Ecology, 97(8), 2016, pp. 1994–2002

Cannibalism amplifies the spread of vertically transmitted pathogens

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Abstract. Cannibalism is a widespread behavior. Abundant empirical evidence demonstrates that cannibals incur a risk of contracting pathogenic infections when they consume infected conspecifics. However, current theory suggests that cannibalism generally impedes disease spread, because each victim is usually consumed by a single cannibal, such that cannibalism does not function as a spreading process. Consequently, cannibalism cannot be the only mode of transmission of most parasites. We develop simple, but general epidemiological models to analyze the interaction of cannibalism and vertical transmission. We show that cannibalism increases the prevalence of vertically transmitted pathogens whenever the host population density is not solely regulated by cannibalism. This mechanism, combined with additional, recently published, theoretical mechanisms, presents a strong case for the role of cannibalism in the spread of infectious diseases across a wide range of parasite–host systems.

Key words: contest competition; density dependence; discrete larval habitats; disease ecology; ecological epidemiology, parental transmission predation; trophic transmission.

Introduction

Vertically transmitted pathogens are broadly important in natural and managed animal populations (Fine 1975, Turelli 1994, Dubey and Lindsay 1996, Tenter et al. 2000, Ebert 2013). Epidemiologically, vertical transmission alone is not sufficient for the persistence of virulent pathogens, because they are selected out of the host population whose healthy members enjoy a greater lifetime reproductive success, and because vertical transmission fidelity is not perfect (Fine 1975, Lipsitch et al. 1995). Thus, in the absence of compensatory mechanisms such as sex-ratio distortions (Hurst 1993) or the prevention of superinfection by more virulent pathogens (Lively et al. 2005, Jones et al. 2007), mathematical models predict that some level of horizontal transmission is also needed for the persistence of the pathogen, and is essential for reaching high levels of endemic prevalence (Lipsitch et al. 1995). Indeed, various virulent, vertically transmitted pathogens have been reported to also employ horizontal transmission (Mims 1981, Andreadis 1985, Shoop 1991, Smith and Dunn 1991, Purcell et al. 1994, Geden et al. 1995, Webster and Kapel 2005, Saito and Bjørnson 2006, Blazquez and Saiz 2010). In fact, the combined (vertical + horizontal) transmission strategy has been suggested as the most common spreading strategy in endosymbionts (Ebert 2013). To better understand the epidemiology of such pathogens, it is important to determine specific mechanisms by which they can be transmitted effectively in the host population.

Cannibalism is a widespread behavior (Fox 1975, Polis 1981, Elgar and Crespi 1992, Richardson et al. 2010) that can involve transmission of parasites from infected victims of cannibalism to cannibals (reviewed by Rudolf and Antonovics 2007). Various vertically transmitted pathogens have been documented also to infect horizontally through cannibalism (e.g., Webster and Kapel 2005, Saito and Bjørnson 2006, Blazquez and Saiz 2010). For example, the microsporidian parasite *Nosema muscidifuracis* is transmitted vertically in the hymenopteran parasitoid *Muscidifurax raptor* from mother to offspring, but also between cannibalistic larvae as they develop within the host (Geden et al. 1995).

However, cannibalistic transmission does not act like a typical horizontal mode of transmission, since it involves, by definition, the death of the victim of cannibalism. Therefore, unlike normal horizontal transmission, the transfer of infection to a cannibal does not generate net new infections in the host population; that is, it is not a "spreading process" (Rudolf and Antonovics 2007). As a result, current theory shows that cannibalism can spread disease only when hosts practice "group cannibalism"; in the absence of this relatively unusual feeding trait, cannibalism cannot be a pathogen's sole mode of transmission (Rudolf and Antonovics 2007). Furthermore, a separate analysis (Sadeh et al., *in press*) indicates that cannibalism combined with an additional mode of horizontal transmission (and assuming a homogenous host population) impedes pathogen spread because cannibalism (1) removes infecteds from the population and (2) suppresses the density of susceptible hosts

Manuscript received 8 May 2015; revised 23 October 2015; accepted 1 December 2015. Corresponding Editor: W. E. Snyder.

