

Geographic range of vector-borne infections and their vectors: the role of African wildlife

M. van Vuuren ⁽¹⁾ & B.L. Penzhorn ^{(1, 2)*}

(1) Department of Veterinary Tropical Diseases, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110, South Africa

(2) Research Associate, National Zoological Gardens, Pretoria, South Africa

*Corresponding author: banie.penzhorn@up.ac.za

Summary

The role of African wildlife in the occurrence of vector-borne infections in domestic animals has gained renewed interest as emerging and re-emerging infections occur worldwide at an increasing rate. In Africa, biodiversity conservation and the expansion of livestock production have increased the risk of transmitting vector-borne infections between wildlife and livestock. The indigenous African pathogens with transboundary potential, such as Rift Valley fever virus, African horse sickness virus, bluetongue virus, lumpy skin disease virus, African swine fever virus, and blood-borne parasites have received the most attention.

There is no evidence for persistent vector-borne viral infections in African wildlife. For some viral infections, wildlife may act as a reservoir through the inter-epidemic circulation of viruses with mild or subclinical manifestations. Wildlife may also act as introductory or transporting hosts when moved to new regions, e.g. for lumpy skin disease virus, Rift Valley fever virus and West Nile virus. Wildlife may also act as amplifying hosts when exposed to viruses in the early part of the warm season when vectors are active, with spillover to domestic animals later in the season, e.g. with bluetongue and African horse sickness.

Some tick species found on domestic animals are more abundant on wildlife hosts; some depend on wildlife hosts to complete their life cycle. Since the endemic stability of a disease depends on a sufficiently large tick population to ensure that domestic animals become infected at an early age, the presence of wildlife hosts that augment tick numbers may be beneficial. Many wild ungulate species are reservoirs of *Anaplasma* spp., while the role of wildlife in the epidemiology of heartwater (*Ehrlichia ruminantium* infection) has not been elucidated. Wild ungulates are not usually reservoirs of piroplasms that affect livestock; however, there are two exceptions: zebra, which are reservoirs of *Babesia caballi* and *Theileria equi*, and buffalo, which are reservoirs of *Theileria parva*. The latter causes Corridor disease when transmitted from buffalo to cattle, but this appears to be a self-limiting condition, at least in southern Africa. Wild animals are important reservoirs of tsetse-transmitted *Trypanosoma* spp. infection. The distribution and abundance of some tsetse species, e.g. *Glossina morsitans* and *G. pallidipes*, are closely related to the occurrence of their preferred wildlife hosts.

Keywords

Africa – African horse sickness – African swine fever – Anaplasmosis – Bluetongue – Bovine ephemeral fever – Buffalo-associated *Theileria parva* – *Ehrlichia ruminantium* – Endemic stability – Heartwater – Lumpy skin disease – *Nagana* – Rift Valley fever – *Theileria parva* – Trypanosomiasis – Vector – Vector-borne infection – Viraemia – West Nile virus – Wildlife.

Introduction

The occurrence of disease agents in free-ranging wildlife may present a risk to the health of domestic animals and human beings as well as to other wild animals. In general, wild animals are susceptible to infection by the same viruses, bacteria and protozoa that infect domesticated animals. Disease transmission can therefore occur in both directions and disease relationships between wild and domestic animals should be viewed as a two-way street. The examples that follow include diseases where wild animals represent a true risk factor, and, less commonly, where they may harbour a significant pathogen while posing little or no threat to other species.

Viral diseases

Rift Valley fever

Rift Valley fever (RVF) is a viral haemorrhagic fever of domestic ruminants and humans. Although a few countries in Africa have not reported the presence of antibodies or documented outbreaks, it is generally accepted that RVF is endemic in most parts of Africa. It has also been reported from the Arabian Peninsula, with the first confirmed outbreak outside Africa occurring in Saudi Arabia and Yemen in 2000 (1, 2). Mosquitoes are biological and mechanical vectors of the virus and are recognised as the primary vectors responsible for the transmission of RVF following the start of the warm or rainy season (3).

The role of wildlife in the maintenance and transmission of the RVF virus (RVFV) is not well defined. Low prevalences of antibody to RVFV but little or no evidence of the disease have been detected in a number of African wildlife species (4, 5, 6). Anderson and Rowe (5) reported antibody in African buffalo (*Syncerus caffer*), black (*Diceros bicornis*) and white (*Ceratotherium simum*) rhinoceroses, waterbuck (*Kobus ellipsiprymnus*), sable antelope (*Hippotragus niger*) and impalas (*Aepyceros melampus*) in Zimbabwe. Similarly, abortion and low death rates have been recorded in water buffalo (*Bubalus bubalis*) in Egypt (7). Antibody to RVFV in camel sera has been reported from Kenya, the sub-Saharan region of Nigeria, Egypt and the Sudan (8). The disease has historically been regarded as virtually absent in free-ranging wildlife and the documented reports have contributed to this perception. This was challenged, however, when abortions occurred in African buffalo confined to a pen in the Kruger National Park, South Africa, in 1999 (9). During the same outbreak, the virus was isolated from a waterbuck and a giraffe (*Giraffa camelopardalis*), which were found dead with lesions compatible with RVFV infection (3). During the major 2010 outbreak in South Africa, deaths were recorded among, *inter alia*, sable antelope, greater kudu (*Tragelaphus*

strepsiceros), springboks (*Antidorcas marsupialis*) and deer. There is no evidence of clinical cases in carnivores. It is now accepted that a serious challenge with high concentrations of virulent virus may, under ideal temporal and spatial conditions, cause clinical RVF in free-ranging wildlife. In the light of documented information, however, this still does not seem to be a regular occurrence.

The role of wildlife in the maintenance and transmission of RVFV on the African continent is probably similar to that of domestic ruminants in some regions where the inter-epidemic circulation of RVFV occurs, with mild or subclinical manifestations (1).

