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Microparasitic disease dynamics in benthic suspension feeders: infective dose, non-focal hosts, and particle diffusion

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Abstract

Benthic suspension-feeders can accumulate substantial numbers of microparasitic pathogens by contacting or filtering particles while feeding, thus making them highly vulnerable to infectious diseases. The study of disease dynamics in these marine organisms requires an innovative approach to modeling. To do so, we developed a single-population deterministic compartmental model adapted from the mathematical theory of epidemics. The model is a continuous-time model, unstructured in spatial or age terms, and configured to simulate the dynamics of diverse dose (body burden)–dependent infectious disease transmission processes in suspension feeders caused by susceptible individuals contacting or absorbing (filtering) infectious waterborne pathogens. Different scenarios were simulated to explore the effect of recruitment, filtration rate, particle loss, diffusion-like processes in the water column and non-focal hosts (i.e. non-susceptible in terms of disease) on disease incidence. An increase in recruitment (i.e. new disease free susceptibles) can reduce the prevalence of infection due to the dilution effect of adding more susceptibles, but the disease can spread faster for the same reason. Lower infective particle accumulation rates or increasing particle loss rates in the environment reduce the prevalence of infection. This effect is trivial when the water is saturated with infective particles released by infected and/or dead animals. Diffusion of particles from the local pool available to suspension feeders to the adjacent remote pool, prompted by a large remote volume and high particle exchange, limits epizootic development. Similarly, the likelihood of an epizootic can be constrained in a large susceptible population when competition for pathogens, more 'active' in active filter feeders than in passive suspension feeders, reduces the per capita infective particle accumulation rate. In passive suspension feeders, decreasing the area of the feeding surface has the same effect in constraining disease development. The effect of competition for infective particles in essence diluting the infective particle concentration in the water column is magnified when the susceptible population is part of a community with non-focal filter feeders, and is particularly effective in limiting disease development in high infective dose systems. At the same time, this active foraging strategy makes filter feeders more vulnerable to epizootics. The model is a suitable framework for studying the disease dynamics and determinants of disease outbreaks in benthic suspension feeders.

Keywords:
disease model, epizootic, transmission, suspension feeders, filter feeders, infective-dose, overfiltration, non–focal host

1. Introduction

Benthic suspension-feeders are among the optimal foragers in the marine context because the energetic cost for capturing prey is almost nil in passive sessile animals and very low in active filter feeders (Riisgård et al., 1993; Riisgård and Larsen, 1995). Suspension feeders play a major role in the structure and function of marine ecosystems (Dame, 1993; Newell, 2004) by transferring energy from the pelagic zone to the benthos (Newell, 2004; Porter et al., 2004). This feeding mode as a foraging strategy has a downside in terms of the transmission and spread of waterborne diseases. In addition to food particles, suspension-feeders can also accumulate a substantial number of infectious pathogens such as bacteria, fungi, protozoans, and viruses from the water. For instance, the scleractinian coral *Madracis mirabilis* can accumulate 1 x 10^7 bacterial cells cm^{-2} h^{-1} and clearance rates in bivalves can be much higher (e.g., 8 L h^{-1} in oysters, Powell et al. (1992)). This potential to accumulate particles, in turn can catalyze major epizootics and massive mortalities causing substantial changes to ecosystem structure and serious losses in shellfish-eries and aquaculture (Lafferty et al., 2015). Among the best characterized, geographically wide-spread, and virulent infectious diseases in suspension feeders are MSX and Dermo in oysters (Villalba et al., 2004), white plague disease and black band disease (BBD) in corals (Sokolow et al., 2009; Zvuloni et al., 2009), and brown ring disease BRD, and Perkinsosis in clams (Paillard, 2004; Dang et al., 2010).

Proliferation–based disease models have been considered sufficient to describe disease impact in benthic suspension feeder populations characterized by rapid non–point–source transmission, such as bivalves (Calvo et al., 2001; Powell et al., 2011, 2012b; Powell and Hofmann, 2015). Host proliferation models alone or together with hydrodynamic models can explore the effect of environmental factors, such as temperature and salinity, on the process of pathogen proliferation and, consequently, on the infection intensity. These models assume high prevalence and simulate epizootic development and host morbidity and mortality based upon population infection intensity as the pathogen proliferates within the host. For these diseases the dynamics of transmission are typically poorly described (Ford, 1992; Ford and Smolowitz, 2007; Gray et al., 2009) and cursorily integrated into disease models (Powell et al., 1996, 1999). Only a few studies have adapted the epidemic *SIR* models (Kermack and McKendrick, 1927; Anderson and May, 1981) to benthic marine organisms, despite the fact that the structure of these models as commonly used for terrestrial diseases is equally applicable and adaptable to describe the epizootiology in benthic animals (McCallum et al., 2004). For this purpose, however, some marine system-specific features such as the buoyancy and the high potential of dispersion and dilution of waterborne pathogens (Strathmann, 1990; McCallum et al., 2003) and certain unique feeding modes such as suspension feeding may need to be incorporated. Kuris and Lafferty (1992) developed a model incorporating both recruitment of the parasite and the host to compare the effect of various management strategies on a hypothetical crustacean parasitized by a parasitic castrator. In this non-point-source host-pathogen system, the number of nearby infected animals is relatively unimportant in comparison with the number of infective pathogens in the water. McCallum et al. (2005) modeled the dynamics of withering syndrome in abalones by incorporating the free-living pathogen stage and disease transmission through contact between this stage and the host. More recently, Sokolow et al. (2009) and Yakob and Mumby (2011) formulated the dynamics of disease in corals describing the transmission of disease by contact between the host and free-living pathogens. (Bidegain et al., 2016)formulated simple compartmental models to yield the basic reproduction number R_0 for a variety of marine host-pathogen systems to explore the relative importance of the host and pathogen traits that determine transmission.

Most compartmental disease models (e.g., SI, SIR, SEIR, etc.) follow the classic mass action approach where disease transmission is a function of the number of contacts between susceptible individuals and infective particles in the water (Regoes et al., 2002; Sokolow et al., 2009; Yakob and Mumby, 2011). However, it may be important to include the well-known dose-effect in disease transmission for some suspension feeders such as oysters (Bushek et al., 1997; Ford et al., 1999; Powell et al., 1999); effective dose is a characteristic with potentially interesting effects on disease transmission that seems crucial to take into account. Active filter feeders can be sufficiently dense to compete for food (e.g., Fréchette et al., 1992; Wilson–Ormond et al., 1997; Widdows et al., 2002) particularly under conditions of slow flow and limited vertical advection. Similarly, they may ‘compete’ for pathogens, presumably reducing the concentration of infective particles sufficiently to limit body burden below the infective dose and, in turn, limiting epizootic development (i.e. the overfiltration scenario – Bidegain et al. in press). Passive filter feeders may show this mechanism less obviously since they more importantly depend on the ambient flow and the body surface exposed to that flow, rather than on an active pumping of water from the environment through a filter (Sebens et al., 1996, 1998).

When host diversity increases, the disease risk can decrease in what is known as the dilution effect. This well-studied effect in Lyme disease (Ostfeld and Keesing, 2000) appears to be a more general phenomenon that relies on the idea that a certain community with high species diversity will contain a proportion of incompetent hosts, in terms of disease, that compete for particles and deflect infectious pathogens away from the susceptible hosts, thereby reducing infection prevalence and disease risk. This marine suspension feeder community could be composed, for instance, by the scleractinian coral *Madracis mirabilis* and the colonial ascidian *Tridemnum solidun*, since data show that both organisms are effective bacterial suspension feeders (Bak et al., 1998). Other examples may by a reef composed by oysters and mussels, since mussels that attach to oyster reefs and shells can double the reef's filtration capacity (Gedan et al., 2014), or by oysters and ascidian tunicates (T. Ben-Horin, unpublished data).

Another interesting characteristic of some diseases of suspension feeders is the importance of dead infected animals as a source of infective particles and, hence, the role of mortality and subsequent scavenging and tissue decay in disease transmission. For instance, the pathogen body burden in dead oysters infected by Dermo disease is commonly higher and the potential release rate upon death is relatively much faster than that of infected live animals (Bushek et al., 2002). Dead corals infected by the black band disease may also release pathogens through breakdown of decaying tissue (Richardson, 2004). Once released, diffusion processes in the water column may have an effect in pathogen dynamics and consequently, in disease transmission. The population turnover rate (i.e. high mortality and recruitment rates) thus is another mechanism of controlling epizootics in suspension feeders. Yakob and Mumby (2011) found that allowing for a more dynamic population turnover in an epizoological model of coral disease not only gives a superior fit to empirical data, but also suggests that emerging coral assemblages could be far less prone to epizootics. The role of predation in the context of SIR models has been considered (Su and Hui, 2011; Wang et al., 2011), but has rarely been applied in the marine context (Liao et al., 2008). In filter feeders such as oysters the important contribution of dead animals releasing infective particles may counterweigh the effect of the population turnover rate as a restraining influence on epizootics (Bidegain et al., unpublished).

A theoretical transmission-based model or a host-population model that incorporates all of these features and describes the dynamics of the host in all possible stages (i.e. susceptible, live infected, dead infected) and the waterborne pathogens does not exist for active or passive suspension feeders. In this paper, we develop an SI model capable of reproducing these processes. For this purpose, we adapt the Kermack and McKendrick (1927) epidemiological theory and the microparasitic epidemic model of Anderson and May (1981). The model reproduces the dynamics of both susceptible and infective stages of the host, whether alive or dead, and the waterborne infective pathogens. Our study system includes dose-dependent (i.e. body burden dependent) disease transmission wherein suspension feeders contact or absorb (filtering) waterborne infective pathogens released by live or dead infected animals. The model contemplates the effect on disease transmission of non-point sources of pathogens and diffusion processes in the water column. Moreover, the model permits the study of potential mechanisms by which suspension feeders might dilute the disease risk, such as competitive interactions among and between species, and the effect of recruitment of new susceptible individuals. Finally, the model yields the formulation of the basic reproduction number R_0 for both active and passive filter feeders, to comprehensively describe the relative importance of some of these processes driving epizootics.

2. The model

2.1. Mathematical theory and model structure

The model is a one-population deterministic compartmental model continuous in time, unstructured in spatial or age terms (Cuddington and Beisner, 2005), and configured to simulate the dynamics of a diversity of infectious disease transmission processes in suspension feeders caused by susceptible individuals contacting or absorbing (filtering) infectious waterborne pathogens such as bacteria, fungi, protozoans, and viruses from the water column.

As a compartmental model, which is the most frequently used class of model in epidemiology (Diekmann et al., 2013), individuals and pathogens can take on a finite number of discrete states. Each state is representative of a subpopulation of individuals or pathogens at a given time (Table 1) which, in turn, together with the corresponding parameters (Table 2) satisfy a system of nonlinear ordinary differential equations (ODEs) describing the dynamics of the host-pathogen association. The incorporation of a dead infected subpopulation stems form the fact that some

suspension feeders such as oysters or corals may also transmit the disease by releasing pathogens upon death (Bushek et al., 2002; Richardson, 2004). A variant of the model incorporates another variable representing an alternate suspension feeder host population which is incompetent in terms of disease (H), that is, the alternate host does not develop the disease and pathogens are inactivated inside the animals acting as an important mechanism of controlling the concentration of infective particles in the water column and as a sink of pathogens.

