Onderstepoort J. vet. Res., 56, 135–139 (1989)

EPIZOOTOLOGY OF WILDEBEEST-DERIVED MALIGNANT CATARRHAL FEVER IN AN OUTBREAK IN THE NORTH-WESTERN TRANSVAAL: INDICATIONS OF AN INTERMEDIATE HOST

B. J. H. BARNARD[1], H. E. VAN DE PYPEKAMP[2] and MONICA D. GRIESSEL[1]

ABSTRACT

BARNARD, B. J. H., VAN DE PYPEKAMP, H. E. & GRIESSEL, MONICA D., 1989. Epizootology of wildebeest-derived malignant catarrhal fever in an outbreak in the north-western Transvaal: indications of an intermediate host. *Onderstepoort Journal of Veterinary Research*, 56 135–139 (1989).

The investigation involved 37 herds of cattle numbering 6 280 animals and 5 groups of blue wildebeest (*Connochaetes taurinus*), consisting of 30–330 wildebeest per group. All the cases of wildebeest-derived malignant catarrhal fever encountered were associated with wildebeest and not with other game animals. Six per cent of the cases were encountered in late summer when the wildebeest calves were 3–4 months old, whereas 73 % occurred in spring, when the wildebeest calves were 8–11 months old and did not excrete virus. The incidence of the disease among cattle born and reared in the vicinity of wildebeest was less than 0,5 %. Among intermittently and directly exposed cattle the incidence was 5,2 %, but the highest incidence was encountered in cattle kept in camps separated from wildebeest by a distance of approximately 100 m.

Alcelaphine herpesvirus-1 (AHV-1), the causal agent of malignant catarrhal fever (MCF) was isolated from the tears, blood and nasal mucus of 8 out of 14 wildebeest calves during their 4th–6th month (April–June), but not subsequently. No sampling was possible before the age of 3 months.

The occurrence of the disease from September–November, when wildebeest calves could not be incriminated because they no longer excreted virus, suggests the involvement of another host or an intermediate host capable of acquiring the infection from young wildebest calves, harbouring the infection until August–September, and then transferring it to cattle.

INTRODUCTION

In South Africa the occurrence of wildebeest-derived malignant catarrhal fever (WD MCF) is often not related to close contact between cattle and blue wildebeest, and most cases are encountered in the spring when wildebeest calves are 8–11 months old (Barnard 1984; Barnard & Van de Pypekamp, 1988). From the year 1984, but particularly during 1986, several farmers in the north-western Transvaal, in an area near Sentrum approximately 40 km north of Thabazimbi, experienced severe losses as a result of WD MCF. The high incidence of the disease, the large numbers of cattle in the area and the involvement of several farms were ideal factors for an epizootological study.

This paper reports on virus excretion by wildebeest calves, the incidence of MCF in cattle of different ages and status of breeding, the seasonal incidence, time of transmission and transfer distance on the occurrence of the disease. It also rules out the possible role played by other species of game in the transmission of WD MCF. The possibility of an intermediate host is also briefly discussed.

History

According to local inhabitants, blue wildebeest were numerous during the first half of the century and WD MCF occurred only sporadically. In later years the wildebeest population declined drastically, but then from about 1970 increased when game farming became popular in the area.

One property, utilized for game farming only, was established in 1972. Initially, both cattle and game were kept on this farm, but since 1983 it was used exclusively for game farming, except for a short period of 2 months during 1984 when cattle were grazed there. Other farms in the area are used for both cattle and game. Game animals, including wildebeest, have occasionally been introduced from other areas or exchanged among the local farms. In South Africa MCF is a notifiable disease and the movement of all wildebeest is restricted to those farms where they already exist and is regulated by a permit system.

Most of the farms included in this investigation are well-managed and, despite a long-standing drought, the grazing was in a reasonable to good condition. However, some farms in the area were uninhabited and could not be farmed owing to the drought.

During 1984–1986 there was an increase in the incidence of WD MCF in the area, but during 1987 only a few cases were recorded.

