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Review article

The ecology, evolution, impacts and management of host–parasite interactions of marine molluscs

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ABSTRACT

Molluscs are economically and ecologically important components of aquatic ecosystems. In addition to supporting valuable aquaculture and wild-harvest industries, their populations determine the structure of benthic communities, cycling of nutrients, serve as prey resources for higher trophic levels and, in some instances, stabilize shorelines and maintain water quality. This paper reviews existing knowledge of the ecology of host–parasite interactions involving marine molluscs, with a focus on gastropods and bivalves. It considers the ecological and evolutionary impacts of molluscan parasites on their hosts and vice versa, and on the communities and ecosystems in which they are a part, as well as disease management and its ecological impacts. An increasing number of case studies show that disease can have important effects on marine molluscs, their ecological interactions and ecosystem services, at spatial scales from centimeters to thousands of kilometers and timescales ranging from hours to years. In some instances the cascading indirect effects arising from parasitic infection of molluscs extend well beyond the temporal and spatial scales at which molluscs are affected by disease. In addition to the direct effects of molluscan disease, there can be large indirect impacts on marine environments resulting from strategies, such as introduction of non-native species and selective breeding for disease resistance, put in place to manage disease. Much of our understanding of impacts of molluscan diseases on the marine environment has been derived from just a handful of intensively studied marine parasite–host systems, namely gastropod–trematode, cockle–trematode, and oyster–protistan interactions. Understanding molluscan host–parasite dynamics is of growing importance because: (1) expanding aquaculture; (2) current and future climate change; (3) movement of non-native species; and (4) coastal development are modifying molluscan disease dynamics, ultimately leading to complex relationships between diseases and cultivated and natural molluscan populations. Further, in some instances the enhancement or restoration of valued ecosystem services may be contingent on management of molluscan disease. The application of newly emerging molecular tools and remote sensing techniques to the study of molluscan disease will be important in identifying how changes at varying spatial and temporal scales with global change are modifying host–parasite systems.

Contents

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1. Introduction

> *"Most biological studies, especially in ecology and evolution, have been done on free-livers. That is, the great bulk of our knowledge of biology comes from studying the minority of species!."*
>
> [Windsor, 1998]

1.1. Host–parasite ecology

The study of diseases and related epidemiological theory in aquatic, and especially marine ecosystems is relatively new, when compared to terrestrial ecosystems (e.g., Harvell et al., 1999, 2002), and the foundation of a lot of the relevant general ecological theory is derived from our long-standing overlap between human and animal hosts (either hunted or later cultivated) and our need to comprehend related diseases (e.g., Harvell et al., 2002; McCallum et al., 2004).

It took till the 1960s for ecologists, and behavioral and evolutionary biologists to begin to embrace the existing parasite literature and to couch the existing observations more broadly, in terms of mounting ecological and evolutionary hypotheses and theory. As this science on host and parasite interactions in the above perspective grew, the perspective expanded, not just on single species parasite–host systems where the host is in essence the 'habitat' (or island, Kuris et al., 1980; but see Lawton et al., 1981) for the parasite, but an ever increasing vision of higher ecological levels of complexity from individuals to even ecosystems. With this expanding interpretation the perspective also expanded spatially, from a single host as a parasite's 'world' to metapopulations, populations, expanding from meters to kilometers and even greater spatial ranges from continents or oceans to global terrestrial, freshwater, and marine biogeographical provinces (e.g., Lafferty et al., 2005, 2010; Morand and Krasnov, 2010; Poulin et al., 2011; Byers et al., 2014; Hopper et al., 2014; Wood et al., 2015). These patterns have been exacerbated significantly by global parasite and host introductions (e.g., Thieltges et al., 2009; Sorte et al., 2010; Sousa et al., 2014). Furthermore studies across natural (e.g., productivity), and anthropogenic gradients of eutrophication, fishing, and disturbance often suggest strong positive relationships between environmental gradients and parasite abundance (e.g., Johnson and Carpenter, 2008; Morand and Krasnov, 2010; Poulin et al., 2011; Lafferty and Harvell, 2014; Wood et al., 2015).

Since the 1950s and particularly in the early 21st century, marine research on host–parasite interactions, and related diseases, has rapidly advanced in scope. Initially, marine host–parasite ecology was to a large extent generally descriptive. It emphasized parasite and host abundance patterns, without directly addressing more complex and often difficult 'ecology' within- or among-hosts and parasite communities (e.g., Ricklefs, 2010; Lafferty and Harvell, 2014; Lafferty et al., 2015). With time, however, marine research has expanded to address:

(1) The biodiversity of marine parasites and their hosts (e.g., Mouritsen and Poulin, 2002a; Hechinger and Lafferty, 2005; Kim et al., 2005; Lafferty and Harvell, 2014).
(2) The role of parasites in food webs (e.g., Lafferty et al., 2008; Byers, 2009; Sonnenholzner et al., 2011; Dunne et al., 2013; Lafferty, 2013; Thieltges et al., 2013; Lafferty and Harvell, 2014).
(3) The relationship between environment, both current and future (climate change), and parasitism and disease (e.g., Harvell et al., 1999; Kim and Powell, 2009; Soniat et al., 2009; Burge et al., 2014).
(4) The relationship between parasites and disease and other natural and anthropogenic stressors (i.e. hurricanes and

other man-made perturbations such as eutrophication and contaminants; Harvell et al., 1999, 2004; Lenihan et al., 1999; Hine et al., 2002; Lafferty and Kuris, 2005; Kim and Powell, 2006; Munroe et al., 2013).

(5) Disturbance agents that influence parasitism and disease, such as fishing and dredging (e.g., Huspeni and Lafferty, 2004; Hine, 1996; Cranfield et al., 1999, 2003, 2005; Powell et al., 2012a).

(6) The role of non-native (exotic) species introductions in influencing spatial and temporal patterns of parasites and disease (e.g., Harvell et al., 2002; Padilla and Williams, 2004; Padilla et al., 2011; Simberloff and Rejmánek, 2011; Burge et al., 2014; Lafferty and Harvell, 2014; Lafferty et al., 2015).

(7) Examples of how parasites and disease have impacted native foundation or ecosystem engineers or biodiversity in complex ways (e.g., Naylor et al., 2001; Ruesink et al., 2005; Byers et al., 2006; Lambrinos, 2007; Molnar et al., 2008; Griffen, 2009; Sousa et al., 2009; Dumbauld et al., 2011).

Numerous lab and field studies of natural systems, along with related experiments have now shown that parasites can impact significantly their host population's life history and behavior (e.g., Barnard and Behnke, 1990; Byers et al., 2008), as well as related community structure through, for example, species interactions (e.g., Rollinson and Anderson, 1985; Price et al., 1986). In some cases these interactions can even alter overall ecosystem functioning (e.g., Mouritsen and Poulin, 2002a; Thomas et al., 2005; Lafferty and Harvell, 2014) potentially through changes in the abundance of foundation or ecosystem engineering species, or the modification of traits associated with habitat provisioning or overall ecosystem engineering (e.g., Thomas et al., 1998, 2005; Dumbauld et al., 2011; Hatcher and Dunn, 2011; Hatcher et al., 2014; Lafferty and Harvell, 2014). Generally, it has been assumed that pathogens and parasites influence the above relationships by affecting life history functions (e.g., growth, reproduction), survival, or via indirect interactions or manipulations (e.g., Toft, 1991; Bush et al., 2001; Moore, 2002; Hatcher et al., 2006, 2014; Lafferty and Kuris, 2009; Hatcher and Dunn, 2011; Lafferty and Harvell, 2014).

McCallum et al. (2004) include a set of differences between marine and terrestrial systems such as: (1) a greater species richness of both hosts and parasites in marine than terrestrial systems; (2) more open life histories (e.g., recruitment) in marine than terrestrial systems; (3) differing modes of parasite transmission (e.g., Bower et al., 1994; Bushek et al., 2002); (4) a greater extent of anthropogenic impact for terrestrial than marine ecosystems and, finally; (5) differences in potential methods and the ability to control native and introduced diseases in the two very broad environments. Based on their open systems, the host–parasite systems of sessile invertebrates and algae may be more similar to plants and their pathogens than animal–pathogen systems within the terrestrial realm. For parasite–host systems in estuarine and marine environments, abiotic factors (e.g., temperature, salinity, dissolved oxygen, etc.) are also particularly critical in shaping the above host–parasite interactions and we focus our attention below on these also.

1.2. Molluscan parasites and diseases

Work on molluscan host–parasite dynamics originated with the study of micro- and macro-parasites in freshwater and terrestrial systems. Gastropods, in particular, were identified early on as hosts for a diversity of parasites under study (Bayne, 1983; Fried, 1997), including intermediate hosts and transmitters of terrestrial and freshwater trematodes (e.g., Foster, 1958; Becker, 1980; Fernandez and Esch, 1991a, 1991b; Esch and Fernandez, 1994; Adema and Loker, 1997; Thompson, 1997; Schmidt and Roberts, 2000). However, interest in marine species followed much later (e.g., Gambino, 1959; Cheng, 1967; Yoshino, 1975; Tallmark and Norrgren, 1976; Lauckner, 1984, 1987; Kuris, 1990; Sousa, 1993; Grosholz, 1994; Mouritsen and Jensen, 1994; Jensen et al., 1996; Calvo-Ugarteburu and McQuaid, 1998; Thomas et al., 1998; Curtis, 2002).

For freshwater and marine molluscan parasitology and related fields (e.g., immunology, physiology, evolution, etc.) there were numerous forerunners in these fields including (though not meant to be exhaustive): J.D. Andrews, B.L. Bayne, S.M. Bower, E.M. Burreson, T.C. Cheng, A. Choi, J. Couch, X. de Montaudouin, R.A. Elston, G.W. Esch, A. Farley, A. Figueras, S.E. Ford, B. Fried, H. Grizel, H.H. Haskins, A.H. Hine, S. Hopkins, P. Korringa, A. Kuris, L. Leibovitz, R. Lester, S.E. McGladdery, J.G. Mackin, E.H. Michelson, R.C. Newell, R. Olsen, R. Overstreet, F.O. Perkins, E.N. Powell, S.M. Ray, C.J. Sinderman, A. Sparks, V. Sprague, L. Stauber, and the graduate students and post-doc cohorts that followed them.

Much research on mollusc–parasite interactions has focused on the role of molluscs as vectors for diseases of humans. Often overlooked, by comparison, is the role that molluscan host–parasite interactions play in shaping estuarine and marine ecology, and economically and ecologically important shellfisheries. As habitats provided by estuarine and marine molluscs are increasingly under threat from a multitude of stressors and food security is an increasingly important global issue, understanding ecological impacts of molluscan parasites and disease is an imperative.

Many molluscs serve as ecosystem engineers, organisms that create, significantly modify, maintain or destroy native and non-native habitats (e.g., Jones et al., 1994; Gutiérrez et al., 2003; Byers et al., 2006; ASMFC, 2007; Sousa et al., 2009). For example bivalve-dominated reefs are not only of high biodiversity value but help to maintain water quality, stabilize shorelines (e.g., mangroves and marsh), enhance fisheries productivity, and cycle nutrients (e.g., Grabowski et al., 2012). Burrowing bivalves can, by bioturbating sediments, modify oxygen concentration, sediment porosity, stability, metal concentration and pH, thereby making sediments more or less habitable to other organisms (Ciutat et al., 2007). Gastropod grazers can exert significant top-down pressure on the biofilms of soft and hard substratum, influencing development of macroalgal communities (Mouritsen and Poulin, 2006). Hence, parasites which influence the distribution, abundance and/or behavior of molluscan species have the potential to have large-scale impacts on communities and ecosystems.

Recent studies suggest that molluscan shellfish habitats may be among the most endangered marine habitats on the planet (Beck et al., 2009, 2011). Overharvesting for food and lime, and habitat destruction associated with the construction of ports and coastal settlements, has decimated populations to, in some cases, less than 10% of their levels prior to industrialization (Beck et al., 2009, 2011). Although disease was perhaps not identified as the driving factor in this early loss of shellfish reefs (prior to the 1960s, Coen and Luckenbach, 2000), it is now seen by some as a significant factor limiting recovery and restoration efforts (cf. Coen and Luckenbach, 2000; Baggett et al., 2015; Coen and Humphries, in press). As interest in protection and restoration of shellfish habitats grows around the world (e.g., Laing et al., 2006; Coen et al., 2007; Beck et al., 2009, 2011; Lallias et al., 2010; Woolmer et al., 2011, *see* http://www.oyster-restoration.org/oyster-restoration-research-reports/ also, last accessed 30 May 2015), it is important to understand how disease impacts the structure and function of these valuable habitats, and how restoration projects can be designed to enhance disease resilience in instances where it is hampering recovery (Coen and Luckenbach, 2000).

Molluscan shellfish aquaculture is replacing wild fisheries either as intensive or semi-intensive aquaculture (e.g., Goulletquer and Heral, 1997; Nell, 2001; Beck et al., 2009, 2011; Groth and Rumrill, 2009). In terms of tonnage, molluscan mariculture now accounts for 35.87% of world aquculture production, with finfish contributing 9.91% (Shinn et al., 2015). Cultivated molluscs in some cases provide many of the same ecosystem services as those of wild shellfish-dominated ecosystems (e.g., filtration; Dumbauld et al., 2009; Bostock et al., 2010; Coen et al., 2011), and the disease dynamics of cultivated and wild populations are often inextricably linked. Diseases and pests are now a major problem for cultivated populations worldwide, where huge investments are made in hatcheries, nurseries, grow-out leases, capital equipment and labor (e.g., Kraeuter and Castagna, 2001; Creswell and McNevin, 2008; Dumbauld et al., 2009; Elston and Ford, 2011; Diana et al., 2013; Shinn et al., 2015). For example, Shinn et al. (2015) estimate that in the UK, parasites account for an annual loss of 5.8–16.5% of the value of aquaculture production across all species, in freshwater and marine systems. As food security becomes an increasing issue for the burgeoning human population, there is increasing pressure to reduce production losses due to disease.

Since the 1950s perhaps, diseases linked to mortality of bivalves, especially protozoans such as *Perkinsus*, *Haplosporidium*, *Marteilia*, and *Bonamia* (e.g., Fernández Robledo et al., 2014), but also others more recently such as viruses, bacteria, phages, macroparasites, non-native species, and even other molluscs have been the focus of numerous studies on parasites, as well as environmental triggers and aspects of the hosts (e.g., Getchell, 1991; Lauckner, 1983; Grizel et al., 1986; Gibbons and Blogoslawski, 1989; Sinderman, 1990; Ford and Tripp, 1996; Ford, 2001; Ford et al., 2002; Burreson and Ford, 2004; Paillard et al., 2004; Renault and Novoa, 2004; Lorenz, 2005; McGladdery et al., 2006; Elston and Ford, 2011; Carnegie and Burreson, 2012).

A number of reviews have focused on the topic of molluscan diseases (see related papers in this volume also). However, the majority of these have either been: (1) inventories of the parasites and diseases influencing particular groups of molluscs (Cheng, 1993; Fryer and Bayne, 1996; AFS-FHS, 2005; Bower, 2006); (2) overviews of specific parasite–host interactions, particular those concerning commercially important species (e.g., Burreson and Ford, 2004; Paillard, 2004; Villalba et al., 2004; Arzul and Carnegie, 2015); or (3) overviews of molluscan diseases that impact humans (e.g., Rippey, 1994; Wittman and Flick, 1995).

Similarly, few reviews have summarized the ecology and diseases of freshwater molluscs (Dillon, 2000, 2006), especially for those groups that are ecologically important such as freshwater bivalves, especially mussels (see Grizzle and Brunner, 2009). Although several excellent papers have provided overviews of how parasites shape marine ecology (e.g. Poulin, 2002; Lafferty and Harvell, 2014), to our knowledge there is no single review that specifically focuses on how parasites influence the ecology of marine molluscs, spanning scales of populations to ecosystems, and considering disease dynamics between wild and cultivated populations.

The need for a review examining ecological impacts of diseases on molluscs is particularly pertinent given coastal development and climate change are increasingly modifying disease cycles, by modifying the ecology of hosts, parasites and their interaction. The Millenium Ecosystem Assessment (MEA, 2005) identified important interactions among disease emergence, climate regulation and ecosystem health and highlighted important related services. Services provided by aquatic ecosystems that are being degraded by a combination of factors include: (1) provision of freshwater for drinking, and other uses; (2) fisheries; (3) air, water and regional climate control; and (4) the regulation of natural threats including 'pests' and diseases. All told, the MEA suggested that perhaps 60% of identified 'ecosystem services' that support all life on the planet have been or are being degraded. Continued modification of these systems will exacerbate future sudden changes in the appearance of new diseases, water quality, the collapse of native fisheries, and regional climate shifts, often in unpredictable paths.

Coincident with MEA report (2005) is a growing body of basic and applied research relating to diseases and host–parasite interactions at all levels of ecological complexity (e.g., Thomas et al., 2005; Poulin, 2002, 2007; Hatcher and Dunn, 2011; Johnson and Paull, 2011; Poulin et al., 2011; Hughes et al., 2012; Lafferty and Harvell, 2014; Dunn and Hatcher, 2015). For instance, we know from the literature that wild populations can serve as reservoirs for infections, that also affect aquaculture, and also aquaculture conditions could amplify diseases resulting in "disease spillback" to wild populations (e.g., Bishop et al., 2006; Kelly et al., 2009; Peeler and Feist, 2011; Poulin et al., 2011). Emerging diseases, new diseases or diseases rapidly expanding in geographic extent or incidence (Okamura and Feist, 2011), are linked to habitat fragmentation, species range shifts, food web modification, climate change and other anthropogenic stressors and contaminants such as pollutagens, metals, antibiotics, PAHs, HABs, pH, CO_2, extreme temperatures and salinities, and low dissolved oxygen (e.g., Harvell et al., 1999, 2002; Powell et al., 1999b; Lafferty et al., 2004; Johnson and Paull, 2011; Patterson et al., 2014; Breitburg et al., 2015).

1.3. This review

This review considers ecological and evolutionary impacts of mollusc–parasite interactions. In fitting with the focus of this Special Issue on Marine Molluscs, our focus is primarily on gastropods and bivalves of marine waters. However, given the importance of many gastropod and bivalve species not only as commercially harvested species but also as key determinants of biodiversity, we consider both gastropods and bivalves in wild settings, irrespective of whether they have commercial value as a shellfishery, as well as those that are being cultivated in intensive or semi-intensive aquaculture. Although our focus is on estuarine and marine species, in some instances we also draw on freshwater examples to illustrate concepts where marine examples are not available and we expect similar processes to operate across biomes.

Our review considers the direct effect of molluscan parasites on their hosts and the populations, communities and ecosystems of which they are a part. It also considers the indirect effect of molluscan parasites that arise as a consequence of management strategies put in place to manage them. As impacts of molluscan parasites are shaped by the spatial and temporal distributions of parasites, hosts and their interaction, we start by summarizing the diversity and nature of host–parasite interactions, and the spatial and temporal patterns of parasites and disease. We consider the abiotic and biotic drivers, including vectors and aquaculture, of these patterns. We go on to examine how parasites impact the growth, survivorship, reproduction and morphological and behavioral traits of individuals and then cascade to influence the ecological patterns and processes at scales ranging from populations to ecosystems. We also include discussions on modeling of oyster diseases and host systems (see Powell and Hofmann, 2015 for further discussion in this volume), and existing and potential management strategies for disease-afflicted wild and cultivated molluscan populations, such as selective breeding and non-native species introductions, and their potential and realized impacts. We conclude with a discussion of emerging diseases, novel approaches, current and novel stressors (including climate change), and their potential ecological impacts. We highlight major research gaps and opportunities.

