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Chytrid infections and diatom spring blooms: paradoxical effects of climate warming on fungal epidemics in lakes

BAS W. IBELINGS*,†, ALENA S. GSELL*, WOLF M. MOOIJ*, ELLEN VAN DONK*,‡, SILKE VAN DEN WYNGAERT† AND LISETTE N. DE SENERPONT DOMIS*

*Department of Aquatic Ecology, Netherlands Institute of Ecology (NIOO-KNAW), Nieuwersluis, The Netherlands

†Department of Aquatic Ecology, Überlandstrasse, Dübendorf, Switzerland

‡Department of Biology, Palaeoeclogy, University of Utrecht, Budapestlaan, Utrecht, The Netherlands

SUMMARY

1. We describe the dynamics of host–parasite interactions over a period of more than 30 years between the freshwater diatom *Asterionella formosa* and two highly virulent chytrid parasites (*Rhizophydium planktonicum* and *Zygorhizidium planktonicum*) in Lake Maarsseveen, The Netherlands. This period is characterised by a significant warming trend which is strongest in spring.

2. The key spring event in lakes, the diatom bloom, was in many years dominated by *Asterionella*. We examine whether and how climate warming has affected the prevalence of infection in *Asterionella* by chytrids.

3. In years with cold winters/early springs, a dense *Asterionella* bloom is followed by epidemic development of disease as high *Asterionella* densities greatly facilitate transmission of chytrid zoospores. This sequence of events is absent in milder winters.

4. Earlier experimental studies have shown that the parasite is almost non-infective at water temperatures below 3 °C, offering a disease-free window of opportunity for growth of *Asterionella*. Climate warming has reduced periods in which water temperature remains <3 °C, narrowing the window of opportunity for uninfected growth. Consequently, *Asterionella* continuously suffers from infection, albeit at low levels.

5. Population reduction as a result of low level infection allows other diatoms to take over as dominant species, possibly through priority effects.

6. In mild winters, chytrid infections no longer reach epidemic levels, but remain at low prevalence since transmission is impaired at low host densities. Climate warming thus affects both host and parasite in intricate ways, with the host denied a bloom and consequently the parasite denied an epidemic.

7. A shift from *Asterionella* to a mixed diatom community in years with mild winters may benefit the food web, because of the poor edibility of *Asterionella*, unless the numerous chytrid zoospores produced during epidemics significantly contribute to zooplankton nutrition.

8. Our study demonstrates the potential complexity of climate change impacts on disease. A reduction in the likelihood of epidemic development of a virulent parasite would seem to be of great benefit to the host, but this was not the case. Unexpected, sometimes paradoxical consequences of climate change can be expected and suggest that the view of a 'warmer hence sicker world' may not always apply.

Keywords: emerging diseases, food webs, global change, lakes, phytoplankton

Correspondence: Bas W. Ibelings, Department of Aquatic Ecology, Netherlands Institute of Ecology (NIOO-KNAW), Rijksstraatweg 6, 3631 AC, Nieuwersluis, The Netherlands. E-mail: b.ibelings@nioo.knaw.nl

Introduction

Climate warming is changing the timing and intensity of the seasons, but not all seasons are affected equally. In the Northern Hemisphere, winters are warming at about twice the rate of summers (Solomon *et al*., 2007). A consequence of a warmer and shorter winter is an earlier onset of spring, which is moving forward by approximately 1.2 days per decade. In nature, typical spring phenomena like migration, nesting and egg-laying in birds have moved forward (Both & Visser, 2005). In aquatic systems, the phytoplankton spring bloom characterises the end of winter. The spring bloom is the first step in the Plankton Ecology Group (PEG) model of phytoplankton succession (Sommer *et al*., 1986), which summarises the seasonal succession of phytoplankton in temperate lakes via the following sequence of events: ample nutrient availability, increasing day length and rising water temperatures stimulate blooms of fast-growing cryptophytes and diatoms. In turn, the phytoplankton spring bloom provides abundant food for herbivorous zooplankton and thus launches the new growing season for the planktonic food web. Intense zooplankton grazing, together with nutrient depletion and increased sedimentation losses (following an increase in water column stability), terminates the spring bloom, making it relatively short-lived. Several authors have shown that the peak of phytoplankton spring biomass is advancing to earlier dates in the year as a consequence of climate warming (e.g. Gerten & Adrian, 2000; Winder & Schindler, 2004). The changing relationships between spring temperature and photoperiod because of the impact of climate warming may strongly affect community composition of the spring bloom and may also lead to uncoupling of spring phytoplankton blooms and peaks in cladoceran densities (Shatwell, Kohler & Nicklisch, 2008).

The focus of our study is on the effects of climate warming on the diatom spring bloom, but from a specific angle, namely the role of spring bloom diatoms as hosts for abundant and virulent chytrid parasites. In particular, we examine whether and how warmer winters and early spring temperatures affect disease prevalence. The UN's Millennium Ecosystem Assessment (2005) identified strong links between climate change and disease emergence, and climate change is predicted to have important effects on disease and parasitism in freshwater and marine ecosystems despite much uncertainty and interactions with other stressors (Marcogliese, 2008). The most dramatic effects are likely to arise from geographical range extension of pathogens. However, longer growing seasons and higher temperatures may increase the number of parasite generations per year and transmission rates. There is a general presumption that climate change will enhance the spread of diseases and increase their occurrence (see references in Hall *et al*., 2006); however, this is still a matter of debate. Thus, Ostfeld (2009) states that 'clear effects of climate change have now been established for several human infectious diseases', while Randolph (2009) claims that 'there is no single infectious disease whose increased occurrence can reliably be attributed to climate change'.

