, the spatial distribution of hosts and pathogens, and the connectivity between host populations, can have significant effects on pathogen transmission and disease prevalence (Ostfeld et al., 2005). Therefore, by affecting the density and connectivity of host populations habitat fragmentation may indirectly influence pathogen transmission and resulting patterns of disease prevalence (Langlois et al., 2001; Allan et al., 2003; McCallum, 2008). However, so far there have been very few studies investigating these effects in natural systems, and general patterns of response are not yet clear (McCallum, 2008; Martinson & Fagan, 2014).

Differences in the composition and formation of host-parasitoid-pathogen communities can also drive qualitatively different host dynamics, leading to shifts in host-cycle periodicity and effects on the risk of population extinction (Begon et al., 1996; Sait et al., 2000). Parasitoids and pathogens also have strongly competitive interactions within hosts, usually resulting in the death of the parasitoid (Begon et al., 1999). Thus, if hosts and their natural enemies respond differently to habitat fragmentation this could lead to changes in host-enemy dynamics, due to altered interspecific interactions within the community. However, these possibilities have yet to be explored in the field.

Species' responses to habitat fragmentation are also dependent on the spatial scale considered (Roland & Taylor, 1997). This is because species

experience landscapes differently at different spatial scales, related to key traits that include their dispersal and foraging abilities, body size and trophic specialisation (Tscharntke & Brandl, 2004). For example, specialist natural enemies appear to respond to habitat structure at smaller spatial scales than generalist natural enemies (Chaplin-Kramer et al., 2011). Consequently, it is important to take a multi-scale approach when exploring the effects of habitat structure. Therefore, this study investigated the scale-dependent effects of habitat fragmentation on an insect host-parasitoid-pathogen community. This was achieved by examining the effects of habitat isolation, measured as the proportion of suitable habitat surrounding sampling sites at a range of spatial scales (Winfree et al., 2005), on population densities of a lepidopteran host and particularly its interactions with two key natural enemies (i.e. the mortality caused by those natural enemies).

The study was conducted on Mainland Orkney, and focused on the magpie moth, whose widespread larval populations suffer substantial larval mortality from the pathogen AbgrNPV and a mobile, *Aleiodes* sp. parasitoid with no known alternative hosts (C. van Achterberg & M.R. Shaw, unpub. data). Although NPVs are primarily horizontally transmitted when larvae consume foliage contaminated with infectious NPV virions (Cory & Myers, 2003), as a mechanism for NPVs to persist at low host densities NPV infections can also be vertically transmitted as non-lethal covert infections, which pass from adults to their offspring before potentially re-emerging as lethal, overt infections (Burden et al., 2002; Burden et al., 2003).

Within the community there are interactions between all the species. Therefore, the host and the parasitoid may be directly affected by habitat isolation, but also indirectly if the other species with which they interact are themselves affected by habitat isolation. There are no clear biological mechanisms for habitat isolation to directly affect the virus AbgrNPV, but indirect mechanisms from the effects habitat isolation can have on adult movement patterns, and/or host density, and/or the parasitoid (thereby altering within-host competition) are all plausible. Therefore, to try and gain a more mechanistic understanding of the effects of habitat isolation a comparative approach was taken involving creating models that either controlled for the effects of species interactions or did not. For each species this meant that models were created to assess the effects of habitat isolation without also controlling for interactions with the other species in the community (i.e. providing an overall measure of the sum of direct and indirect effects of habitat isolation). Additional models were then created for

each species to assess the effects of habitat isolation whilst also controlling for interactions with each of the other species in the community, both separately and then together (i.e. separating out any indirect effects of habitat isolation mediated by species interactions). It was then possible to assess whether variation explained by an overall effect of habitat isolation was actually better explained by the effects of interactions between species, indicating the importance of direct and indirect effects of habitat isolation.

Therefore, the following hypotheses were addressed. (1) Host population density will decrease with greater habitat isolation because of reduced focal habitat area available to support larval populations (Connor et al., 2000), and reduced immigration and inter-population dispersal (Hanski & Thomas, 1994). (2) Parasitism will decrease with greater habitat isolation because of reduced density-dependent parasitism in more isolated, lower density host-populations (Hassell, 2000), and because parasitoids will fail to reach more isolated host populations (Kruess & Tscharntke, 1994). (3) Host mortality from AbgrNPV virus infection will decrease with greater habitat isolation because of reduced density-dependent horizontal transmission in lower density larval populations (Anderson & May, 1981), and also because of reduced inter-population vertical transmission of the virus by adults in more isolated populations.

4.3 Methods

4.3.1 Study area and species

Field work was conducted between 28.05.2012 and 20.06.2012 on Mainland Orkney, the largest of the Orkney Islands (523 km^2). On Mainland Orkney larvae of the magpie moth are found feeding widely on heather, which covers approximately 19.8% of the island. This heather habitat is distributed between three large, separate heather moorlands (with areas of 4490, 1840 and 1570 ha), as well as a number of smaller but still extensive areas of heather (the largest being 519 ha), and over 450 small patches of heather (Fig. 4.1). Magpie moth larvae are polyphagous, but the remaining land area on Mainland Orkney consists almost entirely of pasture with some semi-

natural grasslands and almost no woodland, meaning that suitable larval habitat is overwhelmingly restricted to, and dominated by, heather habitat.

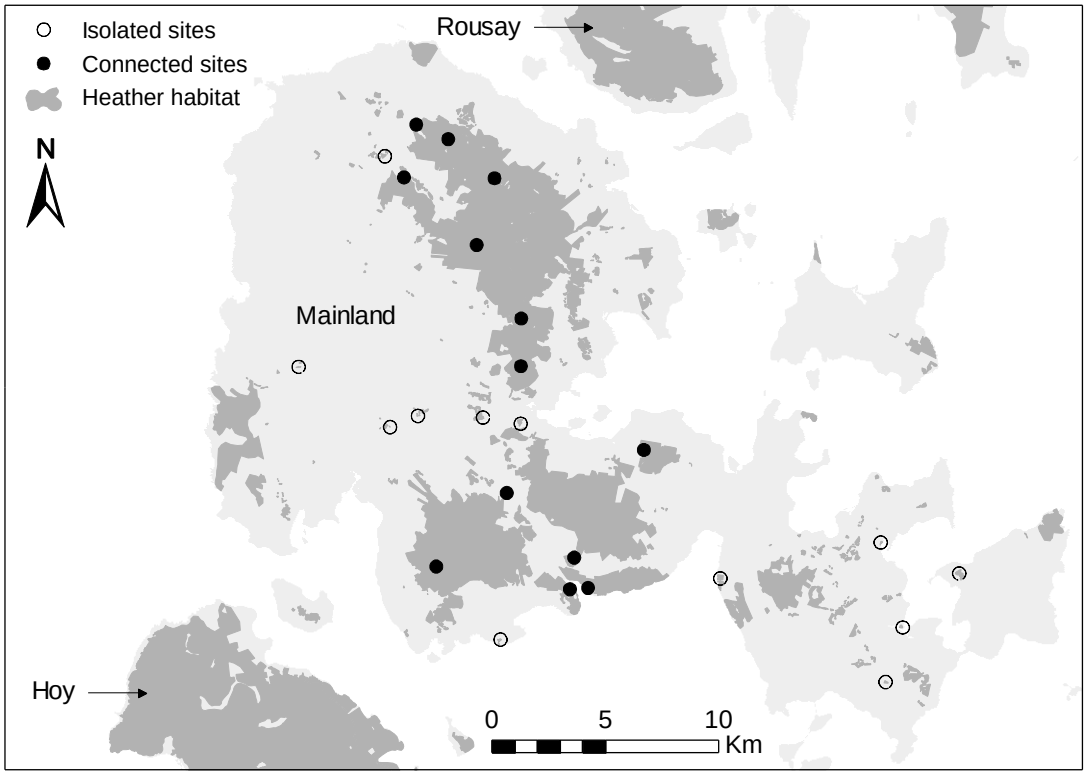

Figure 4.1 Location of the Orkney Islands, Mainland Orkney, the sampling sites on Mainland Orkney and the distribution of heather habitat across Mainland Orkney.

4.3.2 Site selection and sampling protocol

To investigate the effects of habitat fragmentation on the host-parasitoid-pathogen community larvae were sampled from a range of isolated and connected sites within heather habitats. Thirteen connected sites were located within large, continuous areas of heather moorland, ranging in size from 118 to 4486 ha (mean 2723 ± 2050 ha, 1 S.D.). Twelve isolated sites were located in much smaller, discrete patches of heather habitat ranging in size from 0.31 to 5.68 ha (mean 1.79 ± 1.89 ha, 1 S.D.; Fig. 4.1). Isolated sites were also separated from any large, continuous areas of heather habitat (of minimum size 98.86 ha) by between 703 and 6298 m (Fig. 4.1).

Sampling of larvae from heather plants occurred within ten 1 m^2-quadrats, placed at regular intervals along a 100m transect, which was randomly placed within each site. Within each transect any living and dead magpie moth larvae were collected via an exhaustive manual search of heather plants and the ground below the plants. *Aleiodes* parasitoids eventually turn their host into characteristic 'mummies' formed from the host's exoskeleton, which were also collected. Living larvae were then reared individually in 12 ml plastic pots in an outdoor insectary, and provided with non-sterile green heather shoots for food, which were taken from their sampling sites to minimise altering the risk of infection from the virus AbgrNPV. Larvae were checked every 1-3 days, with food replaced as needed until larval pupation, death or development of a parasitoid 'mummy'. All larvae found to have died were tested for infection by the virus AbgrNPV via Giemsa staining (Lacey, 2012), with all cadavers staining negative further tested for AbgrNPV DNA using PCR reactions (Harold, 2009).

4.3.3 Explanatory variables

An area-based buffer index of habitat isolation was used to quantify the degree of habitat isolation at each sampling site, across multiple spatial scales. All non-heather habitat was assumed unsuitable for larval development, and so the index was based on the percentage of heather habitat within nested concentric circles surrounding the centre of each sampling transect, with the following radii used to vary the spatial scale: 0.1, 0.25, 0.5, 1, 2.5 and 5 km. To make it an index of isolation, all percentage values were rescaled by first subtracting them from 100. Therefore, the

index ranged from 0 to 100, or from no isolation (0 = 100% heather habitat within the area considered) to complete isolation (100 = theoretically 0% heather habitat within the area considered). To calculate the habitat isolation index the distribution of heather habitat was mapped across Mainland Orkney using ArcMap 10.0 (ESRI, 2011) and data from the Land Cover Map 2007 for Britain (Morton et al., 2011). Heather habitat was determined based on the habitat classifications 'heather' and 'heather grassland' (Morton et al., 2011). This data was then edited based on recent (2006-2012) aerial imagery from Google Earth (Google, 2011), and verified in the field. Although this type of index is widely used as a measure of habitat isolation it does not take account of the spatial distribution of habitat, only the amount of habitat within a given area (Winfree et al., 2005). Therefore, it is not possible to separate effects related to habitat area from those related to the spatial distribution of habitat. However, this type of area-based buffer measure is a necessary and suitable index of habitat isolation when discrete habitat patches are not well defined (Winfree et al., 2005), which is the situation for the connected sites in this study (Fig. 4.1).

Site-level measures of host density and percentage host-mortality from the parasitoid (parasitism) and the virus AbgrNPV were created by pooling the quadrat-level sampling data within sites, for use as explanatory variables. These variables acted as proxy measures for interactions between species within the community, allowing their effects to be analysed and controlled for. Additionally, site elevation and heather plant height were thought likely to have important influences on all species. Lepidopteran larval density is known to be affected by heather plant height (Haysom & Coulson, 1998), whilst parasitoid foraging efficiency and NPV persistence are also affected by plant height and structure (Raymond & Hails, 2007; Obermaier et al., 2008). Increasing elevation affects both insect density and the level of parasitism due to the changes in abiotic conditions, particularly temperature, associated with changes in elevation (Hodkinson, 2005). Little research has been done on the relationships between elevation and diseases in insect communities, but temperature and UV radiation vary with elevation, and both have important impacts on interactions between hosts and NPVs (Morris, 1971). Plant height and site elevation are also likely to vary with habitat isolation, and so they were measured as explanatory variables so that their effects could be controlled for. Plant height was measured at the quadrat scale by dividing each quadrat into four equal sections, and taking the mean of the four heights of each plant within the centre of each section (measured along the stem from the base to the tip). Site elevation was

measured at the centre of each sampling transect using the *Spatial Analyst* extension in ArcMap 10.0 (ESRI, 2011) and Ordnance Survey Land-Form Profile Digital Terrain Model data (10 m^2 resolution) (Ordnance Survey, 2003).

4.3.4 Statistical analyses

The data were analysed to assess the effects of habitat isolation at different spatial scales on the species within the community. Direct effects of habitat isolation on the host and the parasitoid are likely, because they are independently mobile organisms. However, direct effects are not plausible for the virus AbgrNPV, although indirect effects mediated by the responses of the host and the parasitoid to habitat isolation are. Similarly, indirect effects of habitat isolation on the host and the parasitoid are also plausible, mediated by the responses to habitat isolation of the other species in the community with which they interact. Therefore, data were analysed using generalised linear mixed-effects models (GLMMs) in a MMI process (Burnham & Anderson, 2002). For each species this involved first creating a set of GLMMs to represent different hypotheses about the importance or otherwise of the influence of habitat isolation at each of the different spatial scales, whilst either controlling or not controlling for the effects of site elevation and heather plant height (Table A3.1; Table A3.5; Table A3.9). No effects from the other species within the community were controlled for, which meant that the effects of habitat isolation represented the sum of all direct or indirect influences of habitat isolation.

Therefore, to develop a more mechanistic understanding of the effects of habitat isolation the initial sets of models were constructed again for each species, but including variables to control for the effects of the other species in the community. For each species this was done by first controlling for the effects of each of the remaining species in the community separately and then together, resulting in four sets of models for each species (Table A3.1-A3.12). For each species it was then possible to examine whether variation significantly explained by an overall effect of habitat isolation (i.e. the sum of all direct and indirect effects of habitat isolation) was actually explained by the effects of one or both remaining species in the community. Thus, if an overall effect of habitat isolation was removed once the effects of one or both of the remaining species in the

community were controlled for then an indirect effect of habitat isolation was interpreted as having been mediated by the effects of either one or both of the remaining species in the community. If habitat isolation could still explain variation in species' responses once the effects of the other species in the community were controlled for, then for the host and the parasitoid this was interpreted as a direct effect of habitat isolation; whilst for the virus AbgrNPV this was interpreted as an indirect effect of habitat isolation mediated by changes in adult host-movement patterns, given that the effects of host density and parasitism were controlled for, and this was the remaining plausible mechanism (Langlois et al., 2001; McCallum, 2008).

Host density data were analysed with GLMMs using negative binomial errors and log-link functions, and mortality data for the parasitoid and the virus AbgrNPV were analysed with GLMMs using binomially distributed errors and logit-link functions. All response data was analysed at the scale of the quadrat, and to account for any non-independence due to spatial autocorrelation within sites all models contained site as a random factor (Zuur et al., 2009). Model fitting was done in version 3.0.2 of the statistical software R (R Core Development Team, 2014), with host density models fitted using the *glmmADMB* package (Skaug et al., 2012), and models for mortality due to the parasitoid and the virus AbgrNPV fitted using the *lme4* package (Bates et al., 2014).

Within each of the four model sets for each species models were ranked by their AICc scores, and Akaike weights used to create 95% confidence model sets (Burnham & Anderson, 2002). Inference was then based on model-averaged parameter estimates and their 95% confidence intervals, calculated using all models remaining in each 95% confidence model set. If only one model was retained in the 95% confidence model set, inference was based on parameter estimates and their 95% confidence intervals from this model. Model-averaged parameter estimates were calculated using the natural-average method, and their 95% confidence intervals were calculated based on unconditional standard errors (Burnham & Anderson, 2002).

Explanatory data were rescaled to have a mean of 0 and a standard deviation of 1, so that parameter estimates could be easily compared as unit free predictors on the same scale, and to reduce any multicollinearity (Zuur et al., 2009). Validation of models was based on the best AICc scoring model within each 95% confidence model set. Adequacy of model fit and adherence to relevant statistical assumptions was confirmed using a range

of residual plots following Zuur et al. (2009). Multicollinearity in predictor variables was assessed using variance inflation factors (VIFs), but all VIF scores were <3.5, indicating no issues (O'Brien, 2007). Spline correlograms confirmed there were no issues with between-site spatial autocorrelation in model residuals (Bjornstad & Falck, 2001).

4.4 Results

A total of 927 magpie moth larvae were collected, with individuals found in all sites and 74% of quadrats, highlighting the widespread distribution of magpie moth larvae on heather habitat across Mainland Orkney at both large and small scales. Overall, 38.5% of larvae eclosed as adults, whilst 43.8% died from infection by the virus AbgrNPV, 11% died from parasitism and the remaining 6.7% died from unknown causes (total mortality of 61.5%). These unknown causes were not investigated further, but there was no parasitism found from species other than the *Aleiodes* sp.

Table 4.1 Isolated and connected sites' summary statistics for host density, larval mortality from the virus AbgrNPV, the parasitoid and unknown causes, larval survival, and plant height and site elevation.

	Isolated sites (n = 12)			Connected sites (n = 13)		
Variable	Mean (±1 S.D.)	Range	C.V.	Mean (±1 S.D.)	Range	C.V.
Density (larvae m^{-2})	26.7 ± 18.7	7-54	0.7	45.2 ± 41.3	4-146	0.91
AbgrNPV virus (%)	39.7 ± 22	0-75	0.55	47.1 ± 23	0-82	0.49
Parasitism (%)	7 ± 8.7	0-24	1.24	14.1 ± 15.5	0-56	1.09
Unknown (%)	7.7 ± 9.9	0-30	1.29	6 ± 5.4	0-16	0.9
Survival (%)	45.7 ± 18.3	17-71	0.4	32.8 ± 14.7	15-60	0.45
Plant height	34.8 ± 11.4	5-63	3.05	41.6 ± 11.6	16-76	3.58
Site elevation	22.8 ± 21.1	2-68	1.08	66.8 ± 31.3	16-119	2.13

Statistics are based on quadrat-level data pooled within sites, except for site elevation, which is measured at a site level. Unknown (%) represents the percentage of larval mortality not attributable to the virus AbgrNPV or the parasitoid, and survival (%) represents the percentage of larvae surviving to the pupal stage. C.V. = coefficient of variation.

Site-level host density varied substantially between all sites, but was generally greater in connected sites (Table 4.1). The analysis showed moderately strong, negative relationships between the overall effects of habitat isolation (i.e. the sum of any direct and indirect effects) at the smallest and largest (100 m and 5000 m) spatial scales and host density (Table 4.2). However, when controlling for the effects of mortality from both the parasitoid and the virus AbgrNPV only a single best model was retained in the 95% confidence set of models, which contained an effect of habitat isolation at 100 m, along with effects from site elevation and plant height (Table A3.4 & Table A3.16). This model predicted a decrease in host density from 6.6 to 1.49 (larvae m^{-2}) as habitat isolation at the 100 m scale increased from 0-89.9% (Fig. 4.2). When controlling for host interactions with both natural enemies the effect of habitat isolation at 100 m was largely unchanged from the overall effect of habitat isolation at 100 m (Table 4.2), suggesting a direct mechanism of action.

The effect of habitat isolation at 5000 m when mortality from either the parasitoid or the virus AbgrNPV were controlled for was also very little changed compared to the effect of habitat isolation at 5000 m when mortality from both natural enemies was not controlled for (Table 4.2). Therefore, although no model was retained in the 95% confidence set when mortality from both the natural enemies was controlled for at the same time (Table A3.4), again the results indicated that the important effects of habitat isolation (this time at the 5000 m scale) were best explained as resulting from direct mechanisms. When controlling for interactions with both natural enemies there was also evidence for a moderately strong, negative effect of site elevation, but no effect of plant height, on host density (Table A3.16).

Table 4.2 Parameter estimates and 95% confidence intervals for the effects of habitat isolation at different spatial scales on host density, with or without also controlling for host mortality from the virus AbgrNPV and the parasitoid separately or together.

