Neglected zoonotic helminths: *Hymenolepis nana*, *Echinococcus canadensis* and *Ancylostoma ceylanicum*

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Abstract

The majority of helminth parasites that are considered by WHO to be the cause of 'neglected diseases' are zoonotic. In terms of their impact on human health, the role of animal reservoirs and polyparasitism are both emerging issues in understanding the epidemiology of a number of these zoonoses. As such, *Hymenolepis* (*Rodentolepis*) *nana*, *Echinococcus canadensis* and *Ancylostoma ceylanicum* all qualify for consideration. They have been neglected and there is increasing evidence that all three parasite infections deserve more attention in terms of their impact on public health as well as their control.

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Introduction

Helminth parasites feature prominently in WHO's list of neglected diseases (http://www.who.int/neglected_diseases/diseases/en/). Most are zoonotic with animal reservoirs playing a role in their epidemiology and are a major impediment to control (Table 1). In this article, I have chosen three zoonotic helminth infections that, as well as falling under the 'neglected' umbrella, require re-evaluation in view of emerging issues relating to the epidemiology and impact on public health of the infections they cause.

Hymenolepis nana

Hymenolepis (*Rodentolepis*) *nana* is the most common cestode of humans, particularly in young children [1–3]. It is often referred to as the 'dwarf tapeworm' due to its small size, 2–4 cm long and only 1 mm wide [1] (Fig. 1). The adult tapeworm parasitizes the small intestine of humans, attaching to the mucosal surface between the villi. As the worm matures sexually, the terminal proglottids ('segments') become gravid, detach and disintegrate in the gut releasing the eggs, which are passed in the faeces. The eggs are immediately infective but can survive in the environment for up to 2 weeks [2]. As with most cestodes, the life cycle includes a metacestode, the larval cysticercoid, but atypically an intermediate host is not an obligatory requirement in the life cycle. If humans ingest the eggs, they hatch in the small intestine releasing a motile embryo, the oncosphere, which invades a villus and develops into the larval cysticercoid in approximately 4 days. The cysticercoid then ruptures, completely destroying the villus it occupies, attaches to the mucosal surface and develops into the adult tapeworm, which reaches patency in 4 weeks [1]. Humans can therefore be both definitive and intermediate hosts (Fig. 2).

A true indirect cycle involving an arthropod, usually small beetles such as *Tribolium* that commonly contaminate flour or cereal, also occurs [1,2]. The beetles become infected by ingesting eggs from the environment, and cysticercoids subsequently develop in their body cavity. The accidental ingestion of infected beetles by humans, usually with contaminated food, will lead to the development of the adult tapeworm. The frequency of food-borne transmission is not known but is likely to vary between endemic foci. A recent report considered that the

TABLE 1. Neglected zoonotic helminths

Disease	Causative agent	Life cycle	Definitive hosts	Intermediate hosts/vectors	Geographical distribution/no. of cases	Risk factors
Cysticercosis/Taeniasis	*Taenia* spp.	Indirect	Humans	Cattle, pigs, (Humans for *T. solium*)	Global but most prevalent in South America, Asia, South-East Asia, Africa/highly variable; prevalence up to 20% in some endemic areas	Communities with free-roaming pigs, poor sanitation and unregulated animal slaughter
Dracunculiasis	*Dracunculus medinensis*	Indirect	Humans, occ. other mammals especially dogs	Copepod crustaceans	Now restricted to a few countries in Africa	Communities with poor water quality
Food-borne trematodiases	*Clonorchis**, *Opisthorchis**, *Paragonimus**, *Fasciola*	Indirect	Humans and other mammals	Snails (and freshwater fish*)	Focal—South-East Asia, China, former Soviet Union, Middle East, western Europe, Africa/2−35 million	Eating uncooked fish and other aquatic products
Hydatid disease/echinococcosis	*Echinococcus* spp.	Indirect	Carnivores	Non-carnivorous mammals and humans	Global/~ 1 million	Ingestion of infective stages from the environment or contact with definitive hosts
Lymphatic filariasis	*Brugia* spp.	Indirect	Humans, cats	Mosquitoes	South and South-East Asia/> 10 million	Exposure to mosquito vectors
Schistosomiasis	*Schistosoma* spp.	Indirect	Humans and other mammals	Snails	Africa, Middle East, Caribbean, South America, China, South-East Asia/> 200 million	Children exposed to contaminated freshwater
Soil-transmitted helminthiases	*Ancylostoma ceylanicum*	Direct	Dogs, cats, humans	N/A	Asia, South-East Asia, Australia, South Africa/5−40% in endemic communities	Poor sanitation, promiscuous defecation
Hymenolepiasis	*Hymenolepis nana*	Direct and indirect	Humans and rodents	Beetles		Young children living in areas with poor hygiene and sanitation
Trichinellosis	*Trichinella* spp.	Direct	Mammals including humans	N/A	Global	Eating meat that has not been inspected by regulatory authorities, e.g. hunting

