Wildlife diseases in South Africa: a review

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Summary: Wildlife diseases which have been documented in South Africa, both currently and historically, can be divided into two categories.

The first category includes endemic African diseases which have evolved with and cycle in the various indigenous wildlife populations which, in turn, act as sylvatic reservoirs of the infectious agents. These diseases usually cause only minimal debility or mortality in indigenous wildlife and the infections are often subclinical. In contrast, infection of domestic livestock with these agents usually results in high morbidity and variable mortality. They are thus of considerable agricultural economic importance. African swine fever, African horse sickness, foot and mouth disease (SAT virus types), bovine malignant catarrh, bluetongue, Rift Valley fever, trypanosomiasis and theileriosis are good examples of this disease category.

The second category includes exotic diseases which have been, or are suspected of having been introduced onto the African continent (by domestic animals). These diseases are capable of infecting certain species of indigenous wildlife, often causing large-scale morbidity and mortality. Rinderpest, rabies, anthrax and brucellosis are good examples of this category. This paper reviews our present knowledge and the current status of such diseases in South Africa.

KEYWORDS: Artiodactyla - Bacterial diseases - Epidemiology - South Africa - Viral diseases - Wild animals.

FOOT AND MOUTH DISEASE CAUSED BY SAT TYPES OF VIRUS

This is the most economically important viral disease in South Africa. The SAT types of aphthovirus are confined to the African continent. Surveys and research have shown that the African buffalo (*Syncerus caffer*) is the main sylvatic reservoir and maintenance host of this group of viruses (42, 21, 1, 6, 9, 18, 12, 2), although the greater kudu (*Tragelaphus strepsiceros*) has also been implicated (22). This endemic situation occasionally spills over into other susceptible wildlife species (12, 26, 27, 38, 44) as well as cattle, resulting in small epidemics or large-scale pandemics. Natural outbreaks of FMD have been diagnosed and confirmed in impala (*Aepyceros melampus*), kudu, bushbuck (*Tragelaphus scriptus*), nyala (*Tragelaphus angasii*), warthog (*Phacochoerus aethiopicus*), giraffe (*Giraffa camelopardalis*), sable antelope (*Hippotragus niger*) and roan antelope (*Hippotragus equinus*), living in areas of Southern Africa where infected buffalo are also found.

African elephant (*Loxodonta africana*) and perissodactyls appear to be insusceptible to natural challenge with SAT aphthovirus although elephants have been

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experimentally infected by intra-dermolingual injection of large doses \((2 \times 10^6 \text{ TCID})\) of SAT 2 virus (24). In contrast, elephants placed in contact with these artificially-infected elephants, and (in a later experiment) elephants placed in close contact with infected cattle failed to become infected and no seroconversion occurred (5). This has been confirmed with types SAT 1 and SAT 2.

In South Africa there are essentially three buffalo subpopulations: namely, the Kruger National Park/Eastern Transvaal Lowveld, the Zululand and the Addo National Park populations. Only the first of these populations carries aphthovirus, the other two being free from the disease (17).

An FMD control zone is enforced in Eastern Transvaal, encompassing the areas with infected resident buffalo populations and surrounding agricultural land. The zone is divided into three control categories with diminishing stringency of control measures towards the periphery.

The following important research findings concerning African wildlife and FMD have emerged over the past twenty years:

- **a)** African buffalo appear to be the only long-term carriers of FMD (more than five years) in South Africa, with all three SAT types present in the infected buffalo populations (42, 22, 21, 10).
- **b)** Over 80% of buffalo in any infected herd in South Africa have been exposed to all the SAT types by the age of three years (42).
- **c)** “Carrier buffalo” are unimportant transmitters of FMD to domestic stock (10, 6) but are highly important maintenance reservoirs of FMD virus in a given buffalo population. In the Kruger National Park, buffalo calves are born from December to July with a peak in February-March. As the calves lose their passive (colostral) immunity at 3-7 months of age, some individuals thus become susceptible to FMD infection throughout the year.

Buffalo calves become infected from buffalo which either carry the virus or are experiencing acute primary infection (mainly other calves and yearlings).

