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EFFECTS OF INFECTIOUS DISEASES AND PARASITES ON HOST POPULATIONS AND NATURAL COMMUNITIES: THE USE OF NON-INDIGENOUS SPECIES (NIS) AS MODELS

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ABSTRACT

The use of mathematical models to study the effects of parasites and infectious diseases on host populations is extremely common and helpful. Indeed, these theoretical models can be seen as tools of ecology and epidemiology and can help in the controlling and prevention of infectious diseases. However, testing the predictions of these models has been very difficult, but some experiments of parasite addition and removal certainly have helped to test these theoretical predictions. In this review I explore the possible use of introduced non-indigenous species (NIS) as possible 'natural' experiments of parasite removal, since most of them lose their parasites in the process of introduction, as well as parasite addition, in this last case by looking at biological control studies. These studies indicate that introduced NIS do present lower parasite prevalence and richness, and consequently, have higher population performance than NIS in their native range. This indicates that parasites are able to control host populations. In addition, studies of introduced infectious disease show that the reduction of host abundance can result in secondary effects on communities and ecosystems, and that naïve hosts are in danger of being extirpated by introduced pathogens, especially if reservoir hosts are present. Conversely, hosts will rarely be extinct, even if immunologically naïve, by a parasite, in single host systems, as expected by theoretical models. Lastly, parasites can have key roles in communities when more than one host share the same parasite because of apparent competition.

Keywords: Non-indigenous species; release from parasite; parasite-host dynamics; effects of parasites; emergent diseases.

RESUMO

EFEITOS DE DOENÇAS INFECCIOSAS E PARASITAS NAS POPULAÇÕES DOS HOSPEDEIROS E NA COMUNIDADE NATURAL: O USO DE ESPÉCIES INVASORAS COMO MODELO. O uso de modelos matemáticos para estudar os efeitos de parasitas e de doenças infecciosas na população de hospedeiros é de extrema importância e pode ajudar na elucidação da dinâmica parasita-hospedeiro. Realmente, esses modelos teóricos podem ser vistos como ferramentas ecológicas e epidemiológicas para auxiliar no controle e prevenção de doenças infecciosas. Apesar da dificuldade em testar as predições desses modelos, alguns experimentos de adição e remoção de parasitas tem ajudado a testar essas predições teóricas. Nesta revisão eu exploro a possibilidade de utilizar espécies invasoras como experimentos 'naturais' de remoção de parasitas, uma vez que essas espécies perdem seus parasitas no processo de introdução, assim como de adição de parasitas (nesse último caso através dos estudos de controle biológico). Esses estudos indicam que as espécies invasoras apresentam menor taxa de prevalência e riqueza de parasita, e consequentemente, apresentam maior desempenho populacional na região introduzida do que em sua região nativa. Estudos referentes à introdução

de doenças infecciosas demonstram que a redução na abundância do hospedeiro pode ter efeitos secundários na comunidade e nos ecossistemas. Além do mais, foi observado que hospedeiros que não apresentam resistência aos novos parasitas introduzidos estão em perigo de serem extintos, principalmente se hospedeiros reservatórios estão presentes. Contrariamente, hospedeiros raramente serão extintos, mesmo com baixa resistência à doença, quando o sistema consiste de apenas um parasita-hospedeiro, como esperado em modelos teóricos. Por final, parasitas podem desempenhar papéis-chave na comunidade quando esses conseguem parasitar mais de um hospedeiro devido à competição aparente.
Palavras-chave: Espécies invasoras; liberação de parasitas; dinâmica parasita-hospedeiro; efeitos de parasitas; doenças emergentes.

RESUMEN

EFECTOS DE LAS ENFERMEDADES INFECCIOSAS Y LOS PARÁSITOS EN LAS POBLACIONES DE LOS HOSPEDEROS Y EN LA COMUNIDAD NATURAL: USO DE ESPECIES INVASORAS COMO MODELO. El uso de modelos matemáticos para estudiar los efectos de parásitos y de enfermedades infecciosas en la población de hospederos es de extrema importancia y puede ayudar a elucidar aspectos de la dinámica parasito-hospedero. Realmente, esos modelos teóricos pueden ser vistos como herramientas ecológicas y epidemiológicas para ayudar en el control y prevención de enfermedades infecciosas. A pesar de la dificultad para probar las predicciones de esos modelos, algunos experimentos de adición y remoción de parásitos han ayudado a probar esas predicciones teóricas. En esta revisión exploro la posibilidad de utilizar especies invasoras como experimentos 'naturales' de remoción de parásitos, una vez que esas especies pierden sus parásitos en el proceso de introducción, así como de adición de parásitos (en ese último caso a través de los estudios de control biológico). Esos estudios indican que las especies invasoras presentan menor tasa de prevalencia y riqueza de parásitos, y consecuentemente, presentan mayor desempeño poblacional en la región introducida que en su región nativa. Estudios referentes a la introducción de enfermedades infecciosas demostraron que la reducción en la abundancia del hospedero puede tener efectos secundarios en la comunidad y en los ecosistemas. Además de esto, fue observado que los hospederos que no presentan resistencia a los nuevos parásitos introducidos están en peligro de extinguirse, principalmente si los hospederos que sirven de reservatorio están presentes. Contrariamente, los hospederos raramente se extinguirían, aun con baja resistencia a la enfermedad, cuando el sistema está formado solo por parasito-hospedero, como es esperado en modelos teóricos. Finalmente, los parásitos pueden desempeñar papeles clave en la comunidad cuando consiguen parasitar más de un hospedero debido a la competencia aparente.
Palabras clave: Especies invasoras; liberación de parasitas; dinámica parasito-hospedero; efectos de parasitas; enfermedades emergentes.

INTRODUCTION

Human-induced biodiversity loss is happening at an alarming rate with habitat degradation being considered as the major threat to biodiversity, followed by the introduction of non-indigenous species (Wilcove *et al.* 1998). Biotic invasion is occurring very rapidly as a consequence of global trade increase and these non-indigenous species are responsible for the extinction of many animals and plants through predation, grazing, competition and habitat alteration (Mack *et al.* 2000). In addition, invasions by disease-causing non-indigenous organisms can severely affect native species and humans (Mack *et al.* 2000, Jones *et al.* 2008). For instance, the introduction of the bacteria *Yersinia pestis*, responsible for the 'black plague', in medieval times, killed one-third of the European population (Nentwig 2007). Other examples of non-indigenous diseases affecting humans are HIV, which originated in central Africa, influenza strains, originating in the Far East and many other diseases like malaria and dengue fever (Pimentel *et al.* 2002). Similarly, wildlife is also suffering from introduced pathogenic infectious diseases that have emerged in the past decades (Daszak *et al.* 2000), for example the global decline of amphibian populations due to chytridiomycosis (Daszak *et al.* 1999).

