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Investigations on the incidence of rinderpest virus infection in game animals of N. Tanganyika and S. Kenya 1960/63

By W. PLOWRIGHT and B. McCULLOCH*
E.A. Veterinary Research Organization, P.O. Box 32, Kikuyu, Kenya,
and
Veterinary Investigation Centre, Arusha, Tanganyika Territory

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It is generally considered that all animals of the Order Artiodactyla are susceptible to infection with rinderpest virus, although convincing evidence is lacking for many species (Scott, 1959). There is also a consensus of opinion in veterinary circles that game animals can play a most important role in maintaining the virus in East Africa, even in the absence of susceptible cattle, and furthermore that they have often been responsible for rapid and extensive spread of the disease (see, for example, Lowe, 1942; Thomas & Reid, 1944; Wilde, 1953; Branagan & Hammond, 1965). In addition, from the point of view of the wildlife conservationist or biologist, rinderpest is undoubtedly the most important infectious disease of wild ruminants because of its potential killing power (Blayney Percival, 1918; Simon, 1962), and the impossibility of prophylaxis.

With these considerations in mind all available game animal sera were examined for rinderpest-neutralizing antibody beginning in 1960 and, with the exception of the year 1964, continuing to date. The sera were predominantly from wildebeest, the reservoir host of malignant catarrhal fever virus (Plowright, 1965) but smaller numbers of other species were obtained later. Practical difficulties in obtaining sera unfortunately excluded the African buffalo from this list, although it is undoubtedly most important in the epizootiology of rinderpest. This paper deals with the results obtained in the initial 3 years of the investigation, during the first half of which rinderpest infection was still frequent in some species. A brief account of some of the findings to 1962 was given in an earlier paper (Plowright, 1963*a*).

MATERIALS AND METHODS

Sera

Blood from animals which had been killed by shooting was obtained by severing the neck or other large vessels, whilst animals immobilized by drugs were bled from the jugular vein. The blood was usually allowed to clot and stand overnight at environmental temperature before removing serum for clarification by centrifugation. Serum samples were then stored at 4° C., usually preserved by the addition of a drop of chloroform, and later forwarded on ice; after arrival at the laboratory they were stored until required at −20 to −25° C. and then thawed and inactivated at 56° C. for 30 min.

* Present address: Veterinary Investigation Centre, P.O. Box 129, Mwanza, Tanzania.

Rinderpest neutralization tests

Undiluted serum was tested against $10^{1.6}$ to $10^{2.8}$ TCD 50 of culture-adapted virus of the Kabete 'O' strain (Plowright, 1962). Two tubes were used per sample and the protection of one or both of them against the cytopathic effects of the virus was considered to indicate a significant level of neutralizing activity. The great majority of positive samples were then titrated for neutralizing activity using $10^{1.8}$ to $10^{2.6}$ TCD 50 of virus per tube and an identical technique to that already described (Plowright, 1962). A standard immune cattle serum, not inactivated, was included in each test as a check on the sensitivity.

Log_{10} SN 50 titres were calculated by the method of Thompson (1947); where serum at a final dilution of 1/2 only protected one or two of a total of five tubes, titres were expressed nominally as $10^{0.1}$ and $10^{6.2}$ respectively, for inclusion in calculations of mean titres. Titres were ordinarily written as the reciprocal of the $\log_{10}$ SN 50 end-point.

Animal populations

The populations of blue wildebeest (*Connochaetes taurinus*, Burchell; syn. *Gorgon taurinus taurinus*, Burchell) were in general the same as those already described (Plowright, 1965). However, in the light of more recent studies (Estes, 1966; Watson, 1967) it is apparent that the wildebeest occurring in areas to the west of the Great Rift Valley can be subdivided into several ecologically distinct groups.

By far the largest population is the Serengeti migrant group, which numbers at present 320,000–380,000 head (Watson, 1967), moving in vast hordes on to the central plains and towards Ol Balbal during the wet season (December to June) and dispersing mainly to the west in the remaining dry season (Watson, 1965, 1966; Figure 1). At the western end of the Serengeti National Park is a small group of about 4000 head, resident in the Kirawira area; they disperse during the dry season with migrant animals. In the Ngorongoro Crater there are about 10,000–15,000 resident wildebeest, of which about 35% leave the caldera for the Ol Balbal Plains during the wet season (Estes, 1966; Watson, 1967); whilst there they may intermingle with migrant herds but they return to the Crater in the dry season. A further resident group is found in the Mara-Loita Plains area of Kenya and these animals may establish contact with Serengeti migrants as a result of their southward movement during the dry season; they number about 15,000–20,000 head.

The age of wildebeest was estimated from their dentition and general development, aided by the fact that the great majority of calves are born usually within a period of 6–8 weeks extending from February to April in the Kajiado area and from December to February in areas to the west of the Rift Valley. In ageing very young calves consideration was also given to the state of the umbilical cord, hooves, central incisors and horn buds.

