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The impact of climate change on the parasites and infectious diseases of aquatic animals

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Summary

Climate change is predicted to have important effects on parasitism and disease in freshwater and marine ecosystems, with consequences for human health and socio-economics. The distribution of parasites and pathogens will be directly affected by global warming, but also indirectly, through effects on host range and abundance. To date, numerous disease outbreaks, especially in marine organisms, have been associated with climatic events such as the El Niño-Southern Oscillation. In general, transmission rates of parasites and pathogens are expected to increase with increasing temperature. Evidence suggests that the virulence of some pathogens and parasites may also increase with global warming. The effects of climate change on parasites and pathogens will be superimposed onto the effects of other anthropogenic stressors in ecosystems, such as contaminants, habitat loss and species introductions. This combination of stressors may work cumulatively or synergistically to exacerbate negative effects on host organisms and populations. Climatic effects on parasites and diseases of key species may cascade through food webs, with consequences for entire ecosystems.

Keywords

Aquatic animals – Climate change – Disease – Freshwater – Marine – Parasites – Pathogens – Temperature.

Introduction

All aquatic ecosystems, including freshwater lakes and rivers, coastal estuarine habitats and marine waters, are influenced by climate change (74, 99, 101). For example, relatively small temperature changes alter fish metabolism and physiology, with consequences for:

- growth
- fecundity
- feeding behaviour
- distribution
- migration
- abundance (21, 89, 90, 91, 120).

These biological changes will incur economic consequences for subsistence, commercial and recreational fisheries, along with the people and industries associated with fishing (21, 74, 91, 101). The impacts of climate change have been reviewed and summarised for freshwater and marine ecosystems (74, 96). At this point in time, climate change has led to marked alterations in the distribution of invertebrate, vertebrate and plant species, the timing of their seasonal activities and their physiological responses in both aquatic and terrestrial environments (73, 93, 96, 108, 116). These organisms include marine zooplankton and fish (2, 78). Moreover, threats are imminent, according to recent analyses predicting significant climate warming in the next decade (106). An increase in temperature of some 2°C to 3°C above pre-industrial levels is expected to place 20% to 30%

of plant and animal species at high risk of extinction, while major changes to the structure and function of marine and other aquatic ecosystems are anticipated (22).

Climate change will have a profound impact on the spread of parasites and disease in aquatic ecosystems (28, 29, 59). Not only will climate change affect parasite species directly, but also through changes in the distribution and abundance of their hosts (17, 59). The emergence of disease is linked directly to changes in the ecology of hosts or pathogens, or both (13, 28). The most apparent effects are likely to result from the extension of the geographical range of pathogens (29). In addition, increased temperature may cause thermal stress in aquatic animals, leading to reduced growth, sub-optimal behaviours and reduced immunocompetence (21, 28, 29, 91). Outbreaks of numerous diseases, including those of humans, have been associated with climate, in particular the El Niño-Southern Oscillation (ENSO) (13, 28, 29). In the oceans, diseases are shown to increase in corals, sea urchins, molluscs, sea turtles and marine mammals, although not all can be linked unequivocally to climate alone (48). Disease outbreaks are extremely costly economically. An epizootic causing mass mortalities of pilchard (*Sardinops sagax*) cost the fishery US$ 3 million over three years (13). However, it was recently suggested that diseases may not increase with climate change, although distributions of parasites and pathogens will undoubtedly shift (46). Other factors may dominate over climate in controlling the distribution and abundance of pathogens, including:

- habitat alteration
- invasive species
- agricultural practices
- human activities.

Nevertheless, diseases and parasites can have a grave impact on entire ecosystems. The mechanisms by which the effects of global warming influence disease occurrence are illustrated in Figure 1.

The repercussions of climate change are not limited solely to temperature effects on hosts and their parasites, but also include:

- alterations in water levels and flow regimes
- eutrophication
- stratification
- changes in acidification
- reduced ice cover
- changes in ocean currents
- increased ultra-violet (UV) light penetration
- runoff
- weather extremes.

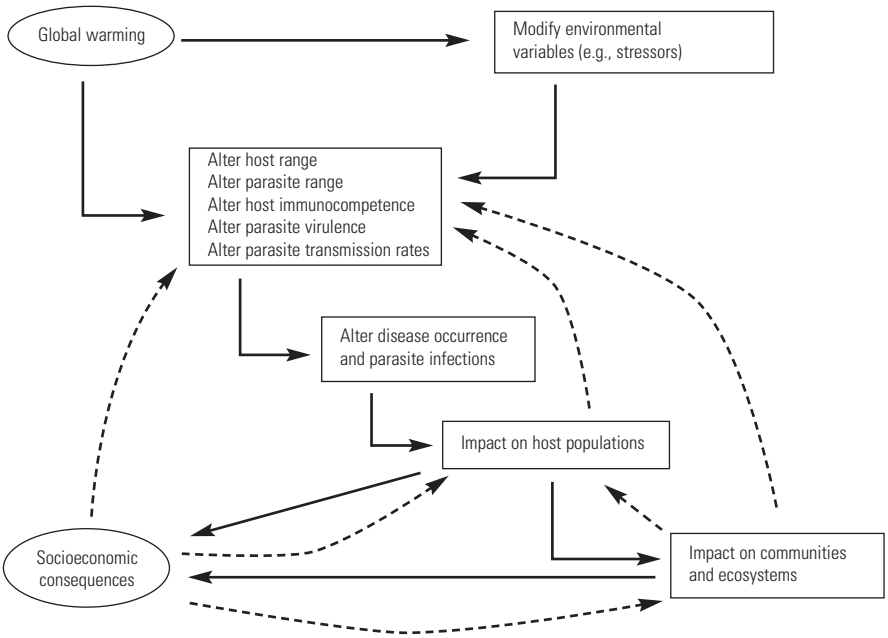

Fig. 1
Schematic representation of effects of global warming on parasites and hosts
Effects will cascade up, leading to impacts on host populations, communities and ecosystems. Effects will be modified by interactions with other stressors and environmental variables. Effects on populations, communities and ecosystems will 'feed back' onto hosts and their parasites (dashed lines)

All these factors will have consequences for entire ecosystems and their food webs (59).