2. Brief overview of eitiological agents of molluscan diseases

2.1. Some relevant disease definitions

Ecologists and parasitologists have for quite some time deliberated over appropriate terms and definitions spanning the gap between these too often considered distinct disciplines. In fact, several review papers actually resolve some of the issues amongst the two disciplines (Margolis et al., 1982; Bush et al., 1997). We provide the following definitions to ensure that the way we employ these terms is consistent throughout. **Pathogens** (sometimes also referred to as **etiological agents** or **infectious agents**) are disease causing agents including viruses, bacteria, fungi, rickettsiae, protozoans, and metazoans. Eukaryotic (protozoa, helminths, other metazoans) pathogens have traditionally been called "parasites" (see also Carella et al., 2015). However, the term parasite is often used more liberally to include bacteria and viruses too. Here, we use the term in its broader sense. We define a **parasite** as an organism that lives in or on another living organism (i.e. host) deriving some benefit from it. Parasites differ from predators in that they attack a single victim during their life-span, whereas predators attack multiple victims (cf. Lafferty and Kuris, 2002 for further insight). **Microparasites** (such as viruses and bacteria) complete their full life cycle within one host whereas **macroparasites** (such as nematodes, cestodes, other metazoans such as helminths, arthropods, primarily) reproduce by infective stages outside the host and are large enough to be seen with the naked eye. Most parasite species are obligate parasites, requiring one or more hosts during their life cycle (e.g., Rohde, 2005; Hatcher and Dunn, 2011; Lafferty and Harvell, 2014) and we focus on these for our review.

We define **disease** as "a negative deviation from normal health, demonstrated by reduced function, changes in form, or both" (cf. Grizzle and Brunner, 2009). Reduced function can encompass reduced feeding, escape from predators or diminished competition, and fitness characteristics such as reproduction, growth, and/or survival. **Infectious diseases** are those caused by one or more biological organisms and that are capable of being transmitted to another host, while **non-infectious diseases** are caused by natural or anthropogenic stressors (chemical or physical), reduced nutritional quality or quantity, or genetic anomalies caused by the aforementioned (Cheng, 1993; Schleyer, 1991). We focus our discussion on infectious diseases.

Transmissibility is the ability to spread a disease or parasite to other organisms. It generally refers to an infected conspecific host, individual, or group spreading a parasite to another conspecific individual or group, irrespective of whether these were previously infected with a parasite or pathogen. Organisms that transmit diseases as pathogens to other organisms are referred to as **vectors**. Pathogens (parasites) can be transmitted **horizontally** (between individuals) and **vertically** (from parent to offspring). **Generalist** parasites can use several to many host species, whereas **specialist** parasites specialize in one or a few host species. **Trophically-transmitted** parasites have complex life cycles with two or more stages that are spread via predator–prey interactions. These typically include Platyhelminthes such as the trematodes, cestodes, nematodes, and acanthocephalans worms. **Directly-transmitted** parasites are species that are spread among conspecific hosts and include crustaceans and monogenean trematodes (Wood et al., 2015). An **enzootic** disease is constantly present in a population, but usually generally affects only a small portion of the 'population' at any one time and at a frequency that is expected in that given time period. An **epizootic** is an epidemic level outbreak of disease in a population at a frequency greater than expected in that given time frame.

A parasite's capacity to initiate a given disease and related parasite production is termed **pathogenicity**, whereas **virulence** is the degree of pathogenicity as indicated by the severity of a given disease and its capacity to invade the host's tissues overall (cf. Fuxa and Tanada, 1987; Chintala et al., 2002). **Prevalence** and **intensity** are two terms commonly used to describe patterns of infection. **Prevalence** is the proportion of individuals in a population having a parasite or disease. **Intensity** is the parasite load within a given infected host. How virulence is maintained or increased given that intuitively increased virulence should decrease host fitness through increased pathogenicity and even death has long been deliberated those who study the field (e.g., Ewald, 1983; Frank, 1996; Sorensen and Minchella, 2001; Choo et al., 2003; Lafferty and Kuris, 2009; Alizon and Michalakis, 2015).

2.2. Ecological and environmental working definitions

In the ecological literature, a **keystone species** (*sensu* Paine, 1966) is one whose impacts within its community or ecosystem are larger and greater than would be expected from either relative abundance or overall biomass. A **foundation species** (*sensu* Dayton, 1972) is a species found at any tropic level that has a disproportionately large influence on the rest of the community or ecosystem through its modification of its environment and influence other species. An **ecosystem engineer** (e.g., Jones et al., 1994) is an organism that creates, significantly modifies, maintains or destroys a native or non-native habitat. All three of these terms have been used to describe several different molluscan activities from oysters (e.g., Coen et al., 1999a, 1999b, 2007; Lenihan, 1999; Coen and Luckenbach, 2000; Grabowski and Peterson, 2007) to giant clams (Neo et al., 2015). **Trophic cascade** (an indirect interaction chain after Carpenter et al., 1985) relates to the impact generally of a predator on its prey's ecology that eventually filters down to at least one or more feeding levels, ultimately affecting prey density and/or behavior down one or more of those levels.

"**Stress**" occurs when an organism is moved from beyond the bounds of its ability to adjust. It is generally regarded as a perturbation with a negative effect. Stress has often been defined as "any environmental factor or several factors that limit a given species fitness characteristics including survival, growth, reproduction, etc. (e.g., Menge and Sutherland, 1987; Menge and Branch, 2001; Elliott and Quintino, 2007). Environmental factors that do not reduce species fitness should not be assumed as stressors. A **stressor** is an abiotic or biotic factor that is the cause of stress. As is obvious there is an inherent degree of circularity as to the definitions of the terms "stress", "stressor", and "stress response". Organisms respond to stressors by initiating some sort of an evolved response that can be physiological and/or behavioral. A **driver** is something that causes change over time. Stress can become a driver, but if the stress is eliminated it may not initiate any change (e.g., Akberali and Trueman, 1985; Elliott and Quintino, 2007).

2.3. Parasites of molluscs

Of marine organisms, molluscs are the next best known taxonomic group after fishes in terms of parasites. The types of parasites that lead to molluscan diseases include: protists, bacteria, viruses, invertebrates such as trematodes and pea crabs, with modes of transmission that range from direct to indirect. The best studied of molluscan–parasite interactions are those that: (1) involve well-studied or abundant species in areas with laboratories, field stations; or (2) influence commercial fisheries value or

human health (e.g., Dillon, 2000; Grizzle and Brunner, 2009; Elston and Ford, 2011; Lafferty et al., 2015).

Among the more common macroparasites of mollucs are:

(1) Small, thin-shelled decapods living in the mantle cavities of numerous molluscs (e.g., Palmer, 1995; Hsueh, 2003; Asama and Yamaoka, 2009; Becker, 2010; Becker and Türkay, 2010; Byers et al., 2014; Neo et al., 2015).
(2) Boring sponges that attach to oysters in particular, but also to other molluscs (e.g., Korringa, 1951; Hopkins, 1956, 1962; Wells, 1959; Guida, 1976; Nicol and Reisman, 1976; Thomas, 1979; Schleyer, 1991; Wesche et al., 1997; Rosell et al., 1999; Stefaniak et al., 2005; Carver et al., 2010; Le Cam and Viard, 2011; Dunn et al., 2014).
(3) Shell-boring polychaete worms (e.g., family Spionidae) that cause shell anomalies that may alter the effectiveness of this protective exoskeleton (Vermeij, 1978, 1983a,b, 1987) and that may reduce host condition (e.g., Lunz, 1941; Loosanoff and Engle, 1943; Korringa, 1951; Murina and Solonchenko, 1991; Schleyer, 1991; Handley, 1997a, 1997b, 1998; Diez et al., 2013; Neo et al., 2015).
(4) Trematodes, a class within the phylum Platyhelminthes. These are internal parasites of molluscs, which can be divided into two subclasses of trematode: Aspidogastrea (~80 species) and Digenea (~18,000 species). Some aspidogastrid species complete their entire life cycle within a given individual mollusc. In contrast, the Digenea, have complex life-histories involving multiple hosts. Molluscs (typically gastropods, but in some instances bivalves too) serve as intermediate hosts, in which sexual reproduction occurs. Vertebrates (e.g., especially fish and birds) are the definitive host, in which sexual reproduction occurs also (reviewed in Grizzle and Brunner, 2009).
(5) Gastropods, including members of the Pyramidellidae (e.g., *Boonea* and *Turbonilla*), the Eulimidae (e.g., genera *Cancellaria and Thyca*), and nine other families, that are either ectoparasites with long modified proboscides which they use for piercing and sucking of host tissue or endoparasites with a reduced or absent radula (reviewed in Lorenz, 2005). These gastropods (see Section 2.4 below) may have major impacts on the growth and survivorship of their wild and cultured molluscan hosts, including oysters, giant clams, limpets and other snails (e.g., Cumming and Alford, 1994; Boglio and Lucas, 1997; Carroll and Finelli, 2015). For example, the gastropod *Boonea impressa*, which feeds on *Crassostrea virginica* and other species, may reach densities as high as 1500 m^{-2} in the southeastern U.S.A. (L. Coen, unpublished data).

Among the more common microparasites of molluscs include:

(1) Protistan diseases, which may have direct or indirect transmission. These are particularly common among marine bivalves and typically come from the genera: *Perkinsus*, *Bonamia*, *Haplosporidium*, *Marteilia*, *Mikrocytos*, *Minchinia*, and *Paramyxa,* along with labyrinthulids (e.g., QPX) (e.g., Lauckner, 1980, 1983; Ford and Tripp, 1996; Bower, 2006; Bower et al., 1994; Carnegie, 2005; McGladdery et al., 2006; Soudant et al., 2013; Carrasco et al., 2015).
(2) Bacterial diseases of particular importance here include *Vibrio tubiashi*, a problematic species in hatcheries (e.g., Elston et al., 2008); and *V. tapetis* (Brown Ring Disease in Manila clams) and *V. splendidus* in *C. gigas* (e.g., Paillard et al., 2004; Travers et al., 2015).
(3) Viruses, such as the norovirus and Hepatitis A, which are acquired by bivalves during feeding in contaminated waters. Like bacterial diseases, they may be vertically transmitted to humans during consumption of raw molluscs. Other viruses, such as oyster herpes virus (OSV-1), which infects Pacific oysters, *Crassostera gigas*, are not transmissible to humans (e.g., Elston, 1997; Renault and Novoa, 2004; Munn, 2006; Mineur et al., 2014; da Silva et al., 2008; Renault, 2008; Degremont and Benabdelmouna, 2014; Mineur et al., 2014; see also Travers et al., 2015).

The first line of defense of molluscs against parasites and predators is the shell itself. However, pathogens that by-pass this line of defense, through shell penetration or ingestion by the host mollusc, will trigger the molluscan immune system that responds to invaders and other foreign materials in much the same way as the vertebrate immune system (Cheng, 1984; Allam and Raftos, 2015). The immunological system involves cellular responses such as phagocytosis and encapsulation, shell overgrowth (pearls), atrophy, necrosis and tissue responses (e.g., Fryer and Bayne, 1996; Lane and Birkbeck, 2000; Bayne et al., 2001; Loker et al., 2004). There are several types of hemocytes (phagocytic cells) in bivalve molluscs, most of which are either granulocytes or agranulocytes (e.g., Chu, 2000). A recent excellent review by Soudant et al. (2013), although focussed on *Perkinsus* spp. and bivalve host–parasite interactions, summarizes much of the more recent and relevant hemocyte and immunological literature.

2.4. Molluscs as parasites

In addition to serving as hosts for parasites (see Section 2.3 above), quite a few molluscs are, themselves, parasites (including of other molluscs). Most molluscan parasites are in the Class Gastropoda, and many are ectoparasites. Some ectoparasitic gastropods (e.g., Eulimidae) have 'jaw-like' modifications that pierce a host's tissue, enabling direct feeding on hemolymph. They may display significant sexual dimorphism unlike any other gastropods and, in some instances, form protective galls like terrestrial insects (Warén, 1983). Other ectoparsitic gastropods have a long proboscis that pierces and sucks host tissue (e.g., Lorenz, 2005). For example, *Cancellaria cooperi* uses its proboscis to suck blood from its host, the California electric ray, *Torpedo californica* (O'Sullivan et al., 1987; Bush et al., 2001). There are also examples of gastropods that are endoparasites of other molluscs, sponges, cnidarians, annelids echinoderms, and even fishes. Many have significant anatomical modifications such that they are almost unrecognizable as molluscs (e.g., Cheng, 1967; Bush et al., 2001).

By contrast, there are relatively few examples of bivalves that have taken up a parasitic lifestyle. Where they have, it is as larvae (i.e. glochidia in unionid clams) that attach to fish gills (e.g., Dillon, 2000; Graf and Cummings, 2007; Grizzle and Brunner, 2009).

3. Distributions of diseases, their prevalence and intensity: Patterns and processes

The distribution, prevalence and intensity of diseases shape their ecological and evolutionary impact on hosts. Diseases, by definition, require the presence of both a host and one or more etiological agent(s) (see also Carella et al., 2015). Spatial and temporal patterns of disease reflect the niche of the host, the niche of the etiological agent (e.g., macro- or micro-parasite), transportation vectors of the etiological agent, environmental factors that influence immune-competence of the host, the life history of the etiological agent and host, as well as biological interactions with other species. The strength of these factors is scale dependent,

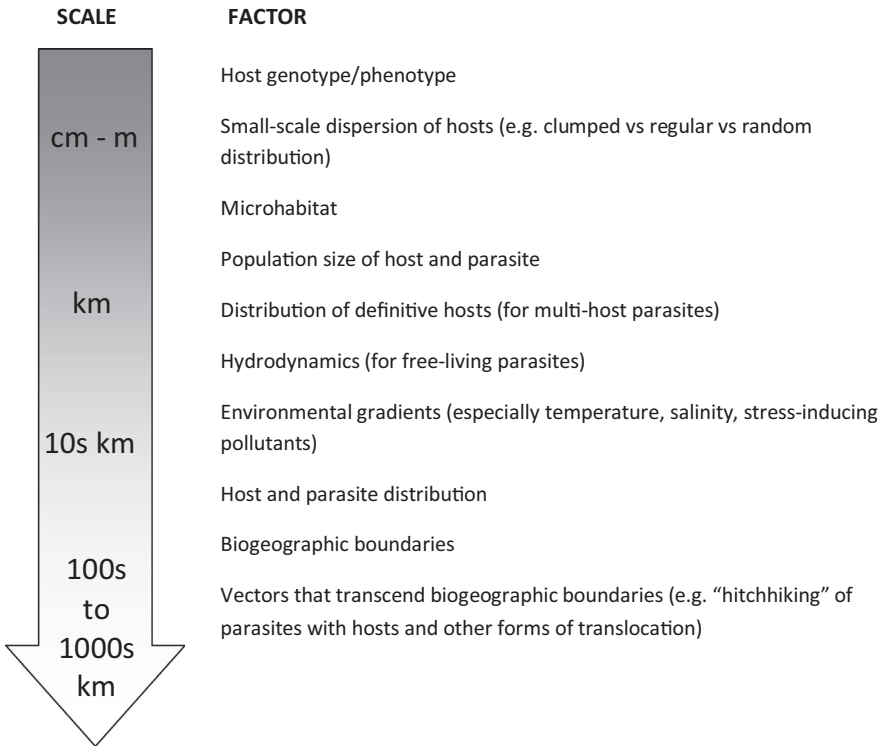

Fig. 1. Summary of the factors that influence the distribution of molluscan diseases at scales ranging from centimeters to thousands of kilometers.

varying from centimeters to thousands of kilometers, and days to years (Fig. 1; Hess et al., 2002; Holt and Boulinier, 2005).

In this section, we provide an overview of the spatial and temporal scales across which molluscan diseases vary, and compare patterns of disease between wild and cultivate populations of molluscs, and native and non-native species. We then consider how vectors, abiotic and biotic factors contribute to these patterns. Spatial and temporal patterns of disease are increasingly being modified by coastal development and climate change (Harvell et al., 2002, 2009; Ruiz-Moreno et al., 2012; Burge et al., 2014; Lafferty et al., 2015).

3.1. Spatial variation

In many instances, local (small) scale factors appear to play bigger roles in determining how many parasites are present in molluscs, and their prevalence, than biogeographic factors (e.g., Poulin and Mouritsen, 2003; Byers et al., 2008, 2014). Despite predictions that the number of diseases might be greater at low latitudes where species richness (and hence, presumably, diversity of hosts and parasites) is greatest, there is relatively little support for biogeographic gradients in the parasites of marine taxa, including molluscs (e.g., Rohde, 1992; Mouritsen et al., 2003; Lafferty et al., 2005; Byers et al., 2008; Thieltges et al., 2009; but see Poulin and Mouritsen, 2003; Rohde, 2005).

Within host populations, many disease-causing parasites display an aggregated distribution. In a cross-site analysis of four parasite species harbored by the cockle, *Cerastoderma edule*, Thieltges and Reise (2007) found that most individuals harbored no or a few parasite individuals (low prevalence). By contrast, a small percentage of individuals had high infection (intensity) levels. In most instances, *C. edule* harbored not just one species of parasite, but multiple.

At scales of 100s of meters to kilometers, patterns of disease may be driven by the spatial distribution of hosts, and, in particular, for multi-species parasites, the distributions of definitive hosts (see Fig. 1; also Section 3.7 below, e.g., White et al., 1989; Wilson et al., 1992; Lagrue and Poulin, 2015).

At larger scales of kilometers or more, the expression of diseases often varies across natural or anthropogenically caused environmental gradients (see Section 7.2 below; Thieltges et al., 2009; Ford et al., 2012; Byers et al., 2014; Hopper et al., 2014). For example, Wilson et al. (1992) found that spatial and temporal variability in the prevalence and infection intensity of *Perkinsus marinus* in *C. virginica* populations in the Gulf of Mexico correlated well with contaminant body burdens. Somewhat differently, within Delaware Bay on the mid-Atlantic coast of the US, MSX activity generally increases along a gradient of increasing salinity (Haskin and Ford, 1982; Burreson and Ford, 2004; Carnegie and Burreson, 2012; Ford and Bushek, 2012).

At larger scales still, biogeographic barriers and host and parasite biogeographic ranges may determine distributions (e.g., Byers et al., 2008, 2015; de Montaudouin and Lanceleur, 2011; Wood et al., 2015). For molluscs, host distribution may reflect modes of larval development (e.g., Scheltema, 1989). For example, within the Ostreidae (true oysters), some genera are larviparous, brooding young (e.g., *Ostrea* and *Tiostrea* spp.), whereas other species are oviparous (e.g., *Crassostrea* and *Saccostrea* spp.) having planktonic larvae whose life cycle prior to settlement is partially at the whim of tides and currents (Jackson, 1974; Buroker, 1985).

