Short and long term suppression of host populations by novel pathogens

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Abstract

Current theory for the regulation of host populations by pathogens suggests that a high level of suppression during the initial epidemic phase will be followed by a population rebound with decreased virulence due to pathogen and host evolution, and the extent of host suppression increases with increasing pathogen transmissibility (R_0) and virulence. Using simple epidemiological models, we explore the effect of three factors on short- and long-term suppression: the strength of density-dependent population regulation (homeostasis); maternal antibodies, and age-dependent mortality. Rapid homeostasis can mitigate long-term population suppression, and surprisingly weak homeostasis can even result in a greater population suppression during the endemic phase compared to the initial epidemic. Maternal antibodies can significantly reduce suppression of the host population if they attenuate rather than block infections. A similar result obtains if the severity of disease is lower in the young than in adults. In both cases, a higher R_0 can result in lower suppression, and the average virulence can decline over time without any (genetic) evolution. Our results suggest the need for a nuanced view of long-term suppression by a new pathogen, with the outcome sensitive to many details even in the absence of evolution.

Introduction

Viruses and other pathogens can cause massive mortality when first introduced into naive animal or human populations [2, 21]. The introduction may be a deliberate attempt to suppress the host population as with myxomavirus and caliciviruses to control Australian rabbits [8, 11]. Or a pathogen may naturally jump from its reservoir host into a new host, as with the 1918 human influenza virus in humans and distemper viruses in lions and in seals [7, 27, 29, 30]. Finally, a pathogen can be introduced via contact with previously isolated populations of the same species, as happened to the Aztecs following contact with Spanish conquistadors [9, 22]. There has considerable attention given to the initial epidemic or pandemic spread of the pathogen and its effect on the host population as illustrated in the examples mentioned above.

Other studies have considered the long-term impact of a pathogen on its host population. These ecologically motivated studies considered the role of pathogens in regulating host populations. In a classic study, Anderson and May [2] showed that, since the transmission (R_0) of the pathogen is proportional to the density of hosts, suppression of the host population is greatest when the density of the host is high.

Most studies that consider the long term impact of pathogens focus on evolution. Evolutionary changes, whether in the pathogen or host, are difficult to predict, but the general view (supported by the myxoma virus released in Australian rabbits) is that an initially highly lethal pathogen will evolve to be less virulent and the host population will evolve resistance.

There are, however, non-evolutionary mechanisms that can change the long term impact of the pathogen from its initial impact, and these have received relatively less attention than pathogen evolution. One factor is a reduction in the average age of infection as the pathogen becomes endemic; the virulence of infections is often lower for youth than for adults [18, 20, 23]. Long term suppression may also be considerably reduced if young are protected by maternal antibodies [12, 31]. Both of these factors can reduce the severity of infections as the disease transitions to endemicity, and for pathogens used to control animal populations, can allow the host population to rebound.

We use mathematical models to explore the impact of a pathogen in suppressing the host population and how this changes between the early, epidemic stage and the late, endemic stage. We focus on non-evolutionary mechanisms and specifically consider the consequences of viral transmissibility (R_0), maternal antibodies, and age-dependence of viral virulence. We show that the extent of density-dependent regulation in the host population, which we term homeostasis, has an unexpectedly large effect on the ability of pathogens to suppress host populations in the long-term. Our findings are relevant to the use of pathogens to control pests as well as to conservation efforts to mitigate natural epidemics. A major goal is to keep the models simple enough to allow the analysis of model structure and the effect of specific parameters on outcomes. As such, the study is not an attempt to match any particular application.

Models and Results

We begin with a simple compartmental model that incorporates susceptible, infected and recovered populations. In the second section we expand the model to include individuals protected by maternally transferred antibodies. In the final section we consider a scenario where the severity of infections depends on age.

Basic model: no maternal antibodies or age dependence

Our most basic model with **S**usceptible, **I**nfected and **R**ecovered (also immune) individuals is given by equation set (1), with notation and parameters in Table 1:

$$\begin{aligned} \text{Susceptible} \qquad \dot{S} &= -\beta SI - d(N)S + b(N)N \\ \text{Infected} \qquad \dot{I} &= +\beta SI - \nu I - d(N)I \\ \text{Recovered} \qquad \dot{R} &= +\nu I(1-f) - d(N)R \end{aligned} \tag{1}$$

where N is total population size. We assume that, in the absence of the pathogen, the population size is maintained by density dependence of the birth and death rates which gives rise to logistic growth. We let the birth rate decrease linearly with density $[b(N) = b_0 - k_b N]$ and the death rate increase linearly with density $[d(N) = d_0 + k_d N]$. Through appropriate choice of parameter values, the carrying capacity (equilibrium population density) in the absence of pathogen is here always scaled to 1 in the absence of the pathogen. The severity of infections is described by the case mortality (f). We choose case mortality rather than a virulence rate [e.g., 15], as it gives a more intuitive description of virulence than a rate of virulence, and it allows exploring the effect of independently changing transmission and disease severity without affecting the lifespan of infecteds. The basic reproductive number (R_0) equals $\beta/(d_0 + k_d + \nu) \approx \beta/\nu$, an approximation that relies on the duration of infection ($1/\nu$) being much shorter compared with the lifespan of the host ($1/(d + k_d)$). The simulations that vary R_0 do so by changing transmission, β.

Symbol	Description	Value
S	Susceptible, born of naive mothers	
I	Infected S	densities, $\leq$ 1
R	Recovered, immune	
N	Total population ($S + I + R$)	
ν	loss of infection rate	50
β	transmission rate constants	for R_0 ($1.5 < R_0 < 10$)
f	case mortality	$0 < f < 1$ per infection
Rapid homeostasis		
b_0, d_0	intrinsic birth and death rates	3.5, 0.2 /year
k_b, k_d	parameters for logistic growth	2.5, 0.8
Slow homeostasis		
b_0, d_0	intrinsic birth and death rates	1.5, 0.8 /year
k_b, k_d	parameters for logistic growth	0.5, 0.2

Table 1: Notation for variables and parameters in eqns (1). We analyze two different sets of values for the four birth-death parameters. Birth and death parameter values are chosen so that the equilibrium population size in the absence of the pathogen is 1, and the parameters for rapid homeostasis are chosen to represent a short-lived, high-fecundity species such as rabbits (see Supplementary file S1 for details); values for slow homeostasis are chosen to contrast with those for rapid homeostasis.

We use parameter values to represent a small mammal, such as a rabbit, which enables us to relate our results to the well documented example of Australian rabbits. We consider two scenario's

for the regulation of the host population in the absence of the pathogen. In the first scenario, the population exhibits rapid regrowth when culled, which we refer to as *rapid homeostasis*. We simulate this by choosing high values for k_b and k_d, the parameters that describe the density dependence of birth and death rates. We explore the effect of *slow homeostasis* by assigning smaller k_b and k_d values so that the birth and death rates exhibit a smaller dependence on population size. The pathogen-free equilibrium is unaffected, however.

We explore the impact of a pathogen on initial and long-term suppression of the host population, specifically comparing effects of rapid and slow homeostasis (Fig 1, top and bottom, respectively). The first column illustrates the dynamics of the host population for specific cases. The second and third columns show the host population size at the initial minima and at steady-state, respectively, which indicate the extent to which the pathogen suppresses the population in the short term and long-term respectively.

