



## Perspective

## The hidden consequences of altering host-parasite relationships during fauna translocations

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## ABSTRACT

Host-parasite relationships are complex, and in wild animal populations individuals are commonly co-infected with various parasite species or intraspecific strains. While it is widely recognised that polyparasitism has the potential to reduce host fitness and increase susceptibility to predation or disease, the role of polyparasitism in influencing translocation success has never been investigated. Here we review the consequences of translocation for the host-parasite infracommunity and demonstrate how translocation-induced perturbations to within-host-parasite relationships may exacerbate the negative impacts of polyparasitism to the detriment of host health and translocation success. We also consider the ecological and immunological effects of altering host-parasite assemblages during translocation, and illustrate how the use of anti-parasitic drugs can further modify parasite infracommunity dynamics, with unintended impacts on target and non-target parasites. Importantly, as the evolutionary and ecological significance of the host-parasite relationship is increasingly recognised, we discuss the benefits of conserving parasites during fauna translocations.

## 1. Introduction

Fauna translocations have become a widely employed conservation tool for the management of threatened species worldwide (IUCN, 2013). Translocations for conservation are occurring at an ever-increasing frequency (Seddon et al., 2007) with their value extending beyond conservation management, by also benefiting conservation and biological research, ecosystem restoration and the wider human community (Parker, 2008). Despite their pertinent role, the success rate of fauna translocations remains poor (Fischer and Lindenmayer, 2000). In a recent assessment of species relocations within Australia (Sheean et al., 2012), only 46% were successful. While there are a range of factors influencing translocation success, it is increasingly (albeit inconsistently) recognised that parasites (using the term broadly to include viruses, bacteria, fungi, protozoa, helminths and arthropods; Viney and Graham, 2013) impose a risk to host fitness and translocation success (Griffith et al., 1993; Viggers et al., 1993; Cunningham, 1996; Sainsbury and Vaughan-Higgins, 2012).

Hosts are usually infected by multiple parasite species (polyparasitism). In essence, therefore, fauna translocations involve the movement of complete “biological packages” from one ecosystem to another, during which the disruption of normal host-parasite

relationships can have various outcomes for both the host and the parasites it carries (Corn and Nettles, 2001; Telfer et al., 2010; Moir et al., 2012). In contrast to the widely recognised disease risks associated with translocating wildlife (see Table 1 for examples), the way in which fauna translocations disrupt the dynamics of within-host parasite communities (infracommunities) is far less well understood, as is the impact of such perturbations on host fitness and translocation success.

We know for example, that a translocated host can acquire novel parasites (using the term “novel” to refer to any parasite that an individual has not previously encountered) within the destination site with devastating consequences for host health and survival. What we don't know is the mechanism behind this outcome. The presence of both canine distemper virus (CDV) and *Sarcoptes scabiei* (mange) reduced pack growth rates of Yellowstone's reintroduced grey wolves (*Canis lupus*), and in severe cases mange was associated with pack extinctions (Almberg et al., 2012). While host density and connectivity appeared to influence the spatio-temporal spread of sarcoptic mange, there were some packs that remained mange-free despite close proximity or territorial overlap with other infected packs. Likewise the spread and severity of mange varied among individuals within the same pack.

Inconsistencies such as these raise a number of important questions with regard to host-parasite dynamics and resistance to infection within

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**Table 1**  
Examples of disease transmission risks during fauna translocations.

Risk	Example	Effect/outcome	References
Translocated host introduces novel parasite into naïve wild population	Parapoxvirus introduced into the United Kingdom by grey squirrels ( <i>Sciurus carolinensis</i> )	Debilitating skin disease, catastrophic mortality and local extinction of native red squirrels ( <i>Sciurus vulgaris</i> )	Tompkins et al. (2002) Sainsbury et al. (2008)
	Crayfish plague ( <i>Aphanomyces astaci</i> ) introduced into Europe by infected North American crayfish.	Local extinction of native European crayfish ( <i>Austropotamobius pallipes</i> ).	Holdich and Reeve (1991) Prenter et al. (2004)
Translocated host exposed to local endemic parasite	Caribou ( <i>Rangifer tarandus</i> ) and moose ( <i>Alces americana</i> ) translocated into areas inhabited by white-tailed deer ( <i>Odocoileus virginianus</i> ) exposed to the meningeal worm <i>Parelaphostrongylus tenuis</i> .	Major morbidity and mortality among translocated hosts due to the development of cerebrospinal nematodiasis (the meningeal worm does not affect local white-tailed deer, which have coevolved with this parasite).	Anderson (1972) Viggers et al. (1993)
	North American elk ( <i>Cervus canadensis</i> ) translocated into the Gila Forest, New Mexico, exposed to the arterial worm ( <i>Elaeophora schneideri</i> ), which is endemic in local mule deer ( <i>Odocoileus hemionus</i> )	Morbidity (blindness, neurological symptoms, facial gangrene and abnormal antler growth) and mortality of elk calves (15–20% survival rate). Arterial worm infection is asymptomatic in mule deer.	Hibler et al. (1969) Viggers et al. (1993)
Parasite spillover from translocated wild host to domestic/human host and vice versa	Translocated bighorn sheep ( <i>Ovis canadensis</i> ) contracted pasteurellosis from direct contact with healthy domestic sheep.	Bighorn sheep developed fatal <i>Mannheimia haemolytica</i> pneumonia.	Foreyt (1989) Kock et al. (2010)
	Brush-tailed possums ( <i>Trichosurus vulpecula</i> ) translocated from Australia to New Zealand acquired bovine tuberculosis ( <i>Mycobacterium bovis</i> ) from infected dairy cattle.	Possums became a new reservoir host for the disease (i.e. amplified parasite transmission) with significant economic consequences for the New Zealand dairy industry.	Coleman (1988) Daszak et al. (2001) Kock et al. (2010)

