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Combined impacts of Allee effects and parasitism

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Despite their individual importance for population dynamics and conservation biology, the combined impacts of Allee effects and parasitism have received little attention. We built a mathematical model to compare the dynamics of populations with or without Allee effects when infected by microparasites. We show that the influence of an Allee effect takes the form of a tradeoff. The presence of an Allee effect in host populations may protect them, by reducing the range of population sizes that allow parasite spread. Yet if infection spreads, the Allee effect weakens host populations by reducing their size and by widening the range of parasite species that lead them to extinction. These results have important implications for predicting the survival of threatened populations or the success of reintroductions, and may help define size ranges within which given populations should be maintained to prevent both epidemics and Allee effects driven extinctions.

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As the focus of population dynamics attention gradually shifts towards smaller populations, the number of studies on the Allee effect has greatly increased. This demographic process, also called depensation, is characterised by reduced fitness leading to positive density-dependence at low densities and potentially affects many populations (Fowler 1991, Courchamp et al. 1999a, Stephens and Sutherland 1999, Stephens et al. 1999). Although some work has shown that Allee effects might play a stabilising and protective role (Gyllenberg et al. 1999, Scheuring 1999, Fowler and Ruxton 2002), most studies have highlighted its negative impact on population persistence (Dennis 1989, McCarthy 1997, Amarasekare 1998, Brassil 2001).

The majority of studies on the Allee effect focus on a single species and do not account for interspecific interactions. However studying the dynamical behaviour of a population without considering interactions with other species may lack realism (Crawley 1992). It is important to consider the consequences of Allee effects in the context of wider community dynamics and interspecific interactions.

Several recent papers have explored the role that Allee effects could play in competition and prey–predator relationships. Wang et al. (1999) highlighted that introducing an Allee effect into a Lotka–Volterra competitor system would destabilise it. Courchamp et al. (2000) have shown that cooperative species, because they are sensitive to an Allee effect, are more sensitive to interactions with natural enemies such as competitors or predators. Some studies have also shown that Allee effects may favour species coexistence within a metapopulation (Hastings 1996, Ferdy and Molofsky 2002).

Another major interspecific interaction type, parasitism, has been less studied in this context. Four studies focus on this subject, but they consider different issues. Scheuring (1999) studies the dynamical stability of populations with stochasticity and concluded that an Allee effect could increase such stability, even in the presence of a parasite. Cruickshank et al. (1999) investigate the characteristics of the infection front

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wave of epidemics and considers that the shortage of infected individuals immediately after epidemics may create a type of Allee effect for the parasite. Regoes et al. (2002) explore the consequences of a sigmoidal dose dependence of the infection rate on host–parasite interactions, mimicking the equivalence of an Allee effect for the microparasite population. Cornell et al. (2004) take into account a mate-finding Allee effect of macroparasite worms (but not on the host) and assess its effect on parasite aggregation and on the likelihood and severity of epidemic.

The combined effects of parasitism and Allee effects on the host population thus remain unstudied, despite the fact that parasitism is one of the most important interspecific interaction types (Anderson and May 1979). Parasites are, according to Anderson, "regulators of natural populations" (Anderson and May 1979), and they affect virtually every species. Besides their obvious theoretical interest (Lafferty and Gerber 2002, Deredec and Courchamp 2003), both Allee effect and parasitism have major practical consequences for population and ecosystem management and conservation. The Allee effect may have been a factor in numerous extinction events (Stephens and Sutherland 1999). Because of the immeasurable number of species currently threatened with disappearance (Fowler 1991), some of which can be particularly sensitive to epidemics (McCallum and Dobson 1995), it has become necessary to explore the impact of the Allee effect on the dynamical fate of potentially infected populations.

The absence of such studies is all the more surprising if one considers that parasitism has several distinctive features in common with the Allee effect. The first one is the importance of density. Infectious diseases can strongly influence host population dynamics (Anderson and May 1979) and host density is a decisive factor in infection behaviour, greatly influencing whether or not a parasite will become established in a host population (Anderson 1982). Another characteristic common to both the Allee effect and parasitism is the existence of population thresholds below which likelihood of host (Dennis 1989) or parasite (Anderson and May 1979, Kermack and McKendrick 1991) extinction increases sharply (reviewed by Deredec and Courchamp 2003). These thresholds have been called the "Allee limit" for the Allee effect (Brassil 2001) and the "density threshold" and the "critical community size" for diseases (Grenfell and Harwood 1997, Knell et al. 1998). In addition, both parasitism and the Allee effect are sensitive to social interactions and to resulting conspecific aggregation. A decrease in social interactions can result in mating shortage (a major cause of Allee effect) but at the same time, it limits parasite transmission. Conversely, aggregation facilitates parasite transmission (Dobson and Poole 1998), but may prevent extinction due to the Allee effect.

The impacts of Allee effects and parasitism are thus likely to interact, although not in an intuitively obvious way. The crucial issue is whether a combination of Allee effects and parasitism increases extinction probabilities for host populations or whether the lower densities induced by Allee effects can protect host populations from parasitism.

In order to characterise the combined effects of parasitism and Allee effects, we compared two similar host–microparasites systems, one of which is subject to an Allee effect. Since the use of the two main classical transmission terms is the subject of a controversy, we considered both of them as well as an additional mixed transmission term, which we deem more appropriate to the modelled situation.

An analytic study of models with and without Allee effect enabled us to identify the conditions leading to parasite disappearance, infection establishment and host extinction. The impact of the Allee effect on the fate of host populations is also measured by comparing equilibrium population sizes. The results are discussed in a conservation perspective.