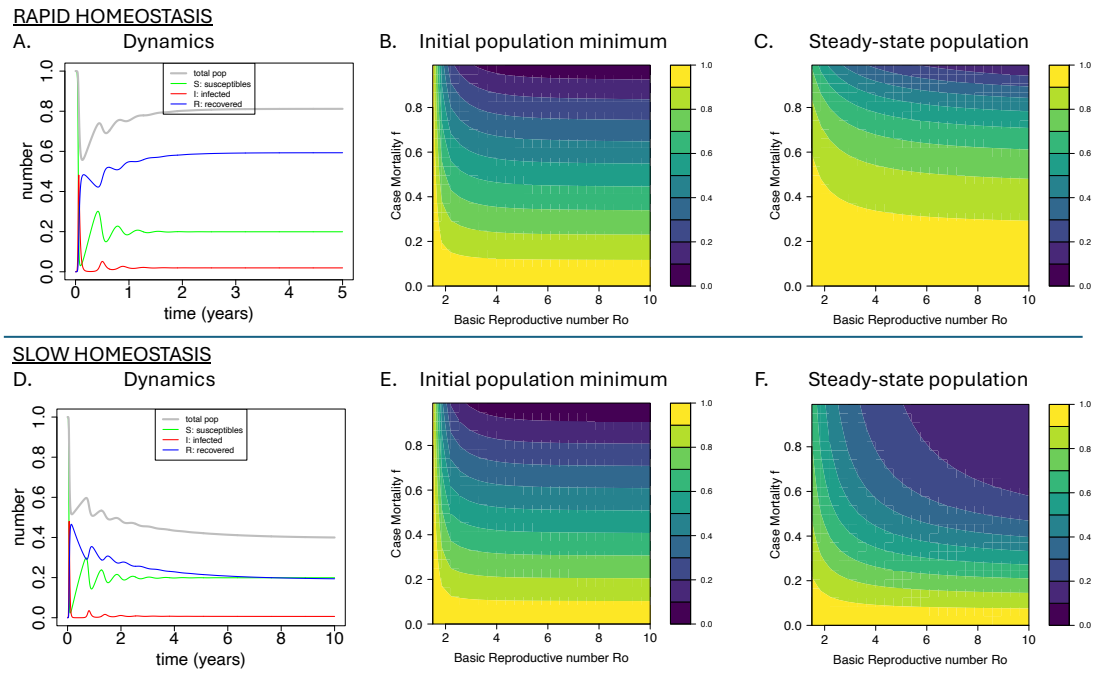

Figure 1: Population suppression following introduction of a pathogen. The top row shows the outcome when the homeostatic regulation of the host population is rapid, the bottom row for slow homeostasis. In the first column (panels A and D) shows the dynamics following introduction of a pathogen with an $R_0 = 5$ and case mortality $f = 0.5$. We see that the initial suppression is similar regardless of the strength of homeostasis, but the population rebounds when homeostasis is rapid (panel A), whereas it declines further when homeostasis is slow (panel D). Panels B-C and E-F show how the initial and steady-state population suppression depend on R_0 and f. We see similar initial population suppression for both rapid and slow homeostasis, but very different outcomes when steady-state is reached. When there is rapid homeostasis the population invariably exhibits a rebound, and the steady-state population (panel C) is higher that at the initial decline (panel B). In contrast when homeostasis is slow, the population often declines further as steady-state is reached (panels E, F). Parameter values are given in Table 1.

Short-term suppression of the host population

Assuming a single set of parameter values ($R_0 = 5$, $f = 0.5$), short-term dynamics of the pathogen and host population are illustrated in Fig 1 (A, D). Following introduction, the pathogen spreads rapidly and kills a large fraction of the host population. The number of susceptible hosts is driven to a low level, whence the pathogen declines, and the total host population reaches a minimum. Comparison of the minima in panels A and D as well as the contours in panels B and E reveals that the initial population suppression is relatively independent of homeostasis; the magnitude of suppression increases with both pathogen transmissibility (R_0) case mortality (f). The initial population suppression is largely independent of homeostasis because it occurs relatively rapidly after introduction of the pathogen on a timescale that is fast compared with the birth and death rates of hosts. This allows estimation of the initial minimum size of host population from the final size formula for an epidemic [24]. Neglecting birth and death and assuming zero case mortality, the final number of recovereds R_f is given by the solution of $R_f = 1-\exp[-R_0(1-R_f)]$. Since a fraction $(1-f)$ of these survive and $(1-R_f)$ susceptibles never get infected, the minimum population of hosts is approximately $(1-R_f) + R_f(1-f)$. For $R_0 > 5$, nearly all the population gets infected ($R_f \approx 1$), and the total density of individuals at the minimum is suitably approximated by $(1-f)$. This expectation is supported by comparisons between the prediction using the final size equation and simulations of eqn (1) (Fig. 6 of Supplement S1). The analytical and simulation results are similar except at very low values of R_0 – when the spread of the infection is so slow that births offset the host losses from infections before the epidemic phase has ended (Supplementary Fig. 6).

Suppression of the host population at steady-state

Following initial population suppression, the host population exhibits oscillations and reaches a steady-state. If the host population exhibits rapid homeostatic regulation, the numbers can rebound to a steady-state level that is significantly higher than the initial suppression – as is seen in the single trial of Fig 1A and by noting that the populations at steady state in 1C are higher than the initial minima in 1B. In contrast, if the host population experiences slow homeostasis, the long term population can decline beyond its initial minimum – as seen for a single trial in Fig 1D and by comparing panels 1E and 1F. A decline beyond the initial minimum does not apply throughout the parameter space, however, and a comparison of panels E and F shows specific regions in which the final population size is larger or smaller than the initial minimum. Relative magnitudes of the initial and long-term population suppression are shown in Supplement S1 Fig 7, where it is seen that, when homeostasis is slow, long-term suppression can exceed both the initial suppression as well as the case mortality. As noted above, case mortality puts an upper bound on early suppression. Suppression exceeding case mortality is not observed when homeostasis is rapid.

Why does homeostasis have such a profound effect on the population of hosts at the steady state? With rapid homeostasis, following the initial suppression of the host population by the pathogen, the birth rate greatly exceeds the death rate, and this is responsible for the population rebound. The more interesting case is slow homeostasis, because the population continues an overall decline after the initial invasion. This case is most easily understood by considering the extreme case of no homeostasis (i.e., $k_b = k_d = 0$, which requires $b_0 = d_0 > 0$). After pathogen invasion and the population has reached its early minimum, there will be no subsequent upward population growth because $b_0 = d_0$. In the short term, pathogen abundance is held in check from a combination of low host density and recovered individuals that cannot be reinfected. However, the ongoing birth and death will gradually replace recovered individuals with susceptible individuals. As susceptibles

increase, despite no population growth, further outbreaks can occur, and the population declines further. Eventually, the population reaches a low point from which the pathogen cannot expand. The process is shown in Supplement S1 Fig 8, for which no homeostasis exists. In less extreme cases, slow homeostasis merely fails to enable enough upward growth to offset the continued pathogen expansion as recovered individuals are replaced by susceptibles.

No virulence changes. In this basic model, the changing level of suppression from the short term to the long term is a population phenomenon: different pathogen parameter values lead to different population sizes, and the population-wide suppression changes over time. Although the average case mortality of infections ('virulence') is varied between trials, it does not change over time within trial. Thus, if the case mortality of an infection is 0.5, it remains at 0.5 during viral invasion and at endemicity. This constancy of average virulence of a single pathogen will not operate in the models considered below.

