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Does infectious disease influence the efficacy of marine protected areas? A theoretical framework

HAMISH McCALLUM*, LEAH GERBER† and ANDREA JANI†

**School of Integrative Biology, The University of Queensland, Brisbane 4072, Australia, and †School of Life Sciences, Arizona State University, College and University Dr Tempe, AZ 85287–1501, USA*

Summary

1. Marine protected areas are increasingly being recommended as an essential component of the management of exploited marine species, but virtually no attention has been given to the influence of parasites. This may be substantial, as a primary effect of marine reserves is to increase the density of an exploited population within the reserve relative to outside the reserve, which may facilitate parasite transmission.

2. We used a simple deterministic model of microparasitic infection in a fishery with a reserve to investigate equilibrium yield and parasite prevalence inside and outside the reserve as a function of three control variables: the proportion of habitat inside the reserve, fishing mortality and the rate of interchange between the stock and the reserve.

3. While our model is generic, we parameterized it with values that may be appropriate to the interaction between abalone and *Rickettsia*.

4. The presence of a pathogen does not necessarily decrease yield when a reserve is present, particularly if the rate of movement of adult hosts between stock and reserve is low.

5. *Synthesis and applications.* Pathogens have important implications for the design of marine reserves. Our modelling identifies two key considerations. First, 'fishing out' a pathogen by reducing the host population density to a level below the threshold for disease maintenance is a potential management strategy that is made more difficult by establishing a reserve. Secondly, the effect of a highly transmissible pathogen without a reserve is to cause a rapid decline in equilibrium yield for efforts beyond those that produce maximum sustainable yield, making the fishery prone to collapse. Introducing a reserve decreases yield in this case, but makes the fishery much more resistant to collapse.

Key-words: conservation biology, host–pathogen models, marine reserves, sustainable harvesting

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Introduction

In the midst of the global effort to establish marine protected areas (Lubchenco *et al*. 2003), the effect of parasites and pathogens on marine protected areas (MPAs) has received virtually no attention. Parasites and pathogens may be important to fisheries, reducing both abundance and yield by increasing mortality, reducing fecundity, affecting the size structure of the population or by reducing the marketability of harvested stocks (Dobson & May 1987). In this paper, we examine the consequences of epidemiological theory for reserve efficacy.

Most empirical evidence for reserve performance is based on studies from within the boundaries of a single reserve. Predictions about the potential for marine reserves to enhance fisheries are therefore based principally on insights from ecological theory (Demartini 1993; Nowlis & Roberts 1999; Neubert 2003). Two generalities to emerge from existing theory are: (1) reserves can increase yield when fishing effort is sufficiently high that recruitment would otherwise decline (Hastings & Botsford 1999) and (2) reserves should provide fewer benefits to species with greater rates of movement (Botsford, Hastings & Gaines 2001). Whether establishing marine reserves increase yields in practice remains controversial (Norse *et al*. 2003), but a recent review of empirical evidence (Gell & Roberts 2003) suggests that reserves have benefited a wide variety of exploited marine

Correspondence: Hamish McCallum (fax: +617 33651655; e-mail: h.mccallum@uq.edu.au).

species. These benefits, which develop rapidly, include increases in density, biomass, average organism size and species richness (Halpern & Warner 2002; Willis, Millar & Babcock 2003). It is certainly the case that marine reserves are being implemented world-wide on an unprecedented scale. For example, the Australian Government has recently set aside one-third of the Great Barrier Reef as no-take zones (Kemp 2004).

Disease dynamics, fishing practices and MPAs may interact in a variety of ways. For example, parasites may contribute to fishery collapse in protected areas of high host density, or a fishery may eliminate a parasite from a fished stock. In many cases, overexploitation has resulted in stressed populations of many species across the globe (Lafferty & Kuris 1999), and parasites that decrease host density have potential to synergistically exacerbate fishing mortality. Theoretically, commercial fisheries that reduce abundance of a fished species should also reduce rates of parasitism on that species. The observation that reports of disease outbreaks in fish are decreasing, in contrast to increasing reports of disease in most other marine taxa (Ward & Lafferty 2004), provides some support for this idea.

A range of marine fisheries, both for finfish and invertebrates, are known to be affected by pathogens. Table 1 lists some examples, together with some key life history traits. Kuris & Lafferty (1992) provide a similar table, concentrating on crustacean fisheries. These pathogens may affect either or both host survival and reproductive success, and may be expected to be of relevance to both conservation and fishery concerns in marine reserve design. In addition, some parasites [e.g. nematodes *Anisakis simplex* and *Pseudoterranova decipiens* infecting cod (Platt 1975)] that are not detrimental to host survival or reproduction may yet be of concern to fishery-orientated reserves because infected hosts lose marketability or pose health hazards when eaten by humans. Because many of these pathogens are transmitted either through direct host contact or via infective stages shed into the water by infected hosts, increases in host density within reserves may facilitate pathogen persistence or growth, thereby dampening positive effects of the reserve on the host species.

Classic epidemiological theory predicts that a pathogen will persist in a host population only if its transmission to new hosts is greater than the loss of infected hosts to death or recovery, with the result that there is a minimum host density below which the pathogen will not persist (Kermack & McKendrick 1927). Models by Dobson & May (1987) suggest that fishing can result in removal of a parasite if it takes the population below the host density threshold for the parasite. This 'fishing out' strategy may be acceptable if the host threshold density is higher than the density for maximum sustainable yield or if host population recovery is anticipated from long-distance dispersal. By maintaining areas of high host density a marine reserve system is likely to make fishing out pathogens more difficult.

