Freshwater Biology

Freshwater Biology (2011) **56,** 676–688 doi:10.1111/j.1365-2427.2010.02425.x

Biological invasions and the dynamics of endemic diseases in freshwater ecosystems

R. POULIN*, R. A. PATERSON*, C. R. TOWNSEND*, D. M. TOMPKINS† AND D. W. KELLY*,†
*Department of Zoology, University of Otago, Dunedin, New Zealand
†Landcare Research, Dunedin, New Zealand

SUMMARY

1. Biological invasions, still occurring worldwide at an alarming rate, are widely acknowledged as threats to the integrity and functioning of ecosystems. In addition to introducing disease, biological invasions have also been linked to sudden increases in the incidence or severity of previously existing diseases. We review and illustrate the potential direct and indirect impacts of introduced species on the dynamics of endemic parasites in freshwater ecosystems.
2. Introduced species may trigger and sustain disease emergence by acting as competent hosts for endemic parasites in which infection is amplified and then 'spilled back' to native hosts. In contrast, if introduced species are not suitable hosts for endemic parasites but become infected anyway, they may act as sinks for parasites and thus dilute disease risk for native hosts.
3. Another mechanism by which introduced species can influence endemic parasitic diseases is by altering the relative abundance of one of the parasite's hosts in ways that could either enhance or reduce disease transmission to other native hosts in the parasite's life cycle.
4. Introduced species may also alter disease incidence and severity in native hosts through trait-mediated indirect effects. For example, the introduced species could change the exposure or susceptibility of native hosts to infection by causing alterations in their behaviour or immunocompetence. Also, by directly changing physicochemical conditions and modifying environmental stressors introduced species may indirectly affect native host exposure and/or resistance to disease.
5. A survey of parasites infecting introduced freshwater fish in four distinct geographical areas revealed that use of non-indigenous hosts by endemic parasites is widespread, mostly involving parasites transmitted via the food chain.
6. We conclude by presenting a framework, based on risk assessment, for the prediction and possible mitigation of the impact of introduced species on endemic diseases and by calling for greater recognition of the potential role of invasive species as triggers of endemic disease emergence.

Keywords: dilution effect, introduced species, parasite spillback, risk assessment, trait-mediated indirect effects

Correspondence: Robert Poulin, Department of Zoology, University of Otago, PO Box 56, Dunedin 9054, New Zealand. E-mail: robert.poulin@stonebow.otago.ac.nz

Introduction

Biological invasions represent a major component of global change, imposing huge economic costs to society (Perrings *et al*., 2005; Pimentel, Zuniga & Morrison, 2005; Hulme *et al*., 2009). The globalisation of modern transport systems ensures that the rate at which invasions occur will remain high (Cohen & Carlton, 1998; Ruiz *et al*., 2000; Drake & Lodge, 2004; Perrings *et al*., 2005). The introductions of non-indigenous species (NIS) pose threats to the integrity and functioning of ecosystems, being (after habitat destruction) the second most important proximate cause of native biodiversity loss worldwide (Wilcove *et al*., 1998; Grosholz, 2002; Clavero & Garcia-Berthou, 2005; Molnar *et al*., 2008). At the same time, emerging infectious diseases and the parasites that cause them are reported with increasing frequency from a wide range of systems, and they too threaten biodiversity and ecosystem functioning (Daszak, Cunningham & Hyatt, 2000; Smith, Acevedo-Whitehouse & Pedersen, 2009). An emerging infectious disease is one that appears for the first time in a population, or a previously existing disease that suddenly increases in incidence or geographic range, or that manifests itself in a new way (Daszak *et al*., 2000).

Biological invasions and emerging infectious diseases are not necessarily independent of each other. The former may trigger the latter and together they may have additive or synergistic effects on ecosystems (Fèvre *et al*., 2006; Brook, Sodhi & Bradshaw, 2008). The most obvious way in which biological invasion can be linked with emerging infectious disease is when NIS introduce and transmit novel parasites to native species (Daszak *et al*., 2000; Taraschewski, 2006; Dunn, 2009). For example, the only freshwater crayfish native to the British Isles, *Austropotamobius pallipes* Lereboullet, once widespread, has suffered several local extinctions since the 1980s, and its geographical distribution is now greatly restricted (Holdich & Reeve, 1991). Its decline appears to have been mediated by the oomycete *Aphanomyces astaci* Schikora introduced in the 1970s along with the invasive American crayfish *Pacifastacus leniusculus* Dana; although asymptomatic in its original host, the fungus causes mortality in native European crayfish (Holdich & Reeve, 1991; Kozubikova *et al*., 2009). Similarly, populations of the native European eel, *Anguilla anguilla* L., have declined markedly following the introduction to Europe of the eel-specific parasitic nematode *Anguillicola crassus* Kuwahara, Niimi & Hagaki, along with its original host from East Asia, via importation of live eels to Germany in the early 1980s (Taraschewski, 2006). Although overfishing and other causes have doubtless played large roles, the severe pathology induced by the nematode in its new European host and its rapid spread are probably key contributing factors (Taraschewski, 2006). Introductions of novel parasites along with their non-indigenous hosts may not be as common as one might think, however, since empirical evidence indicates that during introduction NIS tend to lose most of the parasites they had in their region of origin (Torchin *et al*., 2003).

Interactions between NIS and parasites of native host species may therefore be of greater importance, or at least they may be relevant to a greater proportion of biological invasions. On the one hand, parasitism in native species may facilitate the invasion process by making native species more susceptible to predation or competition from NIS (Prenter *et al*., 2004; Dunn, 2009). In Irish freshwater habitats, for instance, a microsporidian parasite infecting the native amphipod *Gammarus duebeni celticus* Stock & Pinkster reduces its host's capacity to prey on small invasive amphipod species and increases the host's likelihood of being preyed upon by larger invaders (MacNeil *et al*., 2003). On the other hand, under certain circumstances, NIS can directly or indirectly alter the dynamics of endemic parasites, possibly initiating and then sustaining emerging diseases (the 'endemic pathogen' hypothesis of disease emergence; Rachowicz *et al*., 2005). Changes in the environment can affect many steps in the infection process, such as the survival of parasite transmission stages or host resistance, as well as modulating parasite virulence or host recovery rates. The potential of environmental change to alter disease dynamics in the wild has been discussed at length in the context of climate change (Marcogliese, 2001; Harvell *et al*., 2002; Mouritsen & Poulin, 2002; Lafferty, 2009). Similarly, the introduction of NIS may perturb native host–parasite interactions by, for instance, acting as alternative hosts for endemic parasites or by altering the behaviour and subsequent infection risk of native host species. Biological invasions may therefore be an underestimated cause of emerging infectious diseases.