Maternal antibodies can substantially reduce long term suppression

Maternal antibodies, transferred from the mother to offspring, are known to protect the young for a few months after birth [5, 10, 31]. Maternal antibodies have been shown to protect young rabbits from myxoma virus infection [10, 19], and some consequences of this protection to population control have been modeled [12, 13, 14]. Maternal antibodies might provide protection in two ways: by **blocking** – reducing the susceptibility of individuals to becoming infected – or by **attenuation** – reducing the severity of infections while enabling long-lived immunity [17, 19, 26].

Intuition suggests that if maternal antibodies attenuate the infection, considerable population recovery might ensue from an otherwise highly lethal pathogen. This would arise as a result of the few naive individuals surviving an infection having offspring with maternal antibodies. If these offspring are infected during the period of protection, the attenuation improves their survival. Then, having survived infection they now themselves have high levels of antibodies. Consequently they endow their progeny with maternal antibodies that provide the same benefit. If levels of infection are high, this benefit can be propagated as if inherited epigenetically – a type of Lamarckian inheritance. A protected class of the population may thus emerge and expand disproportionately because their survival is much higher than of naive individuals.

The consequences of maternal antibodies that block infection are not so clear: offspring are temporarily blocked from infection but may be infected later in life – suffering the typical case mortality (by assumption here). If blocking is lifelong, the recovered individual is protected for life, but its progeny will not be protected. So the effect of blocking by maternal antibodies may be to reduce the number of susceptible individuals in the population and thereby lower the incidence of the pathogen. But blocking is not inherited because a blocked individual cannot be infected and thus does not develop any phenotype to be passed on.

We model the effect of maternal antibodies by introducing two new classes of individuals into our previous equations. S_M are individuals born with maternal antibodies and thus protected, and I_M are individuals who became infected while in the S_M category. The full set of equations for maternal antibodies is thus:

$$
\begin{aligned}
\text{Susceptible (S)} \qquad \dot{S} &= -\beta S(I+I_M) + \gamma S_M - (d_0 + k_d N)S + (b_0 - k_b N)(N-R) \\
\dot{S}_M &= -\beta_M S_M(I+I_M) - \gamma S_M - (d_0 + k_d N)S_M + (b_0 - k_b N)R \\
\text{Infected (I)} \qquad \dot{I} &= +\beta S(I+I_M) - \nu I - (d_0 + k_d N)I \\
\dot{I}_M &= +\beta_M S_M(I+I_M) - \nu I_M - (d_0 + k_d N)I_M \\
\text{Recovered (R)} \qquad \dot{R} &= +(1-f)\nu I + (1-f_M)\nu I_M - (d_0 + k_d N)R
\end{aligned} \tag{2}
$$

Parameters have the same meaning as above, with one new parameter, γ for the decay of protection conferred by maternal antibodies (Table 2). β and f are subscripted with M to accrue to hosts protected by maternal antibodies.

Symbol	Description	Value
S	Susceptible with no maternal antibodies	
S_M	Susceptible with maternal antibodies	
I	Infected from S	
I_M	Infected from S_M	
R	Recovered and immune	
N	Total population	
γ	maternal antibody decay rate	4 per year

Table 2: Variables used in eqn set (2) along with one new parameter introduced in this model. Other parameters are given in Table 1.

We now apply the model to consider how maternal antibodies affect population suppression, focusing on two cases for maternal antibody protection. In the first case, maternal antibodies block infections; in the second, they attenuate infections – reduce lethality but allow the generation of long-term immunity. We simulate blocking by setting $\beta_M = 0$, attenuation by setting $f_M = 0$ and $\beta_M = \beta$. The parameter for waning of protection from maternal antibody is set as $\gamma = 4$, which corresponds to protection for 3 months. Following our findings for the basic model (1), each analysis of maternal antibodies is run in parallel for rapid and for slow homeostasis.

Short-term suppression is unaffected by maternal antibodies

As was true for the previous section (basic model (1)), the initial level of population suppression with maternal antibody effects is largely independent of homeostasis and of maternal antibodies. Maternal antibodies have minimal effect on initial suppression because the initial suppression occurs prior to the appearance of individuals with maternal antibodies.

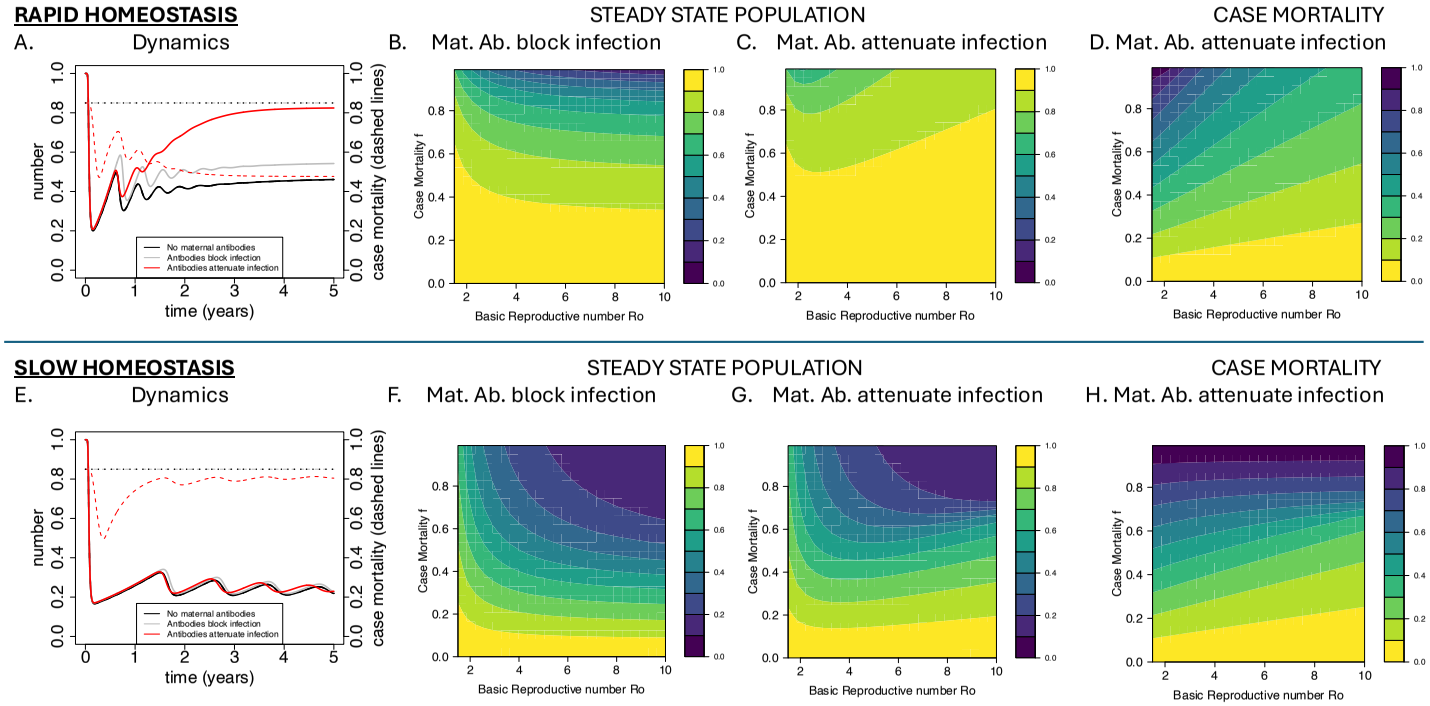

Figure 2: Effect of maternal antibodies on host population suppression and virulence. The top row is for rapid, the bottom row for slow homeostasis. Panels A and E in the first column contrast the dynamics of the total population size when maternal antibodies attenuate infection (red), prevent infection (gray) or have no effect (black) for a pathogen with $R_0 = 5$ and case mortality $f = 0.85$. All other panels are for steady states. As above, population suppression is greater for slow than for rapid homeostasis (B versus F, C versus G), also greater for blocking than for attenuating the infection (B versus C, F versus G), although this difference is much greater for rapid than for slow homeostasis. The rightmost panels (D, H) show the reduction in average case mortality at steady state when maternal antibodies attenuate infection; the upward-sloping contours indicate that average case mortality declines with R_0. Parameter values are given in Tables 1 and 2, and $R_0 = 5$ and $f = 0.85$ for panels A and D. The rate of waning of maternal antibody protection $\gamma = 4$ which corresponds to protection for 1/4 year.