The role of climate warming is likely to be difficult to separate from other drivers since the occurrence and incidence of disease is the outcome of complex, often nonlinear, interactions between host, parasite and the environment. A proper understanding of the network of extrinsic and intrinsic forces driving the ecology (including transmission processes) and epidemiology (observed patterns) of infectious diseases is necessary to understand the role of climate change in disease dynamics. This requires a focus on well-studied host–parasite systems, where long-term field observations are combined with experimental verification of causal relationships between environmental factors and transmission/epidemiology, backed up by mathematical models which aid the interpretation of field observations. We present such a host–parasite system and relate long-term observations on disease occurrence to experimental data and existing models. The host–parasite system we study consists of a colony-forming, freshwater diatom as a host, and two chytrid parasites that exert very similar pathogenic effects.

The study system

Since observations started in 1978 (Van Donk & Ringelberg, 1983), the chytrids *Rhizophydium planktonicum* Canter and *Zygorhizidium planktonicum* Canter have caused regular epidemic infections of the diatom *Asterionella formosa* Hass in Lake Maarsseveen, The Netherlands. Diatoms (Bacillariophyceae) are among the most important primary producers in oceans, lakes and rivers (Armbrust, 2009). *Asterionella formosa* is a pennate diatom and forms stellate colonies of

varying cell number. It is a major component of phytoplankton, especially in spring and autumn, in lakes worldwide. Chytrids (Chytridiomycota) are true fungi characterised by free-swimming motile stages called zoospores that actively find and infect new hosts. Chytrids that infect phytoplankton are highly host specific, and most, if not all phytoplankton species, are infected (see Ibelings *et al*., 2004). The most severe infections seem to occur in larger phytoplankton species, like *Asterionella* (Kagami *et al*., 2007). Zoospores find the diatom host through chemotaxis and settle onto the cell wall. The fungus penetrates the cell, forms an internal rhizoidal system and extracts resources from the host. The zoospores mature into a sporangium, containing many new zoospores that swim out to find new hosts. In *Asterionella*, recurring epidemics caused by *Rhizophydium* and *Zygorhizidium* can reach an extreme prevalence of infection, often exceeding 90%. The development of disease in *Asterionella* following infection by either one of the chytrids is highly comparable (Canter, 1967, 1969), and as every infection kills the host cell, epidemics are a major factor determining host population dynamics (see Ibelings *et al.* (2004) for review). The development of disease depends strongly on environmental conditions, and the effects of temperature, irradiance and nutrients have been studied experimentally (e.g. Bruning, 1991). Of critical importance is the finding that periods in which water temperatures remain below 3 °C offer a virtually disease-free window of opportunity for *Asterionella* growth.

We present observational data on *Asterionella* spring blooms and chytrid epidemics in Lake Maarsseveen (The Netherlands) spanning a period of more than 30 years. This period is characterised by a general warming trend, but contains both relatively warm and cold winters. Previous research (Van Donk & Ringelberg, 1983; Bruning, 1991) on the effect of temperature upon host and parasite dynamics enables us to hypothesise that warmer winter/early spring temperatures shrink the window of opportunity for *Asterionella* and that low, ongoing chytrid infection prevents dense *Asterionella* blooms. Through priority effects (e.g. Louette & De Meester, 2007), diatoms other than *Asterionella* then dominate the spring community. Since the density of susceptible hosts is thus decreased, we further hypothesise that disease is reduced in warmer winters.

Materials and methods

The field data used for analysis in this study were collected by Van Donk from 1978 to 1982 (Van Donk & Ringelberg, 1983), by Bruning from 1984 to 1988 (with the exception of 1987) (see Ibelings *et al*., 2004) and by Gsell from 2007 to 2010 (unpublished data). Samples were collected from the centre of Lake Maarsseveen using standard limnological techniques. *Asterionella* cell numbers and chytrid infections were counted after sedimentation using an inverted microscope (see Van Donk & Ringelberg, 1983). Both attached zoospores and mature sporangia contribute to infection prevalence, calculated as the percentage of the *Asterionella* cells in samples that were infected. In scoring the infections, no distinction was made between *Asterionella* infected by *Rhizophydium* or by *Zygorhizidium planktonicum*. These two species are taxonomically separated on the basis of whether the sporangia are operculate (*Zygorhizidium*) or non-operculate (*Rhizophydium*) (Canter, 1967, 1969). This feature cannot be determined until dehiscence, thus scoring infection by species is not feasible. However, since pathogenic effects and epidemic development are similar, this lack of distinction should not affect our interpretations.

Lake Maarsseveen is a meso-oligotrophic, dimictic man-made lake, located in the centre of the Netherlands, with a surface area of 100 ha and a maximum depth of 34 m (see Swain, Lingeman & Heinis, 1987 for an extensive description). The principal water supplies are precipitation and ground water; there are no direct connections with other surface waters. Ice cover varies from year to year, from no ice cover in mild winters to up to 8 weeks in cold winters. Sampling was conducted from January to May to catch the period of the diatom spring bloom. As phytoplankton blooms are short-lived, sampling was conducted at weekly or two-weekly intervals. Temperature profiles were recorded intermittently using YSI (Yellow Springs, OH, U.S.A.) thermistors. An uninterrupted data set of temperature measurements is lacking.

Continuous data sets of Lake Maarsseveen water temperatures were calculated using the model of Mooij, Domis & Hulsmann (2008), who demonstrated a tight coupling between air temperature in the nearby meteorological station of De Bilt (ca 8 km from the lake) and lake water temperature for Dutch

polymictic lakes. Although Lake Maarsseveen is relatively deep, we show that for the non-stratified period of the year, as well as for the epilimnion under stratified conditions, the model accurately predicts Lake Maarsseveen temperature from air temperature recordings. Regression models of field data sets (8 years of data) were fitted by minimising the sum of squared difference between the observed and fitted values. For *Asterionella* and chytrid infection data series, different regression models were evaluated, using the adjusted coefficient of determination as criterion. Data were checked for normality and equality of variance prior to analyses. Growth of *Asterionella* and chytrids was calculated by fitting exponential growth curves to the abundance data.

