A review of the infectious diseases of African wild ruminants

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ABSTRACT

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The important viral, protozoal and bacterial diseases of wild African ruminants are reviewed. Special attention is paid to the epidemiological factors that determine the role played by these animals in the transmission of diseases to domestic stock. Examples of the converse situation where livestock serve as a source of infection for wild ruminants are also given.

Keywords: African wild ruminants, diagnosis, epidemiology, infectious diseases, review, transmission

INTRODUCTION

In Africa contact between wild ruminants and domestic stock is a significant factor in the occurrence of diseases of livestock as well as those of wild ruminants. Having waned during especially the nineteenth and early twentieth century, when wildlife numbers were drastically reduced by hunters and rinderpest, this contact is again waxing because wildlife conservation and associated ecotourism is currently blooming.

Of crucial importance when considering the subject matter of this paper, is the difference in the epidemiology between diseases caused by agents that have evolved together with African ruminants over long periods of time, and those that have been introduced into Africa comparatively recently (Bigalke 1994). In the former case, the agent and host have generally become adapted to each other and co-exist without causing harmful effects to the host. In such a situation the wild ruminant hosts are usually asymptomatic carriers of the disease agents and represent a source of infection for domestic ruminants or arthropod disease vectors. A typical example is the carrier-state of wildebeest for malignant catarrhal fever (MCF) virus. In contrast, MCF virus causes a serious, usually fatal disease in cattle. Where a disease has been more recently introduced, it may cause severe illness in wild ruminants. This is the case with rinderpest, which caused high mortality in African buffaloes and antelopes during, for example, the great rinderpest epidemic at the turn of the nineteenth century.

Wide ranges of habitat types occur in Africa and some species of ruminants and some arthropod vectors have long-standing restricted habitat ranges thus influencing susceptibility to disease. The heartwater vectors of the genus *Amblyomma* are not found in the virtually treeless, frost-prone Highveld areas of southern Africa. The antelope species that are found predominantly in these areas are generally more susceptible to heartwater than those species that occur in heartwater endemic areas. Animal species that have evolved in countries that are free from particular diseases are often highly susceptible to those diseases. For example, although virtually all African antelopes are highly resistant to the bluetongue virus, American pronghorn antelopes were susceptible to the disease when it was introduced into the United States of America (Hoff & Hoff 1976; Sohn & Yuill 1991).

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It is also worthy of note that very few infectious agents have such a wide host range as *Toxoplasma gondi*, which includes reptiles, birds and mammals (Dubey & Beattie 1988).

In this paper the viral, protozoal and bacterial diseases of African wild ruminants are reviewed with particular reference to the role of these animals in the transmission of diseases to domestic livestock. For many diseases, signs of illness and pathological lesions do not occur in wild ruminants. When they do, they are generally similar to those seen in domestic animals. In this review clinical signs are briefly summarized, generally being those seen in domestic animals. The pathological lesions associated with the infections are not described since they are either similar to those in domestic animals or are not seen. Wild ruminant species have been referred to by their common names, but a list of systematic and common names is given in the Appendix. The term "antelope", as used in this review, does not include buffaloes or giraffes. The term "buffalo" refers to the African buffalo (*Syncerus caffer*). Asian buffaloes and antelopes are only occasionally referred to when relevant or of particular interest.

VIRAL DISEASES

Foot-and-mouth disease

Foot-and-mouth disease (FMD) is a highly infectious disease that affects a wide variety of cloven-hoofed animals and is endemic over large parts of Africa. The virus is fairly resistant to environmental factors and can be transmitted in meat and dairy products (Bengis 1997). The introduction of the disease into previously non-infected countries has lead to vast economic losses. The recent introduction of the disease into Taiwan devastated a thriving pig industry, that according to information published on the Internet (http://www.promedmail.org), had an annual production of US$3.24 billion, and destroyed an export market worth US$1.55 billion. FMD is the disease that is most feared by countries that are free from the infection and a major reason for complicated legislation and conservative trade agreements between countries trading in live animals and animal products.

Aetiological agent

The disease is caused by an *Aphthovirus* belonging to the family Picornaviridae. Serotypes A, O, C, SAT1, SAT2, SAT3, and Asia 1 occur and are immunologically distinct. Several sub-types are known for each serotype. The virus has the capacity to mutate at a rate of about 1.64 % nucleotide substitutions per year in persistently infected carriers (Vosloo, Bastos, Kirkbride, Esterhuysen, Van Rensburg, Bengis, Keet & Thomson 1996) thus leading to the continual development of new strains. The SAT types are particu-

larly important in the wildlife of Africa although types A and O also occasionally occur (Thomson 1994; Chilonda, Woodford, Ahamdu, Samui, Syakalima & Mlangwa 1999).

Susceptible species

The disease affects all cloven-hoofed animals and all antelopes and other Bovidae must be regarded as susceptible, although some species show minimal clinical manifestations when infected. In a serological survey involving 7 970 sera from 14 species of wild ungulates in Zimbabwe, 1.25 % were positive. Positive titres were found in eland, impalas, waterbuck, and sable antelopes (Anderson, Foggin, Atkinson, Sorensen, Madekurozva & Nqindi 1993). Some species of antelopes are particularly susceptible and in Israel 2 000 mountain gazelles died during an outbreak of the disease. Remarkable pancreatic lesions were seen in experimentally infected mountain gazelles (Shimshony, Orgad, Baharav, Prudovsky, Yakobson, Bar Moshe & Dagan 1986; Shimshony 1988; Perl, Yadin, Yakobson, Zuckerman & Orgad 1989).

Incubation period and signs of the disease

The incubation period is usually 2–8 days, sometimes up to 2 weeks in cattle (Thomson 1994). In domestic animals the disease is characterized by fever, inappetance, painful gait and drop in milk production. Vesicles develop in the mouth, on the tongue, teats, on the coronary band and in the interdigital space. The vesicles rupture and progress to form erosions before healing after about 2 weeks. The disease is rarely fatal in adult cattle, but in calves it may cause sudden deaths when heart muscle necrosis occurs. In antelopes and African buffaloes signs of infection are usually milder and some species of antelopes appear to be highly resistant to the disease and show minimal signs of infection. Impalas are more susceptible showing typical signs of infection (Keet, Hunter, Bengis, Bastos & Thomson 1996a). In the Kruger National Park (KNP) in South Africa, impalas usually act as the indicator species when the disease is active amongst wildlife (Keet *et al.* 1996a). In buffaloes the infection is often inapparent (Thomson, Vosloo, Esterhuysen & Bengis 1992).

Carrier state

After recovery from the disease, the virus persists in the pharynx of cattle for up to 2.5 years and perhaps even lifelong in the African buffalo (Condy, Hedger, Hamblin & Barnett 1985; Thomson 1994; Chilonda *et al.* 1999). In a study involving two types of the virus in a small free-living isolated herd of 30–100 buffaloes, the carrier state was shown to persist for at least 5 years in individual buffaloes and was maintained through several generations and for at least 24 years in the herd (Condy *et al.* 1985). Most ante-

lope species probably only carry the virus for short periods after recovery from the acute phase. For example, in experimental infections virus persistence was transitory in sable antelopes and did not occur in eland (Ferris, Condy, Barnett & Armstrong 1989). Similar experiments have not been done for all species of antelopes. However, one authority has suggested that wildebeest and kudus can become carriers of infection (Wittmann 1990) although that author suggests that only carrier buffaloes transmit the virus to their own species and perhaps to cattle.

Transmission

Transmission is by aerosol infection or direct contact between animals excreting virus and susceptible individuals. When winds are favourable, virus-containing aerosols generated by infected domestic animals, particularly pigs, can be transmitted over long distances. For example the virus was shown to cross the English Channel by this means (Donaldson, Gloster, Harvey & Deans 1982). Air samples collected in the vicinity of infected buffaloes contained virus (Gainaru, Thomson, Bengis, Esterhuysen, Bruce & Pini 1986). Virus has been found in blood, nasal secretions, saliva, preputial secretions and faeces of young infected buffaloes, but in lower titres than in cattle. However, excretion in nasal secretions and saliva continued longer in buffaloes than in cattle (Gainaru et al. 1986). The disease can also be transmitted by ingestion of infected material carried on fomites.

Epidemiology

In Africa, the African buffalo is the main maintenance host for the virus (Thomson et al. 1992; Thomson 1994; Thomson 1995; Chilonda et al. 1999). Buffaloes are resistant to infection and show few signs of infection but after becoming infected may carry the infection in their pharynxes for at least 5 years (Condy et al.) and perhaps even for life (Bengis 1997). However, once the viraemic period has passed, buffaloes are not highly infectious and many attempts to transmit the disease by contact with carrier buffaloes have failed (Bengis, Thomson, Hedger, De Vos & Pini 1986; Gainaru et al. 1986). Evidence gathered from experimentally infected buffaloes suggested that they transmitted the disease to cattle and impalas only in the acute stages of infection and when there was direct physical contact between the species (Gainaru et al. 1986). However, buffaloes do carry the agent in their pharynxes for long periods, and as the virus is highly contagious and only a few viable organisms are needed to transmit the disease, they are potentially infectious. Some evidence exists that indicates that transmission from carriers to cattle may occur. In one case experimentally infected buffaloes were held together with cattle on an island in Lake Kariba. The buffaloes developed systemic infections and became carriers. During the acute phase of infection in the buffaloes, the cattle did not show lesions or develop antibodies to the virus. After 5 months the cattle developed clinical FMD and nucleotide sequencing of the viruses isolated from cattle and buffaloes were almost identical (Dawe, Sorensen, Ferris, Barnett, Armstrong & Knowles 1994). In this experiment it seems that the cattle were infected by buffaloes that were carriers of the virus and not by acutely infected animals. In another experiment in which artificially infected buffaloes and non-infected cattle were held together, transmission only occurred after several months in some instances (Vosloo et al. 1996). Linear nucleotide substitutions occurred at a rate of 1.64 % substitutions per year in the SAT2 strain used in the experiment. SAT2 viruses isolated from both buffaloes and cattle differed from the original clone and the nucleotide changes caused a significant change in antigenicity (Vosloo et al. 1996). These results may suggest that when mutation of viruses alters their antigenicity, the virus may be able to escape from the immune suppression exerted by persistently infected animals, thus allowing the virus to multiply more freely.

Overall it must be concluded that buffaloes that are persistent carriers are not highly infectious but are potential transmitters of infection and may occasionally transmit the disease to cattle. Antelopes probably only play a minor role in the maintenance of the disease but clinically infected impalas are infectious. It has also been suggested that the disease could be sexually transmitted from buffaloes to cattle (Bastos, Bertschinger, Cordel, Van Vuuren, Keet, Bengis, Grobler & Thomson 1999). The epidemiological evidence that buffaloes are the main factor in the persistence of the disease is strong (Dawe, Flanagan, Madekurozwa, Sorensen, Anderson, Foggin, Ferris & Knowles 1994).

One suggestion is that persistently infected buffaloes may transmit the infection to susceptible calves in the herd and the disease is then transmitted to other species when young calves are acutely infected. Other than buffaloes, the main species affected in the KNP are impalas, which are the most numerous susceptible animals and also live in herds thus increasing the chance for close contact. Impalas may be involved in spreading the virus to other antelopes and rarely to cattle. However, cattle do not appear to be efficient reservoir hosts of FMD despite the fact that they may carry the virus for a number of years (Thomson 1994).

Diagnosis

Clinical signs of infection are often typical and allow a diagnosis of the disease to be made. Virus isolation, reverse transcriptase polymerase chain reaction (PCR), antigen capture ELISA, and serology including ELISA and virus neutralization are used to

confirm the diagnosis and to identify the strains of virus involved (Donaldson & Kitchen 1996; Chilonda *et al.* 1999).

Rinderpest

Rinderpest is a highly virulent and contagious disease of artiodactylids. It is estimated that, in the great rinderpest epidemic in Africa from about 1889–1905, half of the 5.75 million head of cattle in South Africa died and the effect on wild ruminants was likewise devastating (Bigalke 1994; Vogel & Heyne 1996). Since that great pandemic, rinderpest in wildlife has only occurred in equatorial and East Africa (Anderson 1995). Following a successful campaign to eradicate it, the disease was brought under control in East Africa by the mid 1960s to mid 70s. However, following the breakdown in vaccination campaigns the disease again appeared in cattle in Uganda in 1979 and caused high mortality in buffaloes in Tanzania in 1982 (Rossiter, Jessett, Wafula, Karstad, Chema, Taylor, Rowe, Nyange & Mumbala 1983; Rossiter, Taylor, Bwangamoi, Ngereza, Moorhouse, Haresnape, Wafula & Gumm 1987; Rossiter 1994). Extensive vaccination prevented the spread of the disease southward from Tanzania. The disease continued to smoulder in buffaloes, with animals in the Masai Mara game reserve in Kenya and the Serengeti National Park in Tanzania developing antibodies but not frank disease, at least until 1987 (Anderson, Jage, Mlengeya, Timms, Payne & Hirji 1990; Rossiter 1994). At the same time the disease was causing mortality in cattle in north-eastern Uganda (Rossiter 1994). Following incursions of the disease from countries to the west and north, outbreaks occurred in Kenya in 1986 and 1987 (Wafula & Kariuki 1987). The disease then spread within Kenya in 1988–89 (Wamwayi, Kariuki, Wafula, Rossiter, Mbuthia & Macharia 1992). In Nigeria, after the disease had been eradicated in 1974, it was reintroduced in 1980 and 1983 and caused the death of an estimated one million cattle and mortalities in wildlife including buffaloes, warthogs, waterbuck and bushbuck (Shanthikumar, Malachi & Mayiyagbe 1985; Shanthikumar & Atilola 1990). Currently there are only four foci of rinderpest left in the world, namely southern Sudan, southern Somalia, Yemen and Pakistan.

Between 1993 and 1997 a severe epidemic of rinderpest occurred in wild animals in East Africa. Buffaloes, eland and lesser kudus were highly susceptible. The mortality rate in buffaloes was up to 80%. The virus caused only mild disease in cattle with few mortalities. The strain of virus isolated in this epizootic was related to a strain prevalent in Kenya in the 1960s. It was distinct from the isolations of virus in the regions that had occurred since 1986 (Kock, Wambua, Mwanzia, Rossiter, Wamwayi & Kock 1995; Kock, Wambua, Mwanzia, Wamwayi, Ndungu, Barrett, Kock & Rossiter 1999b; Kock, Kock, Wambua & Mwanzia 1999a).

Aetiological agent

The disease is caused by the rinderpest virus (family: Paramyxoviridae, genus: *Morbillivirus*). Strains may be variable in pathogenicity and many recently isolated strains are of low pathogenicity for cattle. Sequencing a region of the genome around the proteolytic cleavage site of the fusion protein has shown that there are two lineages of virus found in Africa. One line is apparently derived from a strain closely related to the Asian and Middle Eastern strains and includes strains isolated from East and West Africa during the 1960s. The other line consists of isolates from East, West and North Africa made between 1983 and 1993. Virus strains of both lineages circulated in Nigeria during the epizootic of the 1980s (Wamwayi, Fleming & Barrett 1995).

Susceptible species

Cattle are particularly susceptible, but the disease has also been described in sheep, goats, pigs, buffaloes, giraffes, eland and kudus (Rossiter 1994); buffaloes, waterbuck and bushbuck (Shanthikumar *et al.* 1985; Shanthikumar & Atilola 1990); kudus, buffaloes, impalas, eland and kongonis (Kock *et al.* 1995). Neutralizing antibody has also been found in waterbuck, oryxes and impalas (Wafula, Mushi & Karstad 1982). It must be assumed that all antelopes can be infected.

Incubation period and signs of the disease

In acute rinderpest the incubation period is usually 3–9 days in cattle (Scott 1990). Information for individual antelope species was not found. Fever, malaise, nasal discharge, ulceration of oral and nasal mucosa, diarrhoea and high mortality are typical of acute infections with virulent virus strains. However, mild and even asymptomatic infections are frequently seen in African cattle. Similarly infections in wild ruminants may vary from mild or inapparent disease to disease with a high mortality. In buffaloes mortality rates up to 80% have been described (Rossiter *et al.* 1983; Kock *et al.* 1999b).

Carrier state

No carrier state is known, although rare chronic cases occur.

Transmission

Transmission is by droplet infection over a short distance and by contact.

Epidemiology

Although the disease is highly infectious it spreads by contact and carrier animals are not known to occur. For these reasons the disease can be control-

led in livestock by controlling the movement of animals and by the use of vaccination. In less developed areas, however, where movement of livestock cannot be easily controlled and there is contact with wild ruminants, particularly buffaloes, the disease is less easy to control. It has re-emerged after many years in areas where extensive vaccination campaigns appeared to have been successful. It is considered possible that the disease could be eliminated by extensive vaccination and one authority has anticipated that global eradication will be complete by 2010. The disease is able to keep circulating in buffalo populations for at least several years as evidenced by the fact that it remained active in buffalo populations in northern Tanzania and southern Kenya from the early 1980s till at least 1987 (Anderson *et al.* 1990).

Diagnosis

Clinical signs of infection may be typical or inapparent. The disease can be confirmed in the laboratory by virus isolation, PCR, agar gel immunodiffusion for demonstration of antigen and serology including competitive ELISA and virus neutralization (Taylor 1996).

Rift Valley fever

Until recently Rift Valley fever has been confined to Africa and Madagascar and outbreaks have usually been associated with above average rainfall. The disease causes serious economic losses in livestock, and serious disease in some infected humans. An extensive review of the disease has been written by Swanepoel & Coetzer (1994).

Aetiological agent

The disease is caused by Rift Valley fever virus (family: Bunyaviridae, genus: *Phlebovirus*).

The South African virus isolates and strains from other parts of Africa were antigenically similar when analyzed by indirect immunofluorescence tests and neutralization tests, utilizing monoclonal antibodies prepared against the South African prototype virus. However, differences were demonstrated between wild type virus and vaccine strains (Besselaar, Blackburn & Meenehan 1991). Genetic variation between 22 strains collected over 34 years in six countries, showed a 0–4.5 % variation in nucleic acid sequence and a 0–2.4 % variation in amino acid sequence, in a portion of the M segment of the virus (Battles & Dalrymple 1988).

Susceptible species

Rift Valley fever is predominantly a disease of sheep and, to a lesser extent, cattle. The infection causes high numbers of abortions and prenatal deaths in lambs but a much lower mortality in calves. Abortions were described in springbok and blesbok during an outbreak of the disease in livestock. The aetiology of the abortions in springbok and blesbok was not proven, although circumstantial evidence indicated Rift Valley fever was a possible cause (Swanepoel & Coetzer 1994). Six of 50 pregnant buffaloes held in captivity in the KNP aborted and the virus was isolated from all six of them, the pathology being typical of a haemorrhagic disease, but there was no evidence of abortions in free-living buffaloes (Anon. 2000). Low levels of antibody have been found in a few species of antelope and in African buffalo (Swanepoel & Coetzer 1994).

During a serological survey for diseases of wildlife species in Zimbabwe, antibody to Rift Valley fever was found to be most prevalent in white rhinoceroses, buffaloes and waterbuck (Anderson & Rowe 1998). In humans it can cause a serious disease but has a low mortality rate.

Incubation period and signs of the disease

The incubation period in sheep is short, usually only about 1–2 days, and abortions and perinatal deaths in lambs commonly occur (Swanepoel & Coetzer 1994). The phenomenon of abortion is described in detail under "susceptible species".

Carrier state

During the viraemic period, that lasts a few days, very high titres of virus are found in the blood and virus persists in the organs, particularly the spleen, for up to 3 weeks. No long-term carrier state has been observed (Swanepoel & Coetzer 1994).

Transmission

Transmission is by mosquitoes. *Aedes caballus, Aedes circumluteolus/luteolateralis, Aedes juppi, Anopheles cinereus, Anopheles costani, Anopheles mcintoshi, Culex neavi, Culex theileri, Culex zombaensis* and *Eretmapodites quinquevittatus* have been reported as capable vectors of Rift Valley fever (Swanepoel & Coetzer 1994). *Aedes vexans* and *Aedes ochraceus* were described as new vectors in Mauritania (Fontenille, Traore-Lamizana, Thonnon, Digoutte & Zeller 1998) and *Aedes caspius, Culex pipiens, Culex antennatus* and *Culex perexiguus* have been implicated in Egypt (Turell, Presley, Gad, Cope, Dohm, Morrill & Arthur 1996). In Kenya, virus has been isolated from *Cx. zombaensis* and *Mansonia africana* (Logan, Linthicum, Davies, Binepal & Roberts 1991). In South Africa *Aedes unidentatus, Aedes dentatus* and *Culex poicilipes* are possible vectors (Jupp & Comel 1988). Virus has also been isolated from two *Aedes* spp. in Senegal (see "Epidemiology"). At least four species of Australian and ten species of North American mosquitoes have been

shown to be competent vectors, and could potentially act as vectors if the virus were introduced to those countries (Gargan, Clark, Dohm, Turell & Bailey 1988; Turell & Hay 1998). Clearly there must be many more species of mosquitoes that could be vectors. Reports on transovarial transmission of the virus are rare and it probably only occurs in a low number of cases (Swanepoel & Coetzer 1994). It could not be demonstrated for *Ae. juppi* (Gargan, Jupp & Novak 1988).

