IMPORT RISK ANALYSIS

IMPORTED SEROPOSITIVE ANIMALS :

Assurance provided by serological tests

July 1999

Howard J Pharo
Regulatory Authority
Ministry of Agriculture and Forestry
Wellington
NEW ZEALAND

ISBN : 0-478-07981-8

Import Risk Analysis

Imported seropositive animals : Assurance provided by serological tests

Ministry of Agriculture and Forestry
Te Manatu Ahuwhenua, Ngaherehere
ASB Bank House
101-103 The Terrace
P O Box 2526
Wellington
New Zealand

Telephone:+64 4 474 4100
Facsimile:+64 4 474 4133
Internet:http://www.maf.govt.nz

Import Risk Analysis

Imported seropositive animals : Assurance provided by serological tests

July 1999

ISBN : 0-478-07981-8

Howard J Pharo
National Adviser (Agricultural Security)
Animal Health and Welfare Group
MAF Regulatory Authority
email: pharoh@maf.govt.nz

Approved for general release

Dr B D O'Neil
Chief Veterinary Officer

TABLE OF CONTENTS

1. EXECUTIVE SUMMARY

This document examines the biosecurity risks posed by the importation of live cattle, sheep, goats, horses and pigs, should any such animals be found seropositive during post-arrival quarantine after having already tested negative pre-export.

On a number of occasions in the past MAF has detected serologically positive animals in quarantine. Each case has been dealt with on an ad hoc basis and, at times, the decisions made have been criticised by interested parties. This review aims to establish a basis for future incidents when seropositive animals are detected.

Under the Agreement on the Application of Sanitary and Phytosanitary (SPS) Measures, member countries of the World Trade Organisation are obliged to ensure that their sanitary measures are based on a scientific assessment of risk. MAF's policy on serological positive animals constitutes an SPS measure, and as such it must be based on risk analysis.

The approach of this analysis is to consider in detail the epidemiology of each of the relevant animal diseases, in order to reach a decision for each disease as to whether a seropositive animal would constitute a biosecurity threat to New Zealand. The major issue in reaching such a decision is whether an animal which is serologically positive is likely to be harbouring the particular disease agent. Other matters which may also be considered include whether the seropositive animal is likely to be shedding the agent, and whether the introduction of the agent in an imported animal can be expected to result in the establishment of the disease here.

The analysis concludes that for many of the diseases considered of importance for international animal trade there is an identifiable biosecurity risk associated with seropositive animals.

However, for some diseases it is concluded that seropositive animals would not be carrying the infectious agent, and therefore could be safely released from post-arrival quarantine, albeit after a quarantine period of several months in some cases.

For some vector-borne protozoal and rickettsial diseases it is concluded that although the agent is likely to be carried by seropositive animals, the diseases would not be capable of establishment as the necessary insect vectors do not exist in New Zealand. However, with regard to vector-borne diseases on OIE lists A and B, it remains the policy of MAF that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist here.

The analysis concludes that caution is warranted for several diseases which might be transmitted to some extent by the stable fly, *Stomoxys calcitrans*, for which the distribution, population density and ecology in New Zealand are not clearly understood.

The summary of findings is presented in Table 1.

Table 1: Summary of Recommendations

OIE #	*Disease name*	*Safe*	*Not safe*	*Serology not available*	*Caveat emptor*	*Vectors not present*	*Possible mechanical transmission*
A10	Foot and mouth disease		✓				
A20	Vesicular stomatitis	✓					
A30	Swine vesicular disease		✓				
A40	Rinderpest	✓					
A50	Peste des petits ruminants	✓					
A60	Contagious bovine pleuropneumonia		✓				
A70	Lumpy skin disease	✓					
A80	Rift Valley fever	✓					
A90	Bluetongue	✓					
A100	Sheep pox and goat pox	✓					
A110	African horse sickness	✓					
A120	African swine fever		✓				
A130	Classical swine fever		✓				
B051	Anthrax			✓			
B052	Aujeszky's disease		✓				
B053	Echinococcosis (Hydatidosis)			✓			
B055	Heartwater					✓	
B056	Leptospirosis		✓				
B057	Q fever		✓				
B058	Rabies	✓					
B059	Paratuberculosis (Johne's disease)				✓		
B060	Screwworm			✓			
B101	Bovine anaplasmosis					✓	(✓)
B102	Bovine babesiosis					✓	(✓)
B103	Bovine brucellosis		✓				
B104	Bovine genital campylobacteriosis			✓			

OIE #	*Disease name*	*Safe*	*Not safe*	*Serology not available*	*Caveat emptor*	*Vectors not present*	*Possible mechanical transmission*
B105	Bovine tuberculosis		✓				
B106	Cysticercosis			✓			
B107	Dermatophilosis			✓			
B108	Enzootic bovine leukosis		✓				
B109	Haemorrhagic septicaemia			✓			
B110	Infectious bovine rhinotracheitis/ Infectious pustular vulvovaginitis		✓				
B111	Theileriosis					✓	(✓)
B112	Trichomonosis				✓		
B113	Trypanosomosis (Tsetse-borne)					✓	(✓)
B114	Malignant catarrhal fever	✓ (cattle)	✓ (gnu)				
B115	Bovine spongiform encephalopathy			✓			
B151	Ovine epididymitis (*Brucella ovis)*				✓		
B152	Caprine and ovine brucellosis (excl. *Brucella ovis* infection)		✓				
B154	Contagious agalactia		✓				
B153	Caprine arthritis/encephalitis		✓				
B155	Contagious caprine pleuropneumonia		✓				
B156	Enzootic abortion of ewes (ovine chlamydiosis)		✓				
B157	Ovine pulmonary adenomatosis			✓			
B158	Nairobi sheep disease	✓					
B159	*Salmonella abortus ovis*			✓			
B160	Scrapie			✓			
B161	Maedi-visna		✓				
B201	Contagious equine metritis			✓			
B202	Dourine		✓				
B203	Epizootic lymphangitis		✓				
B204	Equine encephalomyelitis (Eastern	✓					

OIE #	*Disease name*	*Safe*	*Not safe*	*Serology not available*	*Caveat emptor*	*Vectors not present*	*Possible mechanical transmission*
	or Western)						
B205	Equine infectious anaemia		✓				
B206	Equine influenza	**?**vacc					
B207	Equine piroplasmosis					✓	**(✓)**
B208	Equine rhinopneumonitis				✓		
B209	Glanders		✓				
B210	Horse pox			✓			
B211	Equine viral arteritis	✓m,f, (vacc)	✓m,f (vacc)				
B212	Japanese encephalitis	✓					
B213	Horse mange			✓			
B214	Salmonellosis (*S. abortus equi*)			✓			
B215	Surra (*Trypanosoma evansi)*						**(✓)**
B216	Venezuelan equine encephalomyelitis	✓					
B251	Atrophic rhinitis of pigs			✓			
B252	Cysticercosis			✓			
B253	Porcine brucellosis		✓				
B254	Transmissible gastroenteritis		✓				
B255	Trichinellosis		✓				
B256	Enterovirus encephalomyelitis (Teschen/Talfan disease)		✓				
B257	Porcine reproductive and respiratory syndrome		✓				
	Akabane & simbu group bunyaviruses	✓					
	Ephemeral fever	✓					
	Palyam group orbiviruses	✓					
	Bovine viral diarrhoea	✓					

Notes:

(✓) There may be some possibility for iatrogenic transmission or for limited mechanical transmission by biting insects, especially *Stomoxys calcitrans*. See main text for details.

?vacc	Even with approved vaccination protocols, safety cannot be guaranteed. See main text for details.
✓m,f (vacc)	The risk for males and females is different, and is influenced by vaccination and breeding status. See main text for details

2. INTRODUCTION

The New Zealand economy is heavily reliant on international trade, and the New Zealand government is committed to free and fair international trade and to maintaining an open, internationally competitive economy, in the clear understanding that zero risk is impossible unless there is a complete absence of trade. Globally the government is working towards rules-based free trade through the World Trade Organisation (WTO).

One principle of rules-based trading is that health-protection measures should be only applied when necessary, and not as a disguised restriction on trade. One of the WTO agreements, the Sanitary and Phytosanitary Agreement (the SPS agreement), establishes principles which WTO members are committed to uphold when they work to protect health while trading in plants, animals and their products. Under article 5.1 of the SPS agreement, countries are obliged to ensure that their sanitary measures are based on a scientific assessment of risk, taking into account the risk assessment techniques developed by the relevant international organisations.

The aim of this document is to analyse the biosecurity risks posed by the trade in live cattle, sheep, goats, horses and pigs, in the event that an imported animal is found to be seropositive during post-arrival quarantine testing in New Zealand.

An imported animal constitutes a biosecurity risk to New Zealand if it carries a disease agent which is likely to result in outbreaks of serious animal disease in New Zealand. Outbreaks may be considered serious if they cause high mortality or losses of production or if they result in disruption of New Zealand's export of animals and animal products. For outbreaks of disease to occur, the imported animal must be shedding the agent of concern, and the agent must be able to be transmitted to susceptible animals in this country.

2.1 Scope

This analysis is based on the principle that free trade in animals and their products should be permitted except where it can be shown that there is an unacceptably high risk of introduction and establishment of exotic disease agents through the product being traded.

The essential objective of serological testing is to detect those animals that have been exposed at some previous time to the disease agent of interest. However, depending on the particular agent, a serological reaction may not indicate that the animal is still infected with that agent. When assessing risk it is therefore necessary to consider not only the serological test result, but also the pathogenesis and epidemiology of the disease agent. For some disease agents, there is clearly no biosecurity risk in the importation of animals which are seropositive and in fact, seropositivity for some agents may indicate a higher level of confidence of freedom from current infection.

A number of diseases of importance for international animal trade are transmitted by specific insect vectors which are confined to precise ecological conditions that are not present in New Zealand. A consideration of the epidemiology of some of these diseases leads to a conclusion that as competent

vectors are not present in New Zealand it would not be possible for the disease agent to become established in this country even if it were introduced. However, the effect of climate change on the possibility that certain insect vectors might become established in this country in the future is not considered in this analysis. Moreover, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

While the conclusions in this analysis may have implications for the wider policy issue of the importation of seropositive animals in general, the paper is not intended to address that wider issue.

In the case of diseases where epidemiological evidence suggests that it is safe to allow imported seropositive animals to be released from quarantine in New Zealand, the disease risk posed by offspring subsequently born from these animals is examined.

In the case of diseases where it is recommended that seropositive animals could not be safely released from PAQ, it should be noted that for some diseases the recommendation may also apply to the cohorts in PAQ, which may have been exposed to the agent concerned.

Permanent identification of imported animals began in 1998, primarily due to concerns of New Zealand's trading partners regarding transmissible spongiform encephalopathies. Such an identification system will have important applications regarding the tracing of imported animals for any reason, if this were considered necessary.

Representatives of the biopharmaceutical industry have registered concerns that the presence of animals in New Zealand which are seropositive to exotic animal diseases might have a negative impact on exports of animal blood products, such as bovine albumin, which are sold to biotechnology companies for use as nutrients for cell lines grown in fermenters for production of human health products such as interferon, interleukin etc. New Zealand is a favoured source for such material because of our freedom from most of the diseases of concern to international trade.

Preliminary consideration of this matter indicates that the market for bovine albumin should not be affected by the presence in New Zealand of animals which are seropositive to exotic diseases. For this product, blood is collected from slaughterhouses. In most circumstances there is no selection of animals, and blood is simply harvested from whichever animals are slaughtered; an exception to this is the harvesting of blood from animals specifically farmed for this purpose. The biopharmaceutical company bulks together blood from several slaughterhouses for processing. The purification processes that this material are submitted to are extremely rigorous, with the result that there is no chance of any antibody being present in the albumin even if it were present in blood. Antibody is, in fact, completely removed and discarded without any tests being applied to it.

In the case of the production of sera or other blood products which were not processed to remove antibody, the titre for the exotic disease agent would be extremely small and probably undetectable due to dilution effects. If such titres were considered to be unacceptable, measures could be adopted to

exclude seropositive animals from those supplying blood. This would be possible by separation of imported seropositive animals, relying on the system of permanent identification of imported animals.

2.2 Diseases considered

The diseases considered in this analysis are the diseases of international trade significance of ruminants, pigs and horses, based the Office International des Epizooties (OIE) Lists A and B. In addition, a small number of unlisted diseases of potential importance to New Zealand are assessed. Diseases of poultry, bees and fish are not considered.

The OIE Manual of Standards for Diagnostic Tests and Vaccines[1] lists diagnostic tests in two categories : 'prescribed' and 'alternative'. 'Prescribed tests' are those that are required by the international animal health code for the testing of animals before they are moved internationally. 'Alternative tests' are those that are suitable for the diagnosis of disease within countries, and these may be used in animal trade under bilateral agreements.

Table 2 lists the diseases considered and the OIE-recommended diagnostic tests.

Reference

(1) Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. OIE, Paris, 1996.

Table 2: Tests for International Trade

OIE #	Disease name	Prescribed test	Alternative test
A10	Foot and mouth disease	ELISA, VN	CF
A20	Vesicular stomatitis	CF, ELISA, VN	-
A30	Swine vesicular disease	VN	ELISA
A40	Rinderpest	ELISA	VN
A50	Peste des petits ruminants	VN	ELISA
A60	Contagious bovine pleuropneumonia	CF	-
A70	Lumpy skin disease	-	VN
A80	Rift Valley fever	-	HI, ELISA, PRN
A90	Bluetongue	AGID, ELISA	VN
A100	Sheep pox and goat pox	-	VN
A110	African horse sickness	CF, ELISA	VN
A120	African swine fever	ELISA	IFA
A130	Classical swine fever	NPLA, FAVN, ELISA	-
B051	Anthrax	-	-
B052	Aujeszky's disease	ELISA, VN	-
B053	Echinococcosis (Hydatidosis)	-	-
B055	Heartwater	IFA	-
B056	Leptospirosis	-	MAT
B057	Q fever	-	CF
B058	Rabies	-	Agent id., FAVN
B059	Paratuberculosis (Johne's disease)	-	CF, DTH, ELISA
B060	Screwworm (Cochliomyia hominivorax)	-	Agent id.
B101	Bovine anaplasmosis	-	CF, Agg. card
B102	Bovine babesiosis	-	ELISA, IFA
B103	Bovine brucellosis	BBAT, CF, ELISA	-
B104	Bovine genital campylobacteriosis	Agent id.	-
B105	Bovine tuberculosis	Tuberculin test	-
B106	Cysticercosis	-	-

OIE #	Disease name	Prescribed test	Alternative test
B107	Dermatophilosis	-	-
B108	Enzootic bovine leukosis	AGID, ELISA	-
B109	Haemorrhagic septicaemia	-	Agent id.
B110	Infectious bovine rhinotracheitis/ Infectious pustular vulvovaginitis	VN, ELISA, Agent id. (Semen only)	-
B111	Theileriosis	Agent id., IFA	-
B112	Trichomonosis	Agent id.	Mucus agg.
B113	Trypanosomosis (Tsetse-borne)	Agent id.	IFA
B114	Malignant catarrhal fever	-	-
B115	Bovine spongiform encephalopathy	-	-
B151	Ovine epididymitis (*Brucella ovis)*	CF	ELISA
B152	Caprine and ovine brucellosis (excluding *Brucella ovis* infection)	BBAT, CF	Brucellin test
B153	Caprine arthritis/encephalitis	AGID	ELISA
B154	Contagious agalactia	-	Growth inhibition
B155	Contagious caprine pleuropneumonia	CF	Agent id.
B156	Enzootic abortion of ewes (ovine chlamydiosis)	-	CF
B157	Ovine pulmonary adenomatosis	Agent id.	-
B158	Nairobi sheep disease	-	-
B159	*Salmonella abortus ovis*		
B160	Scrapie	-	-
B161	Maedi-visna	AGID	ELISA
B201	Contagious equine metritis	Agent id.	-
B202	Dourine	CF	IFA, ELISA
B203	Epizootic lymphangitis	-	-
B204	Equine encephalomyelitis (Eastern or Western)	-	HI, CF, PRN
B205	Equine infectious anaemia	AGID	-
B206	Equine influenza	-	HI
B207	Equine piroplasmosis	CF, IFA	-
B208	Equine rhinopneumonitis	-	VN
B209	Glanders	Mallein test, CF	-

OIE #	Disease name	Prescribed test	Alternative test
B210	Horse pox	-	-
B211	Equine viral arteritis	VN, Agent id. (Semen only)	-
B212	Japanese encephalitis	-	-
B213	Mange	-	Agent id.
B215	Surra (*Trypanosoma evansi)*	Agent id.	-
B216	Venezuelan equine encephalomyelitis	-	HI, CF, PRN
B251	Atrophic rhinitis of pigs	-	-
B252	Cysticercosis	-	-
B253	Porcine brucellosis	BBAT	-
B254	Transmissible gastroenteritis	-	VN, ELISA
B255	Trichinellosis	Agent id.	ELISA
B256	Enterovirus encephalomyelitis (Teschen/Talfan disease)	-	VN
B257	Porcine reproductive and respiratory syndrome	-	-
	Bovine viral diarrhoea	Agent id.	-

Abbreviations:

BBAT	Buffered *Brucella* antigen test
Agent id.	Agent identification
Agg.	Agglutination
AGID	Agar gel immunodiffusion
CF	Complement fixation
CIEP	Counter immunoelectrophoresis
DTH	Delayed-type hypersensitivity
ELISA	Enzyme-linked immunosorbent assay
FAVN	Fluorescent antibody virus neutralisation
HI	Haemagglutination inhibition
IFA	Indirect fluorescent antibody
MAT	Microscopic agglutination test
NPLA	Neutralisation peroxidase-linked assay
PCR	Polymerase chain reaction
PRN	Plaque reduction neutralisation
VN	Virus neutralisation
-	No test designated yet.