*Only Clonorchis, Opisthorchis and Paragonimus include freshwater fish in their life cycles.
Information from references [4,24,29,33,34,40,49] and WHO (http://www.who.int/neglected_diseases/diseases/en/).

proportion of cases attributable to food-borne infection is negligible [4], although no data are available to support this view. In addition to humans, mice can also be definitive hosts of *H. nana* and so act as reservoir hosts.

Epidemiological evidence suggests that direct human-to-human transmission is the most common route of infection with *H. nana*, particularly in environments where the frequency of such transmission is likely to be high due to poor hygiene and inadequate sanitation [2,5−7]. However, it is still considered a zoonosis because infected commensal/synanthropic rodents, such as mice and rats, and arthropod intermediate hosts represent a reservoir of infection that may vary in importance in different environments [2,5,8,9]. The risk of zoonotic transmission may be greater in poor, high-density urban environments and four recent reports found mice and rats in urban environments of Rio de Janeiro, Brazil, Uttarakhand in India, northern England and Kuala Lumpur, Malaysia, to be commonly infected with *H. nana* [10−13]. Molecular evidence is equivocal, suggesting that there may be two strains of *H. nana* that are maintained in zoonotic and non-zoonotic cycles but this remains to be determined. Macnish *et al.* [5] suggested that isolates of *H. nana* in Australia actually exist as two cryptic or sibling (morphologically identical, but genetically different) species, based on a sequence divergence of 5% in the

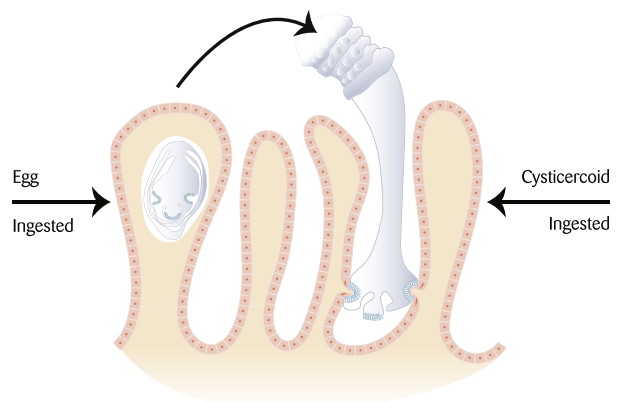**FIG. 1.** Adult tapeworm of *Hymenolepis nana*.

1.0 mm

FIG. 2. Illustration showing direct development of adult *Hymenolepis nana* following ingestion of cysticercoid or indirect with mucosal development of cysticercoid following ingestion of egg.

Egg Ingested

Cysticercoid Ingested

mitochondrial cytochrome *c* oxidase 1 gene. *Hymenolepis nana* has also been shown to infect lower primates and in such cases, humans appear to be the source of infection in a 'reverse zoonosis' scenario [14].

