Population collapse to extinction: the catastrophic combination of parasitism and Allee effect

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Infectious diseases are responsible for the extinction of a number of species. In conventional epidemic models, the transition from endemic population persistence to extirpation takes place gradually. However, if host demographics exhibits a strong Allee effect (AE) (population decline at low densities), extinction can occur abruptly in a catastrophic population crash. This might explain why species suddenly disappear even when they used to persist at high endemic population levels. Mathematically, the tipping point towards population collapse is associated with a saddle-node bifurcation. The underlying mechanism is the simultaneous population size depression and the increase of the extinction threshold due to parasite pathogenicity and Allee effect. Since highly pathogenic parasites cause their own extinction but not that of their host, there can be another saddle-node bifurcation with the re-emergence of two endemic equilibria. The implications for control interventions are discussed, suggesting that effective management may be possible for $\mathcal{R}_0 \gg 1$.

Keywords: fold catastrophe; epizootiological model; Allee effect; wildlife disease; species disappearance

AMS Subject Classification: 92D30 epidemiology; 92D25 population dynamics (general); 37G10 bifurcations of singular points

1. Introduction

Parasites and pathogens are now recognized as integral parts of virtually all ecosystems [18,38,41]. There is a large body of evidence showing that disease agents can substantially affect their host population [4,28,37]. Besides causing a depression in population size, infectious diseases have also been attributed to playing a role in the extinction of species [13,14,30,50,54,60].

Transmission of disease is influenced by aggregation patterns in the host population as well as its social organization and behavioural traits [19,23]. Two different types of incidence (new infections per unit time) are usually distinguished [7,31,43]. Density-dependent (mass action) transmission assumes that the effective contact rate between susceptible and infective individuals increases linearly with population size. In contrast, the number of contacts is independent of population size if transmission is frequency-dependent (also called standard incidence or proportionate mixing).

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Populations subject to density-dependent transmission have been regarded as relatively safe from disease-induced extinction, as infection vanishes with declining population size – but see [15,36,57] for other forces causing extinction at low density. Frequency-dependent incidences are well known to have the capability of driving populations to extinction, see the review in [14]. This is because disease transmission goes on even in small populations due to the constant contact rate.

Since the influential paper by Anderson and May [3] highlighting the role of parasites in regulating natural populations, numerous mathematical models have been developed, which also take the host population as a dynamic variable [5,8,10,27,39,48] rather than fixing it at a constant level (as has been traditionally assumed for human disease models in developed countries). Epidemic models with frequency-dependent incidence typically predict host population extinction in appropriate parameter regimes [11,24,25,61]. The extinction is preceded by a continuous population decline when the control parameters approach the eradication regime. The transition to extinction, therefore, takes place gradually, and small population sizes are typical correlates of imminent species loss.

This paper highlights that extinction can occur directly and abruptly in a catastrophic collapse from high population levels if the host population exhibits a strong Allee effect (AE) (also called critical depensation) [12,55]. The AE describes the phenomenon that populations benefit from large population sizes (e.g. due to higher success rates in finding mating partners, predator dilution or reductions in inbreeding). At low densities, populations experience positive density dependence as they have difficulties in maintaining social functioning, for instance. If the AE is strong (weak), the population growth rate is negative (reduced) at low densities. AEs have been demonstrated in, or proposed for, an increasing number of species, e.g. saiga antelopes [45], polar bears [47] and Atlantic cod [51].

Only recently, the impacts of an AE have been considered in epidemiological models. (Although this paper is mainly motivated by animal diseases and therefore epizootiological problems, it uses epidemiological notation throughout). Deredec and Courchamp [15] as well as Hilker *et al.* [34, 35] conclude that the combination of parasitism and AE generally increases the likelihood of extinction. Thieme *et al.* [57] and Hilker *et al.* [36] consider density-dependent transmission and show that host extinction is possible therein as well. Moreover, these models exhibit a rich dynamical behaviour including super- and sub-critical Hopf bifurcations, homoclinic bifurcations and tristability.

This article reconsiders a simple epidemiological model of SI type with frequency-dependent incidence and a strong AE in the host demographics [35]. While the original publication is primarily concerned with spatio-temporal dynamics (additionally taking into account spatial diffusion), the current focus is on the bifurcation behaviour of the spatially homogeneous model without diffusion. A typical phenomenon in epidemic models with strong AE is bistability, i.e. the outcome (extinction or survival of the population) depends on the initial condition. A super-critical number of infectives can eradicate the host even if its initial state is beyond the minimum viable size defined in the disease-free system. This paper reports that a spontaneous population crash to extinction is possible for all initial conditions. This takes place via a saddle-node bifurcation, in which two endemic equilibria disappear. The loss of the endemic attractor renders the system monostable with extinction as inevitable outcome.

The remainder of this paper is organized as follows. Section 2 briefly introduces the model and derives basic reproduction numbers. They are used in Section 3 to summarize the equilibria and their stability in biologically insightful terms. Section 4 presents a numerical continuation and bifurcation study. The main emphasis is on how varying control parameters impacts the persistence of the host population and the establishment of the disease. Section 5 highlights the relevance for management actions and illustrates the differences to host populations without the AE. Section 6 compares the bifurcation behaviour with a backward bifurcation (BB), pointing out that the two scenarios are fundamentally different, even though both of them involve multiple
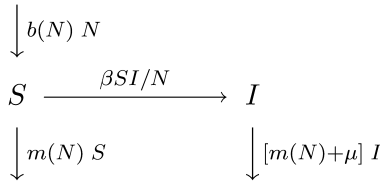
non-trivial stationary states and saddle-node bifurcations. Finally, Section 7 presents the main conclusions and discusses their implications for the understanding of extinction dynamics.

2.　Model description

This section describes the epidemic compartment model with frequency-dependent disease transmission and a strong AE in the host demographics [35]. It also introduces basic reproduction numbers that will be used to express the existence and stability of stationary states in the following section.