Locality and climate

The area under investigation (Fig. 1) is located approximately 40 km north of Thabazimbi at an altitude of 1 000 m. The vegetation is mixed bushveld but varies from farm to farm. The locality has very hot summers and cool winters (South African Weather Bureau). The average daily temperatures vary from about 14,0–28,0 °C, the mean monthly minimum being 9,0 °C, while the mean monthly maximum soars to about 32,7 °C. The winter of 1987, was exceptionally cold however, with an average minimum temperature of 1 °C which occurs for June and July. The mean annual rainfall is 650 mm and is encountered mainly during the summer months.

MATERIAL AND METHODS

Diagnosis

Our diagnosis of WD MCF in cattle was based on clinical examination or on clinical signs provided by reliable farmers. Confirmation was done by virus isolation or histopathology on specimens from one or more cattle from several herds.

Data collection

Farmers were requested to complete a questionnaire. The data obtained were checked with the farmers and, where applicable, cross-checked with neighbouring farmers, as well as with the records of the State Veterinarian for that area (H. J. van de

[1] Veterinary Research Institute, Onderstepoort 0110
[2] Private Bag X138, Pretoria 0001
Received 17 January 1989—Editor

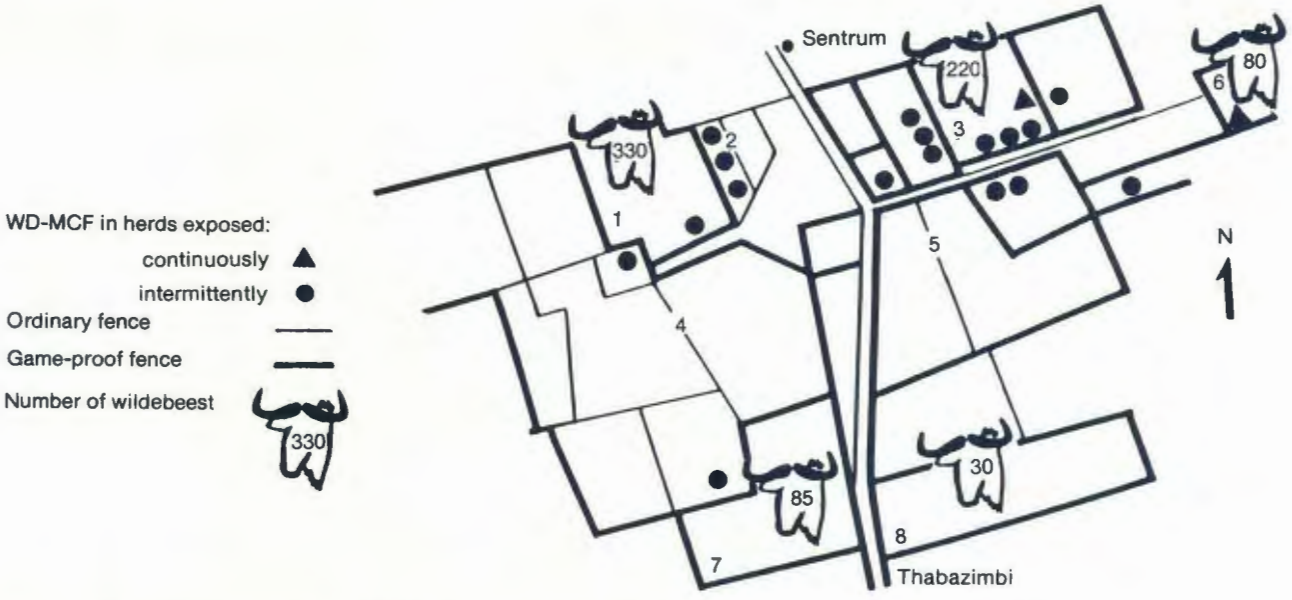

FIG. 1 The occurrence of WD MCF in relation to the distribution of wildebeest in an outbreak in the north-western Transvaal

TABLE 1 The occurrence of WD MCF in cattle exposed to several different species of game on different farms in the north-western Transvaal