3. CONTEXT

3.1 The role of serological tests in disease diagnosis

The serological diagnosis of infectious diseases based on the detection of circulating antibodies is one of the techniques available for the identification of current and previous exposure to infectious agents. Serological tests are by definition carried out on serum, but similar tests may be applied to other body fluids, such as semen or vaginal mucus.

Serological tests detect the presence of antibody to infectious agents, but the results of these tests must be interpreted with care, especially in individual animals. A positive serological result may be a true positive or a false positive. No tests are 100% specific i.e. most tests have some false positives. Moreover, false positives aside, a positive serological test only indicates *exposure* to a particular disease agent, not necessarily that the animal is infected with that agent.

In other cases an antibody response indicates that an animal has been vaccinated in order to stimulate antibody production to protect it from infection and/or disease. For example, MAF requires that dogs imported from certain countries have demonstrable antibodies against rabies to confirm that the dogs have been vaccinated.

Most serological tests are unable to differentiate between antibodies developed in response to infection with a disease agent and antibodies developed as a result of vaccination.

3.2 Exposure[i], infection[ii] and disease[iii]

The definitions given below in footnotes have been established by the International Epidemiological Association(1).

Even if an animal has a positive result to a certain serological test, it does not necessarily mean that the animal is still infected and needs to be considered a biosecurity risk. An imported animal constitutes a risk only if it is able to transmit the organism. That is, the animal must be infected, it must be shedding the agent or be a carrier, and the organism must be able to come into contact with susceptible animals in the destination country.

There are a variety of ways that an animal can respond to exposure to infectious agents, reflecting the virulence[iv] of agent, the animal susceptibility, and the pathological and clinical reaction of the animal(2).

---

[i] Exposure is contact with a source of disease agent in such a way that effective transmission of the agent can occur.

[ii] Infection is the entry and development or multiplication of an infectious agent in the body of a host.

[iii] Disease, or *dis-ease*, the opposite of *ease*, is when there is something wrong (usually physiologically wrong) with a bodily function.

[iv] Virulence is the disease-evoking power of a microorganism in a given host.

Exposure of an animal to an infectious agent may cause no reaction at all, due to the animal being resistant or immune to the agent. Immunity can be natural[i] or acquired[ii]. Further, acquired immunity can be active[iii] or passive[iv].

In many cases, if the animal is immune, then exposure to the agent has no effect, and infection, replication and shedding of the agent will not occur. Such animals are not important in the transmission of disease agents.

However it may be possible for an animal to become infected with an agent when it is immune to a closely related strain. In this case the immunity to the related strain has no protective effect.

It may also be possible for an agent to infect an animal even when it is fully or partially immune to the specific agent. In such cases the immunity is inadequate to prevent infection even though it may be adequate to prevent clinical disease and shedding of the agent.

If exposure of the animal to an agent does result in infection, there can be a number of possible outcomes to that infection. In an inapparent or subclinical infection there are no clinical signs of disease. A positive serological test may result in such animals following the development of an immune response.

If clinical disease does develop as a result of infection with an agent, the ensuing disease may be mild, severe, or fatal.

In the event of recovery from clinical or sub-clinical disease, a sterile immunity may develop following an effective host response, which results in removal of the infectious agent from the body. A positive serological test in such animals would indicate only exposure to the agent, even though the agent is no longer present in the animal.

Immune pregnant females may pass protective antibodies to their offspring in colostrum. The amount of antibody actually transferred via colostrum depends on the antibody level in the dam, the amount of colostrum consumed by the newborn animal, and the period of colostrum consumption. This results in a variable level of antibody against the agent being found in the bloodstream of the newborn, which may last weeks or months. Therefore a positive serological test in such offspring does not necessarily reflect prior exposure to the agent.

3.3 Carriers and Latent Infections

[i] Natural immunity is a species-determined inherent resistance to a disease agent e.g. the resistance of the horse to canine distemper virus

[ii] Acquired immunity is resistance resulting from previous exposure on an animal to an agent.

[iii] Active immunity is usually associated with antibodies which the host develops in response to an infectious agent or a vaccine.

[iv] Passive immunity is resistance gained by the passage of protective antibody from one host to another, such as from dam to offspring via the placenta or colostrum.

The term "carrier" is used loosely to describe several situations. In a broad sense, a carrier is any animal that sheds an infectious agent without at the same time showing clinical signs. Carriers can pose important sources of infection to susceptible animals. There are three types of carriers:

- Incubatory carriers : these are animals which become infected with a disease agent and which shed the agent before clinical signs develop.

- Symptomless carriers : these animals become infected and shed the agent without ever developing clinical signs. Such animals may also be termed subclinical shedders.

- Convalescent carriers : another possible outcome to recovery from a disease is that the recovering or recovered animal becomes a carrier i.e. in spite of an immune response developing, the agent is not cleared from the infected animal.

Carriers may be infectious either continuously or intermittently. The duration of infectiousness can vary widely, but lifelong carriers are rare.

Another term which is frequently used in describing the epidemiology of a disease is latency. Latently infected animals may also be called true carriers. Latency implies that the disease agent is in a dormant state in an animal and that the infectious agent cannot be identified in such animals.

The important feature of latency is that the organism is not in an "infectious" or "replicating" state. True latency is only recognised with certain viral infections, notably with herpes viruses. Following initial infection, the genome of the virus is incorporated in specific cells, often in the central nervous system. No replication or shedding of virus occurs except at intervals when, in response to stressors, reactivation or recrudescence of the viral genome occurs and infectious virus is produced. Shedding may or may not be accompanied by clinical signs; often it is not.

3.4 Disease transmission

Transmission of disease agents may be direct or indirect[1].

Direct transmission is the immediate transfer of an agent from an infected animal to a susceptible animal. This can be by touching, mating, coughing, sneezing, biting, or by passage across the placenta. Contagious diseases are those which require very close contact for transmission.

Indirect transmission includes vehicle-borne transmission, transmission by arthropod vectors, and transmission by airborne spread over long distances.

Vehicle-borne transmission involves spread via contaminated inanimate material (fomites), water, food, and biological products.

Vectors are arthropods (flies, mosquitoes, ticks etc), and they may be subdivided into mechanical vectors and biological vectors. Mechanical vectors can transmit organisms on their feet or mouthparts, or by passage of organisms through their gastrointestinal tract. Biological vectors are those in which some form of development or propagation of the agent is required before transmission can occur. Transmission by biological vectors may be in saliva during biting or by regurgitation or deposition on the skin of faeces or other material capable of penetrating subsequently through the bite wound or through an area of trauma from scratching or rubbing.

3.5 Quarantine

The important point from a quarantine point of view is how long an infected animal remains infected and how long it is capable of transmission. Various diagnostic tests may be carried out in quarantine to detect agents of concern or antibody against the agent.

Serological tests are designed to detect circulating antibody in animals. The accuracy with which this can be done varies between tests, depending on test sensitivity[i] and specificity[ii].

Pre-export quarantine (PEQ) is used to detect incubatory carriers by allowing animals incubating the disease to develop clinical signs and/or antibody to the agent. Convalescent and asymptomatic carriers are detected in PEQ by testing. The length of the quarantine period necessary is determined by the incubation period of the disease under consideration.

Post-arrival quarantine (PAQ) is necessary to detect animals which are exposed to infection prior to or during PEQ, and which are still incubating the disease at the time of export.

Assuming that the animals being tested in PAQ had already been tested with negative results in PEQ, the appearance of seropositive animals in PAQ would be of serious concern as it would suggest one of the following situations had occurred :

a) if the PAQ test result is a true positive, then, assuming the PEQ test was correctly applied, the PEQ test result was a false negative

b) if the PEQ test result is a true negative, then assuming the same test conditions were used the PAQ test result is a false positive

c) if both the PEQ negative result and the PAQ positive result are correct, then it would indicate that the animal had become infected after the final PEQ test - i.e. quarantine had failed either in the country of origin, or during transit.

---

[i] Sensitivity is the proportion of truly infected individuals in the tested population which are identified as infected by the screening test. Expressed as a probability, this is the probability that an animal will be test-positive given that it is infected.

[ii] Specificity is the proportion of truly non-infected individuals which are identified as non-infected by the screening test. Expressed as a probability, this is the probability that an animal will be test-negative given that it is not infected

The resolution of such situations usually requires further testing, including agent isolation, antigen detection, or PCR studies.

In the case of some diseases for which it is considered that seropositive animals cannot safely be released from post-arrival quarantine, it may be necessary to consider whether the positive reaction disqualifies the single animal only or the entire group of animals imported. The sensitivity of the available tests in detecting carrier animals will be the major factor affecting that consideration.

References

(1) Last JM (ed). A Dictionary of Epidemiology. Third Edition. Oxford University Press, New York, 1995.

(2) Thrusfield M. Veterinary Epidemiology. Second Edition. Pp 69-70. Blackwell Science, Oxford, 1995.

4. LIST A DISEASES

A10. Foot and mouth disease

Foot and mouth disease (FMD) is an acute viral infection of cattle, sheep, pigs, goats buffalo, and many species of cloven-hoofed wildlife[1]. It is one of the most contagious of animal diseases[2]. FMD viruses belong to the genus *Aphthovirus* in the family *Picornaviridae*[3].

The respiratory tract is the usual route of infection for FMD virus, and the viraemia which follows results in distribution of the virus to many tissues of the body. Further replication occurs in many of these tissues, giving rise to characteristic lesions of FMD. Significant excretion of the virus in infected animals may occur for 4 or more days before development of clinical signs; milk and semen is of particular importance in this regard. Saliva contains highest quantities of virus in acutely infected animals. High levels of the virus may be found without lesions in many tissues, however the pregnant uterus appears not to be involved. Abortion is rare, and transplacental infection is not reported. Transmission by wind is possible, especially if large pig herds are affected[1].

Viral replication, other than in the mucosa of the pharyngeal region, does not persist for longer than 14 days in infected animals. Persistent infection (in the mucosa of the soft palate, pharynx and cranial oesophagus) occurs in cattle sheep, goats, buffaloes, and African wildlife species, but not in pigs. In cattle the virus may persist in up to 20% of animals for a year, and in some animals for as long as 2 years. In African buffalo the virus may persist even longer - at least 5 years in the pharyngeal region in some animals - and it is considered that persistently infected buffalo probably maintain the virus in inter-epidemic periods[1].

The importance of persistently infected cattle in spreading disease is not clear, as transmission from persistently infected to susceptible cattle has not been demonstrated under natural or experimental conditions[4].

Sheep and goats have a tendency to develop mild or inapparent infections; they have been considered epidemiologically important in Europe and Turkey[1].

Recovered cattle are generally immune to re-infection with homologous virus for 1-3 years. Maternally derived antibody disappears before 6 months of age in calves and piglets. Vaccination results in protection against experimental challenge for 3-6 months. The duration of immunity depends on the vaccine and type of adjuvant[5]. However, although fully immune animals may be protected against clinical disease, they may still be infected and become carriers[4].

Both virus neutralisation and the ELISA are used as serotype-specific serological tests. Virus neutralisation is slower and more complex to carry out, and low titre false positive reactions can occur[6].

Conclusion: A positive serological test would indicate an animal which has recovered from infection or one which had been vaccinated. Such animals could carry FMD virus for years, and although transmission from persistently infected animals has not been demonstrated, caution is warranted.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Thomson GR. Foot-and-mouth disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 825-51. Oxford University Press Southern Africa, Capetown, 1994.

(2) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 116. Australian Government Publishing Service, Canberra, 1995.

(3) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 334, 1995.

(4) Kitching RP. The excretion of foot-and-mouth disease virus by vaccinated animals. State Veterinary Journal 2(3), 7-11, 1992.

(5) Mann JA, Sellers RF. Foot-and-Mouth Disease Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 503-12. Elsevier, Amsterdam, 1990.

(6) Donaldson AI, Kitching RP, Barnett PV. Foot and Mouth Disease. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 47. OIE, Paris, 1996.

A20. Vesicular stomatitis

Vesicular stomatitis (VS) is a disease of horses, cattle and pigs caused by viruses belonging to the genus *Vesiculovirus* in the family *Rhabdoviridae*[1]. Two antigenically distinct vesicular stomatitis viruses, New Jersey and Indiana, have long been recognised, and isolates of both differ in their physical, biological and genetic properties[2]. In addition, Cocal virus from Trinidad and Alagoas virus from Brazil are very closely related to Indiana virus, and were previously known as Indian type 2 and type 3 viruses respectively[2]. Cocal virus is now considered to be a separate species within the genus, along with VS-Alagoas, VS-Indiana, and VS-New Jersey[1].

Vesicular stomatitis is confined to the Americas. Human infections also occur during epidemics, causing an influenza-like disease, sometimes with oral lesions, which lasts 7-10 days[3].

VS-Indiana occurs from the USA through Central America into Brazil. VS-New Jersey ranges from Canada to Argentina. Cocal virus is limited to South Caribbean countries and VS-Alagoas is found only in Brazil[2].

The natural history of VS, including its endemic, maintenance and epizootic patterns, remains enigmatic[3]. The disease is reported at 2 or 3-year intervals in tropical and subtropical countries in Central and South America[2], but outbreaks in the USA occur at 10-15 year intervals, usually beginning in southern states in the spring and moving a variable distance north before disappearing when the first frosts arrive[4].

With the exception of white-tailed deer, there is no evidence of clinical disease occurring in any of the wild mammals that have been found to have specific antibodies. In the USA the racoon, skunk and bobcat have become infected during epidemics. In the tropics, antibodies to Indiana virus are found frequently in arboreal and semiarboreal mammals such as the kinkajou, two- and three-toed sloths, night monkeys, and marmosets. Antibodies to New Jersey virus have not been found in these animals; among terrestrial mammals in the tropics the agouti and the rabbit have antibodies to New Jersey virus but not to Indiana virus. From the wide range of wild animal species affected it could be concluded that disease in livestock is incidental to the perpetuation of the disease, and that transmission is dependent upon some mechanism other than animal-to-animal contact[2].

Numerous observations incriminate biting arthropods as vectors. Outbreaks are strictly seasonal, and spread does not follow human or animal routes. Rather the disease appears to follow natural features such as mountain valleys, river basins or defined ecological zones such as open woodlands or savannas and to stop at mountains, open prairies, and major bodies of water[6]. Attack rates are lower in horses which are protected from insects. Infection of a range of arthropods and spread from arthropods to susceptible animals has been demonstrated experimentally, and the virus has been isolated frequently from wild-caught sandflies in tropical central America and from mosquitoes in New Mexico[3].