Species effects controlled for	None	AbgrNPV virus*	Parasitoid†	AbgrNPV virus + parasitoid
Spatial scale of habitat isolation (m)				
100	0.58 (0.46, 0.72)	0.61 (0.49, 0.77)	0.56 (0.44, 0.71)	0.57‡ (0.45, 0.72)
2500		0.67 (0.53, 0.85)		
5000	0.64 (0.52, 0.77)	0.67 (0.55, 0.82)	0.63 (0.52, 0.77)	

* = Site-level percentage host mortality from the virus AbgrNPV. † = Site-level percentage host mortality from the parasitoid. Parameter estimates and their 95% confidence intervals are based on back-transformed model-averaged coefficients and their standard errors from the MMI analysis. ‡However, the estimated effect of habitat isolation at 100 m when both species were controlled for comes from the single model retained in the relevant 95% confidence set of models. Explanatory data were standardised, and estimates represent the multiplicative change in host density (larvae m^{-2}) given a 1 S.D. increase in habitat isolation at the given scale. Therefore, values >1 indicate a positive effect on host density, values <1 indicate a negative effect. The MMI process for all results also controlled for the effects of site elevation and plant height, and missing values indicate no models containing an effect of habitat isolation at that scale were retained in the relevant 95% confidence set of models (see appendix 2).

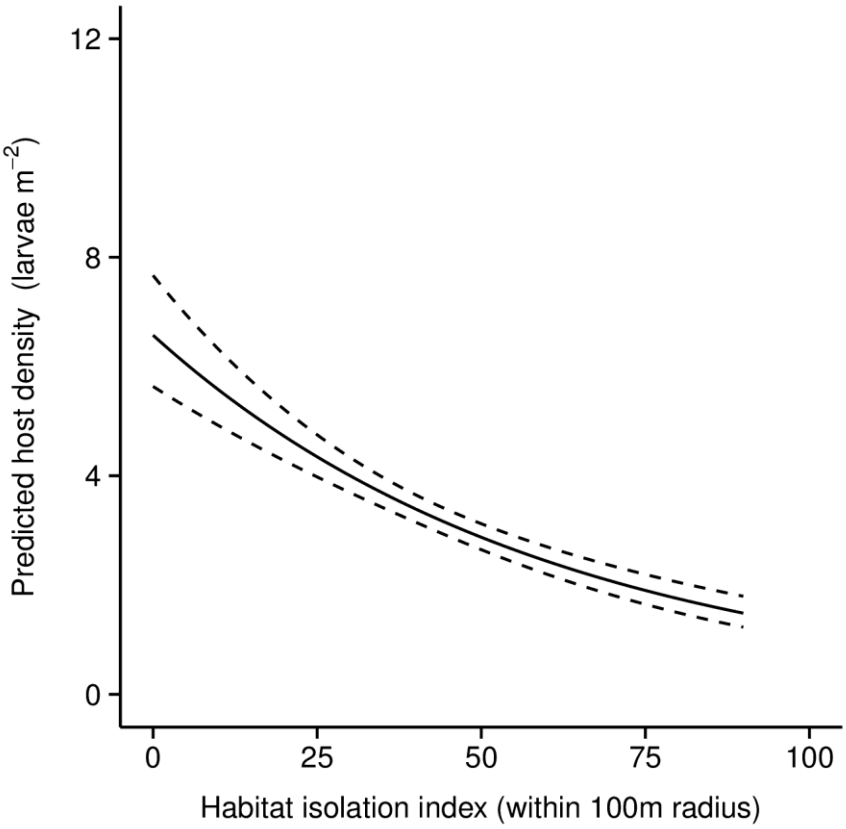

Figure 4.2 Negative relationship between habitat isolation at the 100 m scale and host density (larvae m^{-2}), whilst site-level percentage mortality from the virus AbgrNPV and the parasitoid are controlled for (as well as site elevation and plant height). The habitat isolation index represents the percentage of non-heather habitat surrounding a sampling site within a radius of 100 m, with 0 representing complete connectivity (100% heather habitat with a 100 m radius of a site) and 100 representing (theoretically) complete isolation (0% heather habitat within a 100 m radius of a site). Host density values (± S.E.) are back-transformed model-predictions based on the fixed effects from the single negative binomial GLMM retained in the 95% confidence set of models that controlled for both natural enemies (as well as site elevation and plant height), with all predictor variables other than habitat isolation at 100 m held at their mean values, whilst habitat isolation was varied across the range of measured values.

At a site level, in both isolated and connected sites there was far greater average and maximum levels of mortality from the virus AbgrNPV than the parasitoid (Table 4.1). However, there was also less relative variation in site-level percentage mortality due to the virus AbgrNPV than the parasitoid (Table 4.1). Site-level percentage mortality due to the virus AbgrNPV was also slightly higher in connected sites than in isolated sites (Table 4.1), but the analysis did not provide strong evidence for overall effects of habitat isolation at any scale on the likelihood of mortality from the virus AbgrNPV (Table 4.3). When controlling for site-level percentage mortality from the parasitoid (i.e. within-host competition), with or without controlling for host density, there was evidence for a weak, negative effect of habitat isolation at 5000 m on the likelihood of mortality from the virus AbgrNPV (Table 4.3). Consequently, the model-averaged predicted probability of mortality declined from 0.51 to 0.39 as habitat isolation at 5000 m increased from 48.2%-98.3% at the 5000 m scale when controlling for parasitism and host density (Fig. 4.3).

The effect of habitat isolation at 5000 m, when controlling for species interactions, was little different to the overall effect of habitat isolation at 5000 m, albeit with narrower confidence intervals (Table 4.3). Therefore, given that any effects from changes in host density or competition from the parasitoid, in response to habitat isolation, were controlled for, the effect may be interpreted as being driven by changes to host-movement patterns at large spatial scales. Although weak and highly variable, similar trends in the effects of habitat isolation at smaller spatial scales were also observed, but declined in importance with decreasing scale. When controlling for all species interactions site-level percentage parasitism had a moderately strong, negative effect on the likelihood of mortality from the virus AbgrNPV, whilst host density, heather height and site elevation all had no clear effects (Table A3.20).

Table 4.3 Parameter estimates and 95% confidence intervals for the effects of habitat isolation at different spatial scales on host mortality from the virus AbgrNPV, with or without also controlling for host mortality from the parasitoid and host density separately or together.

Species effects controlled for	None	Parasitoid*	Host density†	Parasitoid + host density
Spatial scale of habitat isolation (m)				
100	0.79 (0.48, 1.31)	0.88 (0.66, 1.17)	0.83 (0.49, 1.4)	0.9 (0.67, 1.21)
250	0.69 (0.4, 1.22)	0.84 (0.64, 1.1)	0.73 (0.4, 1.33)	0.86 (0.65, 1.14)
500	0.79 (0.43, 1.45)	0.84 (0.63, 1.1)	0.99 (0.68, 1.43)	0.85 (0.64, 1.13)
1000	0.70 (0.36, 1.36)	0.8 (0.58, 1.1)	0.74 (0.37, 1.48)	0.82 (0.6, 1.13)
2500	0.67 (0.35, 1.28)	0.75 (0.54, 1.06)	0.71 (0.36, 1.39)	0.77 (0.55, 1.08)
5000	0.74 (0.45, 1.19)	0.73 (0.56, 0.95)	0.76 (0.45, 1.29)	0.74 (0.55, 0.98)

* = Site-level percentage host mortality from the parasitoid. † = Site-level host density. Parameter estimates and their 95% confidence intervals are based on back-transformed model-averaged coefficients and their standard errors from the MMI analysis. Explanatory data were standardised, and estimates represent the multiplicative change in the odds of host mortality from the virus AbgrNPV given a 1 S.D. increase in habitat isolation at the given scale. Therefore, values >1 indicate a positive effect on host density, values <1 indicate a negative effect. The MMI process for all results also controlled for the effects of site elevation and plant height (see appendix 2).

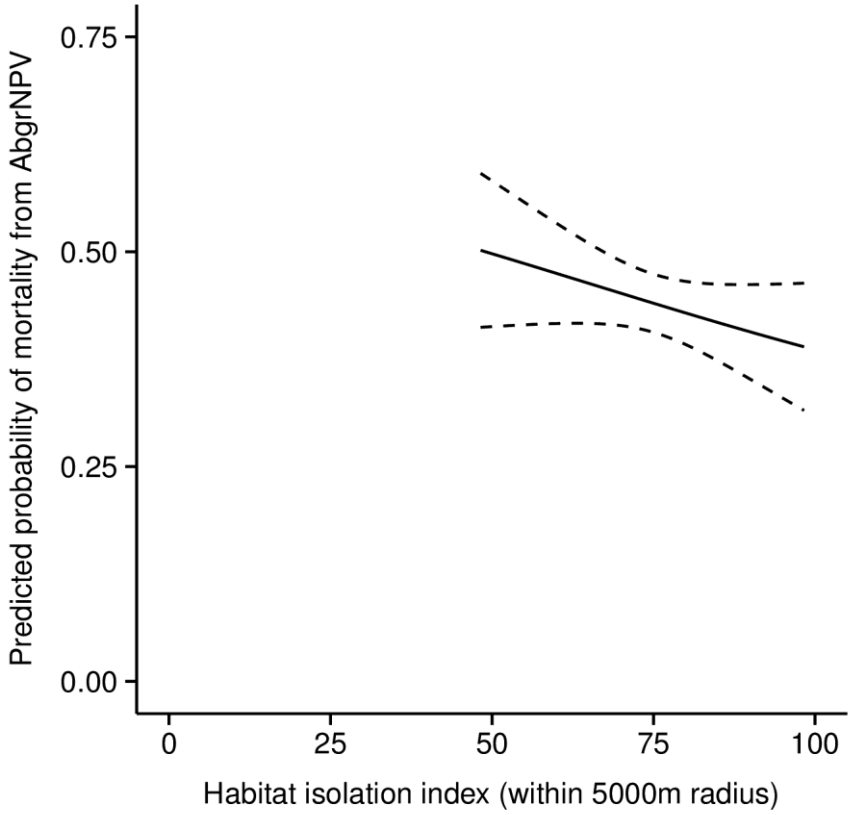

Figure 4.3 Negative relationship between habitat isolation at the 5000 m scale and the probability of host mortality from the virus AbgrNPV, whilst site-level percentage host mortality from the parasitoid and site-level host density are controlled for (as well as site elevation and plant height). The habitat isolation index represents the percentage of non-heather habitat surrounding a sampling site within a radius of 5000 m, with 0 representing complete connectivity (100% heather habitat with a 5000 m radius of a site) and 100 representing (theoretically) complete isolation (0% heather habitat within a 5000 m radius of a site). Values for the probability of host mortality from the virus AbgrNPV (± S.E.) are back-transformed predictions based on the model-averaged fixed effects of the binomial GLMMs retained in the 95% confidence set of models that controlled for parasitism and host density (as well as site elevation and plant height), with all predictor variables held at their mean values, whilst habitat isolation at 5000 m was varied across the range of measured values.

Parasitism varied substantially between all sites, and there were generally higher levels of parasitism in connected sites (Table 4.1). However, contrary to expectations there were strong, positive, overall effects of habitat isolation on the likelihood of parasitism at the 100 and 250 m scales (Table 4.4) when the effects of site elevation and plant height were accounted for (e.g. isolated sites were typically at substantially lower elevations than connected sites, Table 4.1). When controlling for the effects of host density, or host density and site-level percentage mortality from the virus AbgrNPV (i.e. within-host competition), effects of habitat isolation at the 100 and 250 m scales remained, and there was also a smaller positive effect of habitat isolation at the 500 m scale (Table 4.4). These effects were little different in size from the overall effects of habitat isolation at the same spatial scales when not controlling for species interactions (Table 4.4), indicating direct mechanisms. The strongest direct effect was at the 100 m scale, where the probability of parasitism was predicted to increase from 0.03 to 0.15 as habitat isolation increased from 0-89.9% at the 100 m scale (Table 4.4 & Fig. 4.4). When controlling for all species interactions there was a moderate, positive effect of host density on the likelihood of parasitism, and a stronger, negative effect of site-level percentage mortality from the virus AbgrNPV; whilst site elevation had a strong, positive effect, and plant height a moderately strong, positive effect on the likelihood of parasitism (Table A3.24).

Table 4.4 Parameter estimates and 95% confidence intervals for the effects of habitat isolation at different spatial scales on host mortality from the parasitoid, with or without also controlling for host mortality from the virus AbgrNPV and host density separately or together.

Species effects controlled for	None	AbgrNPV virus*	Host density†	AbgrNPV virus + host density
Spatial scale of habitat isolation (m)				
100	3.55 (1.37, 9.19)	2.64 (1.15, 6.03)	4.48 (1.65, 12.17)	3.39 (1.56, 7.34)
250	3.48 (1.36, 8.9)	2.51 (1.09, 5.75)	4.18 (1.65, 10.62)	3.01 (0.55, 5.85)
500	2.67 (0.91, 7.82)	1.99 (0.81, 4.84)	3.09 (1.03, 9.23)	2.39 (1.07, 5.36)
1000	2.42 (0.79, 7.45)	1.6 (0.61, 4.2)	2.92 (0.9, 9.45)	
2500				
5000		0.31 (0.31, 1.31)		

* = Site-level percentage host mortality from the virus AbgrNPV. † = Site-level host density. Parameter estimates and their 95% confidence intervals are based on back-transformed model-averaged coefficients and their standard errors from the MMI analysis. Explanatory data were standardised, and estimates represent the multiplicative change in the odds of host mortality from the parasitoid given a 1 S.D. increase in habitat isolation at the given scale. Therefore, values >1 indicate a positive effect on host density, values <1 indicate a negative effect. The MMI process for all results also controlled for the effects of site elevation and plant height, and missing values indicate no models containing an effect of habitat isolation at that scale were retained in the relevant 95% confidence set of models (see appendix 2).

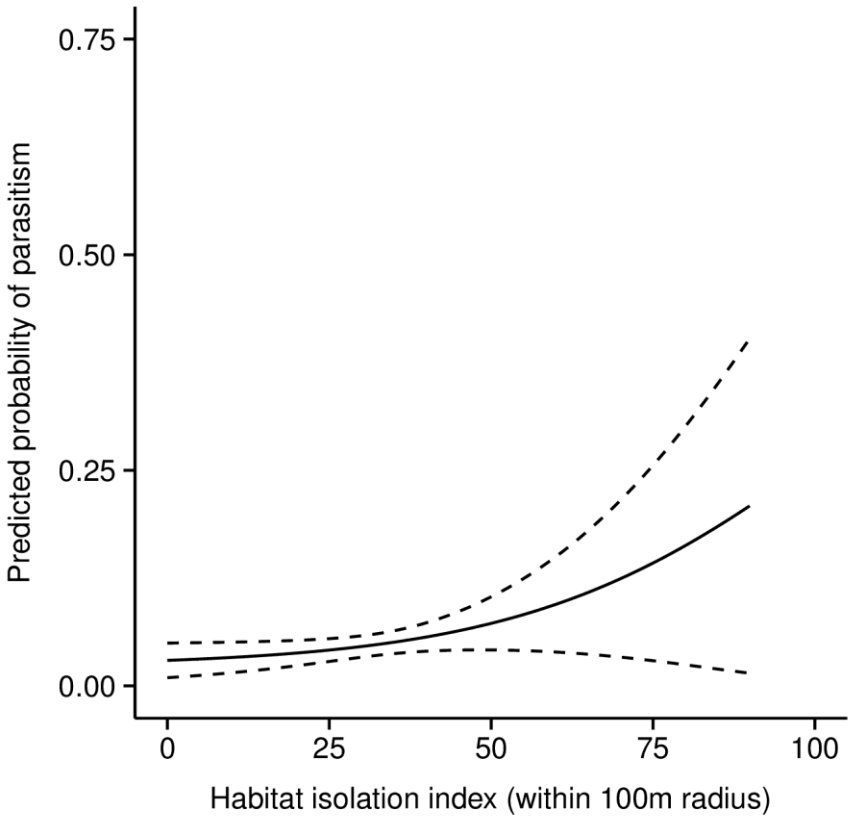

Figure 4.4 Positive relationship between habitat isolation at the 100 m scale and the probability of host mortality from the parasitoid, whilst site-level percentage host mortality from the virus AbgrNPV and site-level host density were controlled for (as well as site elevation and plant height). The habitat isolation index represents the percentage of non-heather habitat surrounding a sampling site within a radius of 100 m, with 0 representing complete connectivity (100% heather habitat with a 100 m radius of a site) and 100 representing (theoretically) complete isolation (0% heather habitat within a 100 m radius of a site). Values for the probability of host mortality from the parasitoid (± S.E.) are back-transformed predictions based on the model-averaged fixed effects of the binomial GLMMs retained in the 95% confidence set of models that controlled for host mortality from the virus AbgrNPV and host density (as well as site elevation and plant height), with all predictor variables held at their mean values, whilst habitat isolation at 100 m was varied across the range of measured values.

4.5 Discussion

There was evidence for a direct negative effect of habitat isolation at the smallest scale (100 m) on host density, and also an overall negative effect of habitat isolation at the largest scale (5000 m) on host density, also probably driven by direct mechanisms. There was also evidence for an indirect negative effect of habitat isolation at the largest scale on the likelihood of mortality due to the virus AbgrNPV. Lastly, in contrast to expectations based on the trophic-level hypothesis and the trophic specialism of the parasitoid (Kruess & Tscharntke, 1994; Holt et al., 1999; Kruess & Tscharntke, 2000), there was evidence for direct positive effects of habitat isolation at small and medium scales (100-500 m) on the likelihood of parasitism, once the effects of site elevation and plant height were also controlled for.

The direct effects of habitat isolation on host density may be due to processes related to habitat area and/or the spatial arrangement of habitat, but it is not possible to distinguish between these possibilities based on the habitat isolation index, and so both must be considered. In insects relationships between population density and habitat area are variable (Bender et al., 1998; Connor et al., 2000; Hambäck & Englund, 2005). Positive relationships have usually been explained by the resource concentration hypothesis (Connor et al., 2000), and the results observed here may indeed be due to larger areas of heather habitat supporting denser larval populations. Alternatively, more recent work has attempted to explain population density-area relationships in terms of the scaling of migration rates with habitat area (Hambäck & Englund, 2005; Hambäck et al., 2007). Applied to moths, as observed here positive relationships between population density and habitat area are typically found, and may be explained by their reliance on primarily olfactory cues to locate suitable habitat for oviposition (Renwick & Chew, 1994), which scale strongly with habitat area (Hambäck & Englund, 2005; Hambäck et al., 2007). Consistent with this hypothesis, butterflies instead locate suitable habitat for oviposition primarily using visual cues (Renwick & Chew, 1994), which scale much less with habitat area (Hambäck & Englund, 2005; Hambäck et al., 2007), and they typically display negative relationships between population density and habitat area.

In terms of the spatial arrangement of habitat, metapopulation theory predicts that population densities should decline with increasing habitat isolation because of reduced immigration (Hanski & Thomas, 1994), and this

has been demonstrated by experimental and observational studies (Hanski & Thomas, 1994; Gonzalez et al., 1998). This represents an alternative mechanism to area-related processes, and could also explain the observed results if more isolated sites receive fewer immigrants due to their isolation from other large areas of heather habitat. The dispersal ability of the magpie moth is not known, and indirect evidence is contradictory. As a relatively large bodied species it should be expected to be a relatively strong disperser (Sekar, 2012), with some anecdotal evidence of large-scale migrations seen a few miles out to sea apparently consistent with this possibility (R. Leverton, pers. comm.). However, as detailed in chapter 2, local populations of magpie moth across Orkney, separated by between approximately 250 m and 40 km, display complete asynchrony in their dynamics, suggesting little inter-population dispersal across such distances. Therefore, although it is not possible to distinguish area-related effects from those due to the spatial arrangement of habitat based on the isolation index, the effect of habitat isolation at the largest spatial scale is consistent with immigration being reduced for sites isolated at large spatial scales, due to the apparent dispersal limitations of the magpie moth. Similarly, the direct negative effect of habitat isolation at the smallest spatial scale is also more consistent with small-scale area-related mechanisms (Fahrig, 2003), reflecting greater larval resources and/or greater immigration with increasing habitat area (Hambäck & Englund, 2005; Hambäck et al., 2007).

