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Allee Effect from Parasite Spill-Back

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abstract: The exchange of native pathogens between wild and domesticated animals can lead to novel disease threats to wildlife. However, the dynamics of wild host-parasite systems exposed to a reservoir of domesticated hosts are not well understood. A simple mathematical model reveals that the spill-back of native parasites from domestic to wild hosts may cause a demographic Allee effect in the wild host population. A second model is tailored to the particulars of pink salmon (*Oncorhynchus gorbuscha*) and salmon lice (*Lepeophtheirus salmonis*), for which parasite spill-back is a conservation and fishery concern. In both models, parasite spill-back weakens the coupling of parasite and wild host abundance—particularly at low host abundance—causing parasites per host to increase as a wild host population declines. These findings show that parasites shared across host populations have effects analogous to those of generalist predators and can similarly cause an unstable equilibrium in a focal host population that separates persistence and extirpation. Allee effects in wildlife arising from parasite spill-back are likely to be most pronounced in systems where the magnitude of transmission from domestic to wild host populations is high because of high parasite abundance in domestic hosts, prolonged sympatry of domestic and wild hosts, a high transmission coefficient for parasites, long-lived parasite larvae, and proximity of domesticated populations to wildlife migration corridors.

Keywords: disease, conservation, depensation, transmission, fisheries, salmon.

Introduction

The spillover of native pathogens from wildlife to domestic hosts and subsequent spill-back to wild host populations is one mechanism underlying the emergence of infectious diseases (Daszak et al. 2000). For example, the transmission of pathogens between wild and farmed animals underlies the global spread of avian flu (Kilpatrick et al. 2006), outbreaks of parasitic copepods in coastal seas (Costello 2006, 2009), and the extinction threat to many mammals (Pedersen et al. 2007). While pathogen transmission between wild and domestic animals may cause decline of wild host populations, the dynamical properties of this process have not been carefully studied, although several studies have focused on analogous processes of cross transmission in multihost systems (Haydon et al. 2002; Holt et al. 2003; Dobson 2004) or on dynamics of diseases where pathogens increase in abiotic reservoirs (Thrall et al. 1997; Rosà et al. 2003). However, precise descriptions of dynamics not only could benefit disease management and biodiversity conservation but also may increase understanding of interactions between parasites and host population regulation.

In particular, there may be potential for parasite spill-back to give rise to Allee effects in the host population. Component Allee effects describe a decline in components of fitness of individuals when a population is small, such as reduced mating opportunities, whereas demographic Allee effects describe the occurrence of an unstable equilibrium at low population size that separates population extinction from persistence (Courchamp et al. 1999; Stephens et al. 1999). There is theoretical and empirical support that generalist predators and parasitoids that have a saturating functional response can generate an Allee effect in their prey (Gascoigne and Lipcius 2004; Courchamp et al. 2008). For parasites, however, work on Allee effects has either assumed a preexisting Allee effect in the host or studied emergence of Allee effects in the parasite, in both cases focusing on their potential influence on parasite transmission dynamics (Regoes et al. 2002; Courchamp et al. 2008; Krkošek et al. 2012). Although it is clear that a parasite can enable extinction of a focal species, either through apparent competition if shared with a (biotic) reservoir host (Holt et al. 2003) or by increasing in an abiotic reservoir, few dynamical models of these processes have been posed (de Castro and Bolker 2005), none with sufficient generality to determine whether we should ex-

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pect parasites to cause Allee effects as do other natural enemies.

We studied two simple mathematical models for parasite spill-back. The first model is a simple extension of a classical host-parasite population model (Anderson and May 1978; Grenfell and Dobson 1995). We give an intuitive graphical representation of the dynamics for the general reader, derive expressions for the magnitude of parasite spill-back relative to other demographic parameters for which demographic Allee effects emerge, and also provide approximate solutions for the location of the unstable equilibrium that divides persistence and extinction of the host population. The second model is tailored to the particulars of pink salmon (*Oncorhynchus gorbuscha*) and salmon lice (*Lepeophtheirus salmonis*), for which parasite spill-back is a conservation and fishery concern (Krkošek 2010). Both models agree that Allee effects may be a fundamental property of the dynamics of parasite spill-back. This generality may extend even farther, for example, to generalist macroparasites exploiting hosts that differ vastly in abundance.

General Model

We begin with a classical Anderson-May type host-macroparasite system that we simplify in standard ways (Anderson and May 1978; Grenfell and Dobson 1995). We then analyze the model (1) when parasites are randomly (Poisson) distributed on the host population and (2) when parasites are aggregated on the host population. In both cases, we derive expressions for the magnitude of parasite spill-back required for demographic Allee effects to occur, where the magnitude of spill-back is expressed relative to the demographic parameters of the host and the parasite. Although the expressions may at first appear technical, they have clear intuitive meanings that are graphically illustrated in the figures.

The host-parasite system is composed of a wild host population (N), free-living parasite larvae (L), and the total number of parasites infecting the host population (P). We include a term for constant immigration (spill-back) of parasite larvae from a reservoir domesticated host population. We assume that the abundance of parasites in the reservoir population is an exogenous variable under human control, reflecting natural processes that regulate parasite abundance as well as management of domesticated populations to comply with animal health and/or environmental regulations. The model is

$$\begin{aligned}
\underbrace{\frac{dN}{dt}}_{\text{rate of change of host population}} &= \underbrace{rN\left(1-\frac{N}{K}\right)}_{\text{logistic growth}} - \underbrace{\alpha P}_{\text{mortality from parasites}},\\
\underbrace{\frac{dL}{dt}}_{\text{rate of change of free-living parasites}} &= \underbrace{L_0}_{\text{immigration from domestic hosts}} + \underbrace{\lambda P}_{\text{reproduction from attached parasites}}\\
&\quad - \underbrace{\beta LN}_{\text{attachment to hosts}} - \underbrace{\mu_L L}_{\text{mortality}},\\
\underbrace{\frac{dP}{dt}}_{\text{rate of change of attached parasites}} &= \underbrace{\beta LN}_{\text{attachment to hosts}}\\
&\quad - \underbrace{(\mu_P + \nu(N))P}_{\text{loss of parasites from (1) direct parasite mortality and (2) non-parasite-associated host mortality}}\\
&\quad - \underbrace{\left[\alpha + \frac{\alpha(k+1)P}{kN}\right]P}_{\text{loss of parasites through parasite-induced host mortality, which includes effect of parasite clumping}},
\end{aligned} \tag{1}$$

where the wild host population has logistic growth with population growth rate r and carrying capacity K and also experiences parasite-induced host mortality at a per-parasite rate α. We assume that density dependence in the host affects the host death rate ν, and so $\nu = d_0 + d_1 N$, where d_0 and d_1 are positive constants that tune the strength of density-dependent mortality in the host. It follows, then, that $r = b - d_0$, where b is the birth rate, and also $K = (b - d_0)/d_1$. Attached parasites produce larvae at rate λ, and the larvae then die at rate μ_L or infect a host at rate β. Once infecting a host, attached parasites die at a per-capita rate μ_P, die when their host dies at its death rate ν, or die with their host because of parasite-induced host mortality at a per-parasite rate α. The distribution of parasites among hosts follows a negative binomial distribution with aggregation parameter k. Larval parasites from domestic hosts immigrate into the area of the wild host population at rate L_0.

