

# 5 Parasites and Biological Invasions

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## 5.1 Introduction

Parasites are increasingly cited as a major threat to wildlife conservation (Daszak et al. 2000; Cleaveland et al. 2002), being implicated in the decline and/or extinction of many high-profile species worldwide, including the thylacine (McCallum and Dobson 1995), amphibians globally (Berger et al. 1998), UK red squirrels (Tompkins et al. 2003), and Hawaiian birds (van Riper et al. 1986, 2002). In the majority of cases, declines are ascribed to “emerging infectious diseases” (EIDs) – those disease-causing agents whose geographical range, host range, or prevalence have increased in recent years. There are several (non-exclusive) causes of disease emergence in wildlife, including changes in habitat and climate, and genomic change. However, it is the translocation of invasive species which appears to be of key importance, being implicated in more than half of all wildlife EID cases recorded (Daszak et al. 2000). A recent example is the exotic parapoxvirus introduced with grey squirrels (*Sciurus carolinensis*) into the UK at the turn of the 20th century, and which has been implicated in the nationwide decline of native red squirrels (*Sciurus vulgaris*; Tompkins et al. 2003).

In this review, we explore how parasitism and species invasions interact to influence natural communities, using New Zealand case studies complemented by global examples of potential relevance. We begin by looking at exotic parasites and how they can impact the native fauna. We then turn to exotic hosts and how, by acting as reservoirs for native parasites, they can exacerbate disease problems for native wildlife. Finally, we review the parasite threats to New Zealand land birds and freshwater fish, evaluate potential future threats to these biota, and propose management recommendations to minimize these threats.

## 5.2 Taxonomic Affiliation of Emerging Infectious Diseases of Wildlife

Helminth and fungal parasites are believed unlikely to “emerge” as new infectious diseases of wildlife, whilst bacteria, protozoans and viruses are (Dobson and Foufopoulos 2001). However, this assumption may be premature due to a bias in the information available. Whereas microparasites can have highly visible effects, diseases caused by macroparasites are often more subtle, yet can still impact the host at the population level (Tompkins et al. 2002). Since such effects are harder to detect, many macroparasite EIDs may simply have yet to be quantified. For example, a survey of the helminth fauna of Hawaiian stream fish revealed three species of exotic parasites introduced by poeciliid fish (a nematode *Camallanus cotti*, a cestode *Bothriocephalus acheilognathi*, and a leech *Myzobdella lugubris*), occurring with higher prevalences and abundances than native parasites (Font 1998). These exotic parasites represent the most significant threat of parasite-induced disease in native stream fishes in Hawaii (Font and Tate 1994). At least one of these three species (*B. acheilognathi*) has recently emerged as a disease-causing agent in other fish species (causing bothriocephaliosis in the humpback chub *Gila cypha* in the USA; Brouder and Hoffnagle 1997).

One fungal pathogen has made a huge impact on native biodiversity globally. In 1998, the disease chytridiomycosis was first described from moribund and dead adult amphibians collected at sites of mass deaths in Australia and Panama from 1993 to 1998 (Berger et al. 1998). The causative agent was subsequently identified from captive frogs in the USA as the fungus *Batrachochytrium dendrobatidis* (Longcore et al. 1999). Since then, this pathogen has been linked to many ongoing population declines, and retrospective studies using museum specimens have found *B. dendrobatidis* infections in amphibians as early as 1974 in North America, 1978 in Australia, 1980 in South America, and 1982 in Central America. The pathogen has also been found in African specimens collected since 1998. Chytrid fungus infects the keratinized tissues of amphibians, including the epidermis of adults and the mouthparts of tadpoles, causing (in adults) abnormal posture, lethargy, and loss of righting reflex (Berger et al. 1998). Evidence suggests that its emergence has been driven by the anthropogenic introduction of the pathogen to new regions and host species (“pathogen pollution”; Daszak et al. 2000). For example, comparisons of ssrRNA sequences indicate that the New Zealand chytrid strain is almost identical to that initially identified in the USA but varies by up to 3 % from the Australian strain (Waldman et al. 2001). This suggests that New Zealand chytrids are recent arrivals, most likely from America via the international pet trade.

