Canine *Babesia* New to North America

Abstract. *A domestic dog residing in New England suffered a fatal febrile illness caused by a* Babesia *infection. The morphology of these intraerythrocytic protozoa and the range of hosts that could be infected experimentally suggested that the parasite was* B. gibsoni. *Although this tick-borne disease is enzootic in wild and domestic Canidae in Africa and Asia, it appears to be new to the Americas.*

A standard poodle in Ridgefield, Connecticut, was found to be infected with a protozoan parasite identified as *Babesia gibsoni*. The dog was born in Massachusetts on 12 September 1977, transferred to New York State 5 weeks later, and brought to Connecticut in November 1977. The dog was taken to Chatham, Massachusetts, and was housed in a kennel in Bedford, New York, during May of 1978. Unidentified ticks were removed from the dog in May. The dog became ill on 7 June 1978, exhibiting vomiting, anorexia, a rectal temperature of 40°C, and, subsequently, severe anemia. *Babesia* parasites were abundant in Giemsa- and Field's-stained blood films (*1*), and the animal died on 21 October 1978. From 7 weeks after the dog became ill until it died, the number of blood cells containing parasites ranged between 0.2 and 1.6 percent.

Three species of *Babesia* infect dogs (*2*), but only *B. canis* has previously been reported from dogs indigenous to the Americas (*3*). The appearance of the parasites in this pet dog was inconsistent with that of *B. canis*. Accordingly, we attempted to isolate and identify the etiologic agent.

Blood was drawn from the dog during the early stages of illness and inoculated intraperitoneally (*4*) into two intact golden hamsters and into one mouse (*Peromyscus leucopus*), one raccoon (*Procyon lotor*), and two mongrel dogs. The last four animals had been splenectomized. All animals had been screened before injection and found to be free of *Babesia*. The raccoon and dogs were at least 1 year of age. Twenty-seven days after inoculation, blood was drawn from the two recipient dogs and stored in ethylenediaminetetraacetic acid or a mixture of anticoagulant, citrate, and dextrose for 6 weeks at 5°C. Portions of blood (5 ml) were then inoculated (second passage) into a splenectomized male and a nonsplenectomized female dog. Blood smears of each mammal were prepared and stained prior to inoculation and at weekly intervals thereafter (*5*). All inoculated dogs became infected. Parasites were similar in appearance to those found in the original pet dog (Fig. 1). The rodents and the raccoon remained uninfected.

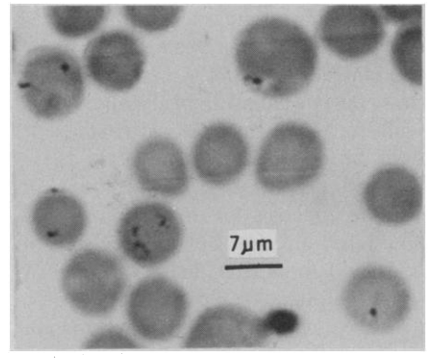
The cytoplasm and chromatin dot of the oval-to-spherical parasites stained light blue and dark red, respectively. Parasites in the original dog measured 1.9 ± 0.1 by 1.2 ± 0.18 μm (N = 10), whereas those in the recipient dogs were 1.8 ± .08 by 1.3 ± 0.1 μm (N = 10). In the first canine passage, parasites were present 6 days after inoculation, infecting less than 0.01 percent of erythrocytes examined (N = 10,000). Parasitemia in one dog (a splenectomized male) increased to 29 percent shortly before death at 27 days after inoculation (Fig. 2); temperature and hematocrit were 41.5°C and 7, respectively. Of 200 infected erythrocytes examined, 75, 21, 3, and 1 percent were infected with one, two, three, and four parasites, respectively. The splenectomized female simultaneously developed a parasitemia of 8.8 percent and had a temperature of 38.5°C and a hematocrit of 40. In a further passage (passage 2) into two more dogs (a splenectomized male and a nonsplenectomized female) parasitemia developed in both animals 7 days after inoculation. Parasitemia increased to 8 percent in the nonsplenectomized dog after 33 days and gradually declined to less than 1 percent by 63 days. In the splenectomized male, parasitemia reached 32.4 percent after 50 days (2 days prior to death).