African horse sickness

African horse sickness virus (AHSV) is transmitted biologically by *Culicoides* spp. Of these, *C. imicola* and *C. bolitinos* have been shown to play an important role in Africa. The prevalence of AHS is therefore influenced by climatic and other conditions which favour the breeding of *Culicoides* spp. (10). African horse sickness virus is endemic to sub-Saharan Africa but periodically makes brief excursions beyond this area. The disease occurred in Yemen in 1930 and in what was then Palestine, Syria, Lebanon and Jordan in 1944. During the summer of 1959, AHS caused by serotype 9 occurred in the south-eastern regions of Iran. This was followed by outbreaks during the spring of 1960 in most of the Middle East (Cyprus, Turkey, Iraq, Syria, Lebanon, Jordan), as well as in Afghanistan, Pakistan and India. Between 1959 and 1961, this region lost more than 300,000 equids due to AHS (11, 12, 13). Between 1987 and 1990, outbreaks of AHS occurred in Spain; the original source of infection was suspected to be ten plains zebras (*Equus quagga*) imported from Namibia (14).

Among African wildlife, antibodies against AHSV have only been found in plains zebras, African elephants (*Loxodonta africana*) and black and white rhinoceroses. The possibility that elephants are reservoir hosts of AHSV has arisen; however, a group of 100 elephants tested in an endemic AHS area in South Africa had high complement-fixing titres to both bluetongue and AHSV, but no neutralising antibodies to these viruses, indicating that the sera of elephants react non-specifically in the complement fixation test. In contrast, in a similar study in Kenya, elephants were shown to have neutralising antibody to AHSV (15).

It has been shown that a continuous transmission cycle of AHSV between *Culicoides* midges and plains zebras exists in the Kruger National Park. Under such circumstances, a sufficiently large zebra population can act as a reservoir for the virus (16). It has also been suggested that donkeys may play a similar role in parts of Africa where there are large donkey populations. Zebras are often implicated as the cause of AHS outbreaks, but the role they play in

the epidemiology of AHS and in the spread of the virus is probably exaggerated. The duration of viraemia in infected zebras is longer than in horses. In an experimental study, low levels of infectious AHSV were isolated intermittently from a zebra for up to 40 days (16). In horses, viraemia does not exceed 21 days. When zebras become exposed to AHSV, they mount a humoral immune response, the infection is cleared from the circulation, and the virus becomes non-infectious to midges. Zebras can, therefore, similarly to horses and donkeys, fulfil the role of maintenance or amplifying hosts in equid populations, but are not major reservoirs of AHSV and do not play an important role in overwintering of the virus. In addition, a carrier state in zebras has never been proven. There are, in other words, no long-term reservoirs of AHSV.

In 1987, zebras imported into Spain caused an outbreak of AHS, yet the disease persisted in the country for four years, in an area where there were no zebras. More examples that exclude zebras as being essential in the epidemiology of AHS include the outbreaks that occurred in the Middle East in the 1960s, and the fact that serotype 9 of the virus is found in West Africa where zebras no longer occur (12). Although the period of viraemia in a zebra is longer than in a horse, the greater susceptibility of horses to AHS and the more frequent movement of horses justify the perception that horses probably play a far greater role in the spread and transmission of AHSV than zebras do. Outbreak data in South Africa have shown that AHS outbreaks start in areas of high horse density where zebras are not necessarily present.

Recently, several wild herbivores were indirectly implicated in the epidemiology of AHS when *C. bolitinos* was described as a second species of *Culicoides* involved in the transmission of AHSV in the field. *Culicoides bolitinos* breeds, among other places, in the dung of cattle, African buffalo and blue wildebeest (*Connochaetes taurinus*). The preference of *C. bolitinos* for the dung of wild bovids may enable it to migrate into areas where horses are present and *C. imicola* may be scarce or absent (17).

Bluetongue

Bluetongue (BT) was described for the first time in South Africa in 1880 (18). The distribution of the virus closely matches the distribution of vector-competent midge species and climatic conditions that support a large population of these insects. The virus therefore occurs more commonly in tropical and sub-tropical regions of the world, between the latitudes of 40°N and 35°S (18, 19). Since 1998 there has been a dramatic change in the distribution of BT, with the disease having spread into countries of north-western Europe and Scandinavia (20, 21). The enzootic nature of BT virus (BTV) in large regions of the African continent and, more specifically, southern Africa is supported by climatic

factors that favour the maintenance and recirculation of the virus in its vertebrate and non-vertebrate hosts. Reservoir and amplifying hosts, such as wildlife, cattle and goats, compounded by the ubiquitous distribution of suitable midge species, contribute to the persistence and transmission of BTV. In areas where the winters are mild, BTV may be transmitted throughout the year (22).

The susceptibility of wild ruminants was first established in South Africa in 1933 by the experimental infection of blesbok (*Damaliscus dorcas phillipsi*) (23). The antelope developed a subclinical infection with a sufficiently high viraemia to infect sheep when they were experimentally injected with infected blood. It is now generally accepted that all ruminant species are susceptible to infection with BTV. Although African antelope do not develop clinical disease, white-tailed deer (*Odocoileus virginianus*), pronghorn (*Antilocapra americana*) and desert bighorn sheep (*Ovis canadensis*) of North America may develop severe clinical disease (24).

The potential role of camels as reservoirs for BTV gained prominence when BT emerged in Morocco in 2004, where it had always been regarded as an exotic disease. Clinical disease derived from BTV had never previously been observed in camels. The experimental infection of three camels with a Moroccan BTV-1 isolate via the subcutaneous, intramuscular and intravenous routes did not yield any clinical signs. Virus was isolated from the blood of all three animals, however, leading to the conclusion that camels may act as a reservoir for BTV and may play a role in its transmission (25). However, the lowest threshold cycle (Ct) value with a real-time reverse-transcription polymerase chain reaction (RT-PCR) in the camels in that study was 31.88 (as opposed to Ct values as low as 20 documented in sheep in other studies), which suggested that BTV replication may not be as efficient in camels as in sheep.

The role of wildlife as maintenance hosts in nature has not yet been resolved. Illness and death as a result of BT in African wildlife are unknown, and infection essentially goes unnoticed. Some wild ruminant species may therefore fulfil the role of reservoirs of BTV. An alternative hypothesis is that the virus may be maintained in a subclinically infected unknown reservoir host population, in which viraemia may be extended or of a relapsing nature (26, 27).

West Nile virus infection

West Nile virus (WNV) is widely distributed throughout Africa, the Middle East, portions of Europe, Australia, the Americas and the Caribbean (28). West Nile virus is usually transmitted among wild birds by bird-feeding mosquitoes, including *Culex* and *Aedes* spp., and is spread by migratory birds. West Nile virus is an example of a disease that entered a country (the United States, 1999) as a foreign disease

which subsequently became established in the wildlife population. It is maintained in wild bird populations, especially American crows. Since 2006/2007 it has also become established in South American birds.

