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# Studies on the epizootiology of rinderpest in blue wildebeest and other game species of Northern Tanzania and Southern Kenya, 1965–7

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Detailed serological evidence presented by Plowright & McCulloch (1967) showed that extensive infections of rinderpest occurred in various populations of blue wildebeest (*Connochaetes taurinus albojubatus* Thomas, syn. *Gorgon taurinus taurinus* Burchell) from N. Tanzania and S. Kenya over the period 1959–62. However, sera from young wildebeest born and collected in 1963 lacked antibodies attributable to rinderpest infection and it appeared possible that epizootics of the disease had ceased.

Work of this nature lapsed during the whole of 1964, but in January 1965 it became possible to resume the collection of wildebeest material. Attempts were made to re-establish the age distribution of animals with rinderpest antibody in the populations previously examined. Although there had been no report of rinderpest in either cattle or game from the area under consideration during 1964, in view of the occurrence of subclinical rinderpest in wildebeest (Plowright, 1963) it was considered necessary to examine animals of all ages.

In December 1965 and March 1966 field outbreaks of bovine rinderpest were confirmed in N. Tanzania from a district where rapid extension to game species was a distinct possibility. Considerable interest attached to the origin of these outbreaks as they brought to a close a 3-year period (1963–5) during which no rinderpest had been detected in the whole of Tanzania. The scope of the present study was therefore enlarged in an attempt to gather evidence of a prolonged maintenance role by local game.

## MATERIALS AND METHODS

### *Sera*

Free-flowing blood was usually obtained from the severed jugular vessels. Serum was separated within 24–72 hr. of shooting, containers in the meantime having been kept as cool as possible. Samples were stored at $-20°$ C., and, where necessary, were transported rapidly on ice.

### *Neutralization tests*

Samples of undiluted sera, previously inactivated at 56° C. for 30 min., were screened for rinderpest-neutralizing antibody in a 5-tube test using $10^{1·2}$–$10^{2·8}$

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TCID 50 of culture-attenuated virus per tube (Plowright, 1962). Samples in which neutralizing activity could be detected were subsequently titrated against a virus dose of $10^{1.8}$–$10^{2.6}$ TCID 50 per tube. Details of the methods and controls employed have been described by Plowright & Ferris (1961) and Plowright (1962). $Log_{10}$ SN 50 titres were calculated by the method of Thompson (1947).

### *Ageing*

Watson (1967) described a reliable method for determining the age of wildebeest based on the number of erupted incisor and cheek teeth, or the amount of wear in full-mouthed animals. Alternatively, females may be aged by counting scars left by corpora lutea of pregnancy. Due to regular annual conception by mature females a good correlation has been found between the two methods; scar counting, however, will underestimate the age of an abnormal individual which fails to conceive in any year of adult life. It was only necessary to use this alternative method of ageing for a small proportion of Serengeti migrant wildebeest shot in 1965.

Species other than wildebeest were aged from a general consideration of dentition, body weight and body measurements, bearing in mind the strictly seasonal nature of the reproductive cycle.

### *Animal populations*

With the exception of the Kajiado group, sera were collected from each of the wildebeest populations described by Plowright & McCulloch (1967). Figure 1 of these authors outlines the topography of the area where the current work was undertaken and the wildebeest populations which were sampled.

In connexion with the 1965/6 field outbreaks of rinderpest it was necessary to collect sera from buffalo, eland, impala, warthog, and resident wildebeest within the Loliondo district of N. Tanzania. The distribution of these animals has not been previously mentioned.

Loliondo resident wildebeest, numbering some 5000 animals, have a wet season range immediately to the south of Loliondo township, and in the dry season disperse into the Loita Hills. Additional small groups are to be found during the wet season in the Loita Hills and eastwards towards Lake Natron. Between December and June the resident population can be greatly augmented by Serengeti migratory animals occupying the northern extremity of their wet season range.

The other species mentioned form loose aggregations of herds or family groups throughout the area.