The population of waterborne pathogens is divided into three classes depending upon their location in the system based on the division of the environment into two volumes of water: The first is defined as the 'local pool' of pathogens P. This pool is the pathogen concentration in the 'local volume' defined as V_l , adjacent to the bottom, within which pathogens are released by infected and dead infected individuals and remain free floating. Pathogens in this volume are susceptible to contact with or being filtered by hosts, and can lose their infective properties after some time or otherwise be lost (i.e advection, diffusion, predation). For example, for oysters, the height of the local volume or the volume available for filtration is considered to be around 10 cm (Wilson–Ormond et al., 1997). This is a typical height for an oyster clump (Powell et al., 1987, 2012a), but this height would exaggerate the effect of an infaunal filter feeder (Ertman and Jumars, 1988; Monismith et al., 1990; Widdows et al., 1998). The second state variable for pathogens is the remote pool U. This second pool is the concentration of pathogens in a second volume contiguous to the local volume, defined as the remote volume V_r, where a remote pool of particles can accumulate without direct interaction with hosts. The size of the remote volume would depend on how deep the water column is above the near-bottom layer directly affected by the filtering benthic population. Diffusional exchange of particles between these two volumes is assumed. The third subpopulation of pathogens is that accumulated in the susceptible population through contact or filtration (F).

The infection state in the model is presence/absence, that is, the animals are infected or are not, and if they are infected, they have the 'average' parasite load. We assume this because microparasites are usually (but not always) unicellular microorganisms, such as viruses, bacteria and protozoans that can multiply rapidly within a host (McCallum et al., 2004) relative to a one year time step. The transfer of individuals from absence to presence of disease is by a dose-dependent or body burden dependent transmission process detailed in section 2.4.1.

2.2. *Miscellany*

Variables related to the host population are defined with respect to the concentration of individuals in a given surface area of the bottom, whereas variables related to the pathogens are defined in number of particles in a given water volume or the number of particles *in vivo*. Note that for simplicity in rendering equations, the reciprocals of the local and remote volumes V_l and V_r, sl and sr respectively, are often used.

Variable	Definition	Unit
S	Susceptible hosts in the population	Number of individuals
I	Infected individuals in the population	Number of individuals
DI	Dead infected individuals in the population	Number of individuals
DS	Dead susceptible individuals in the population	Number of individuals
P	Free-living pathogens in the environment, local pool	Number of particles
F	Total number of pathogens absorbed or filtered by the population	Number of particles
U	Pathogens from a remote volume, remote pool	Number of particles
H	Alternate non–competent reservoir host	Number of individuals

Table 1: Variables of the model. Note that the model has an implicit surface area or volume for the host subpopulations and pathogens, respectively. F is the exception and the corresponding unit is internal particles in the susceptible population.

The mathematical theory of this model contemplates that disease transmission requires the pathogen to exist outside the host and remain infectious for sometime finite period. Thus, the susceptible and infected individuals are

not necessarily in contact or even components of the same local population, since the free-living pathogen stage can drift in the fluid and contact or be filtered by susceptible animals located near or far away from the particle source. Natural mortality of the host and mortality due to disease are also integrated into the formulation of the model. Thus, the infected individuals can die due to both natural causes and disease. The model also assumes an open population since it contemplates demographic turnover; that is, the recruitment process is integrated in the dynamics of the host. The model does not incorporate the process of recovery from disease because only a few examples of marine wild populations recovering from disease are known in marine systems (Gilmour et al., 2013; Paillard et al., 2014; Vega Thurber et al., 2014). Consequently, individuals never recover from the disease and infected individuals remain infected until they die.

2.3. Model scheme

The flow diagram of the model (Fig. 1) shows the most important processes in the disease transmission process in suspension feeders. Disease is transmitted to the susceptible population S thorough filtration (active suspension feeders) or contact (passive suspension feeders) at a rate β by a dose-dependent transmission. Infected animals I die due to both natural mortality m_S and disease mortality m_I, while susceptible individuals S only die due to natural causes. The alternate incompetent (i.e. not susceptible to the disease) suspension feeder host H competing for waterborne pathogens with the susceptible host is also located in the bottom. This alternate host only dies by natural mortality assuming that it is resistant to the disease and does not release particles to the water; that is, they are assumed to be inactivated by the immune system or by diapedesis. An exchange between the local near–bottom pathogen pool P and the remote pool U occurs at a certain exchange rate γ by a diffusion-like process proportional to the difference in concentration between the two pools.

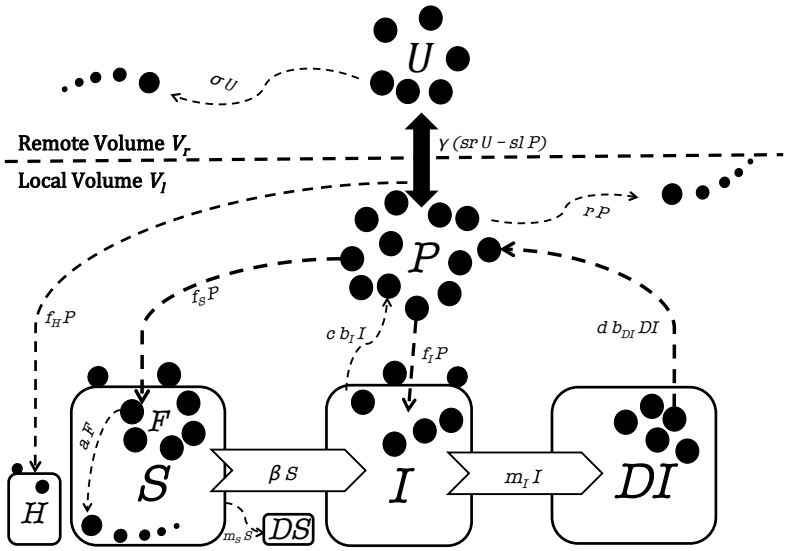

Figure 1: Model flow diagram. The model variables are represented by capital letters: susceptible animals (S), infected animals (I), dead susceptible (DS) and dead infected animals (DI), waterborne pathogens (P), internal pool of pathogens (F), remote pool of pathogens (U) and alternate host population (H). Dashed arrows represent the main processes in the model. The parameters involved in these transmission are described in Table 2.

2.4. Model equations

The equations that follow represent the disease transmission process and the model variables. The host and pathogen states or subpopulations satisfy a system of ODEs describing the dynamic of the host-pathogen association. The numerical model for this ODE system is programmed in Matlab 8.1. The set of coupled differential equations

is solved with a 4^{th}–order predictor corrector scheme using the Adams-Bashforth predictor and the Adams-Moulton corrector.

2.4.1. *Infective dose and disease transmission*

Disease transmission in suspension feeders, at least in filter feeders, most likely occurs via an infective dose (Bushek et al., 1997; Ford et al., 1999; Powell et al., 1999) rather than by unique contact between a single pathogen or fomite and host. Consequently, the transmission process in the model is based on the fact that susceptible animals require some minimum level of body burden of infectious particles, the infective dose, to become infectious. Furthermore, the model assumes that as hosts absorb or filter particles, some susceptible individuals will have a relatively large body burden whereas most will have a smaller body burden. The distribution of the number of susceptible animals (S) with each level of body burden (b) (Fig. 2) is assumed to have the form

$$S(b) = S_0\, e^{-\rho\, b}, \tag{1}$$

which has a simpler expression but similar behavior to the negative binomial distribution, very common in benthic infected hosts (Anderson and Gordon, 1982; Ford et al., 1999)), in having a long tail describing the declining frequency of individuals of increasing infection in the population distribution. S_0 is the total number of susceptible individuals. For body burdens up to several hundred, ρ is a well approximated by $\hat{S}/F$, where $\hat{S}$ is the number of susceptible animals and F is the number of infectious particles housed in the susceptible subpopulation (Eq. (2)). Thus F/S represents the average concentration of infectious particles in susceptible individuals.

$$\frac{d\,F}{d\,t} = -(\beta_F + m_S + a + c_S)\,F + f_S\,A_S\,S\,P. \tag{2}$$

Infectious particles are filtered out (active filter feeders) by or come into contact (passive filter feeders) with susceptible individuals at the filtration or contact rate f_S, being the contact rate proportional to the feeding surface A_S for passive filter feeders (see section 2.4.2 for a more detailed description of particle uptake). Infectious particles can be removed from the susceptible population by four processes: (i) the reduction of the internal pool of particles in the susceptible population due to removal of susceptible individuals that become infected t_F (see details in Eq. (6)), (ii) the background mortality m_S as it removes individuals with incorporated infected particles, (iii) the inactivation inside susceptible animals (a), controlled by some component of the immune response such as phagocytosis, inactivation by oxygen radicles, binding by lectins, etc. (Renwrantz, 1986; Villalba et al., 2004), and (iv) the release of particles from susceptibles through, for instance, faeces. Whether a susceptible becomes infected is a balance between a, the release rate of particles c_S, the filtration of particles by susceptibles $f_s\,S\,P$, and the concentration of infective particles that needs to be reached to generate the infective dose (b_{min}, see Fig. 2).

The distribution of infective particle body burden in susceptible hosts in Fig. 2 has a very long tail so that a few uninfected animals have a high body burden. The portion of the susceptible pool eligible to transition to the infected pool is circumscribed by b_{max}, the maximum body burden for any animal considered to be uninfected and b_{min}, the minimum body burden for animals eligible to transition to the infected pool. At any time, there will be some number of susceptible animals (S) with a total absorbed pool of infectious particles (F). These values are used to determine how many susceptible animals with a body burden between b_{min} and b_{max} should become infected.

The total number of susceptible animals at any time is

$$S = \int_0^{b_{max}} \hat{S}_0\, e^{-\rho\, b}\, db = \frac{\hat{S}_0}{\rho}(1 - e^{-b_{max}}) \tag{3}$$

where $\hat{S}_0$ is the number of susceptibles per body burden. The total body burden for the population is

$$F = \int_0^{b_{max}} \hat{S}_0\; b\, e^{-\rho\, b}\, db = \frac{\hat{S}_0}{\rho^2}\,(1 - (1 + \rho\, b_{max})\, e^{-\rho\, b_{max}}. \tag{4}$$

F and $\hat{S}$ are both known at any step in the model. Consequently, the values of S_0 and ρ can be calculated easily. The total number of susceptible individuals moved to the infected population is

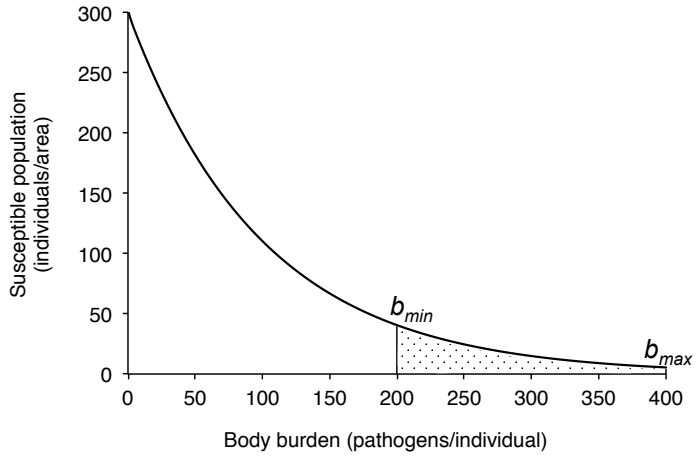

Figure 2: A theoretical distribution of the population with each level of infective particle body burden (b). The minimum body burden at which susceptible animals are considered to be infected and thus transfered to the infected population is b_{min}. The maximum body burden for any susceptible animal considered to be uninfected is b_{max}.

$$t_S = \int_{b_{min}}^{b_{max}} \hat{S}_0\, e^{-\rho\, b}\, db = \frac{\hat{S}_0}{\rho}(e^{-\rho\, b_{min}} - e^{-\rho\, b_{max}}). \tag{5}$$

The total number of infectious particles that should be removed from the susceptible internal pool as a consequence is

$$t_F = \int_{b_{min}}^{b_{max}} b\, \hat{S}_0\, e^{-\rho\, b}\, db = \frac{\hat{S}_0}{\rho^2}((1+\rho\, b_{min})\, e^{-\rho\, b_{min}}) - (1+\rho\, b_{max})\, e^{-\rho\, b_{max}}). \tag{6}$$

Note that since the heavily infected susceptibles are the ones that are moved to the infected pool, a large number of internal infectious particles will be removed from the internal pool of the susceptibles. Based on these calculations, a fraction t_S/S of the susceptibles need to be moved to the infected pool and t_F/F of the internal infectious particles need to be removed from the susceptible internal pool. These changes are assumed to occur at a certain rate α [1/day] depending on the host-pathogen system. For instance, for a change that occurs at a rate of 10% per day, α is 2.3. The terms in the governing equations representing disease transmission thus have the form

$$\beta_S = \alpha\, \frac{t_S}{S} \tag{7}$$

where α is a specific rate to move a fraction of the individuals from the susceptible population S to the infected population I. Similarly, the rate at which the internal particles are removed from the internal pool F is

$$\beta_F = \alpha\, \frac{t_F}{F}. \tag{8}$$

Note that the standard mass action model with infective particles with an instantaneous dose response, typically represented as $\beta\, P\, S$ (Bidegain et al., in press), is a specific case of the dose-response model presented here. For $\rho = 0$, Eq. (5) becomes

$$t_S = \hat{S}_0\, (b_{max} - b_{min}), \tag{9}$$

and consequently, $S = \hat{S}_0\, b_{max}$ and $\beta_S = \alpha\, (1 - \frac{b_{min}}{b_{max}})$. The standard mass action model does not explicitly specify particle accumulation or body burden, so that $b_{min} = 0$; thus, Eq. 7 simplifies for the case of an instantaneous dose response transmission $\beta_S = \alpha$.

