*Import risk analysis:* Diseases of antelope: Risks of introducing live antelope into zoological gardens



30 May 2000

# Diseases of Antelope: Risks of introducing live antelope into zoological gardens

**Bob Worthington** Consultant Wellington

Note: This import risk analysis was conducted and documented by a private consultant working on behalf of would-be importers. It is not an official MAF risk analysis.

Nevertheless, this risk analysis has been subjected to MAF's internal scientific review process and to external expert review. The risk analyst has addressed all the points raised by MAF and the external reviewers.

MAF considers this risk analysis to be technically sound and sufficiently robust to base an Import Health Standard on.

# **Table of Contents**

INTR		TION4
1.0	<b>DISE</b> 1.1	ASE VECTORS CARRIED BY ANTELOPE6 Ticks6
2.0	VIRA	L INFECTIONS OF ANTELOPE14
2.0	2.1	Foot and mouth disease14
	2.2	Rinderpest 19
	2.3	Rift Valley Fever 23
	2.4	Bluetongue
	2.5	Epizootic haemorrhagic disease
	2.6	Rabies
	2.7	Lumpy skin disease
	2.8 2.9	Malignant catarrhal fever41Ephemeral fever46
	2.9	Nairobi sheep disease
	2.10	Crimean-Congo disease
	2.12	Bovine virus diarrhoea
	2.13	Miscellaneous virus diseases 59
3.0	PRO	OZOAL DISEASES OF ANTELOPE64
	3.1	Babesiosis
	3.2	Theileriosis
	3.3	Tsetse fly transmitted trypanosomosis
	3.4	Surra
	3.5 3.6	Besnoitiosis (elephant-skin disease)
4.0		ETTSIAL AND CHLAMYDIAL DISEASES OF ANTELOPE81
	4.1	Heartwater
	4.2 4.3	Anaplasmosis
	4.3	other Rickettsial and Chiamydial Infections
5.0	BAC	FERIAL INFECTIONS OF ANTELOPE92
	5.1	Brucellosis
	5.2	Tuberculosis
	5.3	Johne's disease (paratuberculosis)
		Anthrax 102 Other bacterial infections 106
6.0		ASITES OF ANTELOPE
0.0	FAN	ASITES OF ANTELOFE
7.0	TRAN	SMISSIBLE BOVINE SPONGIFORM ENCEPHALOPATHY. 117
8.0	ARTI	FICIAL INSEMINATION AND EMBRYO TRANSFER
TEC		0GY
	8.1 8.2	Artificial insemination 118 Embryo transfer 122

8.3	Feasibility of using artificial insemination or embryo transfer in antelope	124
RISK	OF INTRODUCTION OF DISEASE	. 125
9.1	Ticks	126
9.2	Infectious diseases	129
9.3	Parasitic diseases	136
Refe	rences	136
	<b>RISK</b> 9.1 9.2 9.3	RISK OF INTRODUCTION OF DISEASE

# **APPENDIX 1**

# **EXECUTIVE SUMMARY**

# DISEASES OF ANTELOPE: RISKS FROM INTRODUCING LIVE ANTELOPE INTO ZOOLOGICAL GARDENS

The diseases carried by antelope have been reviewed.

Forty-four diseases were identified that are carried by or potentially carried by antelope. Six of the diseases are diseases included in the OIE List A. The other diseases range from serious diseases to trivial or potential diseases. The risk of introducing any disease into New Zealand has been identified as extremely low, provided risk reduction procedures based on the following principles are implemented before and during the introduction:

- Selection of antelope from safe sources
- Appropriate quarantine
- Application of suitable diagnostic tests
- Treatment or vaccination where appropriate
- Use of artificial insemination and embryo transfer technology if possible and appropriate

The highest potential risk to animal health of New Zealand livestock is the introduction of ticks that are vectors of 11 of the diseases carried by antelope and many other diseases of other species of animals. Methods used to reduce the risk of introduction of ticks should be carefully planned and scrupulously implemented.

The viruses that cause the OIE List A diseases foot and mouth disease, rinderpest, Rift Valley fever, lumpy skin disease, bluetongue and vesicular stomatitis can infect antelope. It is concluded that the introduction of any of these diseases is extremely unlikely, as the methods used to safeguard against their importation should be highly

effective. In particular, sources of animals can probably be identified in countries in which all or most of the diseases do not occur.

The possibility of disease introduction is probably higher for the chronic diseases, those that have very long incubation periods and those in which long-term carrier states exist. The following 17 diseases that fall into these categories were identified: Johne's disease, tuberculosis, brucellosis, enzootic bovine leucosis, rabies, bovine virus diarrhoea, infectious bovine rhinotracheitis, malignant catarrhal fever, trypanosomosis, babesiosis, anaplasmosis, leptospirosis, toxoplasmosis, sarcocystosis, coccidiosis, cryptosporidiosis and besnoitiosis.

Only one of these diseases, wildebeest-associated malignant catarrhal fever (Alcelaphine herpesvirus-1) is considered to be a moderate risk for introduction. This disease would only be a risk if wildebeest or other members of the sub-family Alcelaphinae were introduced. The consequences of introducing this virus are high for zoos, but insignificant for the livestock industries of New Zealand. It is a recommendation that members of the family Alcelaphinae should not introduced. However, the representatives of the Zoos of New Zealand should make this decision as they represent the group at risk.

All the other chronic disease/long carrier state/long incubation period diseases were not considered to be of importance for one or more of the following reasons:

- There is only a low risk of introduction (all diseases in question)
- The methods used to exclude them would be highly effective for all diseases except Johne's disease. Johne's disease is already endemic in New Zealand and Ministry of Agriculture and Forestry (MAF) considers this disease to be a quality issue for animal owners
- The diseases themselves are unimportant and unlikely to be harmful to New Zealand livestock (toxoplasmosis, sarcosporidiosis, coccidiosis of antelope, besnoitiosis)
- The diseases are already so common in New Zealand livestock that introduction of the disease would not have a significant impact (bovine virus diarrhoea type 1, infectious bovine rhinotracheitis)
- The diseases would be unlikely to spread from the zoos where they were introduced (enzootic bovine leukosis, rabies, brucellosis,

trypanosomosis, babesiosis, anaplasmosis, toxoplasmosis, besnoitiosis, sarcosporidiosis).

Those diseases requiring arthropod vectors such as ticks, mosquitoes and midges are most unlikely to be able to establish in New Zealand, although there is a low risk that endemic mosquitoes could carry some diseases.

Animals in zoos are generally well isolated from farm livestock. For this reason introduction of animals into a zoo situation is less dangerous than the introduction of animals onto farms.

It is concluded that a well managed introduction of antelope species into New Zealand zoos would involve minimal risk of introducing exotic diseases.

# DISEASES OF ANTELOPE: RISKS FROM INTRODUCING LIVE ANTELOPE INTO ZOOLOGICAL GARDENS.

# Introduction

Antelope originate mainly from Africa, fewer species are indigenous to Asia and pronghorn antelope are indigenous to North America. Antelope are able to carry several of the serious diseases of those regions. Many of these diseases have been reviewed in detail in Volumes I and II of the book *Infectious Diseases of Livestock with special reference to Southern Africa*(1) and much of the information contained in this review is derived from that source. This book is generally the best source of comprehensive information as information on diseases in antelope is often fragmentary and some important aspects of the behaviour of the diseases are unknown. For this reason, extrapolation from knowledge of the diseases in cattle is sometimes necessary. This review contains some information on the diseases of giraffe as this was originally requested. It also gives some information on the diseases that occur in African buffalo because this information is often complementary to what is relevant about diseases in antelope and their relationship to diseases of domestic livestock. However, the diseases of African buffalo have not been extensively reviewed, as they were not regarded as antelope in the terms of reference for this review.

Extensive use was made of electronic searches of the literature between 1984 and 1999, but traditional manual searches were also used. However, this review does not claimed to be an exhaustive review and additional information could be found if unlimited time was available.

Antelope are referred to by their common names. A list of generic and common names of antelope is given in Appendix 1.

It was a requirement of the terms of reference for this document that recommendations should be made for reducing the risk of introducing each disease reviewed. General comments and discussion of relevant points are given under the heading "Risk reduction". Specific recommendations are given, where appropriate, under the heading "Recommendations" with the sub-headings "Source of animal", "Quarantine", "Treatment" and "Diagnostic tests". Quarantine may involve pre-entry quarantine in the country of origin and/or post entry quarantine in New Zealand. Quarantine periods in

this document refer to total quarantine periods. It is not appropriate for this reviewer to comment on where that quarantine should be undertaken. It is assumed that a standard quarantine for any animal brought into the country should be 4 weeks. Any recommendations for longer quarantine periods are discussed in the relevant parts of the text. The uses of artificial insemination or embryo transfer are discussed in a separate section. Unreferenced recommendations in this document are those of a consultant acting for the zoos wanting to import antelope. They do not necessarily reflect the views of the MAF and may not reflect the policies that will be implemented by MAF. However, in many cases recommendations with regard to importation of serologically positive animals have already been made by MAF(2) and these recommendations are now accepted as official policy(3). All references to this official policy have been referenced in the text.

#### References

- Infectious Diseases of Livestock with special reference to Southern Africa. Ed. Coetzer JAW., Thompson GR., Tustin RC. Oxford University Press, Cape Town, Oxford, New York. 1994.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.
- 3. MacDiarmid SC. Personal communication.

# 1.0 Disease vectors carried by antelope

Lice, mites and fleas are not vectors of serious diseases carried by antelope. Other arthropod vectors of serious diseases such as mosquitoes and midges (*Culicoides* spp.) are not carried on live animals. Only ticks are of concern, as these are actually carried on antelope.

# 1.1 Ticks

Information in Section 1.1 is basically a summary of two extensive review articles on ticks(1)(2). This section is designed to give background information on ticks and stress the importance of not introducing ticks into New Zealand. Each individual statement concerning ticks is therefore not referenced to a primary source, as this would make the section unnecessarily long and less useful to the reader.

Ticks carry a wide range of serious and less serious diseases and can also cause economic loss, due to loss of condition and tick toxicoses. Annual worldwide losses due to tick borne diseases and tick control are estimated to be several billion dollars(1). Ticks on animals can be easily overlooked in even the most careful inspections. They may be hidden by hair or attached to not easily accessible parts of the body, such as in ears, under the tail and in various skin-folds and crevices. Recently hatched larvae are only pinhead size and almost impossible to see in the hair of their hosts. It is for this reason, that ticks have been inadvertently introduced into New Zealand on a number of occasions, despite most careful inspections(3). Because of these reasons it is suggested in this document that the introduction of ticks on live animals poses the single most important threat of introducing foreign animal diseases to New Zealand if antelope are introduced.

Although ticks are primarily a problem in tropical countries, there are several species of ticks that carry diseases that occur in temperate climates.

Ticks belong to two families: the Ixodidae or hard ticks and the Argasidae or soft ticks. The Ixodidae represent the main cause of concern for herbivores because they are the vectors of several major diseases that occur in antelope. Argasidae are important vectors of poultry diseases and African swine fever.

# 1.1.1 Ixodidae

Ixodidae may be one, two or three-host ticks. The larvae of three host ticks leave the host and moult to become nymphs before seeking a new host. Nymphs similarly leave their host animals before moulting and finding a new host as an adult. In the case of one-host ticks the parasite remains on the same host through all three stages. Some diseases carried by ticks are transmitted through the egg to the next generation (transovarial transmission), while in others, the infectious agents are only transmitted transtadially or even intrastadially.

# 1.1.1.1 Genus: Boophilus

This genus contains five species of small, blue, one host ticks responsible for carrying several diseases, particularly the diseases caused by *Babesia* spp. in a wide variety of mammals, and anaplasmosis (*Anaplasma marginale*) in cattle. They parasitise a large variety of animals(1)(2).

*B. microplus* originated from South-East Asia, but is now present in Asia, South and East Africa, Australia and South and Central America. *B. decoloratus* has remained confined to Africa. *B. annulatus* is present in the Mediterranean region, Russia and Ukraine, the Near and Middle East, parts of West Africa and Sudan, Mexico and the Southern United States. Less important are the species *B. kohlsi* of the Near and Middle East Africa.

# 1.1.1.2 Genus: Dermacentor

This genus contains about 30 species most of which are three host ticks. They occur on all continents except Australia. The main species include *D. marginatus* and *D. reticulatus* in Eurasia and *D. variabilis* and *D. andersonii* in North America. This genus is not important in disease transmission in Africa. Members of the genus carry anaplasmosis in cattle and small ruminants, equine and canine babesiosis and Rocky Mountain spotted fever in humans.

# 1.1.1.3 Genus: *Haemaphysalis*

The genus contains more than 150 species, but most species are not adapted to domestic livestock. They are eyeless three host ticks. *H. longicornis* is an Asian tick

that has spread to Australia, New Zealand and New Caledonia. Both bisexual and parthogenetic strains of this tick occur. *H. bispinosa* occurs on cattle in India. *H. punctata* is common on ruminants in Europe and is the vector for *Babesia major* in cattle, *Babesia ovulata* in cattle in Japan, *Babesia motasi* in small ruminants and the non-pathogenic *Theileria orientalis*, which is also termed *T. buffeli* or *T.sergenti* in Asia. *H. longicornis* carries *Theileria orientalis* in New Zealand and Australia.

# 1.1.1.4 Genus: Hyalomma

There are about 30 species in the *Hyalomma* genus. They are medium to large ticks that are mostly three host ticks, but the genus includes some species that can have either two or three hosts. The large mouthparts of this species may cause necrotic lesions in the skin where the ticks have attached.

*H. anatolicum* and *H. detritum* transmit tropical theileriosis (*T. annulatum*) in tropical regions from China to Spain and Mauritania. Other vectors of this important disease are *H. dromedarii* in Mauritania and *H. marginatum* in Spain. Crimean Congo haemorrhagic fever is carried predominantly by *Hyalomma* species in Europe, Asia and Africa. In Africa *H. truncatum* (the bont-legged tick) causes sweating sickness, a disease caused by a toxin and is a vector for *Babesia caballi* (equine babesiosis).

# 1.1.1.5 Genus: *lxodes*

There are 250 species in this genus. The species of this genus are not of importance in the tropics, but *I. scapularis* (formerly *I. dammini*) is common in North America and *I. ricinus* and *I. persulcatus* are common in Europe and are vectors for *Babesia divergens* in cattle, *Ehrlichia phagocytophilia* (tick-borne fever) in ruminants, and louping ill in sheep. In humans *I. ricinus, I. persulcatus* and *I. scapularis* carry Lyme's disease and *I. ricinus* and *I. persulcatus* are vectors of tick-borne encephalitis. *I. rubicundus* and *I. holocyclus* cause tick paralysis in animals and humans in South Africa and Australia respectively and *I. rubicundus* has caused tick paralysis in a springbok(4).

# 1.1.1.6 Genus: Rhipicephalus

This genus contains about 70 species. They are medium to small, usually three host ticks that are generally confined to the African continent with a few species occurring in Eurasia. They parasitise a large number of animals particularly ruminants.

*R. appendiculatus* (the brown ear tick) and *R. zambeziensis* are the main vectors of East Coast fever (*Theileria parva*) and related theilerioses in Africa. *R. bursa* transmits ovine babesiosis and *R. sanguineus* transmits canine babesiosis and ehrlichiosis and tick-bite fever (*Rickettsia conori*) in humans. *R. eversti* (the red legged tick) transmits anaplasmosis and equine babesiosis as well as a paralytic toxicosis of calves and lambs.

# 1.1.1.7 Genus: Amblyomma

There are more than 100 species in the genus. Species from this genus generally have large mouthparts and coloured patterned scuta. They are three host ticks from tropical and sub-tropical zones. Mammalian, avian, reptilian and amphibian hosts are parasitised. *Amblyomma* spp. are typically ticks of sub-Saharan Africa and islands near Africa such as Reunion, Mauritius and Madagascar. Recently they have spread to some Caribbean Islands. *A. variegatum* and *A. hebraeum* are well adapted to livestock, but also parasitise a wide variety of other animals including antelope. They are the main vectors of heartwater (*Cowdria ruminantium*). Eleven other species of *Amblyomma*, including three American species (*A. maculatum, A. cajennense* and *A. dissimile*) are known to be able to carry heartwater. Further research may yet reveal other capable vectors

# 1.1.2 Argasidae

The members of this family are not important as disease vectors of ruminants. Argasidae are multi-host soft ticks that feed on their hosts for periods of a few minutes up to a few days. They live in nests or burrows in cracks of buildings, buried in shaded sandy places etc., where they mate and lay their eggs. They parasitise host animals repeatedly taking small blood meals during a livespan that can last for several years.

Argas persicus is a parasite of poultry and a vector for spirochaetosis (Borrelia anserina), Ornithodorus moubata and Ornithodorus erraticus are vectors for African

swine fever in pigs. *Ornithodorus savigni* live in shaded areas, which are resting places of livestock, in sandy hot desert areas and feed on the legs of resting livestock. As they only remain on their hosts for short periods and occur in hot arid regions they are unlikely to be introduced by antelope or to establish in New Zealand.

*Otobius megini* the spinous ear tick occurs in India and Africa. They stay in the ears of host animals for several days before leaving the host and mating and completing their life cycle off the host. Spinous ear ticks are not vectors of significant diseases, but are undesirable pests because they cause irritation, damage and stress to their hosts.

# 1.1.3 Antelope as hosts for ticks

Many species of ticks are found on antelope species. No attempt has been made in this review to provide a full host parasite list since this would be unmanageably extensive. Furthermore, if such a list were to be compiled, it would probably be incomplete as many of the host parasite relationships are probably yet to be described. However, it is known that a very large number of tick species are found on antelope and it should be assumed that antelope can act as hosts to most if not all of the ixodid tick vectors of serious animal diseases. Even if the antelope themselves were not carrying a disease, the ticks they carry could be ticks that transmit some diseases transovarially and could therefore introduce diseases such as babesiosis.

Antelope and other wild animals provide a source of ticks to domestic animals, and make the control of ticks by dipping of domestic animals difficult(5). This indicates that ticks are readily transferred from antelope to domestic animals.

Individual animals can carry large numbers of ticks leading to anaemia and loss of condition. A case has been described in which a 3-year-old eland antelope carried about 5,000 ticks(6).

Knowledge of tick species, their hosts and the diseases they carry is not complete, and all ticks should be regarded as potentially harmful and strenuous attempts should be made to prevent their importation.

# 1.1.4 Implications

Introduction of ticks could have serious direct implications as they could transmit diseases to livestock, humans, and zoo and other animals as well as causing discomfort and stress.

# 1.1.5 Risk reduction

Clearly a policy should be followed of trying to prevent the introduction of ticks. The importation policy for antelope be aimed at preventing the introduction of any ticks. It should include:

- Careful inspection of animals at intervals of not more than a week, to ensure that no ticks are present on the animals. The whole skin surface should be inspected visually and palpated, including poorly visible regions such as those around the base of the tail, the anus and perennial region, the interdigital space, areas around the hooves, inside the ears, the udder and all skin-folds.
- Quarantine should be for long enough for the ticks to feed and leave the host. One host ticks are likely to remain on the hosts for up to 3 weeks. Therefore, a quarantine period of 4 weeks is recommended to give any ticks time to complete their growth and leave the host.
- Some stages of three host ticks may only stay on the host for only about 3 days. Therefore, bedding should be removed and burned at least every 3 days so as to destroy any tick larvae or nymphs that have left before they moult and again parasitise quarantined animals.
- Animals should be housed in accommodation that can be easily cleaned and is free from cracks and inaccessible hiding places for insects.
   Walls should be smooth and painted and floors should preferably be concrete.
- Animals should be thoroughly soaked with a currently approved insecticide. The best method of applying insecticides is by immersion dipping. As dip tanks are not available in many quarantine stations,

thorough wetting by spraying will suffice. Pour on insecticides should not be used. The insides of the ears and the areas under the tail and the crevices between the legs and body should be hand dressed. Insecticides currently approved by MAF be applied at recommended concentrations. Dipping should be done shortly before the animals enter the quarantine station, one week after entering the quarantine station and again within one week of being released.

- It is recommended that a shallow trough filled with an insecticide solution should surround the floor of the animal housing. This serves to trap ticks or other parasites leaving the animals. Adequate easily available drinking water must be provided so that thirsty animals do not drink the insecticide. Insecticides of low toxicity such as synthetic pyrethroids are recommended for this purpose.
- An excellent standard of hygiene and management should be maintained throughout.
- When animals are maintained in pre-entry quarantine in countries that are infested with ticks, assurance must be obtained that hay, concentrate rations and bedding are tick-free. This can be assured by using autoclaved or methyl bromide fumigated feed and bedding.

Prevention of the introduction of ticks is probably the most important single measure that should be implemented to prevent introduction of disease when introducing antelope species.

# Sources of information

- 1. Jongejan E., Uilenberg G. 1994. Ticks and control methods. Revue Scientifique et Technique Office des Epizooties, 13(4), 1201-26.
- Norval RAJ. 1994. Vectors: Ticks. In: Infectious Diseases of Livestock with special reference to Southern Africa. Ed. Coetzer JAW., Thompson GR., Tustin RC. Vol 1, pp. 3-24. Oxford University Press, Cape Town, Oxford, New York.

- 3. Fairley R., Heath A. 1997. Exotic ticks intercepted in New Zealand since 1980. Surveillance , 24(1), 21-2.
- 4. Fourie LJ., Horak IG. 1987. Tick induced paralysis of Springbok. South African Journal of Wildlife Research, 17(4), 131-3.
- Colbourne JC., Floyd RB. 1987. The effect of game animals on tick control. ACIAR Proceedings Series, Australian Centre for International Agricultural Research, 17, 149-50.
- Hamel HD., Van Amelsfoort A, Van Amelsfoort A
   1985. Tick infestation and its treatment in an eland antelope (case report). Veterinary Medical Review, 2, 152-7.

# 2.0 Viral infections of antelope

# 2.1 Foot and mouth disease

Foot and mouth disease is potentially the most devastating disease that could be introduced into New Zealand. Many extensive volumes on the disease, its potential impact if introduced into New Zealand and contingency plans to contain and eradicate it if introduced, have been written by or for MAF. It is unnecessary to repeat this information, and only relevant information pertaining to the disease in antelope, is given. An extensive review of various aspects of the disease is given in reference 1.

**Etiological agent:** The etiological agent is the foot and mouth disease virus (Family: Picornaviridae, Genus: *Aphthovirus*). Types A, O, C, SAT1, SAT2, SAT3, and Asia occur and are immunologically distinct. Several sub-types are known for each serotype.

**Susceptible species:** The disease affects all cloven-hoofed animals and all antelope must be regarded as susceptible, although some species show minimal clinical manifestations of the disease. Other species are particularly susceptible and 2,000 mountain gazelle died from the disease in an outbreak in Israel(4). Remarkable pancreas lesions were seen in experimentally infected mountain gazelles(2)(3).

**Incubation period:** The incubation period in cattle is usually 2-8 days, sometimes up to 13 days(1). Information for individual antelope species was not found.

**Carrier state:** After recovery from the disease, the virus persists in the pharynx of cattle for up to 2 years and perhaps lifelong in the African buffalo(1). Most antelope species probably only carry the virus for short periods after recovery. For example, in experimental infections, virus persistence was transitory in sable antelope and did not occur in eland(5). Similar experiments have not been done for all species of antelope, but epidemiological evidence has not been produced to show that they are long term carriers of disease. Outbreaks of the disease in cattle are generally associated with the presence of buffalo or movement of domestic livestock (6).

**Transmission:** Transmission is by aerosol infection or direct contact. When winds are favourable virus-containing aerosols generated by infected animals, can be transmitted

over long distances. For example the virus was shown to cross the English Channel by this means(7). The disease can also be transmitted by ingestion of infected material carried on fomites. Virus can be excreted by infected animals in most body secretions and excretions including milk, semen, faeces, saliva etc.

**Signs of disease:** Signs of the disease include fever, malaise, lameness, salivation, vesicles and erosions in the mouth, feet and udder, and sudden death in young animals.

