Transboundary and Emerging Diseases

ORIGINAL ARTICLE

Assessing the Impact of Climate Change on Disease Emergence in Freshwater Fish in the United Kingdom

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Summary

A risk framework has been developed to examine the influence of climate change on disease emergence in the United Kingdom. The fish immune response and the replication of pathogens are often correlated with water temperature, which manifest as temperature ranges for infection and clinical diseases. These data are reviewed for the major endemic and exotic disease threats to freshwater fish. Increasing water temperatures will shift the balance in favour of either the host or pathogen, changing the frequency and distribution of disease. A number of endemic diseases of salmonids (e.g. enteric red mouth, furunculosis, proliferative kidney disease and white spot) will become more prevalent and difficult to control as water temperatures increase. Outbreaks of koi herpesvirus in carp fisheries are likely to occur over a longer period each summer. Climate change also alters the threat level associated with exotic pathogens. The risk of viral haemorrhagic septicaemia (VHSV), infectious haematopoietic necrosis virus (IHNV) and spring viraemia of carp virus (SVCV) declines as infection generally only establishes when water temperatures are less than 14°C for VHSV and IHNV and 17°C for SCVC. The risk of establishment of other exotic pathogens (epizootic haematopoietic necrosis and epizootic ulcerative syndrome) increases. The spread of *Lactococcus garvieae* northwards in Europe is likely to continue, and thus is more likely to be both introduced and become established. Measures to reduce the threat of exotic pathogens need to be revised to account for the changing exotic diseases threat. Increasing water temperatures and the negative effects of extreme weather events (e.g. storms) are likely to alter the freshwater environment adversely for both wild and farmed salmonid populations, increasing their susceptibility to disease and the likelihood of disease emergence. For wild populations, surveillance and risk mitigation need to be focused on locations where disease emergence, as a result of climate change, is most likely.

Introduction

Climate change is arguably the greatest environmental challenge facing the world today. Current climate patterns are changing and mean temperatures and frequency of extreme weather events are likely to increase. According to published climate change scenarios (Hulme et al., 2002), the mean annual air temperature across the United Kingdom may rise between 0.5 and 1.5°C by 2020 and between 2 and 3.5°C by 2080, depending on the future levels of greenhouse gas emissions. Rainfall patterns will also change: winters are likely to become wetter and summers drier (Hulme et al., 2002). In general, the most affected regions in United Kingdom will be the south and the east, experiencing greater changes than the north and west (Hulme et al., 2002).

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Disease emergence has become a focus for academic research in both human (Krause, 1998) and animal health (Daszak et al., 2001b), including aquatic animals (Harvell et al., 1999). A number of categories of emerging diseases can be defined: diseases which are increasing in incidence or geographical range (e.g. transboundary diseases), causing increased severity or a new pathology, recently moved into a new host, newly discovered or caused by a newly evolved pathogen (Brown, 2000; Daszak et al., 2000). There is good evidence that the rate of disease emergence in human and animal populations has increased substantially in recent decades, which has been in large part attributed to human intervention in the environment (Daszak et al., 2001a) and international trade in live animals (Wilson, 1995; Hedrick, 1996). Considerable evidence exists that climate change may influence disease emergence.

A number of studies have investigated the potential impact of climate change on disease emergence in humans (Patz et al., 2005) and livestock (Baylis and Githeko, 2006; Gale et al., 2009). Vector-borne diseases (e.g. bluetongue virus) have been a focus of attention (Rogers and Packer, 1993; Ogden et al., 2008). In the marine environment, over the last 30 years, an increase in disease in six of nine studied marine taxa has been reported (Ward and Lafferty, 2004); it was concluded that increased global ocean temperatures was likely to have been a factor for disease emergence in turtles and coral. Bally and Garrabou (2007) reported higher sea temperatures appearing to favour bacteria over their marine invertebrate hosts resulting in higher levels of disease. A number of species of abalone in North America have declined because of a combination of increased water temperature and infectious diseases. In particular, high seawater temperatures increased transmission and severity of withering syndrome in red abalone (Braid et al., 2005; Vilchis et al., 2005). A protistan parasite, *Perkinsus marinus*, of oysters (*Crassostrea virginica*) in North America has extended its range northwards as seawater temperatures have risen (Cook et al., 1998). It can be concluded that climate-mediated physiological stress may compromise resistance and increase susceptibility to disease and thus influence disease emergence in marine ecosystems (Harvell et al., 1999; Altekruse et al., 2000; Lafferty et al., 2004; Alborali, 2006; Gubbins, 2006). It is likely that similar conclusions may apply to freshwater ecosystems; however, the impacts of climate change on freshwater aquatic animal diseases have been relatively little studied.