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and, with them, opportunities for spread. No theory currently exists to generate expectations or guide empirical studies regarding the role of cannibalistic transmission in the spread of vertically transmitted pathogens.

Here we developed simple but general mathematical models for the epidemiology of vertically transmitted pathogens in populations of hosts that express cannibalism. We analyze the effects of vertical transmission and cannibalism on the generation of new infected individuals and on their prevalence in the host population. We show that, in the limit case, when the host population is regulated by cannibalism alone, it reaches a steady state in which pathogen prevalence is unaffected by the rate of cannibalism expressed by the hosts. However, whenever the population is also regulated by another environmental factor, pathogen prevalence invariably increases with cannibalism rate. We complement our general mathematical theory with a simple individual-based simulation model, inspired more specifically by organisms with discrete and highly competitive larval habitats, such as stem boring insects, leaf mining insects, tephritid flies, parasitoids, and organisms inhabiting ephemeral pools. Cannibalism in such systems is very common, and relatively well studied (Polis 1981, Elgar and Crespi 1992, Godfray 1994, Wells 2007, Richardson et al. 2010, Sadeh 2012). For example, populations of a stem boring caterpillar are known to be regulated by various factors (Baskauf 2003), but a primary density-dependent regulating factor is cannibalism among larvae (Breden and Chippendale 1989, Baskauf 2003), sometimes resulting in the survival of only one larva out of any number of eggs laid in a single plant. Stem borers are also often plagued by pathogens (Baskauf 2003). Some of these pathogens, e.g., *Nosema* spp. (Inglis et al. 2000), are transmitted vertically (Siegel et al. 1988), as well as through feeding on suspended spores (Solter et al. 1991), suggesting the possibility of cannibalistic transmission. Systems with similar life histories are widespread, and present a familiar relationship between cannibalism and competitive regulation of the population.

Methods

Our models consider a homogenous population of N hosts, each of which may be either susceptible (S) or infected (I). Because vertically transmitted pathogens are often acquired at birth and depend on their host's reproduction to spread further, host recovery rates are generally low or nonexistent (e.g., *Wolbachia* infections in insects, Hong et al. (2002), other symbiont–host systems reviewed in Fine [1975]). Therefore, SI modeling is a biologically appropriate framework for our theory. In addition, Holt and Roy (2007) showed that predation can increase disease prevalence in the prey population due to the removal of recovered and resistant individuals. By excluding recovered individuals, we avoid confounding our results with that effect. For clarity of analysis, we assume that the only modes of transmission are vertical, with fidelity h (representing the proportion of infected offspring born to an infected parent), and cannibalistic transmission with efficiency τ (representing the probability, per cannibalistic event between a healthy cannibal and an infected victim, that the cannibal acquires the infection). While the combination of cannibalism with horizontal transmission is analyzed in greater depth elsewhere (Sadeh et al., *in press*), we also incorporate simple horizontal transmission at the end of our current analysis to shed light on the combined effects of all three modes: vertical, horizontal, and cannibalistic transmission. In all models, we ask how is infection prevalence (I/N) at equilibrium affected by the rate of cannibalism, a, expressed by the hosts? See Table 1 for parameter symbols used in all models. First, we develop a conceptual model of the effects of cannibalism on pathogen spread and prevalence. We then develop two mathematical models that represent opposing, extreme limit cases concerning how the host population is regulated. These limit cases occur rarely in real biological systems, but they are useful to delineate the range of possible effects of cannibalism on disease dynamics. This analysis is followed by two models that represent the intermediate region between the limit cases, and the vast majority of real systems. Finally, we develop an individual-based model based on a specific life history that exemplifies the general rule.

Conceptual model: the effects of cannibalism only on pathogen prevalence

Because cannibalism is a source of mortality, it eliminates individuals from the population, and without group cannibalism cannot generate a net increase in the total number of infecteds. Thus, cannibalism is not a spreading process. However, to illustrate the isolated effect of cannibalism on disease prevalence, we consider the full set of all possible pairwise interactions between cannibals and their victims (Table 2) in a finite, non-reproducing group of n hosts, half of which are infected. In each cannibalistic event between a healthy cannibal and an infected victim, the cannibal contracts the infection with probability τ. We create pairs randomly, allow one member of each pair to consume the other, and calculate the change in disease prevalence.

