Predator evolution in space: Self-shading and complex emergent spatial structure lead to novel effects of ecology

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Abstract

Predation interactions are an important element of ecological communities. Population spatial structure has been shown to influence predator evolution, resulting in the evolution of a reduced predator attack rate; however, its influence on the evolutionary role of traits governing predator and prey ecology is unknown. The evolutionary effect of spatial structure on predators has primarily been explored assuming a fixed metapopulation spatial structure, and understood in terms of group selection. But endogenously generated, emergent spatial structure is common in nature. Furthermore, the evolutionary influence of ecological traits may be mediated through the spatial self-structuring process. Drawing from theory on pathogens, the evolutionary effect of emergent spatial structure can be understood in terms of "self-shading," where a voracious predator limits its long-term invasion potential by reducing local prey availability. Here we formalize the effects of "self-shading" for predators using spatial moment equations. Then, through simulations, we show that in a spatial context "self-shading" leads to relationships between predator-prey ecology and the predator's attack rate that are not expected in a non-spatial context. Some relationships are analogous to relationships already shown for host-pathogen interactions, but others represent new trait dimensions. Finally, since understanding the effects of ecology using existing "self-shading" theory requires simplifications of the emergent spatial structure that do not apply well here, we also develop metrics describing the complex spatial structure of the predator and prey populations to help us explain the evolutionary effect of predator and prey ecology in the context of "self-shading." The identification of these metrics may provide a step towards expansion of the predictive domain of self-shading theory to more complex spatial dynamics.

Key Words

Predator-prey interactions, population spatial structure, evolution, attack rate, “self-shading”

1. Introduction

Predation interactions are a cornerstone of ecological communities. Predators are not only ubiquitous, but also often play the role of keystone species (see Table 1 in [1]), dramatically influencing patterns of community structure including prey distribution, abundance, size, and diversity [2, 3]. In addition, predators are the tools for biological pest control. A predator’s attack rate is a key component of the predation interaction and varies between species (e.g. [4-6]) as well as between populations of the same species (e.g. [7, 8]). Understanding the ecological and evolutionary processes generating variation in predator attack rates thus has important implications for the management of natural and agricultural ecosystems [9, 10].

Population spatial structure, where individuals do not interact randomly but rather according to their spatial proximity, has long been recognized to play an important role in the population dynamics of species, including predators and their prey [11-14]. In addition, spatial structure can influence the evolutionary trajectory of a single species [15-18], as well as interacting species (competing species: [19, 20]; predators: [21-24]; pathogens: [25-31]; parasitoids: [32]). In evolutionary studies, spatial structure is primarily recognized for changing the outcome of selection on a particular trait. For example, in a non-spatial context competing species are expected to evolve maximal growth rates, but in a spatial context they may evolve reduced growth rates [19]. Equally important however, spatial structure can lead to novel evolutionary effects of other traits that characterize the ecology of the species, unexpected in non-spatial contexts. For example, in a non-spatial context, a pathogen's evolutionary stable (ES) transmission rate is not predicted to depend on the reproduction rate of its host, but in a spatial

context, the ES pathogen transmission rate systematically varies with the host reproduction rate [26].

For predators, theory has convincingly shown that spatial structure can lead to the evolution of a reduced predator attack rate [21-24] (in the absence of spatial structure other mechanisms can also constrain predator attack rate evolution; see [33]). Yet little is known about how other aspects of predator or prey ecology influence predator attack rate evolution in a spatial context. Furthermore, the evolutionary effect of spatial structure on a predator's attack rate has primarily been understood through the lens of group selection, where population spatial structure is metapopulation-like [21-24]. The group level framework is not well suited for understanding the effects of population spatial structure that arises endogenously from the behavior of the organisms themselves [34], where groups are not strictly defined. Not only have model predictions been shown to change when spatial structure is modeled at the level of the individual [35], but also the evolutionary influence of some ecological traits may be mediated through the spatial self-structuring process and thus would be missed by a group level framework. The arguably frequent occurrence of emergent spatial self-structuring in the real world (examples in [36-39]) thus begs an alternative framework.

