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Modeling the transmission of *Perkinsus marinus* in the Eastern oyster *Crassostrea virginica*

G. Bidegain[a,*], E.N. Powell[a], J.M. Klinck[a], E.E. Hofmann[b], T. Ben-Horin[c,d], D. Bushek[c], S.E. Ford[c], D.M. Munroe[c], X. Guo[c]

[a] Gulf Coast Research Laboratory, University of Southern Mississippi, 703 East Beach Drive, Ocean Springs, MS 39564, USA
[b] Center of Coastal Physical Oceanography, Old Dominion University, 4111 Monarch Way, Norfolk, VA 23529, USA
[c] Haskin Shellfish Research Laboratory, Rutgers University, 6959 Miller Avenue, Port Norris, NJ 08349, USA
[d] Department of Fisheries, Animal and Veterinary Science, University of Rhode Island, 20A Woodward Hall, 9 East Alumni Avenue, Kingston, RI 02881, USA

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ABSTRACT

Dermo disease caused by the protistan *Perkinsus marinus* in Eastern oysters *Crassotrea virginica* is an important source of mortality impacting oyster population dynamics resulting in substantial losses in fisheries and aquaculture. The rapid transmission and spread of the disease minimized the importance of transmission models and past models (proliferation-based models) assumed simple density-dependent transmission or rapid infection post-settlement. This approach is a good approximation only for low population densities. A transmission model was developed for *P. marinus* in Eastern oysters that accounts for the seasonal change in disease dynamics and density-dependent foraging (of suspended particles) interference among hosts. The model, verified and evaluated against field observations, incorporates parasite release to the water column from live and dead individuals, parasite consumption by living oysters, the diffusion of parasites in the water, body burden-based dose-dependent transmission, recruitment, and disease-caused mortality. The model returns a basic reproduction number R_0 for Dermo much greater than unity ($R_0 = 90$) in accordance with the current persistence and pandemic nature of this disease in oysters. No population density is obtained that is low enough to suppress R_0 below 1 (i.e. disease extinction). R_0 is also estimated for high oyster densities (>300 individuals m^{-2}) and particularly for relatively large oysters (~90 mm), today rare but once common before generalized overfishing occurred on healthy oyster reefs. In this scenario, R_0 drops below 1, indicating that high oyster density can limit disease invasion through foraging interference and depletion of parasites in the water column. High intensity recruitment events allow the oyster population to attain such densities and limit the development of epizootics. These results provide insight into the transition from past populations, where Dermo is inferred to have been limited in its impact, to the current persistent and pandemic nature of this disease. Further coupling of this model into metapopulation and hydrodynamic models could be a promising tool to support management decision-making for bivalve populations impacted by Dermo disease.

1. Introduction

Dermo disease, caused by the protozoan parasite *Perkinsus marinus* (Andrews, 1988; Mackin, 1951; Perkins, 1988), is responsible for extensive epizootics and mass mortalities of the Eastern oyster *Crassostrea virginica* along the Atlantic and Gulf coasts of the United States (Bushek et al., 2012; Ford and Tripp, 1996; Powell et al., 1996). Mortality events associated with this disease impact oyster population dynamics and the structure and ecological function of oyster beds (Coen et al., 2007; Kemp et al., 2005; Powell et al., 2012a), result in severe losses in fisheries and aquaculture (Lafferty et al., 2015), and constitute a major impediment to both commercial production and habitat restoration of oysters (Mann and Powell, 2007). *P. marinus* is transmitted directly from infected to uninfected oysters through filter feeding of infective particles (Ragone Calvo et al., 2003; McCollough et al., 2007; Perkins, 1993). Live and dead infected oysters release *P. marinus* cells through feces and predation and by means of tissue decay, scavenging or vector transfer (e.g., Audemard et al., 2006; Bushek et al., 2002; Hoese, 1962; Villalba et al., 2004; White et al., 1987). The buoyant free-living and metabolically active meront stage can survive

* Corresponding author.
E-mail addresses: gorka.bidegain@usm.edu, gorkabidegain@gmail.com (G. Bidegain).

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for at least two weeks in seawater (Chu et al., 2002) and infective elements are routinely identified in the water column (Audemard et al., 2006). Transmission probably occurs via an infective dose (Bushek et al., 1997; Chu and Volety, 1997; Ford, 1996; Powell et al., 1999). *P. marinus* cells divide and proliferate inside hemocytes facilitating dissemination throughout *C. virginica* tissues (Choi et al., 1989; McCollough et al., 2007; Saunders et al., 1993; Villalba et al., 2004) and heavily infected oysters may harbor tens of millions of *P. marinus* cells (Choi et al., 1989; Saunders et al., 1993).

Dermo disease currently is characterized by high prevalence over large geographic regions (Powell and Kim, 2015; Powell, 2016) strongly modulated by environmental conditions, particularly salinity, and is marked by widespread and rapid transmission (Powell and Hofmann, 2015). This type of transmission minimizes the importance of transmission in the population dynamics of the diseased host population and consequently, the most common models developed to study Dermo disease dynamics are proliferation-based models for *P. marinus* (Ragone Calvo et al., 2001; Hofmann et al., 1995; Powell et al., 2011, 2012b) that parameterize transmission as a function of density of infected animals in the population or simply assume rapid infection post-settlement. However, high host densities may prevent disease spread by consuming enough parasites that the concentration of infective particles decreases and individual host exposure is low enough to limit disease transmission (Bidegain et al., 2016a; Civitello et al., 2013). Unfortunately, transmission of *P. marinus* and other *Perkinsus* spp. in bivalves has received relatively little study (Ford, 1992; Ford and Smolowitz, 2007; Gray et al., 2009) and has been integrated only superficially into disease models (Powell et al., 1996, 1999).

