EXPERIMENTAL INFECTIONS OF BABESIA BIGEMINA IN AMERICAN BISON

Authors: Jerry L. Zaugg, and Kenneth L. Kuttler
Source: Journal of Wildlife Diseases, 23(1) : 99-102
Published By: Wildlife Disease Association
URL: https://doi.org/10.7589/0090-3558-23.1.99
EXPERIMENTAL INFECTIONS OF BABESIA BIGEMINA IN AMERICAN BISON

Jerry L. Zaugg¹ and Kenneth L. Kuttler²

ABSTRACT: Babesia bigemina was experimentally transmitted from cattle to bison and back to cattle. One spleen-intact and two splenectomized American bison (Bison bison) inoculated with a B. bigemina stabilate exhibited clinical and hematological signs of babesiosis within 10 days of exposure. Blood from the infected bison produced disease in a splenectomized bovine steer.

INTRODUCTION

Bovine babesiosis, caused by the erythrocytic protozoan parasite, Babesia bigemina, was eliminated from the United States following the successful eradication of its vector tick, Boophilus annulatus, in 1943 (Kuttler, 1984). However, both the parasite and its Boophilus tick vectors are prevalent within Mexico and elsewhere in the world (McCorker, 1981), providing a continuous threat of re-introduction of the disease into the United States.

Animals other than cattle are known to be susceptible to B. bigemina and may act as inapparent carriers. Water buffalo (Bubalus bubalis) may become infected (Callow et al., 1976). African buffalo (Syncerus caffer) and splenectomized gazelles (Gazella sommermerring) also are susceptible (Robertson, 1976). “Medium-sized” and “large” forms of Babesia, which were morphologically similar to B. bigemina, have been described from dromedaries (Nagay, 1947; Leeflang, 1977).

American bison (Bison bison) in England were found to be very susceptible to B. major, a relatively non-pathogenic babesia for cattle (Findlay and Begg, 1977).

American bison are maintained in ever increasing numbers throughout the United States, and their distribution includes areas previously inhabited by tick vectors of Babesia spp. (Simonton, 1985). Cattle and bison are sometimes grazed together, and as such any mutually infectious disease could result in tragic losses from both animal groups.

The present investigation was undertaken to determine whether American bison are susceptible to experimental infection of B. bigemina.

MATERIALS AND METHODS

The three bison used in this study were obtained as calves from the National Bison Range, Moise, Montana, 18 mo previously for use in anaplasmosis research (Zaugg and Kuttler, 1985). All three bison remained free of any chemical treatment and appeared clinically healthy at the beginning of the present study. Two bison, a male (B1) and a female (B17) were splenectomized as calves (Zaugg, 1984). Another female (B18) remained spleen intact. A 3-yr-old splenectomized Brahman × Jersey cross steer (#45) was kept with the bison in an isolated open pen.

All four initial test animals were inoculated intravenously with a stabilate (Lumsden and Hardy, 1965) made from B. bigemina-infected bovine blood. (Two ml of a 1:10 dilution of blood with a 3% parasitemia, therefore about 5.5 × 10⁷ infected erythrocytes.) The frozen stabilate was thawed and inoculated into each animal within 15 min of thawing. Beginning 3 days after inoculation, blood was obtained from all test animals daily and examined for packed cell volume (PCV) and percent of parasitized erythrocytes (PPE). Daily rectal temperatures were also recorded.

Seven days after inoculation 10 ml of whole blood, pooled from all three bison, were inoculated into a susceptible, splenectomized Holstein steer (R1) to establish a bovine-bison-bo-
| Days after inoculation (IV) | PCV<sup>1</sup> | PPE<sup>1</sup> | TEMP | H<sup>2</sup> | PCV | PPE | TEMP | H  | PCV | PPE | TEMP | H  | PCV | PPE | TEMP | H  | PCV | PPE | TEMP | H  |
|-----------------------------|----------------|------------|------|-----------|------|------|------|----|------|------|------|----|------|------|------|----|------|------|------|
| 3 | 30 | N<sup>4</sup> | 102.0 | - | 32 | N | 101.0 | - | 45 | N | 102.0 | - | 31 | N | 102.8 | - | 30 | N | 101.5 | - |
| 4 | 30 | N | 101.4 | - | 32 | N | 103.0 | - | 44 | N | 102.0 | - | 32 | N | 101.0 | - | 33 | N | 101.8 | - |
| 5 | 30 | <0.1 | 100.4 | - | 31 | 0.8 | 105.0 | - | 44 | N | 101.0 | - | 34 | 0.5 | 99.8 | + | 34 | N | 102.2 | - |
| 6 | 33 | 1.3 | 102.2 | - | 35 | 6.6 | 106.2 | + | 47 | N | 102.9 | - | 35 | 6.3 | 103.2 | + | 32 | 0.2 | 100.0 | - |
| 7 | 31 | 4.9<sup>4</sup> | 102.0 | + | 18 | 12.0 | 106.2 | + | 41 | N | 102.0 | - | 9 | 6.1 | 99.6 | + | 33 | 0.7 | 101.6 | - |
| 8 | 26 | <0.1 | 102.6 | + | 10 | 6.3 | 100.4 | + | 41 | <0.1 | 101.8 | - | 30 | 1.3 | 102.8 | - | 29 | 2.9 | 101.2 | - |
| 9 | 21 | N | 100.6 | + | 42 | 0.1 | 101.2 | + | 25 | 5.1 | 99.8 | + | 33 | 0.7 | 101.6 | - | 29 | 2.9 | 101.2 | - |
| 10 | 21 | N | 99.8 | - | 37 | 2.5 | 103.0 | + | 25 | 5.1 | 99.8 | + | 33 | 0.7 | 101.6 | - | 29 | 2.9 | 101.2 | - |
| 11 | 24 | N | 99.6 | - | 35 | 4.5<sup>4</sup> | 106.0 | + | 22 | 5.5 | 102.4 | + | 33 | 0.7 | 101.6 | - | 29 | 2.9 | 101.2 | - |
| 12 | 25 | N | 101.8 | - | 35 | N | 101.8 | + | 18 | 6.8<sup>4</sup> | 102.6 | + | 15 | N | 101.2 | - | 29 | 2.9 | 101.2 | - |
| 13 | 25 | N | 101.4 | - | 33 | N | 101.2 | - | 41 | <0.1 | 101.8 | - | 29 | 2.9 | 101.2 | - | 29 | 2.9 | 101.2 | - |