In the Ngorongoro Conservation Area during the period early February, 1962 to June of the same year efforts were made to obtain samples from as many dam-

calf pairs as possible. This was usually accomplished by shooting the calf first and then the dam as she turned back towards her offspring. It was thus possible to collect materials for a study of the relationship between serum antibodies in the dam and the calf during the first 4–5 months of life of the latter.

Sera from eland (*Taurotragus oryx* Pallas), Thompson's gazelle (*Gazella thomsoni* Gunther), Grant's gazelle (*Gazella granti* Brooke), and Coke's hartebeest (*Alcelaphus buselaphus cokii* Gunther) were nearly all obtained within the Ngorongoro Conservation area, a proportion of them within the Crater itself. Additional sera from Thompson's gazelle were obtained from the Naivasha area of Kenya, where rinderpest is not enzootic.

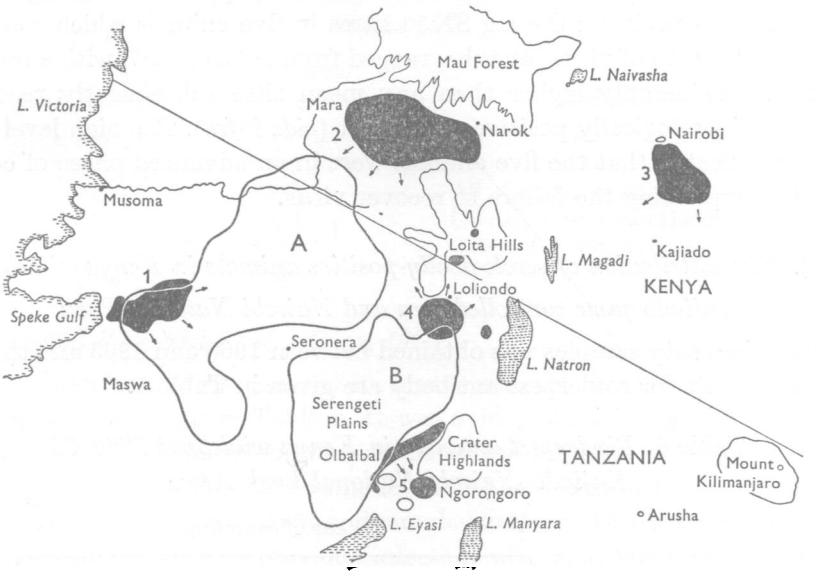

Fig. 1. The main wildebeest populations of East Africa (with acknowledgements to Watson, 1967). A. Dry season dispersal area of the main Serengeti migrant population (July to November). B. Wet season concentration area of Serengeti migrant wildebeest: (1) Kirawira resident group; (2) Mara National Park: Loita Plains group; (3) Kajiado: Nairobi National Park group; (4) Loliondo resident group; (5) Ngorongoro resident group. Arrows indicate the direction of dry-season dispersal.

RESULTS

(1). *Confirmation of rinderpest in the Serengeti National Park—October/November 1960*

In October 1960 an outbreak of severe disease with considerable mortality occurred in 'yearling' wildebeest in the Serengeti National Park; when observed on 6 to 8 November the mortality had already declined considerably, but virtually all 'yearling' wildebeest exhibited a harsh, discoloured pelage with faeces drying on and adherent to the tail and perineal region. There had also been a considerable loss of condition. Five affected animals, whose age was estimated to be 7–9 months, were killed and attempts were made to isolate virus from their spleens and blood by inoculating 5% (w/v) suspensions of the former or leucocyte fractions from

10 ml. of the latter into culture of primary calf kidney cells; the techniques have already been described in detail (Plowright & Ferris, 1962). No cytopathogenic agent was detected.

A sixth yearling animal was found dead, the carcass being partially eaten by predators; its spleen also failed to yield virus but a positive result for rinderpest virus antigens was obtained in agar gel diffusion tests against hyperimmune rabbit serum (Scott & Brown, 1961*). In this animal the only lesion which was suggestive of rinderpest was epithelial necrosis and caseous deposits on the palatal tonsils; in the other five animals no abnormalities indicative of rinderpest infection were seen.

Sera from all six yearlings were tested for rinderpest neutralizing antibody and found to be positive; the log SN 50 titres in five animals which were killed, against $10^{2\cdot4}$ TCD 50 of virus per tube, ranged from 1·8 to $\geqslant$ 2·7, with a mean of at least 2·38, considerably higher than any mean titre subsequently recorded in any group of serologically positive wildebeest (*vide infra*). The high level of antibody also indicated that the five animals were in an advanced phase of convalescence, thus explaining the failure to recover virus.

(2). *The distribution of serologically-positive animals in Kenya wildebeest Kajiado game controlled area and Nairobi National Park*

A total of seventy samples was obtained between 1960 and 1963 and the results of screening tests for rinderpest antibody are given in Table 1.