Within the Mainland Orkney landscape the magpie moth may be seen as a habitat specialist due to the essentially binary suitability of the landscape for its larval stages. Consistent with results found here, habitat specialists are predicted to display more positive density-area relationships than habitat generalists. This is because of their greater risk of population extinction in smaller areas of habitat, given that they cannot utilise alternative habitats (Steffan-Dewenter & Tscharntke, 2000; Hambäck et al., 2007). However, an interaction between habitat specialisation and body size has also been observed, with large bodied (and therefore generally highly mobile) habitat specialists displaying either neutral or negative relationships between population density and habitat area, compared to the more positive relationships displayed by small bodied species (Hambäck et al., 2007). Therefore, the results presented here suggest a more important role for trophic specialisation over dispersal ability in determining species' sensitivity to habitat fragmentation, at least in this species.

Changes in host density and host movement patterns in response to habitat structure are both plausible mechanisms by which habitat fragmentation may indirectly affect patterns in disease (Allan et al., 2003; Brownstein et al., 2005). The effect of habitat isolation at the 5000 m scale on the likelihood of mortality from the virus AbgrNPV was essentially the same whether the effects of host density were controlled for or not, although the effect was only clear when at least controlling for competition with the parasitoid. When controlling for the other species in the community, there was substantial uncertainty associated with the estimated effects of habitat isolation on the likelihood of mortality from the virus AbgrNPV at spatial scales smaller than 5000 m, which declined with decreasing spatial scale. Therefore, the clear negative effects of habitat isolation at 5000 m may be tentatively interpreted as reflecting reduced adult vertical transmission of covert AbgrNPV infections between populations, driven by reduced adult dispersal between populations isolated at large spatial scales (Tscharntke & Brandl, 2004; Sekar, 2012), ultimately leading to reduced larval mortality from emergent overt infections in more isolated populations (Burden et al., 2002; Boots et al., 2003; Burden et al., 2003). Covert vertical transmission of NPV infections appear to be relatively common in Lepidoptera (Burden et al., 2002; Burden et al., 2003; Vilaplana et al., 2010), but their dynamics have been little studied in natural settings, and their importance in this system remains to be determined. However, AbgrNPV is a key mortality agent for magpie moth populations, and may be a principal regulator of local population dynamics (see chapter 2). Therefore, any influence of habitat structure on host-AbgrNPV interactions could influence magpie moth local dynamics, highlighting how habitat structure may have complex effects on important host-pathogen interactions.

Alternatively, studies also suggest that pathogens evolve to become more infective and virulent when inter-population connectivity increases, and transmission is less spatially redistricted, thereby increasing disease prevalence (Boots & Sasaki, 1999, 2000). In systems such as insect-baculovirus communities adult reproduction occurs at much larger spatial scales than horizontal pathogen transmission between larvae, and Boots and Sasaki (2000) have shown that in these contexts self-shading of infected individuals (where an infected individual is surrounded by infected, non-susceptible individuals) is low, which can drive the evolution of highly infective and virulent pathogens. Consequently, if adult dispersal becomes more spatially restricted in more fragmented, isolated habitats, greater self-shading could lead to lower levels of infectivity and virulence, reducing

disease prevalence as seen here. This mechanism has been demonstrated in an insect-baculovirus system, but only in the lab at very small scales (Boots & Mealor, 2007). Similarly, as host reproductive dispersal and pathogen transmission both become increasingly spatially restricted, as will occur in more isolated habitats, co-evolutionary processes can drive increasing host resistance to pathogens (Best et al., 2011). Therefore, covert transmission and evolutionary processes could explain the observed relationship between AbgrNPV mortality and habitat connectivity at the largest spatial scales, and determining their importance in real systems requires will require observational and experimental field studies. More generally though, these results are consistent with habitat fragmentation having important effects on host-pathogen interactions by affecting the connectivity of host populations, as seen in the few field studies to also address this issue (Langlois et al., 2001; Allan et al., 2003; Brownstein et al., 2005).

Unexpectedly, there were also strong positive effects of habitat isolation on the likelihood of parasitism at small and medium spatial scales (100-500 m), possibly reflecting the generally more restricted dispersal abilities of higher trophic levels (Tscharntke & Brandl, 2004; Chaplin-Kramer et al., 2011). These effects appear to be driven primarily by direct mechanisms, rather than being mediated by the responses of the host and/or the virus AbgrNPV to the effects of habitat isolation. Again, the results must be interpreted in the context of the habitat isolation index, which does not allow direct effects related to habitat area to be separated from direct effects related to the spatial arrangement of habitat. Parasitoids are known to increase their oviposition rate and spend longer searching for hosts relative to the distance travelled to locate suitable foraging sites (Tentelier et al., 2006), which can lead to increased rates of parasitism in more isolated areas of habitat (Cronin & Strong, 1999). Parasitoids may also be less willing to disperse from within suitable habitat patches, and more likely to return to them after moving out into unsuitable matrix habitat, leading to increased aggregation of parasitism in more isolated patches (Cronin & Strong, 1999). Insects also commonly exhibit negative responses to the edges of suitable habitat patches (Ewers & Didham, 2006), which may lead to parasitoids becoming 'trapped' in smaller, discrete patches of habitat, again resulting in increased rates of parasitism (Roth et al., 2006). Therefore, the positive relationship between parasitism and habitat isolation observed at small to medium spatial scales is consistent with these behavioural mechanisms, given that the more isolated sites were generally

small, discrete patches of heather habitat, and usually separated from any other areas of heather habitat by substantial distances.

The response of parasitism to habitat isolation therefore revealed that the parasitoid was robust to habitat fragmentation. This was contrary to the trophic-level hypothesis (Kruess & Tscharntke, 1994, 2000), the typically negative responses of parasitoids to increasing habitat fragmentation (Martinson & Fagan, 2014), and the trophic specialisation of the parasitoid, which should make it especially susceptible to habitat fragmentation (Holt et al., 1999). This emphasises that the responses of higher trophic levels can still show striking deviations from predictions based on existing theory (Doak, 2000; Elzinga et al., 2007; Brückmann et al., 2011), even when they are trophic specialists and expected to be especially vulnerable to habitat fragmentation (Schnitzler et al., 2011).

Interestingly, Schnitzler et al. (2011) have also documented a positive response to greater habitat isolation by another specialist *Aleiodes* parasitoid, whilst Doak (2000) demonstrated a positive response to greater habitat isolation and reduced patch size in a further *Aleiodes* sp. parasitoid, although of unknown trophic breadth. Therefore, it is an intriguing but unexplored possibility that common responses to habitat fragmentation, including those contrary to expectations, could be shared by related species at higher trophic levels, driven by shared behavioural responses to habitat structure. At present there is no evidence that the *Aleiodes* sp. parasitoid exerts any regulatory control over magpie moth population dynamics (see chapter 2). However, across Orkney the *Aleiodes* parasitoid is clearly undergoing a rapid expansion in abundance (chapter 2), and as a specialist parasitoid may soon begin to exert population regulation if it becomes sufficiently abundant (Klemola et al., 2014). Certainly, with continued expansion of its population across Orkney there are likely to be complex interactions between the parasitoid and AbgrNPV, with which it will be competing for hosts and possibly transmitting as a vector (Cossentine, 2009), which will likely be mediated by the contrasting influence of habitat structure on the two natural enemies.

The responses seen across all species within the community were likely to result primarily from the effects of habitat structure on host and natural enemy dispersal and foraging behaviours. However, whilst the observed effects were largely as expected for the host they were in opposition to those predicted for the parasitoid, based on existing theory and typical responses (Holt et al., 1999; Martinson & Fagan, 2014). Thus,

despite some clear patterns being evident in the general responses of host-parasitoid interactions to habitat fragmentation (Martinson & Fagan, 2014), a better understanding of the mechanisms driving deviations from predicted responses is required. This may be advanced by looking for shared responses to habitat fragmentation, particularly those contrary to predictions, in taxonomically related suites of higher trophic level species, and then investigating their causes.

Although the response by the virus AbgrNPV to habitat isolation was not unexpected, the responses of host-pathogen interactions to habitat fragmentation have received little attention compared to other trophic interactions (McCallum, 2008; Martinson & Fagan, 2014), despite the increasing recognition of the importance of pathogens for host population dynamics (Bonsall 2004; Myers and Cory 2013). Clearly there is a need to improve understanding of the system-specific mechanisms by which habitat fragmentation can influence host-pathogen interactions in different systems. Improving understanding about these processes will hopefully enable both more general and specific predictions to made.

5. Spatial synchrony and travelling waves in winter moth dynamics

5.1 Abstract

Cyclical species typically display large-scale patterns in their spatio-temporal dynamics, including spatial synchrony and periodic travelling waves. Spatially synchronous population dynamics may be caused by spatially correlated environmental factors (Moran effects), or dispersal processes. It is difficult to disentangle these mechanisms, but comparing the relative extent of spatially correlated environmental fluctuations and spatial population synchrony, along with species' dispersal abilities, can help to indicate plausible causes. Winter moth populations are known to display large-scale spatial synchrony, but the causes have not been explicitly investigated previously. Travelling waves in winter moth abundance also pass across Europe every 9-10 years. However, the long-term dynamics of the winter moth have yet to be studied in Britain at large-scales, and it is not clear whether British winter moth populations form part of the European travelling waves, or whether there are separate winter moth travelling waves within Britain. Therefore, the large-scale spatio-temporal dynamics of the winter moth were analysed within Britain using a 45-year dataset of light trap counts, and spatial correlation in environmental factors compared across the same time frame. Moderate to weak winter moth population synchrony persisted across Britain at large scales, whilst environmental factors were more strongly correlated and across larger spatial scales. Given the relatively poor dispersal abilities of the winter moth, this indicates a Moran effect to be the most likely cause of the population synchrony. British winter moth populations displayed long-term cycles like those in Europe, but analyses indicated no link between winter moth travelling waves in mainland Europe and Britain, suggesting the English Channel and North Sea act as a barrier to their passage. In addition, there were no evidence for any winter moth travelling waves within Britain. Causes of travelling waves are still unclear, but the results are discussed in the context of favoured hypotheses.

5.2 Introduction

As well as displaying strikingly regular local population cycles, cyclical species typically also show spatial synchrony in their dynamics, often over extensive spatial scales. For example, in North America the population dynamics of the cyclically outbreaking gypsy moth display synchrony for over 900 km, and the cyclical snowshoe hare displays population synchrony for over 1000 km (Krebs et al., 2013). Spatial synchrony in cyclical species' dynamics is thought to occur via the entrainment of spatially separate local populations, such that they cycle in phase ('phase-locking') with one another (Royama et al., 2005), and is thought to be generated by two main mechanisms. Firstly, via the influence of spatially autocorrelated fluctuations in climatic factors, known as 'Moran effects' (Moran, 1953), on local populations across large areas. Moran effects are often thought responsible for very large-scale spatial population synchrony (Bjornstad et al., 1999). Secondly, via synchronising inter-population dispersal involving either individuals of the focal species (Peltonen et al., 2002; Tobin & Bjornstad, 2003), or key natural enemies that generate host-cycles through density-dependent regulation (Bjornstad & Bascompte, 2001; Tobin & Bjornstad, 2003). The effects of both mechanisms may also interact to impact population synchrony in complicated ways (Kendall et al., 2000). Untangling their relative effects in field systems is very difficult, unless effective barriers to dispersal are present at scales much smaller than the level of synchrony in climatic factors (Grenfell et al., 1998; Post & Forchhammer, 2002). However, it is also possible to use 'statistical barriers' to help choose between likely casual factors, by comparing the spatial extent of synchrony exhibited by a potential causal mechanism relative to the spatial extent of species' population synchrony (Gouhier & Guichard, 2014). For example, Gouhier et al. (2010) showed that spatial synchrony in environmental forcing extended for less than half the distance at which spatial population synchrony persisted in the mussel *Mytilus californianus* (Conrad), effectively ruling out the influence of environmental forcing on its large-scale population synchrony, which was better explained by a metapopulation model as resulting from linked local dispersal processes.

With the availability of some large-scale (sometimes continental-scale) population datasets spanning many years (sometimes many decades), it has also become clear that the populations of a number of cyclical species', from a range of taxa, display periodic travelling wave behaviour

(e.g. Lambin et al., 1998; Bjornstad et al., 2002; Krebs et al., 2013; Tenow et al., 2013). Well-known examples include the travelling waves in vole (*Microtus* and *Clethrionomys* spp.) abundance, which have been found at both landscape scales in England (Lambin et al., 1998) and regional scales in Fennoscandia and France (Ranta & Kaitala, 1997; Berthier et al., 2014), and the larch budmoth outbreaks that pass across the European Alps (c. 1200 km) every 8-9 years (Bjornstad et al., 2002). In cyclical species, travelling waves occur when synchronised peaks in local population density/abundance move through space in a single direction, but owing to spatially propagating population growth, rather than the mass-migration of individuals (Sherratt, 2001). Consequently, anisotropic (directional) spatial synchrony declines with distance in the direction of wave propagation (and may increase again if the wavelength is shorter than the study area), but remains at similar levels (or declines much less rapidly) with distance parallel to the direction of wave propagation (Sherratt, 2001).

Investigation into the possible causes of travelling wave dynamics has primarily involved theoretical modelling studies (reviewed by Sherratt & Smith, 2008), with models indicating a variety of possible mechanisms capable of causing travelling waves in cyclical populations, including species invasions (Sherratt, 2001), inter-population dispersal (Garvie & Golinski, 2010), gradients in habitat quality (Johnson et al., 2004, 2006; Johnson et al., 2010) and hostile boundaries (Sherratt et al., 2002; Sherratt et al., 2003; Sherratt, 2013). With few examples of travelling waves available though, empirical testing of hypotheses has been very rare. However, modelling and empirical analysis of the larch budmoth system in the European Alps has shown that the observed travelling wave patterns may be explained by habitat quality gradients, with outbreaks originating in epicentres of high-quality habitat, from where they spread to areas of low-quality habitat (Johnson et al., 2004, 2006; Johnson et al., 2010). Similarly, travelling waves in French vole populations appear to emanate from large-scale landscape discontinuities, hostile to the dispersal and survival of voles, consistent with a hostile-boundary driven mechanism (Sherratt & Smith, 2008; Berthier et al., 2014).

Previous research has shown that winter moth populations exhibit extensive spatial synchrony and regional travelling waves in Fennoscandia (Tenow et al., 2007), and it has recently become clear that Fennoscandian winter moth travelling waves are actually part of a much larger process, whereby travelling waves in winter moth abundance pass across Europe in a

broadly WNW direction every 9-10 years, resulting in a rapid wave speed of 330 km $year^{-1}$ and a wavelength of 3135 km (Tenow et al., 2013). The waves appear to begin west of a vast zone of increasingly fragmented broadleaved forest habitat that occurs in E/SE Europe (Tenow et al., 2013). Consequently, based on the hostile boundary hypothesis (Sherratt et al., 2002; Sherratt et al., 2003), Tenow et al. (2013) suggested that this zone acts to prevent effective winter moth dispersal between local populations, preventing enemy-driven cycles from synchronising regional dynamics. However, west of this zone they have argued that as suitable habitat becomes more continuous within-patch dynamics become synchronised by short-distance dispersal of winter moths and their delayed density-dependent mortality factors such as parasitoids. Consequently, it is argued, these populations are then able cycle in synchrony, peaking every 9-10 years, after which they are then suppressed for many years by induced host plant defences and delayed density-dependent parasitism. Further west of these populations though (parallel to the hostile zone) plant defences and levels of parasitism will be low, allowing populations to increase and peak with a lag relative to those further east. In this way Tenow et al. (2013) suggest travelling waves in winter moth abundance are directed across Europe in a broadly WNW direction.

However, it was also suggested by Tenow et al. (2013) that the English Channel (and the European Alps and the Baltic) may have delayed but not stopped the progress of the waves from reaching Britain. This has not been explicitly tested though, and alternatively the English Channel may instead act to prevent the passage of European winter moth waves from 'arriving' in Britain, severing any link between the dynamics of British and mainland European winter moth populations. The population dynamics of the winter moth dynamics within Britain have apparently never been investigated at large spatial-scales though, and so it is unclear whether they display travelling waves, and also spatial synchrony, and if so what the causes might be. Furthermore, there seems no reason why coastlines should not also function as hostile boundaries to dispersal and survival for the winter moth, and thereby generate travelling waves that move away from coastlines and into the European and British mainland. For example, although based on much smaller scales, spatial models of vole predator-prey dynamics based on the Kielder Forest area have shown that the Kielder Water reservoir might act as a wave-generating hostile boundary (Sherratt, 2013), with waves emanating out in all directions from the edge of the lake.

Therefore, in this chapter a 45-year dataset of winter moth light trapping counts from sites across Britain is used to explore the long-term large-scale population dynamics of British winter moth. It is hypothesised that, as in Europe, British winter moth populations will display cyclicity and spatial synchrony in their dynamics. However, although the relative roles of climate and dispersal in generating synchrony in winter moth dynamics have been previously investigated at landscape scales (approximately 40 km) using island populations in Fennoscandia (Ims et al., 2004), there does not appear to have previously been an explicit comparison between the spatial extent of correlated climatic variation and winter moth population dynamics at regional scales. Therefore, the extent of spatial synchrony in climatic variation and winter moth population dynamics within Britain were also compared, and it was hypothesised that spatial synchrony in climatic variation would be at least as great as spatial synchrony in winter moth dynamics, consistent with a Moran effect acting on the relatively poorly dispersing winter moth. Using British and European winter moth data, it was also tested whether British winter moth dynamics were consistent with being part of the pan-European travelling winter moth waves, or whether, as hypothesised, the English Channel instead acts as a barrier to mainland European winter moth waves reaching Britain, contrary to the assertion of Tenow et al. (2013). Finally, it was tested whether there was any evidence for winter moth travelling waves within Britain, something that has apparently not previously been investigated for any insect within Britain previously, but was hypothesised to be the case.

5.3 Methods

The analyses aimed to test three main questions. Firstly, whether British winter moth dynamics display spatial synchrony, and if so whether the extent of spatial synchrony in climatic fluctuations across Britain is at least as great as the extent of spatial synchrony in winter moth dynamics, potentially indicative of a Moran effect (Bjornstad & Bascompte, 2001). Secondly, whether British winter moth population dynamics appear to be part of European winter moth travelling waves or not. Thirdly, whether British winter moth dynamics display travelling waves.

5.3.1 Spatial synchrony

Spatial synchrony and travelling waves in winter moth dynamics within Britain were investigated using yearly abundance data from light trap counts of winter moth, gathered across Britain between 1968 and 2012 by the Rothamsted Insect Survey (Fig. 5.1). The Rothamsted Insect Survey was established in the 1960s as a nationally operated network of light-traps that provide standardised, nightly counts of adult moths from a wide range of habitats (Conrad et al., 2004; Conrad et al., 2006). Consequently, the data are suitable for long-term monitoring of common and widespread insects (Conrad et al., 2004; Conrad et al., 2006), and studies of large-scale spatial synchrony in population dynamics (Koenig, 1999). Data were restricted to those light traps operating for at least 48 weeks a year in all years of operation. Light traps operated for varied periods between 1968 and 2012, with substantial variation in their yearly mean counts. To ensure that light traps producing very few yearly records and/or mainly zero winter moth counts (and therefore providing little or no information on winter moth dynamics) were excluded, only data from those light traps that produced at least ten years' worth of (not necessarily consecutive) non-zero counts were used in all subsequent analyses (Conrad et al., 2004; Conrad et al., 2006).