Cattle in camp number	Estimated number of game species involved							Cattle	
	Impala	Kudu	Harte-beest	Bles-bok	Gems-bok	Water-buck	Wilde-beest	Number exposed	Cases of WD MCF
2	60	20	0	0	0	0	0	200	0
4	600	80	50	0	0	40	0	1 000	0
5	100	60	50	30	20	20	0	900	0
8	200	70	65	0	30	0	30	290	0
7	280	80	100	600	45	0	85	1 100	0
								120[1]	19
6	100	60	50	30	20	0	80	150	2
3	300	40	120	50	20	40	220	920	103
1	450	50	0	10	20	20	330	1 600	137
Total	.	.	.	.	.	.	.	6 280	261

[1] 120 cattle exposed for a short period to game in an adjacent Camp 7

Pypekamp). The data so obtained enabled an identification of cattle herds according to age, time and nature of exposure and breeding status.

Herds of cattle

Although only 13 owners were involved, 37 different herds of cattle consisting of 6 280 individuals were identified. Cattle born and kept on the same farm and in the same camps as wildebeest (4 herds) were regarded as being continuously exposed. Intermittently exposed cattle were regarded as being directly exposed (7 herds), when they shared the same grazing and watering points as wildebeest, and indirectly exposed when they were separated in different ways, such as by game-proof fences (12 herds), a main road and a narrow strip of land 100 m wide (4 herds) or camps and farms of 1 000 m or more in width (10 herds).

The exposure time in the case of intermittently exposed cattle varied from 1 week to almost 11 months. These cattle originated either from local farmers who employed a rotational grazing system or from farmers in other areas who made use of emergency grazing provided by farmers in this location.

Wildebeest calving season

The wildebeest calving season varies from year to year, but usually occurs from approximately 15 December–15 January.

Virus reservoir

In April 1987, 14 3–4-month-old wildebeest calves were captured and hand-reared in a small camp on Farm No. 1 (Fig. 1). Specimens for virus isolation were collected from the calves at monthly intervals from April–October. Leucocytes, obtained from 1 mℓ of osmotically lysed blood, nasal mucus and tears, were co-cultured within 3 h of collection with foetal lamb kidney cells in 25 mℓ plastic flasks[1]. The cultures were incubated at 33 °C–35 °C and examined daily for the appearance of cytopathic foci indicative of AHV-1 multiplication. Cultures not showing cytopathic changes were discarded after 21 days. Isolated virus was identified on a microneutralization test (Mushi & Plowright, 1979), using known positive serum prepared by inoculation of rabbits with the WC 11 isolate of WD MCF virus (Plowright, 1964).

[1] Nunc.

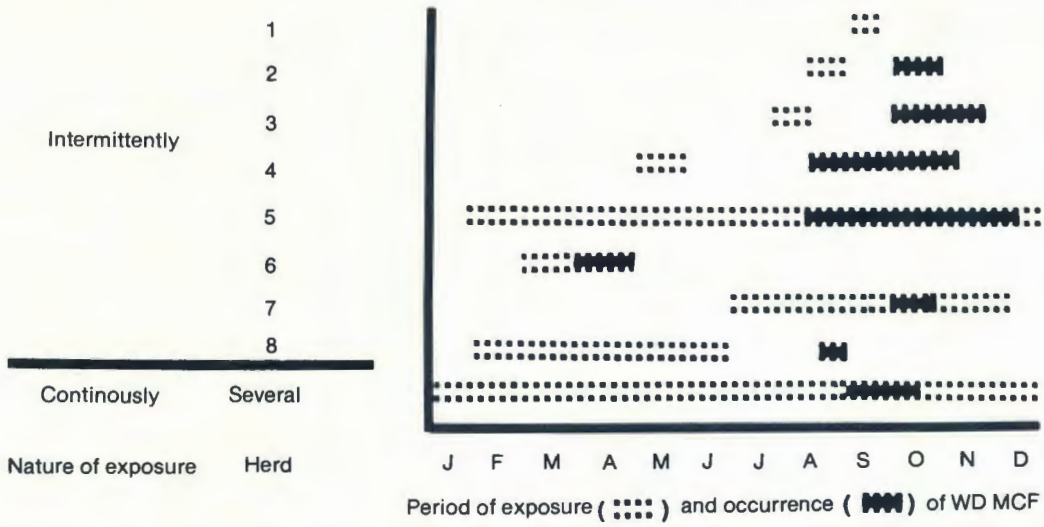

FIG. 2 Occurrence of WD MCF in cattle exposed to wildebeest for different periods in the north-western Transvaal