A common simplification to the model involves a steady state approximation of the larval dynamics, which assumes that larvae are short-lived compared to the longevity of the adult parasitic stages (common for many host-macroparasite systems). For this, we set $dL/dt = 0$ and solve for the abundance of L that quickly equilibrates to changes in the parasite population abundance (Grenfell and Dobson 1995). This simplifies the system to

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - \alpha P,$$
$$\frac{dP}{dt} = \beta N\left(\frac{L_0 + \lambda P}{\beta N + \mu_L}\right) - (\mu_P + \nu(N))P - \left[\alpha + \frac{\alpha(k+1)P}{kN}\right]P. \quad (2)$$

Defining $\overline{P} = P/N$ as the average abundance of parasites per host and applying the quotient rule gives

$$\underbrace{\frac{dN}{dt}}_{\substack{\text{rate of change}\\ \text{of host population}}} = \underbrace{rN\left(1 - \frac{N}{K}\right)}_{\text{logistic growth}} - \underbrace{\alpha\overline{P}N}_{\substack{\text{mortality}\\ \text{from parasites}}},$$
$$\underbrace{\frac{d\overline{P}}{dt}}_{\substack{\text{rate of change of the}\\ \text{average number of}\\ \text{parasites per host}}} = \underbrace{\frac{\beta L_0}{\beta N + \mu_L}}_{\substack{\text{immigration from}\\ \text{domestic hosts}}} + \underbrace{\frac{\beta\lambda N}{\beta N + \mu_L}\overline{P}}_{\substack{\text{reproduction of}\\ \text{parasites on wild hosts}}} - \underbrace{\left(\varphi + \frac{\alpha}{k}\overline{P}\right)\overline{P}}_{\substack{\text{decline in average parasite load}\\ \text{through parasite mortality}}}, \quad (3)$$

where $\varphi = b + \alpha + \mu_P$, recalling that $b = r + d_0$ is the host birth rate.

General Model without Parasite Aggregation

As a simple first case, we assume that parasites are randomly distributed (not aggregated) on the host population and so infect hosts according to a Poisson process, and therefore we let $k \to \infty$. Alternatively, a Poisson approximation also holds if the rate of parasite-induced host mortality α is small relative to the aggregation parameter k, in which case $\alpha/k \ll 1$ and the model simplifies to

$$\frac{dN}{dt} = rN\left(1 - \frac{N}{K}\right) - \alpha\overline{P}N,$$
$$\frac{d\overline{P}}{dt} = \frac{\beta L_0}{\beta N + \mu_L} + \left(\frac{\beta\lambda N}{\beta N + \mu_L} - \varphi\right)\overline{P}. \quad (4)$$

Another simplification that we apply to the host-parasite model is that hosts are typically long-lived relative to the parasites that they carry. This is common for many, but not all, parasites. This yields another pseudo–steady state approximation by setting the derivative of $\overline{P}$ to 0 and solving for the equilibrium abundance of parasites that quickly tracks changes in host abundance,

$$\overline{P}^* = \left(\frac{\beta L_0}{\beta N + \mu_L}\right)\left(\varphi - \frac{\beta\lambda N}{\beta N + \mu_L}\right)^{-1}. \quad (5)$$

Equation (5) indicates that the average abundance of parasites per host increases toward $\beta L_0/\mu_L\varphi$ as the host population approaches 0 (fig. 1), a property that leads to a demographic Allee effect when L_0 is sufficiently strong, as we show below.

The dynamics of the host population can now be understood by writing

$$\frac{dN}{dt} = G(N) - H(N), \quad (6)$$

where host population growth follows the logistic equation in $G(N)$ and host mortality due to parasites is accounted for in the death rate $H(N)$, according to

$$G(N) = rN\left(1 - \frac{N}{K}\right),$$
$$H(N) = \alpha N\left(\frac{\beta L_0}{\beta N + \mu_L}\right)\left(\varphi - \frac{\beta\lambda N}{\beta N + \mu_L}\right)^{-1}. \quad (7)$$

The dynamics of equation (6) can be understood graphically (fig. 2). Regions where host population growth $G(N)$ exceed mortality from parasites $H(N)$ correspond to per-

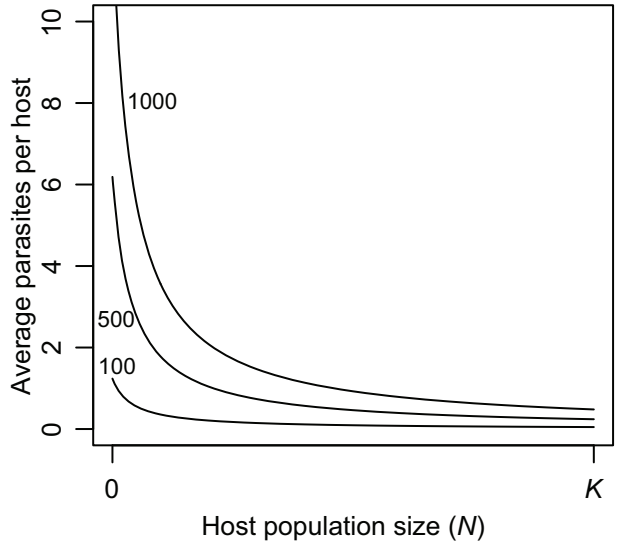

Figure 1: Average abundance of parasites per host $\overline{P}$, given by equation (5), in relation to host population size over a range of magnitudes of parasite spill-back, $L_0 = 100$, 500, and 1,000. Parameters used were $r = 0.01$, $K = 10{,}000$, $\alpha = 0.01$, $\beta = 0.05$, $\mu_L = 1/5$, $\lambda = 20$, and $\varphi = 20.2$.

