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## "The Ecology and Epidemiology of Rinderpest Virus in Serengeti and Ngorongoro Crater Conservation Area"

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**These are preliminary lecture notes, intended only for distribution to participants.**

# The Ecology and Epidemiology of Rinderpest Virus in Serengeti and Ngorongoro Crater Conservation Area.

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Introduction.

The popular perception of Serengeti is a predator-prey system where ecological interactions are dominated by large herbivores and the carnivores that prey upon them (Grizmek & Grizmek, 1961; Schaller, 1972; Kruuk, 1976). This chapter argues that the population density of wildebeest and buffalo have been regulated for most of this century by rinderpest, a virus disease of wild and domestic ungulates. Removal of this pathogen through the vaccination of cattle has led to the eruption of the wildebeest and an increase in buffalo. This eruption in turn led to increases in the numbers of carnivores, particularly lions and hyaenas (Sinclair, this volume; Packer & Pusey, this volume, Heffer & East this volume). This chapter describes the ecology and epidemiology of rinderpest virus and its control in the Ngorongoro Conservation Region. The analysis suggests that maintaining a high level of rinderpest innoculation in the population of cattle that live in and around the park continues to be a crucial component of successful conservation in the whole Serengeti/Ngorongoro region.

Understanding the population biology of rinderpest in the Serengeti is a classic conservation problem. It involves resolving conflicts of interest between the pastoralists and conservation authorities who have complementary interests in the health of domestic and wild animals. Although the virus has now been eradicated from the ecosystem, its reintroduction from endemic sources outside the region poses a continual threat to

the large herds of wild game living in the Ngorongoro Conservation Area and Serengeti (Plowright 1982, 1985). At least two outbreaks have occurred in the last ten years and these have led to significant localized mortality in buffalo and other game species (Rossiter et al, 1987; Prins & Weyerhauser, 1987; Anderson et al, 1990). The main way to ensure the absence of this virus is to maintain high levels of immunity in the cattle population in the surrounding areas. Inoculating cattle in the NCCA and around Serengeti is expensive and perception of the risk associated with a disease usually declines with increasing time since the last outbreak.

The importance of understanding the population biology of diseases in relation to conservation biology has received recent recognition (Dobson & May 1986; Dobson & Miller, 1989; May, 1988; Scott, 1988). Although the majority of recent developments in the population dynamics of infectious diseases have focused on the epidemiology of human pathogens (Anderson & May, 1991), quantitative ecological studies on wildlife diseases are becoming more common (for examples, see Anderson and Trewhella, 1985; Anderson et al., 1981). Similarly, a number of theoretical studies have examined the population dynamics of systems involving one pathogen and two host species (Holt and Pickering, 1985; Dobson and May, 1986; Begon, Bowers, Kadiankis & Hodgkinson, 1992). Understanding the population dynamics of such systems is crucial in systems involving pathogens that infect

both wildlife and domestic species, particularly in situations where economic, health and political factors are important. Determining the most cost-effective and least damaging ways to control pathogens will considerably enhance the ability of wildlife managers to control similar diseases in other situations where they pose a major threat to the preservation of species diversity.

A) Epidemiological history of rinderpest in East Africa.

Evolutionary relationship of rinderpest.

Rinderpest is a member of the Morbillivirus genus in the order Paramyxoviridae. This genus contains four of the most important pathogens of humans and their domestic livestock: the others are canine distemper, measles and Peste de Petite Ruminants. Recent work on the antigenic relationships of the four viruses suggest they show a high degree of epitopic homology (Norrby et al 1985, McCullogh et al, 1986), this can be used to construct a phylogeny for the four species (Fig 1); all the available evidence suggests that the rinderpest virus is the ancestral root of this tree. The majority of the radiation of the species is likely to have occurred in India, the Far East or Europe following the domestication of ungulates and canids ((Plowright 1985, Clutton Brock, 1987). As the ungulate species that live in the Sahara desert exist at very low population densities they would be unlikely to suppport a continual infection of rinderpest virus. The desert would thus act as an efficient barrier for the spread

of rinderpest into the southern half of the African continent (Dobson 1988). The pandemic of rinderpest that occurred at the end of the last century was initiated by the accidental introduction of a few infected cattle into the horn of Africa. The epidemic caused massive mortalities confirming that sub-Saharan ungulate populations had no previous exposure to the pathogen (Plowright 1985).

History of rinderpest control in East Africa.

Throughout this century riderpest has caused disease outbreaks in cattle and a variety of wild ungulates throughout sub-Saharan Africa. Table 1 lists the main outbreaks of rinderpest in East Africa and the main species that were affected since its introduction in 1890 (Simon 1962). Outbreaks of the disease can be recognized by the pathology which is characterized by the onset of a sudden high fever several days after infection, this persists for two to three days when ulcers and erosions appear in and around the mouth. Infected animals then produce profuse nasal and ocular discharges, which may be accomanied by severe diarrhea. The animals quickly become emaciate and a high proportion die from the more virulent strains of the pathogen (Scott 1964).

The first inoculation programs against rinderpest in East Africa were initiated in the early 1940's, by the 1950's wide scale programs were employed throughout Northern Tanzania

(Branagan & Hammond, 1965). These early vaccination programs used the Kenya goat-attenuated virus (KAG) which provided long-lasting immunity to animals which were vaccinated after the maternal immunity conferred by their colostral antibodies had wained (Plowright, 1982; Scott, 1964). Although mortalities associated with the vaccination were low in healthy cattle (<2% within three weeks of innoculation), the vaccination mortality was significantly higher in calves (±20%), particularly if they were suffering from protozoal infections such as coccidosis, theileriasis or trypanosomiasis (Branagan 1965). These mortalities discouraged owners from bring calves for vaccination and produced a significant pool of susceptibles; outbreaks of rinderpest in cattle between 1940 and 1960 invariably affected calves and yearlings almost exclusively (Plowright 1982). This led to the development of culture attenuated rinderpest vaccine which was innocuous to cattle of all age while still producing life long immunity (Plowright & Taylor, 1967). More recent developments have produced a recombinant vaccine (Yilma et al 1988), which leads to some slight reductions in the cost of vaccine production, but these savings are offset by reports of problems with the development of rinderpest symptoms in immuno-compromised human hosts in areas where this vaccine has been tested (Rossiter, pers comm).

The development of an entirely effective rinderpest vaccine led to a large scale innoculation program (JP15) for the control

of rinderpest in sub-saharan Africa. The first three phases of JP-15 were lauched in West Africa in 1962, phase IV began in East Africa in 1968. The program attempted the mass immunization of 80 x $10^6$ cattle in 22 countries. The key success of the program lay in reducing the proportion of young cattle that were susceptible in the population. In areas where vaccination was carried out efficiently, such as Ngorongoro, the proportion of susceptibles fell to about 45%. However, in areas where access made comprehensive coverage more difficult, numbers of susceptibles remained in the 80-90% level (Fig 2).

History of rinderpest in Serengeti.

Rinderpest was first rumoured to be present in the Serengeti in 1930, its presence was not confirmed until 1933 when infectd tissue was collected from a 2-year old wildebeest from near Ngorongoro crater (Plowright & McCullough, 1967). The available records for the region from 1946 until 1961 indicate that rinderpest was present in the Serengeti wildebeest throughout this time with outbreaks occuring at one to two year intervals in yearling and two year-old wildebeest as well as buffalo, warthogs, eland and impala (Plowright & McCullough, 1967).

Following the initiation of the vaccination scheme in the cattle surrounding the national park in the late 1950's, the wildebeest numbers in the Serengeti increased from around 300,000 to around 1.5 million (Campbell, this volume; Sinclair, this

volume). Increases have also occurred in the numbers of buffalo and in the lions and hyeanas that act as the main predators of these two species (this volume). These increases in numbers were matched by the inital disappearance of the virus from wildebeest, buffalo and eland (Figure 3). The dissapearence of rinderpest is matched by the decline in 'yearling' disease which had previously operated as a produced levles of mortality in immature wildebeest than were estimated to be as high as 40% (Talbot & Talbot, 1961).

