# RESEARCH COMMUNICATION

## WILDEBEEST-DERIVED MALIGNANT CATARRHAL FEVER: UNUSUAL EPIDEMIO-LOGY IN SOUTH AFRICA

### B. J. H. BARNARD1 and H. E. VAN DE PYPEKAMP2

#### **ABSTRACT**

BARNARD, B. J. H. & VAN DE PYPEKAMP, H. E., 1988. Wildebeest-derived malignant catarrhal fever: unusual epidemiology in South Africa. Onderstepoort Journal of Veterinary Research, 55, 69-71 (1988).

The epidemiology of wildebeest-derived malignant catarrhal fever in South Africa differs from the world-wide accepted pattern. Here the occurrence of the disease is often not related to close contact between cattle and wildebeest, and most cases are observed during late winter and spring, when wildebeest calves are 8–10 months old. This is in contrast to the situation in Kenya and Tanzania, where most cases are encountered during autumn, when wildebeest calves are 3–4 months old.

### INTRODUCTION

In South Africa, the blue wildebeest (Connochaetes taurinus) plays a major role in the occurrence of malignant catarrhal feven (WD MCF) (Barnard, 1984). The infrequent appearance of the disease created the impression that it was economically a relatively unimportant disease and consequently very little attention was paid to it in South Africa in the past 30 years. The incorporation of game into the farming economy and a greater awareness of game conservation in the past decade has resulted in an increase in the prevalence of the disease. Losses due to the disease have reached the alarming proportions of up to 20 % on individual farms (unpublished observations, Van de Pypekamp, 1986).

Confirmation of a clinical diagnosis of WD MCF is not always possible but the symptoms are sufficiently clear for making a fairly reliable diagnosis.

The validity of such a diagnosis is further strengthened by a typical epidemiology in which cases can be related to close contact between cattle and wildebeest during the wildebeest calving season (Plowright, 1964). In South Africa, however, this is not always the case (Barnard, 1984).

In this report, the occurrence of WD MCF on 6 properties adjacent to or in the proximity of a nature reserve is described. In none of the cases could direct contact have taken place. The disease also occurred when wildebeest calves were 8–10 months old.

#### MATERIALS AND METHODS

## Silkaatsnek Nature Reserve

The Reserve, surrounded by a game-proof fence, is situated on uneven ground and includes part of a mountain range (Fig. 1). In the vicinity are several small farms. Outbreaks occurred on farms on the northern side of the reserve. Animals in the reserve at the outbreak of MCF in August-November 1984 included impala, kudu, eland, zebra, giraffe, blesbok, waterbok and 17 wildebeest, including 9 cows heavy in calf, 2 heifers, 3 bulls, 2 calves 8–10 months of age and a small calf which was born at the end of September 1984. The wildebeest were introduced into the reserve in 1969. Although an extremely dry season had been experienced, the grazing was in fair condition. In June and July preceding the outbreak, 25 mm of rain, spread over several days, was measured in the area. The unusual rain at that time resulted in a much higher humidity than normal, with the

result that during the nights and early mornings fog was blown from the game reserve towards the properties on which MCF was encountered. These conditions lasted for a few days during the first half of July.

### Malignant caterrhal fever cases

Twenty-six cases of MCF were encountered amongst 221 cattle in affected herds on 6 properties adjacent to or in the proximity of the reserve (Fig. 1).

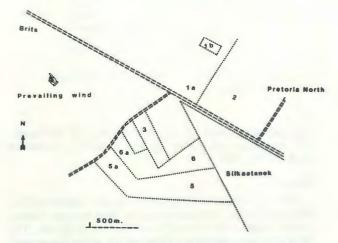


FIG. 1 Silkaatsnek Nature Reserve and 6 properties adjacent to or in the proximity of the reserve

The properties on which WD MCF occurred are numbered 1–6 according to the sequence of the first cases of MCF encountered on each property.