**Epidemiology:** In Africa, the African buffalo is the main maintenance host for the disease. Buffalo are resistant to infection and show few signs of disease, but after becoming infected may carry the infection in their pharynx for life(6)(8). However, once the viraemic period has passed, buffalo are not highly infectious and most attempts to transmit the disease by contact with carrier buffalo have failed. Evidence gathered from experimentally infected buffalo suggested that they transmitted the disease to cattle and impala only in the acute stages of infection and when there was direct physical contact between the species(9). However, it is believed that buffalo initiate the sporadic outbreaks of the disease that occur in endemic areas of Africa, and their exact role as carrier animals remains unclear. One suggestion is that epidemics of disease are initiated when susceptible buffalo calves become infected by close contact with their dams. The main species affected are impala, which are the most numerous susceptible animals in endemic areas and also live in herds, thus increasing the chance for close contact. Impala show typical signs of disease but they are generally inefficient transmitters of disease(10).

An outbreak of foot and mouth disease in the Assam State zoo remained confined to deer and clinical disease did not appear in antelope(11).

**Diagnosis:** The disease is diagnosed from the **c**linical signs of disease, virus isolation, reverse transcriptase polymerase chain reaction, antigen capture ELISA, and serology including ELISA and virus neutralisation(10).

**Implications:** The introduction of the disease into New Zealand would have devastating effects on the animal industries and on trade in animal products. Forbes showed that the disease had the potential to spread rapidly(13). Davidson suggested that an outbreak of disease could cause a loss of \$1,168,000,000 to the national income(14).

**Risk reduction:** The chances of introducing the infection can be reduced to virtually nil if antelope are only imported from countries or zones that are free from the infection. The requirements specified in the *OIE International Animal Health Code*(15) for importation of animals from foot and mouth disease-free zones or countries are that they show no clinical signs of foot and mouth disease and that they have been kept in a foot and mouth disease-free zone for at least 3 months(15). However, in view of the lack of specific information about carrier states in all species of antelope, the catastrophic effect introduction would have, and the fact that there are ample safe sources from which antelope could be introduced, they should only be introduced from disease-free countries or zones.

#### **Recommendations:**

**Source of animals:** Animals should be sourced from zoos in disease-free countries or zones.

**Quarantine:** Provided they are brought from disease-free countries, no quarantine for foot and mouth disease would be necessary.

Treatment: Vaccination is not recommended.

**Diagnostic tests:** No testing for foot and mouth disease would be required if they are brought from safe sources.

**Risk of introduction:** Provided animals are brought from disease-free countries the risk of introduction is negligible and is the same as for bringing sheep or cattle from Australia or wapiti from Canada.

# References

 Thompson GR. 1994. Foot and mouth disease. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 2. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 825-52. Oxford University Press, Cape Town, Oxford, New York.

- 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(3), 765-9.
- 3. Shimshony A. 1986. FMD in mountain gazelles. Wildlife Disease Newsletter, April 2-4. Supplement to Journal of Wildlife Diseases, 22(2).
- Shimshony A. 1988. Foot and mouth disease in mountain gazelle in Israel. Revue Scientifique et Technique Office International des Epizooties, 7(4), 917-23.
- 5. Ferris NP., Condy JB., Barnett ITTR., Armstrong RM. 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(3), 306-16.
- Thompson GR. 1997. The role of carrier animals in the transmission of foot and mouth disease. Conference proceedings of the 64<sup>th</sup> General Session of the OIE, Paris, 20-24 May 1996. Pp. 87-103.
- Donaldson AI., Gloster J., Harvey LD., Deans DH. (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. Veterinary Record, 110(3), 53 -7
- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-165. Oxford University Press, Cape Town, Oxford, New York.
- Gainaru MD., Thompson GR., Bengis RG., Esterhuysen JJ., Bruce W., Pini A. 1986. Foot and mouth disease and the African buffalo (*Syncerus caffer*). Il Virus excretion and transmission during acute infection. Onderstepoort Journal of Veterinary Research, 53(2), 75-85.

- Keet DF., Hunter P., Bengis RG., Bastos A., Thompson GR. 1996. The 1992 foot and mouth disease epizootic in the Kruger national Park. Journal of the South African Veterinary Association, 67(2), 83-7.
- 11. Sarma G., Das SK., Dutta PK. 1983. Outbreak of foot and mouth disease in the Assam state zoo. Veterinary Record, 113(18), 420-1.
- Donaldson AJ., Kitchen RP. 1996. Foot and mouth disease. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 47-56. OIE, Paris.
- Forbes RN. 1992. Foot and mouth disease risk assessment study. Determination of the risks involved . Ministry of Agriculture and Fisheries Report
- 14. Davidson RM. 1991. Foot and mouth disease. Surveillance, 18(3), 13-15.
- 15. OIE. International Animal Health Code. Pp. 87-105. Office International des Epizooties. Paris 1997.

# 2.2 Rinderpest

Rinderpest is a highly contagious OIE List A disease of cattle and other ruminants. In the great rinderpest epidemic in Africa about the turn of the century, half of the 5.75 million head of cattle in South Africa died and the effect on wild ruminants was devastating(1). As recently as 1979 a second African pandemic decimated wildlife populations in western, central and eastern Africa(1). In 1980 1,000,000 cattle were lost in Nigeria(2). Rossiter(3) has reviewed the disease.

**Etiological agent:** The disease is caused by the rinderpest virus (Family: Paramyxoviridae, Genus: *Paramyxovirus*). Individual strains of virus vary in their pathogenicity for various species(3).

**Susceptible species:** Cattle are most often infected but the disease has also been described in sheep, goats, pigs, African buffalo, giraffe, eland, kudu(3); buffalo, waterbuck and bushbuck(4); kudu, buffalo, impala, eland and kongoni(5). Neutralising antibody has also been found in waterbuck, orynx and impala(6). It must be assumed that all antelope can be infected.

**Incubation period:** The incubation period is usually 3-9 days in cattle(7). Information for individual antelope species was not found.

**Carrier state:** No carrier state is known in cattle, although rare chronic cases occur(3)(7).

**Transmission:** Transmission is by droplet infection over a short distance and by contact(3)(7).

**Signs of disease:** In typical acute disease there is fever, malaise, nasal discharge, ulceration of oral and nasal mucosa, diarrhoea and high mortality. However, in both cattle and wild ruminants the disease sometimes occurs in a mild form(3).

**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 controlled in livestock, by controlling the movement of animals together with vaccination. The safety and efficacy of vaccine in antelope is not known.

**Diagnosis:** Provisional diagnoses made from the clinical signs of disease can be confirmed by, virus isolation, polymerase chain reaction, agar gel immunodiffusion and serology including competitive ELISA and virus neutralisation(8).

**Implications:** The disease could be potentially devastating for the susceptible New Zealand cattle population, but would probably be easier to control than foot and mouth disease.

**Risk reduction:** When rinderpest occurs in African wildlife the animals involved cannot be carefully observed or inspected and there are gaps in knowledge concerning the pathogensis and epidemiology of the disease in antelope. After the 1979 outbreak of rinderpest in buffalo in the Serengeti National Park, the infection but not the disease was present until at least 1987 (9)(10). Severe outbreaks of the disease occurred in wild antelope in Kenya as recently as 1997(11). For these reasons no importations of antelope from infected countries particularly the Central African countries should be allowed.

The introduction of the disease should be easily avoided and does not represent a risk if antelope are introduced only from countries that are free from rinderpest. In this case the OIE recommendations for the importation of wild ruminants are only that animals should be free from clinical disease and quarantined for 21 days. OIE recommendations for importation of animals from infected countries are similar, except that animals may be vaccinated(12). No information was found on the vaccination of antelope. Serologically positive animals other than cattle cannot be imported into New Zealand(13). Because of the seriousness of the disease and the gaps in our knowledge about the disease in individual antelope species it is recommended that antelope should only be brought from disease-free countries and zones. There should are several safe sources from which antelope could be obtained.

# **Recommendations:**

**Source of animals:** Animals should be sourced from zoos in disease-free countries or zones.

**Quarantine:** Provided they are brought from disease-free countries no quarantine for rinderpest would be necessary.

Treatment: Vaccination is not recommended.

**Diagnostic tests:** No testing would be required if they are brought from safe sources.

**Risk of introduction:** The risk of introduction is virtually non-existent provided they are brought from safe sources.

# References

- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-165. Oxford University Press, Cape Town, Oxford, New York.
- 2. Shanthikumar SR., Atilola MA. 1990. Outbreaks of rinderpest in wild and domestic animals in Nigeria. Veterinary Record, 126(13), 306-7.
- Rossiter PB. 1994. Rinderpest. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 2. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 735-58. Oxford University Press, Cape Town, Oxford, New York.
- 4. Shanthikumar SR., Malachi SA., Mayiyagbe KA. 1985. Rinderpest outbreak in free-living wildlife in Nigeria. Veterinary Record, 117(18), 469-70.
- Kock RA., Wambua J., Mwanzia J., Rossiter P., Wamwayi H., Kock N. 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, pp. 98-104.
- Wafula JS., Mushi EZ., Karstad L. 1982. Antibodies to rinderpest virus in the sera of some wildlife in Kenya. Bulletin of Animal Health and Production in Africa, 30(4), 363-5.

- Scott GR. 1990. Rinderpest virus. In: Virus Infections of Ruminants. Ed. Dinter Z., Morein B. Pp. 341-54. Elsevier Science Publishers, Amsterdam, Oxford, New York, Tokyo.
- 8. Taylor WP. 1996. Rinderpest. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.69-76. OIE, Paris.
- Rossiter PB., Taylor WP., Bwangamoi B., Moorehouse PD., Haresnape JM., Wafula JS., Nyange JF. Gumm ID. 1987. Continuing presence of rinderpest virus as a threat to East Africa, 1983-1985. Veterinary Record, 120(30), 59-62.
- Anderson EC., Jago M., Mlengeya.T., Timms G., Payne A., Hirji K. 1990. A serological survey of rinderpest antibody in wildlife and sheep and goats in Northern Tanzania. Epidemiology and Infection, 105(1), 203-4.
- Kock RA., Wambua JM., Mwanzia J., Wamwayi H., Ndungu EK., Barrett T., Kock ND., Rossiter PB. 1999. Rinderpest epidemic in wild ruminants in Kenya 1993-7. Veterinary Record ,145(10), 275-83.
- 12.. OIE. International Animal Health Code. Pp 119-29. Office International des Epizooties. Paris 1997.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.3 Rift Valley Fever

Rift Valley fever epidemics occur in years of heavy rainfall in Africa. The disease causes serious economic losses in livestock, and serious disease in humans. Swanepoel(1) has written an extensive review of the disease.

Etiological agent: Rift Valley fever virus (Family: Bunyaviridae, Genus: Phlebovirus).

**Susceptible species:** Rift Valley fever is predominantly a disease of sheep and to a lesser extent, cattle. Abortions were described in springbok and blesbok during an outbreak of the disease in livestock. The etiology of the abortions in springbok was not proven, although circumstantial evidence indicated Rift Valley fever was a possible cause(1). Low levels of antibody have been found in a few species of antelope and in African buffalo(1). During a serological survey for diseases of wildlife species in Zimbabwe, antibody to Rift Valley fever was found to be most prevalent in rhino, buffalo and waterbuck(2). In humans it causes a serious disease and a low mortality rate.

**Incubation period:** The incubation period is short usually only about 1-2 days(1).

**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(1).

**Transmission:** Transmission is by mosquitoes including: *Aedes caballus, Aedes circumluteolus/luteolateralis, Aedes juppi, Anopheles cinereus, Anopheles costani, Anopheles mcintoshi, Culex neavi, Culex theileri, Culex zambaensis and Eretmapodites quinquevittatus* (1). Transovarial transmission of the virus in mosquitoes occurs in a low number of cases(1). Non-mosquito mechanical transmitters of disease such as sandflies are of minor importance.

**Signs of disease:** The infection causes high numbers of abortions and perinatal deaths in lambs and a much lower mortality in calves. In antelope, antibodies have been found, but disease and lesions have not been described except that abortions in springbok and blesbok may have been due to the disease(1). In humans it causes an acute fever with flu-like signs, photosensitivity and sometimes complications such as displaced retina. Deaths are rare in humans.

**Epidemiology:** Epizootics of the disease occur in years of heavy rainfall. In these periods the mosquito population is able to multiply and spread from the permanent water sites where they are normally maintained, to breed in surface water in normally dry areas. It is possible that the disease is maintained by transovarial transmission in mosquitoes during the years when it does not cause endemic disease(1).

**Diagnosis:** A diagnosis may be suspected from the clinical signs, and confirmed by histopathology, virus isolation, and antibody tests such as virus neutralisation and indirect ELISA(3).

**Implications:** Four species of Australian mosquitoes were shown to be capable vectors for the disease under experimental conditions(4). Two of these species of these species, *Aedes notoscriptus* and *Culex quinquefasciatus*, are established in New Zealand(5). The other species established in New Zealand(5) have not been investigated for their vector capability for the virus. It is not known whether the disease could become established in New Zealand where there is a predominantly winter rainfall pattern and the lambing season is in a period of low mosquito activity. If it established, it could cause serious economic repercussions.

**Risk reduction**: Antelope should preferably be introduced from disease-free countries. If animals are brought from infected countries, standard quarantine procedures in insect free premises will prevent the introduction of the disease. According to the *OIE International Animal Health Code*, importation of wild ruminants from infected countries can be undertaken with or without vaccination. If animals are vaccinated they must be quarantined for 30 days prior to shipment. If unvaccinated, they should be subjected to diagnostic testing before and during the 30 day quarantine period(6). This testing would prove that animals did not sero-convert while in quarantine.

In addition to the OIE requirements, animals should not be imported during times when the disease is occurring or when mosquitoes are prevalent. Pre-entry quarantine should be in insect-free premises. Antelope should be serologically tested while in quarantine and any groups of animals held in quarantine where sero-conversions occur should not be imported as this may indicate that the quarantine premises were not insect-free.

#### **Recommendations:**

**Source of animals:** Animals should preferably be sourced from zoos in disease-free countries or zones. Animals should only be brought from countries where the disease occurs during seasons when vectors are inactive and there are no outbreaks of the disease occurring.

**Quarantine:** If they are brought from infected countries they should be quarantined in insect-free premises for 4-weeks.

**Treatment:** Vaccination is not recommended.

**Diagnostic tests:** Animals should be subjected to serological testing (virus neutralisation test or ELISA) before entry into the quarantine premises and again at the end of the quarantine period. It is MAF policy that serologically positive animals may be imported(7). However, when sero-conversions occur during quarantine, the quarantine premises cannot be regarded as insect free and the shipment should be suspended pending upgrading of the quarantine premises and further testing.

**Risk of introduction:** Provided the recommended precautions are taken the risk of introduction of the disease is negligible.

#### References

- Swanepoel R. 1994. Rift Valley fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 688-718. Oxford University Press, Cape Town, Oxford, New York.
- Anderson EC., Rowe LW. 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(2), 441-9.
- OIE International Animal Health Code. Pp 151-154. Office International des Epizooties. Paris 1997.

- 4. Turrell MJ., Kay BH. 1998. Susceptibility of selected strains of Australian mosquitoes (Diptera: Culicidae) to Rift Valley fever virus. Journal of Medical Entomology, 35(2), 132-5.
- 5. Holder PW., Bullians M., Brown G. 1999. The mosquitoes of New Zealand and their animal disease significance. Surveillance, 26(4), 12-5.
- 6. OIE International Animal Health Code. Pp 151-154. Office International des Epizooties. Paris 1997.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.4 Bluetongue

Bluetongue is an OIE List A disease that primarily affects sheep. It occurs in Africa, the Middle East, Asia, the United States and Australia. The virus is carried by *Culicoides* spp. The disease has been reviewed by Verwoerd and Erasmus(1).

**Etiological agent:** The agent is the bluetongue virus (Family: Reoviridae, Genus: *Orbivirus*), 24 antigenically different serotypes of the virus are recognised.

**Susceptible species:** Bluetongue is a serious disease of sheep, but all ruminants are probably susceptible(1). African antelope do not develop clinical disease but inapparent infections occur in many species(1)(2). Pronghorn antelope develop severe clinical disease(2)(3). Bluetongue virus has been isolated from addax, ibex, African buffalo and sable antelope(4) and pronghorn antelope(5). Antibody to bluetongue virus has been found in a wide variety of African antelope(6)(7)(8). Experimental infection of blesbok resulted in asymptomatic infection(9).

**Incubation period:** The incubation period is usually 4-6 days in sheep but can vary from 2-15 days(1).

**Carrier state:** Animals carry the virus for short periods after they recover from infection. Cattle carry the virus for up to at least 49 days(1). Antelope probably carry the virus for shorter periods but the periods during which the various antelope species remain viraemic are not generally known. Viraemia of 17 days in blesbok, 3 days in pronghorn antelope and 35 days in mountain gazelle have been described(2).

**Transmission:** The disease is carried by *Culicoides* spp. Transovarial transmission does not occur(1). About 20 of the 1,400 species of *Culicoides* are known to be competent vectors of bluetongue(10).

**Signs of disease:** The clinmical signs include fever and inappetance. Oedema and hyperaemia of the subcutaneous tissues occur in the head, lips and other parts of the body. A swollen blue tongue, from which the disease gets its name, is an occasional symptom. There may be ulcers in the mouth. Painful muscles due to muscle degeneration may occur. Hyperaemia of the coronary region of the hoof and lameness is common.

**Epidemiology:** In the endemic areas of Africa the disease occurs in late summer and autumn before the first frosts, when the concentration of biting midges is high. It is not clearly understood what acts as reservoir of the disease during the winter months when midges are not active. The virus may survive in frost-free areas where there is permanent water and animals such as cattle or possibly antelope act as reservoirs of infection(1).

**Diagnosis:** Initial diagnosis depends on observation of typical clinical signs of disease and post mortem lesions (particularly haemorrhages in the wall of the pulmonary artery). Virus isolation in embryonated eggs, polymerase chain reaction and antibody detection by agar gel immunodiffusion and competitive ELISA may be used to confirm the diagnosis(11).

**Implications:** If introduced into a New Zealand zoo, the disease is unlikely to spread, because *Culicoides* vectors are not present in New Zealand(12).

Risk reduction: The OIE recommendations for importations of wild ruminants from bluetongue free countries or parts of a country that have no common border with an infected country, are that only a health certificate is required. For importation from infected countries, animals should be kept in an insect-free quarantine station for 60 days (maximum carrier time plus maximum incubation period) and subjected to diagnostic tests for bluetongue. They should be protected from insects during transport(13). It is MAF policy that animals should be guarantined in a vector-free area for 2 months(14). Serological testing before entry into quarantine and before release from quarantine as recommended in the case of Rift Valley fever is not necessary since Culicoides are sylvatic and seldom enter buildings so infection in a guarantine building is very unlikely. Vaccination with bluetongue virus implies vaccination with 23 different strains of virus, usually given in three separate injections of pooled attenuated virus strains, at 3 week intervals. This procedure is unsuitable for the importation of antelope and there might be the risk of importing vaccine strains of virus with the imported animals. Importation of live virus, including vaccine strains, is prohibited under the Biosecurity Act 1993 unless an import permit has been granted. Therefore, vaccination with attenuated live virus is not recommended

#### **Recommendations:**

**Source of animals:** Animals should preferably be sourced from zoos in disease-free countries or zones, but animals could also be sourced from infected countries.

**Quarantine:** If they are brought from infected countries they should be quarantined in vector-free premises for 60 days.

Treatment: Vaccination is not allowed.

**Diagnostic tests:** No serological testing is necessary.

**Risk of introduction:** The chance of introduction of the disease in antelope is negligible if they come from bluetongue-free areas or are suitably quarantined. As long as New Zealand remains free from *Culicoides* even the introduction of viraemic animals would be of little concern, but it remains MAF policy to prohibit the importation of viraemic animals.

#### References

- Verwoerd DW., Erasmus BJ. 1994. Bluetongue. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 443-59. Oxford University Press, Cape Town, Oxford, New York.
- Hoff GL., Hoff DM. 1976. Bluetongue and epizootic haemorrhagic disease: A review of the diseases in non-domestic artiodactyls. Journal of Zoo Animal Medicine, 7, 26-30.
- 3. Sohn R., Yuill TM. 1991. Bluetongue and epizootic haemorrhagic disease in wild ruminants. Bulletin for the Society for Vector Ecology, 16(1), 17-24.
- Castro AE., Rodgers SJ. 1984. Congenital abnormalities in cattle associated with an epizootic of bluetongue virus (serotype II). Bovine Practitioner, 19, 87-91.

- Stott JL., Else KC., McGowan B., Wilson LK., Osburn BI. 1981. Epizootiology of bluetongue virus in Western United States. Proceedings of the United States Animal Health Association, 85, 170-80.
- Hamblin C., Anderson EC., Jago M., Mlengeya T., Hirji K. 1990. Antibodies to some pathogenic agents in free-living wild species in Tanzania. Epidemiology and Infection, 105(3), 585-94.
- Drolet BS., Mills KW., Belden EL., Mecham JO. 1988. Development and application of an enzyme-linked immunosorbent assay (ELISA) for the detection of antibody to bluetongue virus. Proceedings of the United States Animal Health Association, 92, 113-22.
- Anderson EC., Rowe LW. 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(2), 441-9.
- Neitz WO. 1933. The blesbuck (*Damaliscus albifrons*) as a carrier of heartwater and bluetongue. Journal of the South African Veterinary Medical Association, 2, 24-26.
- MacLachlan NJ., Wilson D., Gard G., Mellor PS., Nevill EM. 1998. Supporting document for the OIE International Animal Health Code, Chapter 2.1.9 on Bluetongue. OIE Working Group on bluetongue. Office International des Epizooties, Paris, September 1998.
- 11. Eaton B. 1996. Bluetongue. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.109-18. OIE, Paris.
- 12. Motha J., Hansen M., Irwin G. 1997. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance, 24(4), 18-9.
- 13. OIE. International Animal Health Code. Pp. 155-60. Office International des Epizooties. Paris 1997.

 Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.5 Epizootic haemorrhagic disease

Epizootic haemorrhagic disease is a disease that is caused by an orbivirus and carried by *Culicoides*. It is a disease mainly of deer and pronghorn antelope in the United States of America. Because of the similarities in epizootiology what has been said about bluetongue applies to epizootic haemorrhagic disease and there is little reason to repeat it in this section. The epizootiology, distribution and pathogenesis of the two viruses have been reviewed(1) and an outbreak in which 1,000 deer and pronghorn antelope died has been described(2).

It is recommended that pronghorn antelope from the USA be quarantined for a period of 2-months as recommended for preventing the entry of bluetongue.

# References

- 1. Sohn R., Yuill TM. 1991. Bluetongue and epizootic haemorrhagic disease in wild ruminants. Bulletin for the Society for Vector Ecology, 16(1), 17-24.
- Brodie SJ., Bardsley KD., Diem K., Mecham JO., Norelius SE., Wilson WC. 1998. Epizootic haemorrhagic disease: analysis of tissues by amplification and in situ hybridisation reveals widespread orbivirus infection at low copy numbers. Journal of Virology, 72(5), 3863-71.

## 2.6 Rabies

Rabies is an invariably fatal zoonotic disease. It is carried by carnivores and transmitted to other animals including ruminants and man, when they are bitten by infected carnivores. The disease has been extensively reviewed by Swanepoel(1).

**Etiological agent:** The etiological agent is the rabies virus (Family: Rhabdoviridae, Genus: *Lyssavirus*).

**Susceptible species:** Most mammals, including carnivorous animals, vampire and other bats and ruminants (usually dead-end host) are susceptible.

**Incubation period:** Variable depending on the bite site, infectious dose and probably strain of virus. It can be from weeks to months. Six months is usually taken as the upper limit, but 611 days has been described(2).

**Carrier state:** Infected carnivores transmit the disease while they are viraemic and suffering from the disease, before they die. The Indian grey mongoose may be an asymptomatic carrier of the disease(3) and bats may be carriers of the closely related European bat *Lyssavirus*(4).

**Transmission:** Transmission is by the bite of an infected carnivorous animal. Ruminants and humans are usually dead end hosts, but all infected animals are potentially dangerous.