2.1.　*Model assumptions and equations*

The model structure can be illustrated by the following transfer diagram

$$
\begin{array}{c}
\quad\quad\Big\downarrow b(N)\,N \\
S \xrightarrow{\quad \beta SI/N \quad} I \\
\Big\downarrow m(N)\,S \quad\quad\quad \Big\downarrow [m(N)+\mu]\,I
\end{array}
$$

Note that all quantities are dimensionless. The total host population size $N = N(t)$ at time $t > 0$ is assumed to consist of a susceptible (S) and an infective (I) part, $N = S + I$. That is, there is no recovery from the disease. Disease transmission is frequency-dependent with coefficient β describing the effective contact rate, henceforth referred to as transmissibility. The disease is pathogenic and induces an additional per capita mortality rate μ in the infectives, henceforth referred to as pathogenicity (sometimes also called virulence). The background mortality is described by the density-dependent per capita death rate $m(N)$. There is no vertical transmission, i.e. the offspring of infectives is susceptible. The per capita birth rate $b(N)$ is density-dependent as well. The difference between birth and natural death rate is the net growth rate $g(N) = b(N) - m(N)$ of the population. In the absence of disease, the population dynamics is described by $dN/dt = g(N)N$. In the presence of disease, it is convenient to formulate the model in (N, i) state variables, where $i = I/N$ is the prevalence of the disease (fraction of population being infective). The model equations then read

$$
\frac{\mathrm{d}N}{\mathrm{d}t} = [g(N) - \mu i]N, \tag{1a}
$$

$$
\frac{\mathrm{d}i}{\mathrm{d}t} = [(\beta - \mu)(1 - i) - b(N)]i. \tag{1b}
$$

Following conventional lines, the strong Allee effect is modelled by a quadratic net growth rate

$$
g(N) = r(1 - N)(N - u), \tag{2a}
$$

where r is the intrinsic growth rate, the carrying capacity is scaled to 1 and the Allee threshold (sometimes also called Allee limit or minimum viable population size) is u, $0 < u < 1$. The AE is assumed to be concentrated in the birth rate (reflecting a mate shortage due to encounters based on bimolecular collisions and crowding effects with linearly decreasing offspring survival; see [36]

for more details). The death rate follows density-dependent regulation motivated by intraspecific competition:

$$b(N) = r \left[-N^2 + \left(1 + u + \frac{1}{r} \right) N + \frac{d}{r} \right], \tag{2b}$$

$$m(N) = d + ru + N. \tag{2c}$$

Parameter d determines the baseline levels of fecundity and mortality. Note that d does not affect $g(N)$ and that $b(N)$ is positive at least for all $N \leq 1 + r(1 + u)$, which is larger than the carrying capacity and therefore sufficient for the problem at hand.

2.2. *Reproduction numbers*

The basic reproduction number of the disease is the number of secondary cases produced by a single infective during its entire lifetime when it is introduced into a completely susceptible population (i.e. $N \equiv S$). For models (1) and (2), this yields

$$\mathcal{R}_0(N) = \frac{\beta}{\mu + m(N)} = \frac{\beta}{\mu + d + ru + N}. \tag{3}$$

Note that population size is variable and affects the basic reproduction number. The latter is, therefore, expressed as a function of N. If $\mathcal{R}_0(N) < 1$, the disease cannot spread and will disappear. If $\mathcal{R}_0(N) > 1$, in contrast, the disease can initially multiply and spread. The basic reproduction number thus is a pertinent quantity for parasite invasion.

If the host population is at carrying capacity, the disease can invade if

$$\mathcal{R}_0 := \mathcal{R}_0(1) > 1.$$

It can be shown that the disease-free equilibrium at carrying capacity loses its stability under this condition. Once the disease invades, it can either persist endemically or drive the host population to extinction (see the next section).

If the host population goes extinct ($N \to 0$), the fraction of infectives within the vanishing population can be strictly positive or zero, depending on whether the infectives decay more slowly or more quickly than the total population, respectively. The total population approximately decays at rate ru if N is assumed to be small, whereas the infectives decay at an approximate rate $ru + d + \mu - \beta$ if I is small compared with N. The difference between these two growth rates at zero population density can be expressed in the quantity

$$\mathcal{R}_i = \frac{\beta}{\mu + d},$$

which basically gives the number of secondary infections discounted by the fact that host population size vanishes [32,33,56]. If $\mathcal{R}_i < 1$, the infectives decay faster than the total population with the prevalence approaching zero, whereas if $\mathcal{R}_i > 1$ the infectives remain a positive fraction in the host population and the prevalence approaches a constant value.

3. Equilibria and their stability

The existence and stability of stationary states of system (1) have been studied in [35] for generalized demographic functions yielding a strong AE, that is for $b(N)$ being concave and

$m(N)$ being non-decreasing and convex. This section briefly recalls these results, applied to the demographic functions (2), and reformulates them in terms of reproduction numbers. This allows for a novel, biologically more insightful interpretation, which forms the basis for understanding the bifurcation behaviour investigated in Section 4.

Model (1) and (2) has the following four (semi-)trivial equilibria $E = (N^*, i^*)$.

- $E_0 = (0, 0)$: The trivial extinction state is a stable node if $\mathcal{R}_i < 1$ and an unstable node otherwise:
- $E_u = (u, 0)$: The Allee threshold state is a saddle point if $\mathcal{R}_0(u) < 1$ and an unstable node otherwise:
- $E_1 = (1, 0)$: The carrying capacity state is a stable node if $\mathcal{R}_0 < 1$ and an unstable node otherwise:
- $E_i = (0, i_0^*)$ with $i_0^* = (\beta - \mu - d)/(\beta - \mu)$: The extinction state with positive prevalence in the limit process state exists if $\mathcal{R}_i > 1$ and is always a stable node. Note that E_i coincides with E_0 if expressed in (N, I) or (S, I) state variables.

A necessary condition for non-trivial equilibria is $\beta > \mu$; otherwise, $i \to 0$ as $t \to \infty$, cf. Equations (1b). Endemic equilibria can be found as the intersections of the two quadratic nullclines

$$i = \frac{g(N)}{\mu} \quad \text{and} \quad i = \frac{1 - b(N)}{(\beta - \mu)}. \tag{4}$$

Obviously, there can be up to two non-trivial stationary states $E_{+,-} = (N^*_{+,-}, i^*_{+,-})$. From [35] it is known that E_+ is locally stable and that E_- is a saddle point, if they exist. Periodic solutions can be ruled out. For the demographic functions (2), the endemic equilibria as well as their eigenvalues can be solved explicitly, but are too cumbersome to be presented here. Instead, the remainder of this section shall investigate the conditions under which the endemic equilibria exist, with the goal to formulate them in terms of the basic reproduction number $\mathcal{R}_0$.