RESULTS

Distribution of game and occurrence of malignant catarrhal fever

During the observation period, WD MCF occurred only on farms with wildebeest or in camps adjacent to wildebeest (Fig. 1 and Table 1). No cases of MCF were encountered among cattle utilizing the same grazing and watering points as other game, such as gemsbok (*Oryx gazella*), hartebeest (*Alcelaphus buselaphus*), blesbok (*Damaliscus dorcas*), waterbuck (*Kobus ellipsiprymnus*), kudu (*Tragelaphus strepsiceros*), impala (*Aepyceros melampus*), or other less abundant game. The majority of the MCF cases occurred in cattle exposed to 2 herds of 220 and 330 wildebeest. No cases were encountered among cattle exposed continuously to a small herd of approximately 30 wildebeest or among 1 100 cattle exposed continuously to 85 wildebeest, while 19 cases were encountered among 120 cattle exposed for a short period to the same herd of wildebeest.

Seasonal occurrence of WD MCF

Although 25 % of the cattle were exposed to wildebeest throughout the entire year, most deaths resulting from WD MCF occurred in spring (Table 2). The first cases in the main outbreak were seen in July. The incidence increased in August, reached a peak in September–November, after which it declined. In the first week of January 1987 only 2 cases were recorded. The 17 cases noted in April and May occurred in 1984 in a herd that had grazed on the game farm during March and April. No cases were seen among cattle indirectly exposed during summer and autumn.

TABLE 2 Seasonal occurrence of WD MCF during 1984–1986 in the north-western Transvaal

Cattle		Monthly distribution of WD MCF cases											
Exposed to wildebeest on farm	Number exposed	J	F	M	A	M	J	J	A	S	O	N	D
1	1 600	0	0	0	10	7	0	6	10	42	33	18	11
3	920	0	0	0	0	0	0	0	17	20	31	29	6[1]
6	150	0	0	0	0	0	0	0	0	0	2	0	0
7	120	0	0	0	0	0	0	0	2	11	3	3	0
Total		0	0	0	10	7	0	6	29	73	69	50	17
Percentage		.	.	.	6,5		.	.	.		73,6		.

[1] Including 2 cases encountered in the first week of January 1987

TABLE 3 Nature of contact between cattle and wildebeest and the incidence of WD MCF in the north-western Transvaal during 1984–1986

Nature of exposure	Cattle				Incidence of WD MCF percentage
	Herds		Individuals		
	Exposed	Affected	Exposed	Affected	
Intermittent					
Direct (mutual grazing)	7	6	1 360	71	5,2
Separated by fence	12	8	1 320	133	10,1
Separated by 100 m	4	2	200	51	25,5
Separated by 1 000 m	10	2	1 800	2	0,1
Continuous	4	2	1 600	4	0,3
Total	37	20	6 280	261	4,2

TABLE 4 Occurrence of WD MCF in cattle of different age and breeding status in the north-western Transvaal

Age and breeding status	Number of cattle		
	Exposed	Affected	% affected
Young and dry	1 690	11	0,7
Near-term	200	33	16,5
Cows recently calved a	340	85	25,0
Cows recently calved b	130	26	20,0
Calves of cows b	60	11	18,3
Mixed[1]	3 860	95	2,5

[1] Mostly young and dry cattle

Transmission time of WD MCF

The practice of rotational grazing employed by several farmers and the use of emergency grazing made available by some farmers, enabled us to relate the occurrence of WD MCF to a specific period of exposure (Fig. 2). The data shown clearly illustrate that, with the exception of cases in Herd 6, outbreaks of WD MCF occurred from the last week of July to the first week of January 1987, irrespective of the time and period of exposure. Cattle in Herds 2 and 3, exposed from the middle of August for 2 and 3 weeks respectively, contracted the disease about 6 weeks after the first exposure. The period from the last exposure to the occurrence of the last case, varied from 11 weeks in Herd 2, to 18 weeks in herd 4. A similar minimum period occurred in Herd 6, in which direct exposure took place during March and April.