When inoculated into the skin, vesiculoviruses replicate locally in the lower layers of the epidermis and spread quickly to other sites, presumably via the blood stream. However, only extremely low levels of viraemia are produced in VS infections of horses, cattle and pigs[5].

Therefore, despite the evidence for insect transmission of VS, it has not been established how arthropods could become infected with the virus under natural conditions. One source may be the blood of infected mammals, but vesiculoviruses do not appear to produce viraemias high enough to infect arthropods. Experimental inoculation of VS virus into 55 species of animal failed to produce viraemia in any of them[6].

It has been suggested that insects may become infected from non-domestic animal species which have a prolonged high-level viraemia after infection[4]. It has also been suggested that direct feeding from skin lesions is the most likely route of insect infection[5].

The most favoured transmission hypothesis is that VS is endemic in domestic and wild animals in Central America and possibly the southern states of the USA, that it is capable of insect transmission, and that epidemics occur in North America when climatic conditions favour a northward extension of the insect vector range[4].

Direct animal to animal spread also appears to occur to some extent within a herd[6], although the route is not well understood. The virus may spread by contact when pigs are crowded together for shipping, or when strange pigs are introduced into a pen and fighting occurs. However, in pig herds usually only a small proportion of pigs will develop lesions while many others in the herd will seroconvert[5]. Milking machines are probably important in spreading teat lesions between dairy cattle during outbreaks[4].

The virus does not cross the placenta, and does not cause either seroconversion or abortion in the foetus[6].

There is no evidence for persistence and subsequent shedding of VS virus in livestock[4]. No virus, viral antigens, or viral RNA was found in experimentally infected pigs beyond 6 days post-infection[6]. Immunosuppression of recovered pigs has not produced recrudescence or virus shedding[5].

Antibodies are demonstrated by a liquid-phase blocking ELISA and the virus neutralisation test. Other described tests are the complement fixation test, agar gel diffusion, counter immunoelectrophoresis and a competitive ELISA[7].

In recovered cattle, both complement-fixing and neutralising antibody appear 10-14 days post infection and titres rise for about a month before gradually declining[2]. However, neutralising antibody may persist for as long as 8 years[6], and animals are refractory to re-exposure within a month and for at least a year[2].

In recovered pigs, neutralising and complement-fixing antibodies can be detected 4-5 days after infection and titres rise over the following month. Complement-fixing antibodies recede in 2-4 months, while neutralising antibodies can remain high for years[5].

In recovered horses, antibody titres remain high for at least a year but may fluctuate[6].

Inactivated vaccines for vesicular stomatitis have been developed, and are mostly used in cattle[5], in which species they generate high levels of specific antibodies[7]. However, vaccines are not generally applied for control due to the erratic nature of the disease[4].

Conclusion: A positive serological test would indicate an animal which has recovered from infection or one which has been vaccinated. Viraemia has not been demonstrated in infected animals and there is no evidence for a carrier state in recovered animals.

Recommendation: Seropositive animals could be safely released from PAQ, and the offspring of such animals would not constitute a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 279, 1995.

(2) Hanson RP, McMillan B. Vesicular Stomatitis Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 381-92. Elsevier, Amsterdam, 1990.

(3) Reif JS. Vesicular Stomatitis. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 171-81. CRC Press, Boca Raton, 1994.

(4) Wilks CR. Vesicular stomatitis and other vesiculovirus infections. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 563-6. Oxford University Press Southern Africa, Capetown, 1994.

(5) Carbrey EA. Vesicular stomatitis Virus. In : Pensaert MB (ed). Virus Infections of Porcines. Pp 211-8. Elsevier, Amsterdam, 1989.

(6) Letchworth GJ. Vesicular Stomatitis. In : Studdert MJ (ed). Virus Infections of Equines. Pp 265-79. Elsevier, Amsterdam, 1996.

(7) Fernandez AA. Vesicular Stomatitis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 57. OIE, Paris, 1996.

A30. Swine vesicular disease

Swine vesicular disease (SVD) is a contagious vesicular disease of pigs, caused by viruses belonging to the genus *Enterovirus* in the family *Picornaviridae*[1]. Strains of SVD vary in virulence, and infection may be subclinical or may result in mild or severe disease. The importance of SVD virus in international trade relates to its inclusion in the differential diagnosis for foot and mouth disease[2]. SVD is also a zoonosis. Humans may become infected and develop influenza-like symptoms, aseptic meningitis, or a generalised illness[3].

The incubation period is 2-7 days. Excretions, secretions and many tissues and organs can contain significant amounts of virus before the development of clinical signs. Large amounts of virus are present in the secretions, excretions and lesions of clinically affected animals. Lesions on the feet and in the mouth are the major sources of virus in infected animals. Most virus is produced during the first week of infection and less during the second[4]. The virus can be present in faeces for 20 days or more[5].

In recovered pigs, small quantities of virus may be shed in faeces, urine, and nasal, oral and ocular secretions for several months; the longest period of detection is 13 months, and periods of 3-4 months are probably not rare[6]. However, since lesions are the major source of the virus, subclinically infected animals are usually unable to transmit infection[7].

While most enteroviruses are transmitted by the faecal-oral route, this does not seem to be the case for SVD. Oral transmission is possible only by very large amounts of virus, such as occurs in swill feeding. Transmission via skin abrasions requires 1500 times less virus[7]. When exposed to small amounts of virus, for example in unprocessed waste food, pigs probably become infected through damaged skin since this is the most susceptible tissue. When exposed to large amounts of virus, for example when in contact with infected pen-mates, pigs may become infected by a number of routes[4].

Neutralising antibody can be detected in sera of pigs 4 days after infection. Titres are highest 3-4 weeks after infection, and remain high for years[4]. The prescribed serological test for international trade is the virus neutralisation test. A number of ELISA protocols have been described, but all produce some false positive results[2].

There are currently no vaccines available against SVD[2].

Conclusion: A positive serological test would indicate an animal which has recovered from infection. Convalescent animals shed large amounts of the virus for 2 or 3 weeks after infection. In addition, recovered animals can shed small quantities of virus for extended periods, and although the quantities of virus may not be sufficient to transmit infection between animals, caution is warranted.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 332, 1995.

(2) Donaldson AI, Kitching RP. Swine Vesicular Disease. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 64. OIE, Paris, 1996.

(3) Knowles NJ, Sellers RF. Swine Vesicular Disease. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 437-44. CRC Press, Boca Raton, 1994.

(4) Hedger RS, Mann JA. Swine Vesicular Disease Virus. In : Pensaert MB (ed). Virus Infections of Porcines. Pp 241-50. Elsevier, Amsterdam, 1989.

(5) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 245. Australian Government Publishing Service, Canberra, 1995.

(6) Blaha, T (ed). Applied Veterinary Epidemiology. P 31. Elsevier, Amsterdam, 1989.

(7) Thomson GR. Swine vesicular disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 817-9. Oxford University Press Southern Africa, Capetown, 1994.

A40. Rinderpest

Rinderpest is an acute contagious disease caused by a *Morbillivirus* in the family *Paramyxoviridae*[1]. It is primarily a disease of cattle and buffaloes, and high mortalities are seen in these animals. Infections may also occur in sheep, goats, pigs, and many wild cloven-hoofed animals, without always producing clinical disease[2].

Rinderpest virus has an affinity for lymphoid tissue. In blood the virus is found in mononuclear leukocytes which transport the virus to epithelial tissues, especially those of the alimentary tract. This accounts for the characteristic clinical signs of disease, which include necrotic stomatitis and gastroenteritis. Pregnant animals frequently abort, sometimes weeks or months after the clinical stage of the disease, but transplacental infection of the foetus does not occur[2].

Transmission requires close contact between sick and healthy animals. The virus is excreted in the expired air, nasal and oral secretions, and in the faeces. Recovery from the disease results in lifelong immunity. Surviving animals generally shed the virus only for about 3 weeks post infection[2].

It is generally accepted that recovered cattle are free from infection and that there is no carrier state[3]. The situation in other cloven-hoofed animals is less clear, as small ruminants and pigs have been suggested as reservoirs[3]. This has shown to be the case on the Indian subcontinent, where there are strains of rinderpest virus which cause subclinical infections in sheep and goats, from which the disease can be passed to cattle[4].

Many rinderpest vaccines have been produced[2]. A live attenuated cell culture vaccine is available which confers an immunity that lasts at least 5 years after a single inoculation. The vaccine strain retains its attenuated characteristics during at least five back-passages in cattle, and it lacks the ability to spread by contact[5].

The underlying requirement for the maintenance of the transmission cycle of rinderpest is a sufficiently large population of animals to provide a regular supply of susceptible hosts, and sufficient animal movement to allow animal mixing[2]. Therefore, in Africa and Asia outbreaks of rinderpest are now rarely seen except in areas of civil unrest. However, there is also a pocket of infection in Southern India affecting buffalo, cattle, goats, sheep , pigs and wildlife, and the disease is endemic in the Landhi cattle colony in Karachi, Pakistan[6].

Passive immunity transmitted to offspring via colostrum provides protection for 4-8 months, usually longer[7].

A competitive ELISA is available to detect antibodies to rinderpest in animals that have been infected with field virus or with rinderpest vaccine[4]. It is specific for rinderpest, giving no cross-reactions with antibodies against PPR virus[8].

Conclusion: A positive serological test would indicate either an animal which has recovered from infection or one which has been vaccinated. Recovered cattle do not shed the virus for longer than 3 weeks. There is uncertainty regarding the carrier status in recovered sheep, goats, pigs, and wild cloven-hoofed animals. Vaccination produces a lifelong immunity. In order to exclude the possibility that incubatory or convalescent carriers might pass through quarantine undetected, the combined duration of PEQ and PAQ would have to be at least 1 month.

Recommendation: Given the above quarantine, seropositive cattle could be safely released from PAQ, and the offspring of such animals would not constitute a biosecurity risk. Seropositive animals other than cattle could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 271-2, 1995.

(2) Rossiter PB. Rinderpest. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 735-57. Oxford University Press Southern Africa, Capetown, 1994.

(3) Blaha, T (ed). Applied Veterinary Epidemiology. P 34-5. Elsevier, Amsterdam, 1989.

(4) Anderson EC, Hassan A, Barrett T, Anderson J. Observations on the pathogenicity for sheep and goats and the transmissibility of the strain of virus isolated during the rinderpest outbreak in Sri Lanka in 1987. Veterinary Microbiology 21, 309-18, 1990.

(5) Taylor WP. Rinderpest. In : Office Intemational des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 69-76. OIE, Paris, 1996.

(6) Anderson J, Barrett T, Scott GR. Manual on the Diagnosis of Rinderpest. Second Edition. P 3. Food and Agriculture Organisation of the United Nations, Rome, 1996.

(7) Scott GR. Rinderpest Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 341-54. Elsevier, Amsterdam, 1990.

(8) Anderson J, McKay JA. The detection of antibodies against peste des petits ruminants virus in cattle, sheep and goats and the possible implications to rinderpest control programmes. Epidemiology and Infection 112, 225-231, 1994.

A50. Peste des petits ruminants

Peste des petits ruminants (PPR) is an acute contagious disease caused by a *Morbillivirus* in the family *Paramyxoviridae*[1]. The virus is closely related to the rinderpest virus, and causes clinical disease in goats and sheep with clinical signs similar to rinderpest in cattle[2]. Serological surveys have shown that infection is far more prevalent than clinical disease; many infections, if not most, are subclinical or are insufficiently severe to attract veterinary attention[2].

Goats are generally more dramatically affected than sheep, although this varies with different strains of the PPR virus. There are differences in breed susceptibility in both goats and sheep; European goats are less dramatically affected than West African dwarf breeds[3], and Sahelian breeds of sheep are more resistant than the breeds from the southern humid zone. The virus will infect cattle without clinical signs, and it has been reported in wild ruminants[2].

The incubation period is about 6 days[3]. The virus is present in ocular and nasal discharges, urine and faeces for about a week after the onset of clinical signs[2]. Transmission probably occurs predominantly by the inhalation of aerosols derived from nearby animals, or by nuzzling and licking of infected animals[2].

Climatic effects are involved in outbreaks by influencing the development of secondary bacterial pneumonia, which is a major complication of PPR[2].

As with rinderpest, the requirement for the maintenance of the transmission cycle of PPR appears to be a regular supply of susceptible hosts plus sufficient animal movement to allow mixing of the population. However, unlike rinderpest, PPR is also maintained in flocks at the village and urban level as well as in large nomadic flocks. The ease with which goats and sheep are infected with PPR virus, as opposed to cattle with rinderpest, is a major factor in the spread of this virus. Virtually all outbreaks can be traced to stock movements, either migration to new areas or introduction of new animals. Recovered animals have not been shown to carry the virus[2].

Animals which recover from infection have a lasting immunity. A quarantine period lasting around one month after complete recovery of the last clinical case is considered adequate for control[2]. Colostral antibody protects offspring of immune animals for 4-8 months [4].

Attenuated rinderpest virus vaccine has been used to protect sheep and goats against PPR. This confers immunity against virulent PPR challenge for at least 3 years. Kids born to dams that have been immunised with this vaccine have colostral antibodies to PPR virus for up to 3 months. Attenuated PPR vaccines are also available[2]. There is now a homologous PPR vaccine which allows differentiation of antibodies due to rinderpest infection or PPR vaccination[5].

Besides the routinely used virus neutralisation test and the competitive ELISA, other serological tests, such as counter immunoelectrophoresis, indirect fluorescent antibody tests and a precipitinogen inhibition test may be used[6]. The competitive ELISA is the most widely used test as it allows differentiation between antibodies to rinderpest and PPR viruses[5].

Conclusion: A positive serological test would indicate an animal which has recovered from infection with PPR virus or one which has been vaccinated either with PPR or RP vaccine. As there is no long term carrier state in recovered animals, a quarantine period lasting around one month after recovery of the last clinical case is considered adequate for control. In order to exclude the possibility that incubatory or convalescent carriers might pass through quarantine undetected, the combined duration of PEQ and PAQ would have to be at least 1 month.

Recommendation: Given the above quarantine, seropositive animals could be safely released from PAQ, and the offspring of such animals would not constitute a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 271-2, 1995.

(2) Rossiter PB, Taylor WP. Peste des petits ruminants. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 758-65. Oxford University Press Southern Africa, Capetown, 1994.

(3) Scott GR. Peste-des-Petits-Ruminants (Goat Plague). In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 355-61. Elsevier, Amsterdam, 1990.

(4) Blaha, T (ed). Applied Veterinary Epidemiology. P 37. Elsevier, Amsterdam, 1989.

(5) Anderson J, Institute for Animal Health, Pirbright Laboratory, UK. Personal communication with SC MacDiarmid, March 1999.

(6) Durojjaiye OA, Lefèvre PC. Pest des Petits Ruminants. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 77. OIE, Paris, 1996.

A60. Contagious bovine pleuropneumonia

Contagious bovine pleuropneumonia is a disease of cattle caused by *Mycoplasma mycoides* subsp. *mycoides* (bovine biotype)[1].

Many recovered animals have pulmonary sequestra which result in a chronic, subclinical carrier state. Such animals may carry mycoplasmas for years, and stress may induce the capsule of a sequestrum to break down, with the result that the animal again becomes infectious[1].

The complement fixation test is the most reliable diagnostic method presently available, although it has significant limitations in terms of its sensitivity and specificity[2].

Several vaccines are available[1].

Conclusion: A positive serological test would indicate an animal which has recovered from infection or one which has been vaccinated. A long term carrier state exists in recovered animals.

Recommendation: Seropositive animals could not be safely released from PAQ

References

(1) Schneider HP, van der Lugt JJ, Huebschle OJB. Contagious bovine pleuropneumonia. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1485-97. Oxford University Press Southern Africa, Capetown, 1994.

(2) Regalia J, Lefèvre PC. Contagious Bovine Pleuropneumonia. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 85. OIE, Paris, 1996.

