Which Parasites Should We Be Most Concerned About in Wildlife Translocations?

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In the context of wildlife translocations, it is important to recognize that there is a growing body of evidence that parasites are an essential component of a healthy ecosystem (Hudson et al. 2006). Parasites broadly defined (to include everything from viruses to parasitic arthropods) play an important role in regulating populations (Hudson et al. 1998; Tompkins et al. 2002a), structuring communities (Dobson and Hudson 1986; Marcogliese 2004; Tompkins et al. 2011; Hatcher et al. 2014), and adding complexity and stability to food webs (Lafferty et al. 2006; Smith et al. 2006; Wood 2007). Since ecosystem restoration is typically one of the goals of wildlife translocations, we should strive whenever possible to translocate animals with their native parasites – those that have coevolved with the host in the region and ecosystem in question. The regional context is critical, as some host species with broad geographic distribution could have a parasite that is native to one region, but not another. Such an approach could even reduce the risk of translocation failure, since movement of naïve animals without their native parasites into an environment where exposure will occur could increase the risk of parasite-induced morbidity and mortality (Almberg et al. 2012). While this approach might not be practical in all situations, the potential benefits, and minimal risk, posed by most native parasites to a stable or growing host population should be considered in any risk analysis for wildlife translocations (Gompper and Williams 1998; Gomez et al. 2012).

The primary concern, then, is with the potential for invasion and spread of *non-native* (alien) parasites – those that are either not native to the host, not native ~~or~~ to the local ecosystem, or both – with subsequent negative population-level or species-level impacts. For example, mortality associated with non-native parasites could result in a loss of genetic diversity, a decline in a population or species such that it becomes more vulnerable to stochastic events, additional parasite spillover into other susceptible populations or species, disruption of ecosystem functions, or even species extinction. Recent examples of such wide-ranging impacts include the global spread of the fungus *Batrachochytrium dendrobatidis* in amphibians (Berger et al. 1998; Fisher et al. 2009) and the invasion of the White-nose Syndrome fungus *Pseudogymnoascus destructans* in North American bats (Blehert et al. 2009; Foley et al. 2011; Fisher et al. 2012).

~~There may be other lower-level concerns with d~~Disease associated with non-native parasites~~, such as~~ could also contribute to translocation failure, impacts on individual animal health and welfare, financial or other resource losses, bad publicity, and reduced opportunities for future translocations.

While we should be concerned about the introduction of any non-native parasite because of the potential for negative impacts on populations and ecosystems, ~~the~~

~~characteristics~~some traits of non-native parasites ~~that should increase our level of concern are those that facilitate~~may increase the risk of invasion, spread, and persistence. These ~~characteristics~~ traits are explained below.

Studies of emerging human and animal diseases suggest that we should be more concerned about microparasites (viruses, bacteria, fungi, and protozoa) than macroparasites (helminths and parasitic arthropods) (Cleaveland et al. 2001; Dobson and Foufopoulos 2001; Tompkins et al. 2015). Although both categories of parasites have the potential to invade, persist, and negatively impact populations or species, the microparasites have greater potential for host-switching and adaptation because of their shorter generation times and more rapid evolution (Cleaveland et al. 2001; Woolhouse et al. 2005; Cooper et al. 2012; Engering et al. 2013). RNA viruses are an extreme example of this. Their propensity for spontaneous mutation during replication (Duffy et al. 2008) and potential for reassortment and recombination events (Simon-Loriere and Holmes 2011) enable RNA viruses to very rapidly alter their host range and virulence. The recurring emergence of Morbilliviruses, such as Canine Distemper Virus, and highly pathogenic avian influenza viruses in new hosts are good examples of this (Chen and Holmes 2006; McCarthy et al. 2007; Origgi et al. 2012; Shi et al. 2014).

In contrast, macroparasites tend to have more complex life cycles, longer generation times, slower evolution, and lower virulence overall (Cleaveland et al. 2001). This does not suggest that macroparasites should be ignored, only that they present a lower risk of invasion, epidemic spread, and persistence. Under the right set of circumstances, macroparasites can be drivers of population declines as well (Tompkins et al. 2015). One current example would be the impact of the recently introduced parasitic fly *Philornis downsi* on native birds in the Galapagos Islands (Fessl and Tebbich 2002). There have also been a number of instances where non-native macroparasites have caused significant species-level or population-level impacts in fish, such as reduced fecundity, clutch-size, and survival (Watson 2013; Lymbery et al. 2014). Nevertheless, in the majority of circumstances, microparasites pose the greatest threat.