The classic Kermack–McKendrick formulation for disease models (Anderson and May, 1991) in terms of contact-rate transmission has received little attention for marine diseases (Powell and Hofmann, 2015). Bidegain et al. (2016a,b) recently developed a series of susceptible-infected (*SI*)-based models describing theoretical cases for marine diseases. One limitation of their approach is the assumption that parameters take fixed values independent of time. The assumption of constancy in time has the advantage of simplifying the models, and facilitates use of the well-known basic reproduction number. However, both the prevalence of infection with *Perkinsus* spp. and the transmission of these parasites are strongly tied to temperature and salinity (Burreson and Ragone Calvo, 1996; Bushek et al., 2012; Hofmann et al., 1995). In temperate regions that experience broad annual temperature fluctuations, this dependence leads to a strong annual cycle of *P. marinus* proliferation (Andrews, 1988; Ford et al., 1999; La Peyre et al., 2008) exhibiting (i) an initial increase in the intensities of overwintering infections with increasing temperatures in early summer (Soniat, 1996; Ford et al., 1999; Bushek et al., 2012), (ii) parasite proliferation and oyster mortality increasing through the summer and early fall along with the release of parasites through the feces of live infected oysters and decaying tissues of moribund and dead oysters (Ford et al., 1999; Bushek et al., 2002, 2012), followed by (iii) increases of free *P. marinus* in the water column, leading to transmission (Ragone Calvo et al., 2003; Audemard et al., 2006; Bushek et al., 2012) and (iv) a period of remission over the winter and spring as both parasites and infected oysters die (Bushek et al., 1994; Ragone Calvo and Burreson, 1994; Burreson and Ragone Calvo, 1996). Such observations reinforce the importance for *P. marinus* transmission-based models to incorporate seasonal factors to gain a deeper quantitative understanding of the short- and long-term evolution of disease dynamics, and to better predict outbreaks.

In this paper, a seasonally-varying epizootiological model is developed adapting the Kermack and McKendrick (1927) epidemiological theory to the Eastern oyster–*P. marinus* system and incorporating time factors for known temperature-dependent parameters associated with the oyster, parasite, and disease transmission. In our implementation, the model structure and parameters reflect current understanding of Dermo disease dynamics. The model (1) captures changes in transmission as a function of oyster population density due to high intensity recruitment events and fishing, infective dose, and pathogen *in vivo* inactivation, (2) is verified and evaluated against field mortality data and (3) yields the basic reproduction number R_0 for Dermo. R_0 is used to explain the transition of Eastern oyster populations from a state under-which Dermo disease had limited influence to the current state of persistent and pandemic disease and consider the role played by fishing down of the stock in enabling this transition.

2. The model

2.1. Mathematical theory, model structure and assumptions

We formulate a single-population disease transmission model configured to simulate the dynamics of *P. marinus* transmission in Eastern oysters. Four adult classes or subpopulations of individuals are considered as host model variables: susceptible *S*, infected *I*, dead susceptible *DS*, and dead infected *DI* (Fig. 1 and Eqs. (2)–(5)). Parameterization of the model is standardized to represent a square meter of the environment, so that the model units for these variables are individuals per square meter.

Parasite transmission to new susceptible hosts occurs through filtration (at a rate f_S) of waterborne *P. marinus* cells released by infected and dead infected oysters at a *per capita* rate c_I and c_{DI} respectively and/or imported from an external source (Eq. (6)). An 'average' parasite load for infected (*I*) and dead infected (*DI*) animals is assumed since *P. marinus* cells can multiply rapidly within oysters (Ford et al., 1999; Saunders et al., 1993). The parasite load values used in the model are for a 76 mm-oyster with a moderately heavy or heavy infection intensity (i.e., Mackin 4 for *I* and Mackin 5 for *DI*) (Mackin, 1961; Choi et al., 1989) (see Table 1).

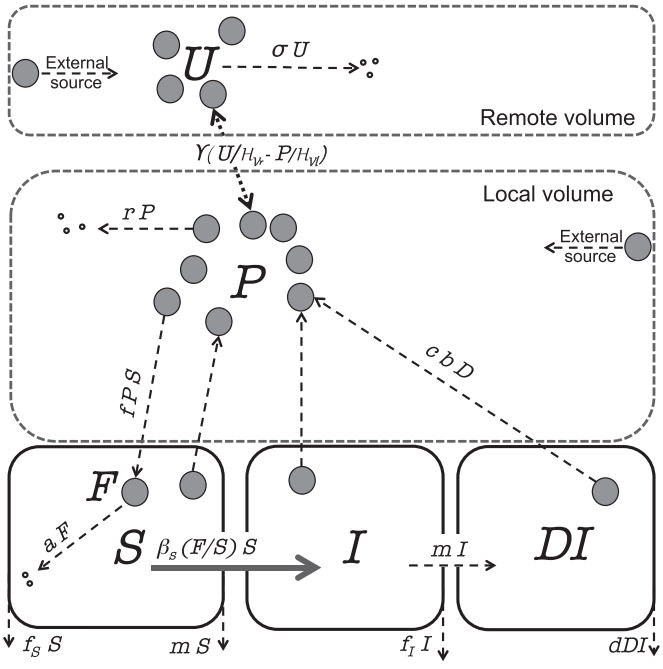

Fig. 1. Model flow diagram. The model variables are represented by capital letters: susceptible (*S*), infected (*I*), dead infected (*DI*) and dead susceptible (*DS*) individuals, local pool of pathogens (*P*), remote pool of pathogens (*U*), and cells accumulated in the susceptible population (*F*). Arrows represent the main processes in the model. The main equation terms and parameters involved in these processes are presented on the correspondent arrows and described in Eqs. (2)–(8) and Table 1.