Fish are poikilothermic, thus their physiology, including the immune response, is directly affected by the ambient temperature (Bowden et al., 2007). Similarly, pathogens also have optimal temperature ranges for replication. For many fish diseases (e.g. furunculosis, koi herpesvirus, spring viraemia of carp), temperature is a key determinant of whether infection results in disease and mortality or immunity and recovery. It is, therefore, highly likely that climate change may affect fish more severely and earlier than homeothermic animals. Increasing freshwater temperature may favour the establishment of introduced non-native aquatic animal species and exotic pathogens, originating in regions with higher ambient temperatures. Extreme weather events, together with higher temperatures and other environmental and anthropogenic factors, are likely to degrade fish habitat and adversely affect water quality (Brander, 2007). In particular, in the United Kingdom, climate change is highly likely to decrease the suitability of the aquatic environment for salmonids and, in particular, Atlantic salmon (*Salmo salar)* (Graham and Harrod, 2009; Johnson et al., 2009). These changes are likely to increase their susceptibility to disease and thus the likelihood of disease emergence.

In this paper, we adapt a risk assessment framework originally developed for terrestrial livestock diseases in Great Britain (Gale et al., 2009) to qualitatively examine how climate change may influence the rate and type of disease emergence in freshwater aquatic animals in the United Kingdom and to identify which species are most likely to be detrimentally affected.

Materials and Methods

A qualitative risk assessment framework has been used to assess the impact of climate change on the emergence of freshwater fish diseases (Table 1). The framework builds upon previously published work (Gale et al., 2009) for terrestrial livestock, whose starting point was the guidelines for import risk analysis published by the World Organisation for Animal Health (Office International des Epizooties, O.I.E.) (O.I.E., 2009). The risk framework seeks to systematically identify the main factors which may influence all types of disease emergence (Table 2).

Qualitative risk assessment framework

Module 1: routes of pathogen introduction to the United Kingdom

Higher water temperatures may lead to increased demand to culture new aquatic animals species in the United Kingdom and increase their chances of survival (and that of their pathogens and parasites) in the wild. Similarly, exotic species adapted to higher water temperatures than those historically occurring in The United Kingdom, introduced as ornamental animals, are more likely to survive and become widely distributed (if released into

Table 1. Qualitative risk assessment framework for disease emergence in fish

Module description	Aim
1. Routes of pathogen introduction into England and Wales	Routes of introduction, assess whether climate change may promote new routes or increase risk of release in existing routes
2. Mutation, replication, virulence and abundance of the pathogen	Potential of pathogen to evolve to exploit new opportunities created by climate change; impact of temperature on rate of replication and virulence.
3. Host susceptibility	Host reservoirs and influence of climate change on their susceptibility
4. Host–pathogen interaction	Influence of climate change on the interaction between host and pathogen (resulting in changes in disease expression)
5. Vectors, intermediate and alternative hosts	Effect of climate change on vectors, intermediate and alternative hosts
6. Animal-to-animal contact and movement	Effect of climate change on animal to animal contact (because of changes in movement and mixing of animals)
7. Environmental routes	Effect of climate change on environmental routes of pathogen spread (e.g. fomites, sediment, water)

Table 2. Association between risk assessment module and category of emerging disease