Table 1. *Rinderpest antibody in Kenya wildebeest 1960–63 Kajiado–Nairobi National Park Areas*

	Year of sampling		
Year of birth	1960	1961	1963
⩽1957	2/9*	—	
1958	0/1	—	2/16
1959	0/3	—	
1960	0/4†	—	
1961	—	3/14‡	3/23§
1962	—	—	

* No. positive/no. tested.
† All calves 3/4 months old.
‡ All calves < 1 month old, of which 7 may not have received colostrum.
§ Titres 0·8, 0·8 and 'trace'.

In 1960 although two of nine adults were positive, no animal less than 3 years old had antibody. In 1963, however, there were three of twenty-three sera from immature animals, i.e. probably 1–2 years old, which did have antibody. The SN 50 titre of two of the positives, 0·8 against $10^{2\cdot4}$ TCD 50 of virus, was sufficiently high to indicate that no error had occurred in the preliminary screening test; in the third animal a titration revealed only a trace of antibody. The presence of anti-

* We are indebted to Dr G. R. Scott for carrying out these tests.

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body in 3/14 young calves captured in 1961 was presumably a reflexion of successful colostral transmission, although all these animals had been abandoned by their dams very early in life (Plowright, 1965) and half of them did not have antibodies to malignant catarrhal fever virus (M.C.F.) although this was universally present in adults. These animals may not, therefore, have received a feed of colostrum.

In the aggregate, it can be seen that seven of fifty-two animals (13·3 %) which were one or more years old showed evidence of previous infection with rinderpest virus. One serum from an adult eland shot in the Kajiado area in 1963 also exhibited an SN 50 titre of 0·8 against $10^{2·2}$ TCD 50 of virus.

Table 2. *Rinderpest antibody in Kenya wildebeest; Mara–Loita Plains 1960*

Year of birth	No. positive/ no. tested	Remarks
⩽1957	12/13	—
1958	3/3	—
1959	5/6	All obtained in March 1960
1960*a*	3/4	Calves up to 3 months old
1960*b*	0/6	Calves 5–8 months old
Total	32	

Mara National Park and Loita Plains, 1960

Between March and September 1960, serum samples were collected from thirty-two wildebeest as shown in Table 2. It was evident in this region that antibody passed via the colostrum of immune dams to the majority of young calves but that it had declined to non-detectable levels in the 5–8 months age group. Of six yearling animals born in early 1959 and sampled in March 1960 there were five which already showed evidence of previous infection with rinderpest virus and a very large proportion (*ca.* 94 %) of the adult animals similarly possessed antibody.

The serological conversion of calves born in 1959 could well have been due to an extension of the rinderpest infection reported to have been present in wildebeest in the Mara area of Tanganyika (Thomas, 1960).

(3). *The distribution of serologically-positive animals in Tanganyika wildebeest Ol Balbal Plains, 1962/63*

The month of collection of serum samples and the results of tests for rinderpest antibody are given in Table 3 for 1962 and Table 4 for 1963.

The majority of samples were obtained during the months January to May when both resident and migratory groups of wildebeest were present but the latter were almost certainly in great predominance and the figures can probably be regarded as typical for the migrant Serengeti population.

Of animals born in or before 1959 seventy-one of 81 (87·7 %) possessed antibody, the proportion of positives in the 1960 and 1961 calf crops falling to 75·8 % and 67·1 % respectively. The rising immunity rate in animals born in earlier years indicated that some individuals which escaped previous annual episodes became

infected in later ones (Table 3). It was evident from figures obtained early in the year 1962 that many calves born one year previously had already been actively immunized; assuming that they had not lost protective levels of colostral antibody until August or September, 1961, the infection must have been widespread in these herds at some time between then and January 1962. No disease or mortality in Serengeti migrant animals was reported during this time, however, although rinderpest 'took a fairly heavy toll of yearling buffalo during October and December' around the Ngorongoro Crater (Kinloch, 1963) and it was stated that the disease was responsible for the usual number of deaths of yearling wildebeest and buffalo in the Narok District of Kenya (Anon, 1962).

Table 3. *Rinderpest antibody in Tanganyika wildebeest; Ol Balbal Plains 1962*

Year of birth	Month of Collection									Positive %
	Jan.	Feb.	Mar.	Apr.	May	July	Aug.	Sept.	Totals	
⩽ 1958	6/6*	15/20	5/5	6/6	3/3	—	1/2	1/2	38/44	86·4
1959	5/5	9/10	12/13	2/2	4/5	1/2	—	—	33/37	89·2
1960	7/9	5/9	6/8	—	4/4	3/3	—	—	25/33	75·8
1961	23/31	11/15	9/16	8/10	2/5	0/1	0/1	—	53/79	67·1
1962	—	14/20	17/17	8/10	5/9	1/7	2/7†	0/2	See text	—
Totals	51	74	59	28	26	13	10	4	265	

* No. positive/no. tested.

