CONCEPTS & SYNTHESIS
EMPHASIZING NEW IDEAS TO STIMULATE RESEARCH IN ECOLOGY

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Parasite spillback: A neglected concept in invasion ecology?

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Abstract. While there is good evidence linking animal introductions to impacts on native communities via disease emergence, our understanding of how such impacts occur is incomplete. Invasion ecologists have focused on the disease risks to native communities through "spillover" of infectious agents introduced with nonindigenous hosts, while overlooking a potentially more common mechanism of impact, that of "parasite spillback." We hypothesize that parasite spillback could occur when a nonindigenous species is a competent host for a native parasite, with the presence of the additional host increasing disease impacts in native species. Despite its lack of formalization in all recent reviews of the role of parasites in species introductions, aspects of the invasion process actually favor parasite spillback over spillover. We specifically review the animal-parasite literature and show that native species (arthropods, parasitoids, protozoa, and helminths) account for 67% of the parasite fauna of nonindigenous animals from a range of taxonomic groups. We show that nonindigenous species can be highly competent hosts for such parasites and provide evidence that infection by native parasites does spillback from nonindigenous species to native host species, with effects at both the host individual and population scale. We conclude by calling for greater recognition of parasite spillback as a potential threat to native species, discuss possible reasons for its neglect by invasion ecologists, and identify future research directions.

Key words: enemy release; infectious disease; invasion ecology; nonindigenous species; parasite; parasite-spillback hypothesis; spillover of infectious agents.

Introduction

The introduction and spread of nonindigenous species (NIS) threatens native biodiversity and the ecosystem function of terrestrial, marine, and freshwater communities globally (Jenkins 2003, Clavero and Garcia-Berthou 2005). While predation and competition have traditionally been considered the key mechanisms underlying NIS impacts (D'Antonio and Kark 2002, Sax et al. 2002), there is now also good evidence for disease impacts (Daszak et al. 2000, Cleaveland et al. 2002, Ladeau et al. 2007). In particular, many studies have documented how infectious agents introduced with NIS can affect native communities, especially when introduced hosts act as reservoirs from which infection can "spill over" to native species. While spillover has received considerable attention (Cleaveland et al. 2002, Prenter et al. 2004), another mechanism of potential importance in NIS-driven disease impacts has not been formalized in invasion ecology. In addition to introducing nonindigenous agents, NIS may also act as new hosts for native infectious agents, from which infection may "spill back" to native fauna (Fig. 1; Daszak et al. 2000, Tompkins and Poulin 2006).

While NIS often leave behind, or lose, their natural parasites (Dobson and May 1986, Torchin et al. 2003, Tompkins and Poulin 2006), they tend to acquire generalist parasites from the local fauna (Poulin and Mouillot 2003). Hence, NIS have the potential to impact disease in native fauna via the spillback of native parasites. Theoretical treatments of predator–prey and biological control systems already recognize dynamics that are analogous to parasite spillback. Modeling studies predict that native prey populations may be at risk of local extinction when native predator populations are enhanced via consumption of NIS prey (Noonburg and Byers 2005). Similarly, introduced biological-control agents are often exploited by native natural enemies whose enhanced numerical response could indirectly affect nontarget native species (Pearson

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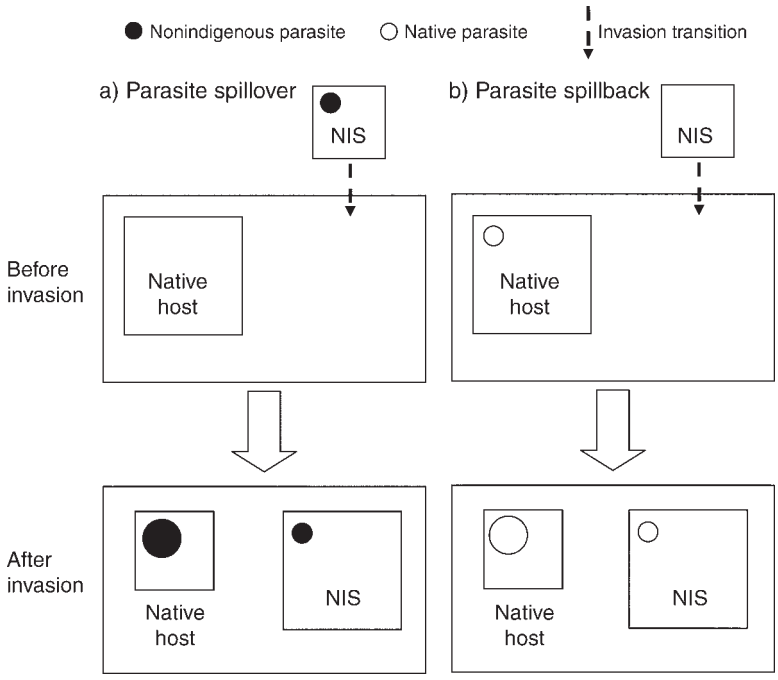