	Emerging disease category				
Module description	New disease	New host	New location/ increased distribution	Increased prevalence/ incidence	Increase severity/new pathology
1. Routes of pathogen introduction into England and Wales		X	X		
2. Mutation, replication, virulence and abundance of the pathogen	X	X			
3. Host susceptibility			X	X	X
4. Host–pathogen interaction			X	X	X
5. Vectors, intermediate and alternative hosts			X	X	
6. Animal-to-animal contact and movement			X	X	
7. Environmental routes			X		

the wild). This will facilitate the introduction of new pathogens and parasites to new naïve host, host-switching and thus disease emergence (Morgan et al., 2004; Brooker et al., 2007). The international trade in ornamental aquatic animals has resulted in the transboundary movements of pathogens. Rosette agent may have been introduced to the United Kingdom with the introduction of top mouth gudgeon (*Pseudorasbora parva*) (Gozlan et al., 2005, 2006). Top mouth gudgeon, now established in the wild in United Kingdom, were originally introduced for the ornamental trade and may also have been accidentally introduced with imports of other ornamental fish species (Domaniewski and Wheeler, 1996). In the United Kingdom, a number of fish macroparasites have been introduced with non-native ornamental fish species (Kennedy, 1993), some of which are considered serious threats (i.e. have been classified as category II[1] parasites). International movement of ornamental fish is believed to be the main reason for the rapid global spread of koi herpesvirus (KHV), a serious transboundary disease, (Gilad et al., 2003) and the spread of emerging iridoviruses (Go and Whittington, 2006).

There is evidence that as seawater temperatures rises, the geographical range of some marine fish species change as they seek to remain at the optimal temperature to maximize growth and thus migrate northwards (Perry et al., 2005). This may be an important pathway for pathogen spread in the marine environment and ultimately to freshwater. Migrations of freshwater species are restricted by natural barriers, and in the case of Great Britain a seawater barrier. In this module disease emergence occurs mainly through expansion of the pathogen's geographical range (i.e. transboundary diseases).

Module 2: mutation, replication, abundance and virulence of the pathogen

The ability of a pathogen to mutate will enable it to respond rapidly to novel opportunities created by climate change, such as the establishment of new host species (Gale et al., 2009). Pathogen evolution lowers the species barrier so new strains are more likely to extend their host range (Kuiken et al., 2006). Negative strand and

[1]The Environment Agency defines category II parasites as those that are not native to fish in the United Kingdom but which have established and are considered as a potential threat to native fish stocks

segmented genome RNA viruses, such as the family Orthomyxoviridae, have higher mutation rates compared with other viruses (Holland et al., 1982) and thus are more likely to emerge as new diseases or in new hosts. Infectious salmon anaemia virus (ISAV), an orthomyxovirus, appears to have evolved from a wild avirulent ancestor on at least two occasions (Cunningham and Snow, 2000; Nylund et al., 2003).

At higher water temperatures, the generation time of bacteria, fungi and parasites with direct lifecycles is shorter (Gubbins, 2006). This may increase the size of pathogen populations (Harvell et al., 2002) and therefore challenge levels and the likelihood that infection results in disease and mortality. *Argulus coregoni*, a crustacean parasite of salmonids, usually has one generational cycle per year but this may double at higher temperatures (Hakalahti et al., 2006). Changes in other environmental variables, driven by climate change, will also affect parasite abundance. Climate change is likely to result in increased acidification in headwater streams, which may result in a decrease in parasite diversity and a loss of trematodes (Marcogliese, 2001). Meanwhile, lakes are likely to become less acid, and thus parasite diversity may increase (Marcogliese, 2001). Lower water levels and flow rates will increase the abundance of free-living infective parasite life stages (Johnson et al., 2009). It is not possible to generalize about the impact of water temperature on viral replication as each has its own optimal temperature range; for example, IHNV grows optimally *in vitro* at 15°C (Mulcahy et al., 1984) and spring viraemia of carp virus (SVCV) grows optimally *in vitro* at 20–22°C (Fijan, 1998).