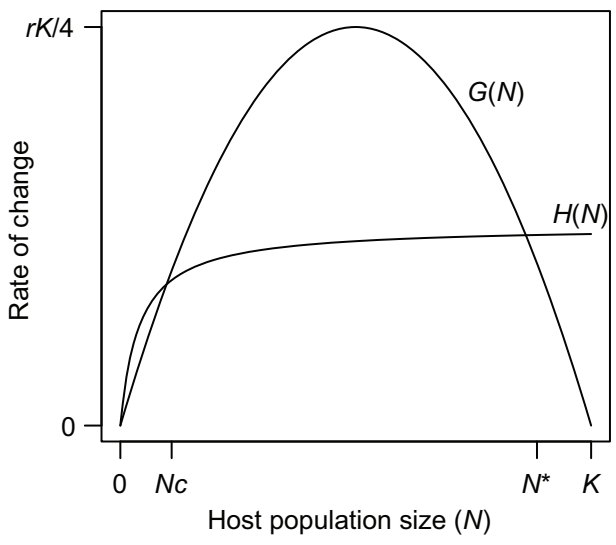

Figure 2: Graphical representation of the dynamics of the host population in equations (6) and (7) under a Poisson approximation, showing the numerical solutions for the rate of population growth via the logistic equation $G(N)$, as well as the rate of population decline due to mortality from parasites $H(N)$. Also shown are the maximum rate of increase $rK/4$ in $G(N)$, the Allee threshold N_c, the stable equilibrium host abundance N^*, and the host carrying capacity in the absence of parasites K. Parameters used were $r = 0.01$, $K = 10{,}000$, $\alpha = 0.01$, $\beta = 0.05$, $\mu_L = 1/5$, $\lambda = 20$, and $\varphi = 20.2$.

sistence of the host population. Regions where host mortality from parasites exceeds host population growth, $H(N) > G(N)$, correspond to extirpation of the host population due to parasite spill-back. The first equilibrium, denoted N_c in figure 2, is unstable and corresponds to the Allee threshold or demographic Allee effect that divides host population extirpation ($N < N_c$) from host population persistence ($N > N_c$). The second equilibrium, denoted N^* in figure 2, is the stable equilibrium for the host population that is smaller than the carrying capacity K because of parasite-induced host mortality. In appendix A, we give the mathematical expressions for the existence of these equilibria, conditions under which a demographic Allee effect occurs, and approximate calculations for the location of the Allee threshold.

General Model with Aggregation of Parasites

Here we consider the more general case where parasites are aggregated on the host population, which is common (Shaw and Dobson 1995). As in the previous section, we assume that hosts are long-lived relative to their parasites and apply a pseudo–steady state approximation to the parasite equation. We then model the dynamics of the host population according to equation (6), where host population growth follows the logistic equation in $G(N)$ and host mortality due to parasites is accounted for in the death rate $H(N)$, according to

$$\begin{aligned} G(N) &= rN\left(1 - \frac{N}{K}\right), \\ H(N) &= \alpha N\overline{P}^*, \end{aligned} \tag{8}$$

where $\overline{P}^*$ is the equilibrium abundance of parasites, the mathematical expression for which is given in appendix B. This gives a slightly modified graphical representation of the dynamics relative to that in the previous section—in particular, aggregation reduces the average number of parasites per host and also reduces the magnitude of Allee effects (figs. 3, 4). Increasing the aggregation of parasites, by decreasing k, causes the abundance of parasites per host to decrease at low population size, although there remains an increase in parasite abundance as the host population approaches 0 (fig. 3). Correspondingly, increasing parasite aggregation causes the Allee threshold to decrease, although the threshold persists over several orders of magnitude of k (fig. 4).

Salmon-Louse Model

In this section, we tailor a simple model for the dynamics of pink salmon (*Oncorhynchus gorbuscha*) and their ec-

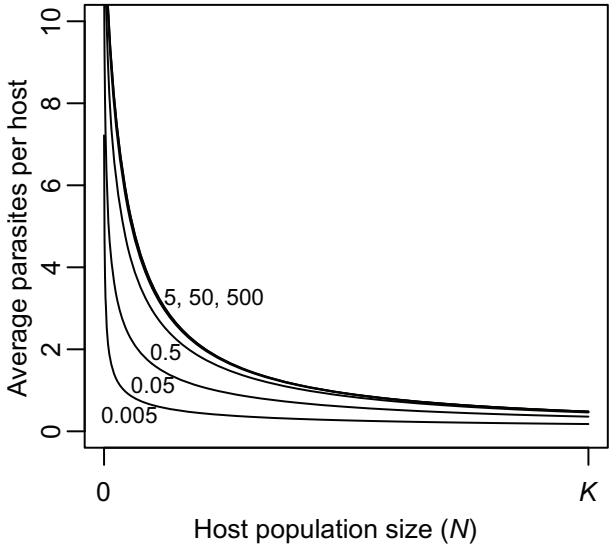

Figure 3: Effect of parasite aggregation on the average abundance of parasites per host $\overline{P}$ across host population size. Parasite aggregation is given by the negative binomial parameter $k = 0.005$, 0.05, 0.5, 5, 50, and 500. Other parameters used were $L_0 = 1{,}000$, $r = 0.01$, $K = 10{,}000$, $\alpha = 0.01$, $\beta = 0.05$, $\mu_L = 1/5$, $\lambda = 20$, and $\varphi = 20.2$.

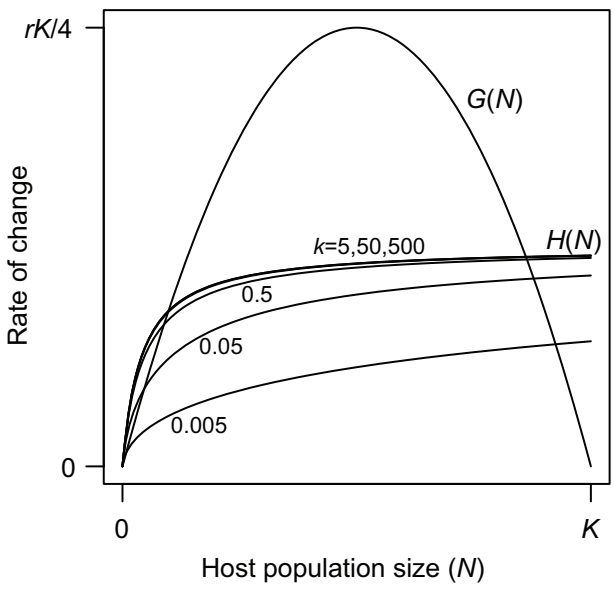

Figure 4: Graphical representation of the dynamics of the host population in equations (8) and (9) under the influence of parasite aggregation, as shown by the aggregation parameter $k = 0.005$, 0.05, 0.5, 5, 50, and 500. Shown are the numerical solutions for the rate of population growth via the logistic equation $G(N)$, as well as the rate of population decline due to mortality from parasites $H(N)$. Also shown are the maximum rate of increase $rK/4$ in $G(N)$, and the host carrying capacity in the absence of parasites K. The Allee threshold N_c occurs where $H(N)$ intersects $G(N)$ to the left of the maximum of $G(N)$, and the stable equilibrium host abundance N^* occurs where $H(N)$ intersects $G(N)$ to the right of the equilibrium. Parameters used were $L_0 = 250$, $r = 0.01$, $K = 10{,}000$, $\alpha = 0.01$, $\beta = 0.05$, $\mu_L = 1/5$, $\lambda = 20$, and $\varphi = 20.2$.

