



Increasing water temperature and disease risks in aquatic systems: Climate change increases the risk of some, but not all, diseases

Anssi Karvonen^{a,*}, Päivi Rintamäki^b, Jukka Jokela^c, E. Tellervo Valtonen^d

^a Department of Biological and Environmental Science, Centre of Excellence in Evolutionary Research, University of Jyväskylä, P.O. Box 35, FI-40014 Jyväskylä, Finland

^b Department of Biology, University of Oulu, P.O. Box 3000, FI-90014 Oulu, Finland

^c EAWAG, Department of Aquatic Ecology, and ETH-Zürich, Institute of Integrative Biology (IBZ), Überlandstrasse 133, P.O. Box 611, CH-8600 Dübendorf, Switzerland

^d Department of Biological and Environmental Science, University of Jyväskylä, P.O. Box 35, FI-40014 Jyväskylä, Finland

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ABSTRACT

Global warming may impose severe risks for aquatic animal health if increasing water temperature leads to an increase in the incidence of parasitic diseases. Essentially, this could take place through a temperature-driven effect on the epidemiology of the disease. For example, higher temperature may boost the rate of disease spread through positive effects on parasite fitness in a weakened host. Increased temperature may also lengthen the transmission season leading to higher total prevalence of infection and more widespread epidemics. However, to date, general understanding of these relationships is limited due to scarcity of long-term empirical data. Here, we present one of the first long-term multi-pathogen data sets on the occurrence of pathogenic bacterial and parasitic infections in relation to increasing temperatures in aquatic systems. We analyse a time-series of disease dynamics on two fish farms in northern Finland from 1986 to 2006. We first demonstrate that the annual mean water temperature increased significantly on both farms over the study period and that the increase was most pronounced in the late summer (July–September). Second, we show that the prevalence of infection (i.e. proportion of fish tanks infected each year) increased with temperature. Interestingly, this pattern was observed in some of the diseases (*Ichthyophthirius multifiliis*, *Flavobacterium columnare*), whereas in the other diseases, the pattern was the opposite (*Ichthyobodo necator*) or absent (*Chilodonella* spp.). These results demonstrate the effect of increasing water temperature on aquatic disease dynamics, but also emphasise the importance of the biology of each disease, as well as the role of local conditions, in determining the direction and magnitude of these effects.

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1. Introduction

Evidence demonstrating the warming of the global climate is indisputable (e.g., Stott et al., 2000; Walther et al., 2002) and it has become clear that climate change presents one of the most serious environmental threats facing global ecosystems. Direct abiotic effects, such as severity of extreme climatic conditions (Knutson et al., 1998; Emanuel, 2005), may be accompanied by a range of indirect biological effects that mediate changes in community structure and species interactions (Parmesan et al., 1999; Peñuelas et al., 2002; Walther et al., 2002; Pounds et al., 2006; Tylianakis et al., 2008). As populations experience direct environmental stress, the risk of parasite and pathogen infections may also increase (e.g., Epstein, 2001; Marcogliese, 2001, 2008; Harvell et al., 2002; Rodó et al., 2002; Lafferty et al., 2004; Hudson et al., 2006; McMichael et al., 2006; Pounds et al., 2006; Lafferty, 2009). In northern latitudes, occurrence and transmission of aqua-

tic parasitic diseases are driven by temperature and primarily take place during the warm summer period (Rintamäki-Kinnunen and Valtonen, 1997; Rintamäki-Kinnunen et al., 2005a,b). This reflects the opportunistic nature of these pathogens; temperature facilitates the multiplication and infection of new hosts as long as the other pathogen-specific conditions for transmission are being met. Elevation in ambient temperature could affect transmission essentially in two ways. First, it could directly enhance parasite metabolism resulting in a higher number of transmission stages being produced, which again would lead to higher parasite fitness and more rapid spread of the disease in a single outbreak. Second, increase in temperature at either end of the natural period of disease occurrence could extend the length of the transmission season resulting in wider overall spread of the disease in the host population. Both of these mechanisms alone, or in combination, would result in higher prevalence of a disease. On the other hand, some diseases could also show the opposite effects with increasing temperature if, for example, their optimal temperature for growth and transmission was lower (e.g., see Pounds et al., 2006). This could lead to decreasing prevalence in such diseases. Several theoretical

* Corresponding author. Tel.: +358 14 260 4250; fax: +358 14 260 2321.