Emergent infectious disease can be a serious threat to endangered and threatened species because infectious agents can cause severe impacts on

host population dynamics. For instance, infectious agents can reduce host density, which in turn can have debilitating secondary effects on ecosystems (McCallum & Dobson 1995, Lafferty & Gerber 2002). In single host-parasite system, parasites should not, in general, lead to the extinction of their host and virulence is expected to be attenuated in the long run (Anderson 1979). If not, then hosts can reach a density threshold where parasite persistence will not be maintained (Deredec & Courchamp 2003). Alternatively, pathogen and parasites can cause host extinction if: they manage to reduce host density to such low levels that they become vulnerable to stochastic events; they are sexually transmitted diseases or vector-borne pathogens (that are usually frequency-dependent), which means that their prevalence (proportion of sampled hosts infected; see Bush *et al.* 1997) are less dependent on host density; if reservoir hosts are present they can serve as a source for pathogen epidemics, and therefore virulence attenuation might not occur (Smith *et al.* 2009); and if hosts are highly susceptible to the pathogen (*i.e*. hosts are immunologically naïve to the pathogen), which can be a common issue when disease-causing organisms are introduced (Daszak *et al.* 2000, Lafferty & Gerber 2002).

Parasites are important population regulators, just like predators can be, and studies with a two-host shared parasite species have shown that parasites are capable of structuring ecological communities, acting as key species through processes such as apparent competition (McCallum & Dobson 1995, Hudson & Greenman 1998, Horwitz & Wilcox 2005). However, experiments of such complex systems are very difficult to conduct.

Lafferty *et al.* (2005) explored the fact that non-indigenous species (NIS) are interesting models to study the impacts that infectious agents (both microparasites, such as bacteria, or macroparasites, such as helminths) have on host's demographic parameters, host shifts and secondary effects on ecosystems. The reason is that when NIS are introduced to a new area, they usually leave behind their native parasites (Dobson 1988), and therefore biotic invasions can be seen as unfortunate natural experiments to look at parasite-host dynamics (Lafferty *et al.* 2005). Here I discuss and extend the exploratory work of Lafferty *et al.* (2005), who reviewed if parasites are capable of regulating host's population dynamics, by looking at the effects of parasite release on NIS, as well as the effects of parasite addition (*i.e.* biological control) to introduced population of NIS. I also look at the effects that introduced infectious agents can have on natural communities and also on humans.

NON-INDIGENOUS SPECIES (NIS) - RELEASE FROM PARASITES

The introduction of NIS provides the chance to study the effects of parasites on host populations by looking at how they perform in the absence of parasites. Despite these 'natural experiments' having much less control than any controlled field experiment, they can still provide good inferences on the role of parasites in natural communities and specially on how parasites regulate host populations (Torchin *et al.* 2003, Lafferty *et al.* 2005).

The escape from natural enemies, such as predators, parasites and pathogens is a very appealing explanation for the overall success of NIS (for both plants and animals). More specifically, it is expected that the limited number of parasites species (or pathogens), both in terms of richness and abundance, which are transported to the new location, die out soon after the species is introduced at their new location (probably because of low host density) or just fail to finish their life cycles in the new introduced locality (Dobson 1988, Torchin *et al.* 2001, Mitchell & Power 2003, Torchin *et al.* 2003, Colautti *et al.* 2004, Torchin & Mitchell 2004). Reasons for NIS to carry a limited subset of parasites can be the differential mortality of infected propagules during transportation when compared to non-infected propagules, transport vectors that may bias the life history stages that it carries (*i.e.* uninfected life history stages such as planktonic larvae and plant seeds), lower host density than the parasite's host-threshold density for maintaining viable populations, and lack of suitable vectors to complete the parasite's life cycle (Torchin *et al.* 2001, Mitchell & Power 2003, Torchin *et al.* 2003, Colautti *et al.* 2004). It is possible that the lower parasitism associated with NIS might enable them to experience a demographic release, which would give these species a competitive edge over the sympatric native species, and allow NIS to rapidly increase in

abundance and distribution and become pests (Keane & Crawley 2002, Torchin *et al.* 2003, Hufbauer & Torchin 2007). The release from the effects of co-evolved parasites and the gaining of new parasites allows for the uncovering of the extent of parasite control on host populations and the structuring of ecological communities (Lafferty *et al.* 2005).

In this context, if NIS indeed lose their parasites, they should present decreased parasite richness and lower prevalence in the NIS' introduced range when compared with their native range (Lafferty *et al.* 2005). In addition, if parasites are directly or indirectly involved in regulating host population, then one would expect to find changes in NIS' demographic parameters, such as fecundity, biomass and survivorship (Anderson & May 1978, May & Anderson 1978, Colautti *et al.* 2004).

In general, NIS in their introduced range will only have a subset of parasite species present in their native range (Dobson 1988, Torchin *et al.* 2003, Torchin & Mitchell 2004). For instance, NIS of plants can escape over half of their native parasites (Torchin & Mitchell 2004) with NIS of plants presenting 77% fewer fungus and viruses in their introduced range when compared with their native range (Mitchell & Power 2003). The same can be said about NIS of animals, which are infected with roughly half of their native parasites in their introduced range when compared with their native range (Dobson 1988, Torchin *et al.* 2003). Additionally, both NIS of animals (Cornell & Hawkins 1993, Torchin *et al.* 2003) and plants (Torchin & Mitchell 2004) tend to present lower parasite prevalence with populations of animals and plants at the introduced range presenting less than half of what is found in populations from the native range (Cornell & Hawkins 1993, Torchin *et al.* 2003). Therefore, in general, NIS present lower parasite richness and prevalence (Table 1).

NIS can become pests once established and present higher density or size as would be expected if they suffered a demographic release (Table 2). Introduced populations do seem to present larger body size than native populations (Torchin *et al.* 2001, Pintor & Sih 2009, Vignon *et al.* 2009). This is important, because body size can be linked to several demographic parameters such as adult survival and life history traits such as fecundity, which in turn, can affect population growth rate (Futuyma 1998). In some cases, the enhancement of demographic performance in introduced populations is associated with parasite loss (Table 2; Lampo & DeLeo 1998, Torchin *et al.* 2001, 2002). However, Colautti *et al.* (2004) argued for a more critical test, since it is necessary to show that parasite loss is linked to demographic performance. For example, increase in body size can be associated with changes in NIS behavioral

Table 1. Release from parasites for several different taxa. Positive symbol indicates parasite release, while negative symbol indicates that parasite release did not occur (adapted from Lafferty *et al*. 2005).