Module 3: host susceptibility

Generally, poikilotherms have an optimum temperature range for reproduction and growth, which differs depending on species and adaptation to the local environment. The fish immune system is optimal at normal summer temperatures for each species (Manning and Nakanishi, 1996). Water temperatures above or below the physiological optimum may cause stress, increase susceptibility to infection and the likelihood of disease and mortality. Salmonids are stenothermal (tolerant to a narrow thermal range), while cyprinids are eurythermal (tolerant to a wide thermal range). Temperatures above 13–16°C stress salmonid species, leading to suboptimal growth rate, decreased swimming performance, higher disease risk and impaired smoltification in Atlantic salmon (Bear et al., 2007; Sutton et al., 2007). Thus, while warm-water fish, such as carp (*Cyprinus carpio*), existing at the northern limit of their geographical range, are likely to be favoured by increased water temperatures in the United Kingdom, cold-water fish, e.g. salmonids, will suffer (Shuter et al., 1998). Stressed populations are more susceptible to opportunistic pathogens, such as *Aeromonas* sp. and *Flavobacterium* sp. (Alborali, 2006). During summer 2003 in the estuary of the river Tyne, migrating adult Atlantic salmon, stressed by high temperatures and low levels of dissolved oxygen, became susceptible to opportunistic bacterial infections (*Vibrio anguillarum*) and died in substantial numbers (St-Hilaire et al., 2005). The rate of temperature increase is also important; sudden, sharp increases (because of heat waves) will adversely affect the fish's immune system, salmonids being particularly affected.

Salmonids are more sensitive to habitat quality than many non-salmonid species. The range of water quality tolerated by fish is related to the species, age class and acclimation temperature in a specific environment (Alborali, 2006). Water temperature depends on air temperature, water depth and flow rate (for running water). Higher water temperatures and more extreme weather events (e.g. droughts, storms and flooding) are predicted (Hulme et al., 2002) and will decrease water quality. Higher temperatures reduce dissolved oxygen levels and increase the rate of primary production and organic matter decomposition. Nutrient run-off will increase the level of eutrophication and frequency of algal blooms. Decreased river flow rates (e.g. during periods of low rainfall) might reduce water quality as contaminants and dissolved solids are less diluted (Johnson et al., 2009). High flow rates (following storms) might also increase leaching of potential contaminants from the soil, increasing the amount of suspended solids and the distribution of contaminants through freshwater systems. The toxicity of common pollutants (e.g. organophosphates and heavy metals) to fish generally increases at higher temperatures (Murty, 1986). High rainfalls in short periods is known to overload the capacity of sewage treatment plants and untreated wastewater may be released into rivers, leading to sudden falls in dissolved oxygen and large-scale fish kills.

Module 4: host–pathogen interaction

Temperature thresholds for infection and disease expression depend on the influence of temperature on the pathogen and the host, as both have optimal temperature ranges. Thus, the threat from endemic and exotic diseases varies with water temperature (Table 3). For diseases with thresholds below which infection and/or disease does not occur, rises in water temperature will increase the period of the year during which infection and disease may occur. For example, proliferative kidney disease (PKD) outbreaks in rainbow trout (*Oncorhynchus mykiss*) normally occur at water temperatures over 15°C (Hedrick et al., 1993), at lower temperatures (<10°C) the fish may become infected but the disease does not express clinically (Clifton-Hadley

Table 3. Optimal temperatures for important endemic and exotic* disease threats to UK freshwater fish