† Positives estimated to be 8 months old, with titres of 0·6 and 0·8 against $10^{2\cdot6}$ TCD 50 of virus. Probably derived from resident population.

Table 4. *Rinderpest antibody in Tanganyika wildebeest; Ol Balbal Plains 1963*

Year of birth	Month of Collection					Totals
	Jan.	Feb.	Mar.	Apr.	June	
1961	—	—	—	—	1/1	1/1
1962	1/6	2/3	0/4	0/2	3/6*	6/21
1963	—	—	—	—	0/1	0/1
Totals	6	3	4	2	8	23

* Probably derived from the resident population.

Two animals estimated as 8 months old were found to be positive in August 1962 and there can be little doubt from the titres recorded that antibody was acquired following active infection; the time of collection made it very probable that these animals were members of a resident herd forming part of the Ngorongoro group. The single positive obtained amongst seven animals in July (Table 3) could have been due to residual colostral antibody; the calf involved was only 5 months old and the titre was very low.

During 1963, six of twenty-one animals born in early 1962 were found to have been infected and of these four had a reasonably high antibody titre (0·8), the other two exhibiting only trace amounts (Table 4). The time of collection of positive samples made it impossible to decide whether the low rate of infection which occurred during 1962 affected both the static (Ngorongoro) and the migratory (Serengeti) herds.

The figures in Table 3 for calves estimated to be less than 5 months old will be discussed in section 4 of the results.

Ngorongoro Crater, 1961/63

Tables 5 and 6 give details for collections made during 1961/62 and 1963 respectively. Whilst 100 % of the wildebeest 4 years old or more possessed antibody, the proportion in 3-year animals (75 %) fell somewhat below that determined in the Serengeti area (89 %) but the number of samples was small. The

Table 5. *Rinderpest antibody in Tanganyika wildebeest; Ngorongoro Crater 1961/62*

Year of birth	Month of collection									Positive %
	Dec. 61	Jan. 62	Apr.	May	June	Aug.	Sept.	Oct.	Totals	
⩽ 1958	16/16*	—	—	—	—	—	3/3	—	19/19	100
1959	—	—	4/5	0/1	2/2	—	—	—	6/8	75
1960	—	—	—	—	2/2	0/1	—	—	2/3	66
1961	0/8	0/10	2/11	1/4	0/11	2/5	1/3	—	6/52	11·5
1962	—	—	7/8	2/4	1/5	3/11†	0/3	0/5	See text	—

* No. positive/no. tested.

† Positives estimated to be 9 months old with SN 50 titres of 0·8, 1·8 and 0·4 against $10^{2\cdot6}$ TCD 50 of virus.

Table 6. *Rinderpest antibody in Tanganyika wildebeest; Ngorongoro Crater 1963*

Year of birth	Month of Collection				Totals
	Jan.	Feb.	Mar.	Nov.	
1962	0/6	0/13	0/3	0/2	0/24
1963	—	—	—	0/26	0/26

difference between the 1961 calf crops in the two localities was, however, very striking. The number of serologically-positive animals in the Crater was 11 % of the total tested, compared with 67 % of the Ol Balbal herds. A very few of the 1962 calf crop had also been infected by August of that year, with serum titres sufficiently high in two of three instances (Table 5) to leave no doubt of recent active infection, comparable to that in animals shot in June on the Ol Balbal Plains. The collections during 1963 failed to reveal any positive animal in fifty tested, of which twenty-four were born in the 1962 calving season (Table 6). It must, therefore, be considered possible that the Crater animals positive in August 1962 became infected during a seasonal move to Ol Balbal grazings.

Kirawira-Ikoma area, 1962/63

A total of thirty-two samples was obtained between December 1962 and July 1963; the results of their examination are given in Table 7. Rinderpest antibodies were distributed in a manner comparable to that already found in the main Serengeti migrant population (Table 3), but no serological conversion had occurred in calves of the 1962 season.

Titration of antibody in wildebeest sera

The titre of neutralizing antibody in groups of adult ($\geqslant$ 3 years old), 2-year-old and yearling wildebeest was determined in order to find if there was a decline with age. As shown in Table 8, yearlings of the 1960 calving season did have a somewhat higher mean titre than older animals, but the variance was very high and the difference was not significant ($t = 0{\cdot}39$; $P > 0{\cdot}5$). If, however, the titres of sixteen yearling sera collected in January 1962 ($1{\cdot}73 \pm 0{\cdot}77$) were compared with eighteen obtained in February to April 1962 inclusive ($0{\cdot}72 \pm 0{\cdot}40$), then there was a highly significant difference ($t = 3{\cdot}99$, $P < 0{\cdot}001$). This, together with the high mean titre ($\geqslant$ 2·38) recorded in 5 yearlings recently recovered from clinically-apparent infection in 1960, suggests that in the majority of animals at least antibody titres may fall rapidly after infection to levels such as those recorded in 2-year-old animals.