Model

Our models are based on simple microparasite SI systems. Two categories of individuals are considered: susceptible individuals (S) and infected ones (I), the total population size being N ($N=S+I$). We assume that the host population occupies a constant area. The terms of population size and density, therefore assumed to be proportional, will be indiscriminately used. Reproductive capacity of an individual is supposed not to be affected by its infective state. Transmission is horizontal (juveniles are born susceptible) and behavioural (through contacts between individuals). Considering $r=b-m$, where r, b and m are respectively the intrinsic growth, birth and death rates, a population of N individuals follows a standard density-dependence law $rf_S(N)$ or an inverse density-dependence law $rf_A(N)$, depending on whether it is subject (f_A) or not (f_S) to an Allee effect. We choose to express these functions as simply as possible: $f_S(N)=(1-N/K)$ and $f_A(N)=f_S(N)(1-L/N)$. Here, K represents the carrying capacity, L is the Allee limit below which the growth rate becomes negative (L is equivalent to K_- and l of previous models (Courchamp et al. 1999b, Brassil 2001, respectively). We thus choose to express the Allee effect as a "strong Allee effect", as described by Brassil (2001) and Wang and Kot (2001) i.e. with a critical threshold. In addition, the infected die from infection at a rate α. The host–parasite system is thus described by (1).

$$\begin{cases} \dfrac{dS}{dt} = bf(N)N - mf(N)S - T(S,I) \\ \dfrac{dI}{dt} = T(S,I) - mf(N)I - \alpha I \end{cases} \quad (1)$$

where T(S,I) stands for the number of transmission events depending on the number of contacts between infected and susceptible individuals.

However, the distinction $r = b - m$ leads to an erroneous division of the population growth rate: the population birth $bf_S(N)$ and death rates $mf_S(N)$ become two linearly decreasing functions of density, null at carrying capacity and negative above it. It assumes no turn-over and thus everlasting individuals at equilibrium. Also, in the presence of an Allee effect, both birth and death rates become negative under the Allee limit.

To circumvent this difficulty, we thus assume that the disease is transmitted both horizontally and vertically: infected hosts infect their newborns rapidly, so that they can be considered to give birth to infected hosts. We thereby avoid the division of the growth rate into birth and survival parts.

Equation 1 becomes:

$$\begin{cases} \dfrac{dS}{dt} = rf(N)S - T(S,I) \\ \dfrac{dI}{dt} = T(S,I) + rf(N)I - \alpha I \end{cases} \quad (2)$$

Traditionally, the horizontal transmission term has been modelled either with a density-dependent term (also called mass action term) or a frequency-dependent one (also called proportionate mixing term). A debate has been raised concerning the use of these two terms (De Jong et al. 1995, McCallum and Dobson 1995). The first assumption has been used more often since it looks more realistic: it satisfies the assumption of mass action law where individuals are mixed homogeneously. But De Jong et al. (1995) pointed out that transmission might be better mimicked under the second assumption. However, no consensus emerges, probably because neither of these terms is suitable for every situation. In both cases, the number of new infected individuals in the population depends on the number of infected I. It also depends on the number of conspecifics encountered by each infected: $\phi(N)$ and on the proportion of susceptible among these encounters S/N. The transmission term is thus described by $\beta\phi(N)SI/N$, where β is the probability of transmission per encounter. The frequency-dependent (fd) model assumes that the number of encounters $\phi(N)$ is a constant. The corresponding transmission term is proportionate to $\beta SI/N$. The density-dependent (dd) transmission term assumes that the number of encounters $\phi(N)$ increases linearly with the population density. Transmission is thus proportionate to βSI.

If one consensus was to emerge concerning the use of these two terms, it is that the dd term is more relevant when the population size remains small enough to enable the number of encounters to increase linearly with the population size. When the population becomes too large, the number of encounters of a single individual saturates (Heesterbeek and Metz 1993, Antonovics et al. 1995) and the fd-term becomes more convenient. A mixed, non-linear, increasing and concave encounter function would thus better cover a large range of population size (Barlow 2000). Therefore, an additional, mixed transmission term mimicking the fd-model at low density and the dd-model at high densities (not explicitly defined, for the sake of generalisation, but see Fig. 1) is considered in this study. Differences in results with this transmission term are presented for the two classical terms when worthy of note. Values of equilibra and conditions are presented for these two terms in Table 1 and 2.

The system thus becomes:

$$\begin{cases} \dfrac{dS}{dt} = rf(N)S - \beta\dfrac{SI}{N}\phi(N) \\ \dfrac{dI}{dt} = \beta\dfrac{SI}{N}\phi(N) + rf(N)I - \alpha I \end{cases} \quad (3)$$

For ease of calculation and for the sake of simplicity we transformed (Eq. 3) into the equivalent system (Eq. 4) using the new variable: $y = I/N$, which represents the prevalence of infection in the population:

$$\begin{cases} \dfrac{dN}{dt} = N[rf(N) - \alpha y] \\ \dfrac{dy}{dt} = y(1-y)(\beta\phi(N) - \alpha) \end{cases} \quad (4)$$

In order to avoid systematic enumeration of the three transmission cases, we will express our results in terms of rate of transmission events $\beta\phi(K)$.