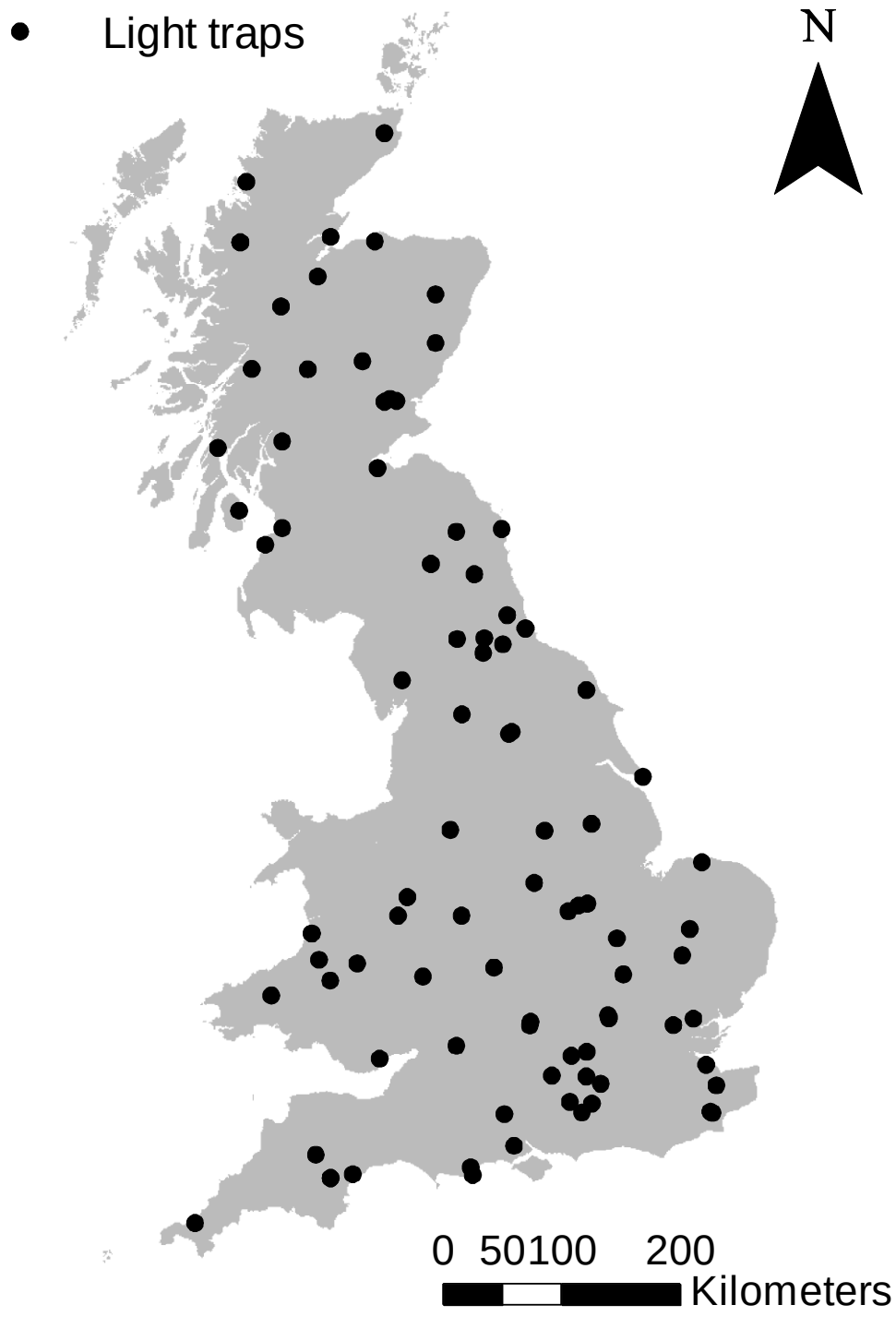

Figure 5.1 Distribution of Rothamsted Insect Survey light traps used in the analyses across Britain.

To analyse the extent of spatial synchrony in British winter moth dynamics, an isotropic (non-directional) spline correlogram was constructed from the light trap data with bootstrapped 95% confidence envelopes calculated from 1000 samples, with missing data handled through pairwise deletion of values when at least one of a pair was missing (Bjornstad, 2009). The isotropic spline correlogram was created using the R package *ncf* (Bjornstad, 2009), and is based on the nonparametric correlation function for spatio-temporal data, which describes how the correlation between pairs of time series vary with the distance between them (Bjornstad & Falck, 2001). Rather than using the original abundance data for the correlogram though, growth rates were used instead, because correlations based directly on abundance can reflect long-term local temporal trends in abundance (Bjornstad et al., 1999). Growth rates were calculated as from the light trap abundance data as: $\ln(N_{t+1}/N_t)$, where N_t is, for each light trap, the total number of adult moths caught in a given year. A constant of 1 was added to all values before calculating the growth rate to ensure non-zero values.

The extent of spatial autocorrelation in climatic variation within Britain was also analysed using isotropic spline correlograms, so that it could be tested whether the spatial extent of winter moth population synchrony within Britain was consistent or not with being driven by a Moran effect (Moran, 1953; Bjornstad et al., 1999). Therefore, isotropic spline correlograms were created for British yearly mean-monthly maximum and minimum temperatures, and British yearly mean-monthly total rainfall values. The climate data were obtained from 34 UK Met Office weather stations distributed across Britain (Fig. 5.2, UK Met Office, 2015), with data obtained from 1968-2012. These three climate variables were the only available for the spatial and temporal extent of the winter moth light trap data.

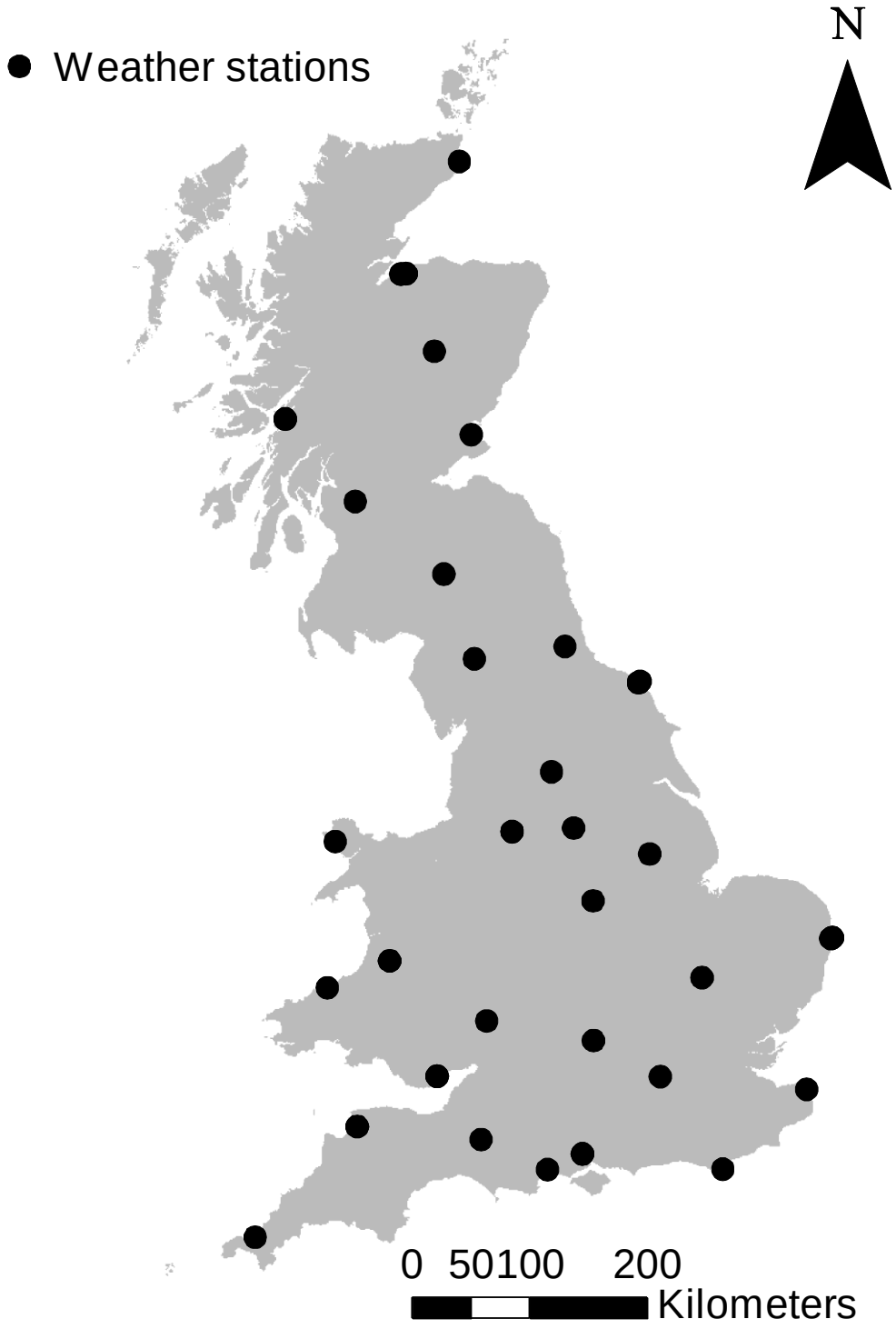

Figure 5.2 Distribution of UK Met Office historic weather stations across Britain.

5.3.2 Travelling waves

To analyse whether there was evidence for travelling waves in winter moth abundance within Britain, anisotropic (directional) spline correlograms were created from the light trap data with bootstrapped 95% confidence envelopes calculated from 1000 samples, and missing data handled through pairwise deletion of values when at least one of a pair was missing (Bjornstad et al., 2002). The anisotropic spline correlograms were created using the R package *ncf* (Bjornstad, 2009). Again, growth rate data, calculated as before, was used instead of the original abundance data. The anisotropic spline correlograms were based on the anisotropic nonparametric correlation function and the anisotropic nonparametric lagged cross-correlation function for spatio-temporal data (Bjornstad et al., 2002). The anisotropic nonparametric correlation function describes how the correlation between pairs of time series vary with the distance between the time series in a given direction (Bjornstad et al., 2002). Opposite angles produce identical correlograms, and therefore only angles between 202.5° and 360° (i.e. westerly angles, which is the broad direction in which European winter moth travelling waves move) in equal 22.5° intervals were assessed. The anisotropic nonparametric lagged cross-correlation function describes how the cross-correlation between lagged time series (i.e. the correlations between pairs of time series when one of them is lagged by one or more years) varies with the distance between time series in a given direction (Bjornstad et al., 2002). With the anisotropic nonparametric lagged cross-correlation function every angle produces a different correlogram (although correlograms for opposite angles are typically very similar), and so correlograms for all angles between 0° and 337.5° in equal 22.5° intervals were assessed.

When a travelling wave is present in spatio-temporal data a spline correlogram based on the anisotropic nonparametric correlation function should display initially high levels of correlation between spatially separate time series, which declines rapidly with distance in the direction of wave propagation (or its opposite angle), but much less rapidly, if at all, parallel to this direction (Bjornstad et al., 2002; Sherratt & Smith, 2008). Following the movement of a wave through space and time a spline correlogram based on the anisotropic nonparametric lagged cross-correlation function should provide evidence of the wave's movement when time series that are lagged by an appropriate amount (e.g. a time period less than the period between

waves) are compared. Such a correlogram should display initially low levels of synchrony between time series in the direction of wave propagation, with synchrony increasing to a peak relative to the distance that the wave's peak has travelled, given its speed and the temporal lag between time series (Bjornstad et al., 2002). Angles parallel to the direction of wave propagation should show little if any synchrony with increasing distance between lagged time series. In this analysis a lag of 1 year was considered in the lagged anisotropic spline correlograms.

To analyse whether British winter moth dynamics appear to be influenced by European winter moth travelling waves or not, the light trap data was also used to determine when regional-scale peaks in British winter moth population cycles occurred between 1968 and 2012 (the temporal range of the data). It was then tested whether these peaks appeared to fit in or not with the spatio-temporal progress of the European winter moth travelling waves, i.e. whether British winter moth peaks were consistent with being part of the European winter moth travelling waves, or whether they occurred substantially after or before the European waves would be expected to arrive in Britain, given their rate of progress across mainland Europe.

This was done following the methods of Tenow et al. (2013), who, using data on European winter moth population peak years between 1950 and 2010 (Tenow et al., 2013, supporting information S2), regressed the last two digits of the years in which European local winter moth populations peaked (i.e. the year in decade) on their perpendicular distances from a 'western baseline'. This baseline consisted of a great circle on the GR80-ellipsoid running N-S around the earth, arbitrarily located to pass through Greenwich (51.477222°, 0°), and rotated 18° from true north. The perpendicular distances of all local winter moth populations from this western baseline were therefore their relative distances along a broadly ESE-WNW axis, which was the approximate direction in which winter moth travelling waves appeared to pass through certain European countries (Tenow et al., 2013). The resulting highly significant negative relationship between the distance from the western baseline and the year in decade of peaks indicated the presence of travelling waves in winter moth population abundance passing across mainland Europe in an ESE-WNW direction every 9-10 years, and moving approximately 330 km $year^{-1}$ (Tenow et al., 2013). By rotating the western baseline through different angles relative to true north and comparing resulting model R^2 values, Tenow et al. (2013)

determined that travelling waves moving perpendicular to a western baseline rotated 18° from true north resulted in the best fit, and therefore this baseline is also used here subsequently.

By combining data on British region-wide winter moth peak years and their locations relative to the western baseline with the European winter moth data of Tenow et al. (2013), it was then possible to test whether British winter moth dynamics appeared to be influenced by European winter moth travelling waves or not. However, a wave front of peaking winter moth populations travelling at 330 km $year^{-1}$ and moving WNW, perpendicular to the assumed western baseline, would pass across Britain in approximately two years, given that the approximate distance such a wave would travel from the point of first contact to the point of final contact with mainland Britain (excluding any islands) is 660 km (Fig. 5.3). Therefore, Britain was first divided into an eastern and a western region, and then region-wide winter moth peak years within each region determined. These two regions were defined by dividing Britain (excluding any islands) in half along an axis rotated 18° from true north (i.e. parallel to the western baseline) and located half-way between where a wave front moving perpendicular to this line would first contact and then leave the British mainland (excluding any islands) (Fig. 5.3). Within the resulting western and eastern regions the relevant available light trap data was then used to create yearly relative abundance indices of regional winter moth dynamics, using the 'time effects model' of the statistical software TRIM (TRends and Indices for Monitoring data Pannekoek et al., 2005).

Figure 5.3 Axes used to divide Britain into a western and eastern region, and to divide each region into two equal halves along an 18° axis, parallel to the western baseline.

The TRIM time effects model uses a generalised linear model (GLM) with Poisson errors and a log link, and estimates parameters and their standard errors for each time period available using generalised estimating equations, which account for the temporal autocorrelation typically present in such data (Pannekoek & Van Strien, 2001). To create the relative abundance index the software arbitrarily sets the estimated abundance of populations in the first year to one, and calculates yearly change-in-abundance parameters relative to the first, suitable for when populations vary highly in their mean abundances (Pannekoek & Van Strien, 2001; Van Strien et al., 2001). TRIM also deals with missing data through an imputation approach (Pannekoek & Van Strien, 2001; Van Strien et al., 2001), and requires at least one count per population, but based on a more conservative approach than previously used with Rothamsted Insect Survey data (Conrad et al., 2004; Conrad et al., 2006), only those light traps that produced at least ten years' worth of non-zero counts were included in the models (Conrad et al., 2004; Conrad et al., 2006).

The resulting relative abundance indices were then used to determine the years in which British eastern and western region-wide winter moth population peaks occurred. Peaks were determined only if there were clear cycles characteristic of a cyclical Lepidoptera, each involving a multi-year increase phase, a peak of one or more years, with the year of greatest relative abundance taken as the peak year for each cycle, and a multi-year decline (or crash) and trough phase (Myers & Cory, 2013). The perpendicular distances from the western baseline of each region was measured relative to two axes, rotated 18° from true north, dividing each region in half parallel to the expected direction of any winter moth travelling waves (Fig. 5.3). The resulting British region-wide peak years in decade and their distance from the western baseline were then added to the Tenow et al. (2013) dataset (Tenow et al., 2013, supporting information S2). However, all records of winter moth population peaks >2300 km from the western baseline were first removed from the original Tenow et al. (2013) dataset (Tenow et al., 2013, supporting information S2), as the original analysis of Tenow et al. (2013) indicated these populations did not form part of the travelling waves. In addition, a number of pre-existing records of British local winter moth population peaks exist in the original dataset, but were also removed first because they were local records and may not accurately represent the effects of any region-wide travelling wave patterns, and would also effectively be duplicated data. Using the resulting dataset, the year in decade in which each winter moth population peak occurred was

then regressed against its distance from the western baseline in a general linear model. However, a second general linear model was then also fitted to the data, which also contained a categorical variable distinguishing mainland European from British records. This allowed an assessment of whether the British peaks occurred when they would be expected to if they were resulting from the 'arrival' of European winter moth travelling waves into Britain, or whether they occurred consistently before (a negative difference between the intercept estimates for the European and British records) or after (a positive difference between the intercept estimates for the European and British records) they would be expected to, assuming they were part of the same travelling waves process.

To assess the relative evidence in support of each model the two models were compared in a MMI approach, which involved ranking them based on their AIC scores, and then calculating their Akaike weights and the evidence ratio between the models (Burnham & Anderson, 2002). The evidence ratio is simply the ratio of the highest to the lowest Akaike weight (i.e. the ratio of model probabilities), and represents the relative weight of evidence in favour of the most probable model (Burnham & Anderson, 2002). Inference was then based on the parameter estimates and their 95% confidence intervals for the most probable model (Burnham & Anderson, 2002). All models were fitted using the statistical software R (R Core Development Team, 2014), with model assumptions checked using a standard range of residual plots (Zuur et al., 2009).

5.4 Results

The isotropic spline correlogram revealed weak synchrony in winter moth dynamics between 1968 and 2012, which declined slowly with distance up to around 580 km (the lower 95% confidence envelope crossed 0 at a lag distance of 584 km), after which there was not strong evidence for any further synchrony (Fig. 5.4). Although synchrony appeared to increase somewhat after approximately 800 km lag distance, this is likely to be a spurious pattern because of the rapidly increasing error at the largest lag distances due to the small number of available populations with which to estimate correlations (Bjornstad & Falck, 2001). The isotropic spline correlograms for all three climate variables indicated similar spatial patterns in the strength of their temporal correlations between 1968 and 2012, with

moderately strong correlation in their temporal variation persisting at quite stable levels until at least 700 km, with relatively rapid declines in the strength of spatial correlation at larger distances (the lower 95% confidence envelopes of the maximum temperature, minimum temperature and precipitation correlograms crossed 0 at 763 km, 695 km and 813 km respectively; Fig. 5.5 (a), (b) and (c)). Therefore, correlation in the climatic variables persisted for substantially greater distances than spatial synchrony in the winter moth's dynamics (Fig. 5.4 & Fig. 5.5 (a), (b) & (c)).

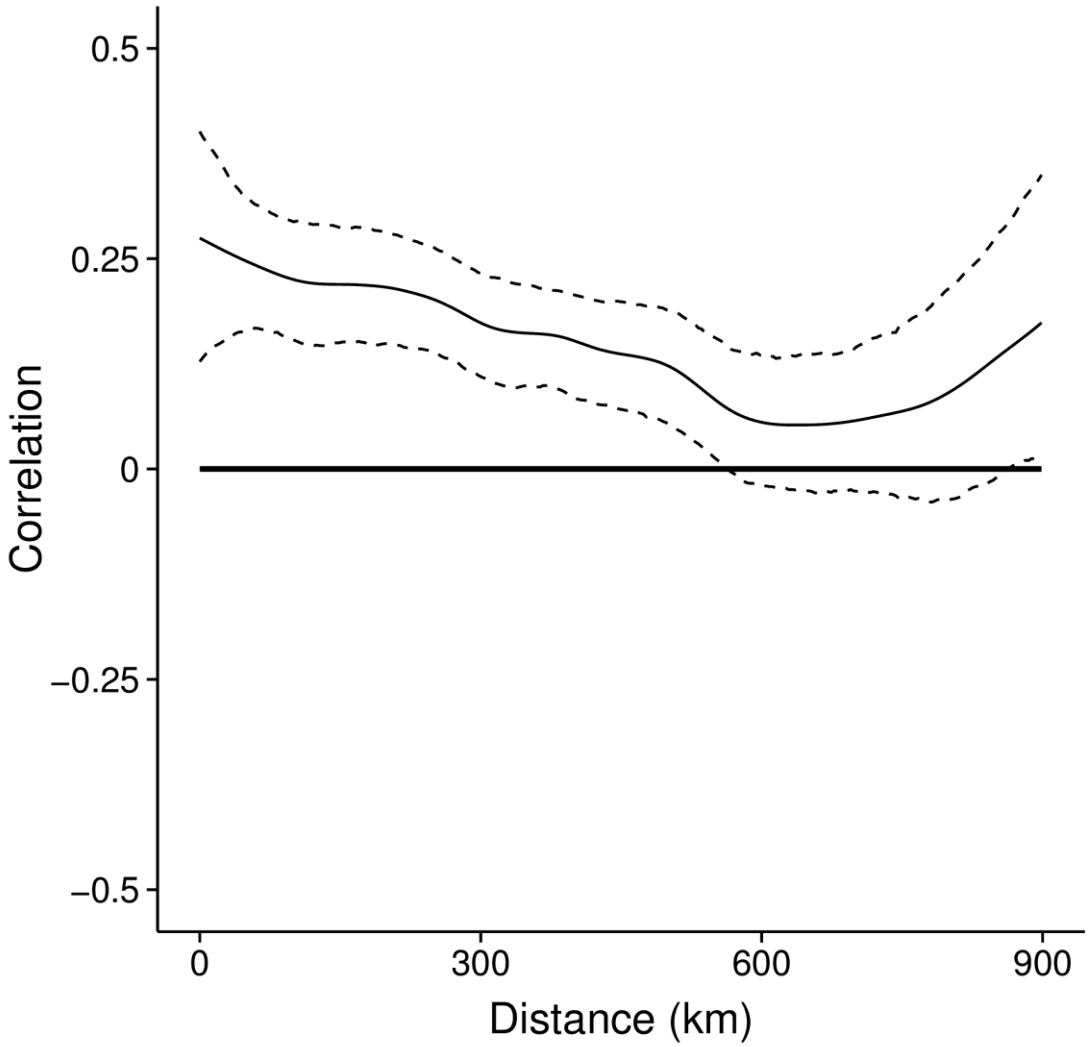

Figure 5.4 Isotropic spline correlogram for winter moth dynamics across Britain, based on population growth rates calculated from yearly light trap abundance data collected across Britain between 1968 and 2012 (Fig. 5.1), representing the estimated mean correlation (synchrony) between the temporal dynamics of winter moth populations separated by increasing lag distances. Dashed lines represent 95% confidence envelopes associated with the estimated level of correlation.

