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

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Abstract

Context

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.

Objectives

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.

Methods

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.

Results

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.

Conclusions

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.

Key-words: biotic interactions • habitat connectivity • habitat loss • host-parasite • host-parasitoid • host-pathogen • landscape epidemiology • natural enemies • species interactions

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 (Ewers and Didham 2006; Fahrig 2003). 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 and 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 and 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 (Martinson and Fagan 2014; McCallum 2008). 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 (Graham et al. 2004; Hawkins et al. 1997). They can have important regulatory influences on their host’s population dynamics (Bonsall 2004; Hassell 2000), and are thought to play a key role in the cyclical outbreaking dynamics of many insect pests (Myers and 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 and Tscharntke 1994, 2000; Martinson and Fagan 2014), studies also reveal both positive and neutral responses to habitat fragmentation by higher trophic levels (e.g. Brückmann et al. 2011; Doak 2000; 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 (Cagnolo et al. 2009; Holt et al. 1999; van Nouhuys 2005). Trophic specialists are predicted to be especially vulnerable to habitat fragmentation, because it can separate them from their prey (Davies et al. 2004), 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 and 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 and 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

(Allan et al. 2003; Langlois et al. 2001; 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 (Martinson and Fagan 2014; McCallum 2008).

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 and 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 and 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 an insect host, the magpie moth (*Abraxas grossulariata*, Lepidoptera: Geometridae), which is not typically considered a pest species. However, on Mainland Orkney there are widespread populations of magpie moth that suffer substantial larval mortality from a pathogen and a parasitoid, making it an ideal model system for exploring these questions. The parasitoid wasp is a specialist, solitary, koinobiont *Aleiodes* (Hymenoptera: Braconidae) sp. (which will be formally named by C. van Achterberg & M.R. Shaw, unpublished data), with no known alternative hosts (C. van Achterberg & M.R. Shaw, unpublished data); whilst the pathogen is a specialist nucleopolyhedrovirus (NPV), AbgrNPV (Harold 2009). NPVs are horizontally transmitted when larvae consume foliage contaminated with infectious NPV virions (Cory and Myers 2003). However, as a mechanism for the pathogen 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 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 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 and 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 and May 1981), and also because of reduced inter-population vertical transmission of the virus by adults in more isolated populations.

