EXPERIMENTAL ANAPLASMOSIS IN AMERICAN BISON: PERSISTENCE OF INFECTIONS OF ANAPLASMA MARGINALE AND NON-SUSCEPTIBILITY TO A. OVIS

Author: Zaugg, Jerry L.

Source: Journal of Wildlife Diseases, 22(2) : 169-172

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-22.2.169

Journal of Wildlife Diseases, 22(2), 1986, pp. 169–172

EXPERIMENTAL ANAPLASMOSIS IN AMERICAN BISON: PERSISTENCE OF INFECTIONS OF *ANAPLASMA MARGINALE* AND NON-SUSCEPTIBILITY TO *A. OVIS*

Jerry L. Zaugg

U.S. Department of Agriculture, Agricultural Research Service, Hemoparasitic Diseases Research Unit, Caldwell Veterinary Teaching Center, Caldwell, Idaho 83605, USA

ABSTRACT: Blood collected 314 and 496 days after experimentally infecting splenectomized and spleen-intact American bison (*Bison bison*) with *Anaplasma marginale* was infective for splenectomized bovine steers. The pathogenesis was identical to that seen in bovine studies using bovine blood inoculations. A splenectomized bison remained normal clinically, hematologically and serologically for 10 mo after repeated inoculation of ovine blood infected with *A. ovis*.

INTRODUCTION

Anaplasmosis is a disease of ruminants, characterized in severe cases by anemia, icterus and death, caused by intraerythrocytic rickettsial agents of the genus *Anaplasma*. While *A. marginale* is the primary pathogen in bovine anaplasmosis, it is not confined to cattle, nor is it the only pathogen of the genus. Another important, but generally less pathogenic, species is *A. ovis*, primarily infecting sheep (Splitter et al., 1956; Magonigle et al., 1981), but also goats (Mallick et al., 1979; Yousif et al., 1983), and some wild animals (Neitz, 1939; Enigk, 1942; Post and Thomas, 1961). A recent review of infections of *Anaplasma* effectively summarized the history and then current knowledge of wild and domestic hosts of species of *Anaplasma* and the associated epizootiology (Kuttler, 1984).

Some wild ruminants are known to be significant reservoirs of the disease for domestic livestock (Christensen and McNeal, 1967; Howarth et al., 1969). To adequately develop and implement effective anaplasmosis control programs, as much information as possible on the epizootiology of each pathogen must be obtained.

Zaugg and Kuttler (1985), reported that two bison calves, one splenectomized and one spleen-intact, were inoculated with an *A. marginale* stabilate prepared from the blood of a bovine steer experiencing acute anaplasmosis. Both bison developed parasitemias and their blood transmitted disease when inoculated into splenectomized bovines 61 and 71 days after the bison were exposed initially. On the other hand, there is little or no evidence that American bison are susceptible to infections by *A. ovis*.

The objectives of the present investigation were to determine if: (1) a long-term *A. marginale* carrier state persisted in experimentally infected bison, and (2) bison are susceptible to experimental infection by *A. ovis*.

MATERIALS AND METHODS

All bison and bovine steers were maintained in open isolated corrals. Alfalfa hay, fresh water and trace mineral salt were provided ad libitum.

Study 1: Whole blood from two *A. marginale*-infected American bison (Zaugg and Kuttler, 1985), was collected in citrated tubes 314 and 496 days after experimental inoculation. Blood from bison B3 obtained on the collection dates for inoculation had a percentage of parasitized erythrocytes (PPE) of 1.6% and 0.6%, respectively. No parasitemia was observed in blood collected from bison B4 on either date. On each collection date 5 ml of blood from each bison was inoculated (IV) into individual splenectomized Holstein-Friesian steers within 15 min of collection. Blood was sampled from all recipient bovines every 3 to 7 days and examined via packed cell volume (PCV), PPE, rapid card ag-

Received for publication 19 July 1985.

TABLE 1. Hematological and serological data summarized on bovine recipients of *Anaplasma marginale*-infected bison blood.

Bison donor	Days after donor inoculation	Bovine recipient	At time of chemotherapy[a] PPP (days)	Days after inoculation (IV)	PCV (%)	RCA	CF	PPE
B3[b]	314	28	21	38	13	+	640	43.6
B4	314	6	35	42	19	+	320	18.0
B3	496	3	30	33	29	+	160	5.5
B4	496	7	30	33	22	+	320	8.2

[a] Oxytetracycline, 20 mg/kg IM; PPP = prepatent period; PCV = packed cell volume; RCA = rapid card agglutination; CF = complement fixation (titers); PPE = percentage of parasitized erythrocytes; + = positive reaction.
[b] Splenectomized.

glutination (RCA) (Amerault and Roby, 1968), and complement fixation (CF) (Anonymous, 1974) tests until therapeutically treated with oxytetracycline (Liquamycin LA-200, Pfizer Inc., New York, New York 10017, USA). Donor bison were observed daily for signs of clinical abnormality and blood was sampled periodically and examined similarly.

