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Baylisascariosis—Infections of animals and humans with 'unusual' roundworms

Christian Bauer*

Institute of Parasitology, Justus Liebig University Giessen, Rudolf-Buchheim-Strasse 2, 35392 Giessen, Germany

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ABSTRACT

The nematode genus *Baylisascaris* (order Ascaridida, superfamily Ascaridoidea) contains nine relatively host-specific, parasite species of carnivores, omnivores, herbivores, carnivorous marsupials or rodents. They have a facultative heteroxenous life cycle, at least under experimental conditions. Eggs passed in faeces embryonate in the environment and the second-stage larva infective for both definitive and intermediate hosts develops. In intermediate hosts larvae migrate extensively through tissues, where they grow and moult to the third-stage, causing extensive damage. All *Baylisascaris* spp. are considered a potential cause of visceral, ocular and/or neural larval migrans in mammals including humans and in birds. This paper summarises our current knowledge on the prevalence, biology, pathogenicity and zoonotic significance of three *Baylisascaris* species: *B. transfuga*, *B. schroederi* and *B. procyonis* which have as definitive hosts bears, giant pandas and raccoons (occasionally dogs), respectively.

1. Introduction

The genus *Baylisascaris* (superfamily Ascaridoidea) contains nine species that can be differentiated using morphological criteria and in the case of four species by genotypic analysis of the mitochondrial genomes (Xie et al., 2011a,b). They are relatively host-specific parasites of the small intestine of carnivores, omnivores, herbivores, carnivorous marsupials or rodents (Table 1). Adult female worms can reach lengths of 14–28 cm and males 7–12 cm (Sprent, 1968). *Baylisascaris* spp. eggs are not embryonated when passed in fresh faeces. The eggs embryonate in the environment and become infective within 2–4 weeks (Sakla et al., 1989; Papini and Casarosa, 1994). Unlike other ascarid species such as *Toxocara canis* (Bruňaská et al., 1995) and *Ascaris suum* (Geenen et al., 1999) in which the third-stage larva is the infective stage within the egg, second stage larvae in *Baylisascaris* spp. eggs are considered to be the infective stage, with the second moult thought to occur in the infected host animal. All *Baylisascaris* spp. have a facultative heteroxenous life cycle, at least under experimental conditions (Sprent, 1953). When ingested by animals such as mice, the embryonated eggs hatch and second-stage larvae start to migrate through organs and tissues. During the migration phase the larvae grow considerably (Bowman, 1987; Goldberg et al., 1993) and moult to the third-stage (*Baylisascaris procyonis* third stage larvae are 1300–1900 μm in length) meaning these animals serve as intermediate hosts. This is in contrast to *T. canis* whose larvae (approximately 400 μm long; Goldberg et al., 1993) do not further develop in mice (paratenic hosts; Schnieder et al., 2011). The migrating *Baylisascaris* spp. larvae can cause extensive tissue damage, and all members of this genus are considered potential causes of visceral, ocular and/or neural larval migrans in mammals including humans and in birds.

This review focuses on three important *Baylisascaris* spp.: *B. transfuga* which occurs worldwide in bears, *B. schroederi*, a species which is pathogenic in its definitive host, the giant panda, and *B. procyonis*, of raccoons, which is

* Tel.: +49 641 9938463; fax: +49 641 9938469.
E-mail address: christian.bauer@vetmed.uni-giessen.de

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Table 1
List of *Baylisascaris* spp., their natural definitive hosts and geographical distribution.