Introducing the auxiliary function

$$\Phi(N) = (\beta - \mu)(g(N) - \mu) + \mu b(N)$$

allows us to determine the total population size N^* in endemic equilibrium as the root of a quadratic equation. Note that $N^* \in (u, 1)$; otherwise $i^* = g(N^*)/\mu$ would be negative. $\Phi(N)$ is a parabola that is open at the bottom, and one has $\Phi(u) < \Phi(1)$. Let $N_{\max}$ be the population size for which $\Phi(N)$ is maximal. The following cases can be distinguished.

First, consider $\Phi(1) > 0$. If $\Phi(u) > 0$, there is no root of $\Phi(N)$ in the interval of interest. Endemic equilibria, therefore, do not exist. If, conversely, $\Phi(u) < 0$, there is a single root N^*_-, with $u < N^*_- < \min\{N_{\max}, 1\}$.

Second, consider $\Phi(1) < 0$, which implies $\Phi(u) < 0$. Then the number of roots depends on the location of the parabola's top $\Phi(N_{\max})$: (i) if $\Phi(N_{\max}) > 0$, there are two roots N^*_- and N^*_+ with $u < N^*_- < N_{\max} < N^*_+ < 1$; (ii) if $\Phi(N_{\max}) = 0$, the previous two roots collide in a unique root $N^* = N_{\max}$; (iii) if $\Phi(N_{\max}) < 0$, there is no root. The critical relation $\Phi(N_{\max}) > 0$ for two endemic equilibria to exist can be solved explicitly in terms of model parameters (see Appendix). Reformulating in terms of the basic reproduction number by substituting one of the epidemiological parameters, one obtains that

$$\mathcal{R}_0 < \mathcal{R}_0^{c1} \quad \text{or} \quad \mathcal{R}_0 > \mathcal{R}_0^{c2}, \tag{5}$$

where $\mathcal{R}_0^{c1} < \mathcal{R}_0^{c2}$. These critical values can be expressed in terms of model parameters, cf. the Appendix. If this is done by substituting transmissibility β, $\mathcal{R}_0^{c2}$ is always unfeasible, which is

why the second relation in Equation (5) never exists in this case. $\mu > \mu^c$ is required for $\mathcal{R}_0^{c1}$ to be feasible, with

$$\mu^c = \frac{r(1-u)^2}{4}. \tag{6}$$

If $\mathcal{R}_0$ is expressed in terms of pathogenicity μ, both critical values $\mathcal{R}_0^{c1,c2}$ are feasible provided that $\beta > \beta^c$, where

$$\beta^c = \frac{A + \sqrt{A^2 - 4B}}{2}, \quad A = 1 + u + 2d + r(1+u^2), \quad B = (d + ru)(1+d) + r^2 u^2. \tag{7}$$

The biological reason behind the existence of one or two critical values $\mathcal{R}_0^{c1,c2}$ is given in the next section.

The above results can be summarized as follows in terms of the basic reproduction number, using the relationships $\Phi(1) = 0 \Leftrightarrow \mathcal{R}_0 = 1$ and $\Phi(u) = 0 \Leftrightarrow \mathcal{R}_0(u) = 1$. Figure 1 shows a corresponding sequence of phase plane illustrations.

First, if

$$\mathcal{R}_0 \leq \frac{\mu + d + ru + u}{\mu + d + ru + 1} = \frac{\mathcal{R}_0}{\mathcal{R}_0(u)} =: \mathcal{R}_0^u,$$
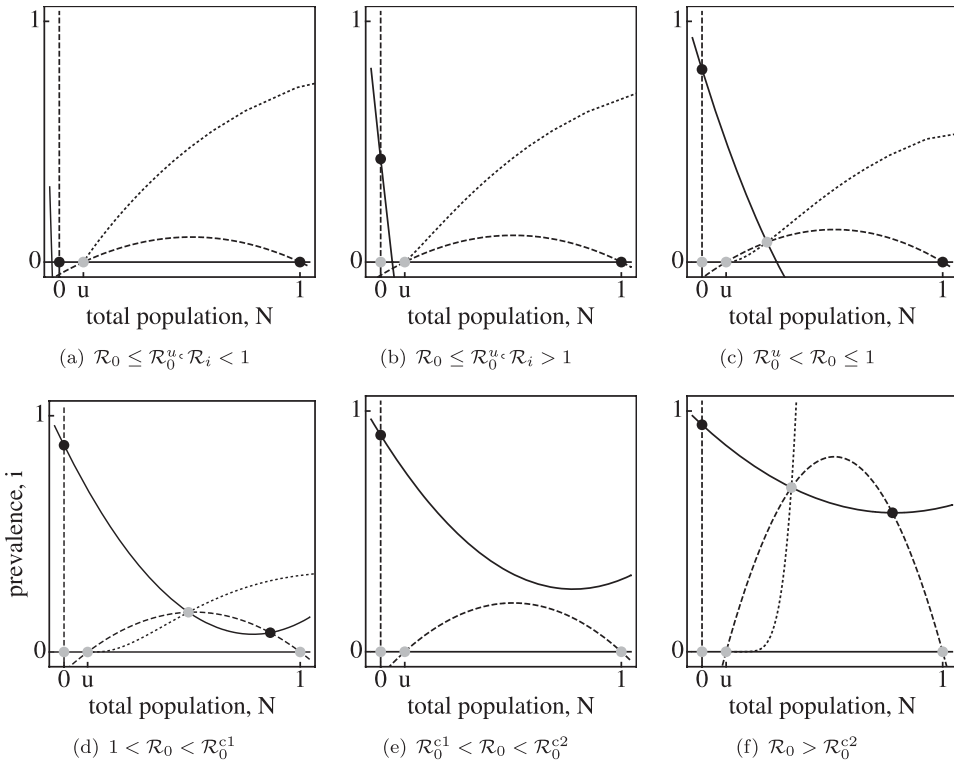
Figure 1. Phase plane illustrations with nullclines and stationary states of model (1) and (2). Solid (dashed) lines are the nullclines of infectives (total populations). Black (grey) points mark stable (unstable) equilibria. If the system is bistable (all panels except [e]), the stable manifolds of the saddle point separating the basins of attraction are shown in dotted lines. Parameter values: (a) $\mu = 3.9$, (b) $\mu = 3.65$, (c) $\mu = 3$, (d) $\mu = 2.4$, (e) $\mu = 2$, (f) $\mu = 0.5$ and $\beta = 4, u = 0.1, r = 2, d = 0.2$.