Infections in humans with *H. nana* are ubiquitous, particularly in children in developing countries [15–20]. Mono-infections are rare and in published surveys *H. nana* is usually reported as co-occurring with a number of other enteric parasites, particularly in community situations where the frequency of faecal–oral transmission is likely to be high [21,22]. *Giardia*, *Entamoeba coli*, *Blastocystis*, *Chilomastix* as well as species of gastrointestinal/soil-transmitted helminth, are the most common co-habiting parasites in such polyparasitic infections [21–27]. Chronic *G. duodenalis* infection is well recognized as contributing to poor growth and development ('failure to thrive') as well as nutritional deficiencies in children, and *H. nana* is thought to exacerbate the clinical impact in polyparasitic infections [6,21]. In this context, the invasive nature of *H. nana* may be more pathogenic than previously appreciated. Heavy infections may cause enteritis [1] but the clinical impact of *H. nana* infection is likely to be greatest in children who are chronically infected and *H. nana* may have been overlooked in such situations where other diseases are ranked higher in terms of their overt impact on health [16]. A recent study in Ethiopia found a much higher prevalence of *H. nana* (34.5%) in stunted (both underweight and dangerously underweight) children than in normal weight children (5.3%), complementing the results of earlier studies in Ethiopia, Egypt and Mexico [6].

Although effective drugs such as praziquantel and albendazole are available to treat infections with *H. nana*, they can do little to control the parasite in endemic foci where the frequency of transmission is high [28,29]. In such circumstances, re-infection will occur rapidly and prevention of re-infection requires education, improved hygiene and sanitation [28,29].

Echinococcus canadensis

Echinococcus canadensis is one of ten species of the 'hydatid tapeworm' *Echinococcus*, which has an obligate two-host life cycle (Table 2) [30]. The adult tapeworm develops in the small intestine of the carnivore definitive host, a canid or felid depending on the species of *Echinococcus*. Intermediate hosts are non-carnivorous mammals including humans, and become infected with the larval metacestode, a hydatid cyst, following the ingestion of eggs from the environment or contact with the definitive host [31]. In humans, infection is referred to as cystic or alveolar hydatid disease/echinococcosis (Table 2) and can be life threatening, particularly with the invasive, metastatic metacestode of *Echinococcus multilocularis* that is responsible for

TABLE 2. Currently recognized species of *Echinococcus*

Species	Main intermediate hosts	Known definitive hosts	Infectivity to humans
Echinococcus granulosus	Sheep, camels, macropod marsupials	Dog, fox, dingo, jackal and hyena	Yes
Echinococcus equinus	Horses	Dog	Probably not
Echinococcus ortleppi	Cattle	Dog	Yes
Echinococcus canadensis	Cervids	Wolves, dog	Yes
Echinococcus intermedius	Camels, pigs, sheep	Dog	Yes
Echinococcus felidis	Warthog	Lion	?
Echinococcus multilocularis	Rodents	Fox, dog, cat, wolf, racoon-dog, coyote	Yes
Echinococcus shiquicus	Pika	Tibetan fox	?
Echinococcus vogeli	Rodents	Bush dog	Yes
Echinococcus oligarthrus	Rodents	Wild felids	Yes

Data from reference [30].

causing alveolar hydatid disease/echinococcosis [32–35]. Effective, curative treatment of symptomatic cystic infection in humans requires surgery, although drug treatment can limit the progression of the disease.

Most cases of cystic echinococcosis in humans are acquired in endemic areas where the parasite is maintained in domestic cycles involving livestock and dogs. However, in northern Canada, Alaska and parts of Scandinavia, *Echinococcus* is maintained in a sylvatic cycle involving wolves and large cervids (moose and caribou) [36–38] (Figs. 3 and 4). The species involved is *E. canadensis* and in the past the number of reported cases in humans has been low, possibly reflecting its wildlife cycle [36]. It has also been considered that cystic infection with *E. canadensis* is less virulent in humans than the domestic species, *Echinococcus granulosus*. However, recent research suggests that *E. canadensis* may have been overlooked from a public health perspective and changing trends in the epidemiology of *Echinococcus* infections require a re-evaluation of the public health significance of *E. canadensis*, particularly in Canada.