The influence of contact on the incidence of WD MCF

Transmission (Table 3) occurred through direct contact in 5,2 % of animals when cattle and wildebeest shared the same camps and watering troughs. Where they were separated by a game-proof fence or where they were separated by a main road and a narrow strip of land 100 m wide, the incidence was 10,1 % and 25,5 % respectively. The incidence was remarkably lower (0,3 %) among cattle born and reared in the same camps as wildebeest. Virtually no cases were encountered where the wildebeest and cattle were separated by 1 000 m or more of densely vegetated grazing.

Breeding status of cattle and the incidence of WD MCF

Some farmers manged their young cattle, near-term cows and recently calved cows as separate herds. This enabled a comparison to be made of the incidence of disease in groups of cattle of different breeding status (Table 4). The data indicate a remarkable difference in mortality rates in the different groups. The highest incidence (20 %–25 %), in recently calved cows, was followed by near-term cows (16,5 %). The incidence of WD MCF among young cattle and dry cows was low (0,7 %), but in 1 herd the incidence among cows and their calves was very similar, being 20 % and 18,3 % respectively. In this herd, which was separated from wildebeest by a game-proof fence, the calves of cows that had died were hand-reared away from wildebeest. These calves died of WD MCF from a few days up to 68 days after their mothers had died.

Virus isolation from wildebeest

AHV-1 was isolated from 8 of the 14 wildebeest calves: Four in April, 3 in May and 1 in June (Table 5). Two isolations were made from nasal mucus, 2 from blood and 5 from tears. Specimens from 9 wildebeest calves, sampled from July–October, yielded no virus. Unfortunately it was impossible to collect specimens from wildebeest calves before April, i.e. calves younger than 3–4 months of age.

DISCUSSION

Outbreaks of WD MCF can usually be related to close contact with wildebeest, which act as reservoirs of infection and indicator hosts (Plowright, 1964). Wildebeest calves less than 3 months old are regarded as the principal reservoirs. The mean virus titres of AHV-1 in their ocular fluid may be as high as $10^{2,25}$ TCID 50/mℓ and up to 40 % of calves may be viraemic (Plowright, Ferris & Scott, 1960; Mushi, Karstad & Jessett, 1980). After the calves are more than 3 months of age, virus shedding declines as a result of the appearance of neutralizing antibody in the nasal cavity (Mushi, Jessett, Rurangirwa, Rossiter & Karstad, 1981). The occurrence of WD MCF in Kenya in April and May and somewhat later in Tanzania can thus be correlated to contact between cattle and wildebeest calves 3–5 months old. The calves in this investigation were slightly older when first sampled and the incidence of virus excretion was 28 % in April and 21 % in May. No virus excretion occurred from July–October.

The present investigation reconfirmed several previously observed aspects of the epizootology of WD MCF in South Africa. All the cases of WD MCF

TABLE 5 Virus isolation from 4–9-month-old wildebeest calves in captivity on a farm in the north-western Transvaal

Calf number	Virus isolated from tears, nasal mucus and blood			
	April	May	June	July–October
1	Tears	—	—	—
2	—	—	—	—
3	—	—	(Escaped)	.
4	—	Tears. Blood	(Escaped)	.
5	—	—	—	(Died)
6	—	—	—	—
7	Tears	—	—	—
8	—	Tears	—	—
9	—	—	Mucus	—
10	—	—	—	—
11	Tears	—	(Escaped)	.
12	—	—	—	—
13	—	Mucus	(Died)	.
14	Blood	—	—	—
Calves positive	4	3	1	0
Calves tested	14	14	10	9

encountered were associated with wildebeest. The absence of cases in cattle in several camps containing hartebeest and gemsbok, as well as other game, but not wildebeest, strengthens the belief that species other than wildebeest do not play an important role in the epizootology of WD MCF (Plowright, 1967; Reid & Rowe, 1973). Previously, hartebeest and gemsbok had been identified as potential transmitters of AHV-1 (Barnard, 1984).