**Signs of disease:** Infected animals typically show signs of nervous disease with typical paralytic or aggressive (furious rabies) syndromes or unnatural behaviour, salivation and incoordination. In ruminants there may be abnormal bellowing, salivation, aggression, incoordination and other signs of nervous disease.

**Epidemiology:** The disease is transmitted from the bite of infected carnivores such as dogs, jackals, foxes, cats, mongoose, vampire bats, racoons etc., depending on the country. Ruminants are generally dead-end hosts. The exception is that in Namibia the disease has been seen as a horizontally sustained infection in kudu in a rabies epizootic that killed thousands of kudu between 1977 and 1983(5)(6). It has been suggested that the disease may have been transmitted in these browsers by saliva

contaminating leaves and twigs of thorn trees, following a disproportionate increase in the kudu population(7).

**Diagnosis:** The diagnosis in suspected cases 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(8).

**Implications:** The introduction of an infected antelope would have serious implications for animal handlers in zoos, but would probably not result in transmission to other animals. Lateral spread as has occurred in kudu would be unlikely. The disease would be unlikely to spread outside the zoo.

**Risk reduction:** The only countries free from rabies, from which animals are likely to be introduced are England and Australia. However, the risk of introduction from countries where the disease occurs would be acceptably low if animals were only brought from zoos, that have had no cases of rabies for prolonged periods of time. Quarantine for long periods is an effective measure for preventing introduction, but is expensive and unpopular with importers. The OIE recommendations for importation of wild mammals from infected countries, that have not been kept in confined conditions, is that they be symptom free and kept in a quarantine station for 6 months prior to shipment. However, animals which have been confined for at least 12 months prior to shipment, in an establishment that is free from rabies for at least a year, can be imported without quarantine(9). In practice this means that animals that have been confined in zoos for long periods would not need to be quarantined. Vaccination of antelope is not recommended as no information was found on the vaccination of various antelope species, although some vaccines are suitable for use in domestic ruminants(1).

## **Recommendations:**

**Source of animals:** Animals should preferably be sourced from zoos in disease-free countries or zones, or from zoos with a history of freedom from rabies for at least a year.

**Quarantine:** Quarantine is unnecessary if they are brought from suitable safe sources. Where doubt exists about the safety of the source they should be quarantined for 6-months.

**Treatment:** Vaccination is not recommended.

Diagnostic tests: No serological testing is necessary.

**Risk of introduction:** The risk of introduction is remote provided the recommendations are adhered to.

- Swanepoel R. 1994. Rabies. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 493-552. Oxford University Press, Cape Town, Oxford, New York.
- Baer GM. 1990. Rabies virus. In: Virus Infections of Ruminants. Ed. Dinter Z., Morein B. Pp. 341-54. Elsevier Science Publishers, Amsterdam, Oxford, New York, Tokyo.
- Everard COR., Everard JD. 1988. Mongoose rabies. Review of infectious diseases (supplement 4), S610-S614. According to Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-165. Oxford University Press, Cape Town, Oxford, New York.
- Peres-Jorda JL., Ibanez C., Munoz-cervera M., Tellez A. 1995. Lyssavirus in *Eptesicus serotinus* (Chiropetra:Vesperatilionidae). Journal of Wildlife Diseases, 31(3). 372-7.
- 5. Schneider HP. Proceedings of an International Conference on rabies control in the tropics. Tunis, October 3-6, 1983.
- Hassel RH. 1984. Rabies in kudu antelope (*Tragelaphus strepsiceros*). Proceedings of the 13th World Congress on Diseases of Cattle. Durban 1984, 1, 65-70.

- 7. Hubschlke OJB. 1988. Rabies in kudu antelope. Reviews of Infectious Diseases, 10, (Supplement 4), S629-33.
- Aubert M., Cliquet F., Barrat J. 1996. Rabies. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.207-17. OIE, Paris.
- 9. OIE. International Animal Health Code. Pp 215-8. Office International des Epizooties. Paris 1997.

# 2.7 Lumpy skin disease

Lumpy skin is an OIE List A disease that causes serious economic loss in cattle in Africa. It has spread to the Arabian Peninsula, and has occurred in Israel but was eradicated. For a review of the disease see Reference 1.

**Etiological agent:** The disease is caused by the lumpy skin disease virus (Family: Poxviridae Genus: *Capripox*).

**Susceptible species:** Cattle, giraffe and impala are highly susceptible to experimental infection(2) and antibody has been found in African buffalo, but two buffalo calves did not develop disease following experimental infection(1). The disease has been described in a captive-bred Arabian oryx at the National Wildlife Research Centre in Saudi Arabia. Two per cent of the oryx in the herd were serologically positive(3).

**Incubation period:** After experimental infection of cattle a local reaction and fever develops in 4-7 days and skin nodules develop in 7-19 days(1).

**Carrier state:** No carrier state is known. Viraemia persists for about 4 days and the virus can persist in skin nodules for 33 days, in the semen for 22 days and saliva for 11 days(4).

**Transmission:** The pattern of infection indicates that the disease is spread by biting flies but the vectors are unknown(1).

**Signs of disease:** Initially there is fever, inappetance and enlarged superficial lymph glands. Typical large pox lesions (lumps) develop on the skin a few days after the initial fever reaction. The lumps may cover the whole body. Sometimes lesions are found in trachea and alimentary tract, particularly the abomasum. Oedema of a limb or limbs occurs in some cases(1).

**Epidemiology:** In Africa the disease occurs in periodic epidemics, the last of which occurred in 1989-90. The vectors are not known but the evidence that it is spread by biting flies is compelling. Antibodies have been found in African buffalo(1). In a study in Tanzania, antibody was not found in buffalo or antelope(5). Giraffe and impala were highly susceptible to experimental infection(2). The African buffalo may be the maintenance host, but definitive proof is lacking.

**Diagnosis:** The clinical picture and pathology are typical. Definitive confirmation is provided by virus isolation or demonstration by electron microscope, identification of antigen by fluorescent antibody, antigen capture ELISA and serological tests such as virus neutralisation(6). Serological test generally do not have high sensitivity or specificity(6).

**Implications:** If infected animals are introduced clinical manifestations of the disease are unlikely to occur in antelope, although the disease has been seen in an Arabian oryx(3). If is not known if suitable vectors occur in New Zealand, but if they do the disease could spread to cattle outside of the zoos.

**Risk reduction:** Animals should preferably be imported from disease-free countries. OIE recommendations for importing wild bovines from infected countries is that they should show no clinical signs of lumpy skin disease and that they should be quarantined for 28 days prior to shipment(7). It is recommended that in addition to the OIE recommendations it should be specified that quarantine should be in insect free premises and that importations should be at times when there is no active outbreak of disease and when potential insect vectors are not active.

#### **Recommendations:**

**Source of animals:** Animals should preferably be from countries or zones that are free from the disease. If brought from an infected country they should be brought at times when biting fly activity is lowest and there are no active outbreaks of the disease.

**Quarantine:** Quarantine is unnecessary if they are brought from suitable safe sources. Animals from infected countries should be quarantined in insect free premises for 28 days.

**Treatment:** Vaccination is not recommended.

**Diagnostic tests:** Serological testing should be done before entry into quarantine and again before release from quarantine to check that sero-conversion did not occur during quarantine. Sero-conversion in quarantine should result in suspension of the importation, pending negotiations about

upgrading the quarantine premises and additional quarantining and testing of the animals. Serologically positive animals may be imported(8).

**Risk of introduction:** The risk of introduction is negligible if recommendations are followed.

- Barnard BJH., Munz E., Dumbell K., Prozesky L. 1994. Lumpy skin disease. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol.
   Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 604-12. Oxford University Press, Cape Town, Oxford, New York.
- Young E, Basson PA., Weiss KE. 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.
- Greth A., Gourreau JM, Vassart M., Vy NB., Wyers M., Leferve PC. 1992. Capripox disease in an Arabian oryx (*Oryx leucoryx*) from Saudi Arabia. Journal of Wildlife Diseases, 28(2), 295-300.
- 4. Weiss KE. 1968. Lumpy skin disease virus. Virology monographs. Vol. 3. New York, Springer Verlag. According to Barnard et al. see reference 1.
- Hamblin C., Anderson EC., Jago M., Mlengeya T., Hirji K. 1990. Antibodies to some pathogenic agents in free-living wild species in Tanzania. Epidemiology and Infection, 105(3), 585-94.
- Kitching RP., Carn V. 1996. Lumpy skin disease. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.93-101. OIE, Paris.
- OIE. International Animal Health Code. Pp 145-49. Office International des Epizooties. Paris 1997.

 Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

## 2.8 Malignant catarrhal fever

Malignant catarrhal fever is the most commonly occurring serious infectious disease of zoo animals. The wildebeest-associated form of the disease has been reviewed in reference 1.

**Etiological agent:** Wildebeest-associated malignant catarrhal fever is caused by alcelaphine herpes virus-1. (Family: Herpesviridae, alcelaphine herpesvirus-1 (AHV1)). In addition to AHV1, a closely related herpesvirus carried by sheep (ovine herpesvirus-2, OHV2) commonly causes the disease in cattle and deer. Isolates from hartebeest and topi showed little nucleic acid homology with typical AHV1 isolates(2)(3). Isolates from topi and hartebeest have been designated alcelaphine herpesvirus-2 (AHV2) and produced only atypical malignant catarrhal fever in cattle after artificial infection and could not be transferred by natural transmission(1). Other closely related herpesviruses may occur in other antelope(4)(5). An isolate from a roan antelope was provisionally designated as being hippotragine herpesvirus-1(HHV1)(6)(7).

**Susceptible species:** The disease occurs in cattle and deer (AHV-1 and OHV-2). In zoos the disease has been reported in kudu, sitatunga , eland and roan(1) and in duiker and gerenuk (8) but is not seen in wild antelope. However, antibody has been found in a wide range of antelope including nyala, roan, sable, tsessebe, waterbuck, gemsbok, red hartebeest, and black and blue wildebeest(1); oryx, topi, addax(3); lechwe, reedbuck(4); and pronghorn antelope(9). Presumably antibody could be found in all species of antelope.

**Incubation period:** The incubation period is usually about 3-7 weeks but can be up to 73 days(10)

**Carrier state:** Blue and black wildebeest commonly carry the AHV1(1)(5)(11). Barnard has suggested that other members of the subfamilies Alcelaphinae and Hippotraginae may also carry the infection. Topi carry AHV2.

**Transmission:** The disease is transmitted by contact over short distances between wildebeest and susceptible animals(1)(11). It has been suggested that separation of wildebeest and cattle by a distance of 1 kilometre is needed to avoid infection(1). Transmission from wildebeest generally occurs when infected young wildebeest are present.

**Signs of disease:** In cattle there is fever, inappetance, mucopurulent nasal discharge (snotsiekte), corneal opacity, and erosions of buccal and nasal cavities. In deer acute, often bloody, diarrhoea is common and the mortality rate is high. Antelope in zoos suffer from a similar disease but clinical disease is not seen in wild antelope.

**Epidemiology:** On cattle farms and in zoos, outbreaks of disease usually correlate with contact with wildebeest(1)(8)(12) or with contact with sheep(OHV2)(12). The virus was apparently endemic to Africa and the indigenous antelope appear to have developed resistance to it under natural conditions, but are susceptible to the disease when kept in captivity. Wildebeest are common asymptomatic carriers in the wild and in zoos. Other related viruses such as AHV2 and HHV1 and possibly other related herpesviruses occur. AHV1 and bovine cytomegalovirus (BHV3) are serologically and immunogenically related(13).

**Diagnosis:** The clinical picture is often typical. The diagnosis can be confirmed by histological examination and demonstration of the typical histopathological lesions or by isolation of virus or the polymerase chain reaction for amplification of viral DNA(14)(5). Serological tests such as virus neutralisation and the immunoperoxidase test can be used to demonstrate antibody(15).

**Implications:** Introduction of the virus could have serious implications for the health of deer and antelope held in zoos. However, the disease would not spread to farmed cattle and deer outside of zoos as there are no wild or farmed maintenance hosts (wildebeest) in contact with cattle and deer.

**Risk reduction:** Outbreaks of malignant catarrhal fever (AHV-1) that have occurred in zoos, have invariably been transmitted by wildebeest. Wildebeest from any source should be regarded with suspicion. However, the official policy of MAF with regard to the importation of wildebeest has already been determined. It is that they should be held in a quarantine premises that are physically separated from cattle and deer(16). This policy should also be applied to other members of the Alcelaphinae and Hippotraginae. This policy implies that infected wildebeest do not constitute a risk to farmed livestock provided they are isolated from them in quarantine facilities or zoos. There is clearly a greater risk for other animals in zoos and steps to safeguard zoo animals are the responsibility of the zoos concerned.

### **Recommendations:**

**Source of animals:** Antelope could be obtained from any source but the risk of wildebeest carrying AHV-1 are high and other Alcelaphinae and Hippotraginae may carry related viruses. Zoos should protect their own interests by not introducing high risk animals (particularly wildebeest).

**Quarantine:** During the time they are quarantined for other diseases, wildebeest and members of the Alcelaphinae and Hippotraginae should be quarantined in premises in which domestic stock are not present.

Treatment: No suitable vaccine is available.

Diagnostic tests: Serological testing is not recommended.

**Risk of introduction:** If wildebeest are introduced, the risks of introducing the disease into animals held in zoos will be high. If other members of the families Acelaphinae or Hippotraginae are introduced the risk is low.

- Barnard BJH., Van der Lugt JJ., Mushi EZ. 1994. Malignant catarrhal fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol.
   Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 946-57. Oxford University Press, Cape Town, Oxford, New York.
- Seal BS., Klieforth RB., Heuschele WP. 1987. Evidence for variation among malignant catarrhal fever virus isolates. Proceedings of the United States Animal Health Association, 91, 527-42.
- Seal BS., Heuschele WP., Klieforth RB. 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(9), 1447-53.

- 4. Hamblin C., Hedger RS. 1984. Neutralising antibody to wildebeest-derived catarrhal fever in African wildlife. Comparative Immunology, Microbiology and Infectious Diseases, 7(3-4), 195-9.
- Lahijani RS., Sutton SM., Klieforth RB., Murphy MF., Heuschele WP. 1994. Application of polymerase chain reaction to detect animals latently infected with agents of malignant catarrhal fever. Journal of Veterinary Diagnostic Investigation, 6(4), 403-9.
- Gulland FM., Reid HW., Buxton D., Lewis JCM., Kock RA., Kirkwood JK. 1989. Malignant catarrhal fever in a roan antelope (*Hippotragus equinus*) at Regent's Park. Veterinary Record, 124(2), 42-3.
- 7. Reid HW., Bridgen A. 1991. Recovery of a herpesvirus from a roan antelope (*Hipppotragus equinus*). Veterinary Microbiology, 28(3), 269-78.
- 8. Meteyer CU., Gonzales BJ., Heuschele WP., Howard EB. 1989. Epidemiologic and pathologic aspects of an epizoootic of malignant catarrhal fever in exotic hoofstock. Journal of Wildlife Diseases, 25(2), 280-6.
- Li H., Shen DT., Jessup DA., Knowles DP., Gorham JR., Thorne T., O'Toole D., Crawford TB. 1996. Prevalence of antibody to malignant catarrhal fever virus in wild and domestic ruminants by competitive inhibition ELISA. Journal of Wildlife Diseases, 32(3) 437-43.
- Plowright W. 1990. Malignant catarrhal fever virus. In: Virus Infections of Ruminants. Ed. Dinter Z., Morein B. Pp. 123-50. Elsevier Science Publishers, Amsterdam, Oxford, New York, Tokyo.
- Castro AE., Ramsay EC., Dotson JF., Schramke ML., Kocan AA., Whitenack DL. 1984. Characteristics of the herpesvirus of malignant catarrhal fever isolated from captive wildebeests calves. American Journal of Veterinary Research, 45(3), 409-15.
- 12. Erasmus BJ. 1986. Malignant catarrhal fever. Biennial report of the Veterinary Research Institute, Onderstepoort 1984-5. Pp. 54-5.

- 13. Rossiter PB., Gumm ID., Mirangi PK. 1988. Immunological relationship between malignant catarrhal fever virus acelaphine herpesvirus and bovine cytomegalovirus (bovine herpesvirus 3). Veterinary Microbiology, 16(3) 211-8.
- Michel AL. 1993. Generation of a nucleic acid probe specific for the alcelaphine 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.
- Reid HW. 1996. Malignant catarrhal fever. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.627-33. OIE, Paris.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.9 Ephemeral fever

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

**Etiological agent:** The disease is caused by the ephemeral fever virus, (Family: Rhabdoviridae, Genus: *Lyssavirus* of the bovine ephemeral fever serogroup).

**Susceptible species:** Amongst domestic livestock the disease is seen only in cattle. Antibody to the virus was found in several antelope species(1)(2). In another study antibody was widespread in 16 species of wildlife including buffalo, eland, nyala, waterbuck and bushbuck(3).

Incubation period: The incubation period varies from 2-10 days(1).

**Carrier state:** No carrier state is known. In cattle viraemia lasts 1-3 days with a maximum of 2 weeks(1).

**Transmission:** The virus is transmitted by *Culicoides* spp. and possibly other biting insects(1).

**Signs of disease:** The disease in cattle is characterised by a phasic fever with two or more peaks. Other signs of disease include inappetance and a stiff painful gait (3 day stiff sickness). Recovery usually takes place after a few days. Occasional cases become recumbent and do not rise, these cases often end fatally.

**Epidemiology:** In summer rainfall areas 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 temperate climates, the disease disappears after the first frost. The natural reservoir of the virus is unknown. As buffalo and several species of antelope have antibody to the virus, it is possible that one or more of these species may be a reservoir species for the virus. However, the disease is endemic in Australia, a country that is essentially free from antelope species and has a very different fauna than Africa. An alternative mechanism could be for the virus to pass through the egg of the vector thus providing a similar mechanism for virus survival to that postulated for Rift Valley fever virus.

As New Zealand has no *Culicoides* spp.(4) and only 16 species of mosquitoes(5) it is possible that the disease could not establish here but this remains unproven.

**Diagnosis:** In cattle the disease can be diagnosed clinically from the typical symptoms and confirmed by virus isolation and serology(1). However, the disease does not occur in antelope and infection of these species is only indicated retrospectively, by the development of antibodies.

**Implications:** Introduction and establishment of the disease might have significant economic implications for the New Zealand cattle industries. It is not known whether competent vectors are present in New Zealand.

**Risk reduction:** No long-term carrier antelope have been described, so it must be assumed that standard quarantine practices would prevent the introduction of the virus. If animals were introduced during times when vector activity is low the risk would be even further reduced. Since the carrier state is not known, it is MAF policy to allow the importation of antibody positive animals. The only reason to test animals serologically would be to show that they had not sero-converted in quarantine

## **Recommendations:**

**Source of animals:** Animals could be brought from infected or uninfected countries. They should be imported during seasons when the vectors are inactive and when no outbreaks of disease are occurring.

**Quarantine:** If brought from disease-free countries no quarantine is necessary. If brought from infected countries a standard quarantine period of 4 weeks will ensure that the animals are no longer viraemic.

Treatment: Vaccination is not recommended.

**Diagnostic tests:** Serological testing is not recommended if animals are brought from disease-free countries. If brought from infected countries serological testing before entry into quarantine and before release from quarantine is recommended to show that the animals did not sero-convert while in quarantine. Animals serologically positive before entering quarantine could be imported without further testing(6). Sero-conversion in quarantine should result in suspension of the importation pending negotiations about upgrading the quarantine premises and additional quarantining and testing of the animals.

**Risk of introduction:** The disease has not been introduced to New Zealand with the importation of Australian cattle. The risk of introduction with introduced antelope is remote.

- St. George T. 1994. Ephemeral fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 553-62. Oxford University Press, Cape Town, Oxford, New York.
- Hamblin C., Anderson EC., Jago M., Mlengeya T., Hirji K. 1990. Antibodies to some pathogenic agents in free-living wild species in Tanzania. Epidemiology and Infection, 105(3), 585-94.
- Anderson EC., Rowe LW. 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(2), 441-9.
- 4. Motha J., Hansen M., Irwin G. 1997. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance, 24(4), 18-9.
- 5. Holder PW., Bullians M., Brown G. 1999. The mosquitoes of New Zealand and their animal disease significance. Surveillance, 26(4), 12-5.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

## 2.10 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(1).

**Etiological agent:** The etiological agent is Nairobi sheep disease virus (Family: Bunyaviridae, Genus: *Nairovirus*).

**Susceptible species:** The disease occurs in sheep and has been described in the blue duiker. No references were found to the disease in other antelope but if duiker are susceptible other species could also be(1).

**Incubation period:** The incubation period is 4-6 days(1).

**Carrier state:** Viraemia in sheep is thought to last only as long as the animal remains febrile, which is 1-7 days. No carrier state has been described(1).

**Transmission:** The virus is transmitted by *Rhipicephalus appendiculatus* in which species transovarial transmission of the disease occurs(1)(2). It is also transmitted by other members of the *Rhipicephalus* genus and by *Amblyomma variegatum* but transoviarial transmission does not occur in these ticks(1).

**Signs of disease:** The disease is characterised by fever, diarrhoea, nasal discharge, abortions and high mortality.

**Epidemiology:** It is a tick-borne disease. Sporadic outbreaks of disease occur when susceptible sheep are moved to areas infested by infected ticks.

**Diagnosis:** A provisional diagnosis may be confirmed by virus isolation and antibody demonstration by the indirect fluorescent antibody test(2).

**Implications:** Introduction of the disease agent would have no impact unless tick vectors were also introduced and became established. If infected tick vectors became established in New Zealand, the disease could have serious financial implications for the sheep industry. Standard quarantine would ensure that introduced antelope are not carrying the virus but introduced ticks would be a more serious cause for concern.

**Risk reduction:** Introduction of tick-free animals, from disease-free countries or zoos and standard quarantine measures would ensure safe introduction of antelope. Animals could be introduced from infected countries and it is MAF policy that serologically positive animals may be introduced(3). However, it must be realised that if animals are serologically positive the ticks they are carrying are likely to be infected since the animals come from an infected environment. Special care must be taken be taken to ensure ticks are not introduced since the virus is transmitted transovarially in the tick.

### **Recommendations:**

**Source of animals:** Animals should preferably from countries or zones that are free from the disease, but introductions from infected countries would be possible.

**Quarantine:** Standard quarantine (4 weeks) will ensure that no antelope that are carrying the virus are introduced. Measures to ensure freedom from ticks should be rigorously carried out.

Treatment: No vaccine is available.

**Diagnostic tests:** In the case of animals coming from infected countries, serological testing before and at the end of the quarantine period should be used to ensure that sero-conversion does not occur in quarantine. Sero-conversion in quarantine should result in suspension of the importation pending negotiations about upgrading the quarantine premises and additional quarantining and testing of the animals. It should also lead to increased efforts to ensure that ticks are not introduced.

**Risk of introduction:** The risk of introduction is remote as there is no reason to import antelope from infected Central African countries. The risk of establishment is nil, unless vectors are also introduced.

#### References

1. Terpstra C. 1994. Nairobi sheep disease. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson

GR., Tustin RC. Pp. 718-22. Oxford University Press, Cape Town, Oxford, New York.

- Davies FG. 1996. Nairobi sheep disease. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 669-72. OIE, Paris.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.11 Crimean-Congo disease

Crimean-Congo disease is important because it is a zoonotic tick borne disease, but it is of little importance to livestock.

**Etiological agent:** The disease is caused by the Crimean-Congo haemorrhagic fever virus (Family: Bunyaviridae, Genus: *Nairovirus*).

**Susceptible species:** A wide range of animals, birds (including ostriches) and humans can be infected. Clinical disease has not been described in antelope. Antibody has been found in giraffe and antelope especially the larger species such as kudu and eland(1). The virus can be transmitted to ostriches(2) and ground feeding birds(3).

**Incubation period:** In humans the incubation period is usually 1-3 days, sometimes up to 7 days after infection by a tick bite and somewhat longer after exposure to blood(1).

**Carrier state:** Cattle carry the virus only for short periods. There is no information about a carrier state in antelope(1).