Methods

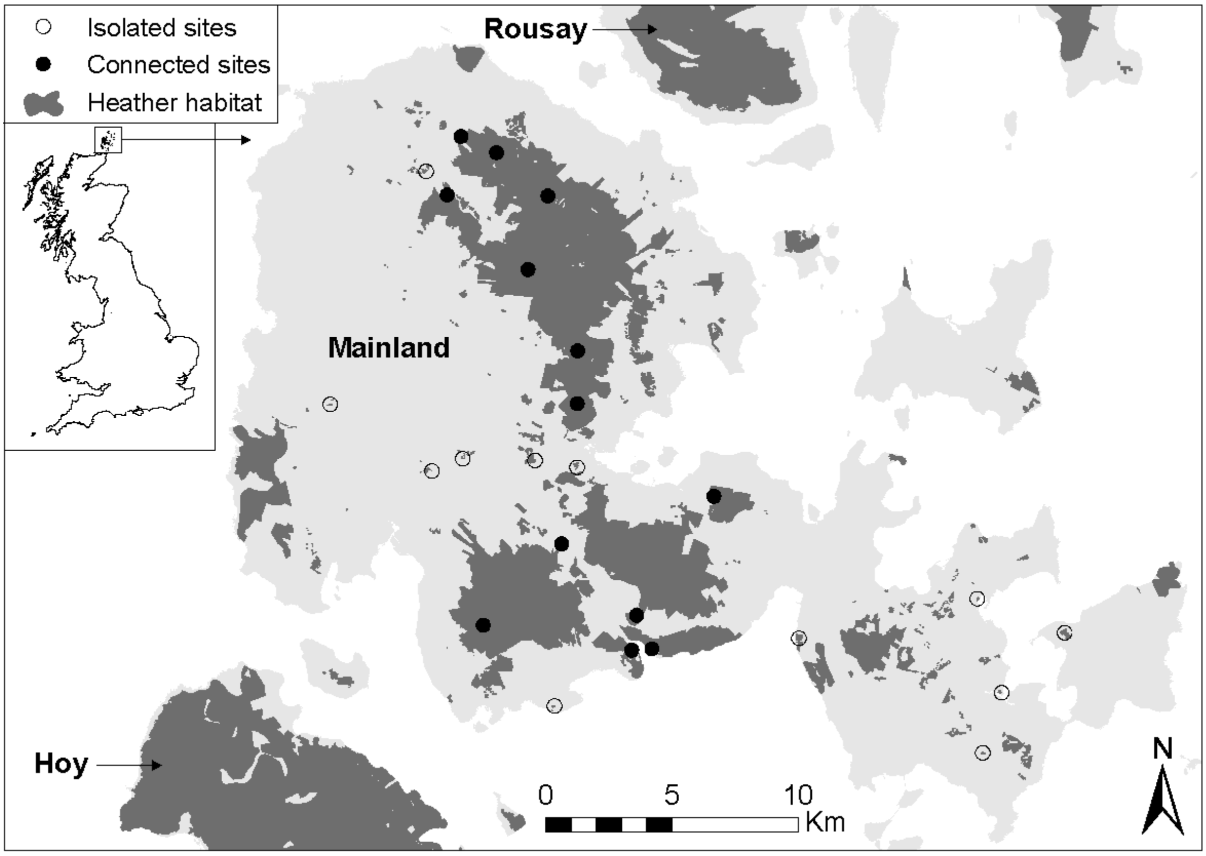
Study area and species

Field work was conducted between 28.05.2012 and 20.06.2012 on Mainland Orkney, the largest of the Orkney Islands (523 km^2), which are situated off the north-east coast of Scotland (Fig. 1). On Mainland Orkney larvae of the magpie moth are found feeding widely on heather (Calluna vulgaris), 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. 1). Magpie moth larvae are polyphagous, and feed on a number of shrubs and trees including Ribes spp., blackthorn (Prunus spinosa) and hazel (Corylus avellana) (Allan 1979). However, 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 1 approximately here#

Fig. 1. Location of the Orkney Islands, Mainland Orkney, the sampling sites on Mainland Orkney and the distribution of heather habitat across Mainland Orkney.

185


○ Isolated sites
● Connected sites
Heather habitat
Rousay
Mainland
Hoy
0
5
10
Km
N

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. 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. 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).

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. 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 and Coulson 1998), whilst parasitoid foraging efficiency and NPV persistence are also affected by plant height and structure (Obermaier et al. 2008; Raymond and Hails 2007). 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 ESRI® ArcGIS™ 10.0 (ESRI 2011) and Ordnance Survey Land-Form Profile Digital Terrain Model data (10 m^2 resolution) (OS, 2003).

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 multimodel inference process (Burnham and 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 S1; Table S5; Table S9). 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 S1-S12). 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 no longer significant 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 still explained a significant amount of variation 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 Team 2010), 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. 2012).

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 and 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 and Anderson 2002). Parameter estimates were considered to show evidence for statistically significant effects if their 95% confidence intervals excluded the null-effect value. 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 and Falck 2001).

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 1 approximately here#

Table 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 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 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 S4 & Table S16). 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. 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 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 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 S4), 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 S16).

#Table 2 approximately here#

Table 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	0.61	0.56	0.57‡
	(0.46, 0.72)	(0.49, 0.77)	(0.44, 0.71)	(0.45, 0.72)
2500		0.67		
		(0.53, 0.85)		
5000	0.64	0.67	0.63	
	(0.52, 0.77)	(0.55, 0.82)	(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 multimodel inference (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, and 95% confidence intervals excluding 1 indicate a significant 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 supplementary material S1 & S2).

#Figure 2 approximately here#

Fig. 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 proportion of 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.