Model 1: host population regulated only by cannibalism

Cannibalism is a density-dependent mortality factor, and therefore has the capacity to regulate the densities of cannibalistic populations (Claessen et al. 2004). We first model a limiting case where the host population is regulated only by cannibalism:

$$dS/dt = b_s S + b_i (1-h) I - dS - aNS - \tau aSI \qquad \text{(Model 1)}$$
$$dI/dt = b_i h I - (d+v) I - aNI + \tau aSI$$

TABLE 1. Parameter symbols.

Symbol	Interpretation	Used in models
S, I	susceptible, infected densities	all models
$N = S + I$	total population density	all models
b_s, b_i	per capita birth rate of susceptibles, infecteds (respectively)	all models
h	vertical transmission fidelity (proportion of offspring)	all models
d	per capita background mortality rate	models 1–4
m	total mortality per larval habitat per time step	model 5
v	mortality rate due to infection (virulence)	models 1–4
a	per capita cannibalism rate, proportion of larvae killed through cannibalism	models 1–4, model 5
τ	probability of pathogen transmission per cannibalistic event	all models
μ	global mortality of entire population	models 2 and 5
K	strict carrying capacity	model 2
K_n	number of available larval habitats	model 5
K_q	number of adults that can emerge from each larval habitat	model 5
p	probability of an infected larva winning a cannibalistic encounter with a susceptible larva	model 5
δ	increase in per capita mortality rate as a function of population density	model 3
α	exponential decay rate of per capita birth rate with population density	model 4
β	horizontal transmission rate	model 6

In this model, susceptible hosts give birth at a rate of b_s offspring per individual per unit time, while the birth rate for infected hosts is b_i ($b_s \geq b_i$). All members of the population are subject to mortality from background causes at a rate of d individuals per capita per unit time. In infecteds, however, mortality rate is increased by v due to the infection. The entire population suffers from cannibalistic mortality, equally expressed by all members of the population at a per capita rate a (per potential victim per unit time), regardless of infection status. When a susceptible cannibal consumes an infected victim, it contracts the infection with probability τ.

In Appendix S1 we present the analytical solution for this system of equations at equilibrium.

Model 2: tight regulation by a factor other than cannibalism

We now consider the opposite limiting case, in which the host population is tightly regulated, regardless of cannibalism rate, by a different ecological factor (e.g., a fixed number of available territories). Thus, the population size is fixed at a constant, strict, carrying capacity, K. Mathematically, we need to track only the dynamics of the infecteds in the population, given that $S = K - I$ at any time (see also Holt and Roy 2007, who modeled a prey population under similar tight regulation).

$$dI/dt = \tau a (K - I) I - (aK + d + v) I + \mu (I/K) h \qquad \text{(Model 2)}$$

Here, the first term represents the infection of susceptible cannibals who consume infected victims, the second term is the removal of infecteds from the population due to cannibalism (aK) and due to background mortality as modified by the survival consequences of being infected ($d + v$). The third term is the birth of new infecteds through vertical transmission. The constant population size implies that global birth rate in the population exactly compensates for global mortality rate ($\mu = aK^2 + dK + vI$). The proportion of these births that is from infected parents is equal to the proportion of infecteds in the population (I/K), and a proportion h of those acquire the infection vertically.

In Appendix S2 we present the analytical solution for this system at equilibrium.

TABLE 2. Outcomes of all possible pairwise interactions between susceptible or infected cannibals and susceptible or infected victims.