FIG. 1. Hypothetical examples of (a) parasite spillover and (b) parasite spillback, illustrating the fundamental differences between the two processes. The large rectangle represents the native environment. The size of the circle relative to host box represents infection burden. The size of the host box represents population size/density. In panel (a), infection of a native host population by a nonindigenous (NIS) parasite occurs after the introduction of that parasite's NIS host. Infection of the native host is maintained by the NIS host, which acts as a reservoir of infection. Infection of the native host by the NIS parasite causes reduced population size, compared to preinvasion levels. Note that the native host may or may not possess native parasites. In panel (b), the NIS acquires a native parasite that is already present in a native host population. The infection burden of the native parasite in the native host is increased by the NIS, which acts as a new reservoir of infection. Elevated infection in the native host causes reduced population size, compared to preinvasion levels.

Notes: Increased infection burden in a native host population in the presence of an NIS reservoir is not solely reliant on that reservoir having a larger population size; this is because the NIS may actually be a more suitable host for native parasites (see *Are NIS competent hosts for native parasites?*). Note also that the NIS host may or may not lose parasites from its native range upon introduction to a new region.

and Callaway 2005). For example, the enhancement of native hyperparasitoid populations upon introduction of parasitoids for biological control, could lead to suppression of native parasitoid populations (e.g., Heimpel et al. 2004). Parasite spillback is thus one form of "apparent competition," where two or more species that do not compete directly for resources, share a natural enemy whose numerical response is predicted to have reciprocal negative population effects (Holt 1977, Holt and Lawton 1993). However, despite a vast literature on apparent competition, parasite spillback has been either overlooked or never considered in recent reviews of the role of parasites in species introductions and as threats to native populations (Prenter et al. 2004, de Castro and Bolker 2005, Hatcher et al. 2006, Pedersen et al. 2007). There are two possible reasons for this. First, studies of NIS impacts have generally focused on introduced parasites, assuming that native parasites are unimportant. Second, native parasites are unimportant, with the potential for impacts on native fauna being rare.

Here we present evidence supporting parasite spillback as a potentially common impact of NIS on native animals, discuss the likely reasons for its prior neglect, and identify avenues for future research. Our aim is to raise awareness and stimulate research effort into what is likely an important, but currently untested, concept in invasion ecology

THE POTENTIAL FOR PARASITE SPILLBACK

The acquisition of native parasites by nonindigenous species (NIS) is the essential precursor for spillback to occur. As noted above, data already show how NIS can acquire native parasites upon introduction or invasion. For example, in their review of "enemy release" by NIS (in this case, the loss of parasites), Torchin et al. (2003) reported that while an average of three helminth parasites were co-introduced with NIS, an average of four helminth species were acquired in the newly colonized region. Moreover, in a meta-analysis of the parasitoids of 87 NIS insect herbivores, an average of 4 native parasitoids were acquired in introduced regions, with over 25% of hosts acquiring >10 parasitoid species (Cornell and Hawkins 1993). To further investigate the extent of this phenomenon in introduced animals, with

the aim of characterizing how broad or narrow the potential for spillback impacts of NIS is, we searched the ISI Web of Science (WOS) database (see Appendix A for a full description of the methods).

Data were obtained from comprehensive parasite faunal surveys for 40 animal NIS, in which a mean of 6.3 native parasite species were acquired per host, with native parasites dominating (67.0%) the total parasite fauna of each nonindigenous animal host (see Appendix B for the list of studies). Of the NIS, 70% acquired $\geq$4 native parasites, and 15% acquired >10 native parasites (Fig. 2). At least 38 of the 40 NIS hosts acquired parasites that were generalists, being reported from more than one native host species. NIS hosts were diverse, and comprised aquatic and terrestrial invertebrates, fish, amphibians, reptiles, birds, and mammals. The types of parasite acquired also were diverse, including helminths, arthropods, protozoa, and parasitoids. While NIS acquired native viruses and bacteria (see Appendix C), the reports probably represent a fraction of the actual acquisition since the literature was often unclear with respect to virus or bacterial status. Indeed, numerous studies documenting the sharing of these and other parasites between native wildlife and NIS were excluded from our main analysis due to uncertain parasite origin (see Appendix D for the full list of studies). The limited availability of data on shared parasites partly biases our selection of NIS, and precludes accurate estimates of the total number of native parasites acquired per NIS. Nonetheless, the breadth of taxonomic coverage, for both animal host and parasite type, is sufficiently comprehensive to illustrate that animal NIS commonly acquire native parasites.