**Transmission:** Ticks of the genus *Hyalomma* are the main vectors of the virus. The disease has been transmitted to humans butchering infected carcasses(2)(4) and as a nosocomial infection in a hospital(5).

**Signs of disease:** The disease causes a haemorrhagic fever in humans with a mortality of about 3%. In livestock and antelope the infection is apparently asymptomatic.

**Epidemiology:** It is a sporadically occurring tick-borne disease in man. Infected antelope do not develop disease. It is believed that the virus is maintained in a rodent/tick cycle in the Crimea(6). In South Africa the infection may be maintained in a complex cycle involving immature ticks feeding on rodents, hares, and guinea fowl and mature ticks feeding on antelope with humans incidentally infected by adult ticks(7).

**Diagnosis:** Infection can be confirmed by virus isolation and serological tests(1).

**Implications:** Establishment of the disease would not be possible unless the vectors were also introduced and became established. Even if vectors were introduced there would be little impact on the health of domestic or zoo animals. Humans would be at risk.

**Risk reduction:** Crimean-Congo haemorrhagic fever is a serious zoonotic disease and there is still uncertainty about the role of antelope and other animals in the epidemiology of the disease. Therefore antelope should preferably be brought from countries that are free from the infection. It seems likely that there is no carrier state in antelope, but this has not been conclusively proven. However, as long as antelope are tick free there is no chance of introducing the virus. Antibody free antelope have probably not been infected or have been infected some time previously and their antibody levels have declined to non-detectable levels. Antelope with detectable antibody have probably been infected and are no longer carriers of the disease. Therefore, both sero-positive and seronegative antelope could be imported with minimal risk provided they did not sero-convert while in quarantine.

### **Recommendations:**

**Source of animals:** Animals should preferably be from countries or zones that are free from the disease, but introductions from infected countries would be possible.

**Quarantine:** Standard quarantine (4 weeks) will provide good assurance that antelope carrying the virus are not introduced. Measures to ensure freedom from ticks should be rigorously enforced.

Treatment: No vaccine is available.

**Diagnostic tests:** Antelope from infected countries should be serologically tested (virus neutralisation test ) before entry into quarantine and if negative, again before release from quarantine. Sero-conversion in quarantine should result in suspension of the importation pending negotiations about upgrading the quarantine premises and additional quarantining and testing of the animals. It should also lead to increased efforts to ensure that ticks are not introduced.

**Risk of introduction:** The chances of an introduced antelope being a carrier are extremely low. No known vectors occur in New Zealand, so that risk of establishment is remote.

- Swanepoel R. 1994. Crimean-Congo haemorrhagic fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 723-32. Oxford University Press, Cape Town, Oxford, New York.
- Swanepoel R., Leman PR., Burt FJ., Jardine J., Verwoerd DJ., Capua I., Bruckner GK., Burger WP. 1998. Experimental infection of ostriches with Crimean-Congo haemorrhagic fever virus. Epidemiology and Infection, 121(2), 427-32.
- Zeller HG., Cornet JP., Camicas JL. 1994. Experimental transmission of Crimean-Congo haemorrhagic fever virus by west African wild ground-feeding birds to *Hyalomma marginatum rufipes* ticks. American Journal of Tropical Medicine and Hygiene, 50(6), 676-81.
- Swanepoel R., Shepherd AJ., Leman PA., Miller GB. 1985. A common source of Crimean-Congo haemorrhagic fever on a dairy farm. South African Medical Journal, 68(9), 635-7.
- Van Eeden PJ., Joubert JR., Van de Wal BW., King JB., De Kock A., Groenewald JH. 1985. A nosocomial outbreak of Crimean-Congo haemorrhagic fever at Tygerberg hospital. South African Medical Journal, 68(9), 635-7.
- Markeshin Sia., Smirnova SE., Evstaf'eV IL. 1992. [An assessment of the status of natural foci of Crimean-Congo haemorrhagic fever in the Crimea] Zh Mikrobiol Epidemiol Immunobiol 4, 28-31.
- Rechov Y. 1986. Seasonal activity and hosts of the vectors of Crimean-Congo haemorrhagic fever in South Africa. South African Medical Journal, 69(6), 364-8.

### 2.12 Bovine virus diarrhoea

Bovine virus diarrhoea virus occurs universally and Type-1 (BVDV-1)is very common in New Zealand. In cattle it causes sporadic cases of mucosal disease and foetal deaths. In sheep, a closely related pestivirus causes hairy shaker disease. The more virulent Type 2 virus does not occur in New Zealand(1). The disease has been reviewed by Harkness and Van der Lugt(2).

**Etiological agent:** The etiological agent is bovine virus diarrhoea virus (Family:Togaviridae, genus: *Pestivirus*). The BVDV-1 virus of cattle and the border disease virus of sheep are similar but distinct viruses. Comparatively recently another pestivirus BVDV-2 has been described. Cytopathogenic and non-cytopathogenic strains of pestiviruses occur.

**Susceptible species:** Cattle, sheep, pigs and deer. Antibody to pestivirus has been found in a wide range of antelope including roan, wildebeest, oryx, kudu, sable, giraffe(3)(4), wildebeest and topi (5), scimitar horned oryx(6); and eland, nyala and bushbuck(7). Antibody is also found in captive exotic ruminants including antelope and giraffe(8). Virus was isolated from five exotic ruminants(9). No information was found on the typing of the virus strains isolated from antelope.

**Incubation period:** As many animals do not show any signs of disease, the incubation period is hard to define. Antibody develops 16-28 days after infection(2).

**Carrier state:** A persistent carrier state occurs in cattle and sheep that have been infected *in utero*. These carrier animals are usually serologically negative. An antibody negative carrier state has also been described in eland(7). Animals that develop antibody are not carriers.

**Transmission:** The disease is spread by contact between animals(2). The infection may be transmitted to the foetus when pregnant animals are infected.(2).

**Epidemiology:** Most infections in cattle 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 the development of antibody negative persistently infected calves(2). A persistently infected eland was found amongst 303 antibody negative eland(7). Mucosal disease in cattle occurs when an antibody negative animal,

persistently infected with a non-cytopathic strain of bovine virus diarrhoea virus is reinfected by an antigenically homologous cytopathic virus. The reinfection may possibly arise from a new infection or from a mutation of the persistent virus.

**Diagnosis:** The presence of pestivirus can be demonstrated by antigen capture ELISA, reverse transcriptase polymerase chain reaction or virus isolation. Antibody can be demonstrated by virus neutralisation or ELISA(10)(11).

**Implications for zoos:** Introduction of BVDV-1 would be of no importance as the virus is already present in most New Zealand cattle herds. The introduction of a Type-2 strain might have an economically significant effect. However, the impact of introducing BVD-2 cannot be predicted, as it might not cause widespread disease in the face of the overwhelming infection of the cattle population with BVDV-1.

**Risk reduction:** As there is no specific information about BVDV-2 infections in antelope it must be assumed that the pathogenesis of BVDV-2 infections is similar to that of BVDV-1 infections. Therefore the same risk reduction measures should be applied for both virus types. It is MAF policy that serologically positive cattle can be released into New Zealand and this policy should also apply to antelope(12). A low percentage of antibody negative animals are likely to be persistent carriers of infection. Therefore, introduced animals should be negative in the antibody capture ELISA test and preferably antibody positive. Quarantine measures would not help to prevent the introduction of the virus by persistent carriers.

## **Recommendations:**

**Source of animals**: All countries must be regarded as infected with BVD. Animals could be introduced from any country.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of virus by persistently infected animals.

**Treatment:** Vaccination of antelope has not been described and it is not recommended.

**Diagnostic tests:** All animals should be negative to the antigen capture ELISA.

**Risk of introduction:** The risk of introduction of a carrier animals is low if introduced animals are tested by the antigen capture ELISA.

- 1. Vilcek S., Bjorklund HV., Horner GW., Meers J., Belak S., 1998. Genetic typing of pestiviruses from New Zealand . New Zealand Veterinary Journal , 46, 35-7,.
- Harkness JW., Van der Lugt JJ. 1994. Bovine virus diarrhoea. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 642-50. Oxford University Press, Cape Town, Oxford, New York.
- 3. Soine C., Uatanaua G., Depner KR. 1992. Prevalence of bovine viral diarrhoea virus in Namibian wildlife. Tropical Animal Health and Production, 24(2), 125-6.
- Depner K., Hubschele OJB., Liess B. 1991. Prevalence of ruminant pestivirus infections in Namibia. Onderstepoort Journal of Veterinary Research, 58(2), 107-9.
- 5 Hyera JMK., Liess B., Anderson E., Hirji KN. 1992. Prevalence of bovine viral diarrhoea virus in some wild ruminants on northern Tanzania. Bulletin of Animal Health and Production in Africa, 40(3), 143-51.
- 6. Frolich K., Flach EJ. 1998. Long term viral serology of free living and captive ungulates. Journal of Zoo and Wildlife Medicine, 29(2), 165-70.
- Anderson EC., Rowe LW. 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(2), 441-9.
- Doyle LG., Heuschele WP., Fowler ME. 1983. Prevalence of antibody to major viral disease in captive exotic ruminants. Proceedings, 1983 Annual Meeting, American Association of Zoo Veterinarians, Tampa Florida, October 24-27, 1983. Pp. 76-82.

- 9. Doyle LG., Heuschele WP. 1983. Bovine viral diarrhoea virus infection in captive exotic ruminants. Journal of the American Veterinary Medical Association, 183(11), 1257-9.
- 10. Brownlie J., Edwards S. 1996. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 651-9. OIE, Paris.
- 11. Nettleton PF. 1996. Border disease. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.678-85. OIE, Paris.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 2.13 Miscellaneous virus diseases

Diseases that have not been described in antelope but could potentially infect antelope include:

- 1. Louping ill virus and other diseases of the tick-borne encephalitis complex. These flaviviruses cause louping ill in Scotland and England . In Europe, Siberia and parts of Asia viruses of the tick-borne encephalitis complex occur. Louping ill has a low mortality rate and occurs mainly in sheep and grouse. Grouse are probably the maintenance hosts. Infected animals are viraemic for about 7 days. The diseases have not been described in antelope although they occur in such a wide range of other animals that this possibility cannot be ruled out. Louping ill is carried by *Ixodes ricinus*(1). Other viruses of the complex are carried by *Rhipicephalus* spp. and *Hyalomma anatolicum* in Asia.
- Wesselsbron disease virus. This arbovirus occurs in sheep but has not been described in antelope. It occurs sporadically in seasons of high rainfall in Africa(2). There may be a maintenance host that has yet to be discovered. This maintenance host could be an antelope species. However, it could equally be maintained by transovarial transmission in mosquitoes as has been postulated for Rift Valley fever.

Antelope have antibody to a number of virus diseases that are of minor importance as far as animal importation is concerned. These include:

1. Infectious bovine rhinotracheitis and infectious pustular vaginitis are caused by bovine herpesvirus-1. Although there are three subtypes of BHV-1 they are all antigenically similar(3). BHV-1 infections are usually asymptomatic but cause lifelong latent infections. In common with other herpesvirus infections latently infected animals do not shed virus except during sporadic periods of virus reactivation generally attributed to stress. Serologically positive animals can be assumed to be latently infected. BHV-1 infection is extremely common in cattle in New Zealand, but the sub-types that cause severe respiratory disease and abortion are not thought to be present(4)(12). Antibody to bovine herpesvirus-1 has been found in several species of antelope(5)(6)(7)(8). The significance of IBR titres in antelope is not known. It has been suggested that only the wildebeest is susceptible to BHV-1(9) and that in this species infection is manifested as a genital infection(10)(11). Antibody to BHV-1 in other antelope may be caused by another herpes virus(9). Since the viruses in question are antigenically similar there is no presently available system of serological testing that can distinguish between the sub-types of BHV1. Excluding all serologically positive animals would reduce the chance of introducing any new strains of herpes viruses into New Zealand. This is the MAF policy for importation of cattle(12).

- 2. **Enzootic bovine leucosis**. Antibodies to enzootic bovine leucosis have been found in antelope(5). The disease occurs in New Zealand and is the subject of a dairy industry eradication campaign. However, the disease is not highly infectious and requires close contact for transmission. It would be unlikely to spread from a zoo to other livestock.
- 3. **Akabane** disease and related simbu virus antibodies occur in the blood of most species of African antelope, but no disease is associated with the infection(13)(14). In cattle, the virus is present in the blood for only about 4 days and is carried by *Culicoides* and mosquitoes. *Culicoides* do not occur in New Zealand and the New Zealand mosquitoes may not be competent vectors. The virus is generally harmless except for causing deformities in the foetus when pregnant females are infected(14). It is unlikely that the virus could be introduced, if standard quarantine measures are enforced.
- Coronavirus infections commonly cause neonatal diarrhoea in calves. The disease occurs universally. Coronavirus has been found in captive sitatunga and waterbuck(15).
- 4. **Vesicular stomatitis** is an insect-borne virus that has remained confined to Central and North America. It is an OIE List A disease by virtue of the similarity of the clinical symptoms to foot and mouth disease. Inability to exclude a diagnosis of foot and mouth disease may

lead to the imposition of expensive disease control measures, closing of export markets etc. until the diagnosis is established. The disease never causes economic losses comparable to foot and mouth disease. Losses are likely to be limited to decreased production that occur on individual farms and involve direct losses from the disease and complications such as mastitits. Since the diagnosis of foot and mouth disease and vesicular stomatitis are now rapid and accurate the classification of vesicular stomatitis as a List A diseases seems to be an artificial one.

Antibody was found in 60% of 139 pronghorn antelope killed by hunters. Experimental infection with the virus showed that the species is susceptible(16). Pronghorn antelope from the United States should be quarantined for a standard 4 week period and moved in times when insect vectors are not active and no outbreaks of vesicular stomatitis are occurring. MAF policy is that serologically positive animals could be safely released from quarantine(12).

- Swanepoel R. 1994. Louping ill. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 671-78. Oxford University Press, Cape Town, Oxford, New York.
- Swanepoel R., Coetzer JAW. 1994. Wesselsbron disease. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 663-70. Oxford University Press, Cape Town, Oxford, New York.
- Van Oirschot JT. Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis.1996. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp.281-90. OIE, Paris.
- 4. Horner GW. 1990. Infectious bovine rhinotracheitis in New Zealand. Surveillance, 17(2), 15-6.

- 5. Hamblin C., Anderson EC., Jago M., Mlengeya T. Hirji K. 1990. Antibodies to some pathogenic agents in free-living wild species in Tanzania. Epidemiology and Infection, 105(3), 585-94.
- Doyle LG., Heuschele WP., Fowler ME. 1983. Prevalence of antibody to major viral disease in captive exotic ruminants. Proceedings, 1983 Annual Meeting, American Association of Zoo Veterinarians, Tampa Florida, October 24-27, 1983. Pp. 76-82.
- 7 Doyle LG., Heuschele WP. 1983. Bovine viral diarrhoea virus infection in captive exotic ruminants. Journal of the American Veterinary Medical Association, 183(11), 1257-9.
- Anderson EC., Rowe LW. 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(2), 441-9.
- Pastoret PP., Thiry E., Broucher A., Schwers A., Thomas I., Dubuisson J. 1988. Diseases of wild animals transmissible to domestic animals. Revue Scientifique et Technique, 7(4), 705-36.
- 10. Mushi EZ, Karstad L., Jesset DM., Rossiter PB. 1979. Observations on the epidemiology of the herpesvirus of infectious bovine rhinotracheitis / infectious pustular vulvovaginitis in wildebeest. Journal of Wildlife Diseases, 15(3), 481-7.
- Mushi EZ, Karstad L. 1979. Experimental infection of Wildebeest with the herpesvirus of infectious bovine rhinotracheitis/infectious pustular vulvovaginits, Journal of Wildlife Diseases, 15(4), 579-83.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.
- Al-Busaidt SM., Hamblin C., Taylor WP. 1987. Neutralising antibodies to Akabane virus in free-living animals in Africa. Tropical Health and Production, 19, 197-202.

- St George TD., Standfast HA. 1994. Diseases caused by Akabane and related Simbu-group viruses. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 681-7. Oxford University Press, Cape Town, Oxford, New York.
- Chasey D., Reynolds DJ., Bridger JC., Debney TG., Scott AC. 1984. Identification of coronaviruses in exotic species of Bovidae. Veterinary Record, 115(23), 602-3.
- Thorne ET., Williams ES., Adrian WJ., Gillin CM. 1984. Vesicular stomatitis in pronghorn antelope: serologic survey and artificial infection. Proceedings of the United States Animal Health Association, 87, 638-43.

# 3.0 Protozoal diseases of antelope

### 3.1 Babesiosis

Babesiosis is a tick-borne disease that is one of the important diseases of African livestock. For a review of the disease see reference 1. Although antelope have antibody to the disease they are generally not thought to be carriers of the infection(1).

**Etiological agent:** Babesiosis is a complex of diseases caused by a number of protozoal parasites including *Babesia bovis*, *B. occulans*, *B. major*, *B. ovata*, and *B. divergens* (in cattle), *B. motasi* and *B. ovis* (in small ruminants) and *B. irvinesmithi* (in sable antelope).

**Susceptible species:** *B. irvinesmithi* is unique to sable antelope and was responsible for an outbreak of babesiosis in captive sable antelope(3). *B. bigemina* has been found in a sable antelope(2) and transmitted to calves with ticks from the sable. 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 infected sable. *B. bovis* has been found in blood smears from asymptomatic Uganda kob(4), but this finding does not seem to have been confirmed by other workers. *B. occulans* and possibly another unnamed species are virtually harmless(1). *B. major* is found in cattle in southern Europe and North Africa, *B. divergens* is important in northern Europe and *B. ovata* occurs in Japan and Asia. *B. motasi* is a parasite of small ruminants. However, because of the difficulties in unravelling the taxonomy and the difficulties of identification, some of the information in published papers may be questionable. *B. bovis* and *B. bigemina* are the most important species in Africa.

**Incubation period:** In cattle the incubation period is from 7-21 days. It is slightly longer for *B. bovis* than for *B. bigemina*(1).

**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(1). Indigenous African cattle tend to carry infections for shorter periods. Antelope may have antibodies

against the parasites but there is no evidence that they are carriers of the parasite(1). However, *B. bovis* were found in blood smears from asymptomatic Uganda kob(4), but independent verification of this finding has not yet been produced.

**Transmission:** Boophilus microplus, Boophilus decoloratus, Boophilus annulatus and probably Boophilus geigyi carry B. bigemina. B. bovis is transmitted by Boophilus microplus but not by Boophilus decoloratus(5). Rhipicephalus evertsi carries B. bigemina. B. occulans is carried by Hyalomma marginatum and Hyalomma truncatum carries an unnamed harmless Babesia. Haemophysalis punctata carries B. major and Haemophysalis longicornis carries B. ovata. It should be noted that Haemophysalis longicornis, is endemic in the warmer parts of New Zealand. No reference was found to Haemophysalis longicornis being a vector for other Babesia infections. The parasite is transmitted transovarially at least in Boophilus spp.

**Signs of disease:** In cattle the disease is characterised by fever, inappetance, anaemia, haemoglobinaemia and haemoglobinuria. Infections with *B. bovis* have a high mortality in European breeds of cattle(1). Sable antelope imported from a zoo in Germany into South Africa developed a disease characterised by a massive haemolytic crisis associated with *B. irvinesmithi(3)*.

**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. However there is no good evidence that serologically positive antelope are carriers of *Babesia* infections(6).

**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(7). In cases of doubt *Babesia* carriers can be identified by inoculating blood into splenectomised calves.

**Implications:** If carrier animals were introduced they would be unlikely to transmit the disease unless competent vectors were also introduced. In warmer parts of New Zealand *Haemaphysalis longicornis* is endemic and could carry *B. ovata.* No references were found to indicate that *Haemophysalis longicornis* is a vector for other species of *Babesias.* 

**Risk reduction:** The first priority should be to prevent the introduction of ticks. Introduced animals should be tick-free and introduced into a tick free environment. It is MAF policy that parasitaemic animals carrying OIE List B diseases may not be introduced into New Zealand. Therefore, serologically positive cattle may not be introduced(8). It is doubtful if sero-positive antelope carry *Babesias* but it is recommended that a conservative approach be followed and the policy should be the same for antelope as for cattle. Ticks on any animals even non-carrier animals could be transovarially infected with *Babesias*.

## **Recommendations:**

**Source of animals**: Animals should be introduced from zoos with a history of freedom from babesiosis. However, it is not necessary to specify that animals come from countries that are free from *Babesias*.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of the disease but are important to ensure freedom from ticks. Standard quarantine of 4 weeks is recommended.

Treatment: Vaccination of antelope is not recommended.

**Diagnostic tests:** Introduced animals should be serologically negative. All animals should be negative to the indirect fluorescent antibody tests for *B. bovis* and *B. bigemina* if they come from infected countries or zones.

**Risk of introduction:** The probability of introducing *Babesias* and allowing the disease to become established is very low, provided introduction of ticks is prevented and introduced animals are serologically negative.

#### References

 De Vos A., Potgieter FT. 1994. Babesiosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 278-94. Oxford University Press, Cape Town, Oxford, New York.

- 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(2), 75-80.
- McInnes EF., Stewart CG., Penzhorn BL., Meltzer DGA. 1991. An outbreak of babesiosis in imported sable antelope (*Hippotragus niger*). Journal of the South African Veterinary Association, 62(1), 30-32.
- 4. Kupper W., Wolters M., Tscharf I. 1983. Observations on Kob antelopes (*Kobus kob*) in Northern Ivory Coast and their epizootiological role in trypanomyiasis transmission. Zeitscrift fur Angewandte Zoologie, 70(3)277-83.
- 5. Jongejan E., Uilenberg G. 1994. Ticks and control methods. Revue Scientifique et Technique Office des Epizooties, 13(4), 1201-26.
- 6. Bigalke RD. 2000. Written statement in a report to MAF.
- De Vos AJ., Jorgensen WK. 1996. Bovine babesiosis. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 305-313. OIE, Paris.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 3.2 Theileriosis

Theilerioisis is caused by *Theileria parva* (East Coast fever, corridor disease and Zimbabwe theileriosis) and *T. annualata* (Mediterranean Coast theileriosis) cause diseases of major economic importance. Other species of *Theilerias* are virtually non-pathogenic to cattle. For reviews of the diseases see references 1-5. A closely related *Cytauxzoon* sp causes disease in some antelope but not in cattle.

Etiological agents and susceptible species: Some Theileria spp. are listed below:

- T. parva (East Coast fever in cattle, benign in buffalo).
- *T. parva lawrenci* (Corridor disease in cattle, buffalo are symptomless carriers of the disease).
- T. parva bovis (Zimbabwe theileriosis in cattle).
- T. annulata (Mediterranean Coast fever in cattle).
- T. mutans (benign infestation of cattle and buffalo).
- T. velifera (benign infestation of cattle and buffalo).
- *T. orientalis* (benign infestation of cattle in Asia, New Zealand, Australia. Probably synonymous with *Theileria sergenti*).
- T. seperata (benign infection of sheep).
- *T. taurotragi* (benign species found in eland, it causes a usually benign infection in cattle but occurs in rare cases of turning-sickness).

Cytauxzoonosis (causes disease in tssesebe, kudu, duiker, roan antelope and giraffe)(6).

Incubation period: The incubation period for East Coast fever is 8-25 days(1).

**Carrier state:** Carrier animals are found in buffalo particularly for corridor disease. Carriers are not known in antelope except possibly for *T. taurotragi* in eland.

**Transmission:** Ticks of the genus *Rhipicephalus* particularly *Rhipicephalus appendiculatus* are the main vectors of East Coast fever and other related theilerioses(1)(2).

**Signs of disease:** East Coast fever is characterised 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. *T*.

*taurotragi* may cause occasional cases of turning sickness, a form of the disease where lymphoblasts containing schizonts block capillaries in the brain. *T. taurotragi* and *T. mutans* were probably confused in the past.

**Epidemiology:** East Coast and Mediterranean Coast fevers are tick-borne diseases but antelope are not considered to be involved in maintaining the disease and are not susceptible. Buffalo may play a small role in maintaining East Coast fever and a major role as maintenance hosts for Corridor disease(1). *T. taurotragi* is found in eland and is a mild infection in cattle except in occasional cases where it causes turning sickness(1).