Once introduced to local mosquitoes, the virus is amplified among susceptible resident birds fed upon by ornithophilic mosquitoes. Various avian species are therefore introductory hosts, amplifying hosts and transporting hosts (29). This pattern allows for the perpetuation and subsequent establishment of the virus in a continuous transmission cycle, as opposed to the infection of dead-end hosts, such as horses (30).

During periods of adult mosquitoes feeding on bird blood, the virus is amplified and spillover infection can take place from the avian–mosquito cycle to mammalian species, such as humans and horses. West Nile virus is now the most widespread and well-travelled arthropod-borne virus in the world and eradicating the disease is no longer a possibility because of its extensive infection of wildlife (31).

Lumpy skin disease

Lumpy skin disease (LSD) is an economically important disease of cattle occurring in Africa and parts of the Middle East. Until 1989, when LSD was reported from Israel, it was regarded as a uniquely African disease (32). Since 2007, when renewed outbreaks of the disease occurred in Israel, the distribution of the disease has changed significantly and LSD has also been reported in Kuwait, Bahrain, Oman, Yemen, the Palestinian territories and Lebanon. Lumpy skin disease broke out in Turkey in August 2013 while outbreaks continued in Israel, Jordan, the Palestinian Authority (West Bank), Lebanon, Iraq and Egypt (where LSD has been endemic since the late 1980s) (33).

It is generally accepted that the most important mode of transmission of LSD virus (LSDV) is probably through mechanical transmission of the virus by blood-feeding vectors. Recent studies have shown that ticks such as *Amblyomma hebraeum* and *Rhipicephalus appendiculatus* can play a role in the mechanical (intrastadial) and transstadial transmission of LSDV (34, 35). The passage of LSDV from infected female ticks through the eggs to the next generation of larvae has been demonstrated in *A. hebraeum*, *R. appendiculatus* and *R. decoloratus*. Transmission to recipient animals by *A. hebraeum* and *R. appendiculatus* larvae has also been shown (36, 37). The finding of the transovarial passage of LSDV in female ticks provided strong evidence of the potential for *A. hebraeum*, *R. appendiculatus* and *R. decoloratus* to be reservoir hosts for LSDV. Although there is a major insect-borne component in the transmission of LSD, the spread of capripoxviruses into new areas is predominantly associated with animal movement through trade.

Since the first description of LSD in Zambia in 1929, several reports of wild antelope species with typical lesions have appeared but the virus has never been isolated in cell cultures from skin lesions (38, 39). These included, *inter alia*, suspected cases in five water buffalo in Egypt (32), in Arabian oryx (*Oryx leucoryx*) in Saudi Arabia (40), in springbok in Namibia and in oryx (*O. gazella*) in South Africa (32). Experimental infections led to severe disease, including mortalities in impala and giraffe, but clinical disease and seroconversion were not detected in black wildebeest (*Connochaetes gnou*) and African buffalo (41). In contrast, Davies reported the detection of antibodies in African buffalo in 1982 (42). Support for this observation was not found in studies completed in the Kruger National Park (P.G. Howell & J.A.W. Coetzer, unpublished data, 1998). The consensus is that wildlife does not play a meaningful role in the spread and maintenance of LSDV (43).

African swine fever

Argasid ticks of the *Ornithodoros moubata* complex are the biological hosts of African swine fever virus (ASFV). They are able to transmit virus when feeding on the blood of pigs but virus excreted in the coxal fluid of the ticks may transmit infection via broken skin (44). African swine fever is an example of a disease that is both vector-borne and contagious. Tick-borne transmission is important in the sylvatic cycle in wildlife and in the transmission from wildlife to domestic pigs. Once domestic pigs are infected, the highly contagious nature of the disease is responsible for its rapid spread.

The role of African wildlife in the maintenance of ASFV is limited to wild pigs, such as the warthog (*Phacochoerus aethiopicus*), bushpig (*Potamochoerus porcus*) and giant forest hog (*Hylochoerus meinertzhageni*), which may act as reservoirs of the virus (45). In countries in southern and eastern Africa, where the virus is present in a sylvatic cycle between wild pigs and ticks of the *O. moubata* complex, domestic pigs become infected when bitten by virus-infected ticks, followed by contagious spread in the herd. Countries where endemicity is confined to the sylvatic cycle include the northern part of South Africa, Zimbabwe, Botswana, Namibia, Mozambique, Malawi, Zambia, Tanzania, Uganda and Kenya. In regions in Africa where wild pigs are not present or play no role in the spread of the disease, the virus is maintained in a cycle involving only domestic pigs, with or without tick involvement (46).

Bovine ephemeral fever

Bovine ephemeral fever (BEF) is an acute, insect-borne, viral disease of cattle and water buffalo that varies in its clinical expression in individual animals. The first isolation and characterisation were described in South Africa in 1967.

The disease is endemic in much of Africa, southern Asia (including the Middle East) and Australia but has never been reported from the western hemisphere (47).

The first isolation from a mixed pool of *Culicoides* took place in Kenya in 1974 and this created a perception that *Culicoides* spp. are the main vectors of BEF virus (BEFV). Multiple vectors are probably involved, but the requirement for intravenous inoculation when conducting experimental infections in cattle makes mosquitoes the most likely vectors. It is now accepted that the virus is principally spread by Anopheline and Culicine mosquitoes (48). Anderson and Rowe (1998) documented positive micro-neutralisation tests for BEF in seven of eight African antelope species tested (5). They point out that they were uncertain whether the antibodies were specific for BEFV or were cross-reacting antibodies from infection with related lyssaviruses. However, other published surveys recorded antibodies to BEFV in a wide variety of free-ranging antelope species. The overwintering mechanism of the virus is unknown but is unlikely to be in cattle (49). Wildlife may therefore be important reservoirs of BEFV.

Protozoal and rickettsial diseases

The major tick vectors of important protozoal and rickettsial diseases in Africa related to wildlife are *A. hebraeum*, *A. variegatum*, *Rhipicephalus (Boophilus) decoloratus*, *R. appendiculatus*, *R. evertsi evertsi* and *R. zambeziensis* (Table I). Many tick species found on domestic animals are frequently more abundant on wildlife hosts and, in fact, some tick species depend on wildlife hosts to complete their life cycle (50). Where a wildlife species serves as a reservoir of infection to domestic animals, e.g. buffalo-associated

Theileria parva, which causes Corridor disease in cattle, wildlife may have a negative impact on the well-being of the cattle. On the other hand, where the endemic stability of a disease depends on a sufficiently large tick population to ensure that domestic animals become infected at an early age and become immune, the presence of wildlife hosts that augment tick numbers in an area may be beneficial.