Study 2: One splenectomized yearling bison bull (B1) was inoculated (IV) three times with fresh whole sheep blood, collected in partially evacuated glass tubes containing EDTA (Vacutainer, Becton-Dickinson, Rutherford, New Jersey 07070, USA) as follows: (1) 15 ml from a ewe experiencing acute ovine anaplasmosis (PCV = 15%, PPE = 21.6%); (2) 100 days later, 20 ml of pooled blood from four ewes known to be carriers of *A. ovis*; and (3) 217 days later, 20 ml from a known carrier. Blood was sampled once per wk via jugular venipuncture and examined hematologically and serologically, as described above, for a total of 10 mo.

RESULTS

Study 1: All four bovine steers inoculated with bison blood developed parasitemias (Table 1) and experienced clinical disease. The prepatent period, defined as the time from exposure to time that a PPE value of 1 is observed (Buening, 1973), lasted 21 and 30 days for the recipients of blood from the splenectomized bison (B3) and 35 and 30 days for recipients of blood from the spleen-intact bison (B4). The single IM injection of oxytetracycline, at 20 mg/kg, was therapeutic as determined by the disappearance of parasitemia and a return to normal PCV values.

Anaplasma marginal bodies could always be found in stained blood samples of B3. The PPE varied between <0.1 and 1.6% throughout the 6-mo test period. No marginal bodies were noted in stained blood samples of B4. Both bison appeared normal clinically. However, each remained RCA and CF test seropositive, showing CF titers between 10 and 1,280 with a mean of 180.

Study 2: The bison bull (B1) inoculated with *A. ovis* parasitemic and carrier blood remained clinically, hematologically and serologically normal for over 10 mo. The PCV values were consistently between 30 and 39%.

DISCUSSION

The prepatent periods of 21 to 35 days in the inoculated steers were consistent with those observed normally in bovine studies using blood inoculations (Blood et al., 1979). Steers 6 and 28 (Table 1) exhibited severe signs of disease and were chemotherapeutically treated to prevent death. Steers 3 and 7 were similarly treated 3 days after a detected PPE so that the animals might be used in another, unrelated study. Judging from the rapid initial rise in PPE values (Table 1) it was felt that had the infections been allowed to progress the steers would have suffered severe anaplasmosis.

Bison B3, but not bison B4, developed acute phase clinical signs of anaplasmosis

58–68 days after exposure (Zaugg and Kuttler, 1985). However, both splenectomized and spleen-intact bison were asymptomatic carriers of *A. marginale* for at least 496 days, as confirmed by passage to susceptible bovine steers. Other investigators have noted a lack of clinical disease in *Anaplasma*-infected North American wild ruminants despite constant parasitemias in splenectomized individuals (Renshaw et al., 1979). However, unlike black-tailed deer (*Odocoileus hemionus columbianus*) which lost their antibody titers within 16 wk after the peak of parasitemia (Christensen et al., 1958; Osebold et al., 1959), bison maintained CF titers at least 15 mo later, the same was observed with cattle infected with *A. marginale* (Todorovic et al., 1977). This immunological similarity may be due to the closer taxonomic relationship bison have with cattle than do the cervids. It also implies that bison may serve effectively as carriers of *Anaplasma* for much longer than proven here, thereby complicating anaplasmosis control programs in areas where bison and cattle intermingle.

The close taxonomic positions of cattle and bison may also be a reason why bison B1 did not become detectably infected with *A. ovis*. Although experimental infections of *A. ovis* have been produced in splenectomized bovine calves (Kuttler, 1981), apparently it is difficult to accomplish as evidenced by the inability of others to do so (Splitter et al., 1956; Ryff et al., 1958; Kreier and Ristic, 1963; Magonigle et al., 1981). The PCV values (between 30 and 39%) observed in bison B1 were substantially lower than the "normal" value of 45.8% reported from 132 wild bison (Peterson and Roby, 1975). The difference was felt to be due to splenectomy because prior to surgery the average PCV value was 43.5%. A similar drop in PCV was also noted in blood samples from bison B3 after surgery and before exposure to *Anaplasma*.

Unfortunately, bison B1 died of unrelated causes before a scheduled inoculation of its blood into susceptible sheep to test for sub-patent infection. While such an inoculation would have enhanced the present study the suggestion that bison are resistant to infection by *A. ovis* may be considered logical from the total lack of hematological and serological responses observed.

LITERATURE CITED

AMERAULT, T. E., AND T. O. ROBY. 1968. A rapid card agglutination test for bovine anaplasmosis. J. Am. Vet. Med. Assoc. 153: 1828–1834.