We should also be more concerned about generalist (multi-host) parasites than specialists (Woolhouse et al. 2001). Generalists will tend to be better invaders by virtue of having a more diverse array of susceptible hosts available during invasion attempts. In addition, multi-host parasites are more prone to causing apparent competition effects (Hudson and Greenman 1998; Tompkins et al. 2000). For example, if a parasite infects two host species and one of those species is more abundant than the other, there is a risk of increased parasite exposure to the less abundant host, resulting in a greater parasite burden and more significant disease impacts. In extreme cases, this could result in population extirpation or species extinction (Tompkins and Wilson 1998; De Castro and Bolker 2005; Hudson et al. 2006). Although generalist parasites may be more prone to competitive exclusion than specialists, they still appear to be better invaders – the majority of emerging disease agents have been classified as generalists (Cleaveland et al. 2001; Tompkins

et al. 2015). Notable examples of generalist pathogens causing significant mortality in wildlife include Canine Distemper Virus in wild carnivores (Viana et al. 2015), avian malaria in Hawaiian forest birds (Warner 1968; De Castro and Bolker 2005), and *Batrachochytrium dendrobatidis* in amphibians (Fisher et al. 2009).

Parasites that have the potential to persist in a reservoir host or the environment are a significant concern because the reservoir or environment provide~~s~~ a persistent source of infection even when the alternate host's population size drops below the threshold required for sustained transmission (McCallum and Dobson 1995; Gog et al. 2002; De Castro and Bolker 2005). Ordinarily, a directly transmitted parasite would go extinct before its host because of this population threshold effect, but the presence of a reservoir turns the tables and enables the parasite to persist until the alternate host becomes extinct. In addition, parasites with reservoirs have no evolutionary pressure to adapt to lower virulence in alternate hosts because the alternate host is not required for persistence (Woolhouse et al. 2001). Furthermore, once established in a reservoir, a parasite can be difficult or impossible to eradicate. These effects of reservoirs can significantly increase extinction risk for small populations (De Castro and Bolker 2005). Squirrelpox virus, which has a reservoir in non-native grey squirrels (*Sciurus carolinensis*) in the UK has caused catastrophic mortality and local ~~extirpation~~ extinction of native red squirrels (*Sciurus vulgaris*) (Tompkins et al. 2002b; Sainsbury et al. 2008).

Vector-borne parasites present a similar concern, since the vector can function as a short-term reservoir, allowing parasite persistence even when few susceptible individuals are available. More importantly, since transmission would typically depend on the frequency of contact between the host and vector rather than on host density (McCallum et al. 2001), transmission can be very efficient even in the low density scenarios we often encounter with endangered species, potentially driving a population or species to extinction (McCallum et al. 2001; De Castro and Bolker 2005). Vector-borne parasites also have the potential to maintain higher virulence because they can continue to be transmitted from immobile hosts (Ewald 1996). Perhaps the most striking example of an non-native vector-borne parasite driving extinction is avian malaria in native Hawaiian forest birds (Warner 1968; Woodworth et al. 2005).

Parasites with long incubation and infectious periods should also be of greater concern because their opportunity for transmission to another host is spread out over a longer period of time, increasing the potential for invasion (André and Day 2005). ~~As a counterexample, if these periods are very short, a parasite could disappear from infected hosts before a transmission opportunity presents itself.~~ Parasites with long incubation periods are also more difficult to detect by conventional surveillance and quarantine methods. Moreover, the longer the incubation and infectious periods are, the more time the parasite has to evolve and adapt to a novel host species,~~-~~ such as through mutations that allow binding to a host receptor (Parrish and Kawaoka 2005), increasing the potential ~~for adaptation to~~ horizontal spread (Antia et al. 2003; Gandon et al. 2013). The long incubation and

infectious periods of *Mycobacterium bovis* almost certainly contributed to its ability to invade and persist in wildlife in East Africa after spillover from domestic animals (Cleaveland et al. 2005).