3.2. Temporal variation

In addition to displaying spatial heterogeneity at scales of centimeters to hundreds to thousands of kilometers, many diseases display significant temporal heterogeneity across scales of days, months, seasons and years (e.g., Altizer et al., 2006; Hudson et al., 2006). Temporal variability might reflect the life cycles of hosts and parasites, or temporal variation in environmental

conditions. For example, ongoing climate change is affecting hosts, parasites and their interaction (e.g., Harvell et al., 2002, 2009; Burge et al., 2014). Likewise, coastal development is driving changes through time in disease, by influencing habitats for molluscs and their parasites, and abiotic conditions (e.g., Harvell et al., 1999; Lafferty et al., 2004).

Many diseases are highly seasonal in their effects (e.g., intensity), despite, in some instances, infecting molluscs year round. For example, Bonamiasis, a disease which affects *Ostrea* spp. and *Saccostrea* spp. (oysters) that is caused by protistan parasites of the genus *Bonamia*, typically has a well-defined infection peak that occurs either in the summer, or autumn months (Hine, 1991; Carnegie et al., 2008). Seasonal infection peaks are often the case for Dermo oyster disease (Bobo et al., 1997), as well as QX oyster disease (caused by *Martielia sydneyi*) which generally peaks during the Austral summer and early fall (Rubio et al., 2013).

Seasonality in diseases may reflect a temperature dependence of physiological and metabolic processes in molluscs (Shumway, 1996), including hemolymph circulation and elimination of foreign substances (e.g., Feng and Stauber, 1968; Feng and Feng, 1974; Barszcz et al., 1978; Yevich and Barszcz, 1983; Kim and Powell, 2007). Other environmental factors which influence immunecompetence of molluscs may also vary seasonally. For example, fresh-water pulses associated with seasonal rainfall weaken phenoxidase production in Sydney rock oysters, *S. glomerata* (Butt et al., 2006), leading to seasonality in outbreaks of QX disease (Rubio et al., 2013).

Very few long-term data sets exist with which to examine interannual and decadal patterns of change in molluscan disease at individual localities. Nevertheless, Bushek et al. (2012) describe a 21 year dataset of Dermo disease (*Perkinsus marinus*) in *Crassostrea virginica* in Delaware Bay, U.S.A. Analyses reveal a potential 7-year cycle in disease, with peaks associated with strong positive anomalies of the North Atlantic Oscillation (NAO), which brings warmer temperatures, heavy rainfall and lower salinities. Similarly, a 10-year study along the coast of California, U.S.A., revealed that significant population declines in the black abalone, *Haliotis cracherodii*, from a Rickettsiales-like prokaryote causing Withering Syndrome, was accelerated during El Niño events as compared to non-El Niño periods (e.g., Moore et al., 2000a; Raimondi et al., 2002; Friedman et al., 2014).

Syntheses of disease records, amassed across multiple studies, reveal poleward range expansions of several molluscan diseases, coincident with climate warming (discussed in Burreson and Ragone Calvo, 1996; Ford and Tripp, 1996; Allison et al., 2011; Burge et al., 2014). In the mid-1980s, the distribution of Dermo disease expanded northward of its historic distribution between the Gulf of Mexico and the mid-Atlantic coast to extend to Great Bay, New Hampshire, U.S.A. (e.g., Ford, 1996; Burge et al., 2014, and references therein). Similarly, MSX disease, caused by the introduced protozoan *Haplosporidium nelsoni*, spread northwards following its initial introduction to the U.S.A. mid-Atlantic coast in the 1960s (e.g., Burreson and Ford, 2004; Ford and Bushek, 2012; Burge et al., 2014). Patterns of *P. marinus* and *H. nelsoni* have also been connected with various large-scale climatic cycles (e.g., Powell et al., 1999a, 2012b; Hofmann et al., 2001; Soniat et al., 2005; Kim and Powell, 2009; Levinton et al., 2011).

Although the number of studies examining long-term change in diseases is relatively few, the establishment of long-term sampling programs (e.g., in the U.S.A., the National Science Foundation's Long-Term Ecological Research, LTER networks) are increasingly producing data sets that can be 'mined'. It is important that these research programs not only sample molluscs but also their parasites through time.

3.3. Disease incidence in cultivated vs. wild populations

Due to the commercial value of cultivated species, much more attention has been given to their parasites and diseases than those of wild populations (Hine and Thorne, 2000; Carnegie, 2009; Morley, 2010a; Lafferty et al., 2015). Nevertheless, in instances where the diseases of cultivated molluscs have been compared to wild populations, often of the same species and growing alongside, the two groups often display very different patterns of infection (e.g., Wilkie et al., 2013; Lafferty et al., 2015). Where cultivated populations are selectively bred for disease resistance, they may be expected to exhibit reduced disease incidence as compared to wild populations (e.g., Nell and Perkins, 2006; see also Degremont et al., 2015). However, several studies indicate that in the absence of such management intervention, cultivated populations often display the reverse pattern of having higher incidence of disease than wild counterparts. For example, simultaneous sampling of adjacent wild and aquaculture populations of Sydney rock oysters *Saccostrea glomerata* along the Hawkesbury River, New South Wales, Australia, revealed that the prevalence of QX disease from *Marteilia sydneyi* among wild oysters peaked at a prevalence of 14%, but was significantly higher at 47% for cultured *S. glomerata* oysters (Wilkie et al., 2013).

Where higher incidence of disease is seen among cultivated than wild populations, this may reflect differences between the two groups in: (1) exposure to disease vectors (covered in the subsequent section of the review); (2) genetic structure; or (3) environment. Selective breeding programs, often aimed at accelerating growth or enhancing resistance to particular diseases, can result in reduced genetic variation among cultivated as compared to wild molluscan populations and in some instances may result in inbreeding depression (e.g., Bower, 1992; Evans et al., 2004; NRC, 2004a; Gaffney, 2006; Hare et al., 2006; Camara and Vadopalas, 2009; Hoffman et al., 2009; Hedgecock, 2011; Rohfritsch et al., 2013; Degremont et al., 2015). This may render cultivated populations more susceptible to diseases for which they have not been selectively bred for resistance against.

Cultivation practices may place molluscs at sufficiently high population densities so as to: (1) stress (e.g., food, space, pollution, habitat degradation) farmed populations; (2) place molluscs in more favorable environments for transmission and survival of the parasite; and/or (3) increase the proximity of molluscs to other disease reservoirs (non-host species) and hosts for parasites (Morley, 2010a; Lafferty et al., 2015). For example, the prevalence and intensity of the turbellarian *Urastoma* cyprinae and the copepod Mytilicola intestinalis in edible mussels was greater in those individuals positioned at the bottom portion of culture ropes, closer to the sea floor habitat of copepods, than in those nearer the surface (Davey and Gee, 1976; Murina and Solonchenko, 1991).

Cultivation practices may also transplant organisms to foreign environments to which they are not adapted. Quahog Parasite Unknown (QPX) is a parasite of concern for the hard clam (*Mercenaria mercenaria*) industry in the USA. The parasite, a fungus-like protest of the Phylum Labyrinthulomycota, causes an inflammatory response by the animal's hemocytes. It has been observed in both cultivated and wild clam populations in Canada (Prince Edward Island) since the 1950s and from the eastern U.S. coast in Massachusetts to North Carolina (e.g., Smolowitz et al., 1998; Ragone Calvo et al., 1998; Kraeuter et al., 2011). In a reciprocal transplant experiment, Ragone Calvo et al. (2007) found that cultivated populations of hard clams, *Mercenaria mercenaria* originating from non-local (more southern, Florida, U.S.A.) sources appeared to be more susceptible to Quahog Parasite Unknown (QPX), than clams from local sources. In addition to genotypic sensitivity related to geography, some have suggested that high planting

densities or poor aquaculture husbandry may enhance locally the occurrence of the infection.

In addition to disease infection processes operating in parallel between cultivated and wild populations, cultivated populations of molluscs may alter disease prevalence in wild molluscs, and wild molluscs may alter disease prevalence in cultivate populations, by introducing parasites into a 'novel' area, or by acting as reservoirs for spread of diseases (Lafferty et al., 2015). These dynamics will be considered in greater detail in the next section on vectors. Overall, however, the relationship between wild and farmed aquatic animals and pathogens is complex (Beveridge, 2001) and poorly researched.

As more molluscs are cultured across the globe, potentially without good genetic practices (inbreeding, small brood stock numbers), populations of wild molluscs, especially those that have significantly depressed population sizes, such as *Ostrea lurida and O. edulis*, may be affected by interbreeding with captively-bred populations that may lack local adaptations and genetic variability to respond to existing, let alone novel introduced diseases and parasites (discussed related Sections here, and in Gaffney, 2006; Camara and Vadopalas, 2009; Hedgecock, 2011; Degremont et al., 2015).

3.4. Disease incidence in native vs non-native molluscs

The incidence of disease in native vs non-native molluscs has received a significant amount of research attention, primarily in the context of a role for parasites in impeding ('biotic resistance') or facilitating ('enemy release') invasion. In the subsequent sections we discuss how diseases can spillover from non-native to native hosts and how, through the process of spillback, non-native hosts may enhance the prevalence of disease in native hosts (see also Section 4.3). We also discuss how diseases can influence interactions between native and non-native species (see also Section 4.3 also). In this section we simply describe observed patterns of disease incidence in both native and non-native taxa.

In many instances, smaller parasite species richness has been identified in native than non-native analogs (Torchin et al., 2003). For example, in a comparison of the trematode assemblages of sympatric populations of the non-native gastropod *Batallaria cumingi* and a native sympatric mud snail, *Cerithidea californica*, Torchin et al. (2005) found that the non-native snail was parasitized only by one introduced trematode species, whereas the native snail was parasitized by 10 native trematode species. Interestingly, however, overall prevalence of trematodes did not significantly differ between the native and non-native snails. The few species of trematode infecting the non-native gastropod appeared to be a release form its natural enemies, as in its native range *B. cumingi* is parasitised by eight different trematode species (Torchin et al., 2005).

The difference in parasite richness observed between species in their native and non-native ranges may be dependent on time since invasion (e.g., Blakeslee and Byers, 2008). The differential is predicted to be greatest soon after invasion, and diminish through time as the probability of subsequent invasions of infected hosts or arrival of parasites through natural vectors increases. Blakeslee and Byers (2008) found greater similarity between North America and Europe in the parasite diversity of *Littorina saxitilis* and L. *obtusa* than the parasite diversity of *L. littorea*. They interpret this as evidence that *L. saxitilus* and *L. obtusa* invading North America from Europe much earlier than *L. littorea*, although this hypothesis was not verified. Patterns of difference in the parasite diversity of native and non-native molluscs may also be dependent on the specificity of the host–parasite relationship (Torchin and Mitchell, 2004). Larval trematode parasites tend to be highly host-specific and it is unclear the extent to which the pattern of greater parasite diversity in native than non-native species might be verified in other groups of molluscan pathogens. 'Naturalized' populations of non-native species, such as *Crassostrea gigas*, or *Mytilus edulis* that have been widely introduced globally might be ideal candidates with which to compare parasite diversity at native and non-native locations, and how these depend on time since introduction (see Solomieu et al., 2015).

3.5. The role of vectors in determining disease distributions

Some etiological agents have free-living stages that are dispersed in the external environment, but others, which remain within their hosts for their entire life-cycle are completely dependent on movement patterns of their hosts for dispersal (reviewed in Sousa and Grosholz, 1991; Combes, 2001, 2005; Simberloff and Rejmánek, 2011). Particularly in the case of parasites that depend on their host for dispersal, species translocations by aquaculture and the aquarium and ornamental trades, and unintentional (accidental "fellow travelers") introductions of "hitchhiking" parasites are key processes influencing disease distributions (e.g., Dinamani, 1986; Aguirre-Macedo and Kennedy, 1999; Renault et al., 2000; Pechenik et al., 2001; Ruesink et al., 2005; Cáceres-Martínez et al., 2012; Degremont and Benabdelmouna, 2014; Paul-Pont et al., 2014). Introductions of parasites have also been attributed to shipping (e.g., Ruiz et al., 1997; Naylor et al., 2001; Padilla and Williams, 2004; Simberloff and Rejmánek, 2011; Lohan et al., 2015).

Largely due to the large number of aquaculture industries they support, molluscs are among the most translocated of aquatic taxa (e.g., Carlton and Mann, 1996; Ruesink et al., 2005; Kochmann et al., 2008; Molnar et al., 2008). At local and regional scales, 'farmers' often move molluscs between hatcheries and grow-out facilities, and within and among water bodies at different stages of their reproductive and developmental cycle in order to take advantage of optimal environmental conditions for growth and survival (e.g., Smolowitz et al., 1998; Ragone Calvo et al., 1998; Nell, 2001; Cranfield et al., 2005; Kraeuter et al., 2011; Herbert et al., 2012).

Moving molluscs from disease-endemic (e.g., *Bonamia*) areas to areas that are presumed to be uninfected can have serious consequences (e.g., Culloty et al., 1999; Hine, 1996; Cranfield et al., 2005; Lynch et al., 2010). In coastal Victoria, Australia, the between-farm pattern of spread of Ganglioneuritis among cultivated abalone followed patterns of abalone movements (Hooper et al., 2007a). Along the east coast of Australia, the spread of outbreaks of QX disease among Sydney rock oysters coincided with the implementation of 'highway farming', where oysters are transported by road among estuaries to optimize growth, in the 1970s (Nell, 2001).

A more concrete example of parasites hitchhiking with molluscs comes from the southeastern USA where seed clams (*Mercenaria mercenaria*) that were relayed between South Carolina to Florida, U.S.A. to maximize growth rates. Inspections of stock revealed non-native 'hitchhikers' in refrigerated sacks of seed (L. Coen & N. Hadley, SCDNR, pers. obs.). This led to a revision of that state's (South Carolina, U.S.A.) importation regulations and expanded inspections and disease assessments of both seed and adults. Concerns that Dermo and MSX diseases may similarly be translocated with *C. virginica* oysters moved from high infection sites (Ford and Tripp, 1996), and that harmful algal blooms (HABs) could also be relayed with molluscs (L. Coen & A. Lewitus, NOAA, pers. obs.; Bobo et al., 1997) led to further restrictions on translocation of molluscs in the state of South Carolina, U.S.A.

A review of oyster diseases (Ruesink et al., 2005) suggests that trans-continental oyster introductions and transplants have been

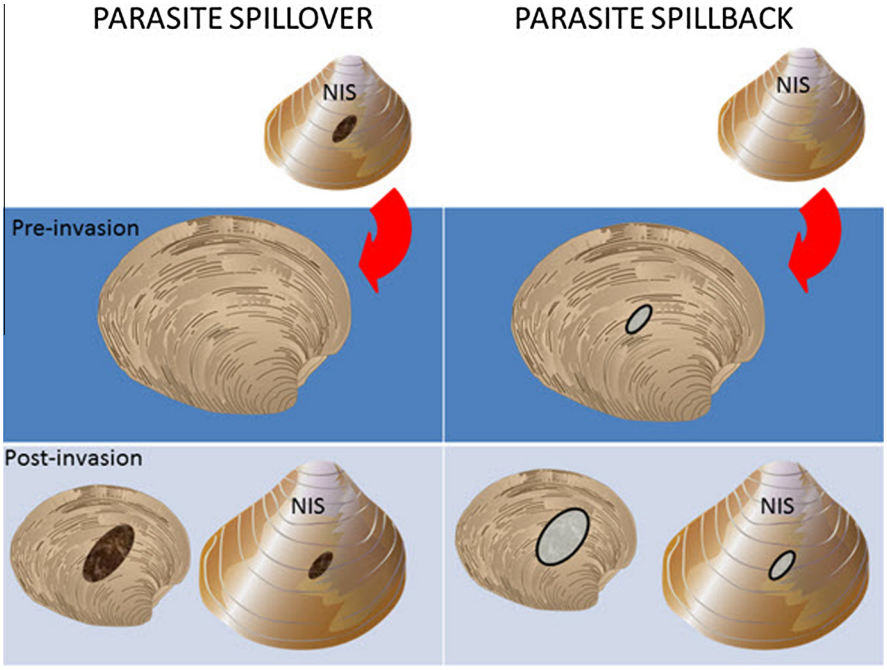

Fig. 2. Conceptual diagrams of *parasite spillover* and *parasite spillback*. The size of a given bivalve is representative of its relative population size with the oval size depicting parasite infection burden. *Parasite spillover* occurs where a non-indigenous species (NIS) introduces a novel parasite into the native's environment to which a native species is then susceptible. The NIS acts as a "reservoir" for the parasite, which in spilling over into the native host, increases the abundance of the parasite and decreases molluscan population size. *Parasite spillback* occurs where a NIS acquires a native parasite following its introduction into a novel environment and, in serving as an additional reservoir for the parasite, increases the overall infection levels in the native host population. The increased level of parasite infection in the native host leads to declines in the host's overall population size (figure adapted from Kelly et al., 2009). Constructed with images from the IAN Image and Video Library; http://ian.umces.edu/imagelibrary/.

a major cause of emerging diseases. For example, the parasitic copepods, *Mytilicola orientalis* and *Myicola ostrae,* which infect molluscan species, are thought to have been transported from Asia to Europe, and in the case of *Mytilicola*, also the Pacific Coast of North America, with infected *C. gigas* oysters imported for culture (e.g., Holmes and Minchin, 1995; Minchin, 1996). *Haplosproidium nelsoni,* a non-native parasite of *C. virginica* on the east coast of the U.S.A. is thought to have originated from *C. gigas* in the Pacific (Burreson et al., 2000). The reintroduction of the native oyster, *Ostrea edulis,* to Spain and France from California, U.S.A. is thought to be responsible for introduction of the non-native parasite, *Bonamia edulis*, to Europe (Comps et al., 1980). Switching of *B. ostreae* from its original host, possibly *C. virginica* (O.I.E., 2009), presumably underpinned its emergence in *O. edulis* (see also Lynch et al., 2010). Nevertheless, these studies linking spread of disease to molluscan movements are correlative and, without causative evidence are just circumstantial; however the evidence is mounting (e.g., Hines et al., 1997; Burreson et al., 2000; Carnegie, 2005, 2009; Torchin and Kuris, 2005; Chapman et al., 2011; Freeman et al., 2013; Lafferty et al., 2015).

At continental scales, introductions of non-native molluscs for aquaculture may negatively influence parasite burdens in native molluscs via the effects of **parasite spillover** and **parasite spillback** (Kelly et al., 2009). Parasite spillover occurs where non-native molluscs introduce novel parasites that subsequently switch to native hosts (see Fig. 2). The less-studied parasite 'spillback' is where non-native species acquire and amplify native parasites, increasing disease impacts in the native species (see Fig. 2; reviewed in Kelly et al., 2009). Invading host species tend to accumulate generalist native parasites from the overall local suite of potential species (e.g., Le Cam and Viard, 2011, native boring sponge and non-native host, *Crepidula*). However, recent reviews have not included parasite spillback when considering non-native parasite species and associated threats to native host populations (e.g., Hatcher et al., 2006).