**Diagnosis:** The diagnosis can be confirmed by identification of the schizonts in lymph node smears or small piros in blood smears. The indirect fluorescent antibody test can be used to identify antibody(7).

**Implications:** There are no implications for domestic animals as antelope are not involved in carrying the pathogenic species of *Theilerias*. *T. taurotragi* carried by eland is of little importance as it causes asymptomatic infections in eland, and the vectors are not present in New Zealand. The disease caused by a *Cytauxzoon* sp. can cause disease in duiker, sable antelope, kudu, roan, giraffe, and tsessebe(6), but not in domestic stock. Itwould be of no importance even in zoos if suitable tick vectors are not also imported.

**Risk reduction:** No risk reduction strategies are required as antelope do not carry virulent species of *Theilerias*.

Risk of introduction: The risk of introduction is remote.

- Lawrence JA., De Vos AJ., Irwin AD. 1994. East Coast fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 309-25. Oxford University Press, Cape Town, Oxford, New York.
- 2. Lawrence JA., De Vos AJ., Irwin AD. 1994. Corridor disease. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed.

Coetzer JAW., Thompson GR., Tustin RC. Pp. 326-28. Oxford University Press, Cape Town, Oxford, New York.

- Lawrence JA., De Vos AJ., Irwin AD. 1994. Zimbabwe theileriosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 329-31. Oxford University Press, Cape Town, Oxford, New York.
- Lawrence JA., De Vos AJ., Irwin AD. 1994. *Theileria taurotragi* infection. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol.
   Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 334-5. Oxford University Press, Cape Town, Oxford, New York.
- Pipano E. 1994. *Theileria annulata* infection. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 341-8. Oxford University Press, Cape Town, Oxford, New York.
- Thomas SE., Wilson DE., Mason TE. 1982. Babesia, Theileria and Anaplasma spp. Infecting sable antelope *Hippotragus niger* (Harris 1938) in Southern Africa. Onderstepoort Journal of Veterinary Research, 49(3), 163-6.
- Dolan TT., Moizaria SP., Katende JM. 1996. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 321-30, OIE, Paris.

# 3.3 Tsetse fly transmitted trypanosomosis

Nagana or trypanosomosis is mainly restricted to the areas of Africa in which tsetse fly (*Glossina* spp.) occur. It is a major disease that has prevented cattle farming in tsetse fly infested areas(1)(2).

**Etiological agent:** The pathogenic species are *Trypanosoma brucei*, *T. congolense* and *T. vivax*. *T. theileri* is a non-pathogenic species. The species pathogenic for man, *T. brucei gambiense* and *T. brucei rhodesiense* are of no importance in the context of importing antelope.

**Susceptible species:** Cattle are susceptible, and buffalo and a variety of antelope carry the parasites(1)(2)(3)(4)(5).

**Incubation period:** The parasites can be found in the blood about 2 weeks after an animal has been bitten by an infected tsetse fly(1).

**Carrier state:** Antelope and buffalo act as asymptomatic carriers of the parasite e.g. kob(3); roan, buffalo, kob, waterbuck(4); and roan, kob, kongoni(5). Other species of antelope have been found to carry the parasite(1).

**Transmission:** Transmission is by tsetse fly. 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 infestation with this parasite has become endemic in South and Central America and the island of Mauritius, in the absence of tsetse flies(1)(2).

**Signs of disease:** It is usually a chronic wasting disease. Animals become weak and anaemic with oedema of the limbs and a gradual loss of condition.

**Epidemiology:** The disease is carried by tsetse flies and restricted to the endemic tsetse fly areas except for *T. vivax* which is also found in South and Central America and Mauritius(1). In South America the disease occurs mainly near low lying swampy areas and may be associated with tabanids as mechanical vectors(6)(7). However, 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 precise factors that allowed the

establishment of *T. vivax* in South America are not known. It therefore seems unlikely that this parasite would establish in New Zealand, but this cannot be proved.

**Diagnosis:** Wet blood film 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(8).

**Implications:** Only *T. vivax* could have some implications for New Zealand as it is found outside of the African tsetse fly areas. Even this parasite is unlikely to establish here. Establishment of the disease would lead to ongoing erosion of farming profitability.

**Risk reduction:** Because of the seriousness of the disease and the impossibility of proving that the infection could not be spread in New Zealand, precautions should be taken to prevent the importation of *T. vivax.* This could most reliably be achieved by importing animals from zoos in countries or zones that are free from the disease. New Zealand successfully imported thousands of Ilama from Chile without importing the parasite. Llamas were imported from areas believed to be free from infection and quarantined and repeatedly tested by the ELISA test. However, there are several countries and zones, that are free from trypanosomosis, from which antelope can be imported and it is recommended that animals should only be brought from these sources.

### **Recommendations:**

**Source of animals**: Animals should be introduced from countries and zones that are free from trypanosomiasis.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of the disease.

**Treatment:** No vaccination is possible and prophylactic chemotherapy is not recommended.

**Diagnostic tests:** Serological or other testing is not necessary if animals are brought from disease-free zones and countries.

**Risk of introduction:** The risk of introduction and establishment of the disease in New Zealand is remote.

# References

- Connor RJ. 1994. African animal trypanosomosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 167-205. Oxford University Press, Cape Town, Oxford, New York.
- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-166. Oxford University Press, Cape Town, Oxford, New York.
- 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 fur Angewandte Zoologie, 70(3), 277-83.
- 4. Truc P., Formenty P., Komoin OkaC. Diallo PB., Lauginie F. 1997. Identification of trypanosomes isolated by KIVI from wild animals in Cote d'Ivoire: diagnostic, taxonomic and epidemiological considerations. Acta Tropica, 67(3), 187-96.
- Verhulst A., Van Meirvenne N., Buscher P., Pandey VS., van Meirvenne N., Uilenberg G., Hamers R. 1993. Wild animals as reservoirs of animal and human trypanosomosis. Resistance or tolerance of animals to disease. Veterinary epidemiology and diagnostic methods, 161, Abstract.

6. Mateus G., Gonzalez M. 1991. Characteristics of a *Trypanosoma vivax* outbreak in Colombia. Revist Cubana Ciencias Veterinarias, 22(3), 167-71. English summary

- Otte MJ., Abuabara JY., Wells EA. 1994. *Trypanosoma vivax* in Colombia: epidemiology and production losses. Tropical Animal Health and Production, 26(3), 146-56.
- Schlater J. 1996. Trypanosomosis (Tsetse-borne). In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 660-4. OIE, Paris.

## 3.4 Surra

Surra, a disease caused by *Trypanosoma evansi,* is an important disease of camels and horses(1). The distribution of the parasite extends from North Africa across large parts of Asia and Russia. It is often fatal in camels, buffaloes, horses, llama and dogs, but it can be mild and sub-clinical in cattle donkeys, goats, sheep, pigs and capybara(2). A search of the literature between 1984 and 1999 yielded no reference to surra in antelope and it is therefore assumed that this disease is not of importance in antelope.

- 1. Fowler ME. 1996. Husbandry and diseases of camelids. Revue Scientifique et Technique. Office International des Epizooties, 15(1), 155-69.
- Kageruka P., van Meirvenne, N. 1996. Surra (*Trypanosoma evans*). In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 686-93. OIE, Paris.

# 3.5 Besnoitiosis (elephant-skin disease)

Besnoitiosis is a disease that affects cattle, goats and horses. It occurs most commonly in the sub-tropical areas of Africa but also occurs more rarely in non-tropical regions. It also occurs in South Korea, Israel, Portugal, France, Venezuela and Russia(1).

Etiological agent: The agent is a protozoan parasite Besnoitia besnoiti.

**Susceptible species:** The primary host is presumed to be a member of the cat family but it has not been identified. Cattle, goats and horses are intermediate hosts. Inapparent infections occur in wildebeest, impala, zebra, donkey, mule and warthog(1)(2).

Incubation period: About 4 days(1).

**Carrier state:** Inapparent infestations with a closely related species or strain occur in wildebeest and impala. The antelope strain is not pathogenic for cattle and has been used as a cattle vaccine(1). The primary host is unknown, but is probably a member of the cat family(1).

**Transmission:** Transmission is presumed to be through the faeces of the primary host(1). The disease can also be transmitted by biting flies but sub-clinical infections are the invariable result(3).

**Signs of disease:** 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 show few signs of disease but inspection of the conjunctiva reveals small, granular cysts in the mucosa. As the disease becomes chronic, some animals develop the typical skin condition with alopoecia and dry thickened skin leading to the common name of olifantvelsiekte or elephant skin disease, which becomes progressive and debilitating. Antelope remain symptomless.

**Epidemiology:** It occurs sporadically particularly in sub-tropical areas. The life cycle for this parasite has not been elucidated. However, for all similar parasites there is a life cycle that involves a carnivorous host that becomes infected while feeding on meat or carcass material and a herbivorous secondary host that is infected from the host's

faeces. Bigalke has suggested that the most likely host for this parasite is one of the cat family(1). The parasite that infests wildebeest and impala does not cause any disease in those animals and is benign for cattle and is used as a vaccine in cattle(3).

**Diagnosis:** In cattle typical signs of disease and the presence of typical cysts in the conjunctiva are usually sufficient to make a diagnosis. The diagnosis can be confirmed by histological examination of skin lesions.

**Implications:** Infected antelope would be asymptomatic carriers of the disease and unlikely to transmit the disease to other animals in a zoo environment. Transmission to a carnivorous primary host is highly unlikely as it should not be zoo practice to feed meat from zoo animals to carnivores. The completion of the life cycle could not occur, making the establishment of the disease in New Zealand impossible.

**Risk reduction:** As infections in antelope are likely to be asymptomatic and would also be benign if transmitted to cattle, there is no reason for concern about this parasite.

**Risk of introduction:** Risk of introduction of the parasite is remote and as long as antelope meat is not fed to carnivores, establishment of the disease would not be possible.

- Bigalke RD., Prozesky L. 1994. Besnoitiosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 245-252. Oxford University Press, Cape Town, Oxford, New York.
- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-166. Oxford University Press, Cape Town, Oxford, New York
- Bigalke RD., Schoeman JH., McCully RM. 1974. Immunization against bovine besnoitiosis with a live vaccine prepared from the blue wildebeest strain of *Besnoitia* grown in cell cultures. 1. Studies in rabbits. Onderstepoort Journal of Veterinary Research, 41(1), 1-6.

# 3.6 Other protozoal diseases

A number of protozoal parasites of antelope are of minor importance because they are unlikely to be introduced, occur rarely or cause insignificant diseases or asymptomatic infections in the countries in which they are endemic. These parasites include:

1. 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 as intermediate hosts. In most species the infection is harmless and they 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 susceptible to infection. Although antelope can be infected as evidenced by serological testing(1)(2), an extensive computer search of the literature revealed no evidence of severe disease in antelope.

Toxoplasmosis is extremely widespread in New Zealand and introduction of antelope carrying *Toxoplasma* cysts would not be significant.

2. 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 infestation of intermediate hosts is usually asymptomatic. Sarcocysts have been found in antelope in zoos in the United States of America(3) and in African (4) and Indian(5) antelope. For a review see reference 6.

Introduction of antelope infested with sarcocystis cysts would be of no significance. The parasites are harmless to the infested animals. In zoos parasites will be unable to complete their life cycles as the definitive hosts are kept isolated from and unable to eat the meat of the infested antelope.

3. Coccidiosis and cryptosporidiosis occur in a wide variety of antelope in zoos(7)(8)(9)(10)(11). Uterine coccidiosis occurs in the uteri of about 1% of impala ewes, in the Kruger National park in South Africa(12). These diseases tend to cause disease in young animals kept in unhygienic conditions. However, the parasites probably occur universally and there is no practical way to prevent their introduction, if live animals are introduced. Introduced antelope

should be adult animals in which oocysts cannot be demonstrated in the faeces. Treatment with coccidiostats could be contemplated although this is unlikely to completely eliminate the parasite. Only if genetic material were introduced as semen or embryos would the parasites be excluded.

- 1. Brillhart DB., Fox LB., Dubey JP., Upton SJ. 1994. Seroprevalence of *Toxoplasma gondii* in wild mammals in Kansas. Journal of the Helminthological Society of Washington, 61(1), 117-21.
- Mohammed OB., Hussein HS. 1994. Antibody prevalence of toxoplasmosis in Arabian gazelles and oryx in Saudi Arabia. Journal of Wildlife Diseases, 30(4), 560-2.
- Stolte M., Odening K., Bockhardt I. 1996. Antelopes kept in European zoological gardens as intermediate hosts of *Sarcocystis* species. Parasitologia Roma, 38(3), 565-70.
- Odening K. Rudolph M., Quandt S., Bengis RG., Bockhardt I., Viertel D. 1998. Sarcocystis spp. in antelopes from southern Africa. Acta Protozoologica, 37(3), 149-58.
- 5. Acharjyo LN., Rao AT. 1988. Sarcocystosis in some Indian wild ruminants. Indian Veterinary Journal, 65(2), 169-70.
- Marcus MB., Van der Lugt JJ. 1994. Sarcocystosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 253-66. Oxford University Press, Cape Town, Oxford, New York.
- Schillhorn van Veen TW., Trapp AL., Daunt D., Richter NA. 1986. Coccidiosis in a kudu. Journal of the American Veterinary Medical Association, 189(9), 1178-9.

- 8. Flach EJ., Blewett DA., Angus KW. 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(4), 446-52.
- Pospischil A., Stiglmair HMT., Von Hegel G., Wiesner H., von Hegel G. 1987.
   Abomasal cryptosporidiosis in mountain gazelles. Veterinary Record, 121(16), 379-80.
- 10. Fenwick BW. 1983. Cryptosporidiosis in a neonatal gazella. Journal of the American Veterinary Medical Association, 183(11), 1331-2.
- 11. Van Winkle TJ. 1985. Cryptosporidiosis in young artiodactyls. Journal of the American Veterinary Medical Association, 187(11), 1170-2.
- Basson PA., McCully RM., Kruger SP., van Niekerk JW., Young E., De Vos V., Keep ME., Ebedes H. 1971. Disease conditions of game in southern Africa: Recent miscellaneous findings. Veterinary Medical Review, 2/3, 313-40.

# 4.0 Rickettsial and chlamydial diseases of antelope

# 4.1 Heartwater

Heartwater is a major disease of livestock in tropical and sub-tropical Africa and has spread to the Caribbean region. A review of the disease is given in Reference 1.

Etiological agent: Heartwater is caused by Cowdria ruminantium.

**Susceptible species:** Cattle, sheep and goats are susceptible. Clinical infections have been reported in black wildebeest, blesbok, springbok and eland and sub-clinical infections have also been reported in giraffe, black wildebeest, blesbok and eland(2). Clinical disease has been reported in a sitatunga(3). The organism could be grown and maintained in endothelial cell cultures from sable and eland(4). It has been suggested that the antelope that are susceptible to heartwater are those such as springbok and blesbok that do not occur in heartwater endemic areas and have not evolved with the organism. Those in endemic areas are generally refractory to the infection and it is postulated that a maintenance host may eventually be identified amongst these animals(5).

**Incubation period:** In cattle the incubation period varies from 9-29days(1).

**Carrier state:** Cattle can carry the infection for a year but sheep only carry it for about 3 weeks(1). Eland, giraffe, kudu and wildebeest have been experimentally infected and the infection was transferred from them to susceptible cattle by *Amblyomma hebraeum*(6). The duration of a carrier state in antelope is not known.

**Transmission:** The disease is carried by ticks of the genus *Amblyomma*, particularly *A. hebraeum* and *A. variegatum*. Generally the infection is transmitted transtadially in the tick. Intrastadial transmission is also likely. There has been one description of transovarial transmission but it is probably very rare(7).

**Signs of disease:** Typical signs of disease include high fever, inappetance, nervous signs and high mortality.

**Epidemiology:** The disease is carried by ticks in sub-tropical and tropical areas of Africa. Young animals are more resistant than adults, and in endemic areas most animals are infected while young and their immunity is maintained by reinfection when ticks are present. It has recently been shown that 1.7% of *Amblyomma hebraeum* from the Kruger National Park carry *Cowdria hebraeum* (8). The Kruger National Park covers an area of nearly 20,000 square kilometres and has been a game reserve free from domestic stock for close to 100 years. This finding provides evidence that heartwater is maintained in a wildlife/vector cycle in the absence of domestic stock.

**Diagnosis:** Signs of 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 polymerase chain reaction can be used to demonstrate *C. ruminantium* DNA in infected tissues. Antibody can be demonstrated by an indirect fluorescent antibody test or by an indirect or a competitive ELISA(9).

**Implications:** There are no implications for New Zealand animal industries as the climate is not suitable for the establishment of *Amblyomma* spp. Many carrier antelope would be asymptomatic and susceptible antelope that are going to develop signs of disease would probably develop the disease while in quarantine. Establishment of the disease would not occur in the absence of *Amblyomma* ticks.

**Risk reduction:** Although *Amblyomma spp.* are unlikely to survive their first winter in New Zealand steps to prevent their introduction should be rigorously enforced. The OIE International Animal Health Code specifies that animals imported from countries infected with heartwater should show no clinical signs of heartwater, be subjected to a diagnostic test for heartwater during the 15 days prior to shipment and be treated with acaricides to be totally tick free(10). MAF policy with regard to serologically positive animals is that they could be carriers of disease and should not be introduced into New Zealand(11). Importation from heartwater free countries and zones would prevent the importation of carrier animals and *Amblyomma* spp. It is recommended that animals should only be brought from heartwater free zones and countries.

#### **Recommendations:**

**Source of animals**: Animals should be introduced from countries and zones that are free from heartwater and *Amblyomma* spp.

**Quarantine:** Standard quarantine and tick control measures should be enforced for prevention of introduction of ticks. Quarantine would be ineffective in preventing the importation of the organism.

Treatment: No vaccination or treatment is recommended.

**Diagnostic tests:** Serological or other testing is not necessary if animals are brought from disease-free zones and countries.

**Risk of introduction:** there would be no risk of introducing the disease if animals are introduced form disease-free zones and countries.

- Bezuidenhout JD. Prozesky L., Du Plessis JL., Van Amstel SR. 1994. Heartwater. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 351-70. Oxford University Press, Cape Town, Oxford, New York.
- 2 Oberem PT., Bezhuidenhout JD. 1987. Heartwater in hosts other than domestic ruminants. Onderstepoort Journal of Veterinary Research, 54, 271-5.
- Okoh AEJ., Oyetunde IL., Ibu JO. 1986. Fatal heartwater in a captive sitatunga. Veterinary Record, 118(25), 696.
- 4. Smith GE., Anderson EC., Burridge MJ., Peter TF., Mahan SM. 1998. Growth of *Cowdria ruminantium* in tissue culture endothelial cells from wild African mammals. Journal of Wildlife Diseases, 34(2), 297-304.
- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-166. Oxford University Press, Cape Town, Oxford, New York.
- Peter TF., Anderson EC., Burridge MJ., Mahan SM. 1998. Demonstration of a carrier state for *Cowdria ruminantium* in wild animal ruminants from Africa. Journal of Wildlife Diseases, 34(3) 567-75.

- Bezuidenhout JD., Oberem PT. 1985. Proof of transovarial transmission of Cowdria ruminantium by Amblyomma hebraeum. Onderstepoort Journal of Veterinary Research, 53(1), 31-34.
- Peter TF., Bryson NR., Perry BD., O'Callaghan CJ., Smith GE., Mlambo G., Horak IG.; Burridge MJ., Mahan SM. 1999. *Cowdria ruminantium* infection in ticks in the Kruger National Park. Veterinary Record,145(11), 304-7.
- 9. Camus E., Uilenberg G. Heartwater. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 229-34. OIE, Paris.
- 10. OIE. International Animal Health Code. Pp 221-2. Office International des Epizooties. Paris 1997.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 4.2 Anaplasmosis

Anaplasmosis is a mainly tick-borne disease that occurs in most tropical and subtropical countries and some temperate parts of the world. Reviews of the disease are given in References 1 and 6.

**Etiological agent:** Anaplasmosis is caused by *Anaplasma marginale* and *A. centrale* in cattle and *A. ovis* in sheep. (Family: Anaplasmataceae, Genus: *Anaplasma*).

**Susceptible species:** Anaplasmosis in cattle is caused by *A. marginale. A. centrale* is relatively benign and is used as a vaccine strain. *A. ovis* is mildly pathogenic for sheep. Naturally occurring *A. marginale* infection (in clinically normal animals) has been described in giraffe, sable, buffalo, black wildebeest(1); buffalo, impala(2); kob(3). Blesbok, duiker and black wildebeest have been experimentally infected(1). The only wild animal in which the disease has been seen is the giraffe(4). It has been suggested that there may be other strains of *Anaplasmas* since transmission to cattle with blood from waterbuck, impala, Grant's gazelle and eland was unsuccessful and isolates from Coke's hartebeest, blue wildebeest and Thompson's gazelle caused mild infections in cattle(1). *A. ovis* has been experimentally transmitted to pronghorn antelope in the United States of America(5). The occurrence of *Anaplasma* infections in wild and domestic ruminants has been reviewed by Kuttler(6).

**Incubation period:** The incubation period is typically from 15-36 days but may be up to 100 days(1).

**Carrier state:** The infection can be carried for long periods and possibly for life by recovered cattle(1). 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 the species was not convincing(8). Mechanical transmission by biting flies such as *Stomoxys calcitrans* and *Tabanus* spp. is possible. Transovarial transmission has been described in *Boophilus* spp. but transtadial transmission is more usual and intrastadial transmission is probably also important(7)(8). Transmission by the only New Zealand cattle tick *Haemophysalis* 

*longicornis,* has not been recorded in reviews on ticks and anaplasmosis by several experts(1)(8)(9)(10).

**Signs of disease:** Signs of disease include fever and inappetance, ruminal stasis and impaction, anaemia and jaundice. The disease tends to be less acute than babesiosis infections and the mortality somewhat lower. Anaplasmosis of sheep generally causes a mild or inapparent infection.

**Epidemiology:** It is a typically tick-borne disease. Those animals that have been born and raised in endemically infected areas are usually immune but animals introduced from non-infected areas are susceptible.

**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 0.0001% infected cells, but 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(8). In case of doubt transmission to splencetomised calves could be attempted.

**Implications:** Imported carrier antelope are unlikely to show any signs of disease. The disease is unlikely to spread in New Zealand due to the absence of tick vectors.

**Risk reduction:** Strict measures to prevent the introduction of ticks are essential. The disease occurs in most tropical and sub-tropical countries and several temperate regions(8). Therefore, sourcing animals from disease-free countries may not be difficult. Since other steps can be taken to ensure that the disease is not introduced and does not become established in New Zealand, introductions from infected countries should be allowed. Introduced animals should be serologically negative(11) and tick free. In addition to these measures it is recommended in the International Animal Health Code that cattle should be treated with an effective drug such as oxytetracycline for five consecutive days at a dose of 22 mg/kg(12).

### **Recommendations:**

**Source of animals**: Animals could be sourced from infected zones and regions but it would be preferable to bring them from disease-free countries.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of the etiological agent but should be strictly enforced to prevent the introduction of ticks.

**Treatment:** Treatment with tetracyclines as recommended by OIE is recommended as an additional supportive measure(12).

**Diagnostic tests:** Animals from infected countries of zones should be subjected to testing with an OIE approved serological test (Complement fixation test or card agglutination test). Serologically positive animals should not be released into New Zealand.

**Risk of introduction:** The risk of introduction is low since and it is unlikely that the disease could establish in New Zealand in the absence of tick vectors.

- Potgieter FG., Stoltsz WH. 1994. Bovine anaplasmosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 351-70. Oxford University Press, Cape Town, Oxford, New York.
- 2. Norval RAI., Fivaz BH., Lawrence JA., Brown AF. 1984. Epidemiology of tickborne diseases of cattle in Zimbabwe. II. Anaplasmosis. Tropical animal Health and Production, 16(2), 63-70.
- 3. 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 fur Angewandte Zoologie, 70(3), 277-83.
- Augustyn NJ., Bigalke RD. 1972. *Anaplasma* infection in a giraffe. Onderstepoort Journal of Veterinary Research, 39, 29.
- 5. Zaugg JL. 1987. Experimental infections of *Anaplasma ovis* in pronghorn antelope. Journal of Wildlife Research. 23(2), 205-10.