Victim	Susceptible cannibal	Infected cannibal
Susceptible	removal of susceptible: two susceptible interactants → one susceptible survivor	removal of susceptible: one susceptible + one infected interactants → one infected survivor
Infected	cannibalistic transfer (τ) or removal of infected ($1 - \tau$): one susceptible and one infected interactant → τ infected survivors and ($1 - \tau$) susceptible survivors	removal of infected: two infected interactants → one infected survivor

Models 3 and 4: co-regulation by cannibalism and other factors

Most realistic systems represent intermediate cases between the above extremes, where both cannibalism and other ecological factors co-regulate the size of the host population. There are many mechanisms through which density-dependent regulation can operate (e.g., Sibly and Hone 2002). In Appendix S3, we present two additional models where the size of the host population is allowed to vary, and is regulated by a combination of cannibalism and another density-dependent process. Model 3 assumes a density-dependent per-capita background mortality rate, and Model 4 assumes a density-dependent per-capita birth rate. We solve both models numerically.

Model 5: organisms with discrete larval habitats

We developed a stochastic, individual-based model that simulates organisms that utilize discrete larval habitats such as stem borers, leaf miners, tephritid flies, parasitoids and organisms inhabiting ephemeral pools. Cannibalism is expressed in the larval stage, within larval habitats, but disease prevalence is measured in the adult population. The model assumes an asexual population of $N(t)$ reproductive adults, consisting of $S(t)$ healthy individuals and $I(t)$ infected individuals, which oviposit into K_n available larval habitats. The time steps of the model are non-overlapping host generations, and we assume that the availability of larval habitats does not change.

Each adult has a complement of b_s or b_i eggs for healthy and infected adults, respectively. Thus, the pathogen can compromise adult fecundity, but it is not lethal to adults. Oviposition is ideal free-distributed, such that the eggs of each adult are distributed uniformly over the available habitats, since habitats are assumed not to vary in quality. Thus, each habitat receives $n = \left[b_s S(t) + b_i I(t)\right] / K_n$ eggs from various different parents.

The pathogen's vertical transmission fidelity, h, is the proportion of eggs of an infected adult that carry the pathogen. We assume that infected eggs maintain their infection status through hatching and maturation to adulthood. There is no horizontal transmission of the pathogen between adults or between larvae within habitats, other than through cannibalism. We also assume that the pathogen does not kill infected larvae, but it may reduce their likelihood of winning a cannibalistic encounter with conspecifics.

We assume that larvae within habitats both compete with, and may cannibalize, each other. Competition resembles intense contest, where only K_q larvae can emerge from each habitat. Typical numbers of successful emergences can be as low as one or two larvae per habitat, as in many stem borers (Baskauf 2003), seed or fruit parasites (Messina 1991), and parasitoids (Godfray 1994). Thus, total larval mortality in each habitat is $\mu = n - K_q$. The rate of cannibalism, a, is defined as the proportion of larval mortality in each habitat that is due to cannibalism. Thus, μa larvae are killed in each habitat as a result of cannibalism. Because cannibalism in many of these systems is an adaptation to avoid competition (Baskauf 2003, Richardson et al. 2010), we assumed that cannibalism operates first, before competition; reversing the order did not change the qualitative results of the model (data not shown). Larvae encounter each other in random pairs, and one larva in each pair consumes the other. The probability that an infected larva wins an encounter with a healthy larva is p. If the healthy larva wins, it may become infected with probability τ, due to cannibalistic transmission. These paired encounters are iterated until μa larvae have been cannibalized. The cannibalism phase is followed by mortality due to non-cannibalistic contest competition, in which $\mu(1-a)$ additional, randomly selected larvae are removed from each habitat.

The surviving larvae emerge to the adult population of the next generation. Thus, the adult population is constant over time after the second generation at $N(t) = K_n K_q$, but the prevalence of the disease, $I(t)/N(t)$, may change over time.

The parameter values used in the simulation results presented here are: $K_n = 100$ larval habitats; $K_q = 2$ larvae emerge per habitat; $h = 0.8$; $p = \tau = 0.5$. To vary the intensity of within-habitat larval competition, we altered adult fecundity, which is also the inverse of the emergence rate (number of emergers / total number of eggs deposited) from each habitat (varying K_q does not change the emergence rate). Thus, setting adult fecundity to $b_s = b_i = 5$, 10 and 20 larvae per adult, we generated low, moderate, and intense competition, respectively. The initial conditions were an adult population of $K_n K_q$ susceptibles, invaded by a single infected adult. We iterated the model for 1,000 replicate populations over 100 generations, and recorded the distribution of disease prevalence over all adult populations. Matlab code for Model 5 is provided in Appendix S4.

