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Rabies in kudu (*Tragelaphus strepsiceros*)

Tollwut bei Kudu-Antilopen (Tragelaphus strepsiceros)

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Summary

Cycles of terrestrial rabies are associated with carnivores. In non-carnivorous species, rabies typically occurs as a spill-over from the carnivore reservoir and quickly encounters a dead end in such species. One major exception to this scenario has been an ongoing epizootic of rabies in the Greater Kudu, an African antelope. These herbivores are found in high densities in southern Africa, but rabies cycles have only been described from Namibia, a vast country located in the South Western region of Africa. Epizootics were first noted in the late 1970's and losses of up to 50 000 animals were estimated by 1985. Between 2002 and 2011, Namibian conservancies again estimated kudu losses ranging from 30–70%, resulting in very significant economic losses to the farming and gaming industries of the country. The sheer magnitude of the epizootic, phylogenetic data and experimental evidence of the particular susceptibility of kudu to rabies infection via mucous membranes are factors in support of a hypothesis that suggests horizontal transmission and maintenance of a rabies cycle within this species. It has become critical to investigate pathways for effective rabies control in Namibia – including the development of a strategy to halt and reverse the devastating epizootic of kudu rabies.

Keywords: rabies, kudu, epidemiology, epizootic, jackal, Namibia

Zusammenfassung

Die terrestrische Tollwut ist eng mit Karnivoren als Reservoirtiere verbunden. Bei anderen nicht-karnivoren Tierarten stellt die Tollwut typischerweise eine Spillover-Infektion aus dem Fleischfresserreservoir dar, wobei diese Tierarten keinen eigenen Infektionszyklus aufbauen können. Eine große Ausnahme scheint dagegen eine anhaltende Tollwutepidemie beim Großen Kudu, einer afrikanische Antilopenart, zu sein. Obwohl diese Pflanzenfresser in hohen Populationsdichten im südlichen Afrika leben, sind beständige Tollwutinfektionszyklen bislang nur bei Kudus in Namibia im Südwesten Afrikas bekannt. Tollwutepidemien wurden das erste Mal in den späten 1970er Jahren beschrieben, denen bis 1985 schätzungsweise 50 000 Tiere zum Opfer fielen. Zwischen 2002 und 2011 wurden die Verluste bei Kudus durch Namibische Schutzorganisationen auf 30–70 % beziffert mit erheblichen wirtschaftlichen Schäden für die Landwirtschaft und den Jagdtourismus. Das schiere Ausmaß der Epidemie, phylogenetische Daten sowie experimentelle Anhaltspunkte, die eine besonders hohe Empfänglichkeit von Kudus gegenüber Tollwutinfektionen über Schleimhautkontakte belegen, unterstützen die Hypothese einer möglichen horizontalen Übertragung des Tollwutvirus und Aufrechterhaltung des Infektionszyklus in dieser Antilopenart. Daher ist die Suche nach effektiven Wegen der Tollwutbekämpfung in Namibia einschließlich der Entwicklung von Strategien zur Beendigung der verheerenden Epidemien in Kudus eine vordringliche zukünftige Aufgabe.