- 6. Kuttler KL. 1984. *Anaplasma* infections is wild and domestic ruminants: A review. Journal of Wildlife Diseases, 20(1), 12-20.
- 7. Potgieter FT. 1979. Epizootiology and control of anaplasmosis in South Africa. Journal of the South African Veterinary Medical Association, 50(4), 367-72.
- Wright IG., Leach G. 1996. Bovine anaplasmosis. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 295-304. OIE, Paris.
- 9. Jongejan E., Uilenberg G. 1994. Ticks and control methods. Revue Scientifique et Technique Office des Epizooties, 13(4), 1201-26.
- Norval RAJ. 1994. Vectors: Ticks. In: Infectious Diseases of Livestock with special reference to Southern Africa. Ed. Coetzer JAW., Thompson GR., Tustin RC. Vol 1, pp. 3-24. Oxford University Press, Cape Town, Oxford, New York.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.
- 12. OIE. International Animal Health Code. Pp 253-4. Office International des Epizooties. Paris 1997.

# 4.3 Other Rickettsial and Chlamydial infections

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

1. *Ehrlichia bovis*. This organism causes a disease syndrome resembling a mild form of heartwater with an incubation period of 3-6 weeks in cattle. Many animals are asymptomatically infected(1). It has not been described in antelope. 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. Vectors include *Hyalomma excavatum* in Iran, *Rhipicephalus appendiculatus* in southern Africa, *Amblyomma variegatum* in West Africa and *Amblyomma cajannense* in Brazil(1). The infection causes the development of antibodies that cross react with heartwater(1).

2. Cytoectes ondiri. Ondiri disease or bovine petechial fever in East Africa is caused by Cytoectes ondiri. The disease is characterised 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 and *Haemophysalis aciculifer* and *Haemaphysalis parmata*, two common ticks of bushbuck are though to be the probable vectors. The disease has not been described in zoo animals and the chances of its introduction are remote if antelope are imported from non-endemic areas and zoos. Bushbuck captured in the wild in East and central Africa should not be imported. The disease has been reviewed in references 1 and 2.

3. *Cytoectes phagocytophilia* causes tick-borne fever in Europe and is carried by *Ixodes ricinus*. The disease has an incubation period of 3-13 days and is characterised by high fever and a drop in milk production. Pregnant animals may abort and mummified foetuses may occur(1). No reference was found to the disease occurring in antelope. 4. 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(3). It is also believed that the organism can be transmitted transovarially in ticks(3). However, 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 animals that have aborted(4). 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(4). If the infection occurs in antelope, they are likely to be asymptomatic as no disease has been described. The probability of introducing the disease into New Zealand directly with antelope importations is, probably low. However, as New Zealand is one of the few countries that is free from the infection, great care should be taken not to introduce ticks with introduced antelope. Only serologically negative antelope should be introduced.

5. Chlamydial infection has been described in a springbok(5) and an outbreak of mortality associated with a chlamydial infection occurred in blackbuck and scimitar oryx, in a zoo in Georgia(6). 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 antelope. 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. Since it has apparently only been described in these two cases it is assumed that it is an uncommon infection. Standard risk reduction procedures including selection of animals from premises that have a history of freedom from disease and standard quarantine should be used to reduce the risk of introduction. Serology could be attempted

but interpretation of the results would be problematical unless clear-cut high titres were found. Since so little information is available concerning this disease no soundly based recommendations can be made for reducing the risks of introducing it. However, because the literature on the condition is so sparse it is probably a rare disease or an infection that only rarely causes a disease syndrome and the risks of introducing the disease are probably low. It is recommended that animals should be introduced without any specific testing for this disease.

- Scott GR. 1994. Lesser know Rickettsias infecting livestock. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 371-7. Oxford University Press, Cape Town, Oxford, New York.
- 2. Davies G. 1993. Bovine petechial fever (Ondiri disease). Veterinary Microbiology, 34, 103-121.
- Scott GR., Herr S. 1994. Q fever. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 390-5. Oxford University Press, Cape Town, Oxford, New York.
- Durand MP. 1996. In: Manual of Standards for Diagnositic tests and vaccines.
   Office International des Epizooties. Pp. 634-41. OIE, Paris.
- Van der Lugt JJ., Kriek JC. 1988. Chlamydiosis in a springbok (*Antidorcas marsupialis*). Journal of the South African Veterinary Medical Association, 59, 33-37.
- Mansell JL., Tang KN., Baldwin CA., Styler EL., Liggett AD. 1995. Disseminated chlamydial infection in antelope. Journal of Veterinary Diagnostic Investigation, 7(3), 397-9.

# 5.0 Bacterial infections of antelope

#### 5.1 Brucellosis

Brucellosis (*Brucella abortus*) is a serious zoonotic disease of cattle that has been eradicated from many developed countries including New Zealand.

**Etiological agent :** The disese in cattle is caused by *B. abortus*. References to *B. suis* and *B. melitensis* in antelope were not found but could potentially occur.

**Susceptible species:** *B. abortus* occurs predominantly in cattle. Antibody has been found in eland, wildebeest, impala, waterbuck and bushbuck(1); eland, impala and giraffe(2): and wildebeest(3) eland, oryx(4). Serological titres occur in buffalo(1)(2)(3) and the organism has also been isolated from buffalo(5).

**Incubation period:** In cattle abortions usually occur in the 5<sup>th</sup> to 9<sup>th</sup> months of pregnancy(6) The time between infection and abortion may vary from weeks to months depending on the stage of pregnancy when the animal is infected(7).

**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(7).

**Transmission:** Transmission occurs by contact with aborted foetuses, placentas and vaginal discharge. Transmission may occur orally or through any mucous membrane.

**Signs of disease:** In cattle, abortion is common and uterine infections and retained afterbirths occur commonly after abortions. In antelope species there is no evidence that the infection causes abortion and generally antelope appear to be well adapted to the infection(1).

**Epidemiology:** Antelope probably play little role in the dissemination of the disease to cattle and the infection appears to be asymptomatic in antelope. However, the infection

does spread to some extent in antelope as evidenced by positive serology in antelope species. It is therefore an infectious disease that could in the right circumstance spread to any susceptible animal. The organism has been isolated from buffalo(5).

**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(6).

**Implications:** The importation of serologically positive antelope should be avoided as the disease could potentially spread to other susceptible zoo animals and represent a possible source of infection for farm animals. Any source of possible infection in the country is unacceptable.

**Risk reduction:** Antelope should be imported from zoos and countries that are free from brucellosis. Only serologically negative antelope should be imported. They should be kept isolated and tested twice with an interval of not less than 30 days between tests, the second test being carried out within 15 days of shipment(8).

### **Recommendations:**

**Source of animals**: Animals should be sourced from disease-free countries and zones.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of the etiological agent.

Treatment: Treatment and vaccination are not recommended.

**Diagnostic tests:** Since zoo animals are not under the same control as domestic stock all introduced antelope, even from *Brucella* free countries, should be serologically tested with an OIE approved test (complement fixation test or ELISA). Serologically positive animals should not be released into New Zealand(9).

**Risk of introduction:** The risk of introduction is negligible if antelope are brought from safe sources and are serologically tested before importation.

- Bigalke RD. 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. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 152-166. Oxford University Press, Cape Town, Oxford, New York.
- 2. Madsen M., Anderson EC. 1995. Serologic survey of Zimbabwean wildlife for brucellosis. Journal of Zoo and Wildlife Medicine, 26(2), 240-5.
- Waghela S., Karstad L. 1986. Antibodies to *Brucella* spp. among blue wildebeest and African buffalo in Kenya. Journal of Wildlife Diseases, 22(2), 189-92.
- Paling RW., Waghela S., Macowan KJ., Heath BR. 1988. The occurrence of infectious diseases in mixed farming of domesticated wild herbivores and livestock in Kenya. Journal of Wildlife Diseases, 24(2), 308-16.
- Gradwell DV., Schutte AP., Van Niekerk CAWJ., Roux DJ. 1977. Isolation of Brucella abortus biotype 1 from African buffalo in the Kruger National Park. Journal of the South African Veterinary Medical Association, 48, 41-3.
- 6. Corbel MJ., MacMillan AP. 1996. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 242-55. OIE, Paris.
- Bishop GC., Bosman PP., Herr S. 1994. Bovine brucellosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 1. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 1053-66. Oxford University Press, Cape Town, Oxford, New York.
- OIE. International Animal Health Code. Pp 227-232. Office International des Epizooties. Paris 1997.
- Pharo H. 1999. Import risk analysis. Imported sero-positive animals: Assurance provided by serological tests. Ministry of Agriculture and Forestry. Wellington New Zealand.

# 5.2 Tuberculosis

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

**Etiological agent:** Bovine tuberculosis is caused by *Mycobacterium bovis* and is the usual cause of tuberculosis in antelope. Infection with *M. tuberculosis* is rare in animals apart from man, and *M. avium* causes lesions in deer but no reference was found to it causing disease in antelope.

**Susceptible species**: Cattle are the main species of domestic animals infected by *M*. *bovis* although the disease is also 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 kudu, duiker and lechwe(1). Recent publications have described tuberculosis in Arabian oryx(2)(3); blackbuck(4)(5); kudu(6); and lechwe, bushbuck(7). The problem in kudu in the Eastern Cape province of South Africa is a long-standing one which was described in 1940(8) and the disease has remained endemic in kudu in that area. Tuberculosis has become a serious problem of wild buffalo in the Kruger National park in South Africa in recent years and has spread to other species(9)(10). All antelope species should be regarded as potentially susceptible to *M. bovis* infection.

**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 often takes years(1).

Carrier state: Since it is a chronic disease, animals usually remain infected for life(1)

**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 eating contaminated material(1).

**Signs of 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 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 disease vary according to where the lesions are located and may include mastitis, enlarged lymph nodes and draining abscesses or sinuses(1)(11).

**Epidemiology:** The epidemiological picture is typical of a slowly spreading infectious disease.

**Diagnosis:** The intradermal tuberculin test is the standard diagnostic method for diagnosis of the disease in cattle. A diagnosis may also be made by demonstration of typical gross and histological lesions, demonstration of organisms in smears or tissue by Ziehl Neelsen staining or isolation of the organism. Polymerase chain reaction methods are now available. It has been suggested that the lymphocyte proliferation assay may be useful in wildlife and zoo animals(11).

**Implications:** New Zealand is endemically infected with bovine tuberculosis, in areas where possums and other wild or feral animals are infected. There is an active campaign to eradicate the disease and the introduction of new foci of infection is unacceptable. Introduction of infected animals could have serious consequences for zoos, because a wide variety of zoo animals could contract the disease, including other antelope, deer, Australian marsupials, cattle, goats, elephants etc. The possibility of transmission to the public would have to be considered. The disease could spread from zoos to possums in the vicinity and they could in turn infect domestic stock. Holding infected animals in zoos is unacceptable.

**Risk reduction:** Antelope should be imported from zoos that are have a history of freedom from infection. They should be subjected to tuberculin testing in a similar manner to deer, even although the tuberculin test has not been standardised and validated for antelope species. The OIE *International Animal Health Code* specifies that cattle should be tested twice with an interval of not less than 60 days, the last test being done within 30 days of shipment. The code also recommends that for wild bovines destined for zoos only one test is required(12) The Australian Quarantine and Inspection Service (AQIS) recommends that the interval between tests should be increased to 90 days to avoid problems with desensitisation. It is recommended that for antelope two tests, with an interval of 90 days, should be done while the animal is isolated from contact with other animals, unless they are of proven health status. Freedom of a country's livestock from tuberculosis would not necessarily guarantee

that zoo animals are free from infection since, they are not subjected to the same levels of control.

# **Recommendations:**

**Source of animals**: Animals should be sourced from disease-free countries and zones and zoos with a history of freedom from tuberculosis.

**Quarantine:** Quarantine measures would be ineffective in preventing the introduction of the etiological agent.

Treatment: Treatment and vaccination are not recommended.

**Diagnostic tests:** Since zoo animals are not under the same control as domestic stock, all introduced antelope, even from bovine tuberculosis free countries, should be tuberculin tested twice with an interval of 90 days, the last test being within 2 weeks of importation into New Zealand.

**Risk of introduction:** The risk of introduction of disease will be low provided animals are brought from disease-free sources and are tuberculin tested.

- Huchzermeyer HFKA., Bruckner GK., van Heerden A., Kleeberg HH., van Rensburg IBJ., Koen P., Loveday RK. 1994. Tuberculosis. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 2. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 1425-44. Oxford University Press, Cape Town, Oxford, New York.
- Rietkerk FE., Griffin FT., Wood B., Mubarak SM., Delima EC., Badri OM., Lindsay NB., Williamson DT. 1993. Treatment of bovine tuberculosis in an Arabian oryx (*Oryx leucoryx*). Journal of Zoo and Wildlife Medicine, 24(4), 523-7.
- Greth A., Flamand JRB., Delhomme A. 1994. An outbreak of tuberculosis in a captive herd of Arabian oryx (*Oryx leucoryx*); management. Veterinary Record, 134(7), 165-7.

- 4. Gupta PP., Singh SP. 1987. Tuberculosis in an Indian black buck. Journal of Research, Punjab Agricultural University, 24(2), 315-6.
- 5. Pathak DC., Rahman H., Upadhyaya TN., Baruah DK. 1987. Tuberculosis in black-bucks (*Antilope cervicapra*). Indian Medical Journal, 11(1), 52-3.
- 6. Weber A., van Hoven W., van Hoven W. 1992. Tuberculosis of the parotid salivary gland in a kudu, *Tragelaphus strepsiceros*. Koedoe, 35(1), 119-22.
- Zieger U., Pandey GS., Kriek NPJ., Cauldwell AE. 1998. Tuberculosis in Kafue lechwe (*Kobus leche kafuensis*) and in a bush buck (*Tragelaphus scriptus*) on a game ranch in Central Province, Zambia. Journal of the South African Veterinary Association, 69(3), 98-101.
- 8. Thorburn JH., Thomas AD. 1940. Tuberculosis in the Cape kudu. Journal of the South African Veterinary Medical Association, 11, 3-10.
- 9. Keet DF., Kriek NPJ., Huchzermeyer H., Bengis RG. 1994. Advanced tuberculosis is an African buffalo (*Syncerus caffer* Sparman). Journal of the South African Veterinary Association, 65(2), 70-83.
- Keet DF., Kriek NPJ., Penrith ML., Michel A., Huchzermeyer H. 1996. Tuberculosis in buffaloes (*Syncerus caffer*) in the Kruger National Park: spread of the disease to other species. Onderstepoort Journal of Veterinary Research, 63, 239-44.
- 11. Haagsma J. 1996. Bovine tuberculosis. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 267-75. OIE, Paris.
- 12. OIE. International Animal Health Code. Pp 235-40. Office International des Epizooties. Paris 1997.

# 5.3 Johne's disease (paratuberculosis)

Johne's disease is a serious disease of domestic animals with a world-wide distribution. It occurs in all species of domesticated ruminant livestock in New Zealand.

**Etiological agent:** The etiological 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(1).

**Susceptible species:** Cattle, sheep, goats, deer and camelids are susceptible. Antelope are susceptible, but the disease has only rarely been reported in antelope species. In an issue of the OIE publication *Revue Scientifique et Technique*(2), devoted entirely to wildlife husbandry and diseases there are no references to Johne's disease in antelope. On a ranch on which antibody was detected in camels and goats, no antibody was found in antelope(3). The disease has been described in a Jimela topi(4) and in a saiga antelope(5). 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.

**Incubation period:** The incubation period is generally from one to several years(6).

**Carrier state:** Animals 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:** Faeco-oral transmission is invariably the method of transmission(6).

**Signs of disease:** Loss of condition, chronic diarrhoea and wasting are the common signs of 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 intestinal mucosa or demonstration of the organism in Ziehl Neelsen

stained smears made from faeces or intestinal mucosa. Typical histological lesions can be demonstrated in infected animal tissues, particularly in the illeum, caecum and mesenteric lymph nodes. Serological methods including especially the absorbed ELISA are useful(6) but have the draw-back of having low sensitivity for detecting nonclinical infections and have not been verified in antelope. Delayed hypersensitivity tests are of limited value(6).

**Implications:** Zoos should avoid introduction of infected animals as once introduced the disease could spread insidiously in susceptible animals and be difficult to eradicate. The disease is a common endemic disease of livestock and MAF have no disease policy to control or eradicate the disease.

**Risk reduction:** Methods for detecting infected carrier animals cannot be relied upon to detect all carrier animals. No measures are presently imposed to prevent the importation of infected livestock into New Zealand. Therefore, no steps to prevent the importation of the organism in antelope are recommended. Steps to prevent the introduction of the organism to zoos should be the responsibility of the importers and zoos concerned.

**Risk of introduction:** The risk of introduction is low because the disease does not appear to be common in antelope.

- Collins DM., Gabric GW., De Lisle GW. 1990. Identification of two groups of *Mycobacterium paratuberculosis* strains by restriction endonuclease analysis and DNA hybridization. Journal of Clinical Microbiology, 28, 1591-6.
- 2. Revue Scientifique et Technique, 15(1), 1996.
- Paling RW., Waghela S., Macowan KJ., Heath BR. 1988. The occurrence of infectious diseases in mixed farming of domesticated wild herbivores and livestock in Kenya. Journal of Wildlife Diseases, 24(2), 308-16.
- Steinberg H. 1988. Johne's disease (Mycobacterium paratuberculosis) in a Jimela topi (Damaliscus lunatus jimela). Journal of Zoo Animal Medicine, 19(1-2), 33-41.

- 5. Dukes TW., Glover GJ., Brooks BW., Duncan JR. 1992. Paratuberculosis in a saiga antelope (*Saiga tatarica*) and experimental transmission to domestic sheep. Journal of Wildlife Diseases. 28(2),161-70.
- Thorel MF. 1994. Paratuberculosis (Johne's disease). In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 218-228. OIE, Paris.
- 7. OIE. International Animal Health Code. Pp 219. Office International des Epizooties. Paris 1997.

# 5.4 Anthrax

Anthrax is an zoonotic disease that is caused by an extremely resistant spore forming bacillus that can survive for many years in the environment. It is believed that New Zealand is free from the infection. An extensive review of the disease with emphasis on anthrax in wildlife is available(1)

Etiological agent: The disease agent is Bacillus anthracis.

**Susceptible species:** Anthrax is a disease of most animals. 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 sub-acute disease(2). Anthrax has been described in at least 17 antelope species(1)

Incubation period: The incubation period is believed to be 1-14 days(1).

**Carrier state:** The organism has been found in the faeces of some animals. Vultures may carry the organism in their faeces for 3 weeks. In experimentally infected impala, lymph nodes remained infected for at least 4 weeks(1).

**Transmission:** Transmission may be by ingestion, or mechanically transmitted by biting flies. In humans working in industries where the spores may become aerosolised (tanners, wool carpet mill workers etc), inhalation of the spores may cause respiratory anthrax(2). In humans skin infection in the form of malignant pustules also occurs(2).

**Signs of disease:** In ruminants the most common form of the disease results in sudden death. In horses, colic, high fever and 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. In this form of the disease the animal may survive for 7 days(2).

**Epidemiology:** Despite the fact that this is a truly ancient disease much remains to be found out about the epidemiology of the disease. In some circumstances the organism survives for many years in the soil. Multiplication of the organism apparently depends mainly on the multiplication that occurs in infected carcasses. 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. Fifty percent of faeces from vultures, jackals and hyaenas collected from the vicinity of carcasses that were believed to have died from anthrax yielded *Bacillus anthracis*, while no spores were found in faeces from sites not associated with anthrax carcasses(3)(4). 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 impala(5). De Vos raised some issues about a possible carrier state in impala, black rats, cattle and pigs but states that "It is unknown whether a carrier state plays a role in the epidemiology of the disease"(1).

It has been suggested that peracute disease was associated with exposure to low numbers of spores while living in a stressful environment rather than exposure to large numbers of spores(6).

**Diagnosis:** Blood smears from infected carcasses are still the main method of diagnosing the disease. A polymerase chain reaction 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(2).

**Implications:** Introduction of a case of anthrax would have serious implications. Apart from the death of the animal involved, the opening of an infected carcass could lead to the development of an infected environment and to the infection of other susceptible animals in the zoo. The zoo environment could remain a source of infection and be a potential source of spread to domestic animals for many years.

**Risk reduction:** Care should be exercised to import animals in clean crates and not to introduce soil and faeces and contaminated bedding with them. Anthrax can survive in the soil for many years and it has been shown to survive in bones for 200 years(1). On arrival all bedding and loose dirt should be incinerated and crates should be steam cleaned. The chances of introduction of spores on cleaned crates is remote and no further action is generally warranted. If animals have died in a crate or arrived showing symptoms suspicious of one of the less acute forms of anthrax, wooden crates should be incinerated and metal crates burned clean with a blow torch. Carcasses must be incinerated without opening them, but blood smears could be made and examined. In environments where anthrax infected carcasses have lain, soil and water has been

shown to be contaminated and spores can survive in such environments for years(3)(4). Animals from such environments may carry spores in faeces and lymph nodes for at least 4 weeks(5). Therefore, a conservative approach should be taken and antelope should be removed from infected environments at least 3 months before being introduced into quarantine. Quarantine should be for 4 weeks. Recommendations in the OIE *International Animal Health Code* are that domestic ruminants should be free from clinical disease, kept for 20 days before shipment in an establishment where no case of anthrax occurred during the time of confinement and vaccinated at least 20 days and not more than 6 months before shipment(6). Information on the use of vaccine in antelope was not found. However, the vaccine is safe in a wide variety of animals and it is recommended that antelope should be vaccinated with Sterne strain, live vaccine. This strain has been extensively used for about 60 years in many millions of animals and has never reverted to virulence(2).

#### **Recommendations:**

**Source of animals**: Animals should be held in an environment (farm, zoo, holding pen etc) where the disease has not occurred for the last 5 years, for 3 months before entering quarantine.

Quarantine: Standard 4 week quarantine would be adequate.

**Treatment:** Animals should be vaccinated at least 3 weeks and not more than 6 months before entering quarantine.

Diagnostic tests: No diagnostic testing is recommended.

**Risk of introduction:** The probability of introducing the disease is remote if they are vaccinated and quarantined as recommended.

### References

 De Vos V. 1994. Anthrax. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 2. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 1262-89. Oxford University Press, Cape Town, Oxford, New York.

- 2. Whitford HW. 1996. Anthrax. In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 170-80. OIE, Paris.
- Turnbull PCB., Carman JA., Lindeque PM., Joubert F. Hubschle OJB., Snoeyenbos GH. 1989. Further progress in understanding anthrax in the Etosha National Park. Madoqua, 16(2), 93-104.
- Lindeque PM., Turnbull PCB. 1994. Ecology and epidemiology of anthrax in the Etosha National Park, Namibia. Onderstepoort Journal of Veterinary Research, 61(1), 71-83.
- De Vos 1990. The ecology of anthrax in the Kruger National Park, South Africa. Salisbury Medical Bulletin, 68 (special supplement), 19-23. As reported by De Vos V. 1994. Anthrax. In: Infectious Diseases of Livestock with special reference to Southern Africa. Vol. 2. Ed. Coetzer JAW., Thompson GR., Tustin RC. Pp. 1262-89. Oxford University Press, Cape Town, Oxford, New York.
- 6. Gainer RS. 1987. Epizootiology of anthrax and Nyasa wildebeest in the Selous Game Reserve, Tanzania. Journal of Wildlife Diseases, 23(1), 175-8.
- 7. OIE. International Animal Health Code. Pp 207-8. Office International des Epizooties. Paris 1997.

# 5.5 Other bacterial infections

There are a range of bacterial infections that occur widely all over the world and often do not cause clearly defined disease entities, or are of only minor importance. They sometimes occur in animals as opportunistic infections. These infections are generally of little importance in considering the importation of animals. Some publications on these organisms are listed in Section 5.5.1. Section 5.5.2 includes bacterial diseases for which no evidence of infection in antelope was found but which might be of concern for regulatory authorities when drafting regulations for importation of antelope.