Table 7. *Rinderpest antibody in Tanganyika wildebeest; Kirawira/Ikoma 1963*

Year of birth	No. positive/ no. tested	Positive %
$\leqslant$ 1959	9/12	75
1960	3/3	100
1961	2/4	50
1962	0/13	Nil
Total	32	

Table 8. *The titre of rinderpest-neutralizing antibody in wildebeest sera*

Age group	No. of samples	Mean titre* and standard deviation	Range	Remarks
Yearling	34	$1{\cdot}19 \pm 0{\cdot}781$	0·1–2·8	All from Ol Balbal in Jan.–Apr. 1961
2 years	23	$0{\cdot}96 \pm 0{\cdot}513$	0·1–1·8	—
$\geqslant$ 3 years	67	$0{\cdot}92 \pm 0{\cdot}513$	0·1–2·6	—

* Reciprocal of $\log_{10}$ SN 50 dilution against $10^{1{\cdot}8}$ to $10^{2{\cdot}6}$ TCD 50 of virus.

(4). *The transfer of rinderpest-neutralizing antibody from wildebeest cows to their calves*

In the case of Ol Balbal and Ngorongoro wildebeest it has already been shown that at least 88 or 100 % respectively of the adults were immune and some animals negative in screening tests may well have had very small quantities of antibody which could have been excreted at a higher titre in the colostrum, as occurs in other ruminants (Brambell, Hemmings & Henderson, 1951; Brown, 1958). It was to be expected, therefore, that at least about 90 % of wildebeest calves which suckled in the first 24 hr. of life would possess rinderpest-neutralizing antibody. Only two foetal sera were examined and, although the dams were positive, no neutralizing activity could be detected.

To investigate this aspect of rinderpest epizootiology in wildebeest, titrations were carried out on fifty pairs of sera obtained from dams and their offspring during the first 5 months of life; in addition, twenty-five other sera were examined

from isolated calves between the ages of 3 weeks and 7 months. Of these twenty-five isolated calves, seven which were 10 weeks of age or less all had antibody and were presumed to have come from serologically-positive dams. Above this age about 10 % of negative calves could theoretically have been born to mothers without antibody.

As shown in Table 9 all of twenty-four calves which were 6–8 weeks old did have neutralizing antibody but four estimated to be $\leqslant 1$ week and two more thought to be 2 weeks old were completely negative. These six animals were referred to in detail in another paper dealing *inter alia* with the colostral transfer in wildebeest of antibody against M.C.F. virus (Plowright, 1967); this antibody also, though present to high titre in the dams (SN 50 $\geqslant$ 1·6), failed to be transferred to the same six calves. Since the failure of maternal antibody to pass to these animals was thought to be possibly associated with abnormal factors such as calf disturbance due to shooting in the herds or severe stress factors in the dams (Plowright, 1967), data for them have not been included in calculating the figures given in columns 3, 4 and 5 of Table 9.

Table 9. *The acquisition and decline of maternally-derived rinderpest antibody in wildebeest calves*

Age of calf (weeks)	No. with antibody	Mean titre*	Mean titre† of dams	No. of calves with titre $\geqslant$ dam
1	3/7	1·47‡	0·93 (3)§	3/3‡
2–4	5/7	1·32‡	0·87 (3)§	2/3‡
6	7/7	1·26	1·00 (6)	6/6
8	17/17	0·98	1·09 (11)	9/11
10	6/8	0·76	1·03 (8)	3/8
12–14	8/12	0·39	1·01 (8)	0/8
16	4/7	0·46	0·70 (4)	1/4
20	4/5	0·24	— (1)	0/1
24	0/2	0·0	—	—
28	0/3	0·0	—	—

* Titres expressed as reciprocal of log $_{10}$ SN 50 dilution.
† Figures in brackets refer to numbers on which calculations are based.
‡ Omitting calves in which colostral transfer of antibody did not take place.
§ Omitting dams whose calves did not acquire any maternal antibody.

The mean figures given in Table 9 show that during the first month of life the titre of antibody in the young was about 0·5 log units or 3 times higher than that in their dams; by the 6th week the mean titre in the calves was still about twice that of the cows but in the 8th week it had fallen below the maternal level and thereafter decreased rapidly. Individual calves, as shown in column 5 of Table 9, nearly always had as much or more antibody than their dams during the first 6 weeks, but by the end of the 3rd month the titre in the calf was almost constantly exceeded by that of its mother.

Some calves which were presumed to have received antibody via the colostrum in the normal manner were becoming serologically negative by the 10th week of

life and by the 6th and 7th months all passively-acquired antibody had disappeared. The rate of decline of colostral antibody in wildebeest was calculated from the regression of mean titres in column 3 of Table 9 and the half-life was found to be 4·4 weeks. Since the maximum titre recorded in any calf was 2·0 it was estimated on this basis that all passively-acquired rinderpest antibody in wildebeest would have passed its extinction point in 29 weeks or about 7 months, which corresponds very well with the observed figures. It also means that wildebeest calves are very largely susceptible to rinderpest by September of the year of their birth.