The evolutionary effect of emergent spatial structure has principally been studied in the context of pathogen-host interactions, where "self-shading" [30] has been identified as a critical process influencing pathogen evolution. For a simple spatial lattice model of host-pathogen interactions, the fitness of a rare invading pathogen is a function of its short-term impact on the local environment [30], suggesting a powerfully simple explanation for the evolution of "prudent" pathogens in a spatial context. When pathogen-host interactions are localized, a pathogen with a high transmission rate can quickly deplete the local availability of susceptible

hosts, hindering its long-term spread and potentially sabotaging its invasion. Such "self-shading" is likely to play a similarly important role in the evolution of other exploitative species including predators, but this has not yet been formally shown.

The "self-shading" framework can be formalized using spatial moment equations, which are coupled equations that describe the mean density and spatial arrangement of individuals. In addition to providing a general understanding, spatial moment equations have further been used to provide insight into the evolutionary influence of pathogen and host ecology [26]. However, this additional theoretical development requires the use of a spatial moment closure approximation method that ignores spatial correlations at a scale above neighboring individuals. For pathogens, predictions based on the most common approximation methods only consistently agree with the results of simulations in the special case of pathogens subject to a transmission-virulence tradeoff [26, 31, 40]; otherwise, it appears that the effect of more complex, larger-scale spatial correlations on "self-shading" is important [26]. Predators are not subject to the analogy of a transmission-virulence tradeoff (i.e. predators with higher attack rates also tend to have higher death rates). Therefore, larger-scale spatial correlations are likely to be important, and by extension, this approach is unlikely to be useful for understanding the evolutionary influence of predator and prey ecology. A potentially fruitful alternative is to use stochastic simulations to study spatial characteristics of predator-prey populations that account for more complex emergent spatial structure.

In this paper we formally extend the "self-shading" concept to predators and elucidate the evolutionary effect of traits governing predator and prey ecology in a spatial context when population spatial structure is endogenously generated. We focus on an individual-based Lotka-Volterra predator-prey model with spatially localized interactions. The model incorporates two

1 important aspects of predator ecology that are not typical of pathogen-host interactions: the prey-
2 to-predator conversion efficiency and predator movement. Though simple relative to real-world
3 predator prey systems, this model can tractably be formalized with spatial moment equations,
4 and provides a limiting case upon which to build future theory incorporating more complexity.
5 Furthermore, it is similar to typical spatially explicit pathogen-host models, allowing us to
6 compare the evolutionary effect of ecology in a spatial context across predators and pathogens.
7 Using this model, we first formalize the effects of "self-shading" using spatial moment
8 equations. We then use comprehensive spatial simulations to show the predicted impact of traits
9 governing predator and prey ecology on the evolution of predator attack rates, making
10 comparison to the equivalent non-spatial model. We delineate the novel effects of the prey-to-
11 predator conversion efficiency, the prey reproduction rate, the predator death rate, and the rate of
12 predator movement. These all provide new potential explanations for natural variation in
13 predator attack rates. We focus on the effects of the prey-to-predator conversion efficiency and
14 predator movement, which are absent from traditional pathogen-host interactions, highlighting
15 both their direct influence (the ES attack rate trades off with efficiency and increases with
16 movement) and also their influence on the effects of ecology that are analogous between models
17 of predator-prey and pathogen host interactions (the prey reproduction rate and predator death
18 rate, which are analogous to host reproduction and infected host death rates respectively).
19 Finally, we develop two metrics, characterizing the complex emergent spatial distribution of the
20 prey population at a larger spatial scale than neighboring individuals, and use them to help
21 explain the novel spatial evolutionary effects of predator and prey ecology in the context of
22 "self-shading."

