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Veterinary and public health aspects of *Toxocara* spp

Paul A.M. Overgaauw*, Frans van Knapen

Institute for Risk Assessment Sciences, Division Veterinary Public Health, Utrecht University, P.O. Box 80175, 3508 TD, Utrecht, The Netherlands

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ABSTRACT

Pet dogs and cats can play an important role in the transmission of zoonotic nematodes such as *Toxocara canis* and *Toxocara cati*, by excreting eggs directly into the human environment, without the involvement of vectors or intermediate hosts. Human toxocarosis remains a hazard despite the availability of highly effective anthelmintics for dogs and cats. A good understanding of the biology and epidemiology of these parasites, and the risk factors that lead to their transmission to humans is required for effective prevention strategies. In this respect, the maintenance of high quality continuing education for veterinarians and the provision of suitably presented information to pet owners are of priority importance. A closer collaboration between veterinary and public health professionals within the 'One Health' concept is also required.

1. Introduction

The close relationship of people with their companion animals, known as the human–animal bond, provides benefits with regard to socialisation, mental health and even physical wellbeing (Paul et al., 2010). But alongside these benefits of pets for the human population, there is also potential health hazards associated with the ownership of a pet. Besides the risk of bites, scratches and allergies, cats and dogs harbour the enteric nematodes *Toxocara canis* and *Toxocara cati*, which can be transmitted to humans. These parasites have an oral–fecal transmission cycle and humans can be infected by ingestion of larvae in undercooked infected organ or muscle tissues (rare); infective eggs from contaminated soil (gardens, sandpits and playgrounds); from unwashed hands or raw vegetables, or by direct contact with pets (Hill et al., 2000; Robertson and Thomson, 2002; Wolfe and Wright, 2003). This review focuses on actual aspects of toxocarosis as a major helminth zoonosis.

2. Toxocarosis

2.1. Occurrence of *Toxocara* spp. in dogs and cats in Europe

Reported infection rates of *T. canis* and *T. cati* in Western Europe vary from 3.5% to 34% for *T. canis* in dogs from different epidemiological environments (pet, shelter, stray, and rural dogs) and from 8% to 76% for *T. cati* in cats (Parsons, 1987; Overgaauw, 1997b; Fok et al., 2001; Habluetzel et al., 2003; Le Nobel et al., 2004; Dubná et al., 2007; Martínez-Carrasco et al., 2007; Lee et al., 2010). Worm burdens and prevalences of patent *Toxocara* spp. infections are highest in puppies and young dogs less than 6 months of age and in kittens. More research is needed on the dynamics of infection in older dogs, so that optimum control strategies can be devised. Age-independent intensity of infection estimates have been obtained for adult dogs lacking any clinical symptoms (Lloyd, 1993; Overgaauw, 1997b; Barutzki and Schaper, 2003; Sager et al., 2006; Martínez-Moreno et al., 2007) and therefore the epidemiological importance of adult dogs as a reservoir of infection should not be underestimated. Increases in vole and other rodent populations in urban areas also attracts predators, including foxes, and can represent a reservoir for *Toxocara* spp. and other

* Corresponding author. Tel.: +31 302535367.
E-mail address: p.a.m.overgaauw@uu.nl (P.A.M. Overgaauw).

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zoonotic helminths of pet dogs and cats (Reperant et al., 2009).

2.2. Transmission of Toxocara spp. eggs to humans

Humans usually become infected by oral ingestion of embryonated *Toxocara* spp. eggs from contaminated soil on unwashed hands or raw vegetables (Glickman and Shofer, 1987). People can also acquire infection by ingesting larvae present in undercooked meat or offal from infected paratenic hosts such as chickens, ruminants or pigs (Nagakura et al., 1989; Stürchler et al., 1990; Baixench et al., 1992; Salem and Schantz, 1992; Taira et al., 2004). Humans act as a paratenic host in which *Toxocara* spp. larvae migrate and then survive for months or years encysted in various body tissues.