there is no non-trivial equilibrium. This condition simply states that $\mathcal{R}_0(u) \leq 1$, i.e. the disease cannot persist in a population that is at the edge of extinction due to the Allee effect. The system is bistable and approaches either the carrying capacity state or one of the extinction states (Figure 1a, 1b).

Second, there is a unique endemic equilibrium $\boldsymbol{E}_- = (N_-^*, i_-^*)$ if $\mathcal{R}_0^u < \mathcal{R}_0 \leq 1$. $\boldsymbol{E}_-$ is always an unstable saddle point. It organizes the effective extinction threshold in the presence of disease. That is, depending on the initial condition the population either goes extinct or survives at carrying capacity. The basins of attraction are separated by the stable manifolds plotted in dotted line in Figure 1c. Host eradication is even possible if the initial population size is larger than the Allee threshold. The disease, therefore, increases the extinction basin beyond u.

Third, consider $\mathcal{R}_0 > 1$. If $\mathcal{R}_0 < \mathcal{R}_0^{c1}$ or $\mathcal{R}_0 > \mathcal{R}_0^{c2}$, provided that these critical values exist, there are two non-trivial stationary states (Figure 1d, 1f). The additional equilibrium is $\boldsymbol{E}_+ = (N_+^*, i_+^*)$ is always locally stable and has a larger population size N_+^*, $N_-^* < N_+^* < 1$. The system remains bistable with either endemicity or host extinction as the eventual outcome. Conversely, if $\mathcal{R}_0^{c1} < \mathcal{R}_0 < \mathcal{R}_0^{c2}$, there is no non-trivial stationary state at all (Figure 1e). This occurs after the two endemic equilibria $\boldsymbol{E}_+$ and $\boldsymbol{E}_-$ coalesce and disappear at one of the critical values. It corresponds to too strong a disease, reducing population size N_+^* and at the same time increasing the effective extinction threshold. As a consequence, neither the disease nor the host can persist, and the system is rendered monostable.

4. Bifurcation behaviour

The bifurcation diagrams in Figure 2 show how the total population and prevalence change with varying basic reproduction number $\mathcal{R}_0$. It is assumed that $\mathcal{R}_0$ is varied by altering transmissibility β. If $\mathcal{R}_0 < 1$, the infection cannot invade the population at carrying capacity. However, if $\mathcal{R}_0 > 1$, the disease-free equilibrium $\boldsymbol{E}_1$ loses stability, and the locally stable endemic equilibrium $\boldsymbol{E}_+$ emerges in a transcritical bifurcation. It coexists with the unstable endemic equilibrium $\boldsymbol{E}_-$, which already arises if $\mathcal{R}_0 > \mathcal{R}_0^u$. The two non-trivial states collide and annihilate each other in a saddle-node bifurcation at $\mathcal{R}_0 = \mathcal{R}_0^{c1}$. The endemic solutions are suddenly lost, and the system abruptly undergoes a transition to a qualitatively very different behaviour. Namely, the population always goes extinct, whereas the population size N_+^* in endemic equilibrium was bounded well away from zero.

The critical basic reproduction number $\mathcal{R}_0^{c1}$ is a tipping point, marking the unexpected population collapse. For $\mathcal{R}_0 > \mathcal{R}_0^{c1}$, the dynamics, therefore, becomes monostable with the extinction state $\boldsymbol{E}_i$ being globally stable. For $\mathcal{R}_0 < \mathcal{R}_0^{c1}$, the system is bistable: one of the attractors always is an extinction state – either $\boldsymbol{E}_0$ or $\boldsymbol{E}_i$ depending on $\mathcal{R}_i \lessgtr 1$ – while the other attractor is either $\boldsymbol{E}_0$ or $\boldsymbol{E}_+$ corresponding to a disease-free or endemic scenario ($\mathcal{R}_0 \lessgtr 1$), respectively.

