

ANTIBODIES TO *BRUCELLA* SPP. AMONG BLUE WILDEBEEST AND AFRICAN BUFFALO IN KENYA

S. Waghela and L. Karstad¹

Veterinary Research Laboratory, Kabete, Kenya

ABSTRACT: A serologic survey of blue wildebeest (*Connochaetes taurinus* Burchell) and African buffalo (*Syncerus caffer* Sparrman) in the Masai Mara area was conducted. Antibodies to *Brucella* spp. were found in 18% of the blue wildebeest and 30% of the African buffalo examined. There were titers in all age groups and in both sexes. Hygromata were seen in both species. The increase in numbers of blue wildebeest and African buffalo which share grazing and watering areas with cattle of the Masai people, makes the presence of infections by *Brucella* spp. in wildlife an important consideration in any program for control of brucellosis.

INTRODUCTION

Evidence of brucellosis in free-living wild animals in Africa has been presented by several authors. Most pertinent to the present investigation are reports of brucellosis in African buffalo in southern Africa (Condy and Vickers, 1976; Gradwell et al., 1977; Herr and Marshall, 1981) and in several species of wild herbivores and carnivores in eastern Africa, including African buffalo and blue wildebeest (Rollinson, 1962; Sachs et al., 1968; F.A.O., 1978). Some of the investigations were limited to serology; however, isolations of *Brucella* spp. have been reported also. *Brucella suis* was isolated from rodents (Heisch et al., 1963), *B. melitensis* from an impala (*Aepyceros melampus*) (Schiemann and Staak, 1971) and *B. abortus* from waterbuck (*Kobus ellipsiprymnus*) (Condy and Vickers, 1969) and from African buffalo (Staak et al., 1968; Kaliner and Staak, 1973; Gradwell et al., 1977). The isolations from buffalo were typed as biotypes 1 and 3. These biotypes are found commonly in cattle in eastern Africa (Hummel and Staak, 1974; Waghela, 1978).

In the present study, the prevalence of antibodies to *Brucella* spp. was examined in African buffalo and blue wildebeest

during a surveillance study for rinderpest in the Masai Mara region of southern Kenya in July, August and September 1982. Masai Mara represents the Kenyan portion of the Serengeti ecosystem. In the Mara, the resident population of approximately 250,000 blue wildebeest comes into contact with the migratory population of nearly 1.5 million. The latter enter Mara from Serengeti in July or August and leave on a southward migration in September. The grazing and watering areas of this region are shared by both wild animals and domestic cattle herds of the Masai and transmission of infectious diseases, such as brucellosis, can occur. The importance of brucellosis in wild animals in relation to the control of the disease in cattle should be considered.

MATERIALS AND METHODS

Animals were captured by drug immobilization using a combination of 2.45 mg etorphine hydrochloride and 10 mg acepromazine maleate/ml (Reckitt and Coleman, Hull, United Kingdom). A reversal of the effect of etorphine hydrochloride and remobilization was accomplished with diprenorphine at a dosage of 3 mg/ml. Delivery was by rifled projectors and projectile syringes (Palmer Chemical and Equipment Company, Atlanta, Georgia 30300, USA). Adult blue wildebeest were immobilized with 1 ml of etorphine-acepromazine solution and adult African buffalo with 2.5 ml. Smaller doses in proportion to estimates of weight were used on immature animals. The ages of blue wildebeest were estimated by measurements of

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¹ Present address: Animal Pathology Division, Food Production and Inspection Branch, 2255 Carling Avenue, Ottawa, Ontario K1A 0Y9, Canada.

TABLE 1. Results of tests for antibodies to *Brucella* spp. in blue wildebeest and African buffalo in Kenya with the complement fixation test (CFT) and the tube serum agglutination test (SAT).

Species	No. of samples tested			CFT* (reciprocal of serum dilution)										SAT* (international units)		
	M	F	Total	2.5	5	10	20	40	80	160	320	640	25	50	100	
African buffalo	14	3	17	0 ^b	3	0	1	1	0	0	0	0	3	1	1	
Blue wildebeest	108	67	175	19	12	10	3	0	2	0	0	2	42	9	5	

* CFT titers of 1/5 or over and SAT titers of 50 IU and higher were considered as positive.

^b No. of animals reacting in each category.

height and width of the exposed crowns of central incisors, as described by Attwell (1980). The ages of African buffalo were estimated by horn growth and conformation (Sinclair, 1977).

The sera were tested for antibodies to *Brucella* spp. using the tube serum agglutination test (SAT), the complement fixation test (CFT) and the rose bengal plate test (RBPT) as described by Philpott and Auko (1972). The interpretations of the tests were those used for bovine brucellosis, i.e., for the SAT, ≥ 50 international units (IU) as "positive"; for the CFT, 50% hemolysis at a dilution of 1/2.5 as "doubtful" and at dilutions of 1/5 or higher as "positive." The RBPT was considered positive when there was slight to complete agglutination.

The RBPT gave a high number of false positives with the wildebeest sera and consequently only 23 blue wildebeest and 17 African buffalo were tested using this method.

TABLE 2. Sex and age groups of blue wildebeest tested for antibodies to *Brucella* spp.