Tabela 1. *Liberação de parasitas para diferentes taxa. Símbolo positivo indica que houve liberação de parasita, enquanto que símbolo negativo indica que não houve liberação de inimigos (adaptado de Lafferty* et al. *2005).*

Taxonomic group	Species #	Parasite release	Mean parasite richness		Mean parasite prevalence		Source
			Native	Introduced	Native	Introduced	
Plants	473	+	4	1	NA	NA	Mitchell & Power 2003
Plant	1	+	9	7.25	NA	NA	Knevel *et al.* 2004
Aquatic invertabrates	10	+/-	6	2	30%	13%	Torchin *et al.* 2002
Insects[a]	87	+	7.74	4.04	30.53%	11.33%	Cornell & Hawkins 1993
Invertebrate and Vertebrate	26	+	16	7	15%	4%	Torchin et. al. 2003
Fish	2	-	7.60	5.38	NA	NA	Poulin & Mouillot 2003
Fish	1	+	10	3	100%	5%	Vignon *et al.* 2009
Fish	2	+	72	22	NA	NA	Kvach & Stepien 2008
Amphibian	1	+/-	8	2	9.53%	41.3%	Marr *et al.*2008
Bird	1	-	37.3	40	NA	NA	Colautti *et al.* 2005

a= data based on medians where the range of values was reported

mechanisms associated with feeding behavior (*i.e.* high foraging activity and better exploitation of resources) or aggressiveness (Petren & Case 1996, Rehage *et al.* 2005, Snyder & Evans 2006, Pintor & Sih 2009). Nevertheless, there are several examples in the literature showing a strong association between parasite loss and demographic performance (Table 2).

For example, the peacock grouper (*Cephalipholis argus*), which presented lower parasite prevalence and richness in the introduced range when compared with its native range, were bigger and presented higher Fulton's index (a good indicator of the general well-being of a fish) in the introduced range than in the species native range. Therefore, release from parasites could allow this species of fish to reallocate energetic resources away from costly defenses towards other more important biological functions (Colautti *et al.* 2004). Torchin *et al.* (2001), also demonstrated that introduced populations of the European green crab (*Carcinus maenas*) were not infected by parasitic castrators, while native populations had a mean prevalence of 16%, which explained 64% of the variation encountered for mean crab size and 34% of the variation in crab biomass for native populations. Thus, possible demographic release can be occurring in the introduced range of the European green crab (Torchin *et al.* 2001). The cane toad (*Rhinella marinus*), which was introduced in Northern Queensland Australia in 1929, may also be presenting a demographic release, for two reasons: (1) introduced populations densities are up to two order of magnitude higher than native populations (Lampo & DeLeo 1998) and; (2) introduced populations of cane toads have only a subset of their native parasites, lacking ectoparasites, which presumably controls toad density in the native populations of South America (Lampo & DeLeo 1998), as well as helminth parasites, with introduced populations harboring less than 30% of the native helminth parasite assemblage (Barton 1997).

There is a clear evidence of parasite release for several NIS with most introduced populations presenting significantly less parasite species and prevalence when compared with native populations. Support was also found, for some species, of a strong association of parasite and pathogen release with host's demographic performance (*i.e.* enhanced body size and higher density). Since these patterns occur

Table 2. Evidence of demographic release for several non-indigenous species and if there were any correlation with parasite release. NA represents that the information was not available (adapted from Lafferty *et al*. 2005).

Tabela 2. *Evidência de liberação demográfica para várias espécies invasoras e se havia correlação com liberação de parasitas. NA indica que a informação não estava disponível (adaptado de Lafferty* et al. *2005).*

Taxonomic group	Species	Demographic parameter compared	Mean % increase	Parasite release?	Source
Plants	*Prunus serotina*	Density (m^2)	83	+	Reinhart *et al.* 2003
Crustacean	*Carcinus maenas*	Biomass (Kg)	59	+	Torchin *et al.* 2001
	Carcinus maenas	Mean size (mm)	29	+	Torchin *et al.* 2001
	Orconectes rusticus	Growth rate (g)	137	NA	Pintor & Sih 2009
	Rhithropanopeus harrisii	Carapace width (mm; males)	110	NA	Czerniejewski 2009
	Rhithropanopeus harrisii	Carapace width (mm; females)	108	NA	Czerniejewski 2009
Marine Invertebrates	15 species	Max. size (mm)	19	NA	Grosholz & Ruiz 2003
	4 species	Max. size (mm)	-4	NA	Grosholz & Ruiz 2003
Fish	*Cephalopholis argus*	Mean size (total length mm)	11	+	Vignon *et al.* 2009
	Cephalopholis argus	Fulton's condition index	44[a]	+	Vignon *et al.* 2009
Amphibian	*Rhinella marinus*	Density ($100m^2$)	3325[b]	+	Lampo & DeLeo 1998
Amphibian	*Eleutherodactylus coqui*	Density ($400m^2$)	222	+	Woolbright *et al.* 2006
Mammals	6 species	Density (Km^2)	424	+	Freeland 1993 cited in Lafferty *et al.* 2005

a= data based on medians using the highest value in Figure 2 of Vignon *et al.* 2009

b= data based on medians

for many different taxa, they provide substantial support to the hypothesis that coevolved parasites are capable of affecting hosts demographic parameter. However, given the complexity of parasite-host dynamics, more studies on the association of parasite release with hosts' demography are still needed. More specifically, it is very difficult to quantify how the number of parasites species, their abundances, if they are generalist or specific, if they are regulatory or compensatory, and their virulence are associated with demographic release.

PARASITE INTRODUCTION - BIOLOGICAL CONTROL

As seen above, NIS are released from the controlling influence of their coevolved parasites and can become pests because the release of parasites is usually associated with demographic release. A way of reducing the number and density of pests is by using management tools such as biological control to reduce the abundance of NIS to a tolerable level. There are several methods of biological control, such as habitat manipulation, introduction of predators and pathogens and fertility control (Hygnstrom *et al.* 1994). In the case of NIS, Dobson (1988) suggests that the use of parasites as a control measure should be very effective because NIS are expected to present high population densities and low genetic variability. Apart from the practical reason of studying the control of pest populations with the use of infectious agents, the intentional introduction of parasites and pathogens can also serve as field experiments of how infectious agents can control host populations and how host specificity, parasite virulence and behavior works. Indeed, it has been shown that infectious agents, when successful, can in fact reduce the abundance of NIS to acceptable economical and ecological levels (Lafferty *et al.* 2005). Also, in the case of NIS species, it would be interesting to see if parasite release is associated with a reduction in the host's resistance to parasite (Dobson 1988).