Results

The model of Mooij *et al*. (2008) accurately reproduces surface water lake temperature (Fig. 1; explained variance $R^2 = 93.8\%$; residual standard deviation = 1.37 °C). Deviations between measured and modelled temperatures showed no significant trend ($P > 0.05$) when plotted against daynumber, i.e. the model performs equally well for the non-stratified situation in early spring and for the epilimnion under stratified conditions. Annual average temperature in the lake rose from 10 °C to above 12 °C between 1960 and 2010 (Fig. 2). The strongest warming was seen in winter and spring, from ca 3 °C to nearly 6 °C in winter and from ca 7 °C to 10 °C in spring. Variation in average temperature was large, especially in winter, however.

In most cases, chytrid prevalences peaked following the development of the host diatom bloom (Fig. 3). How does infection of *Asterionella* by chytrid parasites relate to the warming of the lake? Growth rate of the host *Asterionella* increases almost linearly in batch and continuous culture experiments over temperature ranges from 2 °C to 20 °C (Van Donk & Ringelberg,

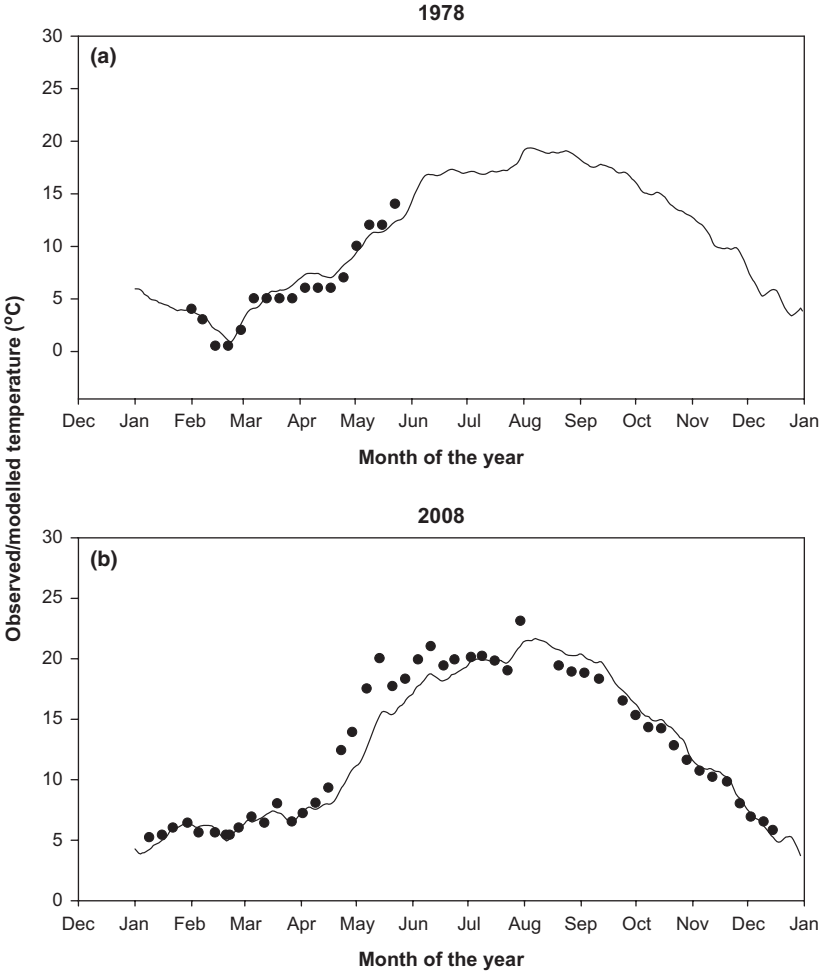

Fig. 1 Observed (black circles) and modelled water temperatures in Lake Maarsseveen (see text for basis of the latter) for an early (winter/spring 1978) and late (2008) year in the data set.

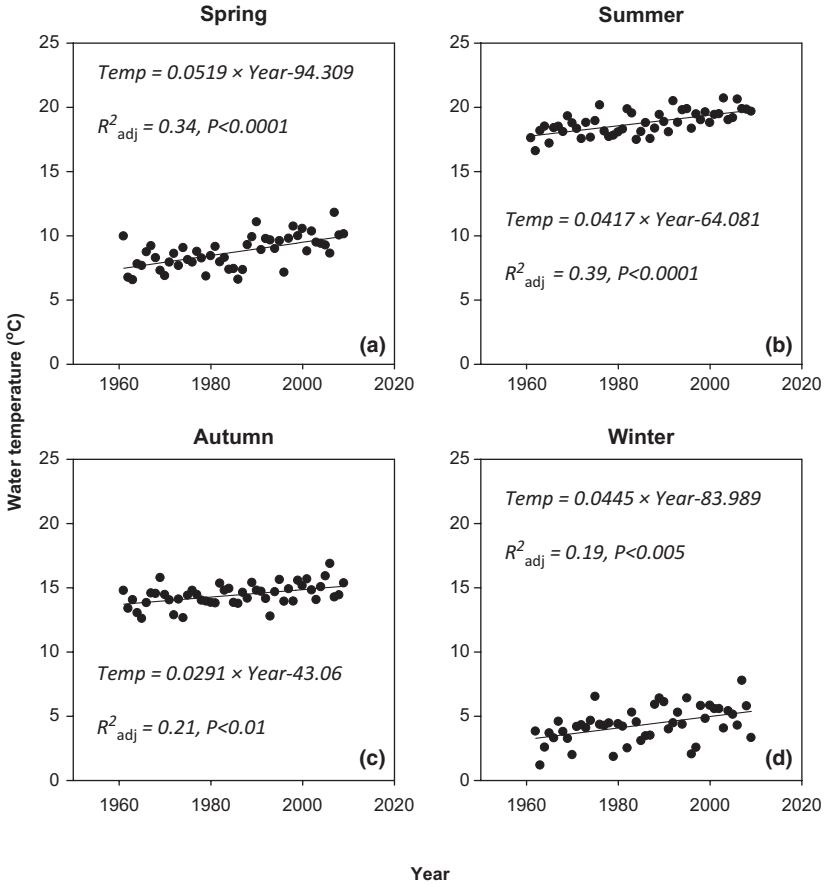

Fig. 2 Warming trend in the surface water temperatures of Lake Maarsseveen, 1960–2010. Data taken from model presented in Fig. 1. Seasons are defined following meteorological seasons of the Northern Hemisphere, e.g. winter months are December, January and February, whereas summer months are June, July and August.

