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Experimental infection in chickens with larvae of *Baylisascaris transfuga* (Nematoda: Ascaridoidea)

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Abstract. The larvae of *Baylisascaris transfuga* (Rudolphi, 1819) were able to penetrate the liver, lungs, carcass and brain of infected chickens, but a great number of larvae accumulated in the liver. No clinical signs were seen. Birds may serve as paratenic hosts of the parasite, but *B. transfuga* seems not to be a possible agent of avian cerebrospinal nematodosis.

The life cycle of species from the genus *Baylisascaris* Sprent, 1968 involves carnivores as definitive hosts, and small mammals, mainly rodents, as intermediate hosts: infectious larvae migrate to and lodge in various organs of the latter. Being abnormal hosts, birds also become infected; the larvae invade the central nervous system, causing disease in emus, partridges, chickens, quails, turkeys and pigeons. Kazacos and Wirtz (1983) reported 100 % mortality in 85 quails, and 6.5 % in 1000 chickens.

Kazakos and Wirtz (1983) studied avian cerebrospinal nematodosis in chickens, which had been experimentally inoculated with *Baylisascaris procyonis* (Stefanski et Zarmowski, 1951) infective eggs.

Experimental studies on infections resulting from ascarid larvae in birds are few: Galvin (1964) reported the infection of chickens and pigeons with *Toxocara canis* (Werner, 1782); Okoshi and Usui (1968) with *T. canis*, *T. cati* (Schrank, 1788) and *Toxascaris leonina* (von Linstow, 1902) in chickens; Pahari and Sasmal (1990) with *T. canis* in quails.

Baylisascaris transfuga (Rudolphi, 1819), the common roundworm found in bears, is the typical species of the genus *Baylisascaris*. The migratory behaviour of the larvae of the parasite was studied by Sprent (1951, 1952, 1953, 1955) and by Matoff and Komandarev (1965). In experimentally infected mice, the larvae migrated into the intestine, liver, lungs and reached the muscles and brain. However, searching through the literature, we found that similar experiments had never been carried out with birds.

As species from the genus *Baylisascaris* are the only known cause of avian cerebrospinal nematodosis, we infected chickens by an oral administration of *B. transfuga* embryonated eggs to establish whether this *Baylisascaris* species may possibly cause avian cerebrospinal nematodosis.

MATERIALS AND METHODS

Specimens of *B. transfuga* were obtained by treating three polar bears, *Thalarctos maritimus* (Phipps, 1774) in the Zoological Garden, Pistoia, Italy, with piperazine adipate. Nematode eggs were obtained by dissecting adult *B. transfuga* females, the eggs were kept in 0.1 N H_2SO_4 at an ambient temperature of 25 °C in Petri dishes for about 30 days until use.

Thirty-five 3-day-old male chickens (*Gallus domesticus* L.) of White-Leghorn breed, obtained from a commercial breeder, were kept in metal cages, in an infection free status and fed a commercial diet and water *ad libitum*.

Each chicken was infected by stomach tube directly into the crop with approximately 5000 embryonated eggs of *B. transfuga*, suspended in 1 ml distilled water.

The chickens were examined daily for clinical signs and were killed in groups of 5 by diethyl-ether on days 3, 5, 7, 10, 15, 20 and 30 post-infection (p.i.).

The liver, lungs, carcass and brain were examined for the presence of larvae: the liver and lungs by the Baermann technique, the brain by flattening it between two glass slides and the carcass by pepsin digestion as described by Sprent (1952).

For each chick, the number of larvae in each organ was recorded; for each group of five chicks, the average number, and percentage, for each organ, were calculated (Table 1).

RESULTS

The results are shown in Table 1. The larvae were able to penetrate the chickens. Of all the examined organs, the liver yielded the maximum number of larvae. The highest recovery rate from the liver was 84.2 % at 3 days p.i., and subsequently remained so. The minimum recovery was 68.2 % at 5 days p.i. and the maximum 91.0 % at 15 days p.i.

The recovery rate from the lung was 7.8 % at 3 days p.i. and thereafter declined to 1.2 % at 15 days p.i. But at 20 days p.i. there was an influx of larvae (4.3 %) before the numbers again decreased to a negligible number (0.4 %) at 30 days p.i.

The carcass recovery rate was already 7.7 % at 3 days p.i., then rose to a peak of 25.2 % at 5 days p.i. and thereafter varied between 7.0 % at 15 days p.i. and 24.2 % at 7 days p.i.