Biological control agents rarely eradicate or extinguish the target pest, although it has frequently managed to reduce and control several host populations (Lafferty *et al.* 2005). Differences in success of different infectious agents are expected, since both target taxa and infectious agents vary in relation to ecological (habitats, functional group, competition among infectious agents, transmission parameters) and evolutionary features (life histories, host-specificity, virulence). With such a complex system, which parasites and pathogens would be the best for controlling targeted pests?

To begin with, natural enemies with high host specificity and habitat fidelity are preferred, since they will ensure that impact, or at least most of the impact, will be directed at the targeted pest species. Thus, guaranteeing lower occurrence, if any, of host shifts (Hoddle 2004). Also, we have to think about how virulent or pathogenic the parasite must be. According to Anderson's (1979) theoretical model, parasites of moderate to low virulence are more suited, because they present higher reduction on population density. There are many other important aspects of host-parasites dynamics that can be studied using biological control as experiments, such as transmission parameters, if there are any differences in the dynamics of micro (bacteria, viruses, protozoans) or macro-parasites (parasitic helminths and arthropod) and host's immune responses. However, the intention here is not to review those issues, but to show that by studying biological control, both cases that failed and succeeded, can help to better understand population dynamics of host-parasites. For example, several biological controls have produced unintended effects on non-target species, with host shifts being the most notoriously common problem (Hoddle 2004). This gives us the opportunity to look at how evolutionary interactions work, such as the interaction between selective pressures (e.g. parasite resistance) and host-parasite population dynamics, thus, how virulence and host defenses evolve (Lafferty *et al.* 2005). Also, other aspects can be looked at, such as how important is host encounter for the maintenance of the parasite. It has been shown for several parasites that a wide range of compatible host can be infected, even for host-specific parasites, under laboratory conditions. However, in nature, parasites will usually infect fewer individuals than under laboratory conditions, suggesting that host encounter can play a role in host specificity (van Lenteren *et al.* 2005).

For example, the introduction of myxomatosis in Tierra del Fogo, Chile, and Australia had different effects on their invasive rabbit population. While in Chile the European rabbit pest was decimated (Jaksic

& Yáñez 1983), the introduction of myxomatosis in Australia did not have the same effect. Initially, the virus was very effective, with an estimated mortality rate of 99%, but its virulence was soon attenuated and rabbits started to present rapid genetic resistance to myxomatosis (Fenner 1983). One of the possibilities is that myxomatosis was too virulent to persist in populations of rabbits in Australia, while in Chile, native rabbit species were asymptomatic to myxomatosis (Fenner 1983, Jaksic & Yáñez 1983), providing a reservoir for this infectious agent and therefore the perpetuation of a highly virulent virus. The introduction of a feline parvo virus was responsible for a reduction of 78% in feral cats at Marion Islands (van Rensburg *et al.* 1987), but the total eradication of feral cats in Marion Islands only happened because hunting and trapping were used after the infection of the parvo virus (Nogales *et al.* 2004). These examples support the idea that an infectious agent is unlikely to extirpate or extinguish its host completely, as theoretical models already hypothesized, especially if transmission is density-dependent (Anderson & May 1978, May & Anderson 1978, Anderson 1979).

Nevertheless, the use of infectious agents is a powerful way of reducing NIS to less harmful densities, if the necessary evaluations such as host-specificity are carefully examined. For example, sexually transmitted diseases may guarantee higher host specificity for vertebrate pests such as mammals, which are usually harder to control with other techniques because of their higher learning capacity and more inconspicuous nature (Hoddle 2004). In addition, sexually transmitted diseases are commonly frequency-dependent, which means that their prevalence can increase even when host population density is low, and could possibly cause host extinction. Another possible way for host extinction to occur is if reservoir hosts are present, since they serve as a source for pathogen epidemics to occur (McCallum & Dobson 1995), like in the case of myxomatosis on rabbit populations of Chile. These are some of the examples and inferences that can be made by studying the introduction of infectious agents as control agents for NIS. These inferences are important because infectious diseases can be a threat to endangered species (Smith *et al.* 2009), such as the on-going decline of global amphibian populations because of chytridiomycosis (Daszak *et al.* 1999, 2003). Thus, with a clearer view of host-population dynamics it is possible to apply the best control strategy, not just to control NIS density but also to reduce the negative effects (*i.e.* disease-mediated extinction) that emergent diseases have upon native wildlife (Smith *et al.* 2009).

SPILLBACK AND SPILLOVER

NIS are also responsible for the emergence of infectious diseases in their introduced range, the most common cited mechanism being the 'spillover' of their parasites onto the native community (Daszak *et al.* 2000, Prenter *et al.* 2004). The introduction of these newly infective agents can cause severe impacts to naïve host populations with decimating effects (Anderson & May 1986, Daszak *et al.* 2000), for example there were several extinctions of Hawaiian native birds associated with the introduction of avian malaria and avian poxvirus (van Riper *et al.* 1986, 2002). Additionally, NIS can acquire native parasites from their novel range and act as reservoirs for these native infectious agents, which can increase incidence of infection (number of new hosts that become infected; see Bush *et al.* 1997) on native hosts because of the 'spillback' of infection to native fauna (Figure 1; Daszak *et al.* 2000). Even though NIS usually lose their parasites, as shown above, they have a tendency to be infected by generalist parasites from the native fauna of the introduced range (Poulin & Mouillot 2003, Kelly *et al.* 2009).

Spillback can be seen as a form of 'apparent competition', which is a situation where two or more species negatively affect one another indirectly, for example through their interaction with a common predator, but in this case with a common parasite (Hudson & Greenman 1998). However, parasite spillback has usually been overlooked in reviews on parasitic role in NIS (Kelly *et al.* 2009). But a recent review by Kelly *et al.* (2009) on parasite acquisition by NIS of 40 introduced animals found that a mean of 6.3 native parasites species were acquired per host with most parasites (67%) belonging to the native parasite community. In addition, 38 out the 40 animals studied had acquired generalist parasites. Therefore, the potential for parasite spillback exists, especially because in some cases the NIS were not

only competent hosts but were also better hosts, since the reproductive rate of the parasites were higher when they infected NIS hosts (Kelly *et al.* 2009).

A possible example of parasite spillback is the displacement of the house gecko (*Lepidodactylus lugubris*) from the Pacific islands, by the introduced *Hemidactylus frenatus* (Kelly *et al.* 2009). The introduced gecko (*H. frenatus*) acquired four parasites from *L. lugubris*, and body condition of the native gecko was negatively correlated with the intensity of infection of *Cylindrotaenia* (Cestoda). The prevalence of *Cylindrotaenia* was higher for *L. lugurbis* when it occurred in sympatry with the introduced gecko (13%) than when in allopatry (5%) (Hanley *et al.* 1995, cited in Kelly *et al.* 2009). Thus, spillback can be a possible reason for the displacement of the native gecko.