Alternative model

In the previous model, disease is transmitted both horizontally and vertically. This mode of transmission is realistic in microparasites with efficient transmission, so that close, prolonged contact between offspring and infected mother will inevitably lead to offspring infection. Such models can thus provide a fair representation of the dynamics of bacteria and viruses in populations of animals with parental care, for example. Obviously, this model also accounts for diseases that are transmitted both vertically and horizontally.

However, this assumption of systematic transmission to progeny is restrictive, while we aimed at providing general results. Therefore we completed the study by a last model that resolves in a different way the problem

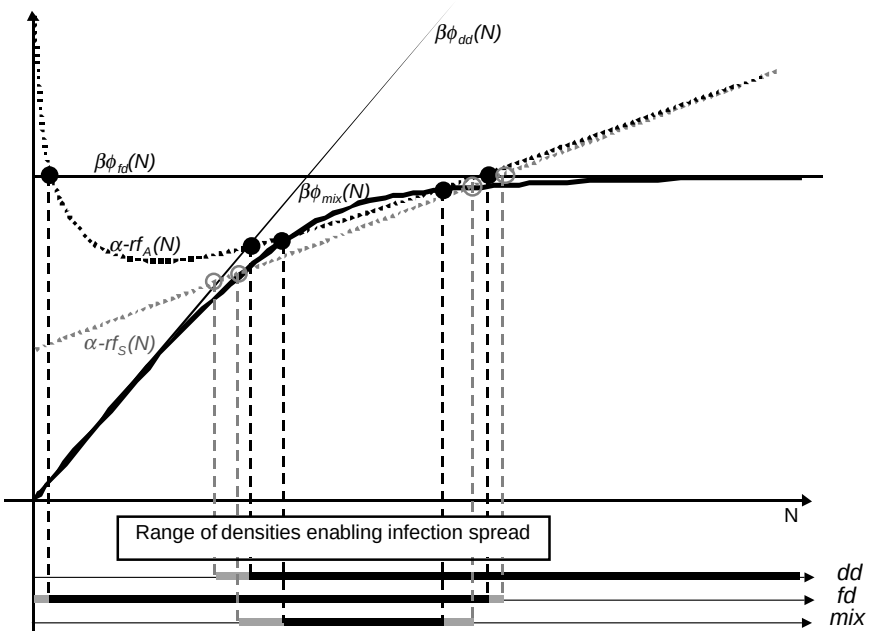

Fig. 1. Ranges of population size enabling infection spread in case of density-dependent (dd), frequency-dependent (fd) and mixed (mix) transmission terms; in the absence (grey) or in the presence (black) of an Allee effect. Infection spreads in a healthy population if transmission events (βφ(N)-continuous line) exceed the reduction in infected hosts ($\alpha - rf(N)$ – dotted line). This happens when host density is above a lower threshold in the dd case, below an upper threshold in the fd case, and between two thresholds in the mix case. This figure also makes sense for the second model provided the dotted line represents ($\alpha + M(N) = \alpha + b - rf(N)$).

raised by the initial model. Here, transmission is strictly horizontal and the growth rate is divided into a constant birth rate and a density-dependent mortality rate, which is biologically more realistic. In this model, the Allee effect acts only on mortality.

The system can then be written:

$$\begin{cases} \dfrac{dS}{dt} = B(N)N - M(N)S - \beta\dfrac{SI}{N}\phi(N) \\ \dfrac{dI}{dt} = \beta\dfrac{SI}{N}\phi(N) - M(N)I - \alpha I \end{cases} \quad (5)$$

where $B(N) = b$ and $M(N) = b - rf(N)$, or equivalently:

$$\begin{cases} \dfrac{dN}{dt} = N[rf(N) - \alpha y] \\ \dfrac{dy}{dt} = y[(\beta\phi(N) - \alpha)(1 - y) - b] \end{cases} \quad (6)$$

We considered the three forms of encounter function φ(N) described above.

System (Eq. 6) was studied graphically, by determining the isoclines $dN/dt = 0$ and $dy/dt = 0$ in the plane (N,y), their intersections and the conditions of stable equilibrium. In addition, equilibria were qualitatively compared. Conditions for infection spread in a wholly susceptible population were also graphically established.

As we aim to remain as general as possible, we do not expand on specific examples of host–parasite pairs to which our models would apply. This would logically require a better specificity of the models. Instead, our models are qualitatively suitable for a large range of infectious diseases, for which there is no recovery and where latency period may be ignored in a first approximation.

Results

Standard population

Equilibrium points

Five stationary points may exist in system (Eq. 4), corresponding to possible equilibria. These equilibria are never simultaneously stable. Their existence and stability depend on the characteristics of the considered host–parasite pair (α/r and β/r). We only consider the stable equilibria (their values are given in Table 1).

When virulence is so high that, at carrying capacity, disease-induced mortality exceeds the rate of transmission events, infected individuals die too rapidly to transmit parasites: the infection disappears, and the population reaches the carrying capacity. Otherwise, the parasite will establish and reduce the host population size, even infecting the whole population, if transmission is high enough and if population growth rate compensates disease induced death (Fig. 2A, Table 1). In the absence of Allee effects, the model never predicts systematic extinction of the host, even very small populations.

With the fd transmission, the weakening effect of parasites is stronger. If transmission exceeds virulence, parasites either infect the whole population, or lead to host extinction when population growth rate does not compensate for disease-induced deaths.

Table 1. Stable equilibria: values and conditions for existence for the cases of mixed (mix), density-dependent (dd) frequency-dependent transmissions. (Note that ϕ^{-1} is the inverse of ϕ, assumed to be continuous, strictly increasing and consequently inversible).