A70. Lumpy skin disease

Lumpy skin disease (LSD) is an acute, subacute or inapparent viral disease of cattle caused by a virus which belongs to the genus *Capripoxvirus* in the family *Poxviridae*[1]. The severity of clinical signs depends on the strain of the virus, but these may include fever, skin nodules, necrotic plaques in the mucous membranes, and swelling of peripheral lymph nodes[2]. The disease was first diagnosed in Zambia in 1929. Since then it has occurred in many African countries and in Madagascar, with considerable variation in mortality rate. In outbreaks over the past 20 years the mortality rate has been less than 5%. In 1989 the disease occurred for the first time outside Africa, in southern Israel[3].

LSD is not particularly contagious, and direct transmission by contact between animals is inefficient[3]. Biting flies have been incriminated in most epidemics, which have been well-defined and have occurred at regular intervals. Outbreaks are more common during the wet summer and autumn months, particularly in low-lying areas and along water courses. The virus has been recovered from *Biomyia fasciata* and *Stomoxys calcitrans* caught while feeding on infected cattle[2].

About 5 days after infection there is a febrile reaction which lasts for 5-12 days. A primary nodule appears at the site of inoculation about 7 days later, and it seems that the virus multiplies in the dermis at the site of insect bites. Viraemia follows, coinciding with the temperature rise, and lasts about 4 days. During the course of disease the virus is present in saliva for 11 days, semen for 22 days, and skin and nodules for 33 days, but not in urine or faeces. Although the saliva contains large amounts of virus, infection by direct contact or by fomites is not of importance, and the period of infectivity will largely depend on the accessibility of the virus to biting flies. It appears that the animal will be most infective during the short viraemic period 2-3 days before and after the appearance of lesions. Since the virus is present in skin nodules for 5 weeks, infected cattle are probably a source of infection during this period[2].

Based on South African field experience it is generally accepted that recovered cattle are not virus carriers[2].

Animals which have recovered from disease develop neutralising antibodies which persist for at least 5 years[2]. The immunity to reinfection is predominantly cell mediated[4]. Cattle vaccinated with attenuated cattle capripox strains or with strains from sheep and goats develop neutralising antibody in 10 days and this persists for at least 3 years. Calves of immune cows acquire maternal antibody via colostrum and are able to resist serious clinical disease for at least 6 months. Immunity in recovered or vaccinated animals is lifelong[2].

The virus neutralisation test is the most specific serological test. The agar gel immunodiffusion test and indirect fluorescent antibody test are less specific due to cross-reactions with other pox viruses. Western blotting is both sensitive and specific, but it is expensive and difficult to carry out[4]. For this reason, a history of freedom from clinical disease of the herd of origin and/or zone, as recommended by the OIE[5], is more appropriate than serology as a safeguard for imported animals.

Conclusion: The interpretation of a positive serological test in a clinically normal animal would depend on the test used. A positive reaction could result from a cross reaction with other pox viruses, such as the parapoxvirus which causes contagious ecthyma (scabby mouth), or it could indicate an animal that was immune through either vaccination or recovery from infection with capripox virus.

Recommendation: Provided the animals complied with the requirements recommended by the OIE, seropositive animals could be safely released from PAQ in New Zealand, and the offspring of such animals would not pose a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 85-6, 1995.

(2) Woods JA. Lumpy Skin Disease Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 53-67. Elsevier, Amsterdam, 1990.

(3) Barnard E, Munz E, Dumbell K, Prozesky L. Lumpy skin disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 604-12. Oxford University Press Southern Africa, Capetown, 1994.

(4) Kitching RP, Carn V. Lumpy skin disease. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 93. OIE, Paris, 1996.

(5) Office International des Epizooties. International Animal Health Code. Special Edition 1997. Pp 146-7. OIE, Paris, 1997.

A80. Rift Valley fever

Rift valley fever (RVF) is a peracute or acute disease of domestic ruminants in Africa, caused by mosquito-borne viruses in the Rift Valley fever complex belonging to the genus *Phlebovirus* in the family *Bunyaviridae*[1]. The disease is most severe in sheep, cattle and goats, producing high mortality rates in newborn animals and abortions in pregnant animals. However, many infections with RVF virus are inapparent or mild[2]. It is also a zoonosis; humans become infected with the virus mainly by contact with tissues of sick animals, and possibly also by insect bites. Infection in humans is usually associated with mild to moderate disease characterised by fever, myalgia, and prostration and which is typically self-limiting after 2-5 days[3].

Apart from recent outbreaks in Sudan (1973, 1976), Egypt (1977, 1978), Senegal and Mauritania (1987), epidemics have been confined to eastern and southern Africa, and have usually been associated with above average rainfall at irregular intervals of 5-18 years[2]. From late October 1997 to January 1998 torrential rains occurred in most of East Africa, resulting in the worst flooding in the region since 1961-63. This included the northern part of Tanzania, northeastern Kenya, southern Somalia and southeastern Ethiopia. The ensuing outbreak in those areas in 1997/98 constituted the outbreak in eastern Africa since the virus was discovered in 1930[4].

The epidemiology of RVF revolves around the natural history and ecology of specific mosquito vectors. In southern and eastern Africa, outbreaks are centred on pans ("dambos") where surface water gathers after abnormally heavy rains. These pans constitute an ideal environment for the breeding of *Aedes* spp mosquitoes of the subgenera *Aedimorphus* and *Neomelaniconion*, which are often referred to as "flood-water *Aedes*"[3]. These mosquitoes attach their eggs to vegetation at the water's edge of these flooded pans; they have drought-resistant eggs which may survive in dried mud for several years without hatching and which require one or more floodings to trigger their further development[2, 3].

Outbreaks of RVF are initiated when abnormally heavy rainfall leads to an explosive increase in mosquito populations in inland floodpans and low-lying grassy areas[2]. The emerging *Aedes* mosquitoes, some of which are infected with the virus transovarially, preferentially feed on cattle, and amplification of the virus in this host leads to infection of other species of mosquito (especially *Culex* spp) which in turn infect other species of vertebrate hosts[5]. Infection rates in vector populations are low even during epidemics, usually below 0.1%. But in these specific ecological conditions, an enormous number of mosquitoes emerge from flooded areas and vertebrates are subjected to an extremely high mosquito-biting frequency[2]. The onset of cold weather puts an end to epidemics, not only by suppressing vector activity and breeding, but also because warm weather appears to be necessary for the development of infectivity in mosquitoes[2].

While the presence of the RVF virus in the Sudan and West Africa has been recognised for decades, disease outbreaks were not seen there until extreme alterations in ecosystems allowed vectors to proliferate to a point where a threshold of RVF transmission was exceeded. Outbreaks in those regions have been independent of rainfall; rather there has been an association with the development of irrigation canals or swamps that provide mosquito breeding sites, such as occurred in Egypt following the building of the Aswan dam[5].

The outbreak in southern Mauritania and adjacent northern Senegal occurred under exactly the same circumstances. Despite the presence of the virus there, serious disease problems did not occur until a large dam was built on the river which forms the border between the two countries[2].

The introduction of the disease into Egypt is considered to have been either by air travel of viraemic humans from the Sudan, or by the movement of viraemic livestock to be slaughtered in southern Egypt. However, the virus disappeared from Egypt after the end of the outbreak, although the reasons for this are not understood[2].

In lambs less than a week old, which are extremely susceptible to clinical disease, viraemia can be seen within 16 hours of infection, and it persists for the duration of the disease which usually terminates fatally in 36-42 hours. In older sheep, goats and cattle, viraemia is detectable 1-2 days after infection and persists for up to 7 days[2]. The viraemia disappears with the development of the immune response, and in recovered animals there is no evidence of a carrier state. The fact that the virus was isolated from the liver and spleen of approximately 50% of clinically normal sheep for up to 21 days after experimental infection is of possible public health significance in endemic areas, but it does not appear to be epidemiologically significant, as in those organs the virus is not accessible to vectors[8].

Infected humans and animals develop specific antibodies which may be demonstrated by virus neutralisation as early as 3 days following infection and after 6-7 days by ELISA and haemagglutination inhibition. Serological tests used less often include immunofluorescence, complement fixation and immunodiffusion[7]. The duration of titres has not been studied in animals, but they remain high for many years in humans, and domestic animals appear to have a long-lasting immunity. Lambs born to immune dams are protected by maternal antibodies for at least 3 months[6].

A wide range of live and inactivated vaccines are available. Vaccination of pregnant dams with inactivated vaccine confers colostral immunity which lasts a similar time to that resulting from natural infection[2].

Many species of mosquito in the genera *Aedes*, *Culex*, *Anopheles*, *Eretmapodites* and *Mansonia* have been shown to be capable of becoming infected with RVF virus under field conditions in Africa. In view of this broad host range, experiments have been carried out in other continents to establish whether local species of mosquitoes should be regarded as potential vectors. North American mosquitoes of the genera *Aedes*, *Anopheles* and *Culex* have been shown to transmit the virus under laboratory conditions[6]. Similarly, experiments carried out on several species of Australian mosquitoes have shown that two tropical/subtropical species which have been introduced and have become established in parts of both islands of New Zealand, *Aedes notoscriptus* and *Culex quinquefasciatus*, are susceptible to infection with RVF virus by feeding on infected hamsters and are also capable of transmitting infection 10-16 days later[9].

However, as the extreme weather and ecological conditions required for the establishment of outbreaks do not occur in New Zealand, and in particular as this country has a winter rainfall pattern, the significance of such experimental findings for this country is unclear. While there is obviously a

possibility that the introduction of a viraemic human or animal into New Zealand could result in cases of RVF in some areas, it is considered unlikely that the disease could become established in this country.

Conclusion: A positive serological test would indicate an animal which has recovered from infection or one which has been vaccinated. The maximum duration of viraemia in infected animals is 7 days. The absence of suitable ecosystems in New Zealand means that the virus would probably not become established even if viraemic animals were imported. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand. To ensure that no viraemic animals were released from quarantine, PEQ should be in a vector-free area or in insect-proof conditions, and the combined length of PEQ and PAQ should be at least 7 days.

Recommendation: Given the above quarantine, seropositive animals could be safely released from PAQ in New Zealand, and the offspring of such animals would not constitute a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 311-12, 1995.

(2) Swanepoel R, Coetzer JAW. Rift valley fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 688-717. Oxford University Press Southern Africa, Capetown, 1994.

(3) Peters CJ, Linthicum KJ. Rift Valley Fever. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 125-38. CRC Press, Boca Raton, 1994.

(4) Sall AA, de A Zanotto PM, Vialat P, Sene OK, Bouloy M. Origin of 1997-98 Rift Valley fever outbreak in East Africa. The Lancet 352, 1596-7, 1998.

(5) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 220. Australian Government Publishing Service, Canberra, 1995.

(6) Wood OL, Meegan JM, Morrill JC, Stephenson EH. Rift Valley Fever Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 481-94. Elsevier, Amsterdam, 1990.

(7) Barnard BJH. Rift Valley Fever. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 102. OIE, Paris, 1996.

(8) Yedloutschnig RJ, Dardiri AH, Walker JS. Persistence of rift valley fever virus in the spleen, liver, and brain of sheep after experimental infection. Contr. Epidem. Biostatist., 3, 72-6, 1981.

(9) Turrell MJ, Kay BH. Susceptibility of selected strains of Australian mosquitoes (Diptera; Culicidae) to Rift Valley fever virus. Journal of Medical Entomology 35(2),132-5, 1998.

A90. Bluetongue

Bluetongue (BT) is an infection of sheep and other domestic and wild ruminants, caused by a member of the genus *Orbivirus* in the family *Reoviridae*[1]. The virus is transmitted only by *Culicoides* midges, which are not present in New Zealand[2]. Cattle are the major vertebrate host of the virus, but sheep and deer are normally the only species to exhibit disease. The disease may vary from peracute to chronic, with mortality rates ranging from 2 to 30%. However, many infections in sheep are clinically inapparent, even in fully susceptible animals[3].

Within the *Orbivirus* genus there are 14 serogroups. Most serogroups are immunologically distinct, but there is considerable cross reaction between members of the Bluetongue virus (BTV) serogroup and the epizootic haemorrhagic disease (EHD) serogroup[3]. Within the BTV serogroup there are currently 24 serotypes recognised[4].

BTV is generally found between the latitudes 40˚N and 35˚S, but the presence of the virus within this band, either seasonal or year round, depends on climate. Although it was considered as an "emerging disease"in the 1960s and 1970s, it is now known that year round BTV activity is restricted to the tropics and subtropics, closely following the spatial and temporal distribution of ruminants and competent *Culicoides* midges, and that disease caused by the virus is limited to seasonal outbreaks in "incursional" zones on the limits of the range of the virus and its vectors[5].

Of the greater than 1400 species of *Culicoides* worldwide, less than 20 are considered actual or potential vectors of BTV, and these vary in their susceptibility to infection and efficiency of virus transmission[6]. In any given environment usually only one or two *Culicoides* species are important[7]. *C. imicola* is believed to be the principal vector in Africa, Europe, the Middle East and Asia, *C. variipennis* in North America, *C. insignis* in Central and South America, and *C. fulvus* and *C. wadai* in Australia. However, both *C. imicola* and *C. variipennis* may be complex species consisting of multiple subspecies, each with differing capacities to transmit disease[4, 8].

Genetic studies of BTV have shown that the virus exists in discrete, stable ecosystems[4], and it is currently considered that strains of BTV from different regions of the world constitute geographically distinct "topotypes" which are the result of prolonged co-evolution of virus strains and vector in each region[6]. When viruses are introduced into a different ecosystem they appear to die out for lack of an efficient vector[4].

The virus may be transmitted throughout the year in the tropics or in areas where the winter is mild, but overwintering of the virus in areas with long, cold winters is more difficult to explain, as transovarial transmission of bluetongue virus in *Culicoides* spp. does not occur. In such areas it is probable that cattle or wild ruminants act as reservoir hosts or infected vectors survive in favourable microclimates[5].

Cattle are the main amplifying host for BT virus, and they are probably also important maintenance hosts. The competent species of *Culicoides* vectors are generally preferential cattle feeders[7]. Outbreaks in sheep occur in late summer and autumn, suggesting that populations of infected midges build up in the primary cycle involving cattle or wild animals during spring and early summer, and that sheep become infected in a secondary cycle as a "spillover"[4].

The disease is not contagious and very little virus is found in the secretions and excretions of infected animals. Oral or aerosol transmission is therefore highly unlikely and animal tissues and products, even from infected animals, can be disregarded as a source of infection[5]. Although BTV can be demonstrated sporadically in the semen of viraemic bulls, the virus is not isolated from semen once the viraemia is terminated[9]. Transmission of BTV by artificial insemination of semen from a viraemic bull has been reported[10], but other studies have failed to demonstrate transmission in bull semen by natural mating[11], and it has been concluded that any such transmission is such a rare event as to be of no consequence to virus ecology[6].

BT has been classified by the International Embryo Transfer Society (IETS) as a category one disease i.e. the risk of transmission by embryo transfer is negligible provided IETS protocols are followed[12]. There is evidence that transplacental infection occurs under some circumstances, resulting in infection of the foetus and/or abortion, but not immunoincompetence[6, 10, 13].

It was previously thought that in the most susceptible species, sheep, the viraemia could last up to 120 days. Cattle were thought to harbour the virus in high concentrations for more than 300 days, with viraemic phases occurring periodically[5]. However, the duration of viraemia has been clarified in recent years. Viraemia begins 3-6 days after infection, reaching a peak 7-8 days after infection[6].

In cattle, the duration of viraemia is usually less than 4 weeks, and in 99% of animals it does not exceed 8 weeks[6]. In sheep, the maximum duration of viraemia appears to be 54 days, but it usually lasts only 6-8 days and is rarely reported longer than 14 days[6]. Earlier reported results which conflict with this are now considered to be explained by natural reinfections with different serotypes which were unknown at the time of the work[5].

Viraemia in deer usually lasts between 2 and 8 days followed by the development of neutralising antibodies[14]. As with cattle, it is considered that the maximum viraemia in deer is not longer than 60 days; viraemia has been shown to last 17 days in blesbok, 16 days in white-tailed deer, 10 days in elk, 3 days in pronghorn antelope, and 35 days in mountain gazelle[15]. Viraemias in buffalo appear of similar duration to cattle[6].