toparasitic copepods, salmon lice (*Lepeophtheirus salmonis*). Pink salmon are distributed throughout the North Pacific Ocean and are characterized by an anadromous and semelparous life cycle of 2 years, with spawning in autumn and emigration of offspring juveniles from rivers to sea the following spring (Heard 1991). Salmon lice are native marine ectoparasites of pink salmon that feed on the surface tissues of host fish and at sufficiently high abundance cause physiological stress (Brauner et al. 2012), behavioral changes (Krkošek et al. 2011), and mortality (Krkošek et al. 2006*a*). Louse transmission occurs primarily through free-swimming nauplius larvae that are hatched from parasitic gravid females and can be dispersed among and between farmed and wild salmon populations (Pike and Wadsworth 1999; Costello 2006). For pink salmon and sea lice, the concern is the effect of increased infection pressure on outmigrating juvenile salmon due to transmission from domesticated salmon populations in aquaculture sea cages situated on migration routes of wild salmon (Krkošek 2010).

We begin with the discrete-time Ricker model, which is commonly used for pink salmon population dynamics (Ricker 1954; Pyper et al. 2001), to which we couple an Anderson-May type submodel for the spread of parasites from farmed salmon to outmigrating wild juvenile salmon. This requires us to track two quantities for host abundance: (1) the abundance of adult spawners for each salmon generation, which follows discrete-time Ricker dynamics and is denoted by n_t, where t is measured in salmon generations, and (2) the within-generation abundance of salmon, which changes in continuous time from juvenile through to adults according to Anderson-May host-parasite dynamics within any particular generation of salmon and is denoted by $N_t(\tau)$, where τ is the time (e.g., days) since seawater entry for juvenile salmon within generation t (e.g., years) of the discrete-time Ricker model. Thus, juvenile abundance $N_t(\tau)$, which occurs in continuous time, is nested within the intergenerational Ricker dynamics.

We assume that parasite abundance on farmed salmon is at equilibrium, reflecting a balance between parasite population growth and fish-health management to comply with regulatory limits on parasite loads on farmed fish. The submodel for parasite spread is similar to the Anderson-May framework, used in the general model, and tracks the spread of one cohort of parasites from farmed salmon to one cohort of outmigrant wild salmon. This is an immigration-death process for both parasites and juvenile wild salmon. We assume that natural processes that would normally regulate the dynamics of the parasite population constrain the population growth of subsequent generations of the parasite after the migrating wild salmon population exits the farming region. Hence, the effects of subsequent generations of parasites on host mortality are subsumed within the dynamics and parameterization of the basic Ricker model, for which parameter values are known (Heard 1991; Myers et al. 1999; Pyper et al. 2001).

Dynamics of the pink salmon population follow a modified form of the Ricker model (Ricker 1954; Dennis et al. 1991),

$$n_{t+1} = n_t \exp(r - bn_t)Q_t, \tag{9}$$

where n_t is the population abundance in time t and t is measured in generations. The reproductive rate is r, which measures the production of juvenile salmon per spawner, and density-dependent mortality from egg to juvenile is determined by b. Survival from juvenile to adult recruit is determined by Q_t, which represents the within-cohort dynamics of mortality due to parasite and nonparasite processes.

For within-cohort dynamics, we represent the abundance of juvenile salmon at time τ after sea entry that were produced by spawners in generation t as $N_t(\tau)$. The

initial abundance of juveniles at the time of sea entry is $N_t(0) = n_t \exp(r - bn_t)$. The quantity Q_t emerges from the solution to a continuous-time submodel for the dynamics of parasite spill-back, on a timescale that is within the life cycle of the host. We give results for Q_t in appendix C and here describe the continuous-time submodel. We assume that the domesticated population initially becomes infected from the outside environment, after which parasite abundance on farms is determined by management factors such as harvest rates or parasiticide treatment. We therefore treat the abundance of parasites on domesticated hosts as an exogenous variable under human control.

The dynamics of larvae emanating from farmed hosts and attaching to and infecting wild juvenile salmon are

$$\begin{aligned}
\underbrace{\frac{dL_t(\tau)}{d\tau}}_{\substack{\text{rate of change of}\\ \text{larval parasite population}}} &= \underbrace{I(\tau)}_{\substack{\text{immigration of larval}\\ \text{parasites from domesticated hosts}}} \\
&\quad - \underbrace{\beta L_t(\tau) N_t(\tau)}_{\substack{\text{attachment of larval}\\ \text{parasites to hosts}}} \\
&\quad - \underbrace{\mu_{\mathrm{L}} L_t(\tau)}_{\text{mortality of larval parasites}}, \\
\underbrace{\frac{dP_t(\tau)}{dt}}_{\substack{\text{rate of change of}\\ \text{attached parasite population}}} &= \underbrace{\beta L_t(\tau) N_t(\tau)}_{\substack{\text{attachment of larval}\\ \text{parasites to hosts}}} \\
&\quad - \underbrace{(\mu_{\mathrm{P}} + \nu) P_t(\tau)}_{\substack{\text{mortality of attached parasites}\\ \text{from parasite and host death}}} \\
&\quad - \underbrace{\left[\alpha + \frac{\alpha(k+1)P_t(\tau)}{kN_t(\tau)}\right] P_t(\tau)}_{\substack{\text{mortality of attached parasites}\\ \text{when they kill their host}}},
\end{aligned} \tag{10}$$

where $L_t(\tau)$ is the density of parasite larvae in habitats of wild juvenile salmon at time τ after sea entry within year t, β is the rate at which individual larvae attach to a host, μ_{L} is the mortality rate of larvae, and $N_t(\tau)$ is the density of wild hosts during exposure to parasite larvae; $P_t(\tau)$ is the total number of adult parasites that are infecting wild salmon at time τ after sea entry, μ_{P} is the intrinsic rate of parasite mortality such that $1/\mu_{\mathrm{P}}$ is the average life span of an adult parasite, ν is the rate of host mortality from nonparasite sources, and α is the rate of parasite-induced host mortality. Immigration of parasite larvae $I(\tau)$ is variable in time to represent host migration and is assumed to have the form

$$I(\tau) = \begin{cases} I & \tau \le \tau_{\mathrm{c}}, \\ 0 & \tau > \tau_{\mathrm{c}}, \end{cases} \tag{11}$$

where τ_{c} is the time when juvenile salmon migrate out of the area of salmon farms.