2.4.2. Uptake of particles by filtration or contact: active and passive suspension feeders

The model is formulated to permit the study of both active (e.g., bivalves) and passive (e.g. corals) suspension feeders. The main feeding difference between these organisms is that active filter feeders pump water actively through a sieve while passive suspension feeders are infected by passively contacting particles transported by water currents. Thus, disease transmission in filter feeders depends on the specific filtration rate (Powell et al., 1999) whereas, for passive suspension feeders, the transmission of disease is a function of the exposed surface area of the individual or colony devoted to food collection and the water flow speed [Sebens et al. (1996) and Sebens et al. (1998) demonstrated that food capture is higher in corals with high polyp and colony exposed surface area in relation to their biomass, and is limited at low flow conditions]. The model incorporates the term f to represent these two foraging strategies. In both cases, we consider that the capture rate of food and infective particles is similar. Consequently, for active filter feeders f represents the rate at which particles are filtered by one individual per unit of time, whereas for passive suspension feeders f represents the rate at which the particles contact the exposed surface area of an individual per unit of time (see Table 2).

Active filter feeders

Particle removal from the water due to active filtration by susceptible individuals (see Eq. (14)) has the form $f_S\, S\, P$, where f_S is the filtration rate (volume filtered) per susceptible individual, S is the number of benthic susceptible individuals, and P is the concentration. Similarly, the term to represent the particle removal due to active filtration of infected individuals (I) and non-focal or alternate incompetent hosts in terms of disease (H) would be $f_I\, I\, P$ and $f_H\, H\, P$, respectively. The incorporation of specific filtration rates for susceptible and infected individuals permits simulation of disease dynamics for cases where infected or severely diseased individuals show a reduction in clearance rate. Similarly, the non-focal host has a specific filtration rate.

Passive suspension feeders

For passive suspension feeders, the particle removal from the water by passive contact (see Eq. (15)) has the form $f\, S\, A_S\, P$, where $f\, S$ is the adrift particle contact rate per susceptible individual and A_S is the exposed surface area per individual. Similarly, the term to represent the particle removal due to passive contact of infected individuals (I) and non-focal or alternate incompetent hosts (H) with particles would be $f_I\, I\, A_I\, P$ and $f_H\, H\, A_s\, P$, respectively. The model also specifies specific contact rates for susceptible and infected individuals, and for non-focal hosts. However, considering that (i) the water movement is the primary mechanism bringing zooplankton into contact with passive suspension feeders and that exposed surface is crucial (Sebens et al., 1998), and (ii) the model already incorporates a term for individual exposed surface area, the term f may be a function of the water flow speed rather than a disease-affected characteristic and, thus, may likely be considered the same for different subpopulations of the same host.

2.4.3. Susceptible Individuals

S is the number of susceptible animals in a given surface area of the bottom. Susceptible individuals are lost by two processes: infection and natural mortality. The disease transmission rate is controlled by β_S which is a function of body burden, allowing for a dose-response to infection, and it is an estimate of the rate at which individuals become infected (see details in section 2.4.1), whereas the natural mortality rate is m_S (Eq. (10)).

The model allows recruitment of new susceptible individuals when the population density (S+I) falls below the carrying capacity K. Susceptible and infected animals can produce recruits at different rates n_S and n_I, respectively allowing for reduced recruitment associated with infected individuals (Yakob and Mumby, 2011). Finally, the model permits an external source of recruits ($S\,RC_S$).

$$\frac{d\,S}{d\,t} = -(\beta_S + m_S)\,S + \Big(1 - \frac{S+I}{K}\Big)(n_S\,S + n_I\,I + S\,RC_S) \tag{10}$$

where, if $1 - \frac{S+I}{K} > 0$, then the carrying capacity is not reached and new individuals can be recruited to the susceptible population as a function of the recruitment rates n_S, n_I, and the potential external source of recruits $S\,rc_S$.

2.4.4. Infected Individuals

Infected individuals are transferred from the susceptible subpopulation to the infected subpopulation at the rate β_S (Eq. (11)). Infected individuals die at a rate controlled by natural mortality m_S and disease mortality m_I.

$$\frac{d\,I}{d\,t} = \beta_S\, S - (m_S + m_I)\, I \tag{11}$$

2.4.5. Dead Susceptible Individuals

Susceptible individuals die at a rate m_S which is a background mortality not related to disease processes (Eqs. (10) and (12)). The subpopulation of dead individuals is reduced at a rate d, which represents bacterial decomposition or scavenging processes. Although true scavengers do not exist in the marine world, many predators scavenge adventitiously (Hoese, 1962; Veale et al., 2000; Morello et al., 2005). These dead susceptible individuals are a diagnostic and do not affect the behavior of the model.

$$\frac{d\,DS}{d\,t} = m_S\, S - d\,DS \tag{12}$$

2.4.6. Dead Infected Individuals

Infected individuals die from disease at a mortality rate m_I and suffer background mortality at rate m_S (Eqs. (11) and (13)). Similarly to dead susceptible animals, these dead infected individuals decay or are scavenged at a rate d.

$$\frac{d\,DI}{d\,t} = (m_S + m_I)\, I - d\,DI \tag{13}$$

2.4.7. Infectious Particles in the local volume

The benthic community comprises susceptible hosts, infected hosts, and alternate hosts, filtering out or contacting particles. The model analyses two situations in terms of the type of benthic system contributing to the removal of particles from the system.

2.4.7.1. Susceptible and infected animals removing and releasing particles.

P is the number of infectious particles in the volume of water immediately accesible to the suspension feeder population (Eqs. (14) and (15)). Infectious particles are mainly added to the water by release from infected individuals at a rate c_I (e.g. by faeces) and from dead infected individuals at a rate c_{DI}. Susceptibles and dead susceptibles can release a small amount of particles through faeces at a rate c_S and upon death through decomposition and scavenging processes at a rate c_{DS}. If dead infected animals release their entire body burden of particles, then $c_{DI} = d$, or they may release a much smaller number depending on the characteristics of the decay or scavenging process removing dead animals, which may inactivate a substantial proportion of or all of the infective particles before they can be released into the water column. Infectious particles are removed from the local volume by one of two processes: (i) inactivation at a rate r, by dilution, transport downstream, or by reduction of infectiousness by inactivation or death, and (ii) filtration by or contact with susceptible and infected individuals.

The local water volume can exchange particles with the vertically adjacent volume if the concentrations are different, through a diffusion-like process controlled by parameter γ (see section 2.2) (Eqs. (14) and (15)). Finally, an unspecified source of infectious particles from another benthic community (SRC_P) is also permitted.

Active filter feeders

At this point, the model differentiates between active filter feeders (e.g., bivalves) (Eq. (14)) and passive suspension feeders (e.g., corals) (Equation 15) (see also section 2.4.2) to represent the distinct removal of particles from the water. Thus, the change in the number of infective particles in a given water volume for active filter feeders is

$$\frac{d\,P}{d\,t} = b_I\,c_I\,I + c_S\,F + b_{DI}\,c_{DI}\,DI\; - (r + f_S\,S\; + f_I\,I + f_H\,H)\,P - \gamma\,(sl\,P - sr\,U) + S\,RC_P \tag{14}$$

where the terms for removal of particles due to filtration have the form $f_S\,S\,P$ or $f_I\,I\,P$, for susceptible and infected individuals respectively, and f_S and f_I are the filtration rate of particles per susceptible and infected individuals, respectively (see units in Table 2). An alternate active filter feeder host population H can also remove particles from the system at the filtration rate f_H (Eq. (14). The alternate host population is assumed to be non–competent in terms of disease. Consequently, the infected and dead individuals of this population do not release infective particles.

Passive suspension feeders

Alternatively, for passive suspension-feeding hosts, the change in the number of infective particles in a given water volume is

$$\frac{d\,P}{d\,t} = b_I\,c_I\,I + c_S\,F + b_{DI}\,c_{DI}\,DI\; - (r + f_S\,A_S\,S\; + f_I\,A_S\,I + f_H\,A_H\,H)\,P - \gamma\,(sl\,P - sr\,U) + S\,RC_P, \tag{15}$$

where the terms for removal of particles due to passive contact of susceptible and infected individuals with particles have the form $f_S\,S\,A_S\,P$ or $f_I\,I\,A_S\,P$, respectively, where A_H represents the particle collection area. An non-focal passive feeder host population H can also contact particles at a rate f_H and function of the particle collection area (Eq. (15)).

2.4.8. *Infectious particles in the remote volume*

U is the number of infectious particles in the remote pool that cannot be directly affected by or affect the host population (Eq. (16)). The incorporation of the remote pool and the exchange of pathogens between pools facilitates the understanding of non-point sources of pathogens and the diffusion processes of waterborne pathogens. The particle exchange γ between the two water volumes describes the process of diffusion of particles. The other process in this equation is controlled by σ, which represents the fact that particles may be removed from the system by loss from the remote pool either through mortality, inactivation, or loss from the system via advection (e.g. tidal exchange). The model also contemplates the fact that there can be an external source of infective particles ($S\,RC_U$) .

$$\frac{d\,U}{d\,t} = \gamma\,(sl\,P - sr\,U)\; - \sigma\,U + S\,RC_U. \tag{16}$$

2.5. R_0 *estimation*

R_0 represents the number of new cases of infection caused by one infected individual in a population of only susceptible individuals. The definition of R_0 in an epidemiological context includes the threshold value of 1, wherein, if $R_0 > 1$, an epidemic can occur and if $R_0 < 1$, an outbreak is not expected (Diekmann et al., 1990; Dietz, 1993). We estimate R_0 using the next-generation matrices (NGM) method for compartmental epidemic models following Diekmann et al. (2010, 2013). Since, R_0 represents the new cases of infection due to one infected animal, likely occurring in a relatively short time scale, we consider the 'close population' variant of the disease dynamic model; that is we do not consider recruitment or non-disease mortality in calculating R_0. The population remains constant, except as far as it is modified by deaths due to the disease itself. The R_0 model considers the two potential infective states: infected individuals and dead infected individuals. We formulate specific R_0 for active and passive filter feeders including the effect of non-focal hosts.

2.5.1. R_0

The basic reproduction number for a system composed of susceptible suspension feeders and non-focal hosts is:

$$R_0 = \sqrt[4]{\frac{\beta_S}{a}\left(\frac{c_I\,b_I}{m_I} + \frac{c_{DI}\,b_{DI}}{d}\right)\frac{f_S A_S N}{r + f_S A_S N + f_H A_H H + \frac{\gamma}{V_l}\left(\frac{1}{1+\frac{\gamma}{\sigma V_r}}\right)}}. \tag{17}$$

This equation permits to formulate R_0 for specific systems regarding the feeding behavior of the host (i.e. active or passive suspension feeder) and the presence of non-focal hosts.