In this review, we explore the potential impacts of introduced species on the dynamics of endemic parasites in freshwater ecosystems. We focus specifically on situations where introduced species have the potential to cause a previously existing endemic parasite to increase in prevalence or severity, thus triggering disease emergence. First, we discuss the different mechanisms by which NIS can influence the dynamics of endemic parasitic diseases. We illustrate each mechanism with case studies from freshwater systems and also consider alternative scenarios where introduced species lead to reductions in infection levels or in their consequences for native hosts. Second, we use published surveys of freshwater fish introductions to provide quantitative estimates of how frequently NIS might serve as alternative hosts of endemic parasites, which is how they affect endemic disease dynamics in most cases. Third, we present a framework for the prediction and possible mitigation of endemic disease emergence because of species introduction. Our overall goal is to expose the under-appreciated but potentially important link between biological invasions and disease emergence in freshwater ecosystems and to address its practical implications.

Impact of invaders on endemic diseases

There are several mechanisms by which NIS might influence endemic diseases. These are not mutually exclusive and they can act in concert in many situations. In addition to acting as hosts of endemic parasites, NIS can increase the severity of endemic diseases by inducing either numerical or functional changes in native species. In other words, introduced species may change, directly or indirectly, the abundance of one or more hosts of endemic parasites in ways that promote parasite transmission or they may induce changes in the behaviour or physiology of native hosts that make them more susceptible to infection. The various scenarios discussed below are illustrated in Fig. 1 using the example of a parasite with a two-host life cycle that is typical of many freshwater parasite taxa such as nematodes, cestodes,

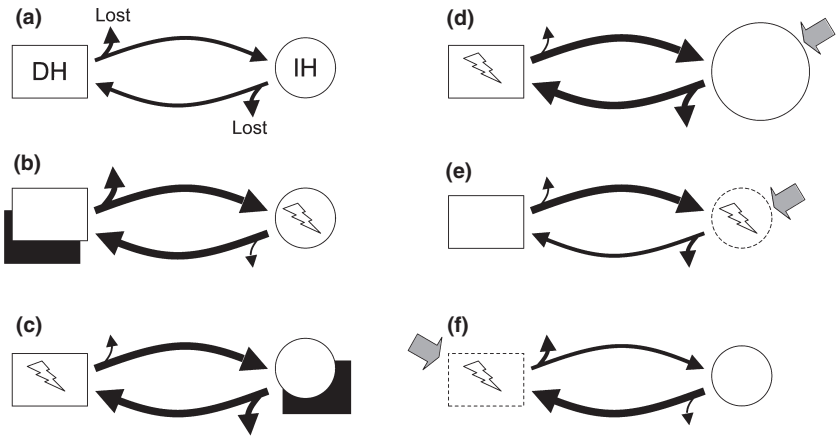

Fig. 1 Summary of possible impacts of NIS on the dynamics of endemic parasitic diseases that might lead to disease emergence. The hypothetical parasite considered here has a two-host life cycle involving a definitive host, DH (white rectangle), and an intermediate host, IH (white circle). During transmission from one host to the other, some parasites are unsuccessful and therefore lost from the system; the thickness of the arrows indicates the relative numbers that are either lost or successfully transmitted. The NIS (black rectangle) can either act as an alternative host for the parasite, or have an indirect effect (shaded arrow) on one of the native hosts. The native host incurring an emergent disease (i.e. an increase in infection rate) is indicated by a lightning bolt. (a) The situation prior to the invasion, providing a benchmark for comparisons. (b) The invader is a suitable alternative definitive host, more parasites therefore reach a definitive host and more infective stages are produced to infect the intermediate host. (c) Same as the previous scenario except that the invader can serve as an alternative intermediate host, leading to greater infection risk for native definitive hosts. (d) The invader indirectly causes the population of native intermediate hosts to increase in size (larger circle), for instance by feeding on their predators or competitors, which leads to reduced losses at that stage of the parasite's life cycle, and greater infection risk for native definitive hosts. (e) The invader indirectly causes intermediate hosts to become more susceptible to infection, for instance by forcing them to change their microhabitat or diet, or via immunosuppression induced by stress. Induced habitat changes may be spatially extensive, such that the native species uses sub-optimal habitats in which infection risk is modified by other stressors. (f) Same as the previous scenario except that the invader indirectly affects the definitive host instead of the intermediate host.

acanthocephalans and myxozoans (scenario a in Fig. 1). Adults or reproductive stages of these parasites typically exploit vertebrate definitive hosts, from which they release eggs or transmission stages that must infect an intermediate host, usually an invertebrate; the life cycle is completed when infected intermediate hosts (or, in the case of myxozoans, further transmission stages released from intermediate hosts) are eaten by a suitable definitive host. The mechanisms discussed later apply equally well to other types of parasites, and equivalent scenarios to those in Fig. 1 could easily be developed for parasites with life cycles involving three or more host species, such as those of many trematodes, or for simple one-host life cycles such as those of most viruses, bacteria, fungi, monogeneans and parasitic copepods.

Parasite spillback: non-indigenous species as hosts of endemic parasites

The most obvious way in which NIS can influence endemic parasite dynamics is by playing an active role in their life cycle and transmission. Following their introduction to a new geographical area, NIS may serve as alternative hosts for endemic parasites, from which infection may 'spill back' to native fauna (Daszak *et al*., 2000; Tompkins & Poulin, 2006; Kelly *et al*., 2009b). It is not unusual for introduced species to acquire parasites from the local fauna. For example, salmonid fishes have been introduced to many parts of the world as fertilised eggs and completely free of the parasites from their area of origin, but following establishment they have accumulated endemic parasites in numbers and taxonomic diversity matching those from their original area (Poulin & Mouillot, 2003). In addition, introduced salmonids often harbour higher abundances of endemic parasites than native host species (Kennedy, Hartvigsen & Halvorsen, 1991). When NIS are competent hosts for endemic parasites (i.e. hosts in which the parasites can develop normally), they may amplify the total number of infective stages to which native hosts are exposed, potentially leading to an emerging disease (scenarios b and c in Fig. 1).

Parasite spillback from NIS to native hosts may be an important but neglected cause of disease emergence. In a review of data available in the literature, Kelly *et al*. (2009b) found that NIS had acquired a mean of 6.3 endemic parasites following their introduction, with 70% acquiring more than four endemic parasites. The non-indigenous taxa in this survey included aquatic and terrestrial invertebrates and vertebrates, while the parasites included protozoa, helminths and arthropods. The literature contains too few rigorous surveys of the frequency at which endemic bacteria and viruses are acquired by NIS to obtain accurate estimates, but we suggest this will be equally common.

A study of the dynamics of fish parasitism in Lake Moreno, Argentina, provides strong evidence for parasite spillback. Two introduced salmonids, rainbow trout (*Oncorhynchus mykiss* Walbaum) and brook trout (*Salvelinus fontinalis* Mitchill), both native to North America, are used as alternative hosts by four endemic parasites acquired from native fish (Rauque, Viozzi & Semenas, 2003). Together, the two introduced salmonids represent only 3% of the total fish abundance in the lake, and yet they now play a very important role in the life cycle and transmission of the endemic acanthocephalan parasite *Acanthocephalus tumescens* von Linstow. Although the parasite does not reach infection intensities as high in the salmonids (<9 worms per fish on average) as in its native hosts (averages in three native fish: 10–27 worms per fish), a higher proportion of female worms reach maturity and produce eggs in the introduced salmonids than in native hosts (Rauque *et al*., 2003). The upshot is that the two salmonids account for approximately a quarter of all parasite eggs produced and released in lake waters. Although it remains to be tested, the boost in parasite reproduction made possible by a relatively small number of exotic hosts should result in greater risk of infection for native species, including both amphipod intermediate hosts and fish definitive hosts.