Long-term recovery can be enabled by maternal antibodies but depends on biology

Fig 2 presents panels reflecting several aspects of our analysis, always contrasting rapid and slow homeostasis (top and bottom rows, respectively), with the columns indicating in order: (i) a representative time course for the dynamics of infection; (ii) and (iii) the steady-state population when maternal antibodies block or attenuate infection respectively, and (iv) the average virulence of the infection when maternal antibodies attenuate infection. Except for the short-term dynamics, these outcomes are evaluated for different combinations of R_0 and case mortality f at steady state.

To help develop a sense of the processes at work and to check the intuitive conjectures offered above, the leftmost panels in Fig. 2 illustrate 5-year population dynamics following introduction of a highly lethal pathogen (case mortality $f = 0.85$) with an $R_0 = 5$. Panel (A) is for rapid homeostasis, (E) for slow homeostasis. Each panel compares (i) no maternal antibodies (black), (ii) maternal antibodies attenuate (red), and (iii) maternal antibodies block the infection (gray). Maternal antibodies attenuating infections results in a striking recovery of the host population, but this happens only when homeostasis is rapid (red curve in panel A). When homeostasis is slow or maternal antibodies block infection, there is no additional rebound compared to a scenario with no maternal antibodies.

Other panels in Fig 2 address the long term effects of maternal antibodies for a broad range of R_0 and case mortality values. Maternal antibodies blocking infection results in only a small increase in the host population at steady state (compare Fig 2 B, F with Fig 1 C, D). In contrast, maternal antibodies attenuating the infection can result in a large increase in the population at steady-state, particularly when homoeostatic regulation of the host population is rapid. The benefit of attenuation and relative lack thereof for blocking is compatible with the intuition offered above.

Any benefit of maternal antibody attenuation can be realized only when offspring are infected prior to losing maternal antibodies. Thus we expect – and observe – the paradoxical effect that population recovery should benefit from high R_0 because the higher transmission leads to a higher incidence of infection and thus earlier infection. As was true of the basic model, rapid homeostasis promotes far greater population recovery than does slow homeostasis – the populations in panel C are larger than in panel G. The same reasoning likely applies here as in the basic model: the steady state population size benefits from more net births.

Antibody duration

It was noted above that, with maternal antibody attenuation of infections, higher R_0 benefits population recovery. This was explained on the grounds that higher R_0 results in infections occurring at earlier ages – prior to the waning of protection by maternal antibodies. Similar intuition suggests that any effect of maternal antibodies on host populations will depend on antibody duration – $1/\gamma$ in our model. If antibodies wane too fast relative to the average age of infection, then few or no individuals will be protected when they are eventually infected. In turn, average age of infection will depend on R_0 and on the birth and death rates at steady-state. Thus, the strong population recovery observed for rapid homeostasis under attenuation as well as the broad lack of recovery under slow homeostasis may change, depending on antibody biology.

Consequences of changing the duration of protection by maternal antibodies are shown in Fig. 3. As expected, increasing antibody duration increases long term population size under attenuation for both rapid and slow homeostasis (panels B, D). A similar but milder trend is also seen for blocking with rapid homeostasis (A), but there is only a very slight effect of antibody duration evident for blocking with slow homeostasis (C). As before, host population suppression is greater

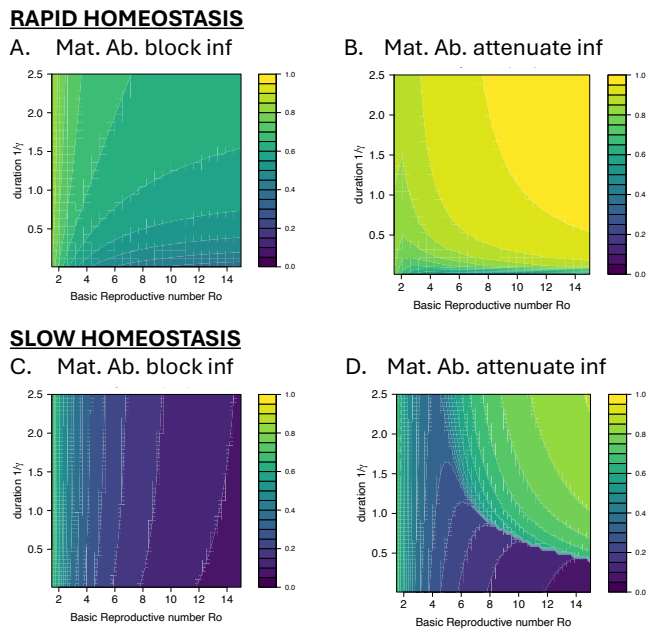

Figure 3: Effect of maternal antibody duration on the suppression of the host population at steady state. Plots vary R_0 (X-axis) and the duration of protection by maternal antibody ($1/\gamma$, Y-axis). As above, suppression is typically greater for slow homeostasis than for rapid homeostasis. The effect of antibody duration is moderately weak except in panel D and at very short durations. The top row is for rapid homeostasis, bottom is for slow homeostasis. Case mortality (f) is 0.85, and the intrinsic lifespan $1/d_0 = 5$ years.

for slow homeostasis than for rapid homeostasis.

Fig. 3 provides some interesting insights to blocking. Whereas any effect of blocking was not necessarily evident in Fig. 2 (because it required a comparison of course-grained contours between Figs. 1 and 2), panels A and C of Fig. 3 reveal subtle, mild effects of antibody duration with blocking – which implies that maternal antibody blocking is affecting long term population size, if only slightly. Furthermore, the effect of R_0 is in the opposite direction as with attenuation – higher R_0 leads to increased suppression. As case mortality is the same for all infections under blocking, higher R_0 must have this directional effect because it leads to more infections.

Perhaps the most interesting result is the sharp boundary observed in panel D: with slow homeostasis and maternal antibody attenuation, population suppression is minimal until reaching a threshold combination of R_0 and antibody duration, beyond which population size rebounds greatly. Although the overall trend is the same for rapid homeostasis (panel B), it is the sharp boundary in panel D that is of interest. A fine-grained analysis of steady state values reveals an extremely sharp transition in all state variables, one that is sensitive to initial conditions. Whether such thresholds would operate under realistic conditions is debatable, but their presence in the idealized context of the model may portend difficulties with prediction in the field, with slight variations in R_0 or antibody duration shifting the outcome from success to failure of population control. Further analysis is obviously required to obtain a deep understanding of the processes at work here.