While changes in the effects of parasites and pathogens, due to modifications in these environmental variables, may ultimately derive from climate change, it will be difficult to determine actual causation in many cases. Other stressors will also increase the effects of climate change (see 'Interactions with other stressors', below) (74). Table I contains examples of how environmental perturbations related to climate change may affect host-parasite systems. Numerous host-parasite systems are affected by climate,

Table I
Examples and predictions of environmental changes or biological effects associated with global warming and resulting alterations to host-parasite relationships in aquatic ecosystems (reviewed in 59, 60, 61)

Effect	General response	Example
Species introductions with changes in host range	Introduced parasites and pathogens	83 potential parasite invaders in the Great Lakes
		Episodic outbreaks of introduced pathogens
	Change in habitat and diet of native species	Change in parasite fauna of native fish after introductions of bait fish
Loss of habitat	Local extirpation of hosts (e.g. cold-water salmonids)	Loss of salmonid parasites
Loss of synchronicity in predator-prey populations	Disruption of parasite life cycles	Reduction in abundance and prevalence of parasites transmitted during fish spawning
Altered hydrology	Lower water levels and flow rates (e.g. in the Great Lakes and St Lawrence River)	Retention of free-living infective stages of parasites
		Promotion of macrophytes and invertebrate intermediate hosts (e.g. molluscs)
		Increased prevalence and abundance of parasites with direct life cycles and eyeflukes, blackspot and white grub in fish
		Increase in swimmer's itch in humans
	Loss of wetlands and increased concentration of waterfowl	Increase in avian cholera
Eutrophication	Shift in host species distributions and composition; anoxia in hypolimnion	Increase in parasites of cyprinids, parasites using birds as definitive hosts, trematodes, cestodes and parasites with relatively short life cycles. Decrease in parasites of salmonids, nematodes, acanthocephalans
		Increased rate of deformities in frogs due to *Ribeiroia*
	Increase in oligochaetes	Increased mortality in wading birds due to *Eustrongylides*
		Increase in myxozoans in fish
Increased stratification	Anoxia in hypolimnion	Increase in zooplankton-transmitted parasites. Reduction in parasites of cold-water stenotherms
	Lower thermocline	
	Reduced hypolimnetic refuge for cold-water species	
Reduced ice cover	Presence of migratory birds year round	Transmission of parasites year round
	Reduced breeding habitat for seals	Reduction of anisakid nematodes in fish in the Gulf of St Lawrence
	Concentrations of ice-breeding seals on islands	Shift in distribution of fish parasites that mature in seals around islands
Increased acidification in headwater streams	Decline in diversity of fish, zooplankton, macro-invertebrates	Decline in parasite diversity
		Change in parasite species composition
	Local extirpation of molluscs	Loss of trematodes
Decreased acidification in lakes	Increased diversity of invertebrates and fish	Increase in parasite diversity
		Increase in trematodes
Altered oceanic circulation	Increase in cold intermediate layer in the Gulf of St Lawrence	Decline in sealworm, increase in other anisakid nematodes in groundfish
Warming of coastal waters	Shift in host ranges	Introductions of pathogens
	More pelagic species with shift of warm currents north	Increase in whale worm (*Anisakis* spp.) in fish
Increase in ultraviolet (UV) radiation	Direct damage to parasites	Decline in parasites with UV-sensitive infective stages (e.g. whirling disease, eyeflukes)
	Increased susceptibility to pathogens	Mortality of frog embryos infected with water mould
Rise in sea level	Salt water intrusion into fresh water	Decline in freshwater parasites, increase in estuarine parasites
		Modification of parasite host specificity and expansion of host spectra
Socio-economic adaptation (reservoir construction)	Replacement of fauna characteristic of lotic conditions with species typical of still or slow-moving waters	Shift from benthic-transmitted parasites to zooplankton-transmitted parasites
		Increased prevalence and abundance of eyeflukes in fish and amphibians

whether ENSO and the North Atlantic Oscillation (NAO) directly affect parasite life cycles or indirectly affect host populations and communities. Many of these effects are summarised by Mouritsen and Poulin (68). These parasites and pathogens include viruses, protozoans, monogeneans, trematodes, cestodes and nematodes infecting cnidarians, molluscs, crustaceans, echinoderms, fish species and anurans in marine and freshwater habitats. How any particular parasite or pathogen will respond is species specific and dependent on context (59), thus making predictability difficult. Predictability is further confounded by the occurrence of extreme events and non-linear thresholds in disease dynamics and climatic processes (29).

However, some general predictions do emerge. Longer growing seasons and higher temperatures should lead to more generations of parasites annually and more frequent outbreaks of disease resulting from increased transmission rates (34, 59). Table II summarises the effects of increased temperature on different aspects of the parasite life cycle and host biology that subsequently affect transmission. Parasites and pathogens with complex life cycles, or those in poikilothermic hosts, may be disproportionately affected by global warming (29). Moreover, disease epizootics in the oceans may become more common if temperature anomalies increase in frequency or intensity (6).

Parasites may respond to increasing temperatures more strongly than their hosts. Trematodes are among the most common parasites of vertebrates. They require a molluscan intermediate host in which infective cercariae are asexually produced and released into the water in search of the next host in the life cycle. In a review of temperature effects on cercarial shedding, it was shown that temperature differentially affected cercarial production (82). The average increase across trematode species was an eight-fold increase with a 10°C rise in temperature, which is much higher than the two-to-three-fold elevation predicted by standard metabolic principles. Small increases in temperature should promote the proliferation of cercariae, with little or no reduction in transmission efficiency. Even short-term spikes in cercarial emergence are sufficient to cause mortality in the next host, with consequences that may cascade through the ecosystem (see 'Ecosystem effects', below) (82). Furthermore, there may be serious consequences for human populations at risk of trematode diseases, such as schistosomiasis, if temperatures increase.