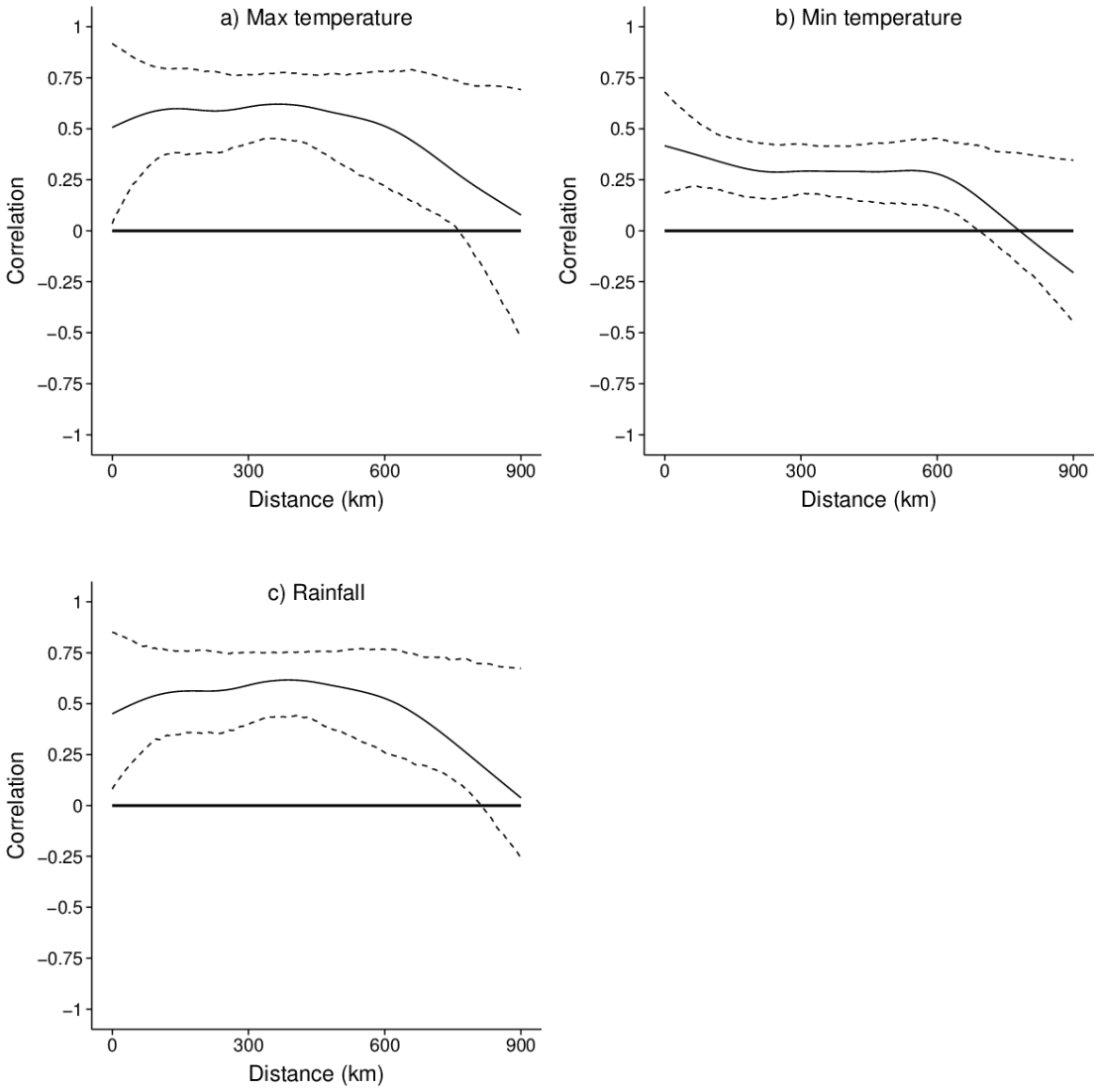

Figure 5.5 Isotropic spline correlograms for the temporal variation in yearly mean-monthly a) maximum and b) minimum temperature and c) the yearly mean-monthly total rainfall across Britain between 1968 and 2012, based on data collected at 34 weather stations across Britain, representing the estimated mean correlation between the temporal variation in climate variables measured at weather stations separated by increasing lag distances. Dashed lines represent the 95% confidence envelopes associated with the estimated mean levels of correlation.

The non-lagged anisotropic spline correlogram indicated no clear directional spatial structure in winter moth population synchrony between 1968 and 2012 at any angle (Fig. 5.6). The anisotropic lagged spline correlograms also provided no indication of any peaks in winter moth abundance moving across Britain in any direction between 1968 and 2012 (Fig. 5.7 & 5.8). Therefore, there was no evidence for travelling waves patterns in the British winter moth abundance data.

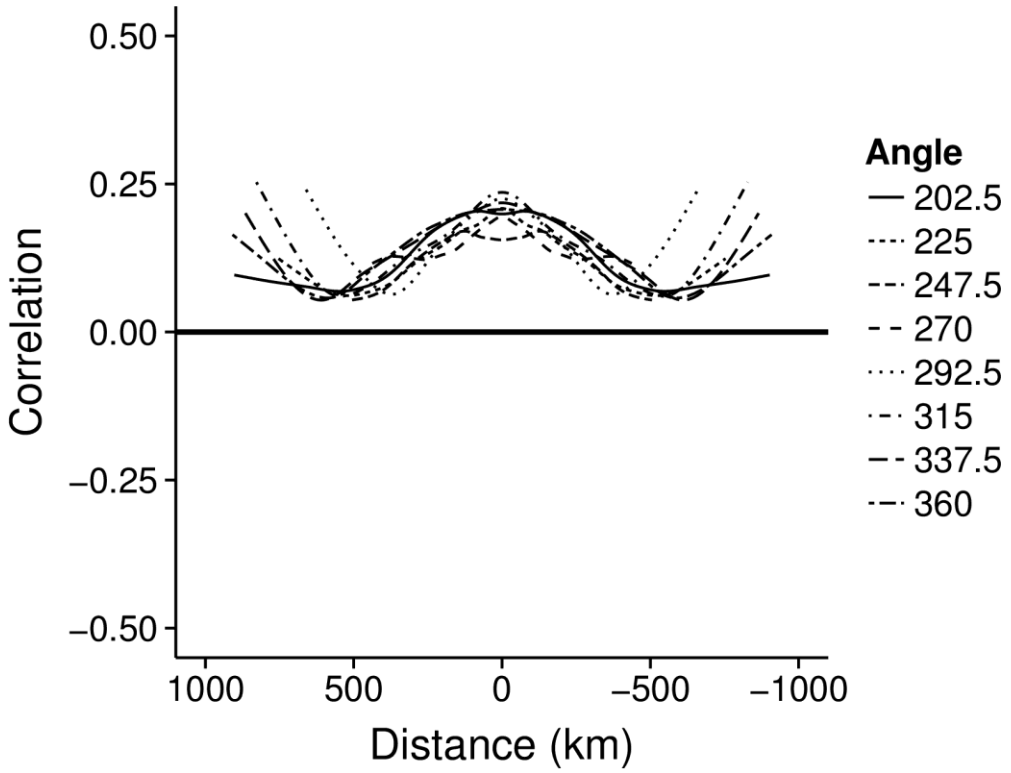

Figure 5.6 Anisotropic (directional) spline correlogram for winter moth dynamics across Britain, based on the nonparametric anisotropic correlation function applied to winter moth population growth rates calculated from yearly light trap abundance data collected across Britain between 1968 and 2012. The spline correlogram represents the estimated mean correlation (synchrony) between the temporal dynamics of winter moth populations separated by increasing lag distances measured along different directional axes. Only westerly angles are presented, as the results from the opposite angles are identical. 95% confidence envelopes are not displayed for clarity, but highlighted no differences between the patterns of spatial correlation calculated for any angle.

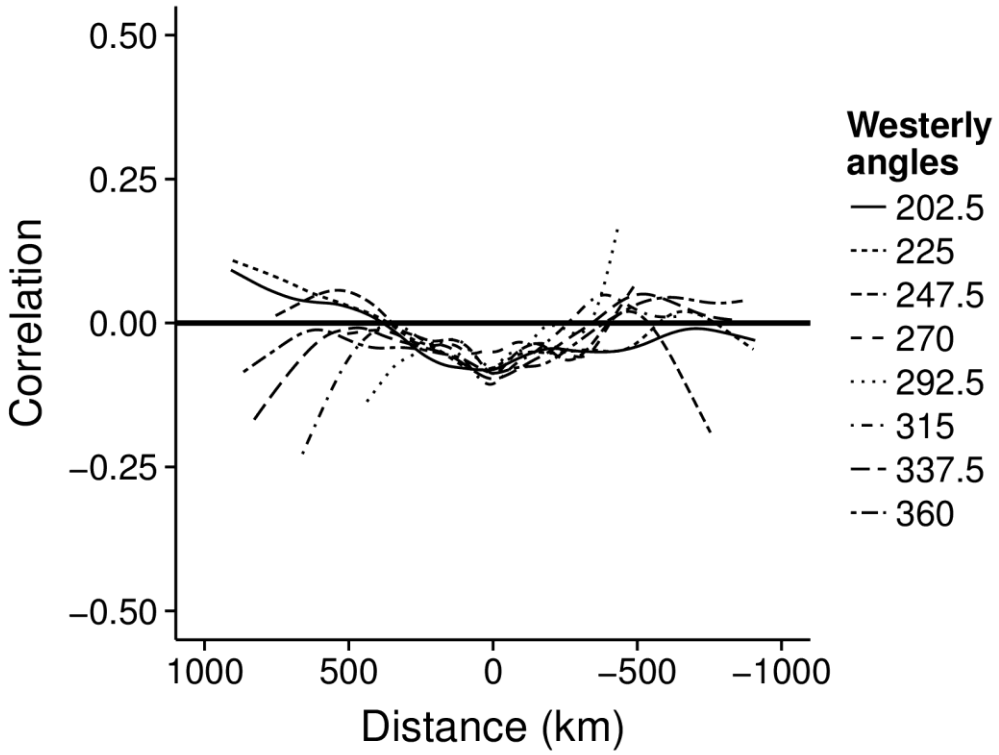

Figure 5.7 Anisotropic (directional) spline correlogram for winter moth dynamics across Britain, for westerly angles based on the nonparametric anisotropic lagged cross-correlation function applied to winter moth population growth rates calculated from yearly light trap abundance data collected across Britain between 1968 and 2012. The spline correlogram represents the estimated mean cross-correlation (synchrony) between the temporal dynamics of winter moth populations lagged in time (by 1 year), and separated by increasing lag distances measured along different directional axes. 95% confidence envelopes are not displayed for clarity, but highlighted no differences between the patterns of spatial lagged cross-correlation calculated for different angles.

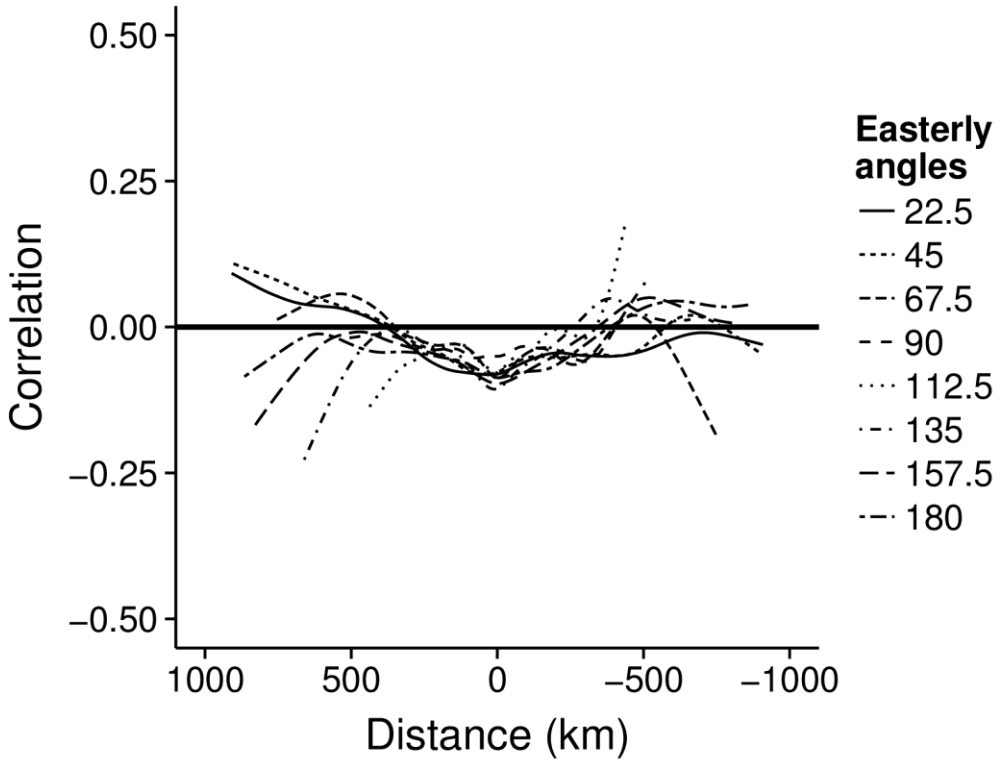

Figure 5.8 Anisotropic (directional) spline correlogram for winter moth dynamics across Britain, for easterly angles based on the nonparametric anisotropic lagged cross-correlation function applied to winter moth population growth rates calculated from yearly light trap abundance data collected across Britain between 1968 and 2012. The spline correlogram represents the estimated mean cross-correlation (synchrony) between the temporal dynamics of winter moth populations lagged in time (by 1 year), and separated by increasing lag distances measured along different directional axes. 95% confidence envelopes are not displayed for clarity, but highlighted no differences between the patterns of spatial lagged cross-correlation calculated for different angles.

Cyclical dynamics were only evident from winter moth light trap catches in the western region of Britain, and so no peaks were determined for the eastern region (Fig. 5.9 (a) & (b)). However, within the western region between 1968 and 2012 there were four clear cycles, with peaks occurring in 1975, 1986, 1994 and 2004 (Fig. 5.9 (a)). Therefore, cycle periodicity in the western region ranged from 8-11 years. The western regional British winter moth peak years and their distances from the western baseline were then combined with the Tenow et al. (2013) dataset of European winter moth peak year in decade and locations (Tenow et al., 2013, supporting information S2), but with all pre-existing local British records removed, along with all records of peaks at locations >2300 km from the western baseline, which do not apparently form part of the European travelling waves (Tenow et al., 2013).

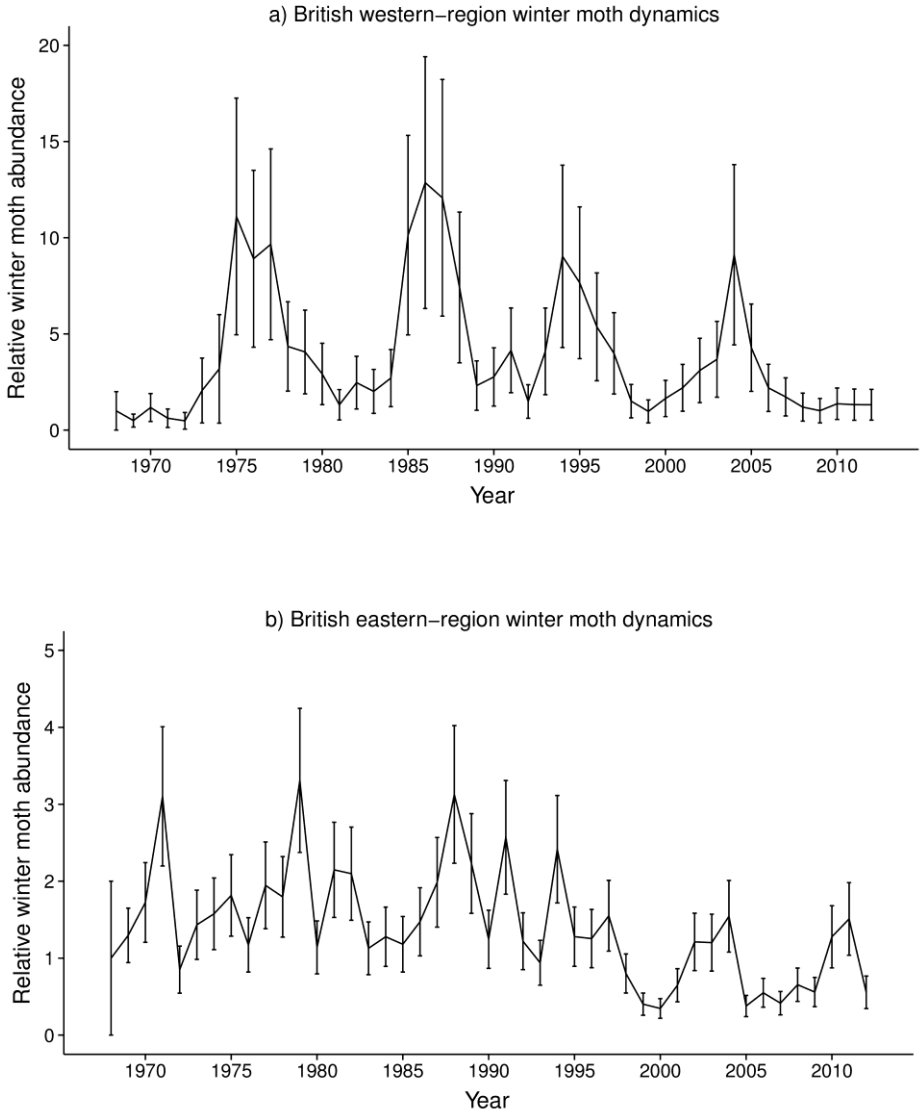

Figure 5.9 Long-term winter moth population dynamics in the western (a) and eastern (b) regions of Britain (Fig. 5.3). Relative winter moth abundance values (and their standard errors) represent the multiplicative change in expected abundance relative to the abundance in the first year of the time series (1968), based on a GLM of winter moth light trap abundance data gathered between 1968 and 2012 from light traps within each region.

A GLM of the relationship between the distance from the western baseline of winter moth peaks and the year in decade in which they occurred was then fitted to the data, along with a second GLM that also contained a parameter estimate for the mean difference between an intercept fitted to all mainland European records, and a second intercept fitted to just the British records (Fig. 5.9). This second model was 13.9 times more likely to be a better model of the data than the initial model (Table 5.1), and was therefore used for subsequent inference. The difference between the intercept for the European records and the British-only records intercept (Table 5.2) indicated that British western-regional winter moth peaks occurred on average 3.2 years before they were predicted to, based on the relationship between the distance from the western baseline and the year in decade in which populations peak when fitted to data for European records only (Table 5.2). Therefore, this indicated that whilst an ESE-WNW directed travelling wave in winter moth population peaks moves across mainland Europe every 9-10 years, by the time it would be expected to reach the western region of Britain (assuming it moves in a constant direction and at a constant rate) winter moth populations across this area of Britain have already peaked on averaged 3.2 years earlier (Table 5.2 & Fig. 5.9).