Although there have been no major epidemics of rinderpest in the Serengeti/Ngorongoro region since the vaccination schemes were modified to use the cell-culture vaccine, there have been a number of outbreaks of the disease that have led to significant localized mortality in cattle, buffalo and eland. In particular, at outbreak at Lobo in March 1982 killed many buffaloes. This was followed by an outbreak in the Ngorongoro crater area in June of the same year when two to four thousand buffalo and a number of giraffes, warthogs and eland also died. Sera collected from both these areas indicated the presence of rinderpest antibody (Rossiter et al 1983). Subsequent serological surveys by Anderson et al (1990) suggested that the disease persisted in the buffalo population for a number of years before fading out (Fig 4). However, it should also be noted that these data, which were collected from animals of different ages over the course of 2-3 years, could also be interpreted as reflecting the continued presence of the virus in the buffalo population. The change in

prevalence with age might simply reflect continued low level exposure of the animals to the virus. The absence of any pathological evidence would suggest that if it is present, it is a very mild strain of the virus. Although serum samples were taken from a 94 wildebeest, no animals with positive titres for rinderpest antibodies were identified. Very low levels of prevalence (<1%) were found in sheep and goats in the area.

Population numbers in Ngorongoro Conservation Region.

The numbers of different game species living in Ngorongoro create have been censusued fairly regularly by both the NCAA and Mweka wildlife college since the early 1960's (Estes & Small 1984, Homewood and Rodgers 1991). Although buffalo numbers have increased significantly since rinderpest was eradicated, the numbers of most species have remained relatively constant, while numbers of wildebeest, eland and rhinoceros have declined (Fig 5a). In contrast to the rest of the Serengeti, there is no evidence of an increase in the wildebeest population. The number of people living in the region has increased by two to three percent each year, while the numbers of cattle have remained roughly constant (Fig 5b). This has led to a decline in the ratio of cattle to human and an increasing reliance on sheep and goats as a source of protein (Homewood & Rodgers, 1991). Nevertheless, cattle are still crucial to the Masai as a source of protein and are an intrinsic part of their culture. Ensuring that rinderpest remains absent from the area is not only

important for wildllife, but crucial for the welfare of the Masai.

Population biology of rinderpest.

Single species models.

A knowledge of the dynamics of the pathogen in a single species population is essential to our understanding of the dynamics of more complex, multispecies host pathogen models. The dynamics of rinderpest may be modelled using standard SIR models (Anderson and May, 1982, 1991, Bailey 1975). These models assume that the host population may be divided into three classes: susceptibles, S, infected, I, and recovered and resistant, R. Three coupled differential equations describe the rates of change of each of these classes of host

$$dS/dt = a(S+I+R) - bS - \beta SI + \delta R \quad (1)$$

$$dI/dt = \beta SI - (\alpha + b + \nu) I \quad (2)$$

$$dR/dt = \nu I - (b+\delta) R \quad (3)$$

Here a is the population birth rate (assuming no effect of the pathogen on host fecundity), b is the death rate of uninfected animals, $\beta$ is the transmission rate between infected and susceptible animals, $\delta$ is the rate at which resistant animals lose their immunity to reinfection ($1/\delta$ is the average time for which resistance lasts), $\alpha$ is the increase in host mortality due to the pathogen and $\nu$ is the recovery rate of infected animals

(1/$\nu$ is the average duration of infectiousness).

Most of these parameters can be estimated from population studies of wild ungulates or from veterinary records for rinderpest. Most wild and domestic ungulates live for 2 to 5 years, so $b$ is of order 0.2 to 0.5; when infected with pathogenic strains of rinderpest virus, most hosts succomb within 10 days so $\alpha$ is of the 30 to 50; if they survive most animals cease to be infectious so $\nu$ is of the order 25. Estimating the rate of disease transmission, $\beta$, is always the hardest part of any epidemiological program. Derivation of two further fundamental parameters of the pathogens epidemiology allow insights to be developed on how this might be undertaken. Equations 1-3 may be rearranged to obtain expressions for the basic reproductive rate of the pathogen, $R_o$

$$R_o = \frac{\beta N}{(\alpha + b + \nu)} \qquad (4)$$

Here $N$ is the total population size (S+I+R), which in a population of susceptibles equals $S$; the basic reproductive rate is the number of secondary infections that a single infectious individual would produce in a population of susceptibles (Anderson and May, 1982). This expression must exceed unity if the pathogen is to persist in the population. The threshold number of hosts required to just maintain an infection of the pathogen, $N_T$, is given by a simple rearrangement of this

expression when $R_0=1$;

$$N_T=\frac{(\alpha+b+\nu)}{\beta} \qquad (5)$$

Even in the absence of any empirical data on the magnitude of the model's parameters, these equations tell us several important things about the dynamics of pathogens. First, it important to notice there is an approximately inverse relationship between the $R_0$ and $N_T$, thus highly transmissible diseases need only small populations to sustain them. Second, notice that parasites with increased virulence $\alpha$, tend to have reduced basic reproductive rates and require large populations to sustain continuous infections. Plowright (1982) suggests that 100,000 susceptibles in a population of 500,000 cattle is sufficient to maintain the virus. This would suggest a preliminary estimate for $\beta$ of 0.001. Analysis of the age prevalence curves for cattle before and after the vaccination scheme was initiated suggest an average age of infection of at around 230 days, this may be used to provide an estimate for $\beta$ in the range 0.002 to 0.005 (Grenfell, pers comm). This suggests that betwwen 20 and 50,000 susceptibles would be required for an outbreak to occur. However, estimates of $\beta$ may have been inflated at this time if significant amounts of transmission occur from wildlife back to cattle.

Examination of the system of equations at equilibrium allows us to derive an expression for the proportion of hosts infected

$$\frac{I^*}{N^*} = \frac{r}{\alpha} \qquad (6).$$

As infected animals of all species tend to live for between one and two weeks once infected and the intrinsic growth rate of most ungulates is between 0.1 and 0.2, we would expect to see less than 1/2 % of the animals infected <u>at any time</u> when the disease is endemic in a host population. Note that this expression implies that the proportion of a population infected <u>declines</u> with the virulence of a pathogen.

The size of the host population in the presence of the pathogen is given by

$$N^* = \frac{\alpha(\alpha+b+\nu)}{\beta[\alpha - r(1+(\frac{\nu}{b+\delta}))]} \qquad (7)$$

This suggests that host population size decreases as pathogen transmission efficiency $\beta$, increases. If rinderpest were restricted to a single species of host, we could eradicate the pathogen by maintaining the number of susceptible hosts below the threshold for disease transmission. In cattle this would be undertaken by vaccination. In the simplest case, the proportion of cattle, p, which would have to be resistant is given by

$$p > 1 - 1/R_o \qquad (8).$$

Anderson & May (1982). The above parameter estimates suggest that this would require between 57 and 91% of cattle to be vaccinated.

The crucial feature of rinderpest is that it is transmitted between species, to examine the population dynamic consequences of this we need to extend our basic model framework and include both intra- and inter specific transmission rates.

Mixed species models.