The basic reproduction number can not only be altered by controlling transmissibility β, but also by varying pathogenicity μ – the other disease-related parameter. Note that an increase in $\mathcal{R}_0$ corresponds to a decrease in μ, cf. Equation (3). Figure 3 shows the corresponding bifurcation diagram of the total host population. (The diagram for the prevalence is not shown but is qualitatively similar.) The first part of the bifurcation diagram is analogous to the one in Figure 2 for changing β. In particular, there is a saddle-node bifurcation for $\mathcal{R}_0 = \mathcal{R}_0^{c1}$, after which the population goes extinct. However, there is another critical reproduction number $\mathcal{R}_0^{c2}$, $\mathcal{R}_0^{c2} > \mathcal{R}_0^{c1}$, for which a second saddle-node bifurcation occurs. This gives rise to two endemic equilibria again. As abruptly as the endemic attractor disappears at $\mathcal{R}_0 = \mathcal{R}_0^{c1}$, it re-emerges after the extinction regime at $\mathcal{R}_0 = \mathcal{R}_0^{c2}$. Note that the phase-plane diagrams in Figure 1 correspond to a sequence of decreasing μ.
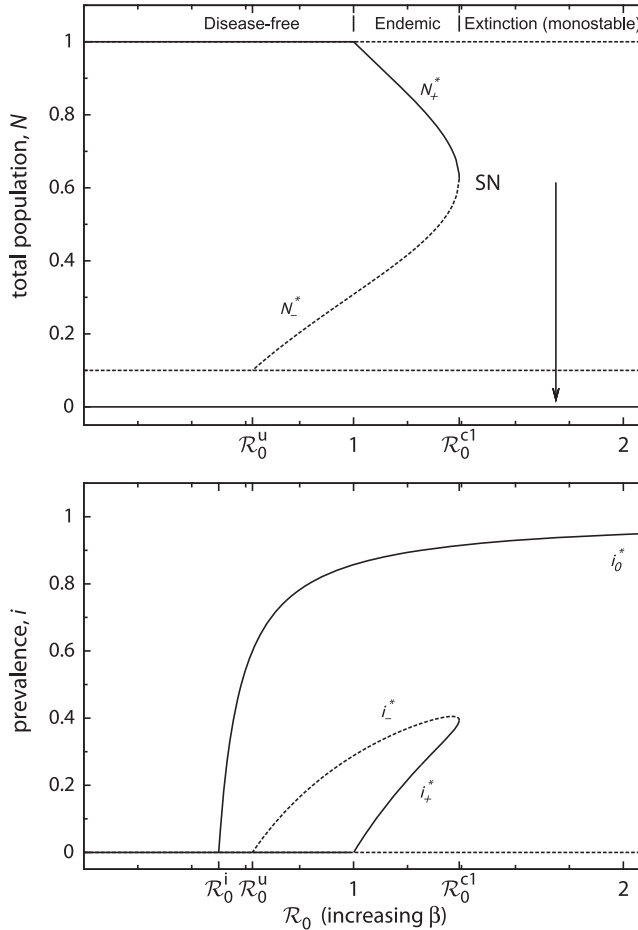
Figure 2. Bifurcation diagrams for varying basic reproduction number (by increasing β). Solid (dashed) lines correspond to stable (unstable) equilibria. The arrow indicates the abrupt population collapse from a level of a large population size N_+^* after a saddle-node (SN) bifurcation. The critical values of $\mathcal{R}_0$ are explained in the main text. Other parameter values: $\mu = 1, u = 0.1, r = 2, d = 0.2$.

It is well known [2,3] that the maximum degree of host population depression (here resulting in extinction) is achieved by parasites with moderate to low pathogenicity, i.e. moderate to high $\mathcal{R}_0$. If pathogenicity is too low (i.e. too large a $\mathcal{R}_0$), the disease cannot cause sufficient case fatalities, enabling the host to persist at endemic equilibrium with large population size ($\mathcal{R}_0 > \mathcal{R}_0^{c2}$). Conversely, if pathogenicity is too high (i.e. too small a $\mathcal{R}_0$), increased mortality of infectives limits their potential to spread the disease ($1 < \mathcal{R}_0 < \mathcal{R}_0^{c1}$) or even leads to their eradication ($\mathcal{R}_0 < 1$).

The sequence of 'endemicity – extinction – endemicity' can be observed in other models with frequency-dependent transmission as well. The novelty in a model with strong AE, however, is that the transition between the regimes takes place via two saddle-node bifurcations. This results in a much more drastic change of behaviour. The transitions in models without AE are induced by transcritical bifurcations, which correspond to a more continuous and smooth evolution.

Figure 4 summarizes the model behaviour in the two-parameter plane (μ, β). The dynamical outcome can, therefore, be characterized in terms of the disease-related parameters. The saddle-node bifurcation conditions define a nonlinear relationship between β and μ, whereas the other critical values of the basic reproduction number define a linear relationship. Fixing a
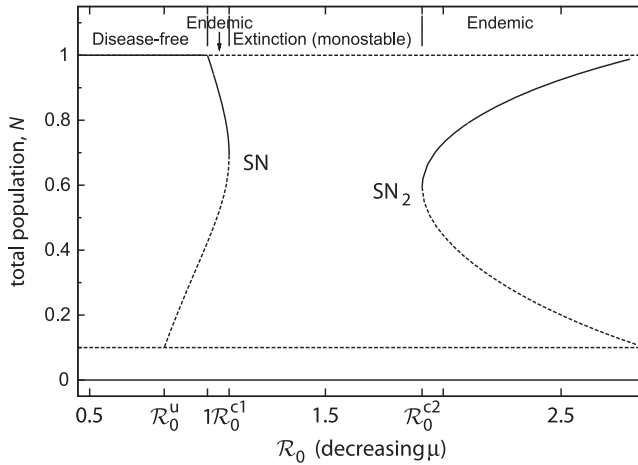
Figure 3.   If the basic reproduction number is varied by changing pathogenicity, the bifurcation diagram reveals a second saddle-node bifurcation (SN$_2$). Parameter values: $\beta = 4, u = 0.1, r = 2, d = 0.2$.
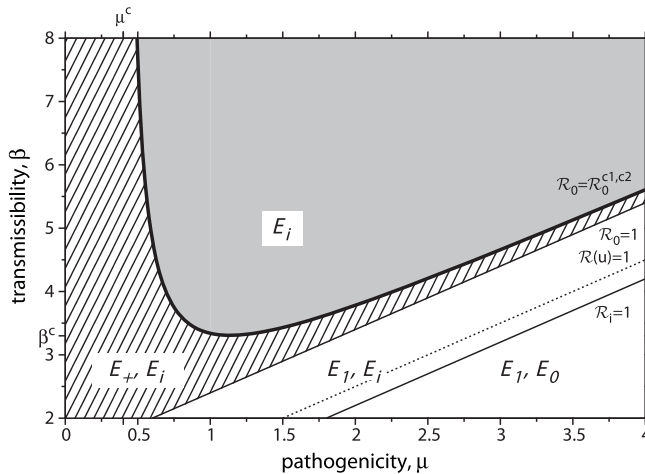

Figure 4.   Domains of model behaviour in the two-parameter plane. Stable equilibria in each domain are indicated. The grey domain is monostable with eventual host extinction. All other domains are bistable. The hatched domain can be endemic, while the remaining white domains can be disease-free. Host extinction is always possible. Bold (thin) lines are saddle-node (transcritical) bifurcation curves. The dotted line marks the emergence of the unstable endemic equilibrium E_-. See the main text for more details. Parameter values: $u = 0.1, r = 2, d = 0.2$.

pathogenicity μ and traversing vertically through Figure 4 by varying β reveals that the saddle-node bifurcation line can be crossed at most once. Note the minimum value of pathogenicity μ^c for saddle-node bifurcations to occur, cf. Equation (6). Similarly, if transmissibility β is fixed and Figure 4 traversed horizontally by changing μ, there is a minimum value of transmissibility β^c as well, cf. Equation (7). The saddle-node bifurcation line can be crossed twice. Mathematically, this can be understood by noting that both nullclines depend on μ, whereas only one nullcline depends on β, cf. Equation (4).