E-mail address: anssi.t.karvonen@jyu.fi (A. Karvonen).

studies have tackled these issues in recent years (see Harvell et al., 2002 for review), but due to the lack of long-term data, empirical understanding of these relationships is limited.

In this paper, we explored the relationships between occurrence of natural pathogenic bacterial and parasitic infections, and water temperature, by analysing a time-series data set collected by a single person from two Finnish fish farms over a period of 21 years (1986–2006). Aquaculture facilities are ideal systems for studies on disease dynamics as they generally harbour a range of directly transmitted disease-causing agents. These pathogens show effective between host transmission at high host densities, which enables their rapid response to changing environmental conditions. Due to the economic loss of infection, diseases are controlled by treating fish with chemicals and drugs. This seemingly stable state where the effective transmission of a disease is controlled by drugs, however, is highly delicate and may easily shift out of balance when the environmental conditions change. One such parameter is water temperature, which promotes the occurrence of the diseases in the wild. As these fish farms draw their water from natural sources, water temperature in the facilities follows the environmental change that the watershed is experiencing. Correspondingly, the disease patterns on the farms reflect the combination of natural disease dynamics of the watershed (pathogens entering with the water) and subsequent pathogen transmission within the farm. We analysed data from two fish farms examining how the mean water temperature changed with time, and how this affected the prevalence of disease spread in the tanks each year. We show that the mean water temperature increased on these farms and that it was positively associated with the prevalence in some of the diseases, whereas in others the effect was negative or absent. Similarly, some of these effects were more pronounced on one of the farms, emphasising the importance of local conditions in determining the direction and magnitude of these effects.

2. Materials and methods

2.1. Study system and data collection

Data were collected from two fish farms (referred to as farms A and B) located in northern Finland between 1986 and 2006 in connection with routine disease monitoring. The farms are located in separate watersheds (River Iijoki and River Oulujoki, ca. 65 km apart, both running into the Baltic Sea) and do not share a water supply. The farms produce smolts of Atlantic salmon (*Salmo salar*) and sea trout (*Salmo trutta*) for stocking purposes by rearing two age cohorts, fingerlings and yearlings, each year. On both farms, the fish were cultured in numerous fibreglass and concrete tanks, as well as in earth ponds, each with an individual water supply. Fingerlings were transferred to new tanks after their first summer where they stayed for the second summer before stocking in the wild. In other words, each tank had a new group of fish each year. Both farms used natural water sources from nearby rivers and therefore changes in water temperature corresponded to natural fluctuations. Water temperature was measured daily at the same time from the incoming water, which ensured that the temperature was the same for all tanks. Incoming water was not heated, cooled or sterilised in any way. Temperatures were recorded daily from the beginning of May to the end of October, from which the mean monthly temperatures were calculated for each year. Furthermore, to explore the within-month variation in temperature during the 21 years, the coefficient of variation was calculated from the daily temperature data for each month.

