Epidemiology of *Toxascaris leonina* infection post-weaning within a colony of dogs

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Abstract

This communication reports incidental observations on *Toxascaris leonina* infections in a beagle breeding colony. Regular faecal monitoring demonstrated that *T. leonina* was endemic in the adult dam population within this colony. Small numbers of *T. leonina* eggs were also detected in the faeces of weaned pups from eight weeks of age possibly produced by a patent infection. This would mean a pre-patent period for *T. leonina* of 56 days or less. Worm counts on 10 pups showed that 60% of pups had acquired a *T. leonina* infection by 12 weeks of age. Since prenatal and lactogenic transmission do not occur and as the pups were kept in an environment which reduced chances of infection with *T. leonina* and there was no apparent source of paratenic hosts, the source of infection must have been embryonated *T. leonina* demonstrate that, if pups are exposed to an infected environment, patent infections may be seen in a younger age group than is normally associated with *T. leonina* infections.

Introduction

Toxascaris leonina has been largely overlooked as a candidate for scientific investigation since the work of Sprent (1959). The parasite is seen as unimportant because it is neither widely implicated as a zoonosis, nor in causing ill health in dogs, whereas *Toxocara canis* has merited investigation on both of these counts. Therefore, relatively less is known about the basic life cycle and epidemiology of *Toxascaris leonina*.

Infection with *T. leonina* is derived from ingestion of infective larvae inside eggs or within paratenic hosts. Once ingested by a suitable final host, worms develop within the intestine without migration, unlike *Toxocara canis*. There appears to be a good deal of variation in the prepatent period. Okoshi & Usui (1968) found, for example, patency to occur 48 days after ingestion of embryonated eggs in some dogs, while Sprent (1959) reported the pre-patent period to be 56 days in dogs and 74 days in cats. Unfortunately, only the longer of these figures was quoted in one of the major veterinary

parasitology reference texts (Soulsby, 1982), and this appears to have led to an understanding that the prepatent period in dogs is longer than it actually is.

Infection is relatively infrequent within the dog population and where it does occur, is generally associated with adolescent or adult dogs, infection being acquired as dogs are introduced to grass runs, for example (Jacobs *et al.*, 1988; Georgi & Georgi, 1991). Endemic infection may be sustained in relatively clean environments, such as colonies of experimental dogs (Georgi & Georgi, 1991).

This communication reports incidental observations on *Toxascaris leonina* infection in a breeding colony of dogs.

Materials and methods

The dog colony contained predominantly beagles and was a closed community. Standards of housing and care of animals were in accordance with the relevant guidelines as required by local and national legislation and codes of practice. All dogs were housed in concrete floored pens with wire screens dividing pens to allow contact. All pens were cleaned with high pressure water once daily. Between litters, adult dams were kept in

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environment.

groups of six to eight, with each dam moved into an individual pen approximately one week prior to the anticipated whelping date. Prior to September 1997, the anthelmintic regimen consisted of a single annual treatment of adult dogs with nitroscanate (Troscan 500, Chanelle). From September 1997 anthelmintic treatment was replaced by regular monitoring of faecal egg counts.

During the period of monitoring from September 1997 to May 1998, 151 faecal samples were taken from dams and examined for nematode eggs using a sensitive centrifugal flotation technique.

Ten pups from five litters born during 1998 were weaned by 6 or 8 weeks of age and thereafter maintained in individual pens that allowed visual and auditory contact with other dogs. Animal technicians regularly socialized with the pups inside their pens. Pens were cleaned out twice daily with water and additionally cleaned once weekly with a dilute solution of Armillatox[®] (Armillatox Ltd., Alfreton, UK) which contains soft soap, potassium hydroxide and 30% high boiling tar and has been shown to destroy ascarid eggs (Armillatox Ltd, personal communication). Faecal samples were collected from individual pups at intervals until the pups were approximately 12 weeks of age. Faecal samples were examined for nematode eggs using a sensitive centrifugal flotation technique. Post-mortem examinations were carried out at approximately 12 weeks of age following an intravenous overdose of pentobarbitone sodium. The contents of the stomach and gastrointestinal tract were washed over a 140 µm sieve. The contents were examined grossly, then re-examined under a $\times 10$ dissecting microscope to ensure retrieval of all worms or worm larvae. Individual worms were examined under a light microscope using $\times 40$ magnification to confirm their identity as T. leonina, to confirm the stage of development and to allow counts to be performed. Toxascaris leonina was distinguished from Toxocara canis primarily by the absence of an oesophageal ventriculus. Other features such as the appearance of the eggs in the adult female and the shape of the male tail were used to confirm identity.

