

## Research

### Can hyperparasitoids cause large-scale outbreaks of insect herbivores?

Hedvig K. Nenzén, Véronique Martel and Dominique Gravel

H. K. Nenzén (<http://orcid.org/0000-0002-0189-4283>) ([hedvig.nenzen@gmail.com](mailto:hedvig.nenzen@gmail.com)), and D. Gravel (<http://orcid.org/0000-0002-4498-7076>), *Dépt de biologie, Univ. de Sherbrooke, Sherbrooke, QC, Canada. HKN also at: Dépt des sciences biologiques, Univ. du Québec à Montréal, PO Box 8888, Stn. Centre-Ville, Montréal, QC, H3C 3P8, Canada.* – V. Martel, *Natural Resources Canada, Canadian Forest Service, Laurentian Forestry Centre, Stn Ste-Foy, Québec, QC, Canada.*

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Synchronous population fluctuations occur in many species and have large economic impacts, but remain poorly understood. Dispersal, climate and natural enemies have been hypothesized to cause synchronous population fluctuations across large areas. For example, insect herbivores cause extensive forest defoliation and have many natural enemies, such as parasitoids, that may cause landscape-scale changes in density. Between outbreaks, parasitoid-caused mortality of hosts/herbivores is high, but it drops substantially during outbreak episodes. Because of their essential role in regulating herbivore populations, we need to include parasitoids in spatial modelling approaches to more effectively manage insect defoliation. However, classic host-parasitoid population models predict parasitoid density, and parasitoid density is difficult to relate to host-level observations of parasitoid-caused mortality. We constructed a novel model to study how parasitoids affect insect outbreaks at the landscape scale. The model represents metacommunity dynamics, in which herbivore regulation, colonisation and extinction are driven by interactions with the forest, primary parasitoids and hyperparasitoids. The model suggests that parasitoid spatial dynamics can produce landscape-scale outbreaks. Our results propose the testable prediction that hyperparasitoid prevalence should increase just before the onset of an outbreak because of hyperparasitoid overexploitation. If verified empirically, hyperparasitoid distribution could provide a biotic indicator that an outbreak will occur.

Keywords: insects, parasitoids, outbreaks, interactions, modeling

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#### Introduction

The causes of synchronous population dynamics, such as outbreaks of insect defoliators, are an enduring ‘ecological mystery’ (Elton 1924), with large consequences for ecosystems. Insect outbreaks lead to high population density and defoliation over large areas (Myers and Cory 2013). Three main mechanisms that synchronize large-scale outbreaks have been proposed (Liebhold et al. 2004, Berryman 1996, Cooke et al. 2007). First, dispersal synchronizes defoliation when individuals spread

and initiate defoliation throughout the landscape. Second, climate variation that is spatially autocorrelated influences the dynamics of isolated populations in similar ways, thus synchronizing their fluctuations. Finally, synchronous fluctuations of natural enemies may produce large-scale outbreaks by modifying species interactions and dispersal (Haynes et al. 2009, Tenow et al. 2012). For defoliating insects, parasitoids are effective natural enemies that often kill more individuals than predators and parasites combined (Hawkins et al. 1997). The relative importance of these three mechanisms for outbreak occurrence is unclear. Yet, improving our understanding of these mechanisms is necessary to anticipate their occurrence and eventually develop management programs that reduce devastation in economically-important forests.

Parasitoids regulate the density of their host (Hawkins et al. 1997), suggesting that the sudden eruption of herbivore density may result from the escape from ‘parasitoid control’. Such loss of control has been observed for various herbivore species (Rosenheim 2001), yet the reasons for it are unknown (Tylianakis and Romo 2010, Myers and Cory 2013). The loss of parasitoid control may be the result of a dilution of parasitoids following a sudden increase in herbivore populations (bottom-up), or it may be the result of a decrease in parasitoid density (top-down). Parasitoids are able to control herbivore population density effectively due to their unique trophic and reproduction lifestyle (Godfray and Shimada 1999, Henri and Van Veen 2011). Primary parasitoids lay their eggs on or inside host eggs, larvae or adults, and parasitoid larvae feeding on hosts eventually causes their death (Eggleton and Gaston 1990). As each successful parasitoid attack simultaneously increases parasitoid growth rates and causes host death, host–parasitoid interactions are highly over-exploitative (Lafferty and Kuris 2002). Theoretical models of host–parasitoid population density generally predict this locally unstable relationship and generate population oscillations of increasing amplitude that can lead to the extinction of both species (Nicholson and Bailey 1935, Mills and Getz 1996). Biological pest management relies on effective parasitoid control (Murdoch et al. 2005), yet traditional models cannot explain why parasitoid control fails or succeeds (Murdoch et al. 2003), and our inability to understand it leads to ‘ecological surprises.’

It has been suggested that successful parasitoid control is inhibited because parasitoids are themselves hosts to other parasitoid species (hyperparasitoids) that regulate their density (Sullivan and Wolfgang 1999, Frago 2016). These hyperparasitoids could therefore indirectly release herbivores by depressing parasitoids (Rosenheim 1998). Parasitoids provide an ecosystem service by controlling herbivorous insects while hyperparasitoids (parasitoids exclusively attacking other parasitoids) may provide an ‘ecosystem disservice’ (Gagic et al. 2012) by interrupting parasitoid control. Obligate hyperparasitoids can either deposit their eggs on or inside immature parasitized hosts or on or inside the immature primary parasitoid (obligate

hyperparasitoids; Müller et al. 1999). Irrespective of their exact mode of attack, all hyperparasitoid larvae cause the death of the primary parasitoid by feeding on the developing primary parasitoid (Gómez-Marco et al. 2015). As hyperparasitoids cause primary parasitoid mortality and primary parasitoids cause herbivore death, the outcome is that hyperparasitoid control also is an intrinsically unstable ecological interaction. In controlled experiments, hyperparasitoids lowered the population density of parasitoids and suppressed parasitoid control of aphids (Van Veen et al. 2001, Schooler et al. 2011). Hyperparasitoids could potentially explain why parasitoid control decreases and biological control fails, as observed during herbivore outbreaks. Traditional models present no consensus on hyperparasitoid impacts and suggest that they either decrease or increase herbivore density (Nicholson and Bailey 1935, Beddington et al. 1978, May and Hassell 1981). Therefore, to predict how hyperparasitoids affect herbivore dynamics, we need new models that include hyperparasitoids.