It has long been thought that the clinical consequences of infection with *E. canadensis* in humans are negligible compared with infection with *E. granulosus*. In part, this may be due to the long progression of the disease in humans, often without symptoms, and the non-specificity of symptoms when they do occur. However, a combination of inadequate surveillance, the limitations of serological tests contributing to human cases being under-diagnosed, and the fact that indigenous people are disproportionately affected and at risk of infection, may have contributed to a lack of awareness of the public health significance of *E. canadensis* [39–42]. Molecular epidemiological studies have shown that *E. canadensis* comprises two genotypes, one of which, G10, appears to have spread rapidly in Canada in recent times [39]. Factors contributing to this include climate change enhancing parasite egg survival as well as the distribution
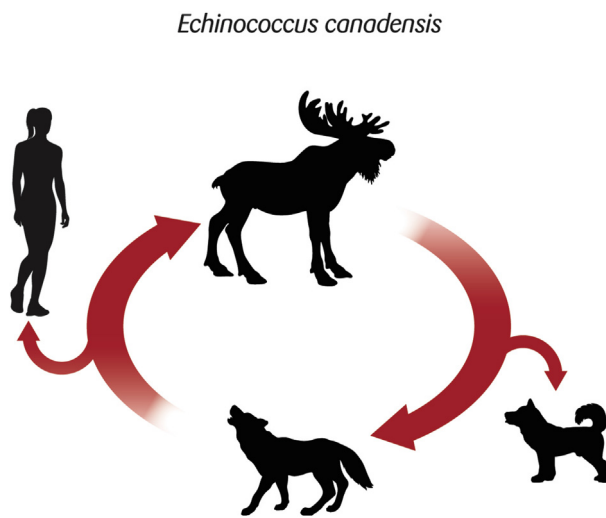
Echinococcus canadensis

FIG. 3.   Life cycle of *Echinococcus canadensis*. In the natural sylvatic cycle, definitive hosts are wolves that become infected by ingestion of the larval, cystic stage (hydatid cyst) in the lungs of moose and other large cervids such as reindeer following predation. Domestic dogs are infected by being fed offal from cervids that are hunted and slaughtered. The adult tapeworm develops in the intestine of the definitive host releasing infective, resistant eggs that contaminate the environment and may be accidentally ingested by cervids when grazing. Humans can become infected by ingesting eggs usually following contact with domestic dogs.

of cervid intermediate hosts. The two genotypes appear to vary in virulence in humans with G8 more pathogenic than previously considered, with two severe cases recently reported [39].

It has always been known that domestic or free-roaming dogs are important 'bridging hosts' between the sylvatic cycle of

E. canadensis and people [43]. Such spill-over from sylvatic foci occurs as a result of subsistence hunting within indigenous communities where dogs have access to offal and carcasses [40]. Recent studies in indigenous communities have demonstrated the occurrence of *E. canadensis* in domestic dogs in communities in northern Canada [44]. A domestic cycle of transmission involving domestic dogs and farmed elk was also recently identified in Western Canada [36]. The risk to public health in both these foci of transmission involving domestic dogs is clearly an issue that should be recognized. Although *E. canadensis* is most widely distributed in Canada, the first report of *E. canadensis* in moose (G8 genotype) in Maine increases the known distribution of *E. canadensis* in the USA from Alaska, northern Minnesota and northern California [45]. The Atlantic regions of the northern USA and Canada were not previously thought to be endemic regions for *E. canadensis* and this suggests that domestic dogs and/or coyotes are the definitive host [45].

Hookworm

Ancylostoma and *Necator* are the two genera of hookworm that infect humans and are a serious cause of morbidity, particularly in children and pregnant women, in developing countries. The most widespread species are *Ancylostoma duodenale* and *Necator americanus* with the latter more common in tropical and subtropical areas whereas *A. duodenale* tends to occur in cooler and drier regions, but the two species do overlap [24,29,46]. Their principle impact on health is as a cause of anaemia with *A. duodenale* a more voracious blood-sucker than *N. americanus* [47] (Fig. 5).

Hookworm along with *Trichuris*, *Ascaris* and *Strongyloides*, collectively comprise the soil-transmitted helminths, and

FIG. 4.   Hydatid cysts of *Echinococcus canadensis* in the lungs of a moose. Some cysts have been cut open to reveal cystic layers on the inside that produce multiple scoleces by asexual budding, which develop into adult tapeworms following ingestion by the definitive host.