A total of eight disease-causing agents occurred on the farms during the study including five bacterial species (*Flavobacterium columnare*, *Yersinia ruckeri*, *Serratia* spp., *Aeromonas salmonicida*

(furunculosis) and atypical *A. salmonicida*) and three ectoparasitic protozoan species (the flagellate *Ichthyobodo necator*, and the ciliates *Ichthyophthirius multifiliis* and *Chilodonella* spp.). Detailed descriptions of each disease and their ecology are given in Rintamäki and Valtonen (1991), Valtonen et al. (1992), Rintamäki et al. (1994), Rintamäki-Kinnunen and Valtonen (1997) and Rintamäki-Kinnunen et al. (1997). It is important to note, however, that if untreated, these infections spread quickly in a tank and cause high mortality among fish under farming conditions. Each year, farms were visited weekly during a period of 17 weeks (beginning of June–end of September) and samples of fish from tanks showing symptoms of a disease were examined by the same person (P. Rintamäki) throughout the study. This included standard microscopic examination of the epidermal tissue (parasitic protozoan infections) as well as bacterial cultivation procedures (Rintamäki and Valtonen, 1991; Rintamäki-Kinnunen and Valtonen, 1997; Rintamäki-Kinnunen et al., 1997). Finding fish with a disease was taken as an indication of infection in the whole tank, which is reasonable as these pathogenic infections quickly spread to all fish individuals in a tank. Detection of a disease was followed by an immediate treatment of the infected tank with a disease-specific chemical or drug. However, it is important to note that these treatments commonly last for only 30–60 min (or a few days in the case of antibiotics) and, in most cases, they are applied at different times of the year due to the seasonality of the diseases (Rintamäki-Kinnunen and Valtonen, 1997). Thus, specific treatments targeted against a disease are unlikely to affect the incidence of the other diseases.

The infrastructure of the farms remained virtually unchanged during the study period, although there were slight changes in the number of tanks used each year (mean number of tanks in use per year (\pm standard error (SE)) = 70 ± 4 (farm A) and 43 ± 3 (farm B)). Furthermore, management practises such as disease treatments remained somewhat constant with two exceptions. The introduction of antibiotics in the early 1990s stabilised the mortality associated with *F. columnare*, but had very little or no effect on the disease occurrence per se (P. Rintamäki, unpublished data). Also, the ban of malachite green in treatment of *I. multifiliis* in 2001 led to the introduction of alternative treatment methods (Rintamäki-Kinnunen et al., 2005a,b). However, these changes were unlikely to affect the results in this study as our data recorded only the first outbreak in each tank each year (see Section 2.2), whereas effects of changes in the treatment protocols would most likely be seen in the number and severity of the subsequent outbreaks in the same tanks, which were not considered here.

2.2. Data analysis

The structure of the data set is such that it records the week of the first outbreak (week 1–17) for each disease, year and tank. In other words, possible subsequent outbreaks of a disease in the same tank were not considered and after being infected, the tank was excluded from the data for that particular disease. The analysis used data only from tanks with salmon, which comprised the majority of the tanks on both farms (on average, 92.2% and 60.4% on farms A and B, respectively). The proportion of infected tanks (disease prevalence) was then calculated for each disease, year and age group of fish. For this purpose, each tank within a farm was treated as a unit of observation. This is relevant because (i) all fish within a tank were likely to be infected and only the first outbreak of each disease in a tank was considered, (ii) each tank was equipped with an individual water source making the disease dynamics separate from the other tanks and (iii) fish in each tank were replaced by a new group each year making consecutive years comparable and independent. It should also be emphasised that the farms were visited weekly for the 17-week period making it

unlikely that an infection in a tank would have been missed (condition of fish in the tanks was also monitored constantly by the farm staff). In other words, a record of absence of a disease in a tank was not based on a single but multiple observations during the season.

The data were analysed using autoregressive moving average (ARMA (p, q)) time-series analysis, which controls for possible temporal autocorrelation in disease occurrence between the years. In the analysis, p and q indicate the order of the autoregressive (AR) and moving average (MA) terms of the model, respectively. The analysis was run for the temperature–year relationship separately for both farms to estimate values of p and q which provided the best fit to the data (the model which had the lowest Akaike's information criterion (AIC) value). Prevalence of each disease in relation to temperature was then estimated for each age group of fish on both farms using linear mixed models with correlation structure (errors) taken from the ARMA model. Some diseases had a sporadic occurrence with infections detected in only a few years, or on one of the farms, or in one of the age groups. In such cases, reliable comparisons could not be made and the diseases were excluded from the analysis. All analyses were run in the R 2.8.0 statistical package using “arma” and “gls” functions with $\alpha = 0.05$.