As is typical of host-macroparasite models, equations (10) assume that the parasite population has an aggregated distribution on the host population, which is described by the aggregation parameter k from the negative binomial distribution (Grenfell and Dobson 1995). However, for our model, we consider only a brief period of transmission relative to the life cycle of pink salmon, which removes some of the processes of host age distribution and repeated exposures that can give rise to aggregated distributions (Anderson and Gordon 1982; Rousset et al. 1996). For simplicity, we therefore assume that transmission follows a Poisson process, for which we let $k \to \infty$ in equations (10). Further, setting $\overline{P} = |P_t(\tau)/N_t(\tau)|$, which is the average number of parasites per wildlife host, and applying the quotient rule gives the dynamics of wildlife host survival from juvenile to adult recruit and average parasites per wildlife host as

$$\begin{aligned}
\frac{dN_t(\tau)}{d\tau} &= -(\nu + \alpha\overline{P}(\tau))N_t(\tau), \\
\frac{d\overline{P}_t(\tau)}{dt} &= \beta L_t(\tau) - (\mu_{\mathrm{P}} + \alpha)\overline{P}_t(\tau).
\end{aligned} \tag{12}$$

The model can be simplified with a quasi–steady state approximation for the dynamics of the larval parasites (Anderson and May 1978; Grenfell and Dobson 1995). Assuming that the rates of larval mortality are high relative to the duration of exposure as well as to the dynamics of parasites in the wildlife host population, the density of larvae in wildlife habitats approaches equilibrium rapidly relative to the timescale of the adult parasites and the host species. Setting $dL/d\tau = 0$ gives

$$L_t(\tau) = \frac{I(\tau)}{\beta N_t(\tau) + \mu_{\mathrm{L}}} \tag{13}$$

for the density of larval parasites. Setting $\theta = \mu_{\mathrm{L}}/\beta$ and substituting equation (13) into equation (12) gives

$$\frac{d\overline{P}_t(\tau)}{d\tau} = \frac{I(\tau)}{N_t(\tau) + \theta} - (\mu_{\mathrm{P}} + \alpha)\overline{P}_t(\tau) \tag{14}$$

for the dynamics of parasites. In appendix C, we give mathematical expressions for the necessary (but not sufficient) conditions for a demographic Allee effect to occur.

To further analyze the model, we proceeded with numerical solutions. Most of the parameters for this model are known and are given in table 1. To calculate the reproductive rate for pink salmon r, we note that for the Ricker spawner-recruit model the reproductive rate is ap-

Table 1: Variables and parameters of the model, with the values used in the model analysis

Model terms	Definition	Value	References
n_t	Annual abundance of salmon returning to spawn in year t	Variable	
$N_t(\tau)$	Abundance of juvenile salmon at time τ in the cohort spawned by n_t adults in year t	Variable	
$P_t(\tau)$	Total number of parasites on wild salmon at time τ within a cohort of salmon in year t	Variable	
$\overline{P}_t(\tau)$	Average abundance of parasites per wild salmon at time τ within a cohort of salmon in year t	Variable	
t	Time, measured in generations of the salmon life cycle	Variable	
τ	Time within a salmon life cycle after the start of exposure to farm parasites	Variable	
r	Salmon reproductive rate	4.2[a]	1
b	Salmon density-dependent mortality	1 spawner^{-1} [b]	
τ_c	Period of exposure of juvenile salmon to sea lice from domesticated salmon	40 days	2
T	Time from seawater entry (~Apr 1) to recruitment (~Aug 1 + 1 year)	488 days	3
ν	Instantaneous natural mortality rate of pink salmon during period of parasite spill-back	.014 day^{-1}	4
γ	Overall natural mortality during pink salmon marine life (νT)	3	4
μ_p	Natural mortality rate of sea lice attached to a host (μ_p^{-1} is the average life span of an attached parasite)	.025 day^{-1}	5
α	Rate of parasite-induced host mortality	.01 day^{-1}	6
ψ	Rate of parasite transmission from domesticated to wild juvenile salmon	Unknown[c]	
θ	Inefficiency of host capture by free-living larval parasites	Unknown[c]	

Note: Parameter estimates are taken from the sea lice (*Lepeophtheirus salmonis*)–pink salmon (*Oncorhynchus gorbuscha*) system.

References 1. Heard (1991); Myers et al. (1999); Dorner et al. (2008); 2. Krkošek et al. (2009); 3. Heard (1991); 4. Parker (1968); Heard (1991); 5. Johnson and Albright (1991); Stien et al. (2005); 6. Krkošek et al. (2006*a*, 2009).

[a] Assuming 5% survival from ocean entry to spawning (Heard 1991); see text for calculations.

[b] Variable among populations, but population size can be scaled by b without affecting other parameters, so we therefore choose $b = 1$.

[c] We analyzed the model in relation to various parameter values/combinations.

proximately 1.2 (Myers et al. 1999; Dorner et al. 2008), which encompasses survival from smolt to adult. However, our reproductive rate represents production of smolts, and subsequent survival to maturity in the absence of parasite spill-back is given by $\exp(-\gamma)$. To get the reproductive rate for our model, we assume that survival from smolt to maturity for pink salmon is 5% (Heard 1991), which gives $\gamma = 3.0$ and $r = 4.2$. An important unknown parameter in the model is θ, which is an inverse measure of transmission efficiency, since it is a ratio of the transmission coefficient (the rate at which parasite larvae die divided by the rate at which parasite larvae attach to fish). For our purposes of understanding the qualitative dynamics, we analyze the model dynamics across a range of potential values of θ. We assume not that the following analyses yield quantitative predictions for Allee effects in pink salmon but rather that with the parameters we used, the analyses give a reasonable characterization of the potential qualitative behavior.