Temperature and virulence

Numerous diseases display greater virulence at higher temperatures. This may be the result of reduced resistance due to stress, increased virulence factors or increased transmission (29, 52). Susceptibility of largemouth bass (*Micropterus salmoides*) to red-sore disease, caused by the bacterium *Aeromonas hydrophila*, increases at warmer temperatures (20). The survival of mosquitofish (*Gambusia affinis*) infected with the Asian fish tapeworm (*Bothriocephalus acheilognathi*) is reduced at high temperatures when compared with that of uninfected fish, and those with high numbers of worms die faster than those with fewer parasites (26). The severity of whirling disease, caused by the myxozoan *Myxobolus cerebralis* in trout (*Oncorhynchus* spp.), is correlated with temperature (31). Blackspot disease in juvenile coho salmon (*O. kisutch*), caused by trematode larvae known as metacercariae, directly correlates with the daily maximum temperature averaged over the previous seven days (8).

Examples also exist of pathogen changes with temperature that may explain increased virulence under warmer conditions. At higher temperatures, virulence genes are expressed in the bacterium *Vibrio shiloi*, a putative causative agent of coral bleaching in *Oculina patagonica* (95). Virulent strains of the bacterium *Flavobacterium columnare*, the causative agent of columnaris disease in fish, adhere more strongly to gill tissue at high temperatures, as well as under conditions of high organic loading (14).

Table II

General effects of increased temperature on parasite life cycles, their hosts and transmission processes

This summary is adapted from Marcogliese (59). Note that, while temperature enhances most effects, once a thermal tolerance limit is approached for parasites or hosts, the effects tend to diminish

Effects on parasites	Effects on hosts	Effects on transmission
Faster embryonic development and hatching	Altered feeding	Earlier reproduction in spring
Faster rates of development and maturation	Altered behaviour	More generations per year
Decreased longevity of larvae and adults	Altered range	Prolonged transmission in the fall
Increased mortality of all stages	Altered ecology	Potential transmission year round
	Reduced host resistance	

Diseases in marine invertebrates

The diseases of aquatic organisms with the strongest links to climate are those of marine invertebrates, including molluscs, echinoderms and cnidarians. Mass mortalities related to high temperature have been documented in sea grasses, oysters, starfish, corals, abalone and sea urchins (28). The populations affected are key constituents in marine habitats, where the effects of disease cascade throughout the local ecosystem, affecting entire communities of organisms, as well as any commercial activities dependent on these natural resources (6, 28).

Among the most dramatic diseases to emerge and increase in recent years are those of corals. Entire reefs are affected across large spatial scales. A number of these diseases have been linked to climate and ENSO events (28, 29). The frequency of warm temperature anomalies is correlated with the occurrence of different diseases of corals over a large area across the Great Barrier Reef (6). Disease agents are often opportunistic facultative pathogens that infect corals suffering from physiological stress resulting from exposure to high temperatures (28, 29, 52). High temperatures may also increase virulence factors in pathogens (52, 95), as well as pathogen growth, development and transmission (29). While coral bleaching is not typically considered a disease, it may be caused by a bacterium (*V. shiloi*) which becomes pathogenic, due to increased sensitivity of corals, increased virulence and greater rates of transmission at high temperatures (95). Aspergillosis, which is caused by a fungus (*Aspergillus sydowii*), is an opportunistic pathogen on a wide range of taxa. Though this disease has declined in recent years in the Florida Keys, two separate epizootics on sea fan corals (*Gorgonia ventalina*) that destroyed more than 50% of sea fan tissue coincided with warm water periods (42, 117). It is suggested that accelerated pathogen growth in conjunction with reduced host resistance at high temperatures is likely to result in a higher frequency of disease outbreaks. It is further thought that the pathogen growth rate accelerates faster at warmer temperatures than host resistance capability (117). The fungal pathogen obtains optimal virulence at 30ºC, whereas warm temperatures stress corals, increasing their susceptibility to attack by pathogens.

Transmission of black band disease (BBD) on staghorn coral (*Acropora muricata*) also increases at higher temperatures at Lizard Island in the Great Barrier Reef (4). Small increases in temperature cause this disease to spread, suggesting that virulence is enhanced while resistance declines (4). Warmer temperatures that lead to disease are close to the upper thermal limit of the coral, suggesting they are stressed. Loss of corals affects entire reef communities, having a profound impact on both ecotourism and those fisheries that depend on reefs and reef organisms as a resource.

The oyster *Crassostrea virginica* represents one of the most important fisheries on the east coast of North America. The fishery endures significant losses due to the protozoan *Perkinsus marinus*, the causative agent of dermo disease. In Chesapeake Bay, oyster beds were reduced to 5% of their former territory after *P. marinus* became established and the harvest was reduced from more than three million bushels (100,000 m^3) to less than half a million (17,000 m^3) (7). The establishment of *P. marinus* in the bay followed consecutive drought years, which resulted in elevated salinities, and warmer winters (7). Development of the pathogen is further prolonged with subsequent warm spring and/or fall seasons. In the Gulf of Mexico, the distribution of this pathogen is linked to precipitation patterns and the maximum monthly temperature experienced in the two previous months. Here, climate shifts are also responsible for annual variations across large spatial scales of > 1,200 km (85). Epizootics in the Gulf are favoured by dry summers followed by warm winters (86). Disease outbreaks have now occurred as far north as Cape Cod, Massachusetts (23, 24), in areas previously undisturbed because of their lower temperatures. Similarly, epizootics of MSX disease on oysters have spread northwards (32). In this case, the causative protozoan, *Haplosporidium nelsoni*, appears limited by low temperature, reducing parasite activity. The range extension and high prevalence of this disease follow seasonally warm winters in New England that permit significant overwinter survival (24). Once the protozoan is established, an important management question for the fishery is whether or not to maintain the ban on introduced oysters for seeding beds (23). Black abalone (*Haliotis cracherodii*) is another mollusc that has experienced mass mortalities and a northward expansion of disease range. The mortalities coincided, to some extent, with periods of warmer sea temperatures and ENSO events (88).