Pathogen	Optimal temperature range for disease (°C)	Comments	References
VIRUSES			
Viral haemorrhagic septicaemia virus	**<15°C**	**Most severe outbreaks occur between 9–12°C. Temperatures above 15°C are inhibitory to virus replication and infection is rarely observed.**	**Vestergaerd and Joergensen, 1982; Wolf, 1988**
Infectious haematopoietic necrosis virus	**<15°C**	**Linear relationship between temperature and time to mortality. At <10°C chronic disease and lower mortalities, at 10–12°C highest mortalities and at >15°C few mortalities.**	**Amend, 1975**
Salmonid alphavirus (sleeping disease in trout)	<11°C	Outbreaks occur up to 10°C, maximum viral replication at 10°C, little replication at 14°C	Villoing et al., 2000
Spring viraemia of carp virus	10–17°C	Over 17°C infected fish develop antibodies. Fry can be clinically ill at temperatures as high as 23°C.	Ahne et al., 2002
Koi herpesvirus	16–28°C	No morbidity observed below 13°C and over 30°C.	Hedrick et al., 2000
Epizootic haematopoietic necrosis virus	**>11°C**	**Longer incubation periods observed at lower temperatures.**	**Whittington and Reddacliff, 1995**
BACTERIA			
Renibacterium salmoninarium (bacterial kidney disease)	>13°C	Most severe outbreaks at temperatures between 15–18°C.	Jones et al., 2007
Lactococcus garvieae	**>16°C**	**Most acute outbreaks appear over 18°C, although acute; outbreaks have been described with water temperature of 14-°C.**	**Vendrell et al., 2006**
Warm water strawberry disease	>16°C	Unknown aetiology. Not observed in the winter in the United Kingdom.	St-Hilaire and Jeffery, 2004
Flavobacterium columnare	>15°C	The bacterium adheres more strongly to gill tissue at higher temperature	Decostere et al., 1999
Yersinia ruckeri (enteric redmouth)	>8°C	Peak of infection seen at 15–18°C	Horne and Barnes, 1999
Aeromomas salmonicida (furunculosis)	>10°C	Outbreaks in salmonids hatcheries occur in spring when water temperatures are rising rapidly. Disease in juveniles can occur at lower temperatures (around 4°C).	Malnar et al., 1988
Cold water strawberry disease	<10°C	Unknown aetiology. Not observed in the summer in the United Kingdom	Verner-Jeffreys et al., 2008
Flavobacterium psychrophilum (rainbow trout fry syndrome)	<10°C	Rainbow trout fry syndrome (RTFS) generally observed in the winter in the United Kingdom	Faruk et al., 2002
PARASITES			
Tetracapsuloides. byrosalmonae (proliferative kidney disease)	>15°C	At lower temperatures fish infected but not clinically ill. Increasingly water temperature has favoured the alternative, bryozoan host.	Wahli et al., 2002; Sterud et al., 2007; Hedrick et al., 1993; Gay et al., 2001
Ichthyophthirius multifiliis (white spot)	>15°C	Salmonids may be affected at temperatures as low as 4°C, but most outbreaks occur at high temperatures (>15°C) (higher temperatures lead to shorter life-cycle).	Buchmann and Bresciani, 1997
OTHERS			
Aphanomyces invadans (epizootic ulcerative syndrome)	**18–22°C**	**In Asia, Australia and Africa, clinical disease does not occur when temperature increases above 22°C.**	**Pathiratne and Jayasinghe, 2001**

*Pathogens exotic to the United Kingdom in bold.

et al., 1986; Gay et al., 2001). Endemically infected farms manage the disease by introducing naïve fish when the water temperature is below <10°C and the fish sufficiently immunocompetent (age-related) to mount an immune response (Ferguson, 1981; de Kinkelin and Loriot, 2001); this becomes increasing difficult as water temperatures rise year on year. At higher water temperatures, diseases normally occurring only in the summer, such as KHV

which occurs above 15°C and below 29°C (Hedrick et al., 2005), may have longer periods of transmission, starting earlier and ending later in the year (Gubbins, 2006). As water temperature increases, parasites with a seasonal occurrence may reproduce and infect fish all year-round (Marcogliese, 2001). White spot (*Ichthyophthirius multifiliis*) is one of the most important diseases affecting farmed rainbow trout in the United Kingdom. The development of the parasite is temperature dependent and most outbreaks occur when the water temperature exceeds 16°C (Buchmann and Bresciani, 1997; Aihua and Buchmann, 2001).

Outbreaks of some bacterial infections of salmonids are related to higher water temperatures. Bacterial kidney disease (BKD) outbreaks are normally seen at 13–18°C (Mitchum et al., 1979; Jones et al., 2007). Temperature is key determinant of the occurrence of furunculosis (one of the most important diseases of farmed salmonids, caused by *Aeromonas salmonicida*); outbreaks generally occurring when water temperatures exceed 10°C (Malnar et al., 1988). Enteric red mouth infection in farmed salmonids (caused by *Yersinia ruckeri*) has a seasonal occurrence, occurring as water temperature rise in spring (Lesel et al., 1983). The bacterium associated with columnaris disease in fish, *Flavobacterium columnare*, adheres more strongly to gill tissue at higher temperatures (Decostere et al., 1999). However, other bacterial diseases, such as rainbow trout fry syndrome (RTFS) (caused by *Flavobacterium psychrophilum*), normally observed below 10°C (Faruk et al., 2002), are favoured by cold water temperatures. Among the diseases of unknown aetiology, warm water strawberry disease is only observed in the spring and summer months in the United Kingdom, when water temperatures are above 10°C (St-Hilaire and Jeffery, 2004). Conversely, cold water strawberry disease, also known as red mark syndrome, is observed at water temperatures less than 15°C (Verner-Jeffreys et al., 2008).