With the three main types of SAT virus and many substrains which have been identified, it would appear that FMD acute primary infection in a free-living buffalo population occurs in young animals, and cycles periodically through susceptible individuals in small epidemics (42).

- **d)** During primary infection, buffalo excrete as much virus in respiratory aerosols, saliva and nasal secretions as do cattle, but for a slightly longer period (18).
- **e)** Impala are highly susceptible to aerosol infection. As little as 10 TCID are required to infect an impala via the respiratory tract (unpublished results). Once infected, impala develop coronet, mouth and occasionally ruminal pillar lesions of varying severity, depending on the virus strain. Impala shed much less virus than cattle or buffalo, but they are capable of infecting contact cattle. No virus can be isolated from impala beyond seven days after development of the initial lesions. Impala are not long-term carriers and their antibody response is brief (6-9 months) (unpublished results).

- **f)** Warthogs are highly susceptible to SAT viruses but the severity of clinical lesions depends very much on the strain of virus present. Acute illness with death due to viral myocarditis has been seen with certain SAT 1 strains. The disease was confined
to mild hoof lesions in the case of some SAT 2 strains. Viral excretion rates are low in comparison with cattle, buffalo and domestic pigs (unpublished results).

\( g \) No FMD antibodies have been detected in sera from white rhinoceros \((Ceratotherium simum)\) or hippopotami \((Hippopotamus amphibius)\) in the endemic area.

**RINDERPEST**

No rinderpest outbreaks have occurred in South Africa since the devastating pandemic of 1898–1903, when the disease entered North Africa from Asia and swept south, killing millions of cattle and countless wild animals. Many of the current anomalies of wildlife distribution in Africa can be traced to this panzootic. Its unexpected sequels included the disappearance of tsetse fly and foot and mouth disease for several decades from large areas of Southern Africa due to the disappearance of the host animals or viral reservoirs of these disease vectors and organisms.

Highly virulent rinderpest strains rapidly burn themselves out among animals with low innate resistance. Outbreaks in highly susceptible species, while spectacular, are relatively unimportant in maintaining the disease. Species possessing high innate resistance, because they may perpetuate the disease unrecognised, are more important in the maintaining and cycling of rinderpest, which may then become endemic, especially if a milder strain of disease is involved (37).

Rinderpest appears to be primarily a disease of cattle, which spills over into adjoining wild ungulate populations. But it can also cycle for a period in wild ungulates, especially if a mild strain is involved or an ungulate species with moderate to low susceptibility is present in large numbers in the infected region (33, 37).

Wildlife have different levels of susceptibility to rinderpest infection (see Table I of the introductory report by P.-P. Pastoret et al.).

It is important to note that the highly susceptible wildlife species are more prone to infection than cattle, and therefore provide a more accurate clinical indication of the disease in a given area.

There is still controversy as to whether the disease can cycle **endemically** in wild ungulates which would then serve as a reservoir of infection for adjacent cattle populations (37, 40). The fact that clinical rinderpest disappeared from wildlife after the disease had been eradicated in cattle would appear to indicate that, in general, cattle remain the ultimate source of infection (37) (the virus cycling in yearlings and partly immune individuals), but it should be remembered that infected wild ungulates can spread the disease by their movements and migrations.

The recent re-emergence and spread of rinderpest in East, Central and West Africa, appears to be linked to several factors:

\( a \) Rinderpest foci were still present in neighbouring territories.

\( b \) Cessation or breakdown of annual cattle vaccination due either to complacency (the disease had been absent for several years) or to financial constraints.

\( c \) Uncontrolled movement of cattle caused by drought or by political unrest and civil wars.
A proposed control strategy to protect any African country under the imminent threat of rinderpest infection would include:

**Phase 1**

1. Mass inoculation of cattle in a cordon sanitaire along the international boundaries under threat, plus quarantine and control of livestock movement.

2. Close monitoring of highly susceptible wild ungulates in the threatened boundary area.

3. Preparation for a possible outbreak by producing or purchasing large quantities of vaccine and other equipment needed for a mass vaccination campaign.