23 **2. The model and extension of "self-shading" theory**

We utilize a relatively simple Lotka-Volterra-like predator-prey model where the prey and predator populations reside on a regular two-dimensional lattice. Lattice sites can be occupied by a predator (P), a prey (N), or neither (O). We assume that predator and prey cannot occupy the same lattice site. Prey and predator reproduction, prey consumption, and predator movement (when allowed) can only occur between the focal lattice site and the four directly adjacent lattice sites. The rate of reproduction for a given prey individual is equal to the intrinsic prey reproduction rate (r) times the number of empty sites next to it. Hence the carrying capacity of the prey population is set by the size of the lattice, here 22,500 sites. For equivalent pathogen-host models, this lattice size is sufficiently large to minimize the effects of accidental extinction [26]. The rate of prey consumption by a given predator individual is equal to the attack rate (α) times the number of prey next to it. Each time a predator consumes a prey individual it produces an offspring in the lattice site that was previously occupied by the prey with probability e (the prey-to-predator conversion efficiency; we assume e <1). Otherwise that site remains empty. Unconsumed prey individuals die at a rate d and predator individuals die at a rate m. When predator movement is allowed, the rate at which a predator individual moves to an adjacent empty site is m_p times the number of empty sites next to it, where m_p is meant to reflect an intrinsic movement ability. We assume for simplicity that the choice of destination sites among multiple empty sites is random.

This type of model is appropriate for sedentary prey whose offspring do not disperse far from their parent. A canonical example is aphids, and many other sap-sucking insects. Another example is cyanobacteria filaments, which often attach and grow on submerged or moist surfaces. In reality, predators of sedentary prey species are unlikely to also be sedentary (have a movement rate of 0); however, examining the limiting case of no predator movement is useful

for understanding the effect of non-zero movement rates. Furthermore, though physical impediment of predator movement by the prey (an implicit assumption of our model, since lattice sites can only be occupied by one individual) is not a general feature of predator-prey interactions, neither is it unrealistic. Consider for example a predator that is similarly sized to its prey, as in the case of some insect predators of aphids. This simplification enables us to formulate the model in terms of spatial moment equations and show that predator fitness is governed by "self-shading." It has the further advantage that in the limit of zero movement (m_p=0), perfect efficiency (e=1), and d/m<1 (predators die more quickly than prey), our model is equivalent to simple lattice models of host-pathogen interactions (e.g. [26, 28, 30-31]). This provides a comparison point to assess the effect of ecology unique to predators.

The dynamics of the model can be quantified by spatial moment equations, which describe the rate of change in the state of pairs of lattice sites. In this model, there are 9 possible state pairs (N-P, N-O, N-N, etc.). The dynamics of the predator and prey alone can be recovered by summing across the spatial moment equations for the appropriate pairs (e.g. for predators, one would sum the rate of change in P-N, P-P, and P-O pairs). The spatial invasion fitness of the predator is the per-capita growth rate of a predator invading a population at equilibrium with a resident predator (eq. 1, see supplement 1 for full analysis).

$$(1) \qquad \frac{1}{P}\cdot\frac{dP}{dt} = \alpha\cdot e\cdot(\hat{q}^{o}_{N|P} - \hat{q}_{N|P})$$

In this equation, P is the density of predators on the lattice, $\hat{q}_{N|P}$ is the equilibrium average density of prey in the neighborhood of a predator (equal to $m/\alpha e$), $\hat{q}^{o}_{N|P}$ is the quasi-equilibrium average density of prey in the neighborhood of a predator (i.e. the average density of prey in the neighborhood of the predator shortly after invasion, during which time the invader has re-shaped

it's local environment but the global equilibrium environment is relatively unchanged), and other parameters are as defined above.