Studies of recurrent mass mortalities in a variety of waterfowl species in North American freshwaters have identified infection by the trematodes *Cyathocotyle bushiensis* Khan and *Sphaeridiotrema globulus* Rudolphi as the probable cause (Hoeve & Scott, 1988; Herrmann & Sorensen, 2009). Transmission to birds occurs when the introduced gastropod *Bithynia tentaculata* L., an intermediate host for both parasites, is eaten by molluscivorous birds. Although *C. bushiensis* was probably introduced, *S. globulus* occurs in several native gastropod intermediate host species in areas where the invasive *B. tentaculata* is absent (e.g. Roscoe, 1983), and is therefore most likely native. The

extremely high densities at which *B. tentaculata* can occur, coupled with high prevalence and intensity of *S. globulus* infection, and heavy feeding activity on snails by birds, increase the probability that waterfowl will ingest a lethal infection dose (Herrmann & Sorensen, 2009). An almost identical situation exists in Belarus, where the introduced zebra mussel, *Dreissena polymorpha* Pallas, has become disproportionately abundant relative to native molluscs. This introduced bivalve now harbours much higher intensities of infection by endemic trematodes than native molluscan hosts and may act as a source of heavy infections for the waterfowl that prey on molluscs and serve as the trematodes' definitive hosts (Mastitsky & Veres, 2010). These systems show how an abundant NIS acting as an intermediate host can increase infection risk to native definitive hosts (scenario c in Fig. 1).

Another apparent but untested case of spillback has been documented in Lake Chichancanab, Mexico, where African cichlid fish, *Oreochromis* spp., were accidentally introduced two decades ago. Over the following few years, as cichlid abundance rapidly increased, population sizes of five native species of the genus *Cyprinodon* declined dramatically and a sixth one became extinct (Strecker, 2006). The cichlids are detritivore–planktivores, and the decline of native fish was therefore not because of predation by the invasive species. All the fish species, both native and introduced cichlids, serve as second intermediate hosts in the transmission of endemic trematodes to the piscivorous birds used by the parasites as definitive hosts. Pre-invasion data show that *Cyprinodon* fish were infected at low prevalence (<25%) by trematodes, but within 6–7 years of the invasion, prevalence reached 90–100% (Strecker, 2006). In this system, birds preyed heavily on the NIS because of their larger sizes, greater abundance and greater use of open-water habitats compared to native fish. Use of cichlids by the parasites augmented the flow of infection from NIS intermediate hosts to native birds, and back to native fish intermediate hosts (Strecker, 2006). This illustrates how parasite spillback from introduced species could potentially affect all host species in a parasite's life cycle.

One of the main criteria for spillback to occur is that the introduced species must be a competent host for the endemic parasites it acquires. If parasites cannot develop in the NIS but infect it anyway, then it may act as a sink for the parasite population. This would reduce infection levels in native hosts via a 'dilution' effect (Keesing, Holt & Ostfeld, 2006). The introduction of European brown trout, *Salmo trutta* L., to New Zealand has apparently had that effect on native fish species. Although many endemic parasites are found in trout (Dix, 1968), the latter are not suitable hosts since most of these parasites do not reach maturity inside trout. A recent study has found a negative relationship across different streams between intensity of infection by endemic trematode species in two native fish, *Gobiomorphus breviceps* Stokell and *Galaxias anomalus* Stokell, and an index of local trout abundance (Kelly *et al*., 2009c). In other words, in sites where trout are abundant, trematode infections in native fish are less severe. One possible mechanism is that, after leaving their snail first intermediate host, the free-swimming infective stages of trematodes that encounter trout infect this host but fail to complete their development; a greater proportion of infective stages would thus be lost in sites with more abundant trout populations (Kelly *et al*., 2009c). However, modelling and experimental studies are required to confirm that availability of infective stages can be limiting in the parasite life cycle and that their wastage by non-competent hosts (akin to 'lost bites' in vector-borne diseases) does lead to reduced infection in other host species. Other studies have recently provided evidence that invasive snails are often not suitable hosts for the trematode infective stages they encounter, which can lead, at least in mesocosm studies, to a dilution of infection for native snail species (Kopp & Jokela, 2007; Genner, Michel & Todd, 2008).

NIS do not necessarily need to kill endemic parasites post-infection for a dilution effect to occur; they may also cause direct mortality of infective stages by feeding on them or causing transmission failure via physical interference (Thieltges, Jensen & Poulin, 2008). Indeed, numerous freshwater invertebrates can actively feed on the infective stages of a range of parasites (e.g. Christensen, 1979; Achinelly, Micielli & Garcia, 2003) and active filter-feeders, such as the dreissenid mussels invasive throughout North America, may be capable of clearing most free-swimming stages of parasites from the surrounding water (MacIsaac *et al*., 1992; Pace, Findlay & Fischer, 1998; Faust *et al*., 2009). Thus, whether an NIS has the potential for positive (spillback) or negative (dilution) effects on

endemic parasites depends on whether it is a suitable host for infection and development of the parasite, as opposed to a sink that causes the loss of infective stages.

Numerical impacts of non-indigenous species on native hosts

The best-documented impacts of NIS are reductions in the abundance or density of one or more native species, sometimes to the point of extinction (Grosholz, 2002; Clavero & Garcia-Berthou, 2005). If negatively impacted species are essential hosts in the life cycle of an endemic parasite, then the parasite's local population will decline, and its other host species may benefit indirectly from the invasion. However, if instead the NIS causes a reduction in the abundance of a predatory species that kept in check the intermediate host of an endemic parasite, then the opposite could happen: the parasite's transmission rate would increase locally with negative consequences for its other hosts (scenario d in Fig. 1).

The impact of invasive dreissenid mussels (i.e. the zebra mussel, *Dreissena polymorpha* Pallas, and quagga mussel, *D. bugensis* Andrusov) provides a good example. Originally from Ukraine and southern Russia, these mussels were introduced in the 1980s to freshwater systems in North America, where their local densities can now be very high. Among the many reported ecosystem impacts of these NIS, there is clear evidence that the abundance of native macrobenthic invertebrates is higher where the invasive mussels are present than where they are not (Botts, Patterson & Schloesser, 1996; Mayer *et al*., 2000). This is probably caused by the physical habitat provided by clumps of mussels and by their faeces and pseudofaeces (mucus pellets in which unfiltered particles are concentrated) enhancing detritus-based benthic food webs. Invertebrates whose abundance is increased include amphipods and oligochaetes that play major roles as intermediate hosts of myxozoans, acanthocephalans, nematodes and cestodes parasitic in fish (Williams & Jones, 1994). At the same time, the filtering activity of dreissenid mussels can cause substantial decreases in the abundance of small-sized zooplankton (MacIsaac *et al*., 1992; Pace *et al*., 1998). These include the small cyclopoid copepods used exclusively as intermediate hosts by cestodes of the genus *Proteocephalus*, which are very common parasites of North American freshwater fish (McDonald & Margolis, 1995; Scholz, 1999). Although the consequences of increases (or decreases) in intermediate host abundance caused by NIS could well include enhanced (or reduced) transmission rates of parasites back to fish, these remain unexplored to date.

