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How do humans affect wildlife nematodes?

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Human actions can affect wildlife and their nematode parasites. Species introductions and human-facilitated range expansions can create new host–parasite interactions. Novel hosts can introduce parasites and have the potential to both amplify and dilute nematode transmission. Furthermore, humans can alter existing nematode dynamics by changing host densities and the abiotic conditions that affect larval parasite survival. Human impacts on wildlife might impair parasites by reducing the abundance of their hosts; however, domestic animal production and complex life cycles can maintain transmission even when wildlife becomes rare. Although wildlife nematodes have many possible responses to human actions, understanding host and parasite natural history, and the mechanisms behind the changing disease dynamics might improve disease control in the few cases where nematode parasitism impacts wildlife.

Humans alter infectious processes

Humans have long battled nematodes, and we have fared well, reducing soil-transmitted helminth prevalence and almost eradicating the human Guinea worm (*Dracunculus medinensis*) through intensive intervention [1,2]. While deliberate anti-parasite campaigns have reduced the human disease burden, introduced species, changing agricultural practices, and habitat degradation have unintentionally affected wildlife diseases. These effects come through two routes. First, humans create new host–parasite dynamics through host and parasite introductions. Second, humans alter existing transmission dynamics by changing host density, parasite survival, and host–parasite contact rates. When such changes increase disease in wildlife or humans, parasitologists have the challenge of developing interventions that work in wildlife. Despite concern for human-mediated increases in wildlife disease, that is not the whole story; human activities can also unintentionally decrease parasitism.

In contrast to the 115 described human nematodes, the ~5000 wildlife nematodes are still little known [3,4]. What we know well comes from work with domestic animals and a few natural host–parasite systems (e.g., grouse, arctic ungulates, raccoon roundworm). These well-studied systems reveal patterns and suggest predictions about wildlife nematodes in general [5]. For example, the raccoon roundworm, *Baylisascaris procyonis*, is a model organism for comparing mechanisms that alter nematode transmission in a wildlife host that interacts with humans. Similarly, the domestic dog, *Canis familiaris*, and its relatively well-studied parasite fauna provide a familiar example of spillover and spillback from an introduced host. Throughout this review we will highlight how these and other well-studied examples help us predict how human actions affect wildlife nematodes.

Creating new host–parasite interactions

Introduced species introduce parasites

Although invasive species do not retain many parasites, compared to populations in their native ranges, an estimated 12% of parasite species do invade along with their hosts [6]. A case in point is the domestic dog, which diverged from wolves over 20 000 years ago then dispersed around the globe with humans [7]. Of the 51 nematode species documented from domestic dogs, about 17 originated in dogs (Figure 1). Two-thirds of those 17 nematodes occur in native wildlife and likely represent spillover from dogs. Repeated host introductions, as have occurred for species such as dogs and rats, increase the probability that parasites will invade. For example, the exotic rat lungworm, *Angiostrongylus cantonensis*, now infects native wildlife, such as the tawny frogmouth (*Podargus strigoides*), in once rat-free regions such as Australia [8,9]. With continued international trade and human movement, introductions like these will only accumulate with time.

An even-greater source of introduced nematodes than accidental introductions is the commerce of pets and livestock. For example, the aquarium trade and mosquito control programs released guppies (*Poecilia reticulata*) and mosquito fish (*Gambusia affinis*) infected with the Asian nematode, *Camallanus cotti*, which is now common in endemic Hawaiian stream fishes [10]. Similarly, Japanese eels (*Anguilla japonica*) imported to Europe for food introduced the swim-bladder worm, *Anguillicoloides crassus*, to the endangered European eel (*Anguilla anguilla*). *Anguillicoloides crassus* is a good example of an introduced parasite that can impact wildlife health. Although infection in