Non-genetic reductions in virulence with attenuation

We first consider maternal antibodies which attenuate infections. Since mortality of infections in individuals with maternal antibodies is low (zero in our simulations), any increase in the incidence of these infections will result in a decrease in the population-wide case mortality – a reduction in the virulence of the pathogen. When the pathogen first invades, the average case mortality per infection (virulence) is f – because maternal antibodies are initially absent. As the prevalence of individuals with maternal antibodies increases there is a reduction in the *average* lethality of infections. Fig. 2 panels D and H show that the average case mortality at steady state decreases with increases in R_0. This is because as R_0 increases there is a decrease in the age of infection and thus a higher probability that individuals with maternal antibodies (S_M) get attenuated infections prior to waning of their maternal antibodies. The patterns are somewhat different between rapid and slow homeostasis – extreme reductions at high f for rapid homeostasis, but for slow homeostasis, virtually no reductions at high f despite obvious reductions at low f.

The dashed red curves in panels 2A and E show the time course of changes in virulence over five years. There is no obvious association of virulence with changes in population size, and in panel E, there is an early major reduction in virulence despite almost none in the long term.

These virulence reductions are not evolutionary, but they could easily and mistakenly be interpreted as evolutionary because they occur as the pathogen invades and becomes established. The reduction in virulence is greatest when the R_0 of the pathogen is high because in this regime, many individuals with maternal antibodies get mild infections before the protection from antibodies wanes.

If maternal antibodies block infection, there is no change in the average virulence of infections – because blocked individuals do not get infected, and once blocking has waned, infections are fully virulent. The dashed black curves in panels 2 A and E show these non-changing virulence curves merely for comparison to the attenuation case.

Age dependence of case mortality

For many pathogens, the severity of an infection in naive hosts varies with age [1, 16, 18, 23, 28]. If the severity of disease in the young is lower than in adults, it is of course expected that more of the population will survive than when lethality is the same for young as for adults. But there may also be an amplification dynamic similar to that with maternal antibodies: a protected class (young) may be uncommon in a naive, equilibrium population, but this class will disproportionately survive to reproduce as the pathogen invades and kills off the unprotected classes. However, in contrast to the population protection derived from maternal antibody attenuation, there no 'inheritance' of age-based protection because all young are endowed with the same benefit regardless of parental status. This means that every individual experiences a protected phase even if their ancestors remained infection-free. Also in contrast to the results from maternal antibodies, age-based *blocking* might yield a substantial benefit. If the age-protected young are able to reproduce before becoming vulnerable, a self-perpetuating, protected class emerges – of youth begetting youth. Each youth will eventually pass out of the protected phase, but if they have already begun reproducing, then they can maintain themselves no matter how prevalent and deadly the pathogen is to adults. Finally for most hosts we expect the duration of protection by maternal antibodies to be shorter than the age when individuals transition from the protected class (young) to adults.

In practice, age dependence in which young are protected may be difficult to discriminate from maternal antibody effects except when the disease agent is first spreading in a population or is

tested experimentally. The combination of maternal antibodies and age dependence could provide a double layer of eventual protection. But here we study age dependence in isolation.

We take a simplified approach to age dependence and modify eqn set (1) modestly. We allow two age classes – one of 'youth' and one of adults (eqn set (3)). As with model (2), the model accommodates allowing protected youth (S_Y) to either block the infection or experience an attenuated infection:

$$
\begin{aligned}
\text{Susceptibles} \quad & \dot{S}_Y = -\beta_Y S_Y (I_Y + I_A) - \alpha S_Y - (d_0 + k_d N) S_Y + (b_0 - k_b N) N \\
& \dot{S}_A = -\beta_A S_A (I_Y + I_A) + \alpha S_Y - (d_0 + k_d N) S_A \\
\text{Infecteds} \quad & \dot{I}_Y = +\beta_Y S_Y (I_Y + I_A) - \nu I_Y - (d_0 + k_d N) I_Y \\
& \dot{I}_A = +\beta_A S_A (I_Y + I_A) - \nu I_A - (d_0 + k_d N) I_A \\
\text{Recovered} \quad & \dot{R} = +(1 - f_Y)\nu I_Y + (1 - f_A)\nu I_A - (d_0 + k_d N) R
\end{aligned} \tag{3}
$$

Subscripts "Y" and "A" refer to young and adults, respectively, and N is the total population. Note that an aging rate (α) replaces the antibody decay rate (γ) of model (2). Table 3 defines the variables and the one new parameter, the other parameters and parameter values being given in Table 1.

Variable	Description	Value
S_Y	Susceptible youth	
S_A	Susceptible adult	
I_Y	Infected youth	
I_A	Infected adult	
R	Recovered, fully immune	
N	Total population	
α	rate of aging (1/longevity of young age category)	2

Table 3: Variables and the one new parameter used in eqn (3). Except for the rate of aging, parameters are given in Table 1.

We acknowledge that model (3) is merely one of the several ways of structuring age dependence. For example, it allows all age classes to reproduce. Limiting reproduction to adults is a simple and realistic alternative, but it then changes the per capita population birth rate and thereby affects homeostasis such that a comparison to models (1) and (2) is no longer straightforward. The model also does not allow *infected* youth to age into infected adults, an assumption that is justified on the grounds that the duration of infection is much shorter than the age range for young. And of course, the model allows but two age classes. Our goal in presenting and analyzing this model is to provide a sense of how age-dependence can influence suppression, but the analysis is decidedly not comprehensive. Our analysis is limited to age-dependent attenuation, not blocking.

Protecting youth reduces population suppression

As with maternal antibody attenuation, age-dependent attenuation can have a large benefit under rapid homeostasis (Fig. 4 A, B). However, in contrast to the results from maternal antibodies,

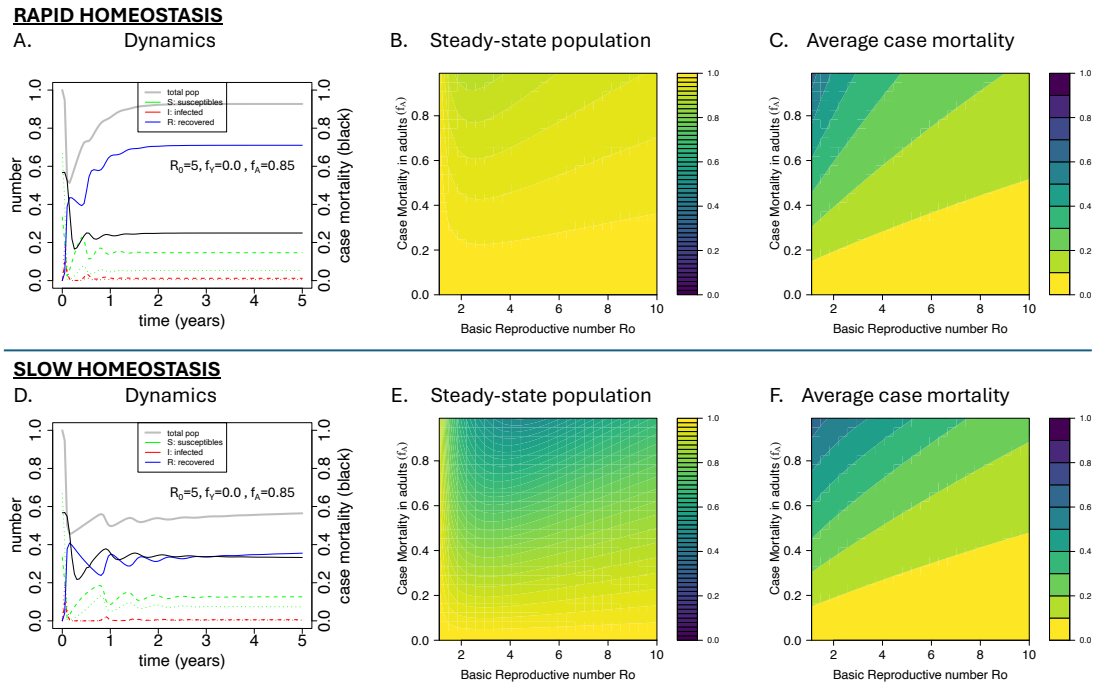

Figure 4: Age-dependent mortality in which young have asymptomatic infections $f_Y = 0$. The top (bottom) row is for rapid (slow) homeostasis. Panels A and D are short term dynamics, B, C, E and F are for steady state; dashed and dotted curves in A and D are for young and adults, respectively. The appropriate comparison for no age dependence is Fig. 1, and it is easy to see that age dependence has a profound effect. Furthermore, there is a profound effect of age dependence even for slow homeostasis, which was not the case for maternal antibodies. Average case mortality is seen to decline by steady state (C, F), increasingly so at high R_0.

age-dependent attenuation also greatly enhances population recovery with slow homeostasis, just not as much as with rapid homeostasis (panels D, E).