## RESULTS

### *Serengeti migrant wildebeest*

A total of 149 sera were examined between January 1965 and April 1966 (Table 1).

Antibodies were found in four samples from 1- to 4-month-old calves born in early 1965 or 1966, but none could be detected in sera of calves born in January 1965 and aged 5–11 months when shot. In three instances it was possible to demonstrate antibodies in the sera of both dam and offspring. It was inferred from

these results that passive transfer of protection took place between cows possessing antibody and their calves, and that this protection was short-lived.

No evidence of active antibody production could be demonstrated in any of 60 samples from animals born since 1963. In contrast rinderpest-neutralizing antibodies were present in 59 of 73 samples (81 %) from animals born before 1962. It seems probable that the last major rinderpest infection of this population occurred in 1961, as approximately 64 % of the animals born in January of that year experienced the disease compared with only 6 % of animals born in 1962. The single positive sample from 1962 was aged by the scar-counting technique.

Table 1. *Distribution of rinderpest antibody in Serengeti migrant wildebeest*, 1965/6

| | Year of sampling | | | |
|---|---|---|---|---|
| Year of birth | 1965 | 1966 | Totals | % positive |
| In or before 1959 | 37/45* | 2/2 | 39/47 | 83·0 |
| 1960 | 12/14 | 1/1 | 13/15 | 86·6 |
| 1961 | 7/9 | 0/2 | 7/11 | 63·6 |
| 1962 | 1/12 | 0/4 | 1/16 | 6·2 |
| 1963 | 0/18 | 0/2 | 0/20 | 0·0 |
| 1964 | 0/5 | 0/8 | 0/13 | 0·0 |
| 1965 | 3/13† | 0/12 | 3/25 | 0·0‡ |
| 1966 | | 1/2† | 1/2 | |
| Totals | 116 | 33 | 149 | |

* No. positive/no. tested.
† Calves aged 1–4 months old; antibody presumed to be passively acquired.
‡ Percentage derived from 1966 sample.

Table 2. *Distribution of rinderpest antibody in the Kirawira, Mara and Ngorongoro resident wildebeest populations*

| | | Year of birth | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Population | Collection date | 1960 | 1961 | 1962 | 1963 | 1964 | 1965 | 1966 | Totals |
| Kirawira | Mar. 1966 | 4/9* | 0/3 | 0/4 | 0/2 | 0/7 | 0/5 | 1/3† | 33 |
| Kirawira | Feb. 1967 | 0/1 | 1/1 | — | 0/1 | 0/1 | 0/4 | 0/19 | 27 |
| Mara | Jan. to Mar. 1966 | 10/12 | 3/4 | 2/13 | 0/11 | 0/13 | 0/7 | 1/3† | 63 |
| Ngorongoro | Apr. 1966 | 9/10 | 1/1 | 4/6 | 0/1 | 0/6 | 0/5 | 1/1† | 30 |

* No. positive/no. tested.
† Calves aged 2–4 months old; antibody presumed to be passively acquired.

*Resident wildebeest populations*

The distribution of rinderpest-neutralizing antibody in three non-migratory populations is presented in Table 2. Antibodies found in young calves were in each instance considered to have been passively acquired.

No evidence of recent infection in animals of the Kirawira resident group could be found among samples collected in March 1966 and February 1967; the last outbreak in this population apparently took place in 1961.

Mara residents were sampled in the first 3 months of 1966. Some 81 % of the animals born in early 1960 and 1961 possessed antibody, together with a much smaller proportion (*ca.* 15 %) of animals born in early 1962. There was no indication of infection in any subsequent year.

Antibodies were found in resident wildebeest from the Ngorongoro crater born in 1960, 1961 and 1962, but animals born since January 1963 have remained susceptible. All samples from this population were taken in April 1966.

### *Field rinderpest*

In November 1965 material submitted from cattle in the Loliondo district of N. Tanzania was found to contain rinderpest virus (W. P. Taylor, unpublished). Reports indicated that the disease affected young stock only without causing mortality. Resident wildebeest and other rinderpest-susceptible ungulates (Scott, 1964) were to be found in close proximity to the primary cattle outbreak, and moreover the disease focus was situated on the eastern edge of the wet season range of the Serengeti migrant population. By this time it was known that this highly mobile group of animals, then some 50 miles to the south-east, contained numerous susceptible individuals.