Transmission term		Standard		Allee Effet	
		Mix and dd	fd	Mix and dd	fd
Values					
	Recovery			$N_R = K$ $y_R = 0$	
	Partial infection	$N^S_{I_p} = \phi^{-1}(\alpha/\beta)$ $y^S_{I_p} = \frac{r f_S(\phi^{-1}(\alpha/\beta))}{\alpha}$	Ø	$N^A_{I_p} = \phi^{-1}(\alpha/\beta)$ $y^A_{I_p} = \frac{r f_A(\phi^{-1}(\alpha/\beta))}{\alpha}$	Ø
	Total infection	$N^S_{I_t} = K\left(1-\frac{\alpha}{r}\right)$ $y^S_{I_t} = 1$		$N^A_{I_t} = \frac{K}{2}\left(1-\frac{\alpha}{r}+\frac{L}{K}+\sqrt{\left(1-\frac{\alpha}{r}+\frac{L}{K}\right)^2-4\frac{L}{K}}\right)$ $y^A_{I_t} = 1$	
	Extinction	Ø	$N_E = 0$	$N_E = 0$	
Conditions					
	Recovery			$\frac{\alpha}{\phi(N_R)} > \beta$	
	Partial infection	$\alpha < r$ and $\frac{\alpha}{\phi(N_R)} < \beta < \frac{\alpha}{\phi(N^S_{I_t})}$ or $\alpha > r$ and $\frac{\alpha}{\phi(N_R)} < \beta$	Ø	$\alpha < r\left(1-\sqrt{\frac{L}{K}}\right)^2$ and $\frac{\alpha}{\phi(N_R)} < \beta < \frac{\alpha}{\phi(N^A_{I_t})}$ or $\alpha > r\left(1-\sqrt{\frac{L}{K}}\right)^2$ and $\frac{\alpha}{\phi(N_R)} < \beta < \frac{\alpha}{\phi(\sqrt{LK})}$	Ø
	Total infection	$\alpha < r$ and $\frac{\alpha}{\phi(N^S_{I_t})} < \beta$		$\alpha < r\left(1-\sqrt{\frac{L}{K}}\right)^2$ and $\frac{\alpha}{\phi(N^A_{I_t})} < \beta$	
	Extinction	Ø	$\alpha > r$ and $\frac{\alpha}{\phi(N^S_{I_t})} < \beta$	$\alpha > r\left(1-\sqrt{\frac{L}{K}}\right)^2$ and $\frac{\alpha}{\phi(\sqrt{LK})} < \beta$	

Table 2. Thresholds for infection spread for the cases of density-dependent and frequency-dependent transmissions.

Characteristics of the host–parasite pair			Conditions for infection's spread	Threshold value
Density-dependent transmission				
Standard	$\beta\phi(K) < r$	$r < \alpha$		No spread
		$r > \alpha$	$N < T_{dd}^{S}$	$T_{dd}^{S} = K\dfrac{r-\alpha}{r-\beta\phi(K)}$
	$r < \beta\phi(K)$	$r < \alpha$	$N > T_{dd}^{S}$	$T_{dd}^{S} = K\dfrac{\alpha-r}{\beta\phi(K)-r}$
		$r > \alpha$		Systematic spread
Allee effect	$\beta\phi(K) < r\left[1-\dfrac{K}{4L}\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}\right)^2\right]$			No spread
		$1+\sqrt{L/k} < \alpha/r$		No spread
	$r\left[1-\dfrac{K}{4L}\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}\right)^2\right] < \beta\phi(K) < r$	$1+\sqrt{L/k} > \alpha/r$	$T_{dd}^{A-} < N < T_{dd}^{A+}$	$T_{dd}^{A-} = \dfrac{K}{2}\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}-\sqrt{\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}\right)^2+4\dfrac{L}{K}\left(\dfrac{\beta\phi(K)}{r}-1\right)}\right)\Big/\left(1-\dfrac{\beta\phi(K)}{r}\right)$
				$T_{dd}^{A+} = \dfrac{K}{2}\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}+\sqrt{\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}\right)^2+4\dfrac{L}{K}\left(\dfrac{\beta\phi(K)}{r}-1\right)}\right)\Big/\left(1-\dfrac{\beta\phi(K)}{r}\right)$
	$r < \beta\phi(K)$	$1+\sqrt{L/k} < \alpha/r$	$N > T_{dd}^{A}$	$T_{dd}^{A} = \dfrac{K}{2}\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}+\sqrt{\left(1+\dfrac{L}{K}-\dfrac{\alpha}{r}\right)^2+4\dfrac{L}{K}\left(\dfrac{\beta\phi(K)}{r}-1\right)}\right)\Big/\left(\dfrac{\beta\phi(K)}{r}-1\right)$
		$1+\sqrt{L/k} > \alpha/r$		Systematic spread

Table 2 (*Continued*)