### 5.3 Determinants of Establishment Success by Introduced and Invading Parasites

A survey of the parasite communities of three species of exotic freshwater poeciliid fishes in Queensland, Australia, revealed that these introduced hosts harbour more species-poor parasite infracommunities and regional faunas than 15 ecologically similar native species (Dove 2000). This is because, while some parasites have been picked up by the exotic hosts from the native fish fauna, many traditional parasites have been lost on introduction to the new geographic region. Other surveys indicate that such losses are the general pattern being documented also in introduced molluscs, crustaceans, birds, mammals, amphibians and reptiles (Freeland 1993; Torchin et al. 2003). In addition to the number of parasite species found in exotic host populations being, on average, only half that found in native populations, introduced hosts also tend to have a lower prevalence of infection. For example, brushtail possums (*Trichosurus vulpecula*) in New Zealand have a very limited endoparasite fauna, compared with Australia (Obendorf et al. 1998; Stankiewicz et al. 1998), and the possum-specific parasites present are patchily distributed nationally (Cowan et al. 2000).

There are several reasons for this loss of parasite species. Primarily, when a host population is subsampled in such a way, the individuals involved are unlikely to support the entire parasite fauna because of the characteristic aggregation of parasites among hosts (Shaw and Dobson 1995). An extreme form of bottleneck occurs when eggs or juvenile hosts are introduced to a new environment, resulting in the loss of all parasites dependent on the adult form. Exotic salmonids in New Zealand are generally not host to any of their traditional parasites, since they were introduced as either fry or eggs (Dix 1968; Margolis and Boyce 1990). The three exceptions to this are the protozoan *Myxobolus cerebralis*, which causes “whirling disease” and is believed to have been introduced in infected bait (Boustead 1993; Champion et al. 2002), the protozoan *Ichthyophthirius multifiliis*, a cosmopolitan pathogen which causes “whitespot” and probably spread from native fish (Hine et al. 2000), and the cestode *Ligula intestinalis*, which appears not to persist but is introduced occasionally by infected birds (the definitive hosts) from Australia (Weekes and Penlington 1986).

Even if parasites are introduced with their hosts, they may still be lost. Small numbers of introduced parasites may disappear due to either stochastic effects, or the new host population initially being of insufficient size or density (below the persistence “threshold”; Swinton et al. 2002). The directly transmitted fish louse *Argulus*, a well-known and widespread parasite of freshwater fish, does not occur in New Zealand. However, when imports of exotic goldfish (*Carassius auratus*) were more frequent (now severely restricted to limit disease entry), *Argulus* did arrive from time to time but

apparently never became established in wild fish populations (Pilgrim 1967). Some parasites may be unable to persist in the new environment, due to either inappropriate abiotic conditions, the absence of other obligate components of the parasites' life cycle (such as suitable intermediate hosts or vectors), or even out-competition by a native parasite (Bauer 1991).

## 5.4 Exotic Parasite Impact on Native Fauna

Introduced parasites can directly cause the extinction of native host populations (Cleaveland et al. 2002). Since infectious agents tend to have threshold levels of host population size below which they are unable to persist ("fade-out"; Swinton et al. 2002), it is those parasites with alternative "reservoirs" available which pose the greatest threat. Indeed, most local extinctions and population crashes in threatened wildlife due to disease have been ascribed to the "spillover" of infection from alternative hosts (Daszak et al. 2000). For example, the decline of the native grey partridge (*Perdix perdix*) in the UK is partly due to the cross-species transmission of the exotic nematode *Heterakis gallinarum* from its introduced traditional host, the ring-necked pheasant (*Phasianus colchicus*; Tompkins et al. 2000). Certain native hosts may also act as "source" populations for the transmission of exotic infectious agents to "sink" populations of other native species. For example, infection trials have demonstrated that whereas chytridiomycosis is fatal for certain amphibians, others (such as the bullfrog *Rana catesbeiana*) can be infected with no apparent harmful effects (Daszak et al. 2003). Introduced parasites can also indirectly cause the extinction of native host populations by suppressing their size or resilience (Cleaveland et al. 2002).