Table 3. *Distribution of rinderpest antibody in various game species from Loliondo District, N. Tanzania, December* 1965 *to January* 1966

| Species | Collection date | Year of birth ⩽1960 | 1961 | 1962 | 1963 | 1964 | 1965 | Totals |
|---|---|---|---|---|---|---|---|---|
| Resident wildebeest | Dec. 1965 | 4/6* | — | 0/2 | 1/2 | 1/7 | 0/5 | 22 |
| Buffalo | Jan. 1966 | 6/7 | — | 0/1 | 1/1 | 0/1 | — | 10 |
| Eland | Dec. 1965 | 0/4 | — | 1/3 | 0/2 | 0/4 | 0/7 | 20 |
| Impala | Dec. 1965 | 0/6 | — | 0/1 | 0/2 | — | 0/7 | 16 |
| Warthog | Dec. 1965 | — | — | — | 0/1 | — | 0/1 | 2 |

* No. positive/no. tested.

Sera were obtained from buffalo, eland, impala, warthog, and Loliondo resident wildebeest some 3–6 weeks after the disease was first diagnosed. Results of examination for rinderpest-neutralizing antibody are given in Table 3. It was apparent that during this interval there had been no rapid extension of rinderpest to contiguous game populations. Of the 70 sera examined positive samples were predominantly among buffalo and wildebeest born in or before 1960. These animals were probably infected early in life as there was no evidence of widespread infection between 1962 and 1965 in any of the species from which samples were taken. In this connexion it should be noted that buffalo infected with rinderpest were reported close to Loliondo district in 1959 (Thomas, 1960) and 1960 (Kinloch, 1961). The single positive eland born in 1962 could also have encountered the disease while young, as it has been demonstrated that rinderpest existed that year, albeit in wildebeest, some 70 miles south at Ngorongoro (Plowright & McCulloch, 1967).

Neutralizing antibody was found in the serum from 1 of 2 wildebeest born in 1963 and from 1 of 7 born in 1964 (Table 3). At the time it was impossible to decide whether these animals had been infected by an extension of the cattle outbreak into wildebeest, or whether their antibody had been acquired at a much earlier stage in life. The same problem was posed by the findings of antibody in a 2-year-old buffalo.

In March 1966 a fresh focus of rinderpest was confirmed, still within cattle of the Loliondo district, but in an area where both Serengeti migrant wildebeest and local resident animals were now concentrated. Additional samples were obtained from both populations some three weeks after this second outbreak was detected. As the migrants had moved about 20 miles south there was no difficulty in differentiating the two types of wildebeest. The distribution of antibodies in the 33 migrant wildebeest collected has already been described (see Table 1, results for 1966), while results for the further 21 resident wildebeest are given in Table 4. There were no indications that the bovine field strain of virus had spread to either population: of the 54 samples the only antibodies found were in the sera of three old Serengeti migrant animals, a 3-month-old calf from this group and a 6-year-old resident animal.

Table 4. *Distribution of rinderpest antibody in Loliondo resident wildebeest, April* 1966

| Year of birth | | | | | | |
|---|---|---|---|---|---|---|
| 1960 | 1961 | 1962 | 1963 | 1964 | 1965 | Total |
| 1/1 | — | 0/2 | — | 0/9 | 0/9 | 21 |