The equivalent of eq. 1 that has been derived for pathogens [30] is slightly different in structure because it is derived from a model that allows some non-local, random interactions, and because it presents $\hat{q}_{N|P}$ in terms of model parameters (i.e. $m/\alpha e$). Our version allows us to understand a predator's invasion fitness through analogy with R^* theory, which predicts that of two competing species, the one that maintains a lower equilibrium level of resource (R^*) will be able to invade the other and not vice versa [41, 42]. In short, at its own R^*, a species population growth rate is 0. If the level of resource is greater than R^*, the growth rate is positive, and if it is lower than R^*, the growth rate is negative. Thus, given a resident species at equilibrium with the resource, an invading species with an R^* lower than that of the resident is able to increase in abundance. This depletes the resources such that the resident species will simultaneously begin to decrease in abundance. Specifically, R^* theory predicts that the invasion growth rate is equal to the R^* of the resident minus the R^* of the invader. For predators, the resource is the prey and, in a lattice model like ours, only local availability matters. Hence R^* theory would predict that the invasion growth rate is proportional to the local equilibrium prey density of the resident predator, minus that of the invader. However, when interactions are local, the invading predator can quickly shape its local environment. Thus, it is not the resident's equilibrium density of prey that determines the long-term invasion growth rate, but the equilibrium density of prey shortly after invasion (the quasi-equilibrium density of prey, $\hat{q}^o_{N|P}$), and the invasion growth rate is proportional to $\hat{q}^o_{N|P} - \hat{q}_{N|P}$. When the invading predator reduces the quasi-equilibrium density of neighboring prey to its own equilibrium value, or lower, the resource is less abundant than

necessary to maintain a positive growth rate, and the invasion fails. This phenomenon is called "self-shading."

The "self-shading" perspective embodied by eq. 3 shows that ecological traits could have important evolutionary influences through their impact on the self-structuring process. First, the predator's death rate and efficiency can influence the evolutionary outcome through the equilibrium density of prey neighboring an invading predator ($\hat{q}_{N|P} = m/\alpha e$). Less obvious, these and other predator and prey ecological traits (specifically predator movement, and prey reproduction and death) can additionally influence the evolutionary outcome through the quasi-equilibrium density of prey ($\hat{q}^{o}_{N|P}$). The quasi-equilibrium density is affected by the spatial structure of the predator and prey population prior to invasion (the "invasion context"), and the impact of the invading predator on the local prey population over a short time frame, both of which can be influenced by predator and prey ecological traits. Indeed, various ecological traits have been shown to influence the evolution of pathogens under spatial self-structuring [26, 28]. In contrast, invasion analysis of a non-spatial model, equivalent to our spatial model except that interactions between individuals are random rather than spatially dependent, predicts that predators evolve an increasing attack rate, to the point of prey and predator extinction, regardless of predator and prey ecology (see supplement 2).

To derive more quantitative relationships between ecological traits and a predator's invasion fitness (eq. 1), one must solve for the quasi-equilibrium local density of prey ($\hat{q}^{o}_{N|P}$), a complex task requiring numerical methods and a spatial moment closure approximation (for a description of the derivation, see supplement 3). Common approximations that ignore complex larger-scale spatial correlations have been shown to be inaccurate except in special cases [26, 28, 31, 40], which do not apply to predator-prey interactions. Hence an alternative approach, that

incorporates larger-scale spatial structure, is needed. Here we use simulations to determine the effect of ecological traits on the ES attack rate. Furthermore, we use the simulations to determine the effect of ecological traits on key larger-scale spatial characteristics of the invasion context. By comparing these, we are able to provide an explanation of how the traits influence evolutionary outcomes, through their effect on the emergent spatial patterning.

3. Model simulations

To simulate the model, prey reproduction and death, and predator consumption and death events are modeled according to a Poisson process using the Gillespie algorithm [43] implemented in C++. To find the predator's ES attack rate, we assume that there is some chance that the offspring of a predator is a mutant with an attack rate slightly different than the parent. The average attack rate of the predator population thus changes over time. When the average attack rate is stable (a linear regression slope over 1000 prey generations of between -0.001 and 0.001), we take the average attack rate over the last 1000 prey generations as the ES attack rate. Other methods of inferring the ES strategy yield the same results (see [26]).