Functional impacts of non-indigenous species on native hosts

To initiate endemic disease emergence, NIS may not have to act as alternative hosts for endemic parasites or even to cause changes in the abundance of native hosts involved in the parasite life cycle. The process could instead involve trait-mediated indirect effects (Werner & Peacor, 2003); NIS might change the exposure or susceptibility of native hosts to infection by causing alterations in their behaviour or immunocompetence (scenarios e and f in Fig. 1). For instance, there is considerable empirical evidence that prey respond to the perceived threat of predation, even predation from novel predators, by changing some aspect of their phenotype such as activity levels, microhabitat choice, prey selection, morphology and life history characters (Boersma, Spaak & De Meester, 1998; Lass & Spaak, 2003; Trussell, Ewanchuk & Matassa, 2006; Wolinska, Loffler & Spaak, 2007; Johansson & Andersson, 2009). Similarly, perceived predation risk can cause physiological stress in prey individuals, measurable as increased levels of stress proteins such as heat-shock proteins or hormones such as cortisol (Kagawa & Mugiya, 2000; Woodley & Peterson, 2003; Slos & Stoks, 2008). Exposure to infection in aquatic organisms depends mostly on diet and microhabitat, since most aquatic parasites are acquired either via ingestion or direct contact with a free-swimming infective stage. Resistance to infection depends on the immunocompetence of the exposed individual and increased stress can depress immunity (Apanius, 1998; Raberg *et al*., 1998; Harris & Bird, 2000). Therefore, if the NIS is a predator or is perceived as such by native species, the latter may experience greater exposure to parasites by shifting to different microhabitats and feeding on different food items, and greater susceptibility to infection because of stress-mediated immunosuppression. In addition, by directly changing physicochemical regimes and modifying environmental stressors, NIS may also indirectly affect host exposure and/or resistance to disease.

These sorts of indirect effects on native freshwater animals have been reported following the introduction of NIS. For instance, the black bullhead *Ameiurus melas* Rafinesque, an ictalurid catfish native to North America, has become one of the most successful exotic fish in European freshwater ecosystems. A series of experiments has shown that the presence of this invader reduces the predation success of *Esox lucius* L. on minnows, not by actively competing with pike for minnows, but simply by interfering with the normal behaviour of pike (Kreutzenberger, Leprieur & Brosse, 2008). Infection levels by parasites normally transmitted trophically from minnows to pike may be reduced as a consequence. However, since pike switch to other food items, they may be exposed to other parasites transmitted via these different preys. In the Laurentian Great Lakes of North America, the large predatory cladoceran *Bythotrephes longimanus* Leydig, introduced from Eurasia in the early 1980s, induces a change in the vertical distribution of its zooplanktonic prey, mostly *Daphnia* spp., causing them to live in deeper and colder waters (Pangle & Peacor, 2006; Pangle, Peacor & Johannsson, 2007). The altered spatial distribution of *Daphnia* induced by a NIS could lead to greater or lower exposure to, and transmission of, parasites (Hall *et al.*, 2005).

Trait-mediated indirect effects also provide alternative hypotheses for the observed pattern in New Zealand of decreasing endemic parasite infection in native freshwater fish with increasing brown trout abundance (see above). Rather than 'parasite dilution', trout may be altering either the behaviour of native fish (on which they prey) or the freshwater environment, in ways that reduce parasite transmission. Behavioural impacts of brown trout on other native species in New Zealand have already been documented; several mayfly species have changed their diel activity patterns and now spend little time grazing on algae during the day in systems where trout have been introduced (McIntosh & Townsend, 1994, 1995). As mayflies and other aquatic insects serve as intermediate hosts for the trematode *Phyllodistomum magnificum* Cribb, which is trophically transmitted to eels, *Anguilla* spp. (Hine, 1978; Cribb, 1987), their altered behaviour in the presence of introduced trout may have consequences for diseases in eels.

Trout invasion highlights another potentially important functional effect of NIS – induced habitat shift – which could interact with other stressors to enhance disease. Populations of non-migratory *Galaxias* fish in New Zealand streams have declined as a result of trout invasion, habitat modification and pollution (McDowall, 2006), but trematode infection may also play a role. Juvenile fish can suffer mortality and severe spinal malformations if infected by free-swimming infective stages (cercariae) of trematodes released by snail intermediate hosts (Kelly *et al.*, 2010). Displaced by trout, the *Galaxias* tend to persist in poor-quality refuges of low flow and higher temperature (Allibone, 2000; Leprieur *et al.*, 2006), marginal habitats that may increase trematode infections by concentrating cercariae, snail intermediate hosts and fish (e.g. Mitchell *et al.*, 2000), with higher temperatures probably promoting snail shedding of cercariae (see Poulin, 2006). Allibone (2000) reported that low flows and high temperatures in streams subjected to water abstraction were associated with high snail densities and a large percentage of malformed adult *Galaxias depressiceps* (McDowall & Wallis, 1996). This example indicates that NIS-driven disease emergence could act in concert with other drivers of environmental change.

There are several additional mechanisms by which NIS could cause disease emergence. First, they could accumulate and biomagnify endemic diseases within the food chain. The zebra mussel invasion of the Great Lakes, and the re-emergence of Type E *Clostridium botulinum* associated with large-scale fish and waterfowl die-offs, provides a good example. Type E botulism is transmitted through diet and proliferates in anaerobic, nutrient-rich sediments. The enormous filtering capacity of zebra mussels has been linked with accumulation of *C. botulinum* and possibly its toxin. It is speculated that waterfowl accumulates *C. botulinum* by preying on invasive round gobies *Neogobius melanostomus* (Pallas), the mussel's main predator (Holeck *et al.*, 2004; Getchell & Bowser, 2006). Second, NIS may enhance disease by altering habitat physicochemistry, illustrated again by zebra mussels. By providing additional nutrients in deposited faeces and pseudofaeces, increasing sediment anoxia because of decomposition of dead mussels and waste and enhancing growth of near-shore benthic algae, mussels create conditions suited to the proliferation of *C. botulinum* (Perez-Fuentetaja *et al.*, 2006; Riley *et al.*, 2008). Because many freshwater NIS, such as crayfish, carp, mitten crabs and beavers, are

ecosystem engineers that cause profound changes in physical habitat, it is probable that the scope for such functional impacts on disease emergence is wide. Indeed, such physicochemical alteration highlights an additional mechanism by which NIS could alter disease, that is, by modifying the strength of environmental stressors of the immune system of native hosts. It is of interest that recent deepwater hypoxic events in parts of the Great Lake Basin have been attributed to zebra mussel invasion (e.g. Kelly, Herborg & MacIsaac, 2009a). Given that immune mechanisms involved in the clearance of pathogens can be highly sensitive to hypoxia (Macey *et al.*, 2008), such hypoxic conditions might lead to the emergence of diseases.