We identified this parasite as *B. gibsoni* because (i) the original host was a dog; (ii) the erythrocytic forms were similar in structure, size, and staining properties to *B. gibsoni* described from domestic and wild canines in Africa and Asia (*1*, *6*–*8*); and (iii) parasitemia developed in one nonsplenectomized and three splenectomized dogs inoculated with infected blood.

The remaining known *Babesia* species that infect dogs, as well as those infecting mammals in the northeastern United States, are excluded as etiologic agents because of their morphology, size, or host range (Table 1). The ring forms of *B.*

Table 1. Characteristics of *Babesia* that infect canines in Africa and Asia or mammals from northeastern United States.

Babesia species	Hosts	Differential morphology of Giemsa-stained intraerythrocytic parasites	Dimensions (μm)	Reference
gibsoni	Canines	One to many lightly stained cytoplasmic rings	1.0 to 3.5	(*8*)
canis	Canines	Paired pyriform bodies	3.0 by 5.0	(*3*, *8*, *11*, *20*)
vogeli	Canines	Paired pyriform bodies	> 3.0 by 5.0	(*3*, *8*, *11*, *20*)
microti	Rodents and primates	One to four intensively stained cytoplasmic rings	1.5 to 2.5	(*9*, *21*)
procyoni	Raccoon	One or more intensively stained cytoplasmic rings	1.9 to 2.1	(*22*)
mephitis	Striped skunk	Paired pyriform bodies	2.5 by 4.8	(*10*)



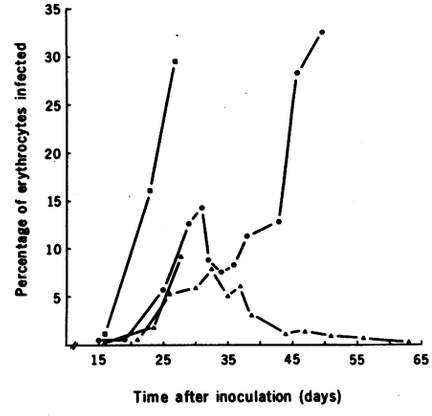


Fig. 1 (left). Field's-stained *Babesia* from a New England dog. Fig. 2 (right). Parasitemia in dogs infected with the New England *Babesia*: ■, male splenectomized dog (first passage) that died 27 days after inoculation; △, female splenectomized dog (first passage) that was killed 27 days after inoculation; and ●, male splenectomized dog (second passage) that died 52 days after inoculation; ▲, female nonsplenectomized dog (second passage) that was alive with a parasitemia of < 0.1 percent at 63 days after inoculation.

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gibsoni contrast with the larger pearlike forms of *B. canis* and *B. vogeli*, the two other *Babesia* species known to infect canines, and *B. mephitis* known from the striped skunk (*Mephitis mephitis*). Lightly staining cytoplasm differentiates *B. gibsoni* from *B. microti* and *B. procyoni*, the other oval *Babesia* in northeastern United States. In addition, our isolate did not parasitize a splenectomized raccoon or rodents, each of which regularly become infected when they are injected intraperitoneally with blood containing parasites of *B. procyoni* and *B. microti*, respectively. Furthermore, previous attempts to infect splenectomized dogs with *B. microti* (*9*) and *B. mephitis* (*10*) were unsuccessful. Serologic tests for identifying *Babesia* species are unavailable (*11*).

Entry of *B. gibsoni* into the United States could have been by way of infected domestic or captive canines or ticks. Although quarantines have been established and ticks have commonly been intercepted at ports of entry (*12*), some ticks have escaped detection and entered the United States (*13*). Both domestic and captive wild canines are allowed entry under federal quarantine restrictions (*14*), but such animals, including those from Asia and Africa, are not routinely examined for babesiasis. At least one pet dog, known to be infected with *B. gibsoni*, was brought into the United States in 1967, prompting the prediction that this parasite might again be introduced here (*15*).

