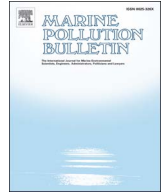




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## Review: Host-pathogen dynamics of seagrass diseases under future global change

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### ABSTRACT

Human-induced global change is expected to amplify the disease risk for marine biota. However, the role of disease in the rapid global decline of seagrass is largely unknown. Global change may enhance seagrass susceptibility to disease through enhanced physiological stress, while simultaneously promoting pathogen development. This review outlines the characteristics of disease-forming organisms and potential impacts of global change on three groups of known seagrass pathogens: labyrinthulids, oomycetes and Phytomyxea. We propose that hypersalinity, climate warming and eutrophication pose the greatest risk for increasing frequency of disease outbreaks in seagrasses by increasing seagrass stress and lowering seagrass resilience. In some instances, global change may also promote pathogen development. However, there is currently a paucity of information on these seagrass pathosystems. We emphasise the need to expand current research to better understand the seagrass-pathogen relationships, serving to inform predicative modelling and management of seagrass disease under future global change scenarios.

### 1. Introduction

Human-induced global change facilitates disease outbreaks on a global scale (Harvell et al., 2002). Mass-mortalities due to disease in keystone species marine species have been reported in corals (Hoegh-Guldberg et al., 2007), marine mammals (Cosby et al., 1988), oysters (Van Banning, 1991), sea-stars (Hewson et al., 2014) and seagrasses (Sullivan et al., 2013). Through the negative impacts on these keystone species, the effects of disease may rapidly cascade through ecosystems, with potentially catastrophic consequences through local species extinction, community regime shifts and disruptions in economically-linked ecosystem services, such as fisheries production (Groner et al., 2015; Sullivan et al., 2013). Despite emerging threats to several ecologically important species, epidemiological studies in marine systems are sparse compared to terrestrial systems (Harvell et al., 1999).

Seagrasses are marine flowering plants that form large swaths of grass-like intertidal and subtidal meadows in coastal areas. Seagrass ecosystems provide vital coastal habitats that are increasingly

recognised for their ecological function and the provision of human services, including reduction in human and wildlife pathogens (Lamb et al., 2017) coastal protection (Christian et al., 2013), fisheries habitat (Jackson et al., 2001) and carbon sequestration (Fourqurean et al., 2012). Seagrasses also provide positive feedback mechanisms for themselves that drive continued provision of suitable habitat for their own survival (De Boer, 2007; Maxwell et al., 2016). Despite increasing attention to seagrasses as a keystone species, researchers estimate nearly 30% of the global seagrass area world-wide was lost between 1879 and 2006 (Waycott et al., 2009).

Under the threat of ongoing global change, ecologists look to understand how marine ecosystems will function, and how community and population dynamics may be altered in the near and distant future. The majority of publications available on the effects of global change in coastal ecosystems have focused on the role of abiotic environmental conditions, namely temperature, on the health of marine systems, and most often at the level of an individual species or population (Harley et al., 2006). Still, research on the role of biologically-mediated

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environmental conditions (Alsterberg et al., 2013; Eklöf et al., 2012; Sunday et al., 2017) suggests that organism-to-organism interactions are important in understanding both direct and indirect effects of global change on the functional capacity of coastal and marine ecosystems.

### 1.1. Known global change impacts to seagrass

Abiotic drivers potentially affecting seagrass resilience under future scenarios include increasing ocean temperatures, sea level rise, increased frequency of storms, ocean acidification (increased CO<sub>2</sub> and decreased pH) and changes to salinity (Duarte, 2002; Harley et al., 2006; Koch et al., 2013; Short et al., 2016). Increasingly, studies are emerging to quantify the effects of global change on seagrasses. These include theoretical investigations of seagrass physiology, communities and populations (Duarte, 2002; Edwards, 1995; Harley et al., 2006; Occhipinti-Ambrogi and Savini, 2003; Orth et al., 2006; Short and Neckles, 1999) and modelling and experimentation (Jordà et al., 2012; Koch et al., 2013; Palacios and Zimmerman, 2007; Rasheed and Unsworth, 2011).

Firstly, elevated seawater temperatures will affect plant physiology, reproduction, growth and geographic distribution (Short and Neckles, 1999). For some seagrasses, warmer waters may be beneficial, resulting in increased growth rates (Short and Coles, 2001) and reproduction (Diaz-Almela et al., 2007). However, plants growing at the edges of their geographical range are found to experience increased mortality due to drought stress (de Fouw et al., 2016) and heat waves (Collier and Waycott, 2014; Fraser et al., 2014).

Secondly, increasing atmospheric CO<sub>2</sub> levels are increasing the amount of dissolved CO<sub>2</sub> in coastal waters, resulting in a lower pH (Orr et al., 2005). Although the effects of ocean acidification are unfavourable to calcifying marine organisms, they are known to enhance resilience of seagrasses (Alsterberg et al., 2013). Seagrasses themselves are expected to benefit from high CO<sub>2</sub> levels that result in increased plant production (Garrard and Beaumont, 2014; Takahashi et al., 2016) and carbon storage (Russell et al., 2013).

Global change may also affect salinity of coastal waters in two ways: either warming will raise coastal salinities by higher evaporation levels and reduced freshwater input, or salinity in coastal areas may decrease for prolonged periods due to increased precipitation. Effects of altered salinities vary among seagrass species (Salo et al., 2014), but extremes of salinity < 10 ppt or > 45 ppt negatively affect seed germination (Pan et al., 2011) and plant performance (Kahn and Durako, 2006; Koch et al., 2007a).

Additionally, there may be important shifts to environmental conditions that may further threaten seagrasses under global change scenarios. One such threat to seagrasses is human-induced eutrophication (Burkholder et al., 2007), which causes an increase in algal cover. Increased nutrient loads in coastal waters may occur as a result of agricultural run-offs and wastewater discharge. Nutrient loading favours fast growing plankton and macroalgal species at the expense of seagrasses (Hauxwell et al., 2003; Kemp et al., 2005). Thus, substantial inputs of additional nutrients to water bodies is known to result in increased turbidity and lower light availability for seagrasses. These conditions undermine general seagrass health and growth (Burkholder et al., 2007) and may impose direct ammonium (van Katwijk et al., 1997) and sulfide stress and toxicity (Lamers et al., 2013). The ability of known seagrass diseases to complete their lifecycles in eutrophic conditions is unknown. Global change thus seems to play a pivotal role in the acute decline of seagrasses worldwide (Orth et al., 2006; Waycott et al., 2009), though those effects are not always simply positive or negative.

### 1.2. Potential increased risk of seagrass disease in the future

The impact and ecological roles of co-occurring biological interactions with seagrasses, such as pathogen presence, are largely unknown.

In fact, while the importance of the microbiome in seagrass is gaining momentum (Fahimipour et al., 2017), the role of pathogenic disease is often left out of seagrass research entirely, and is largely missing or overlooked in reviews and analyses of global change impacts in seagrass ecosystems (Bjork et al., 2008; Brown et al., 2013; Short et al., 2016). Still, there is recognition of the vulnerability of seagrass to global change scenarios (Short et al., 2016), which is alarming, given multiple evidence of accelerating risk of emerging infectious diseases in terrestrial and marine systems, largely through range expansion and human introduction (Anderson et al., 2004; Hoegh-Guldberg and Bruno, 2010).

We theorise that since human-induced changes to global systems pose major threats to seagrasses in the future (Orth et al., 2006; Short et al., 2016), they may decrease seagrass resilience and fitness (De Boer, 2007; Ehlers et al., 2008; Fraser et al., 2014; Ruesink, 2016) and thus, increase the risk of susceptibility to disease (Harvell et al., 2002). Multiple and repeated disturbances, such as those that may occur as global change progresses, are known to further reduce seagrass resilience (Eklöf et al., 2010; Fraser et al., 2014) and may create hotspots of seagrass disease. In addition to enhanced susceptibility of the host, global change may also promote infection and transmission of diseases by promoting pathogen development, survival and reproduction (Burge et al., 2014; Harvell et al., 1999). Combined effects of global change on host, pathogen and host-pathogen relationships in seagrass diseases are understudied. Still, available research points to significant effects for seagrasses from temperature, salinity, eutrophication and biological interactions, such as disease. This research reveals the urgency for continued and inclusive investigations into the potential roles of disease in seagrass ecosystems, especially those under direct stress from global change phenomena.