Tsetse flies (*Glossina* species) evolved in sub-Saharan Africa together with their non-domestic hosts. Although tsetse flies can readily adapt to feeding on domestic animals, the distribution and abundance of some species, e.g. *G. morsitans* and *G. pallidipes*, are closely related to the number of wildlife species present (51).

Theileriosis

Buffalo-associated *Theileria parva* is the only parasite in whose life cycle wildlife, in this case African buffalo (*S. caffer*), play a significant role. Since the natural host of *T. annulata*, which occurs in North and north-eastern Africa, is the water buffalo (*B. bulbalis*), it is not included in this discussion. The known vectors of *T. parva* are *R. appendiculatus*, *R. zambeziensis* and *R. duttoni*. *Rhipicephalus appendiculatus* has a patchy distribution in eastern, central and south-eastern Africa (52). The preferred wildlife hosts of adult ticks are African buffalo; *Tragelaphus* spp. antelope, including eland (*T. oryx*); and waterbuck, while smaller antelope and hares are hosts of immature stages (52). *Rhipicephalus zambeziensis*, which occurs from Tanzania (north of Lake Malawi) to the northern parts of South Africa, generally prefers hotter, drier areas than *R. appendiculatus*; cattle, impala and greater kudu are the preferred hosts of all instars (52). *Rhipicephalus duttoni*, which transmits *T. parva* from African buffalo to cattle in Angola, is a relatively common tick on cattle; its wildlife hosts have not been well documented (52).

Table I
The main wildlife hosts of the most important ticks that transmit protozoal and rickettsial infections to livestock in sub-Saharan Africa (50)

Tick species	Host								
	African buffalo	Giraffe	Eland	Greater kudu	Bushbuck	Nyala (male)	Impala	Sable antelope	Plains zebra
<i>Amblyomma hebraeum</i>	*	*	*						
<i>A. variegatum</i>	*	*	*						
<i>Rhipicephalus appendiculatus</i>	*		*	*		*		*	
<i>R. (Boophilus) decoloratus</i>			*	*	*		*	*	
<i>R. evertsi evertsi</i>			*						*
<i>R. zambeziensis</i>				*			*		

Piroplasmosis

Babesia bigemina and *B. bovis*, the causative agents of bovine babesiosis in Africa, are strictly cattle-related; infections of wildlife species are incidental. *Rhipicephalus (B.) microplus*, the vector of *B. bovis*, was introduced into Africa and is cattle-associated (50). The indigenous *R. (B.) decoloratus*, on the other hand, is widely distributed in sub-Saharan Africa. Although it commonly feeds on cattle, it is also generally present in large numbers of plains zebras, impala, bushbuck (*T. scriptus*), greater kudu, eland and sable antelope, but African buffalo do not appear to be good hosts (50). *Theileria equi* and *B. caballi*, the causative organisms of equine piroplasmosis, also occur in indigenous African equids, e.g. plains zebras and mountain zebras (*Equus zebra*). The two vectors involved occur widely in sub-Saharan Africa: *R. e. evertsi* feeds on large animals such as cattle, horses, zebras and eland, while *Hyalomma truncatum* also feeds on large domestic and wild herbivores; giraffes can be particularly heavily infested (52, 53, 54).

Heartwater

Ehrlichia ruminantium, the organism causing heartwater (also known as cowdriosis), is transmitted exclusively by *Amblyomma* spp. ticks, the main vectors being *A. hebraeum* in southern Africa and *A. variegatum* elsewhere in sub-Saharan Africa. *Amblyomma* spp. prefer savannah and are absent in pure grassland or dwarf shrub vegetation (50). *Amblyomma hebraeum* larvae and nymphs feed on a wide variety of large and small mammals, birds and tortoises. They are very rarely found on murid rodents, even in habitats where large numbers of adults are present (55, 56). The preferred hosts of adult *A. hebraeum*, on the other hand, are larger domestic and wild ungulates (57). The host ranges of the various life stages of *A. variegatum* are similar to those of *A. hebraeum* (58). Heartwater can occur in large and small domestic ruminants wherever the vectors occur, even in the absence of larger wild ungulates. The role of the hosts of the tick larvae and nymphs in the transmission cycle of *E. ruminantium* has not been satisfactorily elucidated (59).

Bovine anaplasmosis

Various tick and haematophagous fly species are vectors of *Anaplasma* spp. Based on the fact that the geographic distribution of *R. (B.) decoloratus* in southern Africa is virtually the same as the area where bovine anaplasmosis is endemic, this tick is regarded as the main vector of *Anaplasma* spp. (60). Various other tick species can also transmit the infection (61). It has long been known that *Anaplasma* spp. can infect certain wild ungulate species, but there is mounting evidence that a wide host range may be involved, e.g. the giraffe, African buffalo, eland, greater kudu, nyala (*T. angasii*), waterbuck, etc. (62, 63, 64, 65, 66, 67). *Anaplasma* infections are readily maintained in cattle

populations. The importance, if any, of wild ungulates as a reservoir for the transmission of *Anaplasma* spp. to cattle is unknown.

