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SHORT COMMUNICATION

CLINICAL FOOT-AND-MOUTH DISEASE IN THE AFRICAN BUFFALO (*SYNCERUS CAFFER*)

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During the course of a recent epizootic of foot-and-mouth disease (FMD) amongst game in the Kruger National Park, a group of young adult buffalo held in captivity became naturally infected. Where circumstances permitted, observations were made on the clinical course of the disease in these animals and this report is published as a record of the first confirmed clinical cases of this disease amongst this species.

Previously attention has been drawn to the complete absence of clinical FMD or other evidence of healed lesions amongst buffalo in Botswana, Rhodesia and Uganda, from which carrier virus had been isolated (Hedger, Condy & Falconer, 1969; Hedger, 1971; Hedger, Forman & Woodford, in press).

During the course of four separate epizootics covering a period of 5 years in the Kruger National Park, characteristic vesicular mouth and hoof lesions have only very rarely been observed in this species, notwithstanding the fact that abundant clinical evidence of the disease was present amongst impala (*Aepyceros melampus*). Between March 1970 and February 1971 while the disease was prevalent in the Park and more detailed examination was possible, it was found that only 22 of 1 285 buffalo carcasses from all areas of the Park showed lesions suggestive of FMD, although on occasions, the examination of small random groups showed that the number of animals with distinguishable lesions could be as high as 25 to 43%. In all these cases however, confirmation was not possible as the lesions were very small and suitable epithelial samples could not be collected.

In the early stages of this epizootic, FMD amongst the free living game population of the Park was confirmed and a strain of SAT 2 virus was identified. Immediately after the disease had been confirmed and in anticipation of the spread of the virus amongst the wild fauna, serum was collected and pharyngeal scrapings taken for the detection of carrier virus in the captive buffalo. Seven weeks later the first symptoms of the disease were observed in these animals.

Initially, three buffalo (No. 3, 5 & 6) were affected. They showed malaise, a degree of anorexia, pyrexia which varied up to 41°C (normal temperature 38,6°C) and a painful gait. The disease spread rapidly and within 7 days all but one (No. 1) of the remaining animals appeared to have become infected. The spread of the disease was apparent from the onset of lameness in one or more feet in seven of the eight animals, some of which also showed salivation. In the most severely affected cases careful chewing movements, intermittent opening and closing of the mouth, protrusion of the tongue and profuse salivation were observed. Salivation persisted for up to 7 days, while the healing lesions in the mouth could still be seen after 13 days. Lameness continued for 10 days and during this period the affected animals showed a marked loss in mass and condition. Buffalo No. 1 showed no obvious symptoms of the disease during the period of observation.

The three animals first found to be infected were tranquillized and examined more closely. Vesicles and ulcers were generally very small, measuring approximately 1 cm in diameter although in one animal a lesion on the hard palate measured 2,5 cm (Fig. 1). These lesions were confined to the dental pad, palate, the dorsum of the tongue and the lips. On the feet, similar small vesicles were found on the coronary band and in the interdigital cleft (Fig. 1). When ruptured, small amounts of clear fluid exuded from the broken epithelium. Specimens of vesicular epithelium from these animals yielded SAT 2 virus and in one of them the titre of virus was found to be $10^{6,5}$ TC ID_{50} per gram. In two animals a viraemia was demonstrated with a virus titre of $10^{3,7}$ TC ID_{50} per ml. After a period of 3 weeks, the animals appeared to have recovered fully and no evidence of previous infection could be detected.

Six months after the first appearance of the disease in the buffalo, serum and pharyngeal scrapings were again collected. The incidence of carrier virus in these and the earlier samples, as well as the titre of neutralizing antibodies at that time, in the recovered animals, are given in Table 1.

It is of interest to note that before infection one of the 8 buffalo (No. 1) estimated to be 3 years of age was a carrier of SAT 3 virus, a type which was last identified as a cause of clinical disease in this region some 11 years previously. Antibody levels in this particular animal also suggested previous infection with and immunity to both SAT 1 and 2 viruses and would explain why this animal did not succumb to clinical disease. However, it did become a carrier of the virus. Although this group of animals had been confined in close association with one another, including buffalo No. 1 which had been introduced 4 months before the collection of the first samples, there was no evidence of the spread of SAT 3 virus within the group. On the other hand clinical infection has resulted in all the animals becoming carriers to SAT 2 virus. In the convalescent sera taken 6 months after infection there is a high titre of homologous antibody and it is significant that no heterotypic response followed infection by the SAT 2 virus.