Our report constitutes the first record of this potentially important *Babesia* of canines being transmitted in North America. The primary tick vector of *B. gibsoni* is *Haemaphysalis bispinosa* (*16*); this tick occurs in India, but not in the Americas. *Rhipicephalus sanguineus*, an introduced tick that is common in kennels and houses in North America (*17*), may transmit the organism (*18*), although the evidence is not conclusive (*16*). Other ticks on dogs in New York and New England include *Ixodes cookei, I. dammini*, and *Dermacentor variabilis* (*19*), but the tick that transmitted this New England infection remains unknown.

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4. The following quantities of blood drawn from the poodle and stored in EDTA at 5°C for 24 to 48 hours were inoculated into recipient mammals: male raccoon, 0.7 ml; *P. leucopus*, 0.1 ml; two golden hamsters, each 0.2 ml; and male and female mongrel dogs, each 1.5 ml.
5. Blood smears were prepared weekly from the raccoon for 10 weeks; from *P. leucopus* for 12 weeks; from the golden hamsters for 5 and 15 weeks; and from the male and female dogs for the first passage, 5 weeks, and for the second passage, for 7 weeks from the male and 10 weeks from the female.
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23. All animals were given humane care during this study. We thank C. Lemmon, A. Whitney, F. Degennaro, E. McDonnell, L. Gallette, W. L. Krinsky, and A. Main for their assistance. The following veterinarians helped in various ways: G. D. Whitney, New Haven Central Hospital for Veterinary Medicine; D. F. Anderson and R. S. Schoemann, Guilford Veterinary Hospital; G. Geering and J. R. Dann, Ridgefield Veterinary Hospital; M. T. Lender, Orange Veterinary Hospital; and R. O. Jacoby, Yale. The Yale Arbovirus Unit provided isolation facilities. This study was supported in part by CDC contract 200-76-0063.

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Laterality of Stereognostic Accuracy of Children for Words, Shapes, and Bigrams: A Sex Difference for Bigrams

Abstract. *Children identified nonsense shapes by touch better with their left hand and words better with their right hand. Bigrams were processed by boys as shapes and by girls as words, which suggests a sexual dimorphism of brain functioning for bigrams. A relative specialization of the hemisphere for stereognostic processing is also suggested, since the accuracy of identification by both hands was greater than chance for all three types of stimuli.*

The right and left sides of the human brain, the two cerebral hemispheres, are joined by the interconnecting (commissural) structure, the corpus callosum. The belief that the left hemisphere plays the dominant role in language functions while the right one does this for processing of spatial stimulation has received strong support from tests of commissurotomized patients, that is, those whose corpus callosum has been cut surgically for sufficient medical reasons. It could be deduced from tests of these adult patients (*1*) that the accuracy of hand stereognosis for linguistic material should be superior by children's right hands and for spatial material superior by their left hands. Of course, one would have to assume that these functions are already sufficiently lateralized in childhood. Indeed, Witelson (*2, 3*) found with a two-handed simultaneous stereognostic test that, if boys felt nonsense shapes with the fingers of their left hand, they recognized them more accurately from a visual display than if they had touched them with the fingers of their right hand. However, although girls were as accurate as boys, girls recognized these same shapes equally well with the fingers of either hand (*3*). Witelson (*2*) also found that boys were equally accurate with both hands in identifying single-letter shapes they had previously felt. This might suggest that linguistic function, as far as stereognostic input is concerned, is not yet lateralized in children.

Several studies (*4-6*), however, show that both boys and girls of the ages tested by Witelson have a right ear superiority for processing linguistic material presented in dichotic listening tests. Moreover, while there is no clear superiority in children's right visual field for processing single Hebrew letters, such has been shown for two-letter Hebrew words (*7*). These results (*4-7*) can be interpreted to mean that even in young children the left (dominant) hemisphere is specialized for linguistic function for stimulus presentation in either the auditory or the visual modalities. Whether Witelson's (*2, 3*)

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