Table 1. Life-history traits of host–pathogen interactions important in marine fisheries. Mode of transmission indicates whether a given pathogen is transmitted by direct contact, indirectly in water, or trophically (usually by ingestion of an alternate host). For non-trophically transmitted pathogens, we specify whether transmission is vertical or horizontal. 'Stage impacted by pathogen' refers to host life stage (egg, larva, juvenile, adult) infected by each pathogen. 'Stage of host movement' indicates the host life stage during which most dispersal or migration takes place. 'Host stage impacted by pathogen' specifies whether each pathogen affects host survival or reproduction

Host	Parasite/pathogen	Mode of transmission	Stage of host movement	Host stage impacted by pathogen	Host vital rate impacted
Abalone	Rickettsiales-like prokaryote	Indirect[1]	Larvae[2]	Adults[1]	Survival[2]
King and Dungeness crabs	Nemertian	Indirect and direct[3,4]	Larvae[18]	Eggs[3,4]	Fecundity[3,4]
Cod	Copepod gill parasite	Indirect[5]	Adults	Adults[5]	Survival[5]
Reef fish	Helminths	Trophic[6]	Larvae	Larvae[6]	Survival[6]
Herring	*Ichthyophonus hofferi*	Trophic[7]	Adults	Adults[8]	Survival[7]
Salmon	Infectious salmon anemia virus	Horizontal, direct and indirect[9,10,11], possibly spread by fish farms[12]	Adults	Adults[13]	Survival[13]
Salmon	*Aeromonas salmonicida*	Horizontal, direct and indirect[14]	Adults	Adults[14]	Survival[14]
Sturgeon	Iridovirus	Vertical and horizontal[15]	Adults	Juveniles[15]	Survival[15]
Cod	Nematodes (*P. decipiens* and *A. simplex*)	Indirect, trophic[16]	Adults	Adults[16]	None[16]
Sea urchin	*Vibrio* sp.	Indirect[17]	Adults	Adults[17]	Survival[17]

[1]Friedman *et al*. 2002; [2]Hobday & Tegner 2000; [3]Kuris *et al*. 1991; [4]Wickham & Kuris 1985; [5]Jones & Taggart 1998; [6]Rigby & Dufour 1996; [7]Jones & Dawe 2002; [8]Patterson 1996; [9]Snow, Raynard & Bruno 2001; [10]Lovdal & Enger 2002; [11]Murray, Smith & Stagg 2002; [12]Nylund & Jakobsen 1995; [13]Bakke & Harris 1998; [14]Austin & Austin 1987; [15]Georgiadis *et al*. 2001; [16]Platt 1975; [17]Tajima *et al*. 1998; [18]Armand Kuris, personal communication.

Kuris & Lafferty (1992) generalized the Dobson & May (1987) approach and showed that fishing out a parasite is possible only for host–parasite interactions where the parasite has a recruitment system that is relatively closed compared to the open recruitment of its host. There are several examples in the literature of parasites being eliminated locally from a population by reduction in the density of their hosts. For example, Amundsen & Kristoffersen (1990) report the elimination of a cestode from a whitefish population by heavy exploitation of the host. Culver & Kuris (2000) describe the apparent eradication of an introduced parasitic sabellid polychaete from an intertidal site in California by removing large numbers of its preferred host.

Fishing out a parasite, however, might not always be effective. For example, crab fisheries are often managed to protect crab brood stock by releasing trapped females. This inadvertently protects some types of parasites, causing such parasites to have a greater than expected effect on the crab population (Kuris & Lafferty 1992).

The feasibility of fishing out pathogens and the influence of reserves on this management strategy will also depend on the life history of both hosts and pathogens. Many pathogens infect a specific life stage of the host. For example, certain viral and bacterial pathogens of salmon (Austin & Austin 1987; Bakke & Harris 1998) as well as nematode and copepod parasites of cod (Platt 1975), and the *Rickettsia*-like prokaryote that infects abalone (Friedman *et al*. 2002), all reduce adult survival. In contrast, iridovirus of sturgeon (Georgiadis *et al*. 2001) and helminth parasites of several reef fishes infect juveniles, while nemertean parasites destroy the eggs of king and Dungeness crabs (Wickham & Kuris 1985; Kuris *et al*. 1991). In many fish species, dispersal and migration are characteristic of adult stages, whereas larvae are responsible for long-distance movement in many invertebrates (e.g. abalone, king crabs, Dungeness crabs), as well as a number of reef fishes (Strathmann *et al*. 2002).

We hypothesize that the presence of parasites and pathogens may dampen the efficacy of a reserve and that the impact of a disease will depend on reserve size and host and pathogen movement rates. Our goal is to examine life-history effects of both host and pathogen species on reserve efficacy to identify when disease will be important in marine reserves. We describe the possible impact of reserves on pathogen transmission for epidemic infections occurring for microparasites, with abalone as a case study. Finally, we suggest areas in which studies of pathogens may advance current theory and provide practical considerations about managing disease in marine reserve management.

Methods

STUDY SYSTEM

As a case study, we consider the dynamics of microparasitic pathogens on the efficacy of marine protected areas using parameters from the abalone and *Rickettsia*-like prokaryote host–pathogen system. Microparasites, such as viral, bacterial, fungal and protozoan parasites, multiply within infected hosts and may produce an immune response in the host. Microparasite models generally divide the host population into susceptible, infected and immune classes of hosts (Anderson & May 1979). In contrast, macroparasites such as helminths must leave the individual host at some point in their life cycle. The frequency distribution of parasites among hosts is of central importance, and a different modelling structure is therefore needed (Anderson & May 1978). Dobson & May (1987) catalogue the effects of a variety of macro- and microparasites on several fish species of economic importance, and suggest that microparasites are generally more important in causing increased mortality rates and reductions in fecundity, in contrast to macroparasites, which may impact more frequently the growth rate or marketability of the fish.