FIG. 5.   Anterior of an adult *Ancylostoma ceylanicum* showing teeth used to lance capillaries in mucosa of small intestine in order to suck blood.

frequently occur together in enteric polyparasitic infections, often with *H. nana* and protozoa such as *Giardia* and *Entamoeba* [21,22,24,48]. However, only hookworms suck blood, leading to iron deficiency anaemia.

Transmission is not direct but occurs via the environment and the infective stage is the third stage larva, which develops in the environment following the passage of unembryonated eggs in the faeces of infected individuals. The development of first stage larvae (L1) in the eggs takes approximately 24 hours, after which the eggs hatch and subsequent larval development from L1 to L2 and then to L3 takes place in the environment within approximately 1 week, after which the larvae can survive in warm damp soil for up to 2 years [46,47]. Humans are infected following skin penetration by the third-stage larvae and in the case of *A. duodenale* also following ingestion of the larvae and penetration of the mucosa. The free-living phase of the hookworm life cycle is an important factor in the epidemiology of hookworm infection, which must be taken into account in control programmes in which regular mass drug administration is used to break the cycle and reduce incidence and lead to eradication [24,28].

Ancylostoma ceylanicum is a hookworm of cats and dogs that has long been known to have the ability to establish patent enteric infections in humans but is considered the most neglected of all human hookworm species [49]. As such, it is the only zoonotic hookworm with this ability and has probably been overlooked in the past because of difficulties in specific diagnosis using morphological criteria [24,49,50]. Until recently, infections with *A. ceylanicum* were considered uncommon in humans. However, because the eggs of all hookworm species cannot be differentiated morphologically it is not possible to determine species based on the microscopic differentiation of eggs in faeces. With the recent development of PCR-based techniques that can differentiate between all species of hookworm in humans, dogs and cats, *A. ceylanicum* has been shown to have a much broader geographical distribution in humans than previously thought [24,50,51]. Recent molecular epidemiological surveys in Asia have shown that *A. ceylanicum* is the second most common species of hookworm infecting humans [50]. Importantly, in developing areas of South-East Asia, the situation is exacerbated by a growing awareness of the role of dogs in the transmission of *A. ceylanicum* in some areas [24]. In many hookworm endemic areas, dogs are common in villages and communities. A number of recent studies have demonstrated infections with *A. ceylanicum* in humans and dogs from the same hookworm endemic foci [24,50,52]. *Ancylostoma ceylanicum* was not the dominant hookworm in these studies with *A. duodenale* or *N. americanus* more prevalent, but 10–20% of people examined were infected with *A. ceylanicum*. Mixed infections are common in both humans (*A. duodenale* ± *N. americanus* + *A. ceylanicum*) and dogs (*A. caninum* + *A. ceylanicum*). Dogs therefore represent a reservoir of human infections that must be considered when using mass drug administration as a means of controlling hookworm infection in the human population. Recent studies in Laos suggest that the limited success of mass chemotherapy programmes targeted at humans in rural communities may have been because the source of some of the human infections was *A. ceylanicum* from dogs [24].

In Australia, a recent study of wild dogs (dingoes and dingo hybrids) in peri-urban areas of northern Australia found that up to 100% were infected with *A. ceylanicum*, so constituting a zoonotic risk to communities in this tropical area of northern Australia [53].

The clinical impact of *A. ceylanicum* in humans is not fully understood. Heavy infection can result in bloody diarrhoea and iron-deficiency anaemia but unlike the other human hookworm, it can also cause severe enteritis as well as cognitive impairment [24,49,54–57]. This complicates the clinical management of hookworm disease in endemic areas where *A. ceylanicum* contributes to the burden of hookworm infection [24].

Concluding comments

Emerging issues with the epidemiology of infections caused by *H. nana*, *E. canadensis* and *A. ceylanicum* demonstrate the importance of continued surveillance in endemic areas, and particularly the value of molecular epidemiological tools. All three parasites serve to highlight the need for greater awareness in endemic areas in terms of ongoing control efforts. However, in all cases anthropogenic factors are key to limiting their impact on public health, particularly in terms of education and hygiene.

Transparency declaration

I have no conflicts of interest.

Acknowledgments

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