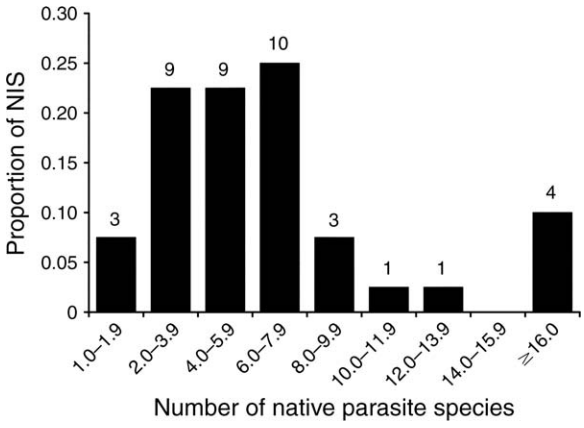

FIG. 2. Frequency distribution of native parasites acquired by the 40 nonindigenous species (NIS) hosts identified in our literature review. The number of parasites acquired is not discrete since there were several NIS for which multiple studies were conducted in different invaded regions. In such cases, the number of parasites acquired was the mean number across multiple studies. The actual number of studies is indicated above each bar.

Are NIS Competent Hosts for Native Parasites?

The acquisition of a native parasite by a nonindigenous species (NIS) will not automatically lead to the spillback of infection to native fauna; the NIS also needs to be a competent host for the parasite, and be capable of disseminating the parasite's infective stages (i.e., be a "reservoir" host in which the parasite can persist and reproduce; Tompkins et al. 2000*b*, Holt et al. 2003, de Castro and Bolker 2005). If this were not the case, the NIS would be a sink of parasite infection that could potentially act to reduce infection levels in the native fauna ("dilution" effect; Heimpel et al. 2003, Keesing et al. 2006).

There is evidence from a range of studies that NIS are not only often competent hosts for native parasites, but can also be better than the original native hosts (i.e., the parasite's basic reproductive rate, R_0, is greater when infecting them). For example, Ross River virus, a mosquito-borne virus native to Australia, is acquired by horses and other domestic stock from native marsupials (Russell 2002), with seroprevalence in horses even exceeding that of native marsupials (Vale et al. 1991, Kay et al. 2007). Another example is eastern equine encephalitis virus (EEEV), a virus native to North America, occurring principally in an enzootic cycle involving mosquito vectors and a variety of native avian hosts, which is often fatal in a range of mammalian and avian species (Komar et al. 1999, Nolen-Walston et al. 2007). In a comparison of reservoir competence of introduced European Starlings (*Sturnis vulgaris*) vs. 11 native bird species, starlings had higher intensities and durations of viraemia, and infected a higher proportion of mosquito vectors than did native birds (Komar et al. 1999). As a result, even though they suffered higher mortality from infection, starlings were twice as competent as reservoirs of infection as any native species.

There are also cases where fitness-associated traits of native macroparasties are higher in NIS vs. native hosts. For instance, in many introduced salmonid fish, native macroparasites occur in equal or higher abundance (percentage of fish infected, mean number of parasites per fish) than in their native fish hosts (Kennedy et al. 1991), with some parasites also being more fecund. This is illustrated by a study on Lake Moreno (Argentina), where four of the five helminth parasites of introduced rainbow trout (*Oncorhynchus mykiss*) and brook trout (*Salvelinus fontinalis*), both native to North America, are acquired from native fish (Rauque et al. 2003; Appendix B). In a comprehensive assessment of host infection by the native intestinal worm *Acanthocephalus tumescens*, both salmonids had similar prevalences (33–50%) but generally lower infection intensities, harboring <9 worms per host, compared with 10–27 worms in three native fish species. Collectively, the salmonids represent a small fraction (2.9%) of the total fish abundance in the lake. However, as a greater percentage of *A. tumescens* mature in the salmonids than in any native fish, the two