The anthropogenic introduction of exotic bird pathogens into Hawaii is perhaps the most cited example of how exotic microparasites can play a key role in the mass decline and extinction of native fauna. It is often overlooked, however, that it was the introduction of an exotic vector, the mosquito *Culex quinquefasciatus*, which is thought to have in fact enabled the introduced pathogens to reach epizootic proportions (van Riper et al. 2002). Both avian pox (*Poxvirus avium*) and avian malaria (*Plasmodium relictum*) have likely been repeatedly introduced to the Hawaiian Islands since their discovery by Captain Cook in 1778, with the importation of domestic avian stock, but it would not have been until the introduction of their principal vector in 1827 that transmission levels were sufficient for establishment to eventually occur. Avian pox is subsequently implicated in the numerous extinctions of endemic birds occurring during the late 1800s (van Riper et al. 2002), and avian malaria in the extinctions of the early 1900s (van Riper et al. 1986). Furthermore, the distribution of *C. quinquefasciatus*, due to its role as the primary vector of these introduced pathogens, is currently a major factor limiting the

distribution of many of the remaining native bird populations across the Hawaiian Islands. This is a clear illustration of how the introduction of exotic vectors (and intermediate hosts) can often trigger greater effects in native communities than those stemming from pathogen introduction, and their subsequent distribution can determine any ongoing effects.

Specialist parasites are less successful than generalists in invading new environments, since the former are less likely to encounter new hosts which they can infect, reducing their potential impact on the native fauna. The implications of a broad host range are seen in examples such as avian malaria and avian pox in Hawaii, where two introduced pathogens with very broad ranges have affected the vast majority of the native avifauna. Likewise, the recent impact of West Nile Virus in North America can be linked to its broad host specificity – since its introduction from the Middle East in 1999, the virus had reached 42 of the 48 southern states in the USA (as well as four of Canada's 10 provinces) by the end of 2002, being found in over 70 bird species and more than 40 mosquito species (Enserink 2002). The impact of exotic infectious agents is also more severe when native species related to introduced hosts are present in the new environment. This is presumably because relatedness facilitates host switching, yet the new hosts lack the adaptive defences against the parasite of the traditional host and, thus, can suffer serious pathogenic effects (Bauer 1991).

## 5.5 Introduced Species as New Hosts for Native Parasites

An alternative disease impact of biological invasions on native fauna, somewhat overlooked in the literature, is when introduced species act as new hosts for native parasites from which potentially deleterious “spillback” of infection to native hosts could occur (Daszak et al. 2000). Whilst many parasites are lost from hosts upon introduction to a new environment, such hosts tend to acquire generalist parasites from the native fauna. For example, a total of 31 parasite species have been recorded from introduced populations of the mallard duck (*Anas platyrhynchos*), 21 of which do not occur in its native range and have evidently been acquired from other hosts worldwide (Torchin et al. 2003). Likewise, of 28 helminth parasites recorded from introduced populations of the cane toad (*Bufo marinus*), 20 do not occur in its native range (Barton 1997). Such acquisition of native parasites is also clear in the Hawaiian and Queensland freshwater fish parasite surveys discussed above. Thus, epizootics of previously undocumented parasites should not, de facto, be considered introduced species simply because they are associated with the introduction of exotic hosts. Rather, they may represent cases where formerly rare local parasites are able to colonize the introduced hosts, the likelihood of which would probably increase if the region invaded con-

tains phylogenetically and ecologically similar native host species (Torchin et al. 2003).

Native parasites can show a relatively low degree of fitness when infecting introduced hosts. Trejo (1992) demonstrated that the fitness of the acanthocephalan parasite *Pomphorhynchus patagonicus* when infecting introduced rainbow trout (*Oncorhynchus mykiss*) in Argentinian waters is far lower than when infecting the traditional host *Patagonina hatchery* (Patagonian pejerrey). Under such circumstances, there would be limited threat to native hosts. In other cases, however, introduced species have proven competent hosts for native parasites. Rauque et al. (2003) demonstrated that the fitness of the parasitic acanthocephalan *Acanthocephalus tumescens* infecting freshwater fish (again in Argentina) is greater in introduced rainbow trout than in many of the parasite's native hosts. Here, the spillback of infection from introduced hosts may prove deleterious to susceptible native species. Such effects can potentially occur whenever introduced host populations act as sources, rather than sinks, of infective stages. In their study, Rauque et al. (2003) concluded that the rainbow trout now harbours a large proportion of the adult component population of *A. tumescens*, effects on native hosts due to raised parasitemia being easily conceivable. Since the acquisition of native generalist parasites by exotic hosts tends to be the rule, rather than the exception, spillback effects have the potential to be more common in the wild than are EIDs caused by introduced parasites.