Massive die-offs of the green sea urchin, *Strongylocentrotus droebachiensis*, in coastal north-eastern North America are associated with warmer sea surface temperatures, as well as higher tropical storm and hurricane activity (100). The pathogen causing the mortalities is the protozoan *Paramoeba invadens*. This parasite cannot survive in waters below 2ºC and is not transmitted below 12ºC. Widespread mortalities occurred during summer and fall when water temperatures exceeded 15ºC. Outbreaks result in wholesale ecosystem changes as urchin-dominated barrens (i.e. areas devoid of seaweeds) are subsequently colonised by kelp, other macro-algae, and their associated fauna (100). On the west coast of North America, the purple sea

urchin (*S. purpuratus*) has experienced numerous epidemics in past decades, caused by unknown pathogens, more common in the warmer waters found off southern California (53). Again, thermal stress may play a role here. These sea urchins are the targets of developing fisheries, so such ecosystem shifts will require corresponding adjustment and changes to those fisheries that use this resource.

Recently, outbreaks of bitter crab disease in snow crabs (*Chionoecetes opilio*) off Newfoundland have been associated with warmer temperatures (104). The disease, caused by parasitic dinoflagellates (*Hematodinium* spp.), has spread rapidly since first reported in 1990, and proves fatal to virtually every infected crab species. This, in turn, affects the marketability of entire catches of live crabs, resulting in severe financial losses to crab fisheries. Global warming may extend the transmission season and increase prevalence in crab populations, thus the disease may spread to additional crab fisheries (104).

The impacts of these outbreaks are severe. They directly affect the fisheries of sea urchins, abalone, crabs and especially oysters. Stress on foundation species, such as corals, that comprise the structure and habitat upon which numerous other organisms depend, and their subsequent loss significantly alters ecosystems and affects established local economies (22). Keystone consumers, such as urchins, actually control the benthic landscape by preventing the establishment of kelp-dominated communities. In all cases, consequences are environmentally and economically severe.

Freshwater systems

There are not as many powerful demonstrations of the impact of climate change on freshwater parasites or pathogens, with the possible exception of chytridiomycosis in amphibians (see below). However, the general effects of increased temperature on parasites include:

– rapid growth and maturation

– earlier onset of spring maturation

– increased parasite mortality

– increased number of generations per year

– increased rates of parasitism and disease

– earlier and prolonged transmission

– the possibility of continuous, year-round transmission (59).

Effects are more profound in northern and southern latitudes, at some distance from the equator, because the occurrence and transmission of parasites are driven by seasonal temperature changes (27). *Argulus coregoni* is a crustacean ectoparasite that infects salmonids. In warmer temperatures, this parasite undergoes two annual generational cycles, as opposed to its usual single generation. This parasite is already problematic for aquaculture. Extended growth could require more applications of treatment for the aquaculturist, doubling the expense (27). Similarly, other parasites proliferate in aquaculture under warmer conditions, demanding more extensive control measures or treatment. Extended growing seasons also permit more rapid growth and more generations per year of intermediate hosts, such as snails. Gastropods transmit trematodes such as *Diplostomum* spp., a parasite which causes cataracts and blindness in fish, resulting in further problems for aquaculture (9). Thus, more rounds of treatment may be required to control vectors of pathogens (27).

Studies of the effects of thermal effluents illustrate the impact of increasing temperature on host-parasite systems (59). These studies demonstrate the difficulty of drawing conclusions or making predictions because effects differ among parasite species and are context-dependent. Parasite populations may remain level, increase or decrease, as may the populations of their intermediate and definitive hosts (80, 81). Indeed, even similar parasites that infect the same snails and fish may display different seasonal transmission patterns under the same thermally enhanced conditions (59). Nor are development and transmission always enhanced. While transmission may be extended into winter months, it may cease during the hottest summer months because temperatures become too warm, as was observed for *Ornithodiplostomum ptychocheilus* and *Tylodelphys scheuringi*, two strigeid trematodes of mosquitofish (*G. affinis*), in heated ponds in South Carolina (reviewed in 59).

Chytridiomycosis, caused by the chytrid fungus *Batrachochytrium dendrobatidis*, is responsible for global mass mortalities, population declines and extinctions of amphibian species (44, 105). This pathogen, known to be more prevalent during cooler months, displays distinct thermal preferences, ceasing to grow at 29ºC and dying at 30ºC to 32ºC (44, 84). Others have speculated that its occurrence may decline with global warming (29). Climatic conditions are linked to chytrid outbreaks and the extinction of at least two frog species in the mountains of Costa Rica (84). Declines in numerous anurans are linked to large-scale and local changes in climate. Conditions in tropical montane environments may be shifting towards optimum for chytrid epizootics. Within the mid-altitudinal range, where extinctions have been observed, days have cooled and nights have warmed. This promotes chytrid transmission in areas where it previously may have been held in check by warm daytime and cool night-time temperatures (84). Chytrid growth may be limited at

higher or lower elevations. Thus, in terms of mountain ecosystems, with climate change, alterations in the altitudinal distribution of chytridiomycosis may be expected, placing species and populations at risk. An extensive survey of chytrid infections in the stoney creek frog (*Litoria wilcoxii*), over a large geographical range in Australia, demonstrated a significant negative relationship between temperature and chytrid prevalence, as well as between prevalence and the previous 30-day rainfall (44). It is suggested that global warming will reduce the impact of chytridiomycosis in low-altitude frog populations in the tropics and subtropics. However, in mountains and temperate lowlands, chytrid infections may be expected to spread, contributing to further amphibian declines and possible extinctions (44).

Ecosystem effects

Any negative effects of climate change on biodiversity may affect disease transmission, because biodiversity may act as a buffer against disease propagation. Basically, the presence of incompetent disease reservoirs decreases the impact of highly competent reservoirs and reduces disease risk, a phenomenon known as the dilution effect (71, 103). Using the terrestrial model of Lyme disease and risk, it is shown that a richness of higher species reduces the risk of transmitting Lyme disease to humans. This model should apply to any disease or parasite:

– that requires horizontal transmission

– that uses a generalist vector or intermediate host, i.e. that is not host specific

– when reservoir hosts differ in their ability to transmit the pathogen

– when the most competent vector or intermediate host is dominant (71, 103).