4. Vaccination of cattle in a belt around any National Park or Game Reserve in the area under threat, followed by close monitoring of highly susceptible wild ungulates in these conservation areas.

Should these measures fail and the disease enter the country, then phase 2 options should be initiated:

**Phase 2**

1. Cordonning off and control of livestock movement out of the infected area.

2. Initial ring vaccination of an area surrounding the infected foci.

3. Mass vaccination of all cattle in the entire surrounding region.

4. Prophylactic vaccination of breeding nuclei of all highly susceptible wildlife species in conservation areas under threat, using drop-out darts or ballistic implants.

Should further spread of the disease occur, then phase 3 options should be exercised:

**Phase 3**

1. Total mass vaccination of the national herd.

2. Total movement control from and into all infected foci.

3. Serological evaluation of vaccination efficacy.

In all three phases, vaccination of domestic stock should be repeated annually (biannually for calves) and if financially feasible, aerial inoculation of highly susceptible wildlife should be expanded.

Annual vaccination of livestock should continue indefinitely after the disease has been controlled, especially if foci of infection are still present in neighbouring countries.

During outbreaks in wildlife, burning of carcasses is of questionable benefit, as the virus is rapidly inactivated by putrefaction, high temperatures and pH changes.

Recent unpublished research in the Kruger National Park has shown that the Onderstepoort attenuated live rinderpest vaccine (incorporating the Kabete O strain) is highly effective and safe for use in buffalo and impala, and affords prolonged protection.
LUMPY SKIN DISEASE
(Bovine herpesvirus 2: Allerton strain)

Among free-living wild animals, this disease has been documented mainly in buffalo in East Africa, where visible clinical symptoms and lesions followed by mortality were reported and the virus isolated. Serological surveys showed almost universal infection of buffalo in East Africa, but only occasional titres in giraffe, waterbuck (*Kobus ellipsiprymnus*), hippopotamus, eland (*Taurotragus oryx*), oryx (*Oryx beisa*), impala, bushbuck and wildebeest (*Connochaetes* spp.). In cattle, contact infection does not occur and transmission by biting flies such as *Byomia fasciata* and *Stomoxys* spp. has been reported.

In naturally occurring cases reported in buffalo, the most striking lesions were well-defined ulcers on the tongue, palate and buccal mucosae (36). An important feature when diagnosing this disease is, therefore, to differentiate it from acute, active foot and mouth disease.

LUMPY SKIN DISEASE
(Neethling strain)

There are no reports of natural cases of this paravaccinia viral disease in free-living wild animals. Young *et al.* (45) succeeded in artificially infecting a young giraffe and an impala, which developed typical lesions and succumbed to the disease. Buffalo and wildebeest were refractory to artificial infection and no seroconversion occurred.

RIFT VALLEY FEVER

The African buffalo is known to be susceptible to Rift Valley fever. Artificial infection resulted in only mild symptoms plus a single abortion (13). Positive serological titres have also been found in hippopotami and elephant in the Kruger National Park. Abortion in wild springbok (*Antidorcas marsupialis*) and blesbok (*Damaliscus albifrons*) occurred during the 1950-51 epizootic in domestic stock in South Africa.

BLUETONGUE

In Africa, the history of bluetongue suggests that it was a viral disease of wildlife which was capable of exploiting the introduced susceptible populations of European livestock.

Antibodies are present in most African artiodactyls (13) and experimental infection of several antelope species resulted in asymptomatic infection (23). Antibodies have also been found in other species ranging from African elephant to various rodents, which opens up an almost unlimited host range with reservoir potential. In North America, however, bluetongue has caused large-scale mortality among white-tail deer and mule deer (23). A closely related viral disease, epizootic haemorrhagic disease, also causes severe mortality among deer in the USA with symptoms and lesions indistinguishable from bluetongue. Although this virus was recently detected in Africa, it has not been associated with disease in wildlife or livestock.
AFRICAN HORSE SICKNESS

As in the case of bluetongue, African horse sickness appears to have originated on the African continent, becoming manifest when susceptible animals were introduced from Europe. It is therefore likely that a primary vertebrate host or reservoir exists among wild animal populations.