## 5.6 Context Dependence of Exotic Parasite Impact

Multiple factors, both biotic and abiotic, are known to interact with parasite emergence, invasion, and impact on native fauna. On this basis, observed disease effects are often "context dependent" (Blaustein and Kiesecker 2002). Climate change is thought to play a major role in the emergence of wildlife diseases by altering the geographic distribution of intermediate hosts and vectors (Harvell et al. 1999). For example, in Hawaii, the decline in endemic forest birds caused by avian malaria and avian pox follows an elevation gradient whereby pathogen transmission is most intense in mid-elevation forests where the mosquito vectors and endemic birds have the greatest overlap (van Riper et al. 1986, 2002). Thus, high-elevation forests currently function as refugia for several of these bird species. Landscape analysis of these refugia, however, shows that climate change is likely to drive many of the remaining endemics extinct, mainly through warmer temperatures increasing the abundance of vectors in the high-elevation forests (Benning et al. 2002). In addition to biting flies, there are also concerns that climate change will alter the distribution of many tick species (Sutherst 2001), acting as vectors for a variety of pathogens of concern to wildlife health (Randolph et al. 2002). Habitat distur-

bance can also lead to, and exacerbate the emergence and spread of infectious diseases, via alterations in intermediate host and vector distributions and dynamics (Patz et al. 2000). On certain Hawaiian islands, the abundance of mosquitoes is strongly increased by the damage caused by feral pigs increasing the number of ephemeral water pools available in which the larvae of these insects develop. Under such circumstances, the best approach available for managing the impact of malaria and pox in the avifauna is to reduce vector abundance by controlling pig numbers.

Factors which increase host susceptibility to infection can make them more prone to introduced parasites. For example, atmospheric pollutants are believed to interact with chytridiomycosis in its impact on amphibian populations (Pounds 2001; Blaustein and Kiesecker 2002). Similarly, experiments have documented how carp (*Cyprinus carpio*) exposed to cadmium are more susceptible to the protozoan parasite *Ichthyophthirius multifiliis* (Combes 2001). Pollution may also increase parasite incidence via effects on intermediate hosts (Lafferty 1997). For example, eutrophication of freshwater can increase the incidence of “swimmers itch” in lakes, caused by the trematode *Trichobilharzia*, by improving conditions for intermediate snail hosts (Beer and German 1993). In New Zealand, disturbance and pollution interacting with macroparasites may increase the susceptibility of waterfowl to infection by microparasites such as avian paramyxoviruses (causative agents of Newcastle disease), influenza, and botulism (Alley 2002).

Stress may also influence host susceptibility, via suppressive effects on immunocompetence (Buchanan 2000). The causes of stress to wildlife are multiple, including habitat alteration, degradation and reduction, anthropogenic translocation, and artificial rearing (Dobson and Foufopoulos 2001). Pathogen emergence fostered by both the increased potential for pathogen transmission and the increased susceptibility of fish hosts in crowded hatchery conditions, although well recognized by aquaculturists, is an acute problem (Kennedy 1994). Such fish-breeding activities are believed to have driven the global emergence of the protozoan *Myxobolus cerebralis* in wild fish populations (Dobson and Foufopoulos 2001). Habitat changes can also increase pathogen impacts if such changes alter or disrupt social systems. For example, both free-living and captive hihi (*Notiomystis cincta*) in New Zealand are believed to be rendered susceptible to infection to the opportunistic fungal pathogen *Aspergillus* by immunosuppression caused by intense social interaction during the breeding season (Alley et al. 1999; Alley 2002). Down-regulation of host immunocompetence can also occur when endangered species suffer reduced genetic diversity as a result of population bottlenecks (Altizer et al. 2003). Such effects may be at least partly responsible for the high incidence of avian pox (*Poxvirus avium*) clinical symptoms in black robin (*Petroica traversi*) populations in New Zealand (Tisdall and Merton 1988).