3. Results

The parameter values in Table 2 were used for simulations in sections 3.1 and 3.2 (Figs. 3 and 5). For the rest of the simulations the varying parameter values are specified in each section. Each simulation has a time step of 0.005 day and runs for 1000 days.

3.1. Host dynamics

We conducted two simulations to evaluate the performance of the model regarding the dynamics of the host population. The first case (Case 1 in Fig. 3) was run with the conditions for parameters given in Table 2, with a transmission coefficient of $\alpha = 0.2$ and the carrying capacity K is 300 individuals. The second case (Case 2 in Fig. 4) was run for a lower transmission coefficient ($\alpha = 0.02$) and a higher carrying capacity ($K = 500$). In both cases, initially there are 300 susceptible benthic individuals and zero infected individuals. Initial conditions include 12,000 infective particles m^{-3} in the near-bottom or local volume V_l (Audemard et al., 2006) and none in the remote volume V_r (i.e. $U = 0$). These simulations also assume a transfer of particles between the two pools, and that particles in the near bottom are removed by animals through filtration or passive contact, or by decay or dilution. Population turnover is function of natural and disease mortality, and recruitment of new susceptible individuals.

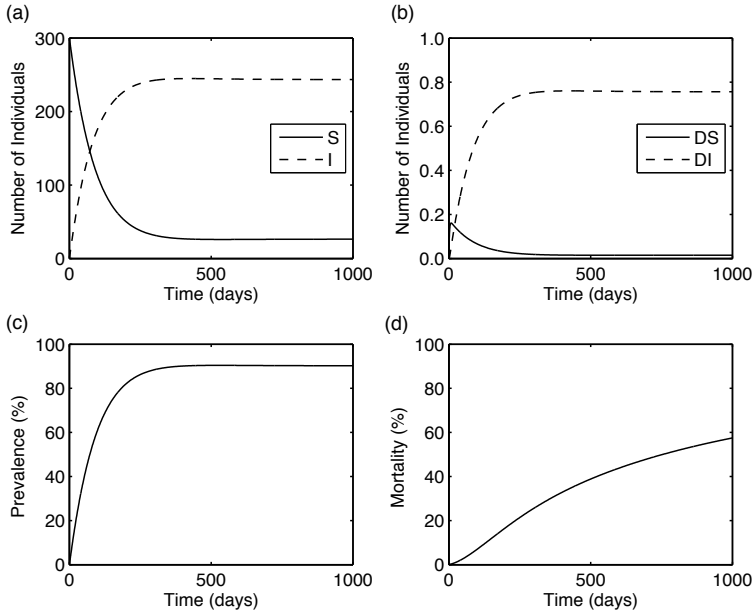

Figure 3: Case 1. Time evolution of host subpopulations (a) susceptible S and infected I, (b) dead susceptible and dead infected, (c) prevalence of infection, and (d) mortality over 1000 days. Mortality is due to both disease and natural causes, assuming a natural mortality of 10% per year. Parameter values in Table 2

In Case 1 (Fig. 3), the susceptible subpopulation decreases as individuals get infected by filtering or contacting infective particles, while the infected subpopulation increases as susceptible individuals get infected and are transfered to the infected subpopulation (Fig. 3a). In Case 2 (Fig. 4) shows more clearly the initial effect of the recruitment increasing the number of susceptible animals (Fig. 4a) as the population reaches carrying capacity. Moreover, the lower transmission rate in the second case gives a larger final susceptible population with respect to the infected

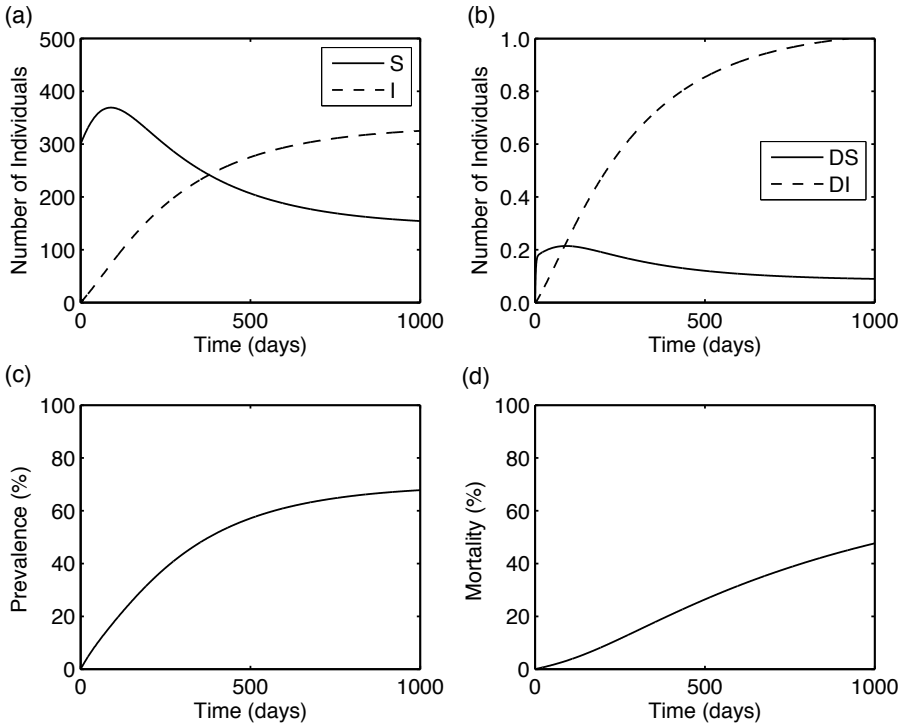

Figure 4: Case 2. Time evolution of host subpopulations (a) susceptible S and infected I, (b) dead susceptible and dead infected, (c) prevalence of infection, and (d) mortality over 1000 days. Mortality is due to both disease and natural causes, assuming a natural mortality of 10% per year. Parameter values as in Table 2 with the exception of the transmission rate coefficient $\alpha = 0.02$ and the carrying capacity $K = 500$

population (Fig. 4b). The dead susceptible and dead infected subpopulations are small due to the high removal rate of dead animals, the number being smaller for the second case because of the lower transmission rate and lower disease mortality (Figs. 3b and 4b). In the first case, the number of dead infected animals increases to a maximum in the second year. As a consequence of rapid infection, almost all dead animals died of infection; an insignificant number of susceptible animals died naturally (Fig. 3b).

Due to the high particle concentration and high transmission rate, in Case 1, the prevalence of infection increases until it reaches a steady and high disease prevalence of almost 90% (Fig. 3c). In Case 2, the prevalence is substantially lower (65%) and the steady state is not yet reached at the end of the simulation (Fig. 4c). The mortality is also higher in Case 1 at the end of the simulation (Fig.3d) compared to Case 2 (Fig. 4d). Disease mortality in both cases greatly exceeds the natural mortality rate of 10% per year; thus mortality in both cases is high due to disease. Case 1, in particular, could be representative of a population heavily impacted by disease both in active filter feeders such as oysters (e.g. Dermo and MSX diseases, (Ford and Tripp, 1996; Ford et al., 2006) and passive suspension feeders such as corals (e.g. white plague disease–Yakob and Mumby (2011)).

Considering the diversity of benthic populations with a wide range of transmission rates and carrying capacities, in addition to these two specific cases described in detail (Figs.3 and 4), we explore the prevalence of infection for a wider range of transmission coefficients α and carrying capacities K (Fig. 5). Initial conditions include 1200 infective particles m^{-3} in the near-bottom or local volume V_l (a much lower initial number of particles than in Cases 1 and 2) in order to better detect and understand the effect of K and α on prevalence of infection in suspension feeders. A very high number of particles in the water column would lead to similarly high prevalence of infections for almost the whole range of values.

Increasing transmission rates leads to higher prevalence of infection in suspension feeders, especially at low carrying capacities (Fig. 5). Increasing carrying capacity can reduce disease transmission even for relatively high

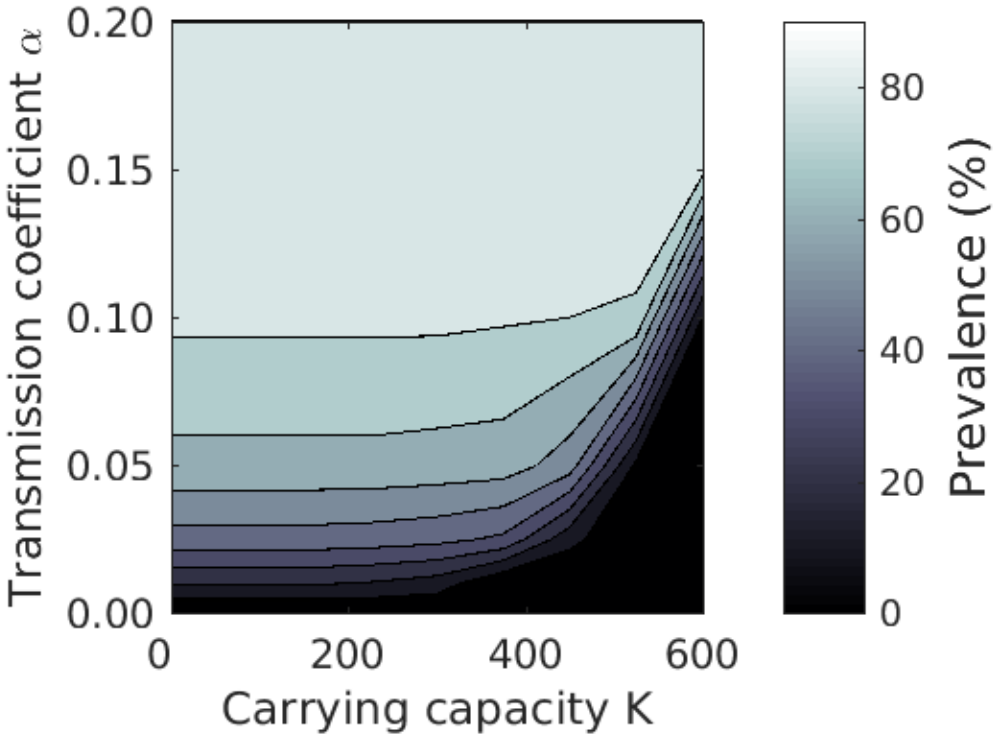

Figure 5: The relationship of prevalence of infection (%) at day 1000 with the transmission coefficient α and the carrying capacity of filter feeders K. Carrying capacity is defined in terms of 76-mm oyster equivalents. X-axis values represent a range of values observed in literature accounts. Other parameter values as in Table 2.

disease transmission rates due to the effect of overfiltration and competition for pathogens, particularly in filter-feeder high-density populations resulting in lower per capita exposure to pathogens.

3.2. Particle dynamics

Particle dynamics corresponding to Case 1 with initial conditions and parameter values given in Table 2 are shown in Fig. 6. The total internal number of particles in the susceptible population increases to a maximum as susceptible individuals accumulate particles without reaching the infective (Fig. 6a). This maximum is followed by a rapid decrease as infective doses are reached in the susceptible population. The decrease in the susceptible population is in turn a consequence of the per capita dose reaching the infective dose. Beyond this point, the total accumulation of particles in the population decreases because the number of susceptible individuals in the population decreases until a steady level corresponding to the steady susceptible population level is reached. Resulting steady state occurs when a balance is reached between disease mortality and recruitment (Fig. 6a).

The per capita particle body burden in susceptible individuals increases (Fig. 6b) to a maximum as a consequence of a combination of both a high concentration of particles in the local volume (Fig. 6c) and a still relatively high density of susceptible individuals. This increase in particle in concentration is due to the release of a large quantity of particles from the increasing subpopulations of infected and dead infected animals (Fig. 6a,b). The dead animals release particles more rapidly than live infected animals ($c_{DI} = 0.5$, $c_I = 0.007$) and thus have a more severe effect on the prevalence of infection. The low particle loss rate ($r = 0.05$) due to decay, diffusion, or advection does not balance

the release of particle from infected animals. The particle dynamics in the remote pool U have the same pattern, but a slightly lower concentration (Fig. 6d) resulting from the diffusion-like process between the two pools.