3. Results

The ARMA model of the relationship between mean water temperature of the 17-week outbreak season (beginning of June–end of September), and time (1986–2006), indicated no significant autocorrelations or partial autocorrelations in temperature between the years, which allowed straightforward analysis of the relationship. The mean water temperature increased significantly on both farms during the study (Pearson correlation: $r = 0.678$, $P < 0.001$ (farm A), $r = 0.639$, $P = 0.002$ (farm B); Fig. 1). On the other hand, the coefficient of variation in water temperature (measuring daily variation in temperature) did not change with time ($r = -0.211$, $P = 0.359$ (farm A), $r = -0.054$, $P = 0.817$ (farm B)). Further analysis of the temperature patterns indicated that the increase was most pronounced in late summer, July–September (Fig. 2). Note that we did not subject the monthly temperature data to statistical analysis due to the possibility of complex two-directional autocorrelations between months and between years. The coefficient of variation in monthly water temperature showed no consistent changes over the years.

Four of the diseases (*I. necator*, *Chilodonella* spp., *I. multifiliis* and bacterium *F. columnare*) were common enough for the analysis whereas the remaining four (*A. salmonicida*, atypical *A. salmonicida*, *Y. ruckeri* and *Serratia* spp.) were observed sporadically on one of

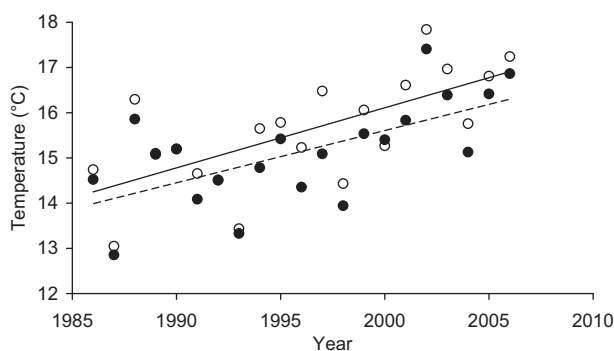


Fig. 1. Mean water temperature (°C) of the disease outbreak season (17 weeks from beginning of June to end of September) on two fish farms in northern Finland (Farm A = open circles, Farm B = solid circles) during a period of 21 years (1986–2006). Fitted lines represent linear regressions (farm A = solid line, farm B = dashed line).

the farms or in one of the age groups (Table 1), and were excluded from further analyses. On both farms, an ARMA model with $p = 1$ and $q = 1$ provided the best fit to the temperature data, i.e. it showed the lowest AIC value. There was a significant positive effect of mean water temperature on the prevalence of *F. columnare* on both farms, indicating that the disease was more prevalent in years of high mean temperature (Table 2, Fig. 3). However, the pattern was more pronounced on farm B, where it was observed in both age groups of fish. The prevalence of *I. multifiliis* was also positively associated with temperature, but this was observed only on farm A. In contrast, the prevalence of *I. necator* in the younger age class of fish on farm B was significantly higher in years of low mean temperature (Table 2, Fig. 3). Similar negative relationships between temperature and the prevalence of *I. necator* were also observed in fingerlings (0+) on farm A, and in yearlings (1+) fish on farm B, but these were not statistically significant (Table 2, Fig. 3). There was no effect of temperature on the prevalence of *Chilodonella* spp. (Table 2, Fig. 3). In four of the 16 analyses, the mixed linear models of the relationship between disease prevalence and temperature produced negative prevalence estimates for some of the lowest temperatures. In such cases, the erroneous estimates were eliminated by forcing the model through origin. However, this procedure had no effect on the statistical significance of the relationships compared with the original models.