## 5.7 Disease Threat of Biological Invasions to New Zealand's Avifauna

In 1918 it was postulated that it “may be possible to connect the comparatively rapid disappearance of New Zealand native birds with the introduction of exotic protozoa through the medium of imported fauna” (Doré 1918). Myers (1923) added to this by stating “the hypothesis of an introduced avian disease which might conceivably commit the same havoc among the indigenous birds as measles among aboriginal races of man supplies perhaps the only theory which can even partially explain the wholesale disappearance of certain species from untouched areas either before weasels or stoats were introduced or before they or any other cause of sufficient magnitude had conceivably reached the area in question”. Although exotic birds in New Zealand have, as one would expect, apparently lost some parasites on introduction (e.g. the absence of *Haemoproteus* blood parasites in silvereyes (*Zosterops lateralis*) and rock pigeons (*Columba livia*); Laird 1950), there are undoubtedly some which successfully established. In his summation of the available evidence, Laird (1950) stated “a very full investigation of the haematozoa of New Zealand birds, both indigenous and exotic, will have to be made before any decision can be reached concerning the effect of these parasites on the native avian fauna”. However, 50 years on, our position regarding the blood parasites of New Zealand birds remains relatively unchanged, with surveys of sufficient size having been conducted on only a few host species (McKenna 1998). Although such surveys would be of limited use in discerning past population and species extinctions, they may inform on cases where populations are currently being impacted by the pathogenic effects of such parasites (Atkinson and van Riper 1991), or indicate regions where such pathogens do not currently occur. The surveys conducted thus far, however, do show that *Plasmodium relictum* occurs in both native and introduced bird species in New Zealand (Table 5.1). As discussed above, such sharing of parasites commonly has deleterious effects for the more susceptible hosts.

The impact of blood parasites on New Zealand birds is likely to increase in the near future, due to the ongoing introduction and range expansion of exotic mosquitoes, primary vectors of such parasites (Atkinson and van Riper 1991). Four exotic species of mosquito have established in New Zealand to date, three of which (*Culex quinquefasciatus*, *Aedes australis* and *A. notoscriptus*) are considered to have been introduced in the 19th and early 20th centuries, and one of which (*A. camptorhynchus*) has only recently established (Laird 1995). Of key concern is *C. quinquefasciatus*, the primary vector of *Plasmodium relictum* in Hawaii. Although it appears that mosquitoes native to New Zealand can also vector *P. relictum*, since avian malaria infections have been recorded in birds outside the ranges of the introduced mosquitoes



**Table 5.1** Parasitic protozoans and helminths known to infect both native and introduced birds in New Zealand (data obtained from McKenna 1998; it is unclear whether any of the parasites listed are introduced)

Parasite	Native hosts	Exotic hosts
<b>Protozoa</b>		
<i>Plasmodium relictum</i> <sup>a</sup>	Dotterel Yellow-eyed penguin Fiordland crested penguin	Song thrush Blackbird House sparrow
<b>Trematoda</b>		
<i>Dendritobilharzia pulverulenta</i>	Grey duck New Zealand shoveler New Zealand scaup Paradise shelduck	Mallard duck Black swan Mute swan
<i>Echinoparyphium recurvatum</i>	Australasian bittern Paradise shelduck New Zealand shoveler New Zealand scaup	Black swan Canada goose Domestic goose
<i>Echinostoma revolutum</i>	Paradise shelduck New Zealand shoveler	Canada goose Domestic goose Rock pigeon
<i>Notocotylus gippyensis</i>	Paradise shelduck Grey duck New Zealand shoveler	Canada goose Domestic goose
<i>Notocotylus tadornae</i> <i>Psilochasmus oxyurus</i>	Paradise shelduck New Zealand shoveler New Zealand scaup	Domestic goose Black swan
<i>Trichobilharzia</i> sp. <sup>b</sup>	Grey duck	Mallard duck
<b>Nematoda</b>		
<i>Echinuria uncinata</i> <i>Heterakis gallinarum</i>	Grey duck Morepork	Mallard duck Ring-necked pheasant Brown quail Domestic fowl Domestic turkey
<i>Porrocaecum ensicaudatum</i>	Kiwi	Blackbird Song thrush
<i>Syngamus trachea</i>	Silvereye Kea	Ring-necked pheasant Chukor partridge Domestic fowl Blackbird Starling
<i>Trichostrongylus tenuis</i>	Paradise shelduck	Brown quail Peafowl