Table 5.1 AIC-based model selection table.

Model	K	AIC	Δ AIC	Akaike weight	Evidence ratio	Adj. R^2
E + B	4	390.727		0.933	13.925	0.305
O	3	396.01	5.283	0.067		0.271

O = Overall intercept + distance from western baseline. E + B = Intercept for mainland European records only and difference to intercept for British records + distance from western baseline. K = no. of model parameters.

Table 5.2 Linear model parameter estimates and their upper and lower 95% confidence intervals for a GLM modelling the relationship between the distance from the western baseline of mainland European and winter moth populations and the year in decade when populations peaked on average, with an additional parameter estimating the difference between the intercept fitted to the European records and the British records (whilst assuming the same relationship between the distance from the western baseline and the year in decade of population peaks for both European and British populations).

Parameter	Estimate	UCI	LCI
European records intercept	7.1	7.87	6.34
Difference between European and British records intercept	-3.24	-0.9	-5.59
Distance from western baseline slope parameter*	-0.23	-0.16	-0.3

* = To aid interpretation the original parameter and confidence intervals were multiplied by 100 to represent the additive effect to the year in decade in which a population is predicted to peak following an addition of 100 kilometres to the population's distance from the western baseline.

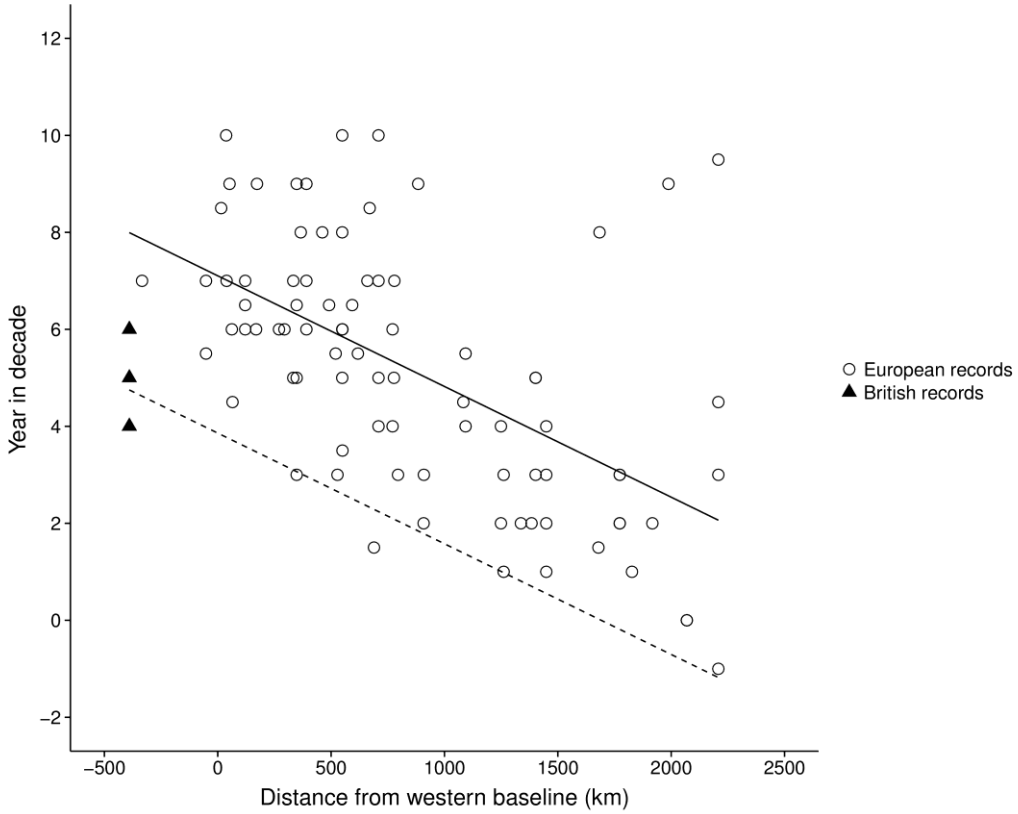

Figure 5.10 Relationship between distance from the western baseline of winter moth populations and the year in decade in which they peaked for winter moth records from mainland Europe and Britain (Fig. 5.9 (a)). The solid line represents a GLM-predicted best fit of this relationship when fitted to mainland European records, whilst the dashed line represents the mean difference in the intercept of this relationship when fitted to just the British records. This clear negative relationship indicates that there is a consistent ESE-WNW movement of winter moth population peaks across mainland Europe every 9-10 years, but when this wave of population peaks is expected to reach the western-region of Britain, winter moth populations in this area have already peaked on average 3.2 years earlier.

5.5 Discussion

The analyses indicated that, like in mainland Europe (Schott et al., 2010; Tenow et al., 2013; Klemola et al., 2014), winter moth populations display long-term (8-11 year) cyclical dynamics within Britain over long time scales, although there was only evidence for these dynamics in the western region of Britain. There was also evidence that the dynamics of winter moth populations are moderately to weakly synchronous over extensive spatial scales within Britain, but there was apparently little evidence of any synchrony between populations extending for distances greater than approximately 580 km or more. Long-term climatic variation within Britain over the same time period was more strongly correlated and over greater spatial scales that varied between maximum lag distances of approximately 700-800 km. However, there was no evidence that the winter moth travelling waves in mainland Europe reach Britain. Instead, British winter moth peaks occurred on average over three-years before these European travelling waves would be expected to arrive in Britain, based on their spatio-temporal progress across mainland Europe. Lastly, there was also no evidence for any winter moth travelling waves within Britain.

Winter moth cycles are generally believed to be caused by delayed density-dependent mortality from specialist natural enemies (chapter 2; Myers & Cory, 2013), but there do not appear to be any obvious mechanisms explaining the strongly differing dynamics between the eastern and western British regions, particularly when winter moth cycles are known to occur over such an extensive area of Europe, encompassing much greater variation in habitat and climate (Tenow et al., 2013). Therefore, possibly more plausibly, the lack of cyclicity in eastern Britain could just be an artefact of the data, and in particular the variation in temporal coverage of different light traps. Although there was no evidence for any travelling waves in winter moth abundance within Britain, there was evidence for spatially synchronous dynamics. Regional-scale spatial synchrony in winter moth dynamics have been documented previously in Fennoscandia (Tenow et al., 2007), where they were assumed to be driven by a Moran effect (i.e. spatially correlated environmental stochasticity) from the North Atlantic Oscillation, although this was not explicitly tested. Therefore, it was explicitly demonstrated for the first time here that the extent of correlation in climatic variation was greater than the extent of spatial synchrony in winter moth dynamics, consistent with a Moran effect driving winter moth spatial

population synchrony within Britain (Moran, 1953; Bjornstad et al., 1999). Determining the precise mechanisms likely to be driving a Moran effect in British winter moth populations is difficult, because there are so many possible climate variables, and they are typically highly correlated with one another (Braunisch et al., 2013), as seen here. However, there are two broad mechanisms by which spatially correlated climatic variation may induce synchrony: either through direct effects on survival and reproduction in winter moth populations, or through indirect effects that impact their key trophic interactions with other species (Bjornstad et al., 1999).

Two potentially important climatic effects on key winter moth trophic interactions involve sunlight and precipitation. It is known that the winter moth suffers from potentially regulatory delayed density-dependent mortality from an NPV in Orkney (chapter 2), and although unknown at present NPV mortality could also be important for population regulation throughout Britain as well. Assuming this to be the case, chapter 2 also found that temperature during larval development in the previous year has a negative effect on winter moth-NPV interactions, which may be explained by the correlation between temperature and sunlight (solar radiation) levels, with greater levels of solar radiation known to inactivate NPV in the environment (Jones et al., 1993; Cory & Myers, 2003). Alternatively, rainfall is known to be involved in the transportation of NPV between branches, plants and out of soil reservoirs, altering the likelihood of NPV transmission and the prevalence of NPV disease (Damico & Elkinton, 1995; Fuxa & Richter, 2001; Fuxa et al., 2007). Therefore, spatially correlated patterns in levels of sunshine and/or precipitation could influence cyclical host-disease dynamics within British winter moth populations, leading to spatial population synchrony. An NPV-precipitation based mechanism has been suggested previously as a possible explanation for similarly large-scale spatial synchrony in gypsy moth populations (Haynes et al., 2013), but has not yet been investigated explicitly in empirical studies. Mechanistic studies are therefore needed to test these hypotheses fully.

Therefore, the results presented here appear broadly consistent with the growing body of evidence that the characteristic extensive spatial synchrony seen in cyclical lepidopteran populations is most likely driven by Moran effects (Peltonen et al., 2002; Haynes et al., 2009; Haynes et al., 2013). It is important to note though that theoretical and laboratory-based mesocosm studies show that even very small (e.g. as little as 0.4% per generation) amounts of dispersal can generate synchrony between linked

populations over relatively large distances (Vasseur & Fox, 2009; Fox et al., 2011; Fox et al., 2013). Therefore, dispersal cannot be completely ruled out as contributing to the observed patterns in winter moth population synchrony in an observational study such as this. However, the winter moth is believed to be a weak disperser, with males having very restricted dispersal abilities (Van Dongen et al., 1996), whilst females are flightless. Early-instar larvae are able to disperse via ballooning for at least several hundred meters though (Edland, 1971), and a recent population genetics study indicated that winter moth populations separated by up to 25 km were actually relatively well mixed, indicating more effective dispersal than generally assumed (Leggett et al., 2011). However, although it seems unlikely that winter moth populations separated by up to 580 km should show synchrony due to dispersal, the importance of dispersal for British winter moth spatial synchrony, as with cyclical insects generally (Myers & Cory, 2013), is still largely unknown.

The lack of evidence for travelling waves in winter moth abundance within Britain may be interpreted in two ways: either winter moth populations do not exhibit large-scale travelling waves in abundance within Britain, or they do occur but could not be detected in this data. It is not immediately clear why winter moth populations should display travelling wave behaviour within mainland Europe but not Britain though. Given the regularity of European winter moth travelling waves, climate is not believed to play a role in their generation (Tenow et al., 2013), making any climate related differences an unlikely explanation. Alternatively, travelling waves in winter moth abundance may occur in Britain, but it may not have been possible to detect them using the current data. In Europe, winter moth travelling waves move at approximately 330 km $year^{-1}$, and have wavelengths of approximately 3135 km (Tenow et al., 2013). Therefore, if waves of a similar nature occurred within Britain, particularly if they moved in a broadly E-W or W-E direction, there would be relatively little geographical area available in which to detect the movement of such a wave (i.e. there would be relatively little area over which the distinct sections of such a wave would be detectable). Consequently, the data may have lacked the necessary degree of spatial resolution needed to detect such patterns. Unfortunately it is not currently possible to distinguish between these two possibilities at present. If present though, travelling waves in winter moth (and other species') abundance could be detected within Britain using a sufficiently large-scale observational study lasting just a few years, if of

appropriate spatial resolution and targeted at the next expected regional peak.

It is currently unclear what drives the European winter moth travelling waves (Tenow et al., 2013), or indeed travelling waves in any species (Sherratt & Smith, 2008). However, primarily based on results from theoretical models, as well as their frequency in nature, hostile boundaries have been advanced as one likely cause (Sherratt et al., 2002; Sherratt et al., 2003; Sherratt, 2013). Hostile boundaries occur in many landscapes, usually when there are abrupt changes in habitat, such as between land and large areas of water, or highly unsuitable habitat for a given species (Sherratt, 2013). Reaction-diffusion models (where the 'reactions' involve host-enemy interactions, and the 'diffusions' involve the dispersal of hosts and enemies) of cyclical populations demonstrate that travelling waves in abundance may be generated by hostile boundaries (Sherratt et al., 2002; Sherratt et al., 2003; Sherratt, 2013). Consequently, Tenow et al. (2013) tentatively proposed that the European travelling waves in winter moth abundance are generated by a huge zone of increasingly fragmented forest in E/SE Europe acting as a hostile boundary. Furthermore, based on their data Tenow et al. (2013) also suggested that the passage of the travelling waves across Europe may have been delayed, but not halted, by possible barriers including the English Channel, the European Alps and the Baltic.

However, the results presented here indicate that winter moth peaks in Britain since the 1970s actually happened substantially (over 3 years) before they would be expected to occur relative to the spatio-temporal progress of European travelling waves in winter moth abundance. Therefore, it seems very unlikely that British winter moth cycles are connected to those on mainland Europe, as there is no obvious mechanism that would cause British populations to peak ahead of the travelling waves on the European mainland. Instead, this finding may be more plausibly interpreted as reflecting separate spatio-temporal winter moth dynamics occurring in Britain. Consequently, this implies that the English Channel (and North Sea) do indeed act as effective barriers to European travelling waves in winter moth abundance passing from mainland Europe into Britain, as predicted. However, these travelling waves are poorly understood, and their extremely rapid movement is currently unexplained, but appears unlikely to be driven by dispersal processes because of its extremely high rate (Tenow et al., 2013). Therefore, it is not clear presently how a barrier such as the English Channel actually functions if not by preventing dispersal, and this is a key

outstanding question both for this system and for travelling wave phenomena more generally.

In addition, it is also not then clear why the zone of highly fragmented forest in eastern Europe acts as a wave-generating hostile boundary, but the coasts of Europe, presumably hostile boundaries for winter moths themselves, do not (e.g. there was no evidence of easterly travelling waves being generated from the much more spatially extensive western coastlines of Europe). In the context of reaction-diffusion models, hostile boundaries are typically modelled as preventing the dispersal and survival of individuals attempting to cross them (Sherratt & Smith, 2008). For example, reaction-diffusion models of vole-predator dynamics that include the Kielder Water reservoir in northern England, which the voles cannot cross, successfully reproduce the broad pattern of travelling waves in vole abundance observed in the area (Sherratt, 2013). Therefore, it can be argued that coastlines should represent hostile boundaries for the winter moth, given that winter moth are unlikely to be able to successfully disperse or survive beyond them (unless the distance to suitable terrestrial habitat is very short). In reaction-diffusion models involving multiple hostile boundaries of differing sizes, the largest boundary dominates the overall behaviour of the resulting travelling waves (Sherratt, 2013). However, the zone of fragmented forest in eastern Europe is much smaller in length than the coastlines of Europe, again raising the question as to why they should not be the primary driver of any travelling wave behaviour in European winter moth populations. Mechanistic spatio-temporal models have not currently been applied to the winter moth system, and further progress in explaining the European travelling waves is likely to depend heavily on refining theoretical work with relevant mechanistic empirical data. In particular, understanding species' behavioural responses to different habitat boundaries is likely to be highly important for explaining why some boundaries can act to generate travelling waves, whilst others apparently do not (Sherratt, 2013).

Therefore, this study provides the first evidence consistent with a Moran effect driving spatial synchrony in winter moth population dynamics within Britain. However, unlike in mainland Europe, there was no evidence for travelling waves in winter moth abundance within Britain, but the reasons for this are not clear, and may be due to the limitations of the data. This study also provides the first evidence that the large-scale travelling waves in European winter moth dynamics do not reach Britain, and are

apparently halted at the Atlantic coast. This raises questions about proposed mechanisms by which these travelling waves are generated, requiring further research to explore possible answers.

6. General discussion

6.1 Host-natural enemy interactions and spatio-temporal dynamics

A core aim of ecology is the understanding of population dynamics and the mechanisms that underpin them. Cyclical dynamics have inspired much ecological attention for over 90 years (Elton, 1927; Myers & Cory, 2013), and one of the primary mechanisms believed to drive these dynamics is natural enemy mortality. More specifically, theoretical models have indicated that delayed density-dependent mortality from specialist predators, parasitoids and pathogens may provide the best explanation for population cycles (Berryman, 2002; Turchin, 2003; Korpela et al., 2014). However, long-term host-enemy datasets gathered at large spatial-scales are very rare, but are necessary to test hypotheses of natural enemy population regulation (e.g. Anderson & May, 1980; Dwyer et al., 2004; Elderd, 2013; Elderd et al., 2013) in real systems. This is particularly true for insect host-pathogen systems, where previous long-term empirical studies have provided inconsistent results relating to direct density-dependent regulation, and have failed to test for delayed density-dependent regulation by pathogens (Kukan & Myers, 1999; Myers et al., 2000; Liebhold et al., 2013).

Therefore, chapter 2 provided the first evidence for delayed density-dependent mortality from a pathogen in a cyclical insect host, consistent with the pathogen playing a role in the generation of the host's cyclical dynamics (Anderson & May, 1980, 1981). Theoretical and empirical studies of population regulation in many cyclical insects have largely or exclusively focused on predators and parasitoids (e.g. Bjornstad et al., 2002; Turchin et al., 2003; Bjornstad et al., 2010; Klemola et al., 2010; Klemola et al., 2014), and it is unclear how important pathogens are more generally for such species, as they are often ignored (T. Klemola, pers. comm.; Myers & Cory, 2013). Similarly, studies in cyclical species in other taxa (such as mammals) have also largely focused on specialist predators (e.g. Krebs et al., 2001; Sherratt, 2001; Sherratt, 2013; Korpela et al., 2014), although more recently some attention has been given to pathogens as possible regulatory agents in

some small mammal populations (Cavanagh et al., 2004; Burthe et al., 2006; Telfer et al., 2008). Therefore, this thesis highlights that it is important to also consider the effects of pathogens on cyclical species' dynamics. Due to their often inconspicuous nature, pathogens can often be overlooked in ecological studies (Roy et al., 2009), but this study demonstrates the need to explicitly explore their effects when trying to understand population cycles. More generally this highlights how important it is to consider pathogens in ecological systems.

However, the results from chapter 2 also provided evidence consistent with pathogen regulation of the non-cyclical magpie moth. Irregular or complex dynamics are the norm in nature (Kendall et al., 1998), and have often been ascribed to weakly regulated populations subject to relatively stronger environmental stochastic effects (Turchin, 2003). Indeed, most populations appear to be weakly regulated (Ziebarth et al., 2010). However, most long-term studies on population dynamics have focused on cyclical species, and data on non-cyclical species' dynamics and their possible regulatory mechanisms, such as their trophic interactions, are rare. However, very strong consumer-resource interactions have been previously documented between the swallow-wort fruit fly (*Euphranta connexa*, Fabricius) and its seed resource, explaining an impressive 96% of the variance in its irregular population dynamics (Solbreck & Ives, 2007).

Therefore, although most species may be non-cyclical and weakly regulated (Kendall et al., 1998; Ziebarth et al., 2010), results from chapter 2 demonstrate that non-cyclical species can also be strongly regulated by non-stochastic mechanisms, even delayed density-dependent processes thought key to generating cyclical dynamics. Therefore, whilst the generation of population cycles may require specific regulatory mechanisms, such as delayed density-dependent trophic interactions, other factors also appear to be necessary. The results relating to the non-cyclical magpie moth also highlight that in terms of population regulation, rather than the largely exclusive focus on classically cyclical species (e.g. Krebs, 1996; Stenseth, 1999; Myers & Cory, 2013; Krebs et al., 2014), a more balanced approach investigating regulatory processes in non-cyclical species could be beneficial for improving understanding of population regulation. For example, where species such as the winter moth and magpie moth share apparently similar regulatory mechanisms within the same environmental context, but display very different dynamics, future research could focus on pinpointing the factors driving the different dynamics observed. Although there were no

clearly obvious reasons for their strikingly different dynamics in the present study, a possible target for future research would be looking at differences in life-history traits thought to be important for population cycles, such as a high level of fecundity/survival during the increase phase (Myers & Cory, 2013).

Chapter 4 highlighted that the spatial structure of habitat can influence host-enemy interactions, which as chapter 2 showed can be key for host population regulation, but as both chapters 4 and 5 showed these effects may be heavily scale dependent. At the landscape scale, the effects of habitat structure on host-enemy interactions may be dependent on the spatial scale of species' responses to habitat structure, in terms of dispersal and population density (chapter 4). These responses may often be hard to predict, and a better understanding of the mechanisms determining them is therefore needed (chapter 4). At much larger scales though the existence of extensive spatial synchrony in winter moth populations throughout Britain, and at similar or larger scales in many other cyclical species, indicates that habitat structure may not have strong effects on certain broad spatio-temporal dynamics, such as spatial synchrony and travelling waves. However, as suggested by hypotheses about the generation of travelling waves (Tenow et al., 2013), and the findings of chapter 5 related to the separating of British and mainland European winter moth dynamics, continental-scale habitat structural features may be highly influential for large-scale travelling waves.