The potential for invasion and spread will also be greater for parasites with the largest basic reproduction number (R_0) (Gandon et al. 2013), which represents the average number of secondary cases arising from a single infectious individual introduced into a uniformly susceptible population. R_0 must be greater than one for successful invasion and spread, and the probability of success would increase as R_0 Increases (André and Day 2005). Parasites with larger R_0 values may be more likely to cause severe declines or extinction in small populations, particularly when reservoirs or frequency-dependent transmission are involved (De Castro and Bolker 2005). A notable example would be the rapid amphibian population declines in Panama due to chytridiomycosis, where entire populations are susceptible to fatal disease and become infected in a relativel narrow window of time (Lips et al. 2006).

Parasite pathogenicity has not been addressed specifically in this review because ~~the variability in~~it varies with host susceptibility and immune response, which makes it difficult to offer generalizations – a parasite that is highly pathogenic in one host, or age-class of host, might be nonpathogenic in another. For similar reasons, we have not discussed the influence of population structure or social contact networks, or the influence of overall biodiversity, on parasite invasion and spread, since these are highly context and species dependent. These issues are critically important, but would need to be addressed in a specific disease risk analysis where the host's demography and ecology are known.

These parasite characteristics are only intended to be a starting point. They can provide a structured means of prioritizing hazards identified during a disease risk analysis (OIE and IUCN 2014), and enable informed predictions in the consequence assessment. We acknowledge that there can be challenges with evaluating or predicting these parasite characteristics in real-world scenarios. We may only be able to make educated guesses about the basic reproduction number and invasion potential of a non-native parasite, its potential for establishment in a reservoir host, or its ability to be transmitted by a new vector species. Indeed some parasites may not have been identified by (be known to) the individuals undertaking the disease risk analysis. In spite of these limitations, efforts should be made to use these or similar criteria in evaluating the risk of disease from non-native parasites for any wildlife translocation. With ongoing anthropogenic changes in ecosystems, climate change, and expansion of global transportation networks, the risk of non-native parasite invasion and spread as a consequence of translocation will only be increasing in the future.

References:

Almberg ES, Cross PC, Dobson AP, et al (2012) Parasite invasion following host reintroduction: a case study of Yellowstone's wolves. Philos Trans R Soc B Biol

Sci 367:2840–2851. doi: 10.1098/rstb.2011.0369

André J-B, Day T (2005) The effect of disease life history on the evolutionary emergence of novel pathogens. Proc Biol Sci 272:1949–1956. doi: 10.1098/rspb.2005.3170

Antia R, Regoes R, Koella J, Bergstrom C (2003) The role of evolution in the emergence of infectious diseases. Nature 426:658–61. doi: 10.1038/nature02177.1.

Berger L, Speare R, Daszak P, et al (1998) Chytridiomycosis causes amphibian mortality associated with population declines in the rain forests of Australia and Central America. Proc Natl Acad Sci U S A 95:9031–9036. doi: 10.1073/pnas.95.15.9031

Blehert DS, Hicks AC, Behr M, et al (2009) Bat White-Nose Syndrome: An Emerging Fungal Pathogen? Science (80-) 323:53706.

Chen R, Holmes EC (2006) Avian influenza virus exhibits rapid evolutionary dynamics. Mol Biol Evol 23:2336–2341. doi: 10.1093/molbev/msl102

Cleaveland S, Laurenson MK, Taylor LH (2001) Diseases of humans and their domestic mammals: pathogen characteristics, host range and the risk of emergence. Philos Trans R Soc Lond B Biol Sci 356:991–999. doi: 10.1098/rstb.2001.0889

Cleaveland S, Mlengeya T, Kazwala RR, et al (2005) Tuberculosis in Tanzanian wildlife. J Wildl Dis 41:446–453. doi: 10.7589/0090-3558-41.2.446

Cooper N, Griffin R, Franz M, et al (2012) Phylogenetic host specificity and understanding parasite sharing in primates. Ecol Lett 15:1370–1377.

De Castro F, Bolker B (2005) Mechanisms of disease-induced extinction. Ecol Lett 8:117–126. doi: 10.1111/j.1461-0248.2004.00693.x

Dobson a, Foufopoulos J (2001) Emerging infectious pathogens of wildlife. Philos Trans R Soc Lond B Biol Sci 356:1001–1012. doi: 10.1098/rstb.2001.0900

Dobson a P, Hudson PJ (1986) Parasites, disease and the structure of ecological communities. Trends Ecol Evol (Personal Ed 1:11–15. doi: 10.1016/0169-5347(86)90060-1

Duffy S, Shackelton L a, Holmes EC (2008) Rates of evolutionary change in viruses: patterns and determinants. Nat Rev Genet 9:267–276. doi: 10.1038/nrg2323