	Characteristics of the host–parasite pair	Conditions for infection's spread	Threshold value
Frequency-dependent transmission			
Standard	$\alpha < \beta\phi(K) + r$	$N < T_{fd}^{S}$	$T_{fd}^{S} = K\left(1 - \frac{\alpha}{r} + \frac{\beta\phi(K)}{r}\right)$
	$\alpha > \beta\phi(K) + r$		No spread
Allee effect	$\alpha < \beta\phi(K) + r\left(1 - \sqrt{1 - L/K}\right)^2$	$T_{fd}^{A-} < N < T_{fd}^{A+}$	$T_{fd}^{A-} = \frac{K}{2}\left(1 + \frac{L}{K} - \frac{\alpha}{r} + \frac{\beta\phi(K)}{r} - \sqrt{\left(1 + \frac{L}{K} - \frac{\alpha}{r} + \frac{\beta\phi(K)}{r}\right)^2 - 4\frac{L}{K}}\right)$
			$T_{fd}^{A+} = \frac{K}{2}\left(1 + \frac{L}{K} - \frac{\alpha}{r} + \frac{\beta\phi(K)}{r} + \sqrt{\left(1 + \frac{L}{K} - \frac{\alpha}{r} + \frac{\beta\phi(K)}{r}\right)^2 - 4\frac{L}{K}}\right)$
	$\alpha > \beta\phi(K) + r\left(1 - \sqrt{1 - L/K}\right)^2$		No spread

(The corresponding table for the alternative model needs only a change of α into $\alpha + b$).

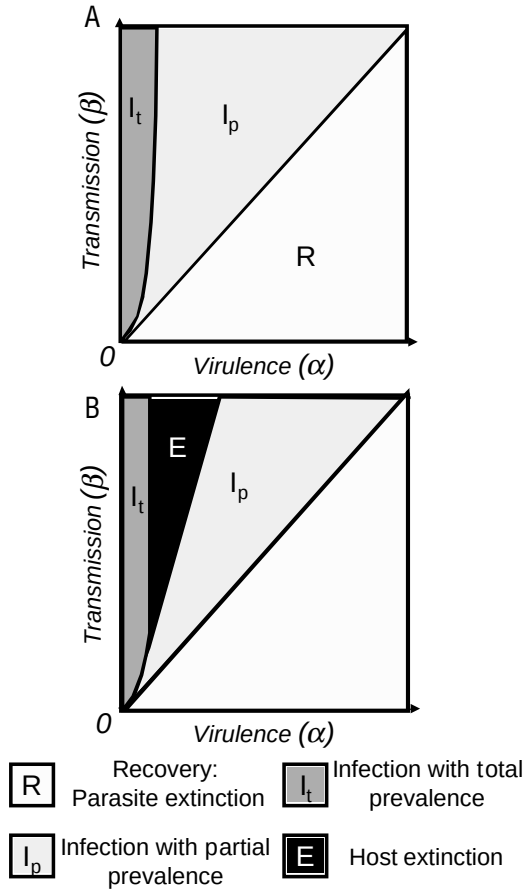

Fig. 2. Fate of the host–parasite system according to the values of the per encounter transmission rate (β) and virulence (α) of the parasite, for a standard population (A) and for a population with an Allee effect (B). This figure is drawn from the models with the mixed transmission term, but results are identical with the density-dependent term and qualitatively similar with the frequency-dependent term. Note that host extinction is always a stable equilibrium in (B).

Conditions for infection spread

When an infected individual is introduced into a healthy population, infection may spread depending on the host population size. The recruitment of infected individuals through either transmission or birth must exceed the number of deaths: $\beta\phi(N) > \alpha - rf_S(N)$.

In the dd-model, the propagation of parasites requires that N either exceeds (if $\beta\phi(K) > r$) or remains below (if $\beta\phi(K) < r$) an infection threshold (Fig. 3). In the fd-model, infection spreads below a given density threshold (see Table 2 for the values of these thresholds).

The number of encounters in the case of mixed transmission term being always smaller than in both other cases, the range of density leading to infection spread for the mixed model is smaller than the intersection of the two ranges of the dd and the fd cases (Fig. 1).

The existence of a threshold above which infection cannot spread can appear intriguing. It simply depends on the assumption of saturation of contacts when density increases. Indeed, this threshold is generated when the increase of the parasite transmission rate is not sufficient to overcome the decrease of the host growth rate. Although implicitly present in some studied models (Getz and Pickering 1983) this threshold has, to our knowledge, never been mentioned before (but see Deredec and Courchamp 2003).

Population with an Allee effect

Equilibrium points

Study of system (Eq. 4) reveals that in the presence of an Allee effect, population extinction is always stable: when the population density is low enough its collapse becomes unavoidable and individual reintroductions cannot generate a new population.

As in the standard case, when virulence is so high that, at carrying capacity, disease-induced mortality exceeds the rate of transmission events, infected individuals die too rapidly to transmit parasites. Infection may then disappear, in which case the population will reach carrying capacity. Otherwise, infection establishes within the population, possibly leading to host population extinction (see Fig. 2B and Table 1 for equilibrium values and conditions of existence).

Conditions for infection spread

In the presence of an Allee effect, infection can spread from a single infected individual only if the host population is between two density thresholds. The Allee effect shortens the range of densities enabling infection spread below the carrying capacity (Fig. 1) and thereby protects the host population.