The evidence that functional changes (e.g. microhabitat use, trophic interactions) in native freshwater animals are induced by NIS is very convincing. However, despite the obvious ways in which these changes can influence both the exposure of native animals to parasites and their resistance to infection, there have been no attempts to quantify the indirect impacts of NIS on disease dynamics. This possible link between biological invasion and emerging diseases remains unexplored empirically, possibly because it may appear less important than other causal pathways. Yet, it may be relevant to a large proportion of invasions, and quantifying the strength of these indirect effects of NIS on endemic diseases should be a top priority.

Importance of non-indigenous species as hosts of endemic parasites

Isolated case studies suggest that NIS can indirectly cause the emergence of endemic diseases by either changing the abundance of native species serving as hosts of parasites, or increasing their exposure or susceptibility to infection. There is no global dataset allowing an evaluation of how frequently disease emergence follows biological invasion. However, surveys and checklists of parasites in freshwater animals, especially fish, are available from several parts of the world, providing the basic data necessary to assess how often the conditions for parasite spillback are met following an invasion. Here, we use compilations of host–parasite associations to determine (i) how many endemic parasite species typically exploit an invasive fish species and (ii) what endemic parasite taxa most commonly use an introduced fish species as an alternative host. These are not definitive analyses, but instead a preliminary demonstration of the very real potential for parasite spillback in freshwater habitats.

Data on endemic parasites of introduced freshwater fish species were obtained from checklists for four geographical areas: Canada (Margolis & Arthur, 1979; McDonald & Margolis, 1995), Mexico (Salgado-Maldonado, 2006), the Czech and Slovak Republics (Moravec, 2001) and New Zealand (Hine, Jones & Diggles, 2000). The parasites considered include only monogeneans, trematodes, cestodes, nematodes, acanthocephalans and copepods, other groups being excluded because they were either ignored in most surveys or were rarely found. In addition, we consider only parasites that are either (i) confirmed as species endemic to the area that only started exploiting the introduced fish following its arrival or (ii) suspected by the authors of the checklists of being endemic to the area though confirmation is lacking. All parasite species known to have been introduced, either with the introduced fish on which they are found or via a different route, were excluded. Finally, we only included introduced fish species for which at least one endemic parasite has been reported. Introduced fish with no known endemic parasites in their new

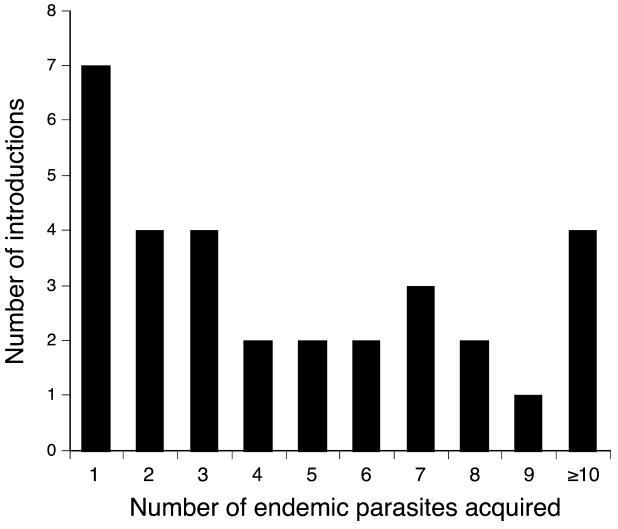

Fig. 2 Frequency distribution of the number of endemic parasite species recorded per species of introduced freshwater fish (n = 31), pooled across four geographical areas. The data include both parasites that are confirmed as endemic species and those that are suspected of being endemic. See text for further details.

area may be 'false negatives': further sampling specifically targeted at finding parasites might reveal that they are indeed used as hosts by endemic parasites.

Well over half of the estimated 50 introduced fish species in the four geographic areas have been found to harbour endemic parasites. Overall, 31 fish introductions, involving mainly Cichlidae, Cyprinidae or Salmonidae, had acquired at least one endemic parasite species, each NIS having acquired between 1 and 15 endemic parasites (mean ± SE: 4.9 ± 0.7 parasite species; see Fig. 2). If only parasites whose endemic status has been independently confirmed are considered, these numbers are reduced by about half (2.3 ± 0.4 parasite species, range 1–9). Overall, the 31 fish introductions led to 151 cases of an endemic parasite using a NIS as an alternative host, with almost half of these cases (i.e. 71) involving parasites confirmed to be endemic. The majority of these were nematodes (23 cases), trematodes (20) or acanthocephalans (14), suggesting that parasites with complex life cycles transmitted via the food chain are more likely to be involved in spillback than those with direct transmission like monogeneans and copepods.

This brief survey suggests that the potential for the amplification of an endemic disease following the use of NIS as alternative hosts, or spillback, is truly considerable for fish parasites. Of course, the widespread use of introduced hosts by endemic parasites does not necessarily imply that spillback (or, for that matter, disease dilution) will ensue; it does, however, set the stage for NIS to alter local parasite dynamics in ways that could lead to disease emergence.

Predicting the risk of emerging endemic disease posed by NIS

The extent of biological invasions is creating major regulatory challenges for government agencies (Hulme *et al.*, 2009). Ecologists need to devise predictive tools for legislators to target prevention and control efforts towards those invasive species most likely to impact ecosystems and the economy, including those probable to initiate emerging diseases. In recent years, ecological risk assessment has become widely used in environmental management and impact assessment (Lackey, 1997; Claassen, 1999). Risk assessment has been used specifically to try to predict which taxa are most likely to successfully invade new geographical areas (Kolar & Lodge, 2002; Landis, 2004). In addition, this approach has previously been applied to the management of diseases in aquatic ecosystems, identifying parasites most likely to spread and have economic impacts (Peeler *et al.*, 2007). Although a lack of data for some critical parameters can limit its full potential, there is little doubt that risk analysis has improved decision making in aquatic animal health management.

In the joint context of biological invasions and infectious diseases, ecological risk assessment could be used to evaluate the likelihood of emerging infectious diseases caused by endemic parasites following the introduction of particular NIS, and to inform decision making and the prioritisation of efforts to eradicate or control invasive species. To achieve this, we would need to build predictive statistical models based on existing data that assign to any given NIS a probability that it will trigger the emergence of an endemic disease; it would then be for decision makers to determine whether the risk is above a threshold (which takes into account not only ecological but also economic factors) for implementation of management actions. Risk (i.e. the probability of an endemic emerging disease following an introduction) can be determined as a function of the number of NIS introduced, using modified logistic regression techniques (see Bender, 1999), or more powerful statistical tools that take into account missing values, uncertainty, nonlinear relationships and higher-order interactions between variables (De'ath, 2002; Elith, Leathwick & Hastie, 2008). Other independent variables probably to matter in such a predictive model include (i) the characteristics of the introduced species, including its phylogenetic affinities and its basic ecological and life history traits, (ii) the characteristics of the native host fauna, such as its diversity and the phylogenetic distance between its component species and the invader and (iii) the properties of the local parasite fauna, including its diversity and its phylogenetic distance to the parasites of the invader in its area of origin. For many biological invasions, data are available for several if not most of these variables. Equipped with models based on these data, it would be relatively easy to quantify the risk of disease emergence associated with a particular NIS after its initial detection in a new habitat. There is an urgent need for this kind of forecasting tool to ensure that the necessary measures to either eliminate the risk of disease or mitigate its consequences are taken before, rather than after, its emergence.