It has always been suspected that infected buffalo may play an important role in the spread of the disease. In these confirmed clinical cases, the results indicate that high concentrations of virus may be shed during the acute phase of the disease. Natural transmission of the disease to cattle took place when three steers in a pen adjoining the buffalo became infected with the same strain of virus 11 to 17 days later. All three of these

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TABLE 1 Recovery of carrier virus and the immune response of buffalo and in-contact cattle

Animal No.	Age (years)	Recovery of carrier virus*		Neutralizing antibody titres					
		Before infection	6 months after infection	Pre-infection			6 months Post-infection		
				SAT 1	SAT 2	SAT 3	SAT 1	SAT 2	SAT 3
Buffalo									
1	3	Pos - SAT 3	Pos - SAT $2\text{-}10^{2,0}$/ml	265**	90	355	178	90	178
2	$1\frac{1}{2}$	Neg	Pos - SAT $2\text{-}10^{2,33}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	256	≤ 6
3	$1\frac{1}{2}$	Neg	Pos - SAT 2 Trace,	≤ 6	≤ 6	≤ 6	≤ 6	178	≤ 6
4	2	Neg	Pos - SAT $2\text{-}10^{2,0}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	355	≤ 6
5	4	Neg	Pos - SAT $2\text{-}10^{2,5}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	256	≤ 6
6	$3\frac{1}{2}$	Neg	Pos - SAT $2\text{-}10^{2,66}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	256	≤ 6
7	$2\frac{1}{2}$	Neg	Pos - SAT $2\text{-}10^{2,5}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	1 024	≤ 6
8	$2\frac{1}{2}$	Neg	Pos - SAT $2\text{-}10^{3,5}$/ml	≤ 6	≤ 6	≤ 6	≤ 6	128	≤ 6
Cow									
1		Neg	Neg	8	≤ 6	≤ 6	≤ 6	32	≤ 6
2		Neg	Neg	≤ 6	≤ 6	≤ 6	≤ 6	64	≤ 6
3		Neg	Neg	≤ 6	≤ 6	≤ 6	≤ 6	≤ 6	≤ 6

*Isolation and assay performed in primary calf thyroid cells
**Reciprocal of the final dilution of serum present in the serum-virus mixture at the 50% end point as determined by the cell metabolic inhibition test (Martin & Chapman, 1961)

animals showed a high temperature as well as lesions on all four feet and in the mouth. Surprisingly however, carrier virus was not isolated from these animals and antibody titres 6 months after infection were of a much lower order than those of the buffalo (Table 1).

The incidence and severity of the disease in domestic animals vary from outbreak to outbreak and experience of the disease in susceptible wild animals under natural conditions has been similar. The reported incidence of clinical infection in buffalo has however always been of a low order. This low incidence of clinical disease in free living buffalo contrasts with the high percentage of animals which become carriers and poses an interesting problem in the natural evolution of the disease. Although previous work has emphasized the fact that the virus of FMD may persist in buffalo for considerable periods in the absence of clinical symptoms, the foregoing observations show that clinical infection can occur. Preferential adaptation of strains of FMD virus of pigs rather than cattle and *vice versa* is well established. A similar situation may exist in wild species. The occurrence of clinical signs may depend on the interplay between strains well adapted to buffalo and the antibodies which many of these animals possess.

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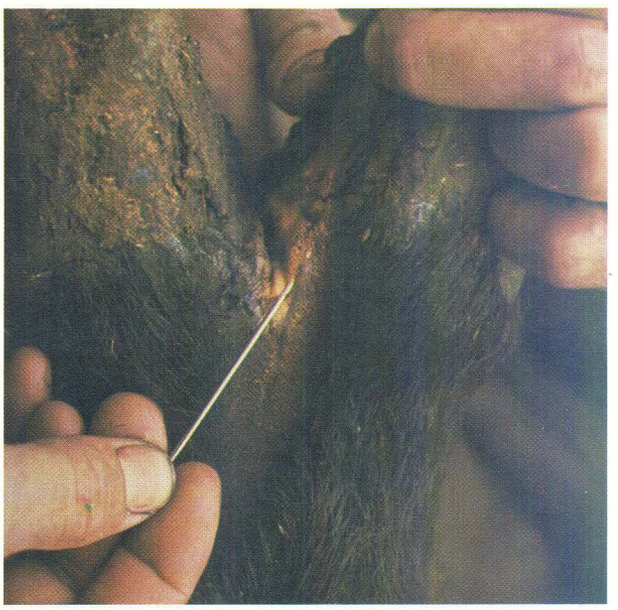

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FIG. 1 (1) Ruptured vesicle on hard palate of buffalo, estimated 3rd day of illness. (2) Ruptured vesicle, interdigital cleft of buffalo showing lameness

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