Properties 1 and 2 are separated from the Reserve by a main road 60 m wide. Six cases occurred amongst 35 stud Brahman cattle which were allowed to graze at night in Camp 1a, the property nearest to the reserve (Table 1). During the day the stud cattle were kept with commercial cattle in a feedlot 1b, 600 m further away. The commercial cattle were not affected. Cattle on property 2 were kept in camps next to the main road. Four contracted MCF. Five of 70 Friesian cows kept in smal paddocks on property 3, 600 m from the reserve, also contracted MCF. On property 5, 2 cattle, and on property 6, 8 cattle succumbed to the disease. Cattle on these properties were allowed to graze camps adjacent to the reserve. However, on each of these properties at least 1 calf kept in a paddock separated by 800 m from the reserve, contracted MCF. The calf on property 6 was only 1 month old when it died on the 31st of September.

The stud cattle on property 1 were first put into the camp next to the main road in June 1984, while those on property 2 grazed the camps from July onwards. The calf

<sup>&</sup>lt;sup>1</sup> Veterinary Research Institute, Onderstepoort 0110

<sup>&</sup>lt;sup>2</sup> Private Bag X82087, Rustenburg 0300

Received 2 October 1987—Editor

TABLE 1 Cases of WD MCF in the 1984 Silkaatsnek Nature Reserve outbreak

Property No.		Nature of contact		Disease in cattle			
	First exposed	Separation in metres	First case	Last case	No.1	Breed	
1a	84.06.15	60	84.08.10	84.10.15	6/35	Brahman	
1b	Prior to <sup>2</sup>	600	_	_	None	Brahman	
2	84.06.15	60	84.08.15	84.10.10	4/80	Brahman	
3	Prior to	500	84.09.20	84.10.15	5/70	Friesian	
4	Prior to	4 000	84.09.28	_	1/10	Dexter	
5a	Prior to	Fence	84.10.05		1/13	Jersey cross	
5b	Prior to	900	84.10.12	_	1/1	Jersey calf	
6a	Prior to	Fence	84.09.30	84.11.08	7/11	Simmental	
6b	84.08.30	900	84.09.30	_	1/1	Simmental calf	

No. dead/No. on property

<sup>2</sup> First exposed at least 3 months prior to 84.06.01

TABLE 2 Confirmed cases of wildebeest-derived malignant catarrhal fever

Property No.	Cattle		Method				
	Case No.	Age in months	Cell- culture	Rabbit	Cattle	Histopathology	
1	1	18	71	24	24	N <sup>2</sup>	
2	2 3	18	6	17	21	N ·	
2	3	7	7	24	24	N	
2 2 2 3	4	36	6	N	N	Positive	
3	5	12	N	N	N	Positive	
4	6 7	30	N	N	N	Positive	
5	7	4	6	N	N	Positive	
6	8	1	N	N	N	Positive	

<sup>1</sup> No. of days from inoculation to cytopathic changes or death

<sup>2</sup> Specimens not available

that died on property 6 was born at the end of August. All the other cattle were on the respective properties prior to July 1984.

## Diagnosis

Affected cattle were submitted to the Veterinary Research Institute, Onderstepoort, for post-mortem examination and/or collection of blood for virus isolation. The diagnosis was confirmed for at least 1 animal from each property (Table 2).

### Virus isolation

For virus isolation, the leucocyte fraction of 5–10 m $\ell$  of blood was cocultured with foetal lamb kidney cells in 25 m $\ell$  plastic flasks¹ within 2 h of collection. The cultures were incubated at 35–37 °C. Isolated virus was identified in a micro-neutralization test using known positive serum prepared by inoculation of rabbits with the WC11 isolate of WD MCF virus (Plowright 1964).

In some cases, cattle and rabbits were injected with  $50 \text{ m}\ell$  and  $10 \text{ m}\ell$  of blood respectively. Cattle were injected intravenously and rabbits intraperitoneally. Both cattle and rabbits were observed daily until death. The results are shown in Table 2.