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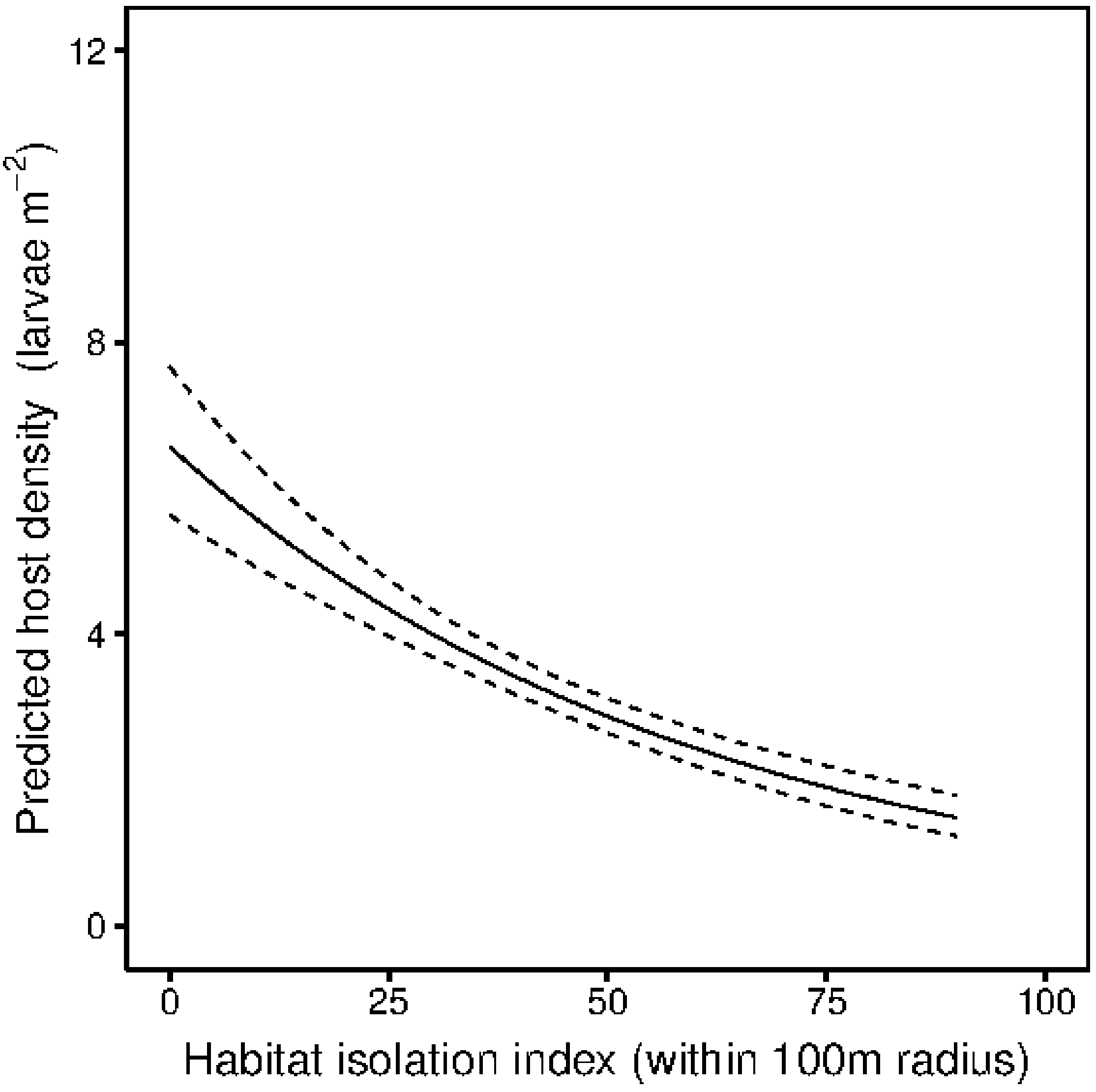

Predicted host density (larvae m^{-2})
Habitat isolation index (within 100m radius)
0
4
8
12
0
25
50
75
100

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 1). However, there was also less relative variation in site-level percentage mortality due to the virus AbgrNPV than the parasitoid (Table 1). Site-level percentage mortality due to the virus AbgrNPV was also slightly higher in connected sites than in isolated sites (Table 1), but the analysis indicated no significant overall effects of habitat isolation at any scale on the likelihood of mortality from the virus AbgrNPV (Table 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 a marginally significant, weak, negative effect of habitat isolation at 5000 m on the likelihood of mortality from the virus AbgrNPV (Table 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. 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 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 not significant, 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 S20).

#Table 3 approximately here#

Table 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.

