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REVIEW ARTICLE

Aquatic disease in New Zealand: synthesis and future directions

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ABSTRACT

Dedicated studies on aquatic disease in New Zealand began in 1974, inspired by a developing aquaculture industry. Since then, two main aquatic disease study areas have emerged: (1) aquatic disease ecology and (2) diseases of commercial species. Progress over the past 20-years has been made by only a small number of researchers and aquatic disease in New Zealand has not received much attention from the wider marine science community. In 2020, the aquaculture industry continues to grow, and disease remains a threat to the industry's viability. However, additional factors such as climate change, invasive species, and pollution, have emerged as future threats for aquatic disease in wild and farmed populations, which are currently understudied. Here, we provide a review of studies on aquatic disease ecology and commercial species carried out in New Zealand. We also present how climate change, pollution and invasive species could influence future aquatic disease dynamics and identify where future research effort is required to address knowledge gaps. The emergence of consequential aquatic diseases overseas highlights wider attention across marine science disciplines is needed to progress and diversify aquatic disease studies in New Zealand.

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Introduction

The detection of whirling disease, caused by the Myxozoa parasite *Myxobolus* (=*Myxosoma*) *cerebralis*, in a Dunedin trout hatchery in 1972 (Hewitt and Little 1972), identified a critical knowledge gap of aquatic diseases in New Zealand (Waugh 1975). As a result, a call went out for more detailed studies on aquatic diseases in the country, principally to support a developing aquaculture industry (Waugh 1975). In 1974, a New Zealand Fish Disease Seminar informed government, the New Zealand research community and other interested parties of current work areas and problems afoot regarding aquatic diseases (Waugh 1975). Topics covered in the seminar included diseases of aquaculture species, aquatic disease management, biosecurity issues of fish importation, and the role of universities in addressing aquatic disease topics (see Waugh 1975). The topics covered at the Fish Disease Seminar are still relevant today, nearly 50 years later. Initial studies on

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aquatic diseases were generally descriptive, dominated by a basic natural history approach of helminth parasites, consisting in the discovery and elucidation of life cycles (Howell 1967). While parasite species discovery continues (Randhawa and Brickle 2011), studies have now increased in scope to include molecular diagnostics (Brosnahan et al. 2019a; Keeling et al. 2012), the application of '-omics' (Nguyen and Alfaro 2020) and epidemiology of commercially important diseases (Pande et al. 2015). Through the influence of ecological theory and the incorporation of diseases into ecological studies, we now better understand how diseases can influence the structure, dynamics and function of natural populations and communities (Mouritsen and Poulin 2005a, 2005b).

The total number of aquatic disease studies in New Zealand has steadily increased since 1991, while the proportion of aquatic disease studies of the total marine science studies has remained stable for the same time period (Figure 1). Advances in aquatic disease in New Zealand have been made by a handful of people, as aquatic disease receives little attention from the wider marine science community. In fact, aquatic disease has received little attention globally (Lafferty and Hofmann 2016). The first marine disease ecology textbook was only released in 2020 (Behringer et al. 2020), having largely been neglected in earlier editions of marine ecology textbooks (Lafferty and Hofmann 2016). Almost 40% of all aquatic animal disease studies in New Zealand has come from one institution (Figure S1) and the top 10 authors by publication count contribute 39% of all aquatic disease research in New Zealand (Figure S2). In contrast, the top 10 authors by publication count for a comparable number of studies on climate change in marine ecosystems in New Zealand contribute just over 10% of all marine climate change research (Figure S2). These contrasting figures indicate that research on aquatic disease is mainly conducted by a small number of researchers, whereas interest in climate change is more broadly shared by marine scientists. In a

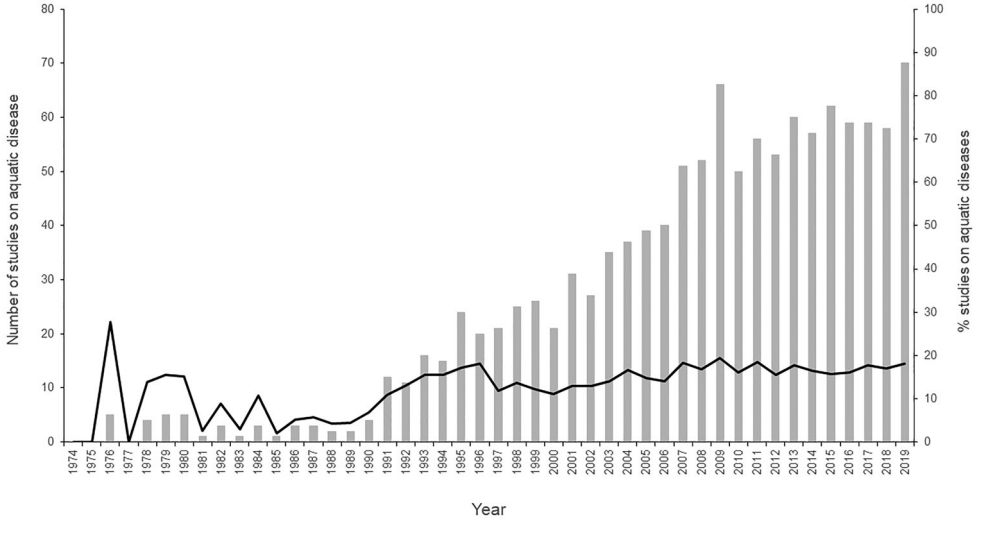

Figure 1. Total number of studies on aquatic disease in New Zealand (bars) and the proportion of aquatic disease studies out of the total number of marine science studies (line) in the period 1974–2019. Refer to supplementary material for search strings.

recent survey of the New Zealand marine science community, aquatic disease was not considered a research priority (Jarvis and Young 2019). Despite this, the aquaculture industry continues to evolve (Heasman et al. 2020), New Zealand's borders are busy through commerce (and tourism pre-COVID-19 border closure) (MPI 2019), and the marine environment continues to change (Law et al. 2018), all of which can alter disease dynamics that can have ecological and socioeconomic consequences (Cranfield et al. 2005; Harvell and Lamb 2020).

In reviewing the literature from New Zealand on aquatic diseases, it is clear there are two main areas of work: (1) studies of parasites (mainly helminths) in natural systems that contribute to our understanding of aquatic ecosystems through hypothesis-driven science; and (2) applied studies, usually of animals of commercial importance, e.g. aquaculture and fisheries species and marine mammals. The central tenet of each area is different: the former strives to understand the role disease has in driving intra- and inter-species interactions and structuring communities, whereas the latter aims to diagnose and manage commercially important diseases. *In lieu* of a second Fish Disease Seminar, we provide a review of studies on aquatic diseases in New Zealand to determine what we know, where knowledge gaps lie, and where we should direct our future focus. To achieve this, we first introduce basic disease concepts, including some definitions and how disease can occur. Second, we review salient studies of disease in natural marine ecosystems and how disease can structure marine communities. We then review studies on diseases of commercially important species (farmed and wild). Our primary focus is on estuarine and marine species, including anadromous species, although where relevant we use freshwater examples. We do not document all parasites detected in New Zealand (Lehnert et al. 2017) nor do we review what diseases are not present in New Zealand (Diggles et al. 2002a). Third, we discuss climate change, invasive species and pollution and how these might influence disease dynamics. Finally, we identify areas of future research and where greater synergy among marine science disciplines should occur to grow and direct aquatic disease research in New Zealand. Our overall goal is to give greater exposure to aquatic disease studies in New Zealand and encourage greater participation and contribution to this field from the New Zealand scientific community.

An introduction to aquatic disease

A quick scan of the syllabus of New Zealand university courses for marine biology or related subjects shows that disease is not commonly taught, so it is presumed that many marine biology and ecology graduates will not be familiar with disease or epidemiological theory and that some background information will be useful. For a more in-depth overview of aquatic diseases, refer to Behringer et al. (2020).

Generally, infectious agents are categorised into two groups, parasites and pathogens, where parasites are comprised of macroparasites like helminths, i.e. worm-like parasites, and microparasites like haplosporidian *Bonamia* parasites, whereas pathogens are comprised of viruses, bacteria, fungi and oomycetes. For the purpose of this review we will use the word parasite in its broader sense, i.e. including macro- and microparasites, bacteria, viruses, fungi and oomycetes, and then state the relevant taxa for specific examples. Parasites, pathogens, infectious agents, and etiological agents are all terms that could be used interchangeably. Parasites are **endemic** if they are known to occur in New Zealand, e.g.

the haplosporidian oyster parasite *Bonamia exitiosa* (Dinamani et al. 1987; Berthe and Hine 2003), and are **exotic** if they are not found in New Zealand prior to a documented introduction, e.g. the salmonid orthomyxovirus infectious salmon anaemia virus (ISAV).

Disease is a negative deviation from normal health, demonstrated by reduced function, changes in form, or both. Reduced function can encompass reduced feeding, socialising, growth, reproduction or survival (Coen and Bishop 2015). How, when, and where disease may occur is the primary concern of the field of **epidemiology**. Central to epidemiology are **transmissibility** and **susceptibility**. Transmissibility is a parasite's or disease's ability to spread to other organisms, while a susceptible host is one that is at risk of becoming infected. Susceptible hosts that become infected may acquire immunity and once recovered from the disease are no longer susceptible. Transmission can be horizontal (between individuals) or vertical (from parent to offspring). Organisms that transmit parasites are known as **vectors**. Helminths are often **trophically transmitted** and have complex lifecycles that use at least two different hosts and transmit via predator-prey interactions (Anglade and Randhawa 2018). Conversely, **directly transmitted** parasites like bacteria, viruses or parasitic copepods, like salmon sea lice (Costello 2006), are transmitted among conspecifics. Refer to Poulin and Randhawa (2015) for an overview of the six general parasitic life strategies. Simply put, the probability of disease increases with a higher density of susceptible hosts, i.e. transmission is more likely to occur. However, the relationship between transmission and susceptible host density is not always linear. For instance, transmission might remain high at low host density for social species (Johnson et al. 2011).

Disease is caused by parasites; however, the presence of a parasite does not necessarily equate to disease. At any one time an animal is likely to be affected by one or many parasites and be perfectly healthy (H. Lane pers. obs.). Disease is largely governed by the interaction of three factors: the host, the parasite, and the environment (Sniesko 1974), known as the **epidemiological triangle** (Figure 2)**.** Recent investigations give consideration to the role of the host and parasite microbiome in the development of disease (Dheilly et al. 2019; Bernardo-Cravo et al. 2020) (see Figure 2), however, for this review we will consider the three components of the epidemiological triangle in the development of disease. The outbreak of disease is the result of a shift in the host, parasite, or environment (Sniesko 1974). A shift may come in the form of **pathogenicity** or **virulence** of the parasite. Pathogenicity is the capacity of the parasite to initiate a given disease and associated related parasite production, whereas virulence is the degree of pathogenicity as indicated by the severity of a given disease and its capacity to invade the host's tissue overall (Coen and Bishop 2015). Alternatively, the host may become more susceptible, e.g. immune-compromised, or the environment becomes less favourable for an organism, moving it beyond the bounds of its ability to adjust, known as stress. Stress may negatively impact the host and/or parasite, but when it negatively impacts the host, the host may become more susceptible to infection, particularly from **opportunistic parasites**. For instance, in humans, we are familiar with wounds that become infected under unsanitary conditions by opportunistic bacteria that are otherwise benign when a person is healthy. Opportunistic parasites are more dependent on environmental factors to cause disease, and the emergence of opportunistic parasites during climate instability is an important future focus (Harvell 2019). An **emerging disease** is a disease that increases in prevalence, geographic range, or virulence, moved into new

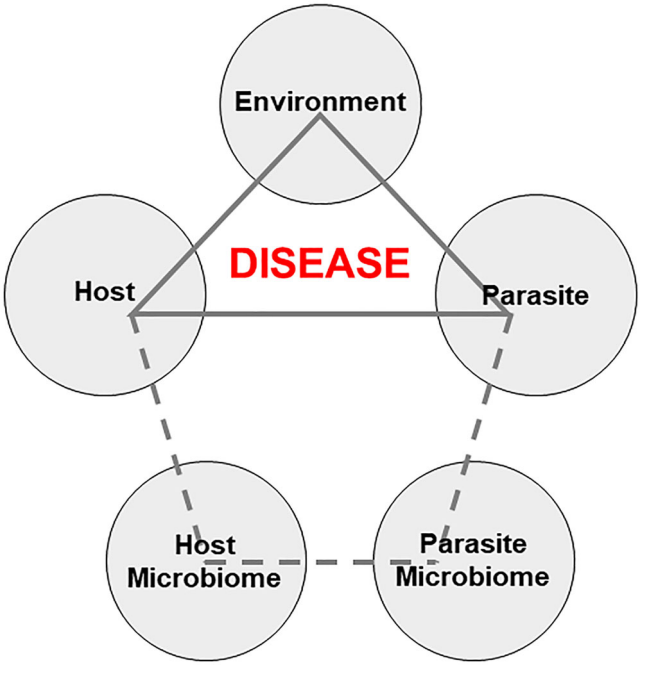

Figure 2. The epidemiological triangle demonstrating the three interacting factors that govern the outbreak of disease, including the prospective epidemiological pentagon (dotted lines) with two proposed additional factors of host microbiome and parasite microbiome.

hosts, newly evolved, or is a new disease (Brown 2000). Examples of emerging aquatic disease include the sea star wasting disease that caused the precipitous decline of keystone North American sea stars (Hewson et al. 2018), or the emergence of new pathogenic viruses associated with the rapid growth of global prawn aquaculture (Walker and Mohan 2009).

Principles of terrestrial veterinary science, epidemiology and disease ecology do not necessarily apply in the aquatic world. For starters, seawater is 800 times denser and 50 times more viscous than air (McCallum et al. 2004). These physical properties keep particles, such as parasites, suspended in the water where they can be transported over long distances, precluding the need for vectors like many terrestrial diseases, e.g. malaria (McCallum et al. 2004). Studies on aquatic disease have lagged behind their terrestrial counterparts, probably because marine organisms were not as readily available to study as terrestrial animals, and economic interests in New Zealand only recently diversified from purely terrestrial agriculture to include aquaculture. Considering the first volume (including six issues) of the New Zealand Veterinary Journal was published in 1952 and aquatic disease studies did not noticeability appear until 1991 (see Figure 1), it is clear our understanding of aquatic disease is in its infancy compared to terrestrial disease.

The World Organisation for Animal Health (OIE) lists important parasites of terrestrial and aquatic animals that pose the greatest economic, social and ecological risks in order to prevent their transboundary spread. The OIE is analogous to the human

health focused World Health Organisation (WHO). By law, the OIE must be notified of the detection of any OIE-notifiable parasites in order to maintain clear reporting and ensure their accurate management. In New Zealand, four OIE notifiable aquatic parasites have been detected: *Bonamia exitiosa* (see Dinamani et al. 1987), *Bonamia ostreae* (see Lane et al. 2016), ostreid herpesvirus-1 microvariant (OsHV-1 μvar) (see Keeling et al. 2014), and *Perkinsus olseni* (see Hine and Diggles 2002). These parasites only affect mollusc species, and none of the OIE notifiable aquatic parasites affecting finfish and crustaceans have yet been reported from New Zealand. A full list of OIE notifiable aquatic animal parasites can be found on the OIE website (https://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2020/).

Aquatic disease ecology

Parasites are naturally occurring in the aquatic environment. They are the most abundant organisms in the marine environment and parasitism the most common lifestyle (Lafferty and Harvell 2014). There are 10 billion viruses per litre of seawater (Fuhrman 1999), although not all of them are capable of causing disease, and the collective biomass of estuarine parasites outweighs that of animals at higher trophic levels (Kuris et al. 2008). Parasites have traditionally been overlooked in marine ecology studies and it was not until their inclusion that their ecological importance attracted wider attention (Marcogliese and Cone 1997). Parasites impact individuals, by affecting fitness components such as growth and reproductive success (Howell 1967), and populations, by lowering the abundance or density of their host species (Cranfield et al. 2005). In addition, a single parasite species can have community-wide impact by modifying the activity of ecosystem engineers or key grazers and indirectly affecting the density and diversity of other free-living species of algae or invertebrates (Thomas et al. 1998). It is clear that the size of parasites is disproportional to the impact they can have on marine ecosystems.

Much of what we know about aquatic disease ecology in New Zealand and overseas comes from studies on helminth parasites, e.g. digenean trematodes. Helminths make ideal model species for testing ecological theories, because they are ubiquitous in the marine environment, use different hosts across trophic levels, and are relatively easy to manipulate in the laboratory (Thomas et al. 1998; Studer and Poulin 2013; O'Connell-Milne et al. 2016b). Using only helminth model species creates blind spots in our understanding of the diversity of other host–parasite interactions (Poulin et al. 2016) (see section 5.1). For instance, how infection and mortality from *B. exitiosa* in the New Zealand flat oyster *Ostrea chilensis* affects the ecology of the Foveaux Strait is unknown. When parasites cause economic harm, the broader ecological effects seem less likely to be researched (but see Chiaradia et al. 2010).

Parasites, food webs and diseases

Food webs are complex networks through which energy and materials move across all trophic levels in an ecosystem. Despite their ubiquity, parasites were late additions to food web studies (Thompson et al. 2005). Parasites affect food webs in diverse ways. For instance, parasites infect across all trophic levels, mediate species interactions, and

either directly or indirectly affect the flow and direction of energy in an ecosystem (Thomas et al. 1998; Bennett et al. 2019; Bennett and Presswell 2019). Parasites are similar to predators by deriving energy from host tissue for their own growth and reproduction. A free-living stage for a parasite can also be an important energy source for lower trophic consumers. For instance, the common New Zealand intertidal anemone *Anthopleura aureoradiata* readily consumes the free-living transmission stages of trematodes (Hopper et al. 2008; Vielma et al. 2019). Parasites indirectly affect food web energy through hosts spending energy to avoid becoming infected or in mounting an immune response to control an infection or repair damaged tissues (Lafferty et al. 2008). Adding parasites to food webs provides a more complex and accurate overview of linkages, energy expenditure within species and energy flow between species (Lafferty et al. 2008).

