iScience

CellPress
OPEN ACCESS

Review

Diseases of marine fish and shellfish in an age of rapid climate change

Andrew F. Rowley,[1,*] Craig Baker-Austin,[2] Annette S. Boerlage,[3] Coline Caillon,[4] Charlotte E. Davies,[1] Léo Duperret,[5] Samuel A.M. Martin,[6] Guillaume Mitta,[7] Fabrice Pernet,[4] Jarunan Pratoomyot,[8] Jeffrey D. Shields,[9] Andrew P. Shinn,[10,11] Warangkhana Songsungthong,[12] Gun Srijuntongsiri,[13] Kallaya Sritunyalucksana,[12] Jeremie Vidal-Dupiol,[5] Tamsyn M. Uren Webster,[1] Suparat Taengchaiyaphum,[12] Ratchakorn Wongwaradechkul,[10] and Christopher J. Coates[1,14,*]

SUMMARY

A recurring trend in evidence scrutinized over the past few decades is that disease outbreaks will become more frequent, intense, and widespread on land and in water, due to climate change. Pathogens and the diseases they inflict represent a major constraint on seafood production and yield, and by extension, food security. The risk(s) for fish and shellfish from disease is a function of pathogen characteristics, biological species identity, and the ambient environmental conditions. A changing climate can adversely influence the host and environment, while augmenting pathogen characteristics simultaneously, thereby favoring disease outbreaks. Herein, we use a series of case studies covering some of the world's most cultured aquatic species (e.g., salmonids, penaeid shrimp, and oysters), and the pathogens (viral, fungal, bacterial, and parasitic) that afflict them, to illustrate the magnitude of disease-related problems linked to climate change.

INTRODUCTION

Earth's human population is projected to exceed nine and 11 billion by 2050 and 2100, respectively,[1] placing inordinate pressure on terrestrial and aquatic environments for greater food production. For millennia, coastal communities have relied on the seas for food and feed. Although this resource was considered boundless by some, we now understand that overfishing has decimated wild stocks, resulting in economic decline and environmental degradation. Aquaculture is also an ancient practice, e.g., freshwater carp husbandry in China is some 2,000 years old,[2] and more recently, the increasing demand for quality food rich in protein, omega 3 polyunsaturated fatty acids, and minerals, has triggered the development of intensive aquaculture (freshwater, brackish, and marine) such that it now exceeds yields from capture fisheries.[3] Examples of marine/brackish fish and shellfish produced globally for human consumption include Atlantic salmon (*Salmo salar*), milkfish (*Chanos chanos*), gilthead seabream (*Sparus auratus*), Pacific whiteleg shrimp (*Penaeus vannamei*), crawfish such *as Procambarus clarkii*, and oysters (e.g., *Crassostrea virginica* and *C. gigas*).[3] In the oceans, ~17% of production comes from mariculture[4] although this may develop in future years with improvements in culture methods and openings in global markets for seafood. As an illustration of the current situation of capture fisheries vs. mariculture, data show that 90.3 million tonnes (live weight equivalent) of global marine capture fishery production were reported in 2020 down from 96.5 million tonnes in 2018 while during the same period mariculture increased production from 30.9 to 33.1 million tonnes.[3] Recent analyses of food production from the oceans conclude that reform in fisheries policies and processes, together with the sustainable expansion of mariculture, could increase production even after considering the diverse and complex challenges that climate change brings.[4,5]

[1]Biosciences, Faculty of Science and Engineering, Swansea University, Swansea SA2 8PP, Wales, UK
[2]Centre for Environment, Fisheries and Aquaculture Science, Weymouth, UK
[3]Centre for Epidemiology and Planetary Health (CEPH), SRUC School of Veterinary Medicine, Inverness, Scotland, UK
[4]Université of Brest, Ifremer, CNRS, IRD, LEMAR, Plouzané, France
[5]IHPE, Université of Montpellier, CNRS, Ifremer, University Perpignan Via Domitia, Montpellier, France
[6]Scottish Fish Immunology Research Centre, School of Biological Sciences, University of Aberdeen, Aberdeen, Scotland, UK
[7]Ifremer, ILM, IRD, UPF, UMR 241 SECOPOL, Tahiti, French Polynesia
[8]Institute of Marine Science, Burapha University, Chonburi 20131, Thailand
[9]Virginia Institute of Marine Science, William & Mary, Gloucester Point, VA 23062, USA
[10]INVE Aquaculture (Thailand), 471 Bond Street, Bangpood, Pakkred, Nonthaburi 11120, Thailand
[11]Centre for Sustainable Tropical Fisheries and Aquaculture, James Cook University, Townsville, QLD, Australia
[12]National Center for Genetic Engineering and Biotechnology (BIOTEC), National Science and Technology Development Agency (NSTDA), Bangkok 10400, Thailand
[13]School of Information, Computer, and Communication Technology, Sirindhorn International Institute of Technology, Thammasat University, Pathum Thani, Thailand
[14]Zoology and Ryan Institute, School of Natural Sciences, University of Galway, H91 TK33 Galway, Ireland
*Correspondence: a.f.rowley@swansea.ac.uk (A.F.R.), christopher.coates@universityofgalway.ie (C.J.C.)
https://doi.org/10.1016/j.isci.2024.110838

Intensification of fish farming (freshwater, brackish, and marine) comes at a price with consequences linked to environmental damage, fish meal extraction from wild fish, waste product toxicity, eutrophication below sea pens, and various pharmaceuticals (e.g., antibiotics and anti-parasitic drugs) leeching into the benthos.[6] In contrast, the cultivation of bivalve molluscs has little environmental footprint as these do not require feed at later stages of development. Regardless of the form of cultivation or the target species, disease limits productivity and increases wastes. Furthermore, spread of pathogens/parasites from captive to wild fish and shellfish (and vice versa) is a major consequence of the expansion of aquaculture, as it is impossible to fully control biosecurity in open water systems (e.g., pens/cages housing fish and trestles and ropes for growing bivalve molluscs). An illustration of this problem comes from the infestation of Atlantic salmon (*Salmo salar*) by ecto-parasitic copepods, the salmon louse *Lepeophtheirus salmonis*, and species of *Caligus*, where there is the potential transfer of these to wild fish in the surrounding waters.[7,8] Parasitism by lice such as *Caligus* spp. and *L. salmonis* is endemic in wild fish and, in the case of the latter, is found all year long, but numbers of lice per fish is generally low such that they do not cause significant mortality. Approaches employing chemical control of lice using various organophosphates are effective but can also have detrimental effects on non-target species[9,10] and lice have become increasingly resistant to certain agents.[11–14] Alternative approaches to chemicals include the use of cleaner fish to consume the lice,[15] mechanical devices such as high-pressure water jetting (e.g., Hydrolicer) for dislodging lice,[16] a suite of physical barriers[17] and novel cage designs to minimize contact with lice; and yet the management of lice in farmed salmon persists among the most prominent issue of parasitic infections in aquaculture. For example, in autumn 2023, an outbreak of *L. salmonis* in cultured salmon in Iceland resulted in the culling of large numbers of grossly affected fish and was reported widely in the media (e.g., https://www.theguardian.com/environment/2023/nov/03/sea-lice-outbreak-icelandic-salmon-farm-welfare-disaster-footage).

Both infectious and non-infectious diseases in wild and captive fish, shellfish, and macroalgae (seaweeds), can devastate production. For instance, economic losses in shrimp production in the Mekong Delta of Vietnam due to the bacterial disease, acute hepatopancreatic disease, and the viral disease, white spot syndrome, were estimated at >US$ 26 million and >US$ 11 million, respectively, in one year.[18] For finfish, parasitic diseases result in the loss of 5.8–16.5% of the total value from aquaculture production in the U.K. alone.[19] In fisheries, the effects of disease can vary in severity from catastrophic collapse of stocks through to reduced growth, fecundity, and at the lowest level, loss of marketability. Regarding the latter, a disease of marine crabs in fisheries caused by the dinoflagellate parasites, *Hematodinium* spp.[20,21] exemplifies this condition. Over 50 species of decapod crustaceans worldwide are infected by these parasites, which can retard growth, discolor the carapace,[22] and lead to mortality. In species including Tanner crabs (*Chionecetes bairdi*) and snow crabs (*C. opilio*), the infected muscle has a bitter taste (termed "bitter crab disease") making it unmarketable.[23,24]

While climate change has been reviewed comprehensively in terms of general effects on fisheries and aquaculture,[4,5,25–31] there are fewer contemporary overviews directed at infectious disease.[32,33] Hence, we aim to evaluate the potential effects of climate change on infectious diseases of fish and shellfish in marine and brackish environments. By means of several "case studies" of notable diseases and their globally dispersed hosts, we also illustrate the potential magnitude of the problems faced with increasing production of high-quality food for an ever-growing population in a time of rapid environmental change. Finally, we briefly explore how these diseases can be mitigated through new approaches to farming fish and shellfish now and in the future.

WHAT IS THE EFFECT OF DISEASE ON THE PRODUCTION OF FOOD FROM THE SEA?

It is difficult to gauge the economic effects of both infectious and non-infectious diseases on wild populations of marine fish and shellfish but there is the potential for serious losses.[34] Mortality in wild fish can be hidden as they are rapidly subject to predation and may decompose before they can be observed.[35] Major fish kills, such as those caused by algal toxins (harmful algal blooms) become apparent when they wash onto the shoreline, but autopsy of such individuals may fail to determine the etiology of the condition that resulted in their death. Pertinent examples of diseases of fisheries with known economic consequences include ichthyophoniasis of herring[36,37] and in Chinook salmon (*Oncorhynchus tshawytscha*) in the Yukon River[38] caused by the oomycete, *Ichthyophonus hoferi*. In this former example, the reduction of populations of Pacific herring (*Clupea pallasii*) in Alaska after 1993 was attributed to a few diseases but principally to ichthyophoniasis. For shellfish, epizootic shell disease outbreaks in New England American lobsters (*Homarus americanus*) coincided with the collapse of the southern New England stock.[39] Similarly, the decline in populations of velvet swimming crabs (*Necora puber*) caught in Brittany, France after 1984 was thought to be linked to the occurrence of hematodiniosis caused by dinoflagellate parasites, *Hematodinium* spp.[40]

In terms of aquaculture, diseases are more readily observed and identified, and the associated economic losses are easier to calculate despite being highly variable in severity across locations, the species affected, and in different seasons. There has been a revolution in molecular "tools" available for pathologists to identify and track the spread of disease-causing agents[41] and these complement traditional approaches such as microscopy and histopathology. Advances in epidemiology, including the modeling of disease dynamics and transmission, are also important when preparing for emerging diseases, and forecasting outbreaks.[42–44]

ELEVATED TEMPERATURE AND CHANGES IN THE PREVALENCE AND SEVERITY OF INFECTIOUS DISEASES IN THE OCEANS

This last year, 2023, was the warmest since modern records began in 1850.[45] Global climate data for January–December 2023 show that oceans were 0.91°C higher than average making this the warmest since 1850.[45] (Figure 1). Similarly, land and ocean + land temperature anomalies, were +1.79 and +1.18°C, respectively, with sea surface temperatures hotter than average in several areas of the world including parts of North and South America, Europe, Africa, Oceania, and Asia.[45]

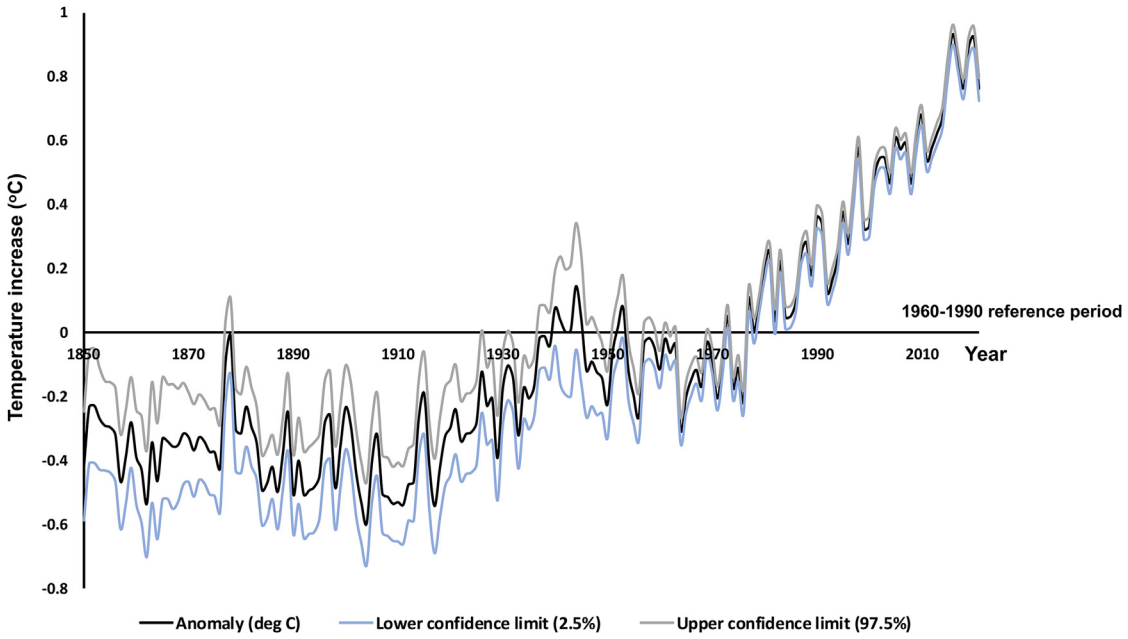

Figure 1. Global ocean temperature trends

Derived from the HadCRUT5 (ensemble means and uncertainties) dataset of Morice et al. (2021)[46] that uses a combination of sea-surface temperatures and near-surface air temperatures over land and compared against the 1961–1990 reference period.

The gradual increase in global temperatures varies geographically, as such, there is a crucial need to examine local temperature trends in each major aquaculture-producing country together with the temperature and thermal tolerance ranges of both pathogens/parasites and their fish and shellfish hosts (Table 1). Such information will allow refined predictions of future changes in disease prevalence and severity.

Increased temperatures are the best understood consequences of climate change in terms of aquatic diseases.[47,48] Higher temperatures have effects on both host and parasite/pathogen normally to the detriment of the host and the relationship is exacerbated if outside their thermal range; Figure 2). In terms of pathogenic bacteria, higher temperatures often stimulate multiplication rates and may also lead to higher virulence (i.e., disease causing ability) caused by changes in the expression profiles of virulence factors such as hemolysins, siderophores, and a variety of endo- and exo-toxins (e.g., refer to studies by Lages M.A. et al., Lopez-C.G. et al., and Lee Y. et al.[49–51]). Hosts may become immune suppressed by direct effects on immune cells or indirectly, as dysbiotic changes in their microbiomes can potentially inhibit the immune system (e.g., refer Liang Q. et al. and Hembron P.S. et al.,[52,53] see also Case study #1 in this review). As a rise in winter sea surface temperatures may permit pathogen multiplication, this can result in all-year round infections,[47] perhaps initiating emerging diseases, or, infectious agents changing their host and geographic ranges.

Examples of how elevated temperature associated with climate change can affect the prevalence and severity of aquatic diseases in fish, shellfish, and seaweeds are shown in Table 2. Three of these examples are discussed further in the following text and in Case studies #2–6. For salmonid fish, the interaction between temperature and ectoparasitic sea lice, *L. salmonis* has been investigated using a range of approaches from experimental through to modeling (Table 2).[54,55] Considering the interaction between the parasite, host, and environmental temperature using an experimental approach revealed that increasing temperatures between 10°C and 22°C reduce the growth, condition, and survival of Atlantic salmon post-smolts exposed to varying infestation levels of lice/fish ranging from zero, low (1.4 lice per fish) to high (*ca.* 6.8 lice per fish) levels.[55] Collectively, these and other data may suggest that control of lice affecting salmon farming in warming waters in the Northern hemisphere may become increasingly difficult in coming years.

Abalone populations in the Pacific Ocean from Mexico to California have been decimated by a condition termed withering syndrome (Table 2). The cause of this disease is an intracellular bacterium that is rickettsiales-like.[90–92] Abalone farming can be a highly lucrative venture as these are some of the highest value shellfish and declines in the populations of black and red abalones of the Californian coastline are attributed, at least partially, to this chronic condition that occurs as epizootics following abnormally high-seawater temperatures associated with El-Niño-southern oscillation (e.g., refer to studies by Tissot B.N. and Moore J.D. et al.[93,94]). Abalones adapted to cool water temperatures are more susceptible to withering syndrome than those with higher thermal ranges.[95]

Putative effects of temperature on multicellular parasites can be difficult to assess as some have multiple life history stages to enable them to finally reach their definitive host, or they may employ reservoirs of infection. For example, digenean parasites of marine animals often have sea birds or fish as their definitive host and various invertebrates, including crustaceans and molluscs, as intermediate hosts. The trematode *Microphallus similis* is a parasite of sea birds (e.g., gulls) and has two intermediate hosts normally littorinid snails and various crabs including the common shore crab, *Carcinus maenas*.[96,97] Changes in the distribution of these intermediate hosts resulting from climate change, or the

Table 1. Pertinent examples of pathogens and parasites affecting marine aquaculture and their temperature ranges, optimal conditions, and critical thresholds affecting their environmental survival

Disease	Pathogen/Parasite (taxonomy)	Hosts	Temperature range (°C)	Optimum temperature (°C)	Thermal tolerances (°C)
AHPND	*Vibrio parahaemolyticus* (G- bacterium)	Shrimp	5–37	20–35	<5–10 & >35-40
Bacterial kidney disease	*Renibacterium salmoninarum* (G+ bacterium)	Salmonid fish	5–22	15–18	<0–4 & >20-25
Bonamiasis	*Bonamia ostreae* (Haplosporidia)	Bivalve molluscs	4–25	12–20	<5–10 &>25-30
Dermo disease	*Perkinsus marinus* (alveolate)	Oysters	>2-28	25	<2
Infectious salmon anemia	ISA virus	Salmonid fish	3–24	10–15	>25
MSX disease	*Haplosporidium nelsoni* (Haplosporidia)	Oysters	5–20	20–22	<4 & >20
Necrotizing hepatopancreatitis	*Hepatobacter penaei* (G-bacterium)	Shrimp	25–32	28–30	<20–25 & 35-40
Salmon louse	*Lepeophtheirus salmonis* (copepod)	Salmonid fish	3–24	10	<3 & >24 (high loss)
White spot disease	White spot syndrome virus	Shrimp	0–50	25–28	>50

appearance of newly arrived non-native snails or crabs, could interrupt the life cycle of the parasite.[98] This is likely to occur as the parasite is a host generalist. There are reports of temperature effects on parasite emergence in marine trematodes—for example, Thieltges and Rick[99] examined the life history of the trematode, *Renicola roscovita*, a parasite of bivalves in the North Sea. The periwinkle, *Littorina littorea*, is the first intermediate host of *R. roscovita* and bivalves, such as mussels and cockles, are the second intermediate host that become infected when cercariae are released. Mesocosm experiments revealed that cercariae infectivity is optimal at 20°C (August temperatures) and the authors concluded that warmer summers in this region will enhance the transmission of *R. roscovita* with resulting ecosystem effects.

Magalhães et al.[100] examined how predicted changes in climate (salinity, pH, and temperature) in Europe may affect the echinostome *Himasthla elongata* in its second intermediate host, the edible cockle, *Cerastoderma edule*. Overall, higher infection success in cockles was found in all three variables under climate change predictions. For example, parasitized cockles at the higher temperature regime of 22°C showed a greater amount of cellular damage because of reduction in their antioxidant levels than those held at 17°C.