Numerical analyses reveal that the dynamics of the model are affected by the force of infection, which is $\Lambda = \beta I/(\beta N + \mu_L) = I/(N + \theta)$ in the salmon-louse model. Changes in the force of infection can occur in several ways: by changing I (the magnitude of parasite spill-back) or life-history characteristics β or μ_L or, equivalently, the inverse measure of transmission efficiency $\theta = \mu_L/\beta$. The changes in the dynamical properties of the salmon-louse model as θ and I change can be seen in the $n(t)$-versus-$n(t + 1)$ plane (fig. 5). There are shifts to lower carrying capacity as θ decreases and I increases, corresponding to reductions in the productivity of the salmon population due to spill-back of parasites from domesticated hosts. In some cases, the carrying capacity is lost, which corresponds to extinction of the wildlife hosts. Also evident from this analysis is the emergence of an unstable equilibrium that corresponds to the critical threshold in the wildlife host population, which differentiates population persistence and extinction. This critical threshold increases as θ decreases or I increases. When combined with the corresponding shifts in the carrying capacity, this produces smaller regions in the phase plane where populations can persist and larger regions where populations become extinct.

Discussion

Our results indicate that a demographic Allee effect may arise in a host population if a reservoir (abiotic or biotic) provides a fixed influx of larval parasites. The effect occurs for macroparasites because each parasite can attack only one host and mortality increases with parasite load, leading to inverse density dependence. This dynamic is analogous to, but distinct from, the mechanism by which a generalist predator with a saturating functional response causes an

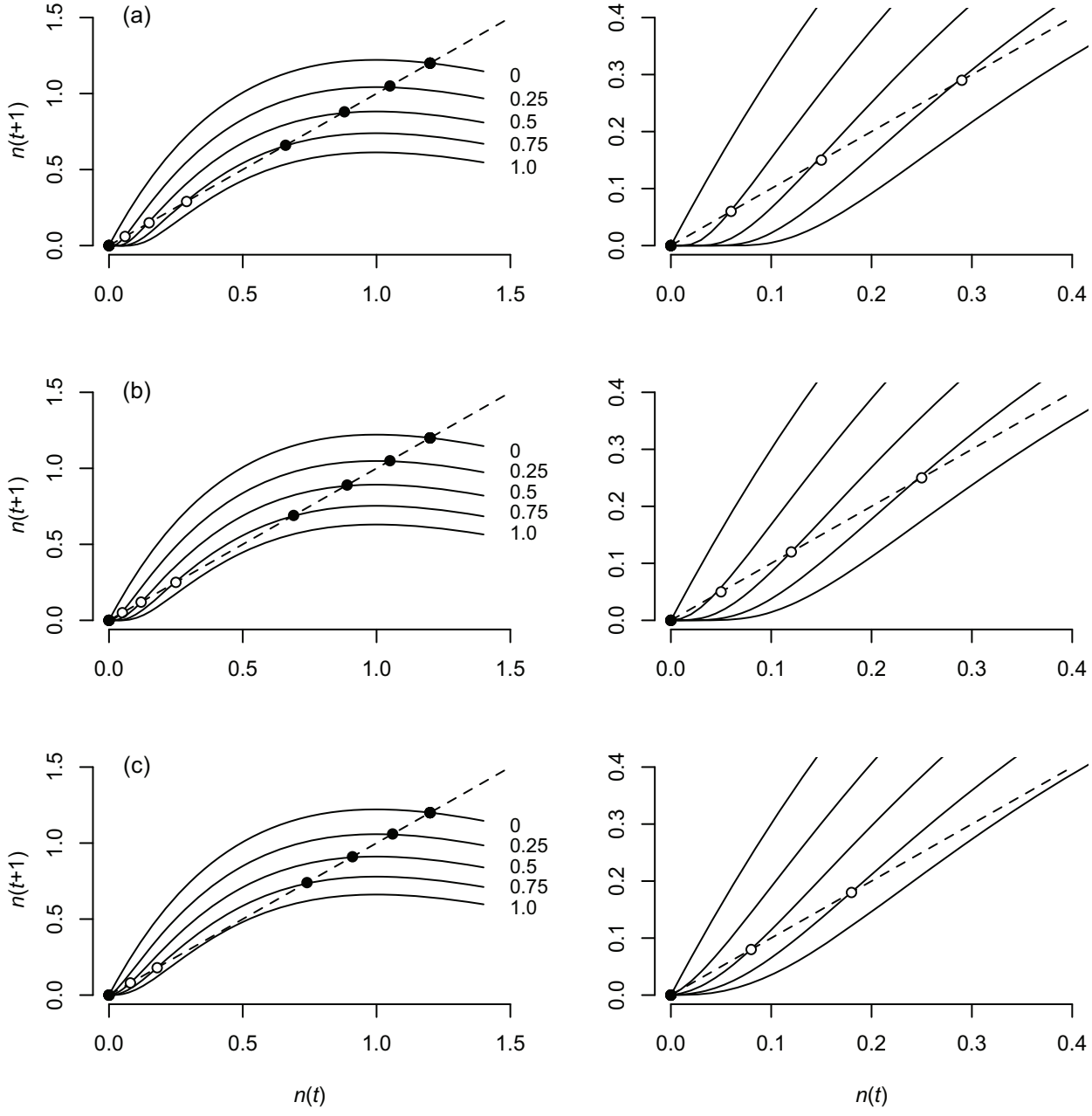

Figure 5: Analysis of the salmon-louse model under the influence of parasite spill-back from domestic hosts (eqq. [9]–[12]). Shown are the $n(t+1)$-versus-$n(t)$ graphs for varying values of transmission inefficiency θ and transmission magnitude I. Lines within panels show results for $I = 0$, 0.25, 0.5, 0.75, and 1.0 for values of $\theta = 0.2$ (*a*), 0.8 (*b*), and 2 (*c*). All other parameter values are given in table 1. Plots on the right are the same as those on the left, but with a different scale to show changes in unstable equilibria. The diagonal dashed line is the 1 : 1 line. Intersections of the model with the 1 : 1 line correspond to equilibria: filled circles show stable equilibria at extinction and carrying capacities, whereas open circles show unstable equilibria that correspond to N_c, the Allee threshold. The unstable equilibrium N_c marks the population size below which population trajectories will trend toward extinction and above which population trajectories will trend toward the carrying capacity.

Allee effect by imposing high per-capita rates of predation on a small prey population (Gascoigne and Lipcius 2004). Although the mechanism is general, we derive it in the context of the population dynamics of parasite spill-back from domesticated animal populations to wildlife populations, where analyses have primarily focused on survival of wild populations (Connors et al. 2010; Krkošek and Hilborn 2011) or the geographic spread of disease (Kilpatrick et al. 2006). Recent work, however, has suggested that spill-back may change the processes governing dynamics of wildlife populations (Ashander et al. 2012), and our results demonstrate one such change: exposure to parasite spill-back from domesticated hosts may cause a demographic Allee effect to emerge in wildlife host populations.