<sup>a</sup> *Plasmodium* sp. (most likely *relictum*) has also been recorded from native grey duck, New Zealand pipit, New Zealand dotterel, and exotic skylark

<sup>b</sup> Identified as a single species unique to New Zealand by Davis (2000)

(Holder et al. 1999), the increasing distribution of *C. quinquefasciatus* is likely to enhance the incidence of avian malaria in three distinct ways.

1. Parasitemia may increase in those species already infected, such as the endangered and endemic Fiordland crested (*Eudyptes pachyrhynchus*) and yellow-eyed (*Megadyptes antipodes*) penguins (Duignan 2001), with an increasing impact on the health of individuals and populations (Graczyk et al. 1995; Reed 1997; Alley 2001).
2. *P. relictum* may increase its host range in the areas in which it is already present. For example, whereas black swans (*Cygnus atratus*) in Australia have tested 50 % positive for *Plasmodium*, the parasite has not been found in black swans in New Zealand, now a common and widespread gamebird since its introduction during the 19th century (Laird 1950). However, increasing vector densities may allow avian malaria to sustain infections in black swans in this country as well, which would then become potential reservoirs of infection to other species.
3. *P. relictum* may increase its geographic range in New Zealand, potentially reaching offshore islands where its impact may be particularly severe due to a lack of prior exposure to blood parasites (McCallum and Dobson 1995). This is likely, since the indigenous portion of the New Zealand avifauna which has been examined for hematozoa appears exceptionally free of these parasites, and no indigenous species of hematozoa have yet been described from New Zealand birds (Laird 1950; McKenna 1998).

Increases in the distribution and/or abundance of other mosquitoes in New Zealand may also lead to increases in the prevalence of other infections such as avian pox (Holder et al. 1999). Furthermore, the threat of mosquitoes carrying exotic parasites being either transported or blown to New Zealand is ever present (Laird et al. 1994). The spread of such infectious agents, and incursions by new mosquito species, could be exacerbated by even moderate increases in average annual temperatures due to global warming (Liehne 1988). Similar considerations also apply to the introduction and spread of exotic pathogens by ticks (Sutherst 2001; Heath 2002; Randolph et al. 2002).

There may be other parasite impacts on native species, caused by biological invasions and which are going unnoticed. This is illustrated in Table 5.1, which lists all known instances of native and introduced birds in New Zealand sharing the same protozoan or helminth parasite species. Thus, the presence in New Zealand of alternative hosts for these parasites could potentially be raising parasitemia in native populations. For example, both introduced gallinaceous birds (pheasant, partridge, quail, fowl, turkey) and waterfowl may be exacerbating disease problems for native species through a range of shared parasites.

## 5.8 Disease Threat of Biological Invasions to New Zealand Freshwater Fish

Few parasites have been associated with the introduction of exotic freshwater fish to New Zealand (although some have come in by other routes – see above). Perhaps the likeliest candidates are the three monogeneans *Dactylogyrus ctenopharyngodonis*, *Gyrodactylus ctenopharyngodontis*, and *Gyrodactylus* sp. Monogeneans transmit horizontally and directly, fish to fish, making them one of the most common groups to be introduced with movements of host fish. Since they are the only three monogenean parasites reported in New Zealand, and two of them have been found infecting only introduced grass carp (*Ctenopharyngodon idella*), they have almost certainly been introduced into New Zealand on exotic cyprinid hosts (Hine et al. 2000).

As discussed above, the lack of traditional parasites on exotic salmonids in New Zealand is primarily due to their introduction as either fry or eggs, excluding parasite species dependent on adult hosts (Dix 1968; Margolis and Boyce 1990). These hosts have evidently acquired new parasite assemblages from the native fish fauna (McDowall 2000; Poulin and Mouillot 2003). Of particular interest is the copepod *Paenodes nemaformis* which, although occurring only in New Zealand, has been recorded infecting only introduced chinook salmon (*Oncorhynchus tshawytscha*) and brown trout (*Salmo trutta*). One suggestion is that this copepod was once a parasite of the now extinct grayling *Prototroctes oxyrhynchus* (McDowall 1990). The only documented harmful effect of parasites associated with introduced salmonids is that of the protozoan causing “whirling disease” (and then, only to the salmonids themselves). However, since the parasites acquired by salmonids in New Zealand waters have (as theory would predict) tended to be highly generalist, there is potential for these new hosts to be impacting native fish via the spillback of infection. This is illustrated in Table 5.2, which lists all known instances of native and introduced freshwater fishes in New Zealand sharing the same protozoan, helminth, or copepod parasite species. The native fishes at greatest risk from such effects are likely to be the galaxiids, including the koaro (*Galaxias brevipinnis*) and the inanga (*G. maculatus*), which share many parasites with the salmonids, and are the most endangered of the indigenous freshwater species (Champion et al. 2002). Such considerations may help explain why salmonids have displaced galaxiids from certain freshwater bodies in New Zealand but not from others (Townsend 2003).

Introduced non-salmonids, such as the cyprinid grass carp (*Ctenopharyngodon idella*) and goldfish (*Carassius auratus*), are also host to parasites shared with native species (Table 5.2). Thus, they also have the potential to drive deleterious disease effects in native fish populations. There are also concerns that the further spread of introduced cyprinid fish within New Zealand

**Table 5.2** Parasitic protozoans, helminths, and copepods known to infect both native and introduced freshwater fish in New Zealand (data obtained from Hine et al. 2000; only the cestode *Ligula intestinalis* has been introduced)

Parasite	Native hosts	Exotic hosts
<b>Protozoa</b>		
<i>Ichthyophthirius multifiliis</i>	Short-finned eel Long-finned eel Common bully Koaro	Grass carp Rainbow trout Chinook salmon
<b>Digenea</b>		
<i>Coitocaecum parvum</i>	Short-finned eel Long-finned eel Koaro Inanga Upland bully Common bully Giant bully Blue-gilled bully Red-finned bully Smelt	Goldfish Brown trout
<i>Telogaster opisthorchis</i>	Short-finned eel Long-finned eel Koaro Dwarf galaxias Banded kokopu Inanga Short-jawed kokopu Common river galaxias Upland bully Common bully Blue-gilled bully Red-finned bully Brown mudfish Smelt	Brown trout
<i>Tubulovesicula angusticauda</i>	Short-finned eel Long-finned eel	Chinook salmon
<b>Cestoda</b>		
<i>Ligula intestinalis</i>	Common bully	Rainbow trout
<b>Nematoda</b>		
<i>Eustrongylides ignotus</i>	Short-finned eel Long-finned eel Inanga Upland bully Giant bully	Rainbow salmon Sockeye salmon Brown trout Tench

**Table 5.2** (*Continued*)

Parasite	Native hosts	Exotic hosts
<i>Hedruris spinigera</i>	Short-finned eel Long-finned eel Inanga Cran's bully Brown mudfish Smelt	Chinook salmon Red-finned perch Brown trout
<b>Copepoda</b> <i>Abergasilus amplexus</i>	Short-finned eel Long-finned eel Inanga Smelt	Goldfish Red-finned perch

may lead to the persistence and range expansion of the introduced cestode *Ligula intestinalis*, for which cyprinids tend to be more typical hosts (McDowall 1990). Potential consequences of this would be the introduction of this generalist parasite to many more native fishes, and an increased impact on the native common bully (*Gobiomorphus cotidianus*), from which infection has already been recorded (Weekes and Penlington 1986). In a similar vein, there is the constant threat that the New Zealand fish fauna, which has largely escaped the disease and parasite problems which have plagued some fish species in other countries, would be highly vulnerable if such organisms were introduced (Champion et al. 2002). As with the threats to the endemic avifauna, habitat degradation and climate change could potentially increase the threat of successful introduction and range expansion of such parasites.

## 5.9 Conclusions

The points highlighted above, regarding the parasite threat from biological introductions, are also applicable to diseases of other biota (notably the chyrid fungus threat to New Zealand's endemic frogs). A common thread is that anthropogenic factors are almost always involved, either as direct causes of parasite emergence or as interacting factors. One relevant example is how the poeciliid fish species introduced to Hawai'i to prey on mosquito larvae, with the goal of reducing the incidence of the introduced mosquito-borne pathogens *Plasmodium relictum* and *Poxvirus avium* in native birds, are now themselves implicated as a major threat to the native Hawaiian freshwater fish fauna via the parasites which were introduced with them (Font and Tate

1994). This illustrates how human introduction of animals to new geographic regions is almost always accompanied by unforeseen deleterious effects on native communities. One would thus hope that the utmost is done to prevent such introductions (either accidental or deliberate) in the future. Recommendations specific to preventing the accidental introduction of exotic pathogens (or vectors) include strict border control biosecurity regimes, and controlling human traffic to remote regions and populations of endangered species (Jackson et al. 2000; Duignan 2001; Alley 2002). In addition, separation should be maintained between captive or domestic and wild animal populations at all time, since domestics commonly act as sources of either novel parasite introduction or increased levels of infection by endemic parasites (Cleveland et al. 2002). Furthermore, contact between closely related species (e.g. in hatcheries where multiple fish species are reared) should also be minimized, since such contact increases the potential for the cross-species transfer of infectious agents (Dobson and Foufopoulos 2001).

Safeguards should be put in place whenever it is necessary to move animals among wild populations, or from captivity into the wild (as is common in endangered species management), since disease problems can also occur under such circumstances. For example, the mass mortalities of the sardine *Sardinops sagax* which occurred throughout its range in Australia during the 1990s were caused by an exotic pathogen introduced with imported fish of the same species (Gaughan 2002). Thus, as stated by many authors (e.g. Jakob-Hoff 2001; Alley 2002), health screening prior to any translocation of animals between populations should be considered a necessity. Also, three important factors to consider when translocating animals into new areas are (1) whether hosts (of the same or other species) already present in the area may be deleteriously affected by parasites which may be introduced, (2) whether parasites present in the native fauna in the area may deleteriously affect the introduced animals, and (3) whether the introduced animals may act as a new biological reservoir for native parasites, from which infection may spill back into existing wild populations.

No matter how successful management regimes are, the increasing level of global movement means that there will undoubtedly always be instances when exotic pathogens or vectors are introduced or invade new geographic regions, host species, or host populations (Daszak et al. 2003). Quantifying the probability of such events is the realm of risk assessment (e.g. Jakob-Hoff 2001). However, certain steps can be taken to ensure that native wildlife populations are more resistant to such incursions if and when they do occur. For example, since habitat reduction and alteration can increase both parasite transmission between host individuals and host susceptibility to infection, management to minimize such effects should be a top priority. Furthermore, although habitat fragmentation may be beneficial to native species faced with exotic parasites (if it acts to limit the spread of infection), it is also more likely to exacerbate disease problems through effects such as crowding and habitat

degradation (McCallum and Dobson 2002). Likewise, pollution has the potential to interact deleteriously with introduced parasites. Thus, unless there is a good reason not to, both the fragmentation and the pollution of native habitat should be minimised to reduce potential host susceptibility to infection, safeguarding against the eventuality that exotic parasites or vectors invade or are inadvertently introduced into new regions.

This chapter has highlighted two broad gaps in our knowledge relevant to the disease effects of biological invasions in New Zealand. First, we still do not have a complete picture of the existing parasite fauna of many native species. Second, there has been no real consideration of the role which already introduced hosts may be playing as biological reservoirs for native parasites. As is often the case in the study of wildlife disease, more fundamental research is required to address these gaps.

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