Other vectors include the sand flies, *Phlebotomus duboscqi, Phlebotomus papatasi* and *Lutzomyia longipalpis,* although there is no evidence of transovarial transmission and apparently no suggestion that they are important vectors in natural epidemics (Hoch, Turrell & Bailey 1984; Turell & Perkins 1990; Turell & Dickson 1992). *Culicoides variipennis* was shown to be only a transient carrier of the virus when infected experimentally (Jennings, Platt & Bowen 1982).

Epidemiology

Epidemics of Rift Valley fever are usually associated with periods of above average rainfall. In these periods the vector populations are able to increase and spread from the permanent water sites where they are normally maintained, to breed in surface water in normally dry areas (Swanepoel & Coetzer 1994). The mechanism for the survival of the virus from one epidemic to another is still unclear. The disease could be maintained as a persistent infection by some as yet unidentified carrier, persist at inapparent levels by circulation amongst the animals and mosquitoes around permanent water sources in dry periods or survive because of transovarial transmission of the virus in mosquitoes.

Although Rift Valley fever virus was recently isolated from buffaloes in the KNP, there is little evidence to implicate wild ruminants as a source of persistent infection. However, because of their close relationship to domestic ruminants they will remain a source for suspicion until the problem is solved. The occurrence of low levels of antibody in certain wildlife species has been referred to above. Outbreaks of abortion in cattle associated with Rift Valley fever virus have occurred in Madagascar, a country in which none of the African wild ruminants occur (Morvan, Rollin, Laventure, Rakotoarivony & Roux 1992). Other species in which the virus can cause infection, that have been suggested as potential carriers of virus, include bats (Boiro, Konstaninov & Numerov 1987; Oelofsen & Van Der Ryst 1999) and other small terrestrial vertebrates particularly the Namaqua rock rat (Pretorius, Oelofsen, Smith & Van Der Ryst 1997). If the problem is to be solved by identifying an animal species that acts as a persistent carrier of virus, it will have to be one that occurs wherever Rift Valley fever is endemic.

The virus has been isolated from *Ae. vexans* and *Ae. ochraceus* in inter-epizootic periods in Senegal (Fontenille, Traore-Lamizana, Zeller, Mondo, Diallo & Digoutte 1995). Antibody to the virus was demonstrated in sheep in South Africa during wet seasons when epizootics of disease did not occur (Van der Riet, Sayed, Barnard, Van Tonder & Crouse 1985). These findings may give some credence to the view that the virus circulates in domestic stock at low levels during inter-epidemic periods.

In Nigeria significantly higher numbers of people with antibodies were found in livestock workers and wildlife rangers than in other categories of the population. Whether the occurrence in game rangers indicates an association between the occurrence of the disease in humans and wild animals is not clear (Olaleye, Tomori, Ladipo & Schmitz 1996). In contrast, an association between the occurrence of disease in domestic stock and humans was clearly demonstrated (Ksiazek, Jouan, Meegan, Le Guenno, Wilson, Peters, Digoutte, Guillaud, Merzoug & Touray 1989; Wilson, Chapman, Hall, Dykstra, Ba, Zeller, Traore-Laizana, Hervy, Linthicum & Peters 1994).

Diagnosis

Rift Valley fever can be diagnosed in cattle and sheep by consideration of the clinical signs of infection and pathology, and confirmed by virus isolation and antibody tests such as virus neutralization and indirect ELISA (Barnard 1996).

Bluetongue

Bluetongue is a major viral disease that primarily affects sheep. It occurs in Africa, the Middle East, Asia, southern Europe, the United States of America and Australia. The causative virus is carried by *Culicoides* spp.. Verwoerd & Erasmus (1994) have reviewed the disease.

Aetiological agent: The disease is caused by the bluetongue virus (family: Reoviridae, genus: *Orbivirus*). Twenty-four antigenically different serotypes of the virus are recognized.

Susceptible species

Although bluetongue is a serious disease of sheep it has been suggested that they may be merely accidental or indicator hosts with the virus being maintained in well adapted African wildlife, cattle and goats (Erasmus 1975). Wild African ruminants do not develop clinical disease, but inapparent infections occur in many species (Verwoerd & Erasmus 1994). Non-African antelope and deer are susceptible to the disease (Hoff & Hoff 1976). Bluetongue virus has been isolated from addaxes, ibexes, African buffaloes and sable antelopes (Castro & Rodgers 1984), and pronghorn antelopes (Stott, Else, McGowan,

Wilson & Osburn 1981). Antibody to bluetongue virus has been found in a wide variety of African antelopes (Simpson 1979; Hamblin, Anderson, Jago, Mlengeya & Hirji 1990; Fomenty, Domenech, Lauginie, Outtara, Diawara, Raath, Grobler, Leforban & Angba 1994; Barnard 1997; Anderson & Rowe 1998). Experimental infection of blesbok resulted in asymptomatic infection (Neitz 1933). The virus has also been described as infecting domestic dogs, shrews and some rodents, and wild African carnivores (Alexander, Maclachlan, Kat, House, O'Brien, Lerche, Sawyer, Frank, Holekamp & Smale 1994).

Incubation period and signs of the disease

The incubation period is usually 4–6 days, but can vary from 2–15 days (Verwoerd & Erasmus 1994). In the sheep there is fever and inappetance, and oedema and hyperaemia of the subcutaneous tissues of the head, lips and other parts of the body. A swollen blue tongue, from which the disease gets its name, is an occasional sign and there are generally excoriations and erosions of the buccal and lingual mucosae. Painful muscles due to muscle degeneration may occur. Hyperaemia of the coronary region of the hoof and lameness is common. Wild African ruminants apparently always remain asymptomatic.

Carrier state

Animals carry the virus for short periods after they recover from infection. Cattle may carry the virus for at least 49 days (Verwoerd & Erasmus 1994). The periods during which the various antelope species remain viraemic are not generally known. Viraemia of 17 days in blesbok, 3 days in pronghorn antelopes and 35 days in mountain gazelles have been described (Hoff & Hoff 1976).

Transmission

The virus is carried by *Culicoides* spp. midges. Transovarial transmission does not occur. About 20 of the 1 400 known species of *Culicoides* are known to be competent vectors of bluetongue (Maclachlan, Wilson, Gard, Mello & Nevil 1998). The most important vector in Africa appears to be *C. imicola* (Verwoerd & Erasmus 1994)

Epidemiology

In the endemic areas of southern Africa bluetongue occurs in late summer and autumn before the first frosts when the concentration of the vector midges is high. It is not clearly understood how the virus overwinters in areas with long cold winters when midges are absent. It is possible that cattle or one or more African antelope species act as reservoirs, but this has not been finally proven. Where winters are mild, bluetongue virus may be transmitted throughout the

year. Recently bluetongue virus infection has been described in dogs in the United States of America and in carnivores in Africa (Alexander *et al.* 1994). It has been suggested that the effect of bluetongue virus on endangered carnivores requires to be investigated and the role of carnivores in the epidemiology of the disease elucidated (Alexander *et al.* 1994). As it is speculated that eating infected meat can infect carnivores, it seems unlikely that they are primary reservoirs of infection.

Diagnosis

The diagnosis depends on observation of typical clinical signs of infection and lesions, particularly haemorrhages in the wall of the pulmonary artery. Virus isolation in embryonated eggs, detection of viral RNA by reverse transcriptase PCR, antibody detection by agar gel immunodiffusion and competitive ELISA are used to confirm the diagnosis (Eaton 1996).

Rabies

Rabies is an invariably fatal, viral disease. It is carried by carnivores and transmitted to other animal species including ruminants, and humans generally, when infected carnivores bite them. The disease has been extensively reviewed (Swanepoel 1994a).

Aetiological agent

The disease is caused by the rabies virus (family: Rhabdoviridae, genus: *Lyssavirus*).

Susceptible species

Probably all mammals are susceptible.

Incubation period and signs of the disease

The incubation period varies depending on the bite site, infectious dose and probably the strain of virus. It can be from weeks to months. Six months is usually taken as the upper limit, but 611 days has been recorded (Baer 1990). Typical paralytic or aggressive (furious rabies) syndromes or unnatural behaviour are usually seen. Salivation and incoordination are common. In cattle there may be abnormal bellowing, salivation, aggression, incoordination or other nervous signs.

Carrier state

Infected carnivores ordinarily transmit the disease by bite, the virus being present in saliva. Only the Indian grey mongoose has been described as an asymptomatic carrier in Carribean countries where it had been introduced to control rats and snakes in sugar cane fields (Everard & Everard 1988 cited by Bigalke 1994 and by Swanepoel 1994a). Bats may be carriers of

the closely related European bat *Lyssavirus* (Peres-Jorda, Ibanez, Munoz-Cervera & Tellez 1995).

Transmission

Transmission is ordinarily by the bite of an infected animal.

Epidemiology

Rabies is generally transmitted by the bite of infected carnivores such as dogs, jackals, foxes, cats, mongooses or other carnivores. Ruminants are generally dead-end hosts. An exception was observed in Namibia where an apparently horizontally-sustained infection killed thousands of kudus between 1977 and 1983 (Barnard & Hassel 1981; Barnard, Hassel, Geyer & De Koker 1982; Hübschle 1988). It has been suggested that the disease may have been transmitted in these browsers by infected saliva contaminating leaves and twigs of thorn trees, following a disproportionate increase in the kudu population (Hübschle 1988). Other than in this exceptional case, ruminants are not thought to play any role in maintaining the disease.

Diagnosis

The diagnosis depends on the demonstration of virus by fluorescent antibody or peroxidase linked antibody tests, mouse inoculation and histological demonstration of Negri bodies in brain tissues (Aubert, Cliquet & Barrat 1996).

Lumpy skin disease

Lumpy skin disease causes serious economic loss during periodic epidemics in cattle in Africa. It has spread to the Arabian Peninsula, and occurred in Israel but was eradicated (Yeruham, Nir, Braverman, Davidson, Grinstein, Hamovitch & Zamir 1995). A comprehensive review of the disease has been written (Barnard, Munz, Dumbell & Prozesky 1994a).

Aetiological agent

The disease is caused by the lumpy skin disease virus (family: Poxviridae, genus: *Capripox virus*).

Susceptible species

Cattle, giraffes and impalas are highly susceptible to experimental infection (Young, Basson & Weiss 1970), but two buffalo calves and adult blue wildebeest failed to react clinically (Barnard *et al.* 1994a). The disease has been described in a captive-bred Arabian oryx at the National Wildlife Research Centre in Saudi Arabia. Two percent of the oryxes in the herd were serologically positive (Greth, Gourreau, Vassart, Nguyen-Ba-Vy, Wyers & Leferve 1992).

Antibodies to the virus have been demonstrated in buffaloes (Davies 1982) and in six other species of free-living wild ruminants (Hedger & Hamblin 1983). Generally the prevalence of antibody in wildlife species was found to be relatively low (Barnard 1997) and in some investigations no antibody was found (Hamblin *et al.* 1990).

Incubation period and signs of disease

The incubation period in cattle is about 5 days. The initial signs include fever, inappetance and enlarged superficial lymph glands. Typical large lumps develop in the skin a few days after the initial fever. The lumps often cover the whole body. In some animals focal lesions are found in the trachea, alimentary tract, particularly the abomasum, and other internal organs. Oedema of a limb or limbs occurs in severely affected cases.

Carrier state

No carrier state is known to exist. Viraemia persists for about 4 days and in cattle the virus can persist in skin nodules for 33 days, in the semen for 22 days and saliva for 11 days (Weiss 1968 cited by Barnard *et al.* 1994a)

Transmission

The mode of transmission has not been established but the pattern of infection indicates that the disease is spread by biting insects. There is some evidence, however, that disease may spread in the absence of insects (Barnard *et al.* 1994a). It has been suggested, on the basis of circumstantial evidence, that *Stomoxys calcitrans* carried by the wind from Northern Sinai or the Nile delta may have introduced the infection into Israel (Yeruham *et al.* 1995).

Epidemiology

In Africa the disease occurs in the form of periodic epidemics. Despite the evidence implicating insects in the transmission, major outbreaks of disease did not occur in the particularly wet seasons in which outbreaks of Rift Valley fever occurred and there is no evidence that ticks act as vectors. Antibodies have been found in African buffaloes (Davies 1982) and the suggestion has been made that the maintenance cycle of the virus may involve buffaloes. However, other authors have found a low prevalence or absence of antibodies in wild ruminants. The mechanism for the persistence of the disease remains unsolved. It is, however, clearly a peculiarly African disease, which causes major outbreaks in cattle but has little effect on wild African ruminants under natural conditions. This suggests that the virus is well adapted to wildlife and they will remain under suspicion until the problem has been resolved.

Diagnosis

The clinical picture and pathology are typical. Definitive confirmation is provided by virus isolation or its demonstration by electron microscope, identification of antigen by fluorescent antibody, antigen capture ELISA and serological tests such as virus neutralization (Kitching & Carn 1996).

Malignant catarrhal fever

Malignant catarrhal fever is a serious, invariably fatal viral disease of cattle and deer that is usually related to contact between cattle and blue or black wildebeest or sheep. An extensive review of the present knowledge of the disease is available (Barnard, Van der Lugt & Mushi 1994b).

Aetiological agent

The disease associated with the presence of wildebeest is caused by acelaphine herpesvirus1 [family: Herpesviridae, acelaphine herpesvirus 1 (AHV-1)]. In addition to AHV-1, a closely related herpesvirus apparently carried by sheep [ovine herpesvirus 2, (OHV-2)] commonly causes a similar disease in cattle and deer. Isolates from hartebeest and topis showed little nucleic acid homology with typical AHV-1 isolates (Seal, Klieforth & Heuschele 1987; Seal, Heuschele & Klieforth 1989). Isolates from topis and hartebeest have been designated alcelaphine herpesvirus 2 (AHV-2). They produced only atypical malignant catarrhal fever in cattle after artificial infection and it could not be transferred by natural transmission (Barnard et al. 1994b). Other closely related herpesviruses may occur in other antelopes (Hamblin & Hedger 1984; Lahijani, Sutton, Klieforth, Murphy & Heuschele 1994). An isolate from a roan antelope was provisionally designated as being hippotragine herpesvirus 1 (HHV-1)(Gulland, Reid, Buxton, Lewis, Kock & Kirkwood 1989; Reid & Bridgen 1991). AHV-1 can be cultured but attempts to culture OHV-2 are invariably unsuccessful and OHV-2 is demonstrated by inoculation of rabbits or by PCR (Michel 1993; Tham, Ng & Young 1994).

Susceptible species

Cattle are highly susceptible to the disease. In zoos the disease has been reported in kudus, situtangas, eland and roan (Barnard et al. 1994b) and in duikers and gerenuks (Meteyer, Gonzales, Heuschele & Howard 1989) but it is not seen in wild antelopes. However, antibodies have been found in a wide range of antelopes including nyalas, roan, sables, tsessebes, waterbuck, gemsbok, red hartebeest, and black and blue wildebeest (reviewed by Barnard et al. 1994b); oryxes, topis, addaxes (Li, Shen, Jessup, Knowles, Gorham, Thorne, O'Toole & Crawford 1996); lechwes, reedbuck (Hamblin & Hedger 1984).

Antibodies formed in response to OHV-2 cross react with antibody formed against AHV-1 and it is not certain if OHV-2 plays a role in any of the antelope species. The reason why some antelope species are susceptible to infection with AHV-1 in zoos but do not appear to be susceptible in the wild is not known.

Incubation period and signs of the disease

The incubation period is usually about 3–7 weeks but can be up to 73 days (Plowright 1990). In cattle the disease is characterized by fever, inappetance, mucopurulent nasal discharge (snotsiekte), corneal opacities, erosions of buccal and nasal mucosa, enlarged lymph nodes, and sometimes diarrhoea.

Carrier state

Blue and black wildebeest commonly carry the AHV-1 (Castro, Ramsay, Dotson, Schramke, Kocan & Whitenack 1984; Barnard et al. 1994). Barnard et al. (1994b) have suggested that other members of the sub-families Alcelaphinae and Hippotraginae may also carry the infection.

Transmission

The disease is transmitted by contact or over short distances between wildebeest and susceptible animals (Castro et al. 1984; Barnard et al. 1994b). It has been suggested that separation of wildebeest and cattle by a distance of 1 km is needed to avoid infection (Barnard et al. 1994b). In one study of 37 herds of cattle and five groups of wildebeest the highest incidence of infection was in cattle kept in camps separated from wildebeest by approximately 100 m (Barnard, Van der Pypekamp & Griessel 1989). An attempt to transmit the disease with an African face fly (Musca xanthomelas) was unsuccessful (Barnard, Bengis & Voges 1990). It has also been postulated that infected cows may transmit the disease to their calves and that intrauterine infection may be possible (Barnard 1990).

Epidemiology

On cattle farms and in zoos, outbreaks of disease usually correlate with contact with wildebeest (Meteyer et al. 1989; Barnard et al. 1994b) or with contact with sheep (Erasmus 1986). There is no suggestion that wild ruminants are involved in the transmission of the OHV-2. Excretion of AHV-1 is generally limited to young calves under the age of 4 months (Barnard, Bengis, Griessel & De Vos 1989). In these animals virus can be recovered from tears, blood and nasal mucus (Mushi, Karstad, Jessett 1980; Mushi, Rossiter, Karstad & Jessett 1980; Barnard et al. 1989a) but not from saliva or urine (Mushi et al. 1980a, b). However, there is a difference

between the epidemiology of the disease in South and East Africa. In East Africa, cattle are usually infected when wildebeest calves are 3–4 months old and in South Africa when they are 8–10 months old (Barnard *et al.* 1989; Barnard & Van der Pypekamp 1988). These findings have lead Barnard to postulate that other not yet identified factors, such as another host or intermediate host, may be important in South Africa (Barnard *et al.* 1989b). Other related viruses such as AHV-2 and HHV-1 and possibly other related herpesviruses occur in species other than wildebeest. AHV-1 and bovine cytomegalovirus (BHV-3) are serologically and immunogenically related and BHV-3 occurs in buffaloes (Rossiter, Gumm & Mirangi 1988; Rossiter, Gumm, Stagg, Conrad, Mukolwe, Davies & White 1989).

Diagnosis

A presumptive diagnosis can be made from the typical clinical picture or from the histopathology. AHV-1 can be identified by isolation of virus and AHV-1 and OHV-2 can be demonstra⁺ed by the PCR for amplification of viral DNA (Michel 1993; Tham *et al.* 1994). Serological tests such as virus neutralization and immunoperoxidase tests are useful for the demonstration of antibody for epidemiological studies (Reid 1996).

Ephemeral fever

Ephemeral fever is caused by an arbovirus. It is usually a non-fatal disease of cattle, but it causes significant production losses particularly in dairy cattle.

Aetiological agent

The disease is caused by the ephemeral fever virus (family: Rhabdoviridae, genus: *Lyssavirus* of the bovine ephemeral fever serogroup).

Susceptible species

Cattle are susceptible to the disease. Antibody to the virus was found in several antelope species (Hamblin *et al.* 1990; St. George 1994; Barnard 1997). In another study antibody was widespread in 16 species of wildlife including buffaloes, eland, nyalas, waterbuck and bushbuck (Anderson & Rowe 1998).

Incubation period and signs of the disease

The incubation period in cattle varies from 2–10 days (St. George 1994). The disease is characterized by a phasic fever with two or more peaks. Other signs of infection include inappetance and a stiff painful gait (3-day stiff sickness). Recovery usually takes place after a few days. Occasionally infected animals become recumbent and do not rise; these cases often end fatally.

Carrier state

No carrier state is known. In cattle viraemia lasts 1–3 days with a maximum of 2 weeks (St. George 1994).

Transmission

The disease is transmitted by *Culicoides* spp. and probably other biting insects (St. George 1994; Nandi & Negi 1999).

