

Title: Dynamic parasite aggregation reduces parasite regulation of host populations and the stability of host-parasite interactions

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Abstract

Macroparasites can have significant effects on the dynamics of host populations that include limiting the growth of a host population and altering the stability of a host-parasite equilibrium. An important factor determining the dynamical effects that parasites have on host populations 5 is how aggregated the distribution of parasites is across hosts in the population. While canonical host-macroparasite models assume that parasite aggregation is fixed, empirical and theoretical-work suggests that fixed aggregation is likely the exception rather than the rule. Here I assumed that parasite aggregation varies according to an empirically and theoretically-supported constraint-based model, in contrast to the canonical assumption of fixed parasite aggregation. I 10 then explored how this assumption alters the ability of parasites to regulate and suppress host populations, as well as stabilize the equilibrium of host-parasite interactions. The model with dynamic aggregation predicts that parasites are less likely to regulate host populations and the host-parasite equilibrium is less likely to be stable compared to the models that assume fixed aggregation. These results suggest that the theoretical ability of parasites to regulate host populations 15 and stabilize the host-parasite equilibrium may be more rare than previously thought, providing an additional explanation for the general lack of empirical evidence for parasites as a primary factor regulating host populations.

1 Introduction

Macroparasites can alter the dynamics of wildlife populations (Hudson *et al.*, 1998; Tompkins & Begon, 1999; Albon *et al.*, 2002). One notable way that macroparasites can do this is by suppressing host population abundance (Anderson & May, 1978; Rosà & Pugliese, 2002; Albon *et al.*, 2002; Tompkins *et al.*, 2002). In simplest terms this means that the presence of a parasite reduces a host population to a lower abundance than would be observed by a similar host population without the parasite. The ability of a parasite to suppress a host population has important implications 20 for managing wildlife populations of conservation and economic interest (Murdoch *et al.*, 1985; Peterson, 2004; Tompkins *et al.*, 2011).

In addition to suppressing the host population, particular macroparasite traits can stabilize or destabilize a host-parasite equilibrium (May & Anderson, 1978; Rosà & Pugliese, 2002; Tompkins *et al.*, 2002). For example, density-dependent interactions of parasites within a host can stabilize
30 the host-parasite equilibrium (Anderson & May, 1978), whereas parasite-induced reductions in host fecundity can destabilize the equilibrium (May & Anderson, 1978; Diekmann O. & Kretzschmar, 1991). While these different dynamical effects have been shown theoretically, empirical examples of parasites directly affecting host dynamics remain limited to a few high-profile studies (Scott, 1988; Hudson *et al.*, 1998; Albon *et al.*, 2002; Redpath *et al.*, 2006). In part, this is due to
35 the logistical difficulties of manipulative experiments that are needed to definitively link parasites to changes in host population dynamics (Tompkins *et al.*, 2002, 2011). Because of this difficulty, epidemiological models are critical for predicting the conditions under which parasites may be expected to suppress and stabilize host-parasite systems (Dobson & Hudson, 1992; Albon *et al.*, 2002; Tompkins *et al.*, 2002).

40 In addition, epidemiological models can provide insight into the conditions under which a parasite can regulate a host population. Following previous work in host-macroparasite systems, I specifically define regulation as the ability of a parasite to prevent a host population from growing without bound (Anderson & May, 1978; Rosà & Pugliese, 2002). While in reality other ecological factors will limit host population size if parasites fail to do so, this simple definition of
45 regulation is often used in host-macroparasite models (Diekmann O. & Kretzschmar, 1991; Albon *et al.*, 2002; Rosà & Pugliese, 2002). In this study I distinguish regulation from suppression, which refers to the ability of a parasite to reduce equilibrium host abundance, conditional on regulation. Particular attributes of macroparasites affect their ability to regulate a host population in models.
50 For example, increasing parasite reproductive rate increases the ability of parasites to regulate host populations, while increasing parasite virulence decreases the ability of parasites to regulate host populations (Anderson & May, 1978).

Parasite aggregation is an attribute of host-macroparasite systems that has implications for the potential of a parasite to regulate a host population (i.e. prevent unbounded growth), suppress a host population (i.e. reduce the equilibrium host abundance) and stabilize the host-parasite

55 equilibrium (Anderson & May, 1978; Kretzschmar & Adler, 1993). Aggregation is the nearly ubiquitous pattern in parasite ecology that many hosts have few parasites and few hosts have many parasites (Shaw & Dobson, 1995; Shaw *et al.*, 1998). Specifically, this means that host-parasite distributions often have a variance to mean ratio greater than one and are highly right-skewed. In host-parasite models, increasing aggregation decreases a parasite's ability to regulate
60 a host population, but, if regulation is possible, then increasing aggregation increases the stability of the host-parasite equilibrium, but decreases the suppression of the host population by the parasite (Anderson & May, 1978). The effect of aggregation on regulation, suppression, and stability also interacts with other processes operating in the host-parasite system. For example, highly aggregated and highly virulent parasites are unable to regulate a host population as many
65 parasites are removed from the population upon the death of a host, which occurs frequently (Anderson & May, 1978). On the other hand, if parasites reduce host fecundity, then higher levels of aggregation are needed to stabilize the host-parasite equilibrium (Tompkins *et al.*, 2002). Thus aggregation plays an important role in host-parasite dynamics both independently and through its interaction with other host- and parasite-related vital rates.

70 Many mechanisms can affect parasite aggregation, including clumped infections (Isham, 1995), parasite-induced mortality (Barbour & Pugliese, 2000), host heterogeneity (Wilson *et al.*, 2002; Gourbière *et al.*, 2015), and the balance between parasite immigration rate and birth rate (Fowler & Hollingsworth, 2016). Due to multiple interacting mechanisms shaping patterns of parasite aggregation, one might assume that predicting aggregation would require highly system-specific models. Surprisingly, this is not the case. Recent work has shown that because many
75 mechanisms interact to affect aggregation, general statistical rules can predict observed patterns of aggregation across different host-parasite systems (Wilber *et al.*, 2017). For example, simple constraint-based models developed in community ecology can predict patterns of aggregation across a range of amphibian (host)-trematode (macroparasite) systems (Wilber *et al.*, 2017).
80 Constraint-based models are rooted in the concept of maximum entropy and predict the most likely distribution given a set of constraints (Haegeman & Etienne, 2010; Harte, 2011; Locey & White, 2013; Harte & Newman, 2014). In host-parasite systems, there are two inherent constraints

on any observed parasite distribution: the total number of parasites P and the total number of hosts H in the distribution (Wilber *et al.*, 2017). This means that the only possible distribution of P
85 parasites amongst H hosts that could be observed is one that is consistent with these constraints. The most-likely distribution given these constraints is the one that can be realized in the largest number of ways (Jaynes, 1982).

An important prediction from constraint-based models is that the level of parasite aggregation changes as the total number of hosts H and parasites P change (Locey & McGlinn, 2013;
90 Johnson & Wilber, 2017). This is important because theoretical results have shown that when aggregation varies with H and P , the conditions for a stable equilibrium in host-parasite systems are altered, compared to the canonical assumption of fixed aggregation (Adler & Kretzschmar,
1992; Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002). For example, Kretzschmar & Adler
1993 showed that aggregation itself is not sufficient to stabilize a host-parasite system. Rather,
95 aggregation needs to be an increasing function of mean parasite load (P/H) to stabilize the host-parasite equilibrium. Despite this well-known importance of aggregation that depends on P and H , there is no theory that predicts the precise shape of this relationship. Constraint-based theory provides such a prediction.