Table 1. Results of experimental infection of 35 chickens (*Gallus domesticus*) with 5000 embryonated eggs of *Baylisascaris transfuga* each on day 0. On days 3, 5, 7, 10, 15, 20 and 30 post-infection (p.i.), batches of 5 chicks were sacrified and their bodies searched for larvae; the average number, and percentage, for each batch of five, are given.

Day p.i.	Average no. (%) of larvae recovered				Total no.
	Liver	Lungs	Carcass	Brain	
3	277.8 (84.2 %)	25.8 (7.8 %)	25.4 (7.7 %)	0.8 (0.2 %)	329.8
5	178.8 (68.2 %)	16.2 (6.1 %)	66.2 (25.2 %)	0.6 (0.2 %)	261.8
7	195.2 (69.7 %)	16.0 (5.7 %)	67.8 (24.2 %)	1.0 (0.3 %)	280.0
10	254.4 (90.1 %)	5.2 (1.8 %)	21.4 (7.6 %)	1.4 (0.4 %)	282.4
15	221.4 (91.0 %)	3.0 (1.2 %)	17.2 (7.0 %)	1.6 (0.6 %)	243.2
20	131.4 (81.5 %)	7.0 (4.3 %)	21.6 (13.4 %)	1.0 (0.6 %)	161.2
30	200.2 (82.4 %)	1.2 (0.4 %)	39.5 (16.2 %)	2.2 (0.8 %)	242.8

The recovery rate from the brain was very low (below 1 %) at all times, even if this progressively increased from 0.2 % at 3 days p.i. to 0.8 % at 30 days p.i.

DISCUSSION

Table 1 shows that the localization of larvae in chickens resembled that in mice (Sprent 1951, 1952, 1953, 1955, Matoff and Komandarev 1965, Papini and Mancianti 1990), with somatic migration occurring early in the infection.

In our results there is some suggestion that *B. transfuga* larvae accumulated in the brain with increasing time but the percentages obtained were constantly small (from 0.25 to 0.8 % at 3-30 days p.i.). During the entire experimental period (30 days) there was no mortality and no clinical signs. Kazakos and Wirtz (1983) reported that chickens inoculated with *B. procyonis* eggs, at dosages of 400-3200 eggs, showed torticollis, ataxia, circling, external rigidity and paralysis, within 17-23 days p.i. *B. procyonis* larvae seem to have a special effect on the central nervous system as observed by Lindquist (1978), who suggested to use this for testing anthelminthics against migrating larvae. In addition, *B. procyonis* larvae are larger than *B. transfuga* larvae. Richardson et al. (1980) reported the recovery from chickens of *B. procyonis* larvae measuring 1560 µm, while the larvae of *B. transfuga* we found in the brain of infected chickens measured on average 788 µm (10 larvae) at 30 days p.i. It is quite likely as put forward by Lindquist (1979, personal communication to Richardson et al. 1980) that chickens provide a better substrate of growth for *B. procyonis* larvae.

In contrast to mice, a large number of larvae were consistently recovered from the liver. The tendency to accumulate in the liver of avian hosts was also seen in chickens, pigeons and quails infected with *T. canis* larvae (Galvin 1964, Okoshi and Usui 1968, Pahari and Sasmal 1990). This could be due to a lower adaptability of larvae to avian tissues than to those of mammals (Okoshi and Usui 1968, Mittra and Sasmal 1985, Pahari and Sasmal 1990).

We did not attempt to determine whether part of the larvae accomplished tracheal migration and returned by this way to the intestine. However, the percentage of larvae recovered from the lung gradually decreased in the course of the infection, while the percentage recovered from the carcass varied independently of that. This suggests that some tracheal migration probably occurred.

The total number of larvae recovered was greatest at 3 days p.i. and thereafter declined. This demonstrates that with time increasing numbers of larvae were destroyed or absorbed by the chickens, which tallies with the results obtained by Agarwal and Johri (1980).

It appears from our observations that *B. transfuga* can persist for relatively long periods in the tissues of birds. Most of the larvae recovered were alive during the examination. Matoff and Komandarev (1965) transferred *B. transfuga* larvae from mouse to mouse after 7 days of infection. The larvae of nematodes infect both avian and mammalian hosts (Okoshi and Usui 1968, Mittra and Sasmal 1985, Pahari and Sasmal 1990). Therefore birds as well as mammals may serve as paratenic hosts of *B. transfuga*.

Our experiments show that although *B. transfuga* is closely related to *B. procyonis* and *B. columnaris* (Leidy, 1856), the evidence for transmission of avian cerebrospinal nematodosis by *B. transfuga* is not consistent.

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