Schlüsselwörter: Tollwut, Kudu, Epidemiologie, Epidemie, Schakal, Namibia

Introduction

Terrestrial rabies is mostly associated with carnivores and well known reservoirs are to be found in wildlife such as foxes, jackals, skunks, raccoons and mongooses (Badrane and Tordo., 2001; Nel et al., 1997). In the developing world, where more than 90% of human rabies cases occur, domestic dog populations are the most important rabies reservoirs (Knobel et al. 2005). Carnivores are well suited as maintenance hosts, given that the rabies virus, present in the saliva of infected animals – particularly during the acute neurological stage of the disease, is predominantly transmitted through bite wounds. Spill-over infections into dead-end hosts can occur due to transmission to non-carnivorous mammals, typically terminating further transmission. There have, however, been three unique circumstances where the infection of non-carnivorous animals has led to the maintenance of a rabies epidemiological cycle, as opposed to a dead-end infection. The first instance was in roe deer (*Capreolus capreolus*) in England in 1885 (Cope 1888). Typical rabies symptoms were observed, including extreme aggressiveness, paralysis and syncope (Tab. 1). Although typical rabies symptoms were noted, and the author diagnosed rabies clinically, Aujezsky's disease (pseudorabies), caused by a herpes virus, has not been ruled out. The symptoms of pseudorabies can be similar to those of rabies, with excess salivation, paresis and paralysis as well as rubbing of wounds (a common name for pseudorabies is "mad itch") being prevalent. However, the incubation period for infection of pseudorabies is commonly much shorter than that seen in rabies. Incubation periods for pseudorabies in rabbits is approximately 2–3 days with death occurring 24 hours after the onset of symptoms (Traub 1933). In an experiment performed by Adami (1889), death in rabbits inoculated with brain material from infected deer occurred 16–17 days after inoculation. Therefore, although pseudorabies cannot be ruled out, the cumulative evidence suggests that rabies was present. Adami (1889) performed an experimental trial where a healthy deer was placed into a room with another infected deer. The animals were observed for possible modes of transmission. It was noted that transmission occurred through infected saliva, and the mode of transmission was thought to be through biting. This was determined by the extremely aggressive behaviour of the animals as well as several attempts by the deer to bite any per-

FIGURE 1: *A Greater Kudu (Tragelaphus strepsiceros) cow.*

son within reach. The time taken for the healthy deer to become infected and show clinical symptoms was 19 days. The second unique occurrence of the maintenance of rabies among a non-carnivorous host species occurred in 1888 at Icksworth Park, Richmond, England, where an estimated 450 of a herd of 600–700 fallow deer (*Dama dama*) died (Adami 1889). These fallow deer showed similar symptoms to those noted by Cope (1888) in the roe deer in 1885.

The only other documented instance of the maintenance of rabies in an herbivorous host comes from Africa – almost 100 years after the above events. This epizootic, in the Greater Kudu (*Tragelaphus strepsiceros*), started in the 1970's in Namibia (Barnard and Hassel, 1981). Rabies cycles in Namibian kudu has ebbed and flowed over the subsequent decades and is still ongoing.
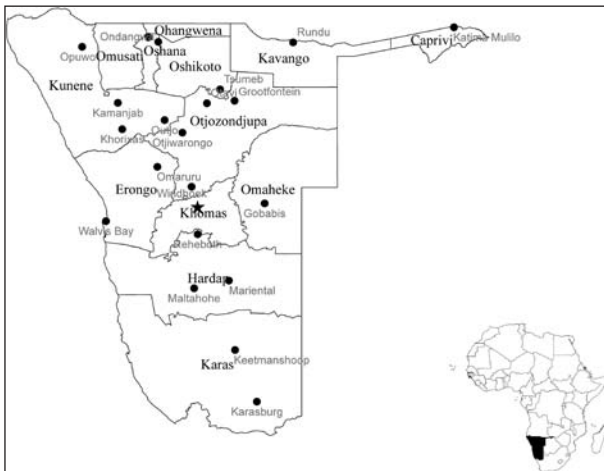
About kudu

The Greater Kudu (*Tragelaphus strepsiceros*) belong to the Order *Artiodactyla* and the *Bovidae* family. The Latin name is derived from the Greek words tragos meaning he-goat, elaphos meaning deer. Thus *Tragelaphus* is the word for antelope. *Strephis* means twisting and keras is the horn of an animal. Thus, the Greater Kudu is the "twisting horn antelope". They have been classified as a species of least concern on the IUCN Red List (IUCN, 2011). Population numbers are estimated to be around 482 000 with the majority of animals being conserved on private land (61%), and 15% in conserved areas, predominantly in Namibia and South Africa (IUCN, 2011).