In this study I explore the effects of the constraint-based aggregation on the dynamics of
100 host-parasite systems. In particular, this study asks two questions. First, how do constraint-based models for parasite aggregation affect the ability of a parasite to regulate and suppress a host population? Second, how does constraint-based aggregation affect the stability of the host-parasite equilibrium? This study shows that accounting for empirically-supported constraint-based models of aggregation substantially reduces the parameter space in which parasites are
105 predicted to regulate host populations and stabilize the host-parasite equilibrium, compared to the standard assumption of fixed aggregation.

2 Models

2.1 Overview

The goal of this study was to understand whether constraint-based aggregation changed how aggregation affected host-parasite dynamics, compared to the canonical assumption of fixed parasite aggregation. To this end, I extended a simple host-macroparasite model developed in previous studies (described below, Anderson & May, 1978; Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002). This simple model excludes important processes affecting host-macroparasite systems such as host heterogeneity (beyond that captured by heterogeneity in parasite intensity), clumped infections, adaptive immunity, and logistic host growth, all of which have been explored previously (Isham, 1995; Pugliese & Rosa, 1995; Pugliese *et al.*, 1998; Rosà & Pugliese, 2002). These processes were excluded to allow for direct comparison between the dynamics of host-macroparasite systems following constraint-based aggregation and those predicted by the canonical host-macroparasite model with fixed aggregation (Anderson & May, 1978; Rosà & Pugliese, 2002). By first analyzing the behavior of the simple model in the absence of the mechanisms mentioned above, the impact of constraint-based aggregation on host-parasite dynamics can be understood without the confounding effects of other stabilizing and destabilizing mechanisms.

In the following sections I describe two host-macroparasite models, one with fixed aggregation and one with aggregation following constraint-based predictions. I then compare and contrast predictions from the two models regarding the ability of a parasite to regulate and suppress the host population and stabilize the host-parasite equilibrium.

2.2 The host-macroparasite model

Consider the following host-macroparasite model where H is the abundance of hosts in a population and P is the abundance of directly reproducing macroparasites across all hosts in the population (Anderson & May, 1978; Diekmann O. & Kretzschmar, 1991; Rosà & Pugliese, 2002)

$$\frac{dH}{dt} = -dH - \alpha P + bH \sum_{i=0}^{\infty} r_i (1 - \xi)^i \quad (1)$$

$$\frac{dP}{dt} = -(d + \mu)P + \phi H - \alpha H \sum_{i=0}^{\infty} i^2 r_i \quad (2)$$

b is host birth rate, *d* is host death rate, α is the rate of parasite-induced host mortality, μ is parasite death rate, and ϕ is the rate at which parasites are acquired from the environment. ϕ takes the functional form $\frac{\lambda P}{H_0 + H}$, which is a result of assuming that dynamics of the free-living stage of
 135 the parasite occur on a much faster time scale than the within-host dynamics (Anderson & May, 1978; Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002). $\sum_{i=0}^{\infty} i^2 r_i = E[i^2]$ is the second moment of the host-parasite distribution where r_i is the probability of a host having *i* parasites. This term is a result of the assumption that parasite-induced death rate increases linearly with the number of parasites per host. $\sum_{i=0}^{\infty} r_i (1 - \xi)^i$ is the probability generating function of the host-parasite
 140 distribution where $0 \leq (1 - \xi) \leq 1$ is the multiplicative reduction of host fecundity due to the parasite ($\xi = 0$ is no reduction). This two dimensional system of equations is an approximation of an infinite series of ordinary differential equations (ODEs) that tracks the number of hosts with $i = 0, 1, \dots, \infty$ parasites (Anderson & May, 1978; Kretzschmar & Adler, 1993). The relationship between equations 1 and 2 and the infinite series of ODEs are discussed at length elsewhere
 145 (Rosà & Pugliese, 2002; Cornell, 2010).

This model makes a number of assumptions, two of which deserve immediate attention. First, the model assumes that there is no density-dependence in host birth or death rates. This means that host population size can only be regulated by parasites and, upon escaping parasite control, hosts will increase exponentially (for $b > d$, Anderson & May, 1978). This is a useful simplification for precisely defining parasite regulation – preventing a host population from increasing exponentially (Rosà & Pugliese, 2002). Second, the model makes the assumption that the host population is homogeneous. In reality host populations have stage- and age-structure and hosts vary in immunity and behavior, all of which have implications on host-parasite dynamics and the patterns of parasite aggregation (Pacala & Dobson, 1988; Isham, 1995; Chan & Isham, 1998;
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¹⁵⁵ Wilson *et al.*, 2002; Calabrese *et al.*, 2011; Johnson & Hoverman, 2014). However, because the goal is to make general conclusions about how constraint-based models of aggregation can affect host-parasite dynamics, I follow the example of similar theoretical studies and ignore host heterogeneity.

2.3 Modeling parasite aggregation

¹⁶⁰ To close the system of equations given above, it is often assumed that the parasite distribution follows a negative binomial distribution – a flexible two parameter distribution that fits observed, aggregated host-parasite distributions (Anderson & May, 1978; Shaw *et al.*, 1998). The second moment of a negative binomial distribution is given by $E[i^2] = \frac{P}{H} + \frac{P^2}{H^2} \frac{k+1}{k}$, where k is the aggregation parameter of the negative binomial distribution and decreasing k indicates increasing aggregation ($k \in (0, \infty)$). The probability generating function for a negative binomial distribution is given by $G(1 - \xi) = \sum_{i=0}^{\infty} r_i (1 - \xi)^i = \left(\frac{kH}{\xi P + kH} \right)^k$.

Plugging these two equations into 1 and 2 leads to the host-macroparasite model with fixed k (henceforth the Fixed k Model; Diekmann O. & Kretzschmar, 1991; Rosà & Pugliese, 2002)

$$\frac{dH}{dt} = -dH - \alpha P + bH \left(\frac{kH}{\xi P + kH} \right)^k \quad (3)$$

$$\frac{dP}{dt} = -(d + \mu)P + \phi H - \alpha H \left(\frac{P}{H} + \frac{P^2}{H^2} \frac{k+1}{k} \right) \quad (4)$$

¹⁷⁰ While assuming a fixed k is a common simplifying assumption (e.g. Dobson & Hudson, 1992; Townsend *et al.*, 2009), it is well-known that parasite aggregation can vary over time (Scott, 1987; Boag *et al.*, 2001). Deterministic host-parasite models have captured the dynamic nature of aggregation using two different approaches. The first approach uses a three dimensional approximation of the infinite series of differential equations such that aggregation (typically the variance to mean ratio) is explicitly modeled as a dynamic state variable (Adler & Krestzschmar, 1992; Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002). While useful, this approach does not obviate the need to assume some distribution (often a negative binomial) to close the three-

dimensional system of equations (Rosà & Pugliese, 2002; Cornell, 2010).

Another approach is to use the two dimensional approximation given by equations 1 and 2, assume a negative binomial distribution, and then allow aggregation (given by k) to be a generic function of the total number of parasites and the total number of hosts in a population (Kretzschmar & Adler, 1993). The advantage to this approach is flexibility – aggregation can be driven by both the dynamics of the system affecting H and P and the assumed relationship between k , H and P . However, this flexibility is also a disadvantage as the assumed relationship between k , P , and H is phenomenological (e.g. Kretzschmar & Adler, 1993). Because constraint-based models provide an empirically- and theoretically-supported relationship for how aggregation varies with P and H (Wilber *et al.*, 2017), they can be used to explore how the varying nature of aggregation affects the dynamics of a host-parasite system.