1983; Butterwick, Heaney & Talling, 2005), although Bruning (1991) found that growth rates dropped at temperatures exceeding 15 °C. When growth rates of *Asterionella* are calculated from the exponential rate of increase during bloom periods[1] in Lake Maarsseveen, the pattern observed in culture experiments is seen (Fig. 4). Growth rate steeply increased when water temperatures exceed 3–5 °C and continues to rise over temperatures encountered in spring, but then dropped abruptly at higher temperatures. Temperature in Fig. 4 is presented as the average temperature in the 8 weeks preceding the *Asterionella* bloom. Algal abundances in lakes reflect average temperature conditions over a certain period whose duration is debatable, but 8 weeks allow sufficient time for a response in growth and loss factors that determine abundance. Peak densities of *Asterionella* are reached at much lower water temperatures than the maximum growth rates (Fig. 4). This apparent inconsistency may be explained by population losses, caused by parasitism at higher temperatures.

The growth and abundance of parasites is primarily determined by the presence of susceptible hosts in sufficient numbers (e.g. Anderson & May, 1979a,b). Disease transmission is greatly facilitated by high host cell densities since the zoospores have a limited infective lifetime during which they need to find and infect a new host (Bruning, 1991). An intimate relationship between host cell density and parasite density emerges when data are combined from all years (Fig. 5a). This relationship may seem counter-intuitive. High *Asterionella* densities indeed stimulate epidemic development of the chytrids, but given the extreme prevalence of infection in many years (Fig. 3), epidemics result in severe host population losses. Epidemic development of the fungus should thus result in low host abundance. This relationship is

[1]We define a bloom as a defined period in which the number of cells rises 'rapidly' and reaches levels that clearly exceed 'background levels' in the periods preceding and following the bloom. In practical terms, *Asterionella* cell density in bloom periods exceeds ca 50–100 cells mL^{-1} in Lake Maarsseveen (cf. Maberly *et al.*, 1994).

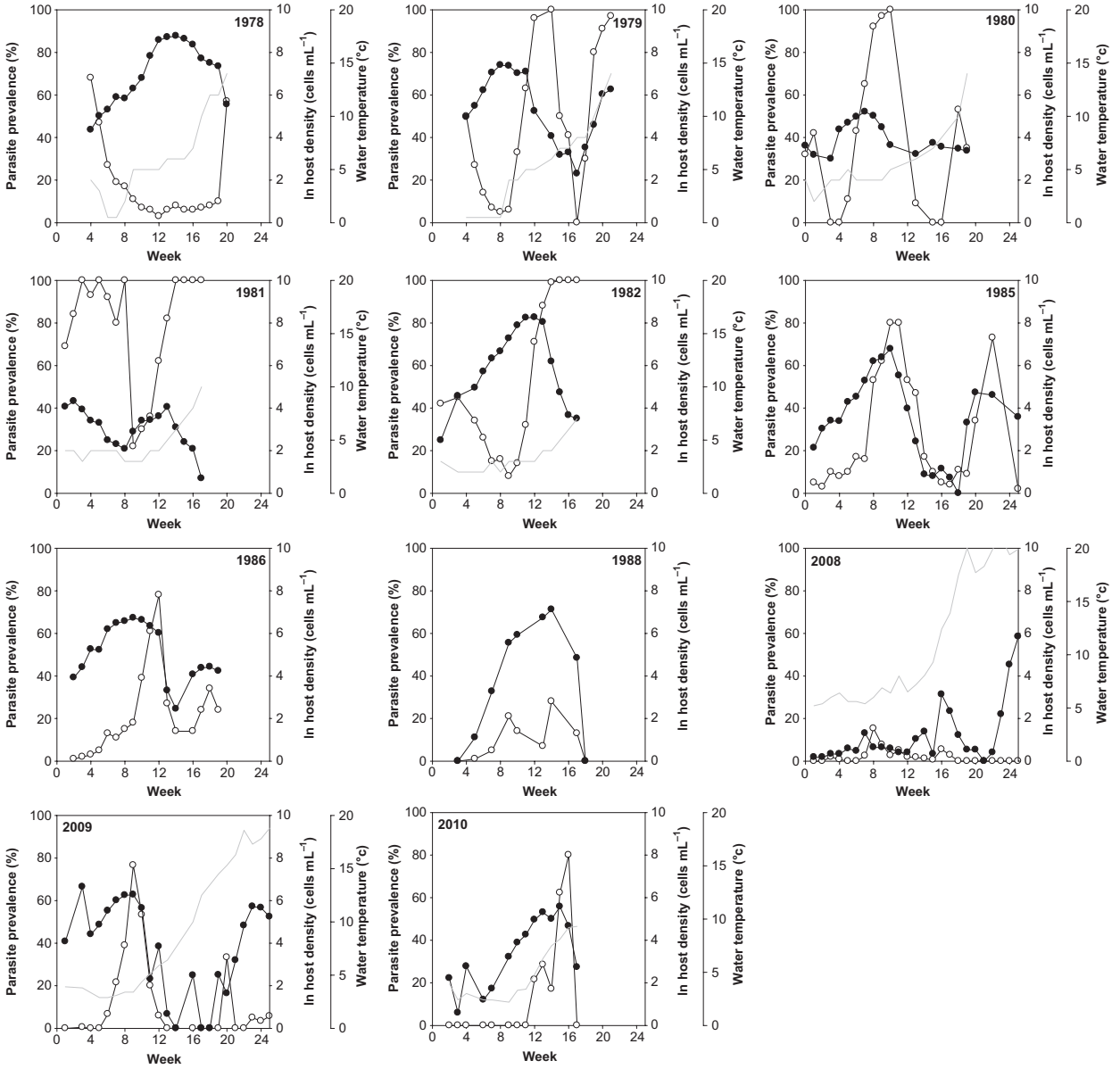

Fig. 3 Seasonal dynamics in cell numbers of *Asterionella* (black circles) and prevalence of chytrid infection (the number of attached zoospores + mature sporangia) (open circles) for eleven years between 1978 and 2010. The grey line indicates water temperature. Data based upon weekly or two-weekly sampling of Lake Maarsseveen..