The study of introduced diseases is generally focused on spillovers, since it is generally assumed that impacts of native parasites on NIS are of no importance (Kelly *et al.* 2009). One of the possible reasons for the relatively high numbers of studies on spillovers is the increase in global trade, which has allowed infectious diseases to cross many biogeographical areas with significant negative impacts (Daszak *et al.* 2000; Lafferty *et al.* 2005). Also, an enormous amount (60.3% of events) of emerging infectious diseases in humans are caused by zoonotic pathogens, many of which have a wildlife origin (Jones *et al.* 2008). I will now present five case studies of introduced infectious diseases and their effects on native communities and one case of an introduced disease that occurs in both birds and humans.

AVIAN POXVIRUS AND AVIAN MALARIA IN HAWAII

NIS have been a problem in Hawaii ever since the arrival of Europeans (Sax *et al.* 2002). Factors such as habitat destruction by humans and introduced ungulates (cattle, sheep and goats), introduced predators (feral house cat and the mongoose *Herpestes javanicus*) and competition among NIS and native fauna have played their role in reducing biodiversity in Hawaii. However, these were not the primary cause of bird extinctions, instead it was hypothesized that it was mainly due to avian diseases (Warner 1968). Indeed, the extinction of many bird species is correlated temporally with the introduction of avian malaria and avian poxvirus (van Riper *et al.* 1986, 2002). In addition, many bird species are restricted in both abundance and distribution, with native birds mainly occurring at higher altitudes where the introduced vector *Culex quinquefasciatus* (probable vector of both avian malaria and poxvirus) occurs at low densities (van Riper *et al.* 1986). Also, native birds were more susceptible to avian malaria and poxvirus, presenting significantly higher intensity of infection (number of individuals of a particular parasite species in a single host; see Bush *et al.* 1997) levels and prevalence than introduced bird species, as well as higher morbidity and mortality (Warner 1968, van Riper *et al.* 1986, 2002).

It seems that vectored blood parasites played a primary role in the extinction of Hawaiian endemic bird fauna. In addition to the important effects that these avian diseases have on Hawaiian avifauna, the studies have also helped to unveil several interesting attributes of vector transmitted diseases. For instance, communities with multiple hosts sharing a common parasite are liable to experience the extinction of certain host species, just as long as other host reservoirs are maintained in the community. In the case of the Hawaiian avifauna, introduced birds are not fully affected by avian malaria and poxvirus, thus the infectious agents remain common and possibly highly virulent. Another important aspect is the difference in susceptibility of native and NIS of birds to parasites, demonstrating the vulnerability of naïve hosts to new infectious agents, and therefore the importance of evolution in hosts' defenses to limit these impacts. Last but not least, vector distribution can change host distribution, in this case host distribution was a direct reflectance of vector distribution with host species being confined to high altitudinal areas where vector density was lowest (Lafferty *et al.* 2005).

RINDERPEST IN AFRICA

Rinderpest was introduced into Africa in 1889 and within a year this highly pathogenic morbillivirus infected various ungulate native species. The panzootic front travelled at a rate of 500 km per year with severe impacts on native and domestic ungulate species. For instance, it is estimated that the virus

decimated 95% of both wildebeest (*Connochaetes taurinus*) and buffalo populations (*Syncercus caffer*) of the Serengeti and 90% of the domestic cattle population of East Africa (McCallum & Dobson 1995, Daszak *et al.* 2000, Plowright cited in Lafferty *et al.* 2005). Consequently, this huge population decline of ungulates had also secondary effects on predator populations and plant composition and biomass (Lafferty *et al.* 2005).

The wild ungulates were blamed for the epidemics, which led to the slaughter of wildlife ungulates near cattle farms. However, after the development of the rinderpest vaccine in 1950, it was established that the domestic cattle were in fact the main reservoir, since soon after the cattle were vaccinated the disease disappeared (Lafferty *et al.* 2005). This shows how important reservoir hosts are in maintaining and spreading diseases to other