2.4 Incorporating constraint-based aggregation

Here I consider the constraint-based model known as “the partition model” for aggregation (Xiao *et al.*, 2015; Wilber *et al.*, 2017). The partition model predicts the most likely host-parasite distribution given the constraints of total host abundance H and total parasite abundance P , as well as unlabeled hosts and unlabeled parasites (see Haegeman & Etienne, 2010; Wilber *et al.*, 2017, for a complete description). In short, this means that, given P parasites and H hosts, there are only a finite number of ways that these parasites can be distributed among the hosts. One realized distribution of parasites among hosts is a configuration. Assuming unlabeled hosts and parasites means that each possible configuration is equally likely. While there is no known analytical solution for the partition model, it can easily be estimated via simulation (Loey & McGlinn, 2013). Fig 1A. shows five predicted host-parasite distributions from a partition model with different numbers of P parasites and H hosts. Perhaps not surprisingly, fitting a negative binomial distribution to the expected prediction from a partition model reveals that the general shape of the partition model can be approximately captured by a negative binomial distribution where k varies as a function of P and H (Fig. 1A.).

Assuming that the partition model can be approximated as a negative binomial distribu-

tion where k is a function of P and H , I computed the $k(P, H)$ surface using the following
205 steps. First, I drew 1000 random partitions (i.e. potential configurations of P parasites among
 H hosts) from all the combinations of partition models with $P = 3, \dots, 500$ and $H = 3, \dots, 500$
(248,004 combinations). For any given (P, H) pair, this resulting draw of partitions resulted in
matrix with 1000 rows (i.e. 1000 random partitions) and H columns. The probability distribution
of a given host having x parasites under the partition model with P and H is $p(x|P, H) =$
210 $\sum_{m \in F} p(x|m, P, H)p(m|P, H)$ where m is a single partition in the feasible set F . Given the 1000
samples, I approximated $p(x|P, H)$ with a negative binomial distribution by first flattening the
1000 $\times H$ matrix into a vector and then fitting this $n = 1000H$ “sample” to a negative binomial
distribution to estimate the aggregation parameter k using a corrected moment estimate for k
(Gregory & Woolhouse, 1993).

215 The above steps provided \hat{k} values for all discrete combinations of P and H on a 498 \times 498 grid
(P and H are both between 3 - 500). However, to use this surface with equations 1 and 2, \hat{k} needed
to be defined when P and H were not integers. To do this, I increased the resolution of the 498
 \times 498 grid such that P and H both had 10,000 equally spaced points between 3 and 500. I used
cubic polynomial interpolation to calculate \hat{k} for all $1 \times 10^8 (P, H)$ on the high resolution grid
220 (Jones *et al.*, 2013). On this high resolution grid, \hat{k} was less than two for 99.9% of the values and
generally only greater than two when H and P were less than or equal to 4. This is consistent
the vast majority of host-parasite systems where $k < 2$ (Shaw & Dobson, 1995; Shaw *et al.*, 1998).
After increasing the resolution, I removed the simulation noise in $\hat{k}(P, H)$ by applying a Gaussian
filter with $\sigma = 75$ (Jones *et al.*, 2013). The choice of σ led to a smooth surface and ensured that
225 all $\hat{k} < 2$. While the choice of σ affects the quantitative conclusions presented below (e.g. the
exact location of the boundaries in parameter space), the qualitative results remain consistent
across all values of σ that I explored. Finally, I assumed that any points beyond the boundary
of the surface had the same value as the nearest point on the boundary. This assumption, along
with $\sigma = 75$, led to $\hat{k} \approx 2$ for all values of $P < 3$ and $H < 3$, which is equivalent to a fixed k
230 assumption for these values of P and H .

Fig. 1B shows the predicted $\hat{k}(P, H)$ surface after increasing the resolution and applying the

Gaussian filter. The surface shows that the partition model predicts that k decreases (aggregation increases) with increasing H and k increases (aggregation decreases) with increasing P , unless P and H are small in which case k briefly decreases with increasing P . Using this predicted $\hat{k}(P, H)$ surface, in combination with the second moment of the negative binomial distribution given above, the second moment of the partition model can be approximated as

$$E[i^2] \approx (P/H) + \frac{P^2}{H^2} \frac{\hat{k}(P, H) + 1}{\hat{k}(P, H)} \quad (5)$$

and the generating function of the partition model as

$$G(1 - \xi) \approx \left(\frac{H\hat{k}(P, H)}{\xi P + H\hat{k}(P, H)} \right)^{\hat{k}(P, H)} \quad (6)$$

Plugging equation 5 and 6 back into equations 1-2 the host-macroparasite model with aggregation following the partition model (henceforth the Feasible k Model) is given by

$$\frac{dH}{dt} = -dH - \alpha P + bH \left(\frac{H\hat{k}(P, H)}{\xi P + H\hat{k}(P, H)} \right)^{\hat{k}(P, H)} \quad (7)$$

$$\frac{dP}{dt} = -(d + \mu)P + \phi H - \alpha H \left(\frac{P}{H} + \frac{P^2}{H^2} \frac{\hat{k}(P, H) + 1}{\hat{k}(P, H)} \right) \quad (8)$$

To answer the questions regarding how constraint-based aggregation affects the host-parasite dynamics, I compared the ability of parasites to regulate and suppress a host population as well as the stability of host-parasite equilibrium under both the Fixed k Model and the Feasible k Model. All analyses were performed in Python v. 2.7.12 and the code to replicate the analyses is provided at [link available from editor].

²⁴⁵ **3 Question 1: How do parasites regulate and suppress a host population?**

To facilitate comparison with previous results, I first assumed that parasites had no effect on host fecundity ($\xi = 0$) and only affected host mortality ($\alpha > 0$). I relaxed this assumption in the following section. Under this assumption, the Fixed k Model predicts that parasites are able to ²⁵⁰ regulate a host population when $\alpha \neq 0$ and $\lambda > b + \mu + \alpha + \frac{(b-d)}{k}$ (Anderson & May, 1978). As aggregation increases (k decreases) it becomes more difficult for parasites to regulate the host population (Fig. 2).

To compare this result to the ability of the Feasible k Model to regulate the host population, I simulated equations 7 and 8 using Euler's method with $\Delta t = 0.01$ and updated P , H and $\hat{k}(P, H)$ ²⁵⁵ for each Δt time step. I ran the model until either the host and parasite populations reached a finite attractor (regulation) or the host population increased above some large upper limit (no regulation).

The ability of a parasite to regulate a host population under the Feasible k Model was reduced compared the Fixed k Model (Fig. 2). However, when parasite reproductive rate λ was low and ²⁶⁰ parasites were highly aggregated under the Fixed k Model (e.g. $k = 0.2$), there were regions of the parameter space in which the parasites in the Fixed k Model did not regulate the host population, but those in the Feasible k Model did. This was only a small portion of the parameter space and as the reproductive rate of the parasite (λ) increased, the Feasible k Model again showed a reduced region in which parasites could regulate the host population (Fig. 2A-C).

²⁶⁵ These differences between the Fixed k Model and the Feasible k Model can be understood in terms of a positive feedback loop between aggregation and the total abundance of hosts. Under the Fixed k Model, an increase in α increases the equilibrium host abundance H^* (Fig. 3A), but has no corresponding affect on aggregation (Fig. 3C). Under the Feasible k Model an increase in α also leads to an increase in H^* (Fig. 3A), which generally leads to a corresponding decrease ²⁷⁰ in k (Fig. 3C). This increase in aggregation reduces the ability of the parasite to suppress the host population which leads to an increase in H^* , which in turn leads to a decrease in k . This

eventually leads to a complete inability of parasites to regulate the host population as small increases in H lead to large decreases in k when H is relatively small (e.g. $H < 100$, Fig. 1B, Fig. 3C).