Abalone fisheries are in decline world-wide (Shepherd & Brown 1993; Tegner, Basch & Dayton 1996; Rogers-Bennett *et al*. 2002). In southern California, spatial depletion has occurred for several abalone species. This depletion has been so severe that white abalone *Haliotis sorenseni* now face extinction (Tegner *et al*. 1996) and have been designated as an endangered species under the US Endangered Species Act of 1973 (Hobday, Tegner & Haaker 2001).

One strategy for recovering white abalone is the aggregation of broodstock within closed marine protected areas (Tegner 1993; Rogers-Bennett *et al*. 2002). Increased aggregation is desirable because fertilization is external (McShane 1995) and may be reduced at low densities (Babcock & Keesing 1999). Rogers-Bennett *et al*. (2002) used spatially explicit data to examine potential egg production of abalone inside and outside MPAs for abalone restoration. They used abundance and size information to calculate egg production and compared estimates inside and outside of reserves for pink abalone. While these studies suggest collectively that marine reserves may be a relevant management tool for recovering abalone, increased aggregation may increase vulnerability to disease.

Withering syndrome in abalone was first detected in the mid-1980s and has been shown to significantly affect US west coast abalone species, especially black abalone *Haliotis cracherodii* (Haaker *et al*. 1992; Steinbeck *et al*. 1992). The bacterium is an emergent disease of unknown origin and an obligate parasite that is difficult to diagnose visually before actually seeing the withering foot. Little is known about the bacteria's distribution (vertical or horizontal) in the water column, its depth range or survival. The bacterium is transmitted by the Rickettsiales-like prokaryote (RLP), infects the post-oesophageal region and is excreted through faeces (Friedman *et al*. 2002). The disease causes changes in the digestive gland and ultimately the foot withers, generally causing mortality within 1 month of the appearance of symptoms. Although evidence suggests that withering syndrome may also affect white abalone,

whether the disease has been a major factor in the decline of the species is unknown.

OBJECTIVES, CONTROL VARIABLES AND RESPONSE VARIABLES

Any approach to understanding the utility of reserves has to be developed in the context of the objective of the reserve (Agardy 1994). Possible objectives that could be assessed through modelling are: to maximize population abundance, to minimize rate of population decline or to minimize the probability that the population falls below some quasi-extinction threshold. In order to compare our results to existing theoretical results, we focus on both fishery objectives (i.e. change in yield) and conservation objectives (i.e. change in population density).

To be useful, models must include control variables that we actually have the potential to influence through management or policy actions. The choice of a particular control variable depends on the objective of the model and will fundamentally influence results and recommendations. Here, the control variables we investigate are: (1) percentage of habitat inside reserve, (2) fishing intensity and (3) reserve configuration.

FORMULATING THE MICROPARASITE MODEL

We commenced with a simple two-patch, density-dependent marine reserve model (see Gerber, Kareiva & Bascompte 2002). While this model was a discrete time model of a population with non-overlapping generations, the very different time scales on which host and pathogen dynamics are likely to operate mean that the system is more tractable reformulated in continuous time. The habitat is divided into reserve (proportion ρ) and non-reserve ($1-\rho$). The host larvae produced in both reserve and unprotected areas migrate into a common pool L. Resource limitation acts to decrease larval production following a Ricker-type density-dependent relationship, with a parameter K. Larvae in the pool mix in the plankton where they die at a rate μ and mature at a rate m, after which they recruit back to both the protected and unprotected areas. The fraction of the common pool recruiting back to each zone depends on the zone's relative area. The common pool model does not mean that all larvae disperse long distances, but it assumes that that the primary mechanism of host movement is larval dispersal. This is a common assumption in marine models (e.g. Holland & Brazee 1996; Gerber *et al*. 2002). Adult hosts may also move at an instantaneous rate γ.

For our case study, we assume that the population is affected by a microparasite, which is carried only by postsettlement (hereafter 'adult') stages (as is *Rickettsia* in abalone). Accordingly, the adult host population is divided into susceptible, infected and resistant classes $S_r, I_r, R_r, S_s, I_s, R_s$, where subscripts r and s designate reserve and stock subpopulations. The disease-free natural mortality rate of the adult hosts μ_H is incremented in infected adults by an amount α. The pathogen may also decrease the rate of larval production from infected adults by a proportion δ. Adult hosts recover from infection at a rate ν, after which they remain resistant for the remainder of their life. In the part of the habitat not in the reserve, adult mortality is further incremented by fishing mortality f, which we assume is proportional to fishing effort. Pathogen transmission occurs through a mass action contact between susceptible hosts and free-living transmission stages T, at a rate with coefficient β. We assume that transmission is proportional to the product of transmission stage density and local susceptible host densities (density dependence). We therefore scale transmission of the pathogen in the reserve by dividing numbers of both infective stages and hosts by the proportion of habitat in the reserve ρ, and scale transmission in the stock similarly by $(1-\rho)$. In addition, β is not a dimensionless quantity: it has units of area/unit time. We must therefore also scale the transmission parameter in proportion to the proportion of the total area occupied by the reserve or stock. The final result of these scaling operations is that transmission in the stock is represented by $\beta T_s S_s/(1-\rho)$, and transmission in the reserve is represented as $\beta I/\rho$.