## 2. History of seagrass disease research

Disease events in seagrass ecosystems have been observed for nearly a century (Fischer-Piette et al., 1932). Initial work in this field was driven by early observations of rapid decline and acute losses to large areas of *Zostera marina* L. on both sides of the Atlantic Ocean in the 1930's (Den Hartog, 1987; Giesen et al., 1990; Rasmussen, 1977). It was estimated that up to 90% of all *Z. marina* of the North Atlantic was lost in a wasting disease epidemic (Muehlstein, 1989). Since that time several wasting disease events have been observed and have resulted in ecologically significant losses of *Z. marina* meadows in Maine, New Hampshire and Massachusetts in the United States (Short et al., 1987) and *Thalassia testudinum* ex Koenig in Florida Bay, USA (Hall et al., 2016; Robblee et al., 1991). There have been other localised disease events around the globe that may be attributed to wasting disease, including observations of the pathogen causing disease symptoms in Japan (Ralph and Short, 2002), Canada, Europe and the United States (Groner et al., 2016a; Short et al., 1988) and New Zealand (Armiger, 1964).

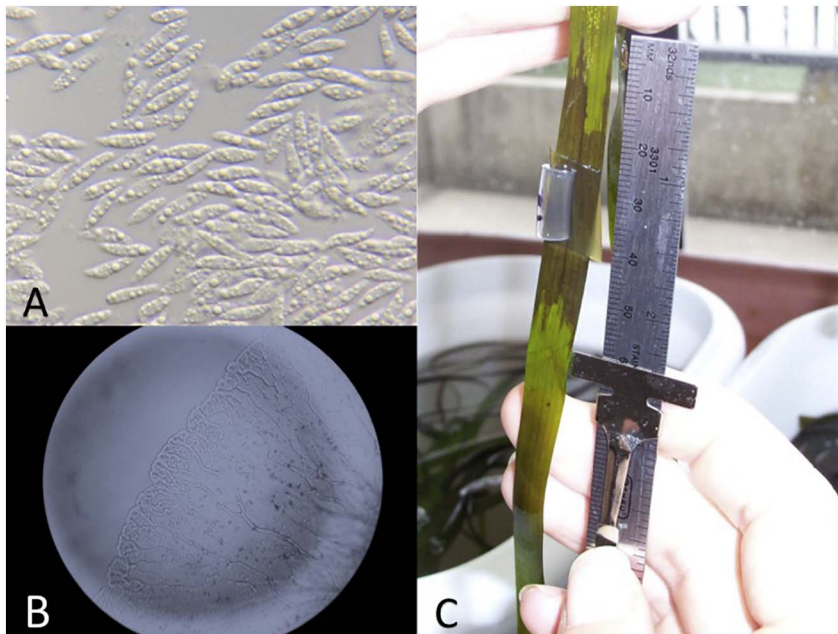
Presumably due to the large scale of losses in the 1930's, seagrass pathology has progressed with a focus on *Labyrinthula*, the etiological agent of seagrass wasting disease. Much of this work has been done in *Z. marina* and other North American and European species. However, more recently, research has emerged on the biology and ecology of *Labyrinthula* at the global level where researchers have investigated phylogeny and global trends in pathogenicity of *Labyrinthula* (Martin et al., 2016), including the most recent work describing isolation, phylogeny (Sullivan et al., 2016) and pathogenicity (Trevathan-Tackett et al., unpublished) of *Labyrinthula* spp. in the southern hemisphere.

Although reports of others seagrass pathogens are rare, *Plasmodiophora bicaudata* and *Plasmodiophora diplantherae* were discovered to cause shoot galls in *Zostera* and *Halodule*, respectively (Den Hartog, 1965, 1989; Walker and Campbell, 2009). Those parasites were described as new species, but there was no comment on their potential to cause diseases or dieback in the populations other than Walker and Campbell (2009) and Den Hartog (1989), who noted increased

**Table 1**  
Overview of known seagrass pathogens and hosts.<sup>a</sup>

Agent of disease	Seagrass host species	Type	Symptoms	References
<i>Halophytophthora</i> sp. <i>Zostera</i>	<i>Zostera marina</i>	Oomycetes, Heterokonta	Reduced seed germination, impaired seedling development	(Govers et al., 2016, Govers et al., 2017)
<i>Labyrinthula</i> sp. including <i>L. zosterae</i>	<i>Zostera marina</i> <i>Zostera muelleri</i> <i>Zostera caulescens</i> <i>Zostera japonica</i> <i>Zostera noltii</i> <i>Zostera pacifica</i> <i>Cymodocea nodosa</i> <i>Posidonia oceanica</i> <i>Ruppia cirrhosa</i> <i>Ruppia maritima</i> <i>Syringodium isoetifolium</i> <i>Thalassia testudinum</i> <i>Zostera marina</i>	Labyrinthulomycetes, Heterokonta	Leaf lesions, mortality if lesion advancement out-grows new leaf growth	(Garcias-Bonet et al., 2011), (Martin et al., 2016), Muehlstein et al. (1991)
<i>Phytophthora gemini</i>	<i>Zostera marina</i>	Oomycetes, Heterokonta	Reduced seed germination (seed pathogen)	(Govers et al., 2016, Govers et al., 2017)
<i>Plasmodiophora diplantherae</i>	<i>Halodule wrightii</i> <i>Halodule uninervis</i>	Phytomyxea, Rhizaria	Shoot galls	(Den Hartog, 1965, Walker and Campbell, 2009)
<i>Plasmodiophora bicaudata</i>	<i>Zostera noltii</i> <i>Zostera capensis</i> <i>Zostera muelleri</i> <i>Zostera japonica</i>	Phytomyxea, Rhizaria	Shoot galls	(Den Hartog, 1989)
<i>Plasmodiophora halophila</i>	<i>Halophila ovalis</i>	Phytomyxea, Rhizaria	Shoot galls	(Karling, 1968)
<i>Tetramyxa parasitica</i>	<i>Ruppia brachypus</i> <i>Ruppia rostellata</i> <i>Ruppia spiralis</i> <i>Ruppia maritimus</i>	Phytomyxea, Rhizaria	Shoot galls	(Karling, 1968)

<sup>a</sup> *Labyrinthula* sp. have been isolated from the following seagrass species but pathogenicity was not tested, or could not be confirmed: *Halodule uninervis*, *Halophila ovalis*, *Thalassodendron ciliatum* (Vergeer and Den Hartog, 1994), *Phyllospadix scouleri*, *Phyllospadix torreyi* (Martin et al., 2016).



**Fig. 1.** A) Spindle-shaped cells of *Labyrinthula* spp., B) Unique ectoplasmic network forms distinctly branching *Labyrinthula* colony, and C) Infected *Zostera marina* leaf with an expanding wasting disease lesion after 4 days.

uprooting of the plants. Very recently, two additional pathogens of seagrasses were discovered: *Phytophthora gemini* and *Halophytophthora zostera* (Govers et al., 2016; Govers et al., 2017; Man in 't Veld et al., 2011). These oomycetes were shown to affect the viability of seeds and restricts seedling development. In light of these novel discoveries, the relevance of additional pathogens in seagrass disease events and resilience of seagrass ecosystems is gaining momentum. A summary of seagrass diseases and their hosts is provided in Table 1.

### 3. The role of environmental parameters in seagrass disease

There is a long history of trying to decouple the influence of environmental factors on prevalence and occurrence of periodic seagrass disease events (Cottam and Munro, 1954; Stevens, 1936; Tutin, 1938). For instance, following the 1930's epidemic, evidence was presented showing correlations of *Zostera marina* declines at the time with extreme drought and flood cycles (Martin, 1954), moon phases, and warm water transgressions (Stevens, 1936). A correlation was later discovered between solar insolation and wasting disease events during that time (Giesen et al., 1990), suggesting reduction in light by increased storm

events or cloud cover may contribute to disease severity. More recent assessments of independent environmental variables, temperature and salinity, have found variable effects of these factors on seagrass disease progression (Burdick et al., 1993; Olsen et al., 2015; Trevathan et al., 2011). Given no studies have positively demonstrated specific environmental factors contribute to presence or severity of disease, it is clear more theory and experimentation in this field is needed.