To determine the effects of ecological parameters on the ES attack rate, we calculated the ES attack rate across a range of prey reproduction rates (up to r=50), for several predator to prey death rate ratios (d/m=1/3, 1/2, 1, and 2), several prey-to-predator conversion efficiencies (efficiency; e=0.2, 0.5, and 1), and a range of predator movement rates (up to m_p=100). (For a comparison of these parameters with values measured in real systems, see supplement 4). We compared trends between the ES attack rate and different demographic rates with the special case of e=1, d/m<1, and m_p=0, which corresponds to a pathogen-host-type interaction.

To quantify the influence of different ecological traits on the "invasion context" (i.e. the equilibrium spatial structure determined by the resident predator and prey), we note that at

equilibrium the prey and predator populations are fragmented into distinct, dynamic clusters of prey and predators separated by empty sites (Fig. 1). We hypothesize that the "invasion context" affects the quasi-equilibrium local density of prey experienced by an invader ($\hat{q}^{o}_{N|P}$) via two key quantities: the size of prey clusters and the frequency with which they join. We expect that in an invasion context where prey clusters are larger, an invading predator is less likely to reduce the local density of prey below its own equilibrium density, which would lead to negative population growth. Likewise, in an invasion context where prey clusters join more frequently an invading predator has greater access to prey outside its immediate cluster and is less likely to reduce the local density of prey below its own equilibrium density. Thus, larger, more frequently joining clusters should reduce "self-shading," and, in the absence of other effects, the invasion process should lead to a higher ES attack rate.

To quantify the size of prey patches and the frequency with which they join, we carried out non-evolutionary simulations where the predator attack rate was fixed at an intermediate value that results in endemism across the relevant ecological parameter space. Across simulations we varied the relevant ecological trait, and measured the average prey cluster size and the rate at which prey clusters join together (details in supplement 5). Guided by these trends and considering the influences that these ecological traits might have on the short-term invasion dynamics, we provide an explanation of the influences of ecological traits on attack rate evolution in the context of "self-shading."

4. Simulation Results

4.1 Predator and prey ecology

In contrast to the evolutionary prediction of a non-spatial model that the predator attack rate increases to the point of prey and predator extinction model regardless of predator and prey

ecology (demonstrated in supplement 2), our spatial simulations predict that the ES attack rate is below the predator-driven extinction threshold (see supplement 6), and that predator efficiency, predator movement, the ratio of predator to prey death rate, and the prey reproduction rate all influence the ES attack rate (Figs. 2 & 3 and supplement 7).

In the absence of predator movement, the relationship between the predator's ES attack rate and the efficiency is a power law-like tradeoff; lower efficiencies lead to higher ES attack rates (Fig. 2), regardless of the host reproduction rate and predator to prey death rate ratio. Also with no predator movement, the ES attack rate decreases with the prey reproduction rate, then at a critical point begins to increase (Fig. 2), The predator death rate has a more consistent influence, generally increasing the ES attack rate (see Fig. 2 and supplement 7), although there is some evidence that it decreases the ES attack rate for very high prey reproduction rates.

The pathogen analogues to the trends with prey reproduction and predator death rate without predator movement have recently been reported [26]. We find that these qualitative trends are unaffected by the ratio of predator to prey death rates (d/m) or efficiency (e), demonstrating a congruence between predator-prey and pathogen-host type interactions. However, there are quantitative differences, which can be seen from a detailed examination of Fig. 2 and supplement 7.