As with the hosts, the pool of transmission stages is divided between reserve and stock areas, with transfer possible between them at a rate $\varepsilon\gamma$. Transmission stages are produced at a rate λ from infected hosts, and have a death rate μ_T. The following equations then represent the dynamics of the system:

$$\frac{dL}{dt} = b(H_r - \delta I_r)\exp\left(\frac{-H_r}{\rho K}\right) + b(H_s - \delta I_s)\exp\left(\frac{-H_s}{(1-\rho)K}\right) - (m+\mu)L \quad \text{eqn 1}$$

$$\frac{dS_s}{dt} = (1-\rho)mL - (f+\mu_H)S_s + \gamma(1-\rho)S_r - \gamma\rho S_s - \frac{\beta T_s S_s}{(1-\rho)} \quad \text{eqn 2}$$

$$\frac{dI_S}{dt} = \frac{\beta T_s S_s}{(1-\rho)} - (f+\mu_H+\alpha)I_s + \gamma(1-\rho)I_r - \gamma\rho I_s - \nu I_s \quad \text{eqn 3}$$

$$\frac{dR_s}{dt} = \nu I_s - (f+\mu_H)R_s + \gamma(1-\rho)R_r - \gamma\rho R_s \quad \text{eqn 4}$$

$$\frac{dT_s}{dt} = \lambda I_s + (1-\rho)\gamma\varepsilon T_r - \rho\gamma\varepsilon T_s - \frac{\beta T_s S_s}{(1-\rho)} - \mu_T T_s \quad \text{eqn 5}$$

$$\frac{dS_r}{dt} = \rho mL - \mu_H S_r - \gamma(1-\rho)S_r + \gamma\rho S_s - \frac{\beta T_r S_r}{\rho} \quad \text{eqn 6}$$

$$\frac{dI_r}{dt} = \frac{\beta T_r S_r}{\rho} - (\mu_H+\alpha)I_r - \gamma(1-\rho)I_r + \gamma\rho I_s - \nu I_r \quad \text{eqn 7}$$

$$\frac{dR_r}{dt} = \nu I_r - \mu_H R_s - \gamma(1-\rho)R_r + \gamma\rho R_s \quad \text{eqn 8}$$

$$\frac{dT_r}{dt} = \lambda I_r - (1-\rho)\gamma\varepsilon T_r + \rho\gamma\varepsilon T_s - \frac{\beta T_r S_r}{\rho} - \mu_T T_r \quad \text{eqn 9}$$

Table 2a. Variables for abalone–*Rickettsia* model (eqns 1–9)

Variable	Definition
L	Larvae (common pool)
H_s	Host population out of reserve (i.e. stock) $I_s + S_s + R_s = H_s$
H_r	Host population within reserve $I_r + S_r + R_r = H_r$
T_s	Infective stages out of reserve
T_r	Infective stages in reserve
I_s	Infected hosts out of reserve
S_s	Susceptible hosts out of reserve
I_r	Infected hosts in reserve
S_r	Susceptible hosts in reserve
R_s	Recovered hosts outside reserve
R_r	Recovered hosts in reserve

Table 2b. Parameters for abalone–*Rickettsia* model (eqns 1–9)

Parameter	Definition
ρ	Proportion of habitat inside reserve
f	Fishing mortality
b	Base rate of larval production
K	Density dependence parameter for larval production
m	Maturation rate of larvae
μ	Death rate of larvae
μ_H	Death rate of hosts
γ	Rate of host adult movement
ν	Rate of host recovery from infection
α	Increase in mortality of infected hosts relative to uninfected hosts
δ	Decrease in host reproduction of infected hosts relative to uninfected hosts
β	Coefficient of disease transmission
ε	Transfer of transmission stages between stock and reserve areas
λ	Rate of production of transmission stages (T) in infected hosts
μ_T	Death rate of transmission stages (T)

Parameters definitions and values are defined in Table 2. Our analyses are restricted to parameter values within the stable domain, so that results are independent of initial conditions.

ESTIMATING R_0

If we assume that the transmission stages T_r and T_s have very short life spans relative to the hosts (i.e. $\mu_T >> \mu_H$), it is possible to derive an algebraic expression for the overall R_0 from the eigenvalue approach described by Dobson & Foufopoulos (2001). However, the expression is too complex to be readily interpretable. In the special case where transmission within reserves and within the fished area is much more important than transmission between reserves and the fished area, we can derive separate simple approximations to R_0 within the fished and reserve areas:

$$R_{0s} = \frac{\dfrac{\beta\lambda}{\mu_T(1-\rho)}H_s^*}{(f + \mu_H + \alpha)} \qquad \text{eqn 10}$$

and

$$R_{0r} = \frac{\dfrac{\beta\lambda}{\mu_T\rho}H_r^*}{(\mu_H + \alpha)} \qquad \text{eqn 11}$$

Here, H_s^* and H_r^* are the equilibrium host sizes in the stock and reserve in the absence of infection. Clearly, as population densities are higher in the reserve than the fished area, and there is the additional fishing mortality term in the denominator of eqn 10, it is possible that in some circumstances $R_0 > 1$ in the reserve, but not in the stock, in which case the reserve will act as a source, and the stock as a sink.

Results

Figures 1 and 2 show numerical solutions of eqns 1–9 for parameter values appropriate for the *Rickettsia*–abalone system (see Table 3). In interpreting our results, we emphasize that this is intended as a generic model to illustrate some of the issues raised by pathogens in marine reserves, and is not intended as a specific model for the management of any particular abalone population affected by *Rickettsia*. We show equilibrium solutions for yield and disease prevalence, together with the equilibrium yield that would be obtained if the pathogen were not present.

Figure 1 shows equilibrium yield and prevalence as a function of three potential control variables: the amount of habitat in the reserve, the fishing mortality in the fished area and the rate of interchange between stock and reserve. This last quantity can be thought of as a control variable, contrasting dividing the habitat into one large reserve and one stock area (small amount of interchange), and many small reserve and stock areas (large amount of interchange). Several general points of interest emerge from this figure.