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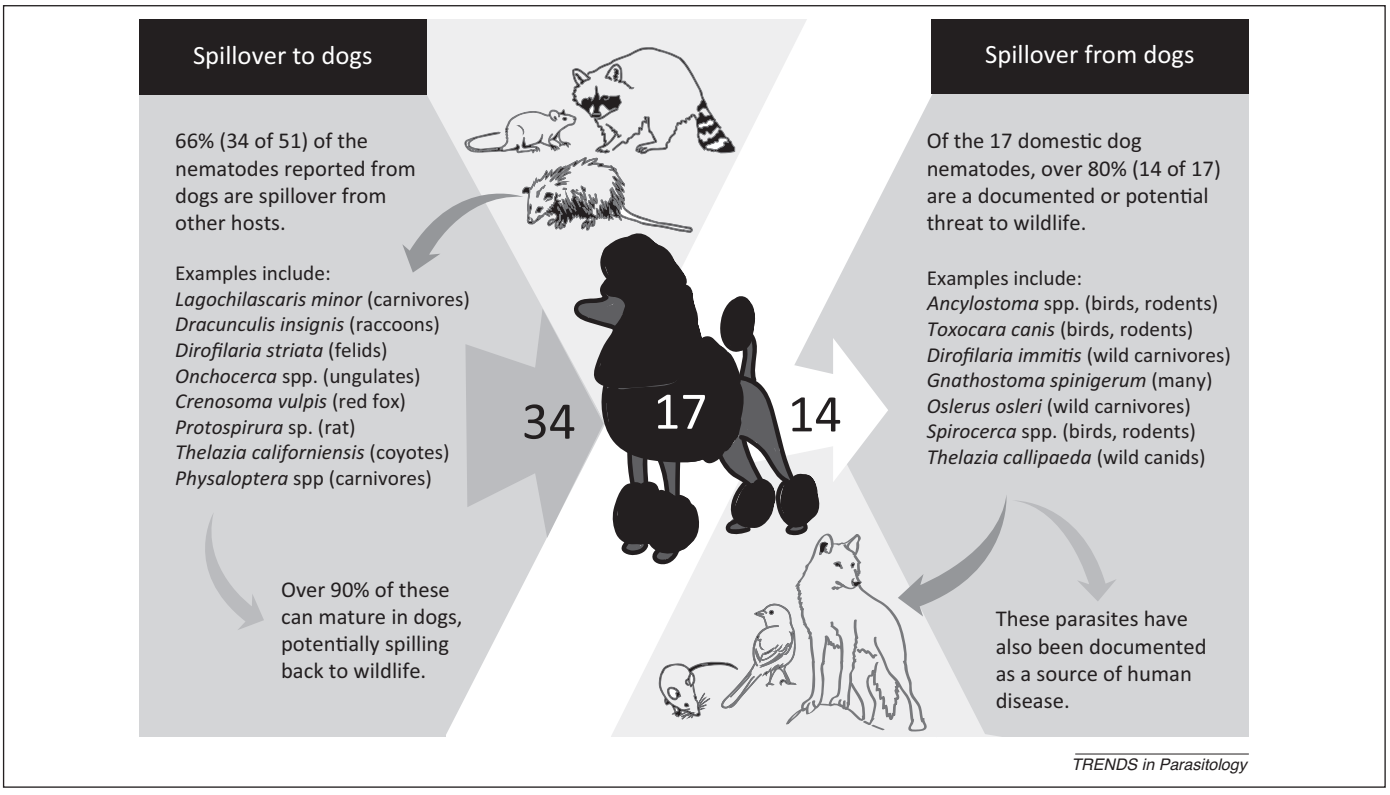

Figure 1. Domestic dog (*Canis familiaris*) nematode parasites: how common is parasite spillover? The domestic dog is host to at least 51 parasitic nematodes. Two-thirds of these infections are spillover from wildlife hosts, and over 90% of these can mature in dogs and can spillback into wildlife. Although only one-third of the nematodes recorded from dogs are dog parasites, 80% represent a disease risk for other wildlife [18,34,75–84].