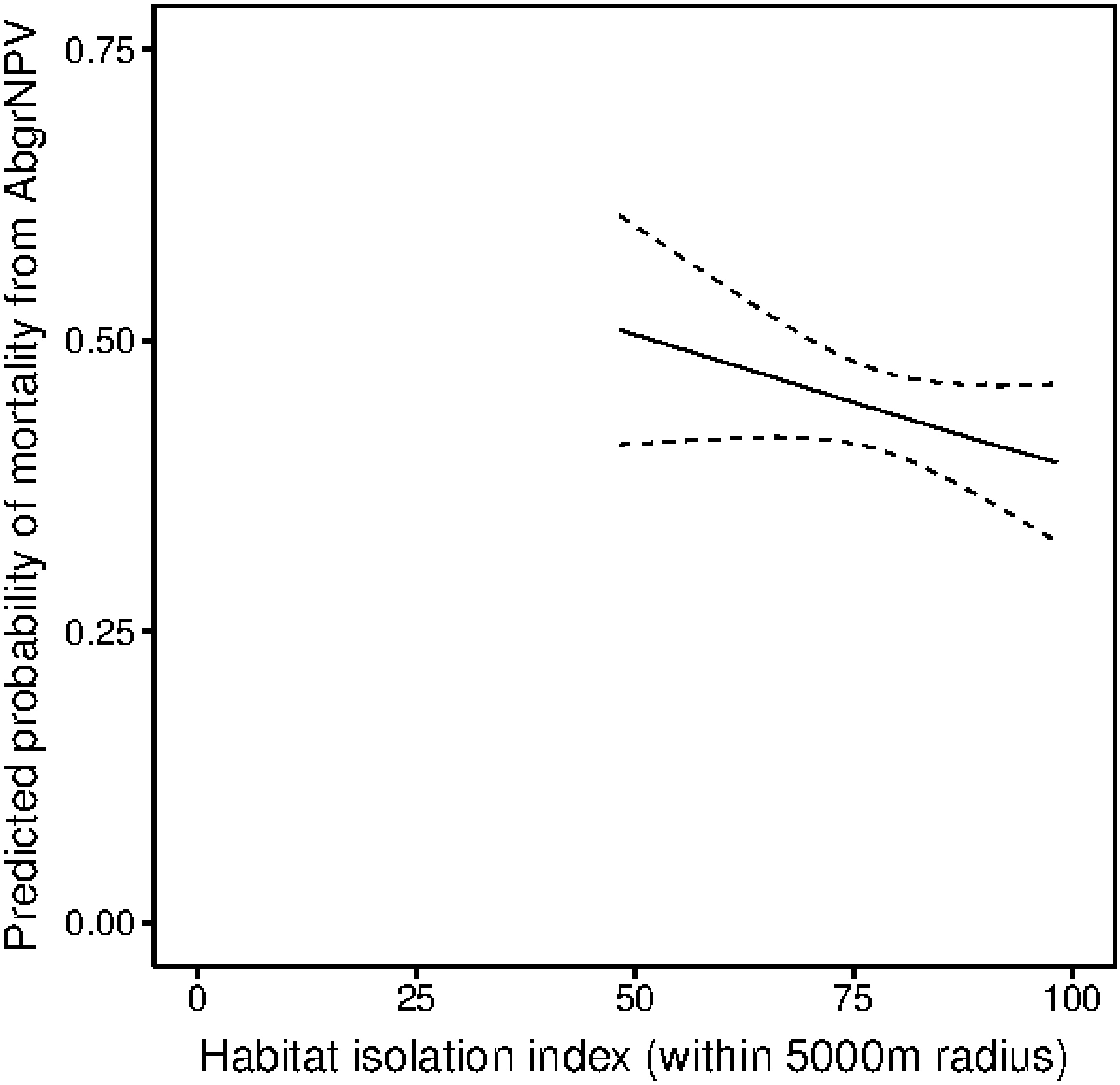
Species effects controlled for	None	Parasitoid*	Host density†	Parasitoid + host density
Spatial scale of habitat isolation (m)				
100	0.79	0.88	0.83	0.9
	(0.48, 1.31)	(0.66, 1.17)	(0.49, 1.4)	(0.67, 1.21)
250	0.69	0.84	0.73	0.86
	(0.4, 1.22)	(0.64, 1.1)	(0.4, 1.33)	(0.65, 1.14)
500	0.79	0.84	0.99	0.85
	(0.43, 1.45)	(0.63, 1.1)	(0.68, 1.43)	(0.64, 1.13)
1000	0.70	0.8	0.74	0.82
	(0.36, 1.36)	(0.58, 1.1)	(0.37, 1.48)	(0.6, 1.13)
2500	0.67	0.75	0.71	0.77
	(0.35, 1.28)	(0.54, 1.06)	(0.36, 1.39)	(0.55, 1.08)
5000	0.74	0.73	0.76	0.74
	(0.45, 1.19)	(0.56, 0.95)	(0.45, 1.29)	(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 multimodel inference (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, and 95% confidence intervals excluding 1 indicate a significant effect. The MMI process for all results also controlled for the effects of site elevation and plant height (see supplementary material S1 & S2).