TABLE 1: *Comparison of symptoms of rabies in kudu with suspected rabies in fallow and roe deer*

Symptoms	
kudu	**fallow/roe deer**
leaving social group	leaving social group
excess salivation	paresis-paralysis
docility	aggressiveness
visiting buildings	rubbing wounds until raw
paresis-paralysis	rapid behavioural changes
bellowing	syncope (unaware of surroundings/feinting)
aggressiveness	charging randomly
tail twitching	biting oneself
pain and depression	biting inanimate objects

The symptoms of the roe deer (*Capreolus capreolus*) have been taken from the 1888 outbreak and the fallow deer (*Dama dama*) from the 1889 outbreak in England. The symptoms in kudu (*Tragelaphus strepsiceros*) have been taken from the 1977 epizootic. The symptoms have been listed from most frequently observed (top) to least frequently observed (bottom).

FIGURE 2: *Map of Namibia.*

Greater Kudu occur throughout Southern and Eastern Africa, however, populations in the northern areas of their range are diminishing and kudu are thought to be extinct from Somalia. Their range extends from South Africa to Angola, and up the eastern part of Africa to Ethiopia, Chad, Sudan and Eritrea (IUCN 2011).

Morphology

The Greater Kudu are the second tallest antelope, after the Eland. Shoulder heights are approximately 1.4 m in males and 1.25 m in females (Mills and Hes, 1997). All males have spiralled horns that can reach lengths of up to 1.8 m. Females generally do not develop horns, although some have been known to. Masses of kudu can vary greatly, depending on their home-range vegetation. For instance, males in Eastern Cape South Africa have been shown to weigh as little as 174 kg whereas males in Namibia have been known to reach masses of 344 kg (Skinner and Chimumba, 2005). Females are smaller than the males and weigh between 112–210 kg. The kudu appears greyish brown, with the males becoming predominantly greyer as they age. Females are typically a more cinnamon brown. Both sexes have distinct humps at the shoulders, distinct white facial markings and large cupped ears (Fig. 1) (Skinner and Chimumba, 2005).

Ecology

Kudu are predominantly gregarious with herds ranging from 6–8 individuals on average. Herds rarely exceed 14 individuals and the larger herds tend to be those of cows. All-male herds do exist and can consist of up to four individuals. Peaks in herd sizes usually occur just before calving and also during the mating season. Home ranges of kudu can vary from 1.6 km² to 21 km² and these home ranges frequently overlap. Kudu bulls are usually not aggressive and a basic hierarchy is formed according to the age of bulls. Females will separate from the herd to give birth in

densely covered bush after a gestation period of 270 days (Skinner and Chimumba, 2005).

Kudu are a savannah-woodland species and prefer dense thickets of succulent trees and bushes. They do not occur in open grassland, desert or forested areas. They are typically classed as concentrate selectors and are predominantly browsers. They are known to browse on a wide variety of bush, typically those avoided by other species such as thorny acacia species and aloe species. Kudu tend to forage during early morning and late afternoon, as well as at midnight. They are also known to browse at midday, however, high temperatures during summer can reduce midday foraging (Skinner and Chimumba, 2005). Kudu forage in their social groups and while foraging, several kudu may individually browse from the same bush over a short period of time, or alternatively, several kudu may simultaneously browse together from the same bush.