The less competent reservoirs deflect the vectors away from those that would serve as better hosts for transmission. Anthropogenic changes that affect biodiversity will increase disease risk (55). There is every reason to suspect that this mechanism may function in aquatic ecosystems as well. Epidemiological models suggest that high species richness may buffer disease occurrence when transmission is dependent on a vector or intermediate host. This will have significant consequences if, with climate change, pathogens extend their distributional range from areas of high diversity, such as the tropics, into areas of low diversity, such as more temperate ecosystems (16).

Not only is diversity important, but food web structure also influences the occurrence of disease and parasitism. Any perturbations that reduce predator abundance may reduce prey mortality rates, resulting in epizootics of density-dependent pathogens in prey populations. This may occur through increases in the occurrence of disease in host populations where the pathogen already exists or via the spread of infection to new host populations (33). Thus, changes at higher trophic levels, that otherwise keep disease in check, may lead to disease outbreaks at lower trophic levels.

Parasites and diseases of foundation species or keystone consumers, considered keystone parasites, may affect their populations in such a way that the impacts cascade through entire communities and ecosystems (19, 57, 64). Diseases of corals and sea urchins fall into this category; corals being important foundation species for reef ecosystems, while urchins are considered keystone consumers (see 'Diseases in marine invertebrates', above). Thus, parasite and disease outbreaks in these organisms may have powerful and long-lasting effects on entire ecosystems (6). These effects can be economically very costly to industries and livelihoods that depend on biological resources from these environments (22).

Mass mortalities of mud snails, *Hydrobia ulvae*, and amphipods, *Corophium volutator*, have been observed on intertidal mudflats in the Danish Wadden Sea (37). These organisms serve as first and second intermediate hosts in the life cycles of microphallid trematodes; shorebirds and waterfowl being the definitive hosts. Numbers of mud snails declined and the amphipods disappeared altogether. Two microphallid trematodes, *Microphallus claviformis* and *Maritrema subdolum*, are implicated in the population crashes, all other factors being ruled out. The declines coincided with abnormally high temperatures. Subsequently, changes were seen in the benthic community and sediments (Fig. 2). The elimination of *C. volutator* transformed a heterogeneous mud flat, consisting of a mosaic of tidal pools and emergent sand bars, into a homogeneous mud flat (67). The median particle diameter of the sediments increased, silt content decreased, and chlorophyll *a* increased. These changes were attributed to the loss of the stabilising influence of amphipod tube construction. Moreover, the amphipod constituted a major food source for millions of migrating shorebirds (69). High temperatures coinciding with the NAO increased the development rate and release of free-living infective stages of the microphallid trematodes, thus causing extensive mortality among the gastropod hosts and eliminating the amphipod population (68).

The NAO is associated with high temperatures, strong winds and precipitation in Europe (68, 72). It is essentially the North Atlantic equivalent of ENSO, and its direct and indirect effects on terrestrial and marine populations are well documented (72). The rate of cercarial emergence

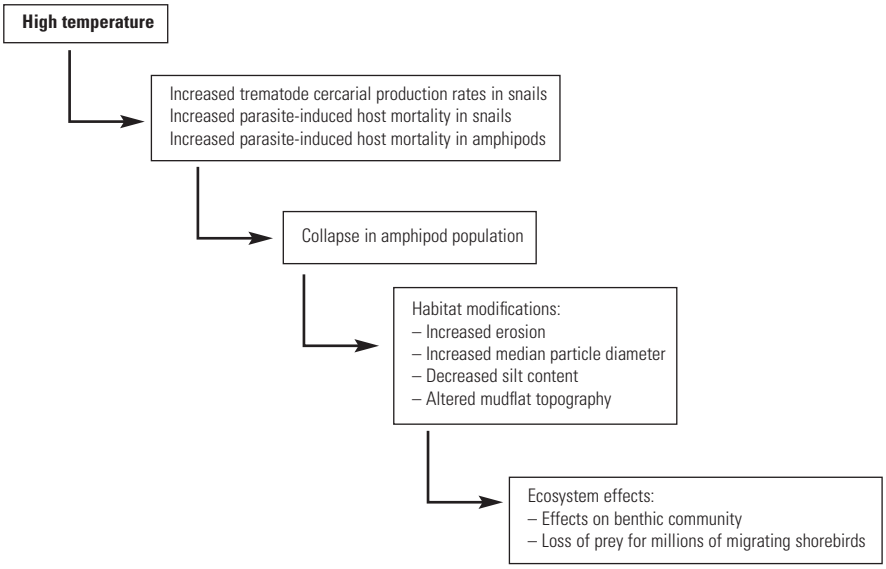

Fig. 2
Cascading effects of increased temperature due to climate change and/or the North Atlantic Oscillation on the Wadden Sea ecosystem in Denmark
Higher temperatures cause increased proliferation of infective stages of the trematodes *Microphallus claviformis* and *Maritrema subdolum* in the gastropod first intermediate host, the mud snail *Hydrobia ulvae*. Increased rates of parasitism cause mortality in both mud snails and the second intermediate host, the amphipod *Corophium volutator*. The collapse of the amphipod population results in habitat modifications that affect the entire Wadden Sea intertidal ecosystem (adapted from 37, 67, 68, 69, 83)

from the first intermediate host, the mud snail, increases with temperature (66). It is experimentally demonstrated that infection levels and parasite-induced mortality in amphipods also increase under warmer conditions. A simulation of host-parasite interactions in the Wadden Sea demonstrated that a 3.8ºC increase in temperature led to a parasite-induced collapse of the amphipod population (69, 83). Essentially, the development and transmission rates of the parasite, under conditions of warmer temperatures and extended growing seasons, are more sensitive than those of their invertebrate intermediate hosts. The parasite can overwhelm the host population under these circumstances, despite increments in the development of the snails and amphipods. Since, in this case, mortality is additive and not density-dependent, higher infection levels can lead to epizootics and cause fluctuations in the amphipod population (66).

