



Invited Review

Parasites and the conservation of small populations: The case of *Baylisascaris procyonis*



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ABSTRACT

Human demands on natural resources result in landscape changes that facilitate the emergence of disease. Most emerging diseases are zoonotic, and some of these pathogens play a role in the decline of vulnerable wildlife species. *Baylisascaris procyonis*, the common roundworm parasite of raccoons (*Procyon lotor*), is a well recognized zoonotic infection that has many of the properties associated with a pathogen capable of driving extinction. It is highly non-specific and frequently pathogenic with regard to paratenic hosts, which contact eggs of *B. procyonis* at raccoon latrines. Eggs accumulate at latrines and remain viable for many years. Transmission of *B. procyonis* is sensitive to changes in land-use, and fragmented habitats increase contact rates between raccoons, potential paratenic hosts, and the parasite. Raccoons, and subsequently *B. procyonis*, have been introduced to Europe and Japan, where naïve vertebrates may be exposed to the parasite. Finally, domestic animals and exotic pets can carry patent infections with *B. procyonis*, thus increasing environmental contamination beyond raccoon latrines, and expanding the area of risk to potential paratenic hosts. This parasite can potentially contribute to extinctions of vulnerable species, as exemplified by the case of the Allegheny woodrat (*Neotoma magister*), a species that has experienced local declines and extinctions that are linked to *B. procyonis*. Conservation strategies for vulnerable species should consider the transmission ecology of parasitic pathogens, like *B. procyonis*.

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1. Introduction

The current human population size and subsequent demands on natural resources have resulted in shifts in land-use patterns

and ecosystem processes that have significant implications for the emergence of disease (Daszak et al., 2001; Plowright et al., 2008; Smith et al., 2009). Links between human and environmental health are well documented, and the emergence of numerous

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zoonotic diseases, which now account for approximately 58% of all human pathogens, have been related to changing land use and resource consumption patterns (Woolhouse and Gowtage-Sequeria, 2005). Since the majority of emerging diseases originate in wildlife populations (~72%, Jones et al., 2008), it is important to determine ecological facilitators of disease transmission among wildlife, and the implications of these emerging diseases on wildlife populations themselves. Infectious diseases are important to consider with regard to conservation efforts, and they may play a role in wildlife extinctions (Smith et al., 2009). While only 3.7% of recorded extinctions are directly linked to disease (Smith et al., 2006), some pathogens have particular characteristics that make them more likely to facilitate extinction (Woodroffe, 1999; Daszak et al., 2000; Altizer et al., 2003; Bradley and Altizer, 2007; Pedersen et al., 2007; Smith et al., 2009; Thompson et al., 2010). Pathogens may play a role in facilitating extinctions when one or more of the following conditions occur: (1) the pathogen is relatively non-specific with regard to host (Pedersen et al., 2007), (2) the pathogen remains viable in the environment or reservoir hosts (Smith et al., 2009; Thompson et al., 2010), (3) habitat loss or overexploitation results in small or fragmented host populations and/or increased contact rates with pathogens (Woodroffe, 1999; Altizer et al., 2003; Bradley and Altizer, 2007; Smith et al., 2009; Thompson et al., 2010), (4) hosts are artificially dispersed via translocation (Daszak et al., 2000; Smith et al., 2009; Thompson et al., 2010), and (5) domestic animals are involved in transmission dynamics of the pathogen (Smith et al., 2009). The majority of pathogens that meet these criteria are zoonotic microparasites including viruses, bacteria, and protists (Taylor et al., 2001; Pedersen et al., 2007); however, there is increasing evidence that multi-host macroparasites contribute to population declines of some vulnerable host species (LoGiudice, 2003; Polley, 2005; Brearley et al., 2013). One such macroparasite that fits the criteria to facilitate extinctions is *Baylisascaris procyonis*, a common nematode parasite of raccoons (*Procyon lotor*) that is a well-recognized zoonotic infection (Murray and Kazacos, 2004; Gavin et al., 2005; Kazacos et al., 2013) and the most common cause of clinical larva migrans among wild and domestic animal species (Kazacos, 2001; Gavin et al., 2005). Several dozen fatal or severe cases of human baylisascariasis (Huff et al., 1984; Fox et al., 1985; Cunningham et al., 1994; Park et al., 2000; Rowley et al., 2000; Moertel et al., 2001; Gavin et al., 2002; Kazacos et al., 2002; Murray and Kazacos, 2004; Wise et al., 2005; Pai et al., 2007; Chun et al., 2009; Hajek et al., 2009; Mehta et al., 2010; Perlman et al., 2010; Haider et al., 2012; Hung et al., 2012; Kelly et al., 2012; Peters et al., 2012; Kazacos, personal communication 2013) have made this parasite a priority for public health and wildlife management officials (Sorvillo et al., 2002; Roussere et al., 2003; Page et al., 2005, 2009b; Sexsmith et al., 2009; Blizzard et al., 2010a,b; Kresta et al., 2010; Chavez et al., 2012; Samson et al., 2012; Hernandez et al., 2013), transmission dynamics (Page et al., 1998, 2008, 2009a, 2001a,b; LoGiudice, 2001; Gompper and Wright, 2005; Kellner et al., 2012) and possible strategies to mitigate risk (Page et al., 2011). Fewer studies have examined the implications of *B. procyonis* with regard to conservation and biodiversity (Evans, 2002a), but the parasite is implicated in population declines and local extirpations of Allegheny woodrats (*Neotoma magister*) (McGowan, 1993; LoGiudice, 2006) and possibly other species. This review will focus on the ecology and transmission dynamics of *B. procyonis* and the implications for conservation of wildlife populations.