In the dd-model, the Allee effect either has no tangible effect (keeping the infection threshold above the carrying capacity: Fig. 3B, 3C), or it reduces the range of population sizes that permits infection spread. This is achieved either by lowering the threshold above which infection fails (Fig. 3D) or, mainly, by increasing the threshold below which infection fails (Fig. 3A). Over these four depicted cases (Fig. 3A–D), one is biologically more frequent: when the transmission rate is higher than both the parasite virulence and the host growth rate (Fig. 3A). Although not impossible, the three other cases are less frequently encountered in natura, since either virulence or host growth rate exceeding transmission would select against the parasite. When the range of host density enabling infection spread is limited by an upper threshold (Fig. 3C, 3D), the Allee effect introduces a second, lower threshold below which infection is not possible either. Note that, although this amounts to protecting the population against infection, host extinction is also likely in this latter case, because of very low

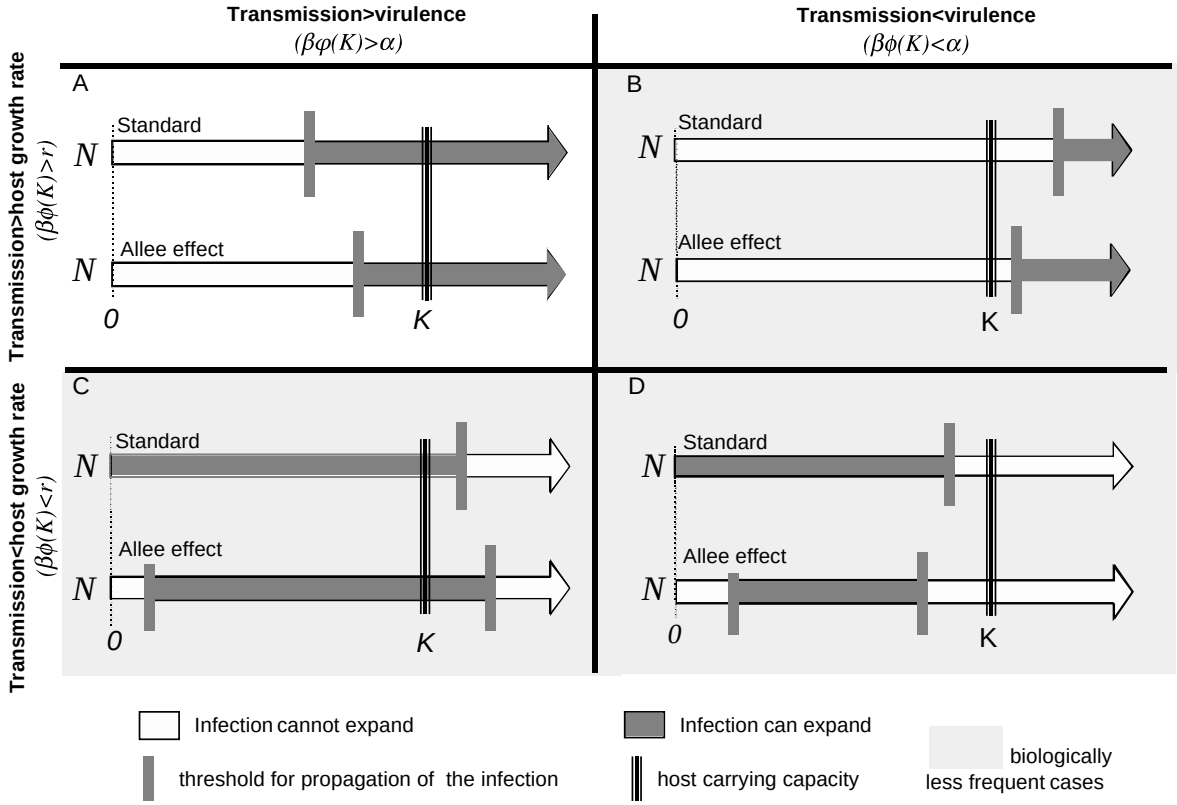

Fig. 3. Ranges of population size enabling infection spread, depending on the characteristics of the host–parasite pair: transmission βϕ(K), virulence (α) and host population growth rate (r), with the density-dependent term. The three shaded boxes are biologically less frequent: the corresponding characteristics of the parasites are not those best promoting selection. The presence of an Allee effect in the biologically most frequent case (transmission exceeding both virulence and host growth rate: $\beta\phi(K) > \alpha$ and $\beta\phi(K) > r$) yields an increase of the threshold below which infection fails, and therefore of the range of populations sizes that are protected from infection. The results obtained with the frequency dependent term are described by the figure C if transmission exceeds virulence, by the figure D if it does not exceed. The corresponding diagram for the alternative model needs only a change of α into α+b.

host density. The same general conclusions are found for the fd-model (Fig. 3C, 3D).

Quantitative impact of the Allee effect

Three types of effects can occur. First, when the parasite becomes established in the whole population, the population reduction is greater in the presence of an Allee effect. This size reduction increases with L, the strength of the Allee effect (Fig. 4). The ratio of population sizes with and without an Allee effect may peak at $1+\sqrt{\alpha/r}$ and may thus approach 2 for the highest values of α/r. An Allee effect could thus reduce some infected population by half. Most of the time, the host population cannot escape extinction for stronger Allee effects (larger L, Fig. 4D).

Second, all the parasites able to drive the host population size under the threshold $\sqrt{LK}$ will systematically eradicate their host, while they would have remained endemic in the absence of Allee effect (Table 1). In the fd-model, the Allee effect increases the range of parasites species (i.e. the values of α and β) driving host populations to extinction. Regardless of the model, the stronger the Allee effect, the larger the range of values for parasite traits that can eliminate their host.

Third, for populations subject to Allee effects, total prevalence is only possible for very low virulence (Fig. 2B). For transmission rates strong enough to produce high prevalence, increasing virulence reduces the host population size such that its growth rate becomes negative. Host extinction is then systematic. Higher virulence leads to rapid death of infected hosts before transmission is fully effective. The prevalence is thus partial. In this latter case, the population size remains unchanged, but the number of infected individuals is reduced, this reduction increasing with the strength of the Allee effect. With the fd transmission, host extinction is the only alternative to the infection of the whole population that establishes when virulence is very low.