The likelihood of establishment of transboundary diseases exotic to the United Kingdom, with temperature thresholds below which infection does not occur, e.g. epizootic haematopoietic necrosis virus (EHNV), *Lactococcus garvieae* and *Aphanomyces invadans*, increases as water temperatures rise. Epizootic haematopoietic necrosis (EHN) epizootics in Australia occur in spring and summer and clinical disease has not been reported at water temperatures below 11–12°C (Whittington and Reddacliff, 1995). Outbreaks of *L. garvieae* occur when water temperatures are over 16°C (Vendrell et al., 2006). The risk of establishment of exotic parasites originating from warmer environments will increase as water temperatures rise.

Conversely, for other viruses, which have thresholds above which disease does not occur (Table 3), the period of the year during which establishment will occur is reduced as water temperatures rise. Examples include IHNV and VHSV (exotic to the UK), spring viraemia of carp virus (SVCV) (occurs only sporadically due to imported infected fish) and salmonid alphavirus (limited distribution in rainbow trout farms in the UK) (Villoing et al., 2000). Adult common carp respond to infection with SVCV by producing protective humoral antibodies at temperatures greater than 17°C (Ahne et al., 2002), above which temperature outbreaks are generally not observed (O.I.E., 2009). In addition, higher water temperatures will allow carp to feed better during winter and thus, be in better condition in spring, the time of the year when most SVC outbreaks occur.

Module 5: vectors, intermediate and alternative hosts

Parasites with indirect life cycles (e.g. digenean trematodes, nematodes) may be particularly sensitive to climate change if the abundance and/or distribution of their intermediate hosts are influenced by water temperature. Outcomes for these diseases are more complex and difficult to predict (Gubbins, 2006). The intermediate hosts of *Tetracapsuloides bryosalmonae* (the myxozoan parasite causing PKD) are freshwater bryozoans. Higher temperatures have been reported to increase the proliferation of both *T. bryosalmonae* and freshwater bryozoans (Tops et al., 2006), leading to an increased level of challenge to the fish in the same environment. Some studies have suggested that the parasite *Argulus foliaceus* may act as mechanical vector of SVCV (Ahne et al., 2002). The effect of climate warming will favour *Argulus* populations (Hakalahti et al., 2006), which may increase transmission of SVC.

Module 6: direct animal to animal transmission

Decreased water quality may reduce macroinvertebrate populations, an important source of food for may fish species. The lack of food, together with the fish habitat degradation (poor water quality and hydrodynamic changes) may reduce salmonids population densities, in particular, and therefore decrease the pathogen transmission rate. Carp will be less affected. By contrast, low river flows in summer (which will occur during droughts which are predicted to increase) hinder the upstream migration of Atlantic salmon and sea trout (*Salmo trutta*) returning to spawn. Consequently, fish congregate in slow flowing water; conditions conducive for pathogen exchange, challenge and disease expression (St-Hilaire et al., 2005). In still stratified water bodies, higher temperatures will decrease the oxygen content of the deeper cool water of the hypolimnion[2] (Coutant, 1987). Higher temperatures in the upper layers and hypoxia in the lower

[2]The dense, bottom layer of water in a thermally stratified lake which is the coldest layer in summer and the warmest layer during winter.

layers of lakes may lead to higher fish densities in the narrowed band where conditions are optimal, resulting in increased rates of disease transmission (Marcogliese, 2008). In farming conditions, poor water quality, in particular low dissolved oxygen, may force trout farmers to decrease stocking density, which may decrease pathogen transmission rates.