The period from first exposure to development of clinical signs of WD MCF may be as short as 6 weeks and the period between last exposure and the occurrence of the last cases varied from 12–18 weeks. The minimum period of 6 weeks is slightly longer than the incubation period of 3–5 weeks after experimental infection (B. J. H. Barnard, unpublished data, 1983). The longest period from the last exposure to development of symptoms is also similar to that observed after experimental infections.

Although WD MCF in South Africa does occur during January–May, when wildebeest calves are less than 5 months old, the majority of cases are encountered from September–November, when the calves are 8–11 months old (Barnard 1984; Barnard & Van de Pypekamp, 1988). In the present outbreak, 77,6 % of the cases were encountered in September–November. This implies the existence of an additional source of virus and/or involvement of an intermediate host responsible for the infection in catle from July–October.

The hypothesis for the existence of an intermediate host is further strengthened by the occurrence of cases of WD MCF among cattle that are separated from wildebeest by fences and distances of several hundred metres (Barnard & Van de Pypekamp, 1988). In the present investigation only 75 of the 261 cases were encountered in cattle using the same grazing and watering points as wildebeest. The incidence of 5,2 % and 0,3 % among these cattle is notably lower than the incidence in cattle separated by a game-proof fence (10,1 %) or when separated by at least 100 m (25,5 %). Cattle kept in the same camps as wildebeest are not necessarily in contact with them. The 2 species are seldom seen to mix or graze together. In fact, because of the large camps any contact is purely coincidental, but close contact at times cannot be ruled out.

It is a pity that there were no herds of cattle that were separated from wildebeest over distances of between 100–1 000 m. However, with the information obtained it seems reasonable to assume that in order to prevent severe losses as a result of WD MCF, cattle must be separated from wildebeest by at least 1 000 m.

The high incidence in near-term and recently calved cows must be evaluated with caution in the light of a similarly high incidence among cows and calves in another herd. The calves succumbed from a few days to 68 days after their mothers, and it is presumed that some cases were the result of *in utero* infections. However, in several other similarly exposed herds, mortalities among calves were rarely encountered. The high incidence in pregnant and recently calved cows does not necessarily indicate a higher susceptibility, but may be due to unkown factors of possible importance in the epizootology of the disease. The low incidence in cattle born and raised in the same camps as wildebeest may be due to the same factors, such as the host preference of an intermediate host or the ability of cattle in certain circumstances or under specific conditions, to avoid contact with an intermediate host.

As wildebeest calves younger than 5 months old are at present the only known source of virus, the occurrence of WD MCF in September–November can only be explained by the existence of an as yet unidentified host or vector.

REFERENCES

BARNARD, B. J. H., 1984. Transmitters and potential transmitters of malignant catarrhal fever: Proceedings of XIII World Congress on Diseases of Cattle, Durban. South Africa, September 1984.

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.

MUSHI, E. Z. & PLOWRIGHT, W., 1979. A microtitre technique for the assay of malignant catarrhal fever virus and neutralizing antibody. *Research in Veterinary Science*, 27, 230–232.

MUSHI, E. Z., KARSTAD, L. & JESSETT, D. M., 1980. 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., JESSETT, D. M., RURANGIRWA, J. F. R., ROSSITER, P. B. & KARSTAD, L., 1981. Neutralizing antibodies to malignant catarrhal fever herpesvirus in wildebeest nasal secretions. *Tropical animal Health Production*, 13, 55–56.

PLOWRIGHT, W., 1964. Studies on bovine malignant catarrhal fever of cattle, D.V.Sc Thesis, University of Pretoria. South Africa.

PLOWRIGHT, W., 1967. Malignant catarrhal fever in East Africa. III. Neutralizing antibody in free-living wildebeest. *Research in Veterinary Science*, 8, 129–136.

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.

REID, H. W. & ROWE, L., 1973. The attenuation of a Herpesvirus (malignant catarrhal fever virus) isolated from hartebeest (*Alcelaphus buselaphus* Cokei) Gunther. *Research in Veterinary Science*, 15, 144–146.