(5). *The distribution of serologically-positive animals in other species*

Eland. The results for sera of forty-seven obtained in the Ngorongoro Conservation area are given in Table 10.

It was apparent that eland suffered widespread infection with rinderpest virus before May and June 1962 when the majority of samples were collected in that year. This was hardly surprising in the case of the older animals, since sick eland

Table 10. *The distribution of serologically-positive eland in the Ngorongoro Conservation Area*

Year of birth	Year of sampling 1962	1963	Totals	Positive %
⩽ 1958	9/9	5/6	14/15	93
1959	3/6	1/2	4/8	50
1960	6/8	1/2	7/10	70
1961	1/2*	1/6	1/6	16
1962	—	1/6*	—	—
Totals	25	22	—	—

* Probably passively-acquired antibody in animals 6 months and 4 months old respectively. These are excluded from totals in column 4.

were observed on the Ol Balbal Plains in 1958 and a strain of rinderpest virus was isolated from one affected bull (Robson, Arnold, Plowright & Scott, 1959). In the case of eland calves born in 1960, they exhibited serological conversion in approximately the same proportion as wildebeest sampled at Ol Balbal or in the Ngorongoro Crater. The eland born in 1961, however, showed a low incidence of past infection, comparable to that in the Ngorongoro wildebeest calves of the same year and, so far as can be judged from small samples, much less than that in migrant Serengeti wildebeest born in early 1961.

Titrations were performed on all positive eland sera and in twenty-three animals which, by virtue of their age, were considered to have been actively immunized the mean log titre was 1·32 with a standard deviation of 0·48 and range of 0·8–2·2. This was somewhat higher than the level in wildebeest (Table 8).

Thompson's and Grant's gazelle, 1962/63. The results for sera from animals of these two species are given in Table 11. All were obtained in the Ngorongoro Conservation area, either in the Crater or on the Ol Balbal Plains. A low rate of infection had apparently occurred in Thompson's gazelle with some serological

conversion probably taking place in the latter half of 1961 or early 1962. There was no evidence of past infection in Grant's gazelle.

The titre of antibody in serologically-positive Thompson's gazelle was probably lower than that in wildebeest; thus in five animals the titre against $10^{2\cdot6}$ TCD 50 of virus varied from a trace to 1·0, the mean log SN 50 being 0·66.

Of ten samples from *G. thompsoni* obtained in the Naivasha area of Kenya, six were from adults but none had demonstrable neutralizing antibody.

Table 11. *Rinderpest antibody in gazelles of the Ngorongoro Conservation Area 1962/63*

	No. with antibody in age group					
Species	1–4 months	5–8 months	9–18 months	2 years	3 years or more	Totals
Thompson's* gazelle	0/2	0/11	2/19	3/23	—	55
Grant's gazelle	—	0/7	0/14	0/18		39

* All obtained in 1962.

Coke's hartebeest and topi. Only four serum samples were examined from hartebeest, these being from animals 1, 2 or 3 years old. All were obtained in the Ngorongoro Conservation area and were negatve in screening tests. Two sera from 3-year-old topi at Ikoma were also negative.

DISCUSSION

The wildebeest has a particular significance in the epizootiology of rinderpest in East Africa, primarily because it is present in very large numbers, the majority of which form big aggregations and migrate regularly over long distances in pursuit of grazing or water. The total number of wildebeest in the Serengeti, Mara and Loita Plains was estimated to be about 220,000 in May 1961 (Stewart & Talbot, 1962), whilst in May 1963 a photographic count revealed over 300,000 head on the Central Serengeti Plains alone (Owen, 1964). Such a prolific, gregarious and mobile species affords excellent opportunities for the maintenance and dissemination of a virus like that of rinderpest.

The Annual Reports of the Veterinary and Game Preservation Departments of Tanganyika reveal the following historical background for rinderpest infection of wildebeest. The disease was rumoured to be present in the western Serengeti in October 1930 (Hornby, 1931), but it was not until March 1933 that the disease in wildebeest was confirmed for the first time by subinoculation into cattle of material from 2-year-old wildebeest found near the Ngorongoro Crater; rinderpest infection of a mild type was simultaneously observed in cattle and sick eland were seen later (Cornell, 1934).

Even at this time rinderpest infection in game animals was reported to be an *annual* occurrence near the Ngorongoro Crater but it was also asserted that it did 'not appear to do much harm' (Teare, 1935). Nevertheless, a considerable rinder-

pest mortality in 7–8 months old wildebeest was reported in the Serengeti area in 1935 (Teare, 1936), thus pointing to previous immunization of older age groups in the migratory population.