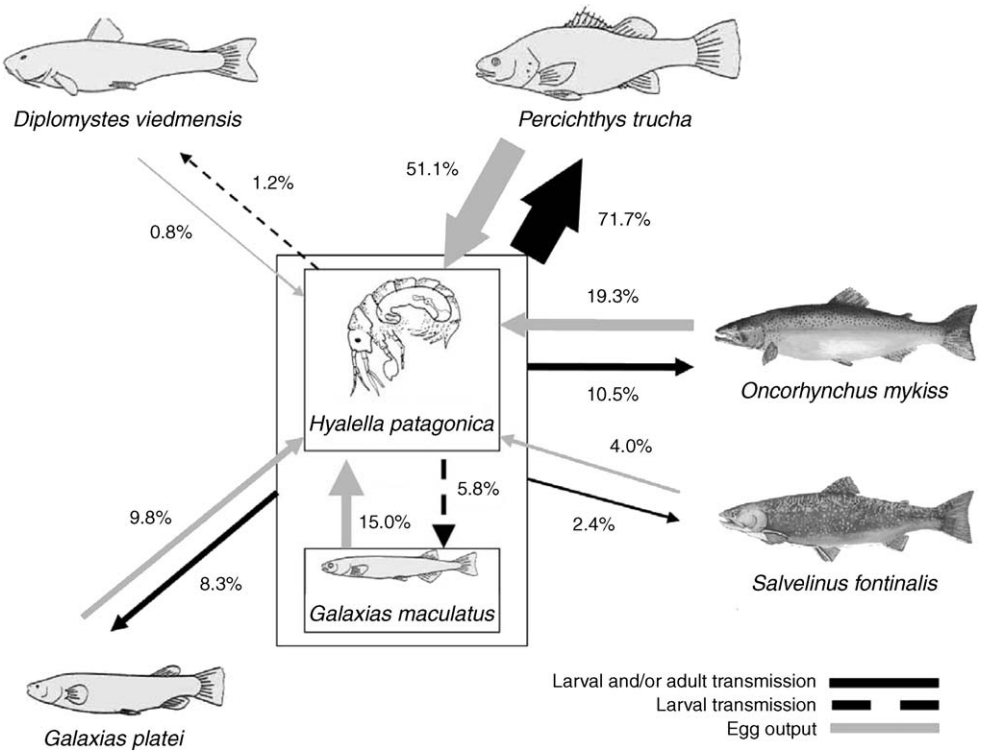

FIG. 3. The relative flow rates of infective stages of the worm *Acanthocephalus tumescens* in native and introduced fish hosts in Lake Moreno, Argentina. Eggs of the adult parasite, which are shed from a definitive fish host, are eaten by the amphipod *Hyalella patagonia*, in which they develop as larval worms. Infected amphipods are eaten by a definitive fish host in which the parasite develops to an adult. Fish become infected with parasite larvae (larval transmission) by feeding on *H. patagonia* or through consumption of adult worms (adult transmission) infecting small fish such as *Galaxias maculatus*. Arrow thickness indicates the relative importance of each fish host for parasite larval/adult transmission, or parasite egg output. The introduced salmonids *Oncorhynchus mykiss* and *Salvelinus fontinalis* are infected by a small proportion of worms (transmission) but account for a high proportion of worm egg output (see *Are NIS competent hosts for native parasites?*). This figure was reproduced from Rauque et al. (2003) by permission of *Folia Parasitologica*.

NIS account for almost a quarter of the parasite's total egg production (Rauque et al. 2003; Fig. 3).

There are thus some compelling examples demonstrating how NIS can be competent hosts for native parasites, from which spillback to native hosts could potentially occur. However, the relative frequency with which NIS are competent hosts, as opposed to "sink" hosts, still needs to be determined.

CAN INFECTION SPILL BACK FROM NIS TO NATIVE HOSTS?

The parasite-spillback hypothesis is consistent with the endemic-pathogen hypothesis proposed for recent emerging infectious diseases of wildlife (Rachowicz et al. 2005). Many wildlife populations may have endured a stable co-existence with endemic diseases, but the disequilibrium often associated with perturbations, such as species introductions, can promote disease emergence (Rachowicz et al. 2005). Interestingly, several viral diseases that are prevalent in domestic cats and dogs have putatively spilled back to wild canids and felids, despite an historic presence in the latter hosts (Steinel et al. 2001, Fiorello et al. 2006, McCarthy et al. 2007). In addition, bacterial haemoplasmas, long associated with domestic cats, are highly prevalent among native wild felids (Willi et al. 2007). For example, studies of domestic cats and dogs in South America indicate that they are important reservoirs for spillover of the apicomplexan *Toxoplasma gondii* (the aetiological agent of Toxoplasmosis) to native wildlife (Fiorello et al. 2004), with the increased ubiquity of *T. gondii* in native felids being attributed to the introduction and increase in abundance of domestic cats since the 16th century (Lehmann et al. 2006). However, recent phylogeographic studies indicate that *T. gondii* has a South American origin in wild native felids (Lehmann et al. 2006, Dubey et al. 2007). If this is indeed the case, then these striking infection patterns are due to spillback, not spillover.