For a vector to be able to transmit BTV, it must first become infected by the ingestion of a viraemic blood meal and then the virus must spread to and replicate in the salivary glands. The blood of an infected animal is more infectious for competent vectors when virus titres are high soon after infection before immune mechanisms develop. As virus titres drop there is less chance of a biting midge imbibing an infectious dose of virus. However, a proportion of highly competent midges will be infected even with low-titred blood after a lengthy period of viraemia and it is possible that the viraemia will always be effective to some extent if the vector's competency and attack rate permit[6]. Therefore, although it is now considered that the maximum duration of viraemia is up to 60 days in cattle and 24 days in sheep, most animals are infectious to vectors for a much shorter period[5].

Group-specific and type specific antibodies are formed within 7-10 days of infection with BT virus. The group-specific antibodies persist for about 6-18 months, whereas type-specific neutralising antibodies can often still be demonstrated after 3 years. Protective immunity is associated with neutralising

antibodies[16]. Thus, recovery from natural infection results in a type-specific humoral immunity which usually lasts at least a year but can be lifelong. However, this immunity protects completely only against homologous serotypes[5]. Lambs born to immune ewes obtain passive immunity via colostrum. The duration of this immunity depends on the initial level and persists for a maximum period of 6 months[16].

Vaccination of sheep with attenuated strains is the most practical and effective control measure, and vaccines have been used for more than 40 years. Annual vaccination of sheep is common in endemic areas[5].

Until recently, tests such as agar gel immunodiffusion (AGID) and indirect ELISA were used to detect BT serogroup-specific antibody, although they were unable to differentiate between antibodies to viruses in the BT and EHD serogroups. The advent of the monoclonal antibody-based competitive ELISA has solved this problem, and competitive ELISAs are now available to specifically detect BT serogroup antibodies[3].

Conclusion: The maximum duration of viraemia is less than 60 days, and there is no evidence for a permanent carrier state in recovered animals. The absence of *Culicoides* midges in New Zealand means that the virus could not be transmitted even if viraemic animals were imported. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand. To ensure that no viraemic animals were released from quarantine, PEQ must be in a vector-free area or in an insect-free environment, and the total duration of PEQ and PAQ should be 2 months.

Recommendation: Given the above quarantine, seropositive animals could be safely released from PAQ in New Zealand and the offspring of such animals would not pose a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 217, 1995.

(2) Motha J, Hansen M, Irwin G. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance 24(4), 18-9, 1997.

(3) Eaton B. Bluetongue. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 109. OIE, Paris, 1996.

(4) Gibbs EPJ, Greiner EC. The epidemiology of bluetongue. Comparative Immunology Microbiology and Infectious Diseases 17(3-4), 207-20, 1994.

(5) Verwoerd DW, Erasmus BJ. Bluetongue. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 443-59. Oxford University Press Southern Africa, Capetown, 1994.

(6) MacLachlan NJ, Wilson D, Gard G, Mellor PS, Nevill EM. 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.

(7) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. Pp 63-4. Australian Government Publishing Service, Canberra, 1995.

(8) Mellor PS, Baylis M, Hamblin C, Calisher CH, Mertens PPC. Preface to supplement on African horse sickness. Archives of Virology [Suppl] 14, v-vi, 1998.

(9) Barratt-Boyes S, MacLachlan NJ. Pathogenesis of bluetongue virus infection of cattle. Journal of the American Veterinary Medical Association 206(9), 1322-9, 1995.

(10) Roberts DH, Lucas MH, Bell RA. Animal and animal product importation and the assessment of risk. In: Walton TE, Osburn BI (eds). Bluetongue, African Horse Sickness and Related Orbiviruses; Proceedings of Second International Symposium, Paris, France, 1991. Pp 916-23. CRC Press, Boca Raton, 1992.

(11) Parsonson IM, Thompson LH, Walton TE. Experimentally induced infection with bluetongue virus serotype 11 in cows. American Journal of Veterinary Research 55(11), 1529-34, 1994.

(12) Office International des Epizooties. International Animal Health Code. Special Edition 1997. P 471. OIE, Paris, 1997.

(13) Parsonson IM. Overview of bluetongue virus infection in sheep. In : Walton TE, Osburn BI (eds). Bluetongue, African Horse Sickness and Related Orbiviruses; Proceedings of Second International Symposium, Paris, France, 1991. Pp 713-24. CRC Press, Boca Raton, 1992.

(14) Hoff GL, Trainer DO. Observations on bluetongue and epizootic haemorrhagic disease viruses in white-tailed deer. Journal of Wildlife Diseases, 10, 25-28, 1974.

(15) Hoff GL, Hoff DM. Bluetongue and epizootic hemorrhagic disease : a review of these diseases in non-domestic artiodactyles. Journal of Zoo Animal Medicine 7, 26-30, 1976.

(16) Erasmus BJ. Bluetongue virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 227-37. Elsevier, Amsterdam, 1990.

A100. Sheep pox and goat pox

Sheep pox and goat pox are acute or subacute contagious and often fatal diseases of sheep and goats, caused by a member of the genus *Capripoxvirus* in the family *Poxviridae*[1]. Most strains of the virus are specific to the host species in which they are isolated, but in some countries strains exist that can infect both sheep and goats[2]. The clinical signs vary considerably with the strain of the virus and the species and breed of host[3]. The morbidity rate in sheep may be as high as 70%; mortality varies from 5 to 50% in adult animals and it may be even higher in lambs. Both morbidity and mortality rates are generally lower in goats. Abortion is rare, except in severe cases which are usually fatal. Mild and inapparent infections can also occur[2].

Pox viruses are epitheliotropic, and the effects of disease are therefore seen especially in the skin and in the lungs. Infected animals shed virus in all excretions and secretions[2]. Transmission may be through inhalation of virus in contaminated water droplets, dust or dry skin scabs or through wounds or scratches on the skin[4]. Infection by contact with lesions or infected milk is of minor importance[2]. Mechanical transmission is possible by the stable fly *Stomoxys calcitrans*[5], which is widespread in New Zealand[6]; the virus may survive on flies for up to 4 days[5].

The disease is regarded as being endemic in most African countries north of the equator, as well as the Middle East, Turkey, Iran, Afghanistan and the Indian subcontinent. In these countries transmission is facilitated by sheep and goats being herded into crowded enclosures at night, and environmental contamination leads to introduction of the virus into small skin lesions. During outbreaks the virus is probably transmitted between animals by aerosols[7]. Disease occurs throughout the year, but severe outbreaks usually occur during the winter or during wet and cold weather and in animals weakened by parasites or other infections[2].

Viraemia starts 3 days after infection and lasts 10-12 days. Peak virus titres in skin nodules persist from day 7 to day 14, after which they decline as serum antibodies develop[4]. Nodules usually scab and persist for several weeks, healing to form a permanent, depressed scar. Lesions within the mouth ulcerate and constitute an important source of virus for infection of other animals[7].

Recovery and healing of skin lesions may take 5-6 weeks[4]. High concentrations of virus occur in lesion material. The quantity and duration of virus excretion, especially in the conjunctival and nasal discharges, seems to depend on the capacity of the particular virus strain to produce well-developed pox lesions. The rate of transmission is therefore probably related to the severity of lesions which develop in clinical cases. Peracute cases usually die before significant amounts of virus are excreted. Recovered animals are immune to reinfection for years[2].

The duration of shedding of the sheep- and goatpox viruses by recovered animals has not been subjected to detailed studies[4]. As with other pox viruses, infectivity is destroyed by exposure to direct sunlight, but it is retained in dark stables for long periods, particularly in scabs shed by infected animals. Infectivity may also be present in the wool or hair of recovering animals[2]. It is generally considered that skin scabs are the main source of shed virus[4], and that infectivity may survive in scab material for at least 3 months[8].

Effective cell culture-derived vaccines containing attenuated capripoxviruses provide immunity which lasts over a year, and will probably provide lifelong protection against lethal challenge[3].

The animals which would constitute a biosecurity threat are those carrying the virus, that is, incubatory or convalescent carriers. Serological tests are of limited value in detecting such animals. The virus neutralisation test is the most specific serological test. Western blotting is both sensitive and specific, but is difficult to carry out and expensive. The AGID and indirect immunofluorescence tests cross-react with antibody to other pox viruses[3]. For this reason, a history of freedom from clinical disease of the herd of origin and/or zone, as recommended by the OIE[9], is more appropriate than serology as a safeguard for imported animals.

Conclusion: The interpretation of a positive serological test in a clinically normal animal would depend on the test used. A positive reaction could result from a cross reaction with other pox viruses, such as the parapoxvirus which causes contagious ecthyma (scabby mouth), or it could indicate an animal that was immune through either vaccination or recovery from infection with capripox virus.

Recommendation: Provided the animals complied with the requirements recommended by the OIE, seropositive animals could be safely released from PAQ in New Zealand, and the offspring of such animals would not pose a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 85-6, 1995.

(2) Munz E, Dumbell K. Sheeppox and goatpox. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 613-5. Oxford University Press Southern Africa, Capetown, 1994.

(3) Kitching RP, Carn V. Sheep pox and goat pox. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 119. OIE, Paris, 1996.

(4) Merza M, Mushi EZ. Sheeppox Virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 43-51. Elsevier, Amsterdam, 1990.

(5) Mellor PS, Kitching RP, Wilkinson PJ. Mechanical transmission of capripox virus and African swine fever virus by *Stomoxys calcitrans*. Research in Veterinary Science 43, 109-12, 1987.

(6) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(7) Fenner F. Poxviruses. In : Fields BN, Knipe DM, Howley PM (eds). Fields Virology. Third Edition. P 2697. Lippincott-Raven, Philadelphia, 1996.

(8) Davies FG. Sheep and Goat Pox. In : Gibbs EPJ (ed) Virus Diseases of Food Animals, a World Geography of Epidemiology and Control. Volume II, Disease Monographs. Pp 733-49. Academic Press, London, 1981.

(9) Office International des Epizooties. International Animal Health Code. Special Edition 1997. Pp 162-3. OIE, Paris, 1997.

A110. African horse sickness

African horse sickness (AHS) is a peracute, acute, subacute or mild infectious but non-contagious disease of equine animals caused by an *Orbivirus* in the family *Reoviridae*, closely related to the bluetongue virus[1]. There are 9 different serotypes of AHS virus[2]. Serotypes 1 to 8 are all highly pathogenic for horses and cause 90 to 95% mortality, but serotype 9 is slightly less pathogenic and results in mortality rates of about 70%. The mortality rate for all serotypes in mules is lower, around 50 to 70%, and in donkeys and zebras most infections are subclinical[2].

AHS virus is transmitted biologically by certain species of *Culicoides* (Diptera: Ceratopogonidae), in particular by *C. imicola* which is in fact a complex of several closely related species. Competent individuals within this complex can transmit the virus by only one bite[3]. Although *C. imicola* is the only confirmed field vector, another species within the complex, *C. bolitinos*, appears to play an important role in transmitting AHS in the cooler parts of South Africa[4]. It is also considered likely that other species within the subgenus *Avaritia*, including *C. pulicaris* and *C. obseletus*, may be competent vectors in sub-Saharan Africa. Experimentally, *C. variipennis* has been shown to transmit the virus[5]. There is no evidence that transovarial transmission occurs [3].

Other arthropods have been suggested as possible vectors, mainly based on experimental evidence. While some workers have shown that the mosquitoes *Anopheles stephensi*, *Culex pipiens* and *Aedes aegypti* can transmit the virus, others have failed in their attempts to infect them[2]. Therefore, it is considered that while mosquitoes, biting flies and ticks may play minor roles in mechanical transmission of the disease during epidemics, they are probably not adequate to maintain the disease in the absence of *Culicoides* spp.[5].

Culicoides spp. are not present in New Zealand[6].

Apart from equine animals, dogs are the only animals to contract a highly fatal form of disease, usually by eating infected horse meat. However it is unlikely that dogs play any role in the spread or maintenance of the disease, as *Culicoides* spp. do not readily feed on them[2].

AHS is endemic in Africa south of the Sahara, where it occurs seasonally during warm and wet weather, in a pattern reflecting the presence of *Culicoides* midges. From the endemic zone in central Africa the disease spreads south every year. The extent of southerly spread depends on the time of the year in which the disease makes its first appearance and the extent of suitable climatic conditions for the breeding of *Culicoides* spp. In summer rainfall areas of southern Africa, the most serious outbreaks occur in March and April, and the disease disappears abruptly with the arrival of the first frosts in May[2]. During outbreaks in endemic areas, different virus serotypes may be active simultaneously within an area, but usually one serotype dominates during a particular season, followed the next year by another serotype[2]. All serotypes, apart from serotype 9, are commonly responsible for outbreaks in South Africa. Serotype 9 is widespread in north and west Africa, and serotypes 3, 4 and 9 have been recorded outside Africa[2].

Although it is not contagious, AHS virus can be spread long distances by air transport of animals incubating the disease, or by movement of infective vectors[5]. However, even if introduced, the virus cannot be transmitted unless both a population of susceptible (non-immune) animals and a sufficient population of *Culicoides* vectors are present in the destination country.

While for some years it was known that *C. imicola* was present across northern Africa and throughout the Middle East as far north as Israel, it was not until 1970 that this insect was discovered in Cyprus. In 1981 it was identified in Turkey, and the next year it was found first on the Greek island of Lesbos, and then finally in mainland Europe, in Spain. By 1992 it was recognised that *C. imicola* occurred widely across south and central Portugal and was present in at least 10 Spanish provinces, up to a northern limit of approximately 40˚N. At the same time it was recognised that *C. imicola* was able to overwinter in frost-free areas in the southern Iberian peninsula. However, this insect is essentially an Afro-Asiatic species, preferring warmer climates than those that are usual in Europe. It remains to be seen whether its expansion and extensive distribution in the Iberian peninsula is a permanent result of general climatic warming, or is rather a temporary incursion as a result of a series of mild winters[7].

Epidemics of AHS north of the Sahara have almost exactly mirrored the distribution of *C. imicola*. Until 1959 periodic short-lived epidemics of AHS were reported from Egypt, Arabia and the Middle East, but in 1959 a widespread epidemic caused by serotype 9 virus occurred in Iran, West Pakistan and Afghanistan and subsequently spread throughout the Middle East and to India in 1960. Although an estimated 300,000 horses, donkeys and mules died in the course of this outbreak, the disease did not establish and had disappeared by 1963. In 1965 an epidemic caused by the same type of virus developed in northern Africa and spread briefly to southern Spain in 1966, where in a period of little more than 3 weeks it caused the deaths either directly or indirectly of over 1000 horses[2, 7]. In 1989 there was an outbreak in Saudi Arabia caused by serotype 9, the first outbreak there in 30 years[2]. These more or less regular incursions by serotype 9 virus into the Iberian peninsula are probably initiated by wind-carried infected *C. imicola* from Africa[5].

Between 1987 and 1991 outbreaks of AHS caused by serotype 4 occurred in Spain, southern Portugal and northern Morocco[2]. In Spain alone, 110 animals died directly as a result of the disease, and more than 900 more were destroyed. Approximately 100 outbreaks were seen in five provinces, and almost 250,000 doses of vaccine were administered to susceptible animals in 12 provinces before eradication was declared in January 1990. In November 1990, another small outbreak caused by serotype 4 virus resulted in 66 deaths and the necessitated the vaccination of the entire equine population of Andalusia[8]. Similar vaccination programs were carried out in Portugal and Morocco, and serotype 4 was eliminated from the whole region in 1991[3].

Thus the distribution of AHS virus mirrors the distribution of its main insect vector, *C. imicola*, and although the potential for other *Culicoides* spp. to transmit AHS viruses in Europe has not been clarified, it appears that the disease has not been able to establish permanently outside Africa. Besides the widespread use of vaccine in to control and limit the spread of outbreaks, this may also be due to climatic differences between Africa and Europe and the variability of winters on the Iberian peninsula, or other factors such as vector range and susceptibility of subpopulations of *Culicoides* spp. which may

influence their importance in the maintenance of outbreaks[5]. Another reason that has been suggested is the absence of a long-term vertebrate reservoir, at least in Europe[7].