The model framework may be readily extended to include a second species of host (Anderson and May, 1986; Holt and Pickering, 1985; Begon et al 1992). The dynamics of infection are again described by a set of coupled differential equations for each population:

$$\frac{dS_i}{dt} = r_i S_i - \beta_{ii} S_i I_i + \beta_{ij} S_i I_j + a_i I_i + e_i R_i \qquad (9)$$

$$\frac{dI_i}{dt} = \beta_{ii} S_i I_i + \beta_{ij} S_i I_j - d_i I_i \qquad (10)$$

$$\frac{dR_i}{dt} = v_i I_i - (\delta - b_i) R_i \qquad (11)$$

Here the number of parameters in the model has been reduced by setting $\underline{r}_i = a_i - b_i$, $\underline{d}_i = \alpha_i + b_i + v_i$ and $\underline{e}_i = a_i + v_i$ (Holt and Pickering, 1985). These equations can again be examined at

equilibrium to determine the influence of the various transmission and virulence rates on the numbers of hosts infected in each population. The numbers of hosts of each species infected at any time is now given by the expression

$$I_i^{**} = \frac{I_i^* - (\beta_{ij}/\beta_{ii})\, I_j^*}{1 - (\beta_{ij}\beta_{ji}/\beta_{ii}\beta_{jj})} \qquad (12)$$

Here the $I_i^*$'s are the number of hosts infected in each population in the absence of interspecific transmission and $I_i^{**}$ is the number of hosts infected when interspecific transmission occurs. This result suggests that the ratio of between to within species transmission rates are as important in determining the numbers of animals infected as are the actual magnitudes of these rates. Where rates of interspecific transmission $\beta_{ij}$, are low compared to rates of intraspecific transmission $\beta_{ii}$, then an increase in the rate of interspecific transmission leads to a decline in the numbers of infected animals. This is mainly because host population size is reduced due to more animals becoming infected and dying. However, as rates of interspecific transmission approach those of intraspecific transmission, the host population first experiences a rapid increase in the numbers of infected, followed by a decline to extinction as all animals become infected and die. Similar effects occur if rates of interspecific transmission are intermediate, but the population of the reservoir host is sufficiently large to ensure that susceptible hosts are always infected. Establishment requires that within species transmission is greater than between species

transmission.

Begon et al (1992) provide a detailed analysis of the population dynamics of pathogens which infect two host species which are regulated to constant carrying capacities, $K_1$ and $K_2$ in the absence of the pathogen. Their analysis shows that if only one of the species, $H_1$, has a population size sufficient to maintain the pathogen ($H_{1T}<K_1$), then persistence of the disease in both species requires either $\underline{\alpha}_2<r_2$ or $\underline{\beta}_{22}<\underline{\beta}_{21}$. In the case of rinderpest, species 1 would correspond to cattle, while species 2 would correspond to wildebeest or buffalo. The second condition is unlikely to be met for either wildebeest or cattle as it requires between species transmission to exceed within species transmission. The first condition is dependent upon the virulence of the virus; if the host population growth rate, $r_2$, exceeds the virulence of the virus, $\underline{\alpha}_2$, then the pathogen will be present in both species, even if the wild species is below its individual threshold for establishment, $H_{2T}$. In contrast where the virulence of the pathogen exceeds the growth rate of the host population ($\underline{\alpha}_2>r_2$), it is possible for the presence of the disease in cattle to drive an alternative wild host to extinction; this will occur when sufficient numbers of species 1 are infected that between host transmission leads to a significant increase in the rate of infection of species 2 (formally $Y_1*\underline{\beta}_{21}/\underline{\beta}_{22} > Y_2$). This is most likely to occur when there are significant differences in the virulence and transmission efficancy of virus between species 1 and 2 (with $\underline{\alpha}_1<$

$\alpha_2$).

Intra-specific and inter-specific disease transmission.

Estimation of transmission rates.

All of the preceding analyses suggest that quantifying rates of transmission is crucial to increasing understanding of the way the pathogen spreads both within a single species population and between different species. A number of different ecological and epidemiological factors are likely to be important in determining the relative rates of within and between species transmission. Population size is a major variable determining a populations ability to maintain a pathogen (Kermack and McKendrick 1927); diseases rapidly die out in species with small scattered populations, they are likely to remain present for a long time in species with large, aggregated populations (Anderson & May 1991). Populations sizes will also be important in the establishment of pathogens that infect several species of host. Here we need to establsih whether the presence of infected individuals of another species can allow a pathogen to establish in a populations in which it would otherwise be unable to persist. Thus we will need to estimate not only rates of transmission between different individuals of different age, sex or social status and members of each individual species, but also transmission rates between different age and sex classes of different species.

This initially daunting task may be simplified if we can

independently quantify ecological associations and interactions between individuals of the same and different species. Distances between animals are likely to be important in determining rates of transmission of respiratory diseases and can be fairly readily quantified in the field. As in other ecological processes, it is not only the mean distance between animals that will be of importance in determining transmission rates, but also the heterogeneity in these distances. In the case of intra-specific transmission the observed population densities and levels of aggregation are determined both by the habitat in which the animal lives and its social system. Physiological factors are a second important component that determines the ability of each species to transmit a pathogen. Here a variety of factors, such as the quality of mucus and saliva produced and the rate at which an infected animal coughs and sneezes all affect its ability to produce viable pathogen transmission stages.

If we assume that net transmission rate consists essentialy of a physiological and a spatial component, the different rates of transmission may be expressed in the simplest case as

$$\beta_{ij} = ( \beta_c + \text{VAR} [ \beta_c ]) \beta_p \qquad (12).$$

Here $\beta_c$ represents the component of transmission due to the average distance between conspecifics, $\underline{\text{VAR}}\ [\beta_c]$ represents an increase in transmission rate due to the tendency of the species

to aggregate into social groups and $\beta_p$ represents the species-specific, physiological component of transmission. Notice that if it is possible to independently quantify the spatial components of transmission, $\beta_c$, using an index of mean crowding such as the reciprocal of nearest neighbor distance, then the physiological component, $\beta_p$, can be used to scale the net transmission rate. Because species with different social systems will exhibit different tendencies to aggregate it will be interesting to see how much of the variation in prevalence between species, is explained by differences in social behaviour. Similarly, it should be possible to use these data to determine how differences in social behaviour <u>within</u> a species are likely to effect expected levels of prevalence and compare these with observed serology data for different social classes of a single host population. The estimates can be rescaled using available serology profiles to produce expected patterns of prevalence in sections of the population with different social or age characteristics.

<u>Interspecific transmission.</u>

The average distance between members of different species and the average group size will also be crucial in determining rates of interspecific disease transmission. Because the ratios of interspecific to intraspecific transmission are important in determining the prevalence of infection in different host species, it seems sensible to initially assume that the relative

magnitudes of interspecific transmission rates scale with some index of average distance between individuals of different species. Figure 7 illustrates the average distances observed between the nearest individuals of different species in one survey fo Ngorongoro crater during August 1989, the data are taken from systematic surveys of the spatial distribution of ungulates in the crater (Dobson, unpublished). These surveys suggest that species with different social systems exhibit different tendencies to aggregate; initial analyses suggest that much of the variation in disease prevalence between species is explained by these differences in spatial distribution. Similarly, the average distances between members of different species are crucial in determining rates of intra-specific transmission. The results of this analyses suggest that disease transmission rates within a species are likely to be much larger than rates between species (Figure 7). However, transmission may occur readily between some species (eg. Thompsons and Grants gazelles) and hardly at all between others (eg. Wildebeest and Impala).

Vaccination coverage in the Ngorongoro region.

Cattle in the Ngorongoro region are vaccinated annually with a live attenuated virus vaccine. During the dry season, cattle are driven by the tribesman to a local 'crush' where as many as four thousand individuals may be vaccinated in a single day. The records for the different regional crushes are kept at the office

of the conservator and at the regional veterinary center at Arusha. These data allow temporal and spatial variation in vaccination coverage to be monitored. Figure 8 illustrates the total number of adult and juvenile cattle inoculated in the entire NCCA region for the years 1971-90. The level of vaccination coverage has increased steadily so that around 90,000 out of 130,000 cattle (around 70%) are protected at any one time. If Plowright's estimate that around 100,000 susceptible cattle are required to maintain the pathogen in the population, then the present level of coverage should be sufficient to prevent the disease re-establishing. However, our estimate of transmission rate using the serology data collected from cattle in the region suggests a threshold value more in the range of 20 to 50,000 animals. If we compare the estimated numbers of susceptible cattle in the Ngorongoro conservation region over the last twenty years (Figure 9), we find that the numbers of susceptibles have exceeded this lower level at least three times.