As noted above and shown in Fig. 3, the ability of the parasite to suppress equilibrium host abundance also varies between the Fixed k Model and the Feasible k Model. For low values of parasite pathogenicity, the Feasible k Model predicts increased host suppression (i.e. reduced equilibrium host abundance) compared to a Fixed k Model with $k = 1$ (Fig. 3A). This is because the Feasible k Model predicts $k > 1$ for these low levels of parasite pathogenicity (Fig. 3C), which leads to increased suppression of the host population relative to $k = 1$. However, as pathogenicity increases the Feasible k Model predicts that k generally decreases (Fig. 3C). Eventually $k < 1$ and the Feasible k Model shows less suppression of host abundance than the Fixed k Model with $k = 1$. This lack of suppression can be further understood by examining how parasite efficiency, defined as the negative log percentage of unparasitized hosts divided by equilibrium parasite abundance (Singh *et al.*, 2009), changes between the Fixed k and Feasible k Model. Fig. 3D shows that while parasites are initially more efficient under the Feasible k Model than the Fixed k Model with $k = 1$, their level of efficiency decreases faster under the Feasible k Model with increasing pathogenicity. This decreasing efficiency with increasing pathogenicity leads to reduced host suppression under both models, but the faster decrease in efficiency under the Feasible k Model leads to parasites failing to regulate host populations at lower levels of parasite pathogenicity (α).

4 Question 2: How does dynamic aggregation affect the stability of the host-parasite equilibrium?

4.1 Stability properties without a parasite reduction in host fecundity ($\xi = 0$)

To understand the nature of the dynamics in the regulated region of Figure 2, I performed a local stability analysis on both the Fixed k Model and the Feasible k Model. I did this by numerically calculating the equilibrium of a given model under a particular set of parameters and

then examining characteristics of the Jacobian matrix at that equilibrium (Nisbet & Gurney, 1982). The results from the Fixed k Model are well-known – when a parasite with aggregation $0 < k < \infty$
300 is able to regulate a host population, the resulting system has a locally stable equilibrium (Fig. 2, Anderson & May, 1978). The Feasible k Model showed similar behavior – when the host-macroparasite system was regulated the resulting host-parasite equilibrium was locally stable for the entire parameter space considered (Fig. 2). Because the parameter space in which the parasite can regulate the host population is reduced under the Feasible k Model compared to the
305 Fixed k Model (Fig. 2), the parameter space in which the host-parasite equilibrium is stable is also reduced under the Feasible k Model.

The stability of the Feasible k Model is consistent with the observation that aggregation is a stabilizing mechanism in host-macroparasite systems (Anderson & May, 1978). While the Feasible k Model predicts that the parasite distribution varies with P and H , the distribution is almost
310 always aggregated (variance / mean > 1 , $k < 2$, Fig. 1). However, aggregation by itself is not sufficient to stabilize the equilibrium of a host-parasite interaction. Rather aggregation must be an increasing function of mean parasite load (Kretzschmar & Adler, 1993). The $\hat{k}(P, H)$ predicted by the partition model largely satisfies this stability criterion and therefore, given an equilibrium exists for a Feasible k Model and parasites do not affect host fecundity, this equilibrium should
315 be stable.

4.2 Stability properties with a parasite reduction in host fecundity ($\xi > 0$)

In the previous section I considered the stability of the host-parasite interaction when there was no parasite-induced reduction in host fecundity ($\xi = 0$). However, in many host-macroparasite systems parasites have larger effects on host fecundity than on host mortality (e.g. Tompkins
320 & Begon, 1999). Therefore, I also consider the stability and dynamics for both the Fixed k and Feasible k Model when $\xi > 0$.

Parasite-induced reduction in host fecundity is a well-known destabilizing mechanism in host-parasite systems (May & Anderson, 1978; Diekmann O. & Kretzschmar, 1991; Dobson & Hudson, 1992). Including parasite-induced reduction in fecundity into the Fixed k Model leads

325 to a region of the parameter space in which the host-macroparasite equilibrium exists, but is
unstable (to the left of the stability region in Fig. 4). Decreasing k (increasing aggregation)
reduces the region in which the equilibrium is unstable (reduces the area of the left hand region
in Fig. 3), but increases the region in which parasites are unable to regulate the host population
(right hand region of Fig. 4). Thus aggregation promotes the stability of the host-macroparasite
330 equilibrium, but decreases the overall parameter space in which an equilibrium can occur. This
result is consistent with the similar Fixed k Model explored by Diekmann O. & Kretzschmar
(1991).

In comparison to the Fixed k Model where $k \leq 1$ (which is an empirically realistic level of host-
macroparasite aggregation, Shaw & Dobson, 1995; Shaw *et al.*, 1998), the Feasible k Model predicts
335 a larger region in which the host-macroparasite has a positive equilibrium that is unstable and,
consistent with the results in the previous section, a larger region in which the host population is
unregulated (i.e. a finite, positive equilibrium does not exist, Fig. 4). This unstable region of the
parameter space either showed stable limit cycles or unlimited host growth under the Feasible k
Model, after perturbation from the equilibrium (Fig. 4). The reason for the increased region of
340 instability in the Feasible k Model compared to the Fixed k Model with $k \leq 1$ is because of the
dynamic link between the equilibrium number of hosts/parasites and parasite aggregation. In
the Feasible k Model, as parasite pathogenicity (α) decreases, the equilibrium number of hosts
also decreases (Fig. 3) and k generally increases (aggregation decreases, Fig. 3C). Increasing k
decreases the stability of the host-macroparasite equilibrium (May & Anderson, 1978) leading to
345 a reduced region in which the host-macroparasite equilibrium is stable.

5 Discussion

This study couples constraint-based models of parasite aggregation with dynamic host-macroparasite
models to show that when parasites follow empirically- and theoretically-supported predictions
of aggregation the ability of a parasite to regulate host populations and stabilize the host-parasite
350 equilibrium is reduced compared to the canonical assumption of fixed aggregation. These results
build on previous studies that show that assuming dynamic parasite aggregation can fundamen-

tally change the behavior of host-macroparasite systems (Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002). However, it has been unclear whether there is a consistent way that real host-macroparasite systems deviate from the assumption of fixed aggregation. Because constraint-based models provide a empirically-supported prediction regarding how parasite aggregation changes with P and H , they may provide a more reasonable starting point when modeling host-macroparasite dynamics than a fixed level of aggregation.

While the Feasible k Model explored here reduced the parameter space in which parasites could regulate host populations, this does not necessarily mean that parasites are less likely to regulate host populations in nature. This is because the reduced parameter space may still be the parameter space in which most host-macroparasite systems reside. In fact, many macroparasites have little effect on the survival probability of their hosts and instead affect other host vital rates such as reproduction (e.g. Dobson & Hudson, 1992; Tompkins & Begon, 1999). Moreover, many macroparasites have high reproductive rates (Anderson & May, 1991), which makes it easier for parasites to regulate host populations under the Feasible k Model. That being said, empirical evidence demonstrating that parasites alone can regulate a host population is uncommon (Scott, 1988; Tompkins *et al.*, 2002, 2011). While this lack of empirical evidence is augmented by the extreme logistical difficulties associated with empirically demonstrating that parasites regulate a host population (Scott & Dobson, 1989; Tompkins *et al.*, 2011), the models presented here suggest that common patterns of parasite aggregation might make the ability of parasites to regulate host populations more rare than previously thought.

In addition to reducing the ability of parasites to regulate a host population, the Feasible k Model, compared to the Fixed k Model, also reduced the stability of the host-parasite equilibrium when parasites reduced host fecundity. It is well-known that by reducing host fecundity macroparasites can augment cycles in host populations (Dobson & Hudson, 1992; Hudson *et al.*, 1998; Rosà & Pugliese, 2002). The Feasible k Model shows that when parasites reduce the fecundity of their host there is an increased region of the parameter space in which an unstable host-parasite equilibrium exists, compared to the assumptions from the Fixed k Model with k in an empirically realistic range of less than 1 (Shaw *et al.*, 1998). In some of this parameter space,

³⁸⁰ perturbations from the unstable Feasible k Model equilibrium resulted in population cycles when the Fixed k Model predicted a stable host-parasite equilibrium.