Toxocara spp. eggs are unembryonated and non-infective when excreted in the faeces of dogs and cats. Eggs can develop to the infective larvated stage within a period of 3 weeks to several months, depending on soil type and environmental conditions such as temperature and humidity. Embryonated eggs have remained viable for at least 1 year under optimal circumstances (Parsons, 1987). Studies from all over the world have demonstrated high rates (10–30%) of soil contamination with *Toxocara* spp. eggs in back yards, sandpits, parks, playgrounds, lake beaches, and other public places (Mizgajska-Wiktor and Uga, 2006). *T. canis* eggs were found to be most common in public parks, while the majority of investigated sand-boxes were contaminated with *T. cati* eggs (Jansen et al., 1993). Mean egg density is highest in faeces from pups less than 8 weeks old. Urban and rural foxes all over Europe commonly carry patent infection, but their overall contribution to environmental contamination has been estimated to be less than one tenth of that attributed to dogs (Morgan et al., 2009). However, the epidemiological impact of *Toxocara* spp. eggs from foxes needs to be quantitatively analysed for different environmental situations. Prevalences are usually higher in cubs under 6 months of age, but they remain high even in adult foxes (Reperant et al., 2007).

Several recent studies have suggested that direct contact with the fur of an infected dog may be an important zoonotic source of *Toxocara* spp. eggs (Wolfe and Wright, 2003; Aydenizöz-Özkayhan et al., 2008; Roddie et al., 2008). Nevertheless, other workers do not consider this to be a significant risk because the eggs need several weeks to become infective (Overgaauw, 1997a; Overgaauw and van Knapen, 2000, 2004; Nagy et al., 2011; Keegan and Holland, 2012). Although a low percentage of eggs in fur have been observed to be embryonated, they are mostly not viable. In addition, *Toxocara* spp. eggs are very adhesive and difficult to remove from dog or cat hair, which lessens the likelihood of them, being accidentally swallowed by a human. Even in the worst-case scenario of an animal with highly contaminated fur, it would be necessary to ingest several grams of hair to present a significant risk of infection (Overgaauw et al., 2009; Keegan and Holland, 2010).

The role of *T. cati* as a zoonotic parasite is not always clearly recognised. Despite the fact that differentiation between *T. canis* and *T. cati* infections is still not feasible in serological surveys, the majority of reported human cases of toxocarosis have been associated with *T. canis* based on epidemiological considerations (Fisher, 2003). The large number of common antigenic fractions shared between *T. canis* and *T. cati* and the similarity in the mode of infection are indications that there is no difference in the zoonotic risk (Cardillo et al., 2009). Furthermore, in Islamic countries, where dogs are avoided for religious reasons, while cats are favoured as pets, the seroprevalence of human toxocarosis can be considerable (Smith and Noordin, 2006). Thus, the potential role of *T. cati* in human toxocarosis should not be ignored or underestimated (Fisher, 2003; Overgaauw, 1997a; Smith and Noordin, 2006).

2.3. Human toxocarosis

A number of different syndromes have been attributed to *Toxocara* spp. infection: visceral larva migrans (VLM), ocular larva migrans (OLM), and covert toxocarosis (CT). In addition, associations with neurological and atopic symptoms have also been described.

The diagnosis of human toxocarosis is based on clinical presentation, laboratory tests and sero-diagnostic techniques. ELISA and WB are currently the most reliable tools for detecting antibodies and circulating antigens (Smith and Noordin, 2006). As anti-*Toxocara* spp. antibodies measured by ELISA persist for up to 2.8 years in infected adults, their presence alone does not distinguish between current and past infections and does not allow a probable or definitive diagnosis of clinically relevant toxocarosis. Therefore, other laboratory tests, mainly peripheral eosinophil count and total serum IgE, are needed in the diagnostic workup of suspected cases (Magnaval and Glickman, 2006). For OLM, serum antibodies are not diagnostic, but intraocular antibodies appear more promising as a diagnostic aid (Taylor, 2006). The major pitfall in the diagnosis of human toxocarosis is the lack of standardized serodiagnostic criteria and case definitions (Smith et al., 2009).

The seroprevalence of *Toxocara* spp. antibodies varies between countries. Anti-*Toxocara* spp.-antibodies were found in 4.6–7.3% of children in the USA (Herrmann et al., 1985), in 2.5% in Germany, and in up to 83% in the Caribbean (Thomson et al., 1986). In the Netherlands, the prevalence was found to be 4–15% in individuals under 30 years of age and 30% in adults older than 45 years, with an average figure of 19% (De Melker et al., 1995). Continuous re-infection is probably the cause of the higher prevalence in adults.