Engering A, Hogerwerf L, Slingenbergh J (2013) Pathogen-host-environment interplay and disease emergence. Emerg Microbes Infect 2:e5. doi: 10.1038/emi.2013.5

Ewald PW (1996) Guarding Against the Most Dangerous Emerging Pathogens: Insights from Evolutionary Biology. Emerg Infect Dis 2is:245–257. doi: 10.3201/eid0204.960401

Fessl B, Tebbich S (2002) Philornis downsi - A recently discovered parasite on the Gal??pagos archipelago - A threat for Darwin's finches? Ibis (Lond 1859)

144:445–451. doi: 10.1046/j.1474-919X.2002.00076.x

Fisher MC, Garner TWJ, Walker SF (2009) Global emergence of Batrachochytrium dendrobatidis and amphibian chytridiomycosis in space, time, and host. Annu Rev Microbiol 63:291–310. doi: 10.1146/annurev.micro.091208.073435

Fisher MC, Henk D a., Briggs CJ, et al (2012) Emerging fungal threats to animal, plant and ecosystem health. doi: 10.1038/nature10947

Foley J, Clifford D, Castle K, et al (2011) Investigating and managing the rapid emergence of white-nose syndrome, a novel, fatal, infectious disease of hibernating bats. Conserv Biol 25:223–231. doi: 10.1111/j.1523-1739.2010.01638.x

Gandon S, Hochberg ME, Holt RD, Day T (2013) What limits the evolutionary emergence of pathogens? Philos Trans R Soc Lond B Biol Sci 368:20120086. doi: 10.1098/rstb.2012.0086

Gog J, Woodroffe R, Swinton J (2002) Disease in endangered metapopulations: the importance of alternative hosts. Proc Biol Sci 269:671–6. doi: 10.1098/rspb.2001.1667

Gomez A, Nichols ES, Perkins SL (2012) Parasite conservation, conservation medicine, and ecosystem health. In: Aguirre AA, Ostfeld R, Daszak P (eds) New Directions in Conservation Medicine: Applied Cases of Ecological Health. pp 67–81

Gompper ME, Williams ES (1998) Parasite Conservation and the Black-Footed Ferret Recovery Program. Conserv Biol 12:730–732. doi: 10.1046/j.1523-1739.1998.97196.x

Hatcher MJ, Dick JT a, Dunn AM (2014) Parasites that change predator or prey behaviour can have keystone effects on community composition. Biol Lett 10:20130879. doi: 10.1098/rsbl.2013.0879

Hudson P, Greenman J (1998) Competition mediated by parasites: Biological and theoretical progress. Trends Ecol Evol 13:387–390. doi: 10.1016/S0169-5347(98)01475-X

Hudson PJ, Dobson a P, Newborn D (1998) Prevention of population cycles by parasite removal. Science 282:2256–2258. doi: 10.1126/science.282.5397.2256

Hudson PJ, Dobson AP, Lafferty KD (2006) Is a healthy ecosystem one that is rich in parasites? Trends Ecol Evol 21:381–385. doi: 10.1016/j.tree.2006.04.007

Lafferty KD, Dobson AP, Kuris AM (2006) Parasites dominate food web links. Proc Natl Acad Sci U S A 103:11211–11216. doi: 10.1073/pnas.0604755103

Lips KR, Brem F, Brenes R, et al (2006) Emerging infectious disease and the loss of biodiversity in a Neotropical amphibian community. Proc Natl Acad Sci U S A 103:3165–3170. doi: 10.1073/pnas.0506889103

Lymbery AJ, Morine M, Kanani HG, et al (2014) Co-invaders: The effects of alien parasites on native hosts. Int J Parasitol Parasites Wildl 3:171–177. doi:

10.1016/j.ijppaw.2014.04.002

Marcogliese DJ (2004) Parasites: Small Players with Crucial Roles in the Ecological Theater. Ecohealth 1:151–164. doi: 10.1007/s10393-004-0028-3

McCallum H, Barlow N, Hone J (2001) How should pathogen transmission be modelled? Trends Ecol Evol 16:295–300. doi: 10.1016/S0169-5347(01)02144-9

McCallum H, Dobson a. (1995) Detecting disease and parasite threats to endangered species and ecosystems. Trends Ecol Evol 10:190–194. doi: 10.1016/S0169-5347(00)89050-3

McCarthy AJ, Shaw M-A, Goodman SJ (2007) Pathogen evolution and disease emergence in carnivores. Proc Biol Sci 274:3165–3174. doi: 10.1098/rspb.2007.0884

OIE, IUCN (2014) Guidelines for Wildlife Disease Risk Analysis. 24.