The outbreak that occurred in 1982 spread into the wildlife and killed large numbers of buffalo. A larger epidemic was prevented due to the mounting of a massive vaccination campaign which may have inoculated around 99% of the cattle in the region. Although the cause of this outbreak has been traced to some illegal cattle movements through the Conservation area, the result underlies the importance of vaccination in containing the disease and in preventing future outbreaks. Furthermore it

should be noted that the outbreak occurred in NCCA where the vaccination coverage is very good (Fig 2); where the vaccination coverage is lower, the potential for rinderpest persistence is higher. This may explain why both Rossiter et al (1987) and Anderson et al (1990) found evidence for rinderpest in buffalo from the Northern part of Serengeti.

## Discussion and conclusions.

The evidence presented in this paper suggests that wildlife do not act as a long-term reservoir of rinderpest virus. Although no wild game were ever vaccinated to protect them from infection, the widescale vaccination of cattle that was initiated in the 1940's ultimately led to the dissapearence of the disease from wildlife. This in turn led to large increases in the wildebeest and buffalo populations, here a six to eight fold increase in wildebeest density failed to produce an outbreak of the pathogen. Although increases in buffalo numbers have also failed to sustained infections of rinderpest in this species, outbreaks of rinderpest have occured in this species on a number of occasions in the Serengeti and Ngorongoro region. Serological surveys suggest that when these outbreaks involve mild strains of rinderpest virus, that then persist for a number of years before fading out. This would imply that wildebeest are well below the threshold density required for them to continuously maintain rinderpest virus, while buffalo may be fairly close to this critical density.

In the absence of the virus in cattle it is unlikely that populations of wild ungulates will maintain the pathogen and rinderpest epidemiology in the region is likely to be characterised by occasional outbreaks in buffalo (and perhaps eland) whenever contact is made with infected animals that may be moved through the region. Rinderpest is therefore still a threat to the integrity of the Serengeti and Ngorongoro conservation, its absence is dependent upon an efficient vaccination program in cattle (Plowright 1985), and vaccination coverage is variable throughout the region and may be low in inaccesible areas (Dobson, in prep). Furthermore, the annual vaccination program is expensive to operate; after road maintenance it is the major budget item in the park's annual budget. Although it is economically cheaper to vaccinate during the dry season, the cattle are on a lower nutritional plane at this time and lower numbers are brought for vaccination. More worringly, cattle are now grazed and watered in Ngorongoro crater at times of prolonged drought; in the years when droughts have led to reduced attendence at vaccination crushes this leads to a higher risk of contact between significant numbers of susceptibles wild and domestic individuals. An outbreak that occured during a drought period, when all species are on a low plain of nutrition and likely to be immunologically compromised, could be disasterous for both the cattle and the wild game of the region.

Finally, it is worth considering the role that poaching of

wild game has had on constarining a rinderpest outbreak and whether systematic culling could be used as a management tool to prevent or contain an outbreak. Evidence presented elsewhere in this volume (Sinclair 1993; Campbell & Hoffer, 1993; Dobson & Georgiadis 1993), suggests that poaching has led to significant reductions in the populations of buffalo, wildebeest, eland and other ungulate species in the Serengeti region. If wild game were the principal hosts of rinderpest then reducing the size of their populations would reduce the possibility of a rinderpest outbreak. However, all the available evidence suggests that cattle are the primary hosts of the virus and successful control of the virus is entirely dependent upon high levels of vaccination coverage in cattle. The increased levels of poaching of wild game have had no discernible effects on the incidence of rinderpest outbreaks. More significantly, Dobson and May (1986) have calculated that the width of a cordon sanitaire required to halt the spread of a rinderpest outbreak in wild game is of the order of forty miles. This would require elimination of most of the game in the Serengeti if an epidemic were spreading from west to east. This and all the above calculations suggest that the best way to control rinderpest in the Serengeti is to continuously prevent an outbreak by maintaining comprehensive vaccination coverage of cattle throughout the region.

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## Figure Legends.

Figure 1. The evolutionary relatedness of the Morbillivirus genus (after McCullogh et al 1986). The estimate of the split between MV (measles virus) and RPV (rinderpest virus) is a rough estimate based on the available evidence for historical plagues whose aetiological descriptions have similar diagnostic characteristics to present day measles.

Figure 2. Serology profiles of cattle before and after vaccination in Ngorongoro District in 1969-1970 and in the Kajiado District in 1970 (after Plowright 1982).

Figure 3. Serology profiles from Buffalo, Eland and Wildebeest in different parts of the Serengeti ecosystem following the onset of vaccination in cattle (data from Plowright & McCullough 1966, and Taylor and Watson, 1966).

Figure 4. Serology profiles of tissue samples collected from buffalo of different ages in the Masai Mara in Kenya (after Rossiter et al, 1987) and in three areas of the Serengeti National Park (after Anderson et al 1990); the Northern samples (74) were taken around Kleins Camp; the West samples were taken around Musabi (85) and also at Kirawira and the Ndabaka plains (34); while a sample was also taken around Seronera and Moru Kopjes (38). In each case buffalo are classified according to their year of birth.

Figure 5. (a) Estimates of population size for the principal ungulate species in Ngorongoro crater between 1966 and 1988 (after Estes & Small 1984 and Rodgers & Homewood, 1991). (b) Estimates of the human population and domestic animals in the NCCA over the period 1966-1988 (after Rodgers & Homewood, 1991).

Figure 6. Population size versus bii.bij ??

Figure 7. The potential transmission rates between the main ungulate species in Ngorongoro Crater. The estimates are based on samples of nearest neighbour distance for each ungulate species in the crater. The estimates were obtained in August 1989 by driving around the crater and estimating the distance to the nearest conspecific and the nearest member of another species for between one and five animals in each social group of ungulate observed each day. The surveys have been repeated in the wet and dry season for each of two years (after Dobson, in prep).