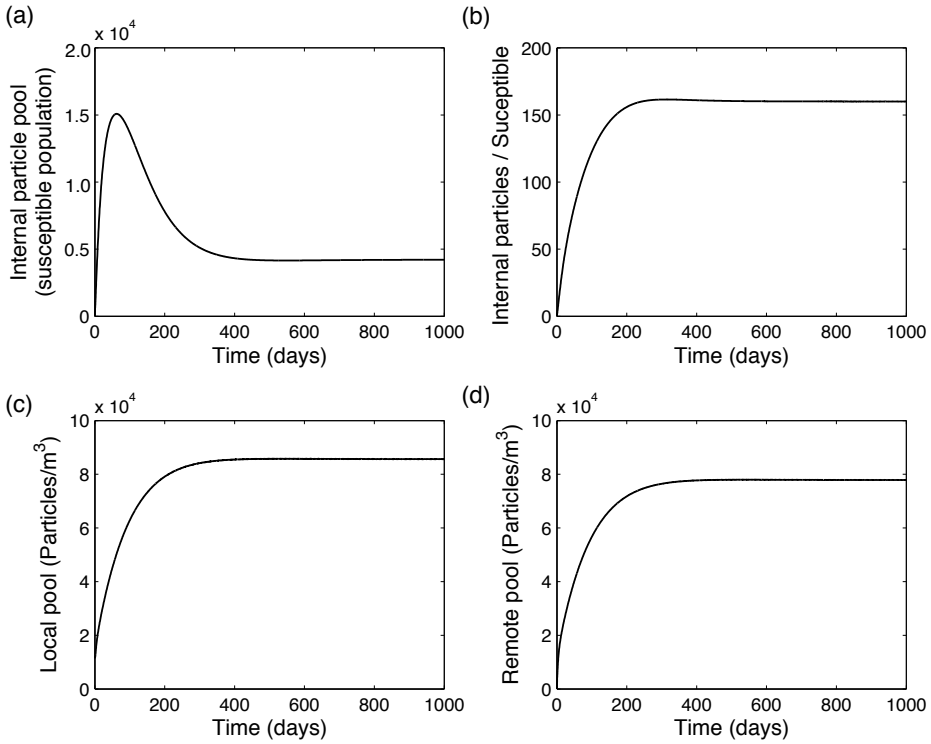

Figure 6: Time evolution of infective particles over 1000 days of simulation: (a) internal particle pool in the susceptible population, (b) internal particles per susceptible individual, (c) concentration of infective particles in the volume available for the benthic population (local pool), (d) concentration of infective particles in the remote volume adjacent to the local volume. Parameter values in Table 2.

3.3. Factors affecting prevalence of infection

We conducted four simulations to evaluate the effect of varying (i) recruitment rate ($n_{S,I}$), (ii) particle filtration or contact rate $f_{S,I}$, (iii) particle loss rate in the local pool (r) and (iv) rates of diffusion of particles by varying the remote pool V_r):local volume (V_l) size ratio, on the prevalence of infection (Fig. 7). Non-varying parameter values are given in Table 2, except that a lower transmission rate β_S was used (i.e. $\alpha = 0.02$).

3.3.1. Recruitment effect

Three recruitment intensities were simulated to compare the relative effect of recruitment rate on the prevalence of infection (Fig. 7a). The simulations with the population at carrying capacity ($K = 300$) and a transmission rate of β_S for a transmission coefficient $\alpha = 0.02$ showed that increasing recruitment intensity limits the prevalence of infection beyond 150 days of simulation with a maximum reduction of 20% between the lowest and highest recruitment scenarios. The similar increase in prevalence of infection at the beginning of the simulation is explained by the fact that the mortality due to disease is too low initially (Fig. 3d) for the population to drop significantly below the carrying capacity. That is, the effect of recruitment on prevalence of infection is visible and increases with the number of new susceptible recruits, which occurs as susceptibles become infected, die and are removed from the system. Together with a decrease in prevalence of infection due to the addition of new individuals, is an increase in the number of individuals becoming infected (Fig. 8b). The addition of individuals by recruitment can also result in a more dense

population filtering out and competing for pathogens, thus reducing the concentration of infective particles in the local pool (Fig. 8c) and decreasing the particle uptake rate per individual (Fig. 8d)

3.3.2. *Filtration/contact rate effect*

To examine the importance of filtration or particle contact rate determining infection prevalence, we varied the filtration/contact rate (Fig. 7b). The simulation showed that the lowest filtration or particle contact rate limits disease transmission because, despite the incorporation of pathogens, the body burden remains below the infective dose (Fig. 7b, gray line). For the two higher filtration rates, initially at day 100, an increasing filtration rate has a clear effect on the prevalence of infection (20%). Beyond this point, the effect of filtration rate continuously decreases and, eventually, beyond a certain point (*ca* day 700) where the prevalence of infection reaches a relatively steady maximum, this effect is almost inappreciable (5%, day 700; 1%, day 1000). The eventual saturation of the local water volume with pathogens (Fig. 6c) released by the increasing infected and dead populations (Fig. 3a,b) makes insignificant the effect of filtration rate on prevalence as the disease progresses in the system.

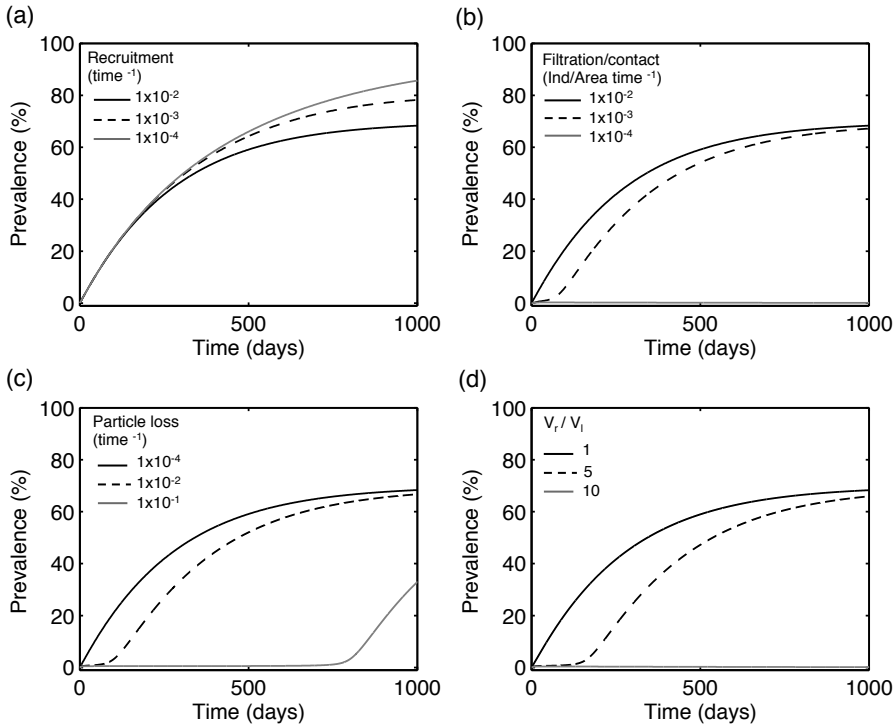

Figure 7: Comparison of the effect of varying (a) recruitment rate, (b) the rate of filtration or contact with infective particles (c) the particle loss rate, and (d) the V_r:V_l ratio on prevalence of infection.

In addition to the contact rate of particles, for passive suspension feeders, such as corals, the accumulation of particles by the individual depends on the exposed surface area per individual A_S (see Eq. 2 and Fig. 11a). Considering Eq. (2), the importance of A_s determining disease prevalence would have the same pattern as the contact/filtration rate f_s. As the exposed surface area increases, more particles contact the animal, consequently prevalence of infection and the likelihood of an epizootic increases (see section 3.4). Similarly to f_s, the saturation of the water with pathogens may minimize the effect of the exposed surface area of the susceptible individual.

3.3.3. *Waterborne pathogen loss*

We examined the potential importance of infective particle loss influencing infection prevalence. For this purpose, we varied the rate of particle loss in the local pool, that is the rate of inactivation of pathogens due to dilution, advection, and mortality. Increasing particle loss limits the prevalence of infection (Fig. 7c) due to the reduced availability of particles to be filtered or contacted. The progressive saturation of particles in the water volume accessible to the benthic population (Fig. 6c) cancels the particle loss effect reducing the infection prevalence. Late in the simulation, the release rate from infected animals clearly overwhelms the inactivation rate of pathogens in the water due to dilution, advection, and natural mortality for the two lower particle loss scenarios tested (see solid and dashed black lines in Fig. 7c). In contrast, at the highest particle loss rate tested (see gray solid line in Fig. 7c), the inactivation rate of pathogens exceeds the release rate, resulting in the absence of infection for the first 800 days and a relatively low prevalence (30%) in the last days of the simulation.

3.3.4. Diffusion of particles

Three rates of particle diffusion were simulated, by means of three remote volume V_r / local volume V_l ratios (Fig. 7d). For most suspension feeders, the volume directly influenced by filter or suspension feeding V_l will be small (e.g., a volume with a height of 0-15 cm for oyster populations (Wilson–Ormond et al., 1997)). Consequently, the size of the remote volume V_r may be a primary determinant of the prevalence of infection. While the local volume ($0.1\,m^3$) was maintained constant, increasing the size of the remote volume from $0.1\,m^3$ to $1\,m^3$ resulted in a decrease in the prevalence of infection of 40% at the beginning of the simulation. Initially, the two lower remote volumes tested showed no infections (see black dashed and gray solid lines in Fig. 7d), but as the simulation progressed only the larger remote volume limited disease transmission (see gray solid line in Fig. 7d). Similarly to the previous case, where particle loss from the local pool was varied, the progressive saturation of particle concentration (Fig. 6a,b) resulted, at the end of simulation, in a suppression of the diffusion effect initially observed for increasing remote volumes.

3.4. Epizootiology and R_0

The generation of an epizootic is regulated by rates involved in the dynamics of both the host and the pathogen, and factors associated with the particle diffusion processes. R_0 increases linearly with increasing transmission rate β_S, and nonlinearly with increasing initial population N (Fig. 9a, black solid line), pathogen body burden and release rate from infected animals $c_I b_I$ and dead infected animals $c_{DI} b_{DI}$, and with increasing filtration or contact rate of particles f_S and local volume sizes V_l (Eq. (17)). The likelihood of an epizootic decreases linearly with increasing inactivation rate of particles inside the body of the animal a, and nonlinearly with increasing disease mortality m_I, the removal of dead individuals from the system d, the remote volume size V_r, the exchange rate of particles between local volume and the remote volume γ, and the loss of particles in the local volume r and the remote volume σ (Eq. (16)). For passive suspension feeders, in addition to filtration or contact rate of particles f_S, transmission of disease increases nonlinearly with the surface area of the susceptible individual exposed to contact with waterborne pathogens A_S.

Host and non-focal host density

In a scenario where an infected animal is introduced into a totally susceptible population, once the population density rises sufficiently to compete for pathogens and lower the per capita body burden in the susceptible subpopulation, the probability of an epizootic developing remains relatively constant with increasing N (Fig. 9a, black solid line). In a scenario with relatively low pathogen body burden, this competition, more likely to occur in active filter feeders than in suspension feeders, results in a reduction of disease risk ($R_0 < 1$) (Fig. 9a, black dashed line).