### **RESULTS AND DISCUSSION**

The Masai in East Africa were among the first people to associate wildebeest with MCF. They believed that WD MCF was contracted when cattle grazed pastures contaminated by hair and afterbirth of wildebeest (Daubney & Hudson, 1936). Attempts to isolate virus from wildebeest placenta or hair have been unsuccessful

(Mushi, cited by Mushi, Rurangirwa & Karstad, 1980). Wildebeest calves acquire the infection in utero (Plowright, Ferris & Scott, 1960; Castro, Ramsay, Dotson, Schramke, Kocan & Whitenack, 1984; Heuschele, Neilson, Oosterhuis & Castro, 1985) and by horizontal spread in the annual calf crop in spite of maternal neutra-lizing antibody. They develop a leucocyte-associated viraemia which is most frequent in the 2nd month of life (Plowright 1984). Virus is excreted in a cell-free state in nasal and ocular secretions at this stage (Mushi, Karstad & Jessett, 1980). The presence of virus in these fluids supports the suggestion that the disease is disseminated by means of these secretions (Rweyemamu, Karstad, Mushi, Otema, Jessett, Rowe, Drevemo & Grootenhuis, 1974). WD MCF virus is only rarely isolated from wildebeest calves more than 3 months old. The fact that virus neutralizing antibody appears in the secretions, accounts for the rapid virus decline after 3 months of age (Rurangirwa, Mushi & Karstad, 1982; Mushi, Jessett, Rurangirwa, Rossiter & Karstad, 1980). Viraemia also declines from the age of 2 months to reach 2 % by the end of the 1st year and is subsequently infrequent (Plowright, 1984). Cattle housed with viraemic wildebeest calves contracted MCF within 5-7 weeks (Plowright, 1964), whereas cattle housed with adult wildebeest with a discontinuous low-level viraemia were not affected even after prolonged exposure (Plowright, 1984).

The annual prevalence of WD MCF in Kenya is at its maximum in the months of April to July, following the wildebeest calving season in February to April (Plowright, 1964). The seasonal occurrence is comparable in Tanzania, where wildebeest start to calf during January. In South Africa where the calving season starts in December, the first cases would be expected in January with a peak prevalence in February–March, but this is not the case. Most cases are concentrated into the months September to November (Barnard 1984).

In the outbreak described in this paper the first case occurred in the middle of August and mortalities continued to take place until the middle of November. This indicates an unusual source of virus, as calves older than 3 months seldom excrete the virus. It is also difficult to explain why the first cases of MCF appeared in 1984, when wildebeest had already been introduced 15 years before. Stress, caused by the unusually cold rainy weather during the winter months, together with poor grazing, might have resulted in increased virus excretion, but adverse climatic conditions have never before been incriminated as being responsible for increased excretion of WD MCF virus by wildebeest.

Close contact is generally regarded as necessary for the transmission of wildebeest virus, but previous observations (Maré, 1977; Barnard, 1984) and events in the

<sup>&</sup>lt;sup>1</sup> Nunc Laboratory Products

recent outbreak proved that direct contact is not essential for transmission. As aerosols are a successful means of transmission (Plowright, 1984), air currents, causing oral and ocular infection, seem likely modes of transfer. Conditions in the outbreak described favoured this idea. High humidity and favourable prevailing winds were encountered during the period of possible infection. This period was clearly identified by the fact that cattle on properties 1 and 2 were first exposed during June and July, 4-8 weeks before the first case were encountered, and the calf on property 6 was born only at the end of August. However, WD MCF virus is very labile and infectivity is associated predominantly with viable cells (Plowright, 1968). Consequently, transmission in an aerosol over distances of at least 800 m is not very likely, especially during the dry winter conditions usually experienced in South Africa.

Another possible mode of transmission is by flies. Several *Musca* spp. are abundant throughout most of the year (Nevill, 1985) and may act as vectors when a suitable reservoir of virus is available. The simultaneous occurrence of WD MCF and *Parafilaria bovicola* ovipositioned blood spots on cattle (Nevill, 1985) may also play a role. Possible vector flies may visit these spots and in this way spread the infection.