It has long been considered likely that zebras may be a reservoir host for the virus, and there is strong circumstantial evidence to suggest that ten zebras imported from Namibia were responsible for introducing the serotype 4 virus to Spain in 1987[9]. In addition, it is considered that populations of donkeys and mules may be involved in the maintenance of the virus in some areas, as they are less susceptible to clinical disease than horses but they do become viraemic[5]. However, there is no evidence of a long term carrier state in any of these animals; experimental infections of zebras have resulted in low viraemias for no more than 27 days[10], and viraemia in donkeys lasts less than 2 weeks[11]. Antibodies to AHS virus have been detected in elephants, dogs, camels, sheep and goats[5], but the significance of these findings are not clear.

In horses the onset of viraemia generally corresponds with the appearance of fever. Horses which recover from natural or experimental infection develop a strong immunity to the homologous virus, associated with neutralising antibody, which develops 15-18 days after infection[5]. Viraemia in horses is usually 4-8 days but does not exceed 21 days[2].

Antibodies can be demonstrated by complement fixation, ELISA, immunoblotting and virus neutralisation[12]. In horses which have survived the disease, high complement fixing antibody titres indicate infection with the virus within the last few months. Neutralising antibodies persist for about 2-4 years. Foals born to immune mares acquire colostral immunity which lasts up to 4-6 months[2].

Vaccination is a very effective control measure. Attenuated vaccines give at least 4 years immunity, and inactivated vaccines are available for annual use. Foals born to immune mares acquire passive immunity via colostrum. This immunity progressively declines in foals until it is completely lost after about 4-6 months[2].

Conclusion: The maximum duration of viraemia in equine animals is less than a month, and there is no evidence for a permanent carrier state. The absence of *Culicoides* midges in New Zealand means that the virus could not be transmitted even if viraemic animals were imported. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand. Therefore, to ensure that no viraemic animals were released from quarantine, PEQ must be in a vector-free area or in an insect-free environment, and the total duration of PEQ and PAQ should be at least 1 month.

Recommendation: Given the above quarantine, seropositive animals could be safely released from PAQ in New Zealand and the offspring of such animals would not pose a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 271, 1995.

(2) Coetzer JAW, Erasmus BJ. African horsesickness. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 460-75. Oxford University Press Southern Africa, Capetown, 1994.

(3) Mellor PS, Baylis M, Hamblin C, Calisher CH, Mertens PPC. Preface to supplement on African horse sickness. Archives of Virology [Suppl] 14, v-vi, 1998.

(4) Barnard BJH. Epidemiology and control of viral diseases transmitted by insects. 1997/98 Annual Report of ARC-Onderstepoort Veterinary Institute. Pp 27-8, 1998

(5) Lagreid WW. African horsesickness. In : Studdert MJ (ed). Virus Infections of Equines. Pp 101-23. Elsevier, Amsterdam, 1996.

(6) Motha J, Hansen M, Irwin G. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance 24(4), 18-9, 1997.

(7) Mellor PS. Culicoides as potential orbivirus vectors in Europe. In : Walton TE, Osburn BI (eds). Bluetongue, African Horse Sickness and Related Orbiviruses; Proceedings of Second International Symposium, Paris, France, 1991. Pp 278-83. CRC Press, Boca Raton, 1992.

(8) Rodriguez M, Hooghuis H, Castano Ma. African horse sickness in Spain. Veterinary Microbiology, 33, 129-42, 1992.

(9) Lubroth J. African horsesickness and the epizootic in Spain 1987. Equine Practice 10(2), 26-33, 1988.

(10) Lubroth J. The complete epidemiologic cycle of African horse sickness : our incomplete knowledge. In : Walton TE, Osburn BI (eds). Bluetongue, African Horse Sickness and Related Orbiviruses; Proceedings of Second International Symposium, Paris, France, 1991. Pp 197-204. CRC Press, Boca Raton, 1992.

(11) Hamblin C, Salt JS, Mellor PS, Graham SD, Smith PR, Wohlsein P. Donkeys as reservoirs of African horse sickness virus. Archives of Virology [Supplement 14, 37-47, 1998.

(12) Sánchez-Vizcaíno JM. African horse sickness. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 128. OIE, Paris, 1996.

A120. African swine fever

African swine fever (ASF) is a disease of domestic pigs caused by a unique virus which has recently been classified as the only member of a new genus, *Asfivirus*, in the new family *Asfarviridae*[1]. There is considerable difference in virulence between strains of the virus. In domestic pigs in Africa ASF is classically a peracute disease, but virus strains of intermediate and low virulence are more common elsewhere[2].

The original vertebrate hosts of ASF virus are African wild swine, especially the warthog and to a lesser extent the bushpig, in which infection is inapparent[3]. Virtually all viruses now occurring outside Africa are considered to be derived from a single introduction to Portugal. The spread of ASF virus to and within Europe (by illicit movement of infected pigs, or, more commonly, infected pig products followed by swill feeding) began in 1957, and it has since appeared periodically in several European countries and a few in the Caribbean[2]. The European Commission recognised Portugal free of ASF in 1994, and Spain's freedom was recognised in 1995. In Sardinia, the last place in Europe where the disease is still present, there were 28 outbreaks in 1998[4].

In Africa the virus is transmitted by argasid ticks (*Ornithidoros* spp.) which live in the same burrows as wild swine[3]. However, once the virus becomes established in domestic pigs, it spreads readily among them by a number of routes and does not require a biological vector[3]. Mechanical transmission may be possible by the stable fly *Stomoxys calcitrans*, which is widespread in New Zealand[5]. Experimentally, ASF virus has been transmitted to susceptible pigs by *S. calcitrans*, and the virus survived on flies for at least 2 days without apparent loss of titre[6].

Inapparent 'carriers' have been recognised as very important in the maintenance and spread of the virus, and serological surveys in various infected countries have indicated that between 0.3% and 8% of sera from slaughtered pigs can be positive. The most recent outbreaks in the Caribbean (1971), Belgium (1985) and Holland (1986) highlighted the difficulty of recognising the insidious spread and non-specific symptoms and pathology of infections by Iberian isolates of the virus in completely susceptible populations. Suspicion and laboratory confirmation of the disease did not occur until 1-5 months after its introduction. Iatrogenic spread by farmers and veterinarians was very common. While in the early stages these outbreaks cases of ASF tended to be acute and rapidly fatal, many infections were subclinical, especially towards the end of outbreaks[3].

In acute infections with African isolates, ASF virus is excreted by the nasopharyngeal route as early as 24-48 hours before the onset of pyrexia. The virus is present in all physiological secretions and excretions, including nasal, oral, pharyngeal, conjunctival, genital, urinary and faecal[3]. Survivor pigs infected with Dominican and Maltese isolates were found to excrete virus intermittently for up to a month, during which time transmission to in-contact animals occurred. In these pigs viraemia persisted for up to 8 weeks, and the virus was recoverable in lymphoid tissues for up to 6 months. Thus it appears that pigs in the acute or early recovery stages of infections may transmit readily but that transmission is infrequent, erratic, and possibly dependent on reactivation by stress for the following period of up to 6 months[3].

Transmission in domestic pigs probably occurs by the oronasal route. Vertical transmission has never been reliably reported. Effective vaccines are not available[3].

Antibodies persist in recovered pigs for long periods, sometimes for life, and a number of tests are available for detecting these antibodies, although only a few of them have been developed for routine use in diagnostic laboratories. The most commonly used is the ELISA. When pigs have been infected with avirulent isolates or those of low virulence, serological tests may be the only way of detecting infected animals[7].

Conclusion: Inapparent 'carriers' are very important in the maintenance and spread of the virus. Survivor pigs may shed the virus for a considerable time.

Recommendation: Seropositive animals could not be safely released from PAQ

References

(1) Seventh Report of the International Committee on Taxonomy of Viruses. Academic Press (in press).

(2) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 37. Australian Government Publishing Service, Canberra, 1995.

(3) Plowright W, Thomson GR, Neser JA. African swine fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 568-99. Oxford University Press Southern Africa, Capetown, 1994.

(4) Chillaud T, Head, Information and International Trade Department, Office International des Epizooties, Paris. Personal communication with HJ Pharo, May 1999.

(5) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(6) Mellor PS, Kitching RP, Wilkinson PJ. Mechanical transmission of capripox virus and African swine fever virus by *Stomoxys calcitrans*. Research in Veterinary Science 43, 109-12, 1987.

(7) Wilkinson PJ. African swine fever. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 141. OIE, Paris, 1996.

A130. Classical swine fever (hog cholera)

Classical swine fever (CSF) or hog cholera is a highly contagious viral disease of pigs, caused by member of the genus *Pestivirus* in the family *Flaviviridae*[1]. There are three members of the *Pestivirus* genus, the other two being bovine viral diarrhoea virus and border disease virus. But although *Pestiviruses* are named after the animal species from which they were first isolated, they may infect and cause disease in other animal species[2].

Transmission of CSF virus is mainly by direct contact. Infected pigs shed large quantities of the virus, especially in saliva. Pig meat and meat products are also important vehicles for virus spread. The movement of infected pigs or pig products (followed by feeding garbage to pigs) has been responsible for outbreaks in countries previously considered free of the disease. Iatrogenic transmission on contaminated instruments carried by farmers, castrators, inseminators, and veterinarians is an important route of transmission during epidemics in areas with high density pig populations[2].

The CSF virus shows considerable strain variation, resulting in a highly variable clinical picture. Infection with virulent strains results in high levels of the virus in blood and other tissues. Viral excretion continues until death, or in pigs which survive, until antibodies have developed. Strains of moderate or low virulence may induce chronic infections in which the virus is shed continuously or intermittently for life[2].

The increasing prevalence of strains of low virulence, particularly in endemic countries where attenuated vaccines are used, has complicated the epidemiology of CSF. Inapparently infected carrier sows have become an important means of spread. The Infection of sows with strains of low virulence leads to infection of the foetuses as the virus can cross the placenta. This generally results in the death of the foetus, but if infection occurs late in gestation the piglets may be born alive. Such piglets will be persistently infected and immunotolerant and they may continue to excrete the virus for several months before showing overt disease[3].

Pigs that recover from CSF possess antibodies to the virus. In comparison with other virus infections of pigs, antibodies appear late in the circulation[4]. Pigs with chronic infections, which eventually die, are also capable of mounting a specific antibody response, resulting in the simultaneous occurrence of virus and antibody in the blood. While pigs that produce a normal antibody response to the virus are immune against a subsequent infection, animals with congenital persistent CSF infections seldom produce antibody[4].

Seropositive sows transmit antibodies via the colostrum to their offspring. Such maternally derived antibodies have a half life of about 14 days. The passive immunity generally protects piglets against mortality during the first 5 weeks of life, but not against virus replication and shedding[4].

The detection of virus-specific antibodies is useful in herds suspected of being infected with CSF strains of low virulence. As antibodies against ruminant pestiviruses are regularly observed in breeding pigs, tests must be able to differentiate between bovine viral diarrhoea and CSF antibodies. For this purpose, the neutralisation peroxidase-linked assay (NPLA), the fluorescent antibody virus neutralisation test (FAVN) and the ELISA using monoclonal antibodies are suitable, as they are specific and sensitive[5].

The viral genome is 66% homologous to that of the bovine virus diarrhoea (BVD) virus, another member of the *Pestivirus* genus. As BVD virus is also infectious for pigs, this antigenic relationship leads to cross-reactions in various diagnostic test systems[(2)].

Conclusion: Seropositive reactions usually indicate a sterile immunity in recovered pigs, but in chronically infected animals and in piglets protected from clinical disease by maternal antibody, seropositive reactions can be found in animals which are shedding the virus.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 421-4, 1995.

(2) Terpstra C. Hog cholera. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 654-7. Oxford University Press Southern Africa, Capetown, 1994.

(3) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 77. Australian Government Publishing Service, Canberra, 1995.

(4) Van Oirschot JT, Terpstra C. Hog cholera virus. In : Pensaert MB (ed). Virus Infections of Porcines. Pp 113-30. Elsevier, Amsterdam, 1989.

(5) Terpstra C. Classical swine fever (hog cholera). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 145. OIE, Paris, 1996.

5. LIST B MULTIPLE SPECIES DISEASES

B051. Anthrax

Anthrax is a peracute, acute or subacute, bacterial disease of domestic and wild animals and humans caused by the bacterium *Bacillus anthracis*. Anthrax occurs all over the world, particularly in warm countries, where the ready production of highly resistant spores favours the persistence of contamination in soil and water. Most transmission is by ingestion of contaminated material. Transmission by biting flies may be possible[(1)]. The disease was last diagnosed in New Zealand in 1954[(2)].

Humans can be infected, especially tannery workers and wool sorters[(3)].

There is no carrier state in animals. Vaccines are commonly used, and produce a solid immunity[(1)].

Diagnosis of anthrax is by isolation of *B. anthracis* from the blood or tissues of a recently dead animal. Several serological tests are available for use in humans, but serology is rarely used for diagnostic purposes in animals[(4)].

Conclusion: Serology is not relevant for this disease.

References

(1) de Vos V. Anthrax. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1262-89. Oxford University Press Southern Africa, Capetown, 1994.

(2) MacDiarmid SC. The importation into New Zealand of meat and meat products: a review of the risks to animal health. P 45. Ministry of Agriculture, Wellington, 1991.

(3) Whitford HW, Hugh-Jones ME. Anthrax. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section A: Bacterial, Rickettsial, Chlamydial and Mycotic. Pp 61-82. CRC Press, Boca Raton, 1994.

(4) Whitford HW. Anthrax. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 171. OIE, Paris, 1996.

B052. Aujeszky's disease

Aujeszky's disease (AD), also known as pseudorabies, is caused by a member of the genus *Varicellovirus* in the sub-family *Alphaherpesvirinae*, family *Herpesviridae*[1]. The AD virus has an extremely wide host range, but it is primarily associated with pigs, which remain latently infected following clinical recovery[2]. Infection of sheep, dogs and cats appears to be invariably fatal. Infected cattle rarely recover[3].

Pigs are the most important source of infection for all species. Acutely infected pigs excrete virus in nasal discharges, aerosols, saliva and semen. Susceptible pigs are infected by inhalation or virus-containing aerosols or by contact with contaminated feed and water. Close contact is required for virus spread[3].

Aujeszky's disease was eradicated from New Zealand in 1997[4].

Pigs which have either recovered from pseudorabies or have been vaccinated and subsequently exposed to wild-type virus may become asymptomatic carriers. Carrier pigs may transmit the virus to their offspring either *in utero* or after birth[3]. Vaccines appear to protect pigs against clinical disease but not against infection. Moreover, latency can result from infection of both fully susceptible pigs and of vaccinated pigs[5].

Virus neutralisation was the standard serological method because of its specificity, but it has been replaced by the ELISA which is more sensitive and more suitable for large scale testing[2].

Conclusion: Clinically recovered animals are carriers of the virus as well as being serologically positive. Vaccinated animals may become latently infected.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 120, 1995.

(2) Toma B, Vannier PH, Eloit M. Aujeszky's disease. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 181-2. OIE, Paris, 1996.

(3) Maré CJ. Aujeszky's disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 958-62. Oxford University Press Southern Africa, Capetown, 1994.

(4) Pannett GR, Motha MXJ, MacDiarmid SC. Eradication of Aujeszky's disease from New Zealand pig herds 1976-97. Veterinary Record 144, 365-9, 1999.

(5) Pensaert MB, Kluge JP. Pseudorabies virus (Aujeszky's disease). In : Pensaert MB (ed). Virus Infections of Porcines. Pp 39-64. Elsevier, Amsterdam, 1989.

B053. Echinococcosis/Hydatidosis

Four species of *Echinococcus* tapeworms are recognised internationally, occurring in the small intestine of dogs or other carnivores. Eggs passing in the faeces of the definitive host are ingested by intermediate hosts, in which the cystic stage develops, usually in offal. Intermediate hosts are usually ruminants and pigs, occasionally horses. Humans may act as a dead-end intermediate host, and cystic hydatid disease in humans is an important zoonosis[(1)].

Echinococcus granulosus is now extremely rare in New Zealand[(2)]. Current import standards require anthelminthic treatment of dogs.

While some progress has been made in the development of serological tests for cysts in humans, there are currently no validated tests for cysts in other intermediate hosts[(1)]. If a serological test of sufficient specificity and sensitivity became available for use in cattle, sheep and pigs, seropositive animals would be regarded as infected. However, it is likely that the sensitivity would be such that the test would be more suitable for identification of infected herds than individuals.