Conclusions

It is clear from our review that empirical evidence supporting NIS as a cause of endemic disease emergence in freshwaters is scant. One of the difficulties in identifying the role of NIS is that parasitism and disease may be driven by multiple stressors, such as climate change, habitat alteration and pollution (Didham *et al*., 2007; Lafferty, 2009), problems that are particularly relevant to freshwaters. However, freshwater systems, when compared to terrestrial habitats, may offer certain advantages in the study of disease emergence. For example, because some NIS are patchily distributed, the often discrete nature of river and lake systems can provide natural spatially replicated experiments that allow a comparison of disease patterns in native hosts in the presence versus absence of an NIS. Lakes and ponds are also more amenable to NIS removal experiments, which would allow temporal assessment of disease dynamics. Such experimental manipulations are vital for demonstrating links between biological invasions and disease emergence. Although many of the direct and indirect interactions leading to disease emergence postulated here have good supporting evidence, conclusive proof is generally still lacking. The challenge is twofold. First, modelling work is required, supported by empirical data from the field and experimental work in the laboratory, to interrogate the complex interactions among parasite fitness and host susceptibility of native and introduced fish and to identify the sets of circumstances under which spillback can be expected to occur. Second, there is a need for well-designed natural field experiments that contrast endemic parasitism of native fish in study sites (e.g. streams or lakes) lacking or containing NIS.

Acknowledgment

Our research is supported by a grant from the Marsden Fund (Royal Society of New Zealand).

References

Achinelly M.F., Micielli M.V. & Garcia J.J. (2003) Pre-parasitic juveniles of *Strelkovimermis spiculatus* Poinar & Camino, 1986 (Nematoda: Mermithidae) predated upon by freshwater copepods (Crustacea: Copepoda). *Nematology*, **5**, 885–888.

Allibone R.M. (2000) Water abstraction impacts on non-migratory galaxiids of Otago streams. *Science for Conservation*, **147**, 1–45.

Apanius V. (1998) Stress and immune defense. *Advances in the Study of Behavior*, **27**, 133–153.

Bender R. (1999) Quantitative risk assessment in epidemiological studies investigating threshold effects. *Biometrical Journal*, **41**, 305–319.

Boersma M., Spaak P. & De Meester L. (1998) Predator-mediated plasticity in morphology, life history, and behavior of *Daphnia*: the uncoupling of responses. *American Naturalist*, **152**, 237–248.

Botts P.S., Patterson B.A. & Schloesser D.W. (1996) Zebra mussel effects on benthic invertebrates: physical or biotic? *Journal of the North American Benthological Society*, **15**, 179–184.

Brook B.W., Sodhi N.S. & Bradshaw C.J.A. (2008) Synergies among extinction drivers under global change. *Trends in Ecology and Evolution*, **23**, 453–460.

Christensen N.O. (1979) *Schistosoma mansoni*: interference with cercarial host-finding by various aquatic organisms. *Journal of Helminthology*, **53**, 7–14.

Claassen M. (1999) Ecological risk assessment as a framework for environmental impact assessments. *Water Science and Technology*, **39**, 151–154.

Clavero M. & Garcia-Berthou E. (2005) Invasive species are a leading cause of animal extinctions. *Trends in Ecology and Evolution*, **20**, 110.

Cohen A.N. & Carlton J.T. (1998) Accelerating invasion rate in a highly invaded estuary. *Science*, **279**, 555–558.

Cribb T.H. (1987) A new species of *Phyllodistomum* (Digenea: Gorgoderidae) from Australian and New Zealand freshwater fishes with notes on the taxonomy of *Phyllodistomum* Braun, 1899. *Journal of Natural History*, **21**, 1525–1538.

Daszak P., Cunningham A.A. & Hyatt A.D. (2000) Emerging infectious diseases of wildlife- Threats to biodiversity and Human health. *Science*, **287**, 443–449.

De'ath G. (2002) Multivariate regression trees: a new technique for modeling species-environment relationships. *Ecology*, **83**, 1105–1117.

Didham R.K., Tylianakis J.M., Gemmell N.J., Rand T.A. & Ewers R.M. (2007) Interactive effects of habitat modification and species invasion on native species decline. *Trends in Ecology and Evolution*, **22**, 489–496.

Dix T.G. (1968) Helminth parasites of brown trout (*Salmo trutta* L.) in Canterbury, New Zealand. *New Zealand Journal of Marine and Freshwater Research*, **2**, 363–374.

Drake J.M. & Lodge D.M. (2004) Global hot spots of biological invasions: evaluating options for ballast-water management. *Proceedings of the Royal Society of London B*, **271**, 575–580.

Dunn A.M. (2009) Parasites and biological invasions. *Advances in Parasitology*, **68**, 161–184.

Elith J., Leathwick J.R. & Hastie T. (2008) A working guide to boosted regression trees. *Journal of Animal Ecology*, **77**, 802–813.

Faust C., Stallknecht D., Swayne D. & Brown J. (2009) Filter-feeding bivalves can remove avian influenza viruses from water and reduce infectivity. *Proceedings of the Royal Society of London B*, **276**, 3727–3735.

Fèvre E.M., Bronsvoort B.M., Hamilton K.A. & Cleaveland S. (2006) Animal movements and the spread of infectious diseases. *Trends in Microbiology*, **14**, 125–131.

Genner M.J., Michel E. & Todd J.A. (2008) Resistance of an invasive gastropod to an indigenous trematode parasite in Lake Malawi. *Biological Invasions*, **10**, 41–49.

Getchell R.G. & Bowser P.R. (2006) Ecology of Type E botulism within *Dreissena* mussel beds. *Aquatic Invaders*, **17**, 1–8.

Grosholz E.D. (2002) Ecological and evolutionary consequences of coastal invasions. *Trends in Ecology and Evolution*, **17**, 22–27.

Hall S.R., Duffy M.A., Tessier A.J. & Caceres C.E. (2005) Spatial heterogeneity of daphniid parasitism within lakes. *Oecologia*, **143**, 635–644.

Harris J. & Bird D.J. (2000) Modulation of the fish immune system by hormones. *Veterinary Immunology and Immunopathology*, **77**, 163–176.

Harvell C.D., Mitchell C.E., Ward J.R., Altizer S., Dobson A.P., Ostfeld R.S. & Samuel M.D. (2002) Climate warming and disease risks for terrestrial and marine biota. *Science*, **296**, 2158–2162.

Herrmann K.K. & Sorensen R.E. (2009) Seasonal dynamics of two mortality-related trematodes using an introduced snail. *Journal of Parasitology*, **95**, 823–828.

Hine P.M. (1978) Distribution of some parasites of freshwater eels in New Zealand. *New Zealand Journal of Marine and Freshwater Research*, **12**, 179–187.