Epidemiology

In South Africa it is a disease of the summer and autumn when *Culicoides* and other biting insects are present in high numbers. It occurs in some seasons and is virtually unknown in others. In Kenya it occurs in seasons of heavy rainfall often coinciding with epizootics of Rift Valley fever (Davies, Ochieng & Walker 1990). In temperate climates, the disease disappears after the first frosts. The natural reservoir of the virus is unknown. As buffaloes and several species of antelopes have antibodies to the virus, it is possible that one or more of these species could be a reservoir species. However, the disease is also widespread in Asia and Australia, mostly in the subtropical and temperate regions (Nandi & Negi 1999). In some of these countries, most notably Australia, the fauna is markedly different from that in Africa, which indicates that wild African animals are not essential for the maintenance of the disease. Alternative possibilities for the mechanism for the persistence of the virus are similar to those for Rift Valley fever.

Diagnosis

The disease may be suspected from the typical symptoms and the diagnosis confirmed by virus isolation and positive serology (St. George 1994).

Nairobi sheep disease

Nairobi sheep disease is a tick-borne disease of sheep occurring mainly in East Africa. It may be zoonotic as it is believed to have been transmitted to a laboratory worker (Terpstra 1994).

Aetiological agent

The disease is caused by Nairobi sheep disease virus (family: Bunyaviridae, genus: *Nairovirus*).

Susceptible species

Sheep are highly susceptible and the disease has been described in the blue duiker (Terpstra 1994). No references were found to the disease in other antelope species. Only low levels of antibody, which was considered to be possibly cross reacting antibody, was found in antelope (Davies 1978).

Incubation period and signs of the disease

The incubation period is 4–6 days and the disease in sheep is characterized by enteritis and high mortality (Davies 1997).

Carrier state

Viraemia is thought to last only as long as the animal remains febrile, which is 1–7 days. No carrier state has been described (Terpstra 1994).

Transmission

The virus is transmitted by *Rhipicephalus appendiculatus,* in which species transovarial transmission of the virus occurs (Davies 1997). Other members of the *Rhipicephalus* genus and *Amblyomma variegatum* may also transmit the virus but transoviarial transmission does not occur in these ticks (Terpstra 1994).

Epidemiology

It is a tick-borne disease causing sporadic outbreaks of disease following the introduction of infected animals and vectors. There is no evidence to incriminate wild ruminants as significant reservoirs of infection.

Diagnosis

The disease can be diagnosed by virus isolation and antibody demonstration using the indirect fluorescent antibody test (Davies 1996).

Crimean-Congo disease

Crimean-Congo disease is a zoonotic disease that causes sporadic cases of serious, sometimes fatal haemorrhagic fever in humans.

Aetiological agent

The disease is caused by the Crimean-Congo haemorrhagic fever virus (family: Bunyaviridae, genus: *Nairovirus*).

Susceptible species

A wide range of animals and birds (including ostriches) and man can be infected. Clinical disease has not been described in ruminants. Antibody has been found in giraffes and antelopes, especially the larger species such as kudus and eland, and buffaloes (Swanepoel 1994b). Ostriches (Swanepoel, Leman, Burt, Jardine, Verwoerd, Capua, Brückner & Burger 1998) and ground-feeding birds have been experimentally infected. A possible cycle of infection between ticks and ground feeding birds has been suggested (Zeller, Cornet & Camicas 1994). In the Crimea, four species of ticks and shrews, voles,

wood mice and European brown hares may be involved in the cycle of infection (Markeshin, Smirnova & Evestaf'ev 1992).

Incubation period and signs of the disease

The incubation period is usually 1–3 days, sometimes up to 7 days. The infection causes haemorrhagic fever in man with a mortality of about 30 %. In livestock and antelopes the infection is asymptomatic.

Carrier state

Cattle only carry the virus for short periods. There is no information about a carrier state in antelopes. Ostriches only carry the infection for a few days (Swanepoel *et al.* 1998).

Transmission

Ticks of the genus *Hyalomma* are disease vectors in Africa (Swanepoel 1994b). There is some evidence that the infection can be transovarially transmitted in ticks (Zeller *et al.* 1994). The disease can also be transmitted from infected carcasses of cattle (Swanepoel, Shepherd, Leman, Shepherd & Miller 1985) and ostriches (Swanepoel *et al.* 1998) to people involved in butchering of carcasses. Incidental infection of humans while butchering wild ruminants is therefore also a possibility. The spread of the disease as a nosocomial infection in a hospital has also been reported (Van Eeden, Joubert, Van De Wal, King, De Kock & Groenewald 1985).

Epidemiology

It is a sporadically occurring tick-borne disease in man. Infected antelopes and other wild ruminants apparently remain asymptomatic. *Hyalomma marginatum* and *Hyalomma truncatum* were studied in a nature reserve, in South Africa, where the disease had occurred in a child (Rechav 1986). It was found that immature stages of the ticks feed on hares, rodents and guinea fowl in the winter, while adults feed on wildebeest, blesbok and springbok during the summer. Since the infection is apparently maintained in a rodent/tick cycle in the Crimea, it is possible that in South Africa small rodents may be carrying the disease. Man, cattle and wild ruminants may be incidentally infected. However, there is a high prevalence of antibodies in cattle in South Africa and in Zimbabwe (Swanepoel, Shepherd, Leman, Shepherd, MacGillivray, Erasmus, Searle & Gill 1987) and a possible role for ruminants in maintaining the infection cannot be ignored.

Diagnosis

The diagnosis can be confirmed by virus isolation and virus neutralization tests (Swanepoel 1994b).

Bovine virus diarrhoea

Bovine virus diarrhoea (BVD) virus occurs universally in cattle and although infections are usually asymptomatic it causes sporadic cases of mucosal disease and foetal deaths.

In sheep, a closely related pestivirus causes hairy shaker disease. The disease has been reviewed (Harkness & Van der Lugt 1994).

Aetiological agent

The disease is caused by the bovine virus diarrhoea (BVD) virus (family: Togaviridae, genus: *Pestivirus*).

The BVD viruses of cattle and the Border disease virus of sheep are similar but distinct viruses. In cattle two distinct pestiviruses occur. Type 1 has been well known for many years and occurs very commonly all over the world. Type 2 has been described more recently and is of higher virulence. Strains of pestivirus may be cytopathogenic or non-cytopathogenic (Ellis, West, Cortese, Myers, Carman, Martin & Haines 1998). The pestivirus of pigs is the cause of classical swine fever but is probably of no importance in wild ruminants.

Susceptible species

Of the domestic animals, cattle, sheep, pigs and deer may be infected with BVD virus. A wide range of antelopes have antibodies to pestivirus, including roan antelopes, wildebeest, oryxes, kudus, sables and giraffes (Depner, Hübschle & Liess 1991; Soine, Uatanaua & Depner 1992), wildebeest and topis (Hyera, Liess, Anderson & Hirji 1992), scimitar horned oryx (Frölich & Flach 1998), eland, nyalas and bushbuck (Anderson & Rowe 1998).

Antibodies have also been found in captive ruminants including antelopes and giraffes (Doyle & Heuschele 1983; Doyle, Heuschele & Fowler 1983). BVD virus has been isolated from eland (Anderson & Rowe 1998) and from giraffes, buffaloes and wildebeest (Nettleton 1990).

Incubation period and signs of the disease

As many animals do not show any signs of infection, the incubation period is hard to define. Antibodies develop 16–28 days after infection.

Carrier state

A persistent carrier state occurs in cattle and sheep that have been infected *in utero*. Sheep that are persistent carriers may also show signs of hairy shaker disease. These carrier animals are usually serologically negative (Harkness & Van der Lugt 1994). An antibody negative carrier state has also been described in eland (Anderson & Rowe 1998).

Transmission

Transmission occurs by contact between animals and congenitally.

Epidemiology

Most infections are asymptomatic. Depending on the stage of pregnancy at which the infection occurs, infection of pregnant cows may lead to foetal death and abortion or to the development of antibody negative, persistently infected calves (Harkness & Van der Lugt 1994). A persistently infected eland was found amongst 303 antibody negative eland (Anderson & Rowe 1998). Mucosal disease in cattle occurs when antibody negative carrier animals are re-infected by a strain of cytopathogenic pestivirus. The second virus must be antigenically similar to the strain of non-cytopathic virus they are already carrying. The virus can clearly be carried by a wide variety of ruminants and the persistent carrier state has been demonstrated in an eland, but mucosal disease has not yet been described in wild ruminants. Infection with Type 1 virus is widespread in cattle, in virtually all countries, and can clearly be maintained in cattle in the absence of wild ruminants. Therefore, it is doubtful if wild ruminants play a significant role in spreading or maintaining the disease in domestic stock. At present there seems to be no evidence that African wild ruminants are carriers of the more highly virulent Type 2 strain of BVD.

Diagnosis

The infection can be diagnosed by virus isolation, antibody capture ELISA, reverse transcriptase PCR, and serological tests such as virus neutralization tests or ELISA (Brownlie & Edwards 1996; Nettleton 1996).

VIRUS DISEASES OF MINOR IMPORTANCE

Antibody to infectious bovine rhinotracheitis (IBR) virus (bovine herpes virus-1) occurs in antelopes and buffaloes (Doyle *et al.* 1983; Doyle & Heuschele 1983; Hamblin *et al.* 1990; Anderson & Rowe 1998). Three types of IBR virus are known but there is only one antigenic type (Van Oirschot 1996). IBR virus occurs world-wide and is maintained in cattle populations that have no contact with wild ruminants. Other ruminants are believed to play no significant role in spreading the disease to domestic cattle (Van Oirschot 1996). A genital form of IBR can cause mild signs of infection in wildebeest. It has been transmitted to them and latent infections were reactivated with cortisone treatment (Mushi & Karstad 1979; Mushi, Karstad, Jessett & Rossiter 1979).

Enzootic bovine leukosis antibodies occur in antelopes and buffaloes (Hamblin *et al.* 1990). The virus

is readily maintained by cattle and close contact is required for its transmission. Therefore, wild ruminants are unlikely to be significant in transmitting the disease to cattle.

Antibody to Akabane disease and related simbu viruses occur commonly in many species of antelope (Al-Busaidt, Hamblin & Taylor 1987; St George & Standfast 1994). However, the virus occurs widely in Asia and Australia, where suitable *Culicoides* spp. vectors occur, and wild ruminants are unnecessary for the persistence of the virus in these cattle populations.

PROTOZOAL DISEASES

Babesioses

Babesiosis is a tick-borne disease that is one of the important diseases of African livestock. The disease has been extensively reviewed by De Vos & Potgieter (1994).

Aetiological agents

Babesiosis is a complex of diseases caused by a number of protozoal parasites including *Babesia bovis*, *Babesia bigemina*, *Babesia occultans*, *Babesia major*, *Babesia ovata*, and *Babesia divergens* in cattle; *Babesia motasi* and *Babesia ovis* in small ruminants; and *Babesia irvinesmithi* in sable antelopes.

Susceptible species

Babesia irvinesmithi has been found in sable antelopes (Thomas, Wilson & Mason 1982) and was responsible for an outbreak of babesiosis in captive sable antelopes (McInnes, Stewart, Penzhorn & Meltzer 1991).

Babesia bigemina has been found in a sable antelope (Hove, Sithole, Munodzana & Masaka 1998) and transmitted to calves with ticks from the sable antelope. However, the finding should be regarded with some suspicion since the ticks involved could already have been transovarially infected with the organism prior to feeding on the sable antelope. Attempts to infect sable antelopes with *B. bovis* and *B. bigemina* were unsuccessful (Thomas *et al.* 1982). *Babesia bigemina* did not establish in splenectomized and non-splenectomized eland, but experimentally infected buffaloes became carriers for at least 5 months (Schreuder, Uilenberg & Tondeur 1977; Karbe, Grootenhuis, Kelley & Karstad 1979).

A large *Babesia* was found in the erythrocytes of a blue wildebeest and a small *Babesia* associated with erythrocyte dyscrasia was found in a tsessebe (Carmichael & Hobday 1975). *Babesia bovis* has been found in blood smears from asymptomatic Uganda kobs (Kupper, Wolters & Tscharf 1983). *Babesia*

occultans and a hitherto unnamed bovine species are apparently harmless in cattle (De Vos & Potgieter 1994). *Babesia major* is found in cattle in southern Europe and North Africa. *Babesia divergens* is important in northern Europe and *B. ovata* occurs in Japan and Asia. *Babesia motasi* is a parasite of small ruminants and not a cause of disease in Africa. No reference was found to any identified pathological species other than *B. bovis*, *B. bigemina* and *B. irvinesmithi* being found in African wild ruminants. However, because of the difficulties in unravelling the taxonomy and identity, some of the information in published papers may be questionable.

Incubation period and signs of the disease

In cattle the incubation period is from 7–21 days. It is slightly longer for *B. bovis* than for *B. bigemina* (De Vos & Potgieter 1994). The disease is characterized by fever, inappetence, anaemia, icterus, haemoglobinaemia and haemoglobinuria. Cerebral babesiosis, manifested by a variety of associated signs, develops in some *B. bovis* infections. Infection with *B. bovis* causes a high mortality in European breeds of cattle (De Vos & Potgieter 1994). Buffaloes showed no signs of the disease when artificially infected (Karbe *et al.* 1979) and there are no reports of naturally occurring disease in these species. Sable antelopes imported from a zoo in Germany into South Africa developed a disease characterized by a massive haemolytic crisis associated with *B. irvinesmithi* (McInnes *et al.* 1991).

Carrier state

European breeds of cattle may carry *B. bovis* for long periods and generally for life and remain infective for ticks for up to 2 years. They carry *B. bigemina* for at least a year but are infective for ticks for only about 4–7 weeks (De Vos & Potgieter 1994).

Indigenous African cattle tend to carry infections for shorter periods. Buffaloes can be carriers of *B. bigemina* for at least 5 months (Karbe *et al.* 1979; Schreuder *et al.* 1977). Antelopes may have antibodies against the parasites but there is no evidence that they are carriers of the pathogenic babesias (De Vos & Potgieter 1994). However, *B. bovis* was reported to have been found in blood smears from asymptomatic Uganda kobs (Kupper *et al.* 1983).

Transmission

Boophilus microplus, *Boophilus decoloratus*, *Boophilus annulatus* and probably *Boophilus geigyi* carry *B. bigemina*. *Babesia bovis* is transmitted by *Boophilus microplus* but not by *B. decoloratus* (Jongejan & Uilenberg 1994). *Rhipicephalus evertsi* carries *B. bigemina*. *Babesias* are transmitted transovarially at least in *Boophilus* spp.

Epidemiology

Babesiosis is a typical tick-borne disease. Cattle that recover from the infection may become long-term immune carriers particularly of *B. bovis*. Serologically positive cattle should be assumed to be carriers of the disease. Buffaloes may carry *B. bigemina* for at least 5 months and can infect ticks. No evidence was found that antelopes are significant carriers of infection. Artificially infected splenectomized and non-splenctomized eland did not become carriers of the disease (Karbe *et al.* 1979). Attempts to transmit *B. bovis* and *B. bigemina* to sables were unsuccessful (Thomas *et al.* 1982). Babesias have occasionally been found in blood smears from wild antelopes (Carmichael & Hobday 1975; Kupper *et al.* 1983) but these parasites have not been rigorously identified.

Diagnosis

The parasite can be identified in blood smears from clinical cases, but it is rarely possible to identify the parasite in blood smears from carrier animals. An ELISA is available for *B. bovis* antibody identification but not for *B. bigemina*. Indirect fluorescent antibody tests are available for both species (De Vos & Jorgensen 1996). In cases of doubt, *Babesia* carriers can be identified by inoculating blood into splenectomized calves.

Theileriosis

Theileria parva (East Coast fever, Corridor disease and Zimbabwe theileriosis) and *Theileria annulata* (Mediterranean Coast theileriosis) cause diseases of major economic importance. Other species of *Theileria* are of low pathogenicity to domestic stock. Detailed reviews of the diseases are available (Lawrence, De Vos & Irvin 1994a, b, c, d; Pipano 1994). A closely related organism, *Cytauxzoon*, causes disease in some antelopes, but attempts to transmit the theilerial piroplasms to cattle by subinoculation of blood were unsuccessful (Thomas *et al.* 1982).

Aetiological agents and susceptible species

Some *Theileria* spp. and the diseases they cause are listed below:

- *Theileria parva* — East Coast fever in cattle, benign in buffaloes (Lawrence *et al.* 1994a)
  - Corridor disease in cattle. Buffaloes are asymptomatic carriers of the organisms (Lawrence *et al.* 1994b)
  - Zimbabwe theileriosis in cattle (Lawrence *et al.* 1994c)
- *Theileria annulata* — Mediterranean Coast fever in cattle (Pipano 1994)
- *Theileria mutans* — benign infestation of cattle and buffaloes
- *Theileria velifera* — benign infestation of cattle and buffaloes
- *Theileria orientalis* — benign infestation of cattle in Asia, New Zealand, Australia. Probably synonymous with *Theileria sergenti*
- *Theileria separata* — benign infection of sheep
- *Theileria taurotragi* — found in eland. It causes a usually benign infection in cattle but rare cases of turning sickness occur (Lawrence *et al.* 1994d)
- *Cytauxzoon* — occasionally causes disease in tsessebes, kudus, duikers, roan antelopes and giraffes (Thomas *et al.* 1982)

Theilerial piroplasms are commonly found in the blood of antelopes and buffaloes (Burridge 1975; Carmichael & Hobday 1975; Thomas *et al.* 1982). Recently PCR methods and oligonucleotide probing have been used to identify four antigenically different species of *Theileria* in the blood of buffaloes in the KNP. The species are *T. parva*, *T. mutans*, *Theileria buffeli* and an unidentified species (Allsopp, Theron, Coetzee, Dunsterville & Allsopp 1999).

Incubation period and signs of the disease

The incubation period for East Coast fever in cattle is 8–25 days (Lawrence *et al.* 1994a). East Coast fever is characterized by enlarged lymph nodes, anaemia, increased respiratory rate, dyspnoea and sometimes diarrhoea. There is a high mortality rate with East Coast fever and Mediterranean Coast fever in cattle. In cattle, *T. taurotragi* may cause occasional cases of turning sickness also referred to as cerebral theileriosis, a form of the disease where lymphoblasts containing schizonts block capillaries in the brain. *Theileria taurotragi* and *T. mutans* were probably confused in the past.

Carrier state

Buffaloes have been shown to remain carriers of particularly Corridor disease for at least 5 years. Carriers of *T. parva* are not known in antelopes. *Theileria taurotragi* and *Cytauxzoon* are probably carried by their antelope hosts for many years.

Transmission

Ticks of the genus *Rhipicephalus,* particularly *R. appendiculatus,* are the main vectors of East Coast fever and other related theilerioses (Lawrence *et al.* 1994a, b).

Epidemiology

East Coast and Mediterranean Coast fevers are tick-borne diseases. Buffaloes act as reservoirs for the pathogenic *T. parva* parasites and transmit them to ticks and thus to cattle. Buffaloes in the KNP in South Africa are infected with four species of *Theileria,* including an unnamed species (Allsopp *et al.* 1999).

Attempts to transmit theilerias of wildebeest, eland and sable antelopes to cattle failed (Burridge 1975; Thomas *et al.* 1982) and antelopes are generally not considered to be important in the epidemiology of *T. parva* infections (Lawrence *et al.* 1994a). However, Stagg, Bishop, Shaw, Wesonga, Orinda, Grooten-huis, Molyneux & Young (1994), infected waterbuck with *T. parva* sporozoites derived from ticks fed on buffaloes. The waterbuck reacted mildly to the infection but were able to infect ticks, which in turn infected cattle. Characterization with monoclonal antibodies and restriction fragment length polymorphism studies on DNA, showed that the waterbuck passaged strain differed from buffalo-derived *T. parva*. It was concluded that a minor parasite population had been selected from the buffalo-derived stock during passage in waterbuck (Stagg *et al.* 1994). Buffaloes may play a small role in maintaining East Coast fever and a major role as maintenance host for Corridor disease (Lawrence *et al.* 1994a).

Theileria taurotragi is found in eland and is a mild infection in cattle except in occasional cases where it causes turning sickness (Lawrence *et al.* 1994d).

Cytauxzoonosis occurs sporadically in some species including tsessebes, kudus, duikers, roan antelopes and giraffes (Carmichael & Hobday 1975; Thomas *et al.* 1982).

Diagnosis

Identification of the schizonts in lymph node smears or "small piros" in blood smears can confirm the diagnosis. The indirect fluorescent antibody test can be used to identify antibodies (Dolan, Moizaria & Katende 1996).

Tsetse fly-transmitted trypanosomosis

Trypanosomosis or nagana is mainly restricted to the areas of Africa where tsetse flies (*Glossina* spp.) occur. It is a major disease that has prevented cattle farming in tsetse fly-infested areas (Bigalke 1994; Connor 1994).