Most zebras (*Equus burchelli*) in the Kruger National Park possess antibodies, and susceptible zebras which were artificially infected developed a febrile reaction and occasional mild supra-orbital oedema. Erasmus (16) reported that viraemia in these zebras lasted 18 days, and on the 20th day virus could still be isolated from lymph nodes.

By day 35, virus could no longer be isolated from lymph nodes. Thus it appears that zebras are in general highly resistant, and that most infections are inapparent. The long viraemic period may be important for arthropod vector uptake and transmission of the virus. Several rodents are susceptible to experimental infection, and may prove to be important reservoirs or amplifiers of this virus.

AFRICAN SWINE FEVER

African swine fever (ASF) was first described in South Africa in 1921 by Montgomery. From 1926 to the present day a cyclical periodicity of outbreaks in domestic swine has been documented, with 10-12 years of disease outbreaks being separated by 10-12 disease-free years (32). Plowright et al. (34, 35) described the importance of argasid ticks in the epidemiology of this disease. It has since been confirmed in East and South Africa (43) that the sylvatic cycle of this disease occurs between argasid ticks (*Ornithodoros moubata* spp.) and wild porcines, of which warthogs appear to be the most important.

In Southern Africa, the distribution of ASF is closely linked to that of warthog and the eyeless tampan (*Ornithodoros moubata*), namely, the Northern, North Western and Eastern Transvaal and the northern part of Zululand. These areas are included in the ASF control zone.

Several heterologous strains of ASF have been isolated, some of which are haemadsorbing and others not. Major differences in the double-stranded DNA molecule of six field isolates have been mentioned by Thomson (43).

Although this disease is subclinical or asymptomatic in wild porcines, it is pathogenic and causes high mortality in domestic pigs, and it is one of the most economically serious diseases of domestic pigs. There is no effective vaccine available, and even following recovery from natural infection, no neutralising antibodies are detectable.

Some of the most important epidemiological aspects of ASF are:

a) In wild porcines, there is neither horizontal nor vertical transmission and no virus is detectable in secretions and excretions of acutely infected individuals.

b) In domestic pigs, large quantities of virus are present in secretions and excretions of infected individuals, and horizontal transmission occurs readily.
c) Infected wild porcines and recovered domestic pigs carry virus for a long time, especially in the lymph nodes.

d) The virus appears to cycle between argasid ticks and young warthogs in their first few months of life. Thomson has shown that primary infection of young warthogs may result in viraemic titres of $> 10^3$, sufficiently high to infect engorging tampans (41).

In turn, Plowright et al. (34, 35) have demonstrated high titres of ASF virus in the coxal fluids and saliva of infected tampans.

e) Plowright et al. (34, 35) have demonstrated that these argasid ticks are highly efficient biological reservoirs, amplifiers and vectors of ASF virus. Transovarial infection of offspring occurred as well as sexual transmission from male to female (but not vice versa).

f) More recently, Mellor et al. (28) have demonstrated that ASF could be transmitted by *Stomoxys* spp. 24 hours after feeding on an infected pig. After 48 hours, the virus titre in these flies showed no significant decrease.

Finally, it would appear that transmission of ASF from wild porcines to domestic pigs can occur in two ways:

- Transmission by an arthropod vector such as the tampan (biological transmission) or the stable fly (mechanical transmission).
- Contact with or ingestion by domestic pigs of raw products or tissues from wild porcines.

### RABIES

This highly lethal viral disease occurs in domestic and wild animals. Historically it appears to have originated in Egypt, spreading north into Europe and Asia and south towards the Cape, probably together with man and his domesticated companion animals.

In Southern Africa today, rabies exists in an urban form, associated with dogs, and a rural form associated with wildlife.