Hine P.M., Jones J.B. & Diggles B.K. (2000) *A Checklist of Parasites of New Zealand Fishes, Including Previously Unpublished Records*. National Institute of Water and Atmospheric Research Technical Report no. 75, Wellington.

Hoeve J. & Scott M.E. (1988) Ecological studies on *Cyathocotyle bushiensis* (Digenea) and *Sphaeridiotrema globulus* (Digenea), possible pathogens of dabbling ducks in southern Quebec. *Journal of Wildlife Diseases*, **24**, 407–421.

Holdich D.M. & Reeve I.D. (1991) Distribution of freshwater crayfish in the British Isles, with particular reference to crayfish plague, alien introductions and water quality. *Aquatic Conservation: Marine and Freshwater Ecosystems*, **1**, 139–158.

Holeck K.T., Mills E.L., MacIsaac H.J., Dochoda M.R., Colautti R.I. & Ricciardi A. (2004) Bridging troubled waters: biological invasions, transoceanic shipping, and the Laurentian Great Lakes. *BioScience*, **54**, 919–929.

Hulme P.E., Pyšek P., Nentwig W. & Vilà M. (2009) Will threat of biological invasions unite the European Union? *Science*, **324**, 40–41.

Johansson F. & Andersson J. (2009) Scared fish get lazy, and lazy fish get fat. *Journal of Animal Ecology*, **78**, 772–777.

Kagawa N. & Mugiya Y. (2000) Exposure of goldfish (*Carassius auratus*) to bluegills (*Lepomis macrochirus*) enhances expression of stress protein 70 mRNA in the brains and increases plasma cortisol levels. *Zoological Science*, **17**, 1061–1066.

Keesing F., Holt R.D. & Ostfeld R.S. (2006) Effects of species diversity on disease risk. *Ecology Letters*, **9**, 485–498.

Kelly D.W., Herborg L.-M. & MacIsaac H.J. (2009a) Ecosystem changes associated with *Dreissena* invasions: recent developments and emerging issues. In: *The Zebra Mussel in Europe* (Eds G. van der Velde , S. Rajagopol & A. bij de Vaate), pp. 221–232. Backhuys Publishers, Leiden.

Kelly D.W., Paterson R.A., Townsend C.R., Poulin R. & Tompkins D.M. (2009b) Parasite spillback: a neglected concept in invasion ecology? *Ecology*, **90**, 2047–2056.

Kelly D.W., Paterson R.A., Townsend C.R., Poulin R. & Tompkins D.M. (2009c) Has the introduction of brown trout altered disease patterns in native New Zealand fish? *Freshwater Biology*, **54**, 1805–1818.

Kelly D.W., Thomas H., Thieltges D.W., Poulin R. & Tompkins D.M. (2010) Trematode infection causes malformations and population effects in a declining New Zealand fish. *Journal of Animal Ecology*, **79**, 445–452.

Kennedy C.R., Hartvigsen R. & Halvorsen O. (1991) The importance of fish stocking in the dissemination of parasites throughout a group of reservoirs. *Journal of Fish Biology*, **38**, 541–552.

Kolar C.S. & Lodge D.M. (2002) Ecological predictions and risk assessment for alien fishes in North America. *Science*, **298**, 1233–1236.

Kopp K. & Jokela J. (2007) Resistant invaders can convey benefits to native species. *Oikos*, **116**, 295–301.

Kozubikova E., Filipova L., Kozak P., Duris Z., Martin M.P., Dieguez-Uribeondo J., Oidtmann B. & Petrusek A. (2009) Prevalence of the crayfish plague pathogen *Aphanomyces astaci* in invasive American crayfishes in the Czech Republic. *Conservation Biology*, **23**, 1204–1213.

Kreutzenberger K., Leprieur F. & Brosse S. (2008) The influence of the invasive black bullhead *Ameiurus melas* on the predatory efficiency of pike *Esox lucius* L. *Journal of Fish Biology*, **73**, 196–205.

Lackey R.T. (1997) Ecological risk assessment: use, abuse, and alternatives. *Environmental Management*, **21**, 808–812.

Lafferty K.D. (2009) The ecology of climate change and infectious diseases. *Ecology*, **90**, 888–900.

Landis W.G. (2004) Ecological risk assessment conceptual model formulation for nonindigenous species. *Risk Analysis*, **24**, 847–858.

Lass S. & Spaak P. (2003) Chemically induced anti-predator defences in plankton: a review. *Hydrobiologia*, **491**, 221–239.

Leprieur F., Hickey M.A., Arbuckle C.J., Closs G.P., Brosse S. & Townsend C.R. (2006) Hydrological disturbance benefits a native fish at the expense of an exotic fish. *Journal of Applied Ecology*, **43**, 930–939.

Macey B.M., Rathburn C.K., Thibodeaux L.K., Burnett L.E. & Burnett K.G. (2008) Clearance of *Vibrio campbellii* injected into the hemolymph of *Callinectes sapidus*, the Atlantic blue crab: the effects of prior exposure to bacteria and environmental hypoxia. *Fish and Shellfish Immunology*, **25**, 718–730.

MacIsaac H.J., Sprules W.G., Johannsson O.E. & Leach J.H. (1992) Filtering impacts of larval and sessile zebra mussels (*Dreissena polymorpha*) in western Lake Erie. *Oecologia*, **92**, 30–39.

MacNeil C., Dick J.T.A., Hatcher M.J., Terry R.S., Smith J.E. & Dunn A.M. (2003) Parasite-mediated predation between native and invasive amphipods. *Proceedings of the Royal Society of London B*, **270**, 1309–1314.

Marcogliese D.J. (2001) Implications of climate change for parasitism of animals in the aquatic environment. *Canadian Journal of Zoology*, **79**, 1331–1352.

Margolis L. & Arthur J.R. (1979) *Synopsis of the Parasites of Fishes of Canada*. Bulletin of the Fisheries Research Board of Canada no. 199, Department of Fisheries and Oceans Canada, Ottawa.

Mastitsky S.E. & Veres J.K. (2010) Field evidence for a parasite spillback caused by exotic mollusc *Dreissena polymorpha* in an invaded lake. *Parasitology Research*, **106**, 667–675.

Mayer C.M., Van De Valk A.J., Forney J.L., Rudstam L.G. & Mills E.L. (2000) Response of yellow perch (*Perca flavescens*) in Oneida Lake, New York, to the establishment of zebra mussels (*Dreissena polymorpha*). *Canadian Journal of Fisheries and Aquatic Sciences*, **57**, 742–754.

McDonald T.E. & Margolis L. (1995) *Synopsis of the Parasites of Fishes of Canada: Supplement (1978–1993)*. Canadian Special Publication of Fisheries and Aquatic Sciences no. 122, National Research Council of Canada, Ottawa.

McDowall R.M. (2006) Crying wolf, crying foul, or crying shame: alien salmonids and a biodiversity crisis in the southern cool-temperate galaxioid fishes? *Reviews in Fish Biology and Fisheries*, **16**, 233–242.

McIntosh A.R. & Townsend C.R. (1994) Interpopulation variation in mayfly antipredator tactics: differential effects of contrasting predatory fish. *Ecology*, **75**, 2078–2090.