Increases in temperature may have profound effects on other host-parasite systems that subsequently cause wholesale ecosystem modifications (68). The New Zealand cockle, *Austrovenus stutchburyi*, is the second intermediate host for the echinostome trematode, *Curtuteria australis*; a whelk being the first intermediate host and shorebirds the final host. Infected cockles are unable to burrow into the mud and suffer heavier predation than uninfected cockles. The emergence and transmission of the infective stages of the parasite are a direct function of temperature. Higher numbers of the parasite in the cockles result in the alteration of the surface topography and reduced bioturbation of the sediments. Biological consequences for the ecosystem include:

– increased predation on cockles by birds

– increased density of certain benthic species

– increased species richness

– more taxonomic and functional groups of benthic organisms (68, 83).

Since numerous trematodes circulate through intertidal ecosystems, it is suggested that warmer temperatures will, in general, lead to increased transmission efficiency and higher infection levels, with possible consequences for entire communities and ecosystems elsewhere (83). Basically, this is because cercarial production is more responsive to temperature than other physiological processes (82, 83). The authors caution that freshwater systems are also susceptible to these effects and that local extinctions will undoubtedly occur elsewhere.

Higher temperature may also increase the development and transmission of *P. invadens*, the parasite of green sea urchins discussed earlier (68). More frequent outbreaks of this parasite will restrict sea urchin populations and ensure the maintenance of kelp forests and their associated community of organisms. High temperature enhances developmental rates of the nematode *Echinomermella matsi*, which infects the same species of sea urchin in Norwegian

waters, causing castration and death. Like *P. invadens*, infection with *E. matsi* can reduce sea urchin populations, leading to the colonisation and establishment of kelp forests (reviewed in 61).

Essentially, these studies and others demonstrate that parasites can regulate host populations, influencing the composition, structure and function of biological communities (61). Such large-scale changes will have serious repercussions for the people and industries which are dependent on these ecosystems and their biological resources, such as fisheries, aquaculture and ecotourism. Moreover, coastal zone management will be challenged to adapt in the face of these environmental and socio-economic pressures (70).

Interactions with other stressors

Climate change will not happen in isolation. Its effects on organisms and ecosystems will be superimposed on the effects of other anthropogenic stressors, such as:

– habitat loss and fragmentation

– pollution

– introductions of alien species

– hypoxia

– altered hydrology (21, 99).

The damage caused by such environmental stressors may be increased by climate change (45, 101), either cumulatively or synergistically. Furthermore, these anthropogenic perturbations may confound the interpretation of the effects of climate change (29, 88). Stress of any kind, of course, increases the susceptibility of fish and other organisms to disease (79, 118).

The combination of direct and indirect effects may result in non-linear responses to climate change by organisms, populations and communities (29, 46, 47). The additional impact of other stressors in conjunction with climate change will further accentuate non-linear responses, perhaps with counter-intuitive results. Thus, disease will be affected by other stressors as well as global warming. These combined effects may act in concert or in opposition, in which case the transmission of parasites and pathogens could conceivably diminish in response to multiple stressors (47). A summary of studies that demonstrate enhanced negative effects on animals exposed to parasites and environmental stressors, which may be associated with climate change (59), is presented in Table III.

Warmer temperatures may reduce stream flow and water volume and, at the same time, lower oxygen below the thermocline of stratified waters in the deeper, cool waters of the hypolimnion (11, 12, 22). High temperatures in surface waters and greater hypoxia in bottom waters will confine fish to narrower bands of tolerable conditions, where they will seek refuge at higher densities. Such high densities may serve to increase disease transmission (21). The reduction in dissolved oxygen together with high temperatures could lead to increased pathogenicity of gill parasites, respiratory problems and even death (81).

Few studies have examined the interaction of parasites and stressors (62). Pollutants are expected to increase in aquatic ecosystems with global warming (45). Combined stressors may affect fish health so that the pathogenic effects of parasites are greater in polluted waters, compared to reference sites (62, 112).

With climate change, hypoxia has been predicted to increase in the hypolimnion of aquatic ecosystems (22). Under these conditions, the survival time of the European eel (*Anguilla anguilla*) is reduced when infected with the introduced nematode parasite *Anguillicola crassus*, when compared to uninfected eels (51, 65). It is suggested that this swimbladder parasite, by interfering with gas exchange during extensive breeding migrations, may be contributing to the decline in European eel stocks (109). Global warming may serve to hasten this decline if eels become exposed to low oxygen levels in the meantime. The ruff (*Gymnocephalus cernuus*), when infected with the nematode *Spinitectus gracilis* or the trematode *Acanthostomum* spp., experiences greater mortality at low oxygen concentrations than ruff with fewer parasites (87). Hypoxic conditions reduce the survival of the saltmarsh snail, *Cerithidea californica*, and the bivalve *Cerastoderma edule*, when infected with trematode metacercariae of the genus *Himasthla* (107, 119). The effects on the saltmarsh snail are context-dependent, in that their intensity varies with parasite species. Mortality is also higher in summer than in winter (107). Population crashes can occur, even under sub-lethal conditions, and the combined effects of infection and hypoxia may explain mass mortalities (119).

Thermal stress is amplified in molluscs and fish, in that their upper thermal limits are reduced, when infected with parasites (50, 56, 58, 111, 114, 115). Thermal tolerance in sunfish (*Lepomis* spp.) correlates with the total numbers of parasites in various body organs, including the liver and mesenteries. In addition, the overall parasite load in the mesenteries, heart, liver, spleen and small intestine is negatively associated with thermal tolerance (56). It is suggested that many parasites may not have an effect on host physiology under optimal conditions but, under certain environmental conditions or stress, they may exert a physiological influence. Thus, parasites may limit the

Table III
Host species at risk of increased stress from combined effects of environmental stress, climate change and parasitic infections