Aetiological agent

The pathogenic *Trypanosoma* spp. are *Trypanosoma brucei*, *Trypanosoma congolense* and *Trypanosoma vivax*. *Trypanosoma theileri* is a non-pathogenic, non-tsetse-borne species. The species pathogenic for man, *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense,* are of no importance in domestic stock and are not part of this review although they may be harboured by antelopes (Songa, Hamers, Rickman, Nantulya, Mulla & Magnus 1991; Guedegbe, Verhulst, Van Meivenne, Pandley & Doko 1992).

Susceptible species

Cattle are susceptible while buffaloes and a variety of antelopes carry the parasites (Drager & Mehlitz 1978; Awan 1979; Dillman & Townsend 1979; Kupper *et al.* 1983; Kariuki, Injairo, Boyce, Wellde & Ngethe 1989; Mattioli, Jean & Belem 1990; Bigalke 1994; Connor 1994; Truc, Fomenty, Komoin, Diallo & Lauginie 1997; Moloo, Orinda, Sabwa, Minja & Masake 1999). Sheep and goats are also susceptible (Connor 1994).

Incubation period and signs of the disease

The parasites can be found in the blood about 2 weeks after an animal has been bitten by an infected tsetse fly (Connor 1994). It usually presents as a chronic wasting disease. Animals become weak and anaemic with oedema of the limbs and a gradual loss of condition.

Carrier state

Antelopes and buffaloes act as asymptomatic carriers of the parasites, for example kob (Kupper *et al.* 1983); roan antelopes, buffaloes, kob, waterbuck (Truc *et al.* 1997); kob, kongonis, sable antelopes, waterbuck (Mattioli *et al.* 1990); oribis, reedbuck (Kariuki *et al.* 1989); waterbuck (Dillman & Townsend 1979); waterbuck, giraffes (Awan 1979); buffaloes, lechwes, kudus, impalas, tsessebes, sable antelopes (Drager & Mehlitz 1978; Moloo *et al.* 1999).

Transmission

Transmission is by the bite of an infected tsetse fly, particularly *Glossina morsitans*, *Glossina pallidipes*, *Glossina brevipalpis*, *Glossina austeni* and *Glossina longipennis* (Connor 1994; Moloo *et al.* 1999). Although mechanical transmission by biting flies is possible, they play no part in the maintenance of the disease caused by *T. congolense* or *T. brucei*. They play a more important role in the transmission of *T. vivax* and infection with this parasite has become endemic in South and Central America, and the island of Mauritius, in the absence of tsetse flies (Connor 1994).

Epidemiology

African trypanosomosis is carried by tsetse flies in association with infected game and is restricted to

the endemic tsetse fly areas. *Theileria vivax,* however, is also found in South America and Mauritius (Connor 1994) in the absence of tsetse flies. In South America the disease occurs mainly near low lying swampy areas and may be associated with tabanids as mechanical vectors (Mateus & Gonzalez 1991; Otte, Abuabara & Wells 1994). In most of Africa *T. vivax* does not occur outside the tsetse fly areas and it has not spread to other parts of the world where tabanids occur. The factors that allowed the establishment of *T. vivax* in South America and Mauritius are not known.

Diagnosis

Wet blood films and thick and thin blood smears may be examined to identify the parasite. The parasites in blood can be concentrated by centrifugation in a microhaematocrit tube and the buffy coat region examined by phase contrast or dark field microscopy. Antibody can be detected by ELISA (sensitive but lacking in specificity) or an indirect fluorescent antibody test (Schlater 1996).

Besnoitiosis (elephant-skin disease)

Besnoitiosis is a disease that affects cattle, goats and horses. It occurs most commonly in the subtropical areas of Africa and occurs more rarely in non-tropical regions. It also occurs in South Korea, Israel, Portugal, France, Venezuela and Russia (Bigalke & Prozesky 1994).

Aetiological agent

The agent of the bovine disease is a protozoan parasite, *Besnoitia besnoiti,* and that of equids *Besnoitia bennetti.* The caprine parasite has recently been named *Besnoitia caprae* (Njenga, Bwangamoi, Kang'ethe, Mugera & Mutiga 1995).

Susceptible species

The primary host is presumed to be a member of the cat family but it has not been satisfactorily identified. Cattle, goats and horses are then the intermediate hosts of the above-mentioned three parasites. Inapparent infections have been observed in blue wildebeest, impalas, zebras, donkeys, a mule and a warthog (Bigalke & Prozesky 1994; Bigalke 1994).

Incubation period and signs of the disease

The incubation period in cattle is probably not longer than about 2 weeks (Bigalke & Prozesky 1994). In cattle, fever, weight loss, anasarca and sometimes death occur in the acute stage. Sterility is common in affected bulls. Most animals survive the acute phase and may subsequently show few signs of disease, but inspection of the eye usually reveals small, granular cysts in the scleral conjunctiva. As the dis-

ease becomes chronic, severely affected animals develop the typical scleroderma stage of the disease characterized by progressive thickening, hardening and prominent folding and puckering of the skin, which is accompanied by progressive loss of hair. These signs have given rise to the common names of "olifantsvelsiekte" or elephant skin disease. However, antelopes, such as blue wildebeest, do not develop such signs.

Carrier state

Inapparent infection with a strain of *B. besnoiti* occurs in blue wildebeest and impalas (Bigalke, Van Niekerk, Basson & McCully 1967; Basson, McCully & Bigalke 1970). The antelope strain is relatively non-pathogenic for cattle and a blue wildebeest isolate is being used in a vaccine to immunize cattle (Bigalke, Schoeman & McCully 1974; Pipano 1997).

Transmission

Transmission is presumed to be through the faeces of a primary carnivorous host (Bigalke & Prozesky 1994). Biting flies can also transmit the disease mechanically but this form of transmission has invariably resulted in subclinical infections (Bigalke & Prozesky 1994).

Epidemiology

Besnoitiosis occurs sporadically, particularly in subtropical areas. The life cycle of this parasite has not been clearly elucidated. However, related parasites, such as *Toxoplasma gondii,* have a life cycle that involves a primary carnivorous host, which becomes infected while feeding on cyst-infected meat or carcass material, and a secondary host that is infected from the host's isosporan-type oocyst-containing faeces. Serological testing indicates a high incidence of clinically inapparent infection (Janitschke, De Vos & Bigalke 1984).

Diagnosis

In cattle, typical signs of the disease and the presence of typical cysts in the scleral conjunctiva are usually sufficient to make a diagnosis (Bigalke & Prozesky 1994). The diagnosis can be confirmed by histological examination of skin lesions for the typical cysts. ELISA and immunofluourescence tests have been used for the demonstration of antibodies (Janitschke *et al.* 1984). A fast skin biopsy smear test for the identification of the cystozoites (= bradyzoites) has been described (Sannusi 1991).

OTHER PROTOZOAL DISEASES

A number of protozoal parasites of antelope are of minor importance because they usually cause insig-

nificant diseases or asymptomatic infections. These parasites include:

Toxoplasmosis

Toxoplasma gondii is found wherever cats are found in the world. Felidae and most often the domestic cat act as definitive host. Most animals, including mammals, birds and reptiles, can be infected and then serve as intermediate hosts. In most species the infection is harmless and infected animals remain asymptomatic. However, some animals such as new world monkeys and Australian marsupials and parrots that evolved in environments where the parasite did not exist, are highly susceptible to infection. Although antelopes can be infected, as evidenced by serological testing (Brillhart, Fox, Dubey & Upton 1994; Mohammed & Hussein 1994), a search of the literature revealed no evidence of severe disease in antelope.

Sarcocystosis

There are a large number of *Sarcocystis* spp. For each parasite species the definitive host is a carnivore and the intermediate host a prey animal of the definitive host. The infection of intermediate hosts is usually asymptomatic. Sarcocysts have been found in antelope in zoos in Europe (Stolte, Odening & Bockhardt 1996) and in African (Odening, Rudolph, Quand, Bengis, Bockhardt & Viertel 1998) and Indian antelope (Acharjyo & Rao 1988). The literature has been reviewed by Marcus & Van Der Lugt (1994).

Coccidiosis and cryptosporidiosis

These parasites occur in a wide variety of antelope in zoos (Fenwick 1983; Van Winkle 1985; Schillhorn Van Veen, Trapp, Daunt & Richter 1986; Pospischil, Stiglmair, Von Hegel, Wiesner & Von Hegel 1987; Flach, Blewett & Angus 1991). Uterine coccidiosis occurs in the uteri of about 1 % of impala ewes in the KNP in South Africa (Basson, McCully, Kruger, Van Niekerk, Young, De Vos, Keep & Ebedes 1971) and an outbreak of intestinal coccidiosis in impalas held in captivity has been described by Pienaar, Bigalke, Tustin & Naudé (1964). Coccidiosis tends to cause enteric disease in young animals kept in unhygienic and/or stressful conditions. Although it is a very common infection in wild animals in Africa, no evidence has hitherto been obtained that cocciodosis causes disease under extensive conditions.

RICKETTSIAL AND CHLAMYDIAL DISEASES

Anaplasmosis

Anaplasmosis is a mainly tick-borne disease that occurs in most tropical and sub-tropical countries and some temperate parts of the world. The disease has been reviewed by Potgieter & Stoltsz (1994).

Aetiological agent

Anaplasmosis is caused by *Anaplasma marginale* and *Anaplasma centrale* in cattle and *Anaplasma ovis* in sheep (family: Anaplasmataceae, genus: *Anaplasma*).

Susceptible species

Anaplasmosis in cattle is caused by *A. marginale*. *Anaplasma centrale* is a relatively benign parasite and is used as a vaccine against the disease. *Anaplasma ovis* is mildly pathogenic for sheep. Naturally occurring asymptomatic *A. marginale* infection (in clinically normal animals) has been described in giraffes, sable antelopes, buffaloes, black wildebeest (Potgieter & Stoltsz 1994); buffaloes, impalas (Norval, Fivaz, Lawrence & Brown 1984); kob (Kupper *et al.* 1983). Blesbok, duikers and black wildebeest have been experimentally infected (Potgieter & Stoltsz 1994). The only wild animal in which frank disease has been seen is the giraffe (Augustyn & Bigalke 1974). It has been suggested that there may be other species of *Anaplasma,* since transmission to cattle with blood from serologically positive waterbuck, impalas, Grant's gazelles and eland was unsuccessful and isolates from Coke's hartebeest, blue wildebeest and Thompson's gazelles caused mild infections in cattle (Stoltsz 1994). *Anaplasma ovis* has been experimentally transmitted to pronghorn antelope in the USA (Zaugg 1987). Recently it has been shown that eland can be infected with both *A. marginale* and *A. ovis* (Ngeranwa, Venter, Penzhorn, Soi, Mwanzia & Nyongesa 1998). It is therefore possible that failure to transmit anaplasmosis from antelope to cattle may have been because the *Anaplasma* involved was *A. ovis* and not *A. marginale*. The occurrence of *Anaplasma* infections in wild and domestic ruminants has been reviewed by Kuttler (1984).

Incubation period and signs of the disease

In cattle the incubation period is typically from 15–36 days but may be up to 100 days (Potgieter 1979). Signs of disease include fever and inappetance, ruminal stasis and impaction, anaemia and jaundice. The disease tends to be less acute than babesiosis and the mortality rate somewhat lower. *Anaplasma ovis* generally causes a mild or inapparent infection in sheep.

Carrier state

The infection can be carried for long periods and possibly for life by recovered cattle (Potgieter & Stoltsz 1994). The length of time antelope remain carriers

is not known. All serologically positive animals should be regarded as carriers.

Transmission

The disease is transmitted biologically and mechanically by arthropod vectors. Fourteen tick species have been listed as capable of carrying the disease, although the evidence for some of them was not convincing (Wright & Leach 1996). Mechanical transmission by biting flies such as *S. calcitrans* and *Tabanus* spp. is possible. Contrary to what has been believed, the two one-host ticks *B. decoloratus* and *B. microplus* are incapable of transovarial transmission, but both transstadial and intrastadial transmission are possible (Potgieter & Stoltsz 1994).

Epidemiology

It is a typically tick-borne disease. Adult cattle that have been born and raised in endemically infected areas are usually immune but animals introduced from non-infected areas are susceptible. Calves under the age of 6 months are resistant to severe clinical disease irrespective of the immune status of their dams. Although other insects can act as mechanical vectors of the organism, the disease is generally confined to areas where competent tick vectors are present.

Diagnosis

A diagnosis can be made by identification of the organism in stained blood smears or fluorescent antibody staining of parasites in blood smears. A PCR method is able to detect as few as 0.0001 % infected cells, but even at this sensitivity only a proportion of carrier animals would be detected. Serological tests include the complement fixation test, a card agglutination test, an ELISA and an indirect fluorescent antibody test (Wright & Leach 1996).

Heartwater

Heartwater is a major disease of livestock in tropical and sub-tropical Africa and has spread to the Caribbean region. Bezuidenhout, Prozesky, Du Plessis & Van Amstel (1994) have written a review.

Aetiological agent

Heartwater is caused by the rickettsial organism *Cowdria ruminantium*.

Susceptible species

Cattle, sheep and goats are susceptible. Clinical infections have been reported in black wildebeest, blesbok, springbok and eland. Sub-clinical infections have also been reported in giraffes, black wildebeest, blesbok and eland (Oberem & Bezuidenhout 1987).

The clinical disease has also been reported in a sitatunga (Okoh, Oyetunde & Ibu 1986).

The organism can be grown and maintained in endothelial cell cultures from sable antelopes and eland (Smith, Anderson, Burridge, Peter & Mahan 1998). It has been suggested that the antelopes susceptible to heartwater are those such as springbok and blesbok that do not occur in heartwater-endemic, *Amblyomma*-infested areas, and have consequently not evolved with the disease (Bigalke 1994). Species from endemic areas remain asymptomatic when infected but may become carriers of the organism.

Experimental infection failed to cause clinical signs of the disease in impalas, wildebeest, buffaloes, kudus, giraffes (Gradwell, Van Niekerk & Joubert 1976) and buffaloes (Keffen 1985); eland, giraffes, kudus and wildebeest (Peter, Anderson, Burridge & Mahan 1998); impalas, sable antelopes and tsessebes (Peter, Anderson, Burridge, Perry & Mahan 1999a).

Incubation period and signs of the disease

In cattle the incubation period varies from 9–29 days (Bezuidenhout *et al.* 1994). Typical signs of the disease include high fever, inappetance, nervous signs and a high mortality rate.

Carrier state

Cattle can carry the infection for a year but sheep only carry it for about 3 weeks (Bezuidenhout *et al.* 1994). Eland, wildebeest, giraffes and kudus were able to infect *Amblyomma hebraeum* 128, 128, 85 and 24 days, respectively, after infection (Peter *et al.* 1998). Buffaloes remained infected for at least 161 days (Andrew & Norval 1989). Experimentally infected sable antelopes transmitted the organism for at least 37 days but tsessebes and impalas failed to transmit the disease 10 days after infection (Peter *et al.* 1999a). Fifty percent of 24 buffaloes captured in the KNP were infected with *Cowdria* (Allsopp *et al.* 1999).

Transmission

The disease is carried by *A. hebraeum* and *A. variegatum*. Generally the infection is transmitted transstadially in the tick. Intrastadial transmission is also likely. There has been one description of transovarial transmission but it probably occurs rarely (Bezuidenhout & Oberem 1985).

Epidemiology

The disease is carried by ticks in subtropical and tropical areas of Africa. Young animals are more resistant than adults are, and in endemic areas most animals are infected while young and their immunity is maintained by re-infection when ticks are present.

It was recently shown that 1.7 % of *A. hebraeum* from the KNP carry *C. ruminantium* (Peter, Bryson, Perry, O'Callaghan, Smith, Mlambo, Horak, Burridge & Mahan 1999b). Another group of workers found 2.8 % of 214 adult *A. hebraeum* taken from buffaloes in the Park to be infected (Allsopp *et al.* 1999). The KNP covers an area of nearly 20 000 km² and has been a game reserve free from domestic stock for close to 100 years. These findings provide irrefutable evidence that heartwater is maintained in a wildlife/vector cycle in the absence of domestic stock. However, wildlife hosts other than buffaloes have not been identified. The disease in domestic stock mainly occurs in the absence of buffaloes.

Diagnosis

Signs of the disease may be typical and the diagnosis can be confirmed by demonstration of the organism in the endothelium of capillaries in smears made from brain tissue. A PCR can be used to demonstrate *C. ruminantium* DNA in infected tissues. Antibodies can be demonstrated by an indirect fluorescent antibody test or by an indirect or a competitive ELISA (Camus & Uilenberg 1996).

OTHER RICKETTSIAL AND CHLAMYDIAL INFECTIONS

Other rickettsial infections of antelope that are of minor importance include the following:

Ehrlichia bovis

In cattle this organism causes a disease syndrome resembling a mild form of heartwater with an incubation period of 3–6 weeks. Originally it was thought to be confined to Senegal and West Africa but it may occur in other parts of Africa, including South Africa. In Senegal it is associated with a syndrome known as nofel in which lymph nodes are enlarged and a suppurative otitis occurs. The infection causes the development of antibodies that cross react with heartwater (Scott 1994). The relationship of this organism to wild ruminants is not known.

Cytoecetes ondiri

Ondiri disease or bovine petechial fever in East Africa is caused by *Cytoecetes ondiri* (Davies 1993). The disease is characterized by widespread petechial and ecchymotic haemorrhages on mucosal and serosal surfaces. The mortality rate may be 50 % in untreated cases. Exotic breeds of cattle are more susceptible to the infection than indigenous cattle. It is endemic in wild ruminants, particularly bushbuck and is restricted to certain well defined ecological areas in Kenya and perhaps other East and Central African countries. It is invariably associated with the presence of bushbuck. *Haemaphysalis aciculifer* and *Haemaphysalis parmata*, two common ticks of bushbuck, are thought to be the probable vectors (Davies 1993).

Coxiella burnetti

No references to Q fever (*Coxiella burnetti*) in antelope were found. However, the organism is widely distributed throughout the world and found in a wide variety of animals and birds. It would be surprising if the organism could not also infect antelope. It has been associated with 35 species of ticks from 11 genera (Scott & Herr 1994). It is also believed that the organism can be transmitted transovarially in ticks (Scott & Herr 1994). The exact role the tick plays in transmission is unclear and it has been suggested that the disease is more likely to be spread by inhaling dust contaminated with the agent derived from placentas of domestic animals that have aborted (Durand 1996). Others have suggested tick faeces in dust as a source of infection. The infection can induce abortion and gynaecological disorders in cows, ewes and goats, but can also sometimes be isolated from placentas from normal births. In humans, it causes a febrile influenza-like condition, pneumonia, hepatitis and endocarditis (Durand 1996). If the infection occurs in wild ruminants they are likely to be asymptomatically infected as no disease has been described.

Chlamydia

Chlamydial infection has been described in a springbok (Van der Lugt & Kriek 1988) and an outbreak of mortality associated with a chlamydial infection occurred in blackbuck and scimitar oryxes in a zoo in Georgia (Mansell, Tang, Baldwin, Styler & Liggett 1995). The disease occurred in blackbuck that had been transported from Texas and it was suggested that the infection could have been caused by the activation of a latent infection by the stress of transport. *Chlamydia* were demonstrated in a wide range of tissues from infected animals.

Little is known about this infection in antelopes. It is not even known which species of *Chlamydia* are involved, how the disease is transmitted, whether latent infections occur, which antelope are susceptible, how widely the infection is distributed and whether the infections described in the two cases reported were caused by the same organism.

BACTERIAL DISEASES

Brucellosis

Brucellosis is a serious zoonotic disease of cattle that formerly had a world wide distribution but has now been eradicated from many developed countries.

Aetiological agent

Brucella abortus causes contagious abortion in cattle. *Brucella suis* occurs predominantly in pigs and *Brucella melitensis* mainly in sheep and goats. In wild African ruminants, *B. abortus* occurs. *B. suis* and *B. melitensis* has not been described but could potentially occur.

Susceptible species

Brucella abortus occurs predominantly in cattle. Antibodies have been found in eland, wildebeest, impalas, waterbuck and bushbuck (Bigalke 1994) eland, impalas and giraffes (Madsen & Anderson 1995); wildebeest (Waghela & Karstad 1986); eland and oryxes (Paling, Waghela, Macowan & Heath 1988). Serological titres occur in buffaloes (Waghela & Karstad 1986; Bigalke 1994; Madsen & Anderson 1995) and the organism has also been isolated from buffaloes (Gradwell, Schutte, Van Niekerk & Roux 1977).

Incubation period

In cattle abortions usually occur in the fifth to ninth months of pregnancy (Corbel & MacMillan 1996). The time between infection and abortion may vary from weeks to months, depending on the stage of pregnancy when the animal is infected (Bishop, Bosman & Herr 1994) The disease is characterized by abortion and retained afterbirth.