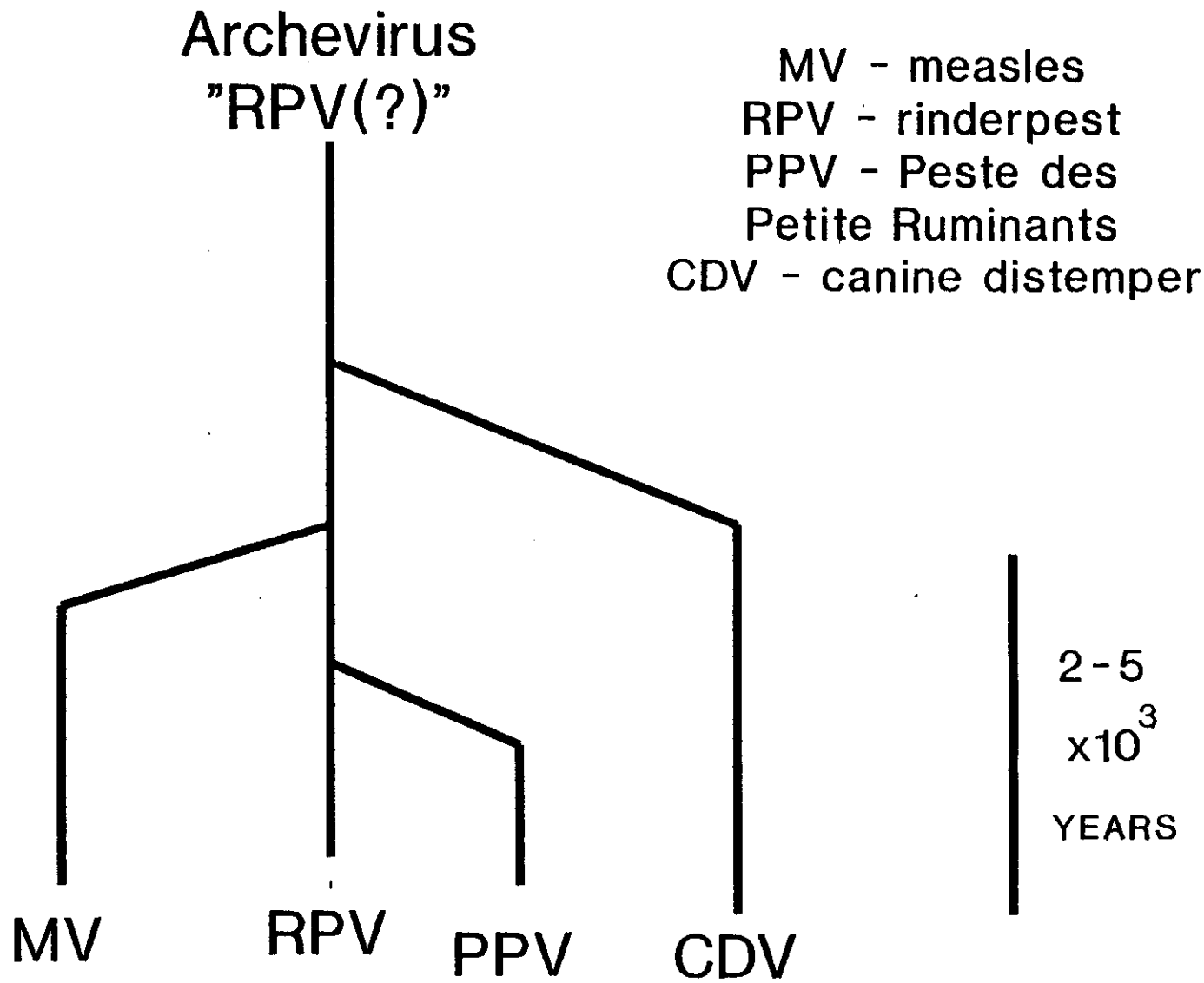
Figure 8. The total numbers of rinderpest vaccinations given to cattle in the Ngorongoro district between 1970 and 1990 (after Dobson, in prep). The vaccination campaign lasts throughout the dry season, from July through until September. The data illustrated are for the total numbers of adult animals, which are branded immediately before vaccination (RB), and calves which are marked with an ear punch (EP).

Figure 9. Estimates of the numbers of susceptible cattle in NCCA in the period 1970 to 1990. The figure gives the numbers of susceptible individuals in the population, before and after vaccination, in each year. These estimates were obtained by assuming a previous innoculation lasts for life, but that the numbers of cattle brought for inoculation include animals who have been inoculated previously. As the average survival and fecundity rates of cattle have been estimated for the region (Homewood & Rodgers, 1991), it is possible to estimate the proportions of susceptible, resistant and multiply innoculated individuals. The dotted lines illustrate two estimates of the potential transmission threshold for rinderpest to establish. The upper value at 100,000 susceptibles represents Plowrights estimate, the lower value at 50,000 is based on an estimate of transmission rate using the serology profile for cattle given in figure 3.

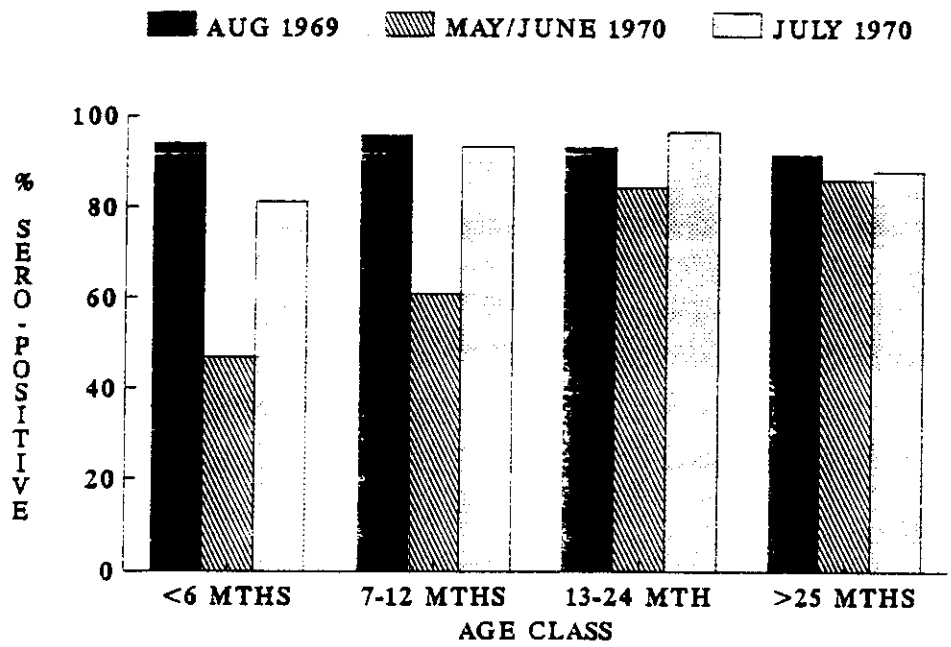
Table 1. The main recorded epidemics outbreaks of rinderpest in East Africa (after Simon 1962).

**Table I**

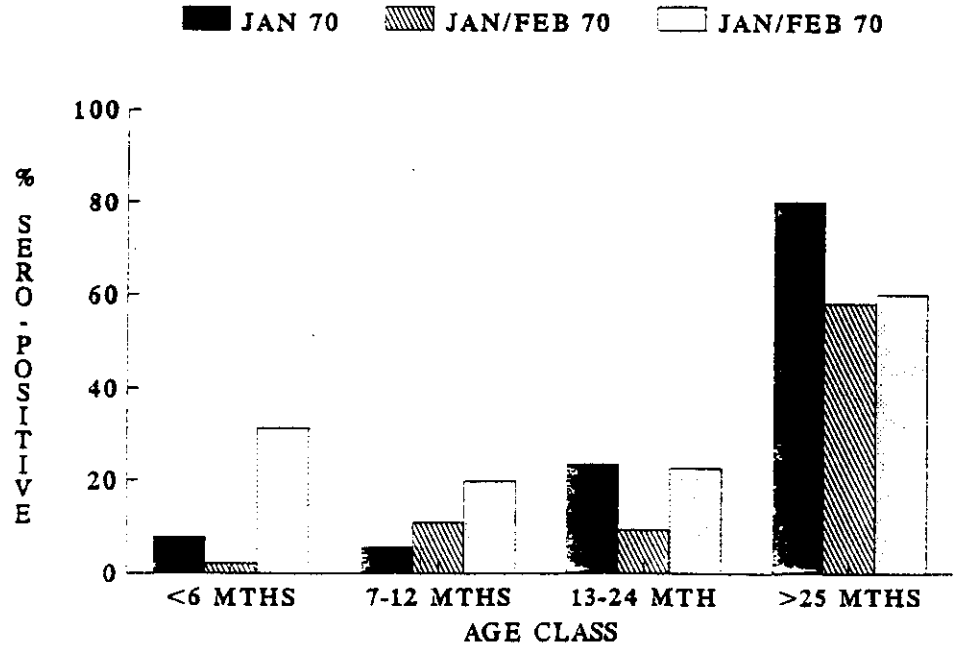
| Year | Species Affected. |
|---|---|
| 1890 | Panzootic - most ungulate species. |
| 1897 | Hartebeest and Kudu. |
| 1913-21 | Eland and Giraffe, then Buffalo, Bushbuck and Reedbuck. |
| 1929 | Buffalo, bushbuck and warthogs, then eland, waterbuck and kob. |
| 1931 | Buffalo, giraffe and wildebeest. |
| 1937-41 | Buffalo, eland and giraffe, then buffalo, eland and kudo. |
| 1949 | Cattle, eland and then wildebeest. |
| 1960 | Eland (60%), kudu & warthogs (60%), buffalo (50%), bushbuck & giraffe (50%), impala (40%) & oryx. |