Host species	Parasite species	Parasite taxa	Environmental stress	Biological effect	Reference
Molluscs					
Biomphalaria glabrata	*Schistosoma mansoni*	Trematoda	Temperature	Lower thermal tolerance	50
Cerastoderma edule	*Himasthla elongata*	Trematoda	Hypoxia	Reduced survival	119
Cerithidea californica	*Himasthla* sp. *Parorchis* sp.	Trematoda	Hypoxia	Reduced survival	107
Hydrobia neglecta	Microphallids	Trematoda	Dessication	Reduced survival	37
Littorea littorea	*Cryptocotyle lingua*	Trematoda	Temperature	Lower thermal tolerance	58
Nassarius obsoleta	*Lepocreadium ovalis* *Zoogonus rubellus*	Trematoda	Temperature	Lower thermal tolerance	115
Nassarius reticulatus	Microphallids	Trematoda	Temperature	Lower thermal tolerance	111
Cnidaria					
Acropora muricata	Black band disease	Unknown	Nutrients Light penetration	Increased mortality	4
Gorgonia ventalina	*Aspergillus sydowii*	Fungus	Nutrients	Increased mortality	5
Teleostei					
Anguilla anguilla	*Anguillicola crassus*	Nematoda	Temperature	Altered physiological response	25
			Hypoxia	Lower thermal tolerance	51, 65
Gymnocephalus cernuus	*Spinitectus gracilis*	Nematoda	Hypoxia	Reduced survival	87
	Acanthostomum sp.	Trematoda			
Lepomis spp.	Total helminths	Varied	Temperature	Lower thermal tolerance	56
			Flow rate	Reduced swimming endurance	56
Notropis hudsonius	*Raphidascaris acus*	Nematoda	Pollution	Enhanced oxidative stress	62
	Apophallus brevis	Trematoda			
	Plagioporus sinitsini	Trematoda	Pollution	Increased melano-macrophages Reduced condition index	112
Oncorhynchus mykiss	*Argulus foliaceus*	Crustacea	Confinement	Altered immune function Impaired stress response	98
	Lepeophtheirus salmonis	Crustacea	Confinement	Altered immune function Impaired stress response	97
Oncorhynchus spp.	Protozoans	Protozoa	Temperature, low flow rate	Increased species richness	102
Salvelinus fontinalis	*Salmincola edwardsii*	Crustacea	Temperature	Lower thermal tolerance	114
Amphibia					
Ambystoma gracile	*Saprolegnia* sp.	Fungus	Nitrate	Increased mortality	92
Bufo boreas	*Saprolegnia ferax*	Fungus	Ultraviolet (UV)-B	Reduced hatching success	41
Hyla versicolor	*Telorchis* sp.	Trematoda	Dessication	Reduced survival and body mass, prolonged development at metamorphosis	40
Rana cascadae	*Saprolegnia ferax*	Fungus	UV-B	Reduced hatching success	41

ability of animals to maintain homeostasis at high environmental temperatures (56).

The mud snail, *H. neglecta*, experiences reduced survival if infected with microphallid trematode metacercariae when exposed to dessication (36). The survival and body mass of the grey treefrog (*Hyla versicolor*) were reduced when infected with metacercariae of the trematode *Telorchis* sp. in temporary ponds which were subject to dessication, but not in permanent ponds (41). In addition, time to metamorphosis was increased in infected frogs in the temporary ponds. Thus, the effects of the parasite are context-dependent. Moreover, these effects will have consequences at the population level by increasing the exposure of tadpoles to predators and reducing over-winter survival.

Eutrophication is predicted to increase in aquatic ecosystems (59), and this is expected to affect parasite distribution and abundance (see Table I), as well as host-parasite interactions. The severity of aspergillosis in sea fans increases under conditions of nutrient enrichment (1, 5). Similarly, nutrient enrichment increases the effects of BBD on staghorn coral, as does greater light penetration at high temperatures (4). Indeed, disease outbreaks in the oceans may be due to climatic changes to the NAO that result in an increased iron supply, altering micronutrients that normally limit the growth and virulence of opportunistic pathogens (30). Deformities in frogs caused by the trematode *Ribeiroia ondatrae* are linked to eutrophication, which subsequently results in an increased abundance of gastropod intermediate hosts and increased production of infective cercariae by those snails (38, 39).

The combined effects of nitrate and the water mould *Saprolegnia* sp. on three species of amphibians further illustrate the importance of context. A combination of *Saprolegnia* sp. and nitrate reduced survival in the northwestern salamander, *Ambystoma gracile*, whereas nitrate prevented mortality from *Saprolegnia* sp. in the red-legged frog, *Rana aurora*. No effect of combined exposure to nitrates and water mould was detected in a third species, the Pacific tree frog (*H. regilla*) (92).

These and other studies indicate that the negative effects of disease will not only increase with global warming, but be compounded by other stressors. Notably, some of these other stressors, such as hypolimnetic hypoxia and eutrophication, will themselves also be enhanced by climate change in various ecosystems throughout the world.

Human diseases

The effects of climate change on human diseases transmitted via aquatic infective stages, including schistosomiasis, are presented elsewhere in this volume (63). Climate change is likely to result in an increase of diarrhoeal diseases (10). The urban poor, elderly and children, traditional societies, subsistence farmers and coastal populations are most at risk. Diseases associated with weather patterns such as ENSO include cholera and cryptosporidiosis (13). Higher temperatures should result in an increase in some water-borne pathogens, such as the bacterium *Vibrio cholerae*, the causative agent of cholera (76). The abundance of bacterial pathogens transmitted to humans through the consumption of shellfish depends on temperature and salinity (10, 94). The consequences of pathogens expanding their range into new areas may be accentuated because new populations of hosts may have no history of exposure. In addition, drug-resistant pathogens have now evolved, which makes control more difficult (18).

A detailed analysis of existing data indicates that the interannual occurrence of cholera in Bangladesh, and the area surrounding the Bay of Bengal, is related to ENSO (75). Furthermore, seasonality in cholera outbreaks is linked to local water temperatures in Peru and India. Other potential climate-driven factors that influence the occurrence of cholera include droughts and floods, sea surface height, salinity and river discharge (75).

Higher rainfall and greater runoff have been associated with increases in cryptosporidiosis and giardiasis, caused by *Cryptosporidium* spp. and *Giardia* spp., respectively (77, 94). In fact, most drinking water outbreaks in the United States have been associated with extreme precipitation (94).