Module 7: environmental routes of disease transmission
Horizontally transmitted pathogens are particularly sensitive to climate change as they have to survive outside the host, if only for a short period, before infecting a new host. However, little data are available on survival of pathogens outside the host at different temperatures. The survival of VHSV in seawater has been shown to be negatively correlated with water temperature increases above 4°C (Parry and Dixon, 1997). Similarly, IHNV [data reviewed by EFSA (EFSA, 2007)] and sleeping disease virus (Graham et al., 2007) survive longer in the environment at lower water temperatures. Increased flooding (Hulme et al., 2002) may increase the dispersal of pathogens (as well as contaminants). Droughts, instead, may affect farming practices. Low flows or stricter limits on water abstraction (because of lower rainfall and increased domestic demand) may result in an increased use of recirculation systems in rainbow trout farms, which increase pathogen concentration (if water treatment does not inactivate infectious agents).

Discussion

In this paper, we have adapted a relatively simple and generic risk framework for terrestrial animals (Gale et al., 2009) to diseases of freshwater finfish. The framework provides a structured, systematic and transparent approach to screen fish pathogens and parasites and thus identify those most likely to emerge as a result of climate change. Disease emergence in the United Kingdom was examined, but the approach could be applied in any location. The scope of this paper was limited to considering the likely impact of climate change on the distribution and frequency of disease; consequences (i.e. costs of aquaculture or ecological implications of declines in wild populations) are not considered.

Disease is the outcome of the interaction between the host, the pathogen and the environment (Martin et al., 1987). Ambient water temperature influences all three elements of the disease triad. Most importantly, interactions between immune response and pathogen replication are temperature dependent. The outcome of infection (disease, recovery or mortality) will be determined by the rate of pathogen replication, the immune response and the resulting level of pathogen challenge (Jun et al., 2009). The pathogen and the host will have an optimal temperature range for replication and immune response, respectively. Increasing water temperatures will differentially benefit either pathogen or host, shifting the outcome towards disease and mortality or immunity and recovery. Thus, host–pathogen interaction is most critical to the risk assessment and needs to be considered for each host-pathogen combination.

A number of endemic diseases of farmed salmonids (e.g. enteric red mouth, BKD, PKD, white spot) are likely to increase in frequency. Second, increases in water temperature and other changes in environmental water quality will adversely affect salmonids and increase their susceptibility to disease, more severely than cyprinids, increasing the risk of disease emergence in wild and farmed populations. There is already evidence from Switzerland (Wahli et al., 2002) and Norway (Sterud et al., 2007) that increased water temperature underlies the increased prevalence and impact of PKD in wild salmonids populations. Pathology associated with *T. byrosalmonae* infection in wild salmonids in the United Kingdom is well recognized (Peeler et al., 2008), and increasing levels of challenge may result in PKD downwardly regulating the wild population levels. Of the diseases affecting cyprinids, KHV is likely to become more widely spread and clinical outbreaks will be observed for over a greater period of the year. Generally, cyprinids will benefit from increasing water temperatures and may as a result be less susceptible to new and emerging diseases; specifically, the threat of SVC will decrease.

The major transboundary diseases which are exotic to the United Kingdom have well-defined temperature thresholds for establishment and while increased water temperature lowers the threat of VHSV and IHNV, the risk of *L. garvieae*, EHNV and epizootic ulcerative syndrome (EUS) will increase (Table 3). *L. garvieae*, a serious cause of mortality and morbidity in European rainbow trout production (Eyngor et al., 2004) is probably the exotic disease whose risk increases most as a result of climate change. As freshwater temperatures continue to rise, its northwards advance across Europe and increased prevalence will inevitably continue (Alborali, 2006), and consequently the likelihood of re-introduction to the United Kingdom) will increase. The rate of replication of the bacteria increases with rising water temperatures which raises the level of challenge and thus likelihood of establishment. The period of the year when introduction is likely to lead to establishment will increase as water temperatures rise and remain above the permissive temperature (15°C) for longer. Climate change might adversely affect host susceptibility due to increased water temperature, and indirectly through decreased water quality. The bacteria are resistant to high temperatures, and increase

in water temperature is unlikely to alter its viability in the environment. The occurrence of the disease in southern England at a single location (Bark and McGregor, 2001) demonstrates the real risk of introduction and establishment.