Conclusion: Serology is currently not relevant for this disease.

References

(1) Acha PN, Szyfres B. Zoonoses and Communicable Diseases Common to Man and Animals. Second Edition. Pp 717-36. Pan American Health Organisation, Washington, 1987.

(2) Pharo H, van der Logt P. Hydatids diagnosed on Arapawa Island. Surveillance 24(2), 8-9, 1997.

B055. Heartwater

Heartwater is a non-contagious tick-borne disease of ruminants caused by the rickettsia *Cowdria ruminantium*. The disease is characterised by high fever, nervous signs, hydropericardium, hydrothorax, oedema of the lungs and brain, and death. Sheep are more susceptible than cattle, and there is some variation in breed susceptibility in both species. Animals which show clinical signs rarely recover. However, many infections are inapparent, and animals with such infections act as reservoirs[1].

The disease only occurs where *Amblyomma* ticks are present. These require a warm and relatively humid climate and bushy grass country for their development. The disease is of major economic importance in Africa south of the Sahara, and it also occurs on several islands in the Caribbean[2]. *Amblyomma* ticks do not exist in New Zealand[3].

Ten species of *Amblyomma* are capable of transmitting the organism in Africa, but not all of these are good vectors. Their importance in the transmission of heartwater depends not only on their vector competence but also on their distribution and adaptation to domestic stock. The abundance and activity of tick vectors is influenced by temperature and humidity. Good rains are often followed by a rise in disease prevalence, but the pattern of disease is not strongly seasonal[4].

The development cycle of *C. ruminantium* in the tick and the infectivity of successive stages of the tick are poorly understood, but there is evidence that the agent undergoes sequential development in the tick. It is thought that after an infected blood meal initial replication of organisms in the tick takes place in the intestinal epithelium and that the salivary glands eventually become parasitised. The agent then multiplies in the salivary glands of the tick, which may remain infected for life[4].

Vaccination protects susceptible animals against the disease, especially when they are first introduced into an endemic area. Vaccines contain live virulent organisms, and their use is therefore not without risk. There is a variable duration of immunity following vaccination; 2 years in cattle without challenge, 6 months to 4 years in sheep[4].

Serological tests include fluorescent antibody tests, ELISA and immunoblotting. Although cross reactions do occur with *Ehrlichia* spp.[5], these blood parasites have never been detected in routine surveillance of blood smears from all species of animals in New Zealand[6], and vectors for *Ehrlichia* spp. are not present in this country[3].

Young calves, lambs and goat kids have a short-lived natural resistance to infection which is independent of the immune status of their dams. The duration of that resistance varies from 1 week for lambs to possibly 6 or 8 months for calves[4].

Conclusion: A positive serological test would indicate an animal which has recovered from infection or one which has been vaccinated. Cross reactions with *Ehrlichia* spp. also occur. However, neither heartwater nor ehrlichiosis are contagious diseases; they can only be transmitted if specific tick vectors are present. The absence of competent vectors in New Zealand means that these agents could not be transmitted even if infected animals were imported. However, with regard to vector-borne diseases on

OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 349. Australian Government Publishing Service, Canberra, 1995.

(2) Robertson A (ed). Handbook on Animal Diseases in the Tropics. 3rd Edition. P 85. British Veterinary Association, 1976.

(3) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

(4) Bezuidenhout JD, Prozesky L, du Plessis JL, van Amstel SR. Heartwater. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 351-70. Oxford University Press Southern Africa, Capetown, 1994.

(5) Camus E, Uilenberg G. Heartwater. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 229. OIE, Paris, 1996.

(6) Thompson J. Blood parasites of animals in New Zealand. Surveillance 25(1), 6-8, 1998.

B056. Leptospirosis

Leptospirosis is a bacterial disease caused by spirochetes of the genus *Leptospira*. The disease is characterised by haemolytic crisis, nephritis, mastitis, abortions stillbirths and reproductive failure in cattle and pigs, agalactia in sheep and goats, and ophthalmia in horses. It is an important zoonosis, and infection in man is usually associated with exposure to urine of infected animals. Leptospirosis is an occupational hazard of persons in close contact with animals, such as farmers, slaughterhouse workers, veterinarians etc[1].

Pathogenic leptospires were formerly classified as serotypes of *Leptospira interrogans*. However the genus has recently been reorganised and pathogenic leptospires are now identified in seven species of *Leptospira*. Antigenically related serovars are placed in serogroups. There are now 198 serovars arranged in 23 serogroups[2].

Leptospiral infection can result in localisation and persistence of leptospires in the kidney and in the male and female genital tract. This can occur with few or no clinical signs. Such animals are of major epidemiological importance as maintenance hosts[1].

Leptospiral antibodies appear within a few days of onset of illness and persist for weeks or months and, in some cases, years. Unfortunately, antibody titres frequently fall to undetectable levels while animals remain chronically infected. Chronic carriers can only be detected by sensitive methods to isolate the organism from urine or the genital tract[2].

Diagnosis of leptospiral infection poses considerable problems. Serological testing is the most widely used method, and the microscopic agglutination test (MAT) is the standard serological test. Minimum antigen requirements are that the test should employ representative strains of all the serogroups known to exist in the particular country of origin plus those known to be maintained elsewhere by the host species under test[2].

The MAT is used primarily as a herd test, and it is useful for screening herds which have a history of abortions or reproductive problems. As an individual animal test, the MAT is useful in diagnosing acute infection, but it has limitations in the diagnosis of chronic infections in individual animals[2]. Another major disadvantage of the MAT is that it requires the maintenance of live cultures of *Leptospira*, which exposes laboratory staff to infection[1].

Serological diagnosis can become very complicated if an animal has been infected with more than one kind of leptospire. Cross reactions are common between serovars within the same serogroup and even between serogroups. Although serological reactions are higher with the homologous serovar it is not possible to be sure of the identity of a particular leptospire even if a comparison is made with large numbers of serovars. To positively identify a leptospire requires microbiological culture, but this involves time-consuming and difficult procedures[1, 2].

With the exception of dogs, the current policy of MAF is to treat imported animals with antibiotics rather that to employ serological or other testing.

Conclusion: Detailed serological testing is complex, but if it were carried out, animals which were found to be serologically positive to exotic serovars of animal health or public health importance would be considered to be infected. Moreover, it would not be possible to be confident that a serologically negative animal was not a carrier.

Recommendation: Animals which were found to be serologically positive to exotic serovars of animal health or public health importance could not be safely released from PAQ.

References

(1) Hunter P, Herr S. Leptospirosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 997-1008. Oxford University Press Southern Africa, Capetown, 1994.

(2) Ellis WA, Bolin CA. Leptospirosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 198-201. OIE, Paris, 1996.

B057. Q fever

Q fever is a zoonosis caused by an obligate intracellular bacterium, *Coxiella burnetii*[1]. The organism infects a wide range of domestic and wild animals in most countries. Infection in animals is usually subclinical, but it can cause abortion in ruminants and is suspected of causing infertility in dairy cattle in Europe. In humans the disease may be subclinical, acute or chronic and may cause influenza-like symptoms, pneumonia, hepatitis, and endocarditis[1].

In nature, *C. burnetii* cycles silently between ticks and small ground-living mammals. Natural infections have found in 35 species of hard and soft ticks of 11 genera, and eight small mammals, six being rodents, one a lagomorph and one a marsupial[2]. Other wild mammals and birds become inapparently infected, either directly by inhaling coxiellae while eating infected prey, or indirectly by exposure to *Coxiella*-laden dust in areas contaminated by infected wild and domestic ruminants. Once infected, ticks remain infected for life, passing coxiellae on to their progeny. Infections similarly persist in mammals and birds[2].

Infection usually gets into domestic animal populations by tick bites or through contact with dried tick faeces. However, once established in herds and flocks of domesticated ruminants, transmission is commonly independent of ticks, and horizontal spread between animals, usually around parturition, maintains the infection in a population. Infection in domestic animals localises in the genital tract and mammary gland. The organism multiples in those sites and in milking animals is shed in the milk either continuously or intermittently throughout the lactation, as well as in the faeces and urine. In pregnant animals the organism colonises the placenta, but abortion is uncommon. At parturition, enormous numbers of organisms are discharged in the foetal and uterine fluids, contaminating pastures, bedding and premises. Susceptible livestock become infected by inhaling aerosols of this material. Between pregnancies the infection tends to become latent so that the organism cannot be isolated[2].

There are some differences between cattle, sheep and goats in their responses to infection with *C. burnetii*, in particular the proportion of infected animals that become carriers. Sheep infections seldom become chronic; they tend to be transient with spontaneous cure. Both cattle and goat infections frequently result in long term shedding of the organism, particularly around parturition. Dogs and cats become infected when they eat contaminated placental membranes, following which the organism is shed in their milk and urine for weeks[2].

Humans are aberrant hosts who, when infected, occasionally develop disease. Infection of humans is by inhalation of aerosols of infectious material in abattoirs and dairies, or exposure to dust containing dried excreta from infected ticks and grazing animals. Spread between humans is rare[1].

Serological tests for *C. burnetii* include complement fixation, the indirect fluorescent antibody test and the ELISA[4].

No evidence of the organism has been found in New Zealand[4], and the small mammals in which infection has commonly been reported abroad (bandicoot, gerbil, porcupine[5]) do not occur in this country .

Although many species of ticks can become infected, not all of them can transmit infection; most carry the organism for only a short time after engorging on contaminated blood[6]. *Haemaphysalis longicornis*, which is the only tick of domestic animals in New Zealand, has been infected experimentally, but is not clear whether it can transmit the agent. Natural infections in this tick have not been reported[7], and transovarial transmission in this tick has not been demonstrated[5].

The absence of suitable tick vectors and specific small mammalian hosts in this country suggests that if *C. burnetii* were introduced into New Zealand, its establishment in the wild would not be a certainty. Nevertheless, if the organism were introduced in imported domestic animals it might possibly become established in farmed ruminant populations.

Conclusion: Serologically positive animals are likely to be carriers of *C. burnetii*.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Williams JC, Sanchez V. Q fever and Coxiellosis. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 429-46. CRC Press, Boca Raton, 1994.

(2) Scott GR, Herr S. Q fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 390-5. Oxford University Press Southern Africa, Capetown, 1994.

(3) Durand MP. Q fever. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 634. OIE, Paris, 1996.

(4) Hilbink F, Penrose M. Q fever is absent from New Zealand. Surveillance 20(3), 39-40, 1993.

(5) Stoker MGP, Marmion BP. The spread of Q fever from animals to man - the natural history of a rickettsial disease. Bulletin of World Health Organisation 13, 781-806, 1955.

(6) Acha PN, Szyfres B. Zoonoses and Communicable Diseases Common to Man and Animals. Second Edition. P 263. Pan American Health Organisation, Washington, 1987

(7) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

B058. Rabies

Rabies is a fatal nervous disease of warm-blooded vertebrates, caused by a *Lyssavirus* in the family *Rhabdoviridae*. Transmission is generally by the bite of diseased animals, most commonly dogs and other carnivores, and vampire bats in Latin America. Apart from dogs and cats, the most commonly affected domestic animal is cattle. Sheep, goats, buffalo, horses and pigs are rarely affected. Rabies is an important zoonosis[(1)].

Vaccination is a very important control measure. A range of highly effective, safe, inactivated veterinary vaccines is available, producing a protective immunity which lasts from one to three years depending on the antigen content of the vaccine. Some of the vaccines may be used in all domestic carnivores and herbivores, while others may be designed for use in specific species[(1)].

Vaccination of dogs and cats from countries which are not free of rabies is an important safeguard against the importation of rabies into New Zealand. In addition, dogs and cats require two serological tests in the 6 months prior to coming to New Zealand, to show that they have protective levels of antibody against the virus.

Chronic rabies, clinically inapparent infections, and recovery from clinical disease with persistent shedding are extremely rare. Strains of rabies virus have been identified in Ethiopia that have been associated with nonfatal clinical disease or symptomless infection, both with excretion of virus. In countries where rabies is endemic, serologic surveys of dogs for antibodies indicative of a non-fatal response to infection have been documented, as have clinical recoveries from laboratory-induced rabies, but unless further evidence comes to light it can be assumed that the carrier state does not exist[(2)].

Conclusion: Vaccination is part of the import protocol for dogs and cats. Vaccines may be used in other species. A positive serological test may be assumed to be due to vaccination.

Recommendation: Seropositive animals could be safely released from PAQ in New Zealand and the offspring of such animals would not pose a biosecurity risk.

References

(1) Swanepoel R. Rabies. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 493-553. Oxford University Press Southern Africa, Capetown, 1994.

(2) Beran GW. Rabies and infections by rabies-related viruses. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 307-57. CRC Press, Boca Raton, 1994.

B059. Paratuberculosis

Paratuberculosis or Johne's disease is a chronic infectious enteritis of cattle, sheep and goats caused by the bacterium *Mycobacterium paratuberculosis*. Both the cattle and the sheep strains are endemic in New Zealand, and although vaccines are used by some farmers, it is not under any form of official control.

Serological tests available are the complement fixation test, the ELISA, and gel immunodiffusion. The skin test and the gamma interferon test are also available[(1)]. However all of the available tests suffer from low sensitivity[(2, 3, 4)]. In particular, when applied to symptomless carriers (subclinical, light-shedding cattle) the sensitivity of commercially available ELISAs may be as low as 15%[(5)]. The specificity of serological tests is generally greater than 99%[(6)].

In view of the low sensitivity of available tests, serological testing is considered to be a herd level test; testing of individual animals is of limited value.

Conclusion: Paratuberculosis is endemic in New Zealand and the testing of imported animals is not a regulatory requirement. However, if serological tests were applied to imported animals, a positive result would indicate an infected animal.

Recommendation: *caveat emptor*

References

(1) Thorel MF. Paratuberculosis (Johne's disease). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 218. OIE, Paris, 1996.

(2) Sherman DM, Markham RJF, Bates BS. Agar gel immunodiffusion test for diagnosis of clinical paratuberculosis in cattle. Journal of the American Veterinary Medical Association 185, 179-82, 1984.

(3) Ridge SE, Morgan IR, Sockett DC, Collins MT, Condron RJ, Skilbeck NW, Webber JJ (1991) Comparison of the Johne's absorbed EIA and the complement fixation test for the diagnosis of Johne's disease in cattle. Australian Veterinary Journal 68:253-7.

(4) van Maanen C, Swinkels M, Kalis CHJ, Barkema HW, van Weering HJ. Evaluation of the sensitivity and specificity of a commercially available absorbed ELISA in the detection of antibodies against *Mycobacterium paratuberculosis* in cattle and performance of this ELISA in a herd certification program. Abstracts of 6th International colloquium on paratuberculosis, Melbourne, 14-18 February 1999. P 69.

(5) Sweeny RW, Whitlock RH, Buckley CL, Spencer PA. Evaluation of a commercial enzyme-linked immunosorbent assay for the diagnosis of paratuberculosis in dairy cattle. J. vet. diagn. invest. 7, 488-93, 1995.

(6) Collin MT. Diagnosis of paratuberculosis. In : Sweeny RW (ed) Paratuberculosis (Johne's disease). Veterinary Clinics of North America : Food Animal Practice 12 (2), 357-71, 1996.

B060. Screwworm (*Cochliomyia hominivorax*)

Screw worm fly (SWF) is an obligate parasite of warm-blooded animals. Myiasis can cause serious production losses to livestock industries. The geographical ranges of the old world SWF (*Chrysomya bezziana*) and the new world SWF (*Cochliomyia hominivorax*) are different, but they are both restricted to the tropics and subtropics[1].

Serological tests are not available.

Conclusion: Serology does not apply to screw worm diagnosis.

References

(1) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 387. Australian Government Publishing Service, Canberra, 1995.

6. LIST B CATTLE DISEASES

B101. Anaplasmosis

Bovine anaplasmosis is a disease caused by either of two species of protozoa in the genus *Anaplasma* ; *A. marginale* and *A. centrale.* Most outbreaks of clinical disease are caused by *A. marginale*[1], and this organism may under certain circumstances also produce latent infections in sheep and goats[2].