4. Discussion

Demonstration of an effect of climate warming on disease dynamics, especially in aquatic systems, has been hampered by the lack of long-term empirical data sets (Harvell et al., 2002; see Harvell et al. (2009) for review on diseases of corals). In this paper, we present one of the first investigations on multi-species disease dynamics in aquatic systems in relation to increasing water temperature. We analysed time-series data from fish farms, where disease dynamics are generally driven by temperature, and kept in equilibrium by high pathogen transmission and effective disease treatment protocols. As such, disease dynamics in these environments rapidly respond to changes in the underlying conditions, which enable the detection of trends in the time frame of the current investigation. We found that increasing water temperature was associated with higher prevalence of some of the diseases (*I. multifiliis*, *F. columnare*), whereas for others, the effect of temperature was negative, or absent, which emphasises the importance of the ecology of each disease. These patterns were also different to some extent between the farms and age groups of fish, which suggests that the effect of temperature on disease dynamics may change with the local conditions or demographic structure of the host population.

There are several, potentially interacting, mechanisms which may underlie the temperature-driven proliferation in disease occurrence. For example, as diseases spread to these facilities with the water supply, to some extent the disease outbreaks on these farms may reflect the incidence of diseases in the natural populations of the watershed upstream of the farm. Under warm conditions, the natural epidemics may be enhanced and therefore more transmission stages may arrive from the upstream watershed, leading to higher disease incidence on the farm (Valtonen and Koskivaara, 1994). This may be accompanied by temperature-driven enhancement in pathogen replication and transmission rate within the farm. Temperature may also have an indirect effect on the establishment probability and spread of the disease on a farm as increasing temperature may become a stressor for salmonid fishes. For example, most of the outbreaks of *F. columnare* and *I. multifiliis* occur during high temperatures in July and August. Increased temperature, particularly in this period (Fig. 2), may have

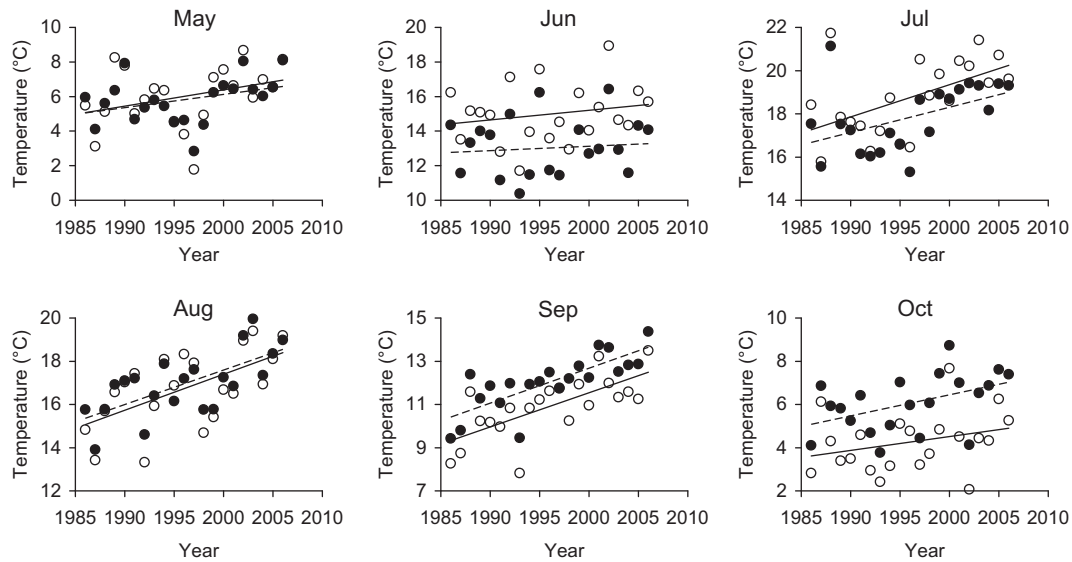


Fig. 2. Monthly (May–October) mean water temperature on two fish farms in northern Finland (Farm A = open circles, Farm B = solid circles) during a period of 21 years (1986–2006). Note differences in y-axis scales. Fitted lines represent linear regressions (farm A = solid line, farm B = dashed line).