Species	Definitive hosts	Geographical distribution
B. ailuri (Wu, He and Hu, 1987)	Red panda (*Ailurus fulgens*)	Asia (China)
B. columnaris (Leidy, 1856)	Skunks (*Mephitis* spp.)	North America
B. devosi (Sprent, 1952)	Mustelids (*Martes* spp.)	North America, eastern Europe, Asia (Russia)
B. laevis (Leidy, 1856)	Rodents (*Marmota* spp., *Citellus* spp.)	North America
B. melis (Gedoelst, 1920)	Badger (*Meles meles*)	Europe
B. procyonis (Stefanski and Zarnowski, 1951)	Raccoon (*Procyon lotor*), other procyonids (*Potos flavus*, *Bassaricyon gabbii*), domestic dog	North and possibly Central America, central Europe, Asia (Japan)
B. schroederi (McIntosh, 1939)	Giant panda (*Ailuropoda melanoleuca*)	Asia (China)
B. tasmaniensis (Sprent, 1970)	Carnivorous marsupials (*Sarcophilus harrisi*, and others)	Australia (Tasmania)
B. transfuga (Rudolphi, 1819)	Bears (*Ursus* spp.)	Arctic, North America, Europe, Asia

the most pathogenic species for intermediate hosts, including man.

2. Baylisascaris transfuga

2.1. Geographic distribution, prevalence and intensity of infection

B. transfuga occurs worldwide in both free-ranging and captive bears such as the American black bear (Foster et al., 2004), European brown bear (De Ambrogi et al., 2011), and polar bear (Testini et al., 2011). In a recent survey 13% of 96 faecal samples from free-ranging European brown bears in Croatia contained ascarid eggs (De Ambrogi et al., 2011). In Florida Foster et al. (2004) reported black bear cubs harboured 1–39 roundworms in their intestines. Infected, captive bears may shed as many as 10,000–20,000 eggs per gram of faeces (Papini et al., 1994) and therefore heavily contaminate their domestic area.

2.2. Life cycle

The life cycle of *B. transfuga* is unclear but it is assumed that infections occur following ingestion of embryonated eggs from the environment. It is not known whether prey animal intermediate hosts are a source of *B. transfuga* infection for omnivorous or carnivorous bears under natural conditions. Because adult worms are first detected in bear cubs from five months of age (Foster et al., 2004) intrauterine or lactogenic transmission of larvae is unlikely to occur. The endogenous development of *B. transfuga* in definitive hosts and the prepatent period remain to be elucidated.

2.3. Pathogenic importance

In bears, the pathogenicity of intestinal infection with *B. transfuga* appears low; one case of granulomatous peritonitis caused by roundworms was described in a cub (Szczepaniak et al., 2012). In contrast, it has been shown that in rodents, e.g. white mice and Mongolian gerbils, which are susceptible to experimental infection with *B. transfuga*, following infection the larvae migrate through different tissues growing and developing to the third-stage, causing various degrees of visceral, neural or ocular larva migrans (Sprent, 1953; Papini et al., 1996; Sato et al., 2004; Cho et al., 2007). Whereas experimentally infected chickens did not show clinical signs although larvae were present in tissues (Papini et al., 1993). There is anecdotal evidence from epidemiological observations that *B. transfuga* was the possible cause of an outbreak of fatal larva migrans in Japanese macaques (Sato et al., 2005). To date however, there is no unequivocal evidence of naturally occurring *B. transfuga* infection in non-ursid animals or humans.

3. Baylisascaris schroederi

3.1. Geographic distribution, prevalence and intensity of infection

Giant pandas are the definitive host of *B. schroederi*, and its geographical distribution is therefore restricted to China. In recent faecal surveys *B. schroederi* infections were detected in 54% of 126 and 48% of 31 free-ranging pandas examined by classical coprological and molecular biology methods, respectively (Zhang et al., 2011, 2012). Surprisingly, the faecal egg output was rather low (Zhang et al., 2011). Intestinal roundworm burdens varied from 1 to 619 worms in 11 animals at post mortem (Xue, 1987, cited by Zhang et al., 2012).

3.2. Life cycle

Because giant pandas are herbivorous animals, infection is assumed to occur by ingestion of embryonated eggs in soil or from faecally contaminated plant material (monoxenous cycle) (Wu et al., 1985, cited by Zhang et al., 2011). There is no evidence of intrauterine or lactogenic transmission of *B. schroederi* larvae from dam to cub. In experimentally infected mice, larvae did not migrate into the placenta (Li, 1990a). From studies in experimentally infected mice (Li, 1990a) and from post-mortem findings in pandas, it is postulated that *B. schroederi* larvae migrate through liver and lungs of pandas and may also