Simulations with predator movement show that increased movement generally increases the ES attack rate (Fig. 3), with some exceptions at low and high reproduction rates, for low and high movement respectively. Movement does not change the qualitative relationship between the ES attack rate and efficiency, but does change the evolutionary influence of the prey reproduction rate (Fig. 4). Specifically, higher movement rates remove the initial decrease in the ES attack rate that characterizes the relationship at low or no movement.

4.2 Spatial population distribution and dynamics and explanation of the effects of ecology

Increasing prey reproduction initially decreases and subsequently increases the prey cluster join rate and the average prey cluster size (Fig. 5). This trend was speculated for pathogens in [26], but was not quantitatively shown. Recall that all else being equal, the effect of "self-shading" mediated through the invasion context is expected to be more severe when prey cluster sizes are small and highly isolated (a low cluster join rate). Thus, this data suggests that "self-shading" is most severe at intermediate prey reproduction rates and explains the qualitative trends shown in Fig. 2.

The effect of movement and efficiency on the prey cluster join rate and prey cluster size does not fully explain their effect on the ES attack rate; however, their effect on these spatial characteristics of the "invasion context" does help us understand their evolutionary influence in terms of "self-shading." In particular, their overall effect on the ES predator attack rate is a balance of effects mediated by the "invasion context" and effects mediated by the invading predator, both of which influence the quasi-equilibrium prey density and hence the severity of "self-shading."

Predator movement decreases the prey cluster join rate and average prey cluster size for high and low efficiency (Fig. 5). Thus, movement mediated by the "invasion context" increases the risk of "self-shading," potentially decreasing the ES attack rate. However, this does not completely explain the trends in Fig. 3. The disparity is perhaps due to the effect of movement on the risk of "self-shading" mediated by the invader. In particular, movement can increase the average neighborhood density of prey experienced by a predator by allowing predators to move into neighboring empty sites that may have neighboring prey. Thus movement mediated by the invader can decrease the risk of "self-shading," potentially increasing the ES attack rate. Though

we have not measured the effect of movement through the invader, we can infer from the trends in the ES attack rate with movement that in most cases it is sufficient to counter the effect of movement mediated by the "invasion context." One exception is when reproduction and movement are very low. This may be because the effect of movement on the invasion context is most dramatic when reproduction is low (when r=5 and e=0.5, the decrease in average clusters size from m_p=0 to m_p =5 is 78.5; in contrast, when r=20, the average cluster size slightly increases by 0.5). The other exception is when reproduction is high and movement rates are intermediate to high. This may be because in this region of parameter space, clusters are small and widely separated such that the risk of "self-shading" is too high to be countered by any amount of movement.

Increasing efficiency has inconsistent effects on prey cluster size and cluster join rate across a range of prey reproduction rates (Fig. 5), in particular leading to increases at low and high prey reproduction. Yet it always decreases the ES attack rate, regardless of prey reproduction (Fig. 2). This indicates that, as with movement, the effect of efficiency is mediated by another factor that dominates over the invasion context, at least at low and high prey reproduction. We explain in greater depth in supplement 8 a potential effect of efficiency mediated by the invader that would explain the trends in the attack rate. Essentially, by pre-empting empty space with its own offspring, a more efficient predator might inhibit prey replenishment within a cluster, intensifying "self-shading".

5. Discussion

Our extension of the "self-shading" concept to predators using spatial moment equations expands the sphere of fitness concepts at our disposal for understanding predator evolution in space. The formerly predominant group selection fitness concept involves a group or "patch"

fitness component, and predicts that evolutionary outcomes are primarily determined by the average number of successful emigrants from "patches" containing the invading predator [44]. In contrast, the "self-shading" fitness concept is focused on the individual, but shortly after invasion, so that it incorporates an invading predator's effect on the local prey population. Hence it explicitly acknowledges that the predator's fitness has a temporal component [24], with the simplification that fitness shortly after invasion will predict long-term evolutionary outcomes. Though we believe that the group and "self-shading" fitness concepts are simply different versions of the same hypothesis regarding the drivers of evolution in a spatial context, the "self shading" fitness concept is more amenable to endogenous spatial structuring [34]. Thus, by extension, the "self-shading" framework is necessary for understanding and predicting the evolutionary effects of ecological traits that may act through that spatial self-structuring process. Though our analysis is based on a relatively simple model, it is an essential stepping-stone toward understanding much more complex models.