suggested in Fig. 5b, which shows that while chytrid growth rate is increasing, the abundance of *Asterionella* decreases (the two lines cross), a relationship that, moreover, depends on temperature. Parasite growth rate does show a tendency to correlate with peak host cell density, but this relationship is not significant at $P < 0.05$ level.

Clearly, the timing of events is important. If newly produced *Asterionella* cells were always immediately infected and killed, the host would never build up to bloom proportions. Low host densities in turn would impede transmission of the zoospores to new host cells, arresting epidemic development. The latter requires a window of opportunity for *Asterionella* in which it can grow more or less unhindered by the chytrids and reach high densities before the parasite becomes established (see timing of host and parasite development in Fig. 3). Observations by Van Donk & Ringelberg (1983) and experiments and models by Bruning (1991) and Bruning, Lingeman & Ringelberg

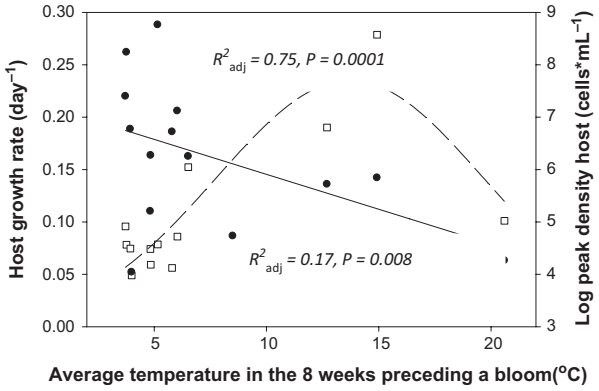

Fig. 4 The net growth rate (open squares; Gaussian curve fit; left *y*-axis) (calculated as the exponential rate of increase over the 8 weeks preceding the bloom) and log peak cell density (black circles; linear fit; right *y*-axis) of the host *Asterionella* (data from Fig. 3.) relative to the water temperature in the 8 weeks preceding a bloom.

(1992) indicate that this window of opportunity can be defined as periods when the water temperature stays below 3 °C. Our field data support this. First, the data show that there is a time lag between the start of the *Asterionella* bloom and the onset of the epidemic (Fig. 6a) with longer time lags between host and parasite development resulting in higher *Asterionella* cell densities. Second, the density of *Asterionella* rises exponentially with an increase in the number of contiguous 3-day periods in which the temperature stays below 3 °C (Fig. 6b). Notably, the number of contiguous 3-day periods below 3 °C has significantly decreased over the period of study (Fig. 6c). Fig. 3 suggests that *Asterionella* spring peaks occur earlier in years with colder winters and later in the year when winters were mild (2008 being an extreme example), a trend supported by the significant linear relationship between temperature and timing of the *Asterionella* spring peak (R^2_{adj} = 0.83, P < 0.0001, data not shown). This relationship is consistent with an effect of parasite-induced losses as discussed earlier.

Overall, our data suggest that climate warming has narrowed the window of opportunity for uninfected growth of *Asterionella*, resulting in a decrease in the occurrence of disease, despite a positive effect of water temperature on growth of both *Asterionella* and the parasite. In an intricate way, climate warming is affecting host and parasite interactions, such that the host is denied a bloom and consequently the parasite is denied an epidemic.

Discussion

Complexity of host–parasite–environment interactions

Our aim for this discussion is to examine the complex interactions between environmental change and parasitism: how do rising temperatures affect the occurrence and severity of disease? For *Asterionella* and the chytrids in Lake Maarsseveen, climate warming has resulted in a reduction in the occurrence and severity of disease, apparently going against the 'warmer hence sicker world' paradigm (see discussion in Hall *et al.*, 2006). Lafferty (2009) concluded that 'it would not be appropriate to build a general theory of climate change and infectious disease around the one-tailed prediction that climate change will increase the problem of infectious diseases'. Our study demonstrates that the effects of climate warming on *Asterionella* are complex: although epidemics have decreased in frequency and intensity, *Asterionella* paradoxically does not benefit. Indeed, arguably the effects of climate warming are detrimental for *Asterionella*, since dense spring blooms have become a rare event, despite the reduced incidence of disease.

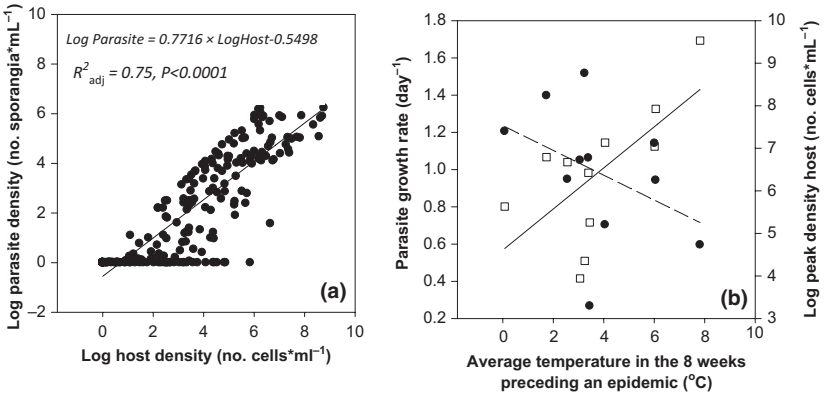

Fig. 5 (a) Intimate relationship between log host density and log parasite density. (b) Parasite growth rates (open squares; linear fit; R^2_{adj} = 0.37, P = 0.03; left *y*-axis) and host densities (closed circles; linear fit; R^2_{adj} = 0.07, P = 0.2; right *y*-axis) weakly associated with water temperature in the 8 weeks preceding the epidemic.