It is challenging to disentangle the effects underlying these recoveries because the case is not directly comparable to the basic model: youth are present at the start, and the protection of youth from serious disease means that adult case mortality does not apply to all infections when the pathogen first invades. This effect alone will lead to a smaller initial suppression of the population than in the basic model (where youth experience the same mortality as adults). However, inspection of the faint contours in panels B and E reveals minima near $R_0 = 2 - 3$ and substantial upward slopes as R_0 increases beyond those minima. The increased population recovery with higher R_0 is necessarily consequence of protected youth that reflects an outcome beyond their presence in the initial population. Higher R_0 results in earlier infections and thus in more youth being infected while they are protected – a similar effect as was seen with maternal antibodies. From the perspective of hoping to suppress a wild population, the effect is unfortunate, because higher recovery with higher R_0 is unintuitive and contrary to the goal. In the basic model, higher R_0 always resulted in greater suppression of the population.

As with maternal antibodies, age-dependent attenuation of infection results in substantial reductions in *average* case mortality in the long term (Fig. 4 C, F). In contrast to the maternal antibody model where the initial virulence equals f, under age-dependent model, if the young are protected (i.e. $f_Y = 0$) then the initial average virulence will equal the f_A times the fraction of the population that is adult. The upward-sloping contours reveal substantial reductions in virulence that increase with higher R_0. As with maternal antibody protection, these changes in virulence are not genetic. They reflect the extent to which infections of youth come to dominate all infections as the pathogen becomes endemic. Note that at steady state, the average virulence (case mortality) is broadly similar between rapid and slow homeostasis despite the (modest) differences in steady state population suppression.

Youth vulnerability increases population suppression

We have so far considered that the youngest class survives the infection. However infections of young can be severe for some pathogens [1, 18]. How are population suppression and virulence affected when youth are exceptionally vulnerable to being killed? Arguing along similar lines as above, if youth have higher mortality than adults, then it is expected the population will be suppressed more than if everyone experienced the adult morality rate. But feedback effects might operate to enhance suppression beyond this simple effect: as youth are killed off, the population of reproductives (adults) will eventually suffer, further depressing the population. (Here we expect an effect of our assumption that all age classes reproduce, because if youth could not reproduce, even greater suppression might result.)

We limit ourselves to the case in which case mortality of youth is extreme – all young infections die ($f_Y = 1$); this is of course just one of several ways to parameterize the model, but this extreme facilitates detecting the effect of youth vulnerability. Now population suppression increases with R_0, no doubt because higher R_0 increases the fraction of youth infected and thus killed (Fig. 5). Again, the population suppression is more extreme with slow homeostasis than with rapid homeostasis. But the effect is large even with rapid homeostasis.

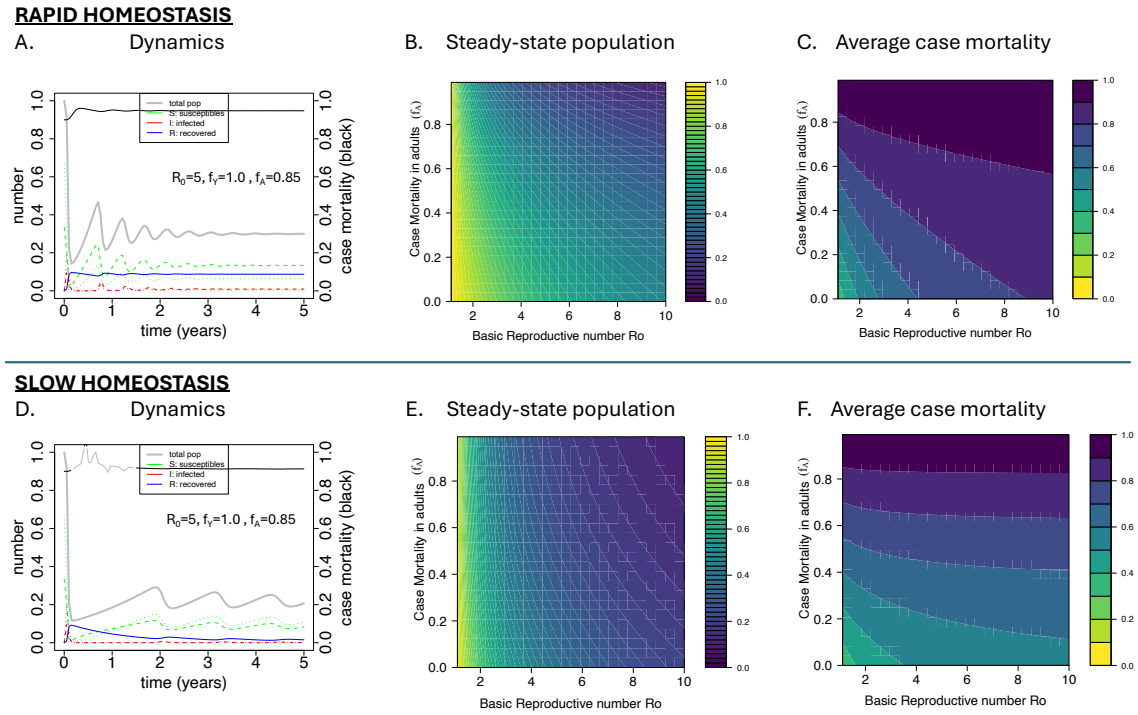

Figure 5: Age-dependent mortality in which young have invariably lethal infections $f_Y = 0$. The top (bottom) row is for rapid (slow) homeostasis. Panels A and D are short term dynamics, B, C, E and F are for steady state; dashed and dotted curves in A and D are for young and adults, respectively. As with Fig. 4, the appropriate comparison for no age dependence is Fig. 1, and it is easy to see that age dependence has a profound effect but now the opposite as when youth are protected. Furthermore, there is a profound effect of age dependence even for slow homeostasis, which was not the case for maternal antibodies. For rapid homeostasis, average case mortality is seen to increase at steady state (C, F), more so at high R_0.

Average case mortality at steady state is interesting (Fig. 5 C, F). With rapid homeostasis, case mortality increases with R_0, consistent with the pattern of population suppression. Since the initial case mortality is unaffected by R_0, the fact that steady state case mortality increases with R_0 indicates that average virulence is increasing as the pathogen invades. However, average case mortality under slow homeostasis appears almost unaffected by R_0, at least for high f_A. Even more surprising is that average case mortality at steady state is mostly less than under rapid homeostasis. Perhaps births are few enough under slow homeostasis that average case mortality is dominated by adult infections.

The main picture emerging from this section is that age dependence can profoundly affect population suppression in either direction. In attempts to suppress populations with pathogens, as was tried with Australian rabbits, understanding age dependence will be critical in anticipating long term effects.