In a system with non-focal suspension-feeding hosts H (Equation 20), with a relatively high filtration/contact rate of particles f_H, the competition effect between host types is intensified and, consequently, the likelihood of an epizootic decreases with increasing initial abundance of the non-focal host (Fig. 9a, gray solid line). Thus, the threshold of the initial host population N for an epizootic to occur may be substantially increased with increasing abundance of the non-focal host population. For instance, in the Fig. 9a scenario, the epizootic threshold for the initial host population in a system without non-focal filter feeders ($N = 30$) is increased to $N = 80$ after adding the same non-focal host

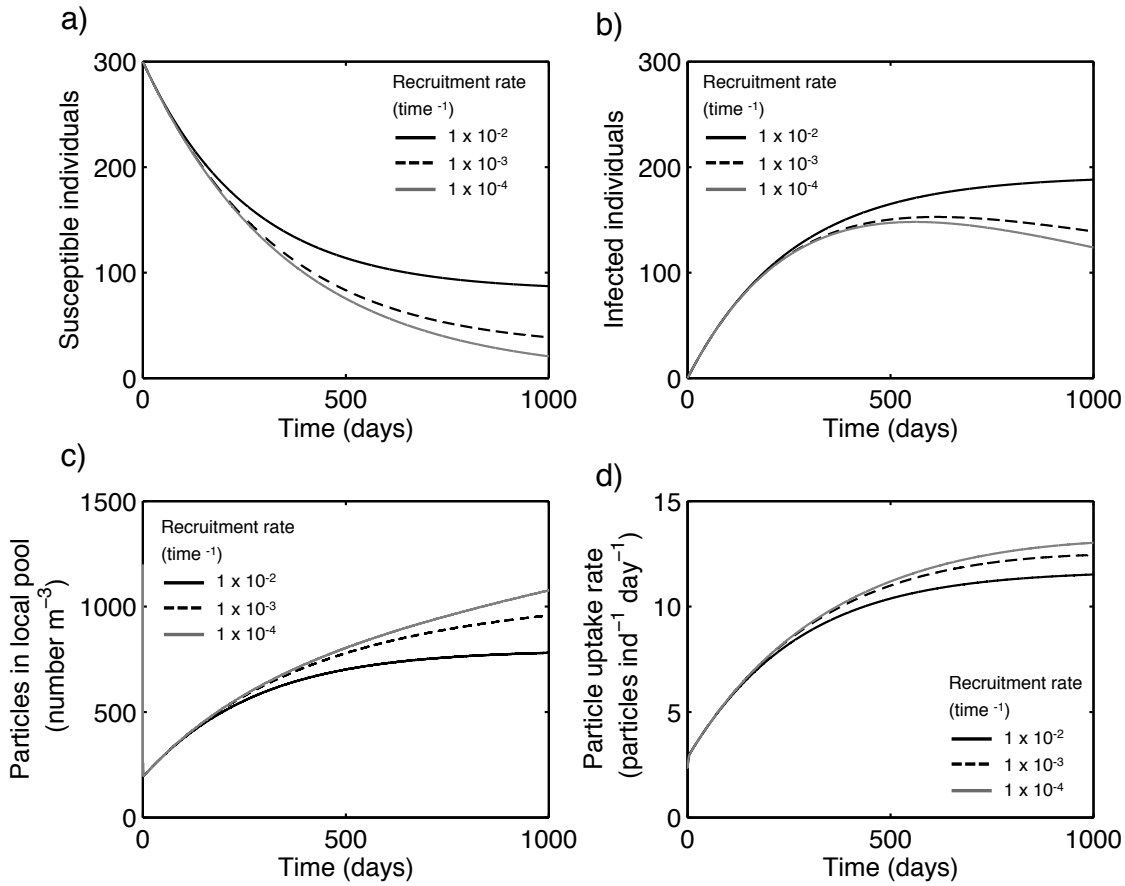

Figure 8: Comparison of the effect of varying recruitment rate on (a) the susceptible population, (b) the infected population, (c) the particle concentration in the local volume, and (d) particle uptake rate.

population ($H = 30$) .

Remote volume size and particle loss

In a scenario with a relatively high exchange rate of particles between the local and remote pools ($\gamma = 1$), an increasing remote volume size, resulting in a diffusion-like transfer of particles to the remote pool, reduces the particle concentration in the local volume available for suspension-feeders and, thus, the likelihood of an epizootic (Fig. 9b, dashed line). The effect of increasing particle loss rate in the remote pool σ on disease development has a similar trend and intensity (Fig. 9b, dash-dot line). Note for comparison that the σ curve is for a remote volume size $V_r = 1$. The effect of increasing internal *in vivo* inactivation rate a also has a similar trend making R_0 estimate much more sensitive to small changes in this parameter (Fig. 9b).

Infective dose

Finally, we explore the similarity of increasing remote volumes and non-focal host densities, specifically focusing on the influence of the infective dose on disease development. When comparing the effect of increasing the remote volume size and non-focal host density on disease development for a high infective dose (200 particles), the R_0 estimate shows that an increase in non-focal host population density of 100 individuals m^{-2} has the same limiting effect

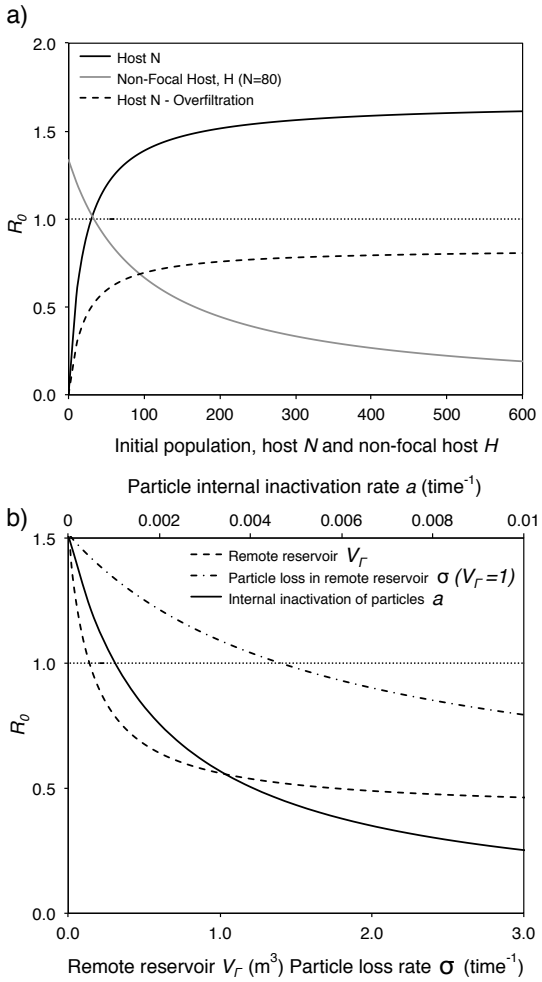

Figure 9: R_0 for increasing (a) initial susceptible N and non-focal host population density (individuals m^{-2}) with the overfiltration scenario for N (with half of the particles), and (b) remote volume size V_r, particle loss rate in the remote pool σ, and particle internal inactivation rate a. The black dotted line represents the outbreak threshold level $R_0 = 1$; above this level the likelihood of an epizootic is high; below this level, an outbreak is not expected.

on epizootics as a remote volume of 0.1 m^3 (Fig. 10). This relationship is nonlinear; thus, a non-focal host population of 500 individuals m^{-2} may have the same limiting effect on epizootics as a remote volume of 2.5 m^3. For lower infective doses (50 particles), the change in non-focal host population abundance or remote volume is much smaller to obtain the same effect on reducing R_0. For instance, an increase in non-focal host population from 0 to 200 individuals m^{-2} or in remote volume from 0 to 0.3 m^3 produces a drop in R_0 from 6 to 3, whereas for a high infective dose (200 particles), the same increase in non-focal host population abundance or remote volume produces a much smaller decrease (R_0 from 1.5 to 0.8). However, if the lower dose disease system has a higher R_0 than the high dose system, a smaller drop in R_0 in the high dose system may contribute more importantly limiting an epizootic than the substantial drop produced by the same change in non-focal host population abundance or remote volume in the low dose system (Fig. 10).

Passive suspension feeders and the exposed surface area

The transmission of disease in passive suspension-feeders not only depends on the contact rate with particles, but also the surface area of the susceptible individual exposed to contact with waterborne pathogens (A_s) (Eq. (17)). First, we conducted a simulation to evaluate the effect of varying A_s on the accumulation of particles. The simulation was run with the conditions for parameters given in Table 2 for Case 2, with a carrying capacity K of 300 individuals. Susceptible individuals accumulate particles (Fig. 11a) with time as disease progresses and the water becomes saturated with particles. The simulation shows an increase in the average host body burden of infective particles as the exposed surface area increases. At the higher exposed surface area tested, the average internal number of particles per susceptible increases to the infective dose (200 particles) by day 150.

Second, we introduced an infected animal in a susceptible population of 300 individuals and calculated R_0 for a range of values for of A_s, including possible realistic feeding surfaces in suspension feeders such as corals (Fig. 11b). These simulations were conducted for low and high infective doses (50 and 200 particles). The likelihood of an epizootic increases nonlinearly with the surface area exposed by the individual to waterborne pathogens A_S, resulting in the requirement for 3 times larger exposed surface area for an epiozootic to occur in the higher infective dose case (Fig. 11b).

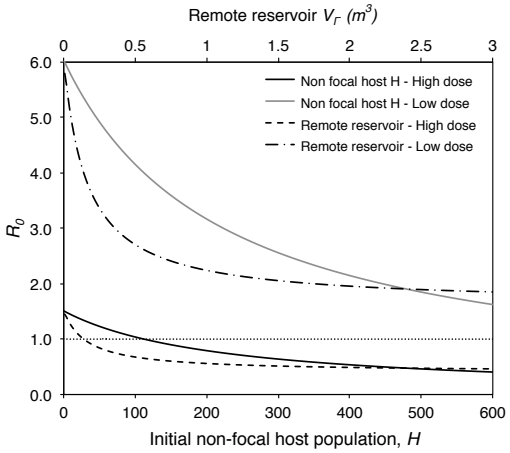

Figure 10: R_0 for increasing non-focal host population density (individuals m^{-2}) and remote volume size (m^{-3}) for a high infective dose (200 infective particles) and for a low infective dose (50 particles). The gray dotted line represents the outbreak threshold level ($R_0 = 1$).

4. Discussion

The deterministic compartmental model accounts for the dynamics and epizootiology of waterborne microparasitc infectious diseases in suspension-feeders, focusing on both the host and the pathogen dynamics. In this model, transmission occurs via particle uptake by contact for passive filter feeders or by filtration for active filter feeders, of waterborne infective pathogens under a body-burden based dose-response mechanism. The model yields a set the basic reproduction numbers R_0 which give insight about the relative importance of the parameters determining the outbreaks of epizootics. The generation of an epizootic is regulated by rates involved in the dynamics of both the host and the pathogen, and factors associated with the particle diffusion processes.

4.1. Host dynamics

Simulation results indicate that a relatively high initial concentration of infective particles available for suspension feeders, where particle inactivation is slow, the prevalence of disease increases rapidly in the first six months to a high and steady level (Fig. 4). Mortality is also relatively high, increasing to a 60% of the population at the end of the simulation. Disease mortality rates in bivalve filter feeders can be 50% or more per year. Rates this high are reported

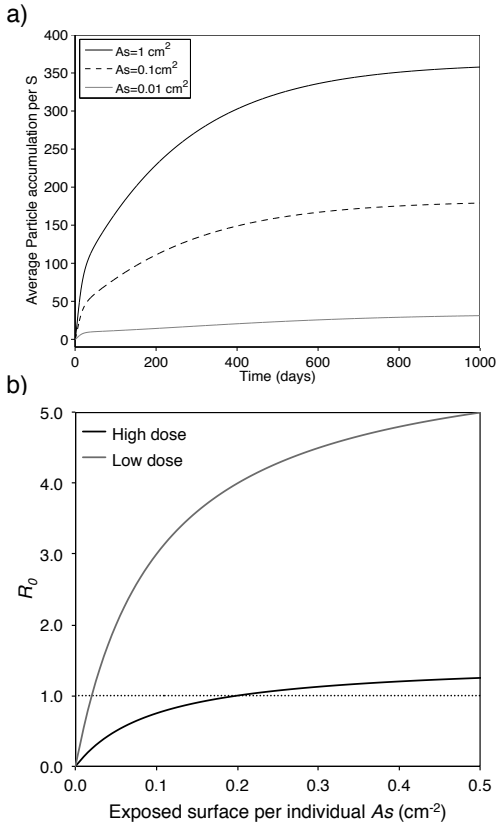

Figure 11: Effect of increasing surface area of susceptible individuals exposed to contact with waterborne pathogens A_s on (a) particle accumulation with time, and (b) disease risk (R_0) for high (200 infective particles) and low (50 infective particles) infective doses. The gray dotted line represents the outbreak threshold level ($R_0 = 1$).

in oysters in the Gulf of Mexico (Soniat and Brody, 1988). In Delaware Bay, the mortality rate is lower; however, dermo disease at least doubles the natural mortality rate of the market-size animals in epizootic years (Ford et al., 2006; Powell et al., 2008). The fungal disease Aspergillosis can impact sea fan corals with mortalities resulting in the loss of >50% of the sea fan colony area in six years (Kim and Harvell, 2004). Worldwide distributed clam species such as *Ruditpes philippinarum* show similar mortality rates associated to brown ring disease (Paillard et al., 2014).