Table 1
Mean prevalence (% of tanks infected annually) of eight pathogen species monitored in salmon (*Salmo salar*) fingerlings (age 0+) and yearlings (age 1+) on two fish farms (A and B) in northern Finland in 1986–2006. Percentages in parentheses indicate the mean proportion of tanks with each age cohort of fish. N years indicate the number of years a pathogen was detected on each farm.

Pathogen	Farm A			Farm B		
	N years	0+ (76.4%)	1+ (23.6%)	N years	0+ (56.6%)	1+ (43.4%)
<i>I. necator</i>	21	34.1	25.2	21	27.7	8.3
<i>Chilodonella</i> spp.	20	13.4	10.6	15	9.1	7.2
<i>I. multifiliis</i>	21	2.8	64.2	21	10.2	51.4
<i>F. columnare</i>	20	17.0	38.3	15	10.8	14.0
<i>A. salmonicida</i>	8	3.3	16.7	4	0.4	4.9
<i>A. salmonicida</i> (at)	3	0.3	0.6	2	0.3	0.4
<i>Y. ruckeri</i>	17	6.2	9.5	2	0.6	–
<i>Serratia</i> spp.	6	–	4.3	15	–	22.7

Pathogens: *Ichthyobodo necator*, *Chilodonella* spp., *Ichthyophthirius multifiliis*, *Flavobacterium columnare*, *Aeromonas salmonicida*, atypical *Aeromonas salmonicida*, *Yersinia ruckeri* and *Serratia* spp.

Table 2
Result of autoregressive moving average (ARMA) time-series models and linear mixed models on the relationship between temperature and prevalence of four diseases (*Ichthyobodo necator*, *Chilodonella* spp., *Ichthyophthirius multifiliis* and *Flavobacterium columnare*) in two age groups of salmon (*Salmo salar*; fingerlings (0+) and yearlings (1+)) on two northern Finland fish farms (A and B).

Disease	Farm	Age group	Model parameters		Temperature			
			Phi1	Theta1	Estimate	SE	t	p
<i>I. necator</i>	A	0+	0.366	0.042	–2.629	2.349	–1.119	0.277
	B	0+	–0.627	1.000	–5.753	2.559	–2.248	0.037
	A	1+	0.890	–0.345	0.162	3.975	0.041	0.968
	B	1+	–0.298	0.544	–2.584	1.919	–1.347	0.194
<i>Chilodonella</i> spp.	A	0+	–0.146	0.179	–0.141	1.911	–0.074	0.942
	B	0+	0.250	0.007	0.961	3.320	0.290	0.775
	A	1+	0.338	–0.185	0.862	2.107	0.409	0.687
	B	1+	0.064	–0.325	2.497	1.905	1.311	0.206
<i>I. multifiliis</i>	A	0+	–0.113	0.251	0.191 ^a	0.066	2.878	0.009
	B	0+	0.743	–0.591	2.955	2.415	1.223	0.236
	A	1+	0.820	–0.275	9.249	4.138	2.235	0.038
	B	1+	1.000	–0.429	–2.510	5.410	–0.464	0.648
<i>F. columnare</i>	A	0+	0.401	0.095	1.165 ^a	0.348	3.349	0.003
	B	0+	–0.157	0.428	0.776 ^a	0.308	2.518	0.020
	A	1+	1.000	–0.762	3.585	6.059	0.592	0.561
	B	1+	–0.172	1.000	1.094 ^a	0.368	2.974	0.008

^a Cases where the model was forced through the origin.