In computer tomography or magnetic resonance imaging (MRI), hepatic lesions may be seen as multiple, ill-defined, oval lesions that measure 1.0–1.5 cm in diameter. In sonography, the lesions appear as multiple, small, oval hypoechoic lesions in the liver parenchyma (Lim, 2008). MRI can be used in patients with neurological syndromes to detect granulomas located cortically or subcortically (Magnaval and Glickman, 2006).

Children are more frequently clinically affected than adults, and severe VLM is mainly seen in children from 1 to 3 years of age. A worldwide review of VLM and OLM cases revealed that more than half of the patients were younger than three years, and only one fifth were adults. Sixty percent were males (Ehrhard and Kernbaum, 1979).

The higher infection risk in children can be explained by their behaviour, since young children play more often in, and thus have closer contact with, potentially contaminated soil in yards and sandpits and often put their fingers into their mouths, sometimes even eating dirt. In a well-conducted study of Irish school children, the prevalence of consultant-diagnosed eye disease was 6.6 cases per 100,000 children (Good et al., 2004). Relationships between *Toxocara* spp. antibody seroprevalence and the incidence of chronic airway disorder (asthma), elevation of serum IgE concentration, the presence of allergen-specific IgE and eosinophilia, have been found (Buijs et al., 1997, Pinelli et al., 2008). Occurrence of asthma or recurrent bronchitis and hospitalization due to asthma were significantly related to seroprevalence, while eczema tended to be more frequent in children aged 4–12 years in the Netherlands. It was concluded that a previous infection with *Toxocara* spp. leads to exacerbation of allergic phenomena in children, who are predisposed to asthma (Buijs et al., 1997; Hakim et al., 1997; Chan et al., 2001; Cooper, 2008; Pinelli et al., 2008). Other studies performed in the USA could not confirm these associations (Sharghi et al., 2001). Interestingly, these findings were recently challenged by a study revealing that these serological associations could also have been due to cross-reactions to *Ascaris suum* (Muñoz-Guzmán et al., 2010). In the Netherlands, data from a seroepidemiological survey indicated that almost 50% of clinical toxocarosis cases were associated with concurrent *Ascaris suum* and *Toxocara* spp. infections (Van Knapen et al., 1992) and the *Ascaris* seropositivity is significantly higher compared to the *Toxocara* spp. seropositivity. Furthermore, while *Toxocara* spp. seropositivity has decreased over time, the *Ascaris* seropositivity has not significantly changed for the past decade (Pinelli et al., 2011). Chronic 'idiopathic' urticaria, chronic pruritus, and miscellaneous eczemas in adults and children have also been associated with toxocarosis (Piarroux et al., 2006; Gavignet et al., 2008). Nevertheless, a systematic population-based study on skin pathology and *Toxocara* spp. seropositivity is lacking (Smith et al., 2009).

2.4. Aspects of Toxocara spp. biology in definitive and intermediate hosts

Infection with *Toxocara* spp. arises therefore from three sources: intrauterine (dog) and lactogenic larval transmission (dog and cat); ingestion of embryonated eggs from the environment; and ingestion of larvae (excreted with infected newborn's faeces and ingested by the mother or via consumed paratenic hosts) (Overgaauw, 1997a).

There are four epidemiological reservoirs: intestinal infections in definitive hosts, eggs in the environment, larvae in paratenic hosts, and somatic larvae in the host (Schnieder et al., 2011).

Somatic larvae gradually accumulate in canine and feline tissues, persisting for long periods in a manner similar to that seen in paratenic hosts. Larvae of *T. cati* prefer to migrate to the muscles of paratenic hosts, while *T. canis* larvae are more likely to be found in the central nervous system of dogs (Sprent, 1958) and paratenic hosts (Cardillo et al., 2009).