Model 6: incorporating both vertical and horizontal modes of transmission

In the models above, the effects of cannibalism and cannibalistic transmission were analyzed when operating only with vertical transmission. In Appendix S4, we extend Model 2 to also include horizontal transmission. Thus, this model explores the effects of cannibalism on a pathogenic infection that is capable of persisting in the host population in the absence of cannibalistic transmission by using a combination of vertical and non-cannibalistic horizontal transmission.

Results

According to our conceptual model, for any $\tau > 0$, cannibalism is a process of differential elimination with respect to infection status: susceptibles are removed at a

greater rate than infecteds. For example, in the first round of cannibalism, we create $n/2$ pairs randomly, and allow cannibalism to occur. This results in $n/2$ survivors, of which at least half (in case $\tau = 0$) are infecteds (see Table 2). Thus, while cannibalism reduces the total number of infecteds in the population (along with the total number of susceptibles), it consistently elevates disease prevalence, as long as the population persists. Reproduction prevents the population from going to extinction. With reproduction comes vertical transmission, and it is reproduction combined with vertical transmission that yields the spreading process (from one infected mother come many infected offspring) that is essential for the invasion of any pathogen. However, vertical transmission alone is not sufficient for parasite persistence, because it is generally a differential generation process with respect to infection status: infected hosts suffer greater mortality due to pathogen virulence, and may also fail to transmit their infection to all of their offspring (imperfect transmission fidelity). Thus, cannibalism and vertical transmission have opposing influences on disease prevalence. Cannibalism is a differential elimination process that pushes prevalence up, whereas vertical transmission is a differential generation process that pushes prevalence down. The outcome of their combination is expected to depend on population density, since the frequency of cannibalism occurring in the host population (aN) increases with its density, while vertical transmission rate does not depend on population density.

In the limiting case of a host population that is regulated by cannibalism exclusively (Model 1), equilibrium population density is inversely related to per-capita cannibalism rate (Appendix S1). Thus, increasing per-capita cannibalism rate (a) by any factor results in a decrease in population density by exactly the same factor, leaving the frequency of cannibalism unchanged (aN). Thus, due to a fully compensatory demographic feedback, pathogen prevalence is indifferent to per-capita cannibalism rate (Fig. 1A). This result holds for the entire space of possible parameter values (Appendix S1).

In the opposite limiting case of a host population that is tightly regulated by an ecological factor other than cannibalism (Model 2), there is no demographic feedback to cannibalism. Therefore, increasing per-capita cannibalism rate also elevates the frequency of cannibalism occurring in the population. As a result, cannibalism increases disease prevalence rapidly (Fig. 1B). This effect saturates, approaching an asymptotic prevalence that is determined by constraints on transmission efficiencies (either vertical, h, or cannibalistic, τ). As in Model 1, this result holds true for the entire space of possible parameter values (Appendix S2).

The analysis above is sufficient to indicate that intermediate, more realistic cases, where the host population is regulated by a combination of cannibalism and other ecological factors, should produce a positive relationship between per-capita cannibalism rate and pathogen prevalence. That is because combined regulation results in a