Table 5. *Titration results for rinderpest neutralizing antibody in sera from Serengeti migrant wildebeest*

| Age group* (years) | No. of samples | Mean titre and standard deviation | Range | *t* Test† |
|---|---|---|---|---|
| > 12 | 7 | 0·86 ± 0·25 | 0·6–1·4 | $t = 1{\cdot}989$, $p > 0{\cdot}05; < 0{\cdot}1$ |
| 10–12 | 6 | 1·12 ± 0·37 | 0·8–1·6 | $t = 1{\cdot}248$, $p > 0{\cdot}2; < 0{\cdot}4$ |
| 7–9 | 14 | 0·97 ± 0·59 | 0·4–2·2 | $t = 0{\cdot}5517$, $p > 0{\cdot}5$ |
| 6 | 9 | 1·11 ± 0·38 | 0·8–1·6 | $t = 1{\cdot}354$, $p > 0{\cdot}1; < 0{\cdot}2$ |
| 5 | 8 | 0·85 ± 0·26 | 0·6–1·4 | $t = 0{\cdot}1122$, $p > 0{\cdot}5$ |
| 4 | 7 | 0·83 ± 0·54 | 0·4–1·8 | — |

* All samples collected during 1965.
† Mean titre for each age group compared to that of 4-year-old animals

The antibodies detected in two young wildebeest in the earlier collection of Loliondo resident samples apparently did not represent the commencement of a fresh wildebeest epizootic. It was concluded that these antibodies were acquired before November 1965 and that they were present in only a small proportion of the resident population.

*Titration results*

Table 5 shows the results of neutralizing-antibody titrations carried out with sera from different age groups of Serengeti migrant wildebeest. When mean titres were compared it was found that antibody levels did not decline with age; however, older animals were undoubtedly exposed to rinderpest virus on more than one occasion.

Animals of the 4-year age group were probably infected in late 1961 (Plowright & McCulloch, 1967). Taking the date of first antibody appearance as January 1962, demonstrable antibodies had persisted for $3\frac{1}{2}$–4 years in the absence of re-infection. Moreover, antibody levels were not significantly lower ($t = 1{\cdot}17$, $P < 0{\cdot}4$, $> 0{\cdot}2$) when compared to figures obtained from the same group of animals some 3 years earlier (Plowright & McCulloch, 1967).

## DISCUSSION

The present survey has demonstrated quite clearly that each of the numerically important wildebeest populations of the Serengeti-Mara region, in S. Kenya and N. Tanzania, has been free of rinderpest infection over the last 3 or 4 years. In reviewing the history of rinderpest in wildebeest of this area Plowright & McCulloch (1967) were able to find evidence of continuously recurring infection in the period between 1947 and 1960, while their own results provided evidence for continuing infection up to 1962. However, the recovery of strains of the virus from cattle in N. Tanzania as recently as March 1966 indicates the continuing risk of further disease in these great herds. From the data of Watson (1967) it is possible to calculate that in the Serengeti migrant population there are at present some 200,000 rinderpest-susceptible individuals, amounting to over 60% of the total. It may be assumed that a similar proportion of each resident population in this area is also at risk. Thus a fresh epizootic of virulent rinderpest in any of these groups could well cause extremely severe losses with the attendant danger of widespread physical dissemination of the virus, and spread to other species.

When the 1965 and 1966 isolations of rinderpest virus were made in the Loliondo area the only other known focus of the disease in East Africa was some 200 miles to the north, in the Isiolo district of Kenya. Strains derived from the Kenya outbreak caused severe clinical signs and some mortality in experimental cattle, whereas the Loliondo strains produced a relatively mild syndrome and no deaths (W. P. Taylor, unpublished). Aside from any difference in strain characteristics it was most unlikely, if not impossible, for infection to have spread between the two localities. Mild rinderpest was reported from the Loliondo district up to the end of 1960 (Roe, 1962) and was still present in 1961 at which time it was reputed to be difficult to detect and to cause only mild symptoms in calves (Branagan & Hammond, 1965). Although a thorough rinderpest vaccination programme was carried out in this district in 1964 (D. Branagan & J. A. Hammond, personal communication) it is suggested that the recent outbreaks were evidence of an enzootic infection that had persisted since 1961 in an undetected form in this isolated part of N. Tanzania.