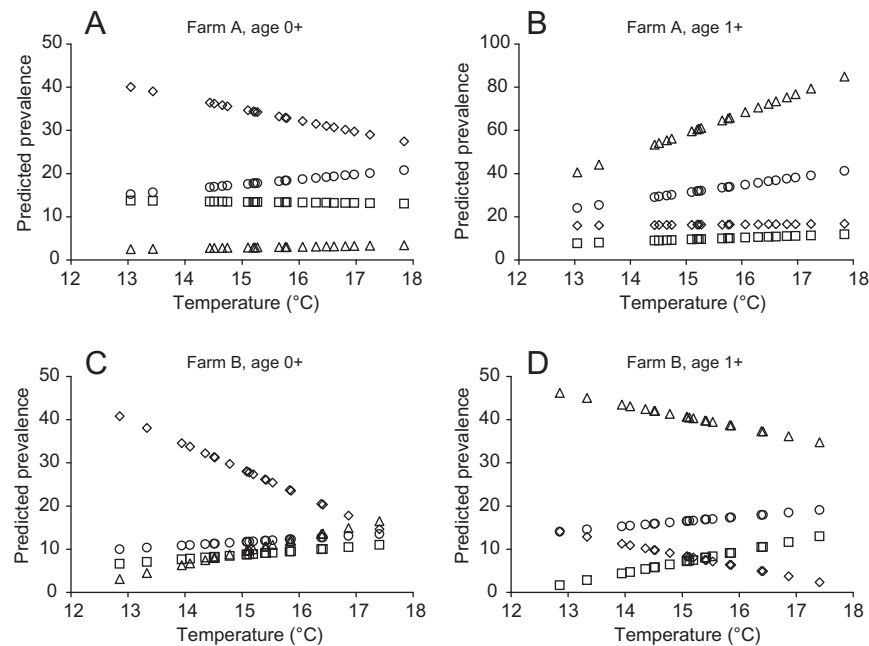


Fig. 3. Predicted prevalence of four disease-causing agents (*Ichthyobodo necator* (diamonds), *Chilodonella* spp. (squares), *Ichthyophthirius multifiliis* (triangles) and *Flavobacterium columnare* (circles)) in relation to temperature. Values are predictions of linear mixed models (Table 2). Analyses were run separately for two age groups of salmon (*Salmo salar*; fingerlings (0+) and yearlings (1+)) on farm A (A and B) and farm B (C and D), both in northern Finland. Note different y-axis scale in B.

favoured higher replication of these pathogens and/or extended the favourable infection period leading to wider seasonal disease spread, i.e. prevalence. It is also possible that the increase in temperature has led to replacement of less virulent pathogen strains with more virulent ones. This is supported by recent findings on *F. columnare* infections in fingerling salmon on one of these farms, indicating an increase in prevalence of more severe disease symptoms in fish (Pulkkinen et al., 2010). In other words, temperature-driven change in the composition of pathogen strains may be one of the mechanisms underlying the observed increase in the prevalence of *F. columnare* (see also Marcogliese (2008) for a review on other diseases). Similar changes in disease symptoms and mortality of fish, however, have not been observed in the other pathogens studied in this system.

The asymmetric pattern of increase in water temperature (late summer) may also partly explain why such a trend was not observed in *Chilodonella* spp. or *I. necator*. The peak of occurrence of these diseases varies to some extent within a season (Rintamäki et al., 1994; Rintamäki-Kinnunen and Valtonen, 1997) and reflects their specific temperature optima. For example, *I. necator* typically shows the highest prevalence in early summer with a lower water temperature (Rintamäki-Kinnunen and Valtonen, 1997), which explains the observed negative effect of temperature. The prevalence of *Chilodonella* spp., on the other hand, was not affected by increasing temperature. These differences in responses of the pathogens could also arise due to mechanisms such as pathogen-specific variation in growth and transmission rates, as well as complex interactions with the host immune system, at different temperatures. Overall, the result emphasises variation in the impact of climate warming on disease dynamics; it may favour some diseases which, for example, coincide with the specific time of temperature increase, while it may also decrease the occurrence of others (see Harvell et al., 2002; Pounds et al., 2006; Lafferty, 2009). It should be emphasised that although these facilities are closely connected to natural systems through the water source, some of the conditions such as host densities are very different compared with the wild. This may lead to different types of disease dynamics e.g., due to differences in host population stress and parasite transmis-