Archevirus
"RPV(?)"
MV - measles
RPV - rinderpest
PPV - Peste des
Petite Ruminants
CDV - canine distemper
MV
RPV
PPV
CDV
2-5
x10^3
YEARS

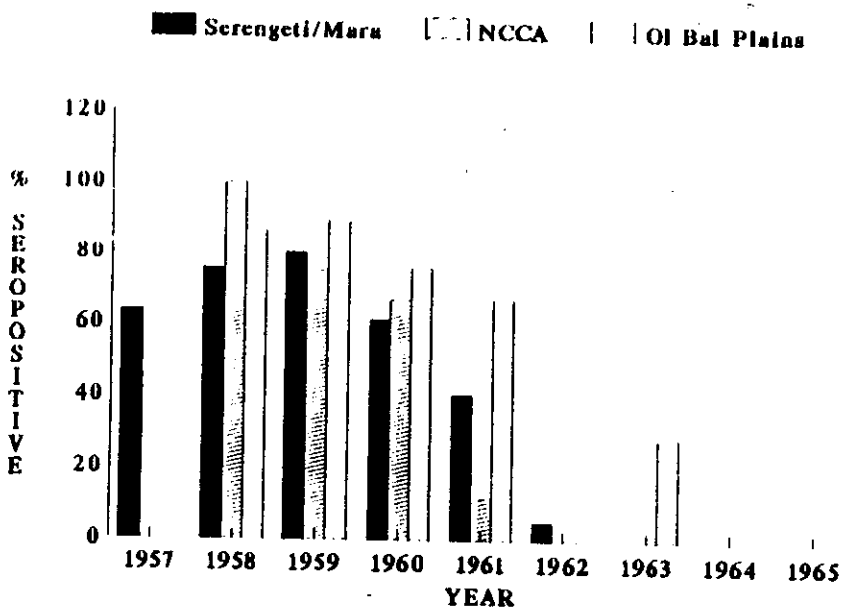
## Ngorongoro District



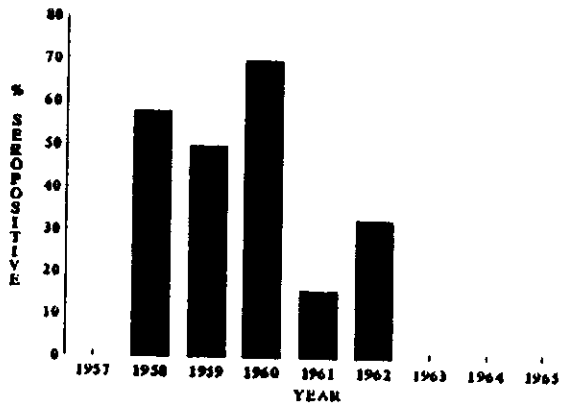

## Kajiado District



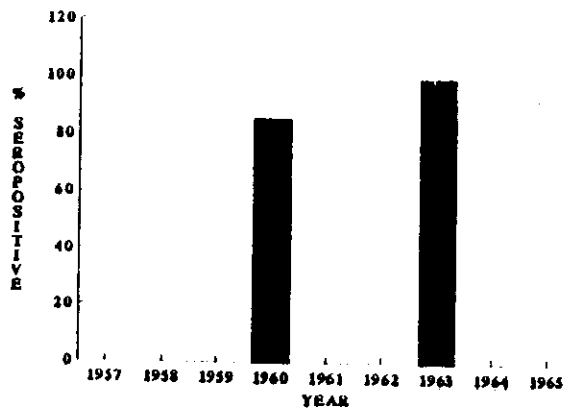