In order to identify the source of the Toxacaris. leonina infection in the whelping environment, faecal samples were taken at intervals from dams with pups, and sellotape strips, each approximately 4.5 cm long by 2.5 cm wide were applied to the floors and whelping boxes in eight whelping pens. The sellotape strips were removed and each strip applied to a microscope slide. Each slide was systematically searched for ascarid eggs using $\times 40$, and where necessary, $\times 100$ magnification.

Results

During the monitoring period from September 1997 to May 1998, 151 faecal samples were taken from dams and of these, 46 were positive for T. leonina; all positive samples occurred in the period from March to May 1998. In most cases where T. leonina was present, it was the only nematode identified, although in three samples there was a mixed infection with Toxocara canis. Of 15 samples collected in March, 73% were positive for Toxascaris leonina. Prevalence in May, at the end of the observation period, had decreased to 29.6%. These

Pup С Е F Η А В D G Ι J Faecal egg counts* 0 0 8 0 Day 0** 2 1 Day 11 _ 0 0 0 0 0 0 Day 17 0 0 0 0 0 3 0 0 0 1 Day 24 0 8 0 1 1 0 0 0 2 1 Day 33 0 0 105 0 3 0 58 4 0 2 Day 40 0 0 221 23 0 0 0 2 13 1 T. leonina worm counts 0 0 0 0 0 0 0 L4 0 2 1 I 5 2 0 40 g 3 0 0 0 0 0 7 Adult female 1 1 1 1 0 0 1 0 0 0 0 0 0 0 Adult male 0 4 0 0 1 Total 43 21 5 3 0 0 0

Table 1. Faecal egg-counts and Toxascaris leonina worm counts

from pups exposed to T. leonina infection in the whelping

*No. of eggs per gram faeces **Day 0: pups aged between 6 and 8 weeks; –, no sample.

1 0

1

figures demonstrate that T. leonina was endemic in the adult dam population within this colony.

Ten pups from five litters born during 1998 were monitored from weaning to approximately 12 weeks of age for nematode infections. In only one of ten monitored pups (pup C) was the dam found to be depositing T. *leonina* eggs. Data obtained from the pups are shown in table 1, including the number of *T. leonina* eggs per gram of faeces on each sampling occasion and individual worm counts. Small numbers of T. leonina eggs were detected in the faeces of pups from 8 weeks of age. At post-mortem examination, adult worms were detected in six pups and immature T. leonina were present in four pups. Of the ten pups examined, 60% had acquired a *T. leonina* infection by 12 weeks of age. All ten pups were infected with *Toxocara canis*, but there did not appear to be any association between levels of infection of *T. canis* and Toxascaris leonina.

To identify the source of the T. leonina infection in the whelping environment, sellotape strips were applied to the floors and whelping boxes in eight whelping pens. No T. leonina eggs but Toxocara canis eggs were identified on the sellotape strips.

Discussion

This communication reports incidental observations on Toxascaris leonina infection in a breeding dog colony. The colony was housed in concrete-floored pens, demonstrating once again the ability of T. leonina to maintain infection in a relatively clean environment. The distinguishing feature of the infection in this colony was that infections were seen in younger dogs than normal. Patent T. leonina infections were seen in dogs immediately after weaning, with the infection apparently derived from the whelping environment.

Results from faecal samples taken from dams during September 1997 to May 1998 demonstrated that T. leonina was endemic in the adult dam population. The reason for the prevalence of patent infections apparently increasing in March 1998 is not clear. It may represent new infections in previously uninfected individuals, or it may be simply that the infection was not detected in previous samples or that a number of dams were at a stage in their oestrous cycle when a patent infection was more likely.

Monitoring a cohort of ten pups kept in an environment which reduced the chances of infection demonstrated that *T. leonina* was also endemic in post-weaning pups of this colony. Small numbers of *T. leonina* eggs were detected in the faeces of pups from 8 weeks of age. These could be either eggs that were simply passing through the intestine of the dog or eggs produced by a patent infection. If this were a patent infection, it would represent a pre-patent period for *T. leonina* of 56 days or less. By 12 weeks of age, 60% of the ten pups had acquired a *T. leonina* infection.

No *T. leonina* eggs were identified on the sellotape strips. So, other than the one pup where the dam was found to have a patent infection, the source of *T. leonina* infection could not be established. However, as prenatal and lactogenic transmission do not occur (Sprent & English, 1958; Okoshi & Usui, 1968) and there was no apparent source of paratenic hosts, the source of the infection must have been embryonated *T. leonina* eggs from the whelping environment. Pups must have begun to ingest eggs from their environment within days of birth for patency to have occurred so early in life.

It is possible that sellotape strip sampling was not an appropriate method to detect \overline{T} . *leonina* eggs in the environment, although the method was successful in the detection of *Toxocara canis* eggs. These observations of *Toxascaris leonina* demonstrate that, if pups are exposed to an infected environment, patent infections may be seen

in a younger age group than is normally associated with *T. leonina* infections.

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