2.4.1. Intrauterine and lactogenic larval transmission

Nearly 100% of puppies are infected *in utero* by re-activated somatic larvae from day 42 of the gestation period (Lloyd et al., 1983). This transplacental migration and intra-uterine infection is the most important mode of transmission in pups. It results in egg excretion after a minimum period of 16 days after birth (Lloyd, 1993). After birth, puppies and kittens also acquire infection through ingestion of larvae in the milk (Parsons, 1987), which can be passed for at least 38 days after parturition (Zimmerman et al., 1985), although this route normally contributes fewer worms than intra-uterine transmission (Coati et al., 2004). Kittens are only infected by lactogenic transmission and will commence faecal egg excretion from 47 days after birth.

2.4.2. Ingestion of larvae

After predation of *Toxocara* spp. infected paratenic hosts by dogs or cats, larvae will be released and develop in most cases directly to adult worms in the intestinal tract (Sprent, 1956, 1958).

2.4.3. Ingestion of embryonated eggs

After ingestion of infective *T. canis* eggs by dogs and cats, hatched larvae migrate by the tracheal route and eggs first appear in dog faeces 4–5 weeks post-infection (Parsons, 1987; Fahrion et al., 2008; Schnieder et al., 2011) and in cat faeces 8 weeks after infection (Sprent, 1956). Patent *T. canis* infections are much less common in dogs older than 6 months than in puppies, and faecal egg-counts are generally much lower (Greve, 1971; Claerebout et al., 2009). The theory of this "age resistance" as a result of acquired immunity in dogs older than 6 months, however, conflicts with the observation that adult dogs, experimentally inoculated with low numbers of embryonated eggs, may also develop patent infections (Fahrion et al., 2008). This suggests that immunity has little influence on the outcome of the experimental infection. To further complicate the picture, inoculation with a larger number of eggs has been found to be less likely to produce a patent infection, even in non-immune dogs. Greve (1971) employed an infective dose of 2000 eggs, which in one year old, ascarid-naive dogs led to only two of four animals developing patent infections. Similarly, Dubey (1978), using 10,000 eggs, reported that no puppies (aged 62–64 days) developed a patent infection. Thus, we may be faced with a therapeutic paradox: that the more effectively exposure is reduced by control measures, the greater the risk of older animals developing patent infections. Experimental data and prevalence studies in dogs worldwide have determined that patent infections are independent of gender and age in adult dogs (see Section 3.1). As in dogs, age resistance seems to be of little importance in older cats (Visco et al., 1978). This may be explained by the fact that older cats catch paratenic hosts (prey animals) more often than dogs. The subsequent direct development of *Toxocara* spp. larvae is not only responsible for a higher prevalence of patent infections, but will also fail to induce immunity as there is no migration stage in the cat's tissues.

2.5. Diagnosis in dogs and cats

Routine diagnosis of patent *Toxocara* spp. infection is achieved by demonstrating the presence of the characteristic eggs in faeces. Dryden et al. (2005) recommend a sedimentation and flotation technique because centrifugation consistently recovers more eggs than other methods. The eggs of *T. cati* and *T. canis* are, however, not clearly distinguishable by microscopy (Uga et al., 2000). A PCR technique utilising the second internal transcribed spacer (ITS-2) of ribosomal DNA (rDNA) was therefore developed to allow species identification (Jacobs et al., 1997). A RAPD-PCR technique and comparison of the ITS-2 sequence were used for the molecular characterisation of *T. canis* isolates from dogs and foxes, which revealed no intraspecific variations (Epe et al., 1999). However, this kind of analysis is not routinely available for diagnostic laboratories. In a recent study (Fahrion et al., 2011) *Toxocara* spp. eggs were isolated and measured and the species determined by PCR. Among the isolates originating from dogs, 24 (68.5%) were determined as *T. canis* and surprisingly 11 (31.5%) as *T. cati*. Coprophagy is suggested as an explanation of this finding. In all samples originating from cats, only *T. cati* eggs were identified.

Following PCR identification, eggs of *T. canis* and *T. cati* were measured, revealing a statistically significant difference ($p < 0.001$) between the mean sizes of 62.3 by 72.7 1-μm for *T. cati* and 74.8 by 86.0 1-μm for *T. canis* eggs. Considering that coprophagy is not unusual for dogs, a considerable percentage of *Toxocara* spp. infections diagnosed in dogs might in fact relate to the intestinal passage of eggs following the uptake of other animal's faeces (Nagy et al., 2011). This observation also highlights potential problems in the interpretation of post-treatment faecal egg-counts.