Trypanosomosis

Shortly after discovering that *nagana* (African animal trypanosomosis) is caused by *Trypanosoma* spp. and that tsetse flies (*Glossina* spp.) are the vectors, Bruce (1897) confirmed the carrier status of African buffalo, blue wildebeest, greater kudu, bushbuck and spotted hyenas (*Crocuta crocuta*) by the sub-inoculation of blood into susceptible dogs (68). Wildlife serves not only as a reservoir of infection, but also as an excellent host for engorging tsetse flies. The distribution and abundance of some tsetse species, e.g. *G. morsitans* and *G. pallidipes*, are closely related to wildlife hosts; the former species disappearing from South Africa when wildlife hosts were decimated by hunting and the 1896 rinderpest pandemic (51). A continent-wide study of tsetse blood meals, comprising nearly 30,000 wild-caught flies, confirmed the important role of wildlife as hosts for tsetse flies (69). *Glossina morsitans* fed mainly on suids, particularly warthogs, although the hippopotamus (*Hippopotamus amphibius*) and ruminants were important hosts in certain areas; *G. austeni*, *G. fuscipennis* and *G. longipennis* also preferred suids, mainly bushpigs; *G. brevipalpis* preferred feeding on hippopotami; *G. pallidipes* fed mainly on ruminants (buffalo, bushbuck and cattle), but also on warthogs; and bushbuck seemed to be the preferred hosts of *G. longipalpis* and *G. fusca*. In the Luangwa Valley, Zambia, infection with trypanosomes was significantly more likely to occur in greater kudu and bushbuck than in other ruminants. Bushbuck were important reservoirs of *T. brucei*, while Bovidae in general appeared to be the most important reservoirs of *T. congolense* (70). Tsetse flies are widely distributed in sub-Saharan Africa, between 14°N and 29°S (51). The occurrence of trypanosomosis is determined largely by tsetse-fly density, which is in turn influenced by the availability of suitable tsetse-fly habitat (71). Where encroachment by people and their livestock leads to the fragmentation of suitable habitat, the abundance of tsetse flies decreases (71).

Distribution géographique des maladies infectieuses à transmission vectorielle et de leurs vecteurs : le rôle de la faune sauvage africaine

M. van Vuuren & B.L. Penzhorn

Résumé

Le rôle joué par la faune sauvage africaine dans les maladies infectieuses à transmission vectorielle qui affectent le bétail domestique suscite un regain d'intérêt, face à l'intensification de l'émergence et de la réémergence de ces maladies à l'échelle mondiale. En Afrique, les stratégies en faveur de la biodiversité et du développement de l'élevage se sont traduites par un risque accru de transmission de ces maladies entre les espèces sauvages et domestiques. Les auteurs mettent l'accent sur les agents pathogènes autochtones africains ayant un potentiel transfrontalier, par exemple les virus de la fièvre de la vallée du Rift, de la peste équine, de la fièvre catarrhale ovine, de la dermatose nodulaire contagieuse et de la peste porcine africaine, ainsi que les parasites transmissibles par voie sanguine.

Rien n'indique que les infections virales à transmission vectorielle soient installées de manière persistante dans la faune sauvage africaine. Celle-ci peut faire office de réservoir pour certains virus pendant la période de circulation virale inter-épidémique, avec des signes cliniques modérés ou absents. Les animaux sauvages déplacés dans de nouvelles régions peuvent également y introduire ou véhiculer les agents pathogènes qu'ils hébergent, comme ce fut le cas pour les virus de la dermatose nodulaire contagieuse et de la fièvre de la vallée du Rift et pour le virus West Nile. Les animaux sauvages peuvent également jouer un rôle d'hôtes amplificateurs lorsqu'ils sont exposés à des virus au début de la saison chaude, la plus active pour les vecteurs, avec un passage ultérieur aux espèces domestiques en tant qu'hôtes incidents plus tard dans la saison, cas de la fièvre catarrhale ovine et de la peste équine.

Certaines espèces de tiques que l'on trouve chez les animaux domestiques sont bien plus abondantes chez leurs hôtes sauvages ; certaines dépendent d'ailleurs de ceux-ci pour achever leur cycle évolutif. La stabilité endémique d'une maladie dépendant de l'existence d'une population de tiques suffisamment abondante pour installer l'infection chez les animaux d'élevage avant l'âge adulte, la présence d'espèces hôtes sauvages joue un rôle important en favorisant l'abondance des tiques. De nombreuses espèces d'ongulés sauvages servent de réservoirs à *Anaplasma* spp. ; en revanche, le rôle de la faune sauvage dans l'épidémiologie de la cowdriose (infection par *Ehrlichia ruminantium*) n'est pas élucidé. En règle générale les ongulés sauvages ne servent pas de réservoirs pour les piroplasmes affectant le bétail domestique, mais il y a deux exceptions : *Babesia caballi* et *Theileria equi* chez les zèbres et *Theileria parva* chez les buffles. *Theileria parva* chez les buffles est responsable de la maladie du Corridor chez les bovins domestiques, affection qui semble s'autolimiter à ceux-ci, du moins en Afrique australe. Les animaux sauvages sont d'importants réservoirs de l'infection par *Trypanosoma* spp. transmise par les glossines. On observe une étroite corrélation entre la distribution et l'abondance de certaines espèces de glossines, par exemple *Glossina morsitans* et *G. pallidipes*, et l'aire de répartition de leurs hôtes préférentiels parmi les espèces sauvages.

Mots-clés

Afrique – Anaplasmose – Cowdriose – Dermatose nodulaire contagieuse – *Ehrlichia ruminantium* – Faune sauvage – Fièvre catarrhale ovine – Fièvre de la vallée du Rift – Fièvre éphémère bovine – Maladie infectieuse à transmission vectorielle – *Nagana* – Parasitose du buffle à *Theileria parva* – Peste équine – Peste porcine africaine – Stabilité endémique – *Theileria parva* – Trypanosomose – Vecteur – Virémie – Virus West Nile.



Distribución geográfica de las infecciones de transmisión vectorial y sus vectores: función de la fauna salvaje africana

M. van Vuuren & B.L. Penzhorn

Resumen

La presencia en todo el mundo y a un ritmo creciente de infecciones emergentes y reemergentes ha suscitado un renovado interés por la intervención de la fauna salvaje africana en la aparición de infecciones de transmisión vectorial en los animales domésticos. En África, la conservación de la diversidad biológica y la expansión de la producción ganadera han acrecentado el riesgo de transmisión vectorial de infecciones entre los animales salvajes y el ganado. Especial atención han merecido, a este respecto, patógenos africanos autóctonos potencialmente transfronterizos como los virus de la fiebre del Valle del Rift, la peste equina, la lengua azul, la dermatosis nodular contagiosa y la peste porcina africana o los parásitos transmitidos por vía sanguínea.

No hay pruebas de la presencia persistente en la fauna salvaje africana de infecciones virales transmitidas por vectores. En el caso de ciertas infecciones víricas la fauna salvaje puede ejercer de reservorio durante las fases de circulación interepidémica de virus que dan lugar a infecciones asintomáticas o clínicamente leves. Los animales salvajes también pueden actuar como anfitriones que introducen o transportan el patógeno al desplazarse a nuevas regiones, como ocurre con los virus de la dermatosis nodular contagiosa, de la fiebre del Valle del Rift y West Nile. Además, la fauna salvaje ejerce a veces de anfitrión amplificador, al verse expuesta a los virus al inicio de la estación cálida, cuando los vectores son activos, y diseminarlos después entre los animales domésticos al ir avanzando la estación, como es el caso de la lengua azul o la peste equina.