Discussion

Our study used mathematical models to investigate the impact of a pathogen on the population-size of its host. The pathogen might be deliberately introduced to control an invasive species, as with rabbits in Australia and France [11, 19], or a pathogen might inadvertently jump to a new species of economic or conservation interest. This study builds on the pioneering work of May and Anderson [2, 21] that considered the role of pathogens in regulating host populations. Their papers focused on how pathogen regulation of the host population depends on the nature of the pathogen – its mode of transmission and the duration of host immunity. That work established some principles that have since become the standard understanding: (i) for a given level of disease-induced mortality, the extent of population suppression increases with increases in the pathogen's basic reproductive number (R_0), and (ii) the population typically rebounds after a high level of initial suppression.

Our analyses explored this question by considering factors that might alter the long term outcome. These include: (i) density-dependent population regulation; (ii) protection of offspring by maternal antibodies; and (iii) age-dependent disease severity (case mortality). Our first and simplest model considered changes in the rate of density-dependent population regulation with no maternal antibodies or age-dependence of case mortality. We observed the outcome described by earlier studies that population suppression increases with increases in both R_0 and case mortality. The most surprising result was that the degree of density-dependent population regulation (what we named 'homeostasis') had a large effect on long term population suppression. If there was rapid homeostatic regulation of the host population in the absence of infection, then the initial decline in the host population was followed by a rebound in the host population. If however there was only weak or slow homeostatic regulation of the host population then the population did not necessarily rebound after the initial phase but instead could experience a further decline. In short we found that at steady state, population sizes for the same pathogen could differ substantially, depending only on the strength of density-dependent regulation intrinsic to the population.

Efforts to predict long term pathogen impact will thus be hampered when density dependent regulation affects population suppression. Considerable effort by ecologists in the 1900s attempted to resolve whether populations were regulated by density-independent versus density-dependent mechanisms [3]. Our results suggest that the degree of density dependence is important, which is a more difficult problem than merely establishing whether regulation is density dependent. One may hope that some level of prediction is possible by merely knowing intrinsic birth rates of a species, since birth rates will set a limit on density-dependent homeostasis. And it should be appreciated that our analysis was limited to just a single case of slow homeostasis and one of rapid homeostasis, so we yet lack a comprehensive picture of density-dependent effects.

Beyond the basic ecological structure of the model, our study introduced two biologically-inspired properties that are expected to affect population suppression: maternal antibody protection of progeny and age-dependent vulnerability to the pathogen. Our analysis suggests that the population-protective effect of maternal antibodies is much greater if they do not prevent infection but attenuate its severity. This allows individuals who get infected while having maternal antibodies to acquire long-term protective immunity and to endow their progeny with the same protection. We found that if maternal antibodies attenuate infections there is a qualitative change in how increases in R_0 affect the population size and virulence (average case mortality). Specifically we found that having a higher R_0 could result in an increase in the population size at steady-state; this arose because a higher R_0 led to an increase in the fraction of individuals with maternal antibodies getting mild, immunizing infections prior to the loss of these antibodies.

Our work on maternal antibodies follows the precedents of three Fouchet et al. papers [12, 13, 14], who tailored their models to myxoma virus in rabbits and included several details that we intentionally omitted: seasonal reproduction by the rabbit plus maternal antibodies affecting three infection properties at once (blocking, attenuation, transmission). As here, Fouchet et al. [12] found that maternal antibodies were protective of the rabbit population at high transmission rates but not at low (their analysis was limited to a 100-fold difference in R_0). We too found the protective population effect of maternal antibodies to be sensitive to transmission as well as other details, although our analyses evaluated each property separately: blocking infection versus attenuation of case mortality, antibody duration, R_0 and density-dependent homeostasis. These combined results suggest that any prediction of maternal antibody effects in a field context will be difficult. Our results highlight the importance of understanding the mode of action of maternal antibodies – specifically whether they block vs. attenuate infections, and indeed whether there is a transition from blocking to attenuation as the titer of maternal antibodies wanes over time.

Age-dependent vulnerability to infection also affects long term population suppression; we considered only the case in which age dependence affected case mortality (without affecting susceptibility to infection) and then only considered simple extremes. Protection of young can greatly offset population suppression, and here, a substantial population recovery was observed for both rapid and slow homeostatic population regulation, contrary to the recovery being largely confined to rapid homeostasis for maternal antibodies. Conversely, extremely high mortality of the young enabled high levels of population suppression. Given the absence of clear biological generalities as to whether young are more or less protected than adults for infectious diseases [1, 18], age dependence presents a serious challenge to anticipating long term consequences.

For both the maternal antibody and the age dependent models, average case mortality ('virulence') changed between the time of pathogen invasion and endemism. For maternal antibodies and for age-dependent protection of young, average virulence of infections declined over time. This change is not evolutionary in a strict sense, rather it reflects changes in the relative abundance of benign versus lethal infections as the pathogen becomes common. Those changes in virulence superficially resemble evolution of reduced virulence. If the pathogen was to die out and then be reintroduced, the process would repeat.

Efforts to control invasive rabbits with viruses in Australia and France should in principle offer relevant case studies for evaluating these models [11, 19]. However, there are important omissions from our simple models that complicate comparisons to rabbit data: ongoing reinfection by myxoma virus so that immunity is not lifelong [19] and seasonal reproduction of rabbits [28]. In France, geographic variation in rabbit suppression from myxoma virus suggests that local factors are important [19]. Those factors could reflect structural omissions from our models or could stem from local variation in parameter values such that our models would match local abundances when parameterized correctly. Regardless, it seems clear that general models such as those here will not be appropriate for specific applications without modification. At the same time, the models point to principles that may apply widely and be important components in any specific application.

Although many aspects of the Australian efforts to suppress rabbits were carefully documented [11], quantitative details of the magnitude of population suppression do not appear to be readily available. Two viruses were introduced: myxoma virus, followed decades later by a calicivirus causing rabbit hemorrhagic fever disease. One survey spanning the introduction of myxoma virus in 1950 to 2013 shows a rapid 90% suppression of the rabbit population with only a small rebound over 3-4 decades [Fig 6 in 6]; a stylized plot from a different study of rabbits in South Australia shows a much faster recovery (until fleas were introduced) from an equally impressive initial suppression

[Fig. 1 in 25]. Details of both surveys are minimal. If maternal antibody protection was operating, or if young experienced attenuated infections, the rebounds should have been far more rapid than is evident in those surveys. Fouchet et al. [12] commented that young rabbits lacked viral receptors for myxoma virus, which would be consistent with blocked infections in young, but a lack of viral receptors is inconsistent with another paper reporting early infection of rabbits [19]. The data from those studies are thus fragmentary and do not provide much basis for a test of the models.

Following the eventual decline in myxoma-based suppression of Australian rabbits, a calicivirus was introduced. In the figures referenced above, the introduction appears to have worked in the short term. But young rabbits experience attenuated infections rather than blocked infections, and Taggart et al. [28] offered the strong caution that the virus should be introduced to populations outside of the birthing season to maximize suppression; apparently such attention to detail was commonly ignored in the calicivirus releases on diffeent properties. Our model omitted seasonal reproduction, and although there can be little doubt that timing of viral introduction is important, it is difficult to appreciate the long term quantitative effect without models tailored to rabbit biology, such as those of Fouchet et al. [12, 13, 14]. The need for periodic introductions of virus implies that it dies out, which should enhance its efficacy when re-introduced, but viral extinction does not occur in our deterministic models.