Our simulations reveal significant effects of predator and prey ecology not predicted by an equivalent non-spatial model. These effects provide potential explanations of variation in predator attack rates found in nature. For example, consider the predicted evolutionary effect of prey reproduction rate. Geographic variation in recruitment is common in many intertidal marine communities [45-47]. Though the observed variation has been proposed to lead to differences in predation rates through ecological mechanisms [48-50], our study suggests that it could also lead to differences in predation rates through evolutionary mechanisms. In fact, for at least one marine predator, a predatory snail, there is good evidence that observed geographic variation in the attack rate is genetically based [8]. While the larval ecology of many marine prey species would preclude the development of spatial structure, the larval ecology of some marine

predators (like snails) would allow it. Though it is unclear to what extent random prey recruitment (rather than spatial recruitment) would change the trend between reproduction rate and the ES attack rate, a previous study showed that random host reproduction did not fundamentally alter the qualitative effect of spatial structure on pathogen evolution [51].

In delineating the novel spatial evolutionary effects of ecology, we highlighted two predator traits with no analogue in pathogen-host theory: the prey-to-predator conversion efficiency and predator movement. Efficiency decreases the ES attack rate, and predator movement typically increases the ES attack rate (there are notable exceptions). We note that our model assumes that a predator's efficiency and attack rate are not related, whereas metabolic theory suggests they should be [52]. Should such a hard-wired relationship exist, it would override the spatially induced evolutionary effect of the efficiency. However, the shape of the relationship could have novel evolutionary effects of its own: for pathogens, the shape of the relationship between the pathogen's transmission rate and virulence has novel evolutionary effects in a spatial context [26].

In the absence of movement, traits with analogues in pathogen-host theory (the prey reproduction rate and predator death rate) have similar effects on the ES attack rate as would be extrapolated from studies specific to pathogens [26]. However, predator movement qualitatively changes the effects of the prey reproduction rate. The multifaceted effect of movement indicates that in extending evolutionary predictions across exploitative species one must be especially careful to consider differences in the spatial ecology of the species. For example, consider a parasitoid that differs from pathogens and predators in that it consumes only one prey per lifetime and is also able to actively seek out prey. According to our data, the former difference is

unlikely to influence the evolutionary outcomes across prey with different reproduction rates, but the latter is.

Though quantifying fitness using the “self-shading” framework is analytically challenging, we show that the framework is useful for gaining conceptual insight into the evolutionary influence of predator and prey ecology on the predator’s attack rate. Specifically, we develop two key metrics of the spatial distribution of the prey population (the mean prey cluster size and the prey cluster join rate) that characterize the "invasion context" and account for correlations at larger spatial scales than neighboring individuals. The effect of ecological traits on these metrics enables us to explain most of their effects on the ES attack rate in terms of the “self-shading” fitness concept. Although difficult, developing a more analytical understanding of these, and other, metrics may prove fruitful [53, 54]. For now, we provide a qualitative understanding of the trends in these metrics with ecology based on observations of simulations, briefly outlined below and illustrated in more detail in supplemental animations 1-3.