Although numerous papers report acquisition of native parasites by non-native hosts (e.g., infection of the zebra mussel *Dreissena polymorpha* by the trematode *Echinoparyphium recurvatum* in Lake Naroch, Mastitsky and Veres, 2010; infection of *Crassostrea ariakensis* by *Bonamia* sp. in North Carolina, U.S.A., Bishop et al., 2006), whether this enhances or reduces parasite burdens in native hosts is less well-studied. The acquisition of a native parasite by a non-native mollusc does not automatically lead to the spillback of infection to the native fauna because in some instances the non-native molluscs may serve as **"dead-end"** hosts that are not capable of disseminating the parasite's infective stages, or may diminish parasite burdens in the native host through "**dilution**" effects (e.g., Krakau et al., 2006; Kopp and Jokela, 2007; Keesing et al., 2006; Randolph and Dobson, 2012). Clever experimental approaches will most likely be needed to validate the **parasite spillback hypothesis** (Kelly et al., 2009).

In addition to intentional introductions of molluscs for aquaculture or the aquarium trade, unintentional introductions and translocations of molluscan species on the hulls of vessels may also lead to the introduction of hitch-hiking etiological agents to new locations (e.g., Hudson and Hill, 1991; Howard, 1994; Carlton and Mann, 1996; Ruiz et al., 2000). The introduction of the parasitic copepod *Mytilicola intestinalis* from the Mediterranean Sea to northern Europe is thought to have been through translocation of blue mussels, *Mytilus galloprovincialis,* on the hulls of ships (Minchin, 1996). Similarly, it is thought that fouling of barges with infected *O. edulis* may have contributed to the spread bonamiosis along the south coast of Britain (e.g., Howard, 1994), although this pathway is unconfirmed.

Additionally, parasites and viruses may be introduced to new locations via ballast water. Bacteria and virus-like particles are

abundant in ballast water (Ruiz et al., 2000), and in some instances, parasites have been found to be particularly prevalent in the vicinity of ports (Torchin et al., 2002; Bishop et al., 2006; Lohan et al., 2015). Nevertheless, direct evidence of ballast water introductions of marine molluscan diseases remains elusive.

3.6. Abiotic determinants of disease

Abiotic factors can greatly influence observed patterns of disease in wild and cultivated populations by influencing: (1) the distribution of hosts (or the parasite's 'macrohabitat'); (2) the 'microhabitat' of a given parasite on or in their hosts; (3) parasite survival during any free-living stages, in which it is away from the host (e.g. the free-swimming cercaria stage of trematode parasites, involved with transmission between first and second if present intermediate hosts); and (4) interactions between a host and one or more parasite(s) (see Fig. 1). These abiotic factors may be naturally occurring or be outcomes of coastal development (e.g., pollution) or climate change (e.g., warming), and may impact the expression of diseases directly or indirectly (e.g., Lafferty and Kuris, 2005; Morley, 2010b). For bivalve molluscs most epizootics appear to be initiated by abiotic conditions (e.g., Hofmann et al., 2001; Soniat et al., 2009). The two prime abiotic variables that have been elicited as having the most impact on bivalve (and especially oyster) diseases are water (and air) temperatures and salinity regimes. In many areas, freshwater releases or diversions, summer evaporation and precipitation rates, food, and climate patterns determine patterns of disease, presumably in large part because they influence temperature and salinity, as well as other environmental factors that influence disease (discussed in La Peyre et al., 2003, 2008, 2009; Soniat et al., 2009; Volety et al., 2009, 2014; Burge et al., 2014; Lafferty and Harvell, 2014).

In many instances, the parasite's larvae (but see Studer and Poulin, 2012) appear to be more sensitive to salinity than adult hosts, constraining disease incidence (e.g., Haskin and Ford, 1982; Ford and Tripp, 1996; La Peyre et al., 2003, 2008; Bushek et al., 2012). For example, the physiological inability of the disease-causing parasite, *H. nelsoni*, to tolerate low salinities (Ford and Haskin, 1988) has resulted in its host, *C. virginica*, experiencing spatial and temporal refuges from the resultant MSX disease at low salinities (Haskin and Ford, 1982; Andrews, 1983; Ford, 1985; Arzul and Carnegie, 2015). Adult bivalves, but not larvae, are able to close their valves to reduce exposure to conditions of unfavorable salinity that extend days to weeks. In normally high salinity environments, lowered salinities in some instances have negative and other instances positive effects on disease. Along the east coast of the U.S.A. it was observed the non-native oyster *Crassostrea ariakensis* is not infected by the parasite *Bonamia* sp. at salinities below 20 ppt (Bishop et al., 2006). In an experiment in which *Bonamia* sp.-infected oysters were exposed to salinities of 10, 20 or 30 ppt, average parasite intensity decreased in oysters placed at salinities of 10 and 20 ppt, but not at the highest salinity (30 ppt) despite the host's tolerance for the entire range of the manipulated salinities (Audemard et al., 2008).

In other instances, however, the same low salinities that cause mortality of the parasite also negatively affect the host, so there is no net benefit to the host of the salinity reduction. For example, in southwest Florida, U.S.A., rainfall and significant freshwater releases (or diversions) that depress normally high salinity environments to salinities as low as 5 ppt for extended periods generally eradicate oyster populations. However, patchy individuals, occurring in refuges such as isolated embayments, can survive these rainy seasons and freshwater releases given that evaporation raises salinities quite rapidly (e.g., L. Coen, pers. data; Milbrandt et al., 2012; Parker et al., 2013). Additionally, where low salinities depress the immune systems of molluscs, but do not negatively affect parasites (such as seen in *S. glomerata*, Butt et al., 2006), low salinities may be associated with increased incidence of disease (e.g. Rubio et al., 2013). In many areas, the rainy season overlaps with oyster's spawning (e.g., *C. virginica* in Florida, U.S.A.) and is often associated with a lower condition of individuals due to investment in reproduction (e.g., Volety et al., 2009, 2014; Milbrandt et al., 2012; Parker et al., 2013).

Temperature may influence the distribution, prevalence and intensity of molluscan diseases by determining the ranges of molluscan hosts, the replication and proliferation of parasites (e.g., Ford and Tripp, 1996; Ben-Horin et al., 2013), the shedding of free-living stages into the environment (e.g., Pietrock and Marcogliese, 2003; Poulin, 2006), and the condition of hosts, which in turn influences their susceptibility to infection (e.g., Parry and Pipe, 2004). Warming winter water temperatures have been implicated in the previously mentioned (see Section 3.2) range expansion of *P. marinus* and *H. nelsoni* epizootics northward of Delaware and Chesapeake Bays (Ford, 1996; Cook et al., 1998; Hofmann et al., 2001; Ford and Chintala, 2006).

Tidal height, aerial exposure (reviewed in Menge and Branch, 2001; Sousa, 2001) and associated parameters (e.g., UV, Studer et al., 2012) have also been found to be a significant factor influencing the distributions of a number of diseases (e.g., James, 1968; Littlewood et al., 1992; Manley et al., 2009). They may do so by influencing the condition of molluscan hosts, the encounter rate of definitive and molluscan hosts of multi-host parasites, or by influencing the suitability of abiotic conditions for parasites (the periodic low-tide drying out of hosts may be deleterious for some). For example, desiccation stress increases and time available for filter-feeding decreases with tidal elevation (Peterson and Black, 1987; ASMFC, 2007; Coen and Grizzle, 2016), potentially modifying the condition of molluscs and hence their capacity to resist disease. Further, filter-feeding bivalves lower on the shore may potentially filter greater volumes (for extended periods) of parasite-laden water, such that the encounter rate between hosts and parasites is greater (Allam et al., 2013; Ben-Horin et al., 2015). The higher prevalence of trematode parasites among *Littorina littorea* higher than lower on the shore is hypothesized to reflect the greater exposure time of high-intertidal snails to feeding birds (see Fig. 5), the definitive host for the parasite (Sindermann and Farrin, 1962). Encounter rates between definitive and intermediate hosts may be particularly great where intertidal habitat-forming species (e.g., clusters of barnacles, gastropods, mussels and oysters) facilitate locally high densities of gastropod intermediate hosts by ameliorating abiotic and biotic stress (e.g., Menge and Branch, 2001; Sousa, 2001). Furthermore, deposited trematode eggs may be less likely to be dislodged if they are in the high intertidal zone (e.g., Byers et al., 2008).

In many instances multiple abiotic factors interact to influence patterns and dynamics of molluscan disease, often in unpredictable ways. For instance, Soniat et al. (2009) mention that colder winter temperatures enable *P. marinus* to tolerate the low salinities associated with freshets since higher tissue osmolality occurs in vivo (La Peyre et al., 2003, 2006). In a related example, salinities greater than 15 ppt are associated with *P.marinus* infection and Dermo disease among subtidal *Crassostrea virginica* oysters (Ford and Tripp, 1996; Elston and Ford, 2011; Burge et al., 2014; Lafferty et al., 2015), but not among intertidal oysters that in many instances appear to be thrive in high salinity waters (O'Beirn et al., 1997; Bobo et al., 1997; Drexler et al., 2013; Volety et al., 2009, 2014). In some instances, abiotic factors interact to influence the production and activity of enzymes associated with cellular defenses (e.g., Akberali and Trueman, 1985; Jenny et al., 2002; Edge et al., 2012; Soudant et al., 2013). Sublethal multi-stressor impacts can be evaluated in molluscs using a suite of 'subtle'

cellular biomarkers, including heat shock proteins (e.g., Akberali and Trueman, 1985; Ringwood et al., 1999, 2002; Sung et al., 2011; Edge et al., 2012).

Abiotic factors may not only determine the niches of parasites and their hosts, but also their interactions. Many diseases are expressed over much smaller areas than might be predicted based on the abiotic tolerances of hosts and parasites alone. For example, along the coast of Australia, the haplosporidian parasite *Marteilia sydneyi* that causes QX disease in Sydney rock oysters, *Saccostera glomerata*, is found in all but one oyster-growing estuary (Adlard and Wesche, 2005), yet it only causes disease mortality in a small subset of these (Green et al., 2011). Studies have revealed that in eastern Australian estuaries in which QX disease is endemic, the activity of the defense enzyme phenoloxidase is inhibited by transient environmental stressors such as low salinity (Butt et al., 2006) or starvation (Butt et al., 2007). Similarly, a link has been established in farmed abalone between increased stress and decreased immune function, which in turn leads to increased rates of bacterial infections (Hooper et al., 2007b; Travers et al., 2015).

3.7. Biotic determinants of disease

While abiotic factors set the niches of parasites and hosts, biotic factors can also be important in influencing the dynamics of disease. In particular, traits of hosts, such as their size, age, density, condition and immune function, and the life-cycle of parasites are of importance in setting patterns of disease.

The relationship between disease prevalence and host age (and size) may be positive, neutral or negative. Susceptibility of bivalves to viral and bacterial infections is generally greater for young than for adults (Lane and Birkbeck, 2000; Renault and Novoa, 2004; Travers et al., 2015). Similarly, a number of diseases caused by protozoans and digeneans appear to more prolific among certain age or size classes. For example, the prevalence of *Bonamia* sp. infection among *Crassostrea ariakensis* deployed in North Carolina was greater among smaller individuals (<40 mm shell height) than larger individuals (Bishop et al., 2006). By contrast, the prevalence of bonamiasis among flat oysters, *Ostrea edulis*, generally increases with age and size (Cáceres-Martínez et al., 1995; Culloty and Mulcahy, 1996). Similarly, a higher infestation prevalence of bucephalid trematodes is frequently observed in larger than smaller mussels (e.g. *Perna perna*, *Mytlis galloprovincialis*; Lasiak, 1993; Villalba et al., 1997).

Older hosts have had more time than younger hosts to accumulate disease-causing parasites and viruses (Lafferty and Kuris, 2009). They also typically eat more than their smaller counterparts, and thus have a greater probability of consuming parasites while feeding (Mouritsen et al., 2003). Further, the greater reproductive output of larger/older individuals may, during times of reproduction, cause a reduction in host 'condition' and reduction of energy available for immunological defenses, thereby rendering them more susceptible to infections (Taskinen and Saarinen, 1999). Conversely, older molluscs have had more time available than juvenile molluscs to develop defense mechanisms, for example thicker shells that are more effective at excluding boring parasites (Stefaniak et al., 2005). There are, however, few studies that have independently manipulated feeding rate, reproductive status of molluscs and immunological defenses to ascertain which of these factors is driving relationships between disease prevalence and size/age.

The density of molluscs can have both positive and negative influences on disease transmission. In the instance of directly transmissible diseases, high densities may increase disease prevalence and intensity by increasing contact between infected and uninfected individuals (Anderson and May, 1991). It has been hypothesized that one of the reasons that cultivated molluscs tend to have much greater incidence of disease than wild molluscs is because they are placed at artificially high densities which enhance disease transmission, and cause stress among individuals (e.g., Ford et al., 2002; Torchin et al., 2002). However, in the instances of diseases that have multiple hosts, the relationship between host density and infection can be negative. Several studies on cockles have observed a reduction in parasite loads at high host densities (Mouritsen et al., 2003; Thieltges and Reise, 2007). This may be explained by infective stages being diverted between several hosts at high densities hence resulting in lower infection intensities of individual cockles.

Common-garden experiments, in which bivalves from different locations are transplanted to a single common site, have revealed significant variation among populations in susceptibility to disease (Culloty et al., 2004; Ford and Bushek, 2012). Although variation in infection may be related to phenotypic differences in the condition of bivalves (Morley, 2010b), genotypic differences linked to cellular or biochemical processes may also play a role (Gaffney and Bushek, 1996). For example, there is good evidence that eastern oysters, *C. virginica*, genetically vary in their resistance to *H. nelsoni* and *P. marinus* (Ford and Haskin, 1987; Ragone Calvo et al., 2003). Similarly, Sydney rock oysters, *S. glomerata* vary in their resistance to both *M. sydneyi* and *Bonamia roughleyi* (Green et al., 2009). Natural variability among individuals in the presence of disease-resistance genotypes has for many decades underpinned selective breeding programs based on mass selection of disease-resistant bivalves (Ragone Calvo et al., 2003; Dove et al., 2013; Degremont et al., 2015). The frequency of natural disease-resistance in bivalve populations tends to increase following exposure to disease mortality (e.g., Ragone Calvo et al., 2003; Yu and Guo, 2006; Carnegie and Burreson, 2011; Lynch et al., 2014).

In wild populations, gene flow between disease-affected and refuge areas may influence the development of disease resistance at the population level (Hofmann et al., 2009). In Delaware Bay, enhanced resistance of Eastern oysters (*C. virginica*) to MSX disease was initially seen following the 1957–1959 epizootic, but has since plateaued between 1960 and 1987, presumably because most of the surviving oysters were in low-salinity 'refugia' where they were protected from sustained selection and continued to contribute susceptible progeny to the population. In 1984–1986, however, an extended period of drought eliminated these low-salinity disease refuges, resulting in significant mortality across Delaware Bay populations, and leading to a major increase in the level of resistance to MSX disease (e.g., Hofmann et al., 2009; Carnegie and Burreson, 2011; Ford et al., 2012; Burge et al., 2014; Arzul and Carnegie, 2015). Unlike *P. marinus* with direct acquisition of the parasite, the transmission of *H. nelsoni* (disease causing agent of MSX) has not been resolved despite over 50 years of research effort, but most assume that there is an 'intermediate' host involved prior to it infecting oysters (Ford and Tripp, 1996; Lafferty and Harvell, 2014; Arzul and Carnegie, 2015).

In addition to influencing host immunity, habitat modification (and related effects) can influence susceptibility to disease by influencing host–parasite transmission. *Bonamia* is a disease-causing parasite in the oyster, *Ostrea chilensis.* Like *P. marinus,* it transmits the parasite directly from oyster to oyster, such that disease is spread as a function of the density of animals. It has been hypothesized that in the Foveaux Strait of New Zealand, extensive dredging (disturbance) contributed to environmental conditions that enhanced disease transmission (reviews in Hine, 1996; Cranfield et al., 1999, 2005). Before oyster dredging began just over 100 years ago, the benthos of Foveaux Strait largely comprised complex communities of sponges, bryozoans, brachiopods and other invertebrates, interspersed with small discreet oyster beds. Subsequent dredging removed large areas dominated by sponge and bryozoan faunas, and greatly increased the extent of the oyster

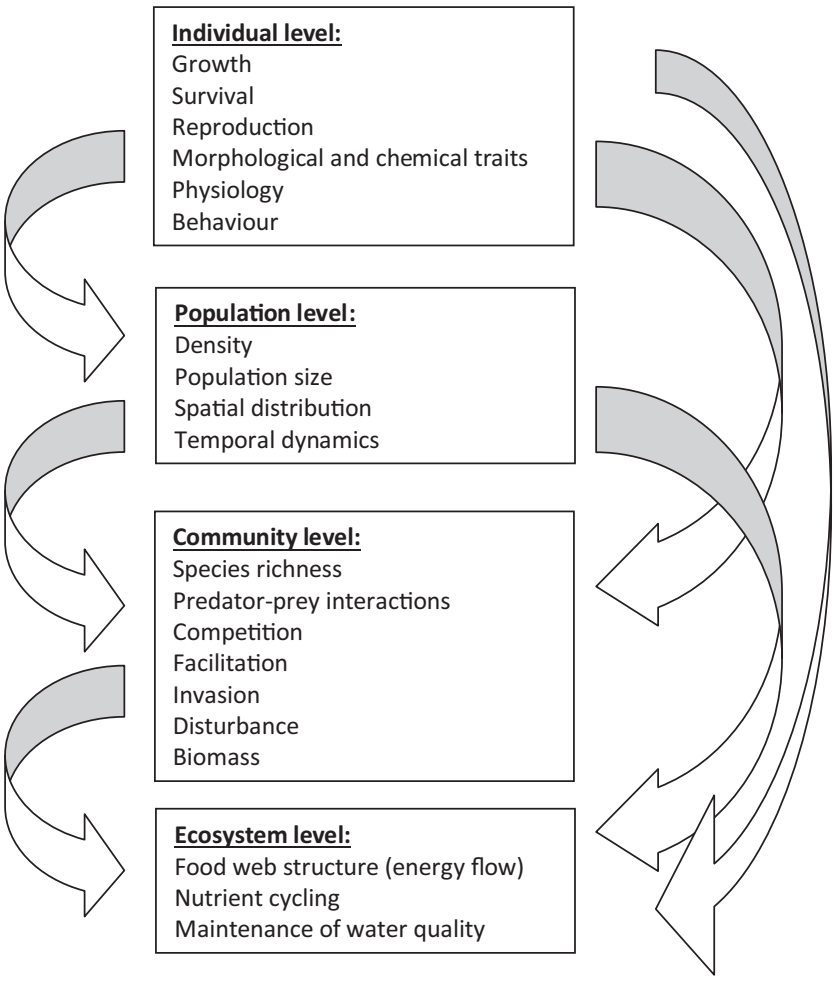

Fig. 3. Summary of the variables disease may affect, from individual, population, community and ecosystem levels. At higher levels of ecological organization, effects of disease(s) may arise directly or, alternatively, indirectly, as a consequence of lower levels of organization affected. For example, disease effects on bivalve sediment burial (=individual level) might influence their ability to provide hard substrate 'habitat' for fouling organisms, or sediment disturbance through bioturbation (=community level). An example of a direct effect of disease(s) on the community level variables might be parasites enhancing overall community species richness.

beds. The net outcome was enhanced densities of oysters and a decreased density of other filter-feeding organisms that may have previously reduced the dispersal stages of *Bonamia* during their filter feeding (see Hine, 1996; Cranfield et al., 1999, 2005). A meta-analysis by Wood and Lafferty (2015) suggests that positive effects of fishing on unfished host species of directly transmitted parasites may be common place. Presumably, this results from densities of the unfished host(s) responding positively to the removal of other competitors.