First, because we have modelled transmission as being density dependent, it is possible to 'fish out' the pathogen, reducing the host density in the stock to below the threshold for disease maintenance, provided there is no (or very little) habitat as reserve (see the results in the first column). For this particular set of parameter values, a fishing mortality of approximately 1·0 y^{-1} is sufficient, which corresponds closely to the effort that generates MSY, but this will not be the case in general. As might be expected, fishing out the pathogen is not possible when a reserve exists, and fishing has a diminishing effect on pathogen prevalence as either the amount of habitat in reserve is increased, or as the number of reserve patches is increased. Fishing has some, but limited, effect on the prevalence of the pathogen in the reserve.

Secondly, the presence of the pathogen causes the yield curve to be shifted to the right, meaning that maximum sustainable yield (MSY) is reached at higher fishing mortality than is the case in the absence of infection. The reason for this is straightforward: fishing reduces host density and thus disease prevalence and hence disease-induced mortality, causing a compensatory

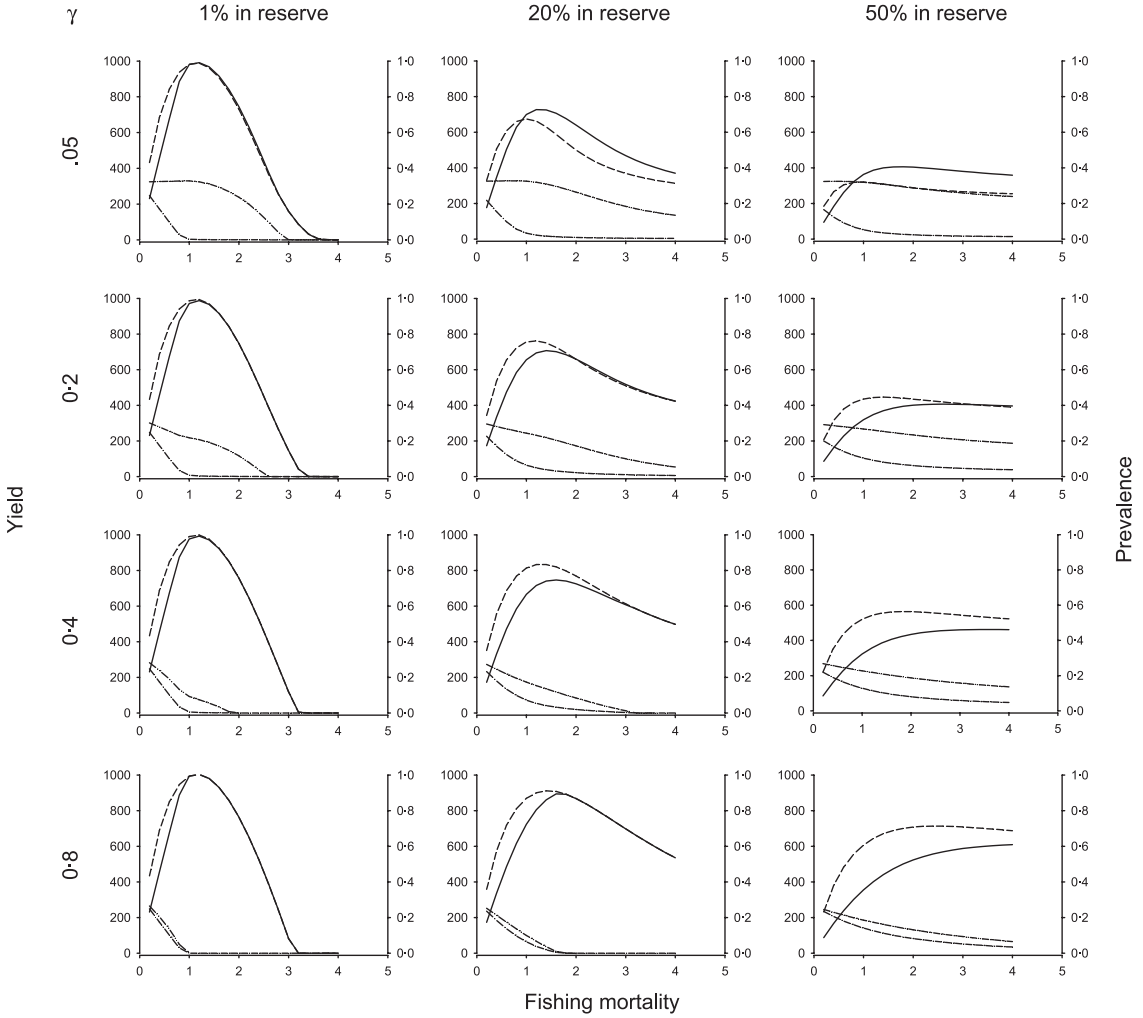

Fig. 1. Equilibrium solutions of the system described by eqns 1–9, as a function of fishing mortality (horizontal axis), % of the habitat in reserves (columns) and the rate of host movement between stock and reserve γ (rows). Yield with pathogens present is shown with a solid line; yield with no pathogens is shown with a dashed line; prevalence of infection in the stock is shown with a dot-dashed line; and prevalence of infection in the reserve is shown with a dot-dot dashed line. Parameter values other than those varied in the figure are: $b = 12{\cdot}25$; $K = 1000$; $m = 36{\cdot}5$; $\delta = 0{\cdot}5$; $\mu = 109{\cdot}5$; $\mu_H = 0{\cdot}15$; $\beta = 0{\cdot}002$; $\alpha = 2{\cdot}0$; $\nu = 0$; $\lambda = 100$; $\mu_T = 73$; $\varepsilon = 200$.

increase in the host rate of increase. The extent of the rightward shift of the yield curve is increased as the amount of habitat in the reserve is increased.

