Filaroides hirthi: Experimental Transmission Among Beagle Dogs Through Ingestion of First-Stage Larvae

Abstract. *Four beagle dogs were found to be infected with sexually mature* Filaroides hirthi *lungworms from 1 to 3 months after they ingested material containing first-stage larvae of this parasite. Infection by first-stage larvae opens the theoretical possibility of autoinfection of which few nematode parasites are capable.*

Infections with the lungworm *Filaroides hirthi* are of great practical importance because (i) the parasite is enzootic in all five of the major research beagle breeding establishments in the United States (*1*); (ii) the associated lesions may be confused with pathological changes induced by drugs, oncogenic influences, and other pathogenic organisms (*2*); and (iii) antemortem diagnosis is impossible because larvae cannot be demonstrated in the blood, secretions, or excretions of infected beagle dogs (*2*, *3*). Infection with *F. hirthi* is thus widespread in beagle dogs used for research and causes lesions that interfere with the pathological evaluation of experimental material. Transmission of this infection is certainly efficient because the infection rate is virtually 100 percent in at least one of the affected establishments (*3*). The diet fed at this establishment was suspected to be the source of infection because it contained uncooked meat. However, when fed for 14 weeks to ten uninfected beagle pups as the only form of nourishment, this diet failed to produce a single infection (*3*). In addition, I have identified *F. hirthi* infection in beagle dogs that were raised from weaning exclusively on commercial dried dog food.

Dorrington (*4*) reported success in transmitting *Filaroides osleri* infection by feeding dogs with first-stage larvae obtained from nodules in the trachea and bronchi of infected dogs. Dunsmore and Spratt (*5*) confirmed this by infecting captive dingoes, dingo–domestic dog crosses, Labrador dogs, and a fox with first-stage larvae, "some hatched, others unhatched." This is an exceptional result because all but a very few nematode parasites (*6*) require a period of development outside of the definitive host or as parasites of an alternate host. For example, all members of the superfamily Metastrongyloidea (to which the genus *Filaroides* belongs) whose life histories have been studied, develop to the infective stage in a mollusk or an annelid. Thus far, in spite of intensive searching at this laboratory, we have been unable to find any evidence that an intermediate host, even a facultative one, is involved in the life cycle of *F. hirthi* (*3*).

Because of *F. hirthi*'s small size (*7*) and widely scattered distribution, isolation of first-stage larvae is tedious and difficult. Therefore, I ground up entire lungs infected with *F. hirthi* and, on 1 August, and again on 15 August 1975, fed them to one of five beagle dogs from an uninfected source. Fifteen other beagle dogs belonging to the same lot had been found free of infection on postmortem examination. On 15 November 1975, I found adult *F. hirthi* male and female worms in the lungs of this dog but none in two controls that I examined at the same time. I now ground up these new infected lungs and fed them to one of the two remaining dogs. A week later I received infected lungs from another source and fed 28 small pieces, each containing at least one gravid female worm, to the same dog. On 20 February 1976, I found *F. hirthi* in the lungs of this dog but none in the control. The infective agents transmitted by feeding lung tissue could only have been the first-stage larvae encased in their eggshells in the uteri of female worms, because the only other life stages that I could demonstrate in this material were pubescent and mature male and female worms. To establish the infectivity of first-stage larvae beyond doubt I completely isolated gravid female worms from lung tissue and administered these by gelatine capsule to two beagle pups. From 10 to 30 worms were thus administered each week for 4 weeks starting on 24 May. On 24 and 25 June, these pups were killed and autopsied and found to be infected with *F. hirthi*, whereas two control pups were found to be free of infection.

I conclude that the infective agents were the first-stage larvae. Thus, autogenous reinfection with *F. hirthi* becomes at least a theoretical possibility. Autogenous reinfection would in turn help to explain the extraordinary apparent longevity of *F. hirthi* infection and the concurrence in the lungs of dogs, both young and old, of living worms surrounded by normal lung tissue and of disintegrating worms surrounded by granulomatous reactions (*3*). In an evolutionary context, this behavior could be viewed as a substitution, by the parasite, of definitive host-tissues for the tissues of an intermediate host as a substrate for the development of larval stages 2 and 3.

JAY R. GEORGI

Department of Pathology, New York State College of Veterinary Medicine, Cornell University, Ithaca, New York 14853

References and Notes

1. I have repeatedly identified *F. hirthi* in beagle lung tissue submitted by two of these establishments. Other workers have reported to me that they have diagnosed this infection in beagle dogs supplied by the other three establishments.
2. R. S. Hirth and G. H. Hottendorf, *Vet. Pathol.* **10**, 385 (1973).
3. J. R. Georgi, W. J. Fleming, R. S. Hirth, D. J. Cleveland, *Cornell Vet.* **66**, 309 (1976).
4. J. E. Dorrington, *Onderstepoort J. Vet. Res.* **35**, 225 (1968).
5. J. D. Dunsmore and D. M. Spratt, abstract of paper presented at the meeting of the Australian Society for Parasitology, Melbourne, Australia, 17 to 18 May 1976.
6. *Probstmayria vivipara* (Oxyurata), *Capillaria phillipinensis* (Trichurata), and the hyperinfective form of *Strongyloides stercoralis* (Rhabditata) are the only nematode parasites of domestic animals and man known to be capable of completing their life histories and thus of multiplying within their host.
7. Male specimens average 2.8 mm in length and 0.038 mm in width; females average 9.9 mm in length and 0.084 in width.

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Antiserum to Brain Gangliosides Produces Recurrent Epileptiform Activity

Abstract. *A single injection of 10 microliters of antiserum to total brain ganglioside onto (and into) the sensorimotor cortex of the rat resulted in recurrent spiking in the cortical electroencephalogram, lasting from 7 to 17 days. Absorption of antibody with pure monosialoganglioside (G_{M1}) completely abolished the effect. Spiking was reactivated after 4 weeks by intramuscular injection of pentylenetetrazole.*

The essential problem in epilepsy is to understand mechanisms underlying discharges which characterize the "hyperphysiological" state at the epileptogenic focus in the cortex (*1*). Experimental models of epilepsy involving tissue damage (freezing, or application of alumina cream or cobalt), pharmacological agents (such as penicillin and strychnine), or electrical stimulation have not provided a clearly defined target for examining mechanisms. In contrast, the immunoneurological model (*2*) provides such a target because of the well-recognized molecular specificity inherent in antigen-antibody reactions. Exploitation