6.2 Host resistance and host-natural enemy interactions in nature

Chapter 3 highlighted that although a huge amount has been achieved in understanding mechanisms of insect resistance through laboratory studies (e.g. Strand, 2008; Wilson & Cotter, 2009), there has been a lack of attention given to how these processes function in natural systems, subject to all the biotic and abiotic variation of nature, and how they interact with other ecological processes, such as changes in population density, to ultimately influence population dynamics. Studies are starting to look at insect resistance, particularly immunological functioning, in wild systems, and are providing interesting insights into the way immunity functions in nature (Pedersen & Babayan, 2011). For example, it has recently been

shown that geographical variation in parasitism, and therefore risk of parasitoid attack, can drive altered investment in insect larval immune systems (Vogelweith et al., 2013a). As highlighted by chapter 2, populations may also be regulated by entirely different natural enemies in different habitats/geographical areas. Consequently, this may lead to geographical variation in population dynamics, due to variation in host survival and investment or costs of resisting different natural enemies in different areas (Fellowes et al., 1998; Kraaijeveld et al., 2002).

There are also some clear links between the results of chapter 3 and 4. Chapter 4 showed how habitat structure can have important influences on host density and host-enemy interactions. Therefore, habitat structure may indirectly influence insect immunity through its effects on host density, because variation in host density can influence insect immunological functioning (chapter 3; Wilson et al., 2002; Silva et al., 2013). This may then influence host dynamics, due to the effects of density-dependent prophylaxis (White & Wilson, 1999; Reynolds et al., 2011). However, by influencing the strength of host-enemy interactions, habitat structure may also influence the effects of direct or delayed costs of host resistance, and/or immunological priming following host defence against parasites (Stoehr, 2007; Zanchi et al., 2011; Moreau et al., 2012). Again, this may ultimately influence host dynamics (Tidbury et al., 2012).

Interestingly, a recent study by Berggren (2009) linked variation in immune functioning to habitat variables, in the form of the size of habitat patches and the amount of linear, connective landscape elements. Both were positively correlated with the strength of assayed immune responses of wild-caught bush crickets (*Metrioptera roeseli*, Hagenbach), and the findings were broadly interpreted as due to costs from dispersal and mating opportunities. Also related to the above processes was a recent study that showed that artificial activation of damselfly immune systems actually resulted in increased dispersal in the wild (Suhonen et al., 2010). Therefore, as highlighted above by the discussed links between chapters 3 and 4, in natural populations there may be complex interactions between host resistance and other ecological processes, which are themselves influenced by factors not necessarily obviously related to host resistance. Clearly potentially very complex interactions between host resistance and ecological processes can occur in natural systems, and it will require some very careful research to fully understand the way these factors interact, and the outcomes for populations.

6.3 The wider context

6.3.1 Global environmental change

If natural enemies have important regulatory roles in the population dynamics of insects (e.g. chapter 2; Myers & Cory, 2013) and other taxa (e.g. Hudson et al., 1998; Krebs et al., 2001; Korpela et al., 2014) there are implications for issues of global change ecology. In the context of this thesis, this was most explicitly explored in chapter 3, where habitat fragmentation was shown to impact host-enemy interactions in variable, scale-dependent ways, highlighting that ongoing habitat change may have implications for populations in terms of their size and dynamics. However, these changes will not be simple to predict, because of the often species- and system- specific responses to habitat fragmentation highlighted by chapter 4. As discussed above, at present this is again particularly the case for host-pathogen interactions. Although effects of spatial structure have been investigated well theoretically (e.g. Park et al., 2001, 2002; Ostfeld et al., 2005), and shown to have important effects on transmission, persistence and pathogen evolution (Boots & Sasaki, 1999, 2000; Boots et al., 2004), there has been very limited study of spatial processes in host-pathogen dynamics within natural insect systems, but see Allan et al. (2003) and Brownstein et al. (2005) for two exceptions looking at habitat fragmentation and disease. Therefore, presently there are no clear general responses of host-pathogen interactions to habitat fragmentation, unlike host-parasitoid interactions, where although there is still often substantial system-specific deviation from theoretically predicted outcomes, as shown in chapter 4, there does appear to be some general patterns in the response of parasitism to increasing fragmentation (Martinson & Fagan, 2014). Consequently, a much better understanding of the influence of habitat fragmentation on pathogens in insect populations is necessary, particularly in terms of the mechanisms by which transmission may be affected, and the responses that occur (chapter 4).

Climate change is also likely to have important influences on key host-enemy interactions, and both chapters 2 and 5 have indirect implications in this area. Climate may influence host-natural enemy interactions in important ways, as demonstrated in chapter 2 where the effect of temperature during larval development on the interactions between

the winter moth and its NPV was as strong as the effect of host density. Therefore, because climatic variables, such as temperature, can have important effects on host-parasite interactions (Thomas & Blanford, 2003), where such interactions are important, such as those between the winter moth and the magpie moth and their NPVs in the present study, climate change may lead to long-term shifts in the host-enemy dynamics (Brooks & Hoberg, 2007). In the case of cyclical insects, long-term climatic variation appears to have important effects on species' dynamics, including the frequency of outbreaks, but responses appear to often be system specific (Haynes et al., 2014). However, whether this is driven by altered host-enemy interactions is not clear.

There is also some evidence that many population cycles across a range of taxa have been collapsing in recent decades, believed to be due to the effects of climate forcing from climate change processes (Ims et al., 2008). Therefore, where species' spatio-temporal population processes are influenced by climate, as may be the case with the large-scale spatial synchrony seen in winter moth dynamics within Britain (chapter 5), changes in climate, such as the frequency of extreme climatic events, may alter these dynamics (Ims et al., 2008). Although the precise climate-related mechanisms behind the apparent loss of cycles are unclear (Ims et al., 2008), as highlighted by chapter 2, where host-enemy interactions regulate population cycles it may be that climate forcing can act through its impacts on these key biotic interactions.

6.3.2 Management implications

Large-scale spatio-temporal dynamics are evident in the populations of most cyclical species, whether it is large-scale spatial synchrony in population fluctuations or travelling waves (Peltonen et al., 2002; Sherratt & Smith, 2008; Myers & Cory, 2013). Chapter 5 showed that there is still much that is not well understood about travelling waves and their mechanisms of generation in particular. One aspect of this is the uncertainty surrounding the causes of local population cycles, given that locally cyclic dynamics are generally needed for travelling waves in models (Sherratt & Smith, 2008). However, whilst chapter 2 provided evidence consistent with specialist natural enemy driven cycles in the winter moth, this involved a pathogen, whilst models producing travelling waves incorporate mobile enemies such

as predators and parasitoids (e.g. Johnson et al., 2004, 2006; Sherratt, 2013), but have apparently not considered pathogens. Therefore, it is not clear how pathogens, dependent on their hosts for transmission, would influence the resulting spatio-temporal dynamics of such models. Given the evidence supporting pathogen driven cycles in certain species (chapter 2; Dwyer et al., 2004; Liebhold et al., 2013), this seems to be something worth investigating, as it could help explain when travelling waves occur or when they do not (Sherratt & Smith, 2008).

Understanding of the spatio-temporal dynamics of pest species like the winter moth is important for the management of habitats in which they live. Traditionally this focus has been on woodlands, forests and orchards, where pest damage from the winter moth has negative economic consequences (Stoakley, 1985; Hunter et al., 1991; Pearsall & Walde, 1994). Like elsewhere in Europe (Tenow et al., 2007; Tenow et al., 2013), within Britain the winter moth displays large-scale cyclical dynamics, with spatially synchronous population fluctuations at large scales relative to the island (chapter 5). This implies that managers may able to use information relating to long-term winter moth dynamics within their region to inform their attempts to plan and mitigate negative consequences of population peaks.

Winter moth have also been increasingly seen as a pest species of heather moorlands (Kerslake et al., 1996; Kerslake & Hartley, 1997; Vanbergen et al., 2003), which are important habitats both in conservation terms for rare and protected species such as hen harriers (*Circus cyaneus*, L.), merlin (*Falco columbarius*, L.) and short-eared owl (*Asio flammeus*, Pontoppidan) (Berry, 2000; Amar & Redpath, 2005), and in economic terms as habitat for game birds. Therefore, the cycles of winter moth on heather moorlands within Britain may have implications in terms of their creation of boom and bust food resources for predators, such as small mammals, which themselves form the primary food source for many of the birds of conservation importance (Ims & Fuglei, 2005; Rydgren et al., 2007). This indicates that management of moorlands with abundant populations of winter moth should be particularly sensitive during periods when winter moth populations have crashed, for example reducing burning and/or cutting of heather, because any insect predators may suffer population declines when their main prey is largely absent. In addition, large-scale outbreaks of winter moth can lead to extensive defoliation of heather plants (Kerslake et al., 1996), the shoots of which form a key part of the diet of the economically important game bird the red grouse. Therefore, now it is clear

that winter moth populations cycle on heather moorlands (chapter 2), like winter moth populations in European deciduous forests (Hogstad, 2005), management of moorlands that have suffered from winter moth defoliation previously may benefit from monitoring their populations in order to predict when peaks are likely to occur, allowing them to apply mitigation measures.

Efforts to control natural systems with biocontrol agents have met with mixed success (Myers, 1988; Hudson et al., 1998), although much better outcomes have been achieved in agricultural systems (Wu, 2010). However, both parasitoids and pathogens are used as biocontrol agents in natural systems such as agroforestry (Myers, 1988), and it is crucial to understand how host-natural enemy dynamics function in such systems to enable the effective use of these tools, otherwise worse outcomes can actually occur from misapplied biocontrol efforts than if no control measures are attempted (Reilly & Elderd, 2014). Consequently, studies like that presented in chapter 2 can provide valuable information for testing the assumptions of models used to predict biocontrol outcomes (Reilly & Elderd, 2014). The fact that populations of the same species may be regulated by fundamentally different natural enemies in different habitats and areas (chapter 2; Klemola et al., 2014) also highlights how important it is to understand the effects of variation in local conditions. When existing host-enemy systems are invaded by different natural enemies, alternative dynamics may result (Begon et al., 1996; Sait et al., 2000). Therefore, predicting management outcomes from models that assume the proposed biocontrol agent is also functionally similar to the agent of natural regulation may provide incorrect conclusions for managers.

Chapter 4 also has implications for the management of natural systems, highlighting how attempts to regulate pests via their interactions with natural enemies may be influenced by the spatial structure of the habitat. In harvested agroforestry systems patterns of harvesting may influence the results of biocontrol, by affecting host-enemy interactions involving biocontrol agents (With et al., 2002). As discussed in chapter 4, the effects of habitat structure on host-enemy interactions are likely to be species specific, and so detailed information on the system being managed is again likely to be very important. In particular, detailed natural history and life-history information on the key species may be crucial for predicting responses, when more general characteristics, such as trophic position and breadth, can sometimes be poor predictors (chapter 4).

6.4 Future directions

6.5 Population regulation and experimental field tests

Unfortunately, long-term and large-scale insect population studies in ecology are inherently logistically difficult, expensive and difficult to obtain funding for, given the length of time before the primary outcomes can be achieved. In addition, they rarely contain experimental controls, but see Klemola et al. (2014) for an exception. Consequently, much of this type of data comes from monitoring programs not directly designed to understand ecological processes (e.g. Dwyer et al., 2004; Haynes et al., 2009). However, although rare, such studies fulfil a very important and necessary role by providing data from temporal and spatial scales that enables the generation and/or testing of hypotheses about population regulation in real systems. It is therefore crucial that such studies are well designed in order to maximise the results generated.

To really rigorously test both the natural enemy hypothesis and other competing hypotheses about the causes of population cycles in insects, much progress could be made with a well designed, multiyear, large-scale experimental study. An excellent model study that could be scaled up and adapted is the four-year parasitoid experimental exclosure experiment of Klemola et al. (2010). However, a more wide ranging study could also experimentally test the effects of pathogens and plant defences on population regulation in addition to parasitism, through experimental treatments utilising plants grown under pest and virus free conditions, which are regularly replaced throughout the trial to prevent impacts of induced plant defences and environmentally transmitted pathogens on host populations. Vertically transmitted pathogen dynamics could also be assessed in subsets of the experimental and control populations, to explore their importance for population dynamics. The study could also be expanded spatially using replicates across a landscape, testing regulation at spatial scales relevant to the large-scale processes in cyclical species' dynamics.

6.6 The importance of dispersal

One key theme that emerged from chapters 2, 4 and 5 that was not explicitly investigated was the importance of dispersal, and the lack of current empirical data in insect populations. Dispersal is believed to be crucial for generating local-scale population synchrony in cyclical populations, although its importance for large-scale population synchrony is not clear (Kendall et al., 2000; Abbott & Dwyer, 2008; Myers & Cory, 2013). Similarly, it is a key component of models generating travelling waves in population abundance (Johnson et al., 2004, 2006; Sherratt & Smith, 2008). Dispersal is also a key factor determining species responses (and the responses of their biotic interactions) to habitat structure (chapter 4; Fahrig, 2001; Tischendorf et al., 2003). However, in studies of insect population spatio-temporal dynamics, dispersal is very rarely explicitly quantified (e.g. Zabel & Tscharntke, 1998; Bjornstad et al., 2002; Brownstein et al., 2005; Jepsen et al., 2009), but see Elzinga et al. (2007) for a rare exception. Therefore, there is a real need for improved understanding about the role and influence that insects' dispersal abilities have on population functioning within natural systems. This information could then be used to test hypotheses. For example, such information could be used to compare the relative influence of dispersal on population synchrony, compared to other potential driving factor such as Moran effects (chapter 2 and 5; Hagen et al., 2008). It could also be used to determine how habitat structure influences host-enemy interactions, allowing a clearer understanding of the mechanistic basis for the responses of host-enemy interactions in terms of the relative importance of changes in host density and host movement (chapter 4; Langlois et al., 2001). Similarly, this information would help to determine what role dispersal of hosts and enemies play in travelling wave behaviour in populations (chapter 5; Bjornstad et al., 2002; Sherratt, 2013).

Traditional field methods, primarily mark-release-recapture (Slade et al., 2013), can be used to estimate species' dispersal abilities, but rely on sufficient recapture rates, and are therefore logistically challenging and potentially fallible. In addition, they cannot define the sources and destinations of individuals without a typically large increase in the logistical effort. However, by measuring the spatial genetic structure of populations at multiple sites the frequency and spatial scale of dispersal can be estimated, as well as the directional movements of individuals between sites (Broquet & Petit, 2009). Consequently, such methods offer an excellent way in which

to indirectly infer much about patterns of dispersal. Recently, James et al. (2015) applied these techniques in combination with detailed local ecological knowledge about the spatio-temporal population dynamics of the cyclical spruce budworm (*Choristoneura fumiferana*, Clemens), and were able to show that outbreaking local populations did indeed act as major sources for surrounding sink populations at a landscape scale, explicitly linking dispersal to spatially correlated cyclical dynamics in an empirical study for the first time. Clearly, such techniques present promising avenues for future empirical research linking dispersal with population processes, also informing more biologically explicit mechanistic models.

6.7 Covert and sublethal vertically transmitted pathogens

As touched on in chapters 2 and 4, although not explicitly investigated in this thesis vertically transmitted covert/sublethal pathogen infections should be given more consideration in future studies of host-pathogen interactions, particularly those involving cyclical insects. It appears that they are common within natural insect populations (Vilaplana et al., 2008; Vilaplana et al., 2010; Kemp et al., 2011), but long-term empirical studies are currently lacking. Theoretical work indicates that they can have important effects on host-pathogen dynamics, and may also generate cyclical dynamics, but only under certain conditions, specifically only when the susceptibility of covertly infected larvae to horizontally transmitted infections is greater than healthy (non-covertly infected) larvae (Bonsall et al., 2005). Therefore, this could help to provide part of the explanation for the lack of cyclicity in magpie moth dynamics, despite apparently strong pathogen regulation (chapter 2), although it does not explain the lack of population synchrony within Orkney local populations. As suggested by chapter 4, the spatial structure of habitat could also have important influences on patterns of horizontal and vertical transmission occurring at different spatial scales, related to the scale of movement of the relevant life stages in which each form of transmission occurs, ultimately influencing host-pathogen dynamics. In addition, given the recent history of the magpie moth's distribution in northern Scotland and Orkney, vertical transmission of AbgrNPV may have been the route by which the pathogen arrived with magpie moth populations in Orkney without any apparent time delay, as was the case for the *Aleiodes* sp. parasitoid. Such a process could be important for the success of economically and ecologically damaging invasive species as well.

Vertically transmitted covert and sublethal infections by definition allow their host to survive, and, at least in the case of sublethal infections, may negatively affect host fitness through costs of immune defence and morbidity (Sait et al., 1994; Myers et al., 2000). It also appears that increasing host density can lead to greater vertical, as well as horizontal, pathogen transmission (Vilaplana et al., 2008). Furthermore, the prevalence of overt infections in the offspring of parents infected with pathogens has been shown to be higher in solitarily rather than gregariously reared parental lepidopteran larvae (Vilaplana et al., 2008). It would therefore be interesting to link immunological functioning with ecological processes involving vertically transmitted pathogens. For example, theoretically both vertically transmitted infections and density-dependent prophylaxis can influence the stability and form of host population dynamics (White & Wilson, 1999; Boots et al., 2003; Bonsall et al., 2005; Reynolds et al., 2011), and there are likely to be important implications for host dynamics where these processes interact.

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Appendix 1. Full list of final model selection tables from the multimodel inference analysis of chapter 2

Table A1.1 AIC-based model selection table for models analysing the effect of larval density in year t and temperature during larval development in year t on the likelihood of parasitism in winter moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD + T	5	6441.777	0	0.843	0.843
Null	3	6445.136	3.358	0.157	1

LD = larval density (year t), T = temperature during early-mid larval instars in year t (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Table A1.2 AIC-based model selection table for models analysing the effect of larval density in year t-1 and temperature during larval development in year t-1 on the likelihood of parasitism in winter moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD_{-1} + T_{-1}	5	3385.520	0	0.948	0.948
Null	3	3391.333	5.813	0.052	1

LD_{-1} = larval density (year t-1), T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Table A1.3 AIC-based model selection table for models analysing the effect of larval density in year t and temperature during larval development in year t on the likelihood of OpbuNPV mortality in winter moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
Null	3	6104.149	0	0.657	0.657
LD + T	5	6105.449	1.300	0.343	1

LD = larval density (year t), T = temperature during early-mid larval instars in year t (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Table A1.4 AIC-based model selection table for models analysing the effect of larval density in year t and temperature during larval development in year t on the likelihood of parasitism in magpie moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD + T	5	1396.376	0	0.652	0.652
Null	3	1397.634	1.258	0.348	1

LD = larval density (year t), T = temperature during early-mid larval instars in year t (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Table A1.5 AIC-based model selection table for models analysing the effect of larval density in year t-1 and temperature during larval development in year t-1 on the likelihood of parasitism in magpie moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD_{-1} + T_{-1}	5	1393.627	0	0.881	0.881
Null	3	1397.634	4.007	0.119	1

LD_{-1} = larval density (year t-1), T_{-1} = temperature during early-mid larval instars in year t-1 (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Table A1.6 AIC-based model selection table for models analysing the effect of larval density in year t and temperature during larval development in year t on the likelihood of AbgrNPV mortality in magpie moth larvae.

Model	K	AIC	ΔAIC	Akaike weight	Cumulative Akaike weight
LD + T	5	4041.855	0	0.829	0.829
Null	3	4045.018	3.163	0.171	1

LD = larval density (year t), T = temperature during early-mid larval instars in year t (i.e. larval development); K = number of parameters in the model. Models are ranked by their AIC scores. Akaike weights represent the relative probability of each model, given the data, to all other models in the set.

Appendix 2. Full list of models considered in the multimodel inference analysis of chapter 3

Table A2.1. Candidate models considered in the multimodel inference analysis of encapsulation score data in chapter 3.