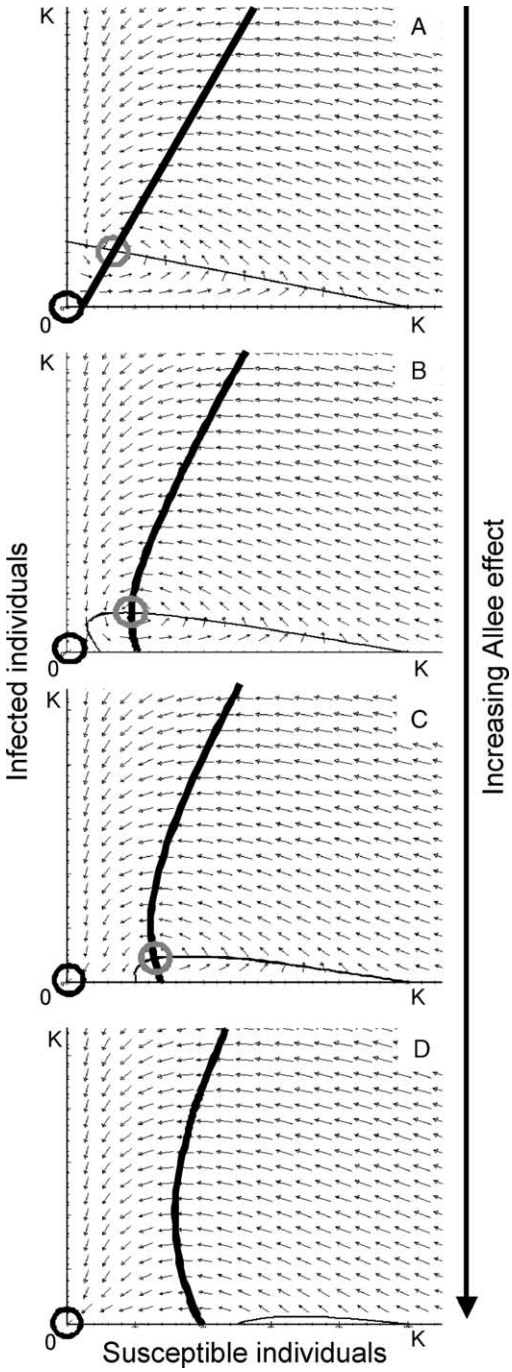

Fig. 4. Phase portraits of host–parasite populations for increasing strength of the Allee effect: susceptible vs infected individuals, with $\alpha = 1.2$, $\beta\phi(K) = 3.5$, $r = 1.1$, $K = 100$ and A. $L = 0$, B. $L = 10$, C. $L = 25$, D. $L = 40$ in case of dd-transmission term. The curves represent the isoclines for the zero growth rate of susceptible individuals (thin line) and infected individuals (thick line). The small arrows are vectors showing towards which stable equilibrium (host extinction, black circles, or infected host persistence, grey circles) the system is led. As the strength of the Allee effect increases (panels (A) to (D), the parasite prevalence decreases until extinction becomes unavoidable (only the (0,0) equilibrium remains, (D)).

Alternative model

System 6 (Eq. 6) globally behaves similarly (see Deredec 2005 for details), to such a point that figures and tables concerning the previous model are mostly applicable to this alternative model, sometimes needing only to replace α by $\alpha + b$ (Table 2, Fig. 1, 3). The only major difference is that, when infected, the reduction of the population by the Allee effect is now systematic. Also, the state of total infection is absent from this model.

Discussion

Regardless of the model considered, the combination of Allee effects and parasitism can have contrasting effects on host population dynamics. On one hand, populations subject to an Allee effect are better protected from parasite infection, as the conditions required for infection spread are restricted: thresholds between which infection is possible are closer together. In addition, when infection establishes in the population, the Allee effect reduces the number of infected individuals. As long as the Allee limit is below a certain value, a stronger Allee effect results in fewer infected hosts in infected populations.

On the other hand, the existence of Allee effects has several detrimental consequences in the presence of parasites. First, the extinction of host populations becomes inevitable at sufficiently low densities, regardless of the life history traits of the parasite. Second, when a parasite invades the host population, it results most of the time in a larger reduction of the total population size than in the absence of Allee effects. From a practical point of view, the future size of a reintroduced population may be overestimated as much as twice its real value if Allee effects are overlooked. Third, if transmission is sufficiently large compared to parasite-induced mortality and if the Allee effect is strong enough, the extinction of the host becomes unavoidable. If they have an Allee effect, host populations can be driven to extinction by a larger range of parasites. For all these points, the stronger the Allee effect, the stronger its impact.

Thus, host populations are better protected from parasite infection when their structure produces an Allee effect, but when infection establishes, the presence of an Allee effect may intensify its detrimental consequences on the host population (Fig. 5). Although unexpected, the existence of such opposite effects is not illogical. The simultaneous protection from parasites invasion and increased vulnerability once some individuals have become infected may be seen as the expression of a modified balance. The same way an organism may exhibit a tradeoff between protection against external enemies and defences against internal ones, a species may exhibit a negative correlation between different