The Allee threshold u (as well as the intrinsic growth rate r) appears in both nullclines, thus also defining two critical tipping points for saddle-node bifurcations. It can be shown that there exists a minimum value of u, for which the 'double-tipping' can occur. That is, the AE needs to be sufficiently severe. However, there are also examples, in which a 'double-tipping' always exists (i.e. including $u \to 0$).

5. Consequences to control interventions and comparison with logistic demographics

The aim of this section is threefold: first, to illustrate the implications of the abrupt host population extinction after a saddle-node bifurcation; second, to show that decreasing $\mathcal{R}_0$ can be disadvantageous; and third, to showcase the difference to populations without AE.
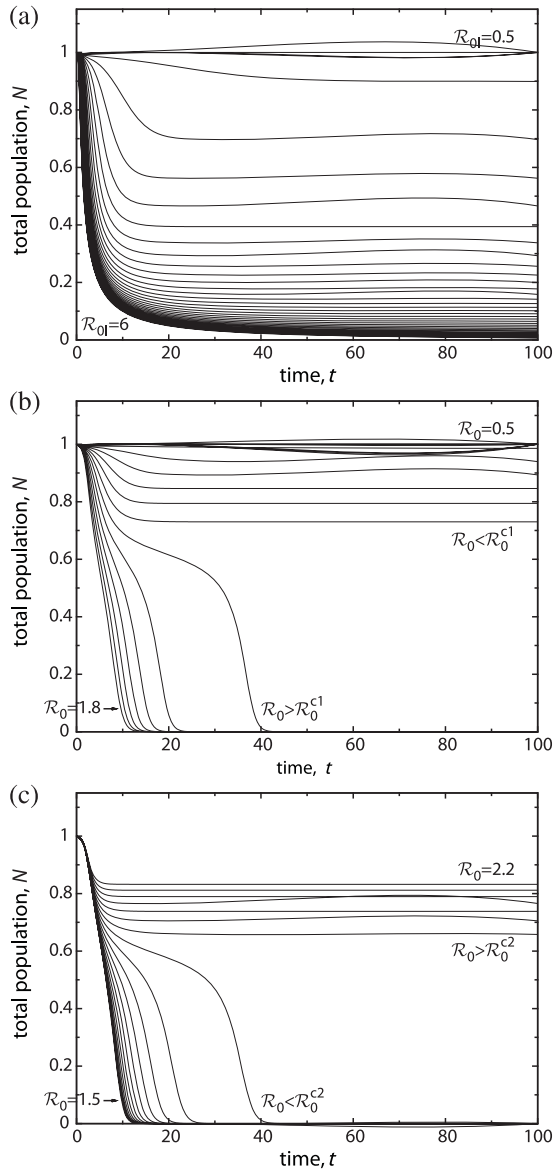
Figure 5. Time evolutions for varied basic reproduction numbers. (a), gradual transition from population persistence to extinction in the logistic model (8), (b) abrupt population collapse in the Allee effect model (2) when increasing $\mathcal{R}_0$ (by increasing β), (c) decreasing $\mathcal{R}_0$ (by varying μ) can lead to a population crash as well. Basic reproduction numbers are varied in equidistant steps. Initial conditions are fixed at $N(t \equiv 0) = 1$, $i(t \equiv 0) = 0.01$. Parameter values: $\mu = 1$, $B = 1$, $M = 0.1$ in the logistic model, $u = 0.1$, $r = 2$, $d = 0.2$ in the Allee effect models with $\mu = 1$ (b) and $\beta = 4$ (c).

System (1) does not exhibit a saddle-node bifurcation if demographics are logistic, e.g.

$$b(N) = B > 0, \tag{8a}$$

$$m(N) = M + (B - M)N, \tag{8b}$$

with $B - M > 0$ being the intrinsic per capita growth rate. There is at most one endemic equilibrium, which emerges if $\mathcal{R}_{0l} = \beta/(\mu + B) > 1$ and disappears again if $\mathcal{R}_{0l}$ is increased to such a value that the disease drives the host extinct, see e.g. [61]. Figure 5a shows various time series for different values of the logistic basic reproduction number. Clearly, the population depression to extinction takes place in a continuous manner.

In a population with strong AE, disease-induced extinction occurs abruptly when increasing $\mathcal{R}_0$, cf. Figure 5b. Host populations with substantial size (for $\mathcal{R}_0 < \mathcal{R}_0^{c1}$) can disappear all of a sudden if the basic reproduction number is increased just a tiny bit ($\mathcal{R}_0 > \mathcal{R}_0^{c1}$).

Figure 5c considers the case when control measures aim at reducing $\mathcal{R}_0$ by increasing pathogenicity. The time plots look similar to the ones in Figure 5b. However, the order of reproduction numbers is reversed. The starting point is an endemic infection with only moderate population depression ($\mathcal{R}_0 = 2.2$). Decreasing $\mathcal{R}_0$ reduces host population size – initially only marginally, but once the tipping point $\mathcal{R}_0^{c2}$ induced by the second saddle-node bifurcation is passed, the population quickly goes extinct. Interventions aimed at altering the basic reproduction number, therefore, need to be planned carefully. Merely reducing $\mathcal{R}_0$ is not always beneficial.

6. Differences to backward bifurcations

In recent years, backward bifurcations (BBs) in epidemic models have received considerable attention in the literature, see e.g. [6,17,21,29,40,42,46,52,53,59]. It is interesting to contrast the bifurcation behaviour of the AE model presented here with a BB, see Figure 6a and 6b, respectively. The discussion in this section is restricted to the case in which $\mathcal{R}_0^{c2}$ is unfeasible, i.e. there is only a single saddle-node bifurcation in the AE model as it occurs with varying transmissibility. Note that the diagrams show the infectives I on the vertical axis. Both scenarios have multiple endemic equilibria and can be bistable. However, the following differences demonstrate that the bifurcation behaviour is fundamentally different.