In antelope species there is no evidence that the infection causes abortion and generally African wild ruminants appear to be well adapted to the infection (Bigalke 1994). Buffaloes appear to be an exception in that artificial infection with *B. abortus* caused abortion. Moreover, naturally infected KNP buffaloes with low antibody titres which were brought into captivity aborted at 7–9 months gestation and *B. abortus* Biotype 1 was isolated from fortal tissues (R.G. Bengis, unpublished data 2001)

Carrier state

Cattle may carry the infection for years and the organism may be excreted in vaginal discharge at the time of calving and in milk. Rare cases of antibody-negative carrier animals occur in calves born to infected dams.

Transmission

Transmission occurs through mucosal surfaces in animals, usually by the oral route, by contact with aborted foetuses, placentas and vaginal discharges.

Epidemiology

Antelopes probably play little role in the dissemination of the disease to cattle and the infection appears to be asymptomatic in antelopes. However, the infection does spread to some extent in antelope as evidenced by positive serology. It is therefore infectious and could in the right circumstances spread to any susceptible animal. Buffaloes, as outlined above, appear to be of greater importance.

Diagnosis

A range of serological tests are used including particularly the complement fixation test and the ELISA. A diagnosis can also be made by isolating the organism (Corbel & MacMillan 1996).

Anthrax

Anthrax is a zoonotic disease that is caused by a spore-forming bacillus. Spores are extremely resistant and can survive for many years in the environment. An extensive review of the disease with emphasis on anthrax in wildlife is available (De Vos 1994).

Aetiological agent

The disease agent is *Bacillus anthracis*. The organism is very stable and claimed to be one of the most molecularly monomorphic bacteria known (Keim, Kalif Schupp, Hill, Travis, Richmond, Adair, Hugh-Jones, Kuske & Jackson 1997). Two distinct genetic lineages were identified amongst 79 isolates studied (Keim *et al.* 1997).

Susceptible species

Anthrax is a disease of numerous animal species. In the most susceptible species such as ruminants it usually occurs as a peracute disease. In less susceptible species it may occur as an acute disease (horses) and in the least susceptible animals such as pigs, it occurs as a subacute disease (Whitford 1996). It has been described in at least 17 antelope species and buffaloes and giraffes (De Vos 1994). Three epidemics of the disease have been recorded in the Etosha National Park in Namibia. These included two epidemics in elephants and one in zebras, wildebeest, gemsbok and springbok (Lindeque & Turnbull 1994). An estimated 4 000 hippopotomi died of the disease in the Luangwa Valley in Zambia (Turnbull, Bell, Saigawa, Munyemyembe, Mulenga & Makala 1991). Apart from these distinct epidemics the disease occurs sporadically in free-living animals.

Incubation period

The incubation period is believed to be 1–14 days (De Vos 1994).

Carrier state

Bacillus anthracis was cultivated from 50 % of vulture, jackal and hyaena faeces from samples col-

lected in the vicinity of confirmed anthrax carcasses (Lindeque & Turnbull 1994). Vultures may carry the organism in their faeces for 3 weeks. In experimentally infected impalas, lymph nodes remained infected for at least 4 weeks (De Vos 1994).

Transmission

Transmission in animals is generally by ingestion, or mechanically by biting flies.

Incubation and signs of the disease

In ruminants the most common form of the disease results in sudden death. In horses, colic, high fever, depression and death within 2–4 days may be seen. Where the organism is introduced under the skin by biting flies, subcutaneous swellings may also occur at the site of the bites. In this form of the disease the animal may survive for 7 days (Whitford 1996).

Epidemiology

There is still much that is unknown about the epidemiology of the disease. In some circumstances spores or the organism survives for many years in the soil. Sandy soil with low pH appeared to be more conducive to sporulation and survival of B. anthracis, than soil with a higher pH (Lindeque & Turnbull 1994). Multiplication of the organism depends mainly on the multiplication that occurs in infected animals. The organism was not generally found in samples of water or mud collected from sites not associated with infected carcasses, but often found in soil samples where carcasses known, or suspected to be infected with anthrax had lain. Higher rates of infection were found in water from water holes when there was an outbreak of anthrax in elephants in the area.

Antibodies to B. anthracis are rare in herbivores but more common in carnivores, antibody titres appearing to reflect the prevalence of anthrax in the ranges of the latter (Turnbull, Doganay, Lindeque, Aygen & McLaughlin 1992). In endemic areas, such as the Etosha National Park, wild carnivores are resistant to anthrax. However, in areas where cyclical epidemics occur, such as the KNP, naïve lions and leopards, for example, develop acute (fatal) or subacute infections, the latter being characterized by swollen faces and tongues and oral ulcers (Bengis 2000).

No spores were found in faeces from sites not associated with anthrax carcasses, but were commonly isolated from faeces of predators that probably fed on anthrax carcasses (Turnbull et al. 1989; Lindeque & Turnbull 1994). Therefore, carnivorous animals may play a role in distributing the organism in the environment. Although the organism can be carried in lymph nodes and faeces of healthy animals, they probably only carry the organism for a short period. A period of at least 4 weeks was demonstrated in

impalas by De Vos (1994) who also raised some issues about a possible carrier state in impalas, black rats, cattle and pigs but states that "It is unknown whether a carrier state plays a role in the epidemiology of the disease".

Diagnosis

Blood smears from infected carcasses are still the main method of diagnosing the disease. A PCR assay has been developed to detect anthrax spores in soil. The organism can also be cultured from infected carcasses. Serological tests are not generally useful for the diagnosis of anthrax (Whitford 1996).

Tuberculosis

Tuberculosis is a serious disease of cattle that occurs in many countries except those where it has been eradicated.

Aetiological agent

Bovine tuberculosis is caused by Mycobacterium bovis, which is also the usual cause of tuberculosis in antelopes. Infection with M. tuberculosis is rare in animals apart from humans, and M. avium causes a mycobacteriosis in deer but its role as a possible cause of lesions in antelopes is not clear.

Susceptible species

Cattle are the main species of domestic animals infected by M. bovis although the disease is found less commonly in pigs and goats and rarely in sheep. The disease has caused serious problems in farmed and wild deer in New Zealand. It has been seen in kudus, duikers and lechwes (Huchzermeyer, Brückner, Van Heerden, Kleeberg, Van Rensburg, Koen & Loveday 1994); Arabian oryxes (Rietkerk, Griffin, Wood, Mubarak, Delima, Badri, Lindsay & Williamson 1993; Greth, Flamand & Delhomme 1994); blackbuck (Gupta & Singh 1987; Pathak, Rahman, Upadhyaya & Baruah 1987); kudus (Weber & Van Hoven 1992); lechwes (Gallager, MacAdam, Sayer & Van Lavieren 1972; Clancy 1977; Stafford 1991; Zieger, Pandey, Kriek & Cauldwell 1998); bushbuck (Zieger et al. 1998). The problem in kudus in the Eastern Cape province of South Africa is a long-standing one which was first described in 1940 (Thorburn & Thomas 1940) and the disease has remained endemic in kudus in that area. The disease also occurs in buffaloes and warthogs in Uganda (Thurlbeck, Butas, Mankiewicz & Laws 1965; Woodford 1982). In recent years tuberculosis has become a serious problem of wild buffaloes in the KNP and Hluhluwe/Umfolozi Park in South Africa and has spread to other species (Keet, Kriek, Huchzermeyer & Bengis 1994; Keet, Kriek, Penrith, Michel & Huchzermeyer 1996; Weyer, Fourie, Durrheim, Lancaster, Haslov & Bryden 1999).

Incubation period

The incubation period cannot easily be defined. Some cattle, particularly if artificially infected, may develop frank disease within a few weeks, but others develop small closed lesions and do not excrete the organism for years. Progression of the disease to an advanced stage may be slow (Huchzermeyer *et al.* 1994).

Carrier state

It is a chronic disease and animals may remain infected for years (Huchzermeyer *et al.* 1994).

Transmission

Infected animals may excrete the organism in expired droplets, faeces, urine, milk or in pus from ruptured abscesses, depending on the location of the lesions. Animals acquire infection by inhalation of infected droplets or ingestion of contaminated material (Huchzermeyer *et al.* 1994). The respiratory route is believed to be the main route of transmission in herbivores (Morris, Pfeiffer & Jackson 1994; O'Reilly & Daborn 1995).

Signs of the disease

Usually the disease runs a chronic course, and infected animals may continue to excrete the organism for months or years. Infected animals may show few signs of the disease, or may have a progressive, wasting, respiratory disease in advanced cases of lung tuberculosis. Emaciation and respiratory distress may occur in the terminal stages of the disease. Other signs of the disease vary according to where the lesions are located and may include mastitis, enlarged lymph nodes and draining abscesses or sinuses (Huchzermeyer *et al.* 1994; Haagsma 1996).

Epidemiology

Although many species of animals can be infected it is generally believed that the disease is not self-maintaining in some hosts (Morris *et al.* 1994; O'Reilly & Daborn 1995). Self-sustaining infections seem to have been established in kudus in the Eastern Cape Province of South Africa (Thorburn & Thomas 1940; Weber & Van Hoven 1992), in buffaloes in the KNP in South Africa (Keet *et al.* 1994; Keet *et al.* 1996b; Weyer *et al.* 1999) and in the Ruwenzori National Park in Uganda (Thurlbeck *et al.* 1965; Woodford 1982), and in lechwes in Zambia (Gallager *et al.* 1972; Clancy 1977; Stafford 1991; Zieger *et al.* 1998). The epidemiological picture is typical of a slowly spreading infectious disease.

Diagnosis

The intradermal tuberculin test is the standard diagnostic test for cattle. A diagnosis may also be made by demonstration of typical gross and histological lesions, demonstration of organisms in smears or tissue sections by Ziehl-Neelsen staining or isolation of the organism. PCR methods are now available. It has been suggested that the lymphocyte proliferation assay may be useful in wildlife and zoo animals (Haagsma 1996). Of the blood-based tests, the gamma interferon test has shown great promise in buffaloes and kudus. (A.L. Michel & J.P. Raath, unpublished data 2001).

Johne's disease (paratuberculosis)

Johne's disease is a serious disease of domestic animals with a world-wide distribution.

Aetiological agent

The aetiological agent is *Mycobacterium paratuberculosis.* There are two main types of *M. paratuberculosis,* a type found predominantly in cattle that is comparatively easy to isolate and a type found in sheep that is very difficult to culture. The two types are distinguishable by electrophoretic analysis of restriction fragments of DNA (Collins, Gabric & De Lisle 1990).

Susceptible species

Cattle, sheep, goats, deer and camelids are susceptible. Antelopes are susceptible, but the disease has only rarely been reported in antelope species.

In an issue of *Revue Scientifique et Technique* 15(1): 996, devoted entirely to wildlife husbandry and diseases, there are no references to Johne's disease in antelopes. On a ranch on which antibody was detected in camels and goats, no antibodies were found in antelopes (Paling *et al.* 1988). The disease has been described in a Jimela topi in a zoo (Steinberg 1988) and in a saiga antelope (Dukes, Glover, Brooks & Duncan 1992), which is not an African species. In the latter case the organism was isolated and the disease was transmitted to sheep. From the description it appears as though the organism was of the difficult-to-isolate type usually found in sheep.

The disease appears to be of no significance in wild African ruminants.

Incubation period

The incubation period is generally from 1 to several years (Thorel 1994).

Carrier state

Cattle may pass the organism in their faeces for years before they become clinically affected. Some animals never become clinically affected but pass organisms in their faeces during their life-time.

Transmission

Johne's disease is transmitted faeco-orally or congenitally (Thorel 1994; Huchzermeyer, Brückner & Bastianello 1994).

Signs of the disease

Loss of condition, chronic diarrhoea and wasting are the common signs of the disease.

Epidemiology

It is a slowly spreading chronic disease. The organism is highly resistant and contaminated pastures may remain infected for long periods.

Diagnosis

The diagnosis can be confirmed by isolation of the organism from faeces, lymph nodes or gut mucosa or demonstration of the organism in Ziehl-Neelsen-stained smears made from faeces or gut mucosa. Typical histological lesions can be demonstrated in infected animal tissues, particularly in the ileum and caecum and mesenteric lymph nodes. Serological methods including especially the absorbed ELISA are useful (Thorel 1994) but have the draw-back of having low sensitivity for detecting non-clinical infections and have not been verified in antelope. Delayed hypersensitivity tests are of limited value (Thorel 1994).

DISCUSSION AND CONCLUSIONS

African wild ruminants play an active role in the maintenance and spread of several important diseases of domestic stock as outlined above and summarized in Table 1.

For several other diseases the role or possible role played by wild ruminants is not clear. These include

Rift Valley fever, lumpy skin disease, Crimean Congo haemorrhagic fever, bovine virus diarrhoea, infectious bovine rhinotracheitis, Nairobi sheep disease, Akabane disease, brucellosis and tuberculosis. In all of these diseases wild ruminants can be infected but the role they play in spreading the disease to domestic animals is either undefined or a minor one.

It is particularly interesting to note that some of the diseases have been introduced into wild animal populations from domestic animals. Such diseases include tuberculosis and rinderpest.

As both wildlife and domestic animals are necessary for the well being of humans on the African continent, the economy and the preservation of the environment, ways must be sought to minimize the effects of the diseases.

Measures that are important include:

- The vaccination of livestock. The vaccination of domestic stock against many diseases is already widely practised and represents an important method for maintaining healthy stock.

- Maintaining naturally immune populations of livestock. Methods of livestock management that ensure that animals are exposed to endemic diseases at a young age and that their immunity is maintained by natural exposure to disease agents results in a population of livestock that is resistant to some diseases.

For tick borne diseases there are two potential control options:

- Intensive dipping to eradicate ticks, thus ending up with a tick-free susceptible population of animals.

- Dipping less intensively/strategically thereby allowing a low number of ticks to maintain natural immunity in the population. Inherently resistant breeds of livestock facilitate the adoption of this

TABLE 1 Economically important diseases of livestock in which wild ruminants play a role

Disease or agent	Wild ruminants involved in maintenance cycle	Transmission/vector*
Foot-and-mouth disease	Buffaloes	Contact and aerosol
Rinderpest	Buffaloes and antelope spp.	Contact
Bluetongue	Several species of antelopes	*Culicoides* spp.
Malignant catarrhal fever	Wildebeest	Contact
Babesiosis	Buffaloes	Tick-borne
Theileria parva infections	Buffaloes	Tick-borne
Ondiriosis	Bushbuck	Tick-borne
Trypanosomosis	Buffaloes and antelope spp.	Tsetse fly
Anaplasmosis	Buffaloes and antelope spp.	Tick-borne
Heartwater	Buffaloes and antelope spp.	Tick-borne
Anthrax	Buffaloes and antelope spp., especially kudus	Environmental contamination

* For details of vectors (where these play a role) see text

model. Where wild and domestic animals share the same grazing eradication of ticks is usually not an option.

- Control of disease vectors has been important for the tick-borne diseases and for trypanosomosis. For several other diseases that are spread by vectors such as *Culicoides* spp. and mosquitoes this type of control is not yet attainable.

- Separation of livestock and wild animals is already an accomplished fact in many parts of Africa and increasingly wild animals will be confined to designated wildlife areas such as national parks and specialist game farms. However, the increased popularity of game ranching also means that wildlife is being re-established in some areas and may threaten the health of livestock on neighbouring farms or *vice versa*. The establishment of "peace parks", such as the envisaged enlarged KNP, may also have the effect of increasing the wildlife/livestock interface thereby facilitating the transmission of diseases from the one to the other.

- Establishing disease-free populations of wild ruminants may in future become a realistic possibility that could have considerable advantages. It seems inevitable that population pressure will eventually mainly confine wild ruminants to large national parks and smaller privately owned reserves and game farms. As this continues to happen the possibility of eradicating some diseases in populations of wild animals may become a realistic possibility. These efforts could be initiated on small game farms that could eventually provide disease-free animals to replace infected populations of animals in larger game reserves. Establishment of small buffalo herds free from FMD, Corridor disease and bovine tuberculosis is indeed already being accomplished in southern Africa.

It should be noted that this article is confined to a discussion of diseases of wild ruminants. However, other wild animals also play important roles in the epidemiology of diseases. Important examples include the carnivores that carry rabies, zebras as maintenance hosts for African horse sickness, and warthogs as carriers of African swine fever. Any overall consideration of the role played by wild animals should embrace all the animals in the eco-system and should not only include external infectious diseases but also parasites and helminths.

ACKNOWLEDGEMENT

REFERENCES

Note 1 – Many of the articles cited were obtained as summaries from the following two sources:

- *CAB Abstracts*. 1984–2000. CAB International, Wallingford, Oxon OX10 8DE, United Kingdom.

- The Internet search engine of the National Centre for Biotechnology/National Library of Medicine, located at:

  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi

Note 2 – Summaries that have been used without the sighting of the whole article have been indicated in the reference list as (Abstract, CAB) for Source 1 and (Abstract, Pubmed) for Source 2

ACHARJYO, L.N. & RAO, A.T. 1988. Sarcocystosis in some Indian wild ruminants. *Indian Veterinary Journal*, 65:169–170 (Abstract, CAB).

AL-BUSAIDT, S.M., HAMBLIN, C. & TAYLOR, W.P. 1987. Neutralising antibodies to Akabane virus in free-living animals in Africa. *Tropical Health and Production*, 19:197–202 (Abstract, CAB).

ALEXANDER, K..A., MACLACHLAN, N.J., KAT, P.W., HOUSE, C., O'BRIEN, S.J., LERCHE, N.W., SAWYER, M., FRANK, L.G., HOLEKAMP, K. & SMALE, L. 1994. Evidence of natural bluetongue virus infection among African carnivores. *American Journal of Tropical Medicine and Hygiene*, 58:568–576 (Abstract, Pubmed).

ALLSOPP, M.T., THERON, J., COETZEE, M.L., DUNSTERVILLE, M.T. & ALLSOPP, B.A. 1999. The occurrence of *Theileria* and *Cowdria* parasites in African buffalo (*Syncerus caffer*) and their associated *Amblyomma hebraeum* ticks. *Onderstepoort Journal of Veterinary Research*, 66:245–249.

ANDERSON, E.C., JAGE, M., MLENGEYA, T., TIMMS, C., PAYNE, A. & HIRJI, K. 1990. A serolgical survey of rinderpest antibody in wildlife and sheep and goats in Northern Tanzania. *Epidemiology and Infection*, 105:203–214 (Abstract, CAB).

ANDERSON, E.C., FOGGIN, C., ATKINSON, M., SORENSEN, K.J., MADEKUROZVA, R.L. & NQINDI, J. 1993. The role of wild animals, other than buffalo, in the current epidemiology of foot-and-mouth disease in Zimbabwe. *Epidemiology and Infection*, 111:559–563 (Abstract: Pubmed).

ANDERSON, E.C. 1995. Morbillivirus infections in wildlife in relation to their population biology and disease control in domestic animals. *Veterinary Microbiology*, 44:319–332.

ANDERSON, E.C. & ROWE, L.W. 1998. The prevalence of antibody to the viruses of bovine virus diarrhoea, bovine herpes virus 1, Rift Valley fever, ephemeral fever and bluetongue and to *Leptospira* sp. in free-ranging wildlife in Zimbabwe. *Epidemiology and Infection*, 121:441–449 (Abstract, CAB).

ANDREW, H.R. & NORVAL, R.A. 1989. The carrier status of sheep, cattle and African buffalo recovered from heartwater *Journal of Parasitology*, 34:261–266 (Abstract, Pubmed).

ANONYMOUS. 2000. Report of the meeting of the working group on wildlife diseases, 4–6 April 2000. Paris: Office International des Epizooties.

AUBERT, M., CLIQUET, F. & BARRAT, J. 1996. Rabies, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 207–217

AUGUSTYN, N.J. & BIGALKE, R.D. 1974. *Anaplasma* infection in a giraffe. *Journal of the South African Veterinary Association*, 45:229.

AWAN, M.A. 1979. Identification by the blood incubation infectivity test *of Trypanosoma brucei* subspecies isolated from game animals in the Luangwa Valley, Zambia. *Acta Tropica* 36: 343–347 (Abstract, Pubmed).

BAER, G.M. 1990. Rabies virus. in *Virus infections in ruminants*, edited by Z. Dinter, B. Morein. Amsterdam, Oxford, New York, Tokyo: Elsevier Science Publishers: 341–354.

BARNARD, B.J. & HASSEL, R.H. 1981.Rabies in kudus (*Tragelaphus strepsiceros*) in South West Africa/Namibia. *Journal of the South African Veterinary Association*, 52:309–314.

BARNARD, B.J.H., HASSEL, R.H., GEYER H.J. & DE KOKER W.K. 1982. Non-bite transmission of rabies in kudu. *Onderstepoort Journal of Veterinary Research* 49:191–192.