Naegleria fowleri is a pathogenic, thermophilic free-living amoeba that inhabits lakes, ponds and rivers worldwide (35, 110). It causes primary amoebic meningo-encephalitis, which is often fatal. The parasite can be found at ambient temperatures but its occurrence is related to elevated temperatures, being more common at temperatures above 25ºC, primarily 30ºC to 40ºC (35). Numerous studies support its association with artificially heated waters, such as thermal effluents (113). Under thermally altered conditions, the concentration of amoebae in water can be amplified 100-fold over that in ambient conditions (113). Disease is relatively rare, with 23 fatalities in the United States between 1995 and 2004, but seven in 2007 alone, which may be attributed to warming trends (http://en.wikipedia.org/wiki/Naegleria; accessed on 4 October 2007). Global warming will most likely lead to the expansion of this disease into temperate waters and more frequent infections in people.

Swimmer's itch, caused by the free-swimming infective stages of avian and mammalian schistosomes, has been noted to be increasing in Europe (15, 49). These increases are attributed to global warming (49), altered hydrology (59) and eutrophication (3, 43, 54). Extended growing seasons for snails (the first intermediate hosts) and residence time for waterfowl (definitive hosts) may lead to further increases in swimmer's itch (59). After the results reported by Poulin (82), demonstrating increased cercarial release with warmer temperatures in general, not only swimmer's itch but other, more serious diseases caused by trematodes, such as schistosomiasis, may also increase.

Others question whether climate change will increase disease. They suggest that the geographic distribution of disease will shift with global warming, but not result in any

net increase, based on recent modelling (46). However, these simulations were basically restricted to vector-borne diseases.

Extreme weather is also associated with increased disease risk. Droughts lead to poor hygiene and malnutrition, while floods result in contamination of drinking water (77). Cholera, cryptosporidiosis, typhoid fever and other diarrhoeal diseases increase after flooding in populations with poor sanitation infrastructure (10, 94). Water availability and access to clean water, rainfall, runoff and temperature are all related to the incidence of diarrhoeal diseases. Humans and ecosystems in coastal areas may experience a decrease in fresh water with climate change (45) that will enhance disease. In particular, access to fresh water will be problematic for developing countries with a large amount of coastal lowland area, semi-arid coastlines and coastal cities, not to mention low-lying small island states (70).

Conclusion

Outbreaks of numerous water-borne diseases in both humans and aquatic organisms are linked to climatic events, although it is often difficult to disentangle climatic from other anthropogenic effects. In some cases, these outbreaks occur in foundation or keystone species, with consequences throughout whole ecosystems. There is much evidence to suggest that parasite and disease transmission, and possibly virulence, will increase with global warming. However, the effects of climate change will be superimposed on a multitude of other anthropogenic environmental changes. Climate change itself may exacerbate these anthropogenic effects. Moreover, parasitism and disease may act synergistically with these anthropogenic stressors to further increase the detrimental effects of global warming on animal and human populations, with debilitating social and economic ramifications.

■

Impact du changement climatique sur les maladies parasitaires et infectieuses affectant les animaux aquatiques

D.J. Marcogliese

Résumé

Le changement climatique aura certainement un impact important sur les maladies et les parasites présents dans les écosystèmes marins et d'eau douce, avec des conséquences sanitaires et socioéconomiques non négligeables pour l'homme. Le réchauffement global affectera directement la distribution des parasites et des agents pathogènes dans ces milieux ; il aura également des conséquences indirectes, liées au spectre d'hôte et à l'abondance des hôtes. À ce jour, de nombreux foyers affectant notamment les organismes marins ont été associés à des évènements climatiques tels que le phénomène ENSO *(El Niño Southern Oscillation)*. En général, le taux de transmission des parasites et des agents pathogènes devrait augmenter en même temps que les températures. Des études indiquent que certains agents pathogènes et parasites deviendront plus virulents en cas de réchauffement global. Aux effets du changement climatique sur les parasites et les agents pathogènes viendront s'ajouter ceux d'autres facteurs anthropiques de stress au sein des écosystèmes, tels que les contaminants, les destructions d'habitats et les introductions de nouvelles espèces. Ces facteurs de stress combinés peuvent agir de manière cumulée ou synergique en exacerbant leurs effets négatifs sur les organismes des hôtes et leurs populations. L'impact du climat sur les parasites et les maladies affectant un certain nombre d'espèces importantes risque ensuite de se propager tout au long de la chaîne alimentaire, avec pour conséquence une altération des écosystèmes entiers.

Mots-clés

Agent pathogène – Animal aquatique – Changement climatique – Eau douce – Écosystème marin – Maladie – Parasite – Température.

■

¿Cómo afectará el cambio climático a los parásitos y las enfermedades infecciosas de los animales acuáticos?

D.J. Marcogliese

Resumen

Según todos los pronósticos, el cambio climático tendrá importantes efectos sobre el parasitismo y las enfermedades ligadas a ecosistemas marinos y de agua dulce, lo que no dejará de influir en la salud y la socioeconomía de las poblaciones humanas. La distribución de los parásitos y patógenos se verá afectada por el calentamiento planetario no sólo directamente, sino también indirectamente por los efectos de éste sobre la distribución y abundancia de los hospedadores. Hasta la fecha numerosos brotes de enfermedades, sobre todo en organismos marinos, han venido asociados a fenómenos climáticos como la Oscilación del Sur El Niño. En líneas generales se supone que los índices de transmisión de parásitos y patógenos aumentarán con la subida de temperaturas. Los datos disponibles parecen indicar que ciertos patógenos y parásitos cobrarán mayor virulencia con el calentamiento planetario. En los ecosistemas, la influencia del cambio climático en parásitos y patógenos se sumará a los efectos de otros factores de estrés de origen antrópico, tales como los contaminantes, la pérdida de hábitat o la introducción de especies foráneas. Esta combinación puede tener efectos acumulativos o sinérgicos y exacerbar las repercusiones negativas para los hospedadores y sus poblaciones. La influencia del clima sobre los parásitos y las enfermedades de especies clave puede surtir efectos en cascada a través de las redes tróficas, e influir así en ecosistemas enteros.

Palabras clave

Agua dulce – Animales acuáticos – Cambio climático – Enfermedad – Marino – Parásito – Patógeno – Temperatura.

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