#Figure 3 approximately here#

Fig. 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 proportion of 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 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.


Predicted probability of mortality from AbgrNPV
0.75
0.50
0.25
0.00
0
25
50
75
100
Habitat isolation index (within 5000m radius)

Parasitism varied substantially between all sites, and there were generally higher levels of parasitism in connected sites (Table 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) 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 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). 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), 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 & Fig. 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 S24).

#Table 4 approximately here#

Table 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.

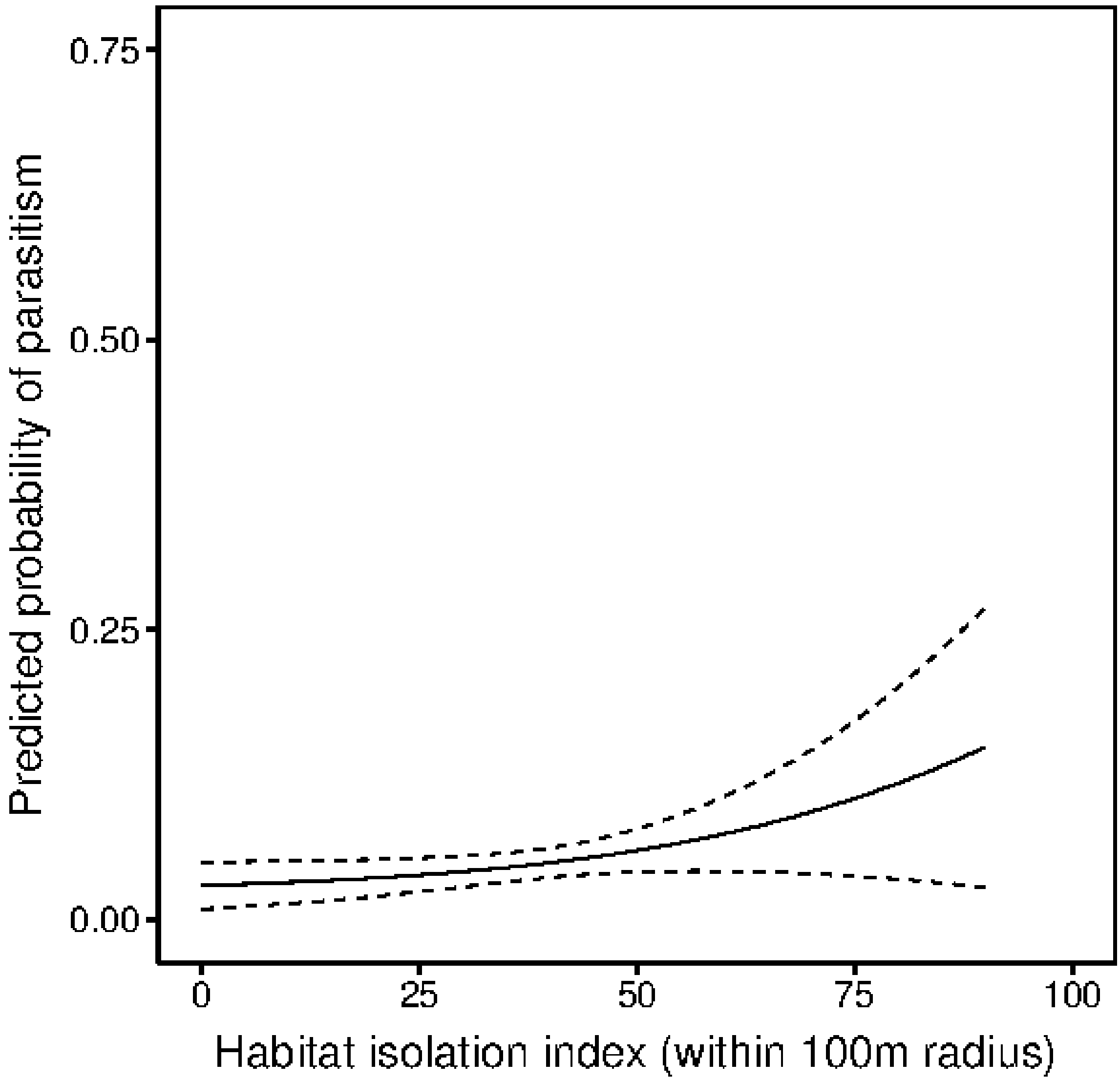
Species effects controlled for	None	AbgrNPV virus*	Host density†	AbgrNPV virus + host density
Spatial scale of habitat isolation (m)				
100	3.55	2.64	4.48	3.39
	(1.37, 9.19)	(1.15, 6.03)	(1.65, 12.17)	(1.56, 7.34)
250	3.48	2.51	4.18	3.01
	(1.36, 8.9)	(1.09, 5.75)	(1.65, 10.62)	(0.55, 5.85)
500	2.67	1.99	3.09	2.39
	(0.91, 7.82)	(0.81, 4.84)	(1.03, 9.23)	(1.07, 5.36)
1000	2.42	1.6	2.92	
	(0.79, 7.45)	(0.61, 4.2)	(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 multimodel inference (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, and 95% confidence intervals excluding 1 indicate a significant 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 supplementary material S1 & S2).