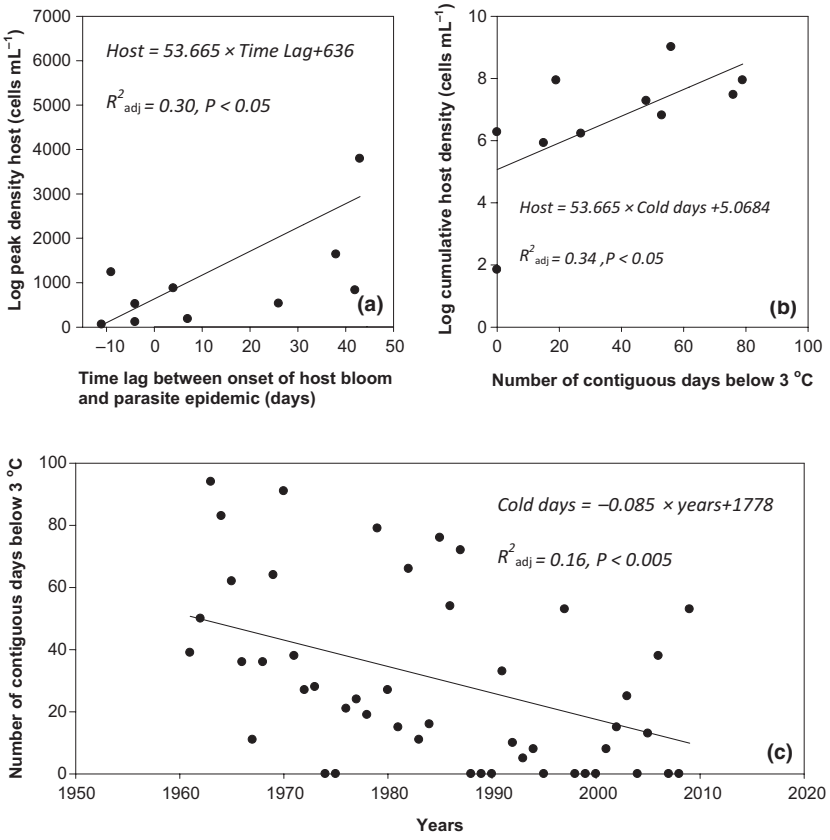

Fig. 6 (a) The time lag between the start of the *Asterionella* bloom and the onset of the epidemic (defined as host peak density). (b) The cumulative *Asterionella* cell density (the total number of cells produced during the bloom) versus the increase in the number of contiguous 3-day periods in which the temperature stays below 3 °C. (c) The number of contiguous 3-day periods below 3 °C from 1960–2010.

The complexities of environmental effects on host–parasite interactions have been investigated by Lafferty & Holt (2003) in an analytical model. They found that environmental stress can have multiple effects on disease, making it unclear what the overall effects of stressors might be. Thus, environmental stressors may (i) increase host susceptibility, for instance because sufficient energy to mount an adequate immune response is lacking; (ii) decrease the density of susceptible hosts, impeding parasite transmission; (iii) reduce the capability of host populations to respond to changes in selection pressure by the parasite, because in dwindling populations, genetic diversity is eroded; and (iv) specifically affect growth and survival of the parasite, reducing the impact of disease on host populations. A stressor like climate change would be most likely to stimulate disease when it enhances host susceptibility without reducing host abundance. Most of the Lafferty & Holt model simulations, perhaps counter-intuitively, indicated that increased stress levels eventually reduce disease, simply because reduced host density impaired efficient transmission. Importantly, this effect is reversed for pathogens that are not host specific, unlike the chytrids infecting phytoplankton.

Temperature, nutrients and mixing

Temperature is an all-pervasive environmental variable, affecting metabolism, growth, reproduction, survival and also biotic interactions. Bruning (1991) conducted a series of meticulous experimental studies using isolates of *Asterionella* and chytrid parasites from Lake Maarsseveen to determine how environmental effects (light, nutrients and temperature) interact and govern the outcome of disease. The specific growth rate of *Rhizophydium planktonicum* is determined by four growth parameters: (i) the number of zoospores produced per sporangium, (ii) the development time of sporangia, (iii) the infective lifetime of zoospores and (iv) the infectivity of zoospores. Increased temperatures consistently decreased the number of zoospores produced per sporangium, both under limiting and saturating light conditions for the host. In contrast, the development time of sporangia sharply decreased with an increase

in temperature. Rising temperatures, however, had a strong negative effect on infective lifetime of the zoospores, which decreased from 12.1 days at 2 °C to 2.1 days at 21 °C (Bruning, 1991). This negative effect of increasing temperature on the infective lifetime of zoospores demonstrates the presupposition (Lafferty, 2009) that increased metabolic costs at higher temperatures are specifically damaging to survivorship of non-feeding free-living stages of parasites. The effect of temperature on the infectivity of zoospores has not been studied yet. However, at low temperatures, the development time of the sporangia of *Rhizophidium* increased. This effect was partly counteracted by an increased number of zoospores per sporangium and an increased infective lifetime of the zoospores at low temperatures (Bruning, 1991). In the temperature range 6–21 °C, the growth rate of the parasite exceeded that of the host, but this was reversed at the lowest experimental temperature of 2 °C (Bruning, 1991). These results imply that at 2 °C, *Rhizophidium* can never become epidemic because more healthy hosts are produced than can be infected. Modelling results (Bruning *et al.*, 1992) have confirmed that low water temperatures are prohibitive for epidemic development of the chytrid, except at very low light conditions which strongly reduce *Asterionella* growth rates.