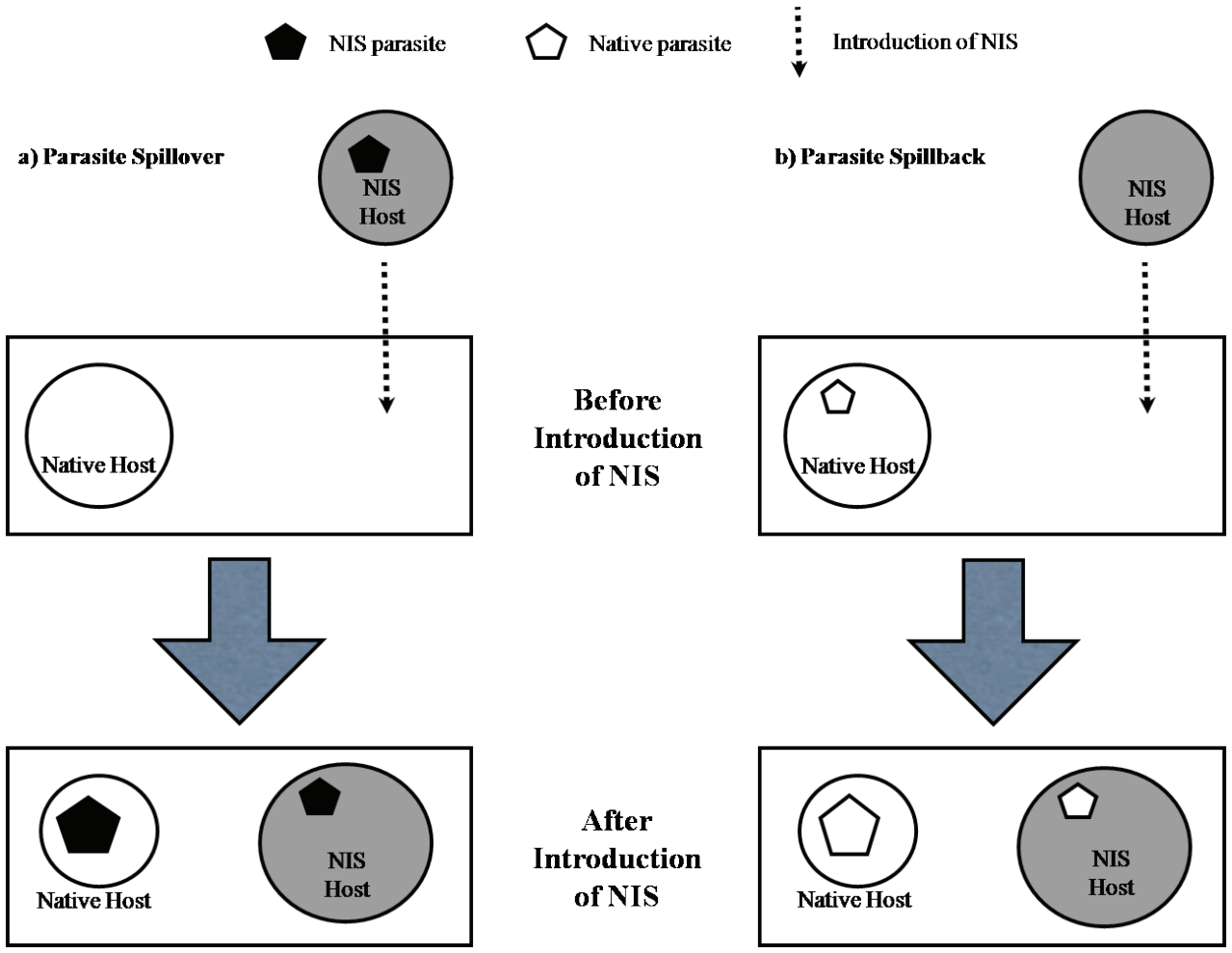

Figure 1. Representation of (a) 'parasite spillover' and (b) 'parasite spillback'. Black hexagonal represents parasites of non-indigenous species (NIS) and white hexagonal represents parasites from native hosts; size of hexagonal is related to infection burden. The size of the circles represents the host's population size or density (filled circles representing NIS hosts and white circles representing native hosts). After the introduction of the NIS, native host can be infected with parasites that were introduced together with the NIS (a) or (b) NIS can serve as reservoir host to native parasites already present (adapted from Kelly *et al*. 2009).

Figura 1*. Representação de (a) 'parasite spillover' e (b) 'parasite spillback'. Hexágonos pretos representam parasitas da espécie invasora enquanto que hexágonos brancos representam parasitas dos hospedeiros nativos; tamanho do hexágono está relacionado ao nível de infecção. O tamanho do círculo representa o tamanho populacional ou a densidade do hospedeiro (círculo cinza representa hospedeiros introduzidos enquanto que círculo branco representa hospedeiros nativos). Após a introdução da espécie invasora, hospedeiros nativos podem ser infectados por parasitas que vieram junto com a espécie invasora (a) ou (b) as espécies invasoras podem servir como hospedeiros reservatórios para parasitas nativos já presente (adaptado de Kelly* et al*. 2009).*

species, which can dramatically affect rare species as well as reduce population sizes of abundant species (Lafferty & Gerber 2002). In this case it also seems that subtle co-evolutionary forces were involved in the development of the disease. Rinderpest was relatively benign to ancient cattle hosts (original host for the introduced strain), but when it passed through sequential wildebeest or buffalo hosts it is possible that its virulence towards these species was attenuated while its virulence to cattle increased (McCallum & Dobson 1995). Consequently, in areas were cattle and wildlife met, it seems that both would be potential reservoir for virulent strains of rinderpest (Lafferty & Gerber 2002). This demonstrates that parasite evolution may occur at a much faster time scale than host evolution, with parasites presenting new characteristics in a time frame that for the host could still be considered as ecological time (Horwitz & Wilcox 2005). After the vaccination in the 1950s and 1960s, wildebeest and buffalo populations began to increase very rapidly because of the reduction in juvenile mortality, thus implicating rinderpest as the cause of ungulate population decline.

Increase in abundance of most ungulate species after rinderpest control was associated with an increase in lion (*Panthera leo*) and hyenas (*Crocuta crocuta*) populations. This had severe consequences in others species such as gazelles who presented a population decline, probably because of higher predation pressure, and wild dogs (*Lycaon pictus*), which declined drastically in numbers due to increased competition with lions and hyenas (Lafferty *et al.* 2005). Also, alteration in plant composition and biomass occurred due to increase in the abundance of grazers. Therefore, it is possible that certain parasites act as keystone species, since marked shifts in community composition and ecosystem functioning can occur after modification of host population size (Horwitz & Wilcox 2005, Lafferty *et al.* 2005).

CHYTRIDIOMYCOSIS AND AMPHIBIAN DECLINE

Chytridiomycosis, which is caused by *Batrachochytrium dendrobatidis* (Bd) an epidermal fungus within the phylum Chytridiomycota, is responsible for major declines in amphibian population in Australia, Europe, North and Central America (Daszak *et al.* 1999, 2003, Kilpatrick *et al.* 2009). This fungus is considered as an emergent disease because it has managed to spread its range recently (over the past couple of decades) and it has also increased in impact with the occurrence of mass mortalities and local extinctions in Australia, New Zealand and North America (Daszak *et al.* 2003).

Preliminary evidence indicates that Bd has most likely been introduced recently in different regions, due to recent movement of Bd between disparate populations of amphibians. Also, isolates from different continents presented identical genetic sequences or poor correlation between origin and position on the phylogenetic tree (Daszak *et al.* 2003, Kilpatrick *et al.* 2009). Thus, it seems that pathogen pollution, which is the anthropogenic introduction of disease to new regions and host species, or simply human mediated pathogen invasion (Daszak *et al.* 2000, Dobson & Foufopoulos 2001), is responsible for the expansion of Bd. Pathogen pollution, like invasive species, can have severe consequences to the ecosystem with significant biodiversity loss (as seen in the earlier examples). For instance, pathogen pollution can cause population crashes, which severely reduce population size of naïve and new host population. It is also possible for introduced pathogens to become enzootic, with initial population declines being followed by chronic depopulation, which could give rise to local extinction if the threshold host density for disease transmission is lowered (Daszak *et al.* 2000) or due to increased likelihood of smaller populations to stochastic events.

In support of the view that Bd is an introduced pathogen, chytridiomycosis has been recognized in different forms of amphibian trade such as laboratory, pet and food trade (Daszak *et al.* 2003). For example, Bd can infect both the American bullfrog (*Rana catesbeiana*) and the African clawed frog (*Xenopus laevi*), with individuals of the former species being relatively resistant to chytridiomycosis (Mazzoni *et al.* 2003, Daszak *et al.* 2004), while the African clawed frog can carry Bd asymptomatically (Rachwoicz *et al.* 2005). The African clawed frog was widely used for pregnancy testing in Europe, Australia and North America in the 1940s and 1950s (Rachowicz *et al.* 2005), while the American bullfrog is farmed for food (Mazzoni *et al.* 2003). Both of these species could be responsible for the global spread of chytridiomycosis,

for instance over 1 million bullfrogs are imported into the United States from South American and Asian farms and are transported alive (Mazzoni *et al.* 2003).

Daszack *et al.* (2003) proposed a theoretical scenario that involved both host's ecological traits (naïve populations of high altitude, stream breeding, low fecundity, habitat specialist amphibians) and Bd's biological traits (more rapid growth in cool temperatures, wide host range, high virulence, potential survival outside the host) to explain why chytridiomycosis has such devastating effects. More specifically, it was shown: populations that declined tended to be naïve; disease impact was higher on low fecundity species; Bd has a wide host range encountered in 13 amphibian families including frogs, toads and salamanders, both in the wild and captivity; variability in host susceptibility, being highly pathogenic for several species of amphibians while it appears not to be virulent in other species; and Bd is capable to persist when host density is low because of asymptomatic infections of larvae and the presence of reservoir hosts, for instance *R. catesbeiana* have been widely introduced around the globe (Daszak *et al.* 2003).

The impact caused by chytridiomycosis may be enhanced because of secondary or 'knock-on' effects on native communities that are hard to predict (Daszak *et al.* 2000). It is possible that chytridiomycosis may increase the success of invading host species such as *R. catesbeiana and X. laevi* because of enhanced parasite mediated competition (McCallum & Dobson 1995, Hudson & Greenman 1998, Horwitz & Wilcox 2005). Other effects on rain forest ecology are also expected because of local extinction of several amphibian species, but have yet to be assessed (Daszak *et al.* 1999, 2000).

CRAYFISH PLAGUE

Crayfish plague was introduced in Europe in 1860 and was responsible for drastic declines in native crayfish in the beginning of the 20th century. The disease is caused by a fungus-like agent (*Aphanomyces astaci*), and it seems that American crayfish such as *Pacifastacus leniusculus*, *Procambarus clarkii* and species of the genus *Orconectes* are probable vectors of *A. astaci* (Edgerton *et al.* 2004). These American crayfish are resistant to *A. astaci* and will only succumb to a full infection under stressful conditions (Philips *et al.* 2007). In Europe, American crayfish have been widely introduced with the intention of replacing native populations of crayfish that were extirpated because of overexploitation, but also unintentionally, for example via ballast water and individuals that managed to escape from farms (Harlioglu & Harlioglu 2006). Therefore, American crayfish species can act as carriers of the disease and it is possible that *A. astaci* was introduced in conjunction with the North American species of crayfish (Edgerton *et al.* 2002).

Displacement of native European crayfish species by the invasive American crayfish can occur through competitive exclusion (Dunn *et al.* 2007) but parasite mediated competition can also be a possible mechanism, since invasive crayfish in Europe are resistant to *A. astaci*, giving these invasive species a competitive edge over the sympatric native crayfish species. The occurrence of reservoir hosts can have important implication in the host-parasite system, for instance parasites can be highly virulent and transmission rate will not depend on the density of a single host species (Laffery & Gerber 2002). In the case of the crayfish plague, reservoir hosts are important because zoospores are usually motile for only up to three days. Also, these zoospores are usually released from zoosporangia of infected crayfish when it is moribund and has recently died (Edgerton *et al.* 2002). Thus, without reservoir hosts it would be very difficult for the disease to maintain itself highly virulent, because zoospores would probably not manage to encounter hosts in time to fulfill its life cycle in declining host populations. However, other pathways of spread can also take place, for instance, boats that have not been dried between watersheds and water containers been emptied in other watersheds or collecting nets and equipment (Edgerton *et al.* 2002).

EFFECTS OF PARAPOXVIRUS IN RED SQUIRRELS AND GREY SQUIRRELS IN THE UK

The grey squirrel (*Sciurus carolinensis*), which is native to North America, was introduced to the United Kingdom in 1876 and rapidly spread and replaced its congener the red squirrel (*S. vulgaris*) (Teangana *et al.* 2000). The subsequent decline of the red squirrel, followed by the rapid expansion of the grey squirrel,

was attributed to competition among the species for food resources (Okubo *et al.* 1989, Bryce *et al.* 2001). Tompkins *et al.* (2003) presented evidence that, in general, the grey squirrel is a stronger competitor than the red squirrel, since grey squirrels have higher growth rates, higher carrying capacity and higher reproductive rates. These researchers modeled several population parameters to elucidate if interspecific competition was the primary cause for the ecological replacement of the red squirrel by the grey squirrel. As expected, the theoretical model indicated that the grey squirrel would manage to replace the red squirrel population in a matter of 15 years. However, when the model was compared with historical data on the expansion of grey squirrels, it did not have a very good fit, since the model's rate of replacement was much lower than the level observed for the available field data. On the other hand, when the model incorporated both interspecific competition and parapoxvirus, it had a very close fit to the available field data. This more detailed model indicated that the ecological replacement of red squirrel would occur in a matter of six years, suggesting that the parapoxvirus leads to a much faster rate of decline, having a close fit to the actual available data.

The enhancement of the decline of red squirrels due to infection of parapoxvirus is pretty intuitive. To begin with, parapoxvirus is highly pathogenic to red squirrels, while grey squirrels are resistant to the virus acting as reservoir hosts (Tompkins *et al.* 2002). When seropositive grey squirrels enter a disease-free area, an outbreak of parapoxvirus in the red squirrels follows. This is supported by the fact that the occurrence of parapoxvirus in red squirrels only takes place in geographical areas where seropositive grey squirrels occur (Sainsbury *et al.* 2008). Furthermore, the disease outbreak leads to a population crash of red squirrels, which in turn reduces the competition pressure on grey squirrels, because more food resources will be available, enabling the increase in numbers of the invading grey squirrels (Tompkins *et al.* 2003). This example reveals the marked effects that introduction of an NIS can have on native species populations because of changes in parasite relationships because of parasite pollution. More importantly, this example shows that attenuation of the virus towards the red squirrel is not happening, probably because of the presence of the grey squirrel, which is an asymptomatic reservoir host (Sainsbury *et al.* 2008). In a similar situation, the introduction of a poxvirus (myxomatosis) in the introduced rabbit in Australia, had devastating effects, but over a short period of time the virus and the rabbit hosts adapted in relation to each other and milder infectious became commonly widespread (Fenner 1983). The attenuation of the myxomatosis virus in Australia happened because no asymptomatic reservoir host was present, if one was, one would expect to see the same effects found for the red squirrel-parapoxvirus example. As expected, myxomatosis did in fact extirpate the introduced rabbit population in Tierra del Fogo, Chile, because native rabbit species were asymptomatic hosts to myxomatosis (Fenner 1983, Jaksic & Yanez 1983).

WEST NILE VIRUS FROM BIRDS TO HUMANS

West Nile Virus (WNV), which is a member of the Family Flaviviridae, has a very widespread distribution in the world occurring in all the continents except Antarctica. Several major outbreaks of the virus have occurred in Africa, Eurasia, Australia and the Middle East (Kramer *et al.* 2007). A more recent outbreak happened in New York City in 1999, where the virus was responsible for the deaths of American crows (*Corvus brachyrhynchos*), fish crows (*C. sifragus*) and several exotic birds from the Bronx zoo, as well as the cause of human encephalitis (Lanciotti *et al.* 1999). This WNV was closely related to a WNV isolated from a dead goose (*Anser* sp.) in Israel and was associated with high pathogenicity for some bird species but not for others (Lanciotti *et al.* 1999, LaDeau *et al.* 2007). It is not yet clear how the virus was introduced to New York, but it is speculated that the virus might have entered through a number of mechanisms such as the pet trade or illegal importation of birds, unintentional introduction of virus-infected mosquitoes or simply by infected human travellers (Lanciotti *et al.* 1999). Nonetheless, this single point introduction is responsible for the current epidemic of WNV in North America and is currently expanding its introduced range in the Western Hemisphere and has already encompassed all 48 lower states of the United States and reached several countries in Central and South America (for reviews see Kilpatrick *et al.* 2007, Kramer *et al.* 2007; CDC 2009).

This spread of WNV happened very quickly (less than 10 years) and is probably associated with the fact that WNV is an ecological generalist virus with many host species (326 bird species; over 30 species of mammals) and vector species (at least 62 species of mosquitoes) (Marra *et al.* 2004, Kilpatrick *et al.* 2007, Kramer *et al.* 2007, CDC 2009). This ecological generalization has probably determined the broad geographical distribution of the virus as well as the high pathogenicity for some animal species and humans (Kramer *et al.* 2007). Although the virus presents wide host and vector range, WNV is believed to be maintained by a bird-to-mosquito-to-bird cycle with *Culex* sp. being the main vector, and migratory birds and human transport (*i.e.* inside planes and train cargos) as possible dispersal pathways for the virus (Rappole *et al.* 2000, Kilpatrick *et al.* 2007, Kramer *et al.* 2007). *Culex* species also feed on both mammals and birds making them a possible bridge vector between bird and human infections (LaDeau *et al.* 2008).

The major concern with WNV is not just the fact that the virus can infect humans, but when it does, it can present high pathogenicity. During 1999 and 2006, 1008 deaths were reported for the United States and Canada with over 20,000 reported cases (Kilpatrick *et al.* 2007). In USA, during 2007 and 2009, 198 fatalities were reported with over 5,000 reported cases of WNV (CDC 2009). Furthermore, the introduction of WNV has led to a substantial decline of seven birds species, being most marked for the American crow (*Corvus brachyrhynchos*), which presented a population decline of up to 45%. Thus the impact of WNV on avian communities should present substantial ecosystem effects (LaDeau *et al.* 2007). For instance, threatened and endangered species could be at serious risks of extinction because of WNV, especially avifauna of islands such as Hawaii, which could be immunologically naïve to the virus, as several species were to avian malaria and smallpox (Marra *et al.* 2004, LaDeau *et al.* 2008). Other indirect consequences may also emerge, such as changes in avian mediated ecosystem services like seed dispersal, nest predation and regulation of insect population (LaDeau *et al.* 2008). These impacts can have serious problem for ecosystem functioning. In addition, impact of WNV can be more problematic because it is a generalist pathogen and therefore, its impact will persist in time and space, since they are not limited by a single host population (Daszak *et al.* 2000, LaDeau *et al.* 2008).

CONCLUSIONS

Ecological theory and mathematical models have been of great help in the understanding of parasite-host dynamics. Not only that, they have also been of great use in controlling and preventing diseases as well as helping with public health policies (Smith *et al.* 2005). For example, theoretical ecology made important contributions to the understanding of the HIV/AIDS epidemiology. Models showed that the rate of spread is related to the variance of the number of sexual encounters of infected individuals. Also, that needle exchange programs (distribution of clean needles to drug users) would be a good policy in the attempt to reduce the spread of HIV (Smith *et al.* 2005).

Theory has also helped to understand disease outbreaks in agricultural communities and how to cope with them, such as the foot and mouth disease outbreak in the UK. In this outbreak, ecologists were responsible for calculating the basic reproductive rate of a pathogen (R_0) and for educating the media and the general public about the importance of reducing R_0. To accomplish this reduction, a rigorous campaign was organized to identify infected farms so the herds of these farms could be quickly culled (Smith *et al.* 2005). In natural communities, infectious diseases have been proposed as a casual threat of animal extinction (Wilcove *et al.* 1998), but is seems that infectious diseases only play a minor role in animal extinction (Smith *et al.* 2006). However, Smith *et al.* (2009) argued that there is now ample evidence that diseases can greatly impact local species population by temporarily or permanently reducing their abundance. Also, pathogens can interact with other factors such as habitat loss, climate change, invasive species (Laffery *et al.* 2005, this study), pollution and habitat alteration to drive species to extinction both locally and globally (Lafferty & Kuris 1999, Lafferty & Holt 2003, Smith *et al.* 2009). For instance, pollutants may increase parasitism by reducing the immunological capabilities of hosts (McDowell *et al.* 1999). However, experimental researches on host-parasite dynamics are still needed and how diseases

can cause extinction is still poorly known (Smith *et al.* 2006).

In this review study I attempted to show some evidence, although without a strict control experiment, that parasites in fact do control host population abundance and can be important key players in the community as well as responsible for extinction of animal species. I did this by looking at the introduction of NIS, which can be seen as natural experiments of how hosts respond in the absence of parasites, and biological control studies, which can be seen as field experiments of how hosts respond to the intentional introduction of parasites. Indeed, these studies have shown that parasites can affect host performance, such as biomass, body size and density, characteristics associated with population performance. In addition, several studies have demonstrated that species can go extinct if the disease can affect several host species at the same time and that parasites can play a keystone role in the community, through apparent competition, which can lead to considerable secondary effects. Furthermore, evolutionary aspects of host-parasite dynamics are also important, since naïve hosts can be extirpated by infectious agents. Lastly, diseases can change the geographical distribution of hosts, since hosts will be absent in areas of high infection risk, which were once part of the hosts range before the emergence of disease.

Parasitical studies of NIS with experimental approaches are needed to better understand host-population dynamics. Also, invasive NIS are major threats to biodiversity and a better understanding of host shifts and host specificity will help to eradicate these species, or to at least reduce them to tolerable levels. Therefore, parasitical studies of NIS can help to elaborate better control programs for NIS, as well as to help prevent emergence of infectious wildlife diseases, which can play a major role in biodiversity alteration.

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