(1) The disease can be endemic in the AE model only for $\mathcal{R}_0 > 1$, whereas it can be endemic in a BB model even if $\mathcal{R}_0 < 1$. Moreover, in a BB model, the disease remains endemic in the host for all $\mathcal{R}_0 > 1$. In the AE model, in contrast, the disease (as well as the host) disappears if $\mathcal{R}_0 > \mathcal{R}_0^{c1}$.
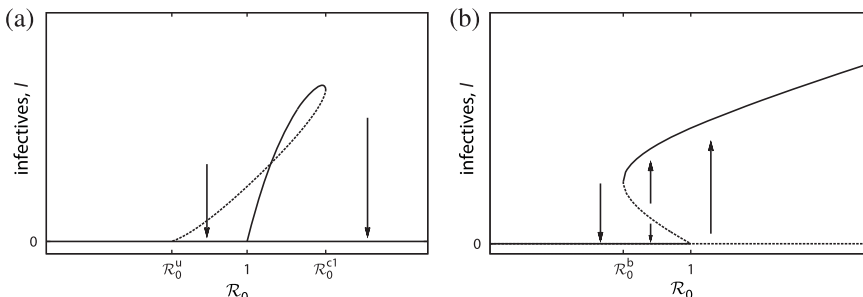

Figure 6. (a) Bifurcation behaviour in the Allee effect model when varying $\mathcal{R}_0$ by increasing β, (b) Stylized backward bifurcation. Note that the total host population goes extinct in (a) if the basic reproduction number is larger than $\mathcal{R}_0^{c1}$. Parameter values in (a): $\mu = 1$, $u = 0.1$, $r = 2$, $d = 0.2$, varying β.

(2) The saddle-node bifurcation occurs in the AE model beyond the disease invasion threshold at $\mathcal{R}_0^{c1} > 1$, whereas it takes place in a BB model before it at $\mathcal{R}_0^b < 1$.

(3) The direction of the saddle-node bifurcation is mirror-inverted. That is, increasing $\mathcal{R}_0$ leads to the disappearance of the endemic equilibria in the AE model and to their emergence in a BB model. The former is the basis for the abrupt population collapse. The latter can lead to an abrupt explosion in infectives if $\mathcal{R}_0$ is increased beyond unity as well as to a sudden eradication of disease if $\mathcal{R}_0$ is reduced below $\mathcal{R}_0^b$.

(4) The backward bifurcation induces a critical behaviour primarily for the infectives I, i.e. whether infection quickly establishes or suddenly disappears. The criticality in the AE model, in contrast, relates mainly to the total host population N, cf. the bifurcation diagram in Figure 2. If $\mathcal{R}_0 > \mathcal{R}_0^{c1}$, the entire population and not only the infectives go extinct. Furthermore, the unstable equilibrium value of the infectives is of interest for the increased likelihood of extinction of smaller populations. Obviously, the infectives always disappear for $\mathcal{R}_0 < 1$ (unless the initial condition is exactly on the unstable equilibrium). The initial number of infectives is actually important for whether the host persists (either at the carrying capacity or endemic equilibrium) or goes extinct.

(5) The bistability and the two endemic equilibria in the AE model are due to the strong AE. Interestingly, infection can render the system monostable. The bistability and the endemic equilibria in a BB model are induced by disease characteristics, see [49] for a review.

7. Discussion and conclusions

Conventional epidemic models (without demographic AE) predict that parasites can eradicate their host if transmission is frequency-dependent. The transition from population persistence to extinction takes place gradually when control parameters vary continuously. There is increasing evidence, however, for positive density dependence leading to strong AEs in host demographics [12]. The mathematical model considered here suggests that extinction occurs abruptly due to a fold catastrophe (saddle-node bifurcation). Marginal changes in model parameters can thus lead to dramatic consequences, namely the spontaneous collapse of the host to deletion.

Extinction research increasingly focuses on synergistic interactions between different processes. In combination, single extinction drivers such as habitat destruction or overexploitation pose a much larger threat to endangered species than in isolation, as they may reinforce themselves in declining populations due to amplifying feedbacks [9]. Parasitism and AEs are two such examples that have long been recognized to increase the risk of extirpations. Their joint interplay acts as an extreme accelerator in the transition towards extinction. In particular, they define a tipping point at which the population collapses all of a sudden.

The subtlety of the tipping point is that the host can be extremely abundant just before it. Figure 5b illustrates that the population crashes from a level of over 75 % of the carrying capacity to nil. Establishing predictors of such spontaneous collapse appears to be difficult, if not impossible. This would be important for identifying potential extinction risks and guiding management actions. Close before the tipping point ($\mathcal{R}_0 = \mathcal{R}_0^{c1}$), the extirpation is accompanied by prolonged transient dynamics [32], cf. Figure 5b and 5c. The population seems to be stable over a considerable time horizon even though it is ultimately commited to its deterministic extinction debt. In nature, however, the transients are likely to be superimposed by perturbations and stochastic effects. These factors could speed up or slow down the extirpation. Recognizing the transient approach to deletion is difficult, and even if it can be identified at all, it might be too late for interventions. Moreover, the transients occur only after the tipping point and not beforehand (from the point of view starting with persistence). Therefore, the imminent disaster is almost impossible to anticipate in practice.

Mathematically, the tipping point is associated with a saddle-node bifurcation. When the two non-trivial equilibria E_+ and E_- annihilate each other, there is no endemic attractor left and extinction is inevitable. E_+ emerges from the disease-free carrying capacity state if $\mathcal{R}_0 > 1$. Obviously, E_+ has a larger population size than E_-, which bifurcates from the Allee threshold state on the disease-free boundary into the interior of the phase plane if $\mathcal{R}_0(u) > 1 \Leftrightarrow \mathcal{R}_0 > \mathcal{R}_0^u$. The emergence of the unstable equilibrium E_- is essential for the saddle-node bifurcation to occur. As a saddle point, E_- organizes the extinction basin in the phase plane (cf. Figure 1). In host populations without critical depensation, it is clearly not possible that an endemic equilibrium arises from the Allee threshold. Abrupt crashes to extinction thus appear to be characteristic for strong AEs.