In South Africa, dog-associated rabies is confined mainly to Natal, Kwazulu and the Northern and Eastern Transvaal (4, 25). Cases of wildlife rabies can be subdivided into four recognisable areas according to the chief vectors in that area:

a) The central plateau including the Orange Free State, Western Transvaal and N.E. Cape. In this area the most common vector is the yellow mongoose (*Cynictis penicillata*).

b) Northern and Eastern Transvaal. In this area the most common vector is the black-backed jackal (*Canis mesomelas*), although feral dogs are also important.

c) North-Western Cape and southern South West Africa. In this area bat-eared foxes (*Otocyon megalotis*), wild cats (*Felis nigrepes*) and genets (*Genetta* spp.) are the most important vectors.

d) In northern South West Africa black-backed jackal and feral dogs are important vectors, but over the last decade, kudu (3) have become the species most often affected by clinical rabies.
Additional epidemiological factors in rabies transmission are:

- The communal burrow-dwelling habits of species such as yellow mongoose, suricates (Suricata suricatta) and bat-eared fox facilitate transmission.
- The aggressiveness of wild cats, genets, feral cats and honey badgers (Mellivora capensis) results in high bite transmission rates.
- Wandering of infected feral dogs transports the infection long distances.
- Non-bite transmission by salivary contamination of leaves occurs in the kudu cycle.
- Rabies has a long incubation period, and virus is shed in the saliva before symptoms develop.

**Control of rabies**

1. Vaccination of all domestic dogs and cats and perhaps cattle in endemic areas and control zones.
2. Vaccination of all dogs and cats entering or passing through control zones.
3. Selective population reduction of important vectors in certain areas, e.g. gassing of mongooses in communal burrows or strychnine baiting of jackals.
4. Shooting of stray dogs and registration and licensing of dogs in urban areas.
5. In Europe, oral vaccination of foxes using live attenuated virus or recombinant vaccines has proved reasonably successful (31).

The choice of vaccine is very important because certain live virus vaccines are capable of causing clinical rabies in wild animals.

In conclusion, rabies has been diagnosed in 35 species of African wildlife, of which the following are of potential epidemiological importance: yellow mongoose, black-backed jackal, wild cat and genets, suricates, bat-eared fox and kudu. Rabies may be responsible for population fluctuations in these species. So far there is no evidence of a true carrier state in any of these animals.

**BRUCELLOSIS**

In Africa, free-living populations of Cape buffalo and hippopotamus have given positive serological tests for brucellosis (14, 11). Tube agglutination, Rose Bengal and complement fixation tests have revealed an 11-13% infection rate in hippopotamus and a 14.2-22.6% infection rate in free-living buffalo populations. Carpal hygromata are also occasionally seen in buffalo, and Gradwell et al. succeeded in isolating *Brucella abortus bovis* biotype 1 from cotyledons in 3 of 68 pregnant buffalo cows (19). Condy et al. (11) isolated the same organism from a waterbuck. A buffalo isolate was capable of infecting a pregnant buffalo cow, which subsequently aborted.

The impact of this disease on the buffalo population appears limited, and very few in utero fetal deaths have been observed following detailed examination of more than 3,000 pregnant buffalo culled over the past five years in the Kruger National Park. This fact is also supported by the 12-15% annual increase in the buffalo population despite moderate predation by lions and hyenas.
Brucellosis appears to be primarily a disease of cattle which has opportunistically entered some wildlife populations. There are no records in Africa of wildlife serving as a source of infection for domestic cattle. In the USA, bison and elk have been reported to have low calving rates as a result of this disease.

TUBERCULOSIS

Tuberculosis is commonly reported in wild free-living animals only where they have been in contact with infected domestic animals or human beings. It has, however, been reported in lechwe (*Kobus leche*) in Zambia (8), Cape buffalo in Uganda (20) and greater kudu and grey duiker (*Sylvicapra grimmia*) in South Africa (30). In each case, the organism isolated was *Mycobacterium bovis* and probably originated from cattle. In captive wild animals, *M. bovis*, *M. avium* and *M. tuberculosis* (human strains) have all been responsible for numerous outbreaks in zoos, involving many species ranging from monkey to elephant. With the exception of free-living badgers in the United Kingdom, wild animals have never been implicated as a reservoir of tuberculosis and appear to be unimportant, incidental victims of the disease.