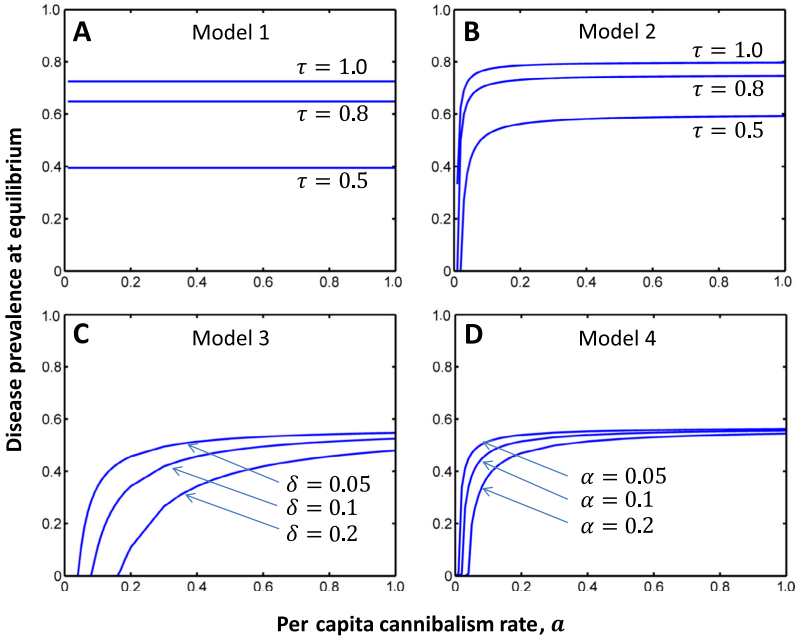

FIG. 1. Disease prevalence at equilibrium as a function of per-capita cannibalism rate, a, in (A) a host population regulated by cannibalism only (curves represent different cannibalistic transmission probabilities); (B) a population regulated tightly by factors other than cannibalism (curves represent different cannibalistic transmission probabilities); (C) a population regulated by cannibalism and density-dependent mortality (curves represent different strengths of density dependent mortality); (D) a population regulated by cannibalism and density-dependent birth (curves represent different strengths of density dependent birth). Parameter values are $b_s = b_i = 5$; $d = v = 0.5$; $h = 0.8$; $\tau = 0.5$ (unless specified otherwise). In Model 2 (Panel B), $K = 100$.

partially compensating demographic feedback to changes in per-capita cannibalism rate. We confirmed this using numerical simulations of populations with density-dependent birth and mortality (Models 3 and 4; Appendix S3), each producing the expected positive relationship (Fig. 1C and D). In both models, stronger density dependence of the non-cannibalistic regulatory factor results in (1) higher threshold rates of cannibalism, below which the pathogen goes extinct, (2) milder effects of cannibalism on disease prevalence when the pathogen becomes endemic in the host population (i.e., the positive effect of cannibalism saturates only at higher cannibalism rates). Both of these patterns are the result of a reduction in host population density, leading to a lower frequency of cannibalistic interactions.

Model 5 explored these general ideas using a more concrete example from a common life history, wherein contest competition and cannibalism occur in the larval habitat. Our simulations showed that disease prevalence in the adult population increases strongly with cannibalism rate (Fig. 2A). For lower cannibalism rates, the pathogen is often lost from the population entirely (Fig. 2B). Under low cannibalism, most larvae die from competition, and the probability that the emerging adults will be infected is low. However, as the cannibalism rate increases, emerging adults are increasingly likely to be the winners of multiple cannibalistic interactions, thus increasing their opportunities to contract an infection. Subsequently, infected adults can spread the disease to several offspring through vertical transmission. The positive effect of cannibalism on disease prevalence is reduced only modestly with decreasing within-habitat competition intensity (Fig. 2). As in Models 3 and 4, insufficient cannibalism rates lead to the extinction of the pathogen. The tipping point (here at $a = 0.4$) between certain pathogen extinction and a nonzero probability of its persistence depends on transmission efficiencies (vertical transmission fidelity, cannibalistic transmission probability, and the probability that an infected larva wins a cannibalistic encounter with a susceptible conspecific), which were kept constant in Fig. 2.

Model 6 extended Model 2 by exploring the effect of cannibalism on the spread of pathogens that employ both vertical and horizontal modes of transmission (Appendix S4). In the absence of cannibalism, the disease can persist in the host population due to horizontal transmission. Low frequencies of horizontal transmission, without cannibalism, lead to low disease prevalence or disease exclusion. In these cases, cannibalism will increase disease prevalence, because many infected individuals that are cannibalized are consumed by healthy conspecifics, facilitating cannibalistic pathogen transfer (Table 2) and the amplification of disease spread. However, particularly high frequencies of horizontal transmission can lead to high disease prevalence even without cannibalism. In that case, many infected victims will be cannibalized by already infected conspecifics, and cannibalistic transfer will be rare. Thus, cannibalism in that case is mostly a source of mortality that accelerates the turnover of hosts (the host population is constant in this model), through which imperfect vertical transmission will lead to the reduction of disease prevalence (Appendix S4: Fig. S1).