The most counterintuitive result is the difference between yields at a given fishing mortality, with and without the pathogen present. One would expect the pathogen, as an additional mortality source, to decrease yield. This is the case in general, particularly when there are high rates of interchange between the stock and the reserve. However, when the rate of interchange between reserve and stock is low (row 1 of Fig. 1), for some effort values the sustained yield is greater if the pathogen is present than if it is absent. In fact, the panel in the top right represents a case in which the maximum sustained yield with the pathogen present exceeds the maximum sustained yield that is possible when the pathogen is absent. This unusual result is probably a consequence of the over-compensatory nature of the Ricker density dependence function, and the fact that in this particular parameter set infection increases mortality, but does not decrease fecundity. With low rates of transfer of infection or adults between stock and reserve, but with complete larval mixing, the pathogen will reduce the adult population in the reserve which, over certain density ranges, will increase larval output.

Figure 2 shows the same variables as a function of the transmission rate β and the amount of habitat in reserve, for a moderate amount of interchange between stock and reserve. When exploiting a fishery affected by a parasite, a key issue is the relationship between the threshold host density for disease transmission and the host density at which MSY would be attained in the absence of infection. The top row of Fig. 2 shows a situation in which β is sufficiently small that the pathogen can effectively be removed from the stock by fishing mortality well below that necessary to maximize yield without infection (note that the prevalence in the stock declines to almost 0 for fishing mortality more than 1 in the first panel). This means that the presence of the pathogen in the system has very little effect on the

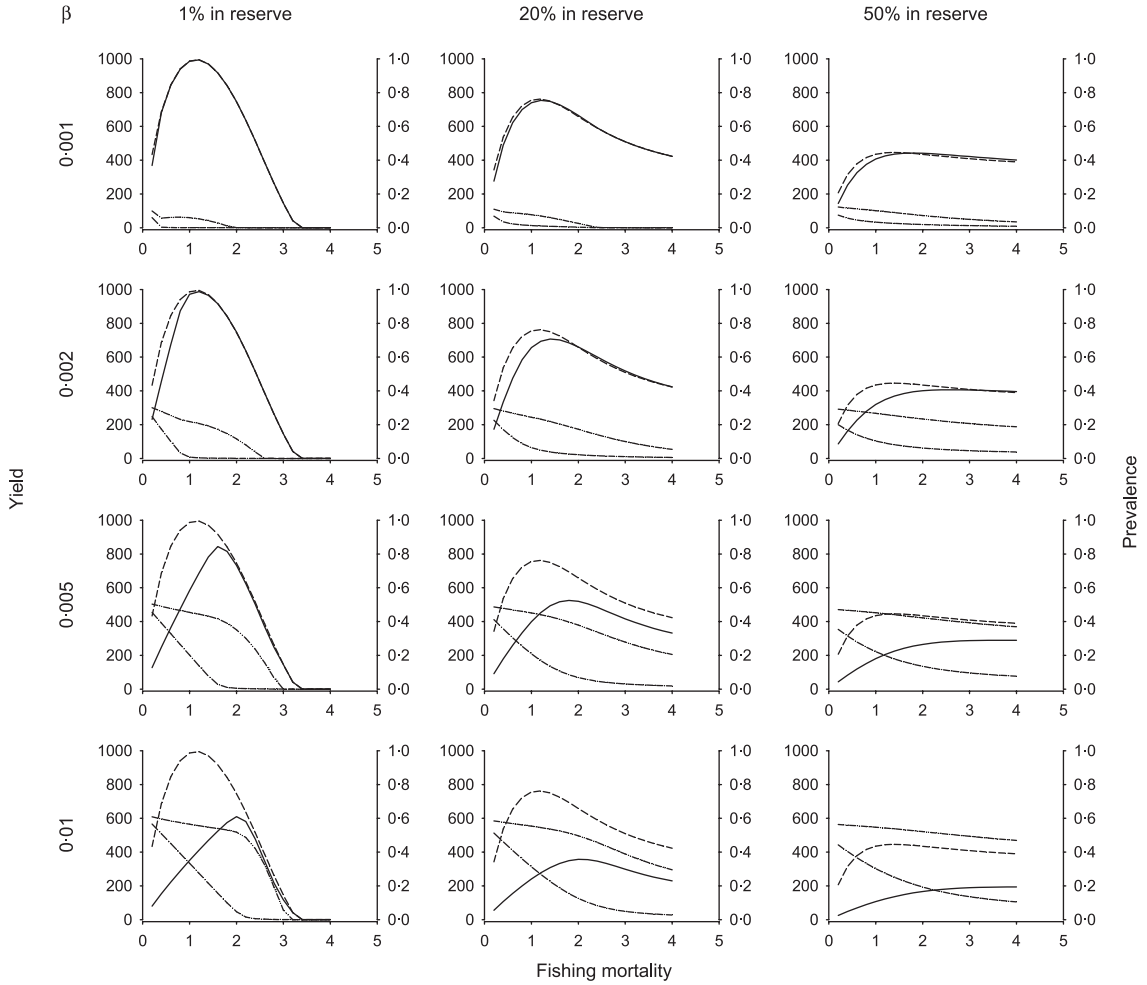

Fig. 2. Equilibrium solutions of the system described by eqns 1–9, as a function of fishing mortality (horizontal axis), % of the habitat in reserves (columns) and the transmission rate β (rows). Yield with pathogens present is shown with a solid line; yield with no pathogens is shown with a dashed line; prevalence of infection in the stock is shown with a dot-dashed line; and prevalence of infection in the reserve is shown with a dot-dot dashed line. Parameter values other than those varied in the figure are: $b = 12{\cdot}25$; $K = 1000$; $m = 36{\cdot}5$; $\delta = 0{\cdot}5$; $\mu = 109{\cdot}5$; $\mu_H = 0{\cdot}15$; $\gamma = 0{\cdot}2$; $\alpha = 2{\cdot}0$; $\nu = 0$; $\lambda = 100$; $\mu_T = 73$; $\varepsilon = 200$.