sion rates (Lafferty and Holt, 2003), which limit extrapolation of the results directly to natural systems.

Disease incidence could also be increased if high temperatures impair the condition of ectothermic hosts due to thermal stress (Harvell et al., 2002). This would seem especially likely in cold water salmonid fish species, which are sensitive to high, but also rapidly fluctuating, temperatures. In general, climate warming has two components. First, it increases the mean temperature and, second, it may increase the incidence of extreme climatic effects such as very high temperatures (Easterling et al., 2000). Interestingly, however, we did not observe consistent changes in within-month water temperature variation on the farms during the study period. This may be because the large watersheds that supply the water to these facilities effectively buffer the daily temperature variation and therefore the temperature shows lower variation compared with measurements taken from the air. The direction and magnitude of the changes in the incidence of extreme high temperatures is also known to vary between different parts of the world (Easterling et al., 2000). Indeed, it has been shown that temperature variation has in fact decreased in some areas due to stronger daily warming in minimum temperatures than in maxima (Easterling et al., 1997). Again, this emphasises the asymmetric nature of climate warming.

The effect of temperature was also different between the farms in some of the diseases, which probably reflects the influence of specific local conditions although, geographically, the farms were closely located. Similarly, there were differences between the age groups of fish, which may be related to factors such as disease ecology, farming practises and immunity. For example, *I. necator* is generally more prevalent among young fish which have not yet acquired immunity against the disease (Rintamäki-Kinnunen and Valtonen, 1997). On the other hand, *I. multifiliis* is more prevalent in older fish because the parasite completes its life cycle more efficiently in larger tanks and earth ponds where these fish are held, which apparently overrides the effects of acquired immunity (see Dickerson and Dawe (1995) and Buchmann et al. (2001) for development and maintenance of immunity against *I. multifiliis*). Overall, this emphasises the role of complex interactions between local

conditions, disease ecology, and host demography and immunity, in determining the direction and magnitude of the temperature effects (see also Hudson et al., 2006; Harvell et al., 2009).

To conclude, the observed effects of climate warming on fish diseases indicate that the direction and magnitude of the changes in disease occurrence are strongly influenced by the ecological features of each disease. In other words, while there are pathogens which benefit from climate warming, the prevalence of others can decrease or remain unaffected. Also, these effects are not spatially universal as seen here in the comparison between the two closely situated fish farms, which emphasises the importance of specific local conditions. Predicting which diseases actually become more common in each particular system, as well as unravelling the mechanisms underlying such changes, represent major challenges for investigations on interactions between disease and temperature dynamics. For example, given the positive effects of temperature on disease dynamics, and the asymmetric warming of the climate (late summer–early autumn), shifts in pathogen occurrence which could prolong disease outbreaks form an interesting field of future research. Furthermore, possible ecological and evolutionary changes triggered by a temperature increase, such as emergence of pathogen strains with higher rates of multiplication (Suomalainen et al., 2006), or alterations in interactions between pathogen species or strains (Read and Taylor, 2001), are of key importance in studies on relationships between global warming and diseases. From an applied perspective, our results suggest that some disease related problems in aquaculture are likely to increase in the future. This could be particularly important given that the industry and its importance in food production continue to increase following the depletion of the wild marine and freshwater fish populations.

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