The propensity of fixed k models to underestimate the region of host-macroparasite cycles compared to more mechanistically based host-macroparasite models has been previously noted (Rosà & Pugliese, 2002). Rosà & Pugliese showed that fixed k models under-predicted the parameter regions in which oscillatory dynamics were expected if the true mechanism leading to aggregated parasite distributions was clumped infections. However, Rosà & Pugliese (2002) also showed that if heterogeneity in host susceptibility was the aggregating mechanism, then a Fixed k Model over-predicted the region of oscillatory dynamics. The Feasible k Model differs from the extensions explored by Rosà & Pugliese (2002) as the aggregation in the parasite distribution is not directly attributable to a specific process. However, because many different processes result in aggregation patterns that follow the partition model, the systematic under-prediction of oscillatory dynamics by the Fixed k Model may be a general result. On the other hand, both models ignore other well-known destabilizing mechanisms in host-macroparasite systems, such as time delays in the macroparasite life cycle (May & Anderson, 1978), such that a more mechanistic approach to parasite aggregation will be needed to understand how often the Fixed k Model over-predicts or under-predicts the stability of the host-parasite equilibrium.

This highlights a notable short-coming of the Feasible k Model: it does not directly model the mechanisms leading to parasite aggregation. This is a limitation because it is well-established that many host-parasite mechanisms affect aggregation (Anderson & Gordon, 1982). For example, increasing parasite pathogenicity α can in turn affect the shape of the host-parasite distribution (Crofton, 1971; Barbour & Pugliese, 2000), above and beyond any effects on aggregation via changes in the total number of parasites and the total number of hosts (Johnson & Wilber, 2017). While changes in aggregation independent of changes in P and H do occur in host-parasite systems (Johnson & Wilber, 2017), changes in aggregation are often well-described by changes in P and H (Wilber *et al.*, 2017). Therefore, the assumption that mechanisms affect aggregation primarily through their affects on P and H may not be unreasonable. Moreover, the Feasible k Model assumption is less stringent than the commonly used Fixed k Model (Anderson & May,

1978; Dobson & Hudson, 1992; Townsend *et al.*, 2009). By allowing the level of aggregation to change with P and H , while still maintaining the flexible negative binomial distribution, the Feasible k Model is mimicking a three-dimensional system of equations where the level of parasite aggregation is also included as a dynamic variable (Adler & Kretzschmar, 1992; Kretzschmar & Adler, 1993; Rosà & Pugliese, 2002).

All of these models (i.e. the Fixed k Model, the Feasible k Model, and the three dimensional model) assume that parasites instantaneously redistribute themselves to either maintain the same level of aggregation or change aggregation according to some function. Considering the biological realism of this assumption, it is clearly impossible for a intra-host helminth population to instantaneously redistribute itself following a change in the host or parasite population. While the biological realism and implications of this assumed instantaneous redistribution of macroparasites is rarely considered in host-macroparasite models, they are much-discussed topics in host-parasitoid systems (e.g. Murdoch & Stewart-Oaten, 1989; Rohani *et al.*, 1994). Depending on the assumptions made about the time scale at which parasitoids redistribute themselves among patches of hosts and whether this redistribution depends on host density, the strength of parasitoid aggregation as a stabilizing mechanism can change (Murdoch & Stewart-Oaten, 1989; Rohani *et al.*, 1994). For example, while density-dependent aggregation is a putative stabilizing mechanisms in discrete time host-parasitoid models (Hassell & May, 1973), Murdoch & Stewart-Oaten (1989) and Rohani *et al.* (1994) showed that this stabilizing effect was reduced when parasitoids were able to continually redistribute themselves among patches in response to host density within a season. This interaction between the stability properties of a system and the time scale at which putative stabilizing process are assumed to act has been shown in other models as well (e.g. Singh & Nisbet, 2007) and highlights that it is important to identify not just how macroparasite aggregation changes with P and H , but also the time scale at which this change in aggregation occurs after changes in P and H . Incorporating time delays in the response of aggregation to changes in host and parasite abundance is one possible approach to account for this mismatch in time scale. Depending on the nature of the time delay, it could potentially increase or decrease the predicted stability of the host-parasite equilibrium in the

Feasible k Model (Nunney, 1985).

These aforementioned challenges stem for the fact that the Feasible k Model, just like the Fixed k Model, is still only approximation of an “exact” model which explicitly tracks the number of hosts with $0, 1, 2, \dots, \infty$ parasites through time (either deterministically or stochastically, Rosà & Pugliese, 2002; Rosà *et al.*, 2003). Because of this the Feasible k Model will inevitably not be able to capture some of the dynamical properties that are seen under an exact model. One open question is how much the dynamics of these “exact” models differ from those of the Feasible k Model. Answering this question is challenging as the Feasible k Model implicitly assumes that “many” mechanisms are interacting to lead to emergent patterns of parasite aggregation predicted by the partition model, but it is not entirely clear how to explicitly model these “many” mechanisms. One approach that could prove useful is to construct individual-based models (IBMs) of empirically well-understood host-parasite systems where empirical parasite aggregation follows partition model predictions. These IBMs could be used to explore what combinations of mechanisms are needed to observe emergent patterns of aggregation consistent with the partition model and whether the resulting IBM dynamics are consistent with the Feasible k Model.

In conclusion, variable parasite aggregation can have large effects on the predicted dynamics of host-parasite systems. Constraint-based models provide an empirically and theoretically-supported way to account for the variation in parasite aggregation and show that models that assume fixed aggregation may over-predict both the ability of parasites to regulate a host population and the stability of the host-parasite equilibrium. Given the lack of evidence that macroparasites are the dominant factor regulating many host populations (Tompkins *et al.*, 2011), these results provide a theoretical rationale, to complement to often-discussed logistical rationale (Scott & Dobson, 1989; Tompkins *et al.*, 2011), that may help explain some of these null results.

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References

- Adler, F.R. & Krestzschmar, M. (1992) Aggregation and stability in parasite-host models. *Parasitology*, **104**, 199–205.
- Albon, S.D., Stien, a., Irvine, R.J., Langvatn, R., Ropstad, E. & Halvorsen, O. (2002) The role of parasites in the dynamics of a reindeer population. *Proceedings of the Royal Society B*, **269**, 1625–32.
- Anderson, R.M. & Gordon, D.M. (1982) Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. *Parasitology*, **85**, 373–398.
- Anderson, R.M. & May, R.M. (1978) Regulation and stability of host-parasite interactions: I. Regulatory processes. *Journal of Animal Ecology*, **47**, 219–247.
- Anderson, R.M. & May, R.M. (1991) *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, Oxford.
- Barbour, A.D. & Pugliese, A. (2000) On the variance-to-mean ratio in models of parasite distributions. *Advances in Applied Probability*, **32**, 701–719.
- Boag, B., Lello, J., Fenton, a., Tompkins, D.M. & Hudson, P.J. (2001) Patterns of parasite aggregation in the wild European rabbit (*Oryctolagus cuniculus*). *International Journal for Parasitology*, **31**, 1421–1428.
- Calabrese, J.M., Brunner, J.L. & Ostfeld, R.S. (2011) Partitioning the aggregation of parasites on hosts into intrinsic and extrinsic components via an extended Poisson-gamma mixture model. *PloS one*, **6**, e29215.
- Chan, M.S. & Isham, V.S. (1998) A stochastic model of schistosomiasis immuno-epidemiology. *Mathematical Biosciences*, **151**, 179–198.
- Cornell, S.J. (2010) Modelling stochastic transmission processes in helminth infections. E. Michael & R.C. Spear, eds., *Modelling Parasite Transmission and Control*, chapter 5, pp. 66–78. Landes Bioscience and Springer Science+Business Media, New York.
- Crofton, H.D. (1971) A quantitative approach to parasitism. *Parasitology*, **62**, 179–193.
- Diekmann O. & Kretzschmar, M. (1991) Patterns in the effects of infectious diseases on population growth. *J Math Biol*, **29**, 539–570.
- Dobson, A.P. & Hudson, P.J. (1992) Regulation and stability of a free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models. *Journal of Animal Ecology*, **61**, 487–498.
- Fowler, A.C. & Hollingsworth, T.D. (2016) The Dynamics of *Ascaris lumbricoides* Infections. *Bulletin of Mathematical Biology*, pp. 1–19.
- Gourbière, S., Morand, S. & Waxman, D. (2015) Fundamental factors determining the nature of parasite aggregation in hosts. *Plos One*, **10**, e0116893.