BARNARD, B.J. & VAN DER PYPEKAMP, H.E. 1988. Wildebeest-derived malignant catarrhal fever: unusual epidemiology in South Africa. *Onderstepoort Journal of Veterinary Research*, 55:69–71

BARNARD, B.J., BENGIS, R.G., GRIESSEL. M.D. & DE VOS, V 1989a. Excretion of alcelaphine herpesvirus-1 by captive and free-living wildebeest (*Connochaetes taurinus*). *Onderstepoort Journal of Veterinary Research*, 56:131–134.

BARNARD, B.J., VAN DER PYPEKAMP, H.E. & GRIESSEL, M.D. 1989b. Epizootiology of wildebeest-derived malignant catarrhal fever in an outbreak in the north-west Transvaal: indications of an intermediate host. *Onderstepoort Journal of Veterinary Research*, 56:135–139.

BARNARD, B.J. 1990. Epizootiology of wildebeest-derived malignant catarrhal fever: possible transmission among cows and their calves in north-west Transvaal. *Onderstepoort Journal of Veterinary Research*, 57:201–204.

BARNARD, B.J., BENGIS, R.G. & VOGES, S.F. 1990. Epidemiology of wildebeest-derived malignant catarrhal fever in South Africa: inability to transfer the disease with an African face fly *Musca xanthomelas* (Diptera: muscidae*)*. *Onderstepoort Journal of Veterinary Research*, 57:89–93.

BARNARD, B.J.H., MUNZ, E., DUMBELL, K. & PROZESKY, L. 1994a. Lumpy skin disease, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 604–612.

BARNARD, B.J.H., VAN DER LUGT, J.J. & MUSHI, E.Z. 1994b. Malignant catarrhal fever, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 946–57.

BARNARD, B.J.H. 1996. Rift Valley fever, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 102–108.

BARNARD, B.J. 1997. Antibodies against some viruses of domestic animals in southern African wild animals. *Onderstepoort Journal of Veterinary Research*, 64:95–110.

BASSON, R.A., MCCULLY, R.M & BIGALKE, R.D. 1970. Observations on the pathogenesis of bovine and antelope strains of *Besnoitia besnoitii* (Marotel, 1912) infection in cattle and rabbits. *Onderstepoort Journal of Veterinary Research*, 37:105–126 (Abstract, Pubmed).

BASSON, P.A., MCCULLY, R.M., KRUGER, S.P., VAN NIEKERK, J.W., YOUNG, E., DE VOS, V., KEEP, M.E. & EBEDES, H. 1971. Disease conditions of game in southern Africa: Recent miscellaneous findings. *Veterinary Medical Review*, 2/3:313–40 (Abstract, Pubmed).

BASTOS, A.D., BERTSCHINGER, H.J., CORDEL, C., VAN VUUREN, C.D., KEET, D., BENGIS, R.G., GROBLER, D.G. & THOMSON, G.R. 1999. Possibility of sexual transmission of foot-and-mouth disease from African buffalo to cattle. *The Veterinary Record*, 145:77–79.

BATTLES, J.K. & DALRYMPLE, J.M. 1988. Genetic variation among geographical isolates of Rift Valley fever virus. *American Journal of Tropical Medicine and Hygiene*, 39:617–631 (Abstract, Pubmed).

BENGIS, R.G., THOMSON, G.R., HEDGER, R.S., DE VOS, V. & PINI A. 1986. 1 Foot-and-mouth disease and the African buffalo (*Syncerus caffer*). 1. Carriers as a source of infection for cattle. *Onderstepoort Journal of Veterinary Research*, 53: 69–73.

BENGIS, R.G. 1997 Animal health risks associated with the transportation and utilisation of wildlife products. *Revue Scientifique et Technique, Office International des Epizooties*, 16: 104–110.

BENGIS, R.G. 2000. An update of anthrax in wildlife in the Kruger National Park. *Proceedings of the 49th Annual Wildlife Diseases Association Conference*, Grand Teton National Park, Wyoming, June 4–8, 2000.

BESSELAAR, T.G., BLACKBURN, N.K. & MEENEHAN, G.M. 1991. Antigenic analysis of Rift Valley fever virus isolates: monoclonal antibodies distinguish wild-type and neurotrophic virus strains. *Research in Virology*, 142:469–474 (Abstract, Pubmed).

BEZUIDENHOUT, J.D. & OBEREM, P.T 1985. Proof of transovarial transmission of *Cowdria ruminantium* by *Amblyomma hebraeum*. *Onderstepoort Journal of Veterinary Research*, 53: 31–34.

BEZUIDENHOUT, J.D., PROZESKY, L., DU PLESSIS, J.L. & VAN AMSTEL, S.R. 1994. Heartwater, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 351–370.

BIGALKE, R.D., VAN NIEKERK, J.W., BASSON, P.A. & MCCULLY, R.M. 1967. Studies on the relationship between *Besnoitia* of blue wildebeest and impala, and *Besnoitia besnoitii* of cattle. *Onderstepoort Journal of Veterinary Research*, 34:7–28.

BIGALKE, R.D., SCHOEMAN, J.H. & MCCULLY, R.M. 1974. Immunization against bovine besnoitiosis with a live vaccine prepared from the blue wildebeest strain of *Besnoitia besnoiti* grown in cell cultures. 1 Studies in rabbits. *Onderstepoort Journal of Veterinary Research*, 41:1–6.

BIGALKE, R.D. 1994. The important role of wildlife in the occurrence of livestock diseases in southern Africa, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 152–166.

BIGALKE, R.D. & PROZESKY, L. 1994. Besnoitiosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 245–252.

BISHOP, G.C., BOSMAN, P.P. & HERR, S. 1994. Bovine brucellosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 1053–1066.

BOIRO, I., KONSTANINOV, O.K. & NUMEROV, A.D. 1987. [Isolation of Rift Valley fever virus from bats in the Republic of Guinea] *Bulletin de la Société de Pathologie Exotique et de ses Filiales*, 80:62–67 (Abstract, Pubmed).

BRILLHART, D.B., FOX, L.B., DUBEY, J.P. & UPTON, S.J. 1994. Seroprevalence of *Toxoplasma gondii* in wild mammals in Kansas. *Journal of the Helminthological Society of Washington*, 61:117–121 (Abstract, CAB).

BROWNLIE, J. & EDWARDS, S. 1996. Bovine viral diarrhoea, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 651–659.

BURRIDGE, M.J. 1975. The role of wild animals in the epidemiology of bovine theilerioses in East Africa. *Journal of Wildlife Diseases*, 11:68–75 (Abstract, Pubmed).

CAMUS, E. & UILENBERG, G. 1996. Heartwater, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 229–234.

CARMICHAEL, I.H. & HOBDAY, E. 1975. Blood parasites of some wild bovidae in Botswana. *Onderstepoort Journal of Veterinary Research*, 42:55–62.

CASTRO, A.E. & RODGERS, S.J. 1984. Congenital abnormalities in cattle associated with an epizootic of bluetongue virus (serotype II). *Bovine Practitioner*, 19:87–91 (Abstract, CAB).

CASTRO, A.E., RAMSAY, E.C., DOTSON, J.F., SCHRAMKE, M.L., KOCAN, A.A. & WHITENACK, D.L. 1984. Characteristics of the herpesvirus of malignant catarrhal fever isolated from captive wildebeest's calves. *American Journal of Veterinary Research*, 45:409–415 (Abstract, Pubmed).

CHILONDA, P., WOODFORD, J.D., AHAMDU, B., SAMUI, K.L., SYAKALIMA, M. & MLANGWA, J.E. 1999. Foot-and-mouth disease in Zambia: a review of the aetiology and epidemiology and recommendation for possible control. *Revue Scientifique et Technique, Office International des Epizooties*, 18: 585–592.

CLANCY, J.K. 1977. The incidence of tuberculosis in Lechwe (marsh antelope). *Tubercle*, 58:151–156.

COLLINS, D.M., GABRIC, G.W. & DE LISLE, G.W. 1990. Identification of two groups of *Mycobacterium paratuberculosis* strains by restriction endonuclease analysis and DNA hybridization. *Journal of Clinical Microbiology*, 28:1591–1596.

CONDY, J.B., HEDGER, R.S., HAMBLIN, C. & BARNETT, J.T. 1985. The duration of the foot-and-mouth disease carrier state in African buffalo (i) in individual animals and (ii) in a free living herd. *Comparative Immunology, Microbiology and Infectious Diseases*, 8:259–265 (Abstract, Pubmed).

CONNOR, R.J. 1994. African animal trypanosomiasis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 167–205.

CORBEL, M.J. & MACMILLAN, A.P. 1996, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 242–255.

DAVIES, F.G. 1978. A survey of Nairobi sheep disease in sheep and goats, wild ruminants and rodents. *Journal of Hygiene (London)*, 81:251–258 (Abstract, Pubmed).

DAVIES, F.G. 1982. Observations on the epidemiology of lumpy skin disease in Kenya. *Journal of Hygiene*, 88:95–102 (Abstract, Pubmed).

DAVIES, F.G., OCHIENG, P. & WALKER, A.R. 1990. The occurrence of ephemeral fever in Kenya. 1968–1988. *Veterinary Microbiology*, 22:129–136.

DAVIES, F.G. 1993. Bovine petechial fever (Ondiri disease). *Veterinary Microbiology*, 34:103–121.

DAVIES, F.G. 1996. Nairobi sheep disease, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 669–672.

DAVIES, F.G. 1997. Nairobi sheep disease. *Parassitologia*, 39: 95–98 (Abstract, Pubmed).

DAWE, P.S., FLANAGAN, F.O., MADEKUROZWA, R.L., SORENSEN, K.J., ANDERSON, E.C., FOGGIN, C.M., FERRIS, N.P. & KNOWLES, N. 1994. Natural transmission of foot-and-mouth disease virus from African buffalo (*Syncerus caffer*) to cattle in a wildlife area of Zimbabwe. *The Veterinary Record*, 134: 230–232.

DAWE, P.S., SORENSEN, K., FERRIS, N.P., BARNETT, I.T., ARMSTRONG, R.M. & KNOWLES, N.J. 1994. Experimental transmission of foot-and-mouth disease virus from carrier African buffalo (*Syncerus caffer*) to cattle in Zimbabwe. *The Veterinary Record*, 134:211–215.

DE VOS, A.J. & POTGIETER, F.T. 1994. Babesiosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 278–294.

DE VOS, A.J. & JORGENSEN, W.K. 1996. Bovine babesiosis, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 305–313.

DE VOS, V. 1994. Anthrax, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 1262–1289.

DEPNER, K., HÜBSCHLE, O.J.B. & LIESS, B. 1991. Prevalence of ruminant pestivirus infections in Namibia. *Onderstepoort Journal of Veterinary Research*, 58:107–109.

DILLMAN, J.S. & TOWNSEND, A.J. 1979. A trypanosomiasis survey of wild animals in the Luangwa Valley, Zambia. *Acta Tropica*, 36:349–356 (Abstract, Pubmed).

DOLAN, T.T., MOIZARIA, S.P. & KATENDE, J.M. 1996, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 321–330.

DONALDSON, A.I., GLOSTER, J., HARVEY, L.D. & DEANS, D.H. 1982. Use of prediction models to forecast and analyse airborne spread during the foot-and-mouth disease outbreaks in Brittany, Jersey and the Isle of Wight in 1981. *The Veterinary Record*, 110:53–57.

DONALDSON, A.I. & KITCHEN, R.P. 1996. Foot-and-mouth disease, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 47–56.

DOYLE, L.G. & HEUSCHELE, W.P. 1983. Bovine viral diarrhoea virus infection in captive exotic ruminants. *Journal of American Veterinary Medical Association*, 183:1257–1259 (Abstract, Pubmed).

DOYLE, L.G., HEUSCHELE, W.P.& FOWLER, M.E. 1983. Prevalence of antibody to major viral disease in captive exotic ruminants. *Proceedings of the Annual Meeting, American Association of Zoo Veterinarians, Tampa Florida*, October 24–27, 1983: 76–82 (Abstract, Pubmed).

DRAGER, N. & MEHLITZ, D. 1978. Investigations on the prevalence or trypanosome carriers and the antibody response in wildlife in Northern Botswana. *Tropenmedizin und Parasitologie*. 29:223–233 (Abstract, Pubmed).

DUBEY, J.P. & BEATTIE, C.P. (Eds). 1988. *Toxoplasmosis of animals and man*. Boca Raton, Florida: CRC Press.

DUKES, T.W., GLOVER, G.J., BROOKS, B.W. & DUNCAN, J.R. 1992. Paratuberculosis in a saiga antelope (*Saiga tatarica*) and experimental transmission to domestic sheep. *Journal of Wildlife Diseases,* 28:161–170.

DURAND, M.P. 1996. Q Fever, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 634–641.

EATON, B. 1996. Bluetongue, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 109–118.

ELLIS, J.A., WEST, K.H., CORTESE, V.S., MYERS, S.L., CARMAN, S., MARTIN, K.M. & HAINES, D.M. 1998. Lesions and distribution of viral antigen following an experimental infection of young seronegative calves with virulent bovine virus diarrhea virus-type II. *Canadian Journal of Veterinary Research*, 62:161–169.

ERASMUS, B.J. 1975. The epizootiology of bluetongue: the African situation. *Australian Veterinary Journal*, 51:196–198.

ERASMUS, B.J. 1986. Malignant catarrhal fever. Biennial report of the Veterinary Research Institute, Onderstepoort 1984–85: 54–55.

EVERARD, J.D. & EVERARD, C.O.R. 1988. Mongoose rabies. Reviews of Infectious Diseases, 10 (Supplement 4):S610–614.

FENWICK, B.W. 1983. Cryptosporidiosis in a neonatal gazella. Journal of the American Veterinary Medical Association, 283: 1331–1332 (Abstract, CAB).

FERRIS, N.P., CONDY, J.B., BARNETT, J.T. & ARMSTRONG, R.M. 1989. Experimental infection of eland (Taurotragus oryx), sable antelope (Ozanna grandicomis) and buffalo (Syncerus caffer) with foot-and-mouth disease virus. Journal of Comparative Pathology, 101:306–316.

FLACH, E.J., BLEWETT, D.A. & ANGUS, K.W. 1991. Coccidial infections of captive red lechwe (Kobus leche) at Edinburgh zoo, with a note on concurrent Trichuris sp. infections. Journal of Zoo and Wildlife Medicine, 22:446–452 (Abstract, CAB).

FOMENTY, P., DOMENECH, J., LAUGINIE, F., OUTTARA, M., DIAWARA, S., RAATH, J.P., GROBLER, D., LEFORBAN, Y. & ANGBA, A. 1994. [Epidemiological study of bluetongue in sheep, cattle, and different species of wild animals in Ivory Coast]. Revue Scientifique et Technique, Office International des Epizooties, 13:737–751.

FONTENILLE, D., TRAORE-LAMIZANA, M., ZELLER, H., MONDO, M., DIALLO, M. & DIGOUTTE, J.P. 1995. Rift Valley fever in western Africa: isolations from Aedes mosquitoes during an interepizootic period. American Journal of Tropical Medicine and Hygiene, 52:403–404 (Abstract, Pubmed).

FONTENILLE, D., TRAORE-LAMIZANA, M., THONNON, J., DIGOUTTE, J.P. & ZELLER, H.G. 1998. New vectors of Rift Valley fever in West Africa. Emerging Infectious Diseases, 4: 289–293 (Abstract, Pubmed).

FRÖHLICH, K. & FLACH, E.J. 1998. Long term viral serology of free living and captive ungulates. Journal of Zoo and Wildlife Medicine, 29:165–170 (Abstract, CAB).

GAINARU, M.D., THOMPSON, G.R., BENGIS, R.G., ESTERHUYSEN, J.J., BRUCE, W. & PINI, A. 1986. Foot- and-mouth disease and the African buffalo (Syncerus caffer). II. Virus excretion and transmission during acute infection. Onderstepoort Journal of Veterinary Research, 53:75–85.

GALLAGER, J., MACADAM, I., SAYER, J. & VAN LAVIEREN, L.P. 1972. Pulmonary tuberculosis in free-living lechwe antelope in Zambia. Tropical Animal Health and Production, 4:204–213 (Abstract, Pubmed).

GARGAN, T.P., CLARK, G.G., DOHM, D.J., TURELL, M.J. & BAILEY, C.L. 1988. Vector potential of selected North American mosquito species for Rift Valley fever virus. American Journal of Tropical Medicine and Hygiene, 38:440–446 (Abstract, Pubmed).

GARGAN, T.P., JUPP, P.G. & NOVAK, R.J. 1988. Panveld ovipositional sites in floodwater Aedes mosquitoes and attempts to detect transovarial transmission of Rift Valley fever virus in South Africa. Medical and Veterinary Entomology, 2:231–236 (Abstract, Pubmed).

GRADWELL, D.V., VAN NIEKERK, C.A. & JOUBERT, D.C. 1976. Attempted artificial infection of impala, blue wildebeest, buffalo, kudu, giraffe, and warthog with heartwater. Journal of the South African Veterinary Association, 47:209–210.

GRADWELL, D.V., SCHUTTE, A.P., VAN NIEKERK, C.A.W.J. & ROUX, D.J. 1977 Isolation of Brucella abortus biotype 1 from African buffalo in the Kruger National Park. Journal of the South African Veterinary Medical Association, 48:41–43.

GRETH, A., GOURREAU, J.M., VASSART, M., NGUYEN-BA-VY, WYERS, M. & LEFERVE, P.C. 1992. Capripox disease in an Arabian oryx (Oryx leucoryx) from Saudi Arabia. Journal of Wildlife Diseases, 28:295–300.

GRETH, A., FLAMAND, J.R.B. & DELHOMME, A. 1994. An outbreak of tuberculosis in a captive herd of Arabian oryx (Oryx leucoryx); management. The Veterinary Record, 134:165–167.

GUEDEGBE, B., VERHULST, A., VAN MEIVENNE, N., PANDLEY, V.S. & DOKO, A. 1992. [Serological evidence of the existence of a wild reservoir of Trypanosoma brucei gambiense in the Pendari biosphere reservation in the republic of Benin]. Annales de la Société Belge de Médecine Tropicale, 72:113–120 (Abstract, Pubmed).

GULLAND, F.M., REID, H.W., BUXTON, D., LEWIS, J.C.M., KOCK, R.A. & KIRKWOOD, J.K. 1989. Malignant catarrhal fever in a roan antelope (Hippotragus equinus) at Regent's Park. The Veterinary Record, 124:42–43.

GUPTA, P.P. & SINGH, S. 1987. Tuberculosis in an Indian black buck. Journal of Research, Punjab Agricultural University, 24: 315–316 (Abstract, CAB).

HAAGSMA, J. 1996. Bovine tuberculosis, in Manual of standards for diagnostic tests and vaccines. Paris: Office International des Epizooties: 267–275.

HAMBLIN, C. & HEDGER, R.S. 1984. Neutralising antibody to wildebeest-derived malignant catarrhal fever in African wildlife. Comparative Immunology, Microbiology and Infectious Diseases, 7:195–199 (Abstract, CAB).

HAMBLIN, C., ANDERSON, E.C., JAGO, M., MLENGEYA, T. & HIRJI, K. 1990. Antibodies to some pathogenic agents in free-living wild species in Tanzania. Epidemiology and Infection, 105:585–594 (Abstract, CAB).

HARKNESS, J.W. & VAN DER LUGT, J.J. 1994. Bovine virus diarrhoea, in Infectious diseases of livestock with special reference to southern Africa. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 642–650.

HEDGER, R.S. & HAMBLIN, C. 1983. Neutralising antibodies to lumpy skin disease virus in African wildlife. Comparative Immunology, Microbiology and Infectious Diseases, 6:209–213 (Abstract, Pubmed).

HOCH, A.L., TURRELL, M.J. & BAILEY, C.L. 1984. Replication of Rift Valley fever virus in the sand fly Lutzomyia longipalpis. American Journal of Tropical Medicine and Hygiene, 33:295–299 (Abstract, Pubmed).

HOFF, G.L. & HOFF, D.M. 1976. Bluetongue and epizootic haemorrhagic disease: a review of these diseases in non-domestic artiodactyls. Journal of Zoo Animal Medicine, 7:26–30.

HOVE, T., SITHOLE, N., MUNODZANA, D. & MASAKA, S. 1998. Isolation and characterisation of a Babesia species from Rhipicephalus evertsi picked off a sable antelope (Hippotragus niger) which died from acute babesiosis. Onderstepoort Journal of Veterinary Research, 65:75–80.

HÜBSCHLE, O.J.B. 1988. Rabies in kudu antelope. Reviews of Infectious Diseases, 10 (Supplement 4):S629–633 (Abstract, CAB).