Inhibition of epidemic development at low water temperatures has been observed under natural conditions. During cold periods, *Zygorhizidium* was inhibited in its parasitic activity because of the formation of thick-walled resting spores (Van Donk & Ringelberg, 1983). The length of the cold period also influences the degree of epidemic development which mainly occurs after prolonged periods of ice cover. When the cold weather period was short, the resting spores had insufficient time for maturation precluding infection upon return to water temperatures >4 °C. However, under the ice and in the absence of snow cover, *Asterionella* grew well and was able to outcompete other diatom species because of the absence of parasitism and its higher affinity for phosphorus (Van Donk & Kilham, 1990).

The effect of environmental change on disease is not restricted to temperature alone, but depends on complex, often nonlinear interactions between several environmental factors, including nutrients. Several authors have pointed out that the warming trends in many temperate lakes are coinciding with re-oligotrophication as a result of lake restoration programmes to reduce phosphorous (Jeppesen, Sondergaard & Jensen, 2003; Van Donk, Santamaria & Mooij, 2003). This complicates the interpretation of the effects of climate change on the functioning of lake ecosystems (Van Donk *et al.*, 2003) particularly as the temporal overlap between climate change and restoration efforts are site specific (Wagner & Adrian, 2009). However, Mckenzie & Townsend (2007) argue that there is mounting evidence that elevated nutrient levels lead to an increased risk of disease even though few studies have directly studied the link between nutrient levels and disease. These complications are likely not to apply in Lake Maarsseveen since historical changes in nutrients are limited compared to most other lakes in The Netherlands. The lake has never undergone strong eutrophication, nor (re)oligotrophication and P concentrations have remained relatively constant (in 1980 total P varied between 8.0 and 17.0 $\mu g\ L^{-1}$ and in 2010 between 7.0 and 14.0 $\mu g\ L^{-1}$).

It is possible that other sources of environmental change may influence the dynamics of *Asterionella* and its chytrid parasites. For instance, global dimming is resulting in a small reduction in insolation in the Northern Hemisphere, and light is an important factor controlling diatom growth rates at the start of the spring bloom (Neale *et al.*, 1991). However, in the Netherlands – if anything – light is slightly increasing although a clear trend is absent (data Royal Netherlands Meteorological Institute). More importantly, elevated temperatures may lead to shallower mixing as a result of microstratification, thus increasing average irradiance for phytoplankton circulating in the near-surface mixed layer (Ibelings *et al.*, 1991). The increase in *Asterionella* growth rate with an increase in water temperature (see Fig. 4) may partially be an outcome of this effect. It is clear, however, that temperature is the main factor driving the interactions between *Asterionella* and the chytrids and the year to year variation in disease. This is supported by both field (Van Donk & Ringelberg, 1983) and experimental investigations (Bruning, 1991), the latter studying temperature effects over a wide range of environmentally relevant levels of irradiance. Experimental studies have indicated that the window of opportunity is present at 2 °C over the range 10–300 μmol photons $m^{-2}\ s^{-1}$, but is absent at higher experimental temperatures, irrespective of irradiance level.

Climate warming is expected to specifically affect larger diatoms like *Asterionella* in yet other ways. Being large and relatively dense organisms, diatoms depend heavily on lake mixing (which is controlled by climatic forcing) to remain in suspension and avoid sedimentation losses. Increased water column stability in a warmer future (e.g. Johnk *et al*., 2008) would select for smaller diatoms. This may partly explain why *Asterionella* is less abundant in warm years in Lake Maarsseveen, but seems unlikely to be particularly relevant for early spring blooms since the *Asterionella* bloom commonly precedes stratification.

Why should *Asterionella* lose its dominant position in mild winters especially as infection prevalence is low when winters are not severe? We hypothesise that the ongoing low level of infection acts as a specific loss factor for *Asterionella* only, lowering the size of the spring inoculum. *Asterionella* populations thus develop too late, and the low population size precludes *Asterionella* being an effective competitor during spring blooms, despite its physiological superiority (Van Donk & Kilham, 1990). Priority effects (Louette & De Meester, 2007) may shift the balance towards other spring diatoms. We are currently working on population dynamic models to clarify the impact of climate warming on the interactions between *Asterionella*, chytrids and other diatoms competing for light and nutrients.

Phenology shifts

Studies on phytoplankton phenology demonstrate a sizeable change in the seasonal timing of chlorophyll maxima. In general, these phenology shifts in aquatic systems appear to be larger than in terrestrial systems (Meis, Thackeray & Jones, 2009). The peak of the spring bloom characterises the pivot point where loss and replication rates are in balance. After the pivot point, loss factors override growth. For diatoms, an earlier onset of SiO_2 limitation resulting from stimulated diatom growth under phosphorous eutrophication has been suggested to explain the advanced centre of gravity of spring bloom chlorophyll maxima, as observed for instance in Lake Windermere in the United Kingdom. (Thackeray, Jones & Maberly, 2008). Loss resulting from parasitism by virulent chytrids should bring the peak in *Asterionella* density forward. However, warmer winters that reduce parasite-induced loss rates may delay peak densities, since the time when losses equal gains is moved back to later in the season. In Lake Maarsseveen, SiO_2 limitation is an unlikely cause for terminating *Asterionella* growth during spring since SiO_2 never drops below 10 μmol L^{-1}. In this system, SiO_2 limitation is therefore unlikely to influence the pivot point where nutrient-limited growth is balanced by loss processes. Notably, the prevalences of chytrid infections of *Asterionella* in Windermere and other lakes in the English Lake remain somewhat below the prevalences in Lake Maarsseveen, e.g.Windermere north basin: 26–63% (1948–51), Windermere south basin: 29–91% (1945–51), Blelham Tarn: 25–50% (1946–51), Esthwaite Water: 25–80% (1946–51) (Canter & Lund, 1953). Hence, a somewhat stronger impact of disease in Lake Maarsseveen and the absence of SiO_2 limitation may explain the difference in phenology shifts for Lake Maarsseveen and lakes in England.