Japanese eels is asymptomatic, severe pathology may contribute to spawning failure in European eels [11,12]. This increased pathology in the acquired host could stem from naïve hosts having little resistance or tolerance. We cannot help but notice harmful introduced parasites [13,14], but many others, such as the gut parasite, *Trichuris muris*, which jumped from introduced black rats to native deer mice in the California Channel Islands [15], cause little damage. Such host-switching appears to be common in nematodes, and is likely to happen when hosts are related and use similar resources [16]. Overall, although some introduced parasites impact native host populations, most introduced nematodes probably have minor impact and go unnoticed.

Introduced species alter disease dynamics of native nematodes

Introduced species can act as alternative hosts or reservoirs for endemic parasites, increasing transmission to native species via spillback [17]. Dogs have picked up most (~34 of 51) of their recorded parasite diversity from native hosts in their introduced range (Figure 1). For these native parasites, dogs increase total host density, which amplifies transmission back to native hosts. For example, the dog heartworm, *Dirofilaria immitis*, was likely endemic in red wolf (*Canis rufus*) populations [18,19]. The parasite persisted even after the red wolf was declared extinct in the wild, suggesting that current infections in the recovering wolf population probably represent parasite spillback from domestic dogs (and the expanding coyote population) [20–22]. The spillover and spillback seen in dog and wildlife nematodes suggests that, for every host introduction, there is likely to be host-switching as introduced parasites invade native hosts and native parasites colonize the introduced host. When introduced species amplify native parasite transmission, this reduces the threshold host density set by the native host, making it possible for sustained parasite transmission even if the native host declines. If an introduced host is much more tolerant to infection than the native host, a parasite can theoretically drive its host extinct [23].

Fortunately, introduced species are often not suitable hosts for native parasite fauna [6], and their presence, under particular conditions, might reduce transmission through a dilution effect. A dilution effect could occur if introduced hosts become a sink for infective stages or vectors. Knowing the extent to which infective stages limit nematode transmission is crucial for understanding the potential for the dilution effect via introduced species. Transmission dilution through introduced species seems most likely to happen for nematodes with frequency-dependent transmission such as vector-transmitted filarial worms [24]. For instance, because filarial worms are host specific in African rainforest birds [25], introduced bird species might (theoretically) divert blood-sucking flies, reducing vector transmission to competent native hosts. The dilution effect is often posited as a biodiversity benefit [26]. Ironically, dilution should be strongest when introduced species dominate communities [27].

Range expansion and human-mediated parasite spillover

As for species introductions, range expansion and increases in anthropophilic wildlife populations can lead to novel host–parasite interactions and conservation

concerns for co-occurring species due to parasite spillover. Some parasite populations have benefited from having human-associated hosts, such as white-tailed deer (*Odocoileus virginianus*) and raccoons (*Procyon lotor*) [28,29]. White-tailed deer have expanded due to a decrease in hunting and reforestation following a switch from firewood to fossil fuels. Deer now overlap with moose (*Alces alces*), and the spillover of the deer meningeal worm, *Parelaphostrongylus tenuis*, causes serious pathology and is predicted to cause moose declines where moose and deer now overlap [30,31]. Similarly, raccoons and raccoon parasites have spread into eastern US forests, resulting in Allegheny woodrat (*Neotoma magister*) extirpation [32]. Woodrats contaminate their food caches with raccoon roundworm eggs as they feed on seeds in raccoon latrines, and then the migrating larval roundworms often kill them [33]. Raccoon roundworm spillover into woodrats is a novel host–parasite interaction, and mammals and birds in Japan, Europe, and Russia may also be at risk (Figure 2). For both raccoon roundworm and the deer meningeal worm, host use is flexible for at least one life stage: the adult meningeal worm infects several ungulates, and larval raccoon roundworms have been recovered from over 100 bird and mammal species [34,35]. This broad host-use increases spillover risk to both wildlife and humans in the parasite's native range and suggests that such nematodes might be the most successful and devastating invaders.