On this basis an explanation can be given for the findings of antibody in the sera of 2- and 3-year-old wildebeest from the Loliondo resident population. Only two such individuals were detected in 36 samples (Tables 3, 4) taken from animals born between 1962 and 1965, so apparently no widespread infection occurred within the population during this period. However, if small groups of animals had encountered infection, possibly when the population was dispersed during a dry season, it would then be possible to sample aggregations during the wet season and find a small number of animals with neutralizing antibody; this is in fact what was done.

At the time of the second field outbreak large numbers of resident and migrant wildebeest were grazing land from which infected stock had recently been withdrawn. Moreover, sick cattle, although in quarantine, remained localized within the territory occupied by wildebeest (P. Jenkins, personal communication). It was therefore somewhat surprising to find that transmission to wildebeest had not occurred. Until further sera can be obtained from young buffalo the epizootiological role of this species in the Loliondo area will remain uncertain.

In general our survey results confirmed and extended the findings of Plowright & McCulloch (1967). From material collected in 1963 they were able to show that Kirawira resident wildebeest had been infected in 1961 but not in 1962. It is now apparent that no rinderpest infection has occurred in this group of animals in the 5-year period 1962 to 1967.

Plowright & McCulloch (1967) found that wildebeest of the Mara group suffered infection during 1959 but not within the first 8 months of 1960. The present results show that this population was subjected to at least one further rinderpest episode, as animals born in early 1960 and 1961 were found to possess actively acquired antibody. Two of 13 animals aged 4 years at the time of shooting had also experienced the disease. Although both these animals were ascribed to the 1962 calf crop, calving in this population is actually spread over a 2–3 months period between December one year and February of the following year (Watson, 1967). It is suggested that these individuals were born and infected in late 1961 when rinderpest was concurrently infecting yearling animals born earlier the same year. It is of course possible that 1961 yearlings, together with small numbers of very young calves, were infected in the first months of 1962; however, the former interpretation gains support from a report of rinderpest in yearling wildebeest of this population in 1961 (Anon, 1962). Maternally derived immunity would probably have protected the majority of the 1962 calf crop for the first 4–6 months of life (Plowright & McCulloch, 1967), so had the outbreak occurred after the loss of this protection a higher proportion of animals with antibody could reasonably have been expected.

In August 1962 Plowright & McCulloch (1967) found antibodies in the sera of three 9-month-old calves in the Ngorongoro Crater, and in two 8-month-old calves from the same population collected on the Ol Balbal Plains; these antibodies were considered to have been actively acquired. When a further series of samples was collected from the crater in 1963 they were unable to repeat their previous finding yet we were able to show that some 67 % of Ngorongoro wildebeest born in 1962 had experienced the disease (Table 2). The coincidence of our result with the

former result of Plowright & McCulloch (1967) clearly established that this group of animals was last infected in 1962.

There is a remarkable agreement between the present survey and that of Plowright & McCulloch (1967) with respect to the percentage of animals possessing neutralizing antibody in the 1959, 1960, and 1961 calf crops of Serengeti migrant wildebeest. While they were able to detect discrete epizootics in 1960 and 1961 they were uncertain whether or not small numbers of these wildebeest were infected in 1962. In the current work there was no evidence of widespread infection in animals of the 1962 calf crop, and in view of the method used to age the donor of the single positive sample it is entirely possible that no infection occurred during 1962 in the migrant group.

Throughout this report it has been assumed that antibody found in the serum of 1- to 4-month-old calves was passively acquired. This interpretation is in agreement with the work of Plowright & McCulloch (1967) where it was shown that young wildebeest acquired antibodies from their dams within the first few days of life, and that thereafter this antibody decayed by 50 % every 4·4 weeks.

Duration of neutralizing antibody in wildebeest has not been studied hitherto, although Plowright, Laws & Rampton (1964) showed that the hippopotamus (*Hippopotamus amphibius* Linnaeus) retained rinderpest antibody for 18 years following a single infection. The absence of a detectable decline in antibody levels in wildebeest between 1962 and 1965 indicates they may persist for some considerable time to come.