3. Control strategies addressing the dog and cat population

3.1. Prevention of environmental contamination

Toxocara spp. eggs are very resistant to adverse environmental conditions and remain infective for years (Parsons, 1987). Since no practical methods exist for reducing environmental egg levels, prevention of initial contamination of the environment is the most important approach. This can be achieved by taking several measures such as eliminating patent infections in dogs and cats, preventing defecation of pets in public areas, hygiene, and educating the public (Glickman and Shofer, 1987). Methods to decrease contamination include: restriction of free-roaming dogs and cats, cleaning up of faeces from soil and on pavements by dog owners, preventing access of dogs and cats to public places (especially children's playgrounds), and the use of strategic anthelminthic treatment of dogs and cats with emphasis on puppies, kittens, nursing bitches and queens.

3.2. Anthelmintic treatment of pups and kittens

The most potent source of *Toxocara* spp. eggs for environmental contamination is *T. canis* from the bitch nursing a litter. In one study, faecal egg-counts up to 107,500 epg were recorded from three-week old greyhound pups. The mean count for pups in the litter reached a maximum of 11,500 epg at 28 days of age with a smaller secondary peak at 6 weeks (Jacobs and Fisher, 1993).

A major aim of long-term prophylactic treatment programmes is to suppress *T. canis* egg output throughout the whole period of puppy-hood using a multidose schedule. Puppies should be treated with appropriate anthelmintics starting at the age of 2 weeks and repeated treatments at suitable intervals are necessary because milk transmission occurs continuously for at least 5 weeks post-partum (Barriga, 1991). The schedule is therefore deworming at 2, 4, 6 and 8 weeks of age and then monthly until 6 months of age (ESCCAP, 2010). Because prenatal infection does not occur in kittens, fortnightly treatment can begin at 3 weeks of age. The preventive treatment of feline hookworm infection can also begin at this. Nursing bitches and queens should be treated concurrently with their offspring since they often develop patent infections at this time.

3.3. Anthelmintic treatment of adult dogs and cats

Periodic treatments with anthelmintics, or treatments based on the results of periodic diagnostic faecal examinations, are of great value for the control of intestinal helminths in dogs and cats. Annual or bi-annual treatments have no significant impact on preventing patent infection within a population (Sager et al., 2006). Thus, an average treatment frequency of 4 times per year is proposed as a general recommendation depending on the life style and life stage of the dogs and cats. Based on the prepatent period in adult dogs, a regular treatment every 4–6 weeks would prevent most patent infections (Fahrion et al., 2008). Monthly administration of macrocyclic lactones suppresses nearly all canine nematodes (Deplazes et al., 2006).

3.4. Anthelmintic treatment of pregnant dogs and cats

Anthelmintics at the recommended doses are not highly effective against inhibited somatic larvae (Epe, 2006), and regular anthelmintic treatment of bitches before mating and 2 weeks before the anticipated whelping date has no useful effect on prenatal transmission (Fisher et al., 1994; Epe et al., 1996). An extended daily treatment with fenbendazole from day 40 of pregnancy to 2 days post-whelping is available in some countries to reduce prenatal transmission of *Toxocara* spp. larvae. Other regimens have been described, but have not been commercialised. Therefore, it is generally not advised to deworm pregnant dogs and cats with the aim of reducing *Toxocara* spp. larval transfer.

3.5. Uniform guidelines for control and treatment of parasites

A treatment schedule individually designed for pets based on certain infection risks (e.g. free roaming, uncontrolled access to rodents or offal, contact with other animals) can improve treatment efficiency. Uniform guidelines for the control and treatment of parasites in pet

animals were developed and published by CAPC in the USA (CAPC, 2012) and ESCCAP in Europe (ESCCAP, 2010). These guidelines give an overview on different worm species, their clinical and zoonotic significance, and suggest rational control measures for the most important species in order to prevent animal and/or human infection. However, even strict compliance by the pet owners will not reduce the environmental contamination with *T. canis* eggs originating from foxes (Deplazes et al., 2004). Coordinated control programmes aimed at minimizing infection pressure from zoonotic parasites attributable to the considerable European fox and stray cat populations have not so far been implemented.

Conflict of interest statement

None.

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