2. *Baylisascaris procyonis* life cycle

Baylisascaris procyonis is an intestinal nematode of raccoons, the definitive host (Kazacos, 2001). Infections are common among raccoons and prevalence of infection can be as high as 82% (Kazacos, 2001). Juveniles are susceptible to direct infection via ingestion of eggs; therefore, prevalence can reach >90% among young raccoons (Kazacos and Boyce, 1989). Prevalence of infection among adult raccoons is lower (37–55%) because they become infected primarily via ingestion of paratenic hosts (Kazacos, 2001). Seasonal changes in prevalence have been noted in some regions (Evans, 2002b; Page et al., 2005; Page et al., 2009b) with marked declines during late winter months suggesting a self-cure (Kazacos, 2001). New infections occur in the spring resulting in highest measurements of prevalence in the fall (Kazacos, 2001; Evans, 2002b). Mature female worms produce, on average, over 100,000 eggs/day resulting in an infected raccoon shedding as many as 20,000–26,000 eggs/g feces (Kazacos, 1982, 2001; Snyder and Fitzgerald, 1985). Under optimum conditions, eggs embryonate to the infective stage within 11–14 days, and can remain viable in the environment for years (Kazacos and Boyce, 1989; Kazacos, 2001). When small mammals or birds ingest eggs, the larvae emerge and begin an aggressive somatic migration (Kazacos, 2001), with a low percentage (5%) entering the central nervous system (CNS) causing damage, clinical disease and death (Tiner, 1953; Sheppard and Kazacos, 1997). Larvae within the CNS or encapsulated in visceral or somatic tissues of paratenic hosts will infect raccoons when the tissue is consumed (Kazacos, 2001).

3. Non-specificity of *B. procyonis* larval infections

Baylisascaris procyonis is highly non-specific with regard to paratenic hosts and over 130 species of vertebrates have been identified with clinical larval infections (Kazacos, 2001; Kazacos, personal communication 2013). Natural infections have been reported across taxa including infections of mammals including rodents, lagomorphs, carnivores, and primates; and birds including galliformes, columbiformes, passeriformes, and psittaciformes (Kazacos, 2001; Evans, 2002a). Captive animals are vulnerable to infection especially when exposed to cages or bedding contaminated with raccoon feces (Sato et al., 2003; Wolf et al., 2007) or when raccoons have access to outdoor exhibits or captive habitats (Ball et al., 1998; Hanley et al., 2006; Gozalo et al., 2008; Thompson et al., 2008). Such outbreaks have affected a range of species including domestic rabbits (Kazacos et al., 1983; Sato et al., 2003), captive birds (Richardson et al., 1980; Kazacos et al., 1982, 1986; Wolf et al., 2007; Thompson et al., 2008), captive rodents (Van Andel et al., 1995; Thompson et al., 2008), and captive primates (Ball et al., 1998; Hanley et al., 2006; Gozalo et al., 2008).

Parasites do not necessarily threaten endangered species when the parasite is a specialist pathogen (Woolhouse et al., 2001); however, generalist parasites that do not have a high level of host-specificity, like *B. procyonis*, could threaten vulnerable populations of (paratenic) hosts (Pedersen et al., 2007). Tiner (1953) demonstrated that larval *B. procyonis* infections resulted in CNS involvement that contributed to 5% mortality of white-footed mice (*Peromyscus leucopus*) populations. While studies of *B. procyonis* induced mortality among wild populations of potential paratenic hosts are limited, all of the >130 noted species of larval infection with *B. procyonis* resulted in CNS involvement usually with severe clinical disease or death (Kazacos, 2001, Kazacos, personal communication 2013), and thus these species could be vulnerable to population declines if coexisting with high densities of raccoons.

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