Our review is the first paper to summarise the roles of global change and diseases in seagrass ecosystems, including 1) detailed descriptions of three known pathogenic groups, 2) investigations into known and potential effects of disease on seagrass in a changing climate, 3) discussions of the need for advancing disease related research and monitoring and 4) providing recommendations for future research considering disease in seagrass resilience and recovery models into the future.

## 4. Known seagrass pathogens

### 4.1. *Labyrinthula*

#### 4.1.1. Description

*Labyrinthula* spp. are unicellular protists (Fig. 1a) in the family Labyrinthulomycetes (at the base of the Stramenopila), which grow in distinctly branching colonies formed by secretion of extracellular mucus, which forms intricate networks the cells utilise for transport (Fig. 1b). Along with thraustochytrids and aplanochytrids, *Labyrinthula* species typically decompose plant material in marine and coastal ecosystems (Tsui et al., 2009). When penetration of *Labyrinthula* into green, live seagrass leaf tissue, does occur it is resulting in chloroplast and cell necrosis. As a result of the necrosis, affected areas of the leaf become blackened while surrounded by green leaf tissue, which is the primary way to identify possible wasting disease infection (Fig. 1c). In the 1930s, the presence of *Labyrinthula* and blackened seagrass leaves closely coupled with a massive die-off of *Z. marina* was affecting > 90% of the seagrass populations in the northern Atlantic Ocean. It wasn't until decades later that the causative agent, *Labyrinthula zosterae* was identified by fulfilling Koch's Postulates (Muehlstein et al., 1988; Muehlstein et al., 1991). The mechanisms behind the spread was shown to be from blade-to-blade contact (Muehlstein, 1992) as well as floating leaf detritus and waterborne introductions (Martin et al., 2016), with lesions bisecting the width of the leaf being detrimental to plant oxygen and nutrient transport. Since then, *Labyrinthula* has been considered primarily an *opportunistic* pathogen under the hypothesis that unhealthy seagrasses would more likely be susceptible to wasting disease and thus larger-scale die-offs.

#### 4.1.2. Host-pathogen relationship

The *Labyrinthula*-seagrass-wasting disease relationship is not entirely well defined. For one, it is unclear why some isolates of *Labyrinthula* are able to infect living seagrass tissues, yet other times, the same isolate may only act as a saprobe of seagrass litter. Martin et al. (2016) made advances in identifying ITS rDNA clades showing pathogenic or non-pathogenic characteristics. The second facet of this relationship is host susceptibility, which can be tested via environmental stressors (decreased health) and availability of a host defence mechanism (secondary metabolites, oxidative burst, hypersensitive response) (Loucks et al., 2013; Trevathan-Tackett, 2011). As previously stated, the majority research thus far has focused on wasting disease in temperate *Z. marina* and (sub) tropical *T. testudinum* (Table 1). However, recently there has been a push to understand the host-pathogen-environment interactions for other *Zostera* species which can infect *Cymodocea nodosa* Asch. and *Posidonia oceanica* Delile (Martin et al., 2016; Olsen et al., 2015; Olsen and Duarte, 2015).

#### 4.1.3. Global change dynamics

Previously, the hypothesised effects of global change and

anthropogenic stressors on seagrass wasting disease were primarily based on seagrass stress studies, absent of infection treatments (e.g. Koch et al., 2007b; Short et al., 2016). More recently, there has been an effort in understanding how the environment influences wasting disease dynamics. These studies have shown that elevated temperatures, particularly above 28 °C, had no effect on wasting disease proliferation for Mediterranean *Cymodocea nodosa* (Olsen and Duarte, 2015) and *Posidonia oceanica* (Olsen et al., 2015). However, testing the effect of warming on disease severity in temperate species, such as *Zostera*, is needed, as these species at the edges of climate tolerance may not be adapted to warmer waters. *Labyrinthula* growth and incidence of wasting disease are generally inhibited by low salinities (e.g. oligo- and mesohaline < 15) and thrive at polyhaline salinities (up to ~30–35). Elevated salinities above 35–40, however, do not increase susceptibility to infection, due to acclimation by the seagrass, as well as detrimental effects of hypersalinity on *Labyrinthula* itself (Bishop, 2013; Burdick et al., 1993; Martin et al., 2009; McKone and Tanner, 2009; Sykes and Porter, 1973; Trevathan et al., 2011). Given that seagrass ecosystems will potentially be subject to several sub-optimal environmental conditions simultaneously, it will be important to understand the combinations of health 'thresholds' within and beyond current climatic conditions (e.g. temperature, salinity). It has been hypothesised that multiple stressors have provided conditions for enhanced seagrass die-off in the past (Koch and Erskine, 2001; Koch et al., 2007b; Koch et al., 2007c), but as of yet the relationship between seagrass and *Labyrinthula* and a changing environment is complex and necessitates further investigation (Bishop, 2013; Groner et al., 2014; Martin et al., 2016).

#### 4.1.4. Current topics

In comparison with other well-studied terrestrial and marine pathogens, there are still many questions to answer about *Labyrinthula* speciation, ecology and seagrass wasting disease (Martin et al., 2016). Firstly, while there are a handful of studies that have tested pathogenicity of *Labyrinthula* of seagrass species other than *Z. marina*, it is critical to understand the wasting disease dynamics of the ~60 other seagrass species world-wide, particularly those that are threatened and/or endemic. It is possible that certain *Labyrinthula* isolates demonstrate host-specific or effective climatic-, species- or genera-specific infection and subsequent responses of seagrass hosts to infection. This could in turn limit wide-spread incidences of wasting disease (Martin et al., 2016; Vergeer and Den Hartog, 1994). There are also large gaps in our knowledge about *Labyrinthula* ecology, particularly, its infection mechanisms from the perspective of enzymatic degradation of live tissue and its own defence against seagrass metabolites. While *Labyrinthula* growth is effectively reduced in vitro by phenolic acids common in seagrass tissues (Jensen et al., 1998; Steele et al., 2005; Trevathan-Tackett et al., 2015), the role of such compounds as an active, induced response to infection is still to be evidenced in vivo. There could also be a biological control of infection through interactions with other epibiotic microbes as seen with marine macroalgal and animal biofilms (Wahl et al., 2012). Lastly, one of the largest research needs surrounding seagrass wasting disease is the detection and monitoring of infectious *Labyrinthula*. Most recent monitoring efforts have been performed with visual lesion identification through a wasting index (Burdick et al., 1993). However, this method can be quite time-consuming and costly as well as unreliable for identifying *Labyrinthula*-based infections, if not followed up with culturing and Koch's Postulates tests. With the growing affordability and accessibility of broader genetic typing techniques such as eDNA profiling of communities on all taxonomic levels (i.e. species, genera, phyla) together with improved detection methods (PCR, qPCR, single cell genomics) large scale screening of the pathogen load and the pathogen community will be viable in the near future (Bockelmann et al., 2013).



## 4.2. *Phytophthora*

### 4.2.1. Description

*Phytophthora gemini* was first discovered on rotting leaves of *Zostera marina* together with another *Phytophthora* species, *Phytophthora inundata* (Man in 't Veld et al., 2011). This was the first report on *Phytophthora* species associated with a seagrass species, but up to that point, nothing was yet known about potential pathogenicity of these *Phytophthora* species (Govers et al., 2016). This knowledge gap is surprising as *Phytophthora* spp. are considered the most vigorous group of terrestrial plant pathogens but virtually nothing is known about freshwater and marine *Phytophthora* species.

### 4.2.2. Host-pathogen relationship

*Phytophthora* species are fungi-like oomycetes that can reproduce both sexually and asexually by means of motile zoospores. Infection by *Phytophthora* species can cause a range of disease symptoms in terrestrial plants including stem rot, root rot and leaf blight (Lamour, 2013). The most infamous *Phytophthora* species is *Phytophthora infestans*, the causative agent of potato blight (Bourke, 1964; Yoshida et al., 2013). Next to causing economic damage through the loss of agricultural crops, several *Phytophthora* species are also known to harm natural ecosystems. *Phytophthora ramorum* is currently threatening forest ecosystems in California by causing mortality in oaks (Rizzo and Garbelotto, 2003; Rizzo et al., 2002), and *Phytophthora cinnamomi* infects and kills many endemic tree species in eucalyptus forests in Southwest Australia (Weste and Marks, 1987). *Phytophthora alni* is a widespread lethal disease of alder that represents a threat to riparian ecosystems in Europe (Brasier et al., 2004). In contrast to this extensive list of examples from terrestrial systems, virtually nothing is known about marine *Phytophthora* species, with the exception of two reports of *Phytophthora* species associated with mangroves (Pegg et al., 1980; Zeng et al., 2009).