Multiple trophic levels of parasitoids have been observed in nature (Müller et al. 1999), and models are needed to explore whether hyperparasitoids may be responsible for the occurrence of large-scale synchronous outbreaks. For example, the spruce budworm, *Choristoneura fumiferana* (hereafter budworm, Lepidoptera: Tortricidae), has large outbreaks (Boulanger et al. 2012) and a parasitoid community with more than three trophic levels that change in density during an outbreak cycle (Eveleigh et al. 2007). At the landscape scale, dispersal between populations may stabilize host–parasitoid dynamics, as it has been shown both in nature (Kruess and Tscharntke 1994, Maron and Harrison 1997, Roland and Taylor 1997) and in the laboratory (Huffaker 1958, Bonsall and Hastings 2004). It is well known that dispersal affects persistence in predator–prey systems (McCann 2000, Gravel et al. 2011a). Conceptual models suggest that biotic interactions are an important driver of spruce budworm outbreaks (Royama 1992, Fleming 1996, Cooke et al. 2007). Quantitative models exist for interactions between insect herbivores and other organisms, such as predators, pathogens, parasitoids and parasites (Anderson and May 1980, Turchin 2002, Dwyer et al. 2004). However, the current challenge is to understand the conditions under which hyperparasitoids contribute to generating landscape-scale outbreaks in host–parasitoid systems.

Here, we present a theoretical model that explores how hyperparasitoids interrupt herbivore suppression and synchronize forest defoliators at the landscape scale. The model was inspired by the spruce budworm, a major defoliator affecting boreal forests in North America and hosting a parasitoid community composed of three trophic levels. To simplify the model, and rather than tracking population density, we constructed a trophic metacommunity model inspired by metapopulation theory (Hanski 1998, Gravel et al. 2011b). In metapopulation models, the possible state in each patch is that a species is either present or absent, i.e. high or low density (Levins 1969). This

corresponds well to herbivore density with high fluctuations between high ‘epidemic’ density, and low density. Spruce budworm density during and between outbreak periods differs by several orders of magnitude (Régnière and Nealis 2007); high density causes defoliation and low density is too low to be noticeable. Outbreaks have previously been modelled as a single-species metapopulation process by coupling defoliation maps and cellular automata models (Nenzén et al. 2018).

In this study, we added additional trophic levels to the classic metapopulation model by considering that herbivore density depends on interactions with other species, i.e. if herbivore density is high compared to parasitoid density, forest defoliation occurs (outbreaks; Fig. 1c). Conversely, we considered that if a parasitoid has a high density, the herbivore is ‘controlled’ and effectively absent (Fig. 1a). As our model includes forest defoliation and regeneration, we included a realistic resource feedback, a link that population models usually ignore (but see Ludwig et al. 1978, Turchin et al. 2003, Økland and Bjørnstad 2006). We also included hyperparasitoid dynamics (Fig. 1b) to the model based on the hyperparasitoid studies described above. We assumed that hyperparasitoid control of primary parasitoids is eventually lost due to over exploitation (Sullivan and Wolfgang 1999) and demographic stochasticity. When hyperparasitoid control is lost, primary parasitoid density low and consequently herbivore density should increase (Fig. 1b–c). At that point in time, there are few of the

parasitised herbivores that the hyperparasitoids requires to complete their life cycle (obligate hyperparasitoids can only attack hosts that are already parasitised). Therefore, it is logical that hyperparasitoid-caused parasitoid mortality should eventually lead to hyperparasitoid mortality.

We investigated whether hyperparasitoids could interrupt parasitoid control and allow herbivore populations to increase synchronously, thereby causing landscape-level eruptions. More specifically, we aimed to identify 1) which parasitoid characteristics, i.e. parameters, are necessary to produce outbreaks, 2) whether herbivore and parasitoid prevalence changes predictably during an outbreak; and 3) whether outbreaks are larger if herbivores spread with a higher probability into a hyperparasitoid controlled forest compared with a parasitoid controlled forest. We asked under which conditions (local colonisation and extinction rates, dispersal) system dynamics are stable or displaying cyclic outbreaks. Our hypothesis was that outbreaks should be larger if herbivores could spread with a higher probability into hyperparasitoid controlled forest than parasitoid controlled forest. We expected this because herbivores cannot be attacked by obligate hyperparasitoids if the primary parasitoid is absent, so herbivores are ‘immune’ to hyperparasitoid attack without the parasitoid intermediate. Finally, we investigated whether the covariation of herbivore, primary parasitoid and hyperparasitoid prevalence could be used as an indicator to anticipate the occurrence of landscape-scale outbreaks.

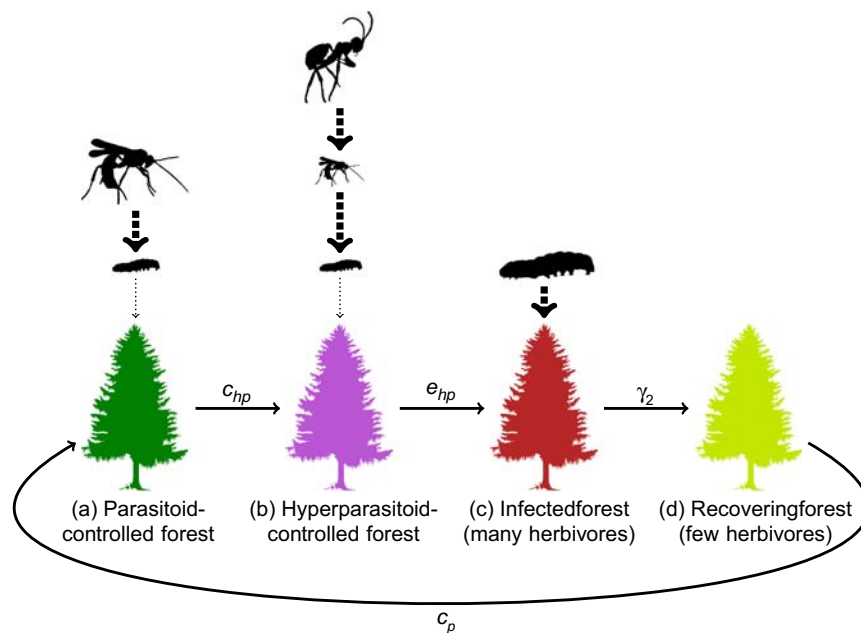


Figure 1. Effect of each trophic level and of underlying trophic levels on forest state dynamics (described further in the text). Insect size is proportional to population density (not body size). Vertical line thickness indicates the effect on the trophic level below. Horizontal arrows indicate transition between states. The forest is non-defoliated in both (a) primary parasitoid controlled forest and (b) hyperparasitoid controlled forest, and is only defoliated when the herbivore is not controlled by any parasitoid (c). After high herbivore density the forest dies and regrows (d), finally becoming parasitoid controlled again (a). Note that hyperparasitoids have a strong effect on both primary parasitoids and herbivores (thick arrows in b), in contrast to trophic relationships, where a stronger effect on one trophic level leads to a weaker effect on the next trophic level.

## Methods

### Model overview

The metacommunity model represents forest as non-defoliated or defoliated; the forest can be in only one of four possible states, which are determined by the abundance of herbivores, primary parasitoids and hyperparasitoids. We assumed that a non-defoliated forest has low herbivore density because it is controlled by either primary parasitoids or hyperparasitoids. Over time, primary parasitoids (P state) are attacked by hyperparasitoids (HP state), and attack by either parasitoids or hyperparasitoids kills the herbivores. The forest remains non-defoliated for as long as densities of parasitoids or hyperparasitoids remain high, causing herbivores to remain low. Eventually, hyperparasitoids decrease in density because they over exploit primary parasitoids. When this occurs, primary parasitoids are at low density, so herbivores can quickly increase in density and defoliate the forest (I state). After defoliation, the forest recovers (R state) and then matures to the primary parasitoid controlled state again (Fig. 2).

We based our model on observed interactions between the budworm and its parasitoids. Parasitoids strongly reduce herbivore survival rates and may inhibit defoliation (Berryman 1996, Klemola et al. 2010). Insect herbivores (i.e. defoliators) such as the budworm, are present at a very low, barely detectable, density between outbreaks (Sanders 1996, Régnière et al.

2013). During the time between outbreaks, the budworm experiences high parasitism (Royama 1992). Between outbreaks, the proportion of individuals that are killed by parasitism was observed to be somewhere between 50–90% (Seehausen et al. 2014, 2016), 40–70% (Cappuccino et al. 1998) or 80% (Cusson et al. 1998). High parasitism rates imply that parasitoid populations are larger relative to herbivore populations, providing abundant hosts to hyperparasitoids between outbreaks. The herbivore mate-finding Allee effect maintains herbivore populations at low levels (Régnière et al. 2013), and parasitoids can contribute to this by further decreasing the already low herbivore populations. As outbreaks occur with 30-year intervals, data on parasitism rates during outbreaks are scarce, but rates are likely lower (McGugan and Blais 1959, see Myers and Cory 2013 for other species). Parasitism rates are higher towards the end of an outbreak (Eveleigh et al. 2007). Régnière and Nealis (2007) measured 0.8% parasitism during outbreaks and 20% during the declining phase of outbreaks. This could be because herbivore density declines due to resource depletion and pathogens (Régnière and Nealis 2007). In other insect species, both higher parasitism rates and resource depletion could potentially cause outbreaks to terminate (Hagen et al. 2010, Schott et al. 2010). Most studies above refer only to parasitoids, and few studies have tracked hyperparasitoids under natural conditions, and especially their role at the beginning of outbreaks. Hyperparasitoids have recently been shown to attack budworm primary parasitoids at low budworm density (V. Martel unpubl.), suggesting that they are present at the beginning of outbreaks. We therefore based model dynamics on field and laboratory studies that have found support for hyperparasitoids causing outbreaks of other herbivores (e.g. aphids, Frago 2016).

### Description of states

Each patch holds a forest stand whose state is controlled by herbivores, primary parasitoids or hyperparasitoids, and stand state indicates how forest condition is affected by their abundance (boxes in Fig. 1, 2). Forest stand state is determined by population density ratios, thus indirectly representing the population fluctuations in classic Nicholson–Bailey host–parasitoid models.

Primary parasitoid controlled forest (state P) indicates regulated herbivore density (‘endemic’ low density) and a healthy forest condition (Fig. 1a). The hyperparasitoid controlled forest (state HP) has a low density of both herbivores and primary parasitoids because hyperparasitoids kill the primary parasitoids that has killed the herbivore (Fig. 1b). In both P and HP states herbivore density is regulated by primary parasitoids and hyperparasitoids, so we assumed that if herbivore density is low the forest was healthy. The infested state I, in contrast, has high herbivore density and no parasitoids (Fig. 1c). Severe defoliation causes forest stand death, and natural regeneration processes eventually lead to state R, a young recovering forest (Fig. 1d). A recovering forest can only support low herbivore density because there is little foliage.

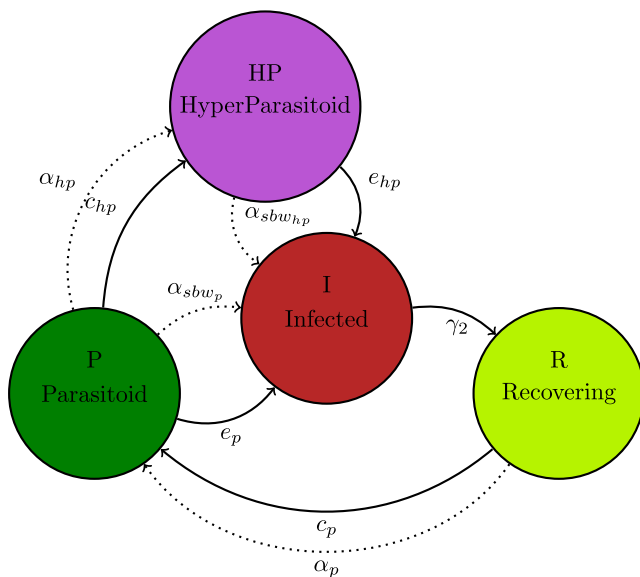


Figure 2. Schematic representation of the metacommunity model showing all the possible states (circles): infested (herbivore-controlled forest), parasitoid controlled and hyperparasitoid controlled forest. Transitions (arrows) show the parameters that control the transitions between states. Solid arrows indicate probabilities that depend on the proportion of only one state, and dotted arrows indicate probabilities that depend on the proportion of both states (law of mass action). See Table 1 for the definition of states and parameters. Note that the colours used for the states in this figure are used in all other figures.



## Description of transitions

In the Levins metapopulation model (Levins 1969), change in the presence and absence of a species occurs due to colonisation and extinction dynamics. Here, we re-interpreted the species' presence-absence dynamics as control (c) and exclusion (e) to emphasise that they occur due to biotic interactions (Fig. 2, arrows). The control and exclusion processes occur at probabilities that are proportional to their own density (law of mass action). In the P state herbivore density is low because parasitoids and thus parasitised herbivores are abundant. Therefore, hyperparasitoids can reproduce successfully and gradually come to increase in density and control primary parasitoids, with the transition from P to HP occurring at rate  $c_{hp}$ . Hyperparasitoids cause primary parasitoid mortality and primary parasitoids cause herbivore mortality so ultimately, hyperparasitoids 'cause' mortality of both herbivores and parasitoids (simultaneous strong effects on two trophic levels, Fig. 1b). Hyperparasitoids are often smaller (Henri and Van Veen 2011) and presumably weaker dispersers (Holt et al. 1999) than parasitoids. Therefore, the P to HP transition from primary parasitoid controlled to hyperparasitoid controlled forest only depends on the proportion of the P state (not on the proportion of any other state).

Parasitoid control can be unstable because parasitoids may over exploit herbivores (Lafferty and Kuris 2002). In such a situation, parasitoids may go extinct and herbivores can increase rapidly (P  $\rightarrow$  I with probability  $e_p$ ). This mechanism corresponds the classic host-parasitoid model. In a model with hyperparasitoids, the parasitoid controlled forest transitions to hyperparasitoid controlled forest state. The hyperparasitoid controlled forest state may transition to infested forest with a high herbivore density because of the co-extinction of hyperparasitoids and parasitoids due to over exploitation (HP  $\rightarrow$  I with probability  $e_{hp}$ ). When both parasitoid and hyperparasitoids go extinct, herbivores increase rapidly in abundance and defoliate forest.

Defoliation caused by high spruce budworm density can occur during 1 to up to 25 years, and tree mortality usually begins in the third to fifth year of severe defoliation (Gray 2013, MacLean 1980). The forest mortality I  $\rightarrow$  R transition rate  $\gamma$  was therefore set to 1/3. As the forest recovers, it switches to mature primary parasitoid controlled forest, R  $\rightarrow$  P, with probability  $c_p$  (Fig. 1a-d). This transition occurs because during forest recovery, parasitoids locate and attack scattered herbivore individuals, thus increasing in density and establishing dominance. We also consider that the parasitoid is present everywhere at low density, so dispersal from another stand is not required.

Once a stand has a high-density spruce budworm population, dispersal is important so that herbivores can disperse to seek food and avoid mortality (Cooke et al. 2007). Even though long-distance spruce budworm dispersal probably occurs (Greenbank et al. 1980, Nenzén et al. 2018), most dispersal occurs between neighbouring stands within 10–80 km (Bouchard and Auger 2014). Therefore, we limit dispersal to neighbouring stands ( $\alpha$ ). Dispersal causes parasitoid

controlled-forest adjacent to an infestation to receive so much dispersing individuals that parasitoids become overwhelmed and the stand becomes infested (HP  $\rightarrow$  I and P  $\rightarrow$  I). We hypothesize that herbivore dispersal can occur into both primary parasitoid and hyperparasitoid controlled forests, but at higher rates into hyperparasitoid controlled forest (HP  $\rightarrow$  I with probability  $\alpha_{sbwhp}$ , mass action). The basis for this hypothesis is that the budworm experiences no mortality in hyperparasitoid controlled stands because the parasitoid density is low. Also, the budworm cannot be attacked by the hyperparasitoid, which cannot lay eggs in the budworm without the primary parasitoid as an intermediate. Herbivore dispersal could also take place into parasitoid controlled forests because herbivore density is several orders of magnitude higher in infested states (P  $\rightarrow$  I dispersal transition with probability  $\alpha_{sbwp}$ , mass action).

Parasitoids can also disperse into neighbouring stands which have lower trophic levels. Once primary parasitoids control the herbivore in a recovering stand ( $c_p R$ ), the population builds up and may also spread to a neighbouring recovering forest ( $\alpha_p R$ ). Similarly, once hyperparasitoids control a previously parasitoid controlled forest ( $c_{hp} P$ ), they may disperse to neighbouring stands ( $\alpha_{hp} P$ ).

## Spatially-implicit metacommunity model formulation

We translated observations of herbivore-parasitoid-hyperparasitoid dynamics into a theoretical metacommunity model representing multiple stands (patches, cells) within a landscape. The model is based on the extension of the traditional Levins metapopulation model (Levins 1969) of colonization-extinction dynamics to include multiple species. Each patch (1–10 km<sup>2</sup>) comprises a forest stand that can only be in one state according to the insect species that are. The model represents one species per trophic level, but can easily be extended to multiple species with known interactions (Gravel et al. 2011b).

We implemented herbivore-parasitoid-model described above with both a system of differential equations with spatially-implicit (global) dispersal and a cellular automata with spatially-explicit dispersal between neighbouring stands. Comparing results from spatially-explicit and -implicit simulations allowed us to explore the role of local dispersal in landscape dynamics. The system of differential equations considered an infinite number of patches and, consequently, probabilities are transformed into rates. Each variable represents the proportion of stands in each forest state, and the landscape contains a fixed number of stands, so  $R + P + HP + I = 1$  and  $R = 1 - (P + HP + I)$ . We represented these dynamics with the following system of differential equations:

$$\begin{aligned} dP/dt = & c_p R + \alpha_p PR - e_p P - (1 - (1 - \alpha_{sbwp} I)^8) \\ & P - c_{hp} P - \alpha_{hp} PHP \end{aligned} \quad (1a)$$

$$dHP/dt = c_{hp} P + \alpha_{hp} PHP - e_{hp} HP - (1 - (1 - \alpha_{sbwhp} I)^8) HP \quad (1b)$$

$$\begin{aligned} dI/dt = & e_p P + (1 - (1 - \alpha_{sbwp} I)^8) P + e_{hp} HP \\ & + (1 - (1 - \alpha_{sbwhp} I)^8) HP - \gamma I \end{aligned} \quad (1c)$$

$$dR/dt = \gamma I - c_p R - \alpha_p PR \quad (1d)$$

Each stand is potentially surrounded by multiple infested stands yet a single successful dispersal event is sufficient to cause an infestation. Therefore, we needed to modify  $I$  in the equation to appropriately describe this probabilistic event. If  $1 - \alpha_{sbwp}$  sets the probability that no dispersal occurs from a given neighbouring stand, then the probability of no-dispersal event across all neighbouring stands is  $(1 - \alpha_{sbwp} I)^8$  and the probability of at least one dispersal event is  $1 - (1 - \alpha_{sbwp} I)^8$  (as in Fukš and Lawniczak 2001, Guichard et al. 2003). This approximation allowed us to consider space implicitly and introduce a more realistic non-linearity in the system, thus making it susceptible to instability (Nenzén et al. 2017). A high proportion of infested forest stands means a high density of insects and population growth and, indirectly, a higher rate of infestation.

### Spatially-explicit metacommunity model formulation

We investigated a spatially-explicit model to reveal the effect of spatially-explicit dispersal by tracking the neighbouring stands of each forest stand. The model we considered is a cellular automaton where each stand can be in only one of four potential states: parasitoid controlled forest,  $P$ , hyperparasitoid controlled forest  $HP$ , infested,  $I$ , or recovering,  $R$ . The total proportion of each state is equivalent to the proportion modelled by the mean field model. Transition rules in each focal stand (and from each infested neighbouring stand in case of dispersal) were implemented on an annual time step and were the following:

$$R \rightarrow P \text{ if primary parasitoid species are present } (c_p) \quad (2a)$$

$$P \rightarrow HP \text{ if hyperparasitoid species controls and primary parasitoids are absent } (c_{hp}) \quad (2b)$$

$$HP \rightarrow I \text{ if hyperparasitoid species are excluded and primary parasitoids are absent } (e_{hp}) \quad (2c)$$

$$P \rightarrow I \text{ if } \alpha_{sbwhp} \geq h \quad (2d)$$

$$HP \rightarrow I \text{ if } \alpha_{sbwhp} \geq h \quad (2e)$$

$$I \rightarrow R \text{ if } \gamma \geq h \quad (2f)$$

The algorithm simulates a dispersal event from each infested neighbouring stand with a random  $h$  drawn from a uniform distribution on the interval  $(0,1)$  for each infested neighbouring stand (maximum of eight neighbouring stands). A susceptible stand can potentially be infested multiple times, but only one successful dispersal event is necessary to cause a switch of states.

### Model implementation and analysis

We investigated the spatially-implicit and -explicit metacommunity model with numerical simulations. More specifically, we looked at the impact of hyperparasitoid control and herbivore dispersal on the occurrence of outbreaks. We defined outbreaks as sudden peaks in the proportion of infested stands at the regional scale.

To illustrate the effect of different trophic levels on outbreaks, we ran simulations with and without different

trophic levels. We simulated forest dynamics in which only primary parasitoids, only hyperparasitoids, and both types of parasitoids could lead to defoliation. With primary parasitoids only, we used the following parameters:  $c_p = 0.025$ ,  $e_p = 0.01$  and  $\alpha_p = 0.003$ , and all hyperparasitoid parameters were set to zero. With both hyperparasitoids and parasitoids, we modified hyperparasitoid parameters to  $c_{hp} = 0.0001$ ,  $e_{hp} = 0.001$  and  $\alpha_{hp} = 0.3$ . Parasitoid parameters were the same, so the exclusion and dispersal of both parasitoids could lead to an infested forest. With only hyperparasitoids we maintained  $c_p = 0.01$  because hyperparasitoids need parasitoid controlled forests and set  $e_p = 0$  so forests could not transition directly from  $P$  to  $I$ , but first had to be in the hyperparasitoid controlled state ( $HP$ ). Other parameters were as shown in Table 1.

We subsequently explored the parameter space with simulations of  $c_{hp}$  in the range of  $0.00001-0.1$ , and two values of  $e_{hp}$ ;  $0.00001$  and  $0.002$ . We also tested the effect of distinct herbivore dispersal probabilities into parasitoid and hyperparasitoid controlled forest,  $\alpha_{sbwp}$  and  $\alpha_{sbwhp}$  ( $0-1$  in increments of  $0.05$ , Eq. 1a–1d, 2a–2f). Other parameters were kept constant based on our knowledge of forest dynamics, and because we wanted to focus on  $c$  and  $e$  (Table 1). The minimum time required to reach a mature forest state in North American boreal forests is 40 years (Burns and Honkala 1990), therefore, we set  $c_p$  to  $1/40$ . Stand mortality usually begins in the fifth year of infestation, and full mortality occurs after roughly 10 years, when the stand is completely defoliated and unable to photosynthesize (MacLean 1980).

We analyzed the spatially-implicit model (Eq. 1a–d) with numerical simulations. We performed numerical integration with a Runge–Kutta fourth-order algorithm with a step size of  $0.1$  year. We ran simulations for 5000 years and discarded the first 100 years as transients.

Table 1. Model states and default parameters used in simulations. Forest states are defined as proportions of the landscape.

Process	Symbol	Value	Description
States	$P$		parasitoid-controlled forest
	$HP$		hyperparasitoid-controlled forest
	$I$		infested forest
	$R$		recovering forest
Parameters	$\gamma$	1/3	forest mortality
	$c_p$	1/40	parasitoid-control of recovering forest with low herbivores
	$e_p$	0	parasitoid exclusion, loss of control
	$c_{hp}$	0.0001	hyperparasitoid-control
	$e_{hp}$	0.001	hyperparasitoid exclusion, loss of control
	$\alpha_p$	0	parasitoid dispersal into recovering forest
	$\alpha_{hp}$	0.3	hyperparasitoid dispersal
	$\alpha_{sbwp}$	0.0	probability of herbivore dispersal into $P$
$\alpha_{sbwhp}$	0.4	probability of herbivore dispersal into $HP$	

The spatially-explicit simulations (Eq. 2a–f) were run on a square lattice of  $X^2$  forest stands with periodic boundary conditions (torus,  $X=200$ ). We used a large landscape to allow complex landscape dynamics to emerge, and present results from a subset of  $10 \times 10$  stands. In each time step (year), the algorithm randomly selects  $X^2$  stands and updates their states. A stand may therefore have no or multiple state changes in a single time step. This method approximates continuous mean-field dynamics (asynchronous updating; Durrett and Levin 1994). Simulations were initiated with an equal proportion of all states (0.25) distributed randomly in space. We ran 10 stochastic simulations for each parameter set. We analyzed simulation results to determine if large-scale outbreaks occurred, and determine their spatial distribution. We recorded the maximum proportion of infested stands (I state) during each simulation as outbreak size. Average parasitism rates were represented by the combined proportions of P and HP states, as this indicates the proportion of stands on which the herbivore density is low.

## Results

Simulations indicated that hyperparasitoids destabilizes community dynamics and causes herbivores to have large, cyclic outbreaks. Outbreaks occurred as hyperparasitoids gradually controlled a larger proportion of the previously primary parasitoid controlled landscape and their inherent instability caused their abundance to drop and an outbreak occur (Fig. 3C, F). Hyperparasitoids persisted during the outbreak even though the model was formulated so there were no hyperparasitoids in defoliated forests. Hyperparasitoid control peaked just before and during outbreaks.

The spatially-implicit model generated outbreaks at the beginning of simulations, while the spatially-implicit model exhibited sustained outbreaks over longer periods. Oscillations were weak in the spatially-implicit model with hyperparasitoids (Fig. 3 C). Large outbreaks covered almost 50% of the landscape in the spatially-explicit model with hyperparasitoids. Outbreaks initiated by hyperparasitoids were 10% larger than outbreaks produced with both

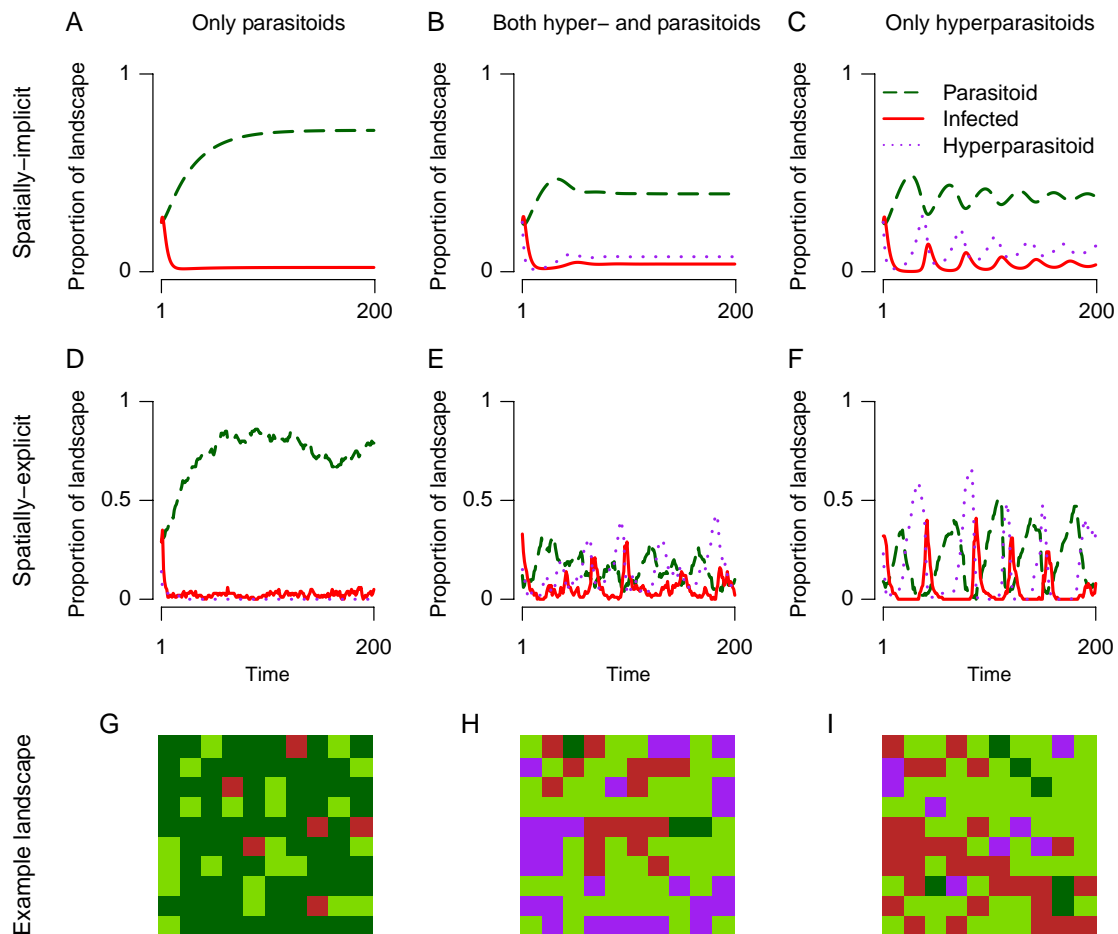


Figure 3. Simulations in which different trophic levels cause outbreaks: only parasitoids (left column, A, D, G), both primary parasitoids and hyperparasitoids (middle column, B, E, H), and only hyperparasitoids (right column, C, F, I), in both spatially-implicit (top row) and -explicit (middle row) models. The bottom row shows an example of landscape from the spatially-explicit simulations. If a (hyper-) parasitoid is present, its exclusion rate,  $e_p$  ( $e_{hp}$ ), is 0.001. Simulations were carried out with all other parameter values set as in Table 1.

hyperparasitoids and primary parasitoids (Fig. 3F versus E). Defoliation was constant and there were no large-scale outbreaks in the absence of hyperparasitoids in both spatially-implicit and -explicit models (Fig. 3A, D).

Hyperparasitoid control and exclusion probabilities determined outbreak size. Either low hyperparasitoid control probabilities,  $c_{hp}$ , or low hyperparasitoid exclusion probabilities,  $e_{hp}$ , increased outbreak size (37% of landscape infested in Fig. 4). Both high hyperparasitoid control and exclusion probabilities allowed hyperparasitoids to quickly control herbivores throughout the landscape over a longer period of time. Then, outbreaks occurred constantly and were therefore smaller as there were only small pockets of susceptible forest (9% of the landscape infested, in Fig. 4).

Outbreaks covered a quarter of the landscape in the absence of dispersal ( $\alpha_{sbwp}$  and  $\alpha_{sbwhp} = 0$ , Fig. 5). Larger outbreaks occurred when herbivores dispersed with a higher probability into hyperparasitoid controlled forests than into parasitoid controlled forests (in terms of proportion of the landscape infested, above the white diagonal, Fig. 5).

## Discussion

We investigated how the joint effects of parasitoids and hyperparasitoids could cause large-scale defoliation. Insect outbreaks have large impacts on ecosystems and economy, and it is therefore critical to better anticipate them. Our metacommunity model replicated unstable and over-exploitative host–parasitoid interactions, and our results suggest that hyperparasitoids destabilise herbivore dynamics and generate outbreaks. We showed that primary parasitoid and hyperparasitoid density build up until hyperparasitoids are excluded and herbivore population increases rapidly (Fig. 3). The spatially-implicit version did not generate

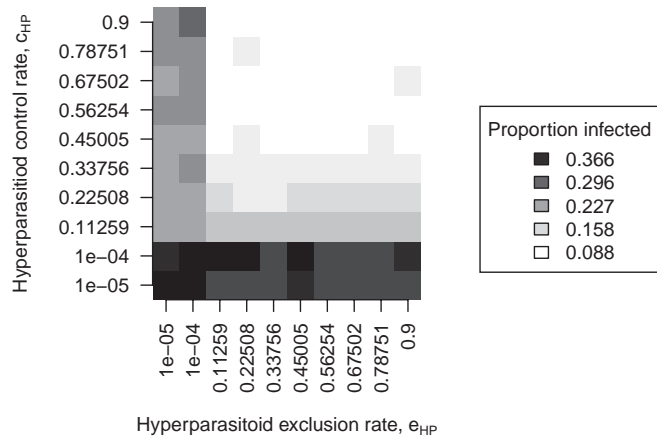


Figure 4. Effect of hyperparasitoid control  $c_{hp}$  and exclusion  $e_{hp}$  probabilities on outbreak size, i.e. the proportion landscape in state I. Each value indicates the average maximum infested proportion from all simulations with that parameter combination. Simulations were carried out with all other parameter values set as in Table 1.

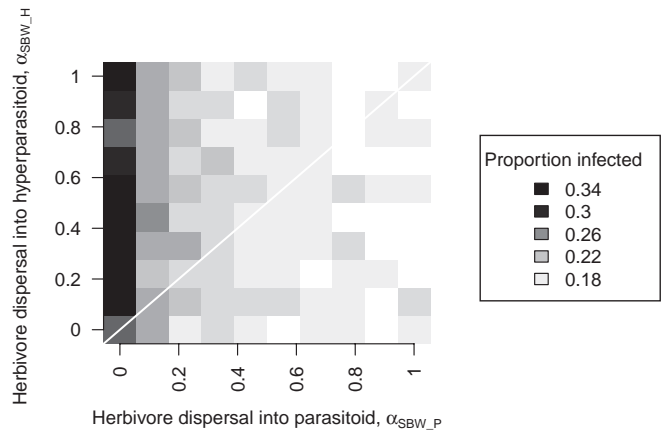


Figure 5. Effect of herbivore dispersal probability on parasitoid and hyperparasitoid controlled forest outbreak size, i.e. the proportion of landscape in state I. The diagonal white line indicates  $\alpha_{sbwp} = \alpha_{sbwhp}$ . Simulations were carried out with all other parameter values set as in Table 1.

sustained outbreaks, only weak oscillations, while the spatially-explicit version did. These results further suggest that spatially-explicit dispersal limitation is a key mechanism to generate outbreaks in nature (Bjørnstad and Bascompte 2001, Roland and Taylor 1997, Nenzén et al. 2017). Hyperparasitoids destabilise herbivore dynamics because they regulate the density of two trophic levels simultaneously, both herbivores and primary parasitoids (Fig. 1). We showed that representing only herbivores and parasitoids was insufficient to generate outbreaks and that a third trophic level, combined with dispersal limitations, was highly destabilising.

We constructed a metacommunity model that quantitatively supports observations that hyperparasitoids may cause outbreaks (Rosenheim 1998). Hyperparasitoid impacts are likely underestimated because they are little studied. Hyperparasitoids are difficult to rear in laboratories and few studies are explicitly designed to sample them at their emergence at the end of the summer (Hawkins 1994, Rosenheim 1998). Studies that quantify their effects corroborate our hypothesis that additional trophic levels are necessary to cause outbreaks. For example, single-species metapopulation models do not capture the dynamics of herbivores that are attacked by parasitoids (Kean and Barlow 2000). Field experiments show that aphids are often controlled by parasitoids, and that hyperparasitoids may disrupt this control (Gómez-Marco et al. 2015, Frago 2016). We found parasitoid density to increase when they were protected from hyperparasitism, suggesting that hyperparasitoids could disrupt top–down regulation. Our study suggests that the regulation of herbivores by their parasitoids may decrease just before an outbreak (Myers and Cory 2013). However, it is possible to alternatively attribute outbreak causes to mechanisms other than trophic regulation, such as bottom–up control. During spruce budworm outbreaks, the observed parasitoid



community could have a higher diversity of hyperparasitoid species just because there are more resources in the form of budworm. Eveleigh et al. (2007) interpreted this as a 'bird-feeder effect', in which higher budworm density attracts a higher primary parasitoid and hyperparasitoid species richness (a bottom-up view of the food web assembly). In our model, hyperparasitoids were more prevalent just before and during outbreaks because they actually add additional instability and cause outbreaks. Our top-down hypothesis that natural enemies drive outbreaks is nonetheless coherent with observed budworm outbreaks because we reproduced the empirically observed long periods without outbreaks punctuated by fast increases in density (Fig. 3).

Once the herbivore has increased in density in one location, higher dispersal probability into hyperparasitoid controlled forests further favors large outbreaks. Simulation results supported the hypothesis that large outbreaks could occur if herbivores experienced less parasitism in hyperparasitoid controlled forests (Fig. 5). The reason for that is that in forest controlled by hyperparasitoids, primary parasitoid density is lower, so herbivores experience less parasitism and population growth is higher.

Our results rest on the assumption that hyperparasitoids control the beginning of outbreaks instead of terminating them. There is no empirical evidence that higher parasitism rates cause the end of outbreaks of other insects (Hagen et al. 2010, Schott et al. 2010). Also, our model did not represent facultative hyperparasitoids that might behave as an intermediate trophic level and stabilise dynamics the way parasitoids do. It is a simplified metapopulation model so it does not explicitly include density-dependent growth or foraging behavior, which may be important (Mackauer and Völkl 1993, Ives et al. 1996). These assumptions should therefore be validated with landscape-level data such as documentation of transition probabilities. We assumed that defoliation was a proxy for high herbivore:parasitoid density ratios, and that a healthy forest indicates high primary parasitoid and hyperparasitoid control probabilities. This allowed us to formulate a presence-absence metacommunity model instead of a population density model. While these assumptions might simplify population dynamics considerably, metacommunity models are advantageous because they require fewer parameters and are more tractable. An additional advantage of using defoliation as a proxy for species control is that the model can be directly compared to available data on parasitoid-caused herbivore mortality, i.e. the percentage of parasitism probability on herbivores caused by parasitoids (Myers and Cory 2013, Eveleigh and Johns 2014, McGugan and Blais 1959). This is useful because in the field it is easier to quantify parasitism probability than to estimate population density (as required by population models, among other parameters, Hassell 2000). Alternatively, molecular methods may reveal hyperparasitoid presence (Gómez-Marco et al. 2015).

## Conclusion

The metapopulation concept was originally developed to improve the biological control of pests (Levins 1969), but it has rarely been used in biological pest management because it did not explicitly include biotic interactions. Here, we extended the Levins metapopulation model to the metacommunity in order to represent interactions between multiple trophic levels. This metacommunity model is more tractable compared with similar population models that represent multiple trophic levels with a greater number of parameters. Results suggest that future empirical and theoretical studies should consider natural enemies from higher trophic levels, as they may have destabilising effects on herbivore dynamics. The model proposes the testable prediction that hyperparasitoid control probability should increase (and consequently parasitism probabilities drop) just before an outbreak occurs. If empirical data reveal the same gradual increase and abrupt decline in hyperparasitoid control probability, hyperparasitoids could serve as indicators to anticipate the occurrence of outbreaks.

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*Author contributions* – HN and DG developed the initial model; HN implemented and analysed the final model and drafted the initial manuscript; all authors co-wrote the manuscript and gave final approval for its publication.

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Supplementary material (available online as Appendix oik-05112 at <[www.oikosjournal.org/appendix/oik-05112](http://www.oikosjournal.org/appendix/oik-05112)>). Appendix 1.