- Gregory, R.D. & Woolhouse, M.E.J. (1993) Quantification of parasite aggregation: A simulation study. *Acta Tropica*, **54**, 131–139.
- 500 Haegeman, B. & Etienne, R.S. (2010) Entropy maximization and the spatial distribution of species. *The American Naturalist*, **175**, E74–90.
- Harte, J. (2011) *Maximum Entropy and Ecology: A Theory of Abundance, Distribution, and Energetics*. Oxford University Press, Oxford, United Kingdom.
- 505 Harte, J. & Newman, E.A. (2014) Maximum information entropy: a foundation for ecological theory. *Trends in Ecology & Evolution*, **29**, 384–389.
- Hassell, M.P. & May, R.M. (1973) Stability in insect host-parasitoid models. *Journal of Animal Ecology*, **42**, 693–726.
- 510 Hudson, P.J., Dobson, a.P. & Newborn, D. (1998) Prevention of population cycles by parasite removal. *Science (New York, NY)*, **282**, 2256–2258.
- Isham, V. (1995) Stochastic models of host-macroparasite interaction. *The Annals of Applied Probability*, **5**, 720–740.
- Jaynes, E. (1982) On the rationale of maximum-entropy methods. *Proceedings of the IEEE*, **70**, 939–952.
- 515 Johnson, P.T.J. & Hoverman, J.T. (2014) Heterogeneous hosts: how variation in host size, behaviour and immunity affects parasite aggregation. *The Journal of Animal Ecology*, pp. 1–10.
- Johnson, P.T.J. & Wilber, M.Q. (2017) The biological basis of Taylor's Power Law revisited: using host-parasite interactions to reveal the drivers of aggregation. *Proceedings of the Royal Society B, In press*.
- 520 Jones, E., Oliphant, T. & Peterson, P. (2013) SciPy: Open Source Scientific Tools for Python.
- Kretzschmar, M. & Adler, F.R. (1993) Aggregated distributions in models for patchy populations. *Theoretical Population Biology*, **43**, 1–30.
- Locey, K.J. & McGlinn, D.J. (2013) Efficient algorithms for sampling feasible sets of macroecological patterns. *PeerJ*, pp. 1–23.
- 525 Locey, K.J. & White, E.P. (2013) How species richness and total abundance constrain the distribution of abundance. *Ecology Letters*, **16**, 1177–85.
- May, R.M. & Anderson, R.M. (1978) Regulation and stability of host-parasite population interactions: II. Destabilizing processes. *Journal of Animal Ecology*, **47**, 249–267.
- Murdoch, W.W., Chesson, J. & Chesson, P.L. (1985) Biological control in theory and practice.
- 530 Murdoch, W.W. & Stewart-Oaten, A. (1989) Aggregation by parasitoids and predators: effects on equilibrium and stability. *The American Naturalist*, **134**, 288–310.
- Nisbet, R.M. & Gurney, W.S.C. (1982) *Modelling Fluctuating Populations*. John Wiley and Sons, New York.

- Nunney, L. (1985) Short time delays in population models: A role in enhancing stability. *Ecology*, **66**, 1849–1858.
- Pacala, S.W. & Dobson, A.P. (1988) The relation between the number of parasites/host and host age: population dynamic causes and maximum likelihood estimation. *Parasitology*, **96**, 197–210.
- Peterson, M.J. (2004) Parasites and infectious diseases of prairie grouse: should managers be concerned? *Wildlife Society Bulletin*, **32**, 35–55.
- Pugliese, A., Rosà, R. & Damaggio, M.L. (1998) Analysis of model for macroparasitic infection with variable aggregation and clumped infections. *Journal of Mathematical Biology*, **36**, 419–47.
- Pugliese, A. & Rosa, R. (1995) A 2-dimensional model for macroparasitic infections in a host with logistic growth. *Journal of Biological Systems*, **3**, 833–849.
- Redpath, S.M., Mousseot, F., Leckie, F.M., Elston, D.A. & Hudson, P.J. (2006) Testing the role of parasites in driving the cyclic population dynamics of a gamebird. *Ecology Letters*, **9**, 410–418.
- Rohani, P., Godfray, H.C.J. & Hassell, M.P. (1994) Aggregation and the dynamics of host-parasitoid systems: a discrete-generation model with within-generation redistribution. *American Naturalist*, **144**, 491–509.
- Rosà, R. & Pugliese, A. (2002) Aggregation, stability, and oscillations in different models for host-macroparasite interactions. *Theoretical Population Biology*, **61**, 319–34.
- Rosà, R., Pugliese, A., Villani, A. & Rizzoli, A. (2003) Individual-based vs. deterministic models for macroparasites: host cycles and extinction. *Theoretical Population Biology*, **63**, 295–307.
- Scott, M.E. (1987) Temporal changes in aggregation: a laboratory study. *Parasitology*, **94**, 583–595.
- Scott, M.E. & Dobson, A. (1989) The role of parasites in regulating host abundance. *Parasitology Today*, **5**, 176–183.
- Scott, M.E. (1988) The impact of infection and disease on animal populations: implications for conservation biology. *Conservation Biology*, **2**, 40–56.
- Shaw, D.J. & Dobson, A.P. (1995) Patterns of macroparasite abundance and aggregation in wildlife populations: a quantitative review. *Parasitology*, **111**, 111–133.
- Shaw, D.J., Grenfell, B.T. & Dobson, A.P. (1998) Patterns of macroparasite aggregation in wildlife host populations. *Parasitology*, **117**, 597–610.
- Singh, A., Murdoch, W.W. & Nisbet, R.M. (2009) Skewed attacks, stability, and host suppression. *Ecology*, **90**, 1679–1686.
- Singh, A. & Nisbet, R.M. (2007) Semi-discrete host-parasitoid models. *Journal of Theoretical Biology*, **247**, 733–742.
- Tompkins, D.M. & Begon, M. (1999) Parasites can regulate wildlife populations. *Parasitology Today*, **15**, 311–313.