### REFERENCES

- BARNARD, B. J. H., 1984. Transmitters and potential transmitters of malignant catarrhal fever. Proceedings of the XIIIth World Congress on Diseases of Cattle. 1, 56-60.
- CASTRO, A. E., RAMSAY, E. C., DOTSON, J. F., SCHRAMKE, M. L., KOCAN, A. A. & WHITENACK, D. L., 1984. Characteristics of the herpes virus of malignant catarrhal fever isolated from captive wildebeest calves. *American Journal of Veterinary Research*, 45, 409-415.
- DAUBNEY, R. & HUDSON, J. R., 1936. Transmission experiments with bovine malignant catarrh. Journal of Comparative Pathology and Therapeutics, 49, 63-89.

- HEUSCHELE, W. P., NEILSON, N. O., OOSTERHUIS, J. E. & CASTRO, A. E., 1985. Dexamethasone-induced recrudescence of malignant catarrhal fever and associated lymphosarcoma and granulomatous disease in Formosan sika deer (Cervus nippon taiouanus). American Journal of Veterinary Research, 46, 1578–1583.
- MARÉ, C. J., 1977. Malignant catarrhal fever, an emerging disease of cattle in the RSA. Proceedings of the 1st Annual meeting of the United State Animal Health Association, 81, 151-157.
- MUSHI, E. Z., JESSETT, D. M., RURANGIRWA, F. R., ROSSITER, P. B. & KARSTAD, L., 1980. Neutralising antibodies to malignant catarrhal fever herpes virus in wildebeest nasal secretions. *Tropical Animal Health and Production*, 13, 55-56.
- MUSHI, E. Z., KARSTAD, L. & JESSETT, D. M., 1980. The isolation of bovine malignant catarrhal fever virus from ocular and nasal secretions of wildebeest calves. *Research in Veterinary Science*, 29, 168-171.
- MUSHI, E. Z. RURANGIRWA, F. R. & KARSTAD, L., 1980. Epidemiology and control of bovine malignant catarrhal fever. Proceedings of the Wild-life Disease Research and Economic Development. Kabete 8 and 9 September 1980. Ed. KARSTAD, L., NESTEL, B. & GRAHAM, M. Ottowa, Canada. pp. 21–23.
- NEVILL, E. M., 1985. The epidemiology of Parafilaria bovicola in the Transvaal Bushveld of South Africa. Onderstepoort Journal of Veterinary Research, 52, 261-267.
- PLOWRIGHT, W., FERRIS, R. D. & SCOTT, G. R., 1960. Blue wildebeest and the aetiological agent of bovine malignant catarrhal fever. *Nature*, *London*, 188, 1167–1169.
- PLOWRIGHT, W., 1964. Studies on bovine malignant catarrhal fever of cattle. Thesis University of Pretoria, South Africa.
- PLOWRIGHT, W., 1968. Malignant catarrhal fever. Journal of the American Veterinary Medical Association, 152, 795-804.
- PLOWRIGHT, W., 1984. Malignant catarrhal fever virus: a lymphotropic herpes virus of ruminants pp. 279–305. Latent herpes virus infections in veterinary medicine. *In:* WITTMAN, G., GASHELL, R. M. & RZIHA, H. J., (eds.). Martinus Nijhoff for the Commission of the European Community.
- RURANGIRWA, F. R., MUSHI, E. Z. & KARSTAD, L., 1982. Malignant catarrhal fever virus specific secretory IgA in nasal secretions of wildebeest calves. Comparative Immunology Microbiology and Infectious Diseases, 5, 429-436.
- RWEYEMAMU, M. M., KARSTAD, L., MUSHI, E. Z., OTEMA, J. C., JESSETT, D. M., ROWE, L., DREVEMO, S. & GROOTENHUIS, J. G. 1974. Malignant catarrhal fever virus in nasal secretions of wildebeest: A probable mechanism for virus transmission. *Journal of Wildlife diseases*, 17, 433–436.