#### 5.5.1 Miscellaneous bacterial and fungal infections

*Yersinia pseudotuberculosis* is an enteric pathogen that causes micro-abscessation of intestinal mucosa in a wide range of ungulates including antelope(1)(2). The organism is also commonly found in non-diseased animals and is very widely distributed in New Zealand. *Yersinia pseudotuberculosis* may also infect man.

*Clostridium perfringens* was associated with an outbreak of haemorrhagic enteritis in crowned duikers(3).

Serological evidence of *Leptospira* infection was found in free-living antelope in Zimbabwe(4). Antelope will undoubtedly be susceptible to infection with *Leptospira* spp. but the lack of reports on the disease indicates that it does not appear to cause serious disease in antelope. Which of the hundreds of serovars of *Leptospira* cause disease in antelope or are associated with them as maintenance hosts, appears to be unknown. Under these circumstances it is recommended that imported antelope should be treated using the antibiotics and treatment regimens currently specified by MAF for other animals.

Listeriosis has been described in an immature blackbuck(5). However, *Listeria* are organisms that occur in the environment all over the world including New Zealand and are opportunistic pathogens. For these reasons, disease should be of no interest to those concerned with regulating the importation of antelope.

Antibody to *Legionella* was detected in the serum of 5 of 23 antelope in Israel(6). Legionella are opportunistic pathogens of humans that occur in the environment,

particularly water, in many countries including New Zealand and are therefore of no importance in the present context.

# References

- 1. Slee KJ., Button C. 1990. Enteritis in sheep, goats, and pigs due to *Yersinia pseudotuberculosis* infection. Australian Veterinary Journal, 67(9). 320-2.
- 2. Welsh RD., Ely RW., Holland RJ. 1992. Epizpootic of Yersinia pseudotuberculosis in a wildlife park. Journal of the American Veterinary Medical Association, 201(1), 142-8.
- Olaleye OD., Oyejide A. 1985. An outbreak of haemorrhagic enterotoxaemia due to *Clostridium perfringens* in crowned duikers (*Sylvicapra grimmia*). Zoologische Garten, 55(5-6), 298-300.
- Anderson EC., Rowe LW. 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(2), 441-9.
- 5. Webb DM., Rebar AH. 1987. Listeriosis in an immature blackbuck antelope (*Antilope cervicapra*). Journal of Wildlife Diseases, 23(2), 318-20.
- 6. Boldur I., Cohen A., Tamarin LR., Sompolinsky D. 1986. Legionellosis in animals in Israel. Veterinary Microbiology, 13(4), 313-20.

#### 5.5.2 Bacterial diseases that have not been described in antelope

*Mycoplasma mycoides* (contagious bovine pleuropneumonia) antibodies were not found in antelope(1). Antibody to *Mycoplasma* sp. (strain F38), now known as *Mycoplasma capricolum* subsp. *capripneumoniae*, the cause of caprine pleuropneumonia, were not found in antelope but were found in buffalo(1).

Despite a electronic search no articles were found describing haemorrhagic septicaemia (*Pasteurella multocida* types B2 or E2) in antelope.

#### References

 Paling RW., Waghela S., Macowan KJ., Heath BR. 1988. The occurrence of infectious diseases in mixed farming of domestic wild herbivores and livestock in Kenya. II. Bacterial diseases. Journal of Wildlife

Diseases, 24(2), 308-16.

# 6.0 Parasites of antelope

Most animals have developed living in balance with a range of parasites. The parasites usually cause little harm to adapted hosts except when they are living in overcrowded, unhygienic conditions resulting in exposure to an excessive number of parasites. Some parasites are host specific, others may have a broad host range. When hosts are exposed to some unusual parasites, with which they did not evolve, they may have no inherited tolerance or resistance to the parasite. In this case they may be extremely susceptible or totally refractory to infestation by the parasite.

The numbers of parasites that are carried by antelope are very large and cannot be described in detail in this paper. Some of the extensive work done on the helminth parasites of Southern African antelope species is listed in references 1-15. This extensive listing only covers the South African parasites and there are undoubtedly many of them still undescribed. There are also certainly many parasites from other antelope species and other geographic regions that are not included in these sources and some parasites still awaiting description.

Some nematodes are specific parasites of particular species of animals. However, even some of the parasites that have a wide host range tend to become adapted to a particular host. It was shown that even *Haemonchus placei, Trichostrongylus colubriformis, Trichostrongylus falculatus* and *Oesophagostomum columbianum* regarded as normal parasites of cattle and sheep were not easily transmissible from antelope to cattle, sheep and goats. Some parasites were successfully transmitted from antelope to domestic ruminants but at least one parasite, *Cooperioides hepaticae,* could not be transferred from antelope to domestic ruminants(16). It was also suggested that there was very little cross-transmission between cattle and impala utilising the same grazing(16). It therefore, seems that introduction of antelope infested with internal parasites would pose a bigger threat to similar species in zoos than to domestic livestock.

There is no reasonable way in which regulations could be drawn up to guard against the introduction of all species of internal parasites. For this reason the only rational strategy is to dose introduced antelope before and during quarantine while monitoring their faeces to try to eliminate all internal parasites. Antelope should be dosed before entry into quarantine and again while in quarantine, with anthelminthics and doses approved by MAF. Proof of the efficacy of the dosing should be provided by doing faecal egg counts before and after treatment. While in quarantine animals should be kept in hygienic conditions and accumulation of faeces should be prevented. All bedding should be regularly removed and concrete floors should be regularly hosed and cleaned.

References 17-28 are a selection of papers on the ectoparasites of antelope. However, many other publications are available and there is no complete check list of hosts and parasites. The principles for the control of ectoparasites are the same as for endoparasites. All animals should be carefully inspected for external parasites or lesions such as mange or warble fly nodules. They should be appropriately dipped or sprayed to eliminate external parasites. While ticks are the most important parasites, others should also be eliminated. Treatments for tick control have been described in Section 1.1.5. This type of treatment will be adequate for the treatment of other ectoparasites.

Some non-tick ectoparasites parasites deserve special mention. These include:

**Mange mites**. *Psoroptes cuniculi* infestation has been seen in blackbuck antelope on a ranch in Texas(29). *Demodex* sp. infestation has been found in eland in a zoo in Czechoslovakia(30). Mange in domestic and wild ruminant has been described in Israel(31). *Psoroptes* and *Sarcoptes* spp. are well known in various antelope species(32)

**Warbles**. Warble fly nodules caused by larvae of *Strobiloestrus* sp. may be found in the skin of reedbuck, kudu and especially klipspringer in South Africa. These parasites are also able to transfer to cattle and cases have been found in cattle in areas where there are many klipspringer in close proximity to cattle(33). The infestation has never become established in cattle populations where there is no contact with susceptible antelope. Cattle are clearly incidental hosts that are ony infested when there is contact with susceptible antelope. The parasites have also been found in sheep but the cysts were sterile(34). Although this is only an isolated problem in South African cattle it would be unwise and unnecessary to introduce the parasites to New Zealand zoos where they could infect antelope in the zoo and might occasionally spread to cattle. All antelope should be inspected while in quarantine and if warble nodules are found they should be treated with a suitable pour on insecticide such as famphur to eliminate the fly larvae.

**Gedoelstiasis**. The normal hosts of *Gedoelstia hassleri* nasal flies are antelope of the Alcelaphinae sub-family, including wildebeest and red hartebeest. They are well adapted to the parasite. The adult fly deposits first stage larvae in the eye of the host. The larvae migrate via the bloodstream to ultimately reach the nasal and paranasal cavities. The host species are well adapted to the parasite and show no apparent ill effects. However, when the parasite enters an aberrant species such as domestic stock significant pathology may result with pronounced ocular, brain and cardiovascular lesions. In the condition know as uitpeuloog (pop-out eye) masses of parasites accumulate behind the eye and cause it to protrude(35)(36)(37). In Africa during centuries of contact between Alcelaphine antelope the parasite has not established itself in domestic stock and only incidental infestations occur when there is contact with the natural hosts. Therefore, this parasite could not establish in New Zealand in the absence of the natural host.

**Screw worm.** The new world screw worm is *Cochliomyia hominivorax* and the old world screw worm is *Chrysomya bezziana*. These flies lay their eggs in wounds where the larvae develop. The parasites digest host tissues causing rapidly developing putrid cavities to develop. They cause significant damage to livestock in tropical climates. The parasites do not tolerate cold and are not found in temperate climates(38). Larvae leave the host within about a week. The wounds infested by parasites rapidly develop into large putrid wounds that would be easily visible. There is virtually no risk of introducing these flies into New Zealand or of them establishing here.

### References

- Boomker J, Horak IG, Gibbons LM, De Vos V. 1983. Haemonchus contortus from the vaal ribbok, Pelea capreolus, and the bontebok, Damaliscus dorcas dorcas, in the Bontebok National Park. Onderstepoort Journal of Veterinary Research, 50(3),179 -81.
- Boomker J, Horak IG, De Vos V. 1986. The helminth parasites of various artiodactylids from some South African nature reserves. Onderstepoort Journal of Veterinary Research, 53(2), 93-102.
- 3. Boomker J, Keep ME, Horak IG. 1987. Parasites of South African wildlife. I. Helminths of bushbuck, *Tragelaphus scriptus*, and grey duiker, *Sylvicapra*

*grimmia*, from the Weza State Forest, Natal. Onderstepoort Journal of Veterinary Research, 54(2),131-4.

- Boomker J, Horak IG, MacIvor KM. 1989. Helminth parasites of grysbok, common duikers and Angora and Boer goats in the Valley Bushveld in the eastern Cape Province. Onderstepoort Journal of Veterinary Research, 56(3),165-72.
- Boomker J, Horak IG, Knight MM. 1991. Parasites of South African wildlife.
   IX. Helminths of kudu, *Tragelaphus strepsiceros*, in the eastern Cape Province. Onderstepoort Journal of Veterinary Research, 58(3), 203-4.
- Boomker J, Horak IG, Flamand JR. 1991. Parasites of South African wildlife.
   X. Helminths of red duikers, *Cephalophus natalensis*, in Natal. Onderstepoort Journal of Veterinary Research, 58(3), 205-9.
- Boomker J, Horak IG. 1992. Parasites of South African wildlife. XIII. Helminths of grey rhebuck, *Pelea capreolus*, and of bontebok, *Damaliscus dorcas dorcas*, in the Bontebok National Park. Onderstepoort Journal of Veterinary Research, 59(3),175-82.
- Boomker J, Booyse DG, Watermeyer R, De Villiers IL, Horak IG, Flamand JR. 1996.
   Parasites of South African wildlife. XIV. Helminths of nyalas (*Tragelaphus angasii*) in the Mkuzi Game Reserve, KwaZulu-Natal. Onderstepoort Journal of Veterinary Research, 63(4), 265-71.
- Horak IG. 1978. Parasites of domestic and wild animals in South Africa. X. Helminths in impala. Onderstepoort Journal of Veterinary Research, 45(4), ,221-8.
- Horak IG. 1981. The seasonal incidence of the major nematode genera recovered from sheep, cattle, impala and blesbok in the Transvaal. Journal of the South African Veterinary Association, 52(3), 213-23.

- Horak IG, Meltzer DG, De Vos V. 1982. Helminth and arthropod parasites of springbok, *Antidorcas marsupialis*, in the Transvaal and Western Cape Province. Onderstepoort Journal of Veterinary Research, 49(1), 7-10.
- Horak IG, Brown MR, Boomker J, De Vos V, Van Zyl EA. 1982. Helminth and arthropod parasites of blesbok, *Damaliscus dorcas phillipsi*, and of bontebok, *Damaliscus dorcas dorcas*. Onderstepoort Journal of Veterinary Research, 49(3), 139-46.
- Horak IG, De Vos V, De Klerk BD. 1982 Helminth and arthropod parasites of vaal ribbok, *Pelea capreolus*, in the western Cape Province. Onderstepoort Journal of Veterinary Research, 49(3),147-8.
- Horak IG, Boomker J, Kingsley SA, De Vos V. 1983. The efficacy of ivermectin against helminth and arthropod parasites of impala. Journal of the South African Veterinary Association, 54(4), 251-3.
- 15. Pletcher JM, Horak IG, de Vos V, Boomker J. 1984. Nodular abomasitis in impala (*Aepyceros melampus*) caused by the nematode *Longistrongylus sabie*. J Parasitol, 70(6), 907-10.
- Horak IG. 1979. Parasites of domestic and wild animals in South Africa. XII. Artificial transmission of nematodes from blesbok and impala to sheep, goats and cattle. Onderstepoort Journal of Veterinary Research, 46, 27-30.
- Horak IG, Boomker J, De Vos. 1980. A description of the immature stages of *Kirkioestrus minutus* (Rodhain; Bequaert, 1915) (Diptera, Oestridae), and the life cycle and seasonal prevalence of this fly in blue wildebeest.
  Onderstepoort Journal of Veterinary Research, 47(1), 23-30.
- Horak IG, Meltzer DG, De Vos V. 1982. Helminth and arthropod parasites of springbok, *Antidorcas marsupialis*, in the Transvaal and Western Cape Province. Onderstepoort Journal of Veterinary Research, 49(1), 7-10.
- 19. Horak IG, Brown MR, Boomker J, De Vos V, Van Zyl EA. 1982. Helminth and arthropod parasites of blesbok, *Damaliscus dorcas phillipsi*, and of bontebok,

*Damaliscus dorcas dorcas*. Onderstepoort Journal of Veterinary Research, 49(3), 139-46.

- 20. Horak IG, Potgieter FT, Walker JB, De Vos V, Boomker J. 1983. The ixodid tick burdens of various large ruminant species in South African nature reserves. Onderstepoort Journal of Veterinary Research, 50(3), 221-8.
- 21. Horak IG, Keep ME, Flamand JR, Boomker J. 1988. Arthropod parasites of common reedbuck, *Redunca arundinum*, in Natal. Onderstepoort Journal of Veterinary Research, 55(1),19 -22.
- 22. Horak IG, Sheppey K, Knight MM, Beuthin CL. 1986. Parasites of domestic and wild animals in South Africa. XXI. Arthropod parasites of vaal ribbok, bontebok and scrub hares in the western Cape Province. Onderstepoort Journal of Veterinary Research, 53(4), 187-97.
- 23. Horak IG, Keep ME, Spickett AM, Boomker J. 1989. Parasites of domestic and wild animals in South Africa. XXIV. Arthropod parasites of bushbuck and common duiker in the Weza State Forest, Natal. Onderstepoort Journal of Veterinary Research, 56(1), 63-6.
- 24. Horak IG, Boomker J, Flamand. 1991. Ixodid ticks and lice infesting red duikers and bushpigs in north-eastern Natal. Onderstepoort Journal of Veterinary Research, 58(4), 281-4.
- 25. Horak IG, Anthonissen M, Krecek RC, Boomker J. 1992. Arthropod parasites of springbok, gemsbok, kudus, giraffes and Burchell's and Hartmann's zebras in the Etosha and Hardap Nature Reserves, Namibia. Onderstepoort Journal of Veterinary Research 59(4), 253-7.
- Horak IG, Boomker J, Spickett AM, De Vos.1992. Parasites of domestic and wild animals in South Africa. Ectoparasites of kudus in the eastern Transvaal Lowveld and the eastern Cape Province. Onderstepoort Journal of Veterinary Research, 59(4), 259-73.
- 27. Horak IG, Boomker J, Flamand JR. 1995. Parasites of domestic and wild animals in South Africa. XXXIV. Arthropod parasites of nyalas in north-

eastern KwaZulu-Natal. Onderstepoort Journal of Veterinary Research, 62(3), 171-9.

- Horak IG, Boomker J. 1998. Parasites of domestic and wild animals in South Africa. XXXV. Ixodid ticks and bot fly larvae in the Bontebok National Park.
   Onderstepoort Journal of Veterinary Research, 65(3), 205-11.
- 29. Wright FC., Glaze RL. 1988. Blackbuck antelope (*Antilope cervicapra*), a new host for *Psoroptes cuniculi* (Acari:Psoroptidae). Journal of Wildlife Diseases, 24(1), 168-9.
- Bukva V., Vitovec J., Moucha P., Vahala J. 1988. Pathological process induced by *Demodex* sp. (Acari: Demodicidae) in the skin of eland, *Taurotragus oryx* (Pallas). Folia Parasitologica, 35(1), 87-91.
- 31. Yeruham I., Rosen S., Hadani A. 1986. Unusual cases of mange in domestic and wild ruminants in Israel. Israel Journal of Veterinary Medicine, 41(1), 49-50.
- Basson PA., McCully RM., Kruger SP., Van Niekerk JW., Young E., De Vos V., Keep ME., Ebedes H. 1971. Disease conditions of game in southern Africa: Recent miscellaneous findings. Veterinary Medical Review, 2/3, 313-40.
- 33. Horak IG., Boomker J. 1981. *Strobiloestrus* sp. larvae in cattle. Journal of the South African Veterinary Association, 52(3), 211-2.
- Brain V, van der Merwe HE, Horak IG. 1983. *Strobiloestrus* sp. larvae in Merino sheep. Journal of the South African Veterinary Association, 54(3),185-6.
- Basson PA. 1966. Gedoelstial myiasis in antelopes of Southern Africa.
   Onderstepoort Journal of Veterinary Research, 33(1), 77-92.
- Basson PA. 1962. Studies on specific ocular myiasis of domestic animals (Uitpeuloog). II. Experimental transmission. Onderstepoort Journal of Veterinary Research, 29, 203-210

- Basson PA. 1962. Studies on specific ocular myiasis of domestic animals (Uitpeuloog). III. Symptomatology, pathology, aetiology and epizootiology. Onderstepoort Journal of Veterinary Research, 29, 211.
- Hall MJR. 1996. Screworm (*Cochliomyia hominivorax*). In: Manual of Standards for Diagnositic tests and vaccines. Office International des Epizooties. Pp. 235-41. OIE, Paris.

# 7.0 Transmissible bovine spongiform encephalopathy

Bovine spongiform encephalopathy (BSE) has been seen in a number of antelope species in British zoos(1)(2)(3). The disease is caused by the feeding of ruminant protein from "infected animals". As the disease epizootic in Britain is now declining and the feeding of ruminant protein is now banned in virtually all countries that have had cases of BSE, the chance of introducing the disease to New Zealand is low. However, the finding of three cases in kudu that were believed to have not been fed ruminant protein, has led to the suggestion that the disease may have been transmitted by other than a food-borne infection. Care should be taken not to introduce antelope from any zoos where the disease has occurred. The incubation period is very long (several years) and there are no suitable approved diagnostic tests that can be used on live animals at this stage, although tests are in the development and validation stage. Therefore, the only method of avoiding the importation of "infected" animals is the careful selection of animals from safe sources. The disease is not generally infectious and would be unlikely to spread if introduced.

#### References

- 1. Cunningham AA. 1991 Bovine spongiform encephalopathy in British zoos. Journal of Zoo and Wildlife Medicine, 22(3), 303-17.
- Kirkwood JK., Wells GAH., Cunningham AA., Jackson SI., Scott AC., Dawson M., Wilesmith JW. 1992. Scrapie-like encephalopathy in a greater kudu (*Tragelapus strepsiceros*) which had not been fed ruminant derived protein. Veterinary Record, 130(17), 365-7.
- 3. Cunningham AA., Wells GAH., Scott AC., Kirkwood JK., Barnett JEF. 1993. Transmissible spongiform encephalopathy in greater kudu (*Tragelapus strepsiceros*). Veterinary Record, 132(3), 68.

# 8.0 Artificial insemination and embryo transfer technology

In domestic animals, an alternative to the introduction of live animals is the introduction of semen or embryos.

# 8.1 Artificial insemination

Where female breeding stock is already available in New Zealand zoos, the use of artificial insemination for the introduction of genetic material might be cheaper than importing live animals. It would also decrease the possibility of introducing diseases and parasites. However, no published information on the use of the technology in antelope species was found. For this reason extrapolation must be made from what is known about artificial insemination in cattle. The review, *Review of quarantine disease risks related to bovine semen* produced by the Australian Quarantine and Inspection Service (AQIS) provides much information on the subject(1)

Semen can become contaminated with infectious organisms during the process of collecting the semen (extrinsic contamination). For example when semen is collected from animals excreting infectious organisms such as *Mycobacterium paratuberculosis* in their faeces, semen can become contaminated with the infectious agent. If semen is collected in a clean hygienic manner and is diluted correctly with the recommended diluents and antibiotic additives as recommended in Appendix 4.2.1.2 of the OIE *International Animal Health Code*(2), the risk of extrinsic contamination will be reduced to minimal levels.

Intrinsic contamination of semen occurs when animals are infected with a particular disease. Although there is a barrier between blood and testes, similar to the blood brain barrier, this barrier can, under some circumstances, be breached at the level of the testes or accessory glands. It is unnecessary to speculate on the mechanisms by which organisms enter the semen, but as a principle it should be assumed that any animal that is viraemic or bacteraemic may potentially excrete organisms in the semen. This is particularly so when inflammatory cells are found in semen.

Adams(1) has used five categories to classify the risk of various diseases being transmitted in semen:

**Category 1** includes diseases that have been transmitted experimentally with semen or in which there is conclusive epidemiological evidence that transmission by semen occurs.

**Category 2** includes diseases in which the etiological agents have been isolated in semen but transmission by artificial insemination has not been demonstrated.

**Category 3**: contains diseases in which there is a presumptive risk, but the presence of organisms in the semen has not been demonstrated. This category is mainly founded on the assumption that if inflammatory cells can enter the semen the infectious agents could be transported with them.

**Category 4**. Diseases where there is no presumptive risk of agent transfer into the semen but where extrinsic transmission cannot be ruled out if the animal is suffering from the disease, are included in this category.

**Category 5** Contains those diseases where the risk of disease transfer is inconceivable.

The classification of the diseases mentioned in this review that were classified into the various categories by Adams(1) are as follows:

**Category 1**: Foot and mouth disease, bluetongue, leptospirosis, bovine brucellosis, bovine tuberculosis, bovine virus diarrhoea, infectious bovine rhinotracheitis.

**Category 2**: Rinderpest, Lumpy skin disease, Johne's disease, enzootic bovine leukosis, bovine malignant catarrh, haemorrhagic septicaemia.

**Category** 3: Rift Valley fever, heartwater, Q fever, rabies, theileriasis, trypanosomosis, anaplasmosis, babesiosis.

Category 4: Vesicular stomatitis, anthrax.

**Category 5**: Ecto and endo parasites and protozoal diseases like coccidiosis would fall into this category.

Other publications on the risk of transmission of diseases through artificial insemination(2)(3) are substantially in agreement with these findings. The only disagreements between the publications are that Eaglesome and Garcia(2) classify bluetongue as a disease in which there is some evidence that the risk of transmission is low and vesicular stomatitis as a disease in which the risk is moderate to high. Classification of other disease by Eaglesome and Garcia are essentially similar to those of Adams(1). The article by Philpot(3) is several years older and is updated by the other publications. Where there are disagreements between the publications 1 and 2, the opinion that classifies the disease into the higher risk category should be used.

An additional advantage with using artificial insemination is that all the normal precautions to ensure that a semen donor is not infected with any diseases can be taken before and after the semen is collected. Semen donors can be quarantined in insect free premises and semen can be collected during seasons when vectors are not active. A prescribed battery of tests can be done on the animal at the time of or before semen collection. The semen can then be frozen and stored pending additional tests to show that the animal has not sero-converted since semen collection, and that the semen contains no infectious organisms.

In practice if the donor animal is not infected with a disease and not in contact with infected animals, the disease cannot be transmitted and the risk is virtually nil. It can also be assumed that all endo and ectoparasites are in Category 5 and of no risk. Importation of semen instead of live animals would therefore exclude the possibility of introducing ecto and endoparasites and most importantly, exclusion of ticks would be guaranteed.