an individual. For example, does the presence of *S. scabiei* directly influence host health? What role does stress and immune function play in enabling *S. scabiei* acquisition and persistence, particularly in the presence of CDV? Does exposure to *S. scabiei* (or any other novel parasite) affect the abundance or pathogenicity of other pre-existing parasites within a host? One can see how this aspect of host-parasite ecology is of great importance, yet there is a lack of research evaluating how fauna translocations influence host-parasite assemblages at the individual level; and the serious, often permanent consequences of using anti-parasitic drugs to remove parasites during translocation.

Our aim in this paper is to illustrate how fauna translocations have the potential to alter within host-parasite relationships and how translocation-induced perturbations to parasite infracommunities may affect host health and translocation success. We also highlight the potential positive and negative consequences of anti-parasitic drug treatment and investigate the possible benefits of conserving parasites during translocation.

## 2. Polyparasitism and infracommunity interactions

In the past, studies have focused on the effects of single parasite species within a host (Bordes and Morand, 2011; Holmstad et al., 2005) despite polyparasitism (co-infection, multiparasitism or concomitant infection) being the norm in wild animal populations (Keusch and Migasena, 1982; Graham, 2008). Theoretical studies have suggested that polyparasitism will reduce host fitness more than single infections (Bordes and Morand, 2011). This may occur because polyparasitism can lead to competitive interactions between parasite species or strains, resulting in increased virulence, which we define as the degree of parasite-induced reduction in host fitness (Lymbrey and Thompson, 2012). For example, experimental coinfection of laboratory rats (*Rattus norvegicus*) with *Trypanosoma lewisi* and *Toxoplasma gondii* resulted in higher numbers of *T. gondii* tachyzoites compared to rats infected with *T. gondii* alone (Guerrero et al., 1997; Catarinella et al., 1998), suggesting that in co-infected hosts *T. gondii* had increased virulence.

In addition, polyparasitism has the potential to reduce host fitness and increase susceptibility to predation or disease through synergistic effects on the course and severity of infection (Irvine, 2006). Observational studies in wildlife have detected a negative correlation between polyparasitism and host body condition (Holmstad et al., 2005;

Lello et al., 2005; Jolles et al., 2008); although a causal connection has rarely been demonstrated experimentally. Gibson et al. (2011) found that California sea lions (*Zalophus californianus*) parasitised with *Sarcocystis neurona* and *T. gondii* succumbed to severe protozoal encephalitis and death, while sea lions with single *S. neurona* infections showed no disease symptoms. Likewise, domestic piglets (*Sus scrofa domestica*) experimentally infected with *Ascaris suum* and *Escherichia coli* displayed severe signs of respiratory disease and weight loss, due to migrating *A. suum* larvae transporting *E. coli* to the lungs (Adedeji et al., 1989).

On the other hand, interactions between parasites may suppress pathogenicity within a host, reducing the impact of disease. In a murine coinfection model, prior infection with the filarial nematode *Litomosoides sigmodontis* protected the host against malarial (*Plasmodium berghei*) pathology via immunomodulation (Ruiz-Fernández, 2008). Mixed trypanosome infections in woylies (*Bettongia penicillata*) also suggest that interspecific competition may sometimes be important in reducing pathogenic effects on the host (Thompson et al., 2014). Woylies initially infected with *Trypanosoma vegrandis* never developed subsequent *Trypanosoma copemani* infections, while several woylies that initially tested positive to *T. copemani*, later also tested positive to *T. vegrandis*. This is of significance because *T. copemani* appears to be more pathogenic than other trypanosome species found in woylies, because of its propensity for intracellular invasion (Botero et al., 2013).

## 3. Fauna translocations and within-host-parasite dynamics

Because wildlife are host to a variety of parasites, there is inherent difficulty in not only determining what parasite species and/or strains are present, but also predicting how these parasites interact with each other and their host during translocation (Sainsbury and Vaughan-Higgins, 2012; Aiello et al., 2014). Translocating fauna can alter existing cycles of parasite transmission among translocated hosts, and establish new transmission cycles between translocated hosts and the recipient host community, thereby altering parasite infracommunity structure and establishing new parasite interactions within hosts. To demonstrate the basic processes by which translocation may influence within-host parasite interactions, parasite persistence and host health, we provide a conceptual framework (Fig. 1), which we refer to in the

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