During the rest of the prewar and war period (1936–45) the available reports do not make possible any reconstruction of the annual disease events in the wildebeest or other specific populations of the Serengeti and adjacent areas. However, deaths in *young* wildebeest were reported in the Musoma district and Northern Province in late 1946 (Reid, 1947), and outbreaks were recorded in 1947 affecting wildebeest and giraffe in the Maswa area (Moore, 1948). The disease in wildebeest was confirmed on the northern range in 1948 (Moore, 1949) and in the Serengeti in November and December 1949 (Burns, 1950); it was suspected in the Serengeti in 1951 (Swynnerton, 1953), confirmed in the Mara area in 1953 (Swynnerton, 1954) and suspected again in the Serengeti in 1954 (Dawe, 1955). In the last year it was confirmed in wildebeest, buffalo and impala to the west of the Serengeti, without apparently gaining access to the park (Swynnerton, 1955). Although no rinderpest was confirmed in Tanganyika game animals in 1955/56 (Swynnerton, 1957), in October 1957 large numbers of wildebeest, 1–2 years old, died in the Musoma and Maswa districts, the disease disappearing by the end of the year (Swynnerton, 1958; Branagan & Hammond, 1965).

In early 1958, rinderpest was again confirmed in yearling wildebeest in Musoma and North Mara to the west of the Serengeti, with no apparent spread to other species; at the same time in the Crater Highlands the disease affected warthogs, buffalo and eland in addition to wildebeest but it had died out by July (Swynnerton, 1959). In 1959, rinderpest was thought to have been present in the Mara area at the beginning of the year, sick animals were seen in the south-east Serengeti during March and a heavy mortality occurred in yearling wildebeest in the northern part of the Ngorongoro Conservation area in December (Thomas, 1960). Only minor outbreaks of rinderpest in unspecified game animals were recorded in 1960 near the Ngorongoro Crater and in wildebeest calves to the west of the Serengeti (Kinloch, 1961). As already noted, the only serious outbreak of rinderpest reported in 1961 by the Game Department, Tanganyika, was one affecting yearling buffalo around the Ngorongoro Crater in October and December (Kinloch, 1963).

In summary of the more recent events it may be said that clinical rinderpest was reported annually in the main wildebeest concentrations of Tanganyika from 1957 to 1960 inclusive, and that it persisted in an easily recognizable form in buffalo until at least 1961. Before 1957, the infrequency of reports cannot be said to indicate the absence of infection for prolonged periods; in fact deaths largely among yearling and 2-year-old wildebeest were a sure indication that widespread infection must have taken place every 1 or 2 years. Talbot & Talbot (1961) referred to a comparable *annual* mortality in yearling wildebeest of the Mara region of Kenya; this began in October or November and faded out during January, by which time 40 % of the total initial calf crop were said to have succumbed to the disease.

The serological results reported in this paper confirm that widespread infection

of yearling wildebeest with rinderpest virus occurred in the Mara–Loita Plains area of Kenya during 1959/60 and on the Serengeti Plains during late 1960. About 76 % of the yearlings in the Serengeti migrant population of late 1960 had probably been infected, as well as a similar proportion (66 %) in the Ngorongoro resident herds. By December 1961 about two-thirds (67 %) of the calves born early in that year to dams of the migrant population had become immunized, but the proportion in the Ngorongoro Crater herds was much lower (11·5 %). The serological conversion in this year was peculiar in that it had not been accompanied by any signs of disease or mortality.

About 29 % of the calves born early in 1962 showed evidence of past infection by mid-1963 on the Ol Balbal Plains (Table 4), but some conversion had probably taken place here by August 1962 (Table 3). No signs of disease were detected in the wildebeest population during 1962 and hence, for the second year, rinderpest infection occurred silently.

The significance of small numbers of apparently silent infections needs to be considered in the light of their undoubted occurrence in the Kajiado area of Kenya (Table 1). They were shown to have occurred there both before and since 1960, affecting no more than about 15 % of the total population; in this district no disease or mortality has been reported in wildebeest in recent years, but mild strains of rinderpest were isolated from cattle in several localities of Kenya Masailand in early 1961 (Plowright, 1963*b*), and the Kajiado wildebeest probably had opportunities to contact such sick cattle. In a similar way Masai cattle, though excluded from the Serengeti Park, share the grazing of the whole of the Ngorongoro Conservation area with the game population and this contact may have been responsible for the infections detected in Ol Balbal and possibly Ngorongoro wildebeest born in 1962. Additional evidence that these infections did, in fact, occur on a significant scale at Ol Balbal was obtained by sampling carried out in 1966 and reported in a companion paper (Taylor & Watson, 1967, personal communication).