## Wildebeest in Serengeti/Mara



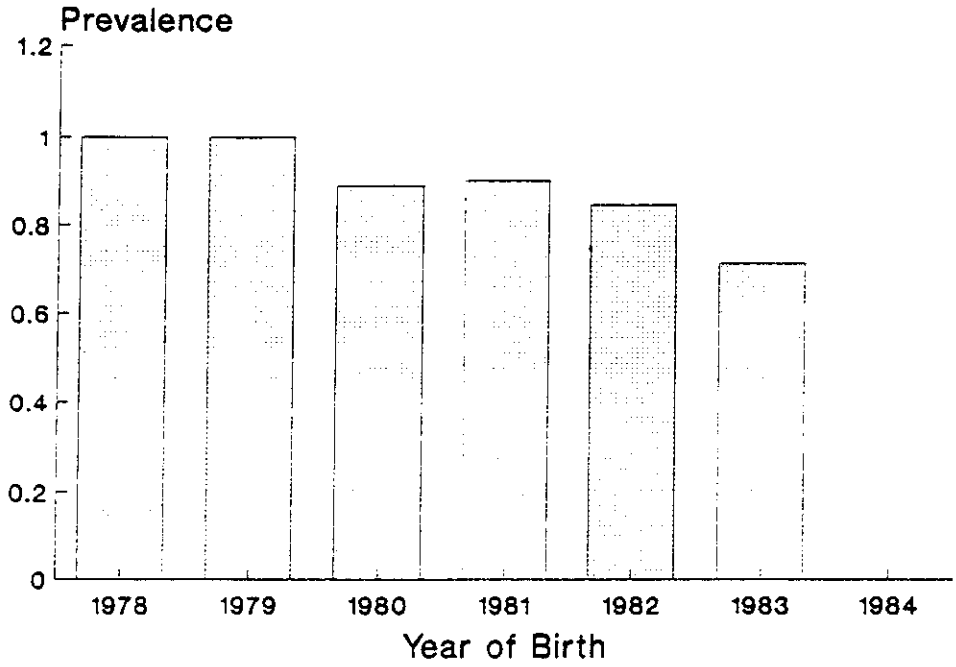

## Eland in NCCA



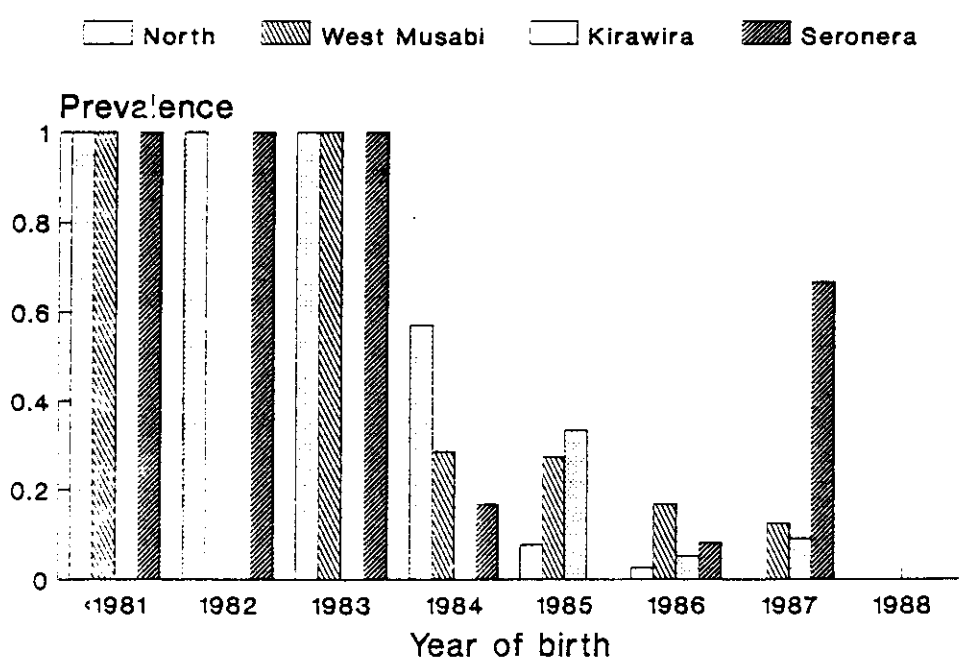

## Buffalo in Loliondo



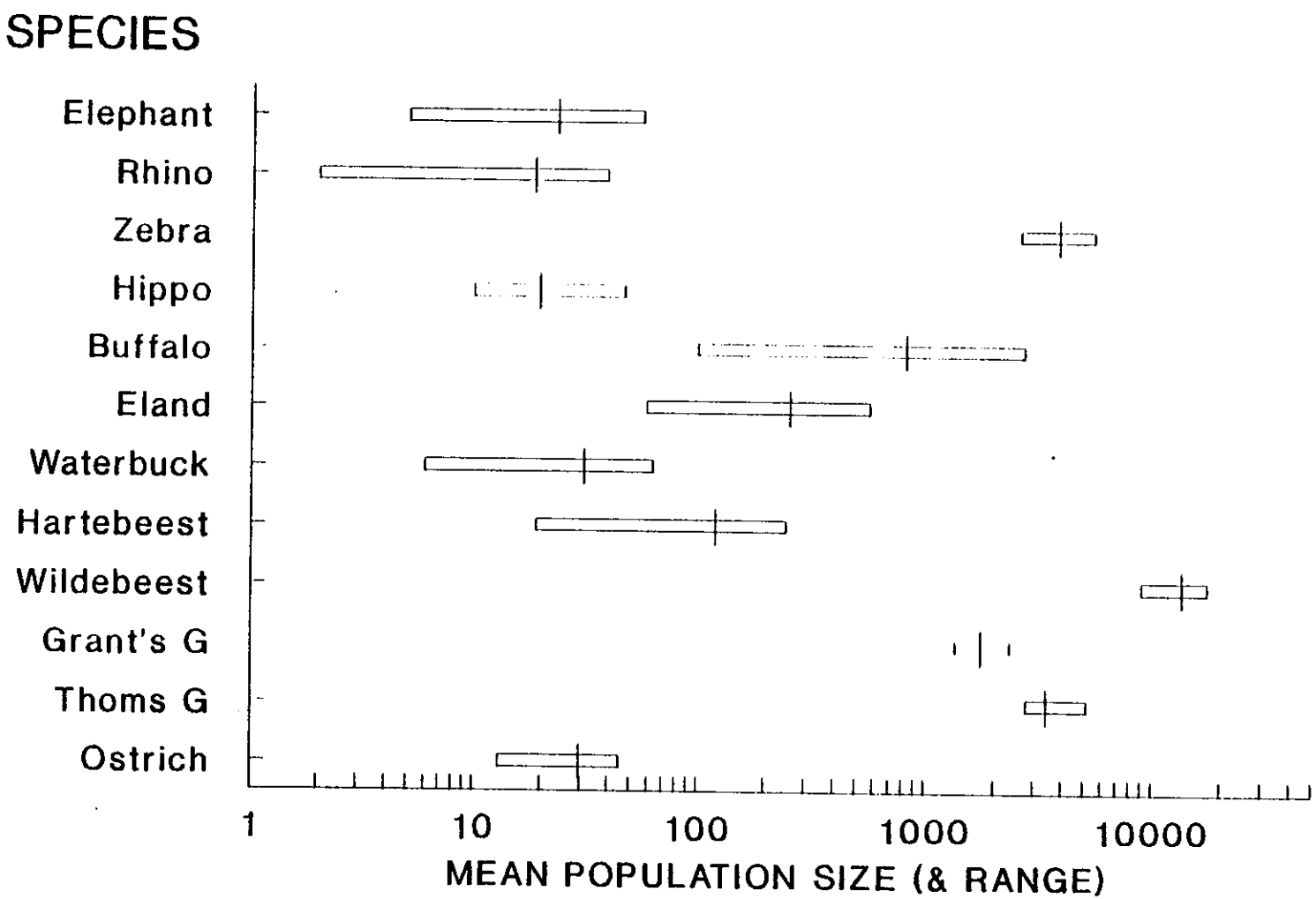
## Buffalo in Masai Mara



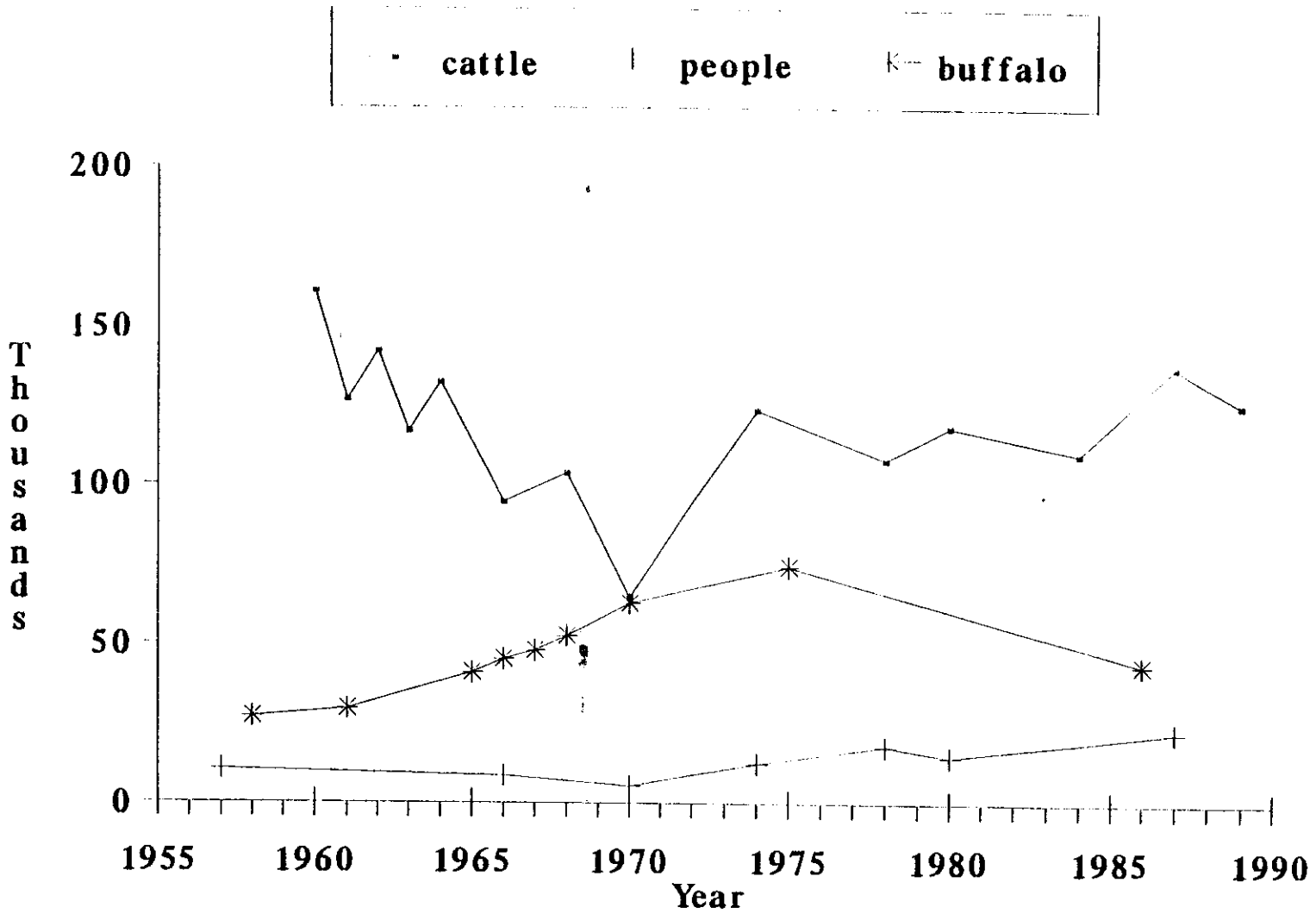