MSY. However, as β is increased, the fishing mortality necessary to eliminate the pathogen eventually exceeds the fishing mortality that produces MSY. This causes a progressive steepening of the declining part of the effort–yield curve (see the first panel in the last row of Fig. 2). Increasing fishing mortality is initially compensated for by decreasing disease induced mortality, but once the pathogen is eliminated the compensation ceases, and increasing fishing mortality leads to a rapid decline in the stock. The effect is that a relatively small increase in effort beyond that which produces MSY will lead to the total collapse of the fishery.

Adding a reserve to each of these scenarios decreases yield, but also markedly decreases the sensitivity of the system to overharvesting. The effort–yield curve becomes more symmetrical, and this is particularly evident in cases where the transmission rate of the pathogen is high.

The transmission parameter β is difficult to measure directly in any host–pathogen system (McCallum 2000). However, assuming density dependent transmission and equilibrium, there is a simple relationship between the basic reproductive number R_0 and the prevalence of infection:

$$R_0 = 1/(S^*/N) \qquad \text{eqn 12}$$

where S^*/N is the equilibrium proportion of susceptible hosts. Further, R_0 is proportional to the population size N. If a microparasite is present at high prevalence in an unexploited population, this indicates that R_0 is high. Given the proportion of susceptible hosts at steady state and estimates of the other parameters, β can be estimated from eqn 10. If independent evidence also shows that the microparasite substantially increases host mortality, then one should be alert to the possibility that the fishery may be prone to sudden collapse as effort increases, as shown in the first panel in the last row of Fig. 2.

Discussion

This study represents the first theoretical treatment in the literature of host–pathogen interactions on marine

Table 3. Estimates for life-history traits used in determining parameter estimates for the abalone–*Rickettsia* model. Most life-history traits in the literature are described as durations, and have been inverted and converted to units of y^{-1} to produce parameter estimates for the model

Trait	Estimate	Rate parameter	Estimated value (y^{-1})
Baseline reproductive rate	12·25[1]	b	12·25
Maturation period of of larvae	9–10 days[2]	m	36·5
Death rate of uninfected hosts	0·15[3]	μ_H	0·15
Mean survival of abalone after infection	6 months[4]	α	2·0
Survival time of transmission stages	5 days[5]	μ_T	73·0
Abalone adult movement	< 10 m[6]		
Development of resistance	0	ν	0·0[7]

[1]Hobday & Tegner (2000) cite fertilization success rates of 48% and 20% for abalone spaced 2 m and 8 m apart, respectively. White abalone density is estimated at 0·002 m^{-2} and is thought to be at densities too low for effective fertilization (Davis, Haaker & Richards 1996). We assumed the following values based on: $4{\cdot}9 \times 10^6$ eggs spawned per female per year; 0·5 for proportion females in abalone population; 0·01% for fertilization success of spawned eggs (Tutschulte & Connell 1981); and 5% for settlement success of fertilized eggs: 5% (Hobday & Tegner 2000), resulting in an estimate of 12·25 for baseline reproductive rate.
[2]Maturation period of larvae according to Hobday & Tegner (2000).
[3]We estimate death rate of hosts based on data for red abalone: mortality estimate, M = 0·15 (Tegner, Breen & Lennert 1989).
[4]Mortality of abalone is estimated to occur 5–7 months after infection with RLP (Carolyn Friedman, personal communication). From this, we calculate $\alpha = 1/0{\cdot}5 = 2$.
[5]Maximum survival time of RLP in seawater has not been empirically determined, but is estimated to be 1 week or longer (Carolyn Friedman, personal communication), but the mean survival will be somewhat less than this (we have estimated it as 5 days).
[6]Juvenile abalone may move tens of metres, and adults are generally more sedentary (Hobday & Tegner 2000). From this, we estimate that adults move less than 10 m.
[7]There is no evidence for development of acquired resistance to *Rickettsia* in abalone.

reserves. In one of the few empirical studies of the effects of marine reserves on parasites, the most abundant and least host-specific monogean in the system (*Lamellodiscus elegans*) was found to be about twice as abundant on white sea bream *Diplodus sargus* inside the reserve as it was outside the reserve (Sasal *et al*. 2004). Although that study was of a macroparasite community, rather than the microparasite system we have modelled here, it does provide evidence to support our key assumption that the higher host densities within reserves will support higher parasite infection levels.

Our model assumes that the rate of pathogen transmission, both inside and outside reserves, is proportional to the respective average host density. If hosts were highly aggregated and transmission occurred primarily on a local scale, then it might be possible for high rates of transmission to be maintained in harvested populations, even though host density overall was reduced. Harvesting affects aggregation, as well as average density. In the case of abalone, in which fishers target aggregations, harvesting decreases the degree of aggregation (Dowling, Hall & McGarvey 2004). One might therefore expect even greater differences in parasite transmission rates between fished and unfished populations than we have assumed in our model. Existence of unharvested reservoir species (whether these exist for withering syndrome is unknown) would decrease any differences in transmission inside and outside reserves.