There is little evidence, however, with which to estimate the frequency at which the spillback of infection from competent NIS to native hosts actually occurs, since obtaining such evidence would normally require an experimental approach. As spillback has not received that kind of attention, here we are restricted to presenting evidence that strongly implies (as with the *T. gondii* example above) that such transmission does

occur. Another compelling case study is provided by the introduction of the muskoxen subspecies *Ovibos moschatus wardi* to northwestern America, and its subsequent acquisition of the lungworm *Protostrongylus stilesi*. After the extirpation of native muskoxen *O. m. moschatus* from this region, *O. m. wardi* was introduced from East Greenland to the Arctic coastal plain in 1900 (Hoberg et al. 2002). *O. m. wardi* occupies a growing range eastward to the Yukon and Northwest Territory and most likely acquired *P. stilesi* from native thinhorned Dall's sheep (*Ovis dalli dalli*). Although the parasite is common in Dall's sheep (see Kutz et al. 2001), a new geographic record in the sheep population of Northwest Territory is coincident with the maximum eastward range expansion of *O. m. wardi* (Hoberg et al. 2002). Thus, it is most likely that the muskoxen transmitted the parasite, initially picked up from the sheep, to this population (Hoberg et al. 2002).

Two more case studies that imply spillback transmission in animal parasite systems are described in the following section. Generally, however, it can be argued that the spillback of native parasites from competent NIS hosts to native fauna is likely to be common, since NIS often possess particular demographic traits that can facilitate the spread of infection. For example, NIS often undergo rapid population growth and achieve high densities relative to ecologically similar native species (e.g., Elton 1958, Ricciardi and Maclsaac 2000, Kelly et al. 2006), with many also spreading rapidly and achieving wide distributions due to their broad environmental tolerance and an association with human dispersal vectors (Kolar and Lodge 2002, Fevre et al. 2006, Muirhead et al. 2006). Indeed, these demographic characteristics have been invoked to explain why some NIS are important dispersers of previously introduced pathogens in certain regions (e.g., the role of the North American Bullfrog in chytrid fungus dissemination globally; Garner et al. 2006). The aforementioned demographic characteristics could be particularly important in facilitating the spillback of acquired native viruses and bacteria (see Appendix C). For example, because most microparasites are directly transmitted, the additional presence of a competent NIS host would increase the threshold density necessary for microparasite persistence (de Castro and Bolker 2005, Pederson et al. 2007).

Can Spillback Impact Native Hosts?

Theoretical support that parasite spillback could impact native hosts already comes from multiple-host shared-parasite systems where local extinction of a focal host is predicted when interspecific transmission equals or exceeds intraspecific transmission, and when parasite virulence in a focal host is higher than in a second host (Hatcher et al. 2006). However, robust empirical data supporting spillback impacts on native animal host populations are lacking.

An empirical example from a plant-pathogen system clearly illustrates the dynamics by which parasite spillback could also impact native animal populations. Malmstrom et al. (2005) investigated the role of introduced annual grasses on the incidence of barley and cereal yellow dwarf viruses (B/CYDVs) in perennial native grasses in California, USA, and the preference and fecundity of the aphid vector of the viruses on the different grass host species. In a field experiment, viral incidence in the native plant more than doubled when grown in sympatry vs. allopatry with the nonindigenous species (NIS) of grass, while aphid densities were significantly higher in plots that included the NIS grass. Laboratory trials showed that aphids preferred feeding on NIS grasses, and had significantly higher fecundity when feeding on the latter hosts. Although the study of Malmstrom et al. (2005) was unclear with respect to virus origin, and did not specifically link amplified infection to negative population impacts on native grasses, it provides a confirmation that parasite spillback can potentially impact native host populations.

Our review of the animal parasite literature uncovered two examples that, in addition to providing further support for the spillback of infection from NIS, also strongly imply that subsequent impact can occur. The first example is Settle and Wilson's classic study of apparent competition in an insect herbivore system (Settle and Wilson 1990). Field observations showed that population declines of the native grape leaf hopper in Californian vineyards were coincident with the introduction and spread of the variegated leaf hopper. Eggs of the grape leaf hopper were naturally parasitized by the hymenopteran *Anagrus epos*, which kills its host upon emergence. Although the variegated leaf hopper was susceptible to *A. epos* attack, it suffered lower rates of parasitism and was less preferred than the native host. Evidence for a parasite-mediated indirect effect of the invader came from observations of positive correlations between its relative density and the proportion of infected native grape leaf hoppers, and between the invader's density and the ratio of the number of parasitoids per native host individual. Crucially, the invader contributed to ~50% of the parasitoid population only when it became the dominant (e.g., 85%) leaf hopper species. This study illustrates how interactions between NIS density, differences in host susceptibility to parasitism, and changes in infection intensity of native hosts, can lead to declines in native host populations.