ANTHRAX

This fatal disease of both domestic and wild animals is the only disease that must kill its host to propagate itself in the environment. There are two distinct epidemiological patterns among wildlife:

a) *Water contamination*

Usually due to animals dying in or near a permanent water-hole, natural pan or gravel pit. Possibly more important is the contamination of water by carrion feeders, especially vultures and marabou storks, which seek to quench their thirst and bathe immediately after feeding. In this way, contaminated blood on the beak, talons and feathers enters the water.

b) *Contaminated vegetation*

Vegetation becomes contaminated directly by carcasses, dung of scavengers and carnivores which have eaten an anthrax carcass, or by blowflies. In the Kruger National Park, blowflies appear to be the most important. Following feeding on an anthrax carcass, blowflies perch on vegetation 1-2 m above ground level and then produce a vomit or fecal droplet, which can be seen with the naked eye as it dries on the leaves.

Pure cultures of the anthrax bacillus can readily be obtained from these leaves in the vicinity of anthrax carcasses. Isotope labelling of blowflies has also shown that they can travel as far as 62 km during two weeks, illustrating their potential for disseminating the disease (unpublished results).

Research into the minimum infectious dose of anthrax spores for different game animal species has been initiated. To date, only impala have been studied fully, and the oral LD$_{50}$ for this species is about 16 million spores. The parenteral LD$_{50}$ is as low as 500 spores (Dr V. de Vos, personal communication).
In the Kruger National Park, rare species such as roan antelope are immunised annually against anthrax by using the Sterne spore vaccine in disposable drop-out darts or ballistic implants (15). Anthrax has become rare in domestic stock in South Africa due to effective annual inoculation.

Q FEVER

This rickettsial disease has been described in many domestic and wild animals, as well as in ticks and man. It would appear that a natural sylvatic cycle occurs between ticks and free-living wildlife, and any infection of man and domestic stock is incidental. The disease is becoming an increasingly important cause of abortions in livestock, as well as a public health hazard.

HEARTWATER

(Cowdria ruminantium infection)

This tick-borne rickettsial disease causes heavy mortality among cattle, sheep and goats in Southern Africa (7). Recent research has shown that guinea-fowl and tortoises can act as sylvatic reservoirs for the causal agent, and that they are also predilection hosts for the larvae and nymphae of the tick vector of this disease, Amblyomma hebraeum. The role of other wild animals, particularly ungulates (buffalo, giraffe and warthogs) which are favourite hosts for the adult Amblyomma tick, has not yet been investigated. Blesbok may become infected asymptomatically. Eland and springbok can be infected clinically and may die (46).

MALIGNANT CATARRHAL FEVER

(Snotsiekte)

With the growth of the game farming industry in Southern Africa, wildebeest-associated snotsiekte has become the single most important cause of wildlife-related mortality in cattle in the region today.

Blue and black wildebeest are asymptomatic carriers of this herpesvirus, and Plowright (33, 36) demonstrated that virtually all wildebeest become infected by the fourth month of life, some of them congenitally. In addition, he showed that some retain the virus late into adult life, and postulated that advanced pregnancy may result in a recrudescence of viraemia. Plowright also mentions that viraemia was most prevalent in wildebeest calves less than four months old.

Mushi et al. (29) demonstrated the presence of malignant catarrhal fever (MCF) virus in a highly infectious cell-free state in the lacrimal and nasal secretions of wildebeest calves less than four months old. Mushi et al. (29) reported that the disappearance of virus from these sites coincided with the appearance of virus-specific IgA with neutralising activity in the nasolacrimal secretions.

It would appear that wildebeest calves are infected in utero or by association with infected wildebeest calves soon after birth. The social behaviour of wildebeest, namely, the formation of nursery herds in the perinatal period, could contribute to the rapid spread of MCF through all susceptible individuals. Rossiter (39) could find no evidence that fetal membranes and fluids of calving wildebeest were infectious.
Since highly infectious cell-free virus is present in the oculonasal secretions for only the first 3-4 months of life, and since the incubation period of MCF is 1-3 months, it would appear that the dangerous contact period for cattle would be within the first 4-5 months after the wildebeest calving peak, which occurs in December-January in the Kruger National Park. In infected cattle which show almost 100% mortality, the virus present in oculonasal secretions is cell-associated with relatively low infectivity. This would explain why MCF is a "dead-end" disease in cattle with no spread between cattle.