Although the focus of this review are diseases of fish and shellfish, brief mention is made of other mariculture products, namely seaweeds, and environments where juvenile fish reside such as coral reefs and seagrass meadows, which are also impacted by climate-driven diseases (Table 2). Seaweed cultivation and harvesting are of increasing importance as sources of various chemicals (e.g., agar and carrageenan), nutraceuticals, cosmetics and feed supplements,[101] and as a tool for carbon sequestration. Disease limits production of seaweeds and examples of pathogens can be found in all the major taxonomic groups including bacteria.[89,102] Two such conditions probably caused by bacteria have been linked to unusually high temperatures including bleaching in the red alga *Delisea pulchra*[83] and "ice-ice" disease in numerous seaweeds.[88] In *D. pulchra*, the disease is associated with dysbiosis where opportunistic bacterial pathogens including *Alteromonas* sp., *Aquimarina* sp., and *Agarivorans* sp. prevail.[84,103] Notably, *Aquimarina* spp. are involved in epizootic shell disease in lobsters that is also triggered by raised sea temperatures (See Table 2 and Case study #5 for further detail). These observations suggest that opportunists such as *Aquimarina* spp. and other members of the phylum Bacteroidota could emerge as threats to a wide range of marine organisms in response to changes in oceanic conditions.[75,104]

Seagrass meadows are vital nurseries for juvenile fish and shellfish[105] and disease-attributed losses can affect these animals. Seagrasses are declining worldwide, and higher temperatures are considered the proximal driver of disease outbreaks, e.g., *Labyrinthula zosterae*, a protistan pathogen of common eelgrass, *Zostera marina*.[106]

Coral reefs provide key habitat to many species of fish and invertebrates and their loss due to climate change and other anthropogenic stressors can have a knock-on effect on fisheries. Coral reef deterioration and bleaching (both microbial and stressor induced) are linked intimately with increased temperatures and predicted to become more prevalent and intense with climate change. Between 1992 and 2018, coral disease prevalence trebled to ~9.9% globally according to a meta-analysis of disease prevalence, coral coverage, and sea surface temperatures.[107] In the same study, 76.8% of global coral reefs are modeled/predicted to be infected/diseased by 2100. If realized, such levels of disease in coral reefs would have devastating consequences for many ecosystem services, notably food provision (biodiversity and productivity[108]) and carbon sequestration.[109]

Of course, not all diseases are likely to become more prevalent in a changing climate as some pathogens/parasites are negatively affected by rising temperature. For example, winter ulcer disease of Atlantic salmon caused by a bacterial consortium (e.g., *Moritella viscosa*, *Aliivibrio wodanis*, and *Tenacibaculum* sp.) occurs in water at 7–8°C or below in Norway[110] and so changes in water temperature now and in the future, may decrease its significance as a key disease in winter months. Other diseases in temperate-Arctic regions that affect fish during the winter

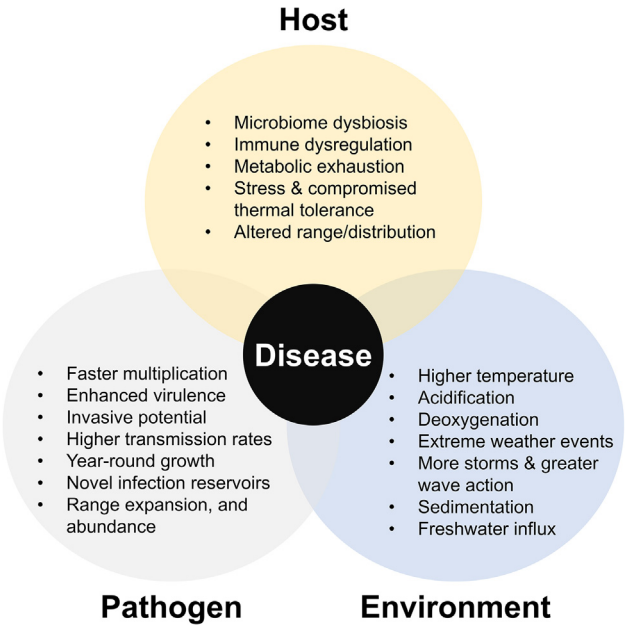

Figure 2. Environmental, host and pathogen/parasite features that co-exist to promote disease outbreaks

include cold water vibriosis caused by *Vibrio salmonicida*.[111] The disease does not typically occur at temperatures above 10°C, which correlates with the temperature profile for siderophore (iron-binding) production—a key virulence factor for this bacterium.[112]

CHANGES IN OTHER ENVIRONMENTAL PARAMETERS AND THEIR POTENTIAL EFFECT ON PREVALENCE AND SEVERITY OF INFECTIOUS DISEASES

Beyond temperature, climate change has other profound effects including sea level rises, ocean acidification, deoxygenation, and increased frequency of storms resulting in storm surges, precipitation resulting in freshwater runoff changing salinity, and pollutants leeching from the land into coastal regions (Figure 2). These are likely to fuel changes in disease occurrence particularly in vulnerable coastal regions, but they are much less understood as drivers than temperature.[113]

White spot disease of crustaceans is an indisputable threat to shrimp cultivation worldwide, and by 2012, was estimated to have cost US$ >18 billion in losses.[114] Shinn et al.[18] attribute annual losses of US$ 4 billion per year between 2009 and 2018. This disease is caused by the white spot syndrome virus (WSSV) that has a wide host range including shrimp and crabs.[115] Higher temperatures favor viral multiplication in general; however, a recent overview concluded that temperatures between 25°C and 28°C favored multiplication of WSSV but above 30°C caused declines in this process.[116] A further observation was that stress caused by rapid temperature and salinity fluctuations was the principal predisposing factor to disease progression rather than higher temperatures alone. Because climate change can alter salinity levels rapidly due to increased incidence of storm activity, such events may be more common in future. Other drivers such as eutrophication, may also play a part in promoting this disease especially in coastal/photic zones.

Bivalves are vulnerable to acidification as their calcium carbonate containing shells are a major barrier to disease-causing agents gaining entry to internal tissues and as a defense to predation.[117] Shells lacking strength and rigidity are potentially more likely to be damaged by wave action resulting in opportunistic pathogens gaining entry to soft underlying tissues[116] although this requires experimental evidence of such correlations. While there are numerous studies examining various scenarios of ocean acidification including changes in physiological processes, only a few have attempted to determine the likely effects of predicted changes to disease susceptibility and/or resistance (e.g., refer to studies by Schwaner C. et al. and Clements J.C. et al[118,119]).

CLIMATE CHANGE AND IMMUNE SYSTEM MODULATION IN FISH AND SHELLFISH

The phagocytosis and subsequent intracellular destruction of pathogens and parasites is a fundamental defense mechanism of all multicellular animals. In shellfish (crustaceans and molluscs), the cells involved are termed haemocytes (because they are housed in the main body cavity the haemocoel) while in fish the equivalents are granulocytes, monocytes/macrophages and other professional phagocytes. Shellfish also depend on a range of antimicrobial factors including antimicrobial peptides (AMPs), lectins, lysozymes, and in some cases, the products of the prophenoloxidase activating system.[117,120] Fish also produce AMPs, lectins, and lysozymes but have additional molecules including immunoglobulins (antibody), a range of immunoregulatory cytokines, and terminal factors of the complement system such as the membrane attack complex, to deal with invasive pathogens and parasites.[121,122]

Table 2. Examples of changes in disease prevalence and/or severity linked to high environmental temperatures

Host(s)	Disease and (causative agent)	Location	Change reported	Key reference(s)
Various marine fish and abalones (*Haliotis rubra* and *H. laevigata*)	Gill damage in fish (diatom, *Chaetoceros coarctatus*)	South Australia	High (*ca.* 27°C) and variable water temperatures (5°C above average) causing mortalities in fish and abalone. Causative agent may be associated with algal blooms. Fish affected in shallow waters near and/or above thermal tolerances	Roberts et al.[56]
Atlantic salmon (*Salmo salar*)	Salmon louse (parasitic copepod, *Lepeophtheirus salmonis*)	Tank-based experiment	Increases in temperature result in faster development of infective stages of parasite	Hamre et al.[54]
Salmon (wild and farmed)	*L. salmonis*	Pacific coast, B.C., Canada	Climate change models show future increase in abundance of sea lice resulting in current control strategies becoming ineffective	Godwin et al.[55]
Salmon (*S. salar*) (migrating wild smolts)	*L. salmonis*	Model based on wild smolt from river Vasso in Norway migrating in coastal waters	Models predict that with increasing temperature lice will cause more damage to migrating salmon smolts and that those fish at the southernmost margins are therefore most vulnerable to the effects of sea lice.	Vollset et al.[57]
Atlantic salmon (*S. salar*)	Gill disease (several non-infectious and infectious conditions)	Various including Chile, Canada and N. Europe	Prevalence of gill damage linked to high water temperatures. See Case study #6	Foyle et al.,[58] Jones and Price[59]
Striped bass (*Morone saxatilis*)	Dermal mycobacteriosis (*Mycobacterium* spp.)	Chesapeake Bay region, USA	Both healthy and diseased bass show a 3-fold increase in mortality with rises in summer surface temperatures of 26°C–28ºC inferring that these fish are close to their thermal tolerance	Groner et al.[60]
Eastern oyster (*Crassostrea virginica*)	Dermo disease (*Perkinsus marinus*)	Gulf of Mexico to Chesapeake Bay, USA	Northwards spread of Dermo linked to increased winter sea surface temperatures	Cook et al.[61]
Pacific oyster (*Crassostrea gigas*)	Summer mortality (*Vibrio* sp.)	Aquarium based experiments with oysters from the Wadden Sea, Germany	Thermal stress and spawning status decrease the disease resistance to vibriosis resulting in higher mortality at 21°C than 17°C	Wendling and Wegner[62]
Pacific oyster (*C. gigas*)	Mass summer mortality (*Vibrio alginolyticus*)	Shandong Province, China	High summer temperatures result in >60% mortality linked to more rapid growth and upregulation of virulence genes in vibrios. No evidence of involvement of other pathogens such as ostreid herpes virus 1	Yang et al.,[63] Li et al.[64]
Pacific oyster (*C. gigas*)	Pacific oyster mortality syndrome (POMS) (polymicrobial, ostreid herpes virus 1 and *Vibrio* spp.)	Europe	A polymicrobial condition where initial infection by the virus causes immune suppression resulting in individuals being more susceptible to secondary bacterial infections by vibrios. Epizootics occur between 16°C and 24°C (See Case study #4)	Petton et al.,[65] Petton et al., 2021,[66] de Lorgeril et al.,[67] Combe et al.,[68] Destoumieux-Garzón et al.,[69] and Oyanedei et al.[70]
Abalone (*Haliotis tuberculata*)	Vibriosis (*Vibrio harveyi*)	Normandy & Brittany, France	Long term higher summer temperatures associated with abalone mortality. Increase in temperature above 17°C associated with vibriosis	Travers et al.[71]

(*Continued on next page*)

Table 2. *Continued*

Host(s)	Disease and (causative agent)	Location	Change reported	Key reference(s)
Black abalone (*Haliotis cracherodii*)	Withering syndrome (Rickettsiales-like bacteria; *Candidatus* Xenohaliotis californiensis)	Laboratory and field-based (California coast)	Animals exposed to high temperature showed disease in tank-based experiments. Extreme environmental temperatures favor disease progression in intertidal zone	Ben-Horin et al.[72]
American lobster (*Homarus americanus*)	Epizootic shell disease (bacterial dysbiosis)	Massachusetts, USA	Increase in temperature results in microbial dysbiosis on shell resulting in erosion, cuticular damage and mortality. (See Case study #5)	Groner et al.,[73] Ishaq et al.,[74] Rowley and Coates,[75] and Shields[76]
Shrimp (including *Penaeus vannamei*)	Acute hepatopancreatic necrosis disease [AHPND, = early mortality syndrome] (*Vibrio* spp. including *V. parahaemolyticus*)	Mexico, Thailand, USA	Strains of vibrios that produce the toxins $PirAB^{vp}$ cause this disease that is characterized by high mortality. Both salinity and high temperature modify the expression of the PirA gene responsible for toxin generation that cause AHPND (See Case study #3)	Estrada-Perez et al.,[77] Lee et al.,[78] and Tran et al.[79]
Shrimp (*Penaeus setiferus* and *Penaeus aztecus*)	Shrimp black gill (ciliate, *Hyalophysa lynni*)	Texas Gulf coast, USA (Gulf of Mexico)	High temperatures (>20°C) and lower salinity (<10 ppt) caused by precipitation events, are environmental drivers of this condition	Swinford et al.[80]
Blue crab (*Callinectes sapidus*)	(dinoflagellate, *Hematodinium perezi*)	Atlantic seaboard, USA	Water temperatures of >25°C and salinity (>30 ppt) favor parasite development in its host. Milder winters facilitate parasite survival	Shields,[76] Huchin-Mian et al.,[81]
Snow crab (*Chionoecetes opilio*)	Bitter crab disease (dinoflagellate, *Hematodinium* sp.)	Newfoundland, Eastern Canada	Warm water masses raising temperatures from −1.4°C to 2.0°C spurred epizootics	Shields et al.[82]
Red seaweed (*Delisea pulchra*)	Bleaching (bacterial dysbiotic condition associated with appearance of *Alteromonas* sp., *Aquimarina* sp. and *Agarivorans*)	Subtidal habitats near Sydney, Australia	High summer temperatures result in bleaching events especially in shallow water. High temperatures appear to drive reduction in levels of host's antibacterial chemicals (halogenated furanones) resulting in bacterial dysbiosis and possible immune suppression	Campbell et al.,[83] Kumar et al.,[84] and Hudson et al.,[85]
Seaweeds (various species)	Ice-ice disease (bacterial dysbiotic condition?)	Widespread including India, China and Zanzibar	Disease appears in hot and dry season. Aquarium based studies show temperatures between 33°C and 35°C trigger disease above thermal tolerance level of some seaweeds	Largo et al.,[86] Largo et al.,[87] Arasamuthu et al.,[88] and Ward et al.,[89]

As fish and shellfish are poikilotherms, their immune systems are influenced by environmental changes such as temperature, salinity, and pH and so climate change is likely to affect their health status. In general, abnormally high and low temperatures act as immune suppressors but this depends on the duration of stress (acute, subacute, and chronic), the thermal tolerance ranges of individual species, and the presence of other environmental stressors such as rapid fluxes in salinity caused by storms and chemical contaminants in runoff.[123] Changes in temperature[124–126], pH[127], and salinity[128,129] have been shown to adversely affect the immune systems of both fish and shellfish and the literature (especially that pertaining to teleost fish) is replete with examples that have been reviewed in detail elsewhere.[130–134] Some pertinent examples are highlighted in Table 3 to illustrate how climate change may affect the immune capacity of shellfish, while examples in fish are discussed in the following text.

Most fish have body temperatures that are isothermal with the surrounding environment. In general, fish at low temperatures, such as those in Arctic-temperate winter, rely more on their non-specific defenses (e.g., phagocytosis, complement fragments, lysozyme, and AMPs) than those as part of their specific defenses (e.g., antibody generation and cell mediated immunity delivered by T cells). Hence antibody levels and some other key regulatory immune molecules, such as cytokines, decrease with declining temperatures in a wide range of fish including those in Arctic, temperate, and tropical climes.[134] With an impaired specific immune system, vaccination in winter temperatures becomes impractical and several cold-water adapted pathogens, including cold-water vibriosis, exploit the impaired immune system.

Fish within their optimal thermal range are more likely to have a "balanced" immune system relying on both non-specific and specific reactions to maintain homeostasis. Short-term (hours-days) sudden elevation in temperature triggers a stress response, while in the

Table 3. Examples of environmental immune modulation in shellfish

Model species	Environmental parameter(s)	Immune and other assays	Overall conclusions	Reference
European abalone (green ormer) (*Haliotis tuberculata*)	Increased temperature as found in summer months	• Phagocytic activity • Phenoloxidase activity • Superoxide dismutase (marker of intracellular antimicrobial generation)	High summer water temperatures coinciding with gametogenesis, causing lower immune capacity, as evidenced by assays, resulting in higher chance of vibrio infections	Travers et al.[71]
Pacific oyster (*Crassostrea gigas*)	Temperature stress (17°C–19°C)	• Phagocytic activity of hemocytes • Survival following vibrio infections	Post-gametogenesis oysters are more susceptible to vibriosis due to immune depression caused by gametogenesis exacerbated by higher temperatures	Wendling and Wegner[62]
Thick shell mussel (*Mytilus coruscus*)	Short term pH and salinity stress	• Hemocyte mortality and counts • Phagocytic activity • Lysosomal content of hemocytes	Decrease in pH results in lower hemocyte counts, phagocytosis and lysosomal content. Overall, high and low salinities and low pH have negative effects on hemocytes	Wu et al.[135]
Blue mussel (*Mytilus edulis*)	Combinations of acidification (apparent pH 7.99 reduced to 7.65) and long term (6 months) temperature stress (12 increased to 15°C)	• Total hemocyte counts • Phagocytic ability • Hemocyte respiratory burst assay	Increase in hemocyte number (a marker of stress?) at higher temperatures. Acidification has limited effects on own or in combination	Mackenzie et al.[127]
American lobster, (*Homarus americanus*)	Temperature stress (16 *vs.* 24°C)	• Phagocytic activity • Hemocyte viability *in vitro*	Immune system compromised above 21°C. Temperature affects viability of hemocytes *in vitro*.	Steenbergen et al.[136]

longer-term, high temperatures lead to immune suppression particularly associated with specific immunity.[137] Temperature shifts in terms of immune reactivity have been extensively studied in a wide range of fish of both commercial and non-commercial significance (see Table 1 in Scharsack and Franke; [134] for examples). There are, however, only a relatively small number of long-term studies likely to reflect climate change scenarios simply because of the difficulty in conducting such experiments in many research aquaria. A valuable example of such long-term studies is that of Magnadottir et al.[137] who carried out an extensive 12-month aquarium-based study on the effect of temperature on Atlantic cod (*Gadus morhua*) where one group of fish was held at 1°C (winter temperature), a second group at 7°C (the optimum temperature for cod), and another at 14°C (at the margins of thermal tolerance for this species). As expected, antibody levels were highest in the 14°C group but hemolytic activity (a marker for the complement activated part of the non-specific immune system) was lowest in this same group and highest in fish at 7°C. As these data were only collected at the end of the experiment (i.e.,12 months) there is no temporal information on the dynamics of these parameters. Nonetheless, fish in both the 1°C and 7°C groups showed the highest growth rates and the overall conclusion was that 7°C was the optimum temperature for Atlantic cod in terms of their immune capacity.

Fish at high temperatures, including those near or above their thermal range, also show evidence of immunosuppression with a generalization of higher dependence of such individuals to rely on their non-specific, as opposed to their specific, defenses.[134] For example, to simulate the effects of climate change, Zanuzzo et al.[138] placed Atlantic salmon (*S. salar*) in aquaria with incremental temperature increases of 1°C per week from 12°C to 20°C and 4 weeks later measured multiple parameters associated with non-specific defenses. They found no evidence of major deleterious effects of these high temperatures on several markers of non-specific immunity (e.g., plasma lysozyme and complement activity) but the short-term nature of their study and lack of infection trials (pathogen challenge), are potential limitations in their experimental design.

CASE STUDIES

This section of our review employs six examples of how climate change may affect aquatic diseases taken from diverse geographic areas, pathogen types and hosts.

Case study #1: Will climate change increase disease susceptibility in fish and shellfish via microbiome perturbation?

In fish and shellfish, host-associated microbiomes are abundant within the intestinal tract and are also associated with all other mucosal surfaces (i.e., gills and skin) and with hemolymph (blood) in invertebrates.[135,139] Microbiota broadly influence host physiology, including nutrient acquisition, metabolism, immune function, and pathogen defense.[140] Relationships between microbiota, the host immune system, and pathogenic agents are complex, dynamic, and can strongly influence disease outcomes.[141] In fish, commensal microbes influence the immune system development and maturation[130,142] while diverse interactions between immune cells and microbiota maintain their symbioses.[141,143] Less is known for shellfish, although crustacean hemolymph microbiota interact with host immune response[144] and microbial metabolites can influence the immune system of crustaceans.[145]

Healthy microbiomes are generally diverse, ecologically stable, and respond dynamically to environmental and host fluctuations.[140,141,146] Commensal and mutualist microbiota can inhibit pathogen colonization, including via competition, niche modification, and/or production of antimicrobial agents.[143] Microbiomes, however, are sensitive to disruption by environmental stressors, often leading to reduced diversity and impaired function (dysbiosis), which can subsequently allow potential pathogens to colonize and proliferate.[147] Pathobiomes can be highly complex, consisting of multiple potential pathogens, and their products, rather than single causative agents of disease.[148] Signatures of dysbiosis have been identified in fish microbiomes following exposure to antimicrobial agents and environmental stressors.[149] These typically include reduction in microbiome diversity and beneficial (anti-inflammatory) bacteria, together with an increased abundance and/or pathogenic activity of opportunistic microbes, influenced by species and mucosal surface.[147,149–151]

Stressors associated with climate change are likely to induce microbiome perturbation and disrupt the critical relationship between microbiome and immune system, subsequently increasing disease risk particularly in aquaculture settings. There is considerable evidence that finfish and shellfish microbiomes are highly sensitive to temperature changes across a range of species. Within poikilothermic hosts, in particular, external temperature directly impacts the microbiome, imposing differential selection pressures within the community and altering microbe physiology and function.[149,152] In addition, temperature-induced changes in host metabolism, immune system, gut function, cortisol, and mucous production, for example, are likely to influence the microbiome indirectly. The effects of temperature change on fish and shellfish microbiomes vary depending on the nature of the stressor. Relatively small thermal fluctuations often cause transitory microbiome changes, whereas more chronic, mild temperature change can induce alternative, but stable, microbiome states.[149,152] Notably, microbiomes have an extensive capacity to develop tolerance of temperature stress, which may also confer benefits to the host.[153] More severe thermal stressors, such as those associated with heatwaves or prolonged temperature elevation, however, are likely to overwhelm this resiliency and induce dysbiotic transitions characterized by reduced diversity and increased presence of opportunistic pathogens.[147–149,151]

Only a small number of studies have to date directly linked temperature stress with dysbiotic transition and increased pathogen abundance in the microbiome and even fewer have documented clinical signs of disease. Some of these studies with shellfish and fish are briefly discussed in the following text. A rapid rise in temperature simulating a heat wave induced changes in the microbiome of Pacific oysters (*C. gigas*) with significant increases in vibrios including *V. harveyi* that resulted in high levels of host mortality.[154] Also, Pacific oysters that had been acclimated to high temperatures, were protected against subsequent rapid temperature stress resulted in lower mortality and vibrio loads than those seen in controls.[155] In another mollusc, the Yesso scallop, *Mizuhopecten yessoensis*, heat stress increased the abundance of pathogenic bacteria, *Tenacibaculum* and *Mycoplasma* in the gut microbiome as well as intestinal pathologies.[156] In fish, increased temperature elevated several opportunistic pathogens in the gut of greater amberjack (*Seriola dumerili*) and this was directly associated with reduced host productivity.[157] Similarly, heat stress in chum salmon (*Oncorhynchus keta*) increased the abundance of several opportunistic *Vibrio* spp.,[158] while estuarine Eastern striped grunters (*Helotes sexlineatus*) exposed to thermal pollution showed evidence of gut dysbiosis including increased prevalence of pathogenic *Photobacterium*.[159] Other studies have demonstrated that heat stress both disrupts the microbiome and exacerbates the effects of experimental pathogen exposure. For example, in the Pacific oyster, hard-shelled mussels and sea cucumbers, elevated temperatures increase susceptibility to experimental *Vibrio* infection and simultaneously induce dysbiosis in gut and hemolymph microbiomes.[160–163] These studies, however, have shown that dysbiotic microbiome changes rarely simply facilitate proliferation of a given target pathogen (i.e., *Vibrio*) but, rather, tend to be characterized by increased abundance of a number of opportunistic pathogens typical of the complex pathobiome state.[148] As such, causal mechanisms linking temperature stress to disease via microbiome disruption are not well established.