The second example is the displacement of the native house gecko *Lepidodactylus lugubris,* from across the Pacific Islands, by the introduced *Hemidactylus frenatus*, a native of Southeast Asia (Case et al. 1994). To investigate whether the pattern observed is mediated by parasitism, Hanley et al. (1995) surveyed helminths and protozoa of both hosts across islands where they occurred in both allopatry and sympatry. We obtained the Hanley dataset to determine if any native parasites were acquired by the NIS host, assuming that a parasite

of the NIS was acquired if it was absent in the native range of *H. frenatus*, present in *L. lugubris* on uninvaded islands, and present in both host species when they occurred in sympatry (K. Hanley, *personal communication*). Under this assumption, the invading *H. frenatus* acquired one protozoan (*Eimeria furmani*), two nematodes (*Hedruris* spp.; *Physaloptera* spp.), and one cestode (*Cylindrotaenia*) from *L. lugubris*. Interestingly, increasing intensity of infection of *Cylindrotaenia* was negatively correlated with body condition in the native gecko. *Cylindrotaenia* also was significantly more prevalent in *L. lugubris* populations when they occurred in sympatry vs. allopatry with *H. frenatus* (13% and 5%, respectively; Hanley et al. 1995). These observations suggest a negative population effect on the native gecko through parasite spillback (Hanley et al. 1995).

These two examples demonstrate that spillback effects can likely impact native animal species. However, a definitive experimental test is still required, and further evidence from multiple systems is needed to determine how widespread and common (and hence important) a mechanism it is. After identifying a system in which parasite spillback is potentially having host community-scale effects, generally achievable through a combined field survey, laboratory trial, and mathematical modeling approach (see Tompkins et al. (2000*a*, *b*) for a parasite-mediated apparent-competition example), proof of concept would require replicated controlled manipulations at the population scale. If experimental reductions in the parasite burdens of NIS hosts alone led to a decrease in infection in a native species, and accompanying increases in both individual fitness and population size of that species, parasite spillback as a mechanism of NIS impact would be proven.

Acquired Parasites and Complex Interactions

In addition to directly amplifying infection of native parasites by acting as a definitive host (spillback), nonindigeneous species (NIS) could also potentially increase infection by native parasites in native hosts by fulfilling other roles in the parasite life cycle, such as that of intermediate host or vector. Such an interaction could be termed "trophically mediated" spillback, with the NIS becoming part of a sequence of energy transfers among multiple hosts. In addition to a parasite numerical response to the presence of a new host, such spillback could also potentially be driven by differences in the behavior or ecology of NIS vs. native intermediate hosts or vectors. This is illustrated in a long-term study by Strecker (2006) on the impact of introduced African cichlid fish *Oreochromis* on endemic *Cyprinodon* species in Lake Chichancanab, Mexico. Cichlids invaded the lake in 1988 and their population size grew rapidly. However, in the years following invasion, six of the seven *Cyprinodon* species declined dramatically and one species, *C. simus*, became locally extinct. Since *Oreochromis* are detritivore–planktivores, this pattern was not due to predation. Of particular interest were the temporal dynamics of parasitism in the endemic fish. The fish are intermediate hosts for transmission of the native trematode *Crassiphiala cf. bulboglossa* to piscivorous definitive bird hosts. Preinvasion data showed that six of the seven species were infected at low prevalence (0–25%) by the trematode but prevalence reached 90–100% by 6–7 years after *Oreochromis* invasion, during which time the invader became a new intermediate host of the parasite. Furthermore, a decline in body size from pre to post cichlid invasion in one endemic fish, *Cyprinodon maya*, was associated with increased parasitism, with predation of *Oreochromis* by the bird hosts augmenting the flow of infective stages from NIS intermediate, to native definitive, to native intermediate host (Strecker 2006). Increased bird predation on the NIS was driven by the invader's larger size, greater population abundance, and use of open-water habitats as compared to the native fish.

In multiple-host shared-parasite systems involving vectors, asymmetries in host and vector competence, and host susceptibility to parasitism, can affect parasite transmission and persistence (Hatcher et al. 2006). Thus, further complexities might be expected in cases where several introduced species fulfill different roles in the life cycle of a shared native parasite. For example, EEEV (eastern equine encephalitis virus) was one of four native viruses isolated from the invasive Asian tiger mosquito *Aedes albopictus* in North America (Mitchell et al. 1992). Patterns of *A. albopictus* infection by EEEV, together with high competence of the vector, suggest that it may be responsible for recent epizootics (Mitchell et al. 1992). The high reservoir competence of NIS starlings for EEEV, described earlier (Komar et al. 1999), suggests a potential interaction with *A. albopictus* or other introduced vectors that would amplify the virus. For instance, *A. albopictus* is a highly competent vector when exposed to viremic starlings (Komar et al. 1999), while another rapidly spreading Asian mosquito, *Ochlerotatus japonicus japonicus*, also is a competent vector of EEEV (Sardelis et al. 2002). Such situations have the potential to increase the spread and impact of a native parasite, perhaps paralleling the invasional-meltdown hypothesis proposed to explain the impacts of many NIS (Simberloff 2006).

Discussion

Current assessment

Here we extend the evidence demonstrating that nonindigenous species (NIS) do generally acquire native parasites during or after introduction or invasion, illustrating that there is great potential for parasite spillback from NIS to impact native communities. We then demonstrate that NIS can be highly competent hosts for native parasites, and present evidence that infection by native parasites does spill back from NIS to native host species, with subsequent effects at both the host individual and population scale. Hence, although still requiring further study to confirm the role of this

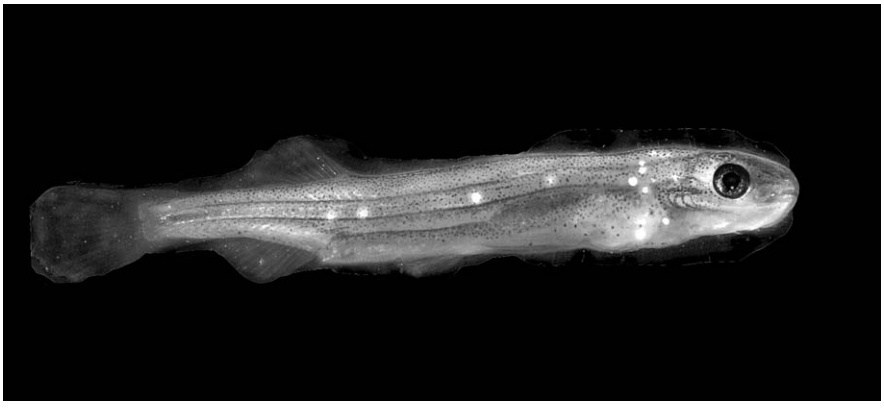

PLATE 1. Larval *Galaxias anomalus*, a native of New Zealand, parasitized by cysts of the trematode *Telogaster opisthorchis*. Could introduced salmonid fish be competent hosts that spill back infection to the detriment of the native species? Photo credit: D. W. Kelly.

mechanism as an impact of NIS on native fauna, and to understand the extent to which the potential for such impacts is actually achieved in the wild, it is likely to be an important mechanism.

So, why has spillback largely been overlooked in the invasion ecology literature? The animal-parasite literature reviewed here indicates that studies of NIS impacts generally assume that native parasites are unimportant. This is illustrated in the study of Smith and Carpenter (2006), investigating threats to the native endemic deer mouse *Peromyscus maniculatus*, on California's Channel Islands (USA), from parasites co-introduced with the nonindigenous black rat *Rattus rattus*. Taking advantage of a patchwork of invaded and noninvaded islands, Smith and Carpenter ascertained that rats introduced the nematode *Trichuris muris* to endemic deer mice through spillover, and discussed the conservation implications. However, closer inspection of their data set reveals that another nematode, *Protospirura peromysci*, was probably acquired by rats from the native mice, because it was absent in global surveys of black rat parasites (Smith and Carpenter 2006: Appendix S1), present in mainland North American deermouse populations, and, in the only two islands in which both hosts were sympatric, reached higher prevalence in rats than the parasite assumed to have spilled over.

The occurrence of spillback is also likely underestimated through failure to identify the status of shared parasites (i.e., native vs. exotic). This issue is highlighted in our review, where many shared parasites had unknown or uncertain status (see Appendix D). Factors hampering identification of parasite status include a lack of parasite faunal surveys of native hosts (particularly records prior to the introduction of NIS), and the presence of shared parasites that were putatively cosmopolitan. Also, parasite faunal lists are often incomplete due to many endangered wildlife species being difficult to sample (Steinel et al. 2001). For example, in Australia, a country renowned for rodent introductions, there is also a diverse assemblage of native rodents, many of which have become extinct or are endangered (Warner 1998). However, while extensive parasite faunal lists have been compiled for introduced rodents (typically after major disease and population epidemics), few studies have focused on native rodents, and it is almost impossible to determine whether parasites are native or introduced (Warner 1998). Other studies appear to have made erroneous assumptions on the status of shared parasites. This problem was recognized by Torchin et al. (2002), who noted that the colonization of more abundant NIS by previously rare native parasites could lead to apparently novel epizootics that are erroneously ascribed to spillover of a co-introduced parasite. In addition to potential impacts being overlooked, the distinction between spillover and spillback is also important for their management. When such impacts are driven by parasite spillover, control of infection in the NIS reservoir host should normally be sufficient to manage disease impacts (e.g., control of canine distemper in lions through domestic dog culls; Cleaveland et al. 2002). However, when such impacts are driven by parasite spillback, control of infection in both native and NIS hosts would likely be required (e.g., control of bovine tuberculosis usually requires management of both domestic stock and wildlife reservoirs; Corner 2006).

Most dramatic outbreaks of disease in native species have been ascribed to spillover infections, where native hosts are exposed to novel infectious agents (Daszak et al. 2000). However, we have shown that, due to uncertainty in parasite origin, several such disease outbreaks may have been caused by parasite spillback. Parasite spillback may have important but less obvious impacts at the population and community scale, since it is well established that parasites causing subtle effects can often regulate host populations (Hudson et al. 1998, Albon et al. 2002, Tompkins et al. 2002). Although such effects are hard to detect, as noted above, they generally require an experimental approach to elucidate.

Future research directions

As is evident from the discussion above, research on parasite spillback is required to provide the definitive proof first that the presence of competent NIS hosts can increase the infection burdens of native parasites in native hosts, and second that such raised levels of infection can impact native populations and communities. In addition, uncertainties over parasite origin demonstrate a need for the resolution of parasite status in studies of shared parasites of native hosts and NIS in general. Such resolution will allow a clearer understanding of the potential of parasite spillback, a more complete understanding of the mechanisms by which parasite interactions associated with NIS impact native fauna, and will influence future conservation management decisions (Rachowicz et al. 2005).

The examples discussed above provide some level of guidance on the native communities in which parasite spillback impacts of NIS are likely occurring, and hence are good candidates for our proposed experimental approach to test the phenomenon. For example, in the freshwater fish community of Lake Moreno in Argentina described above (see *Are NIS competent hosts for native parasites?*), the observation that the remaining individuals of the native fish thought most affected by salmonid introductions, the catfish *Diplomystes viedmensis*, carry the highest abundance of the shared parasite *Acanthosephalus tumescens* of any host (Rauque et al. 2003), is highly suggestive of parasite-spillback effects. Indeed, in other systems where salmonids have been introduced, there may be similar opportunities to test parasite spillback (see Plate 1). Several studies in our review also report the sharing of native parasites among multiple nonindigenous hosts in a particular site or region (e.g., fish, Rauque et al. 2003; deer, Richardson and Demarais 1992; marine invertebrates, Krakau et al. 2006, Thieltges et al. 2006; birds, Miles et al. 1971). For example, in the Wadden Sea, near the coasts of The Netherlands and Germany, all five species of parasite reported in the invading Pacific oyster *Crassostrea gigas* (native to North America) are shared with the native blue mussel *Mytilus edulis* and the common periwinkle *Littorina littorea* (Aguirre-Macedo and Kennedy 1999, Thieltges et al. 2006). Of the parasites acquired, the copepod *Mytilicola intestinalis* and the shell-boring polychaete *Polydora ciliata* appear the most likely candidates for spillback of infection. *M. intestinalis* infection was responsible for historical population declines in native mussels and attains a prevalence of over 50% in *C. gigas*, whereas *P. ciliata* occurs at higher prevalence in *C. gigas* than in *M. edulis* or *L. littorea* where all three hosts are sympatric (Aguirre-Macedo and Kennedy 1999, Thieltges et al. 2006). Interestingly, the slipper limpet *Crepidula fornicata* (native to the Northwest Atlantic coast) is another NIS that has acquired *P. ciliata* in areas of sympatry with *C. gigas* and the native hosts. Both the slipper limpet and the Pacific oyster are increasing in abundance, and spillback of infection could have dramatic consequences because both parasites influence population viability of the native hosts (Thieltges et al. 2006, Buschbaum et al. 2007). Given that the amplifying effect of reservoir hosts may be greater in multiple-host shared-parasite systems (Keesing et al. 2006), such systems are thus perhaps the most suitable candidates for testing the parasite-spillback-from-NIS hypothesis.

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APPENDIX A

Methods used in the ISI Web of Science literature search (*Ecological Archives* E090-142-A1).

APPENDIX B

Studies documenting native parasites that are shared between native and nonindigenous hosts (*Ecological Archives* E090-142-A2).

APPENDIX C

Studies documenting native viruses and bacteria that are shared between native and nonindigenous hosts (*Ecological Archives* E090-142-A3).

APPENDIX D

Parasites of unknown/unspecified origin that are shared between native and nonindigenous hosts (*Ecological Archives* E090-142-A4).