#Figure 4 approximately here#

Fig. 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 proportion of 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.


0.75
0.50
0.25
0.00
Predicted probability of parasitism
0
25
50
75
100
Habitat isolation index (within 100m radius)

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 (Holt et al. 1999; Kruess and Tscharntke 1994, 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 and 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 and 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 and Chew 1994), which scale strongly with habitat area (Hambäck and Englund 2005; Hambäck et al. 2007). Consistent with this hypothesis, butterflies instead locate suitable habitat for oviposition primarily using visual cues (Renwick and Chew 1994), which scale much less with habitat area (Hambäck and 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 1994), and this has been demonstrated by experimental and observational studies (Gonzalez et al. 1998; Hanski and Thomas 1994). 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, but it is a relatively large bodied species, and so may be expected to be a relatively strong disperser (Sekar 2012). 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 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 and 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 (Hambäck et al. 2007; Steffan-Dewenter and Tscharntke 2000). 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 significant when at least controlling for competition with the parasitoid. When controlling for the other species in the community, the effects of habitat isolation on the likelihood of mortality from the virus AbgrNPV were all non-significant at spatial scales smaller than 5000 m, and declined with decreasing spatial scale. Therefore, the significant 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 (Sekar 2012; Tscharntke and Brandl 2004), ultimately leading to reduced larval mortality from emergent overt infections in more isolated populations (Boots et al. 2003; Burden et al. 2002; 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.

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 (Chaplin-Kramer et al. 2011; Tscharntke and Brandl 2004). 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 and 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 and Strong 1999). Insects also commonly exhibit negative responses to the edges of suitable habitat patches (Ewers and 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 and Tscharntke 1994, 2000), the typically negative responses of parasitoids to increasing habitat fragmentation (Martinson and 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 (Brückmann et al. 2011; Doak 2000; Elzinga et al. 2007), 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 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.

Conclusions

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 and Fagan 2014). Thus, despite some clear patterns being evident in the general responses of host-parasitoid interactions to habitat fragmentation (Martinson and 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 (Martinson and Fagan 2014; McCallum 2008), despite the increasing recognition of the importance of pathogens for host population dynamics (Bonsall 2004; Myers and Cory 2013). The proposed mechanism underlying the observed results in this study, involving adult vertical transmission of covert infections, highlights the 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 enable general and specific predictions to made.

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