Allee effects and disease already pose concerns for conservation, but to date, these factors had been conceptually separate (de Castro and Bolker 2005). Our work demonstrates that, for macroparasites, host-parasite dynamics and the "small-population" factor (sensu de Castro and Bolker 2005) can interact to cause disease-induced extinction of small populations. In natural populations, this interaction has implications for conservation or restoration, because an Allee effect due to parasite spill-back may occur in wild populations that otherwise display no Allee effect, entrapping them in an extinction (or extirpation) equilibrium, potentially limiting their recovery. This effect may be particularly insidious in coastal seas, where there is already concern that Allee effects may limit the recovery of fisheries (Liermann and Hilborn 1997) and where depleted fisheries contribute to the socioeconomic rationale for rapid growth of aquaculture (Young and Matthews 2010).

Our results indicate that Allee effects emerging as a result of spill-back are likely to be most pronounced in systems where there is a high force of infection (Λ) due to parasite spill-back. This may be due to high levels of parasite transmission from domestic to wildlife populations (high levels of I) or to high levels of parasite transmission efficiency (low values of θ due to a high transmission coefficient β or a slow larval mortality rate μ_L). Situations where there may be a high force of infection in spill-back dynamics may be those where the parasite has high searching efficiency or where other processes bring the parasite into close contact with the wildlife host population. Examples may include vector-borne diseases, where transmission is mediated by the active search of a vector for natural hosts (Wonham et al. 2006) or where domesticated host populations are situated on constricted migration routes of wild host populations, such as salmon (Krkošek et al. 2006*b*; Heuch et al. 2009). A high force of infection is unlikely to occur in situations where the parasite is relatively immobile and the wildlife host population is widely dispersed and segregated from domesticated animals, such as in some types of terrestrial agriculture of mammals. From a management perspective, it is likely that the magnitude of spill-back I will be the most important because it is amendable to health management in the domesticated population (Saksida et al. 2010; Peacock et al. 2013; Rogers et al. 2013).

Our results relied on two modeling approaches, one that extended a classical Anderson-May host-parasite model (Anderson and May 1978; Grenfell and Dobson 1995) and one that was tailored to the specifics of a pink salmon–sea lice case study. While these modeling approaches agreed on the general qualitative dynamics that parasite spill-back may cause Allee effects in a wild host population, they nevertheless relied on modeling assumptions that may not apply to all parasite spill-back situations. For example, in line with classical models, we assumed that parasites cause a linear per-parasite increase in host mortality rate. While this may be the simplest possible first approach, it may be that some host mortality responses to parasites may be nonlinear, such as a threshold step function. In addition, some parasites may affect host fitness by interfering with birth rates (i.e., castrators) rather than host mortality, a scenario that we did not consider. For our salmon case study, we did not consider heterogeneity in age at maturity, which occurs for some salmon species, nor did we consider iteroparity (although our first model may be appropriate in this case). Thus, our results should not be considered to be applicable to all situations; rather, they constitute a general starting point from which small variations in the details of the host-parasite relationship can be considered.

The Allee effect caused by parasite spill-back in wild host populations is analogous to another mechanism by which demographic Allee effects may emerge, a generalist predator (Gascoigne and Lipcius 2004; Kramer et al. 2009), but the mechanics of parasite spill-back are subtly different: typically, a parasite will infect only one host individual, and when that individual dies, so does its parasite—although ectoparasites such as sea lice may survive (Connors et al. 2008), which could exacerbate Allee effects. Although our theoretical results are general and widely applicable, parasite-mediated interactions between fisheries and aquaculture, such as with salmon (*Salmo salar* and *Oncorhynchus* spp.) and sea lice, are likely to be particularly susceptible to Allee effects associated with parasite spill-back, because of extensive transmission between wild and domesticated populations as well as a tendency of aquaculture operations to be situated where wild fish aggregate and/or migrate.

Previous theoretical models of salmon and sea lice have investigated the effects of parasite transmission between wild and farmed populations in the context of declining

productivity of wild stocks (Frazer 2009), predation risk (Krkošek et al. 2011), and cyclical dynamics of pink salmon (Ashander et al. 2012). Several other studies have applied mathematical models of sea lice and salmon population dynamics to field and experimental data to estimate a number of processes: the magnitude and spatial extent of parasite spill-back (Krkošek et al. 2005, 2006*b*), parasite-induced host mortality of juvenile salmon within a wild salmon cohort (Krkošek et al. 2006*b*, 2009; Morton et al. 2010), and the overall effect of parasite spill-back on the productivity of wild salmon populations (Krkošek et al. 2007; Connors et al. 2010; Krkošek and Hilborn 2011). However, none of these studies have identified the potential of parasite spill-back to induce Allee effects in wild stocks.

In nature, there could be interactions between the mechanisms described here and other ecological processes already thought to produce Allee effects. For example, generalist predators can cause Allee effects in both fishes and other taxa (Neave 1953; Peterman 1977; Gascoigne and Lipcius 2004). If predation is selective on infected hosts and predators are generalists whose abundance is not (or weakly) connected to the prey population, then this would act to intensify mortality of prey at low population size (Krkošek et al. 2011), thereby intensifying Allee effects. At large group sizes, animal aggregation is predicted to be selected for by dilution of predators as well as of parasites (Landeau and Terborgh 1986; Poulin and FitzGerald 1989). Our results suggest that such protection from parasitism at high abundance may cause Allee effects from parasite spill-back because of elevated average infection intensity in small populations. This is similar to dilution of predation risk by prey aggregation, but in our case it is increasing host abundance that dilutes an externally generated infection risk. This could be further compounded by increased predation rates due to infection of individuals as well as increased exposure of individuals in smaller groups to predators.

As populations of humans and our domesticated animals continue to grow, so too does the potential for changes in disease dynamics (Daszak et al. 2000). Since the work of Daszak et al. (2000), much focus has been placed on the role of transmission among humans, wildlife, and domesticated animals in the emergence and spread of infectious diseases. In addition, conservation biologists are increasingly aware of the potential of parasite spill-back to threaten wildlife populations or limit recovery efforts, although most of the focus has been on viral or bacterial pathogens (Lafferty and Gerber 2002; Haydon et al. 2006; Pedersen et al. 2007; Diana 2009). Little attention, however, has been focused on the details of the dynamics of parasite spill-back, particularly for macroparasites. Our results indicate that parasite spill-back may have the predictable effect of reducing the survival of wildlife hosts and that this may also manifest itself in demographic Allee effects and the accompanying risks for population extirpation and recovery.