In South Africa, the peak incidence of MCF in cattle is March-April, which supports the viewpoint mentioned above. Thus a practical method of reducing the incidence of the disease in cattle in South Africa would be to avoid wildebeest/cattle contact from January to May.

Recently, however, a secondary peak of clinical cases of MCF in cattle has emerged in South Africa in September-October, which does not fit the conventional epidemiological picture. It was therefore decided to repeat East African research on the persistence of cell-free virus in the oculonasal secretions of wildebeest calves.

During 1986-87, 103 wildebeest were chemically immobilised in the Kruger National Park, and blood as well as ocular and nasal swabs were collected. During this period 15-20 wildebeest were caught monthly, with particular attention to specific age groups (monthly sampling of the current year's calf crop up to nine months of age). A random sample of juvenile and adult wildebeest of both sexes, including heavily pregnant females (in November), was also obtained.

Our results confirmed the findings of Plowright and others, virus being recovered from 60% of calves aged 2 and 3 months, declining to 15% of calves aged 4 months, and only an occasional animal older than 4 months. No virus was recovered from adult animals including pregnant females, although antibody was universally present in animals over 6 months of age.

Thus the cause of the secondary outbreak peak has yet to be elucidated. Some of these outbreaks occurred without direct contact between cattle and wildebeest, the animals being separated by fences or, in one case, a barrier of more than 200 m. This may well suggest an arthropod vector, and so we are currently investigating the possibility that nasal flies of the genus *Gedoelstia* might be implicated.

Antibodies to MCF have also been detected in the blood of hartebeest (*Alcelaphus* spp.), topi (*Damaliscus korrigum*) and oryx in East Africa. Herpesviruses have also been isolated from hartebeest and topi (29), but these species have never been incriminated as possible causes of outbreaks. Experiments are presently being conducted in East Africa to ascertain the pathogenicity of these strains for cattle, and their antigenicity with regard to vaccine potential. Sheep-associated MCF also occurs on the African continent, and it is clinically and pathologically indistinguishable from the more common wildebeest-associated form. The infectious agent is very difficult to isolate, however, and it may be a related but dissimilar virus.

Finally, outside the African continent, all Cervidae and Bovidae appear to be susceptible, and many develop clinical disease with frequent deaths.

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Resumen: Las enfermedades de la fauna salvaje en Sudáfrica, para las que se dispone de una documentación a la vez histórica y de actualidad, pueden clasificarse en dos categorías:

La primera incluye las enfermedades africanas endémicas que han evolucionado con las diversas poblaciones de especies salvajes autóctonas y tienen un ciclo en estas poblaciones, las que desempeñan un papel de reservorios silvícolas de sus agentes. Por lo general, estas enfermedades son muy poco debilitadoras o mortales para la fauna salvaje autóctona, que suele presentar una infección inaparente. En cambio, la contaminación de los animales domésticos por estos agentes provoca en general una morbilidad elevada y una mortalidad variable. Estas enfermedades tienen pues una importancia económica considerable para la agricultura. La peste porcina africana, la peste equina, la fiebre aftosa (tipos SAT del virus), la coryza gangrenosa, la fiebre catarral maligna, la fiebre del valle del Rift, la tripanosomosis y la theilériose son enfermedades representativas de esta categoría.

La segunda categoría agrupa las enfermedades denominadas «exóticas», que han sido, o se sospecha haber sido, introducidas en el continente africano por
animales domésticos. Dichas enfermedades, entre las que la peste bovina, la rabia, el carbunco bacteridiano y la brucelosis son buenos ejemplos, pueden infectar determinadas especies salvajes autóctonas, provocando generalmente en estas una morbilidad y una mortalidad importantes.

El presente artículo resumirá el estado de nuestros conocimientos y la situación actual de estas enfermedades en Sudáfrica.


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REFERENCES