Ciertas especies de garrapatas presentes en la fauna doméstica son más abundantes en los anfitriones salvajes, y algunas de ellas dependen del anfitrión salvaje para completar su ciclo vital. Toda vez que la estabilidad endémica de una enfermedad depende de la existencia de una población de garrapatas lo bastante numerosa como para que los animales domésticos resulten infectados a una edad temprana, la presencia de anfitriones salvajes que acrecienten el número de garrapatas puede resultar favorable. Muchas especies de ungulados salvajes son reservorio de *Anaplasma* spp., mientras que la función de la fauna salvaje en la epidemiología de la cowdriosis (infección por *Ehrlichia ruminantium*) está aún por dilucidar. Normalmente los ungulados salvajes no son reservorios de piroplasmas que afecten al ganado, pero hay dos excepciones: *Babesia caballi* y *Theileria equi* en la cebra y *Theileria parva* en el búfalo. La infestación por *Theileria parva* en los búfalos causa en el ganado vacuno la enfermedad de Corridor, que parece ser una enfermedad que se autolimita al ganado, por lo menos en el África meridional. Los animales salvajes constituyen un importante reservorio de la infestación por *Trypanosoma* spp., transmitida por la mosca tsetse. La distribución y abundancia de algunas especies de tsetse, como *Glossina morsitans* y *G. pallidipes*, guardan estrecha relación con la presencia de sus anfitriones salvajes predilectos.

Palabras clave

África – Anaplasmosis – Cowdriosis – Dermatosis nodular contagiosa – *Ehrlichia ruminantium* – Estabilidad endémica – Fauna salvaje – Fiebre efímera bovina – Fiebre del Valle del Rift – Infección transmitida por vectores – Lengua azul – *Nagana* – Peste equina – Peste porcina africana – *Theileria parva* – *Theileria parva* asociado al búfalo – Tripanosomosis – Vector – Viremia – Virus West Nile.



References

- Fafetine J., Neves L., Thompson P.N., Paweska J.T., Rutten V.P.M.G. & Coetzer J.A.W. (2013). – Serological evidence of Rift Valley fever virus circulation in sheep and goats in Zambezia Province, Mozambique. *PLoS Negl. Trop. Dis.*, **7** (2), e2065. doi:10.1371/journal.pntd.0002065.
- Gerdes G.H. (2004). – Rift Valley fever. In *Emerging zoonoses and pathogens of public health concern* (L.J. King, ed.). *Rev. Sci. Tech. Off. Int. Epiz.*, **23** (2), 613–623.
- Swanepoel R. & Coetzer J.A.W. (2004). – Rift Valley fever. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1037–1070.
- Pepin M., Bouloy M., Bird B., Kemp A. & Paweska J. (2010). – Rift Valley fever virus (Bunyaviridae: Phlebovirus): an update on pathogenesis, molecular epidemiology, vectors, diagnostics and prevention. *Vet. Res.*, **41**, 61.
- 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. *Epidemiol. Infect.*, **121**, 441–449.
- Evans A., Gakuya E., Paweska J.T., Rostal M., Akoolo L., Van Vuren P.J., Manyibe T., Macharia J.M., Ksiazek T.G., Feikin D.R., Breiman R.F. & Njenga M.K. (2008). – Prevalence of antibodies against Rift Valley fever virus in Kenyan wildlife. *Epidemiol. Infect.*, **136**, 1261–1269. doi:10.1017/S0950268807009806.
- Davies F.G. (2010). – The historical and recent impact of Rift Valley fever in Africa. *Am. J. Trop. Med. Hyg.*, **83**, 73–74.
- Davies F.G., Koros J. & Mbugua H. (1985). – Rift Valley fever in Kenya: the presence of antibody to the virus in camels (*Camelus dromedarius*). *J. Hyg. (Camb.)*, **4** (2), 241–244.
- Paweska J.T., Smith S.J., Wright I.M., Williams R., Cohen A.S., Van Dijk A.A., Grobbelaar A.A., Croft J.E., Swanepoel R. & Gerdes G.H. (2003). – Indirect enzyme-linked immunosorbent assay for the detection of antibody against Rift Valley fever virus in domestic and wild ruminant sera. *Onderstepoort J. Vet. Res.*, **70**, 49–64.
- Coetzer J.A.W. & Guthrie A.J. (2004). – African horse sickness. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1231–1246.
- MacLachlan N.J. & Guthrie A.J. (2010). – Re-emergence of bluetongue, African horse sickness, and other Orbivirus diseases. *Vet. Res.*, **41**, 35–48.
- Mellor P.S. & Hamblin C. (2004). – African horse sickness. *Vet. Res.*, **35**, 445–466.
- Mellor P.S. & Boorman J. (1995). – The transmission and geographical spread of African horse sickness and bluetongue viruses. *Ann. Trop. Med. Parasitol.*, **89** (1), 1–15.
- Lubroth J. (1988). – African horsesickness and the epizootic in Spain 1987. *Equine Pract.*, **10**, 26–33.
- Barnard B.J., Bengis R.G., Keet D.F. & Dekker E.H. (1995). – Epidemiology of African horsesickness: antibodies in free-living elephants (*Loxodonta africana*) and their response to experimental infection. *Onderstepoort J. Vet. Res.*, **62** (4), 271–275.
- Barnard B.J.H. (1998). – Epidemiology of African horse sickness and the role of the zebra in South Africa. *Arch. Virol.*, **14**, 13–19.
- Meiswinkel R. & Paweska J.T. (2003). – Evidence for a new field *Culicoides* vector of African horse sickness in South Africa. *Prev. Vet. Med.*, **60**, 243–253.
- Coetzee P., Stokstad M., Venter E.H., Myrmel M. & van Vuuren M. (2012). – Bluetongue: a historical and epidemiological perspective with the emphasis on South Africa. *Virol. J.*, **9**, 198. doi:10.1186/1743-422X-9-198.
- Gibbs E.P. & Greiner E.C. (1994). – The epidemiology of bluetongue. *Comp. Immunol. Microbiol. Infect. Dis.*, **17**, 207–220.
- Tollersrud T. (2009). – Bluetongue – Europe (06): Norway, first cases detected. Archive No. 20090221.0729. Available at: www.promedmail.org (accessed on 12 May 2014).
- Mellor P.S., Carpenter S., Harrup L., Baylis M. & Mertens P.P. (2008). – Bluetongue in Europe and the Mediterranean Basin: history of occurrence prior to 2006. *Prev. Vet. Med.*, **87**, 4–20.
- Erasmus B.J. & Potgieter C. (2009). – The history of bluetongue. In *Bluetongue* (P. Mellor, M. Baylis & P. Mertens, eds). Elsevier, London, 7–21.
- Neitz W.O. (1933). – The blesbuck (*Damaliscus albifrons*) as a carrier of heartwater and bluetongue. *J. S. Afr. Vet. Med. Assoc.*, **4**, 24–26.
- Hoff G.L. & Hoff D.M. (1976). – Bluetongue and epizootic haemorrhagic disease: a review of these diseases in non-domestic artiodactyles. *J. Zoo Anim. Med.*, **7**, 26–30.
- Batten C.A., Harif B., Henstock M.R., Ghizlane S., Edwards L., Loutfi C., Oura C.A. & El H.M. (2011). – Experimental infection of camels with bluetongue virus. *Res. Vet. Sci.*, **90**, 533–535.
- Verwoerd D.W. & Erasmus B.J. (2004). – Bluetongue. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1201–1220.