The navigation of food webs provides a challenge for parasites, especially for those parasites with complex life cycles (Vielma et al. 2019). During the parasite's free-living stage, they can often end up as food for a non-host organism preventing the completion of the parasite's lifecycle (Vielma et al. 2019). Importantly, some parasites can manipulate host behaviour to facilitate trophic transmission to their final host, increasing the chance the parasite will complete its lifecycle (Poulin 2010). Adaptive parasitic manipulation of a host can be through a physical change in the host's appearance to attract predators or impair escape, such as colour changes making the host more conspicuous (Lagrue et al. 2016). Alternatively, a parasite can manipulate a host to put itself at greater risk of predation by changing its activity (Ruehle and Poulin 2020). A classic example in the New Zealand mud-flat community involves cockles (*Austrovenus stutchburyi*) and the digenean trematode *Curtuteria australis* (Thomas et al. 1998). Usually cockles bury into the benthos, but those heavily infected with *C. australis* remain at the surface because the parasite encysts in the foot of the cockle making it difficult for the shellfish to bury. Shellfish that are on the surface are many times more vulnerable to predation by the pied oystercatcher (*Haematopus longirostris*), the definitive host of *C. australis*, than those buried under the sediment (Thomas and Poulin 1998). This alteration of cockle burying behaviour increases predation rates on shellfish, altering energy flow through the intertidal food web.

Most of what we know about parasites come from nearshore ecosystems, e.g. the shallow intertidal and estuarine mudflats (see Table 1). There are many studies constructing food webs in New Zealand, but almost all do not include parasites (Leleu et al. 2012; Jones et al. 2017). Food webs incorporating parasites into New Zealand ecosystems are limited to a couple of localities: an estuarine mudflat (Dunne et al. 2013; Thompson et al. 2005) and freshwater lakes (Lagrue and Poulin 2015). A comparison of food web structure for the same estuarine mudflat between one with no parasites and one with parasites showed that including parasites increased food chain length, relegated top predators to intermediate status, and increased food web complexity (Thompson et al. 2005). The addition of parasites also modulates the energy transfer through a system, even opening up completely new routes of energy transfers, through behavioural manipulation as described for the cockle-trematode example above (Thomas and Poulin 1998; Thompson et al. 2005) Fundamental to incorporating parasites into a food web is some knowledge of the parasite's lifecycle because it demonstrates ecological linkages (e.g. Anglade and Randhawa 2018). As parasite studies move further offshore new parasite species and lifecycles will become uncovered, elucidating ecological linkages

Table 1. Studies on risk factors associated with climate change, e.g. temperature, ocean acidification, and aquatic disease in New Zealand.

Parasite(s) (Phylum)	Host(s)	Principle Area (s)	Risk factor(s)	Method	Results	Reference
Vibrio parahaemolyticus (Proteobacteria)	*Magallana gigas, Perna canaliculus, Ostrea chilensis* (Mollusca)	Human health and aquaculture	Temperature and salinity	Survey	*V. parahaemolyticus* was detected in 81% of *M. gigas* and 34% of GLM. Putative toxin gene *tdh* was detected in *V. parahaemolyticus* ($n = 3$). Overall, numbers of *V. parahaemolyticus* increased with water exceeding 19 °C.	Cruz et al. 2020; Cruz et al. (2015)
Vibrio vulnificus (Proteobacteria)	*M. gigas, P. canaliculus, O. chilensis* (Mollusca)	Human health and aquaculture	Temperature	Survey	*V. vulnificus* was present in 13.6% of *M. gigas*. Water temperature was a strong predictor of bacterial numbers in shellfish.	Cruz et al. (2016)
Bonamia exitiosa (Haplosporodia)	*O. chilensis* (Mollusca)	Aquaculture and wild fisheries	Temperature, salinity, food availability, disturbance, air exposure	Experimental	Highest mortality and infection intensity were observed in cold and hot water, hypersaline and trough exposure treatments.	Hine et al. (2002)
Matrima novaezealandensis (Platyhelminthes)	*Zeacumantus subcarinatus* (1st intermediate host) (Mollusca) *Paracalliope novizealandiae* (2nd intermediate host) (Arthropoda)	Intertidal ecosystem	Temperature	Experiment	Parasite transmission increased with higher temperatures, directly affecting second intermediate host survival.	Studer and Poulin (2013)
M. novaezealandensis (Platyhelminthes)	*P. novizealandiae* (Arthropoda)	Intertidal ecosystem	Temperature	Modelling	Simulation model investigating increasing temperature with parasitic infection for population dynamics of *P. novizealandiae*. Temperature increases within the forecast range can cause a collapse of *P. novizealandiae* populations.	Studer et al. (2013a)
Digenean trematodes (numerous) (Platyhelminthes)	*Austrovenus stutchburyi, Z. subcarinatus, Z. lutulentus Cominella* spp. (Mollusca)	Intertidal ecosystem	Temperature	Survey	Survey across a latitudinal gradient as a proxy for temperature found no difference in parasite abundance. Instead host prevalence and *A. stutchburyi* foot size were main predictors of parasite infection, suggesting local factors could offset wider ecological factors.	Studer et al. (2013b)
Digenean trematodes (numerous) (Platyhelminthes)	NA	Intertidal ecosystem	UV light	Experiment	Free-living cercariae are highly susceptible to UV damage and have little scope for protection or repair against UV damage	Studer et al. (2012a)

M. novaezealandensis (Platyhelminthes)	*Z. subcarinatus* (Mollusca) *P. novizealandiae* (Arthropoda)	Intertidal ecosystem	UV light	Experiment	Survival of free living cercariae decreased with increased UV light exposure, while infection susceptibility of the 2nd intermediate host increased.	Studer et al. (2012b)
M. novaezealandensis (Platyhelminthes)	*Z. subcarinatus* (Mollusca) *P. novizealandiae* (Arthropoda)	Intertidal ecosystem	Temperature	Experiment	Temperature strongly but differentially affects transmission and disease dynamics (i.e. a non-linear relationship): transmission was lowest at low temperature (<20 °C); transmission was highest at moderate temperature (20–25 °C) and increased risk of intensity-dependent mortality of 2nd int. host; Transmission at high temperature (>25 °C) was lower than moderate, but mortality of 2nd int. host was highest.	Studer et al. (2010)
Digenean trematodes (numerous) (Platyhelminthes)	*Z. subcarinatus* (Mollusca)	Intertidal ecosystem	Ocean acidification (OA)	Experiment	Parasitic infection increases survivorship compared to controls when affected with increasing acidified water	MacLeod and Poulin (2016a, 2016b)
Philophthamus sp. and *Parorchis* sp. (Platyhelminthes)	*Z. subcarinatus*, *Austrolittorina cincta* (Mollusca)	Intertidal ecosystem	OA	Experiment	More acidic water increased production of parasite free-living stages, but less encysted resting stages. Overall, the effects were parasite species-specific. The results suggest OA can affect the biology and transmission of parasites.	Guilloteau et al. (2016)
M. novaezealandensis (Platyhelminthes)	*P. novizealandiae* (Arthropoda)	Intertidal ecosystem	OA	Experiment	Parasite genotypes ($n = 8$) assessed for infection success and parasite-induced mortality showed little variation between them, suggesting limited evolutionary potential.	Harland et al. (2016)
M. novaezealandensis, *Philophthamus* sp., *Acanthoparyphium* sp. (Platyhelminthes)	*Z. subcarinatus* (Mollusca)	Intertidal ecosystem	OA	Experiment	Parasite infection can increase shell growth and dissolution and reduce shell strength compared to non-infected. Differences were observed between different parasite species.	MacLeod and Poulin (2015b)
M. novaezealandensis (Platyhelminthes)	*P. novizealandiae* (Arthropoda)	Intertidal ecosystem	OA	Experiment	Parasite species transmission from first to second intermediate host increased with increasing acidic conditions	Harland et al. (2015)

(*Continued*)

Table 1. Continued.

Parasite(s) (Phylum)	Host(s)	Principle Area (s)	Risk factor(s)	Method	Results	Reference
M. novaezealandensis, Philophthamus sp., *Galactosomum* sp., *Parorchis* sp. (Platyhelminthes)	*Z. subcarinatus*, *Austrolittorina cincta* (Mollusca)	Intertidal ecosystem	OA	Experiment	Overall, a reduced longevity of free-living cercariae and reduced survival of encysted metacercaria. Some parasites showed higher sensitivity to OA compared to other parasites.	MacLeod and Poulin (2015a)
M. novaezealandensis (Platyhelminthes)	*Z. subcarinatus* (Mollusca)	Intertidal ecosystem	Temperature	Experiment	Increased output and longevity of cercariae at elevated temperature. Variation in sensitivity to temperature observed between genotypes.	Berkhout et al. (2014)
M. novaezealandensis, Philophthamus sp. (Platyhelminthes)	*Z. subcarinatus* (Mollusca)	Intertidal ecosystem	Temperature, salinity and water level	Experiment	Different effects were observed for different trematode species. *Maritrema novaezealandensis* released less cercariae at higher temperature than *Philophthamus* sp.	Koprivnikar and Poulin (2009)
M. novaezealandensis (Platyhelminthes)	Various amphipod species (Arthropoda)	Intertidal ecosystem	Temperature	Experiment and Survey	Higher temperatures strongly affected parasitism of amphipod species. There was a negative relationship between parasitism and amphipod species richness.	Mouritsen et al. (2018)
Philophthamus sp. (Platyhelminthes)	*Z. subcarinatus* (Mollusca)	Intertidal ecosystem	Salinity	Experimental	At reduced salinities (25 or 30 PSU), the emergence of cercariae was lower, it took longer for the cercariae to encyst, and lower overall survival of encysted cercariae	Lei and Poulin (2011)
-	Scleractinian coral, soft coral, zoanthid (Cnidaria)	Coral reefs	Temperature	Survey	Baseline survey of disease at 0.33% prevalence across the atoll but was spatially variable and variable among coral genera.	Williams et al. (2011)
Benedenia seriolae, *Zeuxapta seriolae* (Platyhelminthes)	*Seriola lalandi* (Chordata)	Aquaculture	Temperature	Experiment	Between 17.5 and 21.0 °C both parasites have the ability for rapid multiplication.	Tubbs et al. (2005)
Syndesmis kurakaikina n. sp. (Platyhelminthes)	*Evechinus cloroticus* (Echinodermata)	Subtidal ecosystem	Temperature	Experiment	Non-liner relationship of parasite transmission to temperature. Optimal parasite temperature between 18 and 21.5 °C.	Monnens et al. (2019)

pathways (Anglade and Randhawa 2018; Bennett et al. 2019; Lehnert et al. 2019) that can be incorporated into pelagic food web studies.

Disease affects biodiversity

Parasites can modify habitats through infections of keystone species, engineering the ecosystem to become available for other organisms. The most well-known example involves the interaction mentioned above between cockles (*A. stutchburyi*) and the trematode *C. australis*. In areas with high infection levels, a large number of cockles end up unable to bury and stranded at the sediment surface (Thomas et al. 1998). Shellfish at the surface generate hard substrate in an otherwise soft substrate system (Thomas et al. 1998; Mouritsen and Poulin 2002). Limpets prefer to settle on exposed shell, whereas anemones that use cockle shells as an attachment surface are disadvantaged due to desiccation stress associated with increased exposure during low tide (Thomas et al. 1998). Manipulation of the host by the parasite not only allows the parasite to complete its lifecycle through predation of the bivalve host but generates distinct habitat types that can be occupied by different organisms, facilitating coexistence among competing epibionts and increasing species diversity on mudflats (Mouritsen and Poulin 2005b). A reduction in the burrowing by cockles also reduces bioturbation and increases sediment stabilisation that increases species richness and the density of other benthic macroinvertebrates (Mouritsen and Poulin 2005b). Additionally, because benthic macroinvertebrates feed on algae, infected cockles also indirectly decrease primary production (Mouritsen and Poulin 2006). Therefore, in one system (a mudflat), involving parasitism of one shellfish species by one helminth species (actually a few closely related, cryptic trematode species), the complexity and species richness of that system is dramatically changed.

Foundation species such as seagrasses, corals, kelps and mangroves provide food, shelter and substrate for many species. Keystone species, such as sea stars, produce strong interactions that exert impacts disproportionate to their abundance (Paine 1966). In New Zealand, large disease events in foundation and keystone species are unrecorded, except for the report of seagrass wasting disease caused by an opportunistic fungus, *Labyrinthula zostreae*, in Auckland and Christchurch (see Inglis 2003). Marine ecologists are familiar with the importance of foundation species or keystone species for biodiversity and community structure and from global examples we can appreciate the effects and widespread impacts disease can directly have on ecologically important species that can cascade through an ecosystem (Harvell and Lamb 2020).

Diseases of commercial species

Generally, the degree to which parasites and diseases of aquatic animals have been studied reflect the commercial importance of the host concerned and the severity of infection. There is information on the parasites of eels because they were identified as an aquaculture species in the mid-1970s (Hine 1978); see also for snapper (Sharples and Evans 1995a, 1995b, 1995c, 1995d) and kingfish (Sharp et al. 2003). Parasites can increase mortality rates (Cranfield et al. 2005), reduce flesh quality and marketability (Diggles 2003; Lane et al. 2015), reduce fecundity (Howell 1967), incur extra costs

through treatments (Sharp et al. 2004), and in the case of OIE-notifiable diseases, affect international trade (http://www.stuff.co.nz/business/industries/10542544/Canada-bans-NZ-live-mussel-exports-after-parasite-find). Marine mammals are not commercial in the sense of wild capture fisheries or aquaculture production, instead they support eco-tourism and people value their existence.

Studies on diseases of commercial species includes a larger contribution from government agencies and applied studies, such as the development of diagnostic methods for important aquatic parasites (Gias et al. 2011; Keeling et al. 2012, 2013) or the application of epidemiological models to better manage aquatic diseases (Pande et al. 2015). A core function of Biosecurity New Zealand is to investigate and diagnose aquatic diseases. Consequently, detections are often reported (Lane et al. 2015; Brosnahan et al. 2017b), but host–parasite interactions are rarely investigated because government agencies are limited in their research scope. Benefits in operational research between government and research providers have been exemplified in terrestrial disease events such as the New Zealand Veterinary Journal (2016) Special Issue vol. 64: 1 on the bovine parasites *Theileria orientalis* and *Mycoplasma bovis* (https://www.mpi.govt.nz/news-and-resources/media-releases/study-will-reveal-more-about-m-bovis-impact-on-cattle/), however, there are no comparable examples for aquatic diseases that we are aware of.

Outbreaks of disease in wild populations

Disease and parasites mostly go unnoticed in the aquatic environment until a large disease outbreak that draws attention from scientists and media alike. A herpesvirus that emerged in Australian and New Zealand pilchards in 1995 killed ~70% of the pilchard population (Jones et al. 1997). Rafts of floating dead fish up to 10 km long and piles of fish washing ashore occurred along 5000 km of the Australian coastline and 500 km of the New Zealand coastline (Jones et al. 1997). Affected fish died with clinical signs of respiratory distress and were diagnosed to be infected with a novel herpesvirus, later named pilchard herpesvirus (PHV) (Hyatt et al. 1997). The number of fish impacted across a wide geographic range makes this one of the largest and most well-known aquatic disease outbreaks recorded anywhere in the world (Ben-Horin et al. 2020). Within four months of the first disease event, the dying fish spread east and west across the southern Australian coastline and to New Zealand (Jones et al. 1997). The distinct spread of dying fish associated with the disease event was modelled to support the hypothesis that PHV was introduced into Anxious Bay, South Australia, presumably as imported contaminated feed for South Australia tuna farms (Murray et al. 2001a, 2001b). The origins of mass mortality events are often not easy to identify, but the distinct pattern of spread and the severity of PHV provided an opportunity to apply epidemiological models to identify the most likely point source (Murray et al. 2001a, 2001b), and it has since proved to be a textbook example of aquatic disease epidemiology (see Ben-Horin et al. 2020).

Bonamia exitiosa was first detected in the New Zealand flat oyster *Ostrea chilensis* fishery in Foveaux Strait in 1986 (Dinamani et al. 1987; Berthe and Hine 2003). The New Zealand flat oyster from Foveaux Strait, more commonly known as the Bluff oyster, is a revered seafood delicacy. The Bluff oyster fishery is one of New Zealand's oldest and most iconic fisheries and remains one of the last commercially viable

natural populations of flat oysters in the world (K. Michael pers. comm.). *Bonamia exitiosa* has caused three epizootics in New Zealand from 1986 to 1992, 2000 to 2005 and 2011 to 2015, reducing the population size to ~9% of its pre-disease level (Doonan et al. 1994; Fu et al. 2016; Michael et al. 2020). *Bonamia exitiosa* is the main driver of population size within the fishery (Michael 2020), with outbreaks of disease reoccurring, often corresponding with high oyster densities that increases the chance of parasite transmission (Cranfield et al. 2005). Annual surveys of the Bluff oyster fishery collect data on oyster densities and infection prevalence and intensity to predict summer mortality and the effects of mortality on the new oyster fishing season (Michael et al. 2015). Management of the Bluff oyster fishery is unique in New Zealand as the only one to explicitly consider disease-related mortality in stock assessments. There is merit in including disease into other stock assessment models, for instance O'Connell-Milne et al. (2016a) showed a higher level of trematode parasite infection in harvested cockle *A. stutchburyi* beds than non-harvested beds, potentially affecting the host population dynamics. There are few international examples where disease-related mortalities are incorporated into stock assessments, but when disease is considered it can be telling (Wahle et al. 2009; Wood et al. 2010; Hoenig et al. 2017). For example, for the American lobster *Homarus americanus* fishery, the emergence of epizootic shell disease corresponded very closely to recruitment failure (Wahle et al. 2009).