For the important high risk diseases that are in Category 1 other methods of reducing the risk of introduction of the diseases are most important. However, the use of artificial insemination adds another significant layer of security to introductions of genetic material.. This is particularly true for parasitic diseases. It also probably guarantees a high degree of biosecurity for all diseases classified as Category 3 or higher and even possibly for Category 2 diseases. It would also give additional security in cases where knowledge of particular diseases is inadequate to formulate risk reduction steps with a high degree of confidence. As there are significant gaps in our knowledge concerning some diseases of antelopes, the use of artificial insemination would be useful as an additional barrier to

disease entry. In general, since there is no information on the transmission of diseases in the semen of antelope the information available for cattle should be used.

Semen should be:

- Collected only from healthy antelope after conducting recommended tests.
- Collected, diluted and frozen according to OIE specified procedures.
- Stored frozen until such time as it has been shown to be safe before using it to inseminate animals in New Zealand.

# 8.2 Embryo transfer

- The advantages of using embryo transfer to provide a high degree of biosecurity are similar to those for artificial insemination. The risks of transfer of disease by embryo transfer in cattle were assessed by Sutmoller and Wrathall(4). They used a model that calculates the risk as the product of the risk involved in the three basic lines of defence namely:
- The risk of not detecting infection in the donor animals before embryo collection.
- The risk of not removing the infection by the steps involved in the processing of the embryos (washing etc).
- The risk involved in not detecting the disease in donor herds while the embryos are held in storage before transfer.

The most likely probability that a batch of 200 embryos would contain infected embryos was 1 in 1,000 billion for foot and mouth disease, 1 in 4 million for bluetongue and 1 in 500 million for vesicular stomatitis. Although the theory on which quantitative risk analysis is based is sound, estimates made using the theory may be flawed. This occurs due to the necessity when making the calculations, to use estimates and opinions that are not based on sound data. However, in this case the estimated risk is so low that even if some of the data used was inaccurate by several degrees of magnitude, the results still clearly indicate the high degree of safety likely to be achieved if embryo transfer methods are used.

The International Embryo Transfer Society has classified diseases into the following categories(5):

**Category 1**: Diseases for which sufficient evidence has accrued to show that the risk of transmission is negligible if the embryos are properly handled between collection and transfer.

**Category 2**: Disease for which substantial evidence has accrued to show the risk of transmission is negligible provided the embryos are properly handled between collection and transfer.

**Category 3**: Diseases for which preliminary evidence indicates that the risk is negligible.

**Category 4**: Diseases in which preliminary work has been conducted or is in progress.

The categories into which the diseases that have been reviewed in this paper are classified are given below.

**Category 1**: Bluetongue(cattle), *Brucella abortus*(cattle), enzootic bovine leucosis, foot and mouth disease(cattle), infectious bovine rhinotracheitis (provided trypsin treatment is used).

Category 2: Bluetongue(sheep)

**Category 3**: Bovine viral diarrhoea, foot and mouth disease (swine, sheep, goats), rinderpest (cattle).

**Category 4**: Akabane, bovine anaplasmosis, bluetongue (goats), border disease (sheep), *Chlamydia psittaci* (cattle, sheep), vesicular stomatitis.

Clearly in cattle, the safety of importing genetic material is greatly increased if embryo transfer is used. At a minimum it adds another layer of protection to the other risk reduction methods used. Although no data is available for antelope, similar benefits are likely to apply in all species and data obtained in cattle should be used as a guideline for decision making about antelope.

# 8.3 Feasibility of using artificial insemination or embryo transfer in antelope

The possibility of using artificial insemination and embryo transplantation in antelope was not extensively investigated as part of this review. However, Professor Henk Bertschinger, of the Department of Genesiology at the Faculty of Veterinary Science, Onderstepoort, South Africa was consulted. He has worked extensively with reproductive problems in South African wildlife. He said that he was not aware of any published literature on the subject. However, he was aware that insemination of an eland had been successfully carried out at the Johannesburg zoo. Semen had been collected and successfully frozen but had not been used. Similarly embryos had been recovered from antelope but had not been transplanted. There appeared to be no barrier to he development and use of the technology but the handling of animals that are not tame is a practical problem. The question of whether the techniques can be developed and used in antelope is a problem that needs to be investigated by the zoos involved in the importation of embryos. A move to do this, would almost certainly be welcomed by MAF, but it would have to be resourced by the organisations wishing to import antelope.

#### References

- 1. Adams DB. Review of quarantine disease risks related to bovine semen. A report for the Australian Quarantine and Inspection Service. AQIS, Canberra.
- Eaglesome MD., Garcia MM. 1997. Disease risks to animal health from artificial insemination with bovine semen. OIE Revue Scientifique et Epizootique, OIE, 16(1) 215-25.
- 3. Philpott M. 1993. The dangers of disease transmission by artificial insemination and embryo transfer. British Veterinary Journal, 149, 339-63.
- 4. Sutmoller P., Wrathall AE. 1997 The risks of disease transfer by embryo transfer in cattle. Revue Scientifique et Epizootique, OIE, 16(1), 226-39.
- Conclusions of the Research Subcommittee of the International Embryo Transfer Society (IETS) Import/Export Committee, 1998. Revue Scientifique et Epizootique, OIE, 17(3), 839.

# 9.0 Risk of introduction of disease

In the above review 44 viral, protozoal, rickettsial and bacterial diseases that occur in antelope and which could be imported into New Zealand have been discussed. In addition to this, a long list of parasites of antelope could be introduced by live antelope.

Six of the diseases that have been reviewed are OIE List A diseases. Vesicular stomatitis is a List A disease because of the resemblance of its clinical signs of disease to FMD. It does not cause serious, direct economic losses to farmers, but regulatory authorities incur substantial expenses because of the control, management and reporting requirements for a List A disease. Because it is unlikely to cause serious economic losses, is unlikely be introduced into New Zealand and competent vectors are probably not present here, it has only been regarded as being of minor importance in this review. Of the five remaining List A diseases, vectors are not present for bluetongue and the possibility that Rift Valley fever and lumpy skin could establish are doubtful. Foot and mouth disease and rinderpest are the two most serious diseases that could be introduced into New Zealand, but antelope could be introduced from countries where these diseases do not occur. Other risk reduction procedures are also available to ensure that the diseases are not introduced. In particular the more detailed knowledge of the List A diseases, and the well-developed test methods available for them, provides additional reasons for confidence that they can be excluded. Some other diseases and tick vectors represent a higher risk.

An attempt has been made in this paper to discuss the risk of introduction of diseases and the methods to reduce these risks for:

- Ticks.
- The OIE List A diseases.
- Other diseases of economic importance.

An attempt has also been made to identify the parties that would be affected by the introduction of diseases or vectors.

### 9.1 Ticks

#### 9.1.1 Probability and consequences of introduction of ticks

When antelope are imported, the concomitant introduction of ticks is of the greatest concern. At least 11 of the 44 diseases that antelope can carry and many diseases of other animals are tick-borne diseases.

Preventing the introduction of ticks is difficult, and between 1980 and 1995 there were 40 recorded instances when ticks were introduced into New Zealand(1). Fortunately in most incidents a single or a few ticks were introduced and these were quickly discovered and eliminated. However, in one instance a population of *Rhipicephalus sanguineus* ticks became established in a house in Lower Hutt. Although the ticks were eradicated, the incident shows that in a protected environment some species of ticks can become established even as far south as Lower Hutt. Within the cages and animal accommodation in zoos it is possible that some niches, suitable for survival of some tick species, through the winter period, may be found.

If ticks are introduced on host animals that are not carrying any diseases of concern, the ticks could still carry infectious agents that are transovarially or even transtadially transmitted e.g. *Boophilus* spp. ticks could carry *Babesia bovis*, transovarially. Furthermore, if ticks are introduced and become established, the possibility then exists that serious diseases introduced at a later date, could become established in the tick population.

Tick-borne diseases of humans that could be introduced by ticks include: Rocky Mountain spotted fever, tick-bite fever, Lyme's disease, tularaemia, Crimean-Congo haemorrhagic fever, Q-fever and tick-borne encephalitis.

It is concluded that the introduction of ticks is a serious possibility and that steps to reduce this possibility should be carefully designed and enforced.

### 9.1.2 Possibility of ticks becoming established in New Zealand

The possibility of ticks becoming established in New Zealand depends on the species of ticks introduced and the place of introduction. Tick species that occur only in tropical climates and which cannot survive frosts are unlikely to establish in New Zealand except possibly in niche localities in the far north of the country. The establishment of *Amblyomma* vectors of heartwater is therefore unlikely. On the other hand there are several species of ticks that are found in temperate climates. In general, accurate predictions cannot be made about what could or could not survive in New Zealand. Only unfortunate experiences will indicate what is possible. It is therefore important that all ticks should be regarded as dangerous and vigorous attempts made to exclude them.

Introductions of animals into zoos, has the advantage that they are held in relatively confined areas often isolated from other animal species. This should allow them to be carefully observed and if necessary to be treated with suitable insecticides, thereby further reducing the possibility of establishment of ticks in the post quarantine period

# 9.1.3 Risk reduction measure

If artificial insemination or embryo transplant technology is used to introduce genetic material ecto and endo parasites will not be introduced.

Animals should where possible be brought from countries where only limited numbers of ticks occur and countries that are free from the major diseases covered by this review.

Animals should be sprayed or dipped shortly before being moved into pre-entry quarantine or isolation. During the time in quarantine they should be regularly dipped or sprayed using a suitable regime to eliminate ticks.

During quarantine, antelope should be kept on a concrete floor surrounded by a trough filled with a suitable insecticide solution. Bedding should be removed regularly and incinerated and a high standard of hygiene maintained.

Animals should be closely inspected for ticks, other parasites or skin lesions. The inspection should include inspection of the ears, perineal region and all skinfolds not otherwise visible.

On arrival in New Zealand animals should be similarly inspected and all bedding and waste material in crates destroyed by incineration.

In post-entry quarantine similar inspections, insecticide treatments and destruction of bedding should be carried out.

Animals should be released into suitable hygienic accommodation in New Zealand zoos and post entry quarantine inspections carried out at specified intervals.

More details of the recommendations for preventing the importation of ticks are given in Section 1.1.5.

# 9.1.4 Parties that would be affected by the introduction of ticks into New Zealand

Introduction of ticks to New Zealand could have consequences for the health of the residents of New Zealand, as several human diseases could be introduced.

Ticks could have serious negative economic effects on the health of animals and the economic performance of the animal industries. The industries that would be primarily affected by the introduction of ticks brought in by antelopes, would be the dairy and beef industries and to a lesser extent the sheep, deer and goat industries. The pig and poultry industries would probably remain unaffected by ticks brought in by antelope.

The introduction of ticks into zoos would have serious consequences for the zoos and would necessitate them introducing measures to control infestation of zoo animals.

## 9.2 Infectious diseases

#### 9.2.1 Probability of introduction of diseases

The risk reduction measures recommended by OIE and the additional risk reduction measures proposed in this review should ensure that the risk of introduction of diseases is extremely unlikely. Risk reduction strategies for excluding exotic diseases when importing animals are generally based on the following principles:

**A. Selection of animals for importation from safe sources**. The following principles given in order of priority should be used in selecting animals for importation:

- a. Selection from disease-free countries.
- b. Selection from diseas e-free zones.
- c. Selection from safe areas within a country or zone.
- d. Selection from properties (zoos) with a reliable history of freedom from disease.
- e. Importation during safe periods, such as times of low vector activity and times when outbreaks of disease are not occurring.

**B.** Quarantine. The quarantine period should be for the maximum length of the incubation period for the disease plus the maximum period for which recovered animals are known to be capable of carrying the infectious agent. As these periods have often been inexactly determined in insufficient numbers of animals an additional period should be added to provide greater security. Quarantine as a means of providing security from disease introduction is not appropriate for all diseases, particularly chronic diseases, those in which protracted carrier states occur and diseases with long incubation periods. For antelope information is not always available and extrapolation from what is known in cattle is sometimes unavoidable.

**C. Diagnostic testing.** Diagnostic tests, particularly serological tests, but including all available and appropriate tests to recognise infected animals should be used.

**D. Treatment.** Treatment of animals to ensure that infectious agents and parasites are eliminated. Treatments are particularly important for eliminating ecto and endo parasites but also sometimes have a role with infectious diseases as in the use of antibiotics to eliminate *Leptospira*. Vaccination is also recommended for some diseases.

**E. Use of artificial insemination or embryo transfer.** These methods have been discussed in Section 7.

# 9.2.2 Probability of introduction of OIE List A diseases

It should usually be possible to select animals from countries where the OIE List A diseases do not occur. Even when List A diseases are present in a country, it should be possible to source animals from safe areas within the country concerned. Additionally animals should be imported during periods when disease outbreaks are not occurring and vector activity is low e.g. animals should only be brought from the southern parts of Australia where bluetongue does not occur, or from the USA when outbreaks of vesicular stomatitis are not occurring. Adequate diagnostic tests and the ability to quarantine animals and even the possible use of artificial breeding methods would add additional security to the importation process.

Quarantine periods recommended by OIE for vesicular stomatitis, rinderpest, Rift Valley fever and lumpy skin disease are adequate. In the case of foot and mouth disease there is the complication that cattle and African buffalo can carry the infection in their tonsils for years, although they are not usually infectious at this stage. For this reason the OIE recommended protocols for the introduction of cattle from infected countries include the requirement for culture of probang samples. In the case of antelope entering New Zealand it is unlikely that any of them will carry infection in their pharynx for long periods. However it would be more prudent to require that they are brought only from foot and mouth disease-free countries.

Although the OIE recommendations for bluetongue are probably adequate for most species, a viraemic period of 35 days has been described for mountain gazelle. As the incubation period can be up to 15 days a quarantine period of 60 days instead of the 40 days recommended by OIE would probably be safer. For all the insect-borne diseases quarantine should be in insect-free premises.

Diagnostic testing for all the OIE List A diseases has been developed to a high standard and these tests provide additional security when importing animals. No vaccinations or treatments are recommended for any of the OIE List A diseases. For any animals coming from countries where the diseases occur, serological testing should be mandatory.

Given the precautions that can be taken and provided animals are not brought from foot and mouth disease infected countries, it should be possible to introduce antelope into New Zealand with a negligible risk of introducing any of the List A diseases

# 9.2.3 Probability and consequences of establishment of OIE List A diseases in New Zealand

Four of the six List A diseases, Rift Valley fever, bluetongue, lumpy skin disease and vesicular stomatitis, are carried by insect vectors which are not present (*Culicoides* spp.) or the ability of the disease to establish in New Zealand is in doubt. Without competent vectors these diseases would not spread from a zoo into which they were introduced. If the assumptions made about vectors are wrong and competent vectors do occur in New Zealand, the diseases would probably spread from the zoo to farm animals in the vicinity and the disease concerned could become established.

In the case of foot and mouth disease and rinderpest, the ability of the diseases to spread and become established would depend on the speed with which they were diagnosed and with which effective measures were taken to eradicate them. MAF has highly developed contingency plans to eradicate these diseases and if implemented promptly the diseases would probably be contained and eradicated efficiently, but all infected and in contact susceptible animals would be destroyed. Both diseases are highly infectious and if not efficiently controlled, would probably be transferred by staff working in the zoos and visitors to livestock on farms. The diseases could then spread widely and become established in New Zealand.

If foot and mouth disease were introduced there would be extensive embargoes on the export of animals and animal products. For the other diseases there is no logical reason why any exports other than live animal exports should be effected, but there is also no way of predicting the behaviour of importing countries when animal disease is involved.

In addition to export losses there would be direct losses due to mortality and other production losses. Additional high costs would be incurred for the control and eradication of the diseases.

## 9.2.4 Parties that would be affected by the introduction of List A diseases

Several studies done for or by MAF suggest that foot and mouth disease would have a severe impact on the whole New Zealand economy thus having an impact on the whole population. Rinderpest would have lesser effects and the other List A diseases would probably mainly effect the industries concerned.

Zoos would be severely affected by foot and mouth disease or rinderpest as these diseases would probably result in the depopulation of the susceptible animals in the affected zoo. The introduction of other List A diseases by antelope would have less severe effects. The infected antelope would probably be asymptomatic carriers of disease and as the vectors of disease are not present, the presence of the infection might go unnoticed. If a disease was detected, or if it spread within animals in the zoo or outside the zoo, MAF would undoubtedly impose quarantine and control measures. Possible consequences might include depopulation of a range of animals, quarantining of the zoos, possibly for prolonged periods resulting in loss of income, loss of individual animals, and expenses associated with vaccinating animals and vector control. How the Biosecurity Act would be interpreted is unknown. It is possible that the zoos would be considered to be both "exaccerbators" bearing some responsibility for introducing the disease and "beneficiaries" of an eradication programme. Therefore they could be required to pay a proportional share of the costs of disease eradication.

# 9.2.4 Probablility and consequences of the introduction and establisment of non-List A diseases in New Zealand

Each disease has been discussed previously and only some of the main points are summarised in this section.

Nairobi sheep disease, Crimean-Congo haemorrhagic fever, babesiosis, theileriosis, heartwater, anaplasmosis, Q fever, Ondiri disease, louping ill and other diseases of the tick-borne encephalitis complex diseases, ehrlichiosis (*Ehrlichia bovis*) and tick-borne fever (*Cytoectes phagocytophilia*) are tick-borne diseases that are unlikely to be introduced and will not be able to become established in New Zealand unless ticks are

introduced as well. Selection of animals from safe sources, diagnostic testing and in some cases quarantine will also ensure the infectious agents are not introduced.

Ephemeral fever, Akabane and related viruses, trypanosomosis (tsetse-borne) and other trypanosomoses (*Trypanosoma evansi* and *Trypanosoma vivax*) are carried by vectors either known to be absent or probably absent from New Zealand. In addition there is a low risk of introducing these diseases, as the other methods designed to exclude them should be highly effective.

Quarantine measures will be ineffective for preventing the introduction of 17 of the 44 diseases discussed in the review, including:

- Chronic diseases like Johne's disease, tuberculosis, brucellosis and enzootic bovine leucosis.
- Diseases in which long term carrier states occur such as bovine virus diarrhoea, infectious bovine rhinotracheitis, malignant catarrhal fever, trypanosomosis, babesiosis, anaplasmosis, leptospirosis, toxoplasmosis, sarcocystosis, coccidiosis, cryptosporidiosis and besnoitiosis.
- Diseases with long incubation periods such as Johne's disease, brucellosis, rabies, malignant catarrhal fever and anaplasmosis.

Johne's disease is already endemic in New Zealand and is in any case a disease that is unlikely to be found in imported antelope. MAF does not enforce any measures to prevent the importation of this disease in livestock

Tuberculosis is endemic in possums and other feral animals in New Zealand. Judging by the paucity of reports in the literature the prevalence of the disease in antelope is not high except in some endemically infected areas and the chance of introducing the disease is therefore low. The risk would be further reduced by the use of diagnostic testing and careful selection of the source from which animals are introduced. Eradication of the disease would be feasible if it were to be introduced into a New Zealand zoo.

Given the ability to select animals from safe sources and the reliability of diagnostic tests, the introduction of brucellosis with antelope is very unlikely. If introduced the spread to domestic animals from zoos would be unlikely.

Enzootic bovine leukosis is unlikely to be introduced with antelope and unlikely to spread from zoos to domestic stock. It would be of little consequence if it occurred in zoo animals.

Bovine virus diarrhoea and infectious bovine rhinotracheitis, are both so widespread in New Zealand that the introduction of these diseases would have no significant impact on livestock or on zoo animals unless new virulent strains were introduced. Sensitive diagnostic methods are available to identify carrier animals.

There are no competent vectors in New Zealand for babesiosis and trypanosomosis and anaplasmosis is unlikely to establish in the absence of competent tick vectors. Diagnostic testing and selection of animals from safe sources should eliminate the risk of introducing these diseases.

The policy for prevention of the introduction of new *Leptospira* serovars in domestic stock, is to treat introduced animals with antibiotics before introduction. There seems to be no reason why the policy should be different for antelope.

Toxoplasmosis occurs extremely widely in New Zealand and there is no reason to try to prevent antelope carrying toxoplasmosis cysts from entering New Zealand. They will in any case be dead-end hosts unless fed to cats. Sarcocystosis and besnoitiosis are of no consequence and provided carcasses of antelope are not fed to carnivores the life cycle of the parasites will remain uncompleted. Coccidiosis and cryptosporidiosis belong to the category of widespread diseases that possibly cannot be prevented from entering the country with their antelope hosts. The species of parasites introduced would be very unlikely to cause significant new diseases in livestock as this has not occurred elsewhere.

A 6-month quarantine period would be required to prevent the introduction of rabies. However, antelope brought from zoos in which the infection has not occurred could be safely introduced provided they had been confined in a safe environment in the zoo for at least 6 months before introduction. Antelope will almost certainly be dead-end host for the rabies virus and transmission from antelope to other animals is very unlikely although this has occurred in kudu (see Section 2.6). Malignant catarrhal fever (Alcelaphine herpesvirus-1) may have a long incubation period and wildebeest and other members of the Alcelaphine sub-family are frequently long-term carriers of the infection. Quarantine alone can therefore not be relied on to prevent the introduction of infection. The introduction of malignant catarrhal fever is mainly a problem for zoos as without a carrier/host species the disease would not become a problem for livestock. It is recommended that zoos do not introduce species of antelope that could be potential carriers of the virus. The policies with regard to the introduction of possible carrier animals have already been determined by MAF. Potential carriers must be quarantined separately from domestic stock and released into zoos. Zoos must manage the consequences of introducing carrier animals.

# 9.3 Parasitic diseases

Parasitic diseases cannot be regulated for on an individual parasite basis. The exclusion of parasites must be based on the treatment of all introduced antelope for both external and internal parasites while they are in quarantine. A carefully designed programme of dipping, spraying or treating with insecticides for external parasites and drenching for internal parasites must be implemented. Examination of faeces for parasite eggs could be included in the programme. Under these circumstances, the possibility of serious parasites being introduced by antelope and becoming established in domestic stock, are very low

# References

1. Fairley R., Heath A. 1997. Exotic ticks intercepted in New Zealand since 1980. Surveillance , 24(1), 21-22.

# Appendix 1

# Common and systematic names of antelopes.

addax	Adday pagamagulatus
	Addax nasomaculatus
Arabian oryx	Oryx leucoryx
Arabian gazelle	Gazella dorcas
blackbuck	Antilope cervicapra
blesbok	Damaliscus dorcas philipsi*
bongo	Tragelaphus eurycerus
bontebok	Damaliscus dorcas dorcas
buffalo (African or Cape)	Synceros caffer
bushbuck	Tragelaphus scriptus
duiker (red flanked)	Cephalus rufilatus
duiker (blue)	Cephalophus monticolor fusicolor
duiker (crowned)	Sylvicapra grimmia
duiker (red)	Cephalophus natalensis
eland	Taurotragus oryx
four horned antelope	Tetracerus quadricornis
gemsbok	Oryx gazella
gerenuk	Litocranius walleri
giraffe	Giraffa camelopardalis
Grant's gazelle Gaze	lla granti
grysbok	Raphicerus sharpei
impala	Acepyceros melampus
klipspringer	Oreotragus oreotragus
kob	Kobus kob
kongoni/Cokes's hartebeest	Alcephalus busephalus cokei
kudu (greater)	Tragelaphus strepsiceros
lechwe	Kobus leche
mountain gazelle	Gazella gazella
nilgai/Indian antelope	Boselaphus tragocamelus
nyala	Tragelaphus angasi
oribi	Ourebia ourebi
oryx or Beisa oryx	Oryx beisa
oryx or Beisa oryx pronghorn	Oryx beisa Antilocapra americana
	-
pronghorn	Antilocapra americana

roan antelope	Hippotragus equinus
sable antelope	Hippotragus niger **
saiga antelope	Saiga tatarica
sitatunga	Tragelaphus spekei
slender horned gazelle	Gazella leptoceros
Speke's gazelle	Gazella spekei
springbok	Antidorcas marsupialis
steenbok	Raphicerus campestris
suni	Neotragus moschatus
Thompson's gazelle	Gazella thompsoni
topi	Damaliscus korrigum
tsessebe	Damaliscus lunatus
waterbuck	Kobus ellipsiprymnus
wildebeest (blue)	Connochaetes taurinus
wildebeest (Black)	Connochaetes gno

\* Also given as Damaliscus. albifrons \* \*Also given as Ozanna grandicomis