Value

The Namibian economy relies heavily on kudu through the means of trophy hunting, game meat hunting and eco-tourism. Kudu have the third largest asset value of all game farmed animals in Namibia. In 2004, the estimated value of kudu was 31.13 million US dollars, which was greater than the total value of sheep, goats and donkeys combined (Barnes et al. 2004). The game farming industry is also vital for the conservation of the Greater Kudu as these animals are being bred specifically for this purpose. Especially vital is the fact that there is evidence of Namibian kudu being slightly divergent from other kudu, thus adding an important diversity to the current kudu population (IUCN 2011). The conservation of kudu in the southern regions of their distribution range is
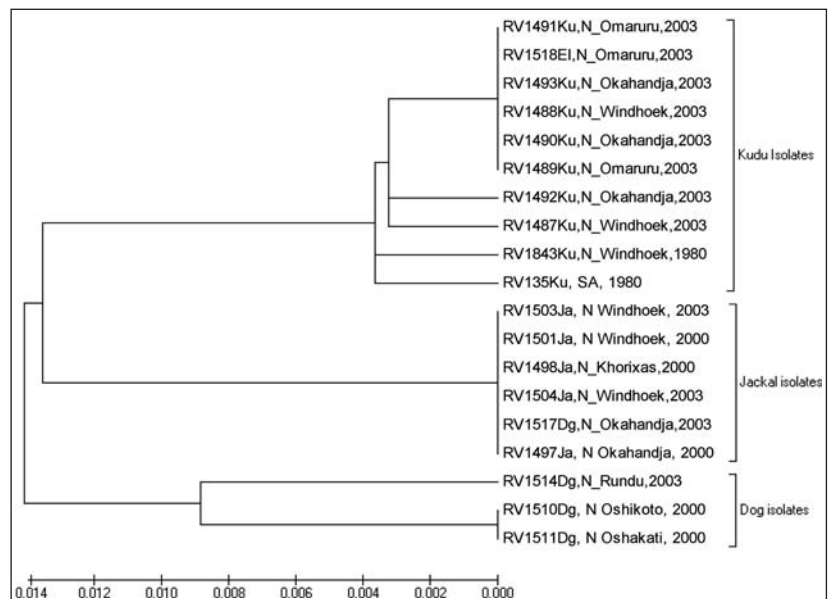
FIGURE 3: *Neighbour-Joining phylogenetic tree with 1000 bootstrap replicates of 400 bp region of nucleoprotein gene showing relationship of kudu isolates to those of canine isolates from Namibia. Branch length represents percentage difference between sequences. All sequences are previously published and were obtained from GenBank. Ku = kudu; Ja = Jackal; El = Eland; Dg = Domestic dog; N = Namibia; SA = South Africa.*

essential as populations in the northern-most ranges are threatened or suspected to have been eradicated.

Rabies in kudu

Historical evidence and current status

The first documented instance of rabies in kudu was in 1975 near Windhoek, Namibia (Barnard and Hassel, 1981) and up to 1976 three kudu were confirmed to have been infected by rabies (Barnard 1979). However, a major epizootic began in 1977, originating in the area of the Okahandja district on a farm near the Swakop River (Fig. 2). During the next two years, 161 animal rabies cases were confirmed in Namibia, of which 93 were kudu originating from the Okahandja district and surrounding areas (Barnard and Hassel, 1981). The epidemiological trend showed a progressive spread of the epizootic in a radial pattern, excepting the eastern side of the Okahandja district where a foot-and-mouth disease game fence prevented the spread in that direction. However, in November 1979 the epizootic crossed the game fence barrier and spread in an easterly direction as well. The epizootic spread at a rate of 40–60 km per year. By 1981 the epizootic peaked and an estimated 10 000 kudu had died of rabies (Barnard and Hassel, 1981). A second peak occurred in 1982 and thereafter, a steady decline in the number of cases was observed (Hübschle, 1988). By 1985 an estimated 30 000 to 50 000 kudu had died, which amounted to approximately 20% of the total kudu population in Namibia (Barnard and Hassel, 1981; Hassel, 1982; Shaw, 1980). Estimates of deaths in kudu were provided by kudu population census counts performed yearly by farmers. Once rabies had been laboratory confirmed on a farm, further deaths were assumed to be due to rabies, provided similar symptoms were observed. No other disease except rabies has ever been diagnosed or associated with kudu die-offs over the past four decades. In 1986 only two cases of rabies in kudu were seen (Hübschle, 1988). As noted by Hübschle (1988) the peaks in the number of rabies cases in kudu and cattle coincided with the peaks in cases in jackals, however, the proportions between jackal and kudu cases are too great to be explained by spillover infections from jackals alone. After 1986, only sporadic cases of rabies in kudu were documented, in line with what could be expected from spillover infections from jackals or other carnivores. However, in 2002 another vast increase in kudu rabies cases was reported (Mansfield et al., 2006). This epizootic also originated near Windhoek and by 2003, an estimated 2500 kudu had died from rabies. In 2006 and 2007, more than 80 laboratory confirmed cases were reported, but significant under-reporting of kudu rabies is expected as kudu are solitary animals and farmers frequently do not report or submit samples any more. Due to the severity of the current epizootic, the MET (Ministry of Environment and Tourism) has reduced the quota of kudu for trophy hunting and game meat hunting during the annual open hunting season. To date, in 2011, farmers have reported losses due to rabies of 30–68% on their farms since the last census in 2009 or 2010 and the rabies epizootic is ongoing.