The recruitment of new susceptible individuals to the system (Case 1, Fig. 4) permits levels of prevalence lower than 100% (e.g., 90%, Fig. 4). This simulation with a low particle loss rate in the local volume of water saturated with infective particles, likely reproduces a "water-tank" system where eventually all the particles in the water are coming into contact with or filtered out by susceptible individuals and so prevalence in the population reaches high levels and remains high, even with recruitment. The development and maintenance of high particle concentrations in the water column (Fig. 6d) resulting in pandemic infection must be one of the reasons for the rapid transmission of Dermo disease and the high mortalities in Eastern oyster populations observed in the early 1990s in Delaware Bay (Ford et al., 2006; Bushek et al., 2012) and in other bays where most newly settled juveniles become infected well within the first year of life (Powell et al., 1996; Ragone Calvo et al., 2003; McCollough et al., 2007; Soniat et al., 2012). Geographically expansive regions with high concentrations of infective particles leading to pandemic infection must occur commonly for diseases like Dermo and other Perkinsus-caused diseases in order to explain the continuously high prevalence of infection (e.g., Park and Choi, 2001; Powell and Hofmann, 2015).

The number of dead animals is small (<1 m^{-2}) in both simulations (Case 1 and 2) due to the high removal rate of these animals from the system. In general, in marine systems, the bacterial decomposition of organic matter (Allison,

1990; Lotz and Soto, 2002; Smith, 1953) or the action of scavengers removing dead animals (Veale et al., 2000; Morello et al., 2005) is a relatively fast process and thus, dead animal tissue is removed quickly from the environment. The initial increase of dead infected individuals to a maximum is related to the increase of infected individuals as the disease transmission progresses in the population. The infected animals and their decay or scavenging can contribute pivotally to transmission by the releasing of particles to the local volume and ultimately supplying the remote pool (Fig. 6c) or they can be inconsequential if the act of decay or scavenging results in the loss of infectivity. Few studies have examined the importance of post-mortem processes in disease transmission.

Simulations show the effect of varying transmission rate and carrying capacity on the host population (Case 1, Fig. 3 vs Case 2, Fig. 4). When the population is below carrying capacity, the susceptible population initially increases (Fig. 4) as the recruitment of new susceptible individuals is faster than the rate of infection. As the disease transmission progresses in the population with a substantial increase of particles in the environment, the reduction of susceptible individuals due to infection exceeds the incorporation of new individuals. The slower transmission rate in Case 2 (Fig. 4) importantly reduces the spread of the disease in the population, lowering the prevalence of infection. Overall, increasing transmission rate leads to higher prevalence of infection in filter feeders, especially at low carrying capacities or at low population densities where the carrying capacity is high. Increasing population density, and particularly in cases where carrying capacity is high, can reduce reduce prevalence of infection even for relatively high disease transmission rates (Fig. 5). In a contact–based density–dependent disease, a population at its carrying capacity has a higher likelihood of disease transmission than when the population is below carrying capacity (Gao and Hethcote, 1992). However, dense filter feeder populations at carrying capacity may be less vulnerable to disease; individual hosts may 'compete' for pathogens reducing the concentration of infective particles sufficiently to limit body burden below the infective dose and, in turn, limiting the probability of epizootic development (i.e. the overfiltration scenario, Bidegain et al., in press).

4.2. Particle dynamics

Many disease models for marine filter-feeders have addressed the proliferation of infection and subsequent disease impact, accepting widespread and rapid transmission to be the norm (Ford et al., 1999; Powell et al., 1999, 1996). Others have examined the effects of diseases on host population dynamics (Kuris and Lafferty, 1992; Yakob and Mumby, 2011). However, models of marine diseases that incorporate particles as a variable, permitting the water column to act as a 'reservoir', conduit, or sink for infective particles, are few and none also include an infective dose effect based on an *in vivo* compartment of infective particles sequestered in the susceptible pool of hosts. The model that we propose here tracks pathogen dynamics in (i) the local volume with a particle pool interacting with the host population, (ii) a remote volume that can act as a source or sink for infective particles depending on the relative concentration between the two volumes, and (iii) a transient pool of infective particles in the susceptible individuals that may ultimately be inactivated or transition the individual to the infected pool.

Simulations show that initial infections of a few susceptible individuals initiates the process (Fig. 3a,b) by which substantial numbers of infective particles are released into the water thereby becoming available to susceptibles and saturating the water with particles (Fig. 6c). This results in an initial diffusion-like transfer of particles to the adjacent remote volume and, eventually, in a 'balance' in particle concentration between the two water compartments (Fig. 6d). A system with a high release rate of pathogens from infected animals or a high rate of release of infective particles upon death, and a limited inactivation rate of infective particles either in the water column or in the susceptible host, might easily lead to be a highly transmissible system characterized by a relatively high incidence of disease outbreaks. This is the case, for instance, for oysters and the pathogen *Perkinsus marinus* (Bushek et al., 2002), but might also occur in corals, where the decay of tissue of infected colonies infected by black-band disease or aspergillosis releases infectious fomites or individual particles into the water which then drift to nearby corals (Jolles et al., 2002; Richardson, 2004; Zvuloni et al., 2009) or in the case of withering syndrome in abalone where infective elements or fomites released by infected individuals before or after death spread infection over wide expanses (Lafferty et al., 2015). We suspect a similar scenario for the disease of long-spined sea urchins that ravaged the Caribbean populations late in the last century (Lessios et al., 1984). Consequently, in these systems, parameters incorporated into the model, such as the body burden of pathogens in infected (b_I) or dead animals (b_{DI}) and the relative importance of the release rate (c) and the removal rate of pathogens in the tissue (a) or water column (r and $sigma$) might have profound influence in determining the frequency and geographic extensiveness of epizootics.

The model present here also tracks particle dynamics inside the susceptible population. The dynamics of the infective dose effect in filter and suspension feeders is poorly understood (e.g., Chu and Volety, 1997; Wang et al., 2010). Our model suggest that the accumulation of particles increases to a maximum in the initial phase of the disease process as susceptible animals take up particles without reaching an infective dose and that a number of *in vivo* and external processes determine the degree of this accumulation and the probability that sufficient infective particles will be accumulated to produce an infection. Among these is the immune response, but equally important are the processes controlling uptake, namely the particle acquisition rate (filtering or surface area impingement) and particle concentration which itself is controlled by the rate of addition and loss of particles from the local water volume. This dynamic is likely enormously important, yet to date has been little studied. Assuming that the disease transmission in filter feeders (Bushek et al., 1997; Ford et al., 1999; Powell et al., 1999), and probably in other suspension feeders, occurs via an infective dose so that susceptible animals need to have some minimum level of body burden of infectious particles before they become infected, some susceptible individuals will then have a relatively high body burden while most will have a small body burden (see section 2.4.1). Presumably, these animals will have some unique attributes either genetically or environmentally that has resulted in their higher body burdens. Understanding the contributing factors that result in a few animals initiating infections than then can propagate by supporting increased particle concentration in the water column is a critical need. In this regard, simulations show that the average per capita dose in susceptible population is 150 particles (Fig. 6b). This demonstrate the adequate performance of dose-based transmission term, considering that the average per capita dose is lower than the minimum dose for susceptibles to become infected b_{min} (200 particles).

4.3. Recruitment

Recruitment introduces new, healthy individuals into the population which have the net effect of reducing disease prevalence. However, in this case, this effect of recruitment on reducing prevalence does not directly limit disease; a decrease in prevalence of infection is not a consequence, for instance, of new individuals increasing population particle filtration rate and then reducing the local pool of infective particles sufficiently to decrease per capita accumulation below the infective dose. The prevalence is lower only due to the addition of more susceptibles to the system. The influence of these new susceptible individuals will depend upon their role as new produces of infective particles or as new 'inactivators' of infective particles. Often these new recruits will promote the disease as the number of individuals becoming infected increases (Fig. 8b) despite the reduction of (i) particles in the water column due to increasing population particle filtration (Fig 8c) and (ii) the particle uptake rate per animal (Fig. 8d). Rather, these individuals provide increased production of infective particles, raising their concentration in the local pool and ultimately the remote pool. This explains the occurrence of pandemic infections in marine filter feeders such as oysters regardless of increasing recruitment intensities (Ford, 1996; Bushek et al., 2012).

However, the effect of recruitment in limiting the occurrence or the duration of disease epizootics can be substantial in both active and passive suspension feeders when enough recruits enter the population to reduce the infective particle concentration in the water column such that the particle uptake rate per animal drops to a point where an increasing number of animals retain a transient body burden below the infective dose. This effect has been suggested to occur in oyster populations following high intensity recruitment events. Even at high salinity, suitable for disease transmission, an oyster population might be able to resist epizootic development as long as recruitment sustains the increase in population density (Hofmann et al., 1995; Powell et al., 1996).

4.4. Filtration/contact rate

Filtration rate in filter feeders, such as bivalves, can be affected by diverse characteristics of the particles filtered, such as the concentration of phytoplankton and suspended solids and the quality and size of food particles (Hofmann et al., 1994; Khalil, 1996). The physical parameters of the natural habitat such as temperature, salinity, and water flow (MacDonald and Thompson, 1986) and the size of the animal can also affect the filtration rate (Dame, 2011). In passive suspension feeders, such as corals, the contact rate depends on the polyp or colony exposed surface area and the flow conditions (Sebens et al., 1996, 1998). Such factors affect the incorporation of pathogens by susceptible animals and, thus, the disease incidence.

We varied filtration/contact rates to explore this effect (Fig. 7b). A relatively low filtration or particle contact rate limits disease transmission, due to lower per capita exposure to pathogens. Increasing filtration rate has an effect on disease transmission when the concentration of particles in the water is relatively low. In this case, as filtration or contact rate increases, the rate of disease transmission increases correspondingly. However, a change in filtration or contact rate ceases to exert a significance influence on transmission when the local volume becomes saturated with infective particles. At this point, higher filtration rates (Fig. 7b) result in the same high prevalence; that is, the per capita dose received by individuals is above the infectious dose over a wide range of population filtration rates. However, active filter feeders in particular, and suspension feeders possibly have two mechanisms, explored here, to reduce the incidence of infection despite having high filtration rates and contact rates with substantive numbers of infective particles in the water column. The first obviously is a higher infective dose which might result from selection for more disease resistant genotypes, for example (Munroe et al., 2015) (Fig. 10) and the second is a dense assemblage of hosts competing for pathogens and reducing the per capita dose (i.e. the overfiltration scenario – Bidegain et al. in press) (Fig. 9a, black dashed line). Because it is well established that dense concentrations of bivalves can become food limited (Heasman et al., 1998; Jiang and Gibbs, 2005; Powell et al., 1995), it stands to reason that the same process would limit per-capita incorporation rates of infective particles. In passive suspension feeder hosts in environments with high particle concentrations and contact rates, reduced exposed feeding areas may result in lowering the accumulation of particles (Fig. 11a), and thus the prevalence of infection. Whether passive suspension feeders can become sufficiently dense to limit per capita particle uptake rate is unclear.