The risk framework presented in this paper is an effective method to initially screen diseases whose risk is likely to increase with climate change. More in-depth prediction of the impact of climate change on disease emergence is problematic because of the large number of interacting factors operating on the host, pathogen and environment. Interactions between factors may be complex and non-additive and either increase or decrease disease incidence (Lafferty et al., 2004). More sophisticated modelling approaches may be used to examine these interactions and in particular to quantify the potential population regulatory effects of higher levels of disease because of climate change. This is probably best illustrated by *Gyrodactylus salaris*, the most important exotic disease threat to wild Atlantic salmon populations in the United Kingdom. Higher water temperatures will increase the parasite's reproductive rate (Appleby and Mo, 1997); but may also reduce the period juveniles (parr) may spend in freshwater, thereby reducing the opportunity for transmission between age classes (Bakke et al., 2007). Mathematical modelling of inter-relationships between changes in environmental parameters and their impact on hosts and pathogens might help to identify plausible outcomes of increased water temperatures on disease epidemiology.

Some impacts of climate change are likely to be spatially heterogeneous. Geographical information systems and remote sensing can be used to assess how the distribution and impact of disease may vary with location. Rogers and Randolph (2006) highlighted the need to adopt a landscape-epidemiological approach in their paper on climate change and vector-borne diseases. This approach has been applied mainly to predict the northern spread of some vector-borne diseases of livestock and humans in Europe (Cook, 1992; Ogden et al., 2005; Patz et al., 2005). These approaches can be applied productively to the study of aquatic animal disease pathogens. They can be employed to predict the northwards migration of marine fish species and the change in the spatial pattern of disease risk with increasing water temperatures (and so identify especially vulnerable populations). For example, mapping the seasonal pattern of water temperature can identify which rivers, and which parts of the river, are at highest risk from the establishment of EHNV if introduced (Peeler et al., 2009). The occurrence and impact of extreme weather events (i.e. storms and droughts) will vary considerably between locations and high risk locations could also be identified.

Intensive and semi-intensive livestock populations can adapt to mitigate environmental changes (i.e. through housing) (Gale et al., 2009). By analogy, land-based recirculation systems can be viewed as largely impervious to the effects of climate change. However, these systems account for only a very small fraction of aquaculture in the United Kingdom. In the long term, aquaculture will respond to climate change by farming species or strains adapted to higher temperatures. The freshwater ecosystem in the United Kingdom is highly managed. Minimizing discharges (e.g. by improving drainage and preventing the release of untreated sewage) and other measures can, to some extent, mitigate the negative impacts of climate change on water quality. Interventions need to take into account the additional stress placed particularly on salmonid species because of changes brought about through increasing water temperature and extreme weather events. For example, provision needs to be made to release sufficient water into rivers (e.g. from reservoirs) during droughts to mimic normal conditions and allow continuous upstream migration of salmonids, thus avoiding crowding, stress and increased pathogen transmission.

Conclusions

Climate change is most likely to influence disease emergence by increasing both the occurrence of some diseases in new locations and their frequency in the existing geographical range (Table 3). Climate change is likely to strengthen drivers for the introduction of exotic fish species and also change the aquatic environment to increase the likelihood of the establishment and spread of exotic parasites and pathogens. National biosecurity may need to be periodically reviewed in the light of the shift in exotic disease threats. Similarly, many endemic diseases have well-established temperature thresholds (for infection and disease expression) and increases in water temperature will lead to increases in both their geographical distribution and prevalence. It is, therefore, necessary to consider the impact of increasing water temperature for each combination of pathogen and host. Broadly, a number of important diseases of farmed salmonids will become more prevalent and changes in farming practices and disease-control measures will be needed. Similarly, wild salmonids populations are likely to experience higher levels of disease. Our very limited capacity to influence disease in wild, compared with farmed populations, reduces options for mitigation. It is also apparent that the impact of climate change, especially when mediated through an increase in extreme weather events, will not be spatially homogenous. Geo-referenced climate data can be used to identify areas of high risk for disease emergence, where surveillance and risk mitigation can be focused. The

potential for mathematical modelling to account for the interaction between host, pathogen and environmental determinants of disease, and thus provide prediction about the impact of climate change on aquatic animal diseases, needs to be explored.

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