## Rinderpest in Serengeti Buffalo



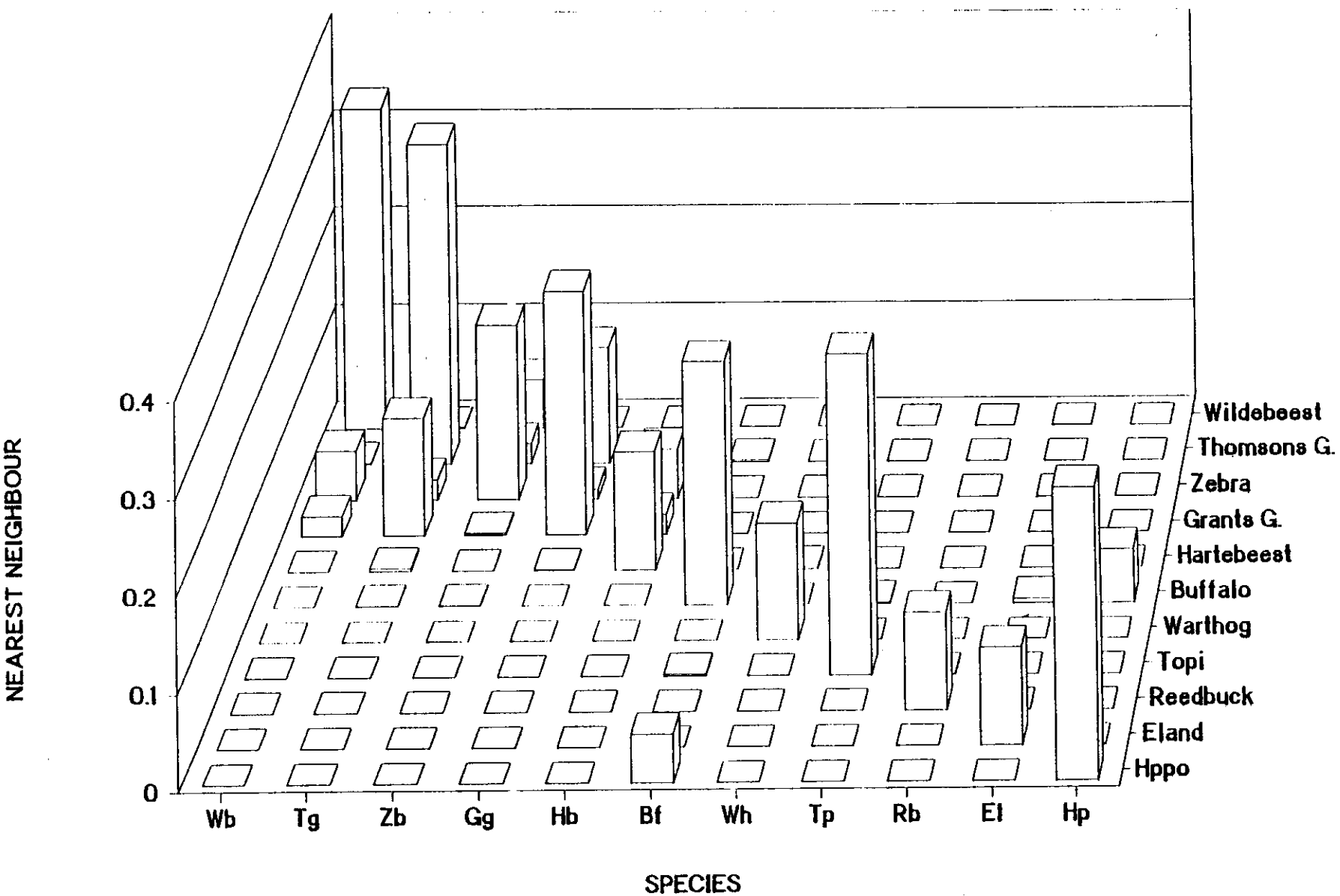
# GAME POPULATIONS IN NGORONGORO CRATER NCCA AND MWEKA COUNTS 1962-88.



# POPULATIONS IN NCCA

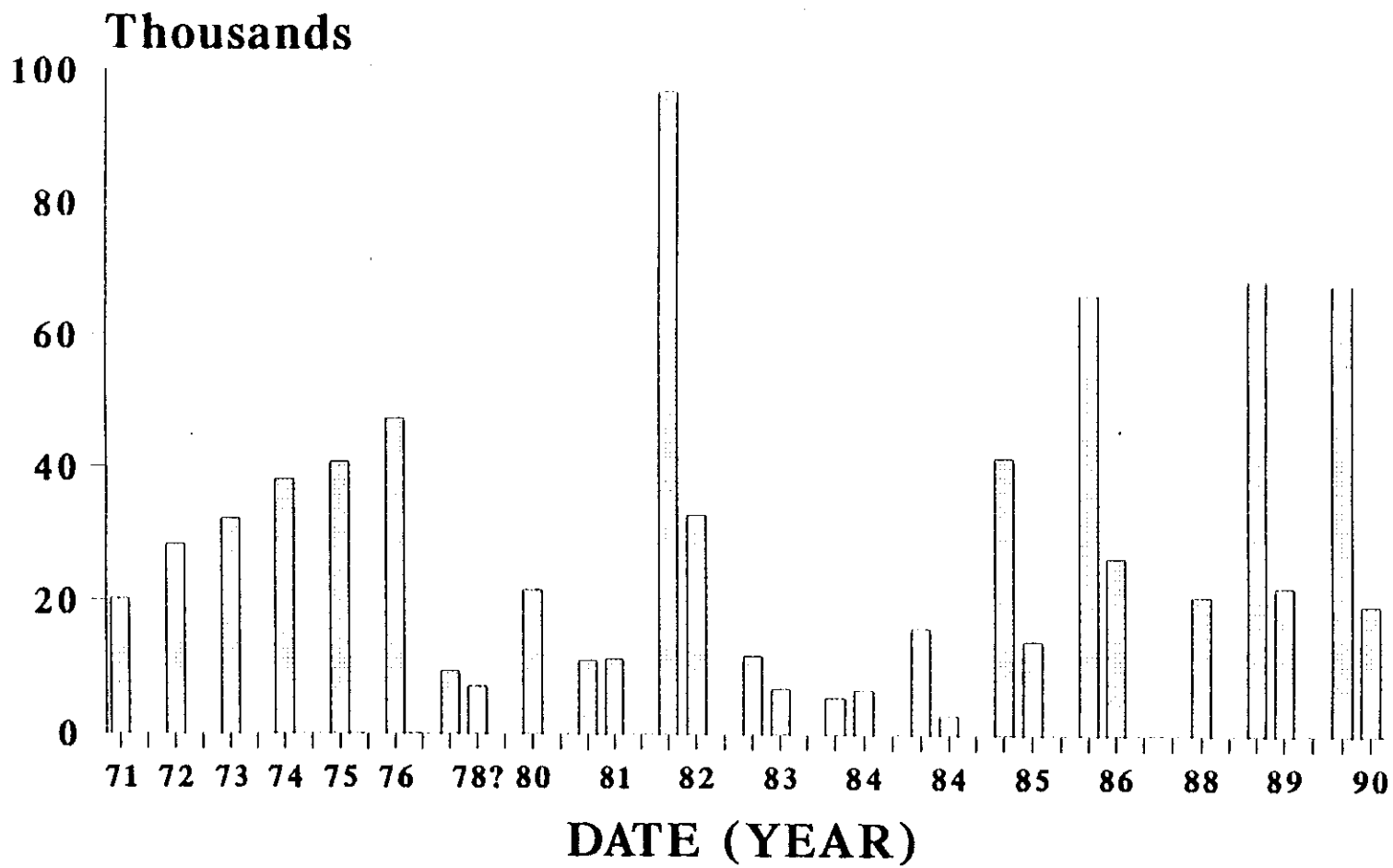


# RINDERPEST TRANSMISSION POTENTIAL



# VACCINATION IN NCAA

## RB - left : EP - right



# POTENTIAL FOR RINDERPEST OUTBREAK