4.5. Particles loss and diffusion

The model incorporates two parameters for particle loss in the water column. The parameter r accounts for the particle loss within the local volume V_l due to natural mortality or other processes of inactivation such as sedimentation, and advection and diffusion to the remote volume V_r. The parameter σ accounts for the subsequent inactivation through natural mortality or other sources of inactivation such as sedimentation in this remote pool or perhaps loss from this compartment through, for example, outwelling. These two volumes in the model are formulated in order to account for particle diffusion by means of concentration gradients between them. Initially, in the simulation when the local volume is not saturated with particles, increasing particle loss through diffusion of particles to the remote pool can limit epizootic development. When, the environment is saturated with particles, this effect becomes inconsequential (Fig. 7c,d) unless the inactivation rate in the remote pool is high (Fig. 9b). A large remote volume together with a high exchange rate of particles between pools γ and a relatively high inactivation rate of pathogens in the remote pool a is an effective mechanism to reduce particle concentration locally and prevent transmission (Bidegain et al., in press) (Fig. 9b). Thus, a scenario similar to what is shown in Fig. 7d (gray line), in which the remote volume is substantially larger than the local volume, thereby leading to a rapid transfer of pathogens from the local pool to the remote pool, is particularly effective at limiting the incidence of infection. This scenario could easily occur in estuaries with short water residence times (Armstrong, 1982; Marshall and Alden, 1997)during periods of increased freshwater inflow or during spring tides where the larger tidal volume results in increased outwelling of particles, thereby reducing particle concentration in the water column (Ellien et al., 2004; Banas et al., 2007; Gray et al., 2009; Narváez et al., 2012).

On the other hand, a high concentration of infective particles in the remote volume could overcome any local loss mechanisms such as overfiltration or the removal by non-focal hosts, and thus maintain high prevalence. The dynamic interchange between local and remote pools if not unique to the marine realm is at least particularly characteristic of the disease transmission processes taking place there. This model is a theoretical model that explores the processes that initiate and terminate an epizootic. The simplification of hydrodynamics to two volumes, the volume directly impacted by the filter feeder and the volume overlying it, with particle diffusion occurring across the interface is sufficient to explain these processes theoretically. The study of a specific case such as the disease transmission on a reef or in an estuary would require the use of similar disease models coupled to a hydrodynamic model. While complex models of larval dispersal using location-specific hydrodynamics have been employed often (North et al., 2008; Ayata et al., 2009; Bidegain et al., 2013), the transmission of disease particles in such applications has been little investigated (Murray and Jackson, 1992).

4.6. Epizootiology and R_0

Simulation results of host and particle dynamics showed that dead infected animals, likely with higher body burden and release rates of infective particles than most live infected animals (Bushek et al., 2002; Richardson, 2004), are determinant in the generation of epizootics in benthic filter and suspension feeders. A relatively small external source of infective particles initially available to a benthic suspension–feeder population may infect few individuals. Although this external source is consumed rapidly or lost and inactivated in the water column, these initially infected individuals can trigger the generation of the epizootic by means of particle release in the live-infected stage and, particularly, upon death.

However, mechanisms potentially exist that might lead to a lowering of the epizootic likelihood in a benthic population of suspension feeders even if a relatively large number of particles is available. High densities of suspension feeders, particularly filter-feeders, may compete for food, but also for waterborne pathogens. Moreover, a non-focal host population ingesting or otherwise collecting infective particles (e.g., bivalves, sponges or tunicates), may enhance the competition effect, which could lead to a lower per capita exposure to pathogens (Peterson and Andre, 1980; Beukema and Cadée, 1996; Powell et al., 2012c). Thus, the threshold of the initial host population for an epizootic to occur may be substantially increased with increasing numbers of non-focal hosts (Fig. 9). This intra- or inter-specific competition (Fig. 9a and 8) by itself or together with particle diffusion (Fig. 9b) may reduce substantially the likelihood of an epizootic.

The effect of increasing particle loss on limiting the epizootic development is similar to that produced by an increase in remote volume size (Fig. 9b). This means that the remote volume is an effective mechanism for limiting an epizootic as it can remove particles from the local pool by diffusion. Thus, an increase in the remote volume size may exert a limiting effect on epizootic development comparable to that produced by inter-specific competition with a non-focal host population. Moreover, an increase in these two disease limiting factors will have a higher impact on disease control in hosts with a relatively high infective dose (Fig. 10). However, an increase in the remote volume needs to be accompanied by particle inactivation or loss (i.e. σ) in order to have an effect on epizootic development. In absence of this particle removal, the remote volume may become saturated with particles and diffuse them back to the local pool thereby making them available to susceptible hosts. In contrast, *in vivo* inactivation rate of particles, by limiting particle body burden below the infective dose, has a more intense effect on disease development than the remote volume size or the particle loss in this volume (Fig. 9b).

Overall, these disease limiting mechanisms apply similarly, but no in equivalent measure, to active and passive suspension feeders. In the case of passive suspension feeders, the competition for particles may be less effective than in active filter-feeders. The exposed surface area of the individuals A_s in a colony of passive suspension feeders (e.g. corals) is as important as the density of individuals; thus, high density populations with a small exposed surface area per individual, resulting in a reduced particle accumulation, may limit the disease outbreak (Fig. 11) just as well as fewer individuals with a large surface area for particle capture. Thus, it seems that the active foraging strategy of filter feeders permits these organisms to effectively limit disease outbreaks in comparison to passive feeders, but the same attribute makes them more vulnerable to epizootics when population densities are inadequate to control local particle concentrations beneath the threshold yielding an effective dose (Fig. 9a and Fig. 11).

5. Conclusions

A large number of marine diseases are transmitted through the water column from one host to another. This water column provides a ‘reservoir’ for infective particles and the mechanisms by which particles are added to it or lost from it exert an important influence on the prevalence of disease and more importantly the difference between a disease exerting a local impact on a host population and pandemic disease affecting the host over large geographic regions. The local population modulates this effect through genetic characteristics that affect the infective dose and through varying local availability by modulating particle incorporation and release rates. The dynamic imposed by this complex interaction between population and water column, potentially over metapopulation scales, is relatively unique to the marine world. Understanding the details of this dynamic is critical to understanding the disease process in host populations and to improving management responses to marine disease challenges. Models that incorporate these processes, such as the one described here, are important tools, little used heretofore in the marine context, to explain the epizootiology of marine diseases. Only when a disease can be understood at the *in vivo* scale of the

individual, the local scale of the population, and the geographic scale of the metapopulation will effective approaches to management become routinely achievable.

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Parameter	Definition	Unit	Values	Key references
β_S	Disease transmission rate by filtration or contact with particles.	Particle^{-1} time^{-1}	Eq. 7	Hofmann et al. (1995); Ford et al. (1999)
β_F	Particle removal rate from F corresponding to the infection of S	Particle^{-1} time^{-1}	Eq. 8	Hofmann et al. (1995); Ford et al. (1999)
α	Transmission rate coefficient (see Eqs.7,8)	Particle^{-1} time^{-1}	$2{\cdot}10^{-1}$	Hofmann et al. (1995); Ford et al. (1999); Sokolow et al. (2009)
m_S	Natural mortality rate	time^{-1}	$3{\cdot}10^{-4}$	Powell et al. (2008, 2009); Sokolow et al. (2009)
m_I	Disease mortality rate	time^{-1}	$8{\cdot}10^{-4}$	Bushek et al. (2012); Choi et al. (1989); Sokolow et al. (2009)
d	Removal rate of dead individuals by scavengers or decay	time^{-1}	$5{\cdot}10^{-1}$	Hoese (1962); Diamond (2012)
n_S	Recruitment rate of S	time $^{-1}$	$1{\cdot}10^{-2}$	Powell et al. (2008, 2009); Sokolow et al. (2009)
n_I	Recruitment rate of I	time^{-1}	$1{\cdot}10^{-2}$	Powell et al. (2008, 2009); Sokolow et al. (2009)
K	Population carrying capacity	Individuals	300	Powell et al. (2012b); Yakob and Mumby (2011)
b_I	Average P per I	time^{-1}	$1{\cdot}10^{6}$	Choi et al. (1989)
b_{DI}	Average IP per dead animal	time^{-1}	$1{\cdot}10^{7}$	Choi et al. (1994); Powell et al. (1996)
b_{min}	Minimum body burden to become I	time^{-1}	200	Adapted from Ford et al. (1999), Guo, unpublished data (oysters)
b_{max}	Maximum body burden to be considered I	time^{-1}	400	400
c_S	Release rate of pathogens from S	time^{-1}	$7{\cdot}10^{-3}$	Bushek et al. (2002)
c_I	Release rate of pathogens from I	time^{-1}	$7{\cdot}10^{-3}$	Bushek et al. (2002); Sokolow et al. (2009)
c_{DI}	Release rate of pathogens from DI	time^{-1}	$5{\cdot}10^{-1}$	Hoese (1962); Diamond (2012)
r	Loss rate of waterborne pathogens from the local pool	time^{-1}	$5{\cdot}10^{-2}$	Sokolow et al. (2009) and this study, experimental data
f_S (*active*)	Filtration rate of infective particles by S	Individual^{-1} time^{-1}	$1{\cdot}10^{-1}$	Powell et al. (1992)
f_I (*active*)	Filtration rate of infective particles by I	Individual^{-1} time^{-1}	$1{\cdot}10^{-1}$	Powell et al. (1992)
f_H (*active*)	Filtration rate of infective particles by the alternate host	Individual^{-1} time^{-1}	$1{\cdot}10^{-2}$	Ben-Horin, unpublished data (tunicates)
f_S (*passive*)	Contact rate of susceptible individuals with particles	Area^{-1} Time^{-1}	$1{\cdot}10^{-1}$	Adapted from Sokolow et al. (2009)
f_I (*passive*)	Contact rate of I with particles	Area^{-1} Time^{-1}	$2{\cdot}10^{-1}$	Adapted from Sokolow et al. (2009)
f_H (*passive*)	Contact rate of non-focal hosts with particles	Area^{-1} Time^{-1}	$1{\cdot}10^{-1}$	Adapted from Sokolow et al. (2009)
A_S	Exposed particle collection area per S or I	Area Individual^{-1}	$1{\cdot}10^{-4}$	Adapted from Sebens et al. (1996, 1998)
A_H	Exposed particle collection area per alternate host	Area Individual^{-1}	$1{\cdot}10^{-4}$	Adapted from Sebens et al. (1996, 1998)
a	Reduction rate of pathogens inside hosts by diapedesis, phagocytosis, apoptosis, etc.	time^{-1}	$1{\cdot}10^{-1}$	Ben-Horin, unpublished data data (oysters)
sl	Local volume factor to normalize concentration of pathogens; it is the reciprocal to the height of the local volume V_l	vol^{-1}	10	Wilson–Ormond et al. (1997)
sr	Remote volume factor to normalize concentration of pathogens; it is the reciprocal to the height of the remote volume V_U	vol^{-1}	1	Model calibration
γ	Exchange rate of free-living pathogens between pools	time^{-1}	$5{\cdot}10^{-1}$	Model calibration
σ	Free-living pathogen lost rate from the remote pool	time^{-1}	$5{\cdot}10^{-2}$	Ben-Horin, unpublished data (oysters)

Table 2: Parameters of the model. These parameters values are considered plausible for active suspension feeders such as bivalves (e.g. oysters, clams) and passive suspension feeders such as corals, and were adapted from literature data and experimental results obtained for Eastern oyster by one of the authors (Ben-Horin, unpublished data), and from model calibration. Note that the model has an implicit surface area or volume for the parameters. The units used in the simulations are days for time, m^2 for area, and m^3 for volume.