Literature has classically categorised the two epizootics as separate incidences due to peaks in numbers of kudu before the epizootic years, originating from two separate introductions into the kudu population from a canid host. However, if rabies has adapted to a new host, it is more likely that the two epizootics are linked to the same epidemiological cycle. If two separate epizootics occurred, then, through molecular epidemiological analysis, one would clearly be able to distinguish the two epizootics from one another, as separate introductions from a carnivorous host would cause the creation of two separate clades of viruses. However, if these epizootics were linked to one another, through continuous transmission of rabies among kudu, the viruses would form a single clade that is diverging from the canine epidemiological cycles. After major epizootics that affect such large numbers, it is expected that, due to the lower population densities, fewer cases will be observed and reported. Thus, this does not mean that the epizootic is over, but it rather suggests that the numbers of animals cannot maintain the same magnitude of epizootic as observed in previous years.

Transmission of rabies among kudu

Horizontal transmission among kudu is believed to occur due to several factors, including both environmental and behavioural. In the year's preceding the first epizootic in 1977, unusually heavy rainfall occurred. In 1973 the rainfall was above average, and in 1975 and 1976 the average rainfall had doubled. This led to an abundance of water as well as food sources due to bush encroachment of thorny bushes into grassland areas (Barnard and Hassel, 1981). This led to a population explosion of kudu in Namibia. Estimated kudu numbers rose from 80 000 animals in 1972 to about 200 000 in 1978. Average population growth rates are four percent whereas, during the epizootic in 1977, the growth rate was approximately 8–10% (Hübschle, 1988). This led to increased population densities of one kudu per 40 ha land. This figure is skewed as kudu were not diffusely distributed due to their social behaviour and their enclosure on game farms. Thus their effective density would have been far greater (Barnard and Hassel, 1981). Other factors leading to increased populations were their protection and breeding for trophy hunting and tourism, as well as a reduction in the number of predators due to farmers protecting their stock (Hassel 1982).

Several factors suggest that non-bite, horizontal transmission of rabies occurs between kudu. Firstly, due to the magnitude in estimated losses of kudu in the first epizootic in 1977, it is unlikely that each kudu was infected separately by a carnivore such as a jackal, dog, surricate, mongoose or wild cat (Hassel 1982). Not only is a healthy kudu an agile and nervy antelope that would easily outrun a jackal under normal circumstances, but absence of corresponding increases of rabies among other species and the restricted spread of rabies through game fences that would restrict passage of rabid kudu (but not of small carnivores) provide support (Barnard and Hassel, 1981). More recently, molecular data supported very close homogeneity among kudu rabies virus isolates. An epidemiological study performed by Mansfield et al. (2006) showed that the majority of kudu isolates, sequenced using partial nucleoprotein amplicons, clustered together, regardless of date isolated. Sequences from 1980 – during the first epizootic – and 2003 – the second epizootic – as well as from years between the two epizootics were included in

the phylogenetic analysis. The jackal isolates from 2003 grouped separately, albeit closely to the kudu isolates. This suggests divergence between the epidemiological cycles and separate maintenance of the cycle within kudu, without further introductions from jackals or other carnivores (Fig. 3). In support, more recent isolates of kudu rabies virus (2008/2009) were found to be genetically separable from jackal rabies virus isolates of the same temporal and spatial origins (unpublished data). Kudu are not aggressive, and very few kudu show signs of aggressiveness when infected with rabies. A study performed by Barnard and Hassel (1981) showed that only 5% of 80 suspected kudu showed any form of aggressiveness. The main symptoms observed were that of docility, excess salivation and visiting domestic households – extremely uncharacteristic behaviour for a wild animal (Tab. 1). It was also stated from interviews with farm owners that domestic dogs would not scare off an infected kudu – a naturally shy animal.