Reports of beach-cast dying shellfish along New Zealand shorelines to the Ministry for Primary Industries biosecurity hotline (0800 80 99 66) and specimens submitted to the Animal Health Laboratory (AHL) for disease investigation have increased over the past few years (J. Howells pers. comm.). Observations of intracellular bacteria in gill and digestive tissue of submitted shellfish led to the hypothesis that the presence of intracellular bacteria is associated with shellfish health (Howells et al. 2019). DNA sequences generated from infected shellfish and confirmed by *in situ* hybridisation, identified the bacteria as *Endozoicomonas* spp. (Howells et al. 2019), a group of bacteria that has been reported as causing disease in King scallops *Pecten maxima* (Cano et al. 2018). *Endozoicomonas* spp. are ubiquitous in the environment, even in the absence of disease, therefore, *Endozoicomonas* spp. are proposed to be an opportunistic parasite that has its greatest effect on stressed hosts (Hooper et al. 2019). The recurrent reports of shellfish mortality events do not follow a pattern of spread characteristic of infectious disease (H. Lane unpub. data.). However, this does not rule-out infectious disease. Diseases in corals and other ectotherms are linked to changes in the organism microbiome that is driven by environmental factors (Harvell and Lamb 2020). Understanding how a changing environment could affect opportunistic parasites and the different taxa they infect is an important line of future enquiry for New Zealand researchers.

Baseline data for aquatic disease is virtually non-existent, which makes it difficult to forecast, track and mitigate disease emergence (but see Williams et al. 2011). In the absence of baselines, trends analyses have tested whether marine diseases have increased, decreased, or remained stable over time (Ward and Lafferty 2004; Tracy et al. 2019). Although useful for macro-scale trends, they do not allow for specific or timely management of response actions. There is a need for health baseline surveys across taxa and localities. Baseline health surveys supplemented by passive observations of diseased animals, such as Microsporidia *Myospora metanephrops* detected in New Zealand scampi *Metanephrops challengeri* (Stentiford et al. 2010) and *Thelohania* sp. in kōura

Paranephrops planifrons (Jones 1980), fungal lesions in pāua *Haliotis iris* (Grindley et al. 1998), the Myxozoa parasite *Myxobolus equisquamalis* in grey mullet *Mugil cephalus* (Lane et al. 2015) and others (Diggles 2003), allow for the assessment of changes in disease prevalence and intensity more easily and quickly.

Outbreaks of disease in New Zealand marine mammals, sea birds and other vulnerable species may conspire against management efforts to conserve population numbers. For example, the bacterium *Klebsiella pneumoniae* has caused mortality in endangered New Zealand sea lions *Phocarctos hookeri* on the Auckland Islands, causing 58% observed seal pup deaths over a five-year period (Roe et al. 2015). In 2004, 90% of mainland and Stewart Island Hoiho (yellow eyed penguin) *Megadyptes antipodes* chicks were infected with the viral disease avian diptheria, with 50% of them dying from the disease (Alley et al. 2017), and an apicomplexan parasite *Leucocytozoon* sp. caused significant mortality of Hoiho chicks on Stewart Island (Hill 2008). Another apicomplexan parasite, *Toxoplasma gondii*, is an emerging parasite of marine mammals from around the world and has been reported from five marine mammal species in New Zealand (Lehnert et al. 2019), most notably the Hector's dolphin *Cephalorhynchus hectori* where it is hypothesised to cause increased mortality rates (Roe et al. 2015). An unusual reddening syndrome has been observed in IUCN red listed New Zealand lamprey *Geotria australis*, where it has been linked to mortalities although no cause has yet been formally identified (Brosnahan et al. 2019d). Because low population numbers beget low genetic diversity, there is a lower chance of adaptability of these species to tolerate outbreaks of diseases. Regrettably, as low population numbers persist the likelihood of deleterious events such as disease may increase (Acevedo-Whitehouse et al. 2009).

Diseases in farmed fish

The introduction of livestock into New Zealand concomitantly introduced pests and diseases that severely impacted New Zealand's fledgling farming industry and led to the genesis of New Zealand veterinary industry (Tenquist 1990). Despite salmonids being introduced into New Zealand around the same time as livestock (late-1800s), salmonids were introduced free of pests and diseases because they were introduced as eggs, the safest life stage from a disease transmission point-of-view (Anderson 1996). This 'fresh start' coupled with New Zealand's geographic isolation and strict import standards, has meant the New Zealand salmon industry has only experienced minor disease events compared with epidemics that have plagued salmon in their Northern Hemispheric range (Costello 2006).

Virtually all commercial fish farmed in New Zealand are Chinook (=King) salmon *Oncorhynchus tshawytscha* and much of what we know of parasites of farmed fish in New Zealand is from Chinook salmon (but see Diggles 2000; Diggles et al. 2000). Commercial farming of Chinook salmon began in freshwater in the mid-1970s then transitioned to on-grow sea cages in the early 1980s (Knowles 1983). Possibly the most serious salmonid disease detected in New Zealand was the first: whirling disease. Whirling Disease, caused by the Myxozoa parasite *M. cerebralis*, is named after the 'whirling' observed in affected salmon, a symptom of neurological damage from the parasite (Hewitt and Little 1972). Mortality rates for this disease can be >50% for fish <1-year-old (Hoffman 1966). Following the detection of whirling disease, the affected hatchery

was depopulated and decommissioned, and a subsequent survey detected the parasite in farmed rainbow trout *O. mykiss* near Christchurch in 1980 (Boustead 1982). Whirling disease was ultimately detected in six establishments across the South Island (Anderson 1996), but has not been detected for over 20 years, which is attributable to an improvement in hatchery facilities (Diggles et al. 2002a). Annual surveillance for whirling disease remains for those New Zealand salmon farms exporting salmon for human consumption to Australia where it is considered exotic (refer to Surveillance Annual Report for testing statistics for salmon export to Australia, e.g. Surveillance (2019) vol. 46 (3) pg. 21).

A 1996 review of parasites detected in New Zealand salmonids presents a short list (Anderson 1996) and very little has been added to it since (Table 2). New Zealand salmon are free of all serious diseases (Boustead et al. 1993). For example, only one virus has been detected in New Zealand salmonids, an aquabirnavirus (Tisdall and Phipps 1987). The aquabirnavirus was first isolated in 1985 during routine sampling of healthy sea-run wild salmon returning up the Rakaia River (Tisdall and Phipps 1987). Initially all aquabirnaviruses were referred to as infectious pancreatic necrosis virus (IPNV), a serious salmonid disease, but serological analyses showed that the New Zealand aquabirnavirus is serologically different from IPNV (Tisdall and Phipps 1987). Consequently, IPNV is exotic to New Zealand. Viruses are some of the most abundant life forms in the aquatic environment (Suttle 2007) that can infect all cellular life forms, so it is inevitable more viruses will be detected in New Zealand fishes, especially with increasing research effort on salmonids and the advent of high throughput sequencing (HTS) (e.g. Mordecai et al. 2019). The challenge lies in understanding the importance of newly discovered viruses to fish health (Mordecai et al. 2019).

Bacteria are ubiquitous in the marine environment with only a small proportion being a primary cause of disease (Austin 2005). The chance of bacterial disease increases with stress caused by excessive handling, crowding or poor water quality (i.e. opportunistic) (e.g. Brosnahan et al. 2017b). *Flavobacterium columnare, F. branchiophilum* and *F. psychrophilum*, *Aeromonas hydrophila, Pseudomonas fluorescens, Nocardia* sp. and *Vibrio* sp. have been associated with bacterial disease in fish hatcheries and on-

Table 2. List of parasites recorded for New Zealand salmon for the period 1977–2020. In bold are parasites detected in the period 1997–2020 following Anderson (1996). Note that aquabirnavirus was originally reported as infectious pancreatic necrosis virus.

Parasite	1977–2020	Reference
Virus	aquabirnavirus	Tisdall and Phipps (1987)
Bacteria	*Flexibacter* (= *Flavobacterium*) sp.	Anderson (1996)
	Aeromonas sp.	Diggles et al. (2002a)
	Hafnia alvei	Anderson (1996)
	Streptococus sp.	Anderson (1996)
	Vibrio sp.	Anderson (1996); Diggles et al. (2002a)
	Yersinia ruckeri O1b	Anderson et al. (1994)
	Flavobacterium psychrophilum	Bingham (2013)
	NZ-RLO-1 -2 -3	Brosnahan et al. (2017a); Brosnahan et al. (2019c)
	Tenacibaculum maritimum	Brosnahan et al. (2019c)
	Lactococcus garvieae	EPA (2020)
	***Nocardia* sp.**	Brosnahan et al. (2017b)
Protozoa	*Chilodenella* sp.	Anderson (1996)
	Icthyophthirus multifilis	Anderson (1996)
	Myxobolus cerebralis	Hewitt and Little (1972)
	Paramoeba sp.	Anderson 1996; Diggles et al. (2002a)

growing facilities (Diggles et al. 2000; Diggles et al. 2002a; Bingham 2013; Brosnahan et al. 2017b). Probably the most significant bacterial disease of New Zealand salmon is caused by *Yersinia ruckeri*. *Yersinia ruckeri* was first isolated in 1989 from dying farmed salmon in Canterbury (Anderson 1996) then later isolated at two other South Island locations (Anderson et al. 1994). *Yersinia ruckeri* serotype O1a (Hagerman strain) is the cause of enteric redmouth disease, a major salmon disease in the Northern Hemisphere, whereas New Zealand *Y. ruckeri* is serotype O1b, a less virulent serotype (Barnes et al. 2016). A comparison of genomes between global isolates of *Y. ruckeri* showed that serotype O1b is endemic in New Zealand and Australia and was not concomitantly introduced with Northern Hemisphere salmon (Barnes et al. 2016). *Yersinia ruckeri* can infect other fish, including sole, turbot and eel (Carson and Wilson 2009) and its status in other wild New Zealand fish, especially salmonids, is not currently known. Other disease-causing bacteria detected in New Zealand salmon include New Zealand rickettsia-like organism strain-type-1 (NZ-RLO-1), -2, and -3, as well as *Tenacibaculum maritimum* (Brosnahan et al. 2019c). The NZ-RLOs were identified for the first time in New Zealand and *T. maritimum* confirmed for the first time during higher mortality levels during summer months. Disease trials carried out on NZ-RLO-1 and NZ-RLO-2 demonstrated their ability to cause disease in salmon smolt (Brosnahan et al. 2019b; Brosnahan 2020).

Vaccinating farmed fish

Vaccines are well-known as a useful tool for managing disease, including in farmed fish populations. Shellfish do not have an adaptive immune system so are not suitable for vaccination strategies (Guo and Ford 2016). New Zealand produces almost 100% of the world's farmed Chinook salmon and has done so without the need for vaccines, unlike the Atlantic salmon industry that has used vaccines to control many viruses and bacteria (see review by Ma et al. 2019). The New Zealand salmon industry has some interest in developing vaccines for different parasites because they are a good preventative measure for disease control compared to antibiotics which is a reactive measure that can lead to the evolution of antibiotic resistance in parasites (http://www.stuff.co.nz/marlborough-express/editors-picks/8599220/Funding-for-salmon-research). Effective vaccinations require a thorough understanding of the pathogen (i.e. the antigen) and the host's ability to mount an immune response to that antigen (Ma et al. 2019). The efficacy of a vaccine ideally needs to be demonstrated *in vitro* and *in vivo*. Globally available commercial vaccines for many salmon parasites may not be effective for New Zealand salmon. For example, NZ-RLOs of New Zealand salmon are different to *P. salmonis* infecting Atlantic salmon from other countries (Brosnahan et al. 2019c). Although a vaccine exists for *P. salmonis* there is no guarantee of its effectiveness in a New Zealand context. Parasites that are geographically separate evolve independently leading to distinct antigens (see Barnes et al. 2016 for *Y. ruckeri*). There are fewer studies on the immunology of Chinook salmon compared to Atlantic salmon *Salmo salar*. A Web of Science search result using the key words immun* AND Atlantic salmon returned >3,000 results whereas immune* AND Chinook salmon returned 365 results. As a result, the likelihood of antigenically different parasites affecting New Zealand salmon and an unknown host immune system means that bespoke vaccines

probably need to be developed. The development of vaccines is necessary, but the biosecurity also needs to be considered, because vaccines can be a biosecurity risk: (1) vaccinated and non-vaccinated fish may not be able to be differentiated during diagnostic tests, increasing the risk of incorrect results (Jansen et al. 2019); (2) vaccines for exotic organisms can change a country's 'disease-free' status, e.g. New Zealand could no longer declare freedom for IPNV if an IPNV vaccine gets implemented; and (3) vaccines can be a vector for introducing non-native parasites, for example, infectious bursal disease, an important chicken virus was introduced into New Zealand via an imported commercial vaccine (Ryan et al. 2000). Currently, New Zealand does not have the data nor capacity to develop fish vaccines or trial their efficacy but remains an important area of future work requiring both empirical research and infrastructure capital investment (see section 5.1).

Diseases of farmed invertebrates

The Pacific oyster *Magallana gigas* is one of New Zealand's top-three aquaculture species by volume and value and is unique among them in being the only species to be regularly affected by a known disease. In the mid-1980s Pacific oysters were considered largely free of parasites (Dinamani 1986) (but see Handley and Bergquist 1997), however, an incursion of a microvariant genotype of ostreid herpesvirus OsHV-1 (OsHV-1 µvar) changed that. In 2009 OsHV-1 affected over 70% of oyster growers in northern New Zealand, including up to 100% mortality in spat and between 5% and 60% mortality in adult oysters (Bingham et al. 2013). Herpes-like infections have been reported from New Zealand Pacific oysters and flat oysters (Hine et al. 1992, 1998), but it was the emergence of the problematic herpesvirus microvariants that caused severe disease and production losses in Pacific oysters around the world since 2008 (Segarra et al. 2010; Jenkins et al. 2013). Epizootics caused by OsHV-1 occur during spring and summer when the seawater is at its warmest (Renault et al. 2014) and mortality is usually higher in spat than adult oysters (Paul-Pont et al. 2014). Interactions between OsHV-1 and other parasites, particularly bacteria *Vibrio* spp. (Pernet et al. 2012; Keeling et al. 2014), are also likely to contribute to disease outbreaks of Pacific oysters. de Lorgeril et al. (2018) demonstrated that OsHV-1 immune-suppressed Pacific oysters lead to a fatal disease from opportunistic bacteria. Although progress in selective breeding has reduced mortality rates from OsHV-1, gaps remain in our knowledge on disease risk factors. For example, the ability of the virus to persist in the environment and the role of other fauna as infection reservoirs are poorly understood (Pernet et al. 2016; Bookelaar et al. 2020).

New Zealand's main aquaculture species by production and value is green-lipped mussels (GLM) *Perna canaliculus* (Castinel et al. 2019). GLM are free from significant production diseases (Castinel et al 2019). Common parasites, the New Zealand pea crab *Nepinnotheres novaezelandiae* and digenean trematode *Cercaria cercaria* rarely induce mortalities although their presence can affect product quality (Hickman 1978; Trottier et al. 2012). Bacterial diseases of hatchery-reared GLM intermittently cause mortality events but are often the result of unsanitary hatchery practice (Kesarcodi-Watson et al. 2009). Jones et al. (1996) identified virus-like particles associated with significant GLM spat loss, but the virus has never been identified.

The protist *Perkinsus olseni* is an important parasite of shellfish and is notifiable to the OIE because of the severity of disease it can cause (e.g. Shimokawa et al. 2010). *Perkinsus olseni* was first detected in New Zealand in wedge shells *Macomona liliana* from Kaipara Harbour in 1999 (Hine and Diggles 2002) and has never been associated with any large mortality event in New Zealand. A health survey carried out in 2001 detected *P. olseni* only in northern New Zealand cockles *A. stutchburyi* (Hine and Diggles 2002). Notably, Hine and Diggles (2002) reported that GLM, scallops *Pecten novaezelandiae*, and pāua *Haliotis iris* were refractory to *P. olseni* infection based on thousands of historically negative samples. Since 2014, *P. olseni* has been detected in GLM, scallops and pāua and has extended its range to the South Island (H. Lane pers. obs.). The emergence of *P. olseni* in commercially important species such as GLM is apparent and further investigation is needed to assess the risk of this parasite to future production. The lifecycle of *P. olseni* is controlled by temperature, peaking during warmer seawater temperatures (Villalba et al. 2005), suggesting infections may continue to become more frequent or severe, or it may continue to extend its range further south. Empirical data on how climate change will affect the pathobiology of *P. oseni* in New Zealand shellfish is an important research gap.

Pāua like other New Zealand aquaculture species are affected by few production diseases. The most important parasite reported from pāua is *P. olseni* (McDonald 2014). An unnamed haplosporidian parasite was reported and its disease progression documented (Diggles et al. 2002b), but has not been reported since. The OIE notifiable abalone herpesvirus, the causative agent of abalone viral ganglioneuritis, failed to establish an infection in pāua during a disease trial carried out in Australia, while blacklip (*Haliotis rubra*) x greenlip *(H. laevigata*) abalone hybrids experience >70% mortality (Corbeil et al. 2017). Abalone herpesvirus first emerged in Australia and spread throughout land-based farms and wild abalone populations, causing high mortalities and considerable financial losses (Hooper et al. 2007). The apparent disease resistance of New Zealand pāua to abalone herpesvirus is unique among the Haliotids, although the mechanism behind it is unknown (Corbeil et al. 2017). New Zealand endemic species like pāua and GLM are not farmed outside of their natural range, so have not been exposed to new parasites, making it difficult to assess their susceptibility to disease. We are familiar with native macrofauna, we are less so with native microorganisms (e.g. Barnes et al. 2016), and there may be a long evolutionary history between pāua and abalone herpesvirus moderating the severity of disease.

Diagnostic testing

Diagnostic testing is critical for accurately detecting parasites and demonstrating proof of freedom from specific diseases (Gias et al. 2011; Keeling et al. 2012, 2013). Histopathology is a classical diagnostic method that provides the ability to detect parasite infections, lesions and the nutritional and reproductive condition of the host (Lane et al. 2016). Other classical methods such as transmission electron microscopy (TEM), scanning electron microscopy (SEM), bacterial culture, immunological assays like immunohistochemistry (IHC) and ELISAs, and viral cell culture are still used in diagnostic testing with many still considered the gold standard method for diagnosis. For instance, the gold standard test for diagnosing OIE-notifiable *P. olseni* and *P. marinus* is a culture method

developed in the 1950s (Ray 1952). Refer to the Manual of Diagnostic Tests for Aquatic Animals for recommended diagnostic methods for OIE notifiable diseases (https://www.oie.int/standard-setting/aquatic-manual/)

The advent of PCR and HTS has unquestionably improved speed, sensitivity and specificity of testing. An inherent shortcoming of PCR is that it only detects nucleic acid and not necessarily a viable organism. However, Brosnahan et al. (2019a) developed and validated a PCR method capable of detecting viable nucleic acid (vPCR) of *Y. ruckeri* and *T. maritimum* and assessed its potential for the detection of aquabirnavirus. Briefly, vPCR detects nucleic acid within an intact cell membrane and therefore derived from a presumably viable organism that is capable of establishing a new infection (Brosnahan et al. 2019a). The implementation of portable hand-held next generation DNA sequencers, such as MinION (Oxford Nanopore Technologies), has demonstrated the potential of HTS in disease diagnostics, particularly outside of a laboratory setting (http://blog.worldfishcenter.org/2020/03/lab-in-a-backpack-rapid-genomic-detection-to-revolutionize-control-of-disease-outbreaks-in-fish-farming/). However, HTS in disease diagnostics requires more validation before its wider application, including assessing its rate of false-positive and false-negatives (OIE 2019). Diagnostic validation for any diagnostic test is labour intensive and expensive, but it is vital for correctly assessing false-positive and false-negative results. False-positives can unnecessarily disrupt trade through non-tariff trade restrictions or unnecessarily culling of farmed stock for biosecurity control (e.g. Qviller et al. 2020), while false-negatives can miss infections theoretically allowing diseases to persist and spread making them harder to control and/or eradicate. The Laboratories for Emergency Animal Disease Diagnosis and Response (LEADDR) (2019) in Australia has released guidelines on validation for HTS in addition to existing guidelines for the development and optimisation of nucleic acid detection assays (OIE 2014).

Although HTS allows us to understand microbial communities better than ever before, it can prove to be a quagmire when a single sequence of an exotic parasite is produced that cannot to be confirmed by other diagnostic tests because of the higher analytical sensitivity of HTS (e.g. Darling et al. 2020). This is a hurdle to overcome in the application of environmental DNA (eDNA) in disease testing. eDNA has been investigated for marine pest surveillance in New Zealand (Wood et al. 2018) but is yet to be thoroughly investigated for disease surveillance. The use of eDNA for parasite detection has been successfully demonstrated overseas (e.g. Merou et al. 2020). eDNA samples are usually complex assemblages of organisms and organic matter that can affect downstream processes, reducing test sensitivity (see Wood et al. 2019 for a comparison of analytical sensitivty for different molecular eDNA methods). The implementation of eDNA in disease surveillance will allow for greater understanding of dispersal of different parasites as well as their persistence in the environment (Merou et al. 2020). Early detection is a critical outcome of disease surveillance and eDNA remains a method with high potential that needs further exploration.

Metabarcoding and metagenomics of eDNA samples are broad-stroke approaches providing a snapshot of species diversity and relative abundance. This 'catch-all' method can saturate users with large amounts data, particularly when only a few parasites are of interest. AmpliSeq Community Panels (ThermoFisher) allow for the detection of specific genetic variations of different human diseases or antimicrobial resistant bacteria, removing all superfluous sequencing data and targeting pre-defined areas of interest.

Existing AmpliSeq Community Panels are all for human diseases, but bespoke panels can be designed that could be applied for animal disease surveillance. Sequencing data (C. Brosnahan unpub. data) from shellfish samples using a Pan-Bacterial Community Panel demonstrated its utility, but other trials will need to be undertaken before specific HTS for aquatic diseases could be implemented in disease surveillance or disease diagnostics.

The Anthropocene and aquatic disease

The Anthropocene will see the largest changes to ecosystems through human-driven impacts. Parasites and disease are natural parts of normal functioning ecosystems, but climate change, invasive species and pollution can alter host and parasite ecology and the co-evolved interactions between them, changing disease dynamics, that can lead to ecosystem or socioeconomic changes (Marcogliese 2008) (Figure 3). For example, the emergence of sea star wasting disease (SSWD) of North American sea stars beginning in 2011 was likely fuelled by warm water temperature anomalies (Hewson et al. 2018). Infected sea stars developed lesions in the dermis that dissolved the tissue from the outside in, leading to arm detachment from the central disc as individuals died. The SSWD was linked to a densovirus (Hewson et al. 2014) and caused the precipitous decline in at least five species of sea stars, including *Pisaster ochraceus* a keystone species of the rocky intertidal (Harvell and Lamb 2020). The outbreak of SSWD significantly changed the seascape of the Pacific coastline of North America, shifting kelp forests to urchin barrens, and releasing previously regulated populations of bivalves from predation (Harvell and Lamb 2020). The outbreak of SSWD demonstrated how the emergence of a novel parasite through environmental changes can so greatly impact an ecosystem.

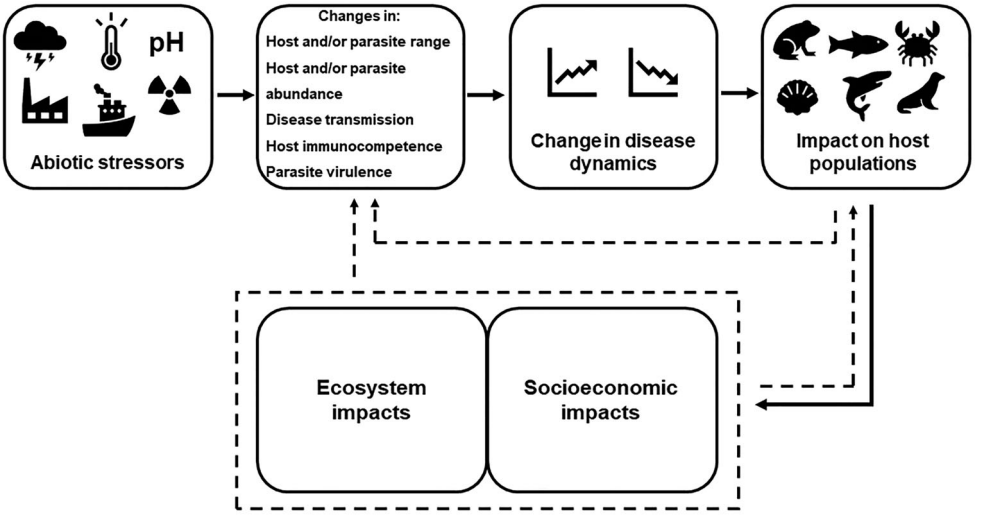

Figure 3. Schematic of how abiotic stressors can cascade through a system resulting in ecosystem and/or socioeconomic impacts. Dashed lines represent feedback interactions.

We know that parasites affect the functioning of food webs, but alterations to food webs can affect parasites that can increase or decrease disease risk. Impacts on hosts populations from disease, fishing, or other perturbations can feedback to alter parasite ecology and disease dynamics (Wood et al. 2010) (Figure 3). For instance, the California sea urchin population exploded following the extirpation of lobsters from overfishing, triggering a phase shift from a predominant kelp-based system to an urchin barren (Lafferty 2004). However, as the population density of sea urchins increased so too did the transmission of density-dependent bacteria, resulting in an outbreak of disease in sea urchins returning urchin barrens back to a more kelp-dominated system (Behrens and Lafferty 2004; Lafferty 2004).

Effect of climate change on parasitic diseases

All hosts and parasites have a temperature optimum, enabling hosts and parasites to extend geographic ranges if favourable conditions emerge outside of their historical native range. For example, the oyster parasite *Perkinsus marinus* has extended its range north by over 500 km from Chesapeake Bay into Maine along the Atlantic coastline of North America as a result of warming seawater temperatures (Pecher et al. 2008). Alternatively, environmental changes can act as stressors, compromising host immunity, increasing the number of susceptible hosts in a population and increasing the chance of disease, especially from opportunistic parasites (Sniesko 1974).

Warming water is a commonly studied stressor of disease dynamics (see Table 1). Empirical data from host–parasite systems demonstrate some general patterns of how warmer water will affect aquatic disease. For instance, warmer seawater temperature is expected to increase parasite transmission, particularly for digenean trematodes. The emergence of trematode infective stages (cercariae) from their mollusc first intermediate host can be many times higher with an increase of just a few °C in seawater temperature (Poulin 2006; Thieltges and Rick 2006; Studer et al. 2010). Higher transmission of free-living stages of digenean trematodes will disproportionately affect intermediate hosts of their lifecycle. For instance, on New Zealand mudflats, greater rates of transmission of the trematode *Matrima novaezealandensis* from the snail first intermediate host *Zeacumantus subcarinatus* to the amphipod second intermediate host *Paracalliope novizealandiae* will result in a higher parasite load in the amphipod leading to higher mortality rates of the intermediate hosts (Studer and Poulin 2013). The relationship between disease transmission and seawater temperature is non-linear, with parasite transmission reducing beyond an optimum seawater temperature (Thieltges and Rick 2006), suggesting that predictions of future disease dynamics will be challenging without empirical data. Temperature range as opposed to maximum temperature experienced has been demonstrated as important to aquatic disease (Ben-Horin et al. 2013), which is an important consideration with an increase in report of marine heatwaves and warm seawater temperature pulses (Claar and Wood 2020).

Ocean acidification is predicted to negatively impact many marine species (Law et al. 2018), so it is conceivable that parasites will act with ocean acidification to amplify those negative impacts. However, disease impacts on calcifying hosts in more acidic seawater can be counterintuitive. MacLeod and Poulin (2016b) found that infected New Zealand snails *Z. subcarinatus* had higher survivorship than uninfected snails under increasing

acidic seawater. The mechanism behind the increased survival is unknown, but it is surmised that because of parasitic castration of the snail, host energy is redirected from gametogenesis to resilience to ocean acidification (MacLeod and Poulin 2016b). Importantly, had MacLeod and Poulin (2016b) not considered parasitic infection in their survivorship study, an important host–parasite interaction would have been overlooked possibly leading to an inaccurate conclusion on species tolerance to ocean acidification. It would be relatively straightforward to add a disease perspective to existing mesocosm studies and in doing so the results could be telling (MacLeod and Poulin 2016b).

Many of the studies on climate change and disease in New Zealand include the digenean trematode *M. novaezealandensis* and the snail host *Z. subcarinatus* (Table 1). Poulin et al. (2016) cautioned against a reliance on a few model systems to understand disease ecology. Considering some of the interspecific and genotype-based intraspecific variation observed across different studies (e.g. Koprivnikar and Poulin 2009; Berkhout et al. 2014), our overall understanding of parasite–host interactions based on a few host–parasite models is limited. For example, the accuracy and utility of disease forecasting is grounded in part on some understanding of the host–parasite interaction under different environmental parameters. It is logistically impossible to test all host–parasite systems, however selecting other host–parasite models across a range of taxa will provide a greater breadth and depth of understanding. Indeed, it is expected that parasites of primary producing marine algae (Safi and Gutierrez-Rodriguez 2017) will have different interactions to parasites of grazing herbivores (Monnens et al. 2019), each affecting the ecology of a system in different ways.

The effect of warming water on the health of aquaculture species, in particular New Zealand salmon, is well publicised (https://www.stuff.co.nz/business/farming/112404171/climate-change-hits-nz-king-salmon-fish-stocks-and-forecast-earnings) but few empirical data for any commercial species are available to accurately assess the true risk (Table 1). A disease trial carried out at the high containment aquarium at New Zealand's Animal Health Laboratory (AHL) serendipitously demonstrated how important water temperature could be to disease progression in salmon. Salmon smolt were inoculated with New Zealand rickettsia-like organism strain-type 2 (NZ-RLO-2) (Brosnahan et al. 2019b) and were held for 30 days to monitor disease progression. At day 22, the aquarium air conditioning failed, causing a spike in water temperature. Fish that had been infected with the bacteria showed a steep increase in cumulative mortality following the increase in water temperature, whereas uninfected control fish experienced no increase in mortality rate (see Brosnahan et al. 2019b). The findings of this study should provide motivation for similar future studies, especially with increasing reports of 'summer mortality syndromes' of aquaculture species.

Outbreaks of disease from OsHV-1 is associated with warmer seawater (Keeling et al. 2014; de Kantzow et al. 2016). Therefore, the continual warming of sea water through climate change may contribute to an increase in the number of disease events from this virus. Whether selective breeding for disease tolerance to OsHV-1 in Pacific oysters is an adequate line of defence remains to be thoroughly tested in New Zealand conditions (Gutierrez et al. 2020). Further, whether the new host and geographic range of *P. olseni* is a consequence of warming waters is an important question that remains to be tested. The dearth of studies addressing these types of questions in New Zealand is obvious. Empirical data is needed because the effect of temperature on

disease is not uniform; for example, the haplosporidian *B. ostreae* prefers cooler more saline waters (Arzul et al. 2009), whereas incidences of bacterial infection across multiple aquaculture species have increased with warmer waters (Reverter et al. 2020). The collection of empirical data of host–parasite interactions under different environmental conditions allows for more accurate forecasting of disease risks and a clearer prioritisation of future research effort to mitigate those risks.

Experimental studies on aquaculture species are accompanied with an extra level of regulatory complexity because often research into diseases of commercial species is under strict biosecure management to avoid the spread of parasites that could affect domestic and international trade and wild ecosystems. The spread of disease from high-density land-based facilities can have severe consequences for wild populations. For example, in 2005 a herpesvirus that causes abalone viral ganglioneuritis first emerged in an aquaculture facility in Australia. In 2006, the disease emerged in wild populations near farms, where it greatly impacted fisheries production (Hooper et al. 2007). The biosecure facility at AHL is the only facility in New Zealand capable of testing disease interactions for unwanted or emerging pathogens. New Zealand needs to develop other biosecure facilities that have the capacity for disease trials while maintain strict biosecurity procedures. The shortage of appropriate facilities may explain the limited number of disease studies for aquaculture species. Indeed, we can learn much from overseas studies on important diseases like OsHV-1 in Pacific oysters (Renault et al. 2014), but this falls short of studies on endemic species such as GLM that are also considered a marine pest in many overseas jurisdictions. Developing biosecure facilities will take time and significant capital investment, but it is necessary infrastructure for future aquatic disease studies.

Effect of pollution on parasitic diseases

Pollutants from terrestrial run-off can introduce new parasites to the marine environment that can affect human and animal health. Most people are familiar with the banning of shellfish gathering following a period of heavy rain to reduce infections of enteric parasites introduced from stormwater. Fish parasites, like the bacterium *Edwardsiella tarda*, the cause of piscine septicaemia, have been reported overseas as introduced to the sea from wastewater (Mohanty and Sahoo 2007). *Toxoplasma gondii* is a common terrestrial parasite that uses felines as a definitive host and recently *T. gondii* has been identified in GLM (Coupe et al. 2018) and five New Zealand marine mammals (Lehnert et al. 2019), most notably Hector's dolphins (Roe et al. 2013). Globally, the detection of *T. gondii* has sparked concerns about the role *T. gondii* has in the health of different aquatic animals, particularly the endangered southern sea otter (*Enhydra lutris nereis*) (Miller et al. 2002). *T. gondii* is shed as environmentally robust oocysts from cat faeces that presumably make their way into the marine environment via wastewater and whether *T. gondii* infects marine mammals through consumption of infected shellfish or through passive dispersal of oocysts is not clear (Lafferty 2015). Although *T. gondii* is likely to cause some disease in southern sea otters, it is thought that infections are quite natural and not necessarily a consequence of anthropogenic pollution. Lafferty (2015) summarises that *T. gondii* is as prevalent in areas of low human-population densities as high human population densities, suggesting wild native cats are just as great a

source of parasites as domestic house cats. Unlike North America, marine mammals in New Zealand have evolved independently of terrestrial mammals, suggesting New Zealand marine mammals may be at greater risk from terrestrial diseases (e.g. Fenwick et al. 2004).

Globally, studies on the interactions between pollution and disease dynamics are scarce (Bojko et al. 2020) and it is similarly the case in New Zealand, where only five studies have been conducted in either freshwater or marine systems (Table 3). Pollution can be biological (Coupe et al. 2018), chemical (Kelly et al. 2010; Hock and Poulin 2012), or physical (Hofmann et al. 2016), and it is expected that each can affect host physiology, parasite multiplication or infectivity to a lesser or greater extent. Coastal fringe habitats like kelp forests, sea grasses and reefs are exposed to greater amounts of pollution than pelagic habitats, increasing their disease risk. Biogenic habitats such as kelp forests and sea grasses provide habitat for many organisms and can also reduce parasites from the water column (Lamb et al. 2017), therefore, their extirpation will have far-reaching ecological consequences, including human health impacts (Lamb et al. 2017). An outbreak of wasting disease of eelgrass *Zostrea marina* caused >90% loss in seagrass meadows along the Atlantic coastlines of North America and Europe in the 1930s (Short et al. 1988). The causative agent was an opportunistic fungus, *Labryinthula zostreae*, that has also been reported from New Zealand (Inglis 2003). Seagrass declines in New Zealand have been linked to anthropogenic pollution (Inglis 2003) and the continued input of pollutants may see a re-emergence of sea grass wasting disease. Records of parasites in seaweed in New Zealand are rare but some have been reported (Preuss and Zuccarello 2018) and how these parasites may interact with current abiotic threats is unknown.

As the terrestrial and marine worlds become increasingly connected, the emergence of aquatic diseases and syndromes could become more likely (Sanderson and Alexander 2020). Several important prawn viruses such as white spot syndrome virus (WSSV) and Taura syndrome virus potentially emerged as a result of insects co-inhabiting terrestrial pond environments (Walker and Winton 2010). A mysterious syndrome called tail fan necrosis (TFN) has emerged in New Zealand crayfish *Jasus edwardsii.* TFN includes the blackening and eroding of the crayfish tail fan, reducing saleability. The cause of TFN is unknown but is probably multifactorial (Zha et al. 2018a, 2018b, 2019). Novel interactions between crayfish and terrestrial contaminants has been postulated as a hypothesis for the emergence of TFN but has yet to be investigated. The concept of One Health interlinks human health and the emergence of disease and the wider environmental health, particularly terrestrial ecosystems. As the marine world and terrestrial world become more connected, it is important to include marine ecosystems into the One Health concept (Sweet et al. 2020).

Parasitic invasive species

Four introduced parasites to New Zealand demonstrate the negative impact non-native parasites can have (Table 4). The introduction of *B. ostreae* to populations of *O. chilensis* led to the depopulation of virtually all flat oyster farms from New Zealand, effectively ending an industry (https://www.mpi.govt.nz/news-and-resources/media-releases/mpi-confirms-flat-oyster-stocks-removal-from-big-glory-bay/). Large disease events can–but not always (Harrison and Duggan 2019)–follow the introduction of non-native

Table 3. Studies on factors associated with pollution and aquatic disease in New Zealand.

Parasite(s) (phylum)	Host(s)	Risk factor	Pollution Type	Result	Reference
Salmonella serotypes (Proteobacteria)	*Phocarctos hookeri* (Chordata)	Run-off	Biological	*Salmonella* serotypes *S.* Cerro and *S.* Newport were isolated from New Zealand sea lions and feral pigs on Auckland Islands, suggesting contamination of marine mammals from run-off from terrestrial animals.	Fenwick et al. (2004)
Toxoplasma gondii (Apicomplexa) *Giardia duodenlais* (Metamonada)	*Perna canaliculus* (Mollusca)	Run-off	Biological	Report of terrestrial parasites in wild *P. canaliculus* at river mouths.	Coupe et al. (2018)
Multiple species	*Potamopyrgus antipodarum* (Mollusca)	Farming and water abstraction	Physical and Chemical	The most common parasite *Microphallus* was most prevalent at high farming intensity and low water abstraction. Antagonising interactions between two agricultural stressors.	Hofmann et al. (2016)
Telogaster opisthorchis (Platyhelminthes)	*Galaxias anomalus, Potamopyrgus antipodarum* (Chordata)	Herbicide	Chemical	Glyphosate (herbicide) can synergistically act on parasites and hosts and can increase disease risk.	Kelly et al. (2010)
Coitocaecum parvum, Apatemon sp., an undescribed renicolid species (Platyhelminthes)	*Potamopyrgus antipodarum* (Mollusca)	Herbicide	Chemical	Increased glyphosate (herbicide) increased emergence of cercariae across all three parasites. Higher survivorship observed in renicolid species. Clear interaction between glyphosate concentrations and parasitism.	Hock and Poulin (2012)

Table 4. Significant introductions of non-native parasites to New Zealand, the hosts infected and the ecological and socioeconomic impact of their introduction. Non-native hosts are shown by **bold** type.

			Impact		
Parasite (phylum)	Host (phylum)	Introduction pathway	Ecological	Commercial	Reference
Ostreid herpesvirus genotype 1 microvariant (OsHV-1 µvar) (Herpesviridae)	***Magallana gigas*** (Mollusca)	Unknown. *M. gigas* was introduced to New Zealand in the 1960s and OsHV-1 µvar emerged in 2008. Possibly subsequently introduced through infected *M. gigas*	The ecological impacts on wild *M. gigas* is unknown nor whether wild *M. gigas* may act as a disease reservoir to aquaculture populations	100% mortally of spat and then up to 70% loss of adult oysters. Ongoing seasonal issues, particularly with warming water.	Bingham et al. (2013); Keeling et al. (2014); Pande et al. (2015)
Pilchard herpesvirus (Herpesviridae)	*Sardinops sadax* (Chordata)	Introduced to Australia via contaminated fish feed and then transmission from pilchard to pilchard from Australia to New Zealand. Understood to have been introduced to Australia independent of host.	Decreased reproductive fitness and population size of predators, e.g. little penguins *Eudyptula minor*. Range expansion of competitors, Australian anchovy *Engraulis australis*.	70% loss of fishing stock, affecting commercial fisheries in the area.	Chiaradia et al. (2010); Jones et al. (1997); Ward (2001); Whittington et al. (1997)
Bonamia ostreae **(Haplosporidia)**	*Ostrea chilensis* (Mollusca)	Unknown, but possibly through infected *O. chilensis* or *O. edulis* or an unknown alternative host.	Unknown, but likely to be negligible because *B. ostreae* was detected in low numbers of wild *O. chilensis.*	Removal of all flat oyster farms from New Zealand, effectively shutting down an industry. Active surveillance is ongoing.	Lane et al. (2018); Lane and Jones (2020); Lane et al. (2016)
Myxobolus cerebralis **(Cnidaria)**	***Oncorhynchus tshawytscha, O. mykiss*** (Chordata)	Imported contaminated fish feed. Understood to have been introduced independent of host.	Unknown status in wild populations.	Depopulation and subsequent closure of affected hatcheries. Active surveillance is ongoing.	Hewitt and Little (1972)

parasites (see Table 4). Less conspicuous parasite introductions are probably more common. Pagenkopp Lohan et al. (2020) argue that because of the microscopic size of parasites, they are often overlooked in species introduction and their detection is usually left to chance, as in the case of the cestode parasite *Ligula* sp. (see Lagrue et al. 2018) and the digenean trematode *Transversotrema patialense* (see Harrison and Duggan 2019), both introduced to New Zealand freshwater ecosystems. Identifying non-native parasites is complicated by slow rates of parasite species descriptions (Poulin et al. 2016) and poor documentation of historical natural ranges of parasites, although checklists are useful 'working inventories' of New Zealand parasites (e.g. Lehnert et al. 2019). Regardless, species-rich and abundant microbial communities detected in high-risk biosecurity vectors like ship ballast water (Pagenkopp Lohan et al. 2016), suggest parasite introductions is an ever-present risk that remains a large gap in biosecurity science and invasion ecology.

The constant movement of consignments across New Zealand's border is a high-risk pathway for parasite introduction. Parasites can be introduced with a host or independently of hosts, as either dispersal stages or dormant stages, meaning there are a variety of routes a parasite introduction may take. The ornamental fish trade is a high-risk pathway for introducing parasites into New Zealand (McDowall 2004), with exotic parasites often detected in post-border quarantine consignments (Edwards and Hine 1974). The trade of live animals for aquaculture is a well-known pathway for parasite introductions overseas (Elston et al. 1986), similarly for animals intended for human consumption, such as the introduction of OIE notifiable WSSV into Australia via the importation of uncooked prawns (Scott-Orr et al. 2017) and the detection of *B. ostreae* in imported consignments of frozen *O. edulis* into China (Feng et al. 2013). Because of its relative geographic isolation, New Zealand has had comparatively fewer introductions than, say, European countries that share multiple land borders and water ways (Peeler et al. 2011). In spite of New Zealand's geographical advantage as well as other aquatic biosecurity measures (e.g. the Craft Risk Management Standard), parasite introductions still occur via pathways that are not always obvious (e.g. Lane and Jones 2020) (see Table 4). The importance of trade and tourism to New Zealand coupled with aquatic disease events in other jurisdictions means parasite introductions are an ongoing threat. As we write these lines, New Zealand has installed strict biosecurity measures to prevent the introduction of the virus that causes COVID-19 highlighting more tangibly the connection between disease, biosecurity and globalisation.

The ability of a parasite to establish in a non-native range is greatly affected by its life-cycle and the capability to persist in the environment. *Myxobolus cerebralis* was probably introduced as dormant spores in contaminated imported fish feed (Hewitt and Little 1972). Similarly, PHV was introduced to Australia via imported bait feed, but then spread to New Zealand likely through a fish, bird or mammal host (Jones et al. 1997). It is not clear how *B. ostreae* or OsHV-1 was introduced to New Zealand, but it likely via boat, most probably through infected hull-fouling organisms (Lane and Jones 2020). Shipping is a well-known vector for marine pests, and it is theoretically a high risk for parasites, but few data empirically demonstrate that shipping acts as a vector for parasites to establish in non-native ranges. Reports of new parasites often occur near ports of entry (e.g. Kroeck and Montes 2005; Lane et al. 2016), however, these data are only correlative. The life cycle and host range for many parasites is unknown

and introductions and disease transmission may occur through previously unsuspected hosts. For example, the European cockle *Cerastoderma edule* was recently demonstrated as a host for OsHV-1 capable of transmitting virus to naïve Pacific oyster (Bookelaar et al. 2020). Exploratory surveillance of ballast water and of alternative host species will provide data on the risk of shipping as a vector for aquatic parasites. Biosecurity is an exercise in risk management; therefore, studies should be directed towards assessing risk of shipping and assigning management efforts accordingly.

Invasion ecology theory tells us that in addition to non-native hosts introducing concomitant non-native parasites, non-native hosts can increase the prevalence or severity of existing native diseases (see Poulin et al. 2011). Introduced hosts may be capable of becoming infected with native parasites, amplifying endemic parasites that 'spill back' to native hosts (Kelly et al. 2009b). In contrast, if introduced hosts are capable of becoming infected by native parasites but the parasite cannot complete its lifecycle then it is a dead-end host that may act as a sink for parasites and thus dilute disease risk for native hosts (Paterson et al. 2013). Alternatively, the introduced host may alter the relative abundance of a native host in a way that could increase or decrease disease transmission to other native hosts in the parasite's lifecycle (Poulin et al. 2011). Introduced hosts may also alter disease prevalence and severity indirectly by changing the behaviour of native hosts or by changing physicochemical conditions that affect native host exposure and/or resistance to disease. Species introduction provides a natural experiment for a different

Table 5. Studies on non-native hosts (shown by **bold** type) and disease dynamics in New Zealand.

Host (phylum)	Parasite(s)	Results	Reference
Salmo trutta, Bullies, Galaxiids. (Chordata)	Numerous (Platyhelminthes)	Parasite spillover is not an issue because brown trout were introduced as eggs (i.e. free of parasites). Presents evidence of parasite dilution (i.e. introduced brown trout acts as a sink for native parasites releasing native hosts from infection).	Kelly et al. (2009a)
Salmo trutta, *Galaxias anomalus* (Chordata)	*Acanthocephalus galaxii* (Platyhelminthes)	Methods used include survey, experimental and modelling. Field observations and experimentation support the hypothesis that brown trout act as an infection sink for this parasite. However, modelling shows trout disrupt infection of galaxiid through altering foraging behaviour or reducing density and not as an actual sink.	Paterson et al. (2011)
Salmo trutta, Oncorhynchus mykiss (Chordata)	*Telogaster opisthorchis, Stegodexamene anguillae* (Platyhelminthes)	Through experimentation and surveys, exotic salmonids are poor hosts of both native trematodes. Exotic salmonids may act as parasite sinks decreasing native parasite flow to native hosts.	Paterson et al. (2013)
Perna canaliculus, Xenostrobus pulex, ***Musculista senhousia*** (Mollusca)	Copepods (*Myicolidae*) *Pinnotheres novaezaleandiae* (Arthropoda)	A survey and experimental study, the invasive *M. senhousia* is less likely to become infected with native parasites compared with native hosts (*P. canaliculus* and *X. pulex*).	Miller et al. (2008)
Charybdis japonica, *Ovalipes catharus* (Arthropoda)	–	A survey revealed different parasite species assemblages between the introduced (*C. japonica*) and native (*O. catharus*) crab. The reported parasite species richness was low.	Miller et al. (2006)

perspective on the role of disease in natural populations. In New Zealand there are over 200 non-native aquatic species of different taxa that have become established, but only five studies (two on marine systems, three on freshwater systems) have investigated how their introduction has interacted with native disease dynamics (Table 5).

Future aquatic disease studies and greater synergy with other marine science fields

Studies on aquatic disease represent an emerging field that has developed through aquatic ecological research, veterinary parasitology, the growth of aquaculture and catastrophic aquatic disease events. The USA has been the epicentre of recent large aquatic disease outbreaks that attracted greater mainstream attention to aquatic disease. Following the emergence of SSWD, a National Science Foundation funded Research Coordination Network brought together individuals with expertise across a range of areas, including pathology, parasitology, fisheries, ecology, oceanography, economics, communications and policy to promote cross-pollination of ideas, knowledge and frameworks for studying and managing marine disease (Lafferty and Hofmann 2016). The idea was based on the principle that individuals from each area brought a different set of skills. For instance, parasitologists understood parasites and their lifecycles, pathologists understood disease, and oceanographers could assist with modelling disease transmission and connectivity. Although the Research Coordination Network was born out of a catastrophic disease event, New Zealand can achieve similar goals through greater studies and recognition of disease in aquatic studies. Collaborative studies across disciplines will promote preparedness in the event SSWD-like events emerge in New Zealand, support our primary industries and progress the field of aquatic disease ecology.

Unexpected disease events and a developing aquaculture industry inspired dedicated studies on aquatic disease in New Zealand starting in 1974. In 2020, the aquaculture industry is still growing, and disease still poses a major threat to the industry's viability. However, additional threats from anthropogenic factors like climate change, invasive species and pollution add an extra layer of complexity that remain under studied. It is apparent that now more than in 1974 a call to arms across science disciplines is needed to progress and diversify aquatic studies in New Zealand. This will not happen overnight, but progress could be made in the exposure of marine science students to aquatic disease during University teaching. There are few marine science courses in New Zealand that teach on aquatic disease, and veterinary courses offer limited aquatic disease modules. Scientists working in current aquatic disease areas such as microbiology, veterinary science, parasitology and epidemiology need to continue to attend marine science meetings and draw similarities across disciplines to promote advancement in aquatic studies in New Zealand. To assist with progression, below we suggest some areas of future work, including some relevant science fields (in italics):

1. Develop disease baselines for taxa across different localities. Collecting data on parasites can be added to any already-planned sampling programme or integrated into any experimental or mesocosm studies. Particular focus should go on taxa that have been less studied in a disease context, such as primary producing organisms and invertebrates (*parasitologists and microbiologists, marine ecologists, marine phycologists*).

2. Through disease trials, test host–parasite interactions across different taxa under different environmental conditions. Combine the empirical data with modelling methods to assist in forecasting future disease events and ecological or socioeconomic impacts (*parasitologists and microbiologists, marine ecologists, marine phycologists, oceanographers, epidemiologists*).
3. Elucidate parasite lifecycles for pelagic fishes, especially commercial fishes, and incorporate parasites and their trophic linkages into food webs to assess how fishing may interact with disease (*parasitologists, fisheries scientists, marine ecologists*).
4. Apply HTS to better understand the interaction between host microbiome, parasite microbiome and a changing environment in the development and progression of disease. This work could be completed in conjunction with point 2 (*bioinformaticians, parasitologists and microbiologists, marine ecologists, marine phycologists*).
5. Identify and prioritise economically and/or ecologically important aquatic diseases and design a surveillance plan for them. Implement and governmentally mandate the surveillance plan, and regularly audit the surveillance operations (*epidemiologists, oceanographers, policy makers, diagnosticians*)

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References

Acevedo-Whitehouse K, Petetti L, Duignan P, Castinel A. 2009. Hookworm infection, anaemia and genetic variability of the New Zealand sea lion. Proceedings of the Royal Society B-Biological Sciences. 276:3523–3529.

Alley MR, Suepaul RB, McKinlay B, Young MJ, Wang JN, Morgan KJ, Hunter SA, Gartrell BD. 2017. Diphtheritic stomatitis in yellow-eyed penguins (*Megadyptes antipodes*) in New Zealand. Journal of Wildlife Diseases. 53:102–110.

Anderson CD. 1996. Distribution of salmonid diseases in New Zealand. Surveillance. 23:23–24.

Anderson C, Knowles G, de Lisle G. 1994. A survey for *Yersinia ruckeri* and *Aeromonas salmonicida* in farmed and wild fish. Surveillance. 21:39–40.

Anglade T, Randhawa HS. 2018. Gaining insights into the ecological role of the New Zealand sole (*Peltorhamphus novaezeelandiae*) through parasites. Journal of Helminthology. 92:187–196.

Arzul I, Gagnaire B, Bond C, Chollet B, Morga B, Ferrand S, Robert M, Renault T. 2009. Effects of temperature and salinity on the survival of *Bonamia ostreae*, a parasite infecting flat oysters *Ostrea edulis*. Diseases of Aquatic Organisms. 85:67–75.

Austin B. 2005. Bacterial pathogens of marine fish. In: Belkin S, Colwell R, editors. Oceans and health: pathogens in the marine environment. Boston (MA): Springer; p. 391–413.

Barnes AC, Delamare-Deboutteville J, Gudkovs N, Brosnahan C, Morrison R, Carson J. 2016. Whole genome analysis of *Yersinia ruckeri* isolated over 27 years in Australia and New

Zealand reveals geographical endemism over multiple lineages and recent evolution under host selection. Microbial Genomics. 2:e000095.
Behrens MD, Lafferty KD. 2004. Effects of marine reserves and urchin disease on southern Californian rocky reef communities. Marine Ecology Progress Series. 279:129–139.
Behringer DC, Silliman BR, Lafferty KD, editors. 2020. Marine disease ecology, 1st ed. Oxford: Oxford University Press.
Ben-Horin T, Bidegain G, de Leo G, Groner ML, Hofmann E, McCallum H, Powell E. 2020. Modelling marine disease. In: Behringer DC, Silliman BR, Lafferty KD, editors. Marine disease ecology. 1st ed. Oxford: Oxford University Press; p. 23.
Ben-Horin T, Lenihan HS, Lafferty KD. 2013. Variable intertidal temperature explains why disease endangers black abalone. Ecology. 94:161–168.
Bennett J, Jorge F, Poulin R, Randhawa H. 2019. Revealing trophic transmission pathways of marine tapeworms. Parasitology Research. 118:1435–1444.
Bennett J, Presswell B. 2019. Morphology and molecules resolve the identity and life cycle of an eye trematode, *Philophthalmus attenuatus* n. sp. (Trematoda: Philophthalmidae) infecting gulls in New Zealand. Parasitology Research. 118:1501–1509.
Berkhout BW, Lloyd MM, Poulin R, Studer A. 2014. Variation among genotypes in responses to increasing temperature in a marine parasite: evolutionary potential in the face of global warming? International Journal for Parasitology. 44:1019–1027.
Bernardo-Cravo AP, Schmeller DS, Chatzinotas A, Vredenburg VT, Loyau A. 2020. Environmental factors and host microbiomes shape host-pathogen dynamics. Trends in Parasitology. 36:616–633.
Berthe FCJ, Hine PM. 2003. *Bonamia exitiosa* Hine et al., 2001 is proposed instead of *B. exitiosus* as the valid name of *Bonamia* sp infecting flat oysters *Ostrea chilensis* in New Zealand. Diseases of Aquatic Organisms. 57:181–181.
Bingham P. 2013. Quarterly report of investigations of suspected exotic marine and freshwater pests and diseases. Surveillance. 40:28–31.
Bingham P, Brangenberg N, Williams R, Van Andel M. 2013. Investigation into the first diagnosis of ostreid herpesvirus type 1 in Pacific oysters. Surveillance. 40:20–24.
Bojko J, Lipp EK, Alex F, Behringer DC. 2020. Pollution can drive marine diseases. In: Behringer DC, Silliman BR, Lafferty KD, editors. Marine disease ecology. 1st ed. Oxford: Oxford University Press; p. 19.
Bookelaar B, Lynch SA, Culloty SC. 2020. Host plasticity supports spread of an aquaculture introduced virus to an ecosystem engineer. Parasites & Vectors. 13:498.
Boustead N. 1982. Whirling disease survey results. Freshwater Catch, Summer. 9–11.
Boustead NC, Meyers TR, Short S. 1993. Absence of infectious hematopoietic necrosis virus (IHNV) in New Zealand sockeye salmon, *Oncorhynchus nerka*. New Zealand Journal of Marine and Freshwater Research. 27:55–60.
Brosnahan C. 2020. Diagnostic investigation into summer mortality events of farmed Chinook salmon (*Oncorhynchus tshawytscha*) in New Zealand. Manawatū: Massey University.
Brosnahan CL, Georgiades E, McDonald C, Keeling SE, Munday JS, Jones B. 2019a. Optimisation and validation of a PCR to detect viable *Tenacibaculum maritimum* in salmon skin tissue samples. Journal of Microbiological Methods. 159:186–193.
Brosnahan CL, Ha HJ, Booth K, McFadden AMJ, Jones JB. 2017a. First report of a rickettsia-like organism from farmed Chinook salmon, *Oncorhynchus tshawytscha* (Walbaum), in New Zealand. New Zealand Journal of Marine and Freshwater Research. 51:356–369.
Brosnahan CL, Humphrey S, Knowles G, Ha HJ, Pande A, Jones JB. 2017b. Nocardiosis in freshwater reared Chinook salmon (*Oncorhynchus tshawytscha*). New Zealand Veterinary Journal. 65:214–218.
Brosnahan CL, Munday JS, Davie PS, Kennedy L, Preece M, Barnes S, Jones JB, McDonald WL. 2019b. Pathogenicity of the bacterium New Zealand rickettsia-like organism (NZ-RLO2) in Chinook salmon *Oncorhynchus tshawytscha* smolt. Diseases of Aquatic Organisms. 134:175–187.

Brosnahan CL, Munday JS, Ha HJ, Preece M, Jones JB. 2019c. New Zealand rickettsia-like organism (NZ-RLO) and *Tenacibaculum maritimum*: Distribution and phylogeny in farmed Chinook salmon (*Oncorhynchus tshawytscha*). Journal of Fish Diseases. 42:85–95.

Brosnahan CL, Pande A, Keeling SE, van Andel M, Jones JB. 2019d. Lamprey (*Geotria australis*; Agnatha) reddening syndrome in Southland rivers, New Zealand 2011–2013: laboratory findings and epidemiology, including the incidental detection of an atypical *Aeromonas salmonicida*. New Zealand Journal of Marine and Freshwater Research. 53:416–436.

Brown C. 2000. Emerging infectious diseases of animals: an overview. In: Brown C, Bolin C, editors. Emerging diseases of animals. Washington (DC): American Society for Microbiology; p. 1–12.

Cano I, van Aerle R, Ross S, Verner-Jeffreys DW, Paley RK, Rimmer GSE, Ryder D, Hooper P, Stone D, Feist SW. 2018. Molecular characterization of an Endozoicomonas-like organism causing infection in the King scallop (*Pecten maximus* L.). Applied and Environmental Microbiology. 84:e00952–e00917.

Carson J, Wilson T. 2009. Yersiniosis in Fish. p. 19.

Castinel A, Webb SC, Jones JB, Peeler EJ, Forres BM. 2019. Disease threats to farmed green-lipped mussels *Perna canaliculus* in New Zealand: review of challenges in risk assessment and pathway analysis. Aquaculture Environment Interactions. 11:291–304.

Chiaradia A, Forero MG, Hobson KA, Cullen JM. 2010. Changes in diet and trophic position of a top predator 10 years after a mass mortality of a key prey. ICES Journal of Marine Science. 67:1710–1720.

Claar DC, Wood CL. 2020. Pulse heat stress and parasitism in a warming world. Trends in Ecology & Evolution. 35:704–715.

Coen LD, Bishop MJ. 2015. The ecology, evolution, impacts and management of host-parasite interactions of marine molluscs. Journal of Invertebrate Pathology. 131:177–211.

Corbeil S, McColl KA, Williams LM, Slater J, Crane MSJ. 2017. Innate resistance of New Zealand paua to abalone viral ganglioneuritis. Journal of Invertebrate Pathology. 146:31–35.

Costello MJ. 2006. Ecology of sea lice parasitic on farmed and wild fish. Trends in Parasitology. 22:475–483.

Coupe A, Howe L, Burrows E, Sine A, Pita A, Velathanthiri N, Vallee E, Hayman D, Shapiro K, Roe WD. 2018. First report of *Toxoplasma gondii* sporulated oocysts and *Giardia duodenalis* in commercial green-lipped mussels (*Perna canaliculus*) in New Zealand. Parasitology Research. 117:1453–1463.

Cranfield HJ, Dunn A, Doonan IJ, Michael KP. 2005. *Bonamia exitiosa* epizootic in *Ostrea chilensis* from Foveaux Strait, southern New Zealand between 1986 and 1992. ICES Journal of Marine Science. 62:3–13.

Cruz CD, Chycka M, Hedderley D, Fletcher GC. 2016. Prevalence, characteristics and ecology of *Vibrio vulnificus* found in New Zealand shellfish. Journal of Applied Microbiology. 120:1100–1107.

Cruz CD, Fletcher GC, Paturi G, Hedderley DI. 2020. Influence of farming methods and seawater depth on *Vibrio* species in New Zealand Pacific oysters. International Journal of Food Microbiology. 325:108644.

Cruz CD, Hedderley D, Fletcher GC. 2015. Long-term study of *Vibrio parahaemolyticus* prevalence and distribution in New Zealand shellfish. Applied and Environmental Microbiology. 81:2320–2327.

Darling JA, Pochon X, Abbott CL, Inglis GJ, Zaiko A. 2020. The risks of using molecular biodiversity data for incidental detection of species of concern. Diversity and Distributions. 26:1116–1121.

de Kantzow M, Hick P, Becker JA, Whittington RJ. 2016. Effect of water temperature on mortality of Pacific oysters *Crassostrea gigas* associated with microvariant ostreid herpesvirus 1 (OsHV-1 μVar). Aquaculture Environment Interactions. 8:419–428.

de Lorgeril J, Lucasson A, Petton B, Toulza E, Montagnani C, Clerissi C, Vidal-Dupiol J, Chaparro C, Galinier R, Escoubas J-M, et al. 2018. Immune-suppression by OsHV-1 viral infection causes fatal bacteraemia in Pacific oysters. Nature Communications. 9:4215–4215.

Dheilly NM, Martinez Martinez J, Rosario K, Brindley PJ, Fichorova RN, Kaye JZ, Kohl KD, Knoll LJ, Lukes J, Perkins SL, et al. 2019. Parasite microbiome project: grand challenges. Plos Pathogens. 15:e1008028.

Diggles BK. 2000. Chemotherapy of the ciliate *Trichodina* sp on juvenile turbot (*Colistium nudipinnis*) with notes on the susceptibility of fish with abnormal pigmentation. New Zealand Journal of Marine and Freshwater Research. 34:645–652.

Diggles BK. 2003. Some pathological abnormalities of New Zealand fishes. New Zealand Journal of Marine and Freshwater Research. 37:705–713.

Diggles BK, Carson J, Hine PM, Hickman RW, Tait MJ. 2000. *Vibrio* species associated with mortalities in hatchery-reared turbot (*Colistium nudipinnis*) and brill (*C. guntheri*) in New Zealand. Aquaculture. 183:1–12.

Diggles BK, Hine PM, Handley S, Boustead NC. 2002a. A handbook of diseases of importance to aquaculture in New Zealand. Wellington: NIWA. p. 200.

Diggles BK, Nichol J, Hine PM, Wakefield S, Cochennec-Laureau N, Roberts RD, Friedman CS. 2002b. Pathology of cultured paua *Haliotis iris* infected with a novel haplosporidian parasite, with some observations on the course of disease. Diseases of Aquatic Organisms. 50:219–231.

Dinamani P. 1986. Potential disease-causing organisms associated with mantle cavity of Pacific oyster *Cassostrea gigas* in northern New Zealand. Diseases of Aquatic Organisms. 2:55–63.

Dinamani P, Hine PM, Jones JB. 1987. Occurrence and characteristics of the hemocyte parasite *Bonamia* sp in the New Zealand dredge oyster *Tiostrea lutaria*. Diseases of Aquatic Organisms. 3:37–44.

Doonan IJ, Cranfield HJ, Michael KP. 1994. Catastrophic reduction of the oyster, *Tiostrea chilensis* (Bivalvia, Ostreidae), in Foveaux Strait, New Zealand, due to infestation by the protistan *Bonamia* sp. New Zealand Journal of Marine and Freshwater Research. 28:335–344.

Dunne JA, Lafferty KD, Dobson AP, Hechinger RF, Kuris AM, Martinez ND, McLaughlin JP, Mouritsen KN, Poulin R, Reise K, et al. 2013. Parasites affect food web structure primarily through increased diversity and complexity. PLoS Biology. 11:e1001579.

Edwards DJ, Hine PM. 1974. Introduction, preliminary handling, and diseases of grass carp in New Zealand. New Zealand Journal of Marine and Freshwater Research. 8:441–454.

Elston RA, Farley CA, Kent ML. 1986. Occurrence and significance of bonamiasis in European flat oysters *Ostrea edulis* in North America. Diseases of Aquatic Organisms. 2:49–54.

EPA. 2020. Self assessment report: advice to the decision-making committee to determine the new organism status of microbial pathogens. Environmental Protection Authority.

Feng C, Lin X, Wang F, Zhang Y, Lv J, Wang C, Deng J, Mei L, Wu S, Li H. 2013. Detection and characterization of *Bonamia ostreae* in *Ostrea edulis* imported to China. Diseases of Aquatic Organisms. 106:85–91.

Fenwick SG, Duignan PJ, Nicol CM, Leyland MJ, Hunter JEB. 2004. A comparison of Salmonella serotypes isolated from New Zealand sea lions and feral pigs on the Auckland Islands by pulsed-field gel electrophoresis. Journal of Wildlife Diseases. 40:566–570.

Fu D, Dunn A, Michael KP, Hills J. 2016. The development and performance of a length-based stock assessment of Foveaux Strait oysters (*Ostrea chilensis*, OYU 5) in southern New Zealand, and application to management. Fisheries Research. 183:506–517.

Fuhrman JA. 1999. Marine viruses and their biogeochemical and ecological effects. Nature. 399:541–548.

Gias E, Johnston C, Keeling S, Spence RP, McDonald WL. 2011. Development of real-time PCR assays for detection of megalocytiviruses in imported ornamental fish. Journal of Fish Diseases. 34:609–618.

Grindley RM, Keogh JA, Friedman CS. 1998. Shell lesions in New Zealand *Haliotis* spp. (Mollusca, Gastropoda). Journal of Shellfish Research. 17:805–811.

Guilloteau P, Poulin R, MacLeod CD. 2016. Impacts of ocean acidification on multiplication and caste organisation of parasitic trematodes in their gastropod host. Marine Biology. 163:96. https://doi.org/10.1007/s00227-016-2871-5.

Guo X, Ford SE. 2016. Infectious diseases of marine molluscs and host responses as revealed by genomic tools. Philosophical Transactions of the Royal Society of London Series B, Biological Sciences. 371:20150206.
Gutierrez AP, Symonds J, King N, Steiner K, Bean TP, Houston RD. 2020. Potential of genomic selection for improvement of resistance to ostreid herpesvirus in Pacific oyster (*Crassostrea gigas*). Animal Genetics. 51:249–257.
Handley SJ, Bergquist PR. 1997. Spionid polychaete infestations of intertidal Pacific oysters *Crassostrea gigas* (Thunberg), Mahurangi Harbour, northern New Zealand. Aquaculture. 153:191–205.
Harland H, MacLeod CD, Poulin R. 2015. Non-linear effects of ocean acidification on the transmission of a marine intertidal parasite. Marine Ecology Progress Series. 536:55–64.
Harland H, MacLeod CD, Poulin R. 2016. Lack of genetic variation in the response of a trematode parasite to ocean acidification. Marine Biology. 163:8.
Harrison KR, Duggan IC. 2019. First record of the parasite *Transversotrema patialense* (Soparkar, 1924) within New Zealand, and its prevalence in *Melanoides tuberculata* (Muller, 1774) among captive and "wild" populations. BioInvasions Records. 8:729–735.
Harvell D. 2019. Ocean outbreak: confronting the rising tide of marine disease. Oakland (CA): University of California Press.
Harvell CD, Lamb JB. 2020. Disease outbreaks can threaten marine biodiversity. In: Behringer DC, Silliman BR, Lafferty KD, editors. Marine disease ecology. 1st ed. Oxford: Oxford University Press; p. 18.
Heasman KG, Scott N, Ericson JA, Taylor DI, Buck BH. 2020. Extending New Zealand's marine shellfish aquaculture into exposed environments – adapting to modern anthropogenic challenges. Frontiers in Marine Science. 7:751.
Hewitt GC, Little RW. 1972. Whirling disease in New Zealand trout caused by *Myxosoma cerebralis* (Hofer, 1903) (Protozoa: Myxosporida). New Zealand Journal of Marine and Freshwater Research. 6:1–10.
Hewson I, Bistolas KSI, Quijano Cardé EM, Button JB, Foster PJ, Flanzenbaum JM, Kocian J, Lewis CK. 2018. Investigating the complex association between viral ecology, environment, and northeast Pacific sea star wasting. Frontiers in Marine Science. 5:77.
Hewson I, Button JB, Gudenkauf BM, Miner B, Newton AL, Gaydos JK, Wynne J, Groves CL, Hendler G, Murray M, et al. 2014. Densovirus associated with sea-star wasting disease and mass mortality. Proceedings of the National Academy of Sciences. 111:17278–17283.
Hickman RW. 1978. Incidence of a pea crab and a trematode in cultivated and natural green-lipped mussels. New Zealand Journal of Marine and Freshwater Research. 12:211–215.
Hill AG. 2008. An investigation of *Leucocytozoan* in the endangered yellow-eyed penguin (*Megadyptes antipodes*). Palmerston North: Massey University.
Hine PM. 1978. Distribution of some parasites of freshwater eels in New Zealand. New Zealand Journal of Marine and Freshwater Research. 12:179–187.
Hine PM, Diggles BK. 2002. The distribution of *Perkinsus olseni* in New Zealand bivalve molluscs. Surveillance. 29:8–11.
Hine PM, Diggles BK, Parsons MJD, Pringle A, Bull B. 2002. The effects of stressors on the dynamics of *Bonamia exitiosus* Hine, Cochennec-Laureau & Berthe, infections in flat oysters *Ostrea chilensis* (Philippi). Journal of Fish Diseases. 25:545–554.
Hine PM, Wesney B, Besant P. 1998. Replication of a herpes-like virus in larvae of the flat oyster *Tiostrea chilensis* at ambient temperatures. Diseases of Aquatic Organisms. 32:161–171.
Hine PM, Wesney B, Hay BE. 1992. Herpesviruses associated with mortalities among hatchery-reared larval Pacific oysters *Crassostrea gigas*. Diseases of Aquatic Organisms. 12:135–142.
Hock SD, Poulin R. 2012. Exposure of the snail *Potamopyrgus antipodarum* to herbicide boosts output and survival of parasite infective stages. International Journal for Parasitology: Parasites and Wildlife. 1:13–18.

Hoenig JM, Groner ML, Smith MW, Vogelbein WK, Taylor DM, Landers DF, Swenarton JT, Gauthier DT, Sadler P, Matsche MA, et al. 2017. Impact of disease on the survival of three commercially fished species. Ecological Applications. 27:2116–2127.
Hoffman GL. 1966. Effects of whirling disease. The Progressive Fish Culturist. 28:151–151.
Hofmann H, Blasco-Costa I, Knudsen R, Matthaei CD, Valois A, Lange K. 2016. Parasite prevalence in an intermediate snail host is subject to multiple anthropogenic stressors in a New Zealand river system. Ecological Indicators. 60:845–852.
Hooper C, Hardy-Smith P, Handlinger J. 2007. Ganglioneuritis causing high mortalities in farmed Australian abalone (*Haliotis laevigata* and *Haliotis rubra*). Australian Veterinary Journal. 85:188–193.
Hooper PM, Ross SH, Feist SW, Cano I. 2019. Shedding and survival of an intracellular pathogenic Endozoicomonas-like organism infecting king scallop *Pecten maximus*. Diseases of Aquatic Organisms. 134:167–173.
Hopper JV, Poulin R, Thieltges DW. 2008. Buffering role of the intertidal anemone *Anthopleura aureoradiata* in cercarial transmission from snails to crabs. Journal of Experimental Marine Biology and Ecology. 367:153–156.
Howell M. 1967. The trematode, *Bucephalus longicornutus* (Manter, 1954) in the New Zealand mud-oyster, *Ostrea lutaria*.
Howells J, Jaramillo D, Pal C, Pande A, Lane H. 2019. Intracellular bacteria in New Zealand shellfish: identifying the unculturable. 5th FRDC Australasian Scientific Conference on Aquatic Animal Health & Biosecurity; Jul 8–12; Cairns, QLD, Australia.
Hyatt AD, Hine PM, Jones JB, Whittington RJ, Kearns C, Wise TG, Crane MS, Williams LM. 1997. Epizootic mortality in the pilchard *Sardinops sagax neopilchardus* in Australia and New Zealand in 1995 .2. Identification of a herpesvirus within the gill epithelium. Diseases of Aquatic Organisms. 28:17–29.
Inglis G. 2003. The seagrasses of New Zealand. In: Green EP, Short FT, editors. World Atlas of seagrasses. Berkeley (CA): University of California Press; p. 288.
Jansen MD, Dong HT, Mohan CV. 2019. Tilapia lake virus: a threat to the global tilapia industry? Reviews in Aquaculture. 11:725–739.
Jarvis RM, Young T. 2019. Key research priorities for the future of marine science in New Zealand. Marine Policy. 106:103539.
Jenkins C, Hick P, Gabor M, Spiers Z, Fell SA, Gu X, Read A, Go J, Dove M, O'Connor W, et al. 2013. Identification and characterisation of an ostreid herpesvirus-1 microvariant (OsHV-1 micro-var) in *Crassostrea gigas* (Pacific oysters) in Australia. Diseases of Aquatic Organisms. 105:109–126.
Johnson MB, Lafferty KD, van Oosterhout C, Cable J. 2011. Parasite transmission in social interacting hosts: monogenean epidemics in guppies. PLoS One. 6:e22634.
Jones JB. 1980. Fresh-water crayfish *Paranephrops planifrons* infected with the microsporidian *Thelohania*. New Zealand Journal of Marine and Freshwater Research. 14:45–46.
Jones JB, Hyatt AD, Hine PM, Whittington RJ, Griffin DA, Bax NJ. 1997. Australasian pilchard mortalities. World Journal of Microbiology & Biotechnology. 13:383–392.
Jones HFE, Pilditch CA, Hamilton DP, Bryan KR. 2017. Impacts of a bivalve mass mortality event on an estuarine food web and bivalve grazing pressure. New Zealand Journal of Marine and Freshwater Research. 51:370–392.
Jones JB, Scotti PD, Dearing SC, Wesney B. 1996. Virus-like particles associated with marine mussel mortalities in New Zealand. Diseases of Aquatic Organisms. 25:143–149.
Keeling SE, Brosnahan CL, Johnston C, Wallis R, Gudkovs N, McDonald WL. 2013. Development and validation of a real-time PCR assay for the detection of *Aeromonas salmonicida*. Journal of Fish Diseases. 36:495–503.
Keeling SE, Brosnahan CL, Williams R, Gias E, Hannah M, Bueno R, McDonald WL, Johnston C. 2014. New Zealand juvenile oyster mortality associated with ostreid herpesvirus 1-an opportunistic longitudinal study. Diseases of Aquatic Organisms. 109:231–239.

Keeling SE, Johnston C, Wallis R, Brosnahan CL, Gudkovs N, McDonald WL. 2012. Development and validation of real-time PCR for the detection of *Yersinia ruckeri*. Journal of Fish Diseases. 35:119–125.
Kelly DW, Paterson RA, Townsend CR, Poulin R, Tompkins DM. 2009a. Has the introduction of brown trout altered disease patterns in native New Zealand fish? Freshwater Biology. 54:1805–1818.
Kelly DW, Paterson RA, Townsend CR, Poulin R, Tompkins DM. 2009b. Parasite spillback: a neglected concept in invasion ecology? Ecology. 90:2047–2056.
Kelly DW, Poulin R, Tompkins DM, Townsend CR. 2010. Synergistic effects of glyphosate formulation and parasite infection on fish malformations and survival. Journal of Applied Ecology. 47:498–504.
Kesarcodi-Watson A, Kaspar H, Lategan MJ, Gibson L. 2009. Two pathogens of Greenshell™ mussel larvae, *Perna canaliculus: Vibrio splendidus* and a *V. coralliilyticus/neptunius*-like isolate. Journal of Fish Diseases. 32:499–507.
Knowles S. 1983. Salmon ranching in New Zealand: biology, economic and policy. Christchurch: University of Canterbury.
Koprivnikar J, Poulin R. 2009. Effects of temperature, salinity, and water level on the emergence of marine cercariae. Parasitology Research. 105:957–965.
Kroeck MA, Montes J. 2005. Occurrence of the haemocyte parasite *Bonamia* sp. in flat oysters *Ostrea puelchana* farmed in San Antonio Bay (Argentina). Diseases of Aquatic Organisms. 63:231–235.
Kuris AM, Hechinger RF, Shaw JC, Whitney KL, Aguirre-Macedo L, Boch CA, Dobson AP, Dunham EJ, Fredensborg BL, Huspeni TC, et al. 2008. Ecosystem energetic implications of parasite and free-living biomass in three estuaries. Nature. 454:515–518.
Lafferty KD. 2004. Fishing for lobsters indirectly increases epidemics in sea urchins. Ecological Applications. 14:1566–1573.
Lafferty KD. 2015. Sea otter health: challenging a pet hypothesis. International Journal for Parasitology: Parasites and Wildlife. 4:291–294.
Lafferty KD, Allesina S, Arim M, Briggs CJ, De Leo G, Dobson AP, Dunne JA, Johnson PTJ, Kuris AM, Marcogliese DJ, et al. 2008. Parasites in food webs: the ultimate missing links. Ecology Letters. 11:533–546.
Lafferty KD, Harvell CD. 2014. The role of infectious disease in marine communities: chapter 5. In: Bertness MD , Bruno J, Silliman BR, Stachowicz JJ, editors. Marine Community Ecology and Conservation. Sunderland, Ma: Sinauer Associates, Inc.; p. 85–108.
Lafferty KD, Hofmann EE. 2016. Marine disease impacts, diagnosis, forecasting, management and policy. Philosophical Transactions of the Royal Society of London Series B. Biological Sciences. 371:20150200.
Lagrue C, Heaphy K, Presswell B, Poulin R. 2016. Strong association between parasitism and phenotypic variation in a supralittoral amphipod. Marine Ecology Progress Series. 553:111–123.
Lagrue C, Poulin R. 2015. Bottom-up regulation of parasite population densities in freshwater ecosystems. Oikos. 124:1639–1647.
Lagrue C, Presswell B, Dunckley N, Poulin R. 2018. The invasive cestode parasite *Ligula* from salmonids and bullies on the South Island, New Zealand. Parasitology Research. 117:151–156.
Lamb JB, van de Water JA, Bourne DG, Altier C, Hein MY, Fiorenza EA, Abu N, Jompa J, Harvell CD. 2017. Seagrass ecosystems reduce exposure to bacterial pathogens of humans, fishes, and invertebrates. Science. 355:731–733.
Lane HS, Booth K, Pande A, Jones JB. 2015. First report of the myxozoan parasite *Myxobolus episquamalis* infecting grey mullet (*Mugil cephalus*) from New Zealand. New Zealand Journal of Marine and Freshwater Research. 49:173–177.
Lane HS, Jones JB. 2020. Low internal transcribed spacer rDNA variation in New Zealand *Bonamia ostreae*: evidence for a recent arrival. Diseases of Aquatic Organisms. 139:121–130.
Lane HS, Jones B, Poulin R. 2018. Comparative population genetic study of an important marine parasite from New Zealand flat oysters. Marine Biology. 165:11.

Lane HS, Webb SC, Duncan J. 2016. *Bonamia ostreae* in the New Zealand oyster *Ostrea chilensis:* a new host and geographic record for this haplosporidian parasite. Diseases of Aquatic Organisms. 118:55–63.
Law CS, Bell JJ, Bostock HC, Cornwall CE, Cummings VJ, Currie K, Davy SK, Gammon M, Hepburn CD, Hurd CL, et al. 2018. Ocean acidification in New Zealand waters: trends and impacts. New Zealand Journal of Marine and Freshwater Research. 52:155–195.
LEADDR. 2019. Guidelines from implementing diagnostic next generation sequencing for animal health laboratories in Australia. Australia.
Lehnert K, Poulin R, Presswell B. 2019. Checklist of marine mammal parasites in New Zealand and Australian waters. Journal of Helminthology. 93:649–676.
Lehnert K, Randhawa H, Poulin R. 2017. Metazoan parasites from odontocetes off New Zealand: new records. Parasitology Research. 116:2861–2868.
Lei FY, Poulin R. 2011. Effects of salinity on multiplication and transmission of an intertidal trematode parasite. Marine Biology. 158:995–1003.
Leleu K, Remy-Zephir B, Grace R, Costello MJ. 2012. Mapping habitats in a marine reserve showed how a 30-year trophic cascade altered ecosystem structure. Biological Conservation. 155:193–201.
Ma J, Bruce TJ, Jones EM, Cain KD. 2019. A review of fish vaccine development strategies: Conventional methods and Modern Biotechnological approaches. Microorganisms. 7:569.
MacLeod CD, Poulin R. 2015a. Differential tolerances to ocean acidification by parasites that share the same host. International Journal for Parasitology. 45:485–493.
MacLeod CD, Poulin R. 2015b. Interactive effects of parasitic infection and ocean acidification on the calcification of a marine gastropod. Marine Ecology Progress Series. 537:137–150.
MacLeod CD, Poulin R. 2016a. Parasitic infection alters the physiological response of a marine gastropod to ocean acidification. Parasitology. 143:1397–1408.
MacLeod CD, Poulin R. 2016b. Parasitic infection: a buffer against ocean acidification? Biology Letters. 12:4.
Marcogliese DJ. 2008. The impact of climate change on the parasites and infectious diseases of aquatic animals. Revue scientifique et technique (International Office of Epizootics). 27:467–484.
Marcogliese DJ, Cone DK. 1997. Food webs: a plea for parasites. Trends in Ecology & Evolution. 12:320–325.
McCallum H, Kuris A, Harvell C, Lafferty K, Smith G, Porter J. 2004. Does terrestrial epidemiology apply to marine systems? Trends in Ecology & Evolution. 19:585–591.
McDonald WL. 2014. Animal Health Laboratory. Surveillance. 41:14–17.
McDowall RM. 2004. Shoot first, and then ask questions: A look at aquarium fish imports and invasiveness in New Zealand. New Zealand Journal of Marine and Freshwater Research. 38:503–510.
Merou N, Lecadet C, Pouvreau S, Arzul I. 2020. An eDNA/eRNA-based approach to investigate the life cycle of non-cultivable shellfish micro-parasites: the case of *Bonamia ostreae*, a parasite of the European flat oyster *Ostrea edulis*. Microbial Biotechnology. 13:1807–1818.
Michael KP. 2020. Recruitment of *Ostrea chilensis* (Philippi, 1844) in Foveaux Strait, Southern New Zealand. Victoria University of Wellington.
Michael KP, Bilewitch J, Forman J, Hulston D, Moss G. 2020. A survey of the Foveaux Strait oyster (*Ostrea chilensis*) population (OYU 5) in commercial fishery areas and the status of Bonamia (*Bonamia exitiosa*) in February 2019. In: 2020/11 NZFAR, editor. p. 78.
Michael KP, Forman J, Hulston D. 2015. A survey of the Foveaux Strait oyster (*Ostrea chilensis*) population (OYU5) in commercial fishery areas and the status of bonamia (*Bonamia exitiosa*) in February 2015. Wellington: Ministry for Primary Industries.
Miller MA, Gardner IA, Kreuder C, Paradies DM, Worcester KR, Jessup DA, Dodd E, Harris MD, Ames JA, Packham AE, Conrad PA. 2002. Coastal freshwater runoff is a risk factor for *Toxoplasma gondi*i infection of southern sea otters (*Enhydra lutris nereis*). International Journal for Parasitology. 32:997–1006.

Miller A, Inglis GJ, Poulin R. 2006. Comparison of the ectosymbionts and parasites of an introduced crab, *Charybdis japonica*, with sympatric and allopatric populations of a native New Zealand crab, *Ovalipes catharus* (Brachyura: Portunidae). New Zealand Journal of Marine and Freshwater Research. 40:369–378.

Miller A, Inglis GJ, Poulin R. 2008. Use of the introduced bivalve, *Musculista senhousia*, by generalist parasites of native New Zealand bivalves. New Zealand Journal of Marine and Freshwater Research. 42:143–151.

Mohanty BR, Sahoo PK. 2007. Edwardsiellosis in fish: a brief review. Journal of Biosciences. 32:1331–1344.

Monnens M, Frost EJ, Clark M, Sewell MA, Vanhove MPM, Artois T. 2019. Description and ecophysiology of a new species of *Syndesmis* Silliman, 1881 (Rhabdocoela: Umagillidae) from the sea urchin *Evechinus chloroticus* (Valenciennes, 1846) Mortensen, 1943 in New Zealand. International Journal for Parasitology: Parasites and Wildlife. 10:71–82.

Mordecai GJ, Miller KM, Di Cicco E, Schulze AD, Kaukinen KH, Ming TJ, Li S, Tabata A, Teffer A, Patterson DA, et al. 2019. Endangered wild salmon infected by newly discovered viruses. Elife. 8: e47615.

Mouritsen KN, Poulin R. 2002. Parasitism, community structure and biodiversity in intertidal ecosystems. Parasitology. 124:S101–S117.

Mouritsen KN, Poulin R. 2005a. Parasites boosts biodiversity and changes animal community structure by trait-mediated indirect effects. Oikos. 108:344–350.

Mouritsen KN, Poulin R. 2005b. Parasitism can influence the intertidal zonation of non-host organisms. Marine Biology. 148:1–11.

Mouritsen KN, Poulin R. 2006. A parasite indirectly impacts both abundance of primary producers and biomass of secondary producers in an intertidal benthic community. Journal of the Marine Biological Association of the United Kingdom. 86:221–226.

Mouritsen KN, Sorensen MM, Poulin R, Fredensborg BL. 2018. Coastal ecosystems on a tipping point: global warming and parasitism combine to alter community structure and function. Global Change Biology. 24:4340–4356.

MPI. 2019. Situation and Outlook for Primary Industries December 2019. Wellington: Ministry for Primary Industries.

Murray AG, O'Callaghan M, Jones B. 2001a. A model of transmission of a viral epidemic among schools within a shoal of pilchards. Ecological Modelling. 144:245–259.

Murray AG, O'Callaghan M, Jones B. 2001b. Simple models of massive epidemics of herpesvirus in Australian (and New Zealand) pilchards. Environment International. 27:243–248.

Nguyen TV, Alfaro AC. 2020. Applications of omics to investigate responses of bivalve haemocytes to pathogen infections and environmental stress. Aquaculture. 518:734488.

O'Connell-Milne SA, Poulin R, Savage C, Rayment W. 2016b. Reduced growth, body condition and foot length of the bivalve *Austrovenus stutchburyi* in response to parasite infection. Journal of Experimental Marine Biology and Ecology. 474:23–28.

O'Connell-Milne S, Savage C, Rayment W. 2016a. The influence of commercial harvesting on parasite infection in the bivalve *Austrovenus stutchburyi*. Canadian Journal of Fisheries and Aquatic Sciences. 73:982–989.

OIE. 2014. Development and optimisation of nucleic acid detection assays. OIE Validation Guidelines. 1–11.

OIE. 2019. Chapter 1.1.2. Principles and methods of validation of diagnostic assays for infectious diseases. Manual of Diagnostic Tests for Aquatic Animals.18.

Pagenkopp Lohan KM, Fleischer RC, Carney KJ, Holzer KK, Ruiz GM. 2016. Amplicon-based pyrosequencing reveals high diversity of protistan parasites in ships' ballast water: Implications for biogeography and infectious diseases. Microbial Ecology. 71:530–542.

Pagenkopp Lohan KM, Ruiz GM, Torchin ME. 2020. Invasions can drive marine disease dynamics. In: Behringer DC, Silliman BR, Lafferty KD, editors. Marine Disease Ecology. 1st ed. Oxford: Oxford University Press; p. 24.

Paine RT. 1966. Food web complexity and species diversity. American Naturalist. 100:65–75.

Pande A, Acosta H, Brangenberg NA, Keeling SE. 2015. Design of a detection survey for ostreid herpesvirus-1 using hydrodynamic dispersion models to determine epidemiological units. Preventive Veterinary Medicine. 119:80–84.
Paterson RA, Lal A, Dale M, Townsend CR, Poulin R, Tompkins DM. 2013. Relative competence of native and exotic fish hosts for two generalist native trematodes. International Journal for Parasitology: Parasites and Wildlife. 2:136–143.
Paterson RA, Townsend CR, Poulin R, Tompkins DM. 2011. Introduced brown trout alternative acanthocephalan infections in native fish. Journal of Animal Ecology. 80:990–998.
Paul-Pont I, Evans O, Dhand NK, Rubio A, Coad P, Whittington RJ. 2014. Descriptive epidemiology of mass mortality due to ostreid herpesvirus-1 (OsHV-1) in commercially farmed Pacific oysters (*Crassostrea gigas*) in the Hawkesbury river estuary, Australia. Aquaculture. 422-423:146–159.
Pecher WT, Alavi MRA, Schott EJ, Fernandez-Robledo JA, Roth L, Berg T, Vasta GR. 2008. Assessment of the northern distribution range of selected *Perkinsus* species in eastern oysters (*Crassostrea virginica*) and hard clams (*Mercenaria mercenaria*) with the use of PCR-based detection assays. Journal of Parasitology. 94:410–422.
Peeler EJ, Oidtmann BC, Midtlyng PJ, Miossec L, Gozlan RE. 2011. Non-native aquatic animals introductions have driven disease emergence in Europe. Biological Invasions. 13:1291–1303.
Pernet F, Barret J, Le Gall P, Corporeau C, Dégremont L, Lagarde F, Pépin JF, Keck N. 2012. Mass mortalities of Pacific oysters *Crassostrea gigas* reflect infectious diseases and vary with farming practices in the Mediterranean Thau lagoon, France. Aquaculture Environment Interactions. 2:215–237.
Pernet F, Lupo C, Bacher C, Whittington RJ. 2016. Infectious diseases in oyster aquaculture require a new integrated approach. Philosophical Transactions of the Royal Society of London Series B, Biological Sciences. 371:20150213.
Poulin R. 2006. Global warming and temperature-mediated increases in cercarial emergence in trematode parasites. Parasitology. 132:143–151.
Poulin R. 2010. Parasite manipulation of host behavior: an update and frequently asked questions. In: Brockmann HJ, Roper TJ, Naguib M, et al., editors. Advances in the study of behavior. Vol. 41. San Diego: Elsevier Academic Press Inc; p. 151–186.
Poulin R, Blasco-Costa I, Randhawa HS. 2016. Integrating parasitology and marine ecology: Seven challenges towards greater synergy. Journal of Sea Research. 113:3–10.
Poulin R, Paterson RA, Townsend CR, Tompkins DM, Kelly DW. 2011. Biological invasions and the dynamics of endemic diseases in freshwater ecosystems. Freshwater Biology. 56:676–688.
Poulin R, Randhawa HS. 2015. Evolution of parasitism along convergent lines: from ecology to genomics. Parasitology. 142(Suppl 1):S6–S15.
Preuss M, Zuccarello GC. 2018. Three new red algal parasites from New Zealand: *Cladhymenia oblongifoliaphila* sp. nov. (Rhodomelaceae), *Phycodrys novae-zelandiaephila* sp. nov. (Delesseriaceae) and *Judithia parasitica* sp. nov. (Kallymeniaceae). Phycologia. 57:9–19.
Qviller L, Kristoffersen AB, Lyngstad TM, Lillehaug A. 2020. Infectious salmon anemia and farm-level culling strategies. Frontiers in Veterinary Science. 6:481.
Randhawa HS, Brickle P. 2011. Larval parasite gene sequence data reveal cryptic trophic links in life cycles of porbeagle shark tapeworms. Marine Ecology Progress Series. 431:215–222.
Ray SM. 1952. A culture technique for the diagnosis of infections with *Dermocystidium marinum* Mackin, Owen, and Collier in oysters. Science. 116:360–361.
Renault T, Bouquet AL, Maurice JT, Lupo C, Blachier P. 2014. Ostreid herpesvirus 1 infection among Pacific oyster (*Crassostrea gigas*) spat: relevance of water temperature to virus replication and circulation prior to the onset of mortality. Applied and Environmental Microbiology. 80:5419–5426.
Reverter M, Sarter S, Caruso D, Avarre J-C, Combe M, Pepey E, Pouyaud L, Vega-Heredía S, de Verdal H, Gozlan RE. 2020. Aquaculture at the crossroads of global warming and antimicrobial resistance. Nature Communications. 11:1870.
Rhodes F, editor. 2016. New Zealand Veterinary Journal Volume 64, Issue 1 [whole issue]. New Zealand Veterinary Journal. 64(1): 1–70.

Roe WD, Howe L, Baker EJ, Burrows L, Hunter SA. 2013. An atypical genotype of *Toxoplasma gondii* as a cause of mortality in Hector's dolphins (*Cephalorhynchus hectori*). Veterinary Parasitology. 192:67–74.
Roe WD, Rogers L, Pinpimai K, Dittmer K, Marshall J, Chilvers BL. 2015. Septicaemia and meningitis caused by infection of New Zealand sea lion pups with a hypermucoviscous strain of *Klebsiella pneumoniae*. Veterinary Microbiology. 176:301–308.
Ruehle B, Poulin R. 2020. Risky business: influence of eye flukes on use of risky microhabitats and conspicuousness of a fish host. Parasitology Research. 119:423–430.
Ryan T, Diprose B, Leong R. 2000. Country-freedom plan for infectious bursal disease: a producer-led national disease control programme. Surveillance. 27:3–5.
Safi K, Gutierrez-Rodriguez A. 2017. Parasitism of diatoms in the Chatham Rise subtropical convergence zone East of New Zealand during summer. New Zealand Marine Sciences Society 56th Annual Conference; Jul 4–6; Christchurch, New Zealand.
Sanderson CE, Alexander KA. 2020. Unchartered waters: climate change likely to intensify infectious disease outbreaks causing mass mortality events in marine mammals. Global Change Biology. 26:4284–4301.
Scott-Orr H, Jones JB, Bhatia N. 2017. Uncooked prawn imports: effectiveness of biosecurity controls. Australian Government Inspector-General of Biosecurity Review report No 2017–18/01.
Segarra A, Pépin J, Arzul I, Morga B, Faury N, Renault T. 2010. Detection and description of a particular ostreid herpesvirus 1 genotype associated with massive mortality outbreaks of Pacific oysters, *Crassostrea gigas*, in France in 2008. Virus Research. 153:92–99.
Sharp NJ, Diggles BK, Poortenaar CW, Willis TJ. 2004. Efficacy of Aqui-S, formalin and praziquantel against the monogeneans, *Benedenia seriolae* and *Zeuxapta seriolae*, infecting yellowtail kingfish *Seriola lalandi lalandi* in New Zealand. Aquaculture. 236:67–83.
Sharp NJ, Poortenaar CW, Diggles BK, Willis TJ. 2003. Metazoan parasites of yellowtail kingfish, *Seriola lalandi lalandi*, in New Zealand: prevalence, intensity, and site preference. New Zealand Journal of Marine and Freshwater Research. 37:273–282.
Sharples AD, Evans CW. 1995a. Metazoan parasites of the snapper, *Pagrus auratus* (Bloch and Schneider, 1801), in New Zealand: 1. prevalence and abundance. New Zealand Journal of Marine and Freshwater Research. 29:195–201.
Sharples AD, Evans CW. 1995b. Metazoan parasites of the snapper, *Pagrus auratus* (Bloch and Schneider, 1801), in New Zealand: 2. Site specificty. New Zealand Journal of Marine and Freshwater Research. 29:203–211.
Sharples AD, Evans CW. 1995c. Taxonomy of the metazoan parasites of the snapper *Pagrus auratus* in New Zealand: 1. Ectoparasites. New Zealand Journal of Zoology. 22:143–161.
Sharples AD, Evans CW. 1995d. Taxonomy of the metazoan parasites of the snapper *Pagrus auratus* in New Zealand: Endoparasites. New Zealand Journal of Zoology. 22:163–174.
Shimokawa J, Yoshinaga T, Ogawa K. 2010. Experimental evaluation of the pathogenicity of *Perkinsus olseni* in juvenile Manila clams *Ruditapes philippinarum*. Journal of Invertebrate Pathology. 105:347–351.
Short FT, Ibelings BW, Den Hartog C. 1988. Comparison of a current eelgrass disease to the wasting disease in the 1930s. Aquatic Botany. 30:295–304.
Sniesko SF. 1974. The effects of environmental stress on outbreaks of infectious diseases of fishes. Journal of Fish Biology. 6:197–208.
Stentiford GD, Bateman KS, Small HJ, Moss J, Shields JD, Reece KS, Tuck I. 2010. *Myospora metanephrops* (n. g., n. sp.) from marine lobsters and a proposal for erection of a new order and family (Crustaceacida; Myosporidae) in the Class Marinosporidia (phylum Microsporidia). International Journal for Parasitology. 40:1433–1446.
Studer A, Cubillos VM, Lamare MD, Poulin R, Burritt DJ. 2012a. Effects of ultraviolet radiation on an intertidal trematode parasite: An assessment of damage and protection. International Journal for Parasitology. 42:453–461.
Studer A, Lamare MD, Poulin R. 2012b. Effects of ultraviolet radiation on the transmission process of an intertidal trematode parasite. Parasitology. 139:537–546.

Studer A, Poulin R. 2013. Differential effects of temperature variability on the transmission of a marine parasite. Marine Biology. 160:2763–2773.
Studer A, Poulin R, Tompkins DM. 2013a. Local effects of a global problem: modelling the risk of parasite-induced mortality in an intertidal trematode-amphipod system. Oecologia. 172:1213–1222.
Studer A, Thieltges DW, Poulin R. 2010. Parasites and global warming: net effects of temperature on an intertidal host-parasite system. Marine Ecology Progress Series. 415:11–22.
Studer A, Widmann M, Poulin R, Krkosek M. 2013b. Large scale patterns of trematode parasitism in a bivalve host: no evidence for a latitudinal gradient in infection levels. Marine Ecology Progress Series. 491:125–135
Suttle CA. 2007. Marine viruses — major players in the global ecosystem. Nature Reviews Microbiology. 5:801–812.
Sweet M, Burian A, Bullling M. 2020. Corals as canaries in the coalmine: towards the incorporation of marine ecosystems into the 'One Health' concept.
Tenquist J. 1990. Wallaceville Veterinary Laboratory: an anecdotal history. Upper Hutt: Ministry of Agriculture and Fisheries.
Thieltges DW, Rick J. 2006. Effect of temperature on emergence, survival and infectivity of cercariae of the marine trematode *Renicola roscovita* (Digenea: Renicolidae). Diseases of Aquatic Organisms. 73:63–68.
Thomas F, Poulin R. 1998. Manipulation of a mollusc by a trophically transmitted parasite: convergent evolution or phylogenetic inheritance? Parasitology. 116:431–436.
Thomas F, Renaud F, de Meeus T, Poulin R. 1998. Manipulation of host behaviour by parasites: ecosystem engineering in the intertidal zone? Proceedings of the Royal Society B-Biological Sciences. 265:1091–1096.
Thompson RM, Mouritsen KN, Poulin R. 2005. Importance of parasites and their life cycle characteristics in determining the structure of a large marine food web. Journal of Animal Ecology. 74:77–85.
Tisdall DJ, Phipps JC. 1987. Isolation and characterization of a marine birnavirus from returning quinnat salmon (*Oncorhynchus tshawtscha*) in the South Island of New Zealand. New Zealand Veterinary Journal. 35:217–218.
Tracy AM, Pielmeier ML, Yoshioka RM, Heron SF, Harvell CD. 2019. Increases and decreases in marine disease reports in an era of global change. Proceedings of the Royal Society B-Biological Sciences. 286:20191718.
Trottier O, Walker D, Jeffs AG. 2012. Impact of the parasitic pea crab *Pinnotheres novaezelandiae* on aquacultured New Zealand green-lipped mussels, *Perna canaliculus*. Aquaculture. 344-349:23–28.
Tubbs LA, Poortenaar CW, Sewell MA, Diggles BK. 2005. Effects of temperature on fecundity in vitro, egg hatching and reproductive development of *Benedenia seriolae* and *Zeuxapta seriolae* (Monogenea) parasitic on yellowtail kingfish *Seriola lalandi*. International Journal for Parasitology. 35:315–327.
Vielma S, Lagrue C, Poulin R, Selbach C. 2019. Non-host organisms impact transmission at two different life stages in a marine parasite. Parasitology Research. 118:111–117.
Villalba A, Casas SM, Lopez C, Carballal MJ. 2005. Study of perkinsosis in the carpet shell clam *Tapes decussatus* in Galicia (NW Spain). II. Temporal pattern of disease dynamics and association with clam mortality. Diseases of Aquatic Organisms. 65:257–267.
Wahle RA, Gibson M, Fogarty M. 2009. Distinguishing disease impacts from larval supply effects in a lobster fishery collapse. Marine Ecology Progress Series. 376:185–192.
Walker PJ, Mohan CV. 2009. Viral disease emergence in shrimp aquaculture: origins, impact and the effectiveness of health management strategies. Reviews in Aquaculture. 1:125–154.
Walker PJ, Winton JR. 2010. Emerging viral diseases of fish and shrimp. Veterinary Research. 41:51.
Ward T. 2001. Effects of the 1995 and 1998 mass mortality events on the spawning biomass of sardine, *Sardinops sagax*, in South Australian waters. ICES Journal of Marine Science. 58:865–875.

Ward JR, Lafferty KD. 2004. The elusive baseline of marine disease: are diseases in ocean ecosystems increasing? PLoS Biol. 2:E120.
Waugh GD. 1975. Proceedings of the fish disease seminar. In: Divsion FR, editor. Wellington: New Zealand Ministry of Agriculture and Fisheries; p. 33.
Whittington RJ, Jones JB, Hine PM, Hyatt AD. 1997. Epizootic mortality in the pilchard *Sardinops sagax neopilchardus* in Australia and New Zealand in 1995 .1. Pathology and epizootiology. Diseases of Aquatic Organisms. 28:1–15.
Williams GJ, Knapp IS, Aeby GS, Davy SK. 2011. Spatial and temporal patterns of scleractinian coral, soft coral, and zoanthid disease on a remote, near-pristine coral reef (Palmyra Atoll, central Pacific). Diseases of Aquatic Organisms. 94:89–100.
Wood CL, Lafferty KD, Micheli F. 2010. Fishing out marine parasites? Impacts of fishing on rates of parasitism in the ocean. Ecology Letters. 13:761–775.
Wood SA, Pochon X, Laroche O, von Ammon U, Adamson J, Zaiko A. 2019. A comparison of droplet digital polymerase chain reaction (PCR), quantitative PCR and metabarcoding for species-specific detection in environmental DNA. Molecular Ecology Resources. 19:1407–1419.
Wood SA, Pochon X, Ming W, von Ammon U, Woods C, Carter M, Smith M, Inglis G, Zaiko A. 2018. Considerations for incorporating real-time PCR assays into routine marine biosecurity surveillance programmes: a case study targeting the Mediterranean fanworm (*Sabella spallanzanii*) and club tunicate (*Styela clava*). Genome. 62:137–146.
Zha H, Jeffs A, Dong Y, Lewis G. 2018a. Potential virulence factors of bacteria associated with tail fan necrosis in the spiny lobster, *Jasus edwardsii*. Journal of Fish Diseases. 41:817–828.
Zha H, Jones B, Lewis G, Dong YM, Jeffs A. 2018b. Pathology of tail fan necrosis in the spiny lobster, *Jasus edwardsii*. Journal of Invertebrate Pathology. 154:5–11.
Zha H, Lewis G, Waite DW, Wu JY, Chang K, Dong YM, Jeffs A. 2019. Bacterial communities associated with tail fan necrosis in spiny lobster, *Jasus edwardsii*. Fems Microbiology Ecology. 95:9.