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APPENDIX A

General Model without Parasite Aggregation

The equilibria of model (3) are given implicitly by

$$N^* = K\left(1 - \frac{\alpha}{r}\overline{P}^*\right),$$
$$\overline{P}^* = \left(\frac{\beta L_0}{\beta N^* + \mu_L}\right)\left(\varphi - \frac{\beta\lambda N^*}{\beta N^* + \mu_L}\right)^{-1} \quad \text{(A1)}$$

and can be visualized graphically as the intersection of the G and H curves (fig. 2). This implies the existence of an unstable equilibrium and the emergence of a demographic Allee effect due to parasite spill-back.

From equations (A1), a sufficient condition for the persistence of the host-parasite system with parasite spill-back can be derived by first noting that the condition for a nonnegative steady state is

$$\varphi > \frac{\beta\lambda N^*}{\beta N^* + \mu_L}. \quad \text{(A2)}$$

The conditions on the magnitude of parasite spill-back L_0 for which a demographic Allee effect in the host population occurs are (1) that the slope at the origin of $H(N)$ is greater than $G(N)$ (fig. 2), which gives the minimum magnitude of L_0 required to produce a demographic Allee effect as

$$L_0 > \frac{r\varphi\mu_L}{\alpha\beta}, \tag{A3}$$

and (2) that the maximum of $G(N)$, which is $rK/4$ and is located at $N = K/2$, is greater than $H(N = K/2)$ (fig. 2), which gives the maximum magnitude of parasite spill-back below which a demographic Allee effect occurs and above which extinction occurs,

$$L_0 < \frac{\varphi r}{4\alpha\beta}[\beta K(\varphi - \lambda) + 2\varphi\mu_L]. \tag{A4}$$

If the condition in equation (A3) is not met, then the host population persists without the existence of a demographic Allee effect, whereas if the condition in equation (A4) is not met, then there is no positive equilibrium for the host population, resulting in extinction.

For the case where conditions in equations (A3) and (A4) are satisfied, there is an approximate expression for the location of the Allee threshold. For this, we study the dynamics of the host when its population size is small (i.e., much below its carrying capacity), and so $N/K \ll 1$, which simplifies the system in equations (6) and (7) to

$$\frac{dN}{dt} \approx rN - \alpha N\left[\frac{\beta L_0}{\beta N(\varphi - \lambda) + \varphi\mu_L}\right] \tag{A5}$$

for small N. Equation (A5) provides an approximate location of the Allee threshold by setting $dN/dt = 0$ and solving for N, which gives

$$N_c \approx \frac{(\alpha\beta L_0 - r\varphi\mu_L)}{r\beta(\varphi - \lambda)}. \tag{A6}$$

APPENDIX B

General Model with Parasite Aggregation

For the case of parasite aggregation given by equations (8) and (9), the mathematics are more complicated, as the average abundance of parasites per host $\overline{P}^*$ is given by the roots of the quadratic equation

$$\frac{\alpha}{k}\overline{P}^2 + \left(\varphi - \frac{\beta\lambda N}{\beta N + \mu_L}\right)\overline{P} - \frac{\beta L_0}{\beta N + \mu_L} = 0. \tag{B1}$$

All roots are real, since all parameters are nonnegative, and applying condition (A2) ensures that $\varphi - \beta\lambda N^*(\beta N^* + \mu_L)^{-1} > 0$, so there will be one positive root given by

$$\overline{P}^* = \frac{k}{2\alpha}\left[\sqrt{B^2 + \frac{4\alpha\beta L_0}{k(\beta N + \mu_L)}} - B\right], \tag{B2}$$

where

$$B = \varphi - \frac{\beta\lambda N}{\beta N + \mu_L}, \tag{B3}$$

and the other root is negative and so not of biological interest.

APPENDIX C

Salmon-Louse Model

To simplify the model defined by equation (14), we apply another pseudo–steady state approximation to the dynamics of parasites, assuming that parasite abundance quickly equilibrates in response to changes in the density of the juvenile salmon population. Therefore, the equilibrium of equation (14) gives the abundance of parasites per host as

$$\overline{P}_t(\tau) = \frac{I(\tau)}{(N_t(\tau) + \theta)(\mu_P + \alpha)}. \tag{C1}$$

Importantly, equation (C1) implies that the abundance of parasites per host increases as host abundance approaches 0—the key element of parasite spill-back in the general model that yields a demographic Allee effect in the host population dynamics.

To study the dynamics of pink salmon in the present model and investigate the potential of demographic Allee effects, note that the fraction of the wildlife host population that survives parasite spill-back is

$$\begin{aligned} Q_t &= \exp\left(-\int_0^T \left(\nu(\tau) + \alpha\overline{P}_t(\tau)\right)d\tau\right) \\ &= \exp\left(-\left(\gamma + \int_0^T \alpha\overline{P}_t(\tau)d\tau\right)\right), \end{aligned} \tag{C2}$$

where T is the within-cohort time of recruitment and $\gamma = \nu T$ is the overall mortality. We can then rewrite equation (9) as

$$n_{t+1} = n_t \exp\left(r - \gamma - bn_t - \frac{\alpha I}{(\mu_P + \alpha)}\int_0^{\tau_c} \frac{1}{N_t(\tau) + \theta}d\tau\right), \tag{C3}$$

where $N_t(\tau)$ is given by the solution at time $\tau \le \tau_c$ to

$$\frac{dN_t(\tau)}{d\tau} = -\left[\nu + \frac{\alpha}{(\mu_P + \alpha)}\frac{I(\tau)}{(N_t(\tau) + \theta)}\right]N_t(\tau), \quad \text{(C4)}$$

with the initial condition that $N_t(0) = n_t \exp(r - bn_t)$. Unfortunately, there is no closed-form solution to equation (C4). However, a necessary, but not sufficient, condition for an Allee effect to emerge is that the slope at the origin for equation (C4) must be less than 1, which is true if

$$r - \gamma - \frac{\alpha I \tau_c}{(\mu_P + \alpha)\theta} < 0 \Leftrightarrow I > \frac{\theta}{\alpha\tau_c}(r - \gamma)(\mu_P + \alpha). \quad \text{(C5)}$$

If relation (C5) is satisfied, then either an Allee effect occurs or there is no persistence of the salmon population at all, resulting in extinction.

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