* Splenectomized.
1 Recipient of pooled infected bison blood.
2 PCV = packed cell volume (%); PPE = percentage of parasitized erythrocytes; TEMP = rectal temperature (F); H = presence of hemoglobinuria (— = not present, + = present).
4 Negative.
5 Treated with diminazene aceturate.
vine sequence. Its condition was monitored daily as described above.

Bison B1 and B18, and steer R1 were treated with diminazene aceturate at 3 mg/kg (Gana-seg, E. R. Squibb and Sons de Mexico, S.A. de C.V., Mexico 20, D.F.) intramuscularly within 4 days of observable signs of acquired infection to prevent death.

RESULTS AND DISCUSSION

All test animals developed acute babesiosis. The prepatent period, defined here as the time from exposure to the time that a PPE of 0.5% or greater was detected, was 5 and 6 days in splenectomized bison B17 and B1 respectively and 10 days in the spleen-intact bison B18 (Table 1). The disease became clinically manifest in steer 45 in 5 days and in steer R1 in 7 days (Table 1). The prepatent periods observed in this study were similar to the 4- to 5-day periods usually seen in blood inoculation studies with bovines (Kuttler, 1984).

Following inoculation, moderate to severe hemolysis and hemoglobinuria were first observed within 5 days in steer 45, 6 days in bison B17, 7 days in bison B1, 9 days in bison B18 and 10 days in steer R1 (Table 1). Hemolysis and hemoglobinuria were moderate in bison B1, but severe in all of the other test animals. Urine appeared as pools of frothy blood and sera were dark red-black. These conditions, however, disappeared within 3 days of treatment in bison B1 and B18.

Figure 1 graphically shows the nearly identical courses of anemia of the two pairs of splenectomized animals receiving similar treatment. No intervention of the disease process was made with bison B17 nor steer 45 and their PCV values plunged simultaneously. Bison B17 died 9 days and steer 45 died 8 days after exposure to B. bigemina. Bison B1 and B18, and steer R1 were therapeutically treated and their PCV values reflected a similar pattern. Further, their parasitemias were reduced to near zero within 24 hr after Gana-seg treatment (Table 1, Fig. 2).

As seen in Figure 1 bison B18 exhibited a notably higher PCV value throughout the study. The higher PCV values were in close agreement with the average value of
45.8% reported from 132 clinically normal wild bison (Peterson and Roby, 1975). Bison B18 was the only spleen-intact bison under study and as such the differences of PCV and prepatent period associated with this animal may possibly be attributed to the function of the spleen. The time of appearance of parasites in all test animals was quite similar (Fig. 2). Of the two animals allowed to proceed to death, the bison B17 actually exhibited the highest PPE value (12.0%) on day 7 post exposure. Dramatically, the observable parasitemias virtually disappeared within 24 hr after treatment in both bison and bovine subjects. The parasitemias did not recrudescence for up to 45 days.

As shown in Table 1, there was a rise in rectal temperature associated with acute infection. Body temperatures of bison B17 and steer 45 were highest concurrently with the detected parasitemia climax, then dropped dramatically the day before they died. All other animals under test exhibited a reduction in temperature within 2 days of treatment. It was difficult to determine “normal” body temperatures, or to ascribe elevated readings to a response to disease because of the physical excitement exhibited by the animals (except steer R1) while working them. Steer R1 was maintained by himself and was much calmer. There was no real variation in his temperature.

American bison were demonstrated to be susceptible to experimental exposure to B. bigemina. Infections were confirmed by passage to a susceptible calf, establishing a bovine-bison-bovine sequence. As a result, bison are potentially at risk should this disease organism regain entry into the United States. Additional precaution should be exercised in babesiosis prevention in connection with the export of bison and/or bison-bovine crosses to B. bigemina endemic areas.

ACKNOWLEDGMENTS
We extend our gratitude to Mr. Jon Malcolm of the National Bison Range, Moise, Montana for making the bison calves available. We thank Burke Newman and Will Harwood for technical assistance.

LITERATURE CITED