Shifting parasitism – effects on higher trophic levels

Although their impact is still undervalued, it is becoming clear that parasites strongly influence the structure, dynamics, stability and function of food webs (review by Lafferty *et al*., 2008). This being the case, how does the reduction in epidemic development of the chytrids affect the food web of Lake Maarsseveen? The issue of whether and which diatoms dominate in spring is not trivial to lake food webs. Diatoms are abundant phytoplankton, major contributors to carbon fixation and the preferred high quality food for many primary consumers (see Armbrust, 2009).

Van Donk & Kilham (1990) studied competitive interactions between *Asterionella* and two other diatoms species that are commonly found in Lake Maarsseveen, *Stephanodiscus hantzschii* Grunow and *Fragilaria crotonensis* Kitton. Although *Stephanodiscus* had a higher maximum growth rate than the other two diatoms, *Asterionella* was the superior competitor for phosphorus. Under the prevailing phosphorus-limiting conditions of Lake Maarsseveen, *Asterionella* is expected to dominate the diatom community in spring unless infection results in population losses on a large scale (Van Donk & Kilham, 1990). Indeed, this occurred in the warm winters of 2007 and 2008 when a mixed phytoplankton community, including several diatom species, was present throughout the spring period (Gsell, unpubl. data). In addition, Reynolds

(1973) observed that infection of *Asterionella* by *Zygorhizidium* changed a spring diatom community into one dominated by small-celled *Stephanodiscus astrea* Grunow. In colder winters, the timing of epidemic development is crucial for the success of competing diatoms. If the *Asterionella* bloom is broken early enough by disease, there may be sufficient time for development of other diatoms before succession favours other groups like green algae and cyanobacteria.

Asterionella has a variable colony morphology, but commonly is too large to be grazed by herbivorous zooplankton and no grazer is known to feed extensively on it (Sen, 2006). Indeed, *Daphnia galeata* G.O. Sars did not survive a diet of *Asterionella* alone (Kagami *et al*., 2004). Hence, in years when *Asterionella* dominates, the spring bloom transfer of nutrients and energy towards higher trophic levels via grazing zooplankton is likely to be severely hampered. A more mixed phytoplankton spring community should benefit the aquatic system as a whole. Interestingly, Kagami *et al.* (2004) also demonstrated that survival and overall fitness of *Daphnia* drastically increased when *Asterionella* was infected with *Zygorhizidium*. The zoospores produced by the mature sporangia are an excellent size and shape for grazing by *Daphnia* and thus should promote the transfer of nutrients from large inedible *Asterionella* to the aquatic food web. This link, named the mycoloop (Kagami *et al*., 2004), may play a relevant role in lake ecosystems but remains to be demonstrated.

Concluding remarks

In this paper, we have attempted to tease apart the relationships between a marked increase in temperature over the last 30 years, changes in timing and abundance of a diatom host and the occurrence of disease. We hypothesised that lake warming has narrowed the window of opportunity for the host to build up a bloom, unhindered by infection, when water temperature remains below a critical value of 3 °C. Unfortunately, we lack data between 1988 and 2008, a period critical for climate warming since the North Atlantic Oscillation synchronised an abrupt climatic regime shift in the spring of 1988 (Wagner & Adrian, 2009). Furthermore, we were unable to perform proper time-series analysis. Nevertheless, pooling data spanning 30 years has allowed us to assess whether and how winter and spring temperatures affect *Asterionella* blooms and chytrid infections. Our analyses together with previous experimental and theoretical work (Van Donk & Ringelberg, 1983; Bruning, 1991; Bruning *et al*., 1992) support the hypothesis that warmer waters result in a reduction of epidemic chytrid development because insufficient hosts are available for efficient transmission. Paradoxically, *Asterionella* does not benefit from the decreased prevalence of infection because warmer temperatures promote earlier infection. The loss of the disease-free window of opportunity available in cold conditions means that a proportion of the *Asterionella* population becomes infected at the start of the growing season. Thus, although infection levels in mild winters remain well below the extreme prevalences that occur in colder years, chytrid infections act as a species-specific loss factor, reducing the size of *Asterionella* inoculums, weakening its competitiveness and promoting priority effects. The role of these priority effects in determining the composition of the diatom spring community is currently the subject of a modelling study (Gsell *et al.* in prep).

So far, we see no evidence that the Lake Maarsseveen ecosystem has shifted to an alternative state in which *Asterionella* blooms or chytrid epidemics have disappeared. The likelihood of these events appears to be decreasing, but cold winters still occur (e.g. winters of 2008–2009 and 2009–2010). During these cold winters, the lake still responds like it did in the 1970s, exhibiting *Asterionella* blooms after ice melt followed a few weeks later by a large-scale chytrid epidemic. Thus, *Asterionella* resting stages still allow a rapid response when a disease-free window of opportunity arises, and the chytrids still respond favourably to the development of large numbers of host cells. Whether this will remain the case if windows of opportunity become increasingly rare is questionable. For instance, there may come a point where the size of the viable *Asterionella* population becomes too small, and *Asterionella* can no longer effectively compete with other spring bloom phytoplankters even in the absence of infection.

Our study presents a scenario that runs counter to the general expectation of a 'warmer hence sicker world'. The body of research on *Asterionella* and chytrid infections in Lake Maarsseveen demonstrates that climate warming is not invariably linked with disease emergence and indeed may result in the

opposite – warmer winters promote a reduction in disease. This diminution of disease promotes changes in the composition of the diatom spring community, leading to enhanced edibility of the spring phytoplankton. This in turn may have positive effects on higher trophic levels. We are only beginning to understand the complex, often nonlinear interactions between components of the food web, including parasites, in relation to environmental change. Further research is required to understand the potential consequences of climate change on population interactions and community composition.

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