All kudu infected with rabies left their social group when symptoms became evident. This then raises the question as how transmission is possible between kudu. As described by Mansfield et al. (2006) there are two main hypotheses regarding non-bite transmission among kudu. The first is that of social browsing. Kudu live in small social groups of between four and six animals and will browse on thorny acacia trees which are capable of causing micro- and macroscopic lesions on the mouth (Hübschle, 1988). An infected kudu may deposit large amounts of virus infected saliva (due to excess salivation) on the branch of an acacia tree. Because kudu are social browsers, a second, healthy kudu may browse from the same branch and come in contact with the infected saliva. The lesions in the healthy kudu's mouth would then provide an entry point for the virus (Mansfield et al., 2006). The second theory is that of mutual grooming. According to Barnard and Hassel (1981), it is a common sight to see cows licking their calves. They also noted that all kudu lick themselves as well as each other (mutual grooming). Two kudu grooming one another would lick the head, neck and shoulders of the other kudu. A healthy kudu grooming an infected kudu may come in contact with the infected kudus saliva (from self-grooming) or an infected kudu grooming a healthy kudu may lick a lesion or wound on the healthy kudu, potentially infecting it through these lesions (Mansfield et al., 2006).

Considering the two aforementioned hypotheses for the transmission of rabies between kudu, an experimental infection was performed by Barnard et al. (1982). Saliva from an infected kudu was placed on the nasal and buccal mucous membranes of four healthy kudu. Two oxen were used as a comparative control. The membranes of the healthy kudu were inspected in order to confirm that no visible lesions were present. Two of the four inoculated kudu died from rabies whereas both of the inoculated oxen survived. These results suggest that kudu are particularly susceptible to rabies infection through mucous membranes and that both hypotheses regarding non-bite transmission are viable. However, it is still not certain as to whether transmission does occur via this route and further experimental trials will need to be performed in order to confirm natural horizontal transmission among kudu.

Control

Kudu is an important species in Namibia, given its role in game farming, trophy hunting and eco-tourism, and the eradication of rabies in kudu will be a major achievement within the rabies control programme in Namibia. While aspects of the transmission and epidemiology of kudu rabies needs to be better understood, solutions in terms of this devastating epizootic need to be found. An integral part of such solution may well rely on the development of a safe, stable vaccine that can be administered to kudu in a bait form. The development of a bait vaccine for kudu is a key factor, as parenteral vaccination for this wildlife species is not feasible. Not only is specialised equipment and staff required to manage such large animals, but kudu are shy animals that are extremely difficult to capture due to their speed, agility and ability to jump over large fences. The inaccessibility and vastness of the terrain in question adds to the enormity of the challenges that will be faced in efforts to vaccinate kudu against rabies. However, oral vaccination – presuming a stable and effective vaccine and attractive bait – can be targeted to waterholes, as kudu will regularly gather at these observable and somewhat controllable locations. Considering the complexity of rabies and the multitude of potential cycles and host species, it is also clear that rabies control in kudu can only be one component of a larger programme that should be directed at rabies control in all associated vector and reservoir species.

Conclusion

The extent of the epizootics, and the continuous number of cases after peaks, lends support to the hypothesis that rabies has crossed the species barrier through an introduction from jackals into kudu. The virus is likely to have adapted to kudu and a new rabies variant is thus infecting kudu. This trend has been evident in several other regions where new host species have been introduced through the adaptation of the virus. Examples include skunks and raccoons in the United States (Badrane and Tordo, 2001); bat-eared foxes (Sabeta et al., 2007) and jackals in Africa and mongooses in southern Africa (Nel et al., 1997). These introductions have posed several major challenges to rabies control and eradication in the countries involved. Thus it is vitally important to scientifically prove that rabies has crossed the species barrier into the kudu and to assess the impact that this has on kudu as well as other ruminant populations.

More effective epidemiological and serological information is also required in order to fully understand the extent of the problem in Namibia and the potential risk to kudu in neighbouring countries. Lack of active epidemiological surveillance and under-reporting of cases are factors which have hindered a comprehensive study and understanding of rabies in kudu. Only through a thorough understanding of the virus, including the confirmation of the means of transmission among kudu, and an extensive effort in rabies control – in both the canine and the kudu populations – can the burden of rabies be relieved.

Acknowledgement

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