Discussion

Our models demonstrate that cannibalistic transmission amplifies the spread of parasites that are also transmitted vertically, unless the population is regulated strictly by cannibalism. While cannibalism has the theoretical potential to regulate population densities (Claessen et al. 2004), and in some cases it plays a considerable role in such regulation (Wagner and Wise 1996, Baskauf

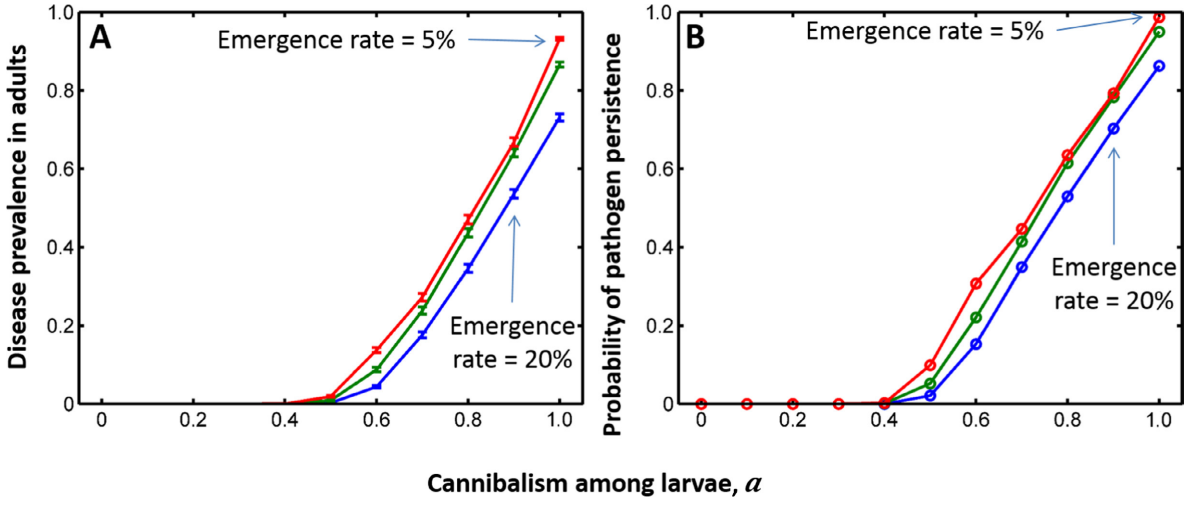

Fig. 2. (A) Disease prevalence and (B) probability of persistence in adults as a function of cannibalism rate among larvae within discrete larval habitats (Model 5). As the intensity of larval competition is reduced (i.e., as successful emergence rates increase: red curve equals 5%, green curve equals 10%, blue curve equals 20%) the positive effect of cannibalism on disease prevalence becomes slightly less pronounced. Error bars are ± SE.

2003), it is extremely rare that a population is regulated strictly by cannibalism and by no other ecological factor, such as limited food resources, space, or the action of predators. Even a slight contribution from any additional regulating factor shifts the effect of cannibalism on disease prevalence from neutral to positive. Therefore, we should expect cannibalism to spread vertically transmitted parasites effectively in the vast majority of parasite–host systems.