HUCHZERMEYER, H.F.K.A., BRÜCKNER, G.K., VAN HEERDEN, A., KLEEBERG, H.H., VAN RENSBURG, I.B.J., KOEN, P & LOVEDAY, R.K. 1994. Tuberculosis, in Infectious diseases of livestock with special reference to southern Africa. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 1425–1444.

HUCHZERMEYER, H.F.K.A., BRÜCKNER, G.K. & BASTIANELLO, S.S. 1994. Paratuberculosis, in Infectious diseases of livestock with special reference to southern Africa. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 1445–1451.

HYERA, J.M.K., LIESS, B., ANDERSON, E. & HIRJI, K.N. 1992. Prevalence of bovine viral diarrhoea virus in some wild rumi-

nants in northern Tanzania. *Bulletin of Animal Health and Production in Africa*, 40:143–151 (Abstract, CAB).

JANITSCHKE, K., DE VOS, A.J. & BIGALKE, R.D. 1984. Serodiagnosis of bovine besnoitiosis by ELISA and immunofluorescence tests. *Onderstepoort Journal of Veterinary Research*, 51: 239–243.

JENNINGS, M., PLATT, G.S. & BOWEN, E.T 1982. The susceptibility of *Culicoides variipennis* Coq. (Diptera:Ceratopgonidae) to laboratory infection with Rift Valley fever virus. *Transactions of the Royal Society for Tropical Medicine and Hygiene*, 76: 587–589 (Abstract, Pubmed).

JONGEJAN, E. & UILENBERG, G. 1994. Ticks and control methods. *Revue Scientifique et Technique. Office des Epizooties*, 13:1201–1026.

JUPP, P.G. & COMEL, A.J. 1988. Vector competence test with Rift Valley fever virus and five South African species of mosquito. *Journal of the American Mosquito Control Association*, 4:4–8 (Abstract, Pubmed).

KARBE, E., GROOTENHUIS, J.G., KELLEY, S. & KARSTAD, L. 1979. Experiments on the *Babesia bigemina* carrier state in East African buffalo and eland. *Tropenmedizin und Parasitologie* 30:313–317 (Abstract, Pubmed).

KARIUKI, D.P., INJAIRO, R., BOYCE, W.L., WELLDE, B.T & NGETHE, S. 1989. Parasite survey for eight wild animals in Ruma National Park. *Annals of Tropical Medicine and Parasitology*, 83 (Supplement 1):115–118.

KEET, D.F., KRIEK, N.P.J., HUCHZERMEYER, H. & BENGIS, R.G. 1994. Advanced tuberculosis in an African buffalo (*Syncerus caffer* Sparman). *Journal of the South African Veterinary Association*, 65:70–83.

KEET, D.F, HUNTER, P., BENGIS, R.G., BASTOS, A. & THOMSON, G.R. 1996a. The 1992 foot-and- mouth disease epizootic in the Kruger National Park. *Journal of the South African Veterinary Association*, 67:83–87

KEET, D.F., KRIEK, N.P., PENRITH, M.L., MICHEL, A. & HUCHZERMEYER, H. 1996b. Tuberculosis in buffaloes (*Syncerus caffer*) in the Kruger National Park: spread of the disease to other species. *Onderstepoort Journal of Veterinary Research*, 63:239–244.

KEFFEN, R.H. 1985. Heartwater challenge of two Cape buffalo (*Syncerus caffer*). *Journal of the South African Veterinary Association*, 56:55.

KEIM, P., KALIF, A., SCHUPP, J., HILL, K., TRAVIS, S.E., RICHMOND, K., ADAIR, D.M., HUGH-JONES, M., KUSKE, C.R. & JACKSON, P. 1997. Molecular evolution and diversity in *Bacillus anthracis* as detected by amplified fragment length polymorphism markers. *Journal of Bacteriology*, 179:818–824 (Abstract, Pubmed).

KITCHING, R.P. & CARN, V 1996. Lumpy skin disease, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 93–101

KOCK, R.A., WAMBUA, J.M., MWANZIA, J., ROSSITER, P., WAMWAYI, H. & KOCK, N.D. 1995. Rinderpest epidemic in the Tsavo National Park in Kenya. *Proceedings of the American Association of Zoo Veterinarians, Wildlife Disease Association, American Association of Wildlife Veterinarians Joint Conference*, East Lansing, Michigan, August 12–17· 98–104 (Abstract, CAB).

KOCK, N.D., KOCK, R.A., WAMBUA, J. & MWANZIA, J. 1999a. Pathological changes in free-living African ungulates during a rinderpest epizootic in Kenya, 1993 to 1997 *The Veterinary Record*, 145:527–528.

KOCK, R.A., WAMBUA, J.M., MWANZIA, J., WAMWAYI, H., NDUNGU, E.K., BARRETT, T., KOCK, N.D. & ROSSITER, P.B. 1999b. Rinderpest epidemic in wild ruminants in Kenya, 1993 to 1997 *The Veterinary Record*, 145:275–283.

KSIAZEK, T.G., JOUAN, A., MEEGAN, J.M., LE GUENNO, B., WILSON, M.L., PETERS, J., DIGOUTTE, J.P., GUILLAUD, M., MERZOUG, N.O. & TOURAY, E.M. 1989. Rift Valley fever amongst domestic animals in the recent West African outbreak. *Research in Virology*, 140:67–77 (Abstract, Pubmed).

KUPPER, W., WOLTERS, M. & TSCARF, I. 1983. Observation on Kob antelopes (*Kobus kob*) in Northern Ivory Coast and their epizootiological role in trypanosomosis transmission. *Zeitschrift für Angewandte Zoologie*, 70:277–283 (Abstract, CAB).

KUTTLER, K.L. 1984. *Anaplasma* infections in wild and domestic ruminants: A review. *Journal of Wildlife Diseases*, 20:12–20.

LAHIJANI, R.S., SUTTON, S.M., KLIEFORTH, R.B., MURPHY, M.F. & HEUSCHELE, W.P. 1994. Application of polymerase chain reaction to detect animals latently infected with agents of malignant catarrhal fever. *Journal of Veterinary Diagnostic Investigation*, 6:403–409 (Abstract, CAB).

LAWRENCE, J.A., DE VOS, A.J. & IRVIN, A.D. 1994a. East Coast fever, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 309–325.

LAWRENCE, J.A., DE VOS, A.J. & IRVIN, A.D. 1994b. Corridor disease, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 326–328.

LAWRENCE, J.A., DE VOS, A.J. & IRVIN, A.D. 1994c. Zimbabwe theileriosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer. G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 329–330.

LAWRENCE, J.A., DE VOS, A.J. & IRVIN, A.D. 1994d. *Theileria taurotragi* infection, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 334–335.

LI, H., SHEN, D.T., JESSUP, D.A., KNOWLES, D.P., GORHAM, J.R., THORNE, T., O'TOOLE, D. & CRAWFORD, T.B. 1996. Prevalence of antibody to malignant catarrhal fever virus in wild and domestic ruminants by competitive inhibition ELISA. *Journal of Wildlife Diseases*, 32:437–43 (Abstract, CAB).

LINDEQUE, P.M. & TURNBULL, P.C.B. 1994. Ecology and epidemiology of anthrax in the Etosha National Park, Namibia. *Onderstepoort Journal of Veterinary Research*, 61:71–83.

LOGAN, T.M., LINTHICUM, K.J., DAVIES, F.G., BINEPAL, Y.S. & ROBERTS, C.R. 1991 Isolation of Rift Valley fever virus from mosquitoes (Diptera:Culicidae) collected during an outbreak in domestic animals in Kenya. *Journal of Medical Entomology*, 28:293–295 (Abstract, Pubmed).

MACLACHLAN, N.J., WILSON, D., GARD, G., MELLOR, P.S. & NEVILL, E.M. 1998. Supporting document for the *OIE International Animal Health Code*, Chapter 2.1.9 on bluetongue. OIE Working Group on Bluetongue. Paris: Office International des Epizooties.

MADSEN, M. & ANDERSON, E.C. 1995. Serologic survey of Zimbabwean wildlife for brucellosis. *Journal of Zoo and Wildlife Medicine*, 26:240–245 (Abstract, Pubmed).

MANSELL, J.L.,TANG, K.N., BALDWIN, C.A., STYLER, E.L. & LIGGETT, A.D. 1995. Disseminated chlamydial infection in antelope. *Journal of Veterinary Diagnostic Investigation*, 7· 397–399.

MARCUS, M.B. & VAN DER LUGT, J.J. 1994. Sarcocystosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 253–266.

MARKESHIN, S.I., SMIRNOVA, S.E. & EVESTAF'EV, I.L. 1992. [An assessment of the status of natural foci of Crimean-Congo hemorrhagic fever in the Crimea]. *Zhurnal Mikrobiologii, Epidemiologii, i Immunobiolgii*, April, 28–31 (Abstract, CAB).

MATEUS, G. & GONZALEZ, M. 1991. Characteristics of a *Trypanosoma vivax* outbreak in Colombia. *Revista Cubana Ciencias Veterinarias*, 22:167–171 (Abstract, CAB).

MATTIOLI, R.C., JEAN, O. & BELEM, A.M. 1990. [Incidence of trypanosomiasis in wildlife of a game ranch in Burkina Faso]. *Revue d' Elevage et de Médecine Vétérinaire des Pays Tropicaux*, 43:459–465 (Abstract, Pubmed).

MCINNES, E.F., STEWART, C.G., PENZHORN, B.L. & MELTZER, D.G.A. 1991. An outbreak of babesiosis in imported sable antelope (*Hippotragus niger*). *Journal of the South African Veterinary Association*, 62:30–32.

METEYER, C.U., GONZALES, B.J., HEUSCHELE, W.P. & HOWARD, E.B. 1989. Epidemiologic and pathologic aspects of an epizoootic of malignant catarrhal fever in exotic hoofstock. *Journal of Wildlife Diseases*, 25:280–286.

MICHEL, A.L. 1993. Generation of a nucleic acid probe specific for Acelaphine herpesvirus 1 and its use for the detection of malignant catarrhal fever virus DNA in blue wildebeest calves (*Connochaetes taurinus*). *Onderstepoort Journal of Veterinary Research*, 60:87–93.

MOHAMMED, O.B. & HUSSEIN, H.S. 1994. Antibody prevalence of toxoplasmosis in Arabian gazelles and oryx in Saudi Arabia. *Journal of Wildlife Diseases*, 30:560–562.

MOLOO, S.K., ORINDA, G.O., SABWA, C.L., MINJA, S.H. & MASAKE, R.A. 1999. Study on the sequential tsetse transmitted *Trypanosoma congolense*, *T brucei*, and *T. vivax* infections in African buffalo, eland, waterbuck, N'Dama and Boran cattle. *Veterinary Parasitology*, 80:197–213 (Abstract, Pubmed).

MORRIS, R.S., PFEIFFER, D.U. & JACKSON, R. 1994. The epidemiology of *Mycobacterium bovis* infections. *Veterinary Microbiology*, 40:153–177.

MORVAN, J., ROLLIN, P.E., LAVENTURE, S., RAKOTOARIVONY, I. & ROUX, J. 1992. Rift Valley fever epizootic in the central highlands of Madagascar. *Research in Virology*, 143:407–415 (Abstract, Pubmed).

MUSHI, E.Z. & KARSTAD, L. 1979. Experimental infection of wildebeest with the herpesvirus of infectious bovine rhinotracheitis/infectious pustular vulvovaginitis. *Journal of Wildlife Diseases*, 15(4):579–583 (Abstract, CAB).

MUSHI, E.Z., KARSTAD, L., JESSETT, D.M. & ROSSITER, P.B. 1979. Observations on the epidemiology of the herpesvirus of infectious bovine rhinotracheitis/infectious pustular vulvovaginitis in wildebeest. *Journal of Wildlife Diseases*, 15:481–487 (Abstract, CAB).

MUSHI, E.Z., KARSTAD, L. & JESSETT, D.M. 1980a. Isolation of bovine malignant catarrhal fever virus from ocular and nasal secretions of wildebeest calves. *Research in Veterinary Science*, 29:168–171.

MUSHI, E.Z., ROSSITER, P.B., KARSTAD, L. & JESSETT, D.M. 1980b. The demonstration of cell-free malignant catarrhal fever herpesvirus in wildebeest nasal secretions. *Journal of Hygiene* (London), 85:175–179.

NANDI, S. & NEGI, B.S. 1999. Bovine ephemeral fever: a review. *Comparative Immunology, Microbiology and Infectious Diseases*, 22:81–91 (Abstract, Pubmed).

NEITZ, W.O. 1933. The blesbuck (*Damaliscus albifrons*) as a carrier of heartwater and bluetongue. *Journal of the South African Veterinary Medical Association*, 2:24–26.

NETTLETON, P.F. 1990. Pestivirus infections in ruminants other than cattle. *Revue Scientifique et Technique, Office International des Epizooties*, 9:31–50.

NETTLETON, P.F. 1996. Border disease, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 678–685.

NGERANWA, J.J., VENTER, E.H., PENZHORN, B.L., SOI, R.K., MWANZIA, J. & NYONGESA. 1998. Characterization of *Anaplasma* isolates from eland (*Taurotragus oryx*). Pathogenicity in cattle and sheep and DNA profiles analysis. *Veterinary Parasitology*, 74:109–122 (Abstract, Pubmed).

NJENGA, J.M., BWANGAMOI, O., KANG'ETHE, E.K., MUGERA, G.M. & MUTIGA, E.R. 1995. Comparative ultrastructural studies on *Besnoitia besnoiti* and *Besnoitia caprae*. *Veterinary Research Communications*, 19:295–308.

NORVAL, R.A.I., FIVAZ, B.H., LAWRENCE, J.A. & BROWN, A.F. 1984. Epidemiology of tick-borne diseases of cattle in Zimbabwe. II. Anaplasmosis. *Tropical Animal Health and Production*, 16:63–70 (Abstract, Pubmed).

O'REILLY, L.M. & DABORN, C.J. 1995. The epidemiology of *Mycobacterium bovis* infections in animals and man: a review . *Tuberculosis and Lung Disease*, 76 (Supplement I):1–46.

OBEREM, P.T & BEZHUIDENHOUT, J.D. 1987. Heartwater in hosts other than domestic ruminants. *Onderstepoort Journal of Veterinary Research*, 54:271–275.

ODENING, K., RUDOLPH, M., QUANDT, S., BENGIS, R.G., BOCKHARDT, I. & VIERTEL, D. 1998. *Sarcocystis* spp. in antelopes from southern Africa. *Acta Protozoologica*, 37:149–158 (Abstract, CAB).

OELOFSEN, M.J. & VAN DER RYST, E. 1999. Could bats act as reservoir hosts for Rift Valley fever virus? *Onderstepoort Journal of Veterinary Research*, 66:51–54.

OKOH, A.E.J., OYETUNDE, I.L. & IBU, J.O. 1986. Fatal heartwater in a captive sitatunga. *The Veterinary Record*, 118:696.

OLALEYE, O.D., TOMORI, O., LADIPO, M.A. & SCHMITZ, H. 1996. Rift Valley fever in Nigeria: infections in humans. *Revue Scientifique et Technique, Office International des Epizooties*, 15:923–935.

OTTE, M.J., ABUABARA, J.Y. & WELLS, E.A. 1994. *Trypanosoma vivax* in Colombia: epidemiology and production losses. *Tropical Animal Health and Production*, 26:146–156 (Abstract, CAB).

PALING, R.W., WAGHELA, S., MACOWAN, K.J. & HEATH, B.R. 1988. The occurrence of infectious diseases in mixed farming of domesticated wild herbivores and livestock in Kenya. *Journal of Wildlife Diseases*, 24:308–316 (Abstract, Pubmed).

PATHAK, D.C., RAHMAN, H., UPADHYAYA, T.N. & BARUAH, D.K. 1987. Tuberculosis in black-bucks (*Antilope cervicapra*). *Indian Medical Journal*, 11:52–53 (Abstract, CAB).

PERES-JORDA, J.L., IBANEZ, C., MUNOZ-CERVERA, M. & TELLEZ, A. 1995. Lyssavirus in *Eptesicus serotinus* (Chiropetra: Vesperatilionidae). *Journal of Wildlife Diseases*, 31: 372–377.

PERL, S., YADIN, H., YAKOBSON, B., ZUCKERMAN, E. & ORGAD, U. 1989. Pathological changes in mountain gazelles challenged with FMD virus, with special reference to pancreatic lesions. *Revue Scientifique et Technique. Office International des Epizooties*, 8:765–769.

PETER, T.E., ANDERSON, E.C., BURRIDGE, M.J. & MAHAN. S.M. 1998. Demonstration of a carrier state for *Cowdria ruminantium* in wild animal ruminants from Africa. *Journal of Wildlife Diseases*, 34:567–575.

PETER, T.E., ANDERSON, E.C., BURRIDGE, M.J., PERRY, B.D. & MAHAN, S.M. 1999a. Susceptibility of impala, sable, and tsessebe for *Cowdria ruminantium* infection (heartwater). *Journal of Parasitology*, 85:468–472 (Abstract, Pubmed).

PETER, T.F., BRYSON, N. R., PERRY, B.D., O'CALLAGHAN, C. J., SMITH, G.E., MLAMBO, G., HORAK, I.G., BURRIDGE,

M.J. & MAHAN, S.M. 1999b. *Cowdria ruminantium* infection in ticks in the Kruger National Park. *The Veterinary Record,* 145:304–307

PIENAAR, J.G., BIGALKE, R.D., TUSTIN, R.C. & NAUDÉ, T.W. 1964. The occurrence of coccidiosis in impala, *Aepyceros melampus* (Lichtenstein 1812). *Journal of the South African Veterinary Medical Association,* 35:333–341

PIPANO, E. 1994. *Theileria annulata* infection, in *Infectious diseases of livestock with special reference to southern Africa.* Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 341–348.

PIPANO, E. 1997 Vaccines against haemoparasitic diseases in Israel with special reference to quality measures. *Tropical Animal Health and Production,* 29(Supplement 4):86s–90s (Abstract, Pubmed).

PLOWRIGHT, W. 1990. Malignant catarrhal fever virus, in *Virus infections in ruminants,* edited by Z. Dinter & B. Morein. Amsterdam, Oxford, New York, Tokyo: Elsevier Science Publishers: 123–150.

POSPISCHIL, A., STIGLMAIR, H.M.T., VON HEGEL, G., WIESNER, H. & VON HEGEL, G. 1987. Abomasal cryptosporidiosis in mountain gazelles. *The Veterinary Record,* 121:379–380.

POTGIETER, F.T 1979. Epizootiology and control of anaplasmosis in South Africa. *Journal of the South African Veterinary Association,* 50:367–372.

POTGIETER, F.T & STOLTSZ, WH. 1994. Bovine anaplasmosis, in *Infectious diseases of livestock with special reference to southern Africa.* Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 351–370.

PRETORIUS, A., OELOFSEN, M.J., SMITH, M.S. & VAN DER RYST, E. 1997. Rift Valley fever virus: a sero-epidemiological study of small terrestrial vertebrates in South Africa. *American Journal of Tropical Medicine and Hygiene,* 57:693–698 (Abstract, Pubmed).

RECHAV, Y 1986. Seasonal activity and hosts of the vectors of Crimean-Congo haemorrhagic fever in South Africa. *South African Medical Journal,* 69:36–48 (Abstract, CAB).

REID, H.W. & BRIDGEN, A. 1991. Recovery of a herpesvirus from a roan antelope (*Hipppotragus equinus*). *Veterinary Microbiology,* 28:269–278.

REID, H.W. 1996. Malignant catarrhal fever. in *Manual of standards for diagnostic tests and vaccines.* Paris: Office International des Epizooties: 627–633.

RIETKERK, F.E., GRIFFIN, F.T., WOOD, B., MUBARAK, S.M., DELIMA, E.C., BADRI, O.M., LINDSAY, N.B. & WILLIAMSON, D.T. 1993. Treatment of bovine tuberculosis in an Arabian oryx (*Oryx leucoryx*). *Journal of Zoo and Wildlife Medicine,* 24:523–527 (Abstract, CAB).

ROSSITER, P.B., JESSETT, D.M., WAFULA, J.S., KARSTAD, L., CHEMA, S., TAYLOR, W.P., ROWE, L., NYANGE, J.C. & MUMBALA, M. 1983. Re-emergence of rinderpest as a threat in East Africa since 1979. *The Veterinary Record,* 113:459–461.

ROSSITER, P.B., TAYLOR, W.P., BWANGAMOI, B., NGEREZA, A.R., MOORHOUSE, P.D., HARESNAPE, J.M., WAFULA, J.S. & GUMM, I.D. 1987. Continuing presence of rinderpest virus as a threat in East Africa, 1983–1985. *The Veterinary Record,* 120:59–62.