The involvement of only a small part of a susceptible population may have a number of implications. For example, between-animal contacts may not be sufficiently close or frequent to ensure adequate exposure of a large proportion of them; with a virus which is probably spread largely by aerosols this could well be true, especially in a tropical country where inactivation of a highly labile agent by desiccation and ultraviolet light would be expected to occur very rapidly. Secondly, the strains of virus involved may not be very invasive for wildebeest or other game species; a lack of invasiveness is well recognized for some strains in experimental cattle (Cooper, 1932; Taylor *et al.* 1965). Invasiveness may not be related to virulence, since it appeared that about two-thirds of the Ol Balbal yearlings suffered infection with a non-virulent strain in 1961; the low rate of infection amongst 1961 calves in the Ngorongoro Crater (Table 5) suggested that the strain of virus which was epizootic in the population outside may not have gained access to this relatively isolated group and that the few serological positives could have been infected during seasonal migration to the Ol Balbal Plains.

Our results for eland sera showed that this species behaved very similarly to

wildebeest in that infection was widespread up to 1960/61 but has since become less frequent. However, the total population of this species in the Serengeti area is very much smaller (6400–9600 according to Stewart & Talbot, 1962) and even if between-herd contacts were adequate, it could hardly maintain rinderpest virus for any prolonged period. Infection of Thompson's gazelle was not infrequent, unless the small number of samples examined gave an inaccurate indication of the rate of infection in the whole of the Serengeti population; assuming that the annual infection rate was approximately the same as that found in the 9–18 months age group—about 10·5 %—then as many as 50,000 infections could have occurred in a year, if we accept the lower census figure of Stewart & Talbot (1962). By this reckoning Thompson's gazelle could contribute materially to the maintenance of the virus.

The decline in the rate of rinderpest infection in game animals in the Serengeti, Ngorongoro and contiguous areas during the years 1962/63 and the continuing absence of widespread infection up to 1967 (Taylor & Watson, personal communication) is no reason for assuming that new and catastrophic invasions of the disease could not occur again. It appears that, for the first time since 1930, no strain of rinderpest virus is present which spreads readily in the most abundant and susceptible species; this is probably due in large measure to the efforts of the Veterinary Departments, which since 1950 have immunized as many as possible of the susceptible cattle in areas where cattle-game contacts are important (Branagan & Hammond, 1965). During and before 1961, in spite of prolonged efforts to immunize all susceptible cattle, it must be concluded that strains of virus persisted which spread readily in susceptible wildebeest, eland and probably also buffaloes and other species. These wild animals, in fact, may have been largely responsible for the maintenance of the virus at that time, whereas in recent years they have played a restricted role. If another strain of rinderpest virus, highly invasive for the predominant species, were to be introduced from outside, or were to emerge, following mutation or otherwise, from an existing undetected focus, then the results could hardly be less than spectacular.

SUMMARY

The incidence of rinderpest infection in game animals in selected localities of South Kenya and North Tanganyika was studied during the period 1960 to 1963. Serum samples from 590 wildebeest (*Connochaetes taurinus*), 48 eland (*Taurotragus oryx*), 65 Thompson's gazelle (*Gazella thompsoni*) and 39 Grant's gazelle (*Gazella granti*) were tested for rinderpest neutralizing antibody.

Rinderpest infection was shown to have been very frequent in yearling wildebeest in the Mara area of Kenya in 1959/60, in the Serengeti National Park of Tanganyika in late 1960 and also in the Serengeti, and some adjacent areas, during the latter half of 1961. In the Ngorongoro Crater in 1961 infection was far less widespread, with only 11 % of the yearlings acquiring antibody, compared to 67 % in the Serengeti. The infections in 1959 and 1960 were clinical epizootics, accompanied by a considerable mortality, whereas no overt disease was reported in the course of 1961. Eland were affected in a similar manner to wildebeest up to

1960 but only a low rate of serological conversion was demonstrated in 1961. Adult Thompson's gazelle showed a low rate (*ca.* 12 %) of infection but no antibody was detected in Grant's gazelle.

Only a small proportion of the wildebeest calves born in early 1962 acquired antibody by mid-1963 and this was due, at least in part, to infection late in 1962; it was not clear, unfortunately, whether the positive animals belonged entirely to resident, as opposed to migratory, groups. No clinical signs or mortality were reported in this year.

A low incidence of rinderpest infection in wildebeest was also demonstrated both before and after 1960 in the Kajiado district of Kenya, where disease of game has not been reported in recent years. It is possible that the positive animals, as also the 1962 cases in Tanganyika, acquired the virus from low-grade infections of cattle.

The transmission of rinderpest antibody from wildebeest dam to calf, presumably via the colostrum, was demonstrated regularly, except in six calves about 1–2 weeks old. No completely satisfactory explanation was obtained for their failure to acquire passive antibody but it may have been due to abnormal disturbance in the herds, associated with the shooting. The antibody titres in calves were initially higher than those in the serum of their dams but by the end of the 3rd month this position had been reversed. Individual calves became serologically negative from about the 10th week of life and all were devoid of antibody by the 6th to 7th month. The half-life of passively-acquired antibody was 4·4 weeks.

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