Should this lead us to expect vertically transmitted pathogens to be more common in cannibalistic hosts? Clearly this is to be expected in the absence of additional, horizontal modes of transmission. In this case, the absence of cannibalism means that the pathogen can only be transmitted vertically, and therefore will generally be selected out of the host population (Lipsitch et al. 1995). However, if an additional mode of horizontal transmission exists, then cannibalistic transmission may not be necessary for the persistence of the pathogen. In fact, frequent horizontal transmission in dense host populations can in itself lead to high infection prevalence, reducing the frequency of cannibalistic pathogen transfers, and the potential for cannibalism to elevate disease spread. Thus, pathogens that are transmitted primarily from parents to offspring and whose other modes of horizontal transmission are weaker will be more common (or occur only) in cannibalistic hosts. Conversely, according to the model presented here, pathogens that are primarily transmitted horizontally, but also employ weak vertical transmission, may be more common in non-cannibalistic hosts (if their populations are dense). It should be noted, however, that the interaction of cannibalism with horizontal modes of transmission can also elevate disease spread when host populations are heterogeneous (Sadeh et al., *in press*). Because the expression of cannibalism usually depends on differences in size and developmental stage between interacting individuals, it tends to transfer pathogens from less developed and often less infectious hosts (typical victims of cannibalism) to more developed and often more infectious hosts (typical cannibals). Thus, in stage-structured host populations, cannibalism is expected to enhance disease spread by amplifying both vertical and horizontal modes of transmission, regardless of their relative strengths.

The theory presented in this study is general, and identifies for the first time a fundamental epidemiological process in cannibalistic host populations that is inherent to vertical pathogen transmission. Suitable empirical systems for testing the theory are parasites of cannibalistic hosts that are known to employ primarily, or only vertical transmission, such as various microsporidians in gammarid crustaceans (Ironside et al. 2003*a*, *b*, Haine et al. 2007). Horizontal transmission is also predicted to facilitate the spread of many sex-ratio distorting, vertically transmitted parasites (Ironside et al. 2011). As suggested for some systems (e.g., MacNeil et al. 2003), cannibalism may constitute a common form of horizontal transmission.

The group of organisms that motivated the development of Model 5 is stem-boring insects such as the southwestern and European corn borers (Baskauf 2003) and their vertically transmitted microsporidian pathogens. Another striking example is the virus LbFV infecting the endoparasitoid *Leptopilina boulardi* (Varaldi et al. 2012). The virus is transmitted vertically from mother to offspring, but also manipulates its host's oviposition behavior such that they tend to superparasitize their *Drosophila* hosts, as opposed to their natural avoidance of hosts that already contain a conspecific egg. As a result, larval competition and cannibalism are facilitated within the Drosophila host, from which only a single *L. boulardi* can emerge. LbFV is also transmitted horizontally among larvae, possibly through cannibalism, thus ensuring the emergence of an infected larva, regardless of who won the contest. Interestingly, it seems that the ability of LbFV to induce superparasitism in infected wasps may have evolved to utilize exactly the mechanism portrayed by Model 5. In this case cannibalism is not a source of additional larval mortality, because even in its absence the habitat enables only up to a single larva to emerge. Therefore, cannibalism has only a positive effect on disease spread, which is not limited by the potential presence of additional horizontal transmission among larvae (as is the case in Model 6). A similar scenario may occur with *Wolbachia* infections in *Trichogramma kaykai* (Huigens et al. 2000).

Our findings add to three other, non-mutually exclusive mechanisms that were previously identified, by which cannibalism can spread parasites. Cannibalism alone is sufficient to spread parasites in host populations where, on average, more than one cannibal consumes each infected victim (i.e., "group cannibalism"; Rudolf and Antonovics 2007). Cannibalism may also elevate the spread of horizontally transmitted pathogens in structured host populations (Sadeh et al., *in press*). Finally, Holt and Roy (2007) showed that predation (they did not model cannibalism) can enhance disease spread in a density-dependent prey population due to the removal of immune individuals and the resulting proliferation of susceptibles that are then vulnerable to contracting the disease. Taken together, theory presents a strong case for the role of cannibalism in the spread of infectious diseases, and points the way for empirical validation across a wide range of parasite–host systems.

Acknowledgments

This research was funded by BSF grant 2013-306 awarded to J. A. Rosenheim and to Moshe Coll, by a Postdoctoral Award No. FI-457-2011 from BARD, The United States—Israel Binational Agricultural Research and Development Fund, awarded to A. Sadeh, and by a Lady Davis postdoctoral fellowship awarded to A. Sadeh by the Hebrew University of Jerusalem. We are grateful to Moshe Coll, Michael Culshaw-Maurer, and four anonymous reviewers for their insightful comments.

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