27. Coetzee P., van Vuuren M., Venter E.H. & Stokstad M. (2014). – A review of experimental infections with bluetongue virus in the mammalian host. *Virus Res.*, **182**, 21–34.
28. Petersen L.R. & Roehrig J.T. (2001). – West Nile virus: a re-emerging global pathogen. *Emerg. Infect. Dis.*, **7**, 611–614.
29. Venter M. & Swanepoel R. (2010). – West Nile virus lineage 2 as a cause of zoonotic neurological disease in humans and horses in southern Africa: a review. *Vector-borne Zoon. Dis.*, **10**, 659–664.
30. Guthrie A.J. (2003). – West Nile virus infection of thoroughbred horses in South Africa (2000–2001). *Equine Vet. J.*, **35**, 601–605.
31. Venter M., Human S., Zaayman D., Gerdes G.H., Williams J., Steyl J., Leman P.A., Paweska J.T., Setzkorn H., Rous G., Murray S., Parker R., Donnellan C. & Swanepoel R. (2009). – Lineage 2 West Nile virus as cause of fatal neurologic disease in horses, South Africa. *Emerg. Infect. Dis.*, **15** (6), 877–884. doi:10.3201/eid1506.081515.
32. Coetzer J.A.W. (2004). – Lumpy skin disease. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1268–1276.
33. World Organisation for Animal Health (OIE) (2013). – Sub regional workshop on lumpy skin disease and other vector borne diseases: final report, 28th February, Lanarca, Cyprus. Available at: www.rr-middleeast.oie.int/download/pdf/Report%20Larnaca%20_LSD_%20final%20JD%2018%20Apr.pdf (accessed on 1 December 2014).
34. Tuppurainen E.S.M., Stoltz W.H., Troskie M., Wallace D.B., Oura C., Mellor P.S., Coetzer J.A.W. & Venter E.H. (2011). – A potential role for hard (ixodid) tick vectors in the transmission of lumpy skin disease virus in cattle. *Transbound. Emerg. Dis.*, **58**, 93–104.
35. Tuppurainen E.S.M., Lubinga J.C., Stoltz W.H., Troskie M., Carpenter S.T., Coetzer J.A.W., Venter E.H. & Oura C.A.L. (2013). – Mechanical transmission of lumpy skin disease virus by *Rhipicephalus appendiculatus* male ticks. *Epidemiol. Infect.*, **141**, 425–430.
36. Lubinga J.C., Tuppurainen E.S.M., Coetzer J.A.W., Stoltz W.H. & Venter E.H. (2014). – Evidence of lumpy skin disease virus overwintering by transstadial persistence in *Amblyomma hebraeum* and transovarial persistence in *Rhipicephalus decoloratus* ticks. *Experim. Appl. Acarol.*, **62**, 77–90.
37. Lubinga J.C., Tuppurainen E.S.M., Coetzer J.A.W., Stoltz W.H. & Venter E.H. (2014). – Transovarial passage and transmission of lumpy skin disease virus by *Amblyomma hebraeum*, *Rhipicephalus appendiculatus* and *Rhipicephalus decoloratus*. *Experim. Appl. Acarol.*, **62**, 67–75.
38. Hedger R.S. & Hamblin C. (1983). – Neutralising antibodies to lumpy skin disease virus in African wildlife. *Comp. Immunol. Microbiol. Infect. Dis.*, **6**, 209–213.
39. Hunter P. & Wallace D. (2001). – Lumpy skin disease in southern Africa: a review of the disease and aspects of control. *J. S. Afr. Vet. Assoc.*, **72**, 68–71.
40. Greth R., Gourreau J.M., Vassart M., Wyers M. & Lefèvre P.C. (1992). – Capripoxvirus disease in an Arabian oryx (*Oryx leucoryx*) from Saudi Arabia. *J. Wildl. Dis.*, **28**, 295–300.
41. Young E., Basson P.A. & Weiss K.E. (1970). – Experimental infection of game animals with lumpy skin disease virus (prototype strain Neethling). *Onderstepoort J. Vet. Res.*, **37**, 79–88.
42. Davies F.G. (1982). – Observations on the epidemiology of lumpy skin disease in Kenya. *J. Hyg. (Camb.)*, **88**, 5–102.
43. Babiuk S., Bowden T.R., Boyle D.B., Wallace D.B. & Kitching R.P. (2008). – Capripoxviruses: an emerging worldwide threat to sheep, goats and cattle. *Transbound. Emerg. Dis.*, **55**, 263–272.
44. Penrith M.-L. & Vosloo W. (2009). – Review of African swine fever: transmission, spread and control. *J. S. Afr. Vet. Assoc.*, **80**, 58–62.
45. Pini A. & Hurter L.R. (1975). – African swine fever: an epizootiological review with special reference to the South African situation. *J. S. Afr. Vet. Assoc.*, **46**, 227–232.
46. Penrith M.-L., Thomson G.R. & Bastos A.D.S. (2004). – African swine fever. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1088–1119.
47. St George T.D. (2004). – Bovine ephemeral fever. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1183–1193.
48. St George T.D. (1988). – Bovine ephemeral fever: a review. *Trop. Anim. Hlth Prod.*, **20**, 194–202.
49. Aziz-Boaron O., Brettschneider S., King R., Gelman B. & Klement E. (2015). – Seroprevalence of bovine ephemeral fever virus in domesticated and wildlife species during epidemic and inter-epidemic periods (2000–2009) in Israel. *Transbound. Emerg. Dis.*, **62**, 183–187. doi:10.1111/tbed.12104.
50. Norval R.A.I. & Horak I.G. (2004). – Vectors: ticks. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 1–42.
51. Phelps R.J. & Lovemore D.F. (2004). – Vectors: tsetse flies. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 43–76.
52. Walker J.B., Keirans J.E. & Horak I.G. (2000). – The genus *Rhipicephalus* (Acari, Ixodidae): a guide to the brown ticks of the world. Cambridge University Press, Cambridge, 643 pp.