Parasite spillover from humans also threatens wildlife. Reverse zoonotic disease risk (i.e., anthropozoonosis) increases as humans move into wildlife habitats [36]. Several human nematodes, including *Ascaris lumbricoides*, *Strongyloides stercoralis*, and *Trichuris trichiura*, infect non-human primates, especially where habitat alteration brings humans and wildlife into contact [37–40]. For example, forest fragmentation leads to higher parasitism in red colobus monkeys (*Piliocolobus tephrosceles*) due to spillover of strongyle nematodes from villagers [41]. However, parasites that use human and non-human primates might not always engage in cross-species transmission. For example, the strongyle *Oesophagostomum bifurcum* infects patas monkeys (*Erythrocebus patas*), mona monkeys (*Cercopithecus mona*), olive baboons (*Papio anubis*), and humans in Ghana. However, each parasite population is structured according to host species [42]. Although proximity to humans should increase human parasite spillover, concurrent changes in wildlife behavior might reduce parasitism. The reduced worm-burden in baboons that raid human crops is attributed to reduced exposure to trophically transmitted infective stages in wild prey and increased resistance due to better body condition [43]. Given the frequency that human nematodes are detected in wild primates, campaigns to reduce nematode infections in humans could also reduce nematode spillover into wildlife, leading to a win–win for biodiversity and human health.

Changing dynamics in established host–parasite interactions

Parasites thrive in intact ecosystems [44]. However, humans often alter natural host–parasite dynamics, which are a complex function of host–parasite contact rates, host density, and parasite survival. Human impacts that increase any of these parameters might also increase wildlife parasitism [45].

Contact rates

Human actions can increase disease transmission through increasing host species contact rates. Contact rates increase