## SUMMARY

A serum neutralization test was used to determine the incidence of rinderpest antibodies in populations of blue wildebeest (*Connochaetes taurinus albojubatus* Thomas) occurring in the Serengeti-Mara districts of N. Tanzania and S. Kenya. By correlating the age of animals at the time of shooting with the presence or absence of antibodies it was possible to outline the course of rinderpest in these animals over the period 1961–7.

Serengeti migrant wildebeest were extensively infected in 1961, but not in 1962 or any subsequent year. No samples have been collected from this group since mid-1966. Kirawira resident wildebeest were last infected in 1961 according to samples collected in February 1967. Two of 13 animals born into the Mara resident population in late 1961 or early 1962 were found to possess antibodies, together with a high proportion (74 %) of animals born in early 1961. This finding was considered indicative of a rinderpest epizootic in this group in the terminal weeks of 1961. No subsequent infection occurred in Mara wildebeest between 1962 and March 1966. Ngorongoro resident wildebeest were infected in the second half of 1962 but have remained free of rinderpest from 1963 until at least April 1966.

The findings of the present survey were compared and contrasted with results of a similar study carried out some 2 to 3 years previously.

Field strains of bovine rinderpest virus were isolated from the Loliondo district of N. Tanzania in November 1965 and March 1966. Serum samples from buffalo, eland, impala, warthog, and resident wildebeest which inhabited areas adjoining

the two outbreaks failed to provide evidence of any recent epizootic in game animals. Circumstantial evidence indicated that the virus could have persisted in the Loliondo area since 1961. Two resident wildebeest born in 1963 and 1964 respectively, were thought to have been infected during a period when the disease went unrecognised. Serengeti migrant wildebeest were not found to have been infected after a period of potential contact with sick cattle in March 1966.

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## REFERENCES

ANON. (1962). *Rep. Game Dep. Kenya*, 1961.

BRANAGAN, D. & HAMMOND, J. A. (1965). Rinderpest in Tanganyika; a review. *Bull. epizoot. Dis. Afr.* **13**, 225–46.

KINLOCH, B. G. (1961). *Rep. Game Dep. Tanganyika*, 1960.

PLOWRIGHT, W. (1962). The application of monolayer tissue culture techniques in rinderpest research. I. Introduction. Use in serological investigations and diagnosis. *Bull. Off. int. Épizoot.* **57**, 1–23.

PLOWRIGHT, W. (1963). The role of game animals in the epizootiology of rinderpest and malignant catarrhal fever in East Africa. *Bull. epizoot. Dis. Afr.* **11**, 149–62.

PLOWRIGHT, W. & FERRIS, R. D. (1961). Studies with rinderpest virus in tissue culture. III. The stability of cultured virus and its use in virus neutralization tests. *Arch. ges. Virusforsch.* **11**, 516–33.

PLOWRIGHT, W. & MCCULLOCH, B. (1967). Investigations on the incidence of rinderpest virus infection in game animals of N. Tanganyika and S. Kenya 1960/63. *J. Hyg., Camb.* **65**, 343–58.

PLOWRIGHT, W., LAWS, R. M. & RAMPTON, C. S. (1964). Serological evidence for the susceptibility of the hippopotamus (*Hippopotamus amphibius* Linnaeus) to natural infection with rinderpest virus. *J. Hyg., Camb.* **62**, 329–36.

ROE, J. E. R. (1962). *Rep. vet. Dep. Tanganyika*, 1960.

SCOTT, G. R. (1964). Rinderpest. *Adv. vet. Sci.* **9**, 113–224.

THOMAS, D. K. (1960). *Rep. Game Dep. Tanganyika*, 1959.

THOMPSON, W. R. (1947). Use of moving averages and interpolation to estimate median effective dose. I. Fundamental formulas, estimation of error and relation to other methods. *Bact. Rev.* **11**, 115–45.

WATSON, R. M. (1967). The population ecology of the wildebeest (*Connochaetes taurinus albojubatus* Thomas) in the Serengeti. Dissertation submitted for the degree of Doctor of Philosophy in the University of Cambridge.