Biologically, the spontaneous population collapse is the consequence of two mechanisms. The first one is the regulatory potential of parasites. This leads to a depression of the host population size N_+^* at endemic equilibrium. The second mechanism is that additional mortality due to the disease increases the likelihood of extinction. That is, the effective extinction threshold becomes larger. The infection, therefore, 'attacks' the host from two ends of the population size spectrum, thus narrowing down the range in which endemic persistence is possible. Extinction takes place if the range of viable population sizes ceases to exist. Metaphorically speaking, the endemic population equilibrium is finally absorbed by the expanding extinction basin.

Sudden host population deletion due to fold catastrophes can also be observed in other epidemic models, in which the strong AE is incorporated differently. Numerical experiments show that the frequency-dependent model by Deredec and Courchamp [15] exhibits a saddle-node bifurcation as well (the authors did not report the possibility of multiple endemic equilibria). Furthermore, if one changes the density-dependent model by Thieme *et al.* [57] to obey a frequency-dependent incidence, two endemic equilibria and their disappearance in a saddle-node bifurcation too become possible.

The abrupt extinction appears to be rather general for AE populations with frequency-dependent transmission. This incidence pattern is typically suggested for sexually transmitted diseases [58] and infections in populations with territorial or social behaviour [1]. In density-dependent transmission models, abrupt host extinction seems to be less general. In [57] multiple equilibria are not possible – this may be due to the assumptions that infectives do not reproduce at all or that mortality is density-independent. The model in [36] – the same as in Equations (1) and (2) but with density-dependent incidence – allows for fold catastrophes, but in a more restricted parameter range (e.g. there exists a maximum pathogenicity below which saddle-node bifurcations are possible). Furthermore, the situation appears to be more complex as three non-trivial equilibria and tristability are possible. The difference between frequency-dependent and density-dependent transmission is that the former can maintain infections in smaller population sizes, whereas the latter cannot promote disease spread if population size is too small [32]. Therefore, the unstable endemic equilibrium emanating from the disease-free Allee threshold state is not generally possible for density-dependent incidences.

The existence of a second saddle-node bifurcation when varying pathogenicity rather than transmissibility is the consequence of the simple fact that highly pathogenic parasites cause their own extinction but not that of their host. The two saddle-node bifurcations, if they exist, are separate from each other (cf. Figure 3). They are not related, e.g. by sharing the same saddle point as in scenarios with three endemic equilibria [26,36,44]. This discontinuity has profound implications for control measures aimed at impacting the reproduction number. Increasing $\mathcal{R}_0$ can be beneficial for the host if it is changed beyond $\mathcal{R}_0^{c2}$, thus facilitating endemic persistence rather extinction. Decreasing $\mathcal{R}_0$ can be disadvantageous if it is reduced below $\mathcal{R}_0^{c2}$, because it can cause host eradication (as illustrated in Figure 5c).

The separate branches of endemic equilibria also complicate a model analysis that is based on numerical continuation of stationary states and bifurcations. Computer software such as

MatCont [16], AUTO [20] or XPPAUT [22] might not detect the second saddle-node bifurcation. The re-emerging non-trivial equilibria can, therefore, remain hidden and be missed if the analysis is restricted to numerical tools.

Emerging and introduced infectious diseases have been implicated in the extinction of a large number of species (cf. references in Section 1). If infected populations are additionally subject to a strong AE, they may be at much higher risk than previously thought. Even if population size is considerably large and close to carrying capacity, tiny parameter variations can trigger an unpredictable and drastic population crash towards eradication. The underlying mechanisms – population depression and critical depensation due to a strong AE – are fairly basic and could occur simultaneously in many animal and plant populations. The insights gained from this study are of particular interest for wildlife management and conservation biology. They also shed new light on the effectiveness of pathogens as potential biological control agents and on the difficulties associated with species reintroductions. Moreover, the dramatic changes occurring for large reproduction numbers call for shifting the focus of disease control away from the threshold $\mathcal{R}_0 = 1$. The tipping points $\mathcal{R}_0^{c1,c2} > 1$ with their catastrophic consequences appear to be much more significant. Small parameter changes around $\mathcal{R}_0^{c1,c2}$ may preserve the population in its existence, while the aim of reducing $\mathcal{R}_0$ below one might be unrealistic and gain relatively little additional benefit.

Acknowledgements

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Appendix A. Existence conditions for two nontrivial stationary states

There are two endemic equilibria if $\mathcal{R}_0 > 1$ and $\Phi(N_{\max}) > 0$, with $N_{\max} = (\mu + \beta r[1 + u])/(2\beta r)$. The latter inequality can be solved explicitly in terms of model parameters. Solving for the transmissibility, one obtains $\beta < \beta_1$ or $\beta > \beta_2$, where

$$\beta_{1,2} = \frac{\mu}{A}(B \pm \sqrt{B^2 + rA}),$$

$A := 4\mu - r(1 - u)^2$ and $B := 1 + u + 2(d + ru + \mu) > 0$. If $\mu > \mu^c$, with μ^c being defined as in Equation (6), then $A > 0$ and consequently $\beta_1 > 0$, while $\beta_2 < 0$. Conversely, if $\mu > \mu^c$, then $A < 0$ and both $\beta_{1,2}$ are either negative or imaginary.

Solving $\Phi(N_{\max}) > 0$ for the pathogenicity, one obtains $\mu < \mu_1$ or $\mu > \mu_2$, where

$$\mu_{1,2} = \frac{r\beta}{1 + 4r\beta}(C \mp \sqrt{C^2 - (1 + 4r\beta)(1 - u)})$$

and $C := 2(\beta - d - ru) - 1 - u$. It can be shown that both $\mu_{1,2}$ are real and positive if $\beta > \beta^c$, with β^c being defined as in Equation (7). Otherwise, $\mu_{1,2}$ are either negative or imaginary.