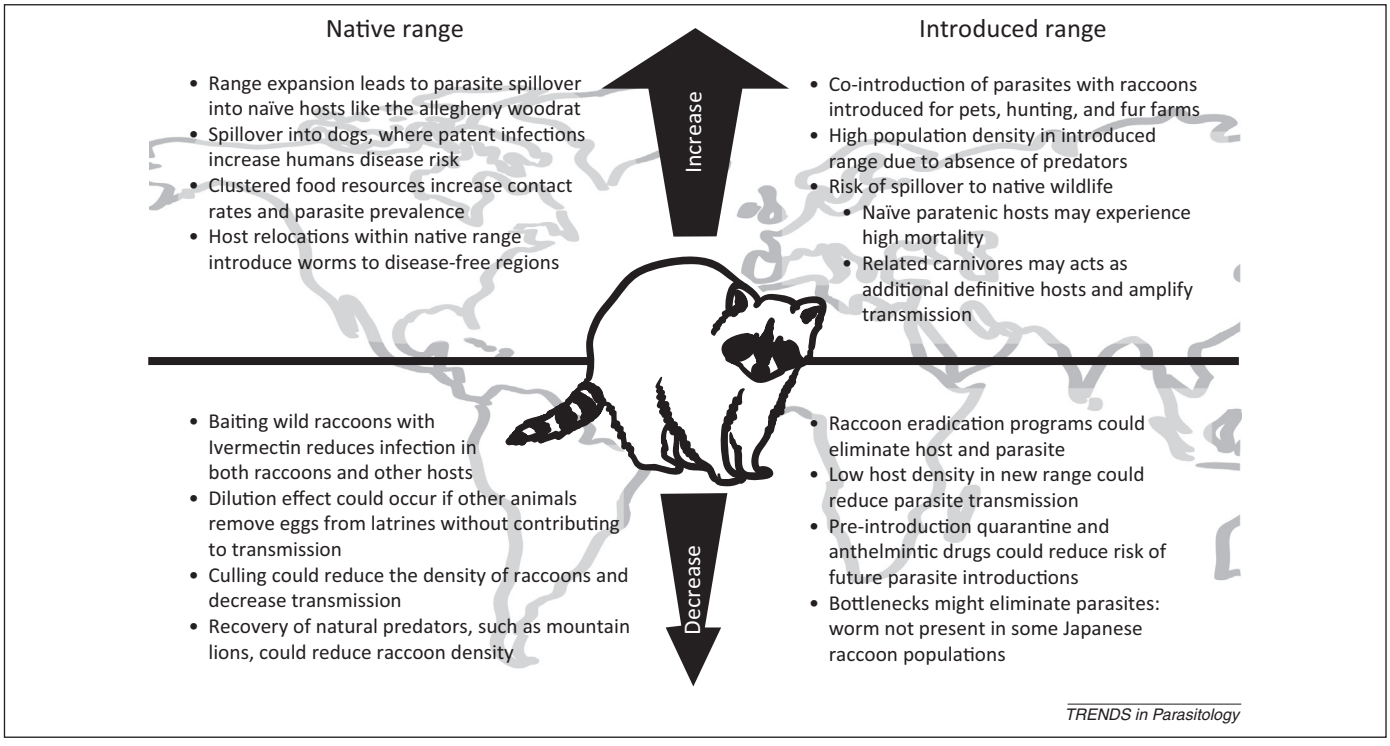

Figure 2. Raccoon roundworm (*Baylisascaris procyonis*): a threat to wildlife in both the native and introduced raccoon range. Human impacts can alter invasion and infection dynamics of the raccoon roundworm, in both its native and introduced ranges, resulting in either amplified or reduced transmission risk to wildlife and humans [32,46,73,85–88].

when wildlife, such as raccoons, aggregate around supplemental food resources, leading to increased parasite transmission [46]. Similarly, Bahamian rock iguanas (*Cyclura cychlura*) aggregate at sites where tourists feed them, and this correlates with higher hookworm and pinworm prevalence and intensity [47]. The impact of supplemental feeding on wildlife is likely a tradeoff for wildlife health. Additional calories might support a larger population, but, when food resources are clumped, increased contact rates are likely to increase parasite transmission. The net effect will depend on host tolerance.

Habitat fragmentation also alters transmission dynamics. For example, mice are more exposed to raccoon roundworm in a fragmented agricultural landscape as a result of changes in the feeding habits in both raccoons and mice. Raccoons forage on crops, and, when mice subsequently forage in raccoon latrines for undigested corn, the increased contact with infective stages results in higher raccoon-roundworm prevalence in mice [48]. Even depleted species can have high local densities in a patchy landscape. For instance, habitat fragmentation has concentrated endangered colobus and mangabey monkeys, and this leads to a higher gastrointestinal nematode prevalence [49]. Although any increase in contact rates should increase parasitism, this is most worrisome when conditions concentrate threatened species and the nematode is pathogenic. When habitat fragmentation increases aggregation, threatened species do not gain the health benefits associated with rarity.

Host density

Wildlife declines as a result of hunting, fishing, and habitat degradation should reduce or even eliminate host-specific nematodes [50]. A striking example is when the swim bladder nematode, *Cystidicola stigmatura*, disappeared from lake trout (*Salvelinus namaycush*) following overfishing [51,52]. In general, parasites with complex life cycles decline under fishing pressure [53]. Moreover, in primates, threatened hosts harbor fewer parasite species, suggesting that parasites are less likely to persist in small, isolated host populations [54]. Host-specific nematodes should be highly sensitive to host population changes, and these relationships become more complex when additional hosts are present in the lifecycle. The more complex the life cycle, the more chances there are for transmission to fail [55].

Even though complex life cycles might be more difficult to complete, increased intermediate host and vector density can increase disease even when wildlife host populations are stable or in decline. Arthropod vectors often proliferate in response to dams and changing agricultural practices. Such human modifications can increase filarial nematode infection in humans [56], and presumably also could for wildlife. Furthermore, at high latitudes, mosquito populations increased with recent increases in temperature and humidity. A warmer climate seems to increase exposure of moose and reindeer to the filarial nematode, *Setaria tundra*, by fueling mosquito abundance and driving ungulates into swampier microhabitats where transmission occurs [57]. A further example is that increasing temperatures result in higher muskox (*Ovibos moschatus*) parasitism by the nematode *Umingmakstrongylus pallikuukensis* because rising temperatures both increase larval parasite development rate and reduce winter mortality of the intermediate-host slug [58,59]. Eutrophication is another environmental factor that can favor intermediate hosts such as oligochaetes. Oligochaetes infected with *Eustrongyloides* nematodes are eaten by fish, which in turn are eaten by birds, and the resulting trophic transmission sequence leads to high infection intensity and increased nestling mortality for birds at eutrophic sites [60]. For these reasons, humans often affect wildlife diseases indirectly through their effects on intermediate hosts and vectors [61].