At equilibrium, prey clusters are characterized by a leading edge, where prey are reproducing and the cluster is expanding, and a lagging edge, where predators are consuming prey and reducing the cluster. The average size of prey clusters on the lattice and the frequency with which they join depends on the rate of cluster expansion (influenced by the prey reproduction rate) relative to reduction (influenced by the predator attack rate) as well as the prevalence of predators on the lattice (influenced by the prey reproduction rate and also by predator efficiency and movement). If predator consumption at the lagging edge of a cluster outpaces growth at the leading edge, the cluster is doomed to collapse and cluster size depends on its size when it first encounters a predator. On the other hand, if predator consumption is less than growth, the cluster can expand at the leading edge and cluster size depends on its size when

the leading edge catches up to the lagging or leading edge of another cluster. However, there are many other complexities that must be considered. For example, clusters that are more densely packed with prey can be consumed more quickly than equally sized clusters that have a more open structure (when the prey reproduction rate is low relative to prey death rate). In addition, the rate at which new clusters are generated relative to the rate at which they collapse significantly affects predator prevalence as well as the number of clusters and their distribution across the lattice.

6. Conclusions

Our study offers a significant first step in extending "self-shading" theory to the evolution of predators. It also suggests sources of variation in predation rates among predators that do not arise from standard non-spatial models, potentially explaining some of the variation among a wide range of predator species, from terrestrial to aquatic and from micro- to mega-fauna, in a unified way. Our study also points to the importance of developing theory of the evolutionary effects of ecology in models specifically designed for predators, by highlighting the effects of ecology novel to predators as compared to pathogens. Finally, our study suggests that we can advance our understanding of the effects of "self-shading" on predators through metrics that incorporate larger-scale spatial correlations at a scale determined by the self-structuring process. Given that predation interactions can exert a strong influence on community structure and stability, as well as their critical role in biological control, developing a better understanding of the processes shaping those interactions by for example further building "self-shading" theory seems a worth future research endeavor.

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Figure Legends

Figure 1. Example spatial distribution of predator and prey. Each image shows a snapshot in time of the predator and prey population on the lattice (red sites are predator, grey sites are prey, and black sites are empty).

Figure 2. Effect of prey conversion efficiency and prey reproduction rate on the evolutionary stable attack rate. Data points are the evolutionary stable attack rate predicted from spatial simulations. Efficiency data (left) are fit with a power law (r=5: $\alpha = 24.74e^{-2.2}$, R^2=0.969; r=10: $\alpha = 15.52e^{-0.65}$, R^2=0.994; r=20: $\alpha = 30.38e^{-0.982}$, R^2=0.952; r=50: $\alpha = 79.725e^{-0.52}$, R^2=0.991). On the right, filled circles represent pathogen-host type interactions ($d/m<1$ and e=1). Decreasing e (non-circle data points) and increasing d/m (shaded data points) does not alter the qualitative relationship. Other parameters: d=1.

Figure 3. Effect of predator movement on the evolutionary stable attack rate. Data points are the evolutionary stable attack rate from spatial simulations across different prey reproduction rates (with spline fit). (a) e=0.5, (b) e=1. In general, movement increases the ES attack rate, though note at high and low prey reproduction rates there is an initial or prolonged decrease in the ES attack rate. Other parameters: d=1, m=2.

Figure 4. Effect of predator movement rate on the relationship between prey reproduction rate and the evolutionary stable attack rate. Data points show the evolutionary stable attack rate predicted by spatial simulations across prey reproduction rates for different predator movement rates (m_p). When m_p=0, the ES α initially decreases with prey reproduction and then increases. As m_p increases, the initial dip disappears. Other parameters: (a) e=0.5; (b) e=1. Other parameters: d=1, m=2, r=20.

Figure 5. Effect of predator and prey ecology on prey cluster size and join rate. In the left column, data points are the average number of times two prey clusters join together per cluster per time at equilibrium and in the right column data points are the average size of prey clusters across the lattice at equilibrium. The first row shows the effect the prey reproduction rate for different predator attack rates (α; e=0.5, d=1, m=2). The second row shows the effect of the prey-to-predator conversion efficiency for different prey reproduction rates (α=40, d=1, m=2). The third row shows the effect of the predator movement rate for different prey-to-predator conversion efficiencies (for e=0.5, α=50 and for e=1, α=30; d=1, m=2, r=20).