Aquaculture microbiomes are also likely to be sensitive to other environmental stressors directly or indirectly associated with climate change, including acidification, hypoxia, salinity, pollution, and nutrient availability. Although these stressors are known to alter fish and shellfish microbiomes (e.g. refer to studies by Xavier R. et al, Dang X. et al., Khan B. et al., and Zhong K.X. et al.[147,164–166]), research examining how microbiome disruption could enhance disease susceptibility has been scarce.[147,166] Interactive effects of combined stressors, newly emerging pathogens, as well as stressor-impacts on other aspects of host physiology all increase complexity and will likely exacerbate disease risk. Ultimately, research is required to better understand how functional changes during microbiome to a dysbiotic pathobiome affect host physiology, such as mucosal surface integrity and inflammatory and immune responses, which subsequently determine disease pathology. This knowledge may help us to better predict and mitigate disease risk in a changing world.

Case study #2: Vibrios are barometers of climate change in the oceans

Climate change influences the dynamics of *Vibrio* bacteria, a genus that includes pathogens of humans such as those causing cholera (e.g., toxigenic *V. cholerae*) and vibriosis (non O1/O139 *V cholerae*, *V. parahaemolyticus*, *V. vulnificus*, and *V. alginolyticus*)[167] and opportunistic pathogens in fish and shellfish (refer to Mohamad N. et al.[168]; see also Case studies #3 and 4 in this review). Vibrios are natural inhabitants

of estuarine, coastal, and marine environments and play an important role in the aquatic ecosystem and biogeochemical cycles.[169] They produce various macromolecule-degrading extracellular enzymes (including chitinase, proteases, and lipases) that degrade carbon substrates into nutrients.[170] Such characteristics allow for their rapid growth such as when climate change increases the level of river discharge with terrestrial nutrients.[171] Rising global temperatures, especially in oceans and coastal regions, create favorable conditions for the proliferation of vibrios relevant to human and animal health. Because these bacteria tend to grow and proliferate in warm, low-salinity conditions, and because vibrios are among the fastest growing bacteria studied to date, they have been dubbed a microbial "barometer" of climate change.[171] Changes in oceanic factors like salinity and sea surface temperatures impact the distribution and abundance of various *Vibrio* species, and compelling long term molecular studies underpin how climate influences the presence and abundance of these bacteria across global scales.[172,173] Life cycles of vibrios can also be complex. They can be found either free living in the water or surface attached to plankton and marine animals.[174] An increase in ocean temperature also increases the abundance of vibrios in plankton-associated bacterial community.[174–176]

As a result of these changes, we are now witnessing the emergence of what were once considered tropical bacterial pathogens into many colder temperate areas. Extreme weather events such as hurricanes[177] and heatwaves[178] can introduce *Vibrio* species into new areas and can greatly increase their concentration in the environment, where they can initiate infections of humans and other animals. Contact between humans and *Vibrio*-contaminated raw shellfish during times of higher-than-normal sea surface temperatures leading to disease appear to be increasing in occurrence.[179,180] Understanding the interplay between *Vibrio* and climate change is crucial for anticipating and mitigating potential human health risks and species under aquaculture cultivation such as oysters and shrimp. Comprehensive surveillance, research, and public health measures are essential to address the emerging challenges posed by the complex relationship between *Vibrio* bacteria and climate-driven environmental changes.

Case study #3: Climate change effects on *Vibrio* infections of shrimp

Acute hepatopancreatic necrosis disease (AHPND) is an emerging disease of shrimp in culture resulting in high mortality. It first emerged in China in 2009 and spread to Vietnam in 2010, Malaysia in 2011, Thailand in 2012, and Mexico in 2013[181] causing a substantial economic loss of USD 44 billion in 2010–2016.[181] *Vibrio parahaemolyticus* was the first species identified to be associated with AHPND. Genome analysis revealed that various *V. parahaemolyticus* isolates recovered from shrimp and pond water during AHPND outbreaks are genetically diverse, indicating that the disease-causing isolates are not clonal.[182] AHPND pathology is brought about by *Photorhabdus* insect-related (Pir) toxins encoded by the pVA1-type plasmid.[78,79,183] *Vibrio* spp. carrying such plasmids can cause AHPND including *V. parahaemolyticus*, *V. harveyi*, *V. campbellii*, *V. owensii*, and *V. punensis*.[184–186] Two toxin homologues to Pir toxins (A and B) are produced and secreted from these species and cause severe cell necrosis in the hepatopancreas (Figures 3A and 3B).

Environmental changes can have profound effects on toxin generation by these vibrios associated with AHPND. Expression of the PirA toxin is altered upon changes in temperature and salt concentration, suggesting the involvement of environmental factors to virulence and pathogenicity.[187] Interestingly, the pVA1-type plasmid spreads horizontally across the *Vibrio* genus by conjugative type IV secretion system encoded on the VA1-type plasmid.[188] The efficacy of conjugation is also regulated by environmental factors such as bacterial density, temperature, and nutrient levels.[188] In addition to impacting the abundance, spread, and species composition of vibrio community, climate change also seems to influence the spread of virulence plasmid like pVA1. Virulence-related genes, including genes involved in adhesion, biofilm formation, toxins, and type III secretion systems, are responsive to climate-dependent environmental cues like temperature, salinity, and pH. For instance, an increase in seawater temperature (from 21 to 27°C and 31°C) upregulates virulence gene expression, enhancing adhesion and biofilm formation in *V. parahaemolyticus*.[189] Expression of a thermostable direct hemolysin, a virulence factor with hemolytic and cytotoxic activity, is also upregulated in *V. parahaemolyticus* upon an increase in seawater temperature.[189] Expression of type III secretion system 2, essential for host colonization and immune modulation, in *V. parahaemolyticus* is also regulated by temperature and salt responsive histone-like nucleoid-structuring protein.[190] Taken together, the occurrence of AHPND during shrimp cultivation seems to be a result of complex interaction of multiple factors—species composition, virulence factors, environmental signals such as temperature and salinity, and host immunity.[189,190]

Case study #4: Pacific oyster mortality syndrome—A polymicrobial and multifactorial disease

Since 2008, massive mortality events have occurred yearly with the emergence of a disease called Pacific oyster mortality syndrome (POMS) severely affecting the oyster industry.[191–193] Within the two following decades, this disease has become panzootic affecting Pacific oyster populations all around Europe, North America, Asia, and the South Pacific.[194–201] POMS is associated with the recurrent detection of Ostreid herpesvirus type 1 μVar (OsHV-1 μVar) in moribund oysters[191–193] as well as strains of the bacterial genus *Vibrio*.[67,70,202–204] Infection by OsHV-1 is the initial step that leads the animal to an immunocompromized state resulting in a bacterial dysbiosis followed by a bacteraemia responsible for oyster death.[67] Several bacterial genera are involved in this secondary infection[67,205]; among them vibrios that behave as opportunistic pathogens causing hemocyte lysis.[70,204]

The risk of POMS outbreaks is dependent on complex interactions between host, pathogens, and environmental factors.[191,206–210] Among the environmental factors that influence POMS, temperature and pH are directly related to climate change. Other factors, indirectly linked to climate change must also be considered including food availability (primary production), energetic status of the host, and salinity. Here, we review how these factors individually affect POMS.

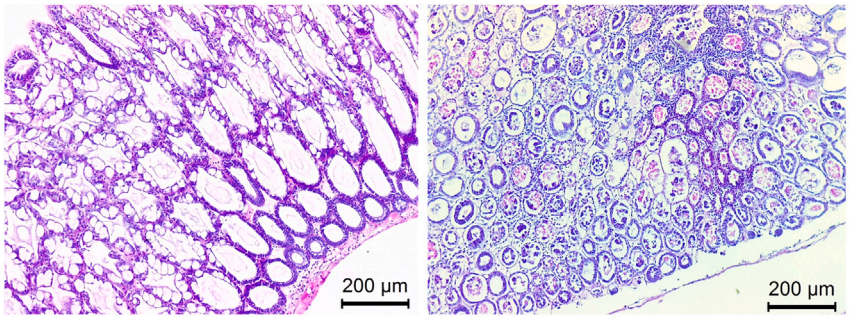

Figure 3. Damage to the hepatopancreas (HP) of shrimp infected with *V. parahaemolyticus* caused by toxins

(A) Normal histological structure of the hepatopancreas.

(B) Appearance of damaged hepatopancreas. Note tubule disruption.

Temperature, ocean warming, and marine heatwaves

In Europe, POMS epizootics occur when the seawater temperature is between 16°C and 24°C[206] Below 16°C, OsHV-1 infections can persist in latent forms or low-replication rates, as reported in other herpesviruses.[211,212] Then, reactivation of the virus can occur several weeks or even months after initial exposure, in response to increases in temperature above 16°C[207,213] Increased temperature results in increased viral replication and shedding by the host, reflecting increased growth metabolism.[214] When the seawater temperature reaches 24°C or more, epizootics are halted in different countries.[206,215–218] Consequently, seawater temperature clearly sets the start and the end of the epizootics in Europe. In Australia, the window of POMS permissiveness is higher, between 18°C and 26°C, even if the upper limit has never been established as clearly as in Europe.[215,216] Finally, in laboratory conditions, viral infections and mortality of oysters occur up to 30°C, which suggests that laboratory conditions differ from those encountered in the natural environment.[207,210]

Ocean warming primarily manifests through species poleward migration. On average, marine species have shifted approximately 30–60 km per decade since 1950.[219] In Europe, the Pacific oyster, originally introduced south of the river Loire in France, now extends its range to the southern regions of Norway, a putative consequence of global warming or translocation of living organisms.[220] In theory, the lower temperature limit is anticipated to be reached earlier in the year, thereby expanding the time window of POMS permissiveness. Conversely, in even more southerly regions of Europe, the upper temperature limit should be surpassed earlier and for more extended periods throughout the year, resulting in a contraction of the time window for POMS permissiveness. Hence, although POMS may advance northward, there is a potential for its gradual decline in southern regions attributed to excessively high temperatures during certain parts of the year. Marine heatwaves, which consist of prolonged periods of abnormally warm water at a particular location,[221] can also influence the expression of POMS. For example, mortality risk is associated with the rate of temperature increase in the weeks before reaching 16°C.[222] Given that marine heatwaves are also associated with rapid increases in temperature, they could increase POMS-related mortality risk as reported in other marine diseases;[223] however, this assumption needs further examination.

Ocean warming and marine heatwaves may increase POMS-related mortality risk through increased growth metabolism. In ectothermic organisms like oysters, increased temperature accelerates growth within the thermal tolerance window of the species. When these organisms are exposed to obligate intracellular parasites that depend on the cellular machinery of the host to replicate and proliferate, like viruses, it is likely that increased temperature promotes viral proliferation. This is clearly what happens with OsHV-1μVar.[207,210,214] Ocean warming and marine heatwaves may also disturb or modulate oyster physiology when the temperature is reaching the upper limit of thermotolerance. *C. gigas* can withstand a wide seawater temperature range. This process was characterized at the transcriptome level with an increase in immune defense genes expression and a drop of metabolomic activities after acclimatization at high temperature.[210] Catabolism, cell growth, and metabolites transport are impaired at 29°C when compared to 21°C. A non-optimal metabolomic environment for virus proliferation, combined with a higher antiviral response to the OsHV-1 μVar infection, leads to a decrease to POMS susceptibility (Duperret et al. unpublished). Furthermore, an overexpression of antiviral functions combined with DNA repair ability in animals acclimated at high temperature offering a better protection against virus exposure (Duperret et al. unpublished). Alternatively, ocean warming, and marine heatwaves, may increase POMS-related mortality risk through dysbiosis. For instance, increased temperature may influence the abundance or the virulence of opportunistic bacterial pathogens responsible for the secondary infection occurring during the disease.[160]

pH and ocean acidification

There is scant evidence of the impact of ocean acidification on the susceptibility of oysters to POMS. Indeed, the only study published to date suggests that short-term exposure to low pH increases the susceptibility of oysters to OsHV-1, and this coincides with lower activity of superoxide dismutase and basal activity of nitric oxide synthase—two enzymes that are involved in the host immune response.[224] This study leads to the conclusion that acute acidification of seawater is likely to induce modulation of immune response in oysters infected by OsHV-1μVar. However, longer-term experiments failed to reproduce this result (F. Pernet personal communication). Further developments should consider

long-term acclimation and multi-generational effects and examine other combined factors such as temperature to fully explore the impacts of ocean acidification POMS-related mortality risk.

Food availability and energetics

Bivalves are filter-feeders which directly rely upon phytoplankton for growth and reproduction. However, phytoplankton community composition and primary production is likely to decline under the effect of climate warming which favors ocean stratification and drought episodes.[225] These two phenomena limit nutrient supplies to phytoplankton in both the open ocean and the coastal zone.[226] The availability of phytoplanktonic food, directly influencing the energetic status of organisms, is one of the most influential factors on susceptibility to POMS[214] and on the biological responses of organisms to global climate change.[227–229]

Unrestricted food supply improves the physiological condition of oysters and lowers their susceptibility to disease, probably reflecting an allocation trade-off between energy available to mount immune response and energy for other fitness-related functions.[214,229] Food scarcity slows the growth and metabolism of the host on which the pathogen depends to proliferate, thus limiting viral proliferation, shedding and host mortality. Reduced primary productivity may mitigate POMS-related mortality risk by reducing host growth metabolism and impeding the ability of the virus to proliferate. However, if energy reserves are severely impaired, this protective effect can be overwhelmed, rendering the organism unable to mount an effective defense against the disease. Therefore, the indirect effect of climate change on primary production on POMS-related mortality risks remains unpredictable and will depend on the intensity of the induced stress. Beyond the quantity of food, the quality of food must also be considered. However, the effect of diet quality on susceptibility to POMS is unknown.

Salinity

Coastal areas where oyster farming is practiced are becoming more saline, reflecting decreasing freshwater inputs, and increasing sea-surface temperature fostering evaporation.[230] The potential impact of hyper-salinity on POMS-related risk of mortality is not fully understood. There is no relationship between the salinity gradient and OsHV-1μVar-associated oyster mortality risk in the field, regardless of the spatial scale of investigation and salinity ranges.[223,231,232] Nonetheless, findings from a laboratory experiment where oysters were acclimated to a range of salinities and subsequently exposed to OsHV-1μVar, reveal that the survival rate of oysters facing OsHV-1μVar at 35‰ is approximately 20% greater than those at 25‰.[233]

Developmental plasticity and rapid adaptation to POMS in a global change context

Recent work demonstrates that genetic and epigenetic mechanisms contribute significantly to oyster's resistance to POMS.[234,235] While epigenetics contribute alone to developmental even transgenerational plasticity,[234] genetic and epigenetic lead to rapid adaptation.[235] These phenomena will continue but global change brings new stressors leading to the needs of multidirectional adaptation for which it is difficult to predict the outcome. Interestingly, several works performed on various models showed that exposure to one stressor can promote an enhanced response toward other stressors.[236] This mechanism of cross-tolerance is still misunderstood but may rely on molecular pathways sharing key actors enabling the upregulation of several pathways in response to a single stressor.[236] These processes would be pre-adaptive in the global change context but this needs to be further studied in the context of oyster/POMS/global change interaction.

Conclusions

The fate of POMS in the face of climate change remains uncertain for three reasons. Firstly, certain climate change factors affecting POMS, such as deoxygenation, have not been examined, and others like ocean acidification and salinity lack comprehensive studies for robust conclusions. Secondly, existing research has predominantly analyzed individual factors in isolation, despite their simultaneous occurrence and potential interactions impacting POMS. Lastly, assessments of climate change factors on POMS have predominantly focused on short-term effects, emphasizing acclimation, without addressing the potential for long-term adaptation. Consequently, it is imperative to embark on a future research path that integrates multiple drivers and adopts a long-term perspective under ecologically realistic conditions.

Case study #5: Epizootic shell disease in the American lobster

The American lobster (*Homarus americanus*) is afflicted by an environmental disease known as epizootic shell disease (ESD). It is caused by bacterial dysbiosis (i.e., an altered bacterial community) on the cuticle that results in severe chitinoclastic damage to the shell of afflicted animals (Figure 4A). The disease is strongly correlated with increasing temperature from climate change and is exacerbated by environmental contaminants known as alkylphenols.[76,237] A recurring suite of bacteria is associated with the disease. This includes *Aquimarina macrocephali* var. *homari* and several other bacterial taxa (*Jannaschia*, *Hirschia*, *Pseudalteromonas*, and *Thalassobius*) that are present at higher prevalence and relative abundance levels on diseased lobsters.[238,239] These bacteria are chitinoclastic, but they appear to require a portal of entry through the cuticle to effect disease.[240] Our current understanding of the etiology is that increased temperatures due to climate change negatively affect host defensive responses, with widespread contaminants (alkylphenols, such as bis-phenol A) interfering with tyrosine-crosslinking (i.e., sclerotisation), thereby lengthening the hardening process after moulting by as much as three times longer than normal.[241,242]

With the longer shell hardening time, the weakened cuticle becomes highly susceptible to colonization by chitinoclastic bacteria which then grow into the exocuticle to form a dysbiotic community. Longer hardening times also make lobsters more susceptible to injury, thereby

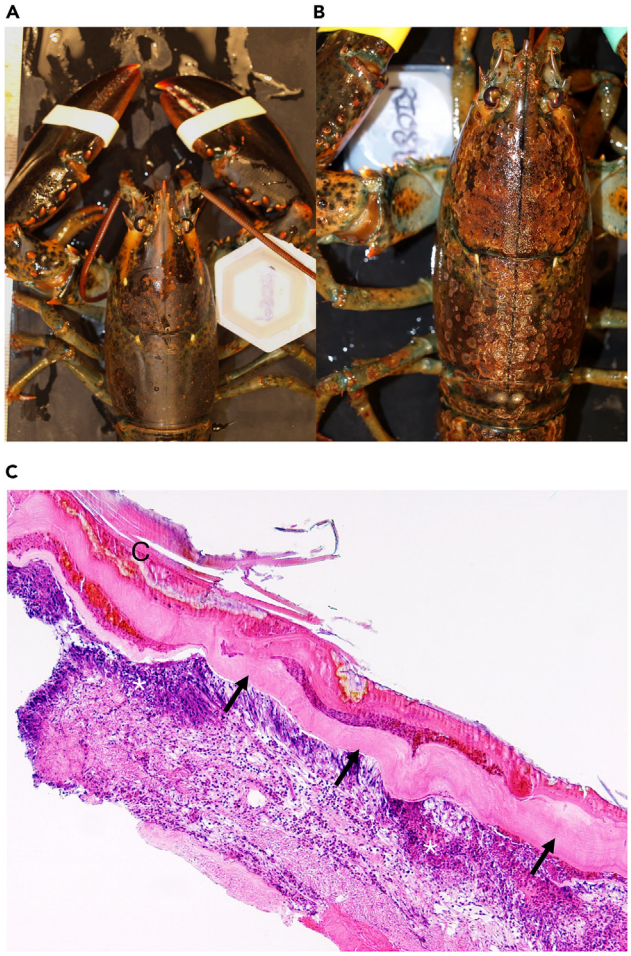

Figure 4. American lobsters (*Homarus americanus*) with epizootic shell disease

(A) Lobster from Rhode Island with a light infection manifesting as pinprick like lesions on the dorsal carapace.

(B) Lobster from Rhode Island, USA with a heavy infection manifesting as coalesced lesions covering >30% of the carapace and limbs.

(C) Histological section through the dorsal carapace of a lobster from the Gulf of Maine showing a large "pseudomembrane" (unlabelled arrows) and intensive cellular infiltration (*) by hemocytes below the affected cuticle (C).

increasing the likelihood of creating a portal of entry for invading bacteria. Ultimately, the dysbiosis can survive at 4°C, thrive at 12°C, and proliferates rapidly at temperatures ≥18°C.[243] Lobsters die at moult presumably from the adherence of pseudomembranes that form on to epidermis prior to ecdysis (Figure 4C). The pseudomembranes form in areas of damage to the shell, where the resulting lesions can form over more than 30% of the cuticle (Figures 4A and 4B).