Additional alternative definitive host species can also increase disease impacts because parasite population dynamics are become less linked to the density of a single host species. One example is the cosmopolitan bird parasite, *Dispharynx spiralis*, which is present in endemic Galapagos finches. Although the origin of the nematode is unclear, disease risk in native birds increases due to spillover from the high worm burdens in island chickens (*Gallus gallus domesticus*). Parasitism is high in the chickens owing to host biology, feeding habits, and captive-rearing methods, and these domestic fowl act as a reservoir for the nematode [62]. Because there are now around 23 billion domestic fowl and five billion domestic ungulates on earth [63], and most domestic animal nematodes are generalists that can spillover to wildlife [64], animal husbandry could be the main way that human actions put wildlife at risk to parasitic nematodes.

Larval parasite survival

Disease transmission should decrease when abiotic conditions reduce larval parasite survival [65–67]. However, such consequences are less likely when crowding or acquired immunity play a strong regulatory role in the parasite population, as seen by comparing the rabbit stomach worm, *Graphidum strigosum*, and the rabbit intestinal worm, *Trichostrongylus retortaeformis* [44]. In other words, parasite intensity is less sensitive to variation in exposure rates when other factors limit infrapopulation abundance.

Because infective stages such as nematode eggs and larvae are exposed to environmental conditions, each species should evolve an optimal physiological tolerance for development and survival. Climate change shifts the optimal temperature of an organism to higher latitudes, and might allow parasites and their intermediate hosts or vectors to invade from lower latitudes [58,68]. At the same time, warming should hasten the mortality rate of eggs and larvae of locally adapted parasites, perhaps excluding them over time. Similarly, unfavorable abiotic conditions associated with environmental degradation should reduce juvenile parasite survival and decrease parasite transmission. For example, petrochemical pollution is correlated with decreased helminth infection in the cotton rat, *Sigmodon hispidus*, and both reduces arthropod intermediate host and free-living nematode juvenile survival [69]. Furthermore, annual burning and herbicide treatments reduce *Syphacia peromysci* and *Nippostrongylus muris* infections in the white-footed mouse, *Peromyscus leucopus*,

by altering mouse behavior and parasite free-living stage survival [70]. However, environmental stressors that increase infective-stage mortality might also stress hosts and increase susceptibility to infection. In addition, stress could reduce the survivorship of infected hosts. Consequently, the idiosyncratic impact on reduced parasite and host survival, versus increased host susceptibility, will determine whether environmental stress reduces or facilitates parasites, but most stressors probably result in parasite declines [71].

Implications for managing wildlife nematodes

For those cases where human actions increase parasitic nematodes in wildlife, effective mitigation at the host population level will require understanding the most important transmission pathways. If an introduced parasite spills over to native wildlife, culling or eradicating the introduced host might reduce the parasite in wildlife hosts. For parasites that spillover from pets or livestock, giving more anthelmintics to domestic animals might be warranted. However, once the parasite establishes transmission within the native fauna, control strategies are much more limited. Baits with anthelmintics are one possible way to control nematodes. Raccoon anthelmintic baiting can reduce roundworm prevalence in both raccoons and rodents, and fenbendazole-laced salt licks can reduce lungworm prevalence in bighorn sheep (*Ovis canadensis*) [72–74]. Such baiting might be easier when wildlife aggregate into reserves or habitat fragments.

Concluding remarks

Although humans introduce many nematodes and alter wildlife–nematode dynamics, impacts to wildlife health are not often noticeable. In part, this is because parasitic nematodes are natural parts of intact ecosystems, and their effects are mostly minor compared with other challenges in wildlife conservation. In those cases where human impacts do increase nematode parasites in wildlife, a better understanding of parasite ecology can point wildlife managers to mitigating solutions.

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