Origgi F-C, Plattet P, Sattler U, et al (2012) Emergence of a Canine Distemper Virus Strain with Modified Molecular Signature and Enhanced Neuronal Tropism Associated with High Mortality in Wild Carnivores. Vet Pathol 146:50. doi: 10.1016/j.jcpa.2011.11.021

Parrish CR, Kawaoka Y (2005) The origins of new pandemic viruses: the acquisition of new host ranges by canine parvovirus and influenza A viruses. Annu Rev Microbiol 59:553–586. doi: 10.1146/annurev.micro.59.030804.121059

Sainsbury AW, Deaville R, Lawson B, et al (2008) Poxviral disease in red squirrels sciurus vulgaris in the UK: Spatial and temporal trends of an emerging threat. Ecohealth 5:305–316. doi: 10.1007/s10393-008-0191-z

Shi Y, Wu Y, Zhang W, et al (2014) Enabling the "host jump": structural determinants of receptor-binding specificity in influenza A viruses. Nat Rev Microbiol 12:822–831. doi: 10.1038/nrmicro3362

Simon-Loriere E, Holmes EC (2011) Why do RNA viruses recombine? Nat Rev Microbiol 9:617–626. doi: 10.1038/nrmicro2614

Smith KF, Sax DF, Lafferty KD (2006) Evidence for the role of infectious disease in species extinction and endangerment. Conserv Biol 20:1349–57. doi: 10.1111/j.1523-1739.2006.00524.x

Tompkins DM, Arneberg P, Begon ME, et al (2002a) Parasites and host population dynamics. The Ecology of Wildlife Diseases. Oxford University Press, p 2002

Tompkins DM, Carver S, Jones ME, et al (2015) Emerging infectious diseases of wildlife: a critical perspective. Trends Parasitol 1–11. doi: 10.1016/j.pt.2015.01.007

Tompkins DM, Draycott R a H, Hudson PJ (2000) Field evidence for apparent competition mediated via the shared parasites of two gamebird species. Ecol Lett 3:10–14. doi: 10.1046/j.1461-0248.2000.00117.x

Tompkins DM, Dunn AM, Smith MJ, Telfer S (2011) Wildlife diseases: From

individuals to ecosystems. J Anim Ecol 80:19–38. doi: 10.1111/j.1365-2656.2010.01742.x

Tompkins DM, Sainsbury a W, Nettleton P, et al (2002b) Parapoxvirus causes a deleterious disease in red squirrels associated with UK population declines. Proc Biol Sci 269:529–533. doi: 10.1098/rspb.2001.1897

Tompkins DM, Wilson K (1998) Wildlife disease ecology:from theory to policy. 13:476–478. doi: 10.1016/S0169-5347(98)01499-2

Viana M, Cleaveland S, Matthiopoulos J, et al (2015) Dynamics of a morbillivirus at the domestic–wildlife interface: Canine distemper virus in domestic dogs and lions. Proc Natl Acad Sci 112:1464–1469. doi: 10.1073/pnas.1411623112

Warner RE (1968) The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. Condor 70:101–120. doi: 10.2307/1365954

Watson MJ (2013) What drives population-level effects of parasites? Meta-analysis meets life-history. Int J Parasitol Parasites Wildl 2:190–196. doi: 10.1016/j.ijppaw.2013.05.001

Wood MJ (2007) Parasites entangled in food webs. Trends Parasitol 23:8–10. doi: 10.1016/j.pt.2006.11.003

Woodworth BL, Atkinson CT, Lapointe D a, et al (2005) Host population persistence in the face of introduced vector-borne diseases: Hawaii amakihi and avian malaria. Proc Natl Acad Sci U S A 102:1531–1536. doi: 10.1073/pnas.0409454102

Woolhouse ME, Taylor LH, Haydon DT (2001) Population biology of multihost pathogens. Science 292:1109–1112. doi: 10.1126/science.1059026

Woolhouse MEJ, Haydon DT, Antia R (2005) Emerging pathogens: The epidemiology and evolution of species jumps. Trends Ecol Evol 20:238–244. doi: 10.1016/j.tree.2005.02.009