ESD has seriously impacted the commercial lobster landings in the southern New England fishery, the third largest lobster fishery for the American lobster, after the Gulf of Maine and Canadian fisheries. The southern New England fishery has not recovered from the influence of this disease. The disease imposes substantial mortality risk to egg-bearing females, which experience a 12× to 16× higher risk of mortality than unaffected lobsters.[244] Female lobsters do not moult annually and, therefore, cannot moult out of the disease before it progresses to an advanced state. This results in a sharp, sustained decline in females, their egg production, and recruitment over time.[73,237,245,246]

ESD has been found at low levels in the Gulf of Maine where sea temperatures are much lower than in the Southern New England fishery. Nonetheless, it has been steadily increasing over the last two decades and is now found at modest levels, <10%, in legal-sized females in the region.[247] Its prevalence in "oversize" females (>126 mm carapace length), however, has been reported at 10–20% and these females may moult less frequently than legal (89–126 mm carapace length) females. The strong association between rising temperature and ESD has promoted surveillance of the disease in the Gulf of Maine. Temperature projections indicate that bottom temperatures ≥12°C are already present in many shallow reaches in the Gulf, particularly in the "Down East" region closest to the Bay of Fundy, and these areas should be targeted for additional surveillance over time.[248]

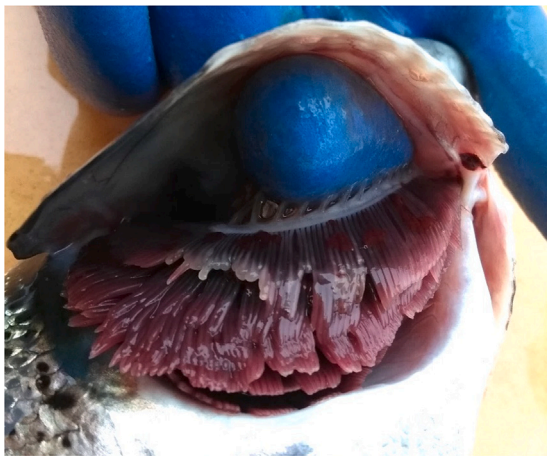

Figure 5. Gross gill appearance—an example of gill disease in Atlantic salmon, *Salmo salar*

Case study #6: Gill health in Atlantic salmon in a warming world

Fish gills are an organ of only one cell layer, consisting of epithelial and mucus producing cells that separates the external environment from the internal bloodstream. They have many critical functions, including respiration, osmoregulation, excretion of nitrogenous waste, pH regulation, hormone production, and protection of internal fish organs against the environment.[249] In parallel to their physiological functions, the gill is recognized as an important immune organ because of its role as a major mucosal barrier. Gill mucus is rich in antibodies, antimicrobial peptides, and signaling molecules that can regulate local immune responses. The interactions between salmon physiology, immunology, the environment, and infectious and non-infectious agents are complex and often not restricted to a single organ such as the gills. Gills are in many cases the primary location of negative effects from the environment due to their small barrier between the fish and the surrounding ocean.

This case study discusses effects of climate change on Atlantic salmon gills. These are the main fish species produced in many countries including the U.K, Norway, and Chile. Atlantic salmon in their sea water stage are mainly cultured in open-net pens and are thus constantly exposed to the ocean and everything in it, therefore we focus on the saltwater production stage because this is most relevant for climate and environmental change.

Gill disease and salmon management

Compromised gill health, or gill disease, occurs when infectious or non-infectious agents result in a detectable change affecting the functionality of the gill (Figure 5). Many inefficiencies in gill functions start with insults to the gills that then lead to colonization of infectious agents, reduced functionality in gills, and in many cases follow-up effects in other organs of the fish. Gill disease in Atlantic salmon can be caused by, or are associated with, many different infectious agents such as the amoeba *Neoparamoeba perurans*, other types of parasitic gill diseases including *Desmozoon lepeophtherii*, bacterial pathogens including *Candidatus* Branchiomonas cisticola and *Tenacibaculum* spp., and viruses including salmon gill pox virus.[250–252] Non-infectious gill diseases are caused by cnidarian nematocysts in zooplankton and jellyfish, gill disease by harmful algae, and chemical/toxin-associated gill disease.[253] The primary interaction with fish is often an insult by a spikey (i.e., irritative/abrasive) agent, which is sometimes followed up by the release of toxins, e.g., by certain plankton species, and secondary infections. Often principal pathological changes are non-specific, often multifactorial and a primary cause is not known. Then, it is referred to as "complex gill disease".[250] Gill disease affects salmon in fresh and saltwater stages, for example Salmon Gill Pox Virus can lead to high mortalities in both of these stages.[254]

The wide variety of causes for gill disease (if known) and clinical signs makes it challenging to standardize the relation between clinical signs, diagnostic test results, and gill health into a single case definition for gill disease that can be quantified. Surveillance for gill disease is most commonly through generic diagnostic tests such as gill scores from gross gill observations (Figure 5), normally assessed on the farm site, histopathology, and molecular tests when specific pathogens are suspected, or where information on underlying molecular mechanisms for gill inflammation is required.[255]

Climate change and salmon gill health

Perhaps the most important consequences of climate change to most salmon producing countries are warmer summers and less cold winters. Warmer summers in which problems arise, and less cold winters in which diseases are not cleared and instead become chronic. Two well-described temporal studies of gill pathogens and gill health have followed fish through a production cycle in commercial Scottish sites[256] and in Norway.[257] In both cases, increases in gill disease and pathology were found to peak in late summer, autumn, and early winter, which then cleared by spring. When season was removed from the analysis a significant relationship was found with water temperature.[256] The impacts of warmer winter temperatures experienced in salmon production areas has meant the fish have been unable to clear their gill parasites and this results in increase gill pathology and hence higher mortalities.

Increased temperatures can affect salmon directly. For example, extreme summer temperatures have been associated with mass mortalities of salmon as was observed off the coast of Newfoundland during 2019 where some sites had 100% mortality.[258] These authors examined salmon physiology and behavior in sea cages during natural warm water events demonstrating increased heart rate with increased water temperature, which reflects increase metabolic activity at warmer water temperature. Any inefficiency in gill function would have a negative outcome under such stress. Warm water also holds less oxygen than cold water, at the same time as fish are cold blooded as water temperature increases, as does their metabolic rate and hence oxygen demand.

Changing environmental conditions can affect salmon gills indirectly through effects on infectious agents of salmon such as amoebic gill disease which grow better at higher temperatures and salinities.[259,260] For instance, fish held at 15°C compared with 10°C showed increased severity of infections, and it has been suggested that the causative agent of amoebic gill disease, *N. perurans*, may be more able to attach to the gills and grow faster at the higher temperature.[261]

Non-infectious agents that can affect salmon gills are zooplankton and phytoplankton blooms, which are correlated with seawater temperatures.[261,262] In temperate regions where Atlantic salmon are farmed warmer waters almost always are reflected by a concurrent increase in planktonic species, often seen as the spring bloom. This increase in productivity also has the potential of increasing the pathogen load. As fish gills become infected and the resulting pathology occurs due to inflammation, the capacity of fish to function physiologically is compromised. Harmful zooplankton and harmful phytoplankton blooms are most likely to be a problem in summer-autumn with higher water temperatures.[263]

Impact of gill disease now and in the future

Given the variety in causes it can be difficult to assess the impact of climate change on overall gill health of Atlantic salmon. Most information comes from public fish mortalities data given the label "due to gill disease", and often these are combined with viral conditions such as cardiac myopathy syndrome. Such data have, however, shown dramatic increases in relation to mortality due to gill health.[264–266] However, they are an underrepresentation of the effects of gill health, because physiological processes in the salmon body can be affected before the fish dies, resulting in reduced productivity, e.g., by poor growth. Little is known about the direct and indirect costs of gill disease and this is needed to better understand the extent of this disease and how this may change because of increased environmental temperature.

MITIGATION AND ADAPTATION STRATEGIES AGAINST DISEASE

Predicting the precise impact of climate change on the frequency and severity of disease episodes in aquaculture and fisheries is inherently complex due to various factors. On a positive note, aquaculture husbandry practices have continuously evolved, encompassing changes in system design, stocking densities, on-site biosecurity protocols, and in management and control strategies. Concurrently, advancements have been made in genetics, enhancing the ability of aquaculture species to resist or tolerate pathogen challenges. Progress has also been evident in diagnostics, pathogen detection and identification, nutrition, including the use of functional feeds and probiotics to bolster the immune status of cultured aquatic species, and the overall health management of the culture environment. This section provides a summary of some of these strategies used to promote health and/or to circumvent the adverse effects of climate change on both fish and shellfish diseases.

Improved biosecurity

Biosecurity measures aim to limit the contact between pathogen/parasite and host such that infection cannot occur. These are superior to control/treatment approaches including use of antimicrobials and anti-parasitic agents as they have less direct impact on the environment. For example, recirculating aquaculture systems (RAS) allow fish cultivation in-land in indoor facilities where the quality of the water in terms of partial sterility, temperature, salinity, and levels of nitrogenous waste products, can be controlled in isolation to inhospitable conditions (e.g., disease-causing agents) in other natural waters. Such systems, though, are expensive to construct, can have high-energy needs and produce greenhouse gas emissions.[267] They do allow a higher stocking density than other flow through systems but despite this advantage they may be economically unworkable based on the price and market demand of the final product.

Improved treatment and control strategies for infectious diseases of fish and shellfish

The development of vaccines and the practical methods for delivery of these has been a success story for fish cultivation over the last few decades. However, despite these successes, some conditions have proven difficult to control. For example, early attempts to develop vaccines to control fish ectoparasites such as *L. salmonis* were disappointing in terms of their efficacy and so alternate strategies (e.g., chemical control) have been developed that may not be as benign as vaccines. Use of new approaches for vaccines including subunit peptides, DNA and RNA-based vaccines (next generation), and new adjuvants show future promise.[268,269] Other emerging approaches to control diseases of both fish and shellfish include biofloc technology,[270] phage delivery,[271,272] and novel immune stimulants.[273,274]

Improved disease surveillance in the oceans

Groner et al.[275] and Maynard et al.[248] argue for an improved approach to disease surveillance in the marine environment utilizing advances in detection and reporting of diseases, management strategies, and developing models to predict outbreaks of disease. However, the practicalities of such improvements by governments and interested parties are unclear.

Genetic selection and breeding of resistant strains

Selective breeding of resistant populations of fish and shellfish to disease has been practiced for nearly a century with some success. Given the scale of available literature on both fish and shellfish in terms of selective breeding programmes, here, we simply point the reader to key reviews on fin-fish and shellfish selective breeding.[275–282] Of particular note here is the finding that triploid oysters (widely used in oyster farming) display enhanced susceptibility to heatwaves in comparison to non-triploids.[283]

Relocation of aquaculture sites

Climate change may make it impossible to culture fish and shellfish in traditional locations especially if water temperatures regularly exceed the physiological buffering capacity of fish and shellfish that leave them vulnerable to infectious and non-infectious diseases. Examples of strategies to move production vary in complexity from simple movement of trestles containing developing Pacific oysters and salmon in cages into deeper water hence avoiding higher summer air temperatures when exposed by the tide through to closure of existing sites and their relocation. Finally, movement of *C. virginica* pre-harvesting from low to high salinities can reduce the risk of vibrio contamination causing food poisoning.[284,285]

CONCLUDING REMARKS

It is 20 years since the publication of the first broad exploration of whether diseases are increasing in the oceans.[286] Their analysis based on Web of Science citations concluded that while the occurrence of disease was increasing in some groups (e.g., corals, marine mammals, urchins, and molluscs) in some there was no apparent change (e.g., crustaceans) while in others, such as bony fish, levels were decreasing. A subsequent analysis in 2019 using available data from 1970 to 2001 and 2001 to 2015, also found increases in disease in corals and urchins and apparent decreases in similar conditions in bony fish and decapod crustaceans.[33] This reduction in disease reporting in bony fish was attributed to population declines (i.e., overfishing) resulting in a lower chance of density dependent disease transmission between individuals of the same species. Our review shows that while these two "broad brush stroke" approaches to changes in disease occurrence in marine dwelling organisms suggest no increase in fish and shellfish in general, there are examples of increased occurrence and severity of diseases in fish and shellfish both in the wild and under cultivation. Episodes of sudden increases in both aerial and sea surface temperature can push animals close to, or beyond, their thermal tolerance resulting in physiological and immunological changes leaving them vulnerable to opportunistic pathogens (Figure 2).[76,127] Sessile animals, such as mussels, oysters, and seaweeds, are at particular risk especially if they are in the intertidal region where temperature extremes can be rapid, severe, and prolonged in the summer months. Of course, the impact(s) of climate-driven cooling on marine disease dynamics remains largely unknown.

The projected increase in humankind population in the coming years will require all our ingenuity in an era of unstable climate if we are to feed ourselves and, at the same time, protect our ecosystems from pervasive, and perhaps irreversible, damage.[287]

ACKNOWLEDGMENTS

A.F.R and C.J.C. acknowledge funding from BBSRC/NERC (BB/P017215/1) and the Ireland-Wales ERDF Interreg project, Bluefish. The POMS studies reported in Case study 4 were supported by the ANR projects DECIPHER (ANR-14-CE19-0023) and DECICOMP (ANR-19-CE20-0004), by Ifremer project GT-huitre, and by the Fond Européen pour les Affaires Maritimes et la Pêche (FEAMP, GESTINNOV project no. PFEA470020FA1000007). These studies are set within the framework of the "Laboratoires d'Excellences (LABEX)" TULIP (ANR-10-LABX-41). W.S. and G.S. acknowledge funding from the National Science, Research and Innovation Fund, Thailand Science Research and Innovation (grant no. 4709364). K.S. and S.T. acknowledge funding from the National Research Council of Thailand: High-Potential Research Team Grant Program (grant no. N42A650869). S.A.M.M. was funded by Scottish Aquaculture Innovation Centre (SAIC grant SL 2017 Nutritional Aspects of Gill Disease in Atlantic Salmon and SAIC grant SL 2020 Spatial and temporal drivers of gill pathology in Atlantic salmon. Case study 6 was supported by the "Gill health in Scottish farmed salmon" project (SAIC grant SL_2017_07).

AUTHOR CONTRIBUTIONS

Initial conceptualization: A.F.R. and C.J.C.; collected and reviewed literature and writing original draft manuscript: All authors.; final editing: A.F.R. and C.J.C.; all authors have read and approved this article.

DECLARATION OF INTERESTS

The authors declare no competing interests.

REFERENCES

1. United Nations (2017). World Population Prospects, The 2017 Revision, Key Findings and Advance Tables. Working Paper ESA/P/WP/248. https://esa.un.org/unpd/wpp/.
2. Costa-Pierce, B.A. (2022). The anthropology of aquaculture. Front. Sustain. Food Syst. *6*, 843743. https://doi.org/10.3389/fsufs.2022.843743.
3. FAO (2022). The State of World Fisheries and Aquaculture 2022. Towards Blue Transformation (FAO), p. 266. https://doi.org/10.4060/cc0461en.
4. Costello, C., Cao, L., Gelcich, S., Cisneros-Mata, M.Á., Free, C.M., Froehlich, H.E., Golden, C.D., Ishimura, G., Maier, J., Macadam-Somer, I., et al. (2020). The future of food from the sea. Nature *588*, 95–100. https://doi.org/10.1038/s41586-020-2616-y.
5. Free, C.M., Cabral, R.B., Froehlich, H.E., Battista, W., Ojea, E., O'Reilly, E., Palardy, J.E., García Molinos, J., Siegel, K.J., Arnason, R., et al. (2022). Expanding ocean food production under climate change. Nature *605*, 490–496. https://doi.org/10.1038/s41586-022-04674-5.
6. Carballeira Brana, C.B., Cerbule, K., Senff, P., and Stolz, I.K. (2021). Towards environmental sustainability in marine finfish aquaculture. Front. Mar. Sci. *8*, 666662. https://doi.org/10.3389/fmars.2021.666662.
7. Torrissen, O., Jones, S., Asche, F., Guttormsen, A., Skilbrei, O.T., Nilsen, F.,

Horsberg, T.E., and Jackson, D. (2013). Salmon lice–impact on wild salmonids and salmon aquaculture. J. Fish. Dis. *36*, 171–194. https://doi.org/10.1111/jfd.12061.

8. Dempster, T., Overton, K., Bui, S., Stien, L.H., Oppedal, F., Karlsen, Ø., Coates, A., Phillips, B., and Barrett, L. (2021). Farmed salmonids drive the abundance, ecology and evolution of parasitic salmon lice in Norway. Aquacult. Environ. Interact. *13*, 237–248. https://doi.org/10.3354/aei00402.
9. Burridge, L.E., Lyons, M.C., Wong, D.K.H., MacKeigan, K., and VanGeest, J.L. (2014). The acute lethality of three anti-sea lice formulations: AlphaMax®, Salmosan®, and Interox®Paramove™50 to lobster and shrimp. Aquaculture *420–421*, 180–186. https://doi.org/10.1016/j.aquaculture.2013.10.041.
10. Strachan, F., and Kennedy, C.J. (2021). The environmental fate and effects of anti-sea lice chemotherapeutants used in salmon aquaculture. Aquaculture *544*, 737079. https://doi.org/10.1016/j.aquaculture.2021.737079.
11. Aaen, S.M., Helgesen, K.O., Bakke, M.J., Kaur, K., and Horsberg, T.E. (2015). Drug resistance in sea lice: a threat to salmonid aquaculture. Trends Parasitol. *31*, 72–81. https://doi.org/10.1016/j.pt.2014.12.006.
12. Barrett, L.T., Oppedal, F., Robinson, N., and Dempster, T. (2020). Prevention not cure: A review of methods to avoid sea lice infestations in salmon aquaculture. Rev. Aquacult. *12*, 2527–2543.
13. Coates, A., Phillips, B.L., Bui, S., Oppedal, F., Robinson, N.A., and Dempster, T. (2021). Evolution of salmon lice in response to management strategies: A review. Rev. Aquacult. *13*, 1397–1422.
14. Godwin, S.C., Bateman, A.W., Kuparinen, A., Johnson, R., Powell, J., Speck, K., and Hutchings, J.A. (2022). Salmon lice in the Pacific Ocean show evidence of evolved resistance to parasiticide treatment. Sci. Rep. *12*, 4775. https://doi.org/10.1038/s41598-022-07464-1.
15. Powell, A., Treasurer, J.W., Pooley, C.L., Keay, A.J., Lloyd, R., Imsland, A.K., and Garcia de Leaniz, C. (2018). Use of lumpfish for sea-lice control in salmon farming: Challenges and opportunities. Rev. Aquacult. *10*, 683–702. https://doi.org/10.1111/raq.12194.
16. Overton, K., Dempster, T., Oppedal, F., Kristiansen, T.S., Gismervik, K., and Stien, L.H. (2019). Salmon lice treatments and salmon mortality in Norwegian aquaculture: A review. Rev. Aquacult. *11*, 1398–1417. https://doi.org/10.1111/raq.12299.
17. Boerlage, A.S., Shrestha, S., Leinonen, I., Jansen, M.D., Revie, C.W., Reeves, A., and Toma, L. (2024). Sea lice management measures for farmed Atlantic salmon (*Salmo salar*) in Scotland: Costs and effectiveness. Aquaculture *580*, 740274. https://doi.org/10.1016/j.aquaculture.2023.740274.
18. Shinn, A.P., Pratoomyot, J., Griffiths, D., Trong, T.Q., Vu, N.T., Jiravanichpaisal, P., and Briggs, M. (2018). Asian shrimp production and the economic costs of disease. Asian Fish Sci. *31S*, 29–58.
19. Shinn, A.P., Pratoomyot, J., Bron, J., Paladini, G., Brooker, E., and Brooker, A. (2015). Economic impacts of aquaculture parasites on global finfish production. Global Aquaculture Advocate *2015*, 82–84.
20. Stentiford, G.D., and Shields, J.D. (2005). A review of the parasitic dinoflagellates *Hematodinium* and *Hematodinium*-like infections in marine crustaceans. Dis. Aquat. Org. *66*, 47–70. https://doi.org/10.3354/dao066047.
21. Li, C., Li, M., and Huang, Q. (2021). The parasitic dinoflagellate *Hematodinium* infects marine crustaceans. Mar. Life Sci. Technol. *3*, 313–325. https://doi.org/10.1007/s42995-020-00061-z.
22. Molto-Martin, I., Neil, D.M., Coates, C.J., MacKenzie, S.A., Bass, D., Stentiford, G.D., and Albalat, A. (2024). Infection of Norway lobster (*Nephrops norvegicus*) by the parasite *Hematodinium* sp.: insights from 30 years of field observations. R. Soc. Open Sci. *11*, 231147. https://doi.org/10.1098/rsos.231147.
23. Meyers, T.R., Koeneman, T.M., Botelho, C., and Short, S. (1987). Bitter crab disease: a fatal dinoflagellate infection and marketing problem for Alaskan Tanner crabs, *Chionoecetes bairdi*. Dis. Aquat. Org. *3*, 195–216.
24. Shields, J.D., Taylor, D.M., Sutton, S.G., O'Keefe, P.G., Ings, D.W., and Pardy, A.L. (2005). Epidemiology of bitter crab disease (*Hematodinium* sp.) in snow crabs *Chionoecetes opilio* from Newfoundland, Canada. Dis. Aquat. Org. *64*, 253–264. https://doi.org/10.3354/dao064253.
25. Tigchelaar, M., Cheung, W.W.L., Mohammed, E.Y., Phillips, M.J., Payne, H.J., Selig, E.R., Wabnitz, C.C.C., Oyinlola, M.A., Frölicher, T.L., Gephart, J.A., et al. (2021). Compound climate risks threaten aquatic food system benefits. Nat. Food *2*, 673–682. https://doi.org/10.1038/s43016-021-00368-9.
26. Islam, M.J., Kunzmann, A., and Slater, M.J. (2022). Responses of aquaculture fish to climate change-induced extreme temperatures: A review. J. World Aquacult. Soc. *53*, 314–366. https://doi.org/10.1111/jwas.12853.
27. Golden, C.D., Koehn, J.Z., Shepon, A., Passarelli, S., Free, C.M., Viana, D.F., Matthey, H., Eurich, J.G., Gephart, J.A., Fluet-Chouinard, E., et al. (2021). Aquatic foods to nourish nations. Nature *598*, 315–320. https://doi.org/10.1038/s41586-021-03917-1.
28. Cao, L., Halpern, B.S., Troell, M., Short, R., Zeng, C., Jiang, Z., Liu, Y., Zou, C., Liu, C., Liu, S., et al. (2023). Vulnerability of blue foods to human-induced environmental change. Nat. Sustain. *6*, 1186–1198. https://doi.org/10.1038/s41893-023-01156-y.
29. Kelling, I., Carrigan, M., and Johnson, A.F. (2023). Transforming the seafood supply system: challenges and strategies for resilience. Food Secur. *15*, 1585–1591. https://doi.org/10.1007/s12571-023-01400-5.
30. Hollowed, A.B., Barange, M., Garçon, V., Ito, S.I., Link, J.S., Aricò, S., Batchelder, H., Brown, R., Griffis, R., and Wawrzynski, W. (2019). Recent advances in understanding the effects of climate change on the world's oceans. ICES J. Mar. Sci. *76*, 1215–1220. https://doi.org/10.1093/icesjms/fsz084.
31. Burge, C.A., Mark Eakin, C., Friedman, C.S., Froelich, B., Hershberger, P.K., Hofmann, E.E., Petes, L.E., Prager, K.C., Weil, E., Willis, B.L., et al. (2014). Climate change influences on marine infectious diseases: implications for management and society. Ann. Rev. Mar. Sci *6*, 249–277. https://doi.org/10.1146/annurev-marine-010213-135029.
32. Salin, K.R., Subasinghe, R.P., Senarathna, D., and Shinn, A.P. (2023). Cage culture of finfish: Its importance, distributions and future modifications in ongoing climate change. In Climate Change on Diseases and Disorders of Finfish in Cage Culture, 3rd ed., P.T. Woo and R.P. Subasinghe, eds. (CABI), p. 536. https://doi.org/10.1079/9781800621640.0001.
33. Tracy, A.M., Pielmeier, M.L., Yoshioka, R.M., Heron, S.F., and Harvell, C.D. (2019). Increases and decreases in marine disease reports in an era of global change. Proc. Biol. Sci. *286*, 20191718. https://doi.org/10.1098/rspb.2019.1718.
34. Hoenig, J.M., Groner, M.L., Smith, M.W., Vogelbein, W.K., Taylor, D.M., Landers, D.F., Jr., Swenarton, J.T., Gauthier, D.T., Sadler, P., Matsche, M.A., et al. (2017). Impact of disease on the survival of three commercially fished species. Ecol. Appl. *27*, 2116–2127. https://doi.org/10.1002/eap.1595.
35. Chapman, J.M., Kelly, L.A., Teffer, A.K., Miller, K.M., and Cooke, S.J. (2021). Disease ecology of wild fish: opportunities and challenges for linking infection metrics with behaviour, condition, and survival. Can. J. Fish. Aquat. Sci. *78*, 995–1007. https://doi.org/10.1139/cjfas-2020-0315.
36. Marty, G.D., Hulson, P.J.F., Miller, S.E., Quinn, T.J., 2nd, Moffitt, S.D., and Merizon, R.A. (2010). Failure of population recovery in relation to disease in Pacific herring. Dis. Aquat. Org. *90*, 1–14. https://doi.org/10.3354/dao02210.
37. Groner, M.L., Bravo-Mendosa, E.D., MacKenzie, A.H., Gregg, J.L., Conway, C.M., Trochta, J.T., and Hershberger, P.K. (2023). Temporal, environmental, and demographic correlates of *Ichthyophonus* sp. infections in mature Pacific herring populations. ICES J. Mar. Sci. *80*, 2342–2355. https://doi.org/10.1093/icesjms/fsad147.
38. Kocan, R., Hershberger, P., and Winton, J. (2004). Ichthyophoniasis: an emerging disease of Chinook salmon in the Yukon River. J. Aquat. Anim. Health *16*, 58–72. https://doi.org/10.1577/H03-068.1.
39. Maynard, J., Van Hooidonk, R., Harvell, C.D., Eakin, C.M., Liu, G., Willis, B.L., Williams, G.J., Groner, M.L., Dobson, A., Heron, S.F., and Glenn, R. (2016). Improving marine disease surveillance through sea temperature monitoring, outlooks and projections Phil. Trans. Soc. Roy. Soc. B. *371*, 1689. https://doi.org/10.1098/rstb.2015.0208.
40. Wilhelm, G., and Mialhe, E. (1996). Dinoflagellate infection associated with the decline of *Necora puber* crab populations in France. Dis. Aquat. Org. *26*, 213–219. https://doi.org/10.3354/dao026213.
41. Bass, D., Rowley, A.F., and Coates, C.J. (2022). Diagnostic approaches in invertebrate pathology. In Invertebrate Pathology, A.F. Rowley, C.J. Coates, and M.M.A. Whitten, eds. (Oxford University Press), pp. 61–72. https://doi.org/10.1093/oso/9780198853756.003.0003.
42. Guilder, J., Ryder, D., Taylor, N.G.H., Alewijnse, S.R., Millard, R.S., Thrush, M.A., Peeler, E.J., and Tidbury, H.J. (2023). The aquaculture disease network model (AquaNet-Mod): A simulation model to evaluate disease spread and controls for the salmonid industry in England and Wales. Epidemics *44*, 100711. https://doi.org/10.1016/j.epidem.2023.100711.

43. Romero, J.F., Gardner, I., Price, D., Halasa, T., and Thakur, K. (2022). DTU-DADS-Aqua: A simulation framework for modelling waterborne spread of highly infectious pathogens in marine aquaculture. Transbound. Emerg. Dis. *69*, 2029–2044. https://doi.org/10.1111/tbed.14195.
44. Yatabe, T., Martínez-López, B., Díaz-Cao, J.M., Geoghegan, F., Ruane, N.M., Morrissey, T., McManus, C., Hill, A.E., and More, S.J. (2020). Data-driven network modelling as a framework to evaluate the transmission of piscine myocarditis virus (PMCV) in the Irish farmed Atlantic salmon population and the impact of different mitigation measures. Front. Vet. Sci. *7*, 385. https://doi.org/10.3389/fvets.2020.00385.
45. NOAA National Centers for Environmental Information, Monthly Global Climate Report for Annual 2023, published online January 2024, retrieved on March 22, 2024 from https://www.ncei.noaa.gov/access/monitoring/monthly-report/global/202313.
46. Morice, C.P., Kennedy, J.J., Rayner, N.A., Winn, J.P., Hogan, E., Killick, R.E., Dunn, R.J.H., Osborn, T.J., Jones, P.D., and Simpson, I.R. (2021). An updated assessment of near-surface temperature change from 1850: The HadCRUT5 data set. JGR. Atmospheres *126*, e2019JD032361.
47. Harvell, C.D., Mitchell, C.E., Ward, J.R., Altizer, S., Dobson, A.P., Ostfeld, R.S., and Samuel, M.D. (2002). Climate warming and disease risks for terrestrial and marine biota. Science *296*, 2158–2162. https://doi.org/10.1126/science.1063699.
48. Harvell, D., Aronson, R., Baron, N., Connell, J., Dobson, A., Ellner, S., Gerber, L., Kim, K., Kuris, A., McCallum, H., et al. (2004). The rising tide of ocean diseases: unsolved problems and research priorities. Front. Ecol. Environ. *2*, 375–382. https://doi.org/10.1890/1540-9295(2004)002[0375:TRTOOD]2.0.CO;2.
49. Lages, M.A., Balado, M., and Lemos, M.L. (2019). The expression of virulence factors in *Vibrio anguillarum* is dually regulated by iron levels and temperature. Front. Microbiol. *10*, 2335. https://doi.org/10.3389/fmicb.2019.02335.
50. López-Cervantes, G., Álvarez-Ruiz, P., Luna-Suárez, S., Luna-González, A., Esparza-Leal, H.M., Castro-Martínez, C., Gámez-Jiménez, C., and Soto-Alcalá, J. (2021). Temperature and salinity modulate virulence and PirA gene expression of *Vibrio parahaemolyticus*, the causative agent of AHPND. Aquacult. Int. *29*, 743–756. https://doi.org/10.1007/s10499-021-00654-0.
51. Lee, Y., Roh, H., Kim, A., Park, J., Lee, J.Y., Kim, Y.J., Kang, Y.R., Kang, H., Kim, S., Kim, H.S., et al. (2023). Molecular mechanisms underlying the vulnerability of Pacific abalone (*Haliotis discus hannai*) to *Vibrio harveyi* infection at higher water temperature. Fish Shellfish Immunol. *138*, 108844. https://doi.org/10.1016/j.fsi.2023.108844.
52. Liang, Q., Li, Z., Ou, M., Wu, X., Qiao, X., Wei, W., Liu, Y., Ye, J., and Wang, W. (2020). Hypoimmunity and intestinal bacterial imbalance are closely associated with blue body syndrome in cultured *Penaeus vannamei*. Aquaculture *522*, 735118. https://doi.org/10.1016/j.aquaculture.2020.735118.
53. Hembrom, P.S., Barik, S., Deepthi, M., Kannoth, S., and Grace, T. (2024). Influence of gut microbiome on health and development of penaeid shrimps. Aquat. Sci. *86*, 4. https://doi.org/10.1007/s00027-023-01018-x.
54. Hamre, L.A., Bui, S., Oppedal, F., Skern-Mauritzen, R., and Dalvin, S. (2019). Development of the salmon louse *Lepeophtheirus salmonis* parasitic stages in temperatures ranging from 3 to 24°C. Aquacult. Environ. Interact. *11*, 429–443. https://doi.org/10.3354/aei00320.
55. Godwin, S.C., Krkosek, M., Reynolds, J.D., and Bateman, A.W. (2021). Sea-louse abundance on salmon farms in relation to parasite-control policy and climate change. ICES J. Mar. Sci. *78*, 377–387. https://doi.org/10.1093/icesjms/fsaa173.
56. Roberts, S.D., Van Ruth, P.D., Wilkinson, C., Bastianello, S.S., and Bansemer, M.S. (2019). Marine heatwave, harmful algae blooms and an extensive fish kill event during 2013 in South Australia. Front. Mar. Sci. *6*, 610. https://doi.org/10.3389/fmars.2019.00610.
57. Vollset, K.W. (2019). Parasite induced mortality is context dependent in Atlantic salmon: insights from an individual-based model. Sci. Rep. *9*, 17377. https://doi.org/10.1038/s41598-019-53871-2.
58. Foyle, K.L., Hess, S., Powell, M.D., and Herbert, N.A. (2020). What is gill health and what is its role in marine finfish aquaculture in the face of a changing climate? Front. Mar. Sci. *7*, 400. https://doi.org/10.3389/fmars.2020.00400.
59. Jones, S.R.M., and Price, D. (2022). Elevated seawater temperature and infection with *Neoparamoeba perurans* exacerbate complex gill disease in farmed Atlantic salmon (*Salmo salar*) in British Columbia, Canada. Microorganisms *10*, 1039. https://doi.org/10.3390/microorganisms10051039.
60. Groner, M.L., Hoenig, J.M., Pradel, R., Choquet, R., Vogelbein, W.K., Gauthier, D.T., and Friedrichs, M.A.M. (2018). Dermal mycobacteriosis and warming sea surface temperatures are associated with elevated mortality of striped bass in Chesapeake Bay. Ecol. Evol. *8*, 9384–9397. https://doi.org/10.1002/ece3.4462.
61. Cook, T., Folli, M., Klinck, J., Ford, S., and Miller, J. (1998). The relationship between increasing sea-surface temperature and the northward spread of *Perkinsus marinus* (Dermo) disease epizootics in oysters. Estuar. Coast Shelf Sci. *46*, 587–597. https://doi.org/10.1006/ecss.1997.0283.
62. Wendling, C.C., and Wegner, K.M. (2013). Relative contribution of reproductive investment, thermal stress and *Vibrio* infection to summer mortality phenomena in Pacific oysters. Aquaculture *412–413*, 88–96. https://doi.org/10.1016/j.aquaculture.2013.07.009.
63. Yang, B., Zhai, S., Li, X., Tian, J., Li, Q., Shan, H., and Liu, S. (2021). Identification of *Vibrio alginolyticus* as a causative pathogen associated with mass summer mortality of the Pacific oyster (*Crassostrea gigas*) in China. Aquaculture *535*, 736363. https://doi.org/10.1016/j.aquaculture.2021.736363.
64. Li, X., Shi, C., Yang, B., Li, Q., and Liu, S. (2023). High temperature aggravates mortalities of the Pacific oyster (*Crassostrea gigas*) infected with *Vibrio*: A perspective from homeostasis of digestive microbiota and immune response. Aquaculture *568*, 739309. https://doi.org/10.1016/j.aquaculture.2023.739309.
65. Petton, B., Pernet, F., Robert, R., and Boudry, P. (2013). Temperature influence on pathogen transmission and subsequent mortalities in juvenile Pacific oysters *Crassostrea gigas*. Aquacult. Envir. Int. *3*, 257–273. https://doi.org/10.3354/aei00070.
66. Petton, B., Destoumieux-Garzón, D., Pernet, F., Toulza, E., de Lorgeril, J., Degremont, L., and Mitta, G. (2021). The Pacific oyster mortality syndrome, a polymicrobial and multifactorial disease: State of knowledge and future directions. Front. Immunol. *12*, 630343. https://doi.org/10.3389/fimmu.2021.630343.
67. 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. Nat. Commun. *9*, 4215. https://doi.org/10.1038/s41467-018-06659-3.
68. Combe, M., Reverter, M., Caruso, D., Pepey, E., and Gozlan, R.E. (2023). Impact of global warming on the severity of viral diseases: A potentially alarming threat to sustainable aquaculture worldwide. Microorganisms *11*, 1049. https://doi.org/10.3390/microorganisms11041049.
69. Destoumieux-Garzón, D., Canesi, L., Oyanedel, D., Travers, M.A., Charrière, G.M., Pruzzo, C., and Vezzulli, L. (2020). Vibrio-bivalve interactions in health and disease. Environ. Microbiol. *22*, 4323–4341. https://doi.org/10.1111/1462-2920.15055.
70. Oyanedei, D., Lagorce, A., Bruto, M., Haffner, P., Morot, A., Labreuche, Y., Dorant, Y., de La Forest Divonne, S., Delavat, F., Inguimbert, N., and Montagnani, C. (2023). Cooperation and cheating orchestrate Vibrio assemblages and polymicrobial synergy in oysters infected with OsHV-1 virus. Proc. Natl. Acad. Sci. USA *120*, e2305195120. https://doi.org/10.1073/pnas.2305195120.
71. Travers, M.-A., Basuyaux, O., Le Goïc, N., Huchette, S., Nicolas, J.L., Koken, M., and Paillard, C. (2009). Influence of temperature and spawning effort on *Haliotis tuberculata* mortalities caused by *Vibrio harveyi*: an example of emerging vibriosis linked to global warming. Global Change Biol. *15*, 1365–1376. https://doi.org/10.1111/j.1365-2486.2008.01764.x.
72. Ben-Horin, T., Lenihan, H.S., and Lafferty, K.D. (2013). Variable intertidal temperature explains why disease endangers black abalone. Ecology *94*, 161–168. https://doi.org/10.1890/11-2257.1.
73. Groner, M.L., Shields, J.D., Landers, D.F., Jr., Swenarton, J., and Hoenig, J.M. (2018). Rising temperatures, molting phenology, and epizootic shell disease in the American lobster. Am. Nat. *192*, E163–E177. https://doi.org/10.5061/dryad.n102b1s.
74. Ishaq, S.L., Turner, S.M., Lee, G., Tudor, M.S., MacRae, J.D., Hamlin, H., and Bouchard, D. (2023). Water temperature and disease alters bacterial diversity and cultivability from American lobster (*Homarus americanus*) shells. iScience *26*, 106606. https://doi.org/10.1016/j.sci.2023.106606.
75. Rowley, A.F., and Coates, C.J. (2023). Shell disease syndromes of decapod crustaceans. Environ. Microbiol. *25*, 931–947. https://doi.org/10.1111/1462-2920.16344.
76. Shields, J.D. (2019). Climate change enhances disease processes in crustaceans: case studies in lobsters, crabs, and shrimps. J. Crust. Biol. *39*, 673–683. https://doi.org/10.1093/jcbiol/ruz072.

77. Estrada-Perez, N., Ruiz-Velazco, J.M.J., Magallon-Barajas, F.J., Campa-Cordova, A.I., and Hernández-Llamas, A. (2020). Dynamic stock model for analysing semi-intensive production of whiteleg shrimp *Litopenaeus* (*Penaeus) vannamei* affected by the acute hepatopancreatic necrosis disease: Assessment of disease severity indicators and relationships with pond water quality parameters. Aquacult. Res. *51*, 242–254. https://doi.org/10.1111/are.14370.

78. Lee, C.T., Chen, I.T., Yang, Y.T., Ko, T.P., Huang, Y.T., Huang, J.Y., Huang, M.F., Lin, S.J., Chen, C.Y., Lin, S.S., et al. (2015). The opportunistic marine pathogen *Vibrio parahaemolyticus* becomes virulent by acquiring a plasmid that expresses a deadly toxin. Proc. Natl. Acad. Sci. USA *112*, 10798–10803. https://doi.org/10.1073/pnas.1503129112.

79. Tran, L., Nunan, L., Redman, R.M., Mohney, L.L., Pantoja, C.R., Fitzsimmons, K., and Lightner, D.V. (2013). Determination of the infectious nature of the agent of acute hepatopancreatic necrosis syndrome affecting penaeid shrimp. Dis. Aquat. Org. *105*, 45–55. https://doi.org/10.3354/dao02621.

80. Swinford, J.L., and Anderson, J.D. (2021). Prevalence of black gill (*Hyalophysa lynni*) in white shrimp *Litopenaeus setiferus* and brown shrimp *Farfantepenaus aztecus* along the Texas gulf coast. Mar. Coast. Fish. *13*, 263–274. https://doi.org/10.1002/mcf2.10153.

81. Huchin-Mian, J.P., Small, H.J., and Shields, J.D. (2018). The influence of temperature and salinity on mortality of recently recruited blue crabs, *Callinectes sapidus*, naturally infected with *Hematodinium perezi* (Dinoflagellata). J. Invertebr. Pathol. *152*, 8–16. https://doi.org/10.1016/j.jip.2018.01.003.

82. Shields, J.D., Taylor, D.M., O'Keefe, P.G., Colbourne, E., and Hynick, E. (2007). Epidemiological determinants in outbreaks of bitter crab disease (*Hematodinium* sp.) in snow crabs *Chionoecetes opilio* from Conception Bay, Newfoundland, Canada. Dis. Aquat. Org. *77*, 61–72.

83. Campbell, A.H., Harder, T., Nielsen, S., Kjelleberg, S., and Steinberg, P.D. (2011). Climate change and disease: bleaching of a chemically defended seaweed. Global Change Biol. *17*, 2958–2970. https://doi.org/10.1111/j.1365-2486.2011.02456.x.

84. Kumar, V., Zozaya-Valdes, E., Kjelleberg, S., Thomas, T., and Egan, S. (2016). Multiple opportunistic pathogens can cause a bleaching disease in the red seaweed *Delisea pulchra*. Environ. Microbiol. *18*, 3962–3975. https://doi.org/10.1111/1462-2920.13403.

85. Hudson, J., Deshpande, N., Leblanc, C., and Egan, S. (2022). Pathogen exposure leads to a transcriptional downregulation of core cellular functions that may dampen the immune response in a macroalga. Mol. Ecol. *31*, 3468–3480. https://doi.org/10.1111/mec.16476.

86. Largo, D.B., Fukami, K., Nishijima, T., and Ohno, M. (1995). Laboratory-induced development of the ice-ice disease of the farmed red algae *Kappaphycus alvarezii* and *Eucheuma denticulatum* (Solieriaceae, Gigartinales, Rhodophyta). J. Appl. Phycol. *7*, 539–543.

87. Largo, D.B., Msuya, F.E., and Menezes, A. (2020). Understanding diseases and control in seaweed farming in Zanzibar. In FAO Fisheries and Aquaculture Technical Paper No. 662 (FAO), p. 64. https://doi.org/10.4060/ca9004en.

88. Arasamuthu, A., and Edward, J.K.P. (2018). Occurrence of ice-ice disease in seaweed *Kappaphycus alvarezii* at Gulf of Mannar and Palk Bay, Southeastern India. Ind. J. Geo. Mar. Sci. *47*, 1208–1216.

89. Ward, G.M., Kambey, C.S.B., Faisan, J.P., Jr., Tan, P., Daumich, C.C., Matoju, I., Stentiford, G.D., Bass, D., Lim, P., Brodie, J., and Poong, S. (2021). Ice-Ice disease: An environmentally and microbiologically driven syndrome in tropical seaweed aquaculture. Rev. Aquacult. *14*, 414–439. https://doi.org/10.1111/raq.12606.

90. Friedman, C.S., and Finley, C.A. (2003). Anthropogenic introduction of the etiological agent of withering syndrome into northern California abalone populations via conservation efforts. Can. J. Fish. Aquat. Sci. *60*, 1424–1431. https://doi.org/10.1139/F03-121.

91. Friedman, C.S., Andree, K.B., Beauchamp, K.A., Moore, J.D., Robbins, T.T., Shields, J.D., and Hedrick, R.P. (2000). Candidatus Xenohaliotis californiensis, a newly described pathogen of abalone, *Haliotis* spp., along the west coast of North America. Int. J. Syst. Evol. Microbiol. *50*, 847–855. https://doi.org/10.1099/00207713-50-2-847.

92. Friedman, C.S., Biggs, W., Shields, J.D., and Hedrick, R.P. (2002). Transmission of withering syndrome in black abalone, *Haliotis cracherodii* Leach. J. Shellfish Res. *21*, 817–824.

93. Tissot, B.N. (1995). Recruitment, growth, and survivorship of black abalone on Santa Cruz Island following mass mortality. Bull. South Calif. Acad. Sci. *94*, 179–189.

94. Moore, J.D., Marshman, B.C., and Chun, C.S.Y. (2011). Health and survival of red abalone *Haliotis rufescens* from San Miguel Island, California, USA, in a laboratory simulation of La Niña and El Niño conditions. J. Aquat. Anim. Health *23*, 78–84. https://doi.org/10.1080/08997659.2011.568860.

95. Crosson, L.M., and Friedman, C.S. (2018). Withering syndrome susceptibility of northeastern Pacific abalones: A complex relationship with phylogeny and thermal experience. J. Invertebr. Pathol. *151*, 91–101. https://doi.org/10.1016/j.jip.2017.11.005.

96. Stunkard, H.W. (1957). The morphology and life-history of the digenetic trematode, *Microphallus similis* (Jägerskiöld, 1900) Baer, 1943. Biol. Bull. Woods Hole *112*, 254–266.

97. Blakeslee, A.M.H., Keogh, C.L., Fowler, A.E., and Griffen, B.D. (2015). Assessing the effects of trematode infection on invasive green crabs in eastern North America. PLoS One *10*, e0128674. https://doi.org/10.1371/journal.pone.0128674.

98. Torchin, M.E., Lafferty, K.D., and Kuris, A.M. (2002). Parasites and marine invasions. Parasitology *124*, S137–S151. https://doi.org/10.1017/s0031182002001506.

99. Thieltges, D.W., and Rick, J. (2006). Effect of temperature on emergence, survival and infectivity of cercariae of the marine trematode *Renicola roscovita* (Digenea: Renicolidae). Dis. Aquat. Org. *73*, 63–68. https://doi.org/10.3354/dao073063.

100. Magalhães, L., de Montaudouin, X., Figueira, E., and Freitas, R. (2018). Trematode infection modulates cockles biochemical response to climate change. Sci. Total Environ. *637–638*, 30–40. https://doi.org/10.1016/j.scitotenv.2018.04.432.

101. McHugh, D.J. (2003). A guide to the seaweed industry. FAO Fisheries Technical Paper 441 (FAO).

102. Ward, G.M., Faisan, J.P., Jr., Cottier-Cook, E.J., Gachon, C., Hurtado, A.Q., Lim, P.E., Matoju, I., Msuya, F.E., Bass, D., and Brodie, J. (2020). A review of reported seaweed diseases and pests in aquaculture in Asia. J. World Aquacult. Soc. *51*, 815–828. https://doi.org/10.1111/jwas.12649.

103. Hudson, J., Kumar, V., and Egan, S. (2019). Comparative genome analysis provides novel insight into the interaction of *Aquimarina* sp. AD1, BL5 and AD10 with their macroalgal host. Mar. Genomics *46*, 8–15. https://doi.org/10.1016/j.margen.2019.02.005.

104. Hudson, J., and Egan, S. (2022). Opportunistic diseases in marine eukaryotes: Could Bacteroidota be the next threat to ocean life? Environ. Microbiol. *24*, 4505–4518. https://doi.org/10.1111/1462-2920.16094.

105. Unsworth, R.K.F., Cullen-Unsworth, L.C., Jones, B.L.H., and Lilley, R.J. (2022). The planetary role of seagrass conservation. Science *377*, 609–613. https://doi.org/10.1126/science.abq6923.

106. Graham, O.J., Stephens, T., Rappazzo, B., Klohmann, C., Dayal, S., Adamczyk, E.M., Olson, A., Hessing-Lewis, M., Eisenlord, M., Yang, B., et al. (2023). Deep habitats and cooler temperatures moderate a climate-driven seagrass disease. Phil. Trans. Roy. Soc. B. *378*, 20220016. https://doi.org/10.1098/rstb.2022.0016.

107. Burke, S., Pottier, P., Lagisz, M., Macartney, E.L., Ainsworth, T., Drobniak, S.M., and Nakagawa, S. (2023). The impact of rising temperatures on the prevalence of coral diseases and its predictability: A global meta-analysis. Ecol. Lett. *26*, 1466–1481. https://doi.org/10.1111/ele.14266.

108. Brandl, S.J., Johansen, J.L., Casey, J.M., Tornabene, L., Morais, R.A., and Burt, J.A. (2020). Extreme environmental conditions reduce coral reef fish biodiversity and productivity. Nat. Commun. *11*, 3832. https://doi.org/10.1038/s41467-020-17731-2.

109. Eddy, T.D., Lam, V.W., Reygondeau, G., Cisneros-Montemayor, A.M., Greer, K., Palomares, M.L.D., Bruno, J.F., Ota, Y., and Cheung, W.W. (2021). Global decline in capacity of coral reefs to provide ecosystem services. One Earth *4*, 1278–1285. https://doi.org/10.1016/j.oneear.2021.08.016.

110. Lunder, T., Eversen, Ø., Holstad, G., and Håstein, T. (1995). 'Winter ulcer' in the Atlantic salmon *Salmo salar*. Pathological and bacteriological investigations and transmission experiments. Dis. Aquat. Org. *23*, 39–49. https://doi.org/10.3354/dao023039.

111. Kasulin, A., Seredkina, N., and Sørum, H. (2017). Cold-water vibriosis. The current status of knowledge. J. Fish. Dis. *40*, 119–126. https://doi.org/10.1111/jfd.12465.

112. Colquhoun, D.J., and Sørum, H. (2001). Temperature dependent siderophore production in *Vibrio salmonicida*. Microb. Pathog. *31*, 213–219. https://doi.org/10.1006/mpat.2001.0464.

113. Burge, C.A., and Hershberger, P.K. (2020). Climate change can drive marine diseases. In Marine Disease Ecology, D.C. Behringer, ed. (Oxford University Press), pp. 83–94. https://doi.org/10.1093/oso/9780198821632.003.0005.
114. Lightner, D.V., Redman, R.M., Pantoja, C.R., Tang, K.F.J., Noble, B.L., Schofield, P., Mohney, L.L., Nunan, L.M., and Navarro, S.A. (2012). Historic emergence, impact and current status of shrimp pathogens in the Americas. J. Invertebr. Pathol. *110*, 174–183. https://doi.org/10.1016/j.jip.2012.03.006.
115. Desrina, Prayitno, S.B., Prayitno, S.B., Verdegem, M.C.J., Verreth, J.A.J., and Vlak, J.M. (2022). White spot syndrome virus host range and impact on transmission. Rev. Aquacult. *14*, 1843–1860. https://doi.org/10.1111/raq.12676.
116. Millard, R.S., Ellis, R.P., Bateman, K.S., Bickley, L.K., Tyler, C.R., van aerle, R., and Santos, E.M. (2021). How do abiotic environmental conditions influence shrimp susceptibility to disease? A critical analysis focussed on white spot disease. J. Invertebr. Pathol. *186*, 107369. https://doi.org/10.1016/j.jip.2020.107369.
117. Coates, C.J., Rowley, A.F., Courtney Smith, L., and Whitten, M.M.A. (2022). Host defences of invertebrates to pathogens and parasites. In Invertebrate Pathology, A.F. Rowley, C.J. Coates, and M.M.A. Whitten, eds. (Oxford University Press), pp. 3–40. https://doi.org/10.1093/oso/9780198853756.003.0001.
118. Schwaner, C., Barbosa, M., Connors, P., Park, T.J., de Silva, D., Griffith, A., Gobler, C.J., Pales Espinosa, E., and Allam, B. (2020). Experimental acidification increases susceptibility of *Mercenaria mercenaria* to infection by *Vibrio* species. Mar. Environ. Res. *154*, 104872. https://doi.org/10.1016/j.marenvres.2019.104872.
119. Clements, J.C., Bourque, D., McLaughlin, J., Stephenson, M., and Comeau, L.A. (2017). Extreme ocean acidification reduces the susceptibility of eastern oyster shells to a polydorid parasite. J. Fish. Dis. *40*, 1573–1585. https://doi.org/10.1111/jfd.12626.
120. Rowley, A.F. (2024). The immune system and immune defence in crustaceans. In Encyclopaedia of Immunology, 2nd. Edn. (Elsevier). https://doi.org/10.1016/b978-0-128-24465-4.00002-8.
121. Flajnik, M.F. (2018). A cold-blooded view of adaptive immunity. Nat. Rev. Immunol. *18*, 438–453. https://doi.org/10.1038/s41577-018-0003-9.
122. Buchmann, K., and Secombes, C.J. (2022). Principles of Fish Immunology. From Cells and Molecules to Host Protection (Springer).
123. Coates, C.J., and Söderhäll, K. (2021). The stress–immunity axis in shellfish. J. Invertebr. Pathol. *186*, 107492. https://doi.org/10.1016/j.jip.2020.107492.
124. Bailey, C., Segner, H., Casanova-Nakayama, A., and Wahli, T. (2017). Who needs the hotspot? The effect of temperature on the fish host immune response to *Tetracapsuloides bryosalmonae* the causative agent of proliferative kidney disease. Fish Shellfish Immunol. *63*, 424–437. https://doi.org/10.1016/j.fsi.2017.02.039.
125. Beemelmanns, A., Zanuzzo, F.S., Sandrelli, R.M., Rise, M.L., and Gamperl, A.K. (2021). The Atlantic salmon's stress- and immune-related transcriptional responses to moderate hypoxia, an incremental temperature increase, and these challenges combined. G3 (Bethesda) *11*, jkab102. https://doi.org/10.1093/g3journal/jkab102.
126. Cheng, W., Hsiao, I.S., Hsu, C.H., and Chen, J.C. (2004). Change in water temperature on the immune response of Taiwan abalone *Haliotis diversicolor supertexta* and its susceptibility to *Vibrio parahaemolyticus*. Fish Shellfish Immunol. *17*, 235–243. https://doi.org/10.1016/j.aquaculture.2005.04.060.
127. Mackenzie, C.L., Lynch, S.A., Culloty, S.C., and Malham, S.K. (2014). Future oceanic warming and acidification alter immune response and disease status in a commercial shellfish species. *Mytilus edulis* L. PLoS One *9*, e99712. https://doi.org/10.1371/journal.pone.0099712.
128. Bussell, J.A., Gidman, E.A., Causton, D.R., Gwynn-Jones, D., Malham, S.K., Jones, M.L.M., Reynolds, B., and Seed, R. (2008). Changes in the immune response and metabolic fingerprint of the mussel, *Mytilus edulis* (Linnaeus) in response to lowered salinity and physical stress. J. Exp. Mar. Biol. Ecol. *358*, 78–85. https://doi.org/10.1016/j.jembe.2008.01.018.
129. Cheng, W., Juang, F.M., and Chen, J.C. (2004). The immune response of Taiwan abalone *Haliotis diversicolor supertexta* and its susceptibility to *Vibrio parahaemolyticus* at different salinity levels. Fish Shellfish Immunol. *16*, 295–306. https://doi.org/10.1016/S1050-4648(03)00111-6.
130. Morshed, S.M., and Lee, T.H. (2023). The role of the microbiome on fish mucosal immunity under changing environments. Fish Shellfish Immunol. *139*, 108877. https://doi.org/10.1016/j.fsi.2023.108877.
131. Makrinos, D.L., and Bowden, T.J. (2016). Natural environmental impacts on teleost immune function. Fish Shellfish Immunol. *53*, 50–57. https://doi.org/10.1016/j.fsi.2016.03.008.
132. Abram, Q.H., Dixon, B., and Katzenback, B.A. (2017). Impacts of low temperature on the teleost immune system. Biology *6*, 39. https://doi.org/10.3390/biology6040039.
133. Mugwanya, M., Dawood, M.A., Kimera, F., and Sewilam, H. (2022). Anthropogenic temperature fluctuations and their effect on aquaculture: A comprehensive review. Aquac. Fish. *7*, 223–243. https://doi.org/10.1016/j.aaf.2021.12.005.
134. Scharsack, J.P., and Franke, F. (2022). Temperature effects on teleost immunity in the light of climate change. J. Fish. Biol. *101*, 780–796. https://doi.org/10.1111/jfb.15163.
135. Wu, F., Xie, Z., Lan, Y., Dupont, S., Sun, M., Cui, S., Huang, X., Huang, W., Liu, L., Hu, M., et al. (2018). Short-term exposure of *Mytilus coruscus* to decreased pH and salinity change impacts immune parameters of their haemocytes. Front. Physiol. *9*, 166. https://doi.org/10.3389/fphys.2018.00166.
136. Steenbergen, J.F., Steenbergen, S.M., and Schapiro, H.C. (1978). Effects of temperature on phagocytosis in *Homarus americanus*. Aquaculture *14*, 23–30.
137. Magnadóttir, B., Jónsdóttir, H., Helgason, S., Björnsson, B., Jørgensen, T.O., and Pilström, L. (1999). Humoral immune parameters in Atlantic cod (*Gadus morhua* L.): I. The effects of environmental temperature. Comp. Biochem. Physiol. B Biochem. Mol. Biol. *122*, 173–180. https://doi.org/10.1016/S0305-0491%2898%2910156-6.
138. Zanuzzo, F.S., Beemelmanns, A., Hall, J.R., Rise, M.L., and Gamperl, A.K. (2020). The innate immune response of Atlantic salmon (*Salmo salar*) is not negatively affected by high temperature and moderate hypoxia. Front. Immunol. *11*, 1009. https://doi.org/10.3389/fimmu.2020.01009.
139. Legrand, T.P., Wynne, J.W., Weyrich, L.S., and Oxley, A.P. (2020). A microbial sea of possibilities: current knowledge and prospects for an improved understanding of the fish microbiome. Rev. Aquacult. *12*, 1101–1134. https://doi.org/10.1111/raq.12375.
140. Lorgen-Ritchie, M., Uren Webster, T., McMurtrie, J., Bass, D., Tyler, C.R., Rowley, A., and Martin, S.A.M. (2023). Microbiomes in the context of developing sustainable intensified aquaculture. Front. Microbiol. *14*, 1200997. https://doi.org/10.3389/fmicb.2023.1200997.
141. Bernardo-Cravo, A.P., Schmeller, D.S., Chatzinotas, A., Vredenburg, V.T., and Loyau, A. (2020). Environmental factors and host microbiomes shape host-pathogen dynamics. Trends Parasitol. *36*, 616–633. https://doi.org/10.1016/j.pt.2020.04.010.
142. Murdoch, C.C., and Rawls, J.F. (2019). Commensal microbiota regulate vertebrate innate immunity-Insights from the zebrafish. Front. Immunol. *10*, 2100. https://doi.org/10.3389/fimmu.2019.02100.
143. Xiong, J.B., Nie, L., and Chen, J. (2019). Current understanding on the roles of gut microbiota in fish disease and immunity. Zool. Res. *40*, 70–76. https://doi.org/10.24272/j.issn.2095-8137.2018.069.
144. Zhang, X., Zhang, M., Zheng, H., Ye, H., Zhang, X., and Li, S. (2020). Source of hemolymph microbiota and their roles in the immune system of mud crab. Dev. Comp. Immunol. *102*, 103470. https://doi.org/10.1016/j.dci.2019.103470.
145. Huang, Z., Aweya, J.J., Zhu, C., Tran, N.T., Hong, Y., Li, S., Yao, D., and Zhang, Y. (2020). Modulation of crustacean innate immune response by amino acids and their metabolites: Inferences from other species. Front. Immunol. *11*, 574721. https://doi.org/10.3389/fimmu.2020.574721.
146. Uren Webster, T.M., Rodriguez-Barreto, D., Castaldo, G., Gough, P., Consuegra, S., and Garcia de Leaniz, C. (2020). Environmental plasticity and colonisation history in the Atlantic salmon microbiome: A translocation experiment. Mol. Ecol. *29*, 886–898. https://doi.org/10.1111/mec.15369.
147. Xavier, R., Severino, R., and Silva, S.M. (2023). Signatures of dysbiosis in fish microbiomes in the context of aquaculture. Rev. Aquacult. *16*, 706–731. https://doi.org/10.1111/raq.12862.
148. Bass, D., Stentiford, G.D., Wang, H.C., Koskella, B., and Tyler, C.R. (2019). The pathobiome in animal and plant diseases. Trends Ecol. Evol. *34*, 996–1008. https://doi.org/10.1016/j.tree.2019.07.012.
149. Guijarro, J.A., Cascales, D., García-Torrico, A.I., García-Domínguez, M., and Méndez, J. (2015). Temperature-dependent expression of virulence genes in fish-pathogenic bacteria. Front. Microbiol. *6*, 700. https://doi.org/10.3389/fmicb.2015.00700.
150. Uren Webster, T.M., Consuegra, S., and Garcia de Leaniz, C. (2021). Early life stress causes persistent impacts on the microbiome of Atlantic salmon. Comp. Biochem. Physiol D Genomics Proteomics *40*, 100888. https://doi.org/10.1101/2020.01.16.908939.

151. Holt, C.C., Bass, D., Stentiford, G.D., and van der Giezen, M. (2021). Understanding the role of the shrimp gut microbiome in health and disease. J. Invertebr. Pathol. *186*, 107387. https://doi.org/10.1016/j.jip.2020.107387.

152. Sepulveda, J., and Moeller, A.H. (2020). The effects of temperature on animal gut microbiomes. Front. Microbiol. *11*, 384–389. https://doi.org/10.3389/fmicb.2020.00384.

153. Kokou, F., Sasson, G., Nitzan, T., Doron-Faigenboim, A., Harpaz, S., Cnaani, A., and Mizrahi, I. (2018). Host genetic selection for cold tolerance shapes microbiome composition and modulates its response to temperature. Elife *7*, e36398. https://doi.org/10.7554/elife.36398.

154. Green, T.J., Siboni, N., King, W.L., Labbate, M., Seymour, J.R., and Raftos, D. (2019). Simulated marine heat wave alters abundance and structure of *Vibrio* populations associated with the Pacific oyster resulting in a mass mortality event. Microb. Ecol. *77*, 736–747. https://doi.org/10.1007/s00248-018-1242-9.

155. Scanes, E., Siboni, N., Rees, B., and Seymour, J.R. (2023). Acclimation in intertidal animals reduces potential pathogen load and increases survival following a heatwave. iScience *26*, 106813. https://doi.org/10.1016/j.isci.2023.106813.

156. Kong, N., Zhao, J., Zhao, B., Liu, J., Li, F., Wang, L., and Song, L. (2023). Effects of high temperature stress on the intestinal histology and microbiota in Yesso scallop *Patinopecten yessoensis*. Mar. Environ. Res. *185*, 105881. https://doi.org/10.1016/j.marenvres.2023.105881.

157. Sánchez-Cueto, P., Stavrakidis-Zachou, O., Clos-Garcia, M., Bosch, M., Papandroulakis, N., and Lladó, S. (2023). Mediterranean Sea heatwaves jeopardize greater amberjack's (*Seriola dumerili*) aquaculture productivity through impacts on the fish microbiota. ISME Commun. *3*, 36. https://doi.org/10.1038/s43705-023-00243-7.

158. Ghosh, S.K., Wong, M.K.-S., Hyodo, S., Goto, S., and Hamasaki, K. (2022). Temperature modulation alters the gut and skin microbial profiles of chum salmon (*Oncorhynchus keta*). Front. Mar. Sci. *9*, 1027621. https://doi.org/10.3389/fmars.2022.1027621.

159. Suzzi, A.L., Stat, M., Gaston, T.F., Siboni, N., Williams, N.L.R., Seymour, J.R., and Huggett, M.J. (2023). Elevated estuary water temperature drives fish gut dysbiosis and increased loads of pathogenic Vibrionaceae. Environ. Res. *219*, 115144. https://doi.org/10.1016/j.envres.2022.115144.

160. Lokmer, A., and Mathias Wegner, K. (2015). Hemolymph microbiome of Pacific oysters in response to temperature, temperature stress and infection. ISME J. *9*, 670–682. https://doi.org/10.1038/ismej.2014.160.

161. Pathirana, E., Whittington, R.J., and Hick, P.M. (2022). Impact of seawater temperature on the Pacific oyster (*Crassostrea gigas*) microbiome and susceptibility to disease associated with Ostreid herpesvirus-1 (OsHV-1). Anim. Prod. Sci. *62*, 1040–1054.

162. Li, X., Shi, C., Yang, B., Li, Q., and Liu, S. (2023). High temperature aggravates mortalities of the Pacific oyster (*Crassostrea gigas*) infected with Vibrio: A perspective from homeostasis of digestive microbiota and immune response. Aquaculture *568*, 739309. https://doi.org/10.1038/s41598-019-52752.

163. Li, Y.-F., Chen, Y.W., Xu, J.K., Ding, W.Y., Shao, A.Q., Zhu, Y.T., Wang, C., Liang, X., and Yang, J.L. (2019). Temperature elevation and *Vibrio cyclitrophicus* infection reduce the diversity of haemolymph microbiome of the mussel *Mytilus coruscus*. Sci. Rep. *9*, 16391. https://doi.org/10.1038/s41598-019-52752-y.

164. Dang, X., Huang, Q., He, Y.-Q., Gaitán-Espitia, J.D., Zhang, T., and Thiyagarajan, V. (2023). Ocean acidification drives gut microbiome changes linked to species-specific immune defence. Aquat. Toxicol. *256*, 106413. https://doi.org/10.1016/j.aquatox.2023.106413.

165. Khan, B., Clinton, S.M., Hamp, T.J., Oliver, J.D., and Ringwood, A.H. (2018). Potential impacts of hypoxia and a warming ocean on oyster microbiomes. Mar. Environ. Res. *139*, 27–34. https://doi.org/10.1016/j.marenvres.2018.04.018.

166. Zhong, K.X., Chan, A.M., Collicutt, B., Daspe, M., Finke, J.F., Foss, M., Green, T.J., Harley, C.D.G., Hesketh, A.V., Miller, K.M., et al. (2024). The prokaryotic and eukaryotic microbiome of Pacific oyster spat is shaped by ocean warming but not acidification. Appl. Environ. Microbiol. *90*, e0005224. https://doi.org/10.1128/aem.00052-24.

167. Baker-Austin, C., Oliver, J.D., Alam, M., Ali, A., Waldor, M.K., Qadri, F., and Martinez-Urtaza, J. (2018). *Vibrio* spp. infections. Nat. Rev. Dis. Prim. *4*, 8–19. https://doi.org/10.1038/s41572-018-0005-8.

168. Mohamad, N., Amal, M.N.A., Yasin, I.S.M., Zamri Saad, M., Nasruddin, N.S., Al-saari, N., Mino, S., and Sawabe, T. (2019). Vibriosis in cultured marine fishes: a review. Aquaculture *512*, 734289. https://doi.org/10.1016/j.aquaculture.2019.734289.

169. Farmer, J.J., III, Michael Janda, J., Brenner, F.W., Cameron, D.N., and Birkhead, K.M. (2015). Vibrio. In Bergey's Manual of Systematics of Archaea and Bacteria, M.E. Trujillo, S. Dedysh, P. DeVos, B. Hedlund, P. Kämpfer, F.A. Rainey, and W.B. Whitman, eds. (Wiley). https://doi.org/10.1002/9781118960608.gbm01078.

170. Li, B., Liu, J., Zhou, S., Fu, L., Yao, P., Chen, L., Yang, Z., Wang, X., and Zhang, X.H. (2020). Vertical variation in Vibrio community composition in Sansha Yongle Blue Hole and its ability to degrade macromolecules. Mar. Life Sci. Today *2*, 60–72. https://doi.org/10.1007/s42995-019-00003-4.

171. Baker-Austin, C., Trinanes, J., Gonzalez-Escalona, N., and Martinez-Urtaza, J. (2017). Non-cholera vibrios: The microbial barometer of climate change. Trends Microbiol. *25*, 76–84. https://doi.org/10.1016/j.tim.2016.09.008.

172. Vezzulli, L., Colwell, R.R., and Pruzzo, C. (2013). Ocean warming and spread of pathogenic vibrios in the aquatic environment. Microb. Ecol. *65*, 817–825. https://doi.org/10.1007/s00248-012-0163-2.

173. Vezzulli, L., Grande, C., Reid, P.C., Hélaouët, P., Edwards, M., Höfle, M.G., Brettar, I., Colwell, R.R., and Pruzzo, C. (2016). Climate influence on *Vibrio* and associated human diseases during the past half-century in the coastal North Atlantic. Proc. Natl. Acad. Sci. USA *113*, E5062–E5071. https://doi.org/10.1073/pnas.1609157113.

174. Vezzulli, L., Brettar, I., Pezzati, E., Reid, P.C., Colwell, R.R., Höfle, M.G., and Pruzzo, C. (2012). Long-term effects of ocean warming on the prokaryotic community: evidence from the vibrios. ISME J. *6*, 21–30. https://doi.org/10.1038/ismej.2011.89.

175. Sheikh, H.I., Najiah, M., Fadhlina, A., Laith, A.A., Nor, M.M., Jalal, K.C.A., and Kasan, N.A. (2022). Temperature upshift mostly but not always enhances the growth of Vibrio species: A systematic review. Front. Mar. Sci. *9*, 959830. https://doi.org/10.3389/fmars.2022.959830.

176. Vezzulli, L., Pezzati, E., Brettar, I., Höfle, M., and Pruzzo, C. (2015). Effects of global warming on Vibrio ecology. Microbiol. Spectr. *3*, 10–1128. https://doi.org/10.1128/microbiolspec.ve-0004-2014.

177. Sodders, N., Stockdale, K., Baker, K., Ghanem, A., Vieth, B., and Harder, T. (2023). Notes from the field: vibriosis cases associated with flood waters during and after Hurricane Ian-Florida, September-October 2022. MMWR Morb. Mortal. Wkly. Rep. *72*, 497–498. https://www.cdc.gov/.

178. Baker-Austin, C., Triñanes, J.A., Taylor, N.G., Hartnell, R.E., Siitonen, A., and Martínez-Urtaza, J. (2013). Emerging *Vibrio* risk at high latitudes in response to ocean warming. Nat. Clim. Change *3*, 73–77. https://doi.org/10.1038/NCLIMATE1628.

179. Venkatesan, P. (2024). Increase in *Vibrio* spp. infections linked to climate change. Lancet Infect. Dis. *24*, e18. https://doi.org/10.1016/S1473-3099(23)00767-3.

180. Trinanes, J., and Martinez-Urtaza, J. (2021). Future scenarios of risk of *Vibrio* infections in a warming planet: a global mapping study. Lancet Planet. Health *5*, E426–E435. https://doi.org/10.1016/S2542-5196(21)00169-8.

181. Tang, K.F.J., and Bondad-Reantaso, M.G. (2019). Impacts of acute hepatopancreatic necrosis disease on commercial shrimp aquaculture. Rev. Sci. Tech. *38*, 477–490. https://doi.org/10.20506/rst.38.2.2999.

182. Chonsin, K., Matsuda, S., Theethakaew, C., Kodama, T., Junjhon, J., Suzuki, Y., Suthienkul, O., and Iida, T. (2016). Genetic diversity of *Vibrio parahaemolyticus* strains isolated from farmed Pacific white shrimp and ambient pond water affected by acute hepatopancreatic necrosis disease outbreak in Thailand. FEMS Microbiol. Lett. *363*, fnv222. https://doi.org/10.1093/femsle/fnv222.

183. Sirikharin, R., Taengchaiyaphum, S., Sanguanrut, P., Chi, T.D., Mavichak, R., Proespraiwong, P., Nuangsaeng, B., Thitamadee, S., Flegel, T.W., and Sritunyalucksana, K. (2015). Characterization and PCR detection of binary, Pir-like toxins from *Vibrio parahaemolyticus* isolates that cause Acute Hepatopancreatic Necrosis Disease (AHPND) in shrimp. PLoS One *10*, e0126987. https://doi.org/10.1371/journal.pone.0126987.

184. Han, J.E., Tang, K., Tran, L.H., and Lightner, D.V. (2015). *Photorhabdus* insect-related (Pir) toxin-like genes in a plasmid of *Vibrio parahaemolyticus*, the causative agent of acute hepatopancreatic necrosis disease (AHPND) of shrimp. Dis. Aquat. Org. *113*, 33–40. https://doi.org/10.3354/dao02906.

185. Xiao, J., Liu, L., Ke, Y., Li, X., Liu, Y., Pan, Y., Yan, S., and Wang, Y. (2017). Shrimp AHPND-causing plasmids encoding the PirAB toxins as mediated by pirAB-Tn903 are prevalent in various *Vibrio* species. Sci. Rep. *7*, 42177. https://doi.org/10.1038/srep42177.

186. Restrepo, L., Bayot, B., Arciniegas, S., Bajaña, L., Betancourt, I., Panchana, F., and

Reyes Muñoz, A. (2018). PirVP genes causing AHPND identified in a new Vibrio species (*Vibrio punensis*) within the commensal Orientalis clade. Sci. Rep. *8*, 13080. https://doi.org/10.1038/s41598-018-30903-x.

187. López-Cervantes, G., Álvarez-Ruiz, P., Luna-Suárez, S., Luna-González, A., Esparza-Leal, H.M., Castro-Martínez, C., Gámez-Jiménez, C., and Soto-Alcalá, J. (2021). Temperature and salinity modulate virulence and PirA gene expression of *Vibrio parahaemolyticus*, the causative agent of AHPND. Aquacult. Int. *29*, 743–756. https://doi.org/10.1007/s10499-021-00654-0.
188. Wang, D., Wang, L., Bi, D., Song, J., Wang, G., Gao, Y., Tang, K.F.J., Meng, F., Xie, J., Zhang, F., et al. (2022). Conjugative transfer of acute hepatopancreatic necrosis disease-causing pVA1-Type plasmid is mediated by a novel self-encoded Type IV secretion system. Microbiol. Spectr. *10*, e0170222. https://doi.org/10.1128/spectrum.01702-22.
189. Billaud, M., Seneca, F., Tambutté, E., and Czerucka, D. (2022). An increase of seawater temperature upregulates the expression of *Vibrio parahaemolyticus* virulence factors implicated in adhesion and biofilm formation. Front. Microbiol. *13*, 840628. https://doi.org/10.3389/fmicb.2022.840628.
190. Pratama, A., Ishii, E., Kodama, T., Iida, T., and Matsuda, S. (2023). The xenogeneic silencer histone-like nucleoid-structuring protein mediates the temperature and salinity-dependent regulation of the Type III secretion system 2 in *Vibrio parahaemolyticus*. J. Bacteriol. *205*, e0026622. https://doi.org/10.1128/jb.00266-22.
191. Segarra, A., Pépin, J.F., Arzul, I., Morga, B., Faury, N., and 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 Res. *153*, 92–99. https://doi.org/10.1016/j.virusres.2010.07.011.
192. Martenot, C., Oden, E., Travaillé, E., Malas, J.P., and Houssin, M. (2011). Detection of different variants of Ostreid herpesvirus 1 in the Pacific oyster, *Crassostrea gigas* between 2008 and 2010. Virus Res. *160*, 25–31. https://doi.org/10.1186/s13567-014-0103-x.
193. Renault, T., Moreau, P., Faury, N., Pepin, J.F., Segarra, A., and Webb, S. (2012). Analysis of clinical ostreid herpesvirus 1 (Malacoherpesviridae) specimens by sequencing amplified fragments from three virus genome areas. J. Virol. *86*, 5942–5947. https://doi.org/10.1128/JVI.06534-11.
194. Lynch, S.A., Carlsson, J., Reilly, A.O., Cotter, E., and Culloty, S.C. (2012). A previously undescribed ostreid herpes virus 1 (OsHV-1) genotype detected in the Pacific oyster, *Crassostrea gigas*, in Ireland. Parasitology *139*, 1526–1532. https://doi.org/10.1017/S0031182012000881.
195. Jenkins, C., Hick, P., Gabor, M., Spiers, Z., Fell, S.A., 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 µ-var) in *Crassostrea gigas* (Pacific oysters) in Australia. Dis. Aquat. Org. *105*, 109–126. https://doi.org/10.3354/dao02623.
196. Burge, C.A., Friedman, C.S., Kachmar, M.L., Humphrey, K.L., Moore, J.D., and Elston, R.A. (2021). The first detection of a novel OsHV-1 microvariant in San Diego, California, USA. J. Invertebr. Pathol. *184*, 107636. https://doi.org/10.1016/j.jip.2021.107636.
197. Delisle, L., Laroche, O., Hilton, Z., Burguin, J.F., Rolton, A., Berry, J., Pochon, X., Boudry, P., and Vignier, J. (2022). Understanding the dynamic of POMS infection and the role of microbiota composition in the survival of Pacific oysters, *Crassostrea gigas*. Microbiol. Spectr. *10*, e01959-22. https://doi.org/10.1128/spectrum.01959-22.
198. EFSA Panel on Animal Health and Welfare AHAW (2015). Oyster mortality. EFSA J. *13*, 4122. https://doi.org/10.2903/j.efsa.2015.4122.
199. Burioli, E.A.V., Prearo, M., and Houssin, M. (2017). Complete genome sequence of ostreid herpesvirus type 1 microVar isolated during mortality events in the Pacific oyster *Crassostrea gigas* in France and Ireland. Virology *509*, 239–251. https://doi.org/10.1016/j.virol.2017.06.027.
200. Burioli, E.A.V., Prearo, M., Riina, M.V., Bona, M.C., Fioravanti, M.L., Arcangeli, G., and Houssin, M. (2016). Ostreid herpesvirus type 1 genomic diversity in wild populations of Pacific oyster *Crassostrea gigas* from Italian coasts. J. Invertebr. Pathol. *137*, 71–83. https://doi.org/10.1016/j.jip.2016.05.004.
201. Abbadi, M., Zamperin, G., Gastaldelli, M., Pascoli, F., Rosani, U., Milani, A., Schivo, A., Rossetti, E., Turolla, E., Gennari, L., et al. (2018). Identification of a newly described OsHV-1 microvar from the North Adriatic Sea (Italy). J. Gen. Virol. *99*, 693–703. https://doi.org/10.1099/jgv.0.001042.
202. Lemire, A., Goudenège, D., Versigny, T., Petton, B., Calteau, A., Labreuche, Y., and Le Roux, F. (2015). Populations, not clones, are the unit of vibrio pathogenesis in naturally infected oysters. ISME J. *9*, 1523–1531. https://doi.org/10.1038/ismej.2014.233.
203. Bruto, M., James, A., Petton, B., Labreuche, Y., Chenivesse, S., Alunno-Bruscia, M., Polz, M.F., and Le Roux, F. (2017). *Vibrio crassostreae*, a benign oyster colonizer turned into a pathogen after plasmid acquisition. ISME J. *11*, 1043–1052. https://doi.org/10.1038/ismej.2016.162.
204. Rubio, T., Oyanedel, D., Labreuche, Y., Toulza, E., Luo, X., Bruto, M., Chaparro, C., Torres, M., de Lorgeril, J., Haffner, P., et al. (2019). Species-specific mechanisms of cytotoxicity toward immune cells determine the successful outcome of *Vibrio* infections. Proc. Natl. Acad. Sci. USA *116*, 14238–14247. https://doi.org/10.1073/pnas.1905747116.
205. Clerissi, C., Luo, X., Lucasson, A., Mortaza, S., de Lorgeril, J., Toulza, E., Petton, B., Escoubas, J.M., Dégremont, L., Gueguen, Y., et al. (2023). A core of functional complementary bacteria infects oysters in Pacific Oyster Mortality Syndrome. Anim. Microbiome *5*, 26. https://doi.org/10.1186/s42523-023-00246-8.
206. Pernet, F., Barret, J., Le Gall, P., Corporeau, C., Dégremont, L., Lagarde, F., Pépin, J., and Keck, N. (2012). Mass mortalities of Pacific oysters *Crassostrea gigas* reflect infectious diseases and vary with farming practices in the Mediterranean Thau lagoon, France. Aquac. Environ. Interact. *2*, 215–237.
207. Petton, B., Boudry, P., Alunno-Bruscia, M., and Pernet, F. (2015). Factors influencing disease-induced mortality of Pacific oysters *Crassostrea gigas*. Aquac. Environ. Interact. *6*, 205–222.
208. Le Roux, F., Wegner, K.M., and Polz, M.F. (2016). Oysters and vibrios as a model for disease dynamics in wild animals. Trends Microbiol. *24*, 568–580. https://doi.org/10.1016/j.tim.2016.03.006.
209. Azéma, P., Lamy, J.B., Boudry, P., Renault, T., Travers, M.A., and Dégremont, L. (2017). Genetic parameters of resistance to *Vibrio aestuarianus*, and OsHV-1 infections in the Pacific oyster, *Crassostrea gigas*, at three different life stages. Genet. Sel. Evol. *49*, 23. https://doi.org/10.1186/s12711-017-0297-2.
210. Delisle, L., Petton, B., Burguin, J.F., Morga, B., Corporeau, C., and Pernet, F. (2018). Temperature modulate disease susceptibility of the Pacific oyster *Crassostrea gigas* and virulence of the Ostreid herpesvirus type 1. Fish Shellfish Immunol. *80*, 71–79. https://doi.org/10.1016/j.fsi.2018.05.056.
211. Segarra, A., Baillon, L., Tourbiez, D., Benabdelmouna, A., Faury, N., Bourgougnon, N., and Renault, T. (2014). Ostreid herpesvirus type 1 replication and host response in adult Pacific oysters, *Crassostrea gigas*. Vet. Res. *45*, 103. https://doi.org/10.1186/s13567-014-0103-x.
212. Divilov, K., Schoolfield, B., Morga, B., Dégremont, L., Burge, C.A., Mancilla Cortez, D., Friedman, C.S., Fleener, G.B., Dumbauld, B.R., and Langdon, C. (2019). First evaluation of resistance to both a California OsHV-1 variant and a French OsHV-1 microvariant in Pacific oysters. BMC Genet. *20*, 96. https://doi.org/10.1186/s12863-019-0791-3.
213. Pernet, F., Tamayo, D., and Petton, B. (2015). Influence of low temperatures on the survival of the Pacific oyster (*Crassostrea gigas*) infected with ostreid herpes virus type 1. Aquaculture *445*, 57–62. https://doi.org/10.1016/j.aquaculture.2015.04.010.
214. Petton, B., Alunno-Bruscia, M., Mitta, G., and Pernet, F. (2023). Increased growth metabolism promotes viral infection in a susceptible oyster population. Aquacult. Environ. Interact. *15*, 19–33. https://doi.org/10.3354/aei00450.
215. de Kantzow, M., Hick, P., Becker, J.A., and Whittington, R.J. (2016). Effect of water temperature on mortality of Pacific oysters *Crassostrea gigas* associated with microvariant ostreid herpesvirus 1 (OsHV-1 µVar). Aquacult. Environ. Interact. *8*, 419–428. https://doi.org/10.3354/aei00186.
216. Delisle, L., Pauletto, M., Vidal-Dupiol, J., Petton, B., Bargelloni, L., Montagnani, C., Pernet, F., Corporeau, C., and Fleury, E. (2020). High temperature induces transcriptomic changes in *Crassostrea gigas* that hinders progress of Ostreid herpesvirus (OsHV-1) and promotes survival. J. Exp. Biol. *223*, jeb.226233. https://doi.org/10.1242/jeb.226233.
217. Petton, B., Pernet, F., Robert, R., and Boudry, P. (2013). Temperature influence on pathogen transmission and subsequent mortalities in juvenile Pacific oysters *Crassostrea gigas*. Aquac. Environ. Interact. *3*, 257–273. https://doi.org/10.3354/aei00070.
218. Paul-Pont, I., Evans, O., Dhand, N.K., Rubio, A., Coad, P., and Whittington, R.L. (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. https://doi.org/10.1016/j.aquaculture.2013.07.038.
219. Chaudhary, C., Richardson, A.J., Schoeman, D.S., and Costello, M.J. (2021). Global warming is causing a more pronounced dip in marine species richness around the equator. Proc. Natl. Acad. Sci. USA *118*, e2015094118. https://doi.org/10.1073/pnas.2015094118.
220. Thomas, Y., Pouvreau, S., and Alunno-Bruscia, M. (2015). Global change and climate-driven invasion of the Pacific oyster (*Crassostrea gigas*) along European coasts: A bioenergetics modelling approach. J. Biogeogr. *43*, 568–579. https://doi.org/10.1111/jbi.12665.
221. Mortensen, S., Strand, Å., Bodvin, T., Alfjorden, A., Skår, C.K., Jelmert, A., Aspán, A., Sælemyr, L., Naustvoll, L.J., and Albretsen, J.A. (2016). Summer mortalities and detection of ostreid herpesvirus microvariant in Pacific oyster *Crassostrea gigas* in Sweden and Norway. Dis. Aquat. Org. *117*, 171–176. https://doi.org/10.3354/dao02944.
222. Hobday, A.J., Alexander, L.V., Perkins, S.E., Smale, D.A., Straub, S.C., Oliver, E.C., Benthuysen, J.A., Burrows, M.T., Donat, M.G., Feng, M., et al. (2016). A hierarchical approach to defining marine heatwaves. Prog. Oceanogr. *141*, 227–238. https://doi.org/10.1016/j.pocean.2015.12.014.
223. Fleury, E., Barbier, P., Petton, B., Normand, J., Thomas, Y., Pouvreau, S., Daigle, G., and Pernet, F. (2020). Latitudinal drivers of oyster mortality: deciphering host, pathogen and environmental risk factors. Sci. Rep. *10*, 7264. https://doi.org/10.1038/s41598-020-64086-1.
224. Fuhrmann, M., Richard, G., Quéré, C., Petton, B., and Pernet, F. (2019). Low pH reduced survival of the oyster *Crassostrea gigas* exposed to the Ostreid herpesvirus 1 by altering the metabolic response of the host. Aquaculture *503*, 167–174. https://doi.org/10.1016/j.aquaculture.2018.12.052.
225. IPCC (2023). Contribution of Working Groups I, II and III to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change, pp. 35–115.
226. Behrenfeld, M.J., O'Malley, R.T., Siegel, D.A., McClain, C.R., Sarmiento, J.L., Feldman, G.C., Milligan, A.J., Falkowski, P.G., Letelier, R.M., and Boss, E.S. (2006). Climate-driven trends in contemporary ocean productivity. Nature *444*, 752–755. https://doi.org/10.1038/nature05317.
227. Melzner, F., Stange, P., Trübenbach, K., Thomsen, J., Casties, I., Panknin, U., Gorb, S.N., and Gutowska, M.A. (2011). Food supply and seawater pCO_2 impact calcification and internal shell dissolution in the blue mussel *Mytilus edulis*. PLoS One *6*, e24223. https://doi.org/10.1371/journal.pone.0024223.
228. Thomsen, J., Casties, I., Pansch, C., Körtzinger, A., and Melzner, F. (2013). Food availability outweighs ocean acidification effects in juvenile *Mytilus edulis*: laboratory and field experiments. Global Change Biol. *19*, 1017–1027. https://doi.org/10.1111/gcb.12109.
229. Pernet, F., Tamayo, D., Fuhrmann, M., and Petton, B. (2019). Deciphering the effect of food availability, growth and host condition on disease susceptibility in a marine invertebrate. J. Exp. Biol. *222*, jeb.210534. https://doi.org/10.1242/jeb.210534.
230. Cooley, S.D., Schoeman, L., Bopp, P., Boyd, P., Donner, S., Kiessling, W., Martinetto, P., Ojea, E., Racault, M.F., Rost, B., and Skern-Mauritzen, M. (2022). Oceans and coastal ecosystems and their services. In Climate Change 2022: Impacts, Adaptation and Vulnerability. Contribution of Working Group II to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change, H.-O. Pörtner, D.C. Roberts, M. Tignor, E.S. Poloczanska, K. Mintenbeck, A. Alegría, M. Craig, S. Langsdorf, S. Löschke, and V. Möller, et al., eds. (Cambridge University Press), pp. 379–550. https://doi.org/10.1017/9781009325844.005.
231. Pernet, F., Fuhrmann, M., Petton, B., Mazurié, J., Bouget, J.F., Fleury, E., Daigle, G., and Gernez, P. (2018). Determination of risk factors for herpesvirus outbreak in oysters using a broad-scale spatial epidemiology framework. Sci. Rep. *8*, 10869. https://doi.org/10.1038/s41598-018-29238-4.
232. Gangnery, A., Normand, J., Duval, C., Cugier, P., Grangeré, K., Petton, B., Petton, S., Orvain, F., and Pernet, F. (2019). Connectivities with shellfish farms and channel rivers are associated with mortality risk in oysters. Aquac. Environ. Interact. *11*, 493–506.
233. Fuhrmann, M., Petton, B., Quillien, V., Faury, N., Morga, B., and Pernet, F. (2016). Salinity influences disease-induced mortality of the oyster *Crassostrea gigas* and infectivity of the ostreid herpesvirus 1 (OsHV-1). Aquac. Environ. Interact. *8*, 543–552.
234. Fallet, M., Montagnani, C., Petton, B., Dantan, L., de Lorgeril, J., Comarmond, S., Chaparro, C., Toulza, E., Boitard, S., Escoubas, J.M., et al. (2022). Early life microbial exposures shape the *Crassostrea gigas* immune system for lifelong and intergenerational disease protection. Microbiome *10*, 85. https://doi.org/10.1186/s40168-022-01280-5.
235. Gawra, J., Valdivieso, A., Roux, F., Laporte, M., de Lorgeril, J., Gueguen, Y., Saccas, M., Escoubas, J.M., Montagnani, C., Destoumieux-Garzón, D., et al. (2023). Epigenetic variations are more substantial than genetic variations in rapid adaptation of oyster to Pacific oyster mortality syndrome. Sci. Adv. *9*, eadh8990. https://doi.org/10.1126/sciadv.adh8990.
236. Rodgers, E.M., and Gomez Isaza, D.F. (2023). The mechanistic basis and adaptive significance of cross-tolerance: a 'pre-adaptation' to a changing world? J. Exp. Biol. *226*, 245644. https://doi.org/10.1242/jeb.245644.
237. Shields, J.D. (2013). Complex etiologies of emerging diseases in lobsters (*Homarus americanus*) from Long Island Sound. Can. J. Fish. Aquat. Sci. *70*, 1576–1587. https://doi.org/10.1139/cjfas-2013-0050.
238. Chistoserdov, A.Y., Quinn, R.A., Gubbala, S.L., and Smolowitz, R. (2012). Bacterial communities associated with lesions of shell disease in the American lobster, *Homarus americanus* Milne-Edwards. J. Shellfish Res. *31*, 449–462. https://doi.org/10.2983/035.031.0205.
239. Meres, N.J., Ajuzie, C.C., Sikaroodi, M., Vemulapalli, M., Shields, J.D., and Gillevet, P.M. (2012). Identification of dysbiotic agents in epizootic shell disease of the American lobster (*Homarus americanus*) using discriminant analysis. J. Shellfish Res. *31*, 463–472. https://doi.org/10.2983/035.031.0205.
240. Quinn, R.A., Cawthorn, R.J., Summerfield, R.L., Smolowitz, R., and Chistoserdov, A.Y. (2013). Bacterial communities associated with lesions of two forms of shell disease in the American lobster (*Homarus americanus*, Milne Edwards) from Atlantic Canada. Can. J. Microbiol. *59*, 380–390. https://doi.org/10.2983/035.031.0216.
241. Jacobs, M., Laufer, H., Stuart, J., Chen, M., and Pan, X. (2012). Endocrine-disrupting alkylphenols are widespread in the blood of lobsters from southern New England and adjacent offshore areas. J. Shellfish Res. *31*, 563–571. https://doi.org/10.2983/035.031.0216.
242. Laufer, H., Chen, M., Johnson, M., Demir, N., and Bobbitt, J.M. (2012). The effect of alkylphenols on lobster shell hardening. J. Shellfish Res. *31*, 555–562. https://doi.org/10.2983/035.031.0215.
243. Barris, B.N., Shields, J.D., Small, H.J., Huchin-Mian, J.P., O'Leary, P., Shawver, J.V., Glenn, R.P., and Pugh, T.L. (2018). Laboratory studies on the effect of temperature on epizootic shell disease in the American lobster *Homarus americanus*. Bull. Mar. Sci. *94*, 887–902. https://doi.org/10.5343/BMS.2017.1148.
244. Hoenig, J.M., Groner, M.L., Smith, M.W., Vogelbein, W.K., Taylor, D.M., Landers, D.F., Jr., Swenarton, J.T., Gauthier, D.T., Sadler, P., Matsche, M.A., et al. (2017). Impact of disease on survival of three commercially fished species. Ecol. Appl. *27*, 2116–2127. https://doi.org/10.1002/eap.1595.
245. Wahle, R.A., Gibson, M., and Fogarty, M. (2009). Distinguishing disease impacts from larval supply effects in a lobster fishery collapse. Mar. Ecol. Prog. Ser. *376*, 185–192. https://doi.org/10.3354/meps07803.
246. Goldstein, J.S., Zarrella-Smith, K.A., and Pugh, T.L. (2022). Recent declines in American lobster fecundity in southern New England: drivers and implications. ICES J. Mar. Sci. *79*, 1662–1674. https://doi.org/10.1093/icesjms/fsac083.
247. Reardon, K.M., Wilson, C.J., Gillevet, P.M., Sikaroodi, M., and Shields, J.D. (2018). Increasing prevalence of epizootic shell disease in American lobster from the nearshore Gulf of Maine. Bull. Mar. Sci. *94*, 903–921. https://doi.org/10.5343/bms.2017.1144.
248. Maynard, J., van Hooidonk, R., Harvell, C.D., Eakin, C.M., Liu, G., Willis, B.L., Williams, G.J., Groner, M.L., Dobson, A., Heron, S.F., et al. (2016). Improving marine disease surveillance through sea temperature monitoring, outlooks and projections. Phil. Trans. Royal Soc. B. *371*, 20150208. https://doi.org/10.1098/rstb.2015.0208.
249. Herrero, A., Thompson, K.D., Ashby, A., Rodger, H.D., and Dagleish, M.P. (2018). Complex gill disease: an emerging syndrome in farmed Atlantic salmon (*Salmo salar* L.). J. Comp. Pathol. *163*, 23–28. https://doi.org/10.1016/j.jcpa.2018.07.004.
250. Boerlage, A.S., Ashby, A., Herrero, A., Reeves, A., Gunn, G.J., and Rodger, H.D. (2020). Epidemiology of marine gill diseases in Atlantic salmon (*Salmo salar*) aquaculture: a review. Rev. Aquacult. *12*, 2140–2159. https://doi.org/10.1111/raq.12426.
251. Marcos-López, M., and Rodger, H.D. (2020). Amoebic gill disease and host response in Atlantic salmon (*Salmo salar* L.): A review. Parasite Immunol. *42*, e12766. https://doi.org/10.1111/pim.12766.
252. Zawisza, M., Chadzinska, M., Steinhagen, D., Rakus, K., and Adamek, M. (2024). Gill disorders in fish: Lessons from poxvirus

infections. Rev. Aquacult. *16*, 234–253. https://doi.org/10.1111/raq.12835.
253. Rodger, H.D. (2007). Gill disorders: an emerging problem for farmed Atlantic salmon (*Salmo salar*) in the marine environment? Fish Vet. J. *9*, 38–48.
254. Gjessing, M.C., Thoen, E., Tengs, T., Skotheim, S.A., and Dale, O.B. (2017). Salmon gill poxvirus, a recently characterized infectious agent of multifactorial gill disease in freshwater- and seawater-reared Atlantic salmon. J. Fish. Dis. *40*, 1253–1265. https://doi.org/10.1111/jfd.12608.
255. Boerlage, A.S., Ashby, A., Eze, J.I., Gunn, G.J., and Reeves, A. (2022). Field evaluation of diagnostic sensitivity (DSe) and specificity (DSp) of common tests for amoebic gill disease (AGD) and complex gill disease (CGD) in cultured Atlantic salmon (*Salmo salar*) in Scotland using Bayesian latent class models. Prev. Vet. Med. *204*, 105654. https://doi.org/10.1016/j.prevetmed.2022.105654.
256. Herrero, A., Rodger, H., Hayward, A.D., Cousens, C., Bron, J.E., Dagleish, M.P., and Thompson, K.D. (2022). Prospective Longitudinal Study of Putative Agents Involved in Complex Gill Disorder in Atlantic salmon (*Salmo salar*). Pathogens *11*, 878. https://doi.org/10.3390/pathogens11080878.
257. Østevik, L., Stormoen, M., Hellberg, H., Kraugerud, M., Manji, F., Lie, K.I., Nødtvedt, A., Rodger, H., and Alarcón, M. (2022). A cohort study of gill infections, gill pathology and gill-related mortality in sea-farmed Atlantic salmon (*Salmo salar* L.): A descriptive analysis. J. Fish. Dis. *45*, 1301–1321. https://doi.org/10.1111/jfd.13662.
258. Gamperl, A.K., Zrini, Z.A., and Sandrelli, R.M. (2021). Atlantic Salmon (*Salmo salar*) Cage-site distribution, behavior, and physiology during a Newfoundland heat wave (2021). Front. Physiol. *24*, 719594. https://doi.org/10.3389/fphys.2021.719594.
259. Benedicenti, O., Pottinger, T.G., Collins, C., and Secombes, C.J. (2019). Effects of temperature on amoebic gill disease development: Does it play a role? J. Fish. Dis. *42*, 1241–1258. https://doi.org/10.1111/jfd.13047.
260. Collins, C., Hall, M., Fordyce, M.J., and White, P. (2019). Survival and growth in vitro of *Paramoeba perurans* populations cultured under different salinities and temperatures. Protist *170*, 153–167. https://doi.org/10.1016/j.protis.2018.11.003.
261. Beaugrand, G., and Reid, P.C. (2012). Relationships between North Atlantic salmon, plankton, and hydroclimatic change in the Northeast Atlantic. ICES J. Mar. Sci. *69*, 1549–1562. https://doi.org/10.1093/icesjms/fss153.
262. Díaz, P.A., Pérez-Santos, I., Basti, L., Garreaud, R., Pinilla, E., Barrera, F., Tello, A., Schwerter, C., Arenas-Uribe, S., Soto-Riquelme, C., et al. (2023). The impact of local and climate change drivers on the formation, dynamics, and potential recurrence of a massive fish-killing microalgal bloom in Patagonian fjord. Sci. Total Environ. *865*, 161288. https://doi.org/10.1016/j.scitotenv.2022.161288.
263. Clinton, M., Ferrier, D.E.K., Martin, S.A.M., and Brierley, A.S. (2021). Impacts of jellyfish on marine cage aquaculture: an overview of existing knowledge and the challenges to finfish health. ICES J. Mar. Sci. *78*, 1557–1573. https://doi.org/10.1093/icesjms/fsaa254.
264. Matthews, C.G.G., Richards, R.H., Shinn, A.P., and Cox, D.I. (2013). Gill pathology in Scottish farmed Atlantic salmon, *Salmo salar* L., associated with the microsporidian *Desmozoon lepeophtherii* Freeman et Sommerville, 2009. J. Fish. Dis. *36*, 861–869. https://doi.org/10.1111/jfd.12084.
265. Mowi (2023). Mowi Integrated Annual Report 2022. https://mowi.com/blog/mowi-annual-report-2023.
266. Sommerset, I., Wiik-Nielsen, J., Henrique Silva de Oliveira, V., Moldal, T., Bornø, G., Haukaas, A., and Brun, E. (2023). Norwegian Fish Health Report 2022. www.vetinst.
267. Ahmed, N., and Turchini, G.M. (2021). Recirculating aquaculture systems (RAS): Environmental solution and climate change adaptation. J. Clean. Prod. *297*, 126604. https://doi.org/10.1016/j.clepro.2021.126604.
268. Adams, A. (2019). Progress, challenges and opportunities in fish vaccine development. Fish Shellfish Immunol. *90*, 210–214. https://doi.org/10.1016/j.fsi.2019.04.066.
269. Bedekar, M.K., and Kole, S. (2022). Fundamentals of fish vaccination. Methods Mol. Biol. *2411*, 1147–1173. https://doi.org/10.1007/978-1-0716-1888-2_9.
270. McCusker, S., Warberg, M.B., Davies, S.J., Valente, C.d.S., Johnson, M.P., Cooney, R., and Wan, A.H.L. (2023). Biofloc technology as part of a sustainable aquaculture system: A review on the status and innovations for its expansion. Aquac. Fish Fish. *3*, 331–352. https://doi.org/10.1002/aff2.108.
271. Liu, R., Han, G., Li, Z., Cun, S., Hao, B., Zhang, J., and Liu, X. (2022). Bacteriophage therapy in aquaculture: current status and future challenges. Folia Microbiol. *67*, 573–590. https://doi.org/10.1007/s12223-022-00965-6.
272. Rowley, A.F. (2022). Bacterial diseases of crustaceans. In Invertebrate Pathology, A.F. Rowley, C.J. Coates, and M.M.A. Whitten, eds. (Oxford University Press), pp. 400–435. https://doi.org/10.1093/oso/9780198853756.003.0015.
273. Bhat, R.A.H., Thakuria, D., Tandel, R.S., Khangembam, V.C., Dash, P., Tripathi, G., and Sarma, D. (2022). Tools and techniques for rational designing of antimicrobial peptides for aquaculture. Fish Shellfish Immunol. *127*, 1033–1050. https://doi.org/10.1016/j.fsi.2022.07.055.
274. García-Beltrán, J.M., Arizcun, M., and Chaves-Pozo, E. (2023). Antimicrobial peptides from photosynthetic marine organisms with potential application in aquaculture. Mar. Drugs *8*, 290. https://doi.org/10.3390/md21050290.
275. Groner, M.L., Maynard, J., Breyta, R., Carnegie, R.B., Dobson, A., Friedman, C.S., Froelich, B., Garren, M., Gulland, F.M.D., Heron, S.F., et al. (2016). Managing marine disease emergencies in an era of rapid change. Phil. Trans. Roy. Soc. B. *371*, 20150364. https://doi.org/10.1098/rstb.2015.0364.
276. Yang, Z., Yu, Y., Tay, Y.X., and Yue, G.H. (2021). Genome editing and its applications in genetic improvement in aquaculture. Rev. Aquacult. *14*, 178–191. https://doi.org/10.1111/raq.12591.
277. Sae-Lim, P., Kause, A., Mulder, H.A., and Olesen, I. (2017). Climate change and selective breeding in aquaculture. J. Anim. Sci. *95*, 1801–1812. https://doi.org/10.2527/jas2016.1066.
278. Boudry, P., Allal, F., Aslam, M.L., Bargelloni, L., Bean, T.P., Brard-Fudulea, S., Brieuc, M.S., Calboli, F.C., Gilbey, J., Haffray, P., et al. (2021). Current status and potential of genomic selection to improve selective breeding in the main aquaculture species of International Council for the Exploration of the Sea (ICES) member countries. Aquac. Rep. *20*, 100700. https://doi.org/10.1016/j.aqrep.2021.100700.
279. Dunham, R.A. (2023). Aquaculture and Fisheries Biotechnology: Genetic Approaches, 3rd Edition (CABI), p. 584. https://doi.org/10.1079/9781789243468.0000.
280. Frank-Lawale, A., Allen, S.K., and Dégremont, L. (2014). Breeding and domestication of eastern oyster (*Crassostrea virginica*) lines for culture in the mid-Atlantic, USA: line development and mass selection for disease resistance. J. Shellfish Res. *33*, 153–165. https://doi.org/10.2983/035.033.0115.
281. Houston, R.D., Bean, T.P., Macqueen, D.J., Gundappa, M.K., Jin, Y.H., Jenkins, T.L., Selly, S.L.C., Martin, S.A.M., Stevens, J.R., Santos, E.M., et al. (2020). Harnessing genomics to fast-track genetic improvement in aquaculture. Nat. Rev. Genet. *21*, 389–409. https://doi.org/10.1038/s41576-020-0227-y.
282. Potts, R.W.A., Gutierrez, A.P., Penaloza, C.S., Regan, T., Bean, T.P., and Houston, R.D. (2021). Potential of genomic technologies to improve disease resistance in molluscan aquaculture. Philos. Trans. R. Soc. Lond. B Biol. Sci. *376*, 20200168. https://doi.org/10.1098/rstb.2020.0168.
283. George, M.N., Cattau, O., Middleton, M.A., Lawson, D., Vadopalas, B., Gavery, M., and Roberts, S.B. (2023). Triploid Pacific oysters exhibit stress response dysregulation and elevated mortality following heatwaves. Global Change Biol. *29*, 6969–6987. https://doi.org/10.1111/gcb.16880.
284. Audemard, C., Cator, H.I., and Reece, K.S. (2018). High salinity relay as post-harvest processing method for reducing *Vibrio vulnificus* levels in oysters (*Crassostrea virginica*). Int. J. Food Microbiol. *279*, 70–79. https://doi.org/10.1016/j.ijfoodmicro.2018.04.043.
285. Elmahdi, S., Parveen, S., Ossai, S., DaSilva, L.V., Jahncke, M., Bowers, J., and Jacobs, J. (2018). *Vibrio parahaemolyticus* and *Vibrio vulnificus* recovered from oysters during an oyster relay study. Appl. Environ. Microbiol. *84*, e01790-17. https://doi.org/10.1128/AEM.01790-17.
286. Ward, J.R., and Lafferty, K.D. (2004). The elusive baseline of marine disease: Are diseases in ocean ecosystems increasing? PLoS Biol. *2*, e120. https://doi.org/10.1371/journal.pbio.0020120.
287. Barange, M., Bahri, T., Beveridge, M.C.M., Cochrane, K.L., Funge-Smith, S., and Poulain, F. (2018). Impacts of climate change on fisheries and aquaculture: synthesis of current knowledge, adaptation and mitigation options. In FAO Fisheries and Aquaculture Technical Paper No. 627 (FAO), p. 628.