The disease is generally characterised by fever, progressive anaemia and icterus[3].

Colostral antibody and non-specific immunity largely protects calves from clinical disease during the first 9 months of life. Susceptibility to severe disease increases with age[3].

Anaplasma spp. have a wide distribution in the world, and is transmitted almost exclusively by Ixodid ticks. *Boophilus microplus* is the only vector in Australia. However, the argasid tick, *Ornithodoros savignyi* can also transmit *Anaplasma marginale*[3].

In the southern USA *Culicidae* and *Tabanidae* have also been shown to be capable mechanical vectors in the absence of ticks, but this appears to be possible only if insect and animal densities are high enough so that there is no more than a few minutes between consecutive feeds by individual insects[3]. Observations in the USA indicate that *Stomoxys calcitrans* plays no role in the natural transmission[4]. In South Africa, limited experimental work has led to the belief that *S. calcitrans* might be able to transmit infection mechanically in conditions where cattle are in close contact, such as feedlots and dairy herds, once a tick-transmitted outbreak has occurred[5], but in Australia biting flies do not appear to play a role in transmission[3].

Anaplasmosis is relatively easily transmitted mechanically by a range of "veterinary" procedures which allow transfer of blood between animals. Needle sharing, dehorning, open castration, and rectal palpation have all been implicated in such spread, but the disease does not persist in populations without the presence of tick vectors[6].

New Zealand has neither capable tick vectors[7] nor the biting flies that spread the disease mechanically in the USA[8].

Anaplasma infections usually persist for the life of the animal. However, except for occasional small recrudescences of parasitaemia, *Anaplasma* cannot readily be detected in blood smears after the initial parasitaemia. A number of serological tests have been developed to detect latently infected animals, the most widely used being the complement fixation test and the card agglutination test[1]. However, these two tests suffer from problems of low sensitivity and poor repeatability respectively[6]. An ELISA, a dot-ELISA and an indirect fluorescent antibody test are also available[1]. A new commercial ELISA is now available[9], but although it is well validated, this test is not in common use for import/export testing[6].

Living and inactivated vaccines are used in several countries. The use of live blood vaccines can lead to clinical anaplasmosis[1].

Conclusion: Animals reacting to serological tests are likely to be either infected or vaccinated. Anaplasmosis is not a contagious disease and it can only be spread effectively if suitable tick vectors are present. The absence of competent vectors in New Zealand means that these agents would not become established even if infected animals were imported. Mechanical transmission might possibly result in isolated cases of disease. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) Wright IG, Leatch G. Bovine anaplasmosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 295. OIE, Paris, 1996.

(2) Stoltsz WH. Ovine and caprine anaplasmosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 431-8. Oxford University Press Southern Africa, Capetown, 1994.

(3) Potgieter FT, Stoltsz WH. Bovine anaplasmosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 408-30. Oxford University Press Southern Africa, Capetown, 1994.

(4) Peterson KL, Raleigh RJ, Stroud RK, Goulding RL. Bovine anaplasmosis studies conducted under controlled natural exposure in a *Dermacentor andersoni*=(*venestus*) indigenous area of Eastern Oregon. American Journal of Veterinary Research 38, 351-4, 1977.

(5) Potgieter FT, Sutherland B, Biggs HC. Attempts to transmit *Anaplasma marginale* with *Hippobosca rufipes* and *Stomoxys calcitrans.* Onderstepoort Journal of Veterinary Research 48, 119-22, 1981.

(6) de Vos B, Tick Fever Research Centre, Wacol, Australia. Personal communication with SC MacDiarmid, February 1999.

(7) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

(8) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(9) Molloy JB, Bowles PM, Knowles DP, McElwain TF, Bock RE, Kingston TG, Blight GW, Dalgliesh RJ. Comparison of a competitive inhibition ELISA and the card agglutination test for detection of antibodies to *Anaplasma marginale* and *Anaplasma centrale* in cattle. Australian Veterinary Journal 77(4), 245-9, 1999.

B102. Babesiosis

Bovine babesiosis, or redwater as it is commonly known, is a tick-borne disease caused by the intra-erythrocytic protozoan parasites, *Babesia bovis*, *B. bigemina*, *B. divergens* and *B. major*[1].

The distribution of bovine babesiosis in the world depends entirely on the distribution of its ixodid tick vectors[1]. The transmission of parasitaemic blood from an infected to a susceptible animal is theoretically possible by biting flies or veterinary instruments, but this appears to be unimportant under natural conditions[2].

B. bigemina occurs in South America, the West Indies, Australia and Africa. *B. bovis* occurs in the tropics including South and Central America, Australia, Asia and southern Europe. *Boophilus microplus* and *Boophilus annulatus* are the major vectors of *Babesia bovis* and *B. bigemina* worldwide, although in Africa, *Boophilus decoloratus* is the vector[1].

B. divergens occurs in north-west Europe, Spain, Eire where the vectors are *Ixodes persulcatus* and *Dermacentor reticulatus*[3]. *B. divergens* is the principal cause of babesiosis in the United Kingdom where *Ixodes ricinus* is the vector. *B. major* occurs in the United Kingdom and Europe[4].

None of these ticks are present in New Zealand[5]. *Haemaphysalis longicornis*, the New Zealand cattle tick, does not transmit *B. bigemina* or *B. major*[6].

Strong immunity develops after natural infection with most *Babesia* spp. Cattle may develop latent infections after recovery, which persist for varying periods of time in different breeds. European breeds of cattle can retain *B. bovis* infections for life, and remain infective for ticks up to 2 years, while most cattle with a significant zebu content lose the infection within 2 years. *Babesia bigemina* infections rarely persist for more than a year, regardless of the host, and infected cattle remain infective for ticks for only 4-7 weeks[1].

The calves born to immune mothers are resistant to infection. After the age of 2 months, a natural, non-specific, innate resistance protects calves. This resistance persists for at least a further 4-6 months and is not dependent on the immune status of the cow[1].

Vaccines consisting of live attenuated strains of *B. bovis*, *B. bigemina* or *B. divergens* are produced in several countries. They are mainly used in calves, as they are not entirely safe in older animals. Protective immunity develops in 3-4 weeks and lasts for several years after a single vaccination. Colostral immunity is not an issue for this disease, as calves fed colostrum from an immune or a non-immune dam are equally resistant to infection[7].

The indirect fluorescent antibody test (IFA) is the most widely used test for the detection of antibodies to *B. bovis* and *B. divergens*. The ELISA is also suitable for antibody detection. The IFA test has been used for the serology of *B. bigemina*, but serological cross-reactions make species diagnosis difficult[7].

Conclusion: Animals reacting to serological tests are likely to be either infected or vaccinated. However, babesiosis is not a contagious disease and it can only become established where competent vectors are present. The absence of competent vectors in New Zealand means that it would not become established even if infected animals were imported. The only way that any transmission could occur in this country would be through needle sharing, which might possibly result in isolated cases of disease. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) de Vos AJ, Potgieter FT. Bovine babesiosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 278-94. Oxford University Press Southern Africa, Capetown, 1994.

(2) Robertson A (ed). Handbook on Animal Diseases in the Tropics. 3rd Edition. P 167. British Veterinary Association, 1976.

(3) Blaha, T (ed). Applied Veterinary Epidemiology. P 116. Elsevier, Amsterdam, 1989.

(4) Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth Edition. P 746. Balliere Tindall, London, 1994.

(5) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

(6) Kuttler KL. Worldwide impact of babesiosis. In : Ristic M (ed). Babesiosis of Domestic Animals and Man. Pp 1-22. CRC Press, Boca Raton, 1988.

(7) de Vos AJ, Jorgensen WK. Bovine babesiosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 305. OIE, Paris, 1996.

B103. Bovine brucellosis *(B. abortus)*

Bovine brucellosis is a highly contagious bacterial disease, caused by the bacterium *Brucella abortus*. It is characterised by abortion and infertility in cows. It is also a serious zoonosis, causing undulant fever in humans. The organism may occasionally cause abortions in sheep and goats, but it does not spread in these species[(1)]. It is present worldwide but has recently been eradicated from a number of countries, including New Zealand[(2)].

Sexually mature heifers (and especially pregnant cattle) are more susceptible to infection than immature heifers. Infected animals usually abort only once; subsequent calves are carried to full term, although they may be infected. 90% of infected cows remain chronically infected, sometimes lifelong, with infection confined to the udder and lymph nodes. Up to 9% of heifers born from seropositive cows may be latently infected but serologically negative until the middle of their first gestation when antibodies are developed. Bulls may become infected *in utero* or in early calfhood and retain the infection into adult life. In bulls, testes and accessory sex glands may be affected, and organisms may be shed in semen[(1)].

Vaccination with strain 19 in animals older than 9 months of age can result in life-long serological reactions which cannot be distinguished from those resulting from natural infections. Moreover, vaccination does not totally prevent infection with field strains of *B. abortus*; it is estimated to be only 70% effective against field challenge[(1)].

Post-vaccinal antibody titres are not as high as a result of vaccination with strain 45/20[(1)] and cattle vaccinated with strain RB51 fail to produce antibodies that are detectable by conventional serological tests[(3)].

Serological tests for international trade are the complement fixation test, the buffered brucella antigen tests and the ELISA[(4)].

Conclusion: Seropositive animals could be carriers of *B. abortus*.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Bishop GC, Bosman PP, Herr S. Bovine brucellosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1053-66. Oxford University Press Southern Africa, Capetown, 1994.

(2) Sabirovic M. Brucella abortus has been eradicated from New Zealand. Surveillance 24(1), 13, 1997.

(3) Steven MG, Hennager SG, Olsen SC, Cheville NF. Serological responses in diagnostic tests for brucellosis in cattle vaccinated with *Brucella abortus* 19 or RB51. Journal of Clinical Microbiology 32(4), 1065-6, 1994.

(4) Corbel MJ, MacMillan. Bovine brucellosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 242. OIE, Paris, 1996.

B104. Bovine genital campylobacteriosis

Bovine genital campylobacteriosis is a venereal disease characterised by infertility, early embryonic death and abortion, caused by the bacterium *Campylobacter fetus*.

The organism is carried on the prepuce of clinically normal carrier bulls. Bulls older than 3 years usually remain permanently infected[1].

Campylobacteriosis is rare in New Zealand cattle, and was last identified in 1992[2]. The disease is not under regulatory control.

Diagnosis in bulls is by culture and identification of the organism from preputial washings. Serological tests (agglutination, ELISA) can be applied to vaginal mucus in infected herds, to assist in the diagnosis of an infertility problem caused by the organism. As sensitivity is low, serological testing is not useful for identifying individual infected cows[3].

Conclusion: Serological tests are not generally applicable to individual animal diagnosis.

References

(1) Schutte AP, van der Walt ML, Bishop GC. Genital campylobacteriosis in cattle. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1010-7. Oxford University Press Southern Africa, Capetown, 1994.

(2) Loveridge R, Gardner E. Campylobacter fetus venerealis infection in cattle. Surveillance 20(4), 26, 1993.

(3) Guerín B. Bovine genital campylobacteriosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 256. OIE, Paris, 1996.

B105. Bovine tuberculosis

Bovine tuberculosis is caused by the bacterium *Mycobacterium bovis*. Transmission between animals is by droplet infection. The disease is endemic in New Zealand, and it is under a compulsory control programme (a Pest Management Strategy) administered by the Animal Health Board[(1)]. One of the rules of the Pest Management Strategy is that animals which react positively to diagnostic tests for tuberculosis must be slaughtered.

A number of serological tests are now recognised by the OIE[(2)]; the lymphocyte proliferation assay, the gamma-interferon test and the ELISA. However, the sensitivity and specificity of these tests have not been fully defined and the delayed hypersensitivity (skin) test is still the standard test for international trade.

Conclusion: If serological tests were to be applied in international animal trade, a positive serological test would be interpreted as indicating an infected animal.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) O'Neil BD, Pharo HJ. The control of bovine tuberculosis in New Zealand. New Zealand Veterinary Journal 43(7), 249-55, 1995.

(2) Haagsma J. Bovine tuberculosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 267. OIE, Paris, 1996.

B106. Cysticercosis (*C. bovis*)

Bovine cysticercosis is caused by the larval stages of the human tapeworm *Taenia saginata.* *Cysticercus bovis* is the name given to the pea-sized cysts in striated muscles of cattle, which are detectable only by postmortem inspection. Infestation of humans arises from eating undercooked meat. The tapeworm in humans is of minor public health significance. A number of attempts have been made to develop serological tests for *C. bovis* but the sensitivity and specificity of such tests has been low, and no practical individual animal test is available[(1)].

Conclusion: Serology diagnosis is not applicable for this parasite.

References

(1) Soulsby EJL. Cysticercosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 314. OIE, Paris, 1996.

B107. Dermatophilosis

Dermatophilosis is an exudative pustular dermatitis, caused by the bacterium *Dermatophilus congolensis.* It affects many domestic animals. The agent is endemic and widespread in New Zealand, and not under any form of regulatory control.

Although serological tests have been developed for research purposes, they are not available for general application[1].

Conclusion: Serology is not applicable for this disease

References

(1) Lloyd DH. Dermatophilosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 318. OIE, Paris, 1996.

B108. Enzootic bovine leukosis

Enzootic bovine leukosis is a disease of adult cattle, and occasionally sheep, caused by the bovine leukemia virus, which is the type species of the genus *"BLV-HTLV retroviruses"* in the family *Retroviridae*[1].

Infection with the virus in cattle is lifelong, giving rise to a persistent antibody response. Most infections are asymptomatic. Up to 30% of infected cattle develop persistent lymphocytosis, but only 1% of infected cattle develop lymphosarcoma, and only after a very long period. Moreover, disease may be confined to susceptible family groups[2].

High antibody titres develop in sheep, but persistent lymphocytosis has not been observed. It is not certain whether goats become infected or not[2].

Infection is endemic in New Zealand. A national control programme is in place for the dairy cattle industry, in which 7.5% of herds are infected although only 0.2% of herds have a within-herd prevalence greater than 10%. There is no programme in the beef cattle industry, but testing of beef animals entering dairy herds suggests that the virus is present at a very low level in beef herds in New Zealand[3].

Transmission between herds is predominantly by movement of infected animals. Iatrogenic transmission at dehorning, blood testing and vaccination is the main route of transmission within herds. Congenital transmission from dam to offspring has been demonstrated although this seems to occur in less than 10% of infected dams, and the mechanism is unclear[2].

Serological methods most widely used are agar gel immunodiffusion (AGID) on serum and ELISA on serum or milk[4].

Antibodies can be detected 3-16 weeks after infection. Maternally-derived antibodies may take up to 6 or 7 months to disappear. There is no way of distinguishing passively transferred antibodies from those developed following infection with the virus. Passive antibody tends to protect calves against infection but may just prolong the incubation period. Cows may have undetectable serum antibody during the periparturient period because of a shift of antibody from the dams circulation to her colostrum. A negative test result on serum taken at this time (2-3 weeks pre- and post-partum) is therefore not conclusive and should be repeated, especially when using the AGID test[4].

Conclusion: Seropositive animals are likely to be infected for life. Offspring of such animals may be infected at birth.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 200, 1995.

(2) Verwoerd DW, Tustin RC. Enzootic bovine leukosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 778-82. Oxford University Press Southern Africa, Capetown, 1994.

(3) Hayes D, Burton L. Enzootic bovine leucosis eradication scheme. Surveillance 25(4), 3-5, 1998.

(4) Lucas MH. Enzootic bovine leukosis. Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 276-7. OIE, Paris, 1996.

B109. Haemorrhagic septicaemia

Haemorrhagic septicaemia (HS) is an acute, highly fatal bacterial septicaemia of cattle and buffaloes caused by *Pasteurella multocida* serotype B or E. It occurs almost exclusively in Asia (serotype B) and Africa (serotype E), in countries with a high and very seasonal rainfall. Various stresses (especially the sudden onset of the rainy season, with an associated drop in temperature) appear to be associated with outbreaks. Carrier animals probably play an important role in maintaining a reservoir of the agent through other seasons[(1)].

P. multocida serotype A is present in many species in New Zealand, where it is involved in respiratory diