Highlights

Effect of Migrations on Synchrony in Host-Parasitoid system

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Effect of Migrations on Synchrony in Host-Parasitoid system

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Abstract

Insect outbreaks can cause large scale defoliation of forest trees or destruction of crops, leading to ecosystem degradation and economic losses. Some outbreaks occur simultaneously across large geographic scales and some outbreaks occur periodically every few years across space. Parasitoids are a natural enemy of these defoliators and could help mitigate these pest outbreaks. A holistic understanding of the host-parasitoid interactions in a spatial context would thus enhance our ability to understand, predict and prevent these outbreaks. We use a discrete time deterministic model of the host parasitoid system with populations migrating between 2 patches to study spatial host outbreaks. We show that whenever populations persist indefinitely, host outbreaks in both patches occur alternatively (out of phase) at low migration between patches whereas host outbreaks occur simultaneously (in phase) in both patches at high migration between patches. We show that our results are robust across different modelling approaches and give an analytical expression for the period of oscillations when the migration is low i.e. when host outbreaks in both patches are out of phase. We end our paper by showing that we get the same results whether we include the biologically rooted formulations from May et al. (1981) or a general cellular automata model with qualitative rules.

Keywords: Population dynamics, Insect oscillations, Host-parasitoid interactions, Functional responses, Cellular Automata models

1. Introduction

It is long known that insect herbivores are agents causing great threats to ecosystems - threatening irreversible changes to food security, forest cover etc Balla et al. (2021) Boyd et al. (2013) Gandhi and Herms (2010). These insect populations can remain at a low density for many years, often going unnoticed. However, every few years, these insect populations explode in numbers, causing large scale loss of forest cover Liebhold and Bentz (2011) Hunter and Dwyer (1998). Many factors including growth rates, predation, environmental factors and changes in food quality are known to influence the population trends of these herbivores (Barbosa et al. (1987)) Myers (1988), however the exact reasons still remain elusive. Several time-lagged mechanisms like interactions with predators, diseases, maternal effects etc., have been identified to cause such cyclical dynamics Umbanhowar and Hastings (2002).

Classical biological control is the phenomenon of purposefully introducing and establishing a foreign species (natural enemy) with the aim of suppressing the outbreaks of the native species Briese et al. (2000) Caltagirone (1981). Pests which can be controlled by introducing natural enemy includes invertebrates, vertebrates and weeds. The organisms that function as natural enemy include vertebrates (birds, reptiles) and invertebrates (parasitoids). Here, we focus our attention on herbivore insect populations and parasitoids

Parasitoids have long been a subject of ecological interest for several reasons. Roughly 14% of all insect species are parasitoids Hassell and Waage (1984). The female parasitoid searches for an immature stage of the herbivore insect (host) and lays eggs inside it. These eggs hatch inside the body of the host and the parasitoid larvae feed and grow at the expense of hosts, inside the host. At a later stage, they emerge from the host, typically killing it in the process. Thus, parasitoids are considered agents of biological control as they can keep the herbivore insect populations to low numbers by direct mortality Waage and Hassell (1982), Wang et al. (2019) Godfray et al. (1994) Strand and Obrycki (1996).

There are several simplifications which make the study of host-parasitoid system more suited to study enemy-victim dynamics than more gen-

eral predator prey dynamics. Many parasitoids are highly specific to the hosts they predate Strand and Obrycki (1996), allowing us to consider the host-parasitoid system as a closed system, independent of the influence of other populations. Furthermore, this specificity also leads to synchronized life cycles of host and parasitoid species, allowing the use of discrete time equations appropriate. Since hosts are attacked only by adult parasitoids, we can further ignore age structure in our modelling Mills and Getz (1996).

Migration is of crucial importance in unraveling the dynamics of spatial synchrony in insect populations Abbott and Dwyer (2008) Liebhold et al. (2004). Migration can introduce traveling population waves, leading to complex relationships between synchrony and distanceLiebhold et al. (2004),Hassell et al. (1991). It shapes the abundance and demographic properties of insect populations by influencing reproductive patterns, mortality rates, and population characteristics such as size and age distributions. Moreover, migratory movements can synchronize the dynamics of different species, including those with direct trophic interactions and shared resourcesLiebhold et al. (2004). Investigating migration patterns can lead to insights into the mechanisms driving spatial synchrony which is vital for predicting and managing insect outbreaks, understanding ecological interactions, and effectively conserving and controlling insect populations.

Given the rather strong assumptions made in most spatial host-parasitoid models, it could appear that drawing conclusions about a specific biological system could be difficult. We address this gap here by trying to demonstrate robust results that should apply across a range of systems. In this paper, we study coupled dynamics of a host-parasitoid system with a focus on exploring the role of migration in ensuing dynamics. We use a 2 patch host-parasitoid system, with the dynamics within each patch as given by Umbanhowar and Hastings (2002), with hosts and parasitoids migrating between the patches after reproduction. Within each patch, the hosts are limited by intraspecific competition and parasitism Umbanhowar and Hastings (2002). We show that at low migration, the host outbreaks in the 2 patches occur alternatively (out of phase) and at high migration, the host outbreaks in both patches occur simultaneously (in phase). While similar results for host-parasitoid systems have been shown before Adler (1993), we focus our attention to the robustness and cause of such oscillations. We address the robustness by testing our results across different biologically relevant functional forms for intraspecific competition and parasitism. We then show that similar results are obtained in a cellular automata model devoid of biological detail, which only qualitatively captures host-paraisioid system, thus establishing the role of migration independent of other biological interactions. Lastly, we provide an analytical expression for calculating the time period of the oscillations of the system, when the two patches oscillate out of phase.

2. Single patch dynamics

We use the general formulations from May et al. (1981), which models 3 phenological processes - reproduction, intraspecific competition amongst hosts and parasitism (by parasitoids). This gives a general form for annual densities for hosts (H_t) and parasitoids (P_t)

$$\begin{aligned} H_{t+1} &= \lambda F(H_t) G(H_t, P_t) \\ P_{t+1} &= cF(H_t)(1 - G(H_t, P_t)) \end{aligned} \tag{1}$$

Here λ is the intrinsic growth rate of the hosts and c is the no of parasitoids that emerge from a single larvae. Following the analysis done in Umbanhowar and Hastings (2002), we assume the following form of non-dimensionalized equations for the host-parasitoid dynamics within each patch, where the min function is used to model intraspecific competition. It is exactly compensatory, as opposed to Ricker or many other forms of intraspecific competition which are over compensatory. This model has an unstable fixed point and thus leads to oscillations as shown in Figure 1.

$$\begin{aligned} h_{t+1} &= \lambda \min(h_t, 1) e^{-p_t} \\ p_{t+1} &= \gamma \min(h_t, 1)(1 - e^{-p_t}) \end{aligned} \tag{2}$$

The oscillations produced by this model can be divided into 3 phases - 1) Buildup - which is marked by low host and parasitoid densities (i.e. $h_t \to 0, p_t \to 0$), followed by 2) Outbreak - which is marked by high host density but low parasitoid density (i.e. $h_t \to \lambda, p_t \to 0$), which is followed by 3) Crash - where the parasitoid population is large enough to reduce host population significantly (i.e. $p_t > 1, h_t \to 0$).

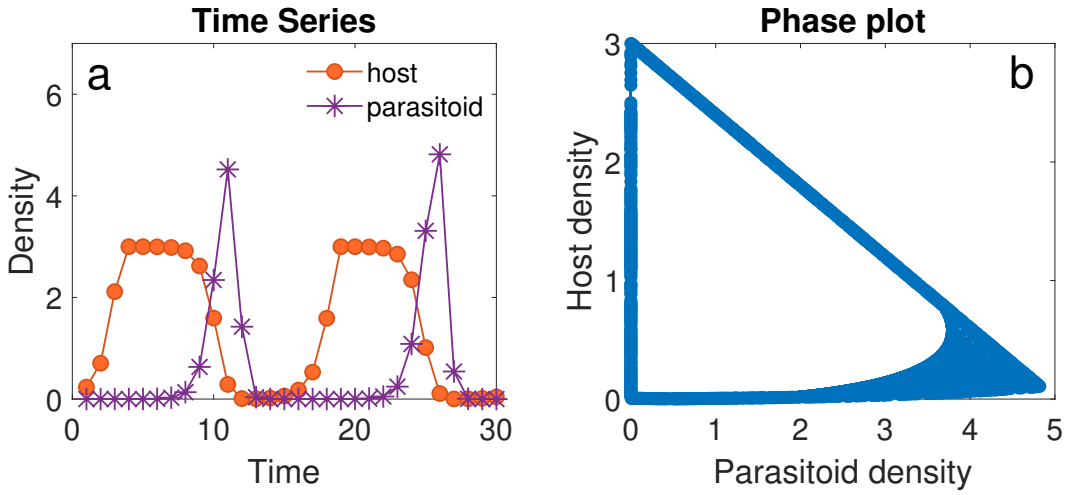

Figure 1: a) Time series and b) phase plot of a single host-parasitoid population at steady state is shown. As shown, the densities show a quasi-periodicity, where they are confined within a manifold as shown in (b).

3. Two patch dynamics

Let h_t^i, p_t^i be the host and parasitoid population in patch i at time t. Let λ be the growth rate of the host and γ be the growth rate of parasitoid. Let ϵ be the fraction of population migrating from one patch to another. The equations for the resulting dynamical system is:

$$\begin{aligned} h^1_{t+1} &= h^1_{tm}(1-\epsilon) + \epsilon\, h^2_{tm} \\ h^2_{t+1} &= h^2_{tm}(1-\epsilon) + \epsilon\, h^1_{tm} \\ p^1_{t+1} &= p^1_{tm}(1-\epsilon) + \epsilon\, p^2_{tm} \\ p^2_{t+1} &= p^2_{tm}(1-\epsilon) + \epsilon\, p^1_{tm} \end{aligned} \tag{3}$$

Here h_{tm}, p_{tm} represent the host and parasitoid densities at t before migration after competiton, parasitism and reproduction have taken place i.e.

$$\begin{aligned} h_{tm} &= \lambda \min(h_t, 1) e^{-p_t} \\ p_{tm} &= \gamma \min(h_t, 1)(1 - e^{-p_t}) \end{aligned} \tag{4}$$

In our model, reproduction is given by a multiplicative factor, thus, the order of migration and reproduction can be interchanged. Biologically, it'll refer to the scenario where the surviving adults after competition and parasitism have taken place, migrate and then reproduce.

When $\epsilon \to 0$, both patches oscillate almost independently i.e. the effect of coupling due to migration between patches is negligible. Note that the parameters are identical in both the patches.

As we increase the strength of coupling due to migration i.e. ϵ from 0, we first get out of phase solutions (as shown in Figure 2 (b)), where the peaks in host densities (i.e. host outbreaks) in 2 patches occur alternatively (out of phase). These out of phase oscillations persist indefinitely in our simulations as long as the initial conditions in the 2 patches aren't very identical. If the initial conditions are identical, then both patches behave as one single patch and thus, they oscillate in unison (see Figure 2 (d)). In our simulations, initial conditions were selected randomly and the host densities of 2 patches at steady state is shown in Figure 2.

These out of phase solutions occur for a wide range of migrations as shown in Figure 2 (b,c)), ranging several orders of magnitude. With increasing ϵ, we find that the length of the outbreaks decrease until the outbreaks occur for 1-2 generations. Further increasing ϵ, beyond this point leads to a transition towards in phase solutions, where the host outbreaks occur simultaneously in both patches (as shown in Figure 2 d). We find that, the transition occurs in a very small window for ϵ i.e. (in $0.01 < \epsilon < 0.025$). The exact nature and window of transition is complex and depends on other parameters i.e. λ and γ. Out of phase solutions are further characterized by periods of near absence of hosts are accompanied by sudden outbreaks which last for few generations in each patch. However, if we look at the overall sum of host densities in both patches, they remain unchanging for most of the generations as shown in Figure 3 (a). However, the

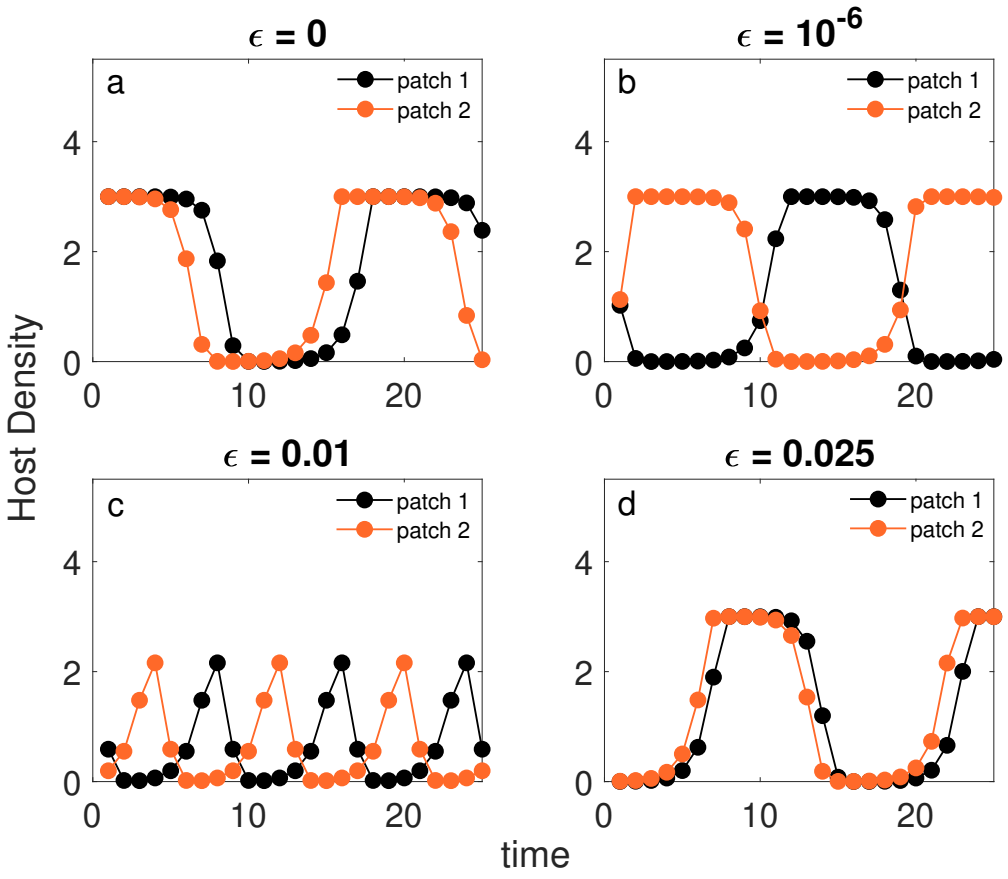

Figure 2: Host densities at steady state are shown for different migration rates. The parameters $\lambda = 3$, $\gamma = 6$ were fixed. In (a), there is no coupling between the patches. However, when we increase ϵ, we see out of phase solutions (b,c), where the host outbreaks in the 2 patches occur alternatively. These out of phase solutions exist for a large range of ϵ values. Increasing ϵ, the time period of host outbreaks decrease till the outbreak occurs for 1-2 generations (as shown in (c)). With further increase in migration, the host outbreaks in both the patches occur simultaneously as shown in (d).

in phase solutions are different as periods of near absence of hosts are accompanied by sudden outbreaks lasting few generations, both at individual patch level and overall sum over both patches as shown in Figure 3 (b).

At higher values of ϵ, we see in phase solutions, where the peaks in host densities (i.e. host outbreaks) in 2 patches occur simultaneously (as shown in Figure 2b) i.e. both patches oscillate in phase. High migration rates i.e. ($\epsilon \to 1$) lowers any difference in population in 2 patches. This happens till both patches oscillate in phase after which the effect of migration is negligible, as shown below.

$$h^1_{t+1} - h^2_{t+1} = \lambda(1-2\epsilon)\delta \tag{5}$$

where

$$\delta = \left(\min(h^1_t, 1)e^{-p^1_t} - \min(h^2_t, 1)e^{-p^2_t}\right)$$

When

$$\epsilon \to 1, \qquad \lambda(1-2\epsilon) < 1$$

and

$$|(\min(h^1_t, 1)e^{-p^1_t} - \min(h^2_t, 1)e^{-p^2_t})| \leq |h^1_t - h^2_t|$$

Thus, the difference in population in 2 patches decreases every generation

$$|h^1_{t+1} - h^2_{t+1}| < |h^1_t - h^2_t| \tag{6}$$

Thus, at steady state, both patches oscillate in phase, i.e.

$$\lim_{t\to\infty} h^1_t = h^2_t$$

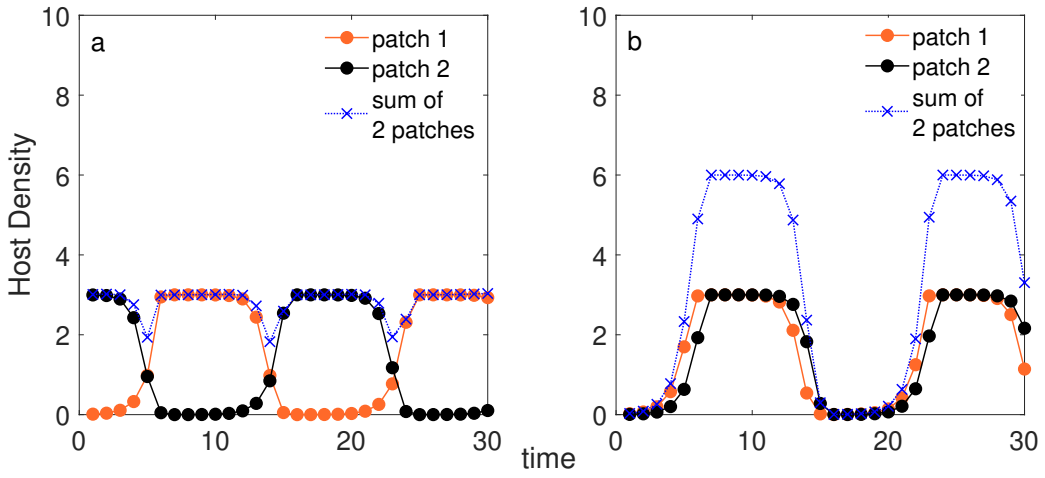

Figure 3: Host densities at steady state are shown for different migration rates. At low migration (a) i.e. $\epsilon = 10^{-6}$, we see that the host outbreaks in the 2 patches occur alternatively. The sum of the host densities remains constant with small fluctuations every few generations. In contrast, at high migration rates i.e $\epsilon = 0.025$, host outbreaks in both the patches occur simultaneously as shown in b). We see cycles of large overall host outbreaks followed by generations of near host absence, as shown by the sum of the host densities in the 2 patches. The parameters used were $\lambda = 3$, $\gamma = 6$.

Table 1: List of all different functions we've tested in our 2 patch model

Description	Mathematical form	Other parameters involved
Ricker map for hosts intraspecific competition	$F(H_t) = e^{r(1-\frac{H_t}{K})}$	Growth rate r, Carrying Capacity K
Smith map for hosts intraspecific competition	$F(H_t) = \frac{H_t}{1+(\lambda-1)(\frac{H}{K})^b}$	Growth factor λ, Carrying capacity K, strength of competition b
Independent search by parasitoids for hosts	$G(H_t, P_t) = e^{-aP_t}$	Per capita searching efficiency a
Aggregated attacks by parasitoids on hosts	$G(H_t, P_t) = \left(1 + \frac{aP(t)}{k}\right)^{-k}$	Per capita searching efficiency a, Degree of aggregation k

4. Robustness across choice of functions

Our results from the previous section are robust across different choices of intraspecific competition i.e. $F(H_t)$ and parasitism $G(H_t, P_t)$. For $F(H_t)$, we chose Ricker map and a function described by Maynard Smith, for annual insects amongst choice of intraspecific competition (as shown in the table below). For $G(H_t, P_t)$, we used both independent and aggregated parasitoid search for hosts.

We found similar out of phase and in phase oscillations at low and high values of migration for any combination of functions mentioned above. Figure 4 shows a few specific cases of our findings.

5. Length of the cycle

When migration rate is high, both patches oscillate in phase, behaving like a single patch. Thus, any measure of length of cycle i.e. time between successive host outbreaks, can be calculated using the equations derived for a single isolated patch Umbanhowar and Hastings (2002). We provide an approximate expression for length of cycle when migration rates are low and both patches oscillate out of phase. We define $T_{b,i}$, $T_{o,i}$ and $T_{c,i}$ as the number of generations patch i undergoes buildup, outbreak and crash respectively. We use the following observations (without losing generality):

- Duration of build up in 1 patch must be no more than the combined duration of outbreak

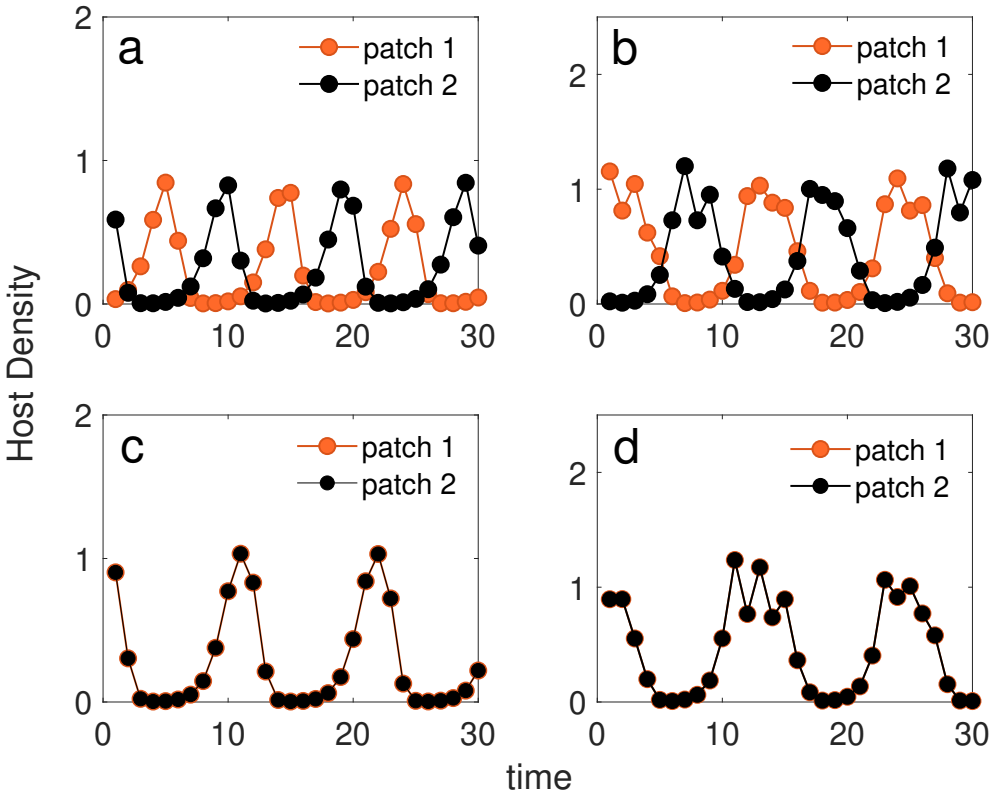

Figure 4: Host densities in 2 patches are plotted at steady state. Top (bottom) column represents cases for low (high) migration rates respectively. a) and c) are examples where host intraspecific competition was modelled using Ricker map and the parasitoid attack was aggregated. b) and d) are examples where intraspecific competition was modelled using Smith map and parasitoid search for hosts are independent.

and crash in other patch i.e.

$$T_{b,1} \leq T_{o,2} + T_{c,2}$$

If this condition isn't satisfied, then both the patches will have build-up simultaneously, which isn't the case in out of phase oscillations.

- Duration of build up in 1 patch must be no less than the duration of outbreak in other patch i.e.

$$T_{b,1} > T_{o,2}$$

If this condition isn't satisfied, then both the patches will have outbreaks simultaneously, which isn't the case in out of phase solutions.

Since a crash occurs for 2-3 generations, the time for build up is given by

$$T_{b,1} = T_{o,2} + 1 \text{ or } 2 \tag{7}$$

Because both the patches are symmetric, we have $T_{b,1} = T_{b,2}$ and $T_{o,1} = T_{o,2}$. Henceforth, we will drop the patch numbers and just talk about build up (T_b), outbreak (T_o), and crash phases (T_c) and the total length of the cycle (T).

$$T = T_b + T_o + T_c \tag{8}$$

From $T_o = T_b - 1 \text{or} T_b - 2$ (from Equation [4]). Thus, adding that the crash is usually 2-3 generations

$$T = 2T_b \text{ or } 2T_b + 1 \tag{9}$$

The host density at the start of build up is $H_1 = \lambda^2 e^{-\gamma\left(2-\frac{1}{\lambda}\right)} + \epsilon\lambda$, where the former term is a result of parasitism (as shown in Umbanhowar and Hastings (2002)) and the latter is the host population which migrated into this patch from the patch undergoing an outbreak. Since γ is usually large, $\lambda^2 e^{-\gamma\left(2-\frac{1}{\lambda}\right)} \approx 0$. This is supported by the intuition that after the crash, the host den-

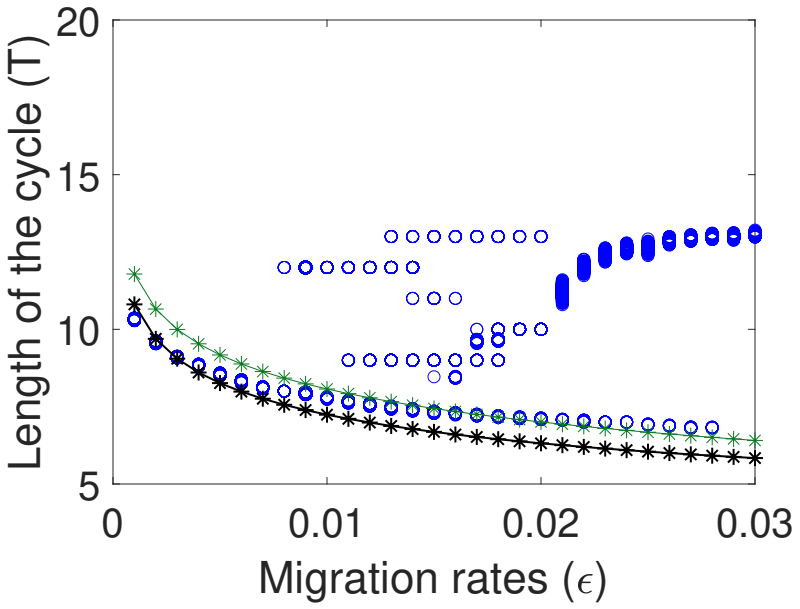

Figure 5: Length of cycle (numerically and approximate expressions) are plotted. Each blue dot represents length of cycle numerically obtained for a single iteration, after taking the mean of 1000 different time points after steady state was reached. The green and black curves represent the approximate theoretical expressions derived in the text. Here $\lambda = 3.375$ and $\gamma = 4.875$.

sities are reduced to very small values due to parasitism. Thus, for very small values of migration i.e. $\epsilon \leq \lambda e^{-\gamma\left(2-\frac{1}{\lambda}\right)}$, we don't see any affect of migration and the two patches behave as if they are isolated patches. But for $1 >> \epsilon >> \lambda e^{-\gamma\left(2-\frac{1}{\lambda}\right)}$, $H_1 \approx \epsilon\lambda$. Given the geometric nature of the growth of host density during buildup (as parasitoid density is low, leading to no mortality due to parasitism) and host migration from the other patch undergoing an outbreak, we have

$$H_t = \epsilon(\lambda^t + \lambda^{t-1} + \cdots + \lambda) = \epsilon\lambda\frac{(\lambda^t - 1)}{\lambda - 1} \quad (10)$$

As long as the other patch is undergoing an outbreak, the host density in the patch in the build up phase will continue to grow according to the expression above. Assuming that the outbreak lasts for $T_o = T_b - 2$ generations, we would have

$$H_{T_b-2} = \epsilon\lambda\frac{(\lambda^{T_b-2} - 1)}{\lambda - 1} \quad (11)$$

Following an outbreak, the other patch undergoes crash which has high parasitoid density, reducing host densities in that patch. Thus, we can ignore the effect of migration for the remainder of the 2 generations of buildup, giving us the expression for host density at the end of the build up

$$H_{T_b} = \epsilon\lambda^3\frac{(\lambda^{T_b-2} - 1)}{\lambda - 1} \quad (12)$$

Similarly, at the end of buildup the host density is $H_{T_b} \approx 1$ (as shown in Umbanhowar and Hastings (2002)). Equating the expected host densities at the start of outbreak, we get

$$\epsilon\lambda^3\frac{(\lambda^{T_b-2} - 1)}{\lambda - 1} \approx 1 \quad (13)$$

This gives us an approximate expression for T_b and $T = 2T_b$ as follows

$$T_b = 2 + \frac{\ln\left(1 + \frac{\lambda-1}{\epsilon\lambda^3}\right)}{\ln(\lambda)} \quad (14)$$

$$T = 4 + 2\frac{\ln\left(1 + \frac{\lambda-1}{\epsilon\lambda^3}\right)}{\ln(\lambda)} \quad (15)$$

Furthermore, if instead we take $T_o = T_b - 1$ generations and $T = 2T_b + 1$, we get the approximate expression as

$$T_b = 1 + \frac{\ln\left(1 + \frac{\lambda-1}{\epsilon\lambda^2}\right)}{\ln(\lambda)} \quad (16)$$

$$T = 3 + 2\frac{\ln\left(1 + \frac{\lambda-1}{\epsilon\lambda^2}\right)}{\ln(\lambda)} \quad (17)$$

To test our expression for T against numerical simulations, we plot these expressions against the length of cycle numerically observed in out of phase solutions for different migration rates (for fixed λ and γ). To find the length of the cycle numerically, we measure the no of generations between peak parasitism (during crash) within each patch. We measure this 1000 times from either patch at random times after steady state was reached. The length of the cycle is the mean of these 1000 instances. Furthermore, for each value of ϵ, we repeat this for 10,000 different iterations, with different initial conditions, to account for any dependency on initial conditions. We plot the length of a cycle for all of these 10,000 iterations (for a given ϵ) in the same plot. When $\epsilon \approx 0$ or $\epsilon \approx 1$, the length of cycle should be same as that of a single patch. For intermittent values of ϵ, we expect the length of cycle to first decrease and then increase as $\epsilon \approx 1$, in agreement with what we see in Figure 5. As shown in Figure 5, the numerical and approximate analytical expressions (shown in green and black) are in agreement. Furthermore, as ϵ increases, we see that length of cycle increases as both patches are in phase, albeit there are some instances in which system still ends up out of phase, implying the dependence of initial conditions.

6. Cellular Automata model

In this section, we show that our main results - patches oscillating out of phase at low migration rates and in phase at higher migration rates - is independent of the exact biological details of our modelling of host parasitoid system. We introduce a cellular automata model which qualitatively captures the different phases of host-parasitoid system i.e. the buildup, outbreak and crash. Our model is sequential i.e. it stays in buildup phase for m generations, then in outbreak for n generations and then in crash for o generations and the cycle continues. For simplicity of presenting results later, we say outbreak is state 1, crash is state 0 and buildup is state -1.

We consider 2 patches, each with this cellular automata model, with diffusive migration between them. We only consider scenarios where at least one patch has high host or parasitoid density, so that migration could cause change in the other patch. The 2 patch dynamics can be summarized as follows (since both patches are symmetric):

- If patch 1 is in buildup and patch 2 is in outbreak, then patch 1 inches closer to outbreak phase, depending on the migration rate. The reasoning for considering this is that the host density in 2 is high and thus migration could cause a change in host density in patch 1, which is undergoing buildup. Any change in parasitoid densities is ignored because the densities in both patches are low.

- If patch 1 is in outbreak and patch 2 is in crash, the patch 1 inches closer to crash, depending on the migration rate. The parasitoid density in 2 is high and thus any migration could cause a change in parasitoid density in patch 1, which is undergoing outbreak. Any change in host densities in either patches is ignored because of high parasitoid density in patch 2 and low parasitoid density in patch 1.

Analogous to our host-parasitoid system, when $\epsilon \approx 0$, both patches should have no change in their state due to migration. Conversely, when $\epsilon \approx 1$, the patches should exchange their current state. Thus, we implement the effect of migration in a discrete steps between $[0, 1]$, in between these two extremes. For example, if patch 1 is $k1$ generations into build up and patch 2 is $k2$ generations into outbreak and ϵ is the migration rate, then patch 1 inches forward by approximately $\epsilon(k2 + m - k1)$ generations. Similarly, if patch 1 is $k1$ generations into outbreak and patch 2 is $k2$ generations into crash and ϵ is the migration rate, then patch 1 inches forward by approximately $\epsilon(k2 + n - k1)$ generations.

Figure 6 shows the results for different values of migration. We see that at low migration values, we see both patches oscillate out of phase, which changes at higher migration rates. We used $m = 10$, $n = 9$ and $o = 3$ for our figure. But our results are valid for many values of m,n and o as long as $m, n >> o$. This is an important condition because crash is often short-lived compared to build up or outbreak phases.

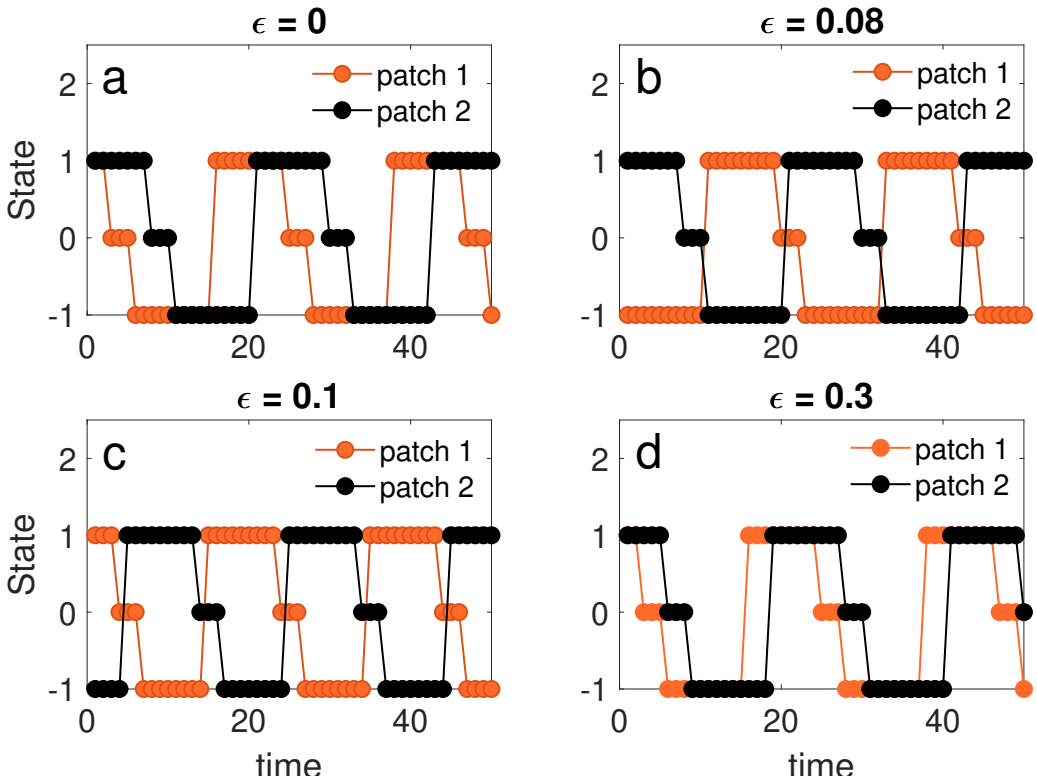

Figure 6: An example of the results for different migration rates from our cellular automata model is shown. Here $m = 10$, $n = 9$, $o = 3$. State 1 represents outbreak, state 0 represents crash and state -1 represents buildup. a) migration rate is 0 and hence both patches oscillate independently. At intermediate values of migration ((b) and (c)), we see both patches out of phase. At higher values of migration i.e. (d), we see both patches closer to being in phase.

7. Discussion and Conclusion

We analyze the robustness of the oscillations that arise when 2 patches, each having a host-parasitoid system, are coupled with density independent migration. Although, previous studies have shown the presence of in-phase and out of phase solutions in host-parasitoid systems Adler (1993), those models lacked important details such as effect of inter specific competition of hosts for resources or different parasitoid foraging behaviors (parasitism). It is now known that the outbreaking insect herbivores are often resource limited and thus including such interactions are key to the overall emergent dynamics. We surveyed the literature of host-parasitoid systems and used a suite of different functional forms to model intraspecific competition for resources among hosts and parasitism, relevant to the literature. Our analysis shows that the result i.e. the two patches oscillate out of phase when migration is low, is robust across the modelling choices for intraspecific competition and parasitism. Robustness across modelling choices is useful whenever direct comparison with data is lacking or difficult, which is often the case in host-parasitoid systems. This also presents avenues of further work where this robustness is mathematically shown starting from a modified version of Eq. (1).

In addition, we also show an approximate way to derive an analytical expression for length of a cycle (Eq. (4-5)), when the two patches oscillate out of phase. We define length of a cycle as the expected number of generations between the peaks of parasitoid population in a single patch. Our approach is different from the standard analyses, where Eq.(3) is linearized and eigenvalues are calculated. We used approximations grounded in our understanding of different stages (i.e. buildup, outbreak and crash) that a host parasitoid undergoes Umbanhowar and Hastings (2002) and found that our theoretical expressions are in agreement with the simulation results as shown in Figure 5. Further improvements to the analytical work can be made by including parasitoid migrations from the outbreaking patch to the patch undergoing

buildup, as that will give a better estimate for the onset of outbreak in the latter patch.

A range of different models can be employed to look at the questions studied here, the spatio-temporal dynamics of host-parasitoid systems. One way to demonstrate that results obtained are robust is by showing similar conclusions for different models with different underlying implicit assumptions. This can also determine what biological features lead to observed dynamics. Thus, lastly, we also analyzed if the oscillations we studied were rooted in the specific biological details of a host-parasitoid system. We use a 3 state cellular automata model devoid of any biological details (like competition, parasitism etc.), which qualitatively undergoes buildup, outbreak and crash for m, n and o time steps respectively. We show that as long as $m \approx n >> o$, the two patches oscillate out of phase when migration is low (Figure 6). Although similar cellular automata model have been used in studies before (see Hassell et al. (1991)), our work presents new insights. Our analysis shows that out of phase oscillations could arise as long as the population in each patch undergoes a cycle of 3 stages, where one stage (i.e. crash) is much smaller than the other two (buildup and outbreak). This result could also hold true for populations other than host-parasitoid system. Our work could be extended to a more general framework which could map oscillations across patches to oscillations in individual patches, a problem which is of great interest in metapopulation studies.

Declaration of Competing Interest

The authors declare that there was no competing interests, either financially or personally, that could influence the results presented in this work.

Code availability

All codes used for this project can be found here.

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Contributions

Research question developed by both. Analysis done by me with guidance by Alan. manuscript written by me, edited by both authors.

References

References

RM May, MP Hassell, RM Anderson, and DW Tonkyn. Density dependence in host-parasitoid models. *The Journal of Animal Ecology*, pages 855–865, 1981.

Amel Balla, Allaoua Silini, Hafsa Cherif-Silini, Ali Chenari Bouket, Warren Keith Moser, Justyna Anna Nowakowska, Tomasz Oszako, Farida Benia, and Lassaad Belbahri. The threat of pests and pathogens and the potential for biological control in forest ecosystems. *Forests*, 12(11):1579, 2021.

Ian L Boyd, PH Freer-Smith, Christopher A Gilligan, and Hugh Charles J Godfray. The consequence of tree pests and diseases for ecosystem services. *Science*, 342(6160): 1235773, 2013.

Kamal JK Gandhi and Daniel A Herms. Direct and indirect effects of alien insect herbivores on ecological processes and interactions in forests of eastern north america. *Biological Invasions*, 12:389–405, 2010.

A Liebhold and B Bentz. Insect disturbance and climate change. *US Department of Agriculture, Forest Service, Climate Change Resource Center. www. fs. usda. gov/ccrc/topics/insectdisturbance/insect-disturbance*, 2011.

Alison F Hunter and Greg Dwyer. Outbreaks and interacting factors: insect population explosions synthesized and dissected. *Integrative Biology: Issues, News, and Reviews: Published in Association with The Society for Integrative and Comparative Biology*, 1(5):166–177, 1998.

Pedro Barbosa, Jack C Schultz, et al. *Insect outbreaks.* Academic Press, Inc., 1987.

Judith H Myers. Can a general hypothesis explain population cycles of forest lepidoptera? In *Advances in Ecological Research*, volume 18, pages 179–242. Elsevier, 1988.

James Umbanhowar and Alan Hastings. The impact of resource limitation and the phenology of parasitoid attack on the duration of insect herbivore outbreaks. *Theoretical Population Biology*, 62(3):259–269, 2002.

David T Briese et al. Classical biological control. *Australian weed management systems*, pages 161–192, 2000.

LE Caltagirone. Landmark examples in classical biological control. *Annual Review of Entomology*, 26(1):213–232, 1981.

MP Hassell and JK Waage. Host-parasitoid population interactions. *Annual review of entomology*, 29(1):89–114, 1984.

JK Waage and MP Hassell. Parasitoids as biological control agents–a fundamental approach. *Parasitology*, 84(4):241–268, 1982.

Zhi-Zhi Wang, Yin-Quan Liu, SHI Min, Jian-Hua Huang, and Xue-Xin Chen. Parasitoid wasps as effective biological control agents. *Journal of Integrative Agriculture*, 18(4):705–715, 2019.

Hugh CJ Godfray, Michael P Hassell, and Robert D Holt. The population dynamic consequences of phenological asynchrony between parasitoids and their hosts. *Journal of Animal Ecology*, pages 1–10, 1994.

Michael R Strand and John J Obrycki. Host specificity of insect parasitoids and predators. *BioScience*, 46(6):422–429, 1996.

Nicholas J Mills and Wayne M Getz. Modelling the biological control of insect pests: a review of host-parasitoid models. *Ecological modelling*, 92(2-3):121–143, 1996.

Karen C Abbott and Greg Dwyer. Using mechanistic models to understand synchrony in forest insect populations: the north american gypsy moth as a case study. *The American Naturalist*, 172(5):613–624, 2008.

Andrew Liebhold, Walter D Koenig, and Ottar N Bjørnstad. Spatial synchrony in population dynamics. *Annu. Rev. Ecol. Evol. Syst.*, 35: 467–490, 2004.

Michael P Hassell, Hugh N Comins, and Robert M Mayt. Spatial structure and chaos in insect population dynamics. *Nature*, 353 (6341):255–258, 1991.

Frederick R Adler. Migration alone can produce persistence of host-parasitoid models. *The American Naturalist*, 141(4):642–650, 1993.