- 570 Tompkins, D.M., Dobson, A.P., Arneberg, P., Begon, M., Cattadori, I.M., Greenman, J.V., Heesterbeek, J.A.P., Hudson, P.J., Newborn, D., Pugliese, A., Rizzoli, A.P., Rosa, R., Rosso, F. & Wilson, K. (2002) Parasites and host population dynamics. P.J. Hudson, A. Rizzoli, B.T. Grenfell, H. Heesterbeek & A.P. Dobson, eds., *The Ecology of Wildlife Diseases*, chapter 3, pp. 45–62. Oxford University Press, Oxford.
- Tompkins, D.M., Dunn, A.M., Smith, M.J. & Telfer, S. (2011) Wildlife diseases: From individuals to ecosystems. *Journal of Animal Ecology*, **80**, 19–38.
- 575 Townsend, S.E., Newey, S., Thirgood, S.J., Matthews, L. & Haydon, D.T. (2009) Can parasites drive population cycles in mountain hares? *Proceedings Biological sciences / The Royal Society*, **276**, 1611–7.
- Wilber, M., Johnson, P.T.J. & Briggs, C.J. (2017) When can we infer mechanism from parasite aggregation? A constraint-based approach to disease ecology. *Ecology*, **98**, 688–702.
- 580 Wilson, K., Bjoernstad, O.N., Dobson, A.P., Merler, S., Poglayen, G., Read, A.F. & Skorping, A. (2002) Heterogeneities in macroparasite infections: patterns and processes. P.J. Hudson, A. Rizzoli, B. Grenfell, H. Heesterbeek & A. Dobson, eds., *The Ecology of Wildlife Diseases*, chapter 2, pp. 6–44. Oxford University Press, Oxford.
- Xiao, X., Locey, K.J. & White, E.P. (2015) A process-independent explanation for the general form of taylor's law. *The American Naturalist*, **186**, E51–60.

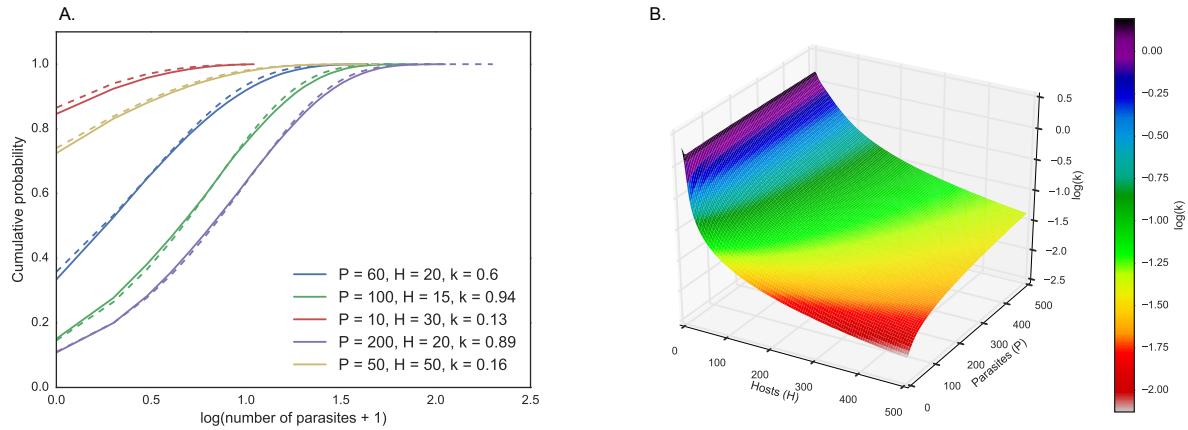


Figure 1: **A.** Predicted parasite distributions from the partition model with different numbers of parasites P and hosts H (solid lines) The corresponding best-fit negative binomial distributions are also plotted (dashed lines). The negative binomial distribution with varying levels of k can generally capture the aggregation predictions of the partition model. **B.** The surface $\hat{k}(P, H)$ as predicted by the partition model after interpolation and smoothing. For visual clarity, B. shows $\hat{k}(P, H)$ interpolated over 1000×1000 points with H and P between 3 and 500 rather than the $10,000 \times 10,000$ points used in the analyses.

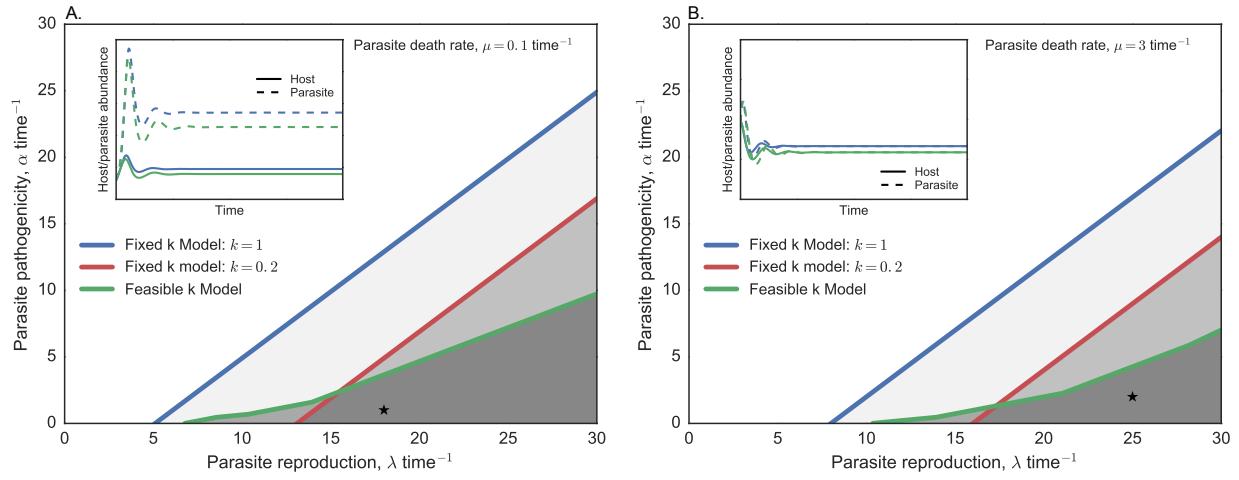


Figure 2: **A.** - **B.** The boundaries at which a parasite can regulate a host population under the Fixed k Model and the Feasible k Model. Below the line of a particular model is where a parasite can regulate a host population. For the parameter space shown, when a parasite was able to regulate the host population the system had a locally stable equilibrium (the host-parasite equilibria in all gray shaded regions are stable). The inset plots in A. and B. give examples of the host-parasite dynamics when the models have values of λ and α given by the black star in the respective plots. For clarity, the inset plots do not show the dynamical predictions for the Fixed k Model with $k = 0.2$. All other parameters are $\mu = 0.1$ and 3 time^{-1} , $H_0 = 10$, $b = 3 \text{ time}^{-1}$, $d = 1 \text{ time}^{-1}$, and $\xi = 0$.