In the case of "vectored" (examples where some organism transmits the pathogen) parasites, habitat utilization, and hence spatial distributions of definitive hosts often influences the spatial patterns of both the infection and the disease among various intermediate hosts (e.g., Kuris, 1990; Sousa, 1990; Sousa and Grosholz, 1991). In a study spanning several hundred kilometers of coastline, Byers et al. (2008) found that the abundance of gulls, the definitive host for a trematode parasite, was the key correlate of spatial patterns of variation in the prevalence among *Littorina littorea*. Similarly a strong relationship was found between the abundance of the diamondback terrapin turtle (*Malacemys terrapin*), the definitive host for several trematode parasites and the parasite load of another gastropod *Illyanassa obsoleta* (Byers et al., 2011).

4. Ecological impacts of diseases

Diseases affect all levels of ecological organization from individuals to ecosystems. In regulating host abundance, and modifying physiological, reproductive, morphological and behavioral traits, they can have indirect effects on species interactions and community structure (Fig. 3; Poulin, 1999; Bush et al., 2001; Mouritsen and Poulin, 2002a; Lafferty and Harvell, 2014). Non-lethal effects of parasites (also called "trait-mediated effects") for example, can include induced changes in hosts habitat usage, foraging or general activity, and life history traits (i.e. growth, survival, reproduction; Hatcher and Dunn, 2011). By directly and indirectly affecting trophic relationships parasites can be key determinants of food web typology and stability (e.g., Lafferty et al., 2006, 2008; Hechinger et al., 2011).

The vast majority of studies investigating ecological impacts of molluscan diseases have focused on the effects of disease agents at the individual- and population-level, for economically-important species (e.g., oysters, mussels). However, examples of their effect on communities and whole ecosystems are now accumulating for marine and analogous freshwater systems (e.g., Mouritsen and Poulin, 2002a, 2002b, 2010; Poulin, 2002; Wood et al., 2007; Lafferty and Harvell, 2014; Lagrue and Poulin, 2015).

4.1. Individual-level effects

A wide range of etiological agents have been linked to changes in the condition of individual molluscs and their phenotypes (Combes, 1991; Poulin, 2007). The most extreme manifestation of disease at the individual-level is death of the molluscan host. This may by a direct effect of disease, where the parasite destroys body tissues of the host or depletes energy resources available for vital cellular functions (Thieltges, 2006). For example, infection of cultivated giant clams (*Tridacna gigas*) with high densities (10–20 ectoparasite snails per giant clam) of parasitic *Turbonilla* sp. impacts clam growth rate by as much as 25% and in some cases causes high mortality rates (Boglio and Lucas, 1997). Alternatively (or additionally) death may result indirectly from diseases enhancing the susceptibility of molluscs to stressors, predation, and/or infection by additional pathogens (Thomas and Poulin, 1998; Poulin et al., 1998). For example, the shell-boring polychaete *Polydora ciliata* weakens the shell of its gastropod host *Littorina littorea*, rendering it more susceptible to gastropod predation (Buschbaum et al., 2007).

In addition to these lethal effects, a broad range of non-lethal effects of disease have been seen at the individual-level (see Fig. 3; Combes, 1991; Poulin, 2007). These may include phenotypic changes such as: (1) predator or competition avoidance and related behaviors (e.g., Hatcher et al., 2006; Hatcher and Dunn, 2011); (2) movement and microhabitat use (e.g., Miller and Poulin, 2001; Mouritsen, 2002, 2004; O'Dwyer et al., 2014a, 2014b); (3) reproductive investment (e.g., Sousa, 1983; Lafferty, 1993; Bernot and Lamberti, 2008) and/or (4) size, resulting from changes in rate of growth (Sousa, 1983; Ballabeni, 1995; Lafferty and Kuris, 2009). Additionally, the effect can (5) even be a change in phenotypic appearance (e.g., shell shape and snail 'spinosity') of the host (e.g., Lagrue et al., 2007; Miura and Chiba, 2007). In some instances the manipulation of molluscan phenotypes by the etiological agent represents an adaptation that enhances parasite proliferation and transmission through various manipulative "behaviors" (e.g., Poulin, 1995, 2007; Moore, 2002; Thomas et al., 2005; Lefèvre et al., 2008; Hughes et al., 2012). Other phenotypic changes, however, do not have any adaptive significance for the etiological agent and are simply pathological side-effects of infection.

Trematode parasites, many of which use molluscs as an intermediate host in their complex life cycles, have served as models for studying how parasites influence behavior (overviews in Combes, 2001, 2005; Moore, 2002; Mouritsen, 2002, 2004). Trematodes may induce behaviors in intermediate hosts that enhance their risk of predation by definitive hosts, and hence parasite transmission. Perhaps the most widely studied example of host manipulation by disease agents is the inhibition in many, but not all instances (see Fermer et al., 2011), of cockle burrowing by the metacercariae of trematode parasites that encyst the foot of cockles and stunt foot growth (Thomas and Poulin, 1998; Mouritsen, 2002). Heavily infected cockles remain on the surface sediments where they are more susceptible to predation by their definitive host, birds (Thomas and Poulin, 1998; Mouritsen, 2002, 2004), by species unaffected by the parasite, including fish (Mouritsen and Poulin, 2003a, 2003b), and are more susceptible to environmental stressors such as heat and desiccation. Other etiological agents, such as the marine bacterium, *Pseudomonas fluorescens*, have also been found to impede burial of certain cockle species (Blanchet et al., 2003). Irrespective of the agent that causes inhibition of burial, unburied cockles suffer up to seven times the mortality rate of normally buried individuals (e.g., Poulin, 1999; Desclaux et al., 2002; Blanchet et al., 2003).

In addition to their effects on cockle burial, trematode parasites have also been found to modify the behavior and habitat utilization of gastropod hosts, in some instances influencing rates of mortality. Miller and Poulin (2001) found that the trochid gastropod *Diloma subrostrata* when parasitized by trematodes moved smaller distances than similar-sized non-parasitized snails. Moreover, the mean direction moved by parasitized snails was almost parallel to the water's edge, whereas non-parasitized snails moved toward the habitat refuge in the upper portion of the intertidal zone. On New Zealand rocky shores, philophthalmid trematode parasites altered the microhabitat use of their littorinid hosts, thus increasing occupancy of rock surfaces, and reducing the distance travelled by host snails, particularly in a down-shore direction (O'Dwyer et al., 2014a). Additionally, they were found to reduce the attachment strength of the littorinids on rocky wave-impacted shores, thereby indirectly affecting mortality through a second pathway (O'Dwyer et al., 2014b).

Reproduction is also a trait of hosts that is commonly manipulated by parasites. Trematodes (e.g. Sousa, 1983; Huxham et al., 1993) and pinnotherid (pea) crabs (e.g., Yoo and Kajihara, 1985; Ocampo et al., 2014) are among the parasites that may castrate their molluscan hosts. In reducing or completely inhibiting reproduction of hosts through castration, parasites can redirect energy from host reproduction to parasite growth (e.g., Sousa, 1983; Lafferty, 1993; Bernot and Lamberti, 2008). There is even evidence that trematode parasites may physiologically-reinstate somatic growth of fully mature batellariad snails that would have ordinarily ceased growth (Miura et al., 2006).

Where 'gigantism' (enlarged size) does occur in infected hosts, it could affect: (1) the overall size structure of the population; (2) how individuals utilize resources; and even (3) intraspecific competition among hosts (e.g., Miura et al., 2006). 'Gigantism' is, however, not a general response to parasitic castration because some castrators reduce the growth of their hosts, sometimes modestly, sometimes markedly (Lafferty and Kuris, 2009). Further, studies noting 'gigantism' (i.e. non-parasitized hosts larger than unparasitized hosts of the same age) based on size differences alone can be problematic, as the effect of the parasite(s) itself (themselves) on host size can be confounded with the effect of individual host size on parasite level (Sousa and Gleason, 1989). Older (and generally larger) hosts also accumulate more parasites as they age (e.g., Sorensen and Minchella, 2001).

Etiological agents may have negative effects on growth rates where they damage tissue, shell or interfere with feeding. The metacercarial trematode parasite, *Renicola roscovita* negatively influences growth rate of blue mussels *Mytilus edulis* at mid intertidal elevations (Thieltges, 2006). The negative effect may result from direct tissue disruptions, interference of feeding by metacercariae (located in palps and visceral mass), and growth of metacercarial cysts within the host. For many marine species with lifespans >4 yrs, infection by trematodes appears to have limited or no growth effect, or even stunts growth (e.g., Sousa, 1983; Mouritsen and Jensen, 1994; Sorensen and Minchella, 2001; Miura et al., 2006; Lafferty and Kuris, 2009).

Shell borers such as sponges and polychaete worms may profoundly alter shell integrity, which is critical to ensure protection from predators (e.g. Stefaniak et al., 2005), to reduce mechanical stress (e.g., Marin et al., 2005) and to better control an individual mollusc's internal environment (e.g., Shumway, 1977). Infested molluscs respond by producing extra shell material to stem the inward progression of the intruder (e.g., Hoeksema, 1983; Rosell et al., 1999; Diez et al., 2013). However, as a consequence of responding to the infestation an individual's living space may be reduced or damaged, and, because of the costs of calcification (Palmer, 1992), energy may be allocated away from somatic growth and reproduction (Stefaniak et al., 2005; although see Le Cam and Viard, 2011 for examples where this was not the case).

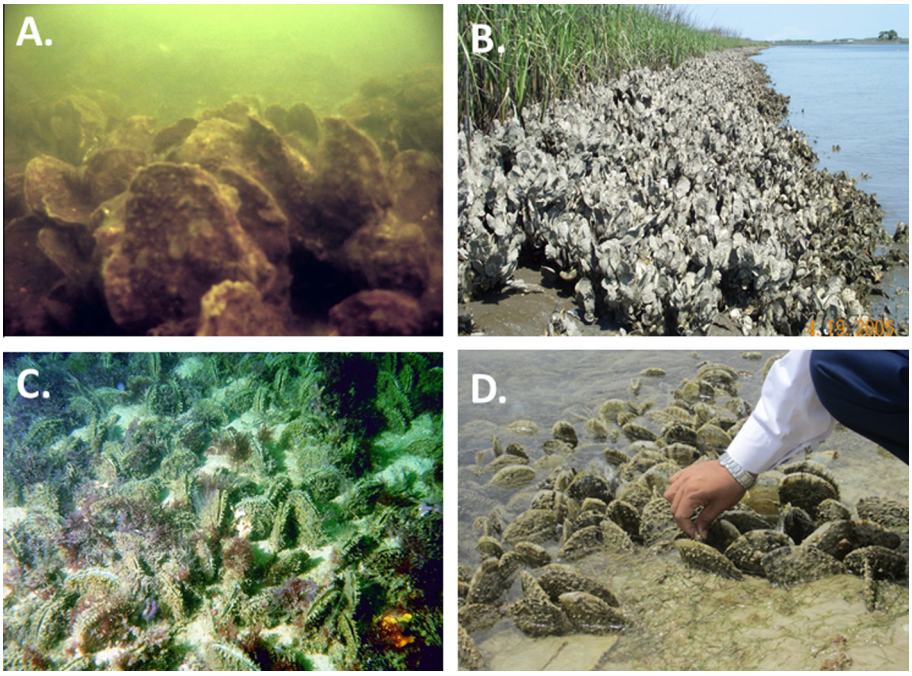

Fig. 4. Composite figure showing examples of intertidal and subtidal bivalve molluscs which, in forming reefs or dense aggregations, act as foundation species or ecosystem engineers. (A) Restored *Crassostrea virginica* oyster reef in the Great Wicomico River, Virginia, on the western shore of lower Chesapeake Bay, USA. The high-relief reef harbored about 1000 oysters m^{-2} of four age classes (Schulte et al., 2009) and is thought to resemble historical reefs from Colonial times. (*Source:* R.P. Burke and R.N. Lipcius, VIMS, VA, USA, image from ROV video); (B) restored fringing intertidal *Crassostrea virginica* oysters reefs adjacent to salt marsh, in South Carolina, U.S.A. (*Source:* L. Coen, SCDNR); (C) beds of the pen shell, *Atrina zelandica* in shallow New Zealand waters, forming epibenthic hard substrate for sessile organisms and acting as a foundation species (*Source:* Simon Thrush, Institute of Marine Science, University of Auckland, New Zealand) and (D) dense pen shell aggregation in an intertidal seagrass bed in Dubai. (*Source:* R. Grizzle, UNH, Durham, NH, U.S.A.)

4.2. Population-level effects

Parasites have been shown to interfere significantly with host populations (reviewed in Magalhães et al., 2015) by initiating diseases that affect reproduction in complex manners, as well as affecting absolute host population numbers (see Fig. 3). Negative relationships between the prevalence of diseases and population size and density are not, however, always apparent because pelagic larval dispersal can decouple rates of recruitment from the status of adult populations at a given locality and the broadcast spawning strategy of many molluscs makes relationships between reproductive adults and settlement weak. In general, relationships between disease prevalence and host density are more common among species with closed recruitment, such as estuarine gastropods with crawl away larva, than among species with open recruitment (e.g., Lafferty, 1993; Fredensborg et al., 2005). Only where there is widespread infection of adult hosts, across multiple localities, may relationships between parasitism and the density of hosts with pelagic larval dispersal be seen (Lafferty and Kuris, 2009).

Causation between diseases and significant mortality events are often difficult to establish in aquatic environments, so firm evidence for population-level effects of disease on wild populations of molluscs remain relatively scarce. Nevertheless, strong relationships between the timing of significant mortality events and parasite loadings in surviving individuals are highly suggestive of disease-mediated population control. For example, following a mass mortality of 73% of a Narragansett Bay (Rhode Island, U.S.A.) sea scallop, *Placopecten magellanicus* population in the winter of 1979–1980, 88% of surviving individuals were observed to have intracellular prokaryotes on their gills, suggesting an epizootic (Gulka et al., 1983). Similarly, following heavy mortality of *Ostrea chilensis* in the Foveaux Strait, New Zealand in 1986, histological examination revealed infection by the haplosporidian microcell parasite *Bonamia* sp. (later named *B. exitiosa*; Berthe and Hine, 2003; Carnegie and Cochennec-Laureau, 2004).

Spatial contrasts of mortality rates between infected and non-infected populations of a diverse array of molluscs have also been used as evidence of disease-control of molluscan population dynamics (e.g., Sousa, 1990; Villalba et al., 1993; Smith, 2001; Thieltges and Reise, 2007; Ramilo et al., 2014). The mortality rate of *C. virginica* infected by *P. marinus* is 2–3 times the non-epizootic rate, and has been accompanied by observations of significant population declines (Powell et al., 2012b; Lafferty et al., 2015). Overall, however, records of epizootics affecting wild populations of molluscs are relatively scarce as compared to records of epizootics affecting cultivated populations in aquaculture facilities (e.g., Cranfield et al., 2005; Lafferty et al., 2015).

In the Foveaux Strait, New Zealand, the cyclical pattern of mass mortality of the oyster *Ostrea chilensis* every 20–30 years is thought to have been caused by density-dependent infection of by the directly transmissible parasite, *Bonamia* sp. (Hine, 1996). It has been posited that high densities of oysters facilitate an increase in the prevalence and intensity of the directly-transmissible parasite, which in turn leads to mass oyster mortality. In reducing oyster density, mass mortality leads to a decrease in parasite transmission and, hence, its impact oysters (see also Solomieu et al., 2015). This allows oyster populations to build, and the cycle starts again.

4.3. Community-and ecosystem-level effects

In modifying individual- (e.g., morphology, movement) and population-level (e.g., density) traits of hosts, etiological agents have the potential to directly and indirectly modify interactions among species (e.g., see Fig. 3; Lafferty, 1992; Kuris and Lafferty, 1994; Thomas et al., 1997, 1998). Research (especially by Mourit-

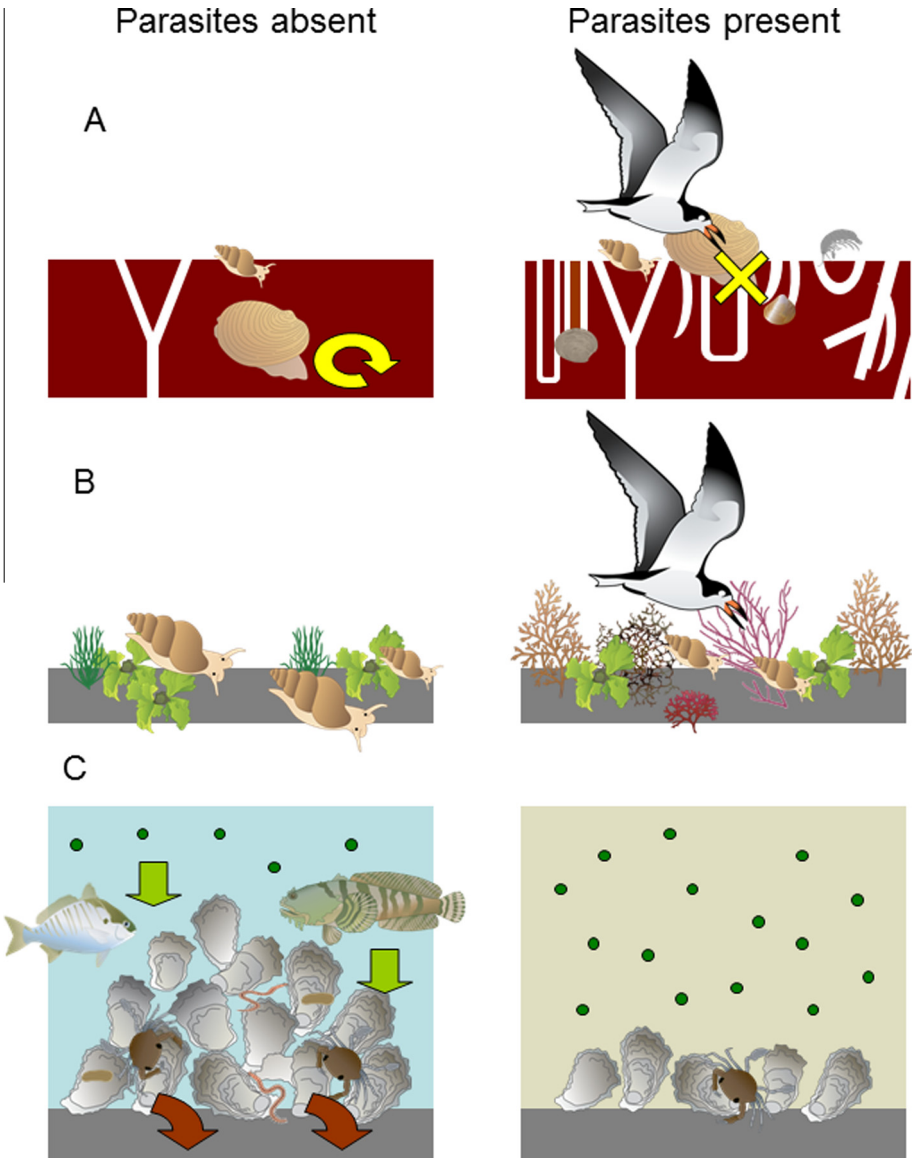

Fig. 5. Conceptual diagram illustrating how parasites may have particularly large impacts on community structure and ecosystem service provision where they affect ecosystem engineers. (A) Trematode parasites that infect the foot of cockles can prevent them from burrowing, making them more susceptible to predation from the definitive host, and modifying community structure by decreasing bioturbation by cockles which in some instances dislodges co-habiting taxa (Mouritsen and Poulin, 2002a); (B) trematode parasites that castrate gastropod hosts may alter community structure on rocky shores by reducing grazing pressure on algal communities through affects on gastropod density and size. Especially where grazing suppresses competitive dominants, major changes in community structure may occur following reduction in grazing (Mouritsen and Poulin, 2002a); (C) oysters are ecosystem engineers that support dense and diverse communities of fish and invertebrates and provide important regulating services by cycling nutrients and maintaining water quality (Beck et al., 2011). Hence, parasites that reduce the abundance of oysters may be expected to have large effects on ecosystem services. Constructed with images from the IAN Image and Video Library; http://ian.umces.edu/imagelibrary/.

sen, Poulin and their colleagues) has shown that parasites, especially trematodes, are important in structuring both soft bottom and rocky intertidal communities (e.g., Mouritsen and Poulin, 2002a, 2005, 2006; Wood et al., 2007; Mouritsen and Haun, 2008).

Etiological agents can have particularly large effects on community structure and function where they infect molluscs that are foundation species or ecosystem engineers (Fig. 4 habitats) and modify traits that are directly involved in their ecosystem engineering (see Fig. 5; Thomas et al., 1999, 2005). They may also have particularly large effects where they modify competitive (between hosts or even parasites) or predator–prey interactions (see Fig. 6; Sousa, 1983, 1990, 1993; Kuris, 1990; Lafferty and Kuris, 2005; Hatcher and Dunn, 2011; Dunn et al., 2012). Because of the large trait-mediated (indirect) effects etiological agents can have on community structure, many authors consider them equivalent to ecosystem engineers (e.g., Poulin, 1999; Thomas et al., 1998, 1999, 2005). Molluscs can act as 'ecosystem engineers' within a given community through a variety of interesting and often extraordinarily unpredictable interactions. In sedimentary environments, burrowing or epifaunal bivalves influence community structure through the bioturbation of sediments (e.g., Ciutat et al., 2007), and by providing often the only hard substrate for attachment of sessile (fouling) organisms (e.g., see Figs. 4 and 5; Ciutat et al., 2007; Norkko et al., 2006; Gribben et al., 2009).

Some of the most interesting effects demonstrated to date involve trematode parasites modifying ecosystem engineering by their cockle hosts. Trematode parasites that inhibit the burial of infected cockles can have large indirect effects on community structure because they modify the very traits of cockles associated with ecosystem engineering (reviewed in Combes, 2001; Moore,

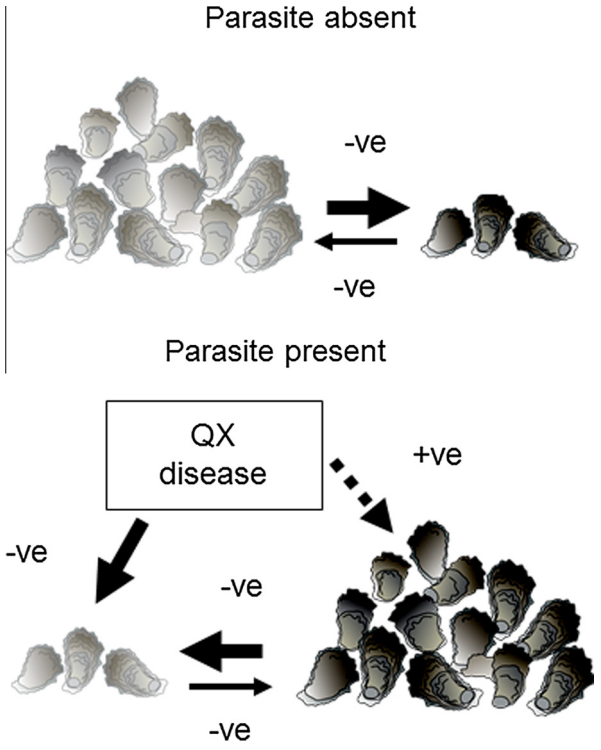

Fig. 6. Conceptual diagram illustrating how parasites may alter the outcome of competitive interactions where they have greatest impact on the competitive dominant. Along the Georges River, New South Wales, Australia, *Marteilia sydneyi* casuses mortality from QX disease in native Sydney rock (gray symbols), but not non-native Pacific (black symbols) oysters. The outcome is that space pre-empted by the native species is freed allowing the non-native oyster to proliferate (Nell, 2001). Constructed with images from the IAN Image and Video Library; http://ian.umces.edu/imagelibrary/.

2002; Hughes et al., 2012). Infaunal diversity and abundance in communities profits from decreased bioturbation (Fig. 5A; Mouritsen and Poulin, 2005). The 'immobilization' of cockles on the sediment surface provides a greater surface area of exposed shell for colonization (Thomas et al., 1998). In detecting a relationship between benthic community structure and cockle parasitism across 17 intertidal flats, Mouritsen and Poulin (2010) suggest that trematode parasites can modify community structure at scales spanning many kilometers. After bioturbation by ghost shrimp, parasitism of *Austrovenus* was the best predictor (positively) of abundance for 8 of the 49 (16%) most widespread species.

In another example, on mud flats of the Wadden Sea parasitism of the mud snail *Hydrobia ulvae*, can have community-wide effects as a result of changes to snail activity (Mouritsen and Haun, 2008). The mud snail, which can exceed densities of 15,000 m^{-2} facilitates local microphytobenthic biomass, perhaps through nutrient enrichment of sediments or its bioturbation (reworking the sediments) periodically exposing epipsammic (sediment-bound) diatoms to the surface and higher light. Mud snails infected by trematodes have reduced activity, which in turn indirectly decreases microphytobenthic (microscopic algae living in or on benthic sediments) biomass. Species composition of these microphytobenthos communities, as well as the resident infauna, also varies spatially according to the extent of snail infection in the area.

Diseases may also have large effects on communities where they modify the abundance of habitat-forming species. Oysters and mussels form complex three-dimensional structure that provides shelter and food to a variety of species, while also modifying the physico-chemical environment through filtration of water, benthic-pelagic coupling, and denitrification and nutrient assimilation (Fig. 5C; Gutiérrez et al., 2003; Newell, 2004; Beck et al., 2011). These important functions of oysters can be dependent on density of bivalves (zu Ermgassen et al., 2012; Green and Crowe, 2014; but see Dame et al., 2002), such that diseases that significantly reduced oyster and mussel densities could have large negative effects on biodiversity (Fig. 5C; Lafferty et al., 2015). In a modeling study, Powell et al. (2012a) suggest that Dermo disease, caused by the protistan parasite, *Haplosporidium nelsoni*, may play a role in preventing recovery of oyster reefs historically overharvested. Nevertheless, the effect of disease on the biodiversity and ecosystem services of oyster reef has not been directly quantified. A lot more work needs to be done to assess the assumptions and underlying datasets that are employed in modeling oysters and related mortality factors (e.g., Baggett et al., 2014). Empirical studies are needed directly assessing effects of disease on the shell accretion rates of bivalve-dominated reefs (e.g., Casas et al., 2015).

In addition to modifying the individual- and population-level traits of ecosystem engineers, modifying the outcomes of competitive interactions is another mechanism by which diseases can influence community structure (Fig. 6). On rocky shores, oysters and mussels are often the dominant occupants of space. Hence, diseases that influence their abundance or reduce their size can modify community structure by freeing space for colonization by other organisms. In the Georges River, New South Wales, Australia, up to 90% annual mortality of Sydney rock oysters, *S. glomerata*, from QX disease has feed space formerly pre-empted by the native oyster (Nell, 2001). This has allowed the non-native Pacific oyster, *C. gigas*, which is not affected by QX disease to colonize such that the non-native now accounts for 80% of the oysters on the foreshore of the upper reaches of the river (Fig. 6; Nell, 2001). In South Africa, two native trematode parasites that affect the native mussel *Perna perna,* but not the invasive mussel *Mytilus galloprovincialis* have helped to facilitate the invader by reducing the native mussel's occupation of space (Calvo-Ugarteburu and McQuaid, 1998). Impacts of parasites on competitive interactions will be greatest where the affected species is the dominant competitor.

Predator–prey interactions, such as grazing, are also important in structuring communities, and food-web structure, and may similarly be influenced by disease. Along the Atlantic coast of North America, *Littorina littorea* that are infected by the trematode *Cryptocotyle lingua*, consume 29% less ephemeral macroalgae than their uninfected counterparts (Wood et al., 2007). The net outcome is an altered macroalgal community composition, which in turn influence other organisms that depend on macroalgae for food or habitat (see also Fig. 5B).

In southern California, mortalities of black abalone (Lafferty and Kuris, 1993) from a rickettsia (intracellular bacterium) has permitted the colonization of fouling organisms, presumably because of reduction in abalone grazing (Miner et al., 2006). The intertidal species assemblage has shifted from one of bare rock and crustose coralline algae, which is conducive to abalone recruitment, to one of sessile invertebrates and sea urchins which is inhibitory – thereby creating a negative feedback to the abalone, and a state change in community.

Trophic transmission of pathogens from molluscs to their predators may result in disease among higher trophic levels or altered foraging behavior. As well as influencing existing predator–prey relationships, parasites may facilitate new trophic interactions. For example, trematodes that encyst the foot of cockles may not only prevent the bivalve from burying, but may also cause the bivalve to expose its foot to predators such as fish, as well as other opportunistic predators (Mouritsen and Poulin,

2003a, 2003b). Hence, these trematodes divert cockle biomass to other members of the food web.

Additionally, free-living parasites may serve as a food resource for mesopredators. Brachyuran crabs (*Carcinus maenas*) and shrimps (*Crangon crangon*) were observed to actively prey on free-living cercariae of trematode species that use the cockle *Cerastoderma edule*, as an intermediate host (Thieltges et al., 2008). Similarly, in an example involving molluscan parasites, but non-mollusc hosts, eulimid gastropod snails (*Sabinella shaskyi* and *Pelseneeria* spp.) that parasitize the pencil urchin, *Eucidaris galapagensis,* are preyed upon by a majid crab, *Mithrax nodosus* that is also commensal on urchins (e.g., Sonnenholzner and Molina, 2005; Sonnenholzner et al., 2011). Lafferty et al. (2006) estimated that perhaps 44% of links within one well-studied estuarine food web involved predation on parasites.

Parasites that are host-specific or differentially affect native and non-native species may influence the outcome of biological invasions through enemy release or through biotic resistance. On average introduced species escape at least 75% of the parasites of their native range (Torchin et al., 2003) and in many instances may not acquire the parasites of native species. In Lake Malawi, a highly invasive introduced morph of the gastropod *Melanoides tuberculata* is free from trematode infection, including the opisthorchid trematodes that sterilize and induce gigantism within populations of the native morph of *M. tuberculata* (Genner et al., 2008). It has been speculated that this enemy release may contribute to the invasion success of the non-native morph, by giving it a survivorship and reproductive advantage over the parasitized native morph, although this has not been empirically tested.

Conversely, where native parasites are able to infect non-native hosts they may impede the invasion through 'biotic resistance' (Dunn and Hatcher, 2015). In the North Sea, up to 80% of introduced *Crassostrea gigas* and *Ensis americanus* were infected with native trematode parasites (Krakau et al., 2006). Although there is a growing number of studies comparing the parasite diversity of native and non-native species (e.g., Torchin et al., 2005; Blakeslee et al., 2012), it is unclear how this relationship changes with time since invasion or how it influences invasion success and the structure and function of the recipient community. Torchin et al. (2005) speculate that where molluscan invaders with few parasites displace those with diverse parasitic communities, the increased number of nodes and links in the food web formed by the invader and its parasites in food web topology might fail to compensate those lost through displacement. Widely translocated species such as *C. gigas* might provide ideal candidates with which to test hypotheses about when and where parasites help and hinder biological invasion.

Aside from research on trematode parasites and how they modify ecological interactions, relatively little research has directly addressed how molluscan diseases influence biodiversity and ecosystem services. The effect of parasites on molluscan ecosystem engineers, which generate biogenic habitats (e.g., oysters and mussels), or on "keystone predators" and grazers that have large cascading effects on multiple trophic levels needs to be evaluated more broadly and rigorously. More widespread monitoring of disease as part of molluscan restoration programs might help to assess how this factor influences ecosystem service provision. Inclusion of parasites in biodiversity assessments might help to advance our understanding on how they influence food-web structure.

5. Evolutionary impacts of molluscan diseases

In the previous sections we have considered how parasites by modifying either the survivorship, morphology, behavior or reproduction of hosts, can have individual, population, community and even ecosystem-level ecological impacts. In this section we consider how parasites can impact host fitness, how they can drive evolutionary change, and how the evolution of hosts may in turn drive parasite evolution. We also consider how the genotypic changes in hosts, which occur as a result of parasitism, can drive the observed phenotypic changes described in Section 4.1.

5.1. Arms race between hosts and parasites

As May and Anderson (1979) point out in their Nature article (Part II), any discussion on the relationships among populations that characterize infectious diseases "must ultimately take account of the evolutionary pressures on both hosts and parasites." Where there is a fitness cost to parasitism, there is selection for hosts with greater resistance to pathogens. However, as parasites must be able to infect hosts to survive, there may be subsequent selection for parasites with increased virulence and pathogenicity. The net effect is co-evolution of parasite and host.

A great deal of effort (e.g., Grosholz, 1994; Langand and Morand, 1998; Sorensen and Minchella, 2001) has been directed in order to better understand how resistance by hosts against their parasites and pathogens evolves and is preserved (reviewed in Frank, 1996). The numerous models that have been generated to examine host–parasite interactions assume that there is significant genetic variation among host individuals in resistance (Frank, 1996). The assumption is that resistance has a cost so will be selected for where parasites are numerous, but selected against where parasites and pathogens have significantly less affect. In this way susceptible and resistant individuals may co-exist. Where selection is variable and diverse one would predict heritability, h^2 (Falconer, 1981) for resistance and susceptibility to be significant.

5.2. Experimental evidence of selection for and heritability of host resistance to parasites

There are a number of examples, from marine and freshwater habitats, in which exposure of naïve molluscan hosts to macroparasites has been experimentally manipulated to examine selection for and heritability of host resistance to parasites. Most of these studies have involved larval trematodes infecting gastropods (Lafferty and Harvell, 2014). Gastropods and trematodes provide an ideal system with which to investigate the effects of parasitism on evolutionary processes because they are abundant and easy to manipulate, and because gastropod hosts may be infected by multiple life-history stages of trematode that vary in their effect on fitness.

Many groups such as the echinostome digenean trematodes have a complex life cycle with three hosts, two of which are intermediates with great specificity of host, along with a generalist final host (Fried, 1997; Sorensen and Minchella, 2001). Predator–prey transmission is involved in these latter two hosts, as is common for many helminths (nematodes, cestodes, and trematodes; Sousa, 1994). Asexual reproduction of the trematode takes place in the first intermediate host, and typically results in host castration. Trematode infection is significantly less pathogenic in the second intermediate host (most commonly also a gastropod), and even less pathogenic in the final vertebrate host (e.g., Fried, 1997; Moore, 2002).

An elegant set of experiments by Langand and Morand (1998) made use of the complex life cycle of trematodes, manipulating exposure of a naïve host, the South American freshwater gastropod *Biomphalaria glabrata,* to larval miracidia of the African digenean parasite, *Echinostoma caproni* to assess heritability of resistance and susceptibility in this system. The snail, which is the first intermediate host for the trematode, is castrated by the parasite, so it

was predicted that there would be a large fitness benefit of resistance. Sexually mature snails were exposed to the parasite for 20–35 days and offspring from susceptible and resistant individuals retained, and similarly exposed to the parasite once they, in turn, reached sexual maturity. Using a quantitative genetic model they estimated heritability of resistance and susceptibility among hosts. Unexpectedly, their results showed high, but unequal, heritability of both resistance and susceptibility, possibly due to the dominance of resistance. A follow on and later study suggested that the cost of resistance is also delayed maturity (Langand and Morand, 1998).

In a simpler marine system, Grosholz (1994) used related families of the bivalve mollusc *Transennella tantilla,* cultured in the laboratory and then exposed them in the field to metacercaria of the digenian parasite *Parvatrema borealis* to quantify heritable variation in parasite resistance. He found moderate, but significant, heritability (0.36) for resistance to the parasite in the bivalve – a second intermediate host for the parasite. This was despite the fact that infection of molluscs with metacercaria typically has much lower fitness consequences than infection with miracadia, as in the first example. Motile miracidia settle in hosts to become sporocysts that then asexual reproduce. The host is typically castrated or has greatly reduced reproduction, whereas metacercaria generally manifest at a low disease level, only increasing with greater infestation.

In contrast to macroparasites, the microparasites that many molluscs are exposed to are much more difficult to experimentally manipulate so as to examine co-evolution of parasite and host. Nevertheless, selective breeding programs that successively expose multiple generations of molluscs to pathogens, and keep and breed the survivors (e.g., Ford and Haskin, 1987; Ragone Calvo et al., 2003; Dove et al., 2013), suggest that resistance of molluscs to microparasites is also likely to be heritable. In many instances, these selectively bred molluscs display greater resistance to disease than conspecifics from unselected lines (e.g., Ragone Calvo et al., 2003; Samain et al., 2007; Dove et al., 2013; Lynch et al., 2014; Degremont et al., 2015). Furthermore, long-term descriptive studies also suggest co-evolution of microparasites and hosts.

For example, long-term field sampling in Delaware Bay is suggestive that the development by Eastern oysters, *Crassostrea virginca*, of resistance to MSX disease, caused by *Haplosporidium nelsoni*, is dependent on the extent of freshwater refuges from the parasite (Hoffman et al., 2009). Enhanced resistance of Eastern oysters (*C. virginica*) to MSX disease was seen following the 1957–1959 epizootic, but plateaued between 1960 and 1987, presumably because most of the surviving oysters were in low-salinity 'refugia' where they were protected from sustained selection and continued to contribute susceptible progeny to the population. In 1984–1986, however, an extended period of drought eliminated these low-salinity disease refuges, resulting in significant mortality across the Bay populations, and leading to a major increase in the level of resistance to MSX disease (e.g., Hofmann et al., 2009; Carnegie and Burreson, 2011; Ford et al., 2012; Burge et al., 2014; Arzul and Carnegie, 2015).

5.3. Modeling studies that address host resistance

In addition to the above experimental and observational studies, gene-based population dynamic models have been used to simulate the development of disease resistance in molluscs under various scenarios (see Powell and Hofmann, 2015 and Degremont et al., 2015 for further discussion this volume). A model's utility is to characterize data, phenomena, or processes logically and objectively. By doing this it can simplify the complex interactions associated with diseases and host populations. However, one typically must simplify the system one is trying to model by reducing the number of parameters (=variables) that one presumes to be well understood to a smaller number of 'state variables' and associated mathematical functions. Models obviously have few or many basic assumptions. As in all assessments, models are only as good as the data that are used to construct them, and later test them and it is critical that the relevant statistics and related assumptions be followed.

For example, modeling approaches have been used to consider how host genetics and population dynamics interact to influence development of resistance to Dermo disease, caused by *Perkinsus marinus,* among *Crassostrea virginica* oysters (e.g., Powell et al., 2011; Lafferty et al., 2015; Powell and Hofmann, 2015 for further discussion this volume). *Perkinsus marinus* is an obligate parasite of *C. virginica*, which infects its oyster host during feeding, entering the hemolymph where it spreads and proliferates (see Ben-Horin et al., 2015). The disease typically kills oysters following at least one spawning cycle, such that animals can spawn several times before death (Powell et al., 2012b). Hosts shed the parasite into the water through either their feces or death and resulting decomposing (e.g., Bushek et al., 2002). Infection rate (prevalence) varies both temporally and spatially with infections as high as 100% (Bobo et al., 1997; Kim and Powell, 2006, 2007; Lafferty et al., 2015).

In simulated oyster populations exposed to *P. marinus* (Dermo disease) over multiple generations, resistance to the parasite in *C. virginica* increased with time, and in proportion to the extent with which the disease influenced mortality (Powell et al., 2011; Lafferty et al., 2015). As overall mortality rates in the population declined as a result of increasing disease resistance among individuals, the rate at which disease resistance was acquired, in turn, diminished. The net effect was a relatively slow rate of evolution of disease resistance to *P. marinus* (Dermo disease). Subsequent modeling showed that development of resistance was also slowed by periodic decreases in exposure to the pathogen (Powell et al., 2012b). Subsequent modeling needs to consider how the evolution of disease resistance among oysters might be influenced by the cyclic nature by which the abundance of *P. marinus* responds to variation in temperature and salinity (see Section 3.2; Bushek et al., 2012).

The above models assume that *Perkinsus marinus* is a passive player in the interaction such that selection is unidirectional. In this system, the parasite is obligate parasite on its host, and hence it seems unlikely, that a co-evolutionary arms race would not be involved, with simultaneous co-evolution of the host. Alternatively, in contrast to the assumptions of the model, selection may not be on oysters that 'resist' the parasite, but instead oysters that continue to be infected, 'tolerating' the parasite by suffering only reduced physiological impacts. This is a much more evolutionarily stable state (ESS) as it benefits both parasite and host. Irrespective of the mechanism, some development of disease resistance among *C. virginica* exposed to Dermo over multiple generations is supported by field observations. Notably, we know that when naïve "sentinel" oysters are deployed in the field, they more rapidly acquire intense and ultimately lethal *Perkinsus* infections than oysters from areas long-exposed to *P. marinus* (R. Carnegie, pers. comm.). Additionally, subsequent modeling needs to consider how the evolution of disease resistance among oysters might be influenced by the cyclic nature by which the abundance of *P. marinus* responds to variation in temperature and salinity (see Section 3.2; Bushek et al., 2012).

6. Disease management, its ecological costs and benefits

In many regions of the world, disease is viewed as a major contributing factor to the decline of wild mollusc populations

(e.g., Moyer et al., 1993; Ford and Tripp, 1996; NRC, 2004a; Elston and Ford, 2011; Lafferty et al., 2015), and an impediment to native or non-native molluscan aquaculture and restoration efforts (e.g., Coen and Luckenbach, 2000; Carnegie, 2005, 2009). In response to this perceived disease issue, increased biosecurity measures have been put in place to reduce the translocation of disease, and aquaculture production has increasingly shifted to cultivation of animals selectively bred for disease resistance or non-native species that are less susceptible to native parasites (e.g., Littlewood et al., 1992; Launey et al., 2001; Culloty et al., 2004; Encomio et al., 2005; Harding, 2007; Elston and Ford, 2011; Kraeuter et al., 2011; Ford et al., 2012). As selectively bred and non-native molluscs have been increasingly adopted by aquaculture industries, there has also been interest in applying them to restoration projects (e.g., Allen et al., 2003; Arnold et al., 2005; Crane et al., 2013). The application of selective breeding and non-native species to aquaculture and restoration has been highly controversial, due to the mixed success of such approaches and their potential to produce major changes to aquatic food webs (e.g., Carlsson et al., 2006; Gaffney, 2006; Hare et al., 2006; Camara and Vadopalas, 2009). In this section, we review disease management strategies and, in keeping with the ecological focus of this review paper, discuss the direct and indirect ecological effects of these.

6.1. Biosecurity

Increasingly, government agencies around the world are initiating and implementing biosecurity programs in response to escalating disease outbreaks among molluscan and other aquaculture industries. Typically, these programs include: (1) disease prevention; (2) disease monitoring; (3) cleaning and disinfection between production cycles; and (4) general security precautions.

Some programs are primarily aimed at minimizing the translocation of diseases with stock movements. Others such as the Interstate Shellfish Sanitation Conference (ISSC) and U.S. Food and Drug Administration's National Shellfish Sanitation (NSSP) (see also Leonard and Macfarlane, 2011) programs in the U.S.A. are aimed at preventing the trophic transmission of diseases from molluscs to humans (*see* http://www.issc.org/NSSP/, http://www.fda.gov/Food/GuidanceRegulation/FederalStateFoodPrograms/ucm2006754.htm). In addition to mollusc-specific management programs, ballast water management plans and other quarantine procedures have also been implemented to minimize translocation of disease by vessels, either in ballast water or in hull-fouling species (e.g., International Convention (IMO) for the Control and Management of Ships' Ballast Water and Sediments: IMO, 2004). Concerns about the movement of diseases and other hitchhikers with shell-stocks used for replanting or restoration efforts have brought about routine 4–6 month minimum quarantine of shell stocks before use (Bushek et al., 2004; Cohen and Zabin, 2009).

Disease prevention may include sourcing stock and feed from certified disease-free locations, or holding animals under quarantine conditions until they can be verified as disease-free. Disease monitoring involves regular assessments of the ''quality ''of the water in which molluscs are growing and the health of animals, using lethal and/or non-lethal sampling (see Rodgers et al., 2015). Non-lethal techniques for detecting pathogens in molluscs may include gill or hemolymph sampling, and immunological assays, while lethal sampling may include bacterial cultures, viral isolation and histopathology (e.g., other chapters in this volume). Where pathogens are detected in the water and/or in animals, areas may be closed to harvest of molluscs, and translocation of stock to non-infected sites might be prohibited. Furthermore, equipment, such as vessels, molluscan bags and tongs may need to be disinfected prior to use at other locations. In some instances, harvested molluscs are routinely relayed to clean seawater for 'depuration' prior to being sent to market, so as to minimize risk of trophic transmission of bacterial diseases and other pathogens (e.g., http://www.fda.gov/downloads/food/guidanceregulation/federalstatefoodprograms/ucm350344.pdf).

Biosecurity measures may have unintended flow-on effects on ecological communities where they introduce biocidal agents into the environment. For example, there is a rich literature on how antifouling paints, such as tributyltin, impact on the growth, development, reproduction and survival of organisms ranging from bacteria to fish and mammals (e.g., Alzieu, 1991; Antizar-Ladislao, 2008). With growing international awareness of the ecological impacts of biocides, the most noxious of these anti-fouling products are now banned by most countries (e.g., International Maritime Organization Anti-Fouling Strategy; IMO, 2001).

6.2. Use of selectively bred, disease-resistant, molluscs for aquaculture and restoration

Historically, molluscan aquaculture involved the collection of wild-caught juveniles and grow-out of these using farming practices that accelerate growth rate (reviewed in NRC, 2009, 2010; Lafferty et al., 2015). As disease epidemics have threatened the viability of molluscan aquaculture industries, they have increasingly turned to cultivation of animals selectively bred for disease resistance and rapid growth (e.g., Ragone Calvo et al., 2003; Dove et al., 2013; Degremont et al., 2015). Selective breeding programs in many instances take advantage of natural variation in the susceptibility of individuals to disease, and the heritability of this trait (discussed in Elston and Ford, 2011; Hedgecock, 2011; Camara and Vadopalas, 2009).

Selective breeding programs have been widely applied to oyster aquaculture industries to produce lines that are resistant to diseases caused by protozoan parasites (e.g., Littlewood et al., 1992; Culloty et al., 2004; Elston and Ford, 2011; Kraeuter et al., 2011). These programs typically use a mass selection approach, where multiple generations of animals are exposed to epizootics and the survivors are successively mated with one another. Selective breeding programs have been used to produce lines of Eastern oysters (*Crassostrea virginica*) that are resistant to MSX (e.g., Ford and Haskin, 1987; Arzul and Carnegie, 2015), Dermo diseases (e.g., Ragone Calvo et al., 2003), and Juvenile Oyster Disease (e.g., Farley et al., 1998), Sydney rock oysters (*Saccostrea glomerata*) that are resistant to QX disease or Winter Mortality (Dove et al., 2013), as well as Pacific oysters (*Crassostrea gigas*) that are resistant summer mortality (e.g., Hershberger et al., 1984; Samain et al., 2007; Dégremont et al., 2010) and Ostreid Herpes virus 1 (OsHV-1). Use of selectively bred oysters in aquaculture has in many instances led to enhanced performance (survival, growth and resistance) against particular parasites, as compared to conspecifics from unselected lines (e.g., Ragone Calvo et al., 2003; Samain et al., 2007; Dove et al., 2013; Lynch et al., 2014).

In several instances, restoration projects have also capitalized on lines of oysters selectively bred for disease resistance by the aquaculture industry. In the Chesapeake Bay, the parasites *Perkinsus marinus* (Dermo disease) and *Haplosporidium nelsoni* (MSX disease) are viewed by some as major impediments to the restoration of native Eastern oyster, *Crassostrea virginica* (NRC, 2004a; Coen and Luckenbach, 2000). Consequently, domesticated lines of Eastern oysters, bred for the aquaculture industry for disease resistance and rapid growth (Ragone Calvo et al., 2003), have been used for seeding oyster reef restoration projects since 1999 (e.g., Luckenbach et al., 1999; Brumbaugh et al., 2000; Coen and Luckenbach, 2000; Brumbaugh and Coen, 2009; Baggett et al., 2014). The rationale is that these disease resistant lines might survive and reproduce for longer than wild-type seed, and in bays with high rates of larval retention, may serve as persistent natural

incubators for local recruitment of disease tolerant progeny (e.g., Hare et al., 2006; Carlsson et al., 2008).

Both the use of selectively bred oysters in aquaculture and restoration has been controversial. Selectively bred stocks may display reduced allelic diversity (e.g., Gaffney et al., 1992) and heterozygosity (e.g., Allendorf, 1986; Dillon and Manzi, 1987) relative to wild populations. This may result in inbreeding depression, which lowers survivorship and fitness of the inbred individuals (e.g., Bierne et al., 1998; Launey and Hedgecock, 2001) and may diminish the genetic 'health' of wild populations if the selectively bred and wild oyster populations interbreed (Wang and Ryman, 2001). Further although the selectively bred line(s) may exhibit superior performance when exposed to a particular parasite for which resistance has been selectively bred, genotype by environment interactions may result in this line being sub-optimal under alternative 'environmental' or 'biological' conditions (e.g., Mallet and Haley, 1983; Rawson and Hilbish, 1991; Camara and Vadopalas, 2009; Hedgecock, 2011; Degremont et al., 2015). For example, Sydney rock oysters selectively bred for disease resistance appear to be less resilient to enhanced pCO_2 than wild oysters (Thompson et al., 2015). Additionally, molluscan species selectively bred for resistance against one disease, are frequently still susceptible to others (Dove et al., 2013) and may potentially lose the genetic diversity required for development of resistance to new epidemics of emerging diseases (reviewed in Camara and Vadopalas, 2009).

Among molluscs (especially bivalves), the extent of introgression between selectively bred aquaculture and wild populations has not been investigated. However, there is evidence that interbreeding between cultivated salmon that have escaped from farms and wild salmon has alteration the genetic integrity of the wild population, possibility reducing adaptation to local conditions (e.g., Bourret et al., 2011). Several studies have investigated the contribution of selectively bred oysters to natural recruitment in estuaries where the selectively bred oysters have been seeded (e.g., Hare et al., 2006; Carlsson et al., 2008). They have found very limited introgression, which in some instances does not statistically deviate from zero (Hare et al., 2006; Carlsson et al., 2008). It has been hypothesized that the low genetic introgression may be due to: (1) high rates of predation on the oyster seed; (2) the relatively small contribution of the selectively bred genotypes (e.g., DEBY) to total oysters in these estuaries (i.e. the genotype is being swamped); and/or (3) low fitness under natural conditions caused by the selection process (e.g., Carlsson et al., 2008). Such results question the feasibility of genetically enhancing disease-afflicted wild populations through seeding.

Based on the results of modeling and sampling some (e.g., Powell et al., 2012a; Lafferty et al., 2015) have suggested that *C. virginica* reef accretion is unfeasible, and hence the benefits of oyster reef restoration negligible, in any estuary where Dermo disease is a controlling influence on population dynamics. Nevertheless, the success of oyster reef restoration projects at a variety of sites in the mid-Atlantic, and southeastern Atlantic, U.S.A., including some that are in high-salinity (over 30 ppt) and Dermo-affected with 100% prevalences (e.g., Rodney and Paynter, 2006; Gregalis et al., 2008; Beck et al., 2009; Powers et al., 2009; Schulte et al., 2009; Baggett et al., 2014; Coen and Humphries, in press) suggests that this is an unduly pessimistic view-point.

6.3. Introduction of non-native molluscs for aquaculture and/or ecological restoration

An alternative approach to mollucsan aquaculture and restoration in disease-afflicted waters is to use non-native species, which are not susceptible to endemic diseases (e.g., Mann et al., 1994; Schlaepfer et al., 2011). Molluscs are among the most translocated species in the world for aquaculture, with oysters alone introduced to over 73 countries (reviewed in Ruesink et al., 2005). Although the rationale for introducing non-native species may be multiple, resistance to native pathogens that plague native analogs is commonly among the arguments put forward (reviewed in NRC, 2004a; Keiner, 2010).

For example, disease was a major driver in the proposal to introduce *Crassostrea ariakensis* to the Chesapeake Bay (reviewed in NRC, 2004a). Unlike the native oyster, *Crassostrea virginica, C. ariakensis* is not susceptible to MSX and Dermo (Calvo et al., 2001). The introduction was proposed as a way to rebuild the oyster industry, ravaged by decades of overharvest and declining water quality, and more recently disease, and return ecosystem services once provided by the native oyster. The proposal was, however, eventually rejected on the basis of its ecological risks (reviewed in NRC, 2004a; Ruesink et al., 2005), and the uncertainty of economic and ecosystem benefits in part due to the susceptibility of the oyster to a local *Bonamia* sp. parasite and native predators (e.g., Burreson et al., 2004; Bishop and Peterson, 2006; Carnegie et al., 2008; Grabowski et al., 2007; Moss et al., 2007; USACE, 2009).

Despite some redundancy in the biodiversity and services supported by closely related native and non-native molluscs (Bishop and Peterson, 2006; Harwell et al., 2010; Wilkie et al., 2012), introductions of non-native species can have large unintended consequences (e.g., Ruesink et al., 2005). Introductions of non-native species for aquaculture have in some instances resulted in establishment of feral populations (Diederich et al., 2005; Bishop et al., 2010), that may outcompete native species (Diederich, 2006; Krassoi et al., 2008). Additionally, movement of molluscs may introduce non-native organisms, including parasites, diseases and harmful algae to new environments (Ruesink et al., 2005; Hégaret et al., 2008).

Adherence to the International Council for Exploration of the Sea's (ICES) code of practice for marine introductions may reduce the risk of unintended consequences of molluscan introductions (NRC, 2004a). The protocol requires that there is strong rationale for a species introduction and that reviews of the biology and life history of the organism have been conducted, without uncovering areas of significant risk. If the introduction is to proceed, the F1 or subsequent generations of quarantined brood stock should be transplanted to the new environment, to minimize risk of unintended species translocations. Nevertheless, even following these protocols, introductions are not without risk. Introduction of triploid molluscs, that are theoretically sterile, may further reduce the risk of non-native species attaining pest status through proliferation and spread (e.g., Allen and Guo, 1996; NRC, 2004a, 2004b), and may be an option where the introduction is intended to support an aquaculture industry based on hatchery supply of seed. However the production of triploids is not totally failsafe, as individuals may revert to reproductive diploids through time (e.g., Blankenship, 1994; Dew et al., 2003; NRC, 2004a, 2004b).

7. Emerging diseases, stressors and associated ecological impacts

Reports of disease in the ocean are on the rise (Ward and Lafferty, 2004; Harvell et al., 1999). The molecular revolution has produced new techniques that allow rapid diagnosis of pathogens (e.g., Reece et al., 1997, 2008; De Faveri et al., 2009; Wight et al., 2009; Wilbur et al., 2012). Whereas in the past, epidemiologists to a large extent relied on often ineffective culturing techniques and histology to identify marine pathogens, species-specific DNA probes now enable screening for a broad range of pathogens (e.g., Harvell et al., 1999). Although this trend may, in part, be

explained by increasing search effort and the development of powerful molecular tools that enable rapid diagnosis of pathogens, these factors along cannot explain the increasing rate of pathogen detection in molluscs (Ward and Lafferty, 2004, see Gómez-Chiarri et al., 2015). In this section we consider how globalization and the rise of aquaculture, climate change and coastal development might be contributing to the emergence of diseases and global epidemics within molluscs.

7.1. The role of globalization and the rise of molluscan aquaculture in contributing to emergence of disease

As discussed previously in this review (see Sections 3.2 and 3.3) globalization and aquaculture have in combination been implicated in increasing rates of pathogen translocation at local and global scales. Every day, thousands of species are passively translocated across the world's oceans by ballast water or on the hulls of ships (e.g., Carlton and Mann, 1996; Ruiz et al., 2000). Translocation of fish, invertebrates and algae by shipping has received the most attention, but microorganisms, including pathogens, are also among those species translocated (e.g., Ruiz et al., 2000). Although international ballast water initiatives have been instigated to curb the rate of passive introductions (see Section 6.1), it is inevitable that some organisms, particularly those at the smaller end of the spectrum, will continue to get through. At smaller scales, movement of molluscan aquaculture stocks from site to site to maximize rates of growth has also resulted in unintentional translocations of pathogens (e.g., Culloty et al., 1999; Hine, 1996; Cranfield et al., 2005; Lynch et al., 2010).

The unintentional translocation of aquatic molluscan pathogens (and their nonnative hosts) is particular problematic given that the rise of aquaculture has in some instances resulted in biotic homogenization, with the same productive and commercially valuable species cultivated globally. Per capita supply from aquaculture increased from 0.7 kg in 1970 to 7.8 kg in 2006, an average annual growth rate of 6.9%, and several species of mollusc, especially, oysters have contributed to this trend (FAO, 2015). For example, the Pacific oyster *Crassostrea gigas*, which is native to Japan, is now cultivated in at least 42 countries and in 2013, had an annual production of 4.38 million tonnes, more than any other species of fish, mollusc or crustacean (FAO, 2015). Increasing distributions of hosts enhances the probability that translocated parasites will find a match with host species when arriving at new locations.

The rapid global spread of oyster herpesvirus type I (OsHV-1) is an example of how global trade might facilitate the rapid spread of diseases that infect widely distributed hosts. OsHV-1 causes mortality of larval and juvenile bivalves from the species *Crassostrea gigas*, *Ostrea edulis*, *Venerupis* (formerly *Ruditapes*) *decussatus*, *V. philippinarum*, and *Pecten maximus*. The virus is also found in adult bivalves, presumably under a latent form. In 2008, a variant (OsHV-1 µvar) of the virus was detected that caused up to 100% mortality of *Crassostrea gigas* in France, Ireland and England (Segarra et al., 2010). Subsequently, mortality attributable to OsHV-1 µvar was detected among C. *gigas* in New Zealand (November 2010), Australia (December 2010) and the Netherlands (2011), and in Spain and Italy OsHV-1 µvar was also detected among *C. gigas*, although without oyster mortality (Herpes virus OsHV-1, EU Reference Laboratories, 2012, http://www.eurl-mollusc.eu/Main-activities/Tutorials/Herpes-virus-OsHV-1). Furthermore, herpesvirus OsHV-1, which is very close to the OsHV-1 µvar from France was reported from China (2002) and Japan (2010), although without mortality (Herpes virus OsHV-1, EU Reference Laboratories, 2012). Similarly an ostreid herpes virus that is related but distinct to OSHV-1 has caused mortalities of juvenile *C. gigas* in Tomales Bay, California, U.S.A. (Friedman et al., 2005).

As international vessels movements continue to grow with the world's population, aquaculture industries continue to expand, and live molluscs are moved around the world for food, globally coordinated strategies will be required to ensure that epidemics of marine species do not become increasingly frequent and compromise food security and the ecological values provided by molluscan species.

7.2. Climate change and disease

In addition to globalization, climate change is among the factors implicated in shifting patterns of marine diseases (e.g., Harvell et al., 2002, 2009; Baker-Austin et al., 2013; Burge et al., 2014). Warming, ocean acidification, and other associated physical impacts such as altered patterns of rainfall, ocean currents and stratification may impact molluscan disease by influencing distributions and abundances of hosts and parasites, as well as immune responses, which influence host–parasite interactions. Despite the large demonstrated effects of both climate change (reviewed by Harley et al., 2006) and disease (reviewed by Ward and Lafferty, 2004) on marine taxa, their combined effects have, however, received relatively little research attention to date (see reviews by Harvell et al., 2002, 2009; Burge et al., 2014).

Of climate stressors, warming has by far received the most attention in terms of the impacts it might have on parasite–host systems (e.g., Harvell et al., 2002, 2009; Burge et al., 2014). Biological reaction rates are strongly influenced by temperature, with enhanced metabolism at high temperatures in some instances enhancing growth, development and reproduction, but in other instances reducing survivorship where rates of food consumption do not keep up with increased metabolic needs (Lafferty, 2009). The strong seasonality of many molluscan diseases (see Section 3.2) suggests that temperature plays an important role in controlling infection, and that consequently climate warming may have significant effects on molluscan disease. Several authors including Poulin (2006) have found evidence of relationships between temperature and trematode cercarial shedding rates, but is unclear how survivorship is simultaneously affected by temperature. Growth rates of both marine bacteria and fungi are positively correlated with temperature (Holmquist et al., 1983; McManus et al., 2004) such that warming coastal waters may be expected to enhance the frequency of bacterial diseases, such as *Vibrio* spp. in molluscs (e.g., Harvell et al., 2002; Elston et al., 2008; Travers et al., 2009, 2015; Vezzulli et al., 2010, 2012, 2013).

Unfortunately, with few exceptions (e.g. Bushek et al., 2012), long-term data are not yet available to test hypotheses about relationships between warming waters and incidence of molluscan disease. Consequently effects of warming have in many instances been inferred from variation in disease incidence across temperature gradients, decadal patterns of climate oscillation (e.g., ENSO), or from mesocosm experiments investigating effects of temperature on parasite–host systems (e.g., Moore et al., 2000b; Raimondi et al., 2002). Inferences about climate warming made from such studies assume that there is little or no evolution of parasites or their hosts across the longer time scales at which climate warming is occurring.

Other major consequences of climate warming include poleward shifts in species distributions and changes in the timing of key events such as reproduction and migration (Parmesan, 1996; Walther et al., 2002). In some instances these changes may result in greater overlap in the distributions between parasites and their hosts. For example, the already observed poleward range expansion in several *Vibrio* spp. (e.g., Vezzulli et al., 2010, 2012, 2013; Baker-Austin et al., 2013) may result in molluscs, and their predators, being more susceptible to these bacterial infections at localities where they were not found before. In other instances,

however, asynchrony in the range and/or shifts in the phenology of parasites and their hosts may decrease overlap in distributions and hence reduce the incidence of disease. Parasites such as trematodes that display a high degree of host-specificity, and depend on multiple hosts to complete their life cycle may be particularly sensitive to such shifts as it may be difficult for them to switch hosts rapidly.

Altered rainfall patterns associated with climate change may also be expected to have a large effect on molluscan disease due to the strong relationships between salinity and disease seen for many molluscan parasite–host systems (see Section 3.6). As described for temperature above, shifting salinity gradients may result in changes to molluscan host–parasite relationships by altering overlap between parasite and host distributions, by altering the abundance of parasites and/or hosts and by influencing parasite immune systems (see Allam and Raftos, 2015). As previously noted (see Section 3.6), among Sydney rock oysters, *Saccostrea glomerata*, phenoloxidase, a key component of the oyster immune system, is inhibited by transient environmental stressors such as low salinity (Butt et al., 2006).

As calcifiers, molluscs may be particularly prone to the effects of ocean acidification, especially in marine environments where organisms are not adapted to fluctuating pH levels (e.g., Fabry et al., 2008). Where shell dissolution occurs as a result of acidification, molluscs may be more susceptible to shell borers such as sponges and *Polydora* polychaetes. Duckworth and Peterson (2013) found that decreased pH had minimal effect on the survivorship of a boring sponge *Cliona celata*, but increased the degree of shell boring into scallop (e.g., *Argopecten irradians*) shells. Other symptoms of enhanced pCO_2, such as hypercapnia (an increased amount of CO_2 in the haeolymoph) may alter susceptibility to disease by influencing immune function. Among blue mussels, *Mytilis edulis*, enhanced exposure to CO_2 suppressed phagocytosis, a key immune response (e.g., Bibby et al., 2008). Where climate change influences the occurrence of other physical stressors such as, for example, hypoxia, changes in the condition of molluscs may also result in impairment of immune response (see Boleza et al., 2001 for an example for where this has occurred in fish).

Changes in ocean currents as a result of climate change might also result in altered parasite–host interactions by changing dispersal patterns of hosts, parasites or both. To date, however, most research on effects of climate change on the spatial and temporal patterns of molluscan diseases has considered only effects of temperature. In order to prepare for and manage changing patterns of molluscan diseases, further research is needed that couples observations of long-term changes in diseases, at a variety of spatial scales, with manipulative experiments that investigate the mechanisms by which associations are altered.

7.3. Coastal development and disease

Estuaries, coasts, and lakes are among the most rapidly changing environments on earth. Globally, 23% of people live within 100 km of the coast and population densities in coastal regions are about three times higher than the global average (e.g., Small and Nicholls, 2003). As the human population continues to grow, anthropogenic impacts to aquatic environments, including pollution, over-extraction of fish, deterioration of water quality (including through eutrophication) and habitat modification will continue to increase (reviewed in Jackson et al., 2001; Lotze et al., 2006; Halpern et al., 2008; Burge et al., 2014) with concomitant increases in diseases, both old and new (reviewed in Naylor et al., 2001; Lafferty et al., 2004, 2015; Lafferty and Harvell, 2014).

Both alteration of the abiotic and biotic environment may result in shifts to host–parasite interactions. Pollutants, even at low concentrations, can have large effects on the physiology, immunology and ecology of molluscan populations (Rittschof and McClellan-Green, 2005). In many instances, parasites are less affected by pollutants than their hosts, resulting in large synergistic effects of pollutants and parasites on molluscan populations (reviewed by Morley, 2010b).

Over-harvesting of finfish can influence molluscan host–parasite relationships by reducing predation pressure on molluscs which may, in turn, facilitate parasites by increasing the abundance of hosts (Van Bocxlaer et al., 2012, 2014; see also 2015). By contrast where molluscs are themselves the target fishery, host populations of molluscs may be reduced to sufficiently low levels that their parasites are diminished or eliminated (see also Kuris and Lafferty, 1992; Ward and Lafferty, 2004; Wood et al., 2010; Lafferty et al., 2015). Where fishing gear is used across multiple waterways, including those that are disease-afflicted, parasites may be translocated. The establishment of no-take marine protected areas provide an opportunity to test hypotheses about the role of fishing in influencing parasite communities where there are no other confounding variables (Wood et al., 2010, 2015).

8. Conclusions and future directions

In our review, it is clear that the most well-studied molluscan host–parasite systems are those that involve commercially cultivated or harvested species. For example, the devastating economic effects *Haplosporidium nelsoni* (MSX) and *Perkinsus marinus* (Dermo) have had on eastern oyster, *Crassostrea virginica*, fisheries have driven research programs investigating the environmental factors that influence the dynamics of parasite and host, and those biological and environmental factors that influence expression of disease. These studies have enhanced our understanding of how disease shapes marine molluscan ecology at population-levels, but have seldom considered impacts of disease at higher levels of ecological organization.

More recently, with the increasing realization that there may be cascading effects of parasites on community structure, and even whole ecosystems and their services, studies of molluscan diseases have begun to extend beyond commercially important species. Many of the examples of how parasites modify community structure have come from study of gastropod–trematode and cockle–trematode interactions in intertidal environments. These studies have demonstrated how parasites can modify interactions such as competition, facilitation and predation, and in doing so may have indirect impacts that cascade to influence all aspects of community structure. Nevertheless, our knowledge remains largely confined to a small subset of molluscs and their parasites, which have been extensively studied by a handful of productive research groups.

Various methods (e.g., hatchery related animals, mark recapture, transplant experiments, sampling various populations) can be employed with field populations of molluscs to explore the effects of parasitism on host selection (easier for macroparasites), growth rates and behavior, especially if there is significant existing natural history and related empirical data (e.g., Sousa, 1983; Miura et al., 2006). For many molluscan host–parasite systems, particular those involving microparasites, our knowledge of host–parasite dynamics has come largely from descriptive studies, often conducted in response to mass-mortality events in cultivated populations (e.g., Elston and Ford, 2011). Although these types of studies have been very useful in generating hypotheses about those biological and environmental factors that may lead to disease outbreaks, without complementary experimental work, it is difficult to pinpoint disease as the cause of mortality. In many instances there is no biological or environmental sampling from prior to the disease outbreak and by the time sampling is conducted,

animals are dead. This severely limits any inferences that can be made. As Grizzle and Brunner (2009) emphasize, and we concur, one of the most difficult things to achieve when working with diseases is to capture a disease event in wild molluscs.

For most molluscan species the occurrence and level of infections in the field are based on sampling often a very limited set of populations with wide ranging environmental conditions thus only providing a partial picture of the degree of impacts and abiotic and biotic environmental controlling conditions of a given host–parasite relationship (e.g., Perrigault et al., 2010). Enhanced monitoring, both inside of and outside of mortality events, at affected and unaffected locations, is required to advance our understanding of when and where disease outbreaks occur, what their environmental drivers are, and how these translate to ecosystem level impacts (e.g., Lafferty and Harvell, 2014; Lafferty et al., 2015).

Studies monitoring disease in wild and cultured populations often sample dissimilar-sized individuals across space or time (e.g., O'Beirn et al., 1997). Consequently, any seasonal or spatial patterns they detect may be confounded with differences in age and exposures of animals to disease. Often low sample sizes are collected ($n \leqslant 10$) because of logistics and related costs for sample workup. These low sample sizes can often miss rare infection levels (Gregory and Blackburn, 1991; Jovani and Tella, 2006). Additionally, because of changing climatic norms, sampling that is limited to a single season in which disease is assumed to peak may miss atypical infection patterns of critical interest. Because of budget cuts many entities (e.g., South Carolina, U.S.A.) no longer sample and have a dedicated disease staff to react to or even sample regular norms (P. Kingsley-Smith, SCDNR, pers. comm.).

Enhanced monitoring assessing the occurrence of disease outbreaks in various species' populations, along with better approaches that zero in on the actual causative agents of these existing and novel diseases must be developed to detect both the frequency and range of disease epizootics, especially on species that are foundation or ecosystem engineers in coastal systems (e.g., Lafferty and Harvell, 2014; Lafferty et al., 2015). This will be especially difficult as local, state and federal budgets significantly shrink, along with many agencies mandates modified to ignore climate change, especially in the U.S.A. and now Australia and Canada.

Perhaps scrutiny of potential alien invaders with trade across the planet will provide some measure of surveillance? Molluscs, especially bivalves, are inadvertently introduced through shipping, fouling, ballast water, natural dispersal on floating debris and directed introductions via mariculture (e.g., Padilla and Williams, 2004; Ruesink et al., 2005; Molnar et al., 2008; Padilla et al., 2011). The rapid expansion of aquaculture may also provide possibilites for developing wide-spread monitoring disease programs that are required by permitting authorities.

A diverse array of monitoring and remote sensing technologies are now available that provide new opportunities for monitoring biological and environmental correlates of disease at large spatial and temporal scales. These include optical instrumentation on satellites, aircraft, moored and floating systems, gliders and autonomous vehicles, that may measure environmental variables such as air and water temperature, salinity, dissolved oxygen, wave and current patterns, as well as aspects of habitat structure, for example the extent of key habitat units and their spatial configuration. Increasingly these techniques are providing the basis of national and international observation systems (e.g. the Integrated Marine Observing System in Australia, and the National Estuarine Research Reserve (NERR) System and the National Marine Sanctuaries in the U.S.A.) that provide continuous and in some cases real-time data streams. 'Water quality' (e.g., circulation, temperature, salinity, nutrients, seston, food quality and quantity) is critical for molluscan aquaculture, as well as wild populations, and as discussed above it can be important as major stressors of hosts and enhance diseases.

One potential disease driver that is rarely considered is submarine groundwater discharges (or SGD), which serve as a source of freshwater, nutrients and related contaminants, especially karst or carbonate (limestone) areas (e.g., Florida and South Carolina, U.S.A., Brazil, Thailand; see Moore, 1996, 2010; Burnett et al., 2003; Charette et al., 2013). Overlapping hypoxia stressor events have been tied to SGDs (reviewed in Moore, 1996, 2010). The impacts of SGDs in near coastal systems are just beginning to be explored, especially as this is also where molluscan wild and cultured populations generally occur (e.g., Hwang et al., 2010). We need to have the ability to sample and evaluate the potential for extensive (or SGD) from inshore aquifers into shellfish growing waters (e.g., Hwang et al., 2010).

When accompanied with direct or indirect monitoring of disease, remote sensing technologies provide powerful opportunities to assess relationships between environmental change and disease outbreaks, at a diversity of spatial and temporal scales. Additionally, they may also prove powerful tools with which to assess relationships between disease outbreaks and ecosystem-scale changes that are predicted to occur as a result of ecosystem engineering by molluscs and other taxa (e.g., Griffen, 2009; Dumbauld et al., 2011; Repetto and Griffen, 2011; Lafferty et al., 2015).

Geographic information system (GIS)-based modeling can be used to address questions and hypotheses related to biogeography, conservation, evolutionary ecology, invasive species, and the focus here, disease ecology (e.g., Hay et al., 2000; Estrada-Peña, 2002). For example, the environmental requirements of hosts and of parasites, inferred from present distributions, can be used to produce maps that indicate habitat suitability under different scenarios of environmental change or management (e.g., Stensgaard et al., 2006). This may not only be a critical tool for guiding disease management, but also in generating hypotheses that can then be empirically tested about when and where the effects of disease will be greatest. Recently, interactive maps on the web at various institutions have been available for researchers to begin to take advantage of web-based novel technologies for use in evaluating human parasite epidemiology (e.g., see ParaSite, Parasites and Pestilence, based at Stanford University http://web.stanford.edu/class/humbio153/InteractiveMaps/Analysis.html, and directed by P.J. Hubbard, P. Sud, and H. Lee). Another site is GeoParasite.org, a geospatial approach to the ecology, distribution and vectorial role of parasitic Arthropoda (see http://www.geo-parasite.org/about.html).

In parallel to the explosion in remote sensing technologies and GIS tools, has been the molecular revolution which has enable high-throughput rapid-detection of pathogens within molluscs (e.g., Reece et al., 1997, 2008; Blakeslee et al., 2008; Dungan and Bushek, 2015; Thompson et al., 2015). In particular, this new technology provides exciting possibilities for the study of microparasites, which historically have been difficult and costly to sample. In addition to opening the door for extensive monitoring programs of parasites, molecular technologies might make manipulative experiments with micro-parasites more feasible (see Gómez-Chiarri et al., 2015). For example, molecular screening techniques can enable rapid identification of populations of mollusc that differ in their level of parasitic infection, that could then be the subject of various experimental manipulations that assess effects of abiotic and biotic factors on disease development and impact (see Dungan and Bushek, 2015).

Overall, the limited number of experiments examining cause–effect relationships between disease and ecological change at larger spatial scales and levels of ecological organization remains a major research gap. Conducting whole-ecosystem experiments is not only logistically challenging, but can be ethically problematic.

Nevertheless, as humans continue to modify the planet at large scale, and in planned, as well as unplanned ways, there is real opportunity to use human interventions as manipulative experiments. For example, activities such as ecological restoration and the establishment of marine protected areas (MPAs) might enable test of how trophic and/or habitat structure influence ecological impacts of disease. A comparison of molluscan (especially bivalve) restoration projects that use disease-susceptible and disease-resistant stock may enable examination of how disease influences ecosystem functions such as filtration and shoreline stabilization. The cultivation of molluscan species in new locations may enable test of hypotheses of how the environment influences disease expression. Changing shipping routes, genetics and patterns of species translocation can be used to address questions about the role of vectors in influencing spatial patterns of disease (e.g., Blakeslee and Byers, 2008). The manipulation of molluscan densities and spatial arrangements by cultivation and dredge harvest may enable test of how the spatial ecology of host populations influences disease. Ideally sampling of hosts, parasites, and environmental factors should occur before and after the intervention at multiple control and impact sites (i.e. beyond BACI designs, Underwood, 1992) in order to establish causation between the intervention and variables of interest.

As rapidly expanding aquaculture operations, global climate change and coastal development continue to modify the distribution and abundance of hosts and parasites and the ways in which they interact, it will be important to understand how broadly concepts gained from model host–parasite systems apply to other associations and to novel environmental contexts.

Conflicts of Interest

The authors confirm that there are no known conflicts of interest associated with this publication.

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