Test results ^a	No. animals in each age class (yr)					Ud ^b	Total
	<3	3-5	6-10	11-15	>15		
Male							
Negative	6	10	29	8	3	33	89
Positive	3	2	9	1	1	3	19
	9	12	38	9	4	36	108
Female							
Negative	2	15	17	9	2	8	53
Positive	2	1	3	1	0	7	14
	4	16	20	10	2	15	67
Total	13	28	58	19	6	51	175

* CFT titers of 1/5 or over and SAT titers of 50 IU and higher were considered as positive.

^b Age was not determined.

RESULTS AND DISCUSSION

Thirty-one of 175 blue wildebeest and five of 17 African buffaloes were considered positive based on reactions in either the SAT or CFT or both (Table 1). Antibodies were detected in all ages and both sexes, although the numbers of positive immature animals were small (Table 2).

There was not complete agreement among the three tests. Of 152 blue wildebeest, 36 were either doubtful or positive in the CFT, of which only four were also positive in the SAT; three were doubtful in the SAT and the rest were negative in the SAT. The latter group, i.e., CFT positive, but SAT negative included one wildebeest with a swollen hock and a CFT titer of 1/5, and another animal with a carpal hygroma and a CFT titer of 1/10.

Seven of the 17 African buffaloes were positive in the CFT, five of which were also positive in the RBPT. One of these five was positive and one was doubtful in the SAT. The remaining 10 sera were negative in all the tests.

In general, titers were higher in the blue wildebeest sera than in the African buffalo. Blue wildebeest sera reacted at dilutions of up to 1/640 in the CFT and 100 IU in SAT, while the highest titers in CFT and SAT in the African buffalo were 1/40 and 25 IU, respectively. This is not in complete agreement with the observations by Condy and Vickers (1972) who reported titers of 20 IU in SAT for two of 38 blue wildebeest and 20-1,280 IU in SAT

for 15 of 102 African buffalo tested in a survey. However, the antibody response of individual animals vary according to the severity of infection, length of infection and the serologic test used.

Hygromata were observed in both blue wildebeest and African buffalo, but specimens for culture were not obtained. There seemed to be no serious effect on reproduction judging by the number of calves present of both the species. Populations of both African buffalo and blue wildebeest are expanding possibly as a result of a ban on hunting of wildlife in Kenya.

Data from previous studies in Kenya by us and co-workers (F.A.O., 1978) contain information on positive sera among several wildlife species. These included nine of 69 African buffalo, eight of 55 eland (*Taurotragus oryx* Pallas) of which the positive animals were from a semi-domesticated herd in contact with cattle and 17 of 85 zebra (*Equus burchelli* Gray). Positive sera were also found in only two of 249 blue wildebeest of which most were from a separate population in Kajiado District, eastern Masailand. We concluded that wildlife in Kenya was not a significant reservoir of infections by *Brucella* spp. This assumption may have been incorrect. In the present study, the prevalence of positive sera in blue wildebeest was 18% and in African buffalo was 30%. Cattle sera from Narok District, which includes Masai Mara, had a prevalence of 4%, while only 1.3% positives were found in cattle in the neighbouring Kajiado District, where the prevalence in blue wildebeest was lower, approximately 1% (F.A.O., 1978; Waghela, 1978).

Working in northern Tanzania, in areas adjacent to Masai Mara, Sachs and Staak (1966) and Sachs et al. (1968) found 10.5% of the blue wildebeest to have significant serological reactions to *Brucella* antigens. Four of 23 African buffalo also reacted but at serum dilutions of 1/10 and 1/20. They recorded four cases of orchitis in blue

wildebeest. Cattle owned by the Masai in northern Tanzania had a prevalence of 5% (Staak et al., 1967; cited by Sachs et al., 1968). Sachs et al. (1968) concluded that infection of *Brucella* spp. in the Serengeti blue wildebeest were significant. Furthermore, they found a higher prevalence in the population of migratory blue wildebeest of the Serengeti (12%) than in three resident herds of blue wildebeest located in the Tanzanian Mara (5%), Loliondo (6%) and Ngorongoro (3%) areas.

Our survey was conducted during the period July through September 1982, when the Mara resident populations and Serengeti populations of migratory blue wildebeest were co-mingling in Masai Mara. The fact that 15 yr after the studies by Sachs et al. (1968) we have found a higher prevalence is a cause for concern. Furthermore, the migratory blue wildebeest has meanwhile increased approximately three-fold. It has been suggested that factors contributing to the spread of brucellosis in drier areas like Masailand of Kenya, are the movement of livestock for grazing and concentrating them around water-holes. Obviously, any plan to control brucellosis in domestic animals must take into account the sharing of grazing and watering areas with the blue wildebeest, African buffalo and other wild animals such as the antelopes, gazelles and zebras. Sachs et al. (1968) found serologic reactors among zebras and various carnivores such as jackals (*Canis mesomelas* Schreber) and hunting dogs (*Lycaon pictus* Temminck) which prey and scavenge on blue wildebeest, African buffalo, and zebra.

Herr and Marshall (1981) concurred with Condy and Vickers (1976) that *Brucella* is ineffective as a culling agent in African buffalo, but infected herds should be considered as a likely source of reinfection for domestic stock where they intermingle. Brucellosis is most likely to be the next disease for obligatory control in

Kenya and the increasing populations of blue wildebeest and African buffalo in areas where there is increasing use of semi-arid land for agriculture means that wildlife must be considered significant in any control measures developed for livestock.

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