Recent studies on seagrasses have shown that *Phytophthora gemini* infection of *Zostera marina* seeds is widespread across the North Atlantic with infection rates as high as 74%. Infection strongly reduces seed

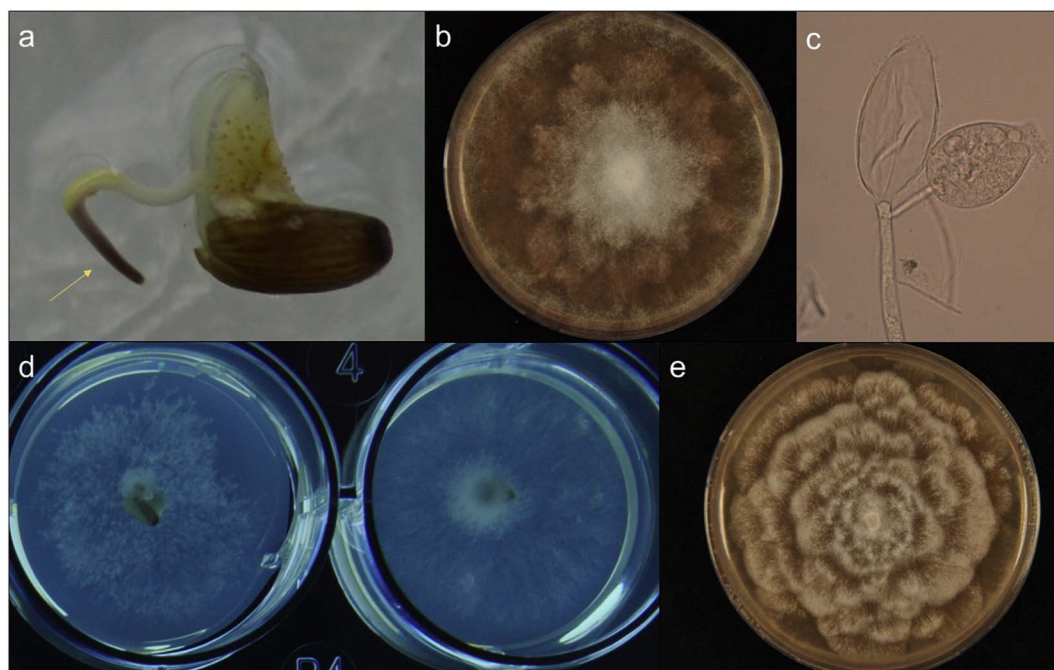
germination (by 6 times) and impairs seedling development (Fig. 2a). (Govers et al., 2016; Govers et al., 2017) of *Z. marina*. Although *Phytophthora inundata* has also been found on *Zostera marina* leaf and seed material (Man in 't Veld et al., 2011), pathogenicity trials with this *Phytophthora* species have yet to be performed. Another, newly described *Phytophthora* species, *Phytophthora chesapeakensis* was recently found on *Zostera marina* seeds from Chesapeake Bay, U.S. (Man in 't Veld et al., in review). Based on the pathogenic character of the *Phytophthora* genus, it is very likely that both *P. inundata* and *P. chesapeakensis* may cause disease in *Zostera marina*, but this needs further investigation. So far, *Phytophthora* species have only been found associated to *Zostera marina* (Govers et al., 2016; Man in 't Veld et al., 2011) and very recently with *Zostera noltii* Hornem. growing intermingled with infected *Zostera marina* (Man in 't Veld et al., in review). This may indicate that other seagrass species, especially from the *Zostera* genus may also be susceptible to infection by *Phytophthora* species.

Although *Phytophthora* spp. have been isolated from both *Zostera marina* leaves and seeds, no symptoms of infection have been found on leaves so far (Govers et al., 2016). Instead, *Phytophthora* spp. seem to be seed pathogens, killing dormant seeds and newly emerging pre-seedlings (Govers et al., 2017).

Infection numbers may be extremely high in dense seagrass meadows (Govers et al., 2016), but transmission pathways have not yet been studied. However, presence of oomycetes such as *Phytophthora* spp. in aquatic systems are often baited for by using pollen grains (Goldstein, 1960). Therefore, it may be expected that infection of *Zostera marina* by *Phytophthora* spp. zoospores takes places in the young sporophyte and gametophyte stages (Gleason et al., 2013).

### 4.2.3. Global change dynamics

Global change is known to negatively affect seagrass performance (see above), thereby likely enhancing susceptibility of seagrasses to disease. In addition, global change may very likely also affect *Phytophthora* survival, motility and infection potential. Climate warming may affect plant diseases by altering stages and rates of pathogen development, leading to range shifts and altered infection



**Fig. 2.** a) Impaired seedling development of *Zostera marina* due to *Phytophthora gemini* infection, b) Colony morphology of *Phytophthora gemini* grown on cherry decoction agar (CHA), c) Microscopic detail of *Phytophthora gemini* sporangia characterized by the occasional formation of two neighbouring sporangia on the zoospore, d) *Phytophthora gemini* (left) and *Halophytophthora* sp. *Zostera* and *Phytophthora gemini* mycelium (right) growing from infected *Zostera marina* seeds on a ParpH medium, d) colony morphology of *Halophytophthora* sp. *Zostera* grown on CHA. Photos are reproduced from Govers et al. (2016) and Govers et al. (2017).

patterns (Coakley et al., 1999). In many high-temperature optimum *Phytophthora* species, winter is generally considered the period of major mortality. Increasing winter sea temperatures due to global warming is therefore expected to lessen the effects of this bottleneck period of the *Phytophthora* growth cycle, thereby expanding the range of these pathogens. In addition, warming is predicted to increase pathogen activity at existing disease locations (Bergot et al., 2004; Brasier, 1996; Sturrock et al., 2011; Thompson et al., 2014). Although no research has yet been done on *Phytophthora* species that infect seagrasses, growth curves of *Phytophthora gemini* and *Phytophthora inundata* show optimum mycelium growth rates around 27 °C and steeply increasing growth rates between 6 and 27 °C (Man in 't Veld et al., 2011). In contrast, *Phytophthora gemini* infection of *Zostera marina* seeds seems to be higher in seeds wintering in low temperature sediments (5 °C) than high temperature sediments (12 °C) (Govers et al., 2016). This ambiguity indicates that temperature effects on seagrass-*Phytophthora* interactions may be hard to predict and will depend on both plant and pathogen life stage and seasons.

Although nothing is known about marine *Phytophthora* species in relation to seawater pH, many terrestrial *Phytophthora* species can disperse via waterways and are affected by water quality (Granke et al., 2009). These common, water dispersing *Phytophthora* species such as *P. alni*, *P. kernoviae*, *P. ramorum*, generally have a broad pH tolerance, which is species specific (Kong et al., 2012). pH levels outside the species-specific tolerance zones negatively affected motility, encystment and germination of *Phytophthora* zoospores (Kong et al., 2012). However, ocean acidification is only expected to change seawater pH by 0.1–0.2 pH points (Pachauri et al. IPCC). Such a change will very likely fall within the pH tolerance of marine *Phytophthora* species. We, therefore, expect no effects of ocean acidification on *Phytophthora* infection in seagrasses.

In contrast, high salinities generally seem to facilitate *Phytophthora* as a result of increased sporangium production under high saline conditions (Bouchibi et al., 1990). Salinity extremes due to global change may negatively affect seagrass performance (Kahn and Durako, 2006), thereby enhancing susceptibility to disease, including *Phytophthora* as is seen in many terrestrial plant species (Blaker and MacDonald, 1986; Sanogo, 2004; Snapp et al., 1991). However, during periods of extremely low salinity (e.g. high precipitation or flooding events) may have opposite effects on *Phytophthora* spp. Infection at salinities as low as 0.5 ppt have been observed to completely eliminate infection of *Zostera marina* seeds, possibly because *Phytophthora* are weak competitors in these conditions (Govers et al., 2017).

#### 4.2.4. Current topics

As *Phytophthora* species have only recently been discovered as seagrass pathogens (Govers et al., 2016; Govers et al., 2017), current topics on *Phytophthora*-seagrass interactions cover every aspect of this host-pathogen interaction, from a plant physiological to ecosystem level, including the use of novel techniques for pathogen detection (Menning et al. unpublished data). Research on seagrass-*Phytophthora* interactions is still in a pioneering phase and additional research is urgently needed as *Labyrinthula* infection of eelgrass has shown that massive, disease-driven die-offs may occur in seagrass beds, affecting both seagrasses and associated fauna (Den Hartog, 1987; Rasmussen, 1977). Many stringent questions have arisen. 1) Ecology: What other seagrass species may be vulnerable to *Phytophthora* infection? What conditions determine infection and pathogen transmission? How widespread are *Phytophthora* spp. associated with seagrasses? 2) Phytopathology: How do seagrass plants get infected by *Phytophthora* species? How does *Phytophthora* kill seagrass seeds? What kind of defence mechanisms do seagrasses have against infection? Finally, 3) Global change: How do temperature, ocean acidification and extreme salinities affect seagrass-*Phytophthora* interactions.

### 4.3. *Halophytophthora*

#### 4.3.1. Description

Just like *Phytophthora* species, *Halophytophthora* species are oomycetes, with similar morphology (Fig. 2c–d) and life cycles. The genus of *Halophytophthora* is not monophyletic and so far, only 14 species have been named (Robideau et al., 2011). Some *Halophytophthora* species even group with *Pythium*, another genus of plant parasites (Cooke et al., 2000). *Halophytophthora* are commonly found inhabiting brackish and saltwater and were only very recently positioned in a genus separate from *Phytophthora* based on rDNA-ITS sequences (Cooke et al., 2000).

#### 4.3.2. Host-pathogen relationship

*Halophytophthora* species were previously unknown as pathogens and were only described as saprophytes, decomposing dead mangrove and saltmarsh plant leaves (Ho and Jong, 1990; Nakagiri, 2000; Newell, 1996). Some saprophytes may however become pathogenic under favourable conditions (Freitag et al., 2009; Marois and Mitchell, 1981), and *Halophytophthora* species have very recently indeed been found to be pathogenic to seagrasses (Govers et al., 2016). In total, three *Halophytophthora* species (sp. 1, 3, 4) have been isolated from *Zostera marina*, which have not yet been named officially (Man in 't Veld et al., in review). Potential pathogenic effects of *Halophytophthora* species on other higher marine plant species (mangroves, salt marsh plants) have not yet been discovered.

As indicated above, *Halophytophthora* species have traits very similar to *Phytophthora* species and infection rates in the field can be as high as 67% (Govers et al., 2016). In addition, *Halophytophthora* spp. may be equally pathogenic, causing seed mortality of infected seeds and impaired seedling development (Govers et al., 2016; Govers et al., 2017). *Halophytophthora* infection of *Zostera* is as widespread as *Phytophthora* infection (Govers et al., 2016; Govers et al., 2017; Man in 't Veld et al., in review) and *Halophytophthora* spp. have also been isolated from both leaves and seeds of *Zostera marina* (Govers et al., 2016; Man in 't Veld et al., in review). So far, *Halophytophthora* species have only been found to infect *Zostera marina*, but since this group of oomycetes is very widespread across ecosystems and climatic zones (Nakagiri, 2000; Newell, 1996; Nigrelli and Thines, 2013), it is very likely that other seagrasses may also be at risk of infection.

#### 4.3.3. Global change dynamics

Most reports of *Halophytophthora* species come from tropical to subtropical coastal ecosystems (Fell and Master, 1975; Nakagiri and Newell, 1994; Pegg and Alcorn, 1982), until their recent discovery in temperate coastal waters (Govers et al., 2016; Man in 't Veld et al., in review; Nigrelli and Thines, 2013). These recent discoveries are probably not a consequence of biogeographic expansion due to global change, but rather a result of increased sampling efforts for eukaryotic microbes. This is because growth rates of temperate *Halophytophthora* spp. are similar (between 5 and 25 °C), whereas tropical *Halophytophthora* spp. showed optimum growth rates around 30 °C (Nigrelli and Thines, 2013). Thus, climate warming may not necessarily benefit temperate *Halophytophthora* species. However, the range of common tropical and sub-tropical *Halophytophthora* species may expand because of increased temperatures.

Potential effects of ocean acidification on *Halophytophthora* have not yet been studied, but there is one study reporting on the effects of pH on *Halophytophthora* growth (Leano et al., 1998). The best-studied species in this group is *Halophytophthora vesicula*, commonly isolated from mangrove leaves. It has a narrower pH range for growth than most *Phytophthora* species (Kong et al., 2012), growing between pH 6 and 8.5, with an optimum of pH 7 (Leano et al., 1998). For this particular species, ocean acidification would thus provide more suitable vegetative growth conditions. The *Halophytophthora* species infecting seagrasses however may have very different pH tolerances and hence generic effects of ocean acidification on *Halophytophthora* are hard to



predict.

Salinity extremes are likely to occur in shallow coastal zones where seagrasses generally grow and *Halophytophthora* pathogens (and decomposers) may be affected by such conditions. Although “*Halo*”, coming from the Greek word “Háls” – meaning salt, would indicate that *Halophytophthora* are salt-loving *Phytophthora* relatives, many *Halophytophthora* have a broad salinity tolerance ranging from 0 to 50 ppt (Leano et al., 1998; Nakagiri and Newell, 1994). Optimum levels for vegetative growth are however somewhat more specified (between 15 and 30 ppt) and requirements for sporulation (production of zoospores for dispersal) are even more specific and depend on species (Leano et al., 1998; Nakagiri and Newell, 1994). Thus, *Halophytophthora* will very likely survive both high and low salinity extremes, although growth and dispersal of these pseudo-fungi may be limited during such events.

#### 4.3.4. Current topics

*Halophytophthora* species in temperate waters have long been overlooked (Nigrelli and Thines, 2013) and only very recently, *Halophytophthora* species were discovered to cause disease in seagrasses (*Zostera marina*). Current themes in seagrass-*Halophytophthora* research therefore comprise the same basic questions as for seagrass-*Phytophthora* research. In addition, stringent questions that need further investigation is if the same *Halophytophthora* species that act as decomposers may also act as pathogens and under what conditions these saprophytes turn pathogenic.

#### 4.4. Phytomyxea

##### 4.4.1. Description

Phytomyxea are obligate biotrophic parasites known to infect vascular plants, brown algae, diatoms and oomycetes. Phytomyxea are currently classified in the Phytorhiza, a sub-group of the Rhizaria, a protistan supergroup (Sierra et al., 2016). The best known species is *Plasmodiophora brassicae*, the causal agent in the development of clubroot in cabbage (Dixon, 2014) (Fig. 3a–b). They are able to infect and reinfect host species in multiple kingdoms, but there seems to be a separation between marine and terrestrial species (Neuhauser et al., 2014). However, the role of Phytomyxea beyond their

phytopathological potential is less well-known and data about their abundance and occurrence in natural ecosystems are largely missing (Neuhauser et al., 2011). In seagrass environments, four phytomyxid species have been described which all have been identified as the causal agents in the formation of shoot galls (Fig. 3c–d), which are hypertrophic enlargements of the cortical tissues in seagrass rhizomes (Den Hartog, 1965). Enlargements in host root tissues can appear as a ‘string of beads’ forming from the roots tissues while appearing to advance up the stem (Fig. 3d). They greatly alter the morphology of affected tissues, however, the uninfected part of the plant may appear normal. Morphologically phytomyxids are characterized by their distinct resting spores. In their vegetative form, they can be identified as large, amoeba-like, multinucleate single-celled plasmodia that fill the entire host cell before cleaving into spores, which then release flagellated motile zoospores and are free to infect new hosts.

##### 4.4.2. Host-pathogen relationship

All described phytomyxid pathogens of seagrasses cause enlargements, or hypertrophies, of the rhizomes, internodes or shoots of the seagrasses. From genetic studies of the clubroot pathogen, it is known that these hypertrophies are linked to a pathogen-induced alteration of the auxin and cytokinin metabolism of the host (Ludwig-Müller, 2014), so it is likely that similar strategies will be employed in seagrasses. Two species of *Plasmodiophora* have been well documented and have been repeatedly linked to shoot gall disease in seagrasses of both temperate and tropical regions of both northern and southern hemispheres (Den Hartog, 1965, 1989; Walker and Campbell, 2009). Infection of seagrasses by *Plasmodiophora bicaudata* J. Feldman was confirmed across a wide range of *Zostera* species in temperate regions of the globe, including *Z. muelleri* ‘complex’ in Australia, *Z. capensis* Setch. in South Africa, *Z. noltii* in Europe and *Z. japonica* in Asia and the United States (Den Hartog, 1989). *Plasmodiophora diplantherae* (F & W) Iv. Cook affects *Halodule* species, including *Halodule wrightii* Asch., *Halodule uninervis* Forssk., *Halodule tridentata* Steinh., *Halodule pinifolia* Hartog, and *Halodule beaudettei* Hartog in tropical regions of the world (Karling, 1968). *Plasmodiophora halophila* was described in 1913 from herbarium material of *Halophila ovalis* (r.Br) Hook.f., and the species is now considered to be doubtful (Karling, 1968). The fourth known species, *Tetramyxa parasitica*, forms galls on *Ruppia* spp. and seems to be

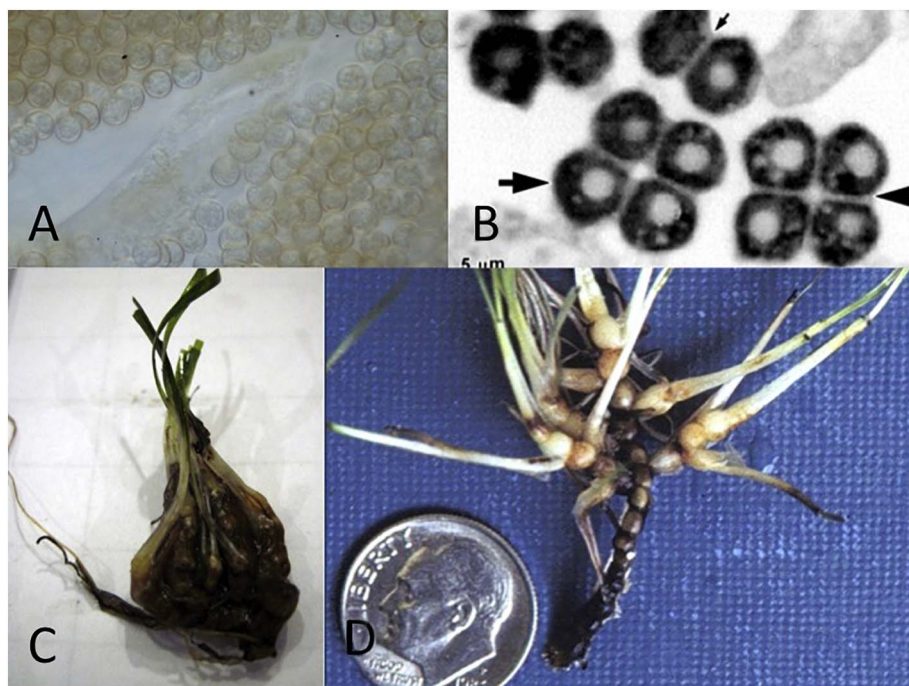


Fig. 3. a) Resting spores of *Plasmodiophora diplantherae* a parasite of *Halodule wrightii*, light microscopy. b) Resting spores of *Tetramyxa parasitica*, parasite of *Ruppia* spp. TEM picture (picture from James Braselton). c) *Plasmodiophora bicaudata* infected *Zostera noltii*. d) *Plasmodiophora diplantherae* infected *Halodule* (picture from James Braselton).

widespread in northern Europe (Karling, 1968). Like in all phytomyxids, no direct antagonistic effect between the parasite and the host can be observed, although infected plants often show dwarfed growth as a result of the enlarged internodes which divert the energy otherwise directed into lateral growth into the formation of gall tissue and the parasite. Additionally, Den Hartog (1989) reported that he could not find any inflorescences on *Halodule* spp. infected with *P. diphantherae*.

#### 4.4.3. Global change dynamics

Gall formation from Phytomyxea infection is accompanied by alterations of the seagrass rooting tissues, and in some species largely eliminates rhizome elongation at the internodes. This effect has been discussed to reduce resilience of individual plants to existing hydrodynamic forces (Walker and Campbell, 2009). This suggests even further risk of losses to seagrasses, including *Zostera* and *Halodule*, under global change scenarios that predict increased storm events, including larger and stronger waves and currents in seagrass habitats (Short and Neckles, 1999). As seagrasses are championed for their ability to reduce coastal erosion and alleviate storm surges (De Boer, 2007), impacts from phytomyxid infection on seagrasses could have cascading effects on ecosystems and existing conservation and management plans. Acidification and changes in chemical properties can potentially impact the disease prevalence and abundance: the clubroot pathogen *P. brassicae* causes the largest damage in agricultural ecosystems when the soil pH is 6.5 or lower and is often kept below the detection limit when the pH is higher (Dixon, 2014; Gossen et al., 2014). Whether or not a similar effect of increased disease severity and abundance will be observable in seagrass ecosystems needs to be tested, as not all phytomyxid show such a marked pH dependence than *P. brassicae* (Dixon, 2014).

#### 4.4.4. Current topics

There is a paucity of information surrounding seagrass-infecting phytomyxids. There are large gaps in our understanding of the life cycle of these parasites, as well as of the host-parasite interactions, including infection mechanisms and the impact of infection on plant physiology in the marine environment. While infection does not seem to affect the development of leaf tissues (Den Hartog, 1989) it is known that phytomyxids impact on the photosynthesis of their host (Ludwig-Müller, 2014). Also potential alterations of inflorescence were described in an anecdotal report and a greater understanding of physiological effects on root tissues are also needed (Den Hartog, 1989). In land plants the seed quality of clubroot infected plants is inferior to plants free of infection (Gossen et al., 2014), an effect which has not been studied to date but which would be dramatic in the context of restoration efforts. As species in this genus are capable of host shifts across kingdoms, and have very resistant and long-lived resting spores, the persistence of spores and potential reservoirs in natural systems must be explored.

### 5. Potential risk of increasing disease for seagrasses in future

Through an analysis of known relationships between host, pathogen, and host-pathogen effects under given climate thresholds (Table 2), we may begin to predict key drivers of increasing disease events under future global change scenarios. Generally, with current and predicted alterations to marine environments globally, the likelihood of infection due to decreased defences and likewise increased susceptibility of the host, is hypothesised to increase (Harvell et al., 1999; Harvell et al., 2002).

We developed a novel framework for using known responses of hosts and pathogens to described global change parameters to determine a risk factor for global change stressors contributing to disease in seagrasses (Fig. 4). Here, depending on positive or negative responses of hosts and pathogens under a given parameter level (Table 2), we were able to deduce one of three potential levels of disease risk associated with that parameter. These risk levels are 1) High, when host

immunity is suppressed and pathogen is enhanced (e.g. warming temperatures and ocean acidification), 2) Moderate, when both or neither host or pathogen are enhanced (e.g. freshwater inputs) and 3) Low, when the host is enhanced and the pathogen is suppressed (e.g. increased atmospheric CO<sub>2</sub>, potentially). The results of our investigation suggest the highest overall risk of infectious disease outbreaks in seagrass ecosystems may be related to ocean acidification, hypersalinity and warming temperatures. Still, Table 2 demonstrates the lack of basic research into the roles of several environmental parameters on host, pathogen and host pathogen relationships. Thus, further research in these areas is urgently needed to develop and test these theories, and to ultimately better understand the risks and mechanisms of disease and severity related to these parameters.

### 6. Expanding capacity and resilience in seagrass disease research

Similar to human diseases, the onset of disease in plants and animals can be sudden and widespread (Sullivan et al., 2013; Woodroffe, 1999), causing long-term loss or alteration of function (Cottam and Munro, 1954; Dexter, 1944), and thus losses of associated ecosystems services that are beneficial to humankind (Fisher et al., 2012; Harvell et al., 2002). Given the lack of knowledge about seagrass diseases, it is critical to develop a strategy for managers and researchers to use when prioritising resources for monitor existing populations and preparing for future outbreaks.

When strategising responses to emerging threats to human health from infectious disease, researchers have identified key focus areas to promote preparedness for outbreaks of infectious disease (Gostin et al., 2002). Other researchers have reviewed critical predictors of infectious disease outbreaks under global change regimes (Altizer et al., 2013). We have used these considerations as a building block for prioritising activities that increase our capacity to respond to global outbreaks of seagrass diseases. Thus, we offer five key recommendations, or action items, summarising priorities for building our capacity to respond to outbreaks and advance resilience of seagrasses in the future.

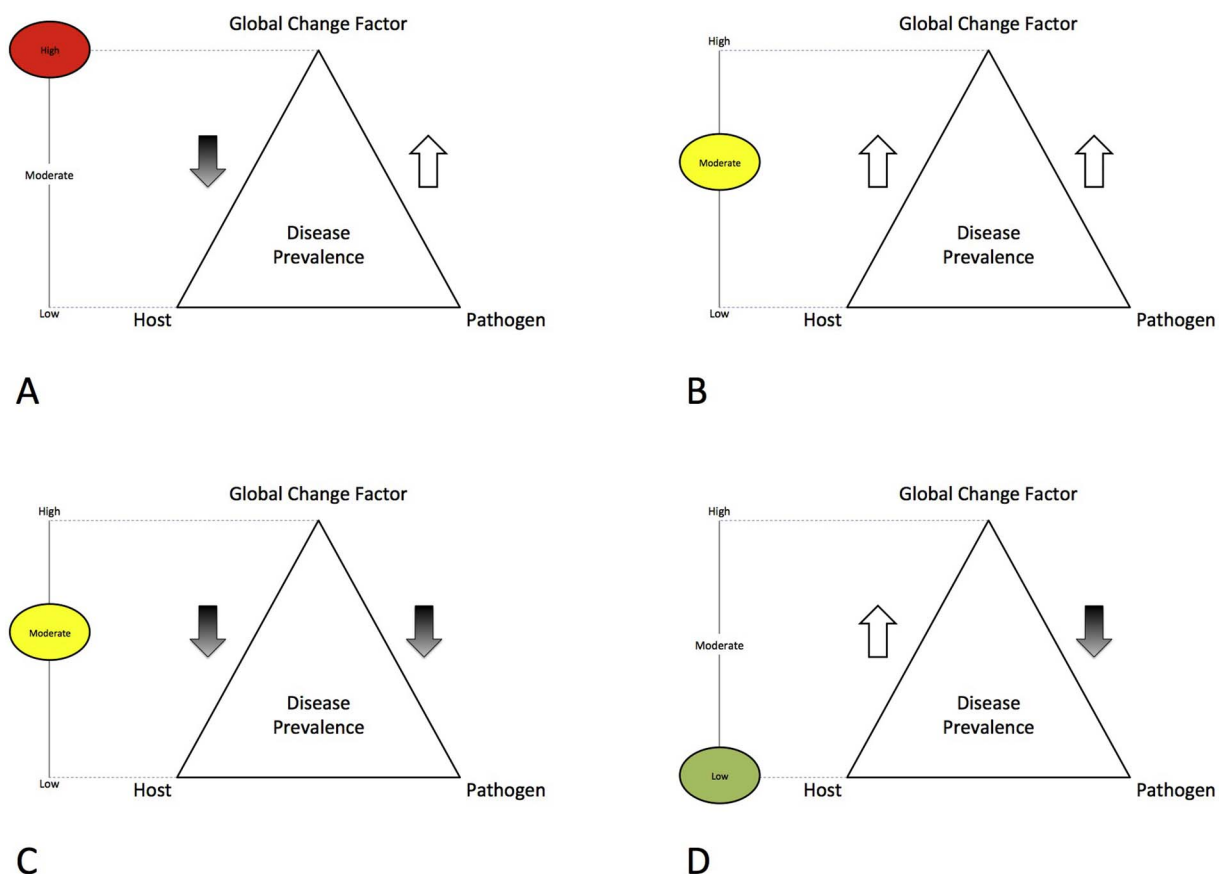
1. *Continue support for basic seagrass disease research.* The lack of basic research, management and monitoring for marine disease escalation under global change scenarios are presumably due to a lack of specific evidence that disease events in seagrass may be exacerbated under future global change scenarios. In fact, as noted above, some studies even point to some contrary evidence in results. Still, evidence in multiple host-pathogen fields indicate that the role of climate is exacerbating disease in marine and coastal ecosystems around the world and warrants support of research, monitoring and management programs for these ecologically important seagrass ecosystems. Successful development of pathogenesis is a function of properties of both the host and pathogen. The immune response of the host and virulence of the pathogen both play a part in the resulting severity of disease (Casadevall and Pirofski, 2003). We also know that seagrasses are expected to be impacted by global change conditions (Short and Neckles, 1999). Impacts from global change are increasing instances of disease and these increases are expected to continue in ecosystems worldwide (Altizer et al., 2013). In fact, attention to climate-related increases of disease in other marine ecosystems, such as corals and fisheries aquaculture, are gaining momentum. We find the role of global change phenomenon in exacerbating the threat of disease in seagrass ecosystems around the world has not been sufficiently investigated to warrant the current omissions in research and management schemes. Further, we suggest that continued research will help build models to identify potential disease hotspots and areas of vulnerability or resilience around the world. We offer a framework for advancing future seagrass disease research that responds to host, pathogen and environmental conditions (Fig. 4).



**Table 2**

Summary of predicted host and pathogen responses to anthropogenic global change. Potential responses of seagrass and known pathogens to anticipated future global change scenarios and identification of research gaps. (“↑” = positive relationship, “↓” = negative relationship, “±” = neutral relationship, and “?” = relationship unknown). Global change stressors derived from Short and Neckles (1999), Duarte (2002), Harley et al. (2006), and Koch et al. (2013).

Global change stressor	<i>Labyrinthula</i> Responses	<i>Phytophthora</i> Responses	<i>Halophytophthora</i> Responses	Phytophyxea Responses	Seagrass Responses
Warming temperatures	↑ (Watson and Ordal, 1951; Pokorny, 1967; Olsen et al., 2015)	↑ (Bergot et al., 2004)	± to ↑	↑ to ↓	↓ (Short and Neckles, 1999)
Hypersalinity	↑ to ↓ (Young, 1943; Pokorny, 1967; Bishop, 2013)	↑ (Govers et al., 2017, Bouchibi et al., 1990)	± to ↓ (Leano et al., 1998; Nakagiri and Newell, 1994)	?	↓ (Kahn and Durako, 2006; Touchette, 2007)
Eutrophication/increased plankton and algal blooms	↑ (Jepps, 1931; Armstrong et al., 2000)	↑ ?	?	?	↓ (van Katwijk et al., 2009)
Ocean acidification	± (Pokorny, 1967) Wide ranging pH tolerances	± (e.g. Kong et al., 2012)	? ↑ (Leano et al., 1998)	? ↑ (Gossen et al., 2014)	± (Beer et al., 2006; Mealey Hon theis)↓
Freshwater inputs	↓ (Young, 1943)	↓ (Govers et al., 2017)	± to ↓ (Leano et al., 1998; Nakagiri and Newell, 1994)	±	↓ (Lirman and Cropper, 2003)
Increased hydrogen sulfide	↓ (Vishniac, 1955)	↓ (Govers et al., 2016)	↓ (Govers et al., 2016)	?	↓ (Holmer and Bondgaard, 2001; Koch and Erskine, 2001)
Increased atmospheric CO <sub>2</sub>	?	?	?	?	↑ (Zimmerman et al., 2017) Negative (Arnold, 2012)
UV-B radiation	?	?	?	?	↓ (Larkum and Wood, 1993)
Increased storm frequency and intensity		?	?	?	↓ (Short and Neckles, 1999)



**Fig. 4.** Conceptual framework demonstrating three potential levels of disease risk. These include, 1) high (when the host is suppressed and pathogen is enhanced), 2) moderate (when both or neither host or pathogen are enhanced) and 3) low (when the host is enhanced and the pathogen is suppressed). Here we evaluate the results of potential co-effects of described global change stressors on known seagrass diseases (summarised in Table 2).

2. *Establish emergency funding sources.* Traditional models of funding, such as those that rely on lengthy annual applications or specific collaborations with identified institutions, may not be successful in relieving emerging infectious disease events (Groner et al., 2016b; Woodroffe, 1999). When access to immediate funding may be required to investigate an emerging disease event, we suggest an international organization could be tasked to collect and quickly release funds, as is the case for diseases that directly affect humans through the oversight of the World Health Organization. Funding opportunities generally must include both long-term and short-term solutions, including availability of emergency funding, alongside traditional grant funding schemes that support long-term basic research objectives. Provision of emergency funds will allow researchers to engage with active epidemics and derive real-world data to compliment the seagrass disease research framework and fill current research gaps.
3. *Enhancing global expert collaborations and perspectives.* We suggest international collaborations are key to getting ahead of recognised marine infectious diseases, especially those known to have global reach. We suggest the creation of a seagrass task force aimed at advancing global understanding of issues in marine plant diseases. This task force should specifically oversee the coordination of conference sessions and workshops at the International Seagrass Biology Workshop on current topics in seagrass disease. In addition, this task force could connect to similar groups, such as the Coastal and Estuarine Research Federation and individual researchers studying disease in other marine plants such as mangroves or salt marsh plants. By encouraging an exchange of knowledge and greater awareness of disease issues, conference sessions may help in generating evidence of global disease occurrences while also helping form expert research collaborations. Through the exchange of ideas, we believe greater progress in basic research and modelling of global trends is more likely, and is warranted for this critical ecosystem.
4. *Develop an adaptive framework for responding to emerging seagrass disease events.* When a monitoring event reveals an epidemic disease event is underway or is at risk of developing, a response framework for effective action would aid managers and researchers in attempts to harness emerging infectious disease events in seagrass ecosystems and thus potentially prevent or reduce acute losses to ecosystem function and human services. For instance, we know transmission rates and severity of disease may be reduced by mitigation measures such as optimising host resilience, managing environmental conditions known to facilitate disease and reducing pathogen abundance (Groner et al., 2016b; Lamb et al., 2017). In addition, transmission rates globally may also be reduced through enhanced international biosecurity measures. For example, a newly described species *Phytophthora gemini* is very likely an invasive species in the Wadden Sea as genotypic variation in this species is very low. In Alaska, the same species was isolated displaying a great deal more genotypic diversity, and as such, it may have been the source of introduction to the Wadden Sea (Man in 't Veld et al., in review). The potential transmission of seagrass disease agents through transference of ballast water or distribution of plant materials is unknown.
5. *Development of an on-going global seagrass disease database.* We propose the development of a world-wide GIS-database of seagrass monitoring and disease events, similar to those developed to understand the spread of human diseases (Hightower and Klein, 1995). A centralised data source could assist researchers and managers in regional areas and facilitate further international collaborations. The potential to link to existing seagrass citizen science mapping programs like Seagrass Spotter (<https://seagrassspotter.org/>) may also be effective at encouraging engagement and collecting large

amounts of monitoring data.

In summary, there are three known groups of pathogenic organisms currently known to cause disease symptoms in seagrass, including labyrinthulids, oomycetes and phytomyxids. Global change is likely to affect seagrass-pathogen relationships, for instance through climate warming, hypersalinity and eutrophication. Ocean acidification, low salinity events and increased sulfide production may also have some effects on seagrass-pathogen dynamics. However, research into the specific impacts and mechanisms of these effects, and co-effects, are largely unknown. We summarise three key areas of research needed to advance our understanding of disease phenomenon, especially as we face a future of global changes.

1. *Biological Research.* Many of the research gaps revealed in our assessment can be described as simple lack of basic research into key biological features and interactions of hosts, pathogens or host-pathogen interactions and environmental parameters. These gaps include unknown mechanisms of infection and transmission, genetic precursors to disease, and host defence or immune systems. With regards to *Labyrinthula*, even after decades of research, we are still unclear about the basic mechanisms by which the host is able to enter and infect the host leaf tissues. Several theories have been presented (Muehlstein, 1992), but no conclusive evidence has been obtained. It will be vital in the future to establish model seagrass systems needed to study disease ex-situ. We suggest analysing basic biological interactions in a targeted and systematic way to understand the processes that form the basis of the pathogenesis. Additionally, the role of biota outside of the immediate host-pathogen relationship may play a role in disease presence, transmission and severity. However, there is no research into the potential role of parasitism in reducing pathogen loads (Gleason et al., 2014), or for mesograzers to facilitate disease in seagrasses, as has been shown in several terrestrial and saltmarsh plants through host tissue damage (Daleo et al., 2009). Additionally, pathogenic microbes can have direct predators, which could further reduce pathogen loading (Schmeller et al., 2014). However, food web dynamics at smaller scales and identification of key players in the microbiome of seagrass communities are only just beginning to be explored (Fahimipour et al., 2017). Biological control and biological causes of disease transmission and severity in seagrasses, has not been studied.
2. *Pathogen discovery.* There are roughly 60 recognised species of seagrass globally. So far, only pathogens belonging to three different eukaryotic groups have been identified, and these mostly affect the most extensively studied seagrass species (e.g. *Zostera marina*). The recent discovery of new and widespread seagrass pathogens, such as *Phytophthora* spp. and *Halophytophthora* species, are indications that diseases may be present that have been previously overlooked. Additionally, research suggests there may be a pathogenic role of the Ascomycete, *Lindra thalassiae*, described as causing dieback in the seagrass *Thalassia testudinum* (Porter, 1986). More recently, researchers have found the oomycetes *Salisapilia* sp. on *Zostera noltii* (Man in 't Veld et al., in review) and the fungus *Phialophora malorum* on *Zostera marina* (Govers et al., 2017) may generate disease symptoms. Bacteria and viruses also threaten marine sessile organisms like algae, corals and sponges (Harvell et al., 1999; Suttle et al., 1990). However, other research demonstrates bacteria and viruses can moderate eukaryotic diseases (Gil-Turnes et al., 1989; Rohwer and Thurber, 2009). Thus, it is worthwhile to investigate the effects bacterial and viral pathogens and symbionts may have on seagrass ecosystems. These discoveries indicate that there are potentially many more seagrass pathogens to be identified. Since global change will very likely also affect these unknown pathogens and their relationship with seagrass hosts, it is very important to increase efforts

to discover, characterise and understand symbiotic relationships of eukaryotic microorganisms and seagrasses.

**3. Modelling.** Using results from basic biological and ecological research, we can build mathematical models to assist our understanding of the complex environmental and biological conditions that lead to disease. Additionally, disease is shaped by, and may in turn shape, host demography with population level consequences (Metcalf et al. 2015). Modelling may assist in the identification of populations that are more or less vulnerable to disease emergence, or to predict disease transmission and severity in an emerging epidemic. We know the ability to model the emergence and transmission of disease are key to preparing for, and potentially mitigating, disease events in human and animal systems (Groner et al., 2016a; Heesterbeek et al., 2015). We believe the time is right to begin modelling efforts in seagrass disease as well.

**4. Monitoring.** In order to respond to an emerging disease event, consistent monitoring of hosts and environmental conditions are required (Groner et al., 2016b). Observations should include improving existing monitoring of ambient environmental conditions known to promote disease, to include rapid diagnosis methods for targeted diseases, such as rt-qPCR and analysis of eDNA (Thomsen and Willerslev, 2015). Consistent observations of vulnerable populations are needed. In order to appropriately identify monitoring targets, continued support of basic research into host, pathogen and host-pathogen biology is essential. This basic research aids in the development of disease transmission and expression models that may help identify environmental thresholds and key hotspots or seasonal factors where infection may lead to disease emergence.

In the face of future global change phenomenon, such as climate disruptions, marine ecosystems are believed to face several challenges. We predict seagrasses are likely to be increasingly vulnerable to disease under projected global change scenarios. Thus, there is a need to investigate a variety of potential environmental conditions, including disturbance thresholds and co-occurring effects, as effects may be accumulative and non-linear. In addition, climate related disease effects are likely to be species and region specific, therefore international and cross disciplinary collaborations on seagrass disease issues, such as this one, are key to ensure adequate use of existing and novel techniques for understanding, monitoring, and hopefully in the future, managing the role of diseases in seagrass ecosystems.

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