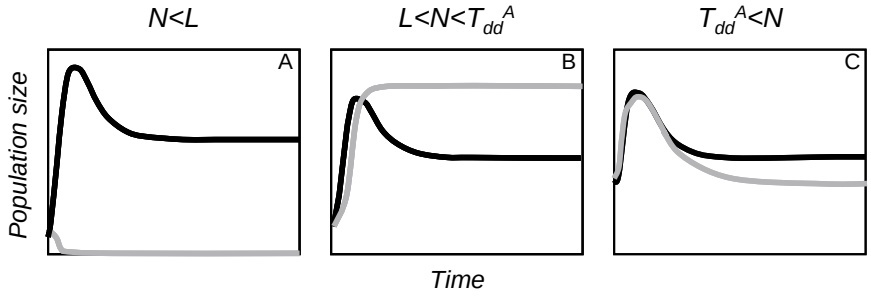

Fig. 5. Propagation of infection from a single infected individual depending on the population size for populations with (grey) or without (black) an Allee effect in case of dd-transmission term. In a situation where the standard population cannot escape infection spread, the Allee population may experience different scenarios. When N is below the Allee limit (A), infection spreads and leads to population extinction. When N is above this limit, but below the infection threshold (B), Allee effect populations are protected from infection while classical populations are reduced in size. When N is above both thresholds (C), host populations with an Allee effect suffer a greater size reduction than standard populations. Here, $K = 100$; $L = 12$; $N_{0(A)} = 10$; $N_{0(B)} = 15$; $N_{0(C)} = 40$; $r = 2.4$; $\alpha = 1.03$; $\beta\phi(K) = 2.5$, giving $T_S < 0$ and $T_A = 17.2$.

stages of defence of populations or social groups from parasitism. The presence of an Allee effect might thus result in a modification of the repartition of defences among these different stages. For example, the social structure and behaviour of African wild dogs (*Lycaon pictus*), a species with a strong Allee effect (Courchamp and MacDonald 2001, Courchamp et al. 2002) is such that contact rates between packs are low, thus limiting contact with parasites (and thus pack infection), but contact within packs are very high, often dooming the pack if a virus gets in (Kat et al. 1995).

Although this was not the primary aim of this paper, it may also be interesting to consider these results from the parasites point of view. Regardless of the model and the transmission term used, the Allee effect of the host population has only detrimental effects on the parasite population: (i) it hinders parasite propagation and then establishment into a healthy population; (ii) it more often leads to parasite extinction through host extinction; (iii) in infected populations, it reduces the number of infected hosts, i.e. the size of the parasite population. This may have important consequences in conservation biology since host populations subject to Allee effects may submit specialist parasites too strong selection with important evolutionary consequences.

As in any modelling exercise, we must ask whether our results correspond to actual biological effects or are driven by our implicit assumptions. For instance, the mathematical form of the function we chose for the Allee effect might contribute to increase the classical infection threshold and to reduce the number of infected hosts (Deredec 2005). Indeed, this function chosen to reduce population growth rate at low density, also inevitably alters it slightly at higher density. Any other continuous function would behave so. The question is thus whether this change in the growth rate at high density is disturbingly unrealistic. The definition of an Allee effect does not imply any impact at higher density, but to our knowledge, no research has been done in order to evaluate the precise form of the growth rate at higher density. It is likely that in many cases the physiological or social mechanisms responsible for an Allee effect at low density will also affect the population dynamics at higher densities.

Comparison of the two classical transmission terms (frequency- and density-dependent transmission terms) highlights that their main conclusions are identical. However some differences remain (Deredec 2005). Since neither of these classical transmission terms are valid for all densities, it is important to distinguish which property is related to low density, and which to high density. Therefore our mixed transmission term is a necessary tool to capture the entire behaviour of transmission upon the large range of densities that the system may take. Being bound between two extremes, this mixed function produced smoothened results: the range of densities leading to infection spread is shorter than the range defined by the intersection of the two ranges defined in each classical transmission term case. The use of such a mixed transmission term is probably a novelty in model analysis (but see Barlow 2000, Fenton et al. 2002). It is certainly the best way to capture the system dynamics for all the host density, which is especially crucial in systems with varying densities.

Because the basic model (system (1)) had an intrinsic flaw, we have presented the study of two different models that are complementary in both their approaches of the complex reality of our biological system and in their way of solving the original problem. Although it may appear unsatisfying to present more than one model, be it only because it does not ease the reading of the manuscript, we deem the congruence of conclusions obtained through two models comforting with regards to its robustness, and therefore worthy of presentation.

This study may provide important insights for the management of threatened populations. One obvious precaution to be taken, regardless of parasite effects, is to prevent host population sizes from dropping below a critical threshold. Our results also concur on the importance of increasing the carrying capacity of the habitat to protect populations from collapsing, since decreasing L/K (i.e. increasing the distance between the carrying capacity and the Allee limit) appears to be an effective means for decreasing the range of fatal parasites

species. As infection thresholds depend on the life history characteristics of the parasite, our work may also help quantify the threat of a given parasite to a host population. Generally, it appears that protection from easily transmitted parasites (high β) is more important than protection from virulent parasites (high α). Preventing parasite transmission can be achieved by a number of management actions in various contexts.

Our work emphasises the necessity of simultaneously taking into account parasitism and possible Allee effects when developing conservation strategies for threatened populations. Our study could also be a stepping-stone for further, more specific models aiming to improve the management of pest species, by helping to select an effective parasite for biological control (Hopper and Roush 1993).

With simple models, we showed that the Allee effect has opposite effects on host populations: it can protect them from infection, but when it fails to do so, the impact of parasites is much heavier. This study adds to the growing framework on population dynamics of interacting species, partly filling a gap in the understanding of the impact of natural enemies on populations subject to Allee effects. We believe further theoretical studies on these aspects are strongly needed.

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