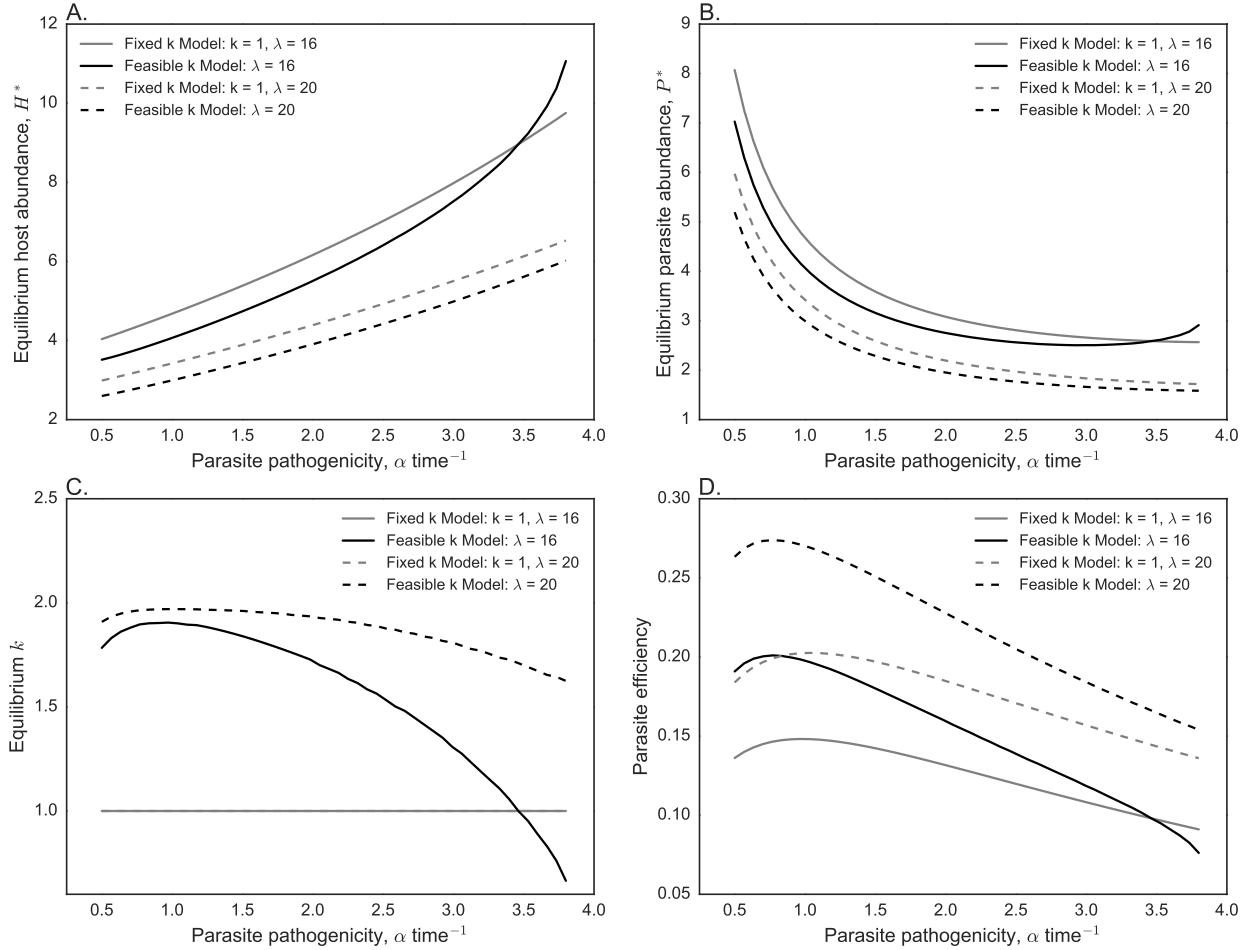


Figure 3: **A.** The host equilibrium under the Fixed k Model and the Feasible k Model with different values of parasite reproduction λ and varying levels of parasite pathogenicity α . **B.** The parasite equilibrium under the Fixed k and Feasible k Models. **C.** The equilibrium level of k under the Fixed k and Feasible k Models. The k for the Fixed k Model, by definition, does not change. **D.** Parasite efficiency, defined as the negative log percentage of unparasitized hosts divided by equilibrium parasite abundance, under the Fixed k and Feasible k Models. The other parameters are $\mu = 0.1 \text{ time}^{-1}$, $H_0 = 10$, $b = 3 \text{ time}^{-1}$, $d = 2 \text{ time}^{-1}$, and $\xi = 0$.

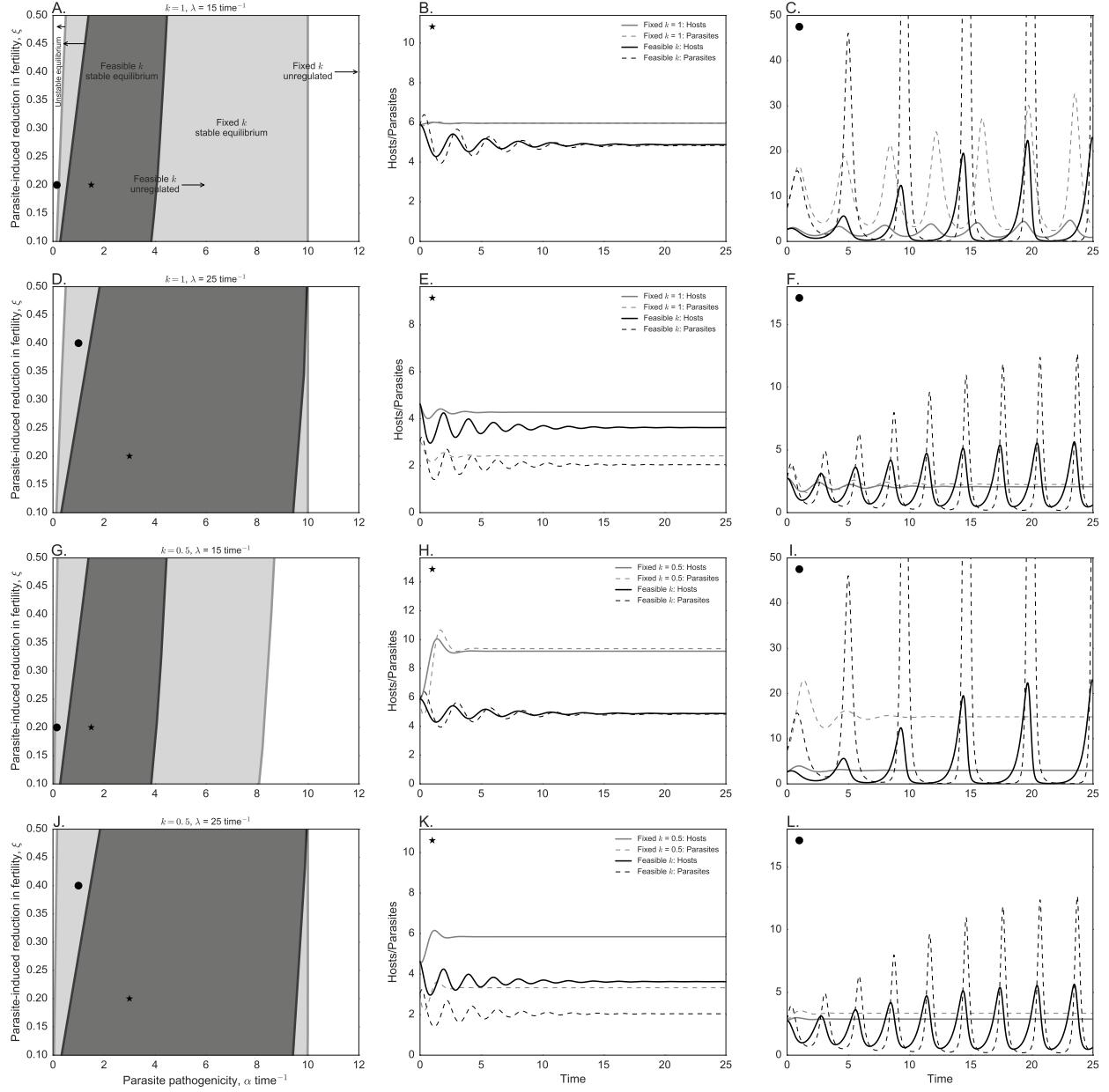


Figure 4: Stability properties and dynamics of the Fixed k and Feasible k Models when parasites cause a multiplicative, load-dependent reduction in host fecundity. A., D., G., J. The parameter space in which the host-parasite equilibrium is stable for varying parasite pathogenicity α , parasite reduction in fecundity ξ , parasite reproduction $\lambda = 10 \text{ time}^{-1}$, and k for the Feasible k and Fixed k Model. The dark gray regions show where the Feasible k Model has a stable equilibrium and the light gray regions show where the Fixed k Model has a stable equilibrium. To the right of the shaded regions the parasite fails to regulate the host population. To the left of the shaded regions the parasite can regulate the host, but the resulting equilibrium is unstable. The dynamics from these different regions are shown in B., C., E., F., H., I., K., L. which correspond to the black circles and stars shown in A., D., G., J.. The y-axis is truncated on C. and I. for clarity. All other parameters are $\mu = 0.1 \text{ time}^{-1}$, $H_0 = 10$, $b = 3 \text{ time}^{-1}$, and $d = 1 \text{ time}^{-1}$.