53. Apanaskevich D.A. & Horak I.G. (2008). – The genus *Hyalomma*. VI. Systematics of *H. (Euhyalomma) truncatum* and the closely related species, *H. (E.) albiparvum* and *H. (E.) nitidum* (Acari: Ixodidae). *Experim. Appl. Acarol.*, **44**, 115–136. doi:10.1007/s10493-008-9136-z.
54. Walker A.R., Bouattour A., Camicas J.-L., Estrada-Peña A., Horak I.G., Latif A.A., Pegram R.G. & Preston P.M. (2003). – Ticks of domestic animals in Africa: a guide to identification of species. *Bioscience Reports*, Edinburgh, 1–221.
55. Braack L.E.O., Horak I.G., Jordaan L.C., Segerman J. & Louw J.P. (1996). – The comparative host status of red veld rats (*Aethomys chrysophilus*) and bushveld gerbils (*Tatera leucogaster*) for epifaunal arthropods in the southern Kruger National Park, South Africa. *Onderstepoort J. Vet. Res.*, **63**, 149–158.
56. Howell D.J., Petney T.N. & Horak I.G. (1989). – The host status of the striped mouse, *Rhabdomys pumilio*, in relation to the tick vectors of heartwater in South Africa. *Onderstepoort J. Vet. Res.*, **56**, 289–291.
57. Horak I.G., MacIvor K.M., Petney T.N. & de Vos V. (1987). – Some avian and mammalian hosts of *Amblyomma hebraeum* and *A. marmoreum* (Acari: Ixodidae). *Onderstepoort J. Vet. Res.*, **54** (3), 397–403.
58. Petney T.N., Horak I.G. & Rechav Y. (1987). – The ecology of the African vectors of heartwater, with particular reference to *Amblyomma hebraeum* and *Amblyomma variegatum*. *Onderstepoort J. Vet. Res.*, **54**, 381–395.
59. Peter T.F., Burridge M.J. & Mahan S.M. (2002). – *Ehrlichia ruminantium* infection (heartwater) in wild animals. *Trends Parasitol.*, **18**, 214–218.
60. Bigalke R.D., de Vos A.J. & Barrowman P.R. (1976). – The control of some tick-borne diseases in South Africa. *Bull. Off. Int. Epiz.*, **86**, 89–100.
61. Potgieter F.T. & Stoltz W.H. (2004). – Bovine anaplasmosis. In *Infectious diseases of livestock*, 2nd Ed. (J.A.W. Coetzer & R.C. Tustin, eds). Oxford University Press, Cape Town, 594–616.
62. Augustyn N.J. & Bigalke R.D. (1974). – *Anaplasma* infection in a giraffe. *J. S. Afr. Vet. Assoc.*, **45**, 229.
63. Berggoetz M., Schmid M., Ston D., Wyss V., Chevillon C., Pretorius A.-M. & Gern L. (2014). – Tick-borne pathogens in the blood of wild and domestic ungulates in South Africa: interplay of game and livestock. *Ticks Tick-borne Dis.*, **5**, 166–175.
64. Kuttler K.L. (1984). – *Anaplasma* infections in wild and domestic ruminants: a review. *J. Wildl. Dis.*, **20**, 12–20.
65. Ngeranwa J.J.N., Shompole S.P., Venter E.H., Wambugu A., Crafford J.E. & Penzhorn B.L. (2008). – Detection of *Anaplasma* antibodies in wildlife and domestic species in wildlife-livestock interface areas of Kenya by major surface protein 5 competitive inhibition enzyme-linked immunosorbent assay. *Onderstepoort J. Vet. Res.*, **75** (3), 199–205. doi:10.4102/ojvr.v75i3.95.
66. Ngeranwa J.J.N., 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. *Vet. Parasitol.*, **74** (2–4), 109–122. doi:10.1016/S0304-4017(97)00161-1.
67. Pfitzer S., Oosthuizen M.C., Bosman A.-M., Vorster I. & Penzhorn B.L. (2011). – Tick-borne blood parasites in nyala (*Tragelaphus angasii*) from KwaZulu-Natal, South Africa. *Vet. Parasitol.*, **176**, 126–131. doi:10.1016/j.vetpar.2010.11.006.
68. Bruce D. (1897). – Further report on the tsetse fly disease or *nagana*, in Zululand. Harrison & Sons, London, 67 pp.
69. Clausen P.-H., Adeyemi I., Bauer B., Breloer M., Slachow F. & Staak C. (1998). – Host preference of tsetse (Diptera: Glossinidae) based on bloodmeal identification. *Med. Vet. Entomol.*, **12**, 169–180.
70. Anderson N.E., Mubanga J., Fevre E.M., Picozzi K., Eisler M.C., Thomas R. & Welburn S.C. (2011). – Characterisation of the wildlife reservoir community for human and animal trypanosomiasis in the Luangwa Valley, Zambia. *PLoS Negl. Trop. Dis.*, **5** (6), e1211. doi:10.1371/journal.pntd.0001211.
71. Ducheyne H., Mweempwa C., De Pus C., Vernieuwe H., De Deken R., Hendrickx G. & Van den Bossche P. (2009). – The impact of habitat fragmentation on tsetse abundance on the plateau of eastern Zambia. *Prev. Vet. Med.*, **91**, 11–18. doi:10.1016/j.prevetmed.2009.05.09.