McIntosh A.R. & Townsend C.R. (1995) Impacts of an introduced predatory fish on mayfly grazing in New Zealand streams. *Limnology and Oceanography*, **40**, 1508–1512.

Mitchell A.J., Salmon M.J., Huffman D.G., Goodwin A.E. & Brandt T.M. (2000) Prevalence and pathogenicity of a heterophyid trematode infecting the gills of an endangered fish, the fountain darter, in two central Texas spring-fed rivers. *Journal of Aquatic Animal Health*, **12**, 283–289.

Molnar J.L., Gamboa R.L., Revenga C. & Spalding M.D. (2008) Assessing the global threat of invasive species to marine biodiversity. *Frontiers in Ecology and the Environment*, **6**, 485–492.

Moravec F. (2001) *Checklist of the Metazoan Parasites of Fishes of the Czech Republic and Slovak Republic*. Academia Publishers, Prague.

Mouritsen K.N. & Poulin R. (2002) Parasitism, climate oscillations and the structure of natural communities. *Oikos*, **97**, 462–468.

Pace M.L., Findlay S.E.G. & Fischer D. (1998) Effects of an invasive bivalve on the zooplankton community of the Hudson River. *Freshwater Biology*, **39**, 103–116.

Pangle K.L. & Peacor S.D. (2006) Non-lethal effect of the invasive predator *Bythotrephes longimanus* on *Daphnia mendotae*. *Freshwater Biology*, **51**, 1070–1078.

Pangle K.L., Peacor S.D. & Johannsson O.E. (2007) Large nonlethal effects of an invasive invertebrate predator on zooplankton population growth rate. *Ecology*, **88**, 402–412.

Peeler E.J., Murray A.G., Thebault A., Brun E., Giovaninni A. & Thrush M.A. (2007) The application of risk analysis in aquatic animal health management. *Preventive Veterinary Medicine*, **81**, 3–20.

Perez-Fuentetaja A., Clapsadl M.D., Einhouse D., Bowser P.R., Getchell R.D. & Lee W.T. (2006) Influence of limnological conditions on *Clostridium botulinum* type E presence in Eastern Lake Erie sediments (Great Lakes, USA). *Hydrobiologia*, **563**, 189–200.

Perrings C., Dehnen-Schmutz K., Touza J. & Williamson M. (2005) How to manage biological invasions under globalization. *Trends in Ecology and Evolution*, **20**, 212–215.

Pimentel D., Zuniga R. & Morrison D. (2005) Update on the environmental and economic costs associated with

alien-invasive species in the United States. *Ecological Economics*, **52**, 273–288.

Poulin R. (2006) Global warming and temperature-mediated increases in cercarial emergence in trematode parasites. *Parasitology*, **132**, 143–151.

Poulin R. & Mouillot D. (2003) Host introductions and the geography of parasite taxonomic diversity. *Journal of Biogeography*, **30**, 837–845.

Prenter J., MacNeil C., Dick J.T.A. & Dunn A.M. (2004) Roles of parasites in animal invasions. *Trends in Ecology and Evolution*, **19**, 385–390.

Raberg L., Grahn M., Hasselquist D. & Svensson E. (1998) On the adaptive significance of stress-induced immunosuppression. *Proceedings of the Royal Society of London B*, **265**, 1637–1641.

Rachowicz L.J., Hero J.M., Alford R.A., Taylor J.W., Morgan J.A.T., Vredenburg V.T., Collins J.P. & Briggs C.J. (2005) The novel and endemic pathogen hypotheses: competing explanations for the origin of emerging infectious diseases of wildlife. *Conservation Biology*, **19**, 1441–1448.

Rauque C.A., Viozzi G.P. & Semenas L.G. (2003) Component population study of *Acanthocephalus tumescens* (Acanthocephala) in fishes from Lake Moreno, Argentina. *Folia Parasitologica*, **50**, 72–78.

Riley S.C., Munkittrick K.R., Evans A.N. & Krueger C.C. (2008) Understanding the ecology of disease in Great Lakes fish populations. *Aquatic Ecosystem Health and Management*, **11**, 321–334.

Roscoe D.E. (1983) Fatal enteritis caused by *Sphaeridiotrema globulus* (Trematoda: Psilostomidae) in a Whistling Swan. *Journal of Wildlife Diseases*, **19**, 370–371.

Ruiz G.M., Rawlings T.K., Dobbs F.C., Drake L.A., Mullady T., Huq A. & Colwell R.R. (2000) Global spread of microorganisms by ships. *Nature*, **408**, 49–50.

Salgado-Maldonado G. (2006) Checklist of helminth parasites of freshwater fishes from Mexico. *Zootaxa*, **1324**, 1–357.

Scholz T. (1999) Life cycles of species of *Proteocephalus*, parasites of fishes in the Palearctic region: a review. *Journal of Helminthology*, **73**, 1–19.

Slos S. & Stoks R. (2008) Predation risk induces stress proteins and reduces antioxidant defense. *Functional Ecology*, **22**, 637–642.

Smith K.F., Acevedo-Whitehouse K. & Pedersen A.B. (2009) The role of infectious diseases in biological conservation. *Animal Conservation*, **12**, 1–12.

Strecker U. (2006) The impact of invasive fish on an endemic *Cyprinodon* species flock (Teleostei) from Laguna Chichancanab, Yucatan, Mexico. *Ecology of Freshwater Fish*, **15**, 408–418.

Taraschewski H. (2006) Hosts and parasites as aliens. *Journal of Helminthology*, **80**, 99–128.

Thieltges D.W., Jensen K.T. & Poulin R. (2008) The role of biotic factors in the transmission of free-living endohelminth stages. *Parasitology*, **135**, 407–426.

Tompkins D.M. & Poulin R. (2006) Parasites and biological invasions. In: *Biological Invasions in New Zealand* (Eds R.B. Allen & W.G. Lee), pp. 67–86. Springer, Berlin, Heidelberg.

Torchin M.E., Lafferty K.D., Dobson A.P., McKenzie V.J. & Kuris A.M. (2003) Introduced species and their missing parasites. *Nature*, **421**, 628–630.

Trussell G.C., Ewanchuk P.J. & Matassa C.M. (2006) Habitat effects on the relative importance of trait- and density-mediated indirect interactions. *Ecology Letters*, **9**, 1245–1252.

Werner E.E. & Peacor S.D. (2003) A review of trait-mediated indirect interactions in ecological communities. *Ecology*, **84**, 1083–1100.

Wilcove D.S., Rothstein D., Dubow J., Phillips A. & Losos E. (1998) Quantifying threats to imperiled species in the United States. *BioScience*, **48**, 607–615.

Williams H. & Jones A. (1994) *Parasitic Worms of Fish*. Taylor & Francis, London.

Wolinska J., Loffler A. & Spaak P. (2007) Taxon-specific reaction norms to predator cues in a hybrid *Daphnia* complex. *Freshwater Biology*, **52**, 1198–1209.

Woodley C.M. & Peterson M.S. (2003) Measuring responses to simulated predation threat using behavioral and physiological metrics: the role of aquatic vegetation. *Oecologia*, **136**, 155–160.

(Manuscript accepted 19 February 2010)