Our study leaves unexplored many obvious extensions of the models. Some specific extensions are relevant to the field observations mentioned above: seasonal host reproduction and stochastic (non-deterministic) viral abundance, model properties which might accommodate the observed viral extinctions. More generally, what properties of density dependence are important for long term suppression or recovery? Our model allowed 4 birth and death parameters, and we analyzed only two sets of values. Analysis of more parameter combinations will shed light on just which properties of density dependence are important. Indeed, our preliminary trials suggest that a model with a single density-dependent parameter may reproduce the qualitative results reported here. Furthermore, it will be desirable to consider different model structures for density dependence than the one we applied (which was linear in population size). Another important consideration is that our model decoupled case mortality and recovery from infection. A more common approach is to assign mortality and recovery rates from infection, whereby a high mortality rate shortens the duration of infection and thus reduces R_0 [4, 15]. If the model structure of case mortality greatly affects suppression, then it adds a further challenge in predicting the long term impact of a lethal pathogen.

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File S1: Supplementary Information

0.1 Parameter values

0.1.1 Estimating the parameters for small animal (rabbit)

1. From Google AI : 30 female offspring per year. Lifespan in the wild ie. at equilibirum in the absence of the pathogen is about 1 year. Choose max lifespan in the wild of 5 years though in captivity they can live a bit longer.
2. Rescale carrying capacity c=1.
3. Maximum lifespan 5 years gives $d_0 = 1/5$.
4. To get natural lifespan about 1 year $d_0 + k_d * c = 1$ gives $k_d = 1 - 1/5 = 0.8$
5. 30 female offspring per year gives $b_0 = \ln(30) \approx 3.5$
6. At steady-state (i.e. $c = 1$): $(b_0 - k_b * c) = (d_0 + k_d c) = 1$ which gives k_b 2.5
7. We can maintain the same eqm death rate by $d_0 = 0.8 \quad k_d = 0.2$
8. We can maintain the same eqm birth rate by $b_0 = 1.5 \quad k_b = 0.5$

0.1.2 Estimating the parameters for lower homeostasis

1. We keep the same carrying capacity $c = 1$
2. Let new $b_0 = 1.5$ and $d_0 = 4/5 = 0.8$
3. so new $k_d = 1 - 4/5 = 1/5 = 0.2$
4. and new $k_b = 1.5 - 1 = 0.5$

Note for simulations: During the initial epidemic phase the simulations often resulted in very low number of infecteds and populations becoming negative in simulations. We thus added a very small term equivalent to constant infections from outside of the order of 10^{-5} as can be seen in the code.

0.2 Supplementary Figures

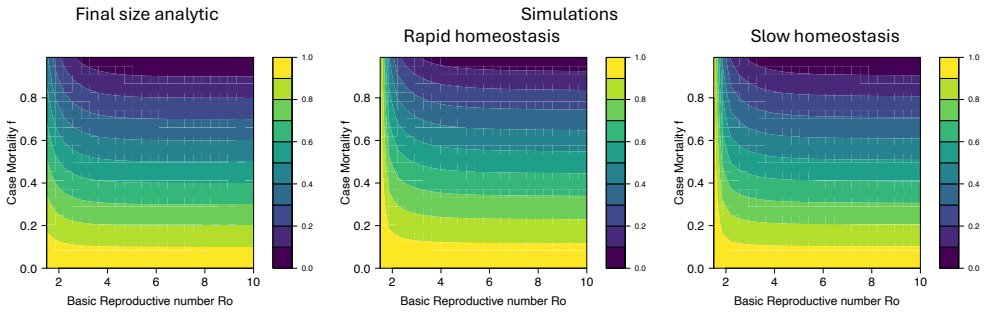

Figure 6: For the basic model (1), plots compare the analytic approximation for the initial minimum (left) and the actual initial minimum (middle and right). They are virtually indistinguishable except at the left margin. The discrepancy at low R_0 arises because, for these R_0, the pathogen spreads slowly and allows significant numbers of births during the initial phase.

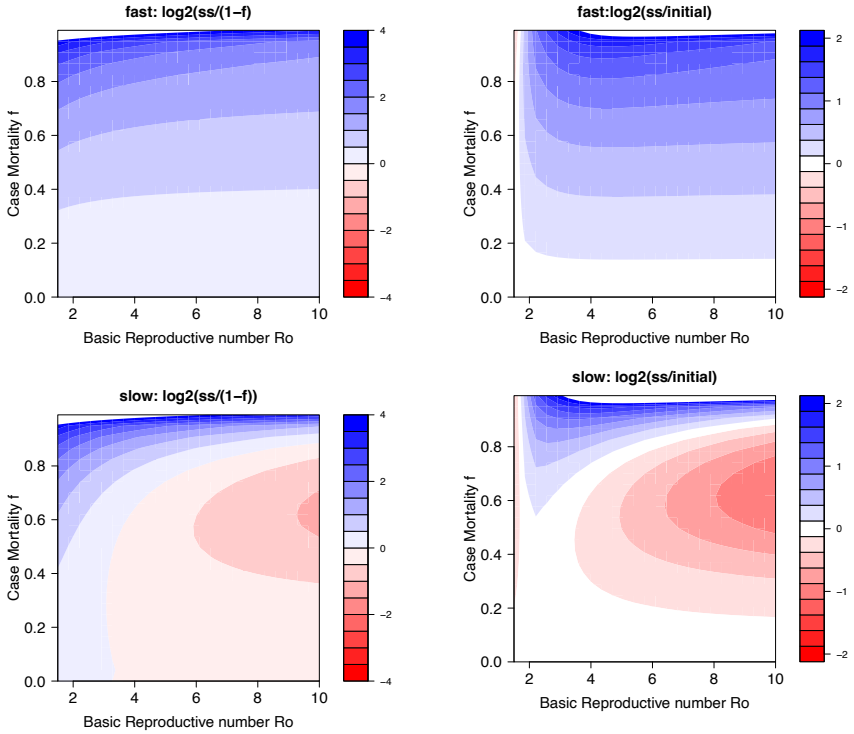

Figure 7: Population suppression at steady state can sometimes exceed case mortality, but not at the initial minimum. Red/pink shows the zones in which population size is less than $1 - f$, which is the fraction of cases surviving infection. Thus, red and pink show the zones in which the population is suppressed beyond the level that would be imagined from experimental studies of case mortality. Top plots show the log2(Steady-state/initial minima). Bottom plots show log2(steady-state/(1-f)). Parameter values are the same as for text Fig. 1.

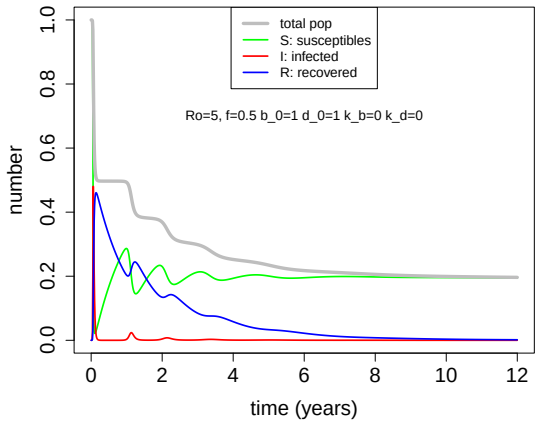

Figure 8: In the absence of homeostasis the total population declines until the reproductive number falls below 1. The early process involves stepwise decreases in population size, each preceded by an increase in the number of susceptibles that eventually enables the pathogen to expand further; recovereds decline as the susceptibles increase.