ANONYMOUS. 1974. A Microtiter Technique for the Complement-Fixation Test for Anaplasmosis. USDA, APHIS, Beltsville, Maryland, 6 pp.

BLOOD, D. C., J. A. HENDERSON, AND O. M. RADOSTITS. 1979. Veterinary Medicine, 5th Ed. Lea and Febiger, Philadelphia, Pennsylvania, pp. 67–70.

BUENING, M. G. 1973. Cell-mediated immune responses in calves with anaplasmosis. Am. J. Vet. Res. 34: 757–763.

CHRISTENSEN, J. F., AND D. W. MCNEAL. 1967. *Anaplasma marginale* infection in deer in the Sierra Nevada foothill area of California. Am. J. Vet. Res. 28: 599–601.

———, J. W. OSEBOLD, AND M. N. ROSEN. 1958. The incidence of latent *Anaplasma marginale* infection in wild deer in an area where anaplasmosis is enzootic in cattle. Proc. Annu. Meet. U.S. Livestock San. Assoc. 62: 59–65.

ENIGK, K. 1942. Susceptibility of the eland antelope for *Anaplasma ovis* and *Eperythrozoon ovis*. Dtsch. Tropenmen. Z. 46: 48–52.

HOWARTH, J. A., T. O. ROBY, T. E. AMERAULT, AND D. W. MCNEAL. 1969. Prevalence of *Anaplasma marginale* infections in California deer as measured by calf inoculation and serologic technique. Proc. Annu. Meet. U.S. Anim. Health Assoc. 73: 136–147.

KREIER, J. P., AND M. RISTIC. 1963. Anaplasmosis. VII. Experimental *Anaplasma ovis* infection in white-tailed deer (*Dama virginiana*). Am. J. Vet. Res. 21: 565–572.

KUTTLER, K. L. 1981. Infection of splenectomized calves with *Anaplasma ovis*. Am. J. Vet. Res. 42: 2094–2096.

———. 1984. *Anaplasma* infections in wild and domestic ruminants: A review. J. Wildl. Dis. 20: 12–20.

MAGONIGLE, R. A., W. P. ECKBLAD, S. D. LINCOLN, AND F. W. FRANK. 1981. *Anaplasma ovis* in Idaho sheep. Am. J. Vet. Res. 42: 199–201.

MALLICK, K. P., S. K. DWIVEDI, AND M. N. MALHO-

TRA. 1979. Anaplasmosis in goats: Report on clinical cases. Indian Vet. J. 56: 693–694.

NEITZ, W. O. 1939. Ovine anaplasmosis: The transmission of *Anaplasma ovis* and *Eperythrozoon ovis* to the blesbuck (*Damaliscus albifrons*). Onderstepoort J. Vet. Sci. Anim. Ind. 13: 9–16.

OSEBOLD, J. W., J. F. CHRISTENSEN, W. M. LONGHURST, AND M. N. ROSEN. 1959. Latent *Anaplasma marginale* infection in wild deer demonstrated by calf inoculation. Cornell Vet. 49: 97–115.

PETERSON, K. J., AND T. O. ROBY. 1975. Absence of *Anaplasma marginale* infection in American bison raised in an anaplasmosis endemic area. J. Wildl. Dis. 11: 395–397.

POST, G., AND G. M. THOMAS. 1961. A study of anaplasmosis in elk. J. Am. Vet. Med. Assoc. 139: 357–358.

RENSHAW, H. W., R. A. MAGONIGLE, AND H. W. VAUGHN. 1979. Evaluation of the anaplasmosis rapid card agglutination test for detecting experimentally-infected elk. J. Wildl. Dis. 15: 379–386.

RYFF, J. F., C. S. GILBERT, J. L. WEIBEL, AND H. BREEN. 1958. Anaplasmosis and concurrent copper intoxication of sheep. J. Am. Vet. Med. Assoc. 133: 312–315.

SPLITTER, E. J., H. D. ANTHONY, AND M. J. TWIEHAUS. 1956. *Anaplasma ovis* in the United States. Am. J. Vet. Res. 17: 487–491.

TODOROVIC, R. A., R. F. LONG, AND B. R. MCCALLON. 1977. Comparison of rapid card agglutination test with the complement fixation test for diagnosis of *Anaplasma marginale* infection in cattle in Colombia. Vet. Microbiol. 2: 167–177.

YOUSIF, Y. A., R. A. DIMITRI, S. K. DWIVEDI, AND N. J. AHMED. 1983. Anemia due to anaplasmosis in Iraqi goats: I. Clinical and hematological features under field conditions. Indian Vet. J. 60: 576–578.

ZAUGG, J. L., AND K. L. KUTTLER. 1985. *Anaplasma marginale* infections in American bison: Experimental infection and serologic study. Am. J. Vet. Res. 46: 438–441.