Model terms
Null model (intercept only)
S + W + AD
S + W + AD + L14
S + W + AD + L13
S + W + AD + L14 + L13
S + W + AD + P13
S + W + AD + P14
S + W + AD + P13 + P14
S + W + AD + V13
S + W + AD + V14
S + W + AD + V13 + V14
S + W + AD + P13 + V13
S + W + AD + P14 + V14
S + W + AD + P13 + V13 + P14 + V14
S + W + AD + L13 + P13 + V13
S + W + AD + L14 + P14 + V14
S + W + AD + L13 + P13 + V13 + L14 + P14 + V14

Variable codes represent the following: S = sex; W = pupal weight; AD = assay date; L13 = 2013 larval density; L14 = 2014 larval density; P13 = 2013 % parasitism; P14 = 2014 % parasitism; V13 = 2013 AbgrNPV mortality; V14 = 2014 AbgrNPV mortality. All models also contained site as a random factor.

Table A2.2. Candidate models considered in the multimodel inference analysis of pupal weight data.

Model terms
Null model (intercept only)
S + AD
S + AD + L13
S + AD + L14
S + AD + L14 + L13
S + AD + P13
S + AD + P14
S + AD + P13 + P14
S + AD + V13
S + AD + V14
S + AD + V13 + V14
S + AD + P13 + V13
S + AD + P14 + V14
S + AD + P13 + V13 + P14 + V14
S + AD + L13 + P13 + V13
S + AD + L14 + P14 + V14
S + AD + L13 + P13 + V13 + L14 + P14 + V14

Variable codes represent following: S = sex; W = pupal weight; AD = assay date (smoothed effect); L13 = 2013 larval density; L14 = 2014 larval density; P13 = 2013 % parasitism; P14 = 2014 % parasitism; V13 = 2013 % AbgrNPV mortality; V14 = 2014 % AbgrNPV mortality. All models also contained site as a random factor.

Appendix 3. Full list of model selection tables and corresponding model-averaged parameter estimate tables from the multimodel inference analysis of chapter 4

Table A3.1 Results of the AICc-based selection of models explaining host density in terms of the overall effects of habitat isolation at different spatial scales, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	5	0	0.731
2*						+	+	+	5	2.2	0.24
3					+		+	+	5	8.3	0.012
4		+					+	+	5	9.1	0.008
5				+			+	+	5	10.1	0.005
6	+								3	11.8	0.002
7						+			3	12.6	0.001
8			+				+	+	5	13.3	0.001
9		+							3	15.9	0
10					+				3	17.3	0
11†									2	17.7	0
12				+					3	17.7	0
13			+						3	18.1	0
14							+	+	4	20.6	0

* = Models retained in 95% confidence set of models. † = Null model. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.2 Results of the the AICc-based model selection for models explaining host density in terms of the effects of habitat isolation at different spatial scales, host mortality from the virus AbgrNPV, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*AbgrNPV mortality*‡	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	+	6	0	0.67
2*						+	+	+	+	6	1.8	0.267
3*					+		+	+	+	6	6.8	0.022
4	+						+			4	8.6	0.009
5		+					+	+	+	6	8.9	0.008
6				+			+	+	+	6	8.9	0.008
7						+	+			4	9.1	0.007
8			+				+	+	+	6	11.1	0.003
9		+					+			4	12.2	0.001
10							+			3	12.5	0.001
11					+		+			4	12.6	0.001
12				+			+			4	13.2	0.001
13			+				+			4	13.4	0.001
14							+	+	+	5	15.3	0
15†										2	20	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage host mortality from the virus AbgrNPV. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.3 Results of the AICc-based model selection for models explaining host density in terms of the effects of habitat isolation at different spatial scales, parasitism, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Parasitism*‡	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	+	6	0	0.638
2*						+	+	+	+	6	1.3	0.33
3					+		+	+	+	6	8.1	0.011
4						+	+			4	8.8	0.008
5		+					+	+	+	6	9.7	0.005
6				+			+	+	+	6	10.6	0.003
7	+						+			4	11.3	0.002
8			+				+	+	+	6	13.9	0.001
9					+		+			4	15	0
10		+					+			4	15.1	0
11				+			+			4	16.2	0
12†										2	16.3	0
13			+				+			4	16.8	0
14							+			3	17	0
15							+	+	+	5	20.4	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage host mortality from the parasitoid. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.4 Results of the AICc-based model selection for models explaining host density in terms of the effects of habitat isolation at different spatial scales, host mortality from the virus AbgrNPV, parasitism, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*AbgrNPV mortality*‡	*Parasitism*§	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	+	+	7	0	0.979
2						+	+	+	+	+	7	8.5	0.014
3		+					+	+	+	+	7	11.8	0.003
4					+		+	+	+	+	7	12.8	0.002
5				+			+	+	+	+	7	14	0.001
6	+						+	+			5	14.6	0.001
7			+				+	+	+	+	7	15.6	0
8						+	+	+			5	15.7	0
9							+	+			4	18.2	0
10		+					+	+			5	18.4	0
11					+		+	+			5	19.1	0
12				+			+	+			5	19.5	0
13			+				+	+			5	19.6	0
14							+	+	+	+	6	20.1	0
15†											2	24.5	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage mortality from the virus AbgrNPV. § = Site-level percentage mortality from the parasitoid. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.5 Results of the AICc-based model selection for models explaining host mortality from the virus AbgrNPV in terms of the overall effects of habitat isolation at different spatial scales, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*†									2	0	0.129
2*					+		+	+	5	0.3	0.114
3*		+					+	+	5	0.3	0.113
4*						+	+	+	5	0.7	0.092
5*				+			+	+	5	0.9	0.084
6*						+			3	1.5	0.062
7*	+						+	+	5	1.7	0.055
8*		+							3	1.7	0.055
9*	+								3	1.8	0.052
10*					+				3	1.8	0.052
11*				+					3	1.9	0.049
12*			+				+	+	5	1.9	0.049
13*			+						3	2	0.047
14							+	+	4	2	0.047

* = Models retained in 95% confidence set of models. † = Null model. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.6 Results of the AICc-based model selection for models explaining host mortality from the virus AbgrNPV in terms of the effects of habitat isolation at different spatial scales, parasitism, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Parasitism* ‡	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*						+	+			4	0	0.338
2*						+	+	+	+	6	1.8	0.136
3*					+		+			4	2.1	0.116
4*				+			+			4	3.1	0.071
5*							+			3	3.2	0.067
6*			+				+			4	3.7	0.052
7*		+					+			4	3.7	0.052
8*					+		+	+	+	6	3.9	0.049
9*	+						+			4	4.5	0.036
10*							+	+	+	5	5.3	0.024
11*				+			+	+	+	6	5.8	0.019
12		+					+	+	+	6	6.1	0.016
13			+				+	+	+	6	6.5	0.013
14	+						+	+	+	6	7.1	0.01
15†										2	15	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage host mortality from the parasitoid. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.7 Results of the AICc-based model selection for models explaining host mortality from the virus AbgrNPV in terms of the effects of habitat isolation at different spatial scales, host density, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Host density*‡	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*†										2	0	0.229
2*							+			3	1.2	0.126
3*					+		+	+	+	6	2.3	0.071
4*		+					+	+	+	6	2.4	0.07
5*						+	+	+	+	6	2.8	0.057
6*						+	+			4	2.9	0.052
7*				+			+	+	+	6	3	0.052
8*		+					+			4	3.1	0.049
9*					+		+			4	3.1	0.048
10*	+						+			4	3.2	0.047
11*				+			+			4	3.2	0.046
12*			+				+			4	3.3	0.045
13*							+	+	+	5	3.5	0.04
14*	+						+	+	+	6	3.8	0.035
15			+				+	+	+	6	4	0.032

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level host density. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.8 Results of the AICc-based model selection for models explaining host mortality from the virus AbgrNPV in terms of the effects of habitat isolation at different spatial scales, parasitism, host density, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Parasitism*‡	*Host density*§	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*						+	+	+			5	0	0.304
2*					+		+	+			5	1.7	0.127
3*						+	+	+	+	+	7	2	0.111
4*							+	+			4	2.4	0.089
5*				+			+	+			5	2.7	0.079
6*			+				+	+			5	3.3	0.058
7*		+					+	+			5	3.4	0.056
8*					+		+	+	+	+	7	3.9	0.043
9*	+						+	+			5	4	0.04
10*							+	+	+	+	6	4.4	0.033
11*				+			+	+	+	+	7	5.7	0.018
12		+					+	+	+	+	7	6	0.015
13			+				+	+	+	+	7	6.2	0.014
14	+						+	+	+	+	7	6.5	0.012
15†											2	13.1	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage host mortality from the parasitoid. § = Site-level host density. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.9 Results of the AICc-based model selection for models explaining host mortality from the parasitoid in terms of the overall effects of habitat isolation at different spatial scales, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	5	0	0.427
2*		+					+	+	5	0.1	0.396
3*			+				+	+	5	3.8	0.065
4*				+			+	+	5	4.6	0.043
5*							+	+	4	4.9	0.037
6					+		+	+	5	6.4	0.018
7						+	+	+	5	7	0.013
8†									2	15.4	0
9						+			3	15.8	0
10					+				3	15.8	0
11				+					3	16.5	0
12			+						3	16.6	0
13		+							3	17.3	0
14	+								3	17.4	0

* = Models retained in 95% confidence set of models. † = Null model. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.10 Results of the AICc-based model selection for models explaining host mortality from the parasitoid in terms of the effects of habitat isolation at different spatial scales, host mortality from the virus AbgrNPV, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*AbgrNPV mortality*‡	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	+	6	0	0.398
2*		+					+	+	+	6	0.5	0.312
3*			+				+	+	+	6	3.1	0.085
4*							+	+	+	5	3.4	0.074
5*						+	+	+	+	6	4.1	0.05
6*				+			+	+	+	6	4.5	0.042
7					+		+	+	+	6	5.3	0.028
8						+	+			4	8.8	0.005
9					+		+			4	9.8	0.003
10							+			3	11.6	0.001
11				+			+			4	12.2	0.001
12			+				+			4	12.8	0.001
13		+					+			4	13.4	0
14	+						+			4	13.6	0
15†										2	19.1	0

* = Models retained in 95% confidence set of models. † = Null model. ‡ = Site-level percentage host mortality from the virus AbgrNPV. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.11 Results of the AICc-based model selection for models explaining host mortality from the parasitoid in terms of the effects of habitat isolation at different spatial scales, host density, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*Host density*[§]	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1[*]	+						+	+	+	6	0	0.48
2[*]		+					+	+	+	6	0.3	0.421
3[*]			+				+	+	+	6	4.9	0.041
4[*]				+			+	+	+	6	5.7	0.027
5							+	+	+	5	6.8	0.016
6					+		+	+	+	6	8	0.009
7						+	+	+	+	6	8.9	0.006
8[†]										2	15.3	0
9							+			3	17.2	0
10						+	+			4	17.2	0
11					+		+			4	17.5	0
12				+			+			4	18.2	0
13			+				+			4	18.4	0
14		+					+			4	19.1	0
15	+						+			4	19.2	0

* = Models retained in 95% confidence set of models. † = Null model. § = Site-level host density. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.12 Results of the AICc-based model selection for models explaining host mortality from the parasitoid in terms of the effects of habitat isolation at different spatial scales, host mortality from the virus AbgrNPV, host density, site elevation and plant height.

Rank	*Isolation (100 m)*	*Isolation (250 m)*	*Isolation (500 m)*	*Isolation (1000 m)*	*Isolation (2500 m)*	*Isolation (5000 m)*	*AbgrNPV mortality*‡	*Host density*§	*Site elevation*	*Plant height*	K	ΔAICc	Akaike weight
1*	+						+	+	+	+	7	0	0.499
2*		+					+	+	+	+	7	0.4	0.412
3*			+				+	+	+	+	7	4.8	0.044
4							+	+	+	+	6	7	0.015
5				+			+	+	+	+	7	7	0.015
6						+	+	+	+	+	7	8.3	0.008
7					+		+	+	+	+	7	9.1	0.005
8						+	+	+			5	13.3	0.001
9					+		+	+			5	14.1	0
10							+	+			4	15.9	0
11				+			+	+			5	16.6	0
12			+				+	+			5	17.1	0
13		+					+	+			5	17.7	0
14	+						+	+			5	17.9	0
15†											2	21.4	0

* = Models retained in 95% confidence set of models. † = null model. ‡ = Site-level percentage host mortality from the virus AbgrNPV. § = Site-level host density. + = variable included in the model. K = number of parameters in the model. Models are ranked based on the difference in AICc values (ΔAICc) between a given model and the best model in the set. Akaike weights represent the probability of each model given the data, relative to all other models in the set.

Table A3.13 Model-averaged results from the 95% confidence set of negative binomial GLMMs explaining host density in terms of possible overall effects of habitat isolation (without controlling for the effects of any other species in the community) at different spatial scales, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.55	0.11	-0.77	-0.32
Isolation 5000 m	-0.45	0.10	-0.65	-0.26
Elevation	-0.40	0.10	-0.61	-0.20
Plant height	-0.05	0.10	-0.26	0.15

Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.1).

Table A3.14 Model-averaged results from the 95% confidence set of negative binomial GLMMs explaining host density in terms of possible effects of habitat isolation at different spatial scales, percentage host mortality from the virus AbgrNPV, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.49	0.12	-0.72	-0.26
Isolation 2500 m	-0.4	0.12	-0.63	-0.16
Isolation 5000 m	-0.4	0.1	-0.6	-0.20
AbgrNPV mortality*	0.20	0.09	0.01	0.38
Elevation	-0.36	0.10	-0.57	-0.16
Plant height	-0.01	0.11	-0.22	0.2

* = Site-level percentage mortality from the virus AbgrNPV. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.2).

Table A3.15 Model-averaged results from the 95% confidence set of negative binomial GLMMs explaining host density in terms of possible effects of habitat isolation at different spatial scales, percentage parasitism, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.58	0.12	-0.82	-0.34
Isolation 5000 m	-0.46	0.10	-0.65	-0.27
Parasitism*	0.01	0.13	-0.25	0.28
Elevation	-0.42	0.12	-0.65	-0.18
Plant height	-0.05	0.12	-0.28	0.18

* = Site-level percentage mortality from the parasitoid. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.3).

Table A3.16 Results from the single negative binomial GLMM retained in the 95% confidence set of models (Table A3.4) explaining host density in terms of possible effects of habitat isolation at different spatial scales, percentage host-mortality from the virus AbgrNPV, percentage parasitism, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.56	0.07	-0.79	-0.33
AbgrNPV mortality*	0.36	0.11	0.14	0.58
Parasitism†	0.3	0.12	0.07	0.52
Elevation	-0.49	0.11	-0.71	-0.27
Plant height	-0.08	0.1	-0.27	0.11

* = Site-level percentage mortality from the virus AbgrNPV. † = Site-level percentage mortality from the parasitoid.

Table A3.17 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the virus AbgrNPV in terms of possible overall effects of habitat isolation (without controlling for the effects of any other species in the community) at different spatial scales, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.24	0.26	-0.74	0.27
Isolation 250 m	-0.36	0.29	-0.93	0.20
Isolation 500 m	-0.23	0.31	-0.84	0.38
Isolation 1000 m	-0.36	0.34	-1.03	0.31
Isolation 2500 m	-0.40	0.33	-1.06	0.25
Isolation 5000 m	-0.31	0.25	-0.79	0.18
Elevation	-0.46	0.27	-1.00	0.07
Plant height	-0.18	0.12	-0.42	0.06

Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.5).

Table A3.18 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the virus AbgrNPV in terms of possible effects of habitat isolation at different spatial scales, percentage parasitism, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.13	0.15	-0.42	0.16
Isolation 250 m	-0.17	0.14	-0.45	0.10
Isolation 500 m	-0.18	0.14	-0.46	0.10
Isolation 1000 m	-0.22	0.16	-0.54	0.10
Isolation 2500 m	-0.28	0.17	-0.62	0.06
Isolation 5000 m	-0.32	0.14	-0.58	-0.05
Parasitism*	-0.72	0.17	-1.05	-0.39
Elevation	-0.12	0.21	-0.53	0.30
Plant height	-0.14	0.11	-0.36	0.08

* = Site-level percentage mortality from the parasitoid. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.6).

Table A3.19 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the virus AbgrNPV in terms of possible effects of habitat isolation at different spatial scales, host density, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.19	0.27	-0.71	0.34
Isolation 250 m	-0.32	0.31	-0.92	0.28
Isolation 500 m	-0.01	0.19	-0.39	0.36
Isolation 1000 m	-0.30	0.35	-0.99	0.39
Isolation 2500 m	-0.35	0.35	-1.03	0.33
Isolation 5000 m	-0.27	0.27	-0.80	0.26
Host density*	0.10	0.19	-0.28	0.47
Elevation	-0.42	0.30	-1.01	0.17
Plant height	-0.17	0.12	-0.41	0.07

* = Site-level host density. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.7).

Table A3.20 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the virus AbgrNPV in terms of possible effects of habitat isolation at different spatial scales, percentage parasitism, host density, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	-0.10	0.15	-0.40	0.19
Isolation 250 m	-0.15	0.14	-0.44	0.13
Isolation 500 m	-0.16	0.14	-0.44	0.12
Isolation 1000 m	-0.20	0.16	-0.52	0.13
Isolation 2500 m	-0.26	0.17	-0.60	0.08
Isolation 5000 m	-0.31	0.14	-0.59	-0.02
Parasitism*	-0.73	0.17	-1.06	-0.39
Host density †	0.07	0.13	-0.18	0.32
Elevation	-0.09	0.24	-0.55	0.37
Plant height	-0.14	0.11	-0.36	0.08

* = Site-level percentage host mortality from the parasitoid. † = Site-level host density. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.8).

Table A3.21 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the parasitoid in terms of possible overall effects of habitat isolation (without controlling for the effects of any other species in the community) at different spatial scales, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	1.27	0.49	0.31	2.22
Isolation 250 m	1.25	0.48	0.30	2.19
Isolation 500 m	0.98	0.55	-0.10	2.06
Isolation 1000 m	0.88	0.57	-0.25	2.01
Elevation	1.40	0.50	0.42	2.38
Plant height	0.71	0.21	0.30	1.13

Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.9).

Table A3.22 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the parasitoid in terms of possible effects of habitat isolation at different spatial scales, percentage host-mortality from the virus AbgrNPV, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	0.97	0.42	0.14	1.80
Isolation 250 m	0.92	0.42	0.08	1.75
Isolation 500 m	0.69	0.45	-0.21	1.58
Isolation 1000 m	0.47	0.49	-0.50	1.44
Isolation 5000 m	-0.45	0.37	-1.17	0.28
AbgrNPV mortality*	-0.80	0.31	-1.41	-0.19
Elevation	0.93	0.47	0.00	1.86
Plant height	0.64	0.21	0.22	1.05

* = Site-level percentage host mortality from the virus AbgrNPV. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.10).

Table A3.23 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the parasitoid in terms of possible effects of habitat isolation at different spatial scales, host density, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	1.50	0.51	0.50	2.50
Isolation 250 m	1.43	0.48	0.50	2.37
Isolation 500 m	1.13	0.56	0.03	2.23
Isolation 1000 m	1.07	0.60	-0.11	2.25
Host density*	0.40	0.29	-0.17	0.97
Elevation	1.63	0.49	0.67	2.59
Plant height	0.76	0.22	0.33	1.18

* = Site-level host density. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.11).

Table A3.24 Model-averaged results from the 95% confidence set of binomial GLMMs explaining host mortality from the parasitoid in terms of possible effects of habitat isolation at different spatial scales, host mortality from the virus AbgrNPV, host density, site elevation and plant height.

Predictor variable	Parameter estimate	Standard error	Lower 95% C.I.	Upper 95% C.I.
Isolation 100 m	1.22	0.39	0.44	2
Isolation 250 m	1.10	0.34	0.43	1.77
Isolation 500 m	0.87	0.41	0.06	1.68
AbgrNPV mortality*	-0.90	0.27	-1.44	-0.36
Host density†	0.48	0.20	0.09	0.87
Elevation	1.21	0.36	0.51	1.92
Plant height	0.67	0.21	0.25	1.09

* = Site-level AbgrNPV mortality. † = Site-level host density. Model-averaged parameter estimates were calculated using the natural-average method, with unconditional standard errors and 95% confidence intervals (C.I.), based on the 95% confidence set of models selected from the full model set (Table A3.12).