ROSSITER, P.B., GUMM, I.D. & MIRANGI, P.K. 1988. Immunological relationship between malignant catarrhal fever virus, acelaphine herpesvirus and bovine cytomegalovirus (bovine herpesvirus 3). *Veterinary Microbiology,* 16:211–218.

ROSSITER, P.B., GUMM, I.D., STAGG, D.A., CONRAD, P.A., MUKOLWE, S., DAVIES, F.G. & WHITE, H. 1989. Isolation of bovine herpesvirus-3 from African buffaloes (*Syncerus caffer*). *Research in Veterinary Science,* 46:337–343 (Abstract, Pubmed).

ROSSITER, P.B. 1994. Rinderpest, in *Infectious diseases of livestock with special reference to southern Africa.* Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 735–758.

SANNUSI, A. 1991. A simple field diagnostic smear test for bovine besnoitiosis. *Veterinary Parasitology,* 39:185–188 (Abstract, Pubmed).

SCHILLHORN VAN VEEN, T.W., TRAPP, A.L., DAUNT, D. & RICHTER, N.A. 1986. Coccidiosis in a kudu. *Journal of the American Veterinary Medical Association,* 189:1178–1179 (Abstract, CAB).

SCHLATER, J. 1996. Trypanosomosis (tsetse-borne), in *Manual of standards for diagnostic tests and vaccines.* Paris: Office International des Epizooties: 660–664.

SCHREUDER, B.E., UILENBERG, G. & TONDEUR, W. 1977 Studies on Theileriidae (Sporozoa) in Tanzania. viii. Experiments with African buffalo (*Syncerus caffer*). *Tropenmedizin und Parasitologie,* 28:367–371 (Abstract, Pubmed).

SCOTT, G.R. 1990. Rinderpest virus, in *Virus infections in ruminants,* edited by Z. Dinter & B. Morein. Amsterdam, Oxford, New York, Tokyo: Elsevier Science Publishers: 341–354.

SCOTT, G.R. 1994. Lesser know rickettsias infecting livestock, in *Infectious diseases of livestock with special reference to southern Africa.* Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 371–377.

SCOTT, G.R & HERR, S. 1994. Q fever, in *Infectious diseases of livestock with special reference to southern Africa.* Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 390–395.

SEAL, B.S., KLIEFORTH, R.B. & HEUSCHELE, W.P. 1987 Evidence for variation among malignant catarrhal fever virus isolates. *Proceedings of the United States Animal Health Association,* 91:527–542 (Abstract, CAB).

SEAL, B.S., HEUSCHELE, W.P. & KLIEFORTH, R.B. 1989. Prevalence of antibodies to acelaphine herpesvirus 1 and nucleic acid hybridization analysis of virus isolated from captive exotic ruminants. *American Journal of Veterinary Research,* 50:1447–1453 (Abstract, CAB).

SHANTHIKUMAR, S.R., MALACHI, S.A. & MAYIYAGBE, K.A. 1985. Rinderpest outbreak in free-living wildlife in Nigeria. *The Veterinary Record,* 117:469–470.

SHANTHIKUMAR, S.R. & ATILOLA, M.A. 1990. Outbreaks of rinderpest in wild and domestic animals in Nigeria. *The Veterinary Record,* 126:306–307.

SHIMSHONY, A., ORGAD, U., BAHARAV, D., PRUDOVSKY, S., YAKOBSON, B., BAR MOSHE, B. & DAGAN, D. 1986. Malignant foot-and-mouth disease in mountain gazelles. *The Veterinary Record,* 119:175–176.

SHIMSHONY, A. 1988. Foot-and-mouth disease in mountain gazelle in Israel. *Revue Scientifique et Technique. Office International des Epizooties,* 7:917–923.

SIMPSON, V.R. 1979. Bluetongue antibody in Botswana's domestic and game animals. *Tropical Animal Health and Production,* 11:43–49.

SMITH, G.E., ANDERSON, E.C., BURRIDGE, M.J., PETER, T.F. & MAHAN, S.M. 1998. Growth of *Cowdria ruminantium* in tissue culture endothelial cells from wild African mammals. *Journal of Wildlife Diseases,* 34:297–304.

SOHN, R. & YUILL, T.M. 1991. Bluetongue and epizootic haemorrhagic disease in wild ruminants. *Bulletin for the Society for Vector Ecology,* 16:17–24 (Abstract, CAB).

SOINE, C., UATANAUA, G. & DEPNER, K.R. 1992. Prevalence of bovine viral diarrhoea virus in Namibian wildlife. *Tropical Animal Health and Production*, 24:125–126 (Abstract, CAB).

SONGA, E.B., HAMERS, R., RICKMAN, R., NANTULYA, V.M., MULLA, A.F. & MAGNUS, E. 1991. Evidence for the widespread asymptomatic *Trypanosoma rhodesiense* human infection in the Luangwa Valley (Zambia). *Tropical Medicine and Parasitology*, 42:389–393 (Abstract, Pubmed).

ST. GEORGE, T.D. 1994. Ephemeral fever, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 553–562.

ST. GEORGE, T.D. & STANDFAST, H.A. 1994. Diseases caused by Akabane and related Simbu-group viruses, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 681–687.

STAFFORD, K.J. 1991. A review of the diseases of and parasites of the Kafue lechwe (*Kobus leche kafuensis*). *Journal of Wildlife Diseases*, 27:661–667.

STAGG, D.A., BISHOP, R.P., SHAW, M.K., WESONGA, D., ORINDA, G.O., GROOTENHUIS, J.G., MOLYNEUX, D.H. & YOUNG, A.S. 1994. Characterisation of *Theileria parva* which infects waterbuck (*Kobus defassa*). *Parasitology*, 108:543–54 (Abstract, Pubmed).

STEINBERG, H. 1988. Johne's disease (*Mycobacterium paratuberculosis*) in a Jimela topi (*Damaliscus lunatus jimela*). *Journal of Zoo Animal Medicine*, 19:33–41 (Abstract, CAB).

STOLTE, M., ODENING, K. & BOCKHARDT, I. 1996. Antelopes kept in European zoological gardens as intermediate hosts of *Sarcocystis* species. *Parasitologia Roma*, 38:565–570 (Abstract, CAB).

STOLTSZ, W.H. 1994. Ovine and caprine anaplasmosis, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York : Oxford University Press : 431–438.

STOTT, J.L., ELSE, K.C., MCGOWAN, B., WILSON, L.K. & OSBURN, B.I. 1981. Epizootiology of bluetongue virus in Western United States. *Proceedings of the United States Animal Health Association*, 85:170–180 (Abstract, CAB).

SWANEPOEL, R., SHEPHERD, A.J., LEMAN, P.A., SHEPHERD, S.P. & MILLER, G.B. 1985. A common-source outbreak of Crimean-Congo haemorrhagic fever on a dairy farm. *South African Medical Journal*, 68:635–637.

SWANEPOEL, R., SHEPHERD, A.J., LEMAN, P.A., SHEPHERD, S.P., MACGILLIVRAY, G.M., ERASMUS, M.J., SEARLE, L.A. & GILL, D.E. 1987. Epidemiological and clinical features of Crimean-Congo haemorrhagic fever in southern Africa. *American Journal of Tropical Medicine and Hygiene*, 36:120–132.

SWANEPOEL, R. 1994a. Rabies, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 493–552.

SWANEPOEL, R. 1994b. Crimean-Congo haemorrhagic fever, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 723–732.

SWANEPOEL, R. & COETZER, J.A.W. 1994. Rift Valley fever, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 688–718.

SWANEPOEL, R., LEMAN, P.A., BURT, F.J., JARDINE, J., VERWOERD, D.J., CAPUA, I., BRÜCKNER, G.K. & BURGER, W.P. 1998. Experimental infection of ostriches with Crimean-Congo haemorrhagic fever virus. *Epidemiology and Infection*, 121: 427–432 (Abstract, Pubmed).

TAYLOR, W.P. 1996. Rinderpest, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 69–76.

TERPSTRA, C. 1994. Nairobi sheep disease, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 718–722

THAM, K.M., NG & YOUNG, L.W. 1994. Polymerase chain reaction amplification of wildebeest-associated malignant catarrhal fever virus DNA. *Archives of Virology*, 135:355–364.

THOMAS, S.E., WILSON, D.E. & MASON, T.E. 1982. *Babesia, Theileria* and *Anaplasma* spp. infecting sable antelope, *Hippotragus niger* (Harris, 1838), in southern Africa. *Onderstepoort Journal of Veterinary Research*, 49:163–166.

THOMSON, G.R, VOSLOO, W., ESTERHUYSEN, J.J. & BENGIS, R.G. 1992. Maintenance of foot-and-mouth disease viruses in buffalo (*Syncerus caffer* Sparrman, 1779) in southern Africa. *Revue Scientifique et Technique, Office International des Epizooties*, 11:1097–1107.

THOMSON, G.R. 1994. Foot-and-mouth disease, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 2, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 825–852.

THOMSON, G.R. 1995. Overview of foot-and-mouth disease in southern Africa. *Revue Scientifique et Technique, Office International des Epizooties*, 14:503–520.

THORBURN, J.H. & THOMAS, A.D. 1940. Tuberculosis in the Cape Kudu. *Journal of the South African Veterinary Medical Association*, 65:70–83.

THOREL, M.F. 1994. Paratuberculosis (Johne's disease), in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 218–228.

THURLBECK, W.M., BUTAS, C.A., MANKIEWICZ, E.M. & LAWS, R.M. 1965. Chronic pulmonary tuberculosis in the wild buffalo (*Syncerus caffer*) in Uganda. *American Review of Respiratory Diseases*, 92:801–805 (Abstract, Pubmed).

TRUC, P., FORMENTY, P., KOMOIN OKA, C., DIALLO, P.B. & LAUGINIE, F. 1997. Identification of trypanosomes isolated by KIVI from wild animals in Cote d'Ivoire: diagnostic, taxonomic and epidemiological considerations. *Acta Tropica*, 67:187–196 (Abstract, CAB).

TURELL, M.J. & PERKINS, P.V. 1990. Transmission of Rift Valley fever virus by the sand fly, *Phlebotomus dubosqui* (Diptera: Psychodidae). *American Journal of Tropical Medicine and Hygiene*, 42:185–188 (Abstract, Pubmed).

TURELL, M.J & DICKSON, D.L. 1992. Recoverability of Rift Valley fever and sandfly fever Sicilian viruses from infected *Phlebotomus papatasi* (Diptera: Psychodidae) trapped in various oils. *Journal of the American Mosquito Association*, 8:92–94 (Abstract, Pubmed).

TURELL, M.J. & HAY, B.H. 1998. Susceptibility of selected strains of Australian mosquitoes (Diptera:Culicidae) to Rift Valley fever virus. *Journal of Medical Entomology*, 35:132–135.

TURELL, M.J., PRESLEY, S.M., GAD, A.M., COPE, S.E., DOHM, D.J., MORRILL, J.C. & ARTHUR, R.R. 1996. Vector competence of Egyptian mosquitoes for Rift Valley fever virus. *American Journal of Tropical Medicine and Hygiene*, 54:136–139 (Abstract, Pubmed).

TURNBULL, P.C., BELL, R.H., SAIGAWA, K., MUNYEMYEMBE, F.E., MULENGA, C.K. & MAKALA, L.H. 1991. Anthrax in wild-life in the Luangwa Valley, Zambia. *The Veterinary Record*, 128:399–403.

TURNBULL, P.C., DOGANAY, M., LINDEQUE, P.M., AYGEN, B. & MCLAUGHLIN, J. 1992. Serology and anthrax in humans, livestock and Etosha National Park wildlife. *Epidemiology and Infection*, 108:299–313 (Abstract, Pubmed).

VAN DER LUGT, J.J. & KRIEK, J.C. 1988. Chlamydiosis in a springbok (*Antidorcas marsupialis*). *Journal of the South African Veterinary Association*, 59:33–37.

VAN DER RIET, F.D., SAYED, A.R., BARNARD, B.J., VAN TONDER, E.M. & CROUSE, W.J. 1985. Arthropod-borne virus zoonosis surveillance in the Cape Province: 1. Prospective serological investigations for virus activity in the Beaufort West and Middelburg Districts during 1981. *Journal of the South African Veterinary Association*, 56:25–29.

VAN EEDEN, P.J., JOUBERT, J.R., VAN DE WAL, B.W., KING, J.B., DE KOCK, A. & GROENEWALD, J.H. 1985. A nosocomial outbreak of Crimean-Congo haemorrhagic fever at Tygerberg Hospital. Part 1. Clinical features. *South African Medical Journal*, 68:711–717 (Abstract, Pubmed).

VAN OIRSCHOT, J.T. 1996. Infectious bovine rhinotracheitis, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 281–290.

VAN WINKLE, T.J. 1985. Cryptosporidiosis in young artiodactyls. *Journal of the American Veterinary Medical Association*, 187: 1170–1172 (Abstract, Pubmed).

VERWOERD, D.W. & ERASMUS, B.J. 1994. Bluetongue, in *Infectious diseases of livestock with special reference to southern Africa*. Vol. 1, edited by J.A.W. Coetzer, G.R. Thomson & R.C. Tustin. Cape Town, Oxford, New York: Oxford University Press: 443–459.

VOGEL, S.W. & HEYNE, H. 1996. Rinderpest in South Africa—100 years ago. *Journal of the South African Veterinary Association*, 67:164–170.

VOSLOO, W., BASTOS, A.D., KIRKBRIDE, E., ESTERHUYSEN, J.J., VAN RENSBURG, D.J., BENGIS, R.G., KEET, D.W. & THOMSON, G.R. 1996. Persistent infection of African buffalo (*Syncerus caffer*) with SAT-type foot-and-mouth disease viruses. Rate of fixation of mutation, antigenic change and interspecies transmission. *Journal of General Virology*, 77:1457–1467.

WAFULA, J.S. & KARIUKI, D.P. 1987. A recent outbreak of rinderpest in East Africa. *Tropical Animal Health and Production*, 19: 173–176 (Abstract, Pubmed).

WAFULA, J.S., MUSHI, E.Z. & KARSTAD, L. 1982. Antibodies to rinderpest virus in the sera of some wildlife in Kenya. *Bulletin of Animal Health and Production in Africa*, 30:363–365 (Abstract, CAB).

WAGHELA, S. & KARSTAD, L. 1986. Antibodies to *Brucella* spp. among blue wildebeest and African buffalo in Kenya. *Journal of Wildlife Diseases*, 22:189–192 (Abstract, Pubmed).

WAMWAYI, H.M., KARIUKI, D.P., WAFULA, J.S., ROSSITER, P.B., MBUTHIA, P.G. & MACHARIA, S.R. 1992. Observations on rinderpest in Kenya. *Revue Scientifique et Technique, Office International des Epizooties*, 11:769–784.

WAMWAYI, H.M., FLEMING, M. & BARRETT, T. 1995. Characteristics of African isolates of rinderpest virus. *Veterinary Microbiology*, 44:151–163.

WEBER, A. & VAN HOVEN, W. 1992. Tuberculosis of the parotid salivary gland in a kudu, *Tragelaphus strepsiceros*. *Koedoe*, 35:119–122 (Abstract, CAB).

WEYER, K., FOURIE, P.B., DURRHEIM, D., LANCASTER, J., HASLOV, K. & BRYDEN, H. 1999. *Mycobacterium bovis* as a zoonosis in the Kruger National Park, South Africa. *International Journal of Tuberculosis and Lung Disease*, 3:1113–1119 (Abstract, Pubmed).

WHITFORD, H.W. 1996. Anthrax, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 170–180.

WILSON, M.L., CHAPMAN, L.E., HALL, D.B., DYKSTRA, E.A., BA K., ZELLER, H.G., TRAORE-LAIZANA, M., HERVY, J.P., LINTHICUM, K.J. & PETERS, C.J. 1994. Rift Valley fever in northern Senegal: human factors and potential vectors. *American Journal of Tropical Medicine and Hygiene*, 50:663–675 (Abstract, Pubmed).

WITTMANN, G. 1990. [Virus carriers in foot-and-mouth disease]. *Berliner und Münchner Tierärztliche Wochenschrift*, 103:145–50 (Abstract, Pubmed).

WOODFORD, W.M. 1982. Tuberculosis in wildlife in the Ruwenzori National Park, Uganda. *Tropical Animal Health and Production*, 14:155–160 (Abstract, Pubmed).

WRIGHT, I.G. & LEACH, G. 1996. Bovine anaplasmosis, in *Manual of standards for diagnostic tests and vaccines*. Paris: Office International des Epizooties: 295–304.

YERUHAM, I., NIR, O., BRAVERMAN, Y., DAVIDSON, M., GRINSTEIN, H., HAMOVITCH, M. & ZAMIR, O. 1995. Spread of lumpy skin disease in Israeli dairy herds. *The Veterinary Record*, 137:91–93.

YOUNG, E., BASSON, P.A. & WEISS, K.E. 1970. Experimental infection of giraffe (*Giraffa camelopardalis* [Linnaeus 1962]), impala (*Aepyceros melampus* [Lichtenstein, 1812]) and the Cape buffalo (*Syncerus caffer* [Sparman, 1779]) with lumpy-skin disease virus. *Onderstepoort Journal of Veterinary Research*, 37:78–88.

ZAUGG, J.L. 1987. Experimental infections of *Anaplasma ovis* in pronghorn antelope. *Journal of Wildlife Research*, 23:205–210 (Abstract, CAB).

ZELLER, H.G., CORNET, J.P. & CAMICAS, J.L. 1994. Experimental transmission of Crimean-Congo heamorrhagic fever virus by west African wild ground-feeding birds to *Hyalomma marginatum rufipes* ticks. *American Journal of Tropical Medicine and Hygiene*, 50:676–681 (Abstract, CAB).

ZIEGER, U., PANDEY, G.S., KRIEK, N.P.J. & CAULDWELL, A.E. 1998. Tuberculosis in Kafue lechwe (*Kobus leche kafuensis*) and in a bushbuck (*Tragelaphus scriptus*) on a game ranch in Central Province, Zambia. *Journal of the South African Veterinary Association*, 69:98–101.

APPENDIX

Common and systematic names of antelopes referred to in text

Addax	*Addax nasomaculatus*	Kudu (greater)	*Tragelaphus strepsiceros*
Arabian oryx	*Oryx leucoryx*	Lechwe	*Kobus leche*
Arabian gazelle	*Gazella dorcas*	Mountain gazelle	*Gazella gazella****
Blackbuck	*Antilope cervicapra ****	Nilgai/Indian antelope	*Boselaphus tragocamelus****
Blesbok	*Damaliscus pygarthus philipsi**	Nyala	*Tragelaphus angasi*
Bongo	*Tragelaphus eurycerus*	Oribi	*Ourebia ourebi*
Bontebok	*Damaliscus pygarthus dorcas*	Oryx or Beisa oryx	*Oryx beisa*
Buffalo (African or Cape)	*Syncerus caffer*	Pronghorn	*Antilocapra americana****
Bushbuck	*Tragelaphus scriptus*	Reedbuck	*Redunca arundinum*
Duiker (red flanked)	*Cephalus rufilatus*	Roan antelope	*Hippotragus equinus*
Duiker (blue)	*Philantomba monticola*	Sable antelope	*Hippotragus niger ***
Duiker (common or grey)	*Sylvicapra grimmia*	Saiga antelope	*Saiga tatarica ****
Duiker (red)	*Cephalophus natalensis*	Sitatunga	*Tragelaphus spekei*
Eland	*Taurotragus oryx*	Slender horned gazelle	*Gazella leptoceros*
Four horned antelope	*Tetracerus quadricornis****	Speke's gazelle	*Gazella spekei*
Gemsbok	*Oryx gazella*	Springbok	*Antidorcas marsupialis*
Gerenuk	*Litocranius walleri*	Steenbok	*Raphicerus campestris*
Giraffe	*Giraffa camelopardalis*	Suni	*Neotragus moschatus*
Grant's gazelle	*Gazella granti*	Thompson's gazelle	*Gazella thompsoni*
Grey rhebuck	*Pelea capreolus*	Topi	*Damaliscus korrigum*
Grysbok	*Raphicerus sharpei*	Tsessebe	*Damaliscus lunatus*
Impala	*Acepyceros melampus*	Waterbuck	*Kobus ellipsiprymnus*
Klipspringer	*Oreotragus oreotragus*	Wildebeest (blue)	*Connochaetes taurinus*
Kob	*Kobus kob*	Wildebeest (black)	*Connochaetes gnou*
Kongoni/Cokes's hartebeest	*Alcephalus busephalus cokei*		

* Also sometimes given as *Damaliscus albifrons*
** Also sometimes given as *Ozanna grandicomis*
*** Not indigenous to Africa