To date, modelling studies on marine reserves have addressed two questions relevant to the potential effects of reserves for fisheries: (1) can reserves maximize yield and/or spawning stock biomass; and (2) can reserves minimize the effects of uncertainty, such as variability in yield and likelihood of extinction? In this paper we show that the presence of a pathogen may influence the answer to both of these questions. For example, establishing a reserve system has the effect of increasing the population density of the harvested species within the reserve relative to the population density before the reserve was established. Similarly, once the reserve has been established, the density of the harvested species in the reserve will exceed that outside the reserve. Unless pathogens are transmitted in a frequency-dependent fashion, there is a threshold host population density N_T, below which the disease cannot persist. The most obvious potential effect of a reserve system on a pathogen is therefore that the host density may be above the threshold for disease persistence N_T in the reserves, but may have been below it in the fished population before the reserve was established. This means that establishing a reserve system may introduce a pathogen problem into a system from which it has previously been absent.

The problem of reserves leading to disease emergence is unlikely to be so serious as to preclude establishing reserves, for several reasons. First, the mean population density in reserves will not usually exceed the mean density in the pristine state, before the fishery was established. We should therefore not anticipate that reserves should cause the emergence of a novel parasite, although they may permit the re-emergence of a previously existing parasite or the establishment of an exotic parasite that had failed to establish while the fishery was in operation. Secondly, given that the host

equilibrium density maintained by a pathogen always exceeds the threshold for its introduction, an endemic disease problem resulting from establishing a reserve system will not cause the host population inside the reserve to be reduced below the mean density that existed before the reserve was established, nor below that outside the reserves. This means that if the goal of the reserve is the conservation of the host population a disease may reduce, but not eliminate, the benefit of the reserve system. However, if the purpose of the reserve is fisheries management and the pathogen has impacts on the host other than by reducing population density (such as by reducing the marketability of the resource), then spillover from a reserve into the harvestable stock may be of significant concern.

We emphasize that this is not a complete treatment of the impact of pathogens on marine reserves. We have considered only microparasitic infections, and while our model allows for the possibility of effects on fecundity, we have explored only parameter sets in which infection influences mortality alone. Many parasites of commercial importance in fisheries are macroparasites (see Table 1). Investigation of the effect of reserves on marcroparasites requires a different model structure that we could not include here. Parasites of commercial crustaceans are frequently castrators (Kuris & Lafferty 1992), and may even increase survival while eliminating reproductive output. Parasitic castrators themselves are usually crustaceans, and as such are macroparasites in terms of size. However, it is possible to understand their dynamics using a 'microparasite' framework, in which hosts are represented as 'susceptible' or 'infected', because one parasite is sufficient to castrate its host, so that the parasite burden per host is not important (Kuris & Lafferty 1992). There is no resistant class. Our framework can therefore be adapted easily to deal with parasitic castrators.

We have dealt only with an equilibrium analysis of a deterministic model. This means that our results help to understand the impact of endemic disease on sustained yield, but do not deal directly with the questions of epidemic disease, variability in yield or extinction risk, although we can certainly make some inferences about such problems. For example, the very steep decline in yield with increasing effort beyond MSY in the first panel of the last row of Fig. 2 shows that the presence of a highly transmissible pathogen in a system without a reserve may make the population very susceptible to extinction through overharvesting. Addition of a reserve to the system does decrease the MSY, but makes the system much more resistant to overharvesting.

What can we recommend to managers who wish to set up a reserve in a situation where a pathogen is known to be significant in the fishery? First, and most fundamentally, consideration of pathogens is important when setting up reserves. Pathogens shift yield–effort curves to the right, meaning that MSY is reached with higher efforts than will be the case in the absence of infection. Establishing a reserve makes the system more resistant to overharvesting, whether or not a pathogen is present, but because fisheries impacted by highly transmissible pathogens are particularly prone to overharvesting reserves are more important in such cases. A reserve does make it more difficult to 'fish out' a pathogen, a management option that may be attractive if pathogen dispersal is more limited than that of host larvae. However, it is not always the case that establishing a reserve decreases yield more if a pathogen is present than if a pathogen is absent. In some cases, the combination of a pathogen and reserve may even produce higher yields than would be obtained by the reserve in the absence of the pathogen. The potential range of results illustrated in Figs 1 and 2 emphasizes that it will be necessary to examine the joint effects of pathogens and reserves on a case-by-case basis, with appropriate parameter estimates for the specific situation.

Our initial results suggest that there is clearly potential for fruitful integration between the fields of epidemiology and marine reserve theory. Most empirical evidence for reserve performance is based on studies from within the boundaries of a single reserve, often focused on the response of a single species. Consequently, predictions about the potential for marine reserves to enhance fisheries are based principally on insights from ecological theory. The most recent synthesis of theoretical studies (Gerber *et al*. 2003) concluded that reserves can increase yield when fishing effort is sufficiently high that recruitment would otherwise decline without reserves (i.e. when populations are recruitment overfished). Secondly, this review concluded that reserves should provide fewer benefits to species with greater rates of movement, particularly as adults. Our results suggest that inclusion of a pathogen may change these theoretical predictions. For example, yield may actually be higher for a smaller reserve in the presence of a pathogen, and reserves are most effective for moderate rates of movement (Fig. 1). As a first exploration of the effects of disease, our study does not consider other factors known to affect marine efficacy, and as such is not meant to be applied directly to reserve design. However, our model results represent a useful first step to considering the extent to which host–pathogen dynamics might extend the theoretical predictions of marine reserves. With this work as a basis, we plan to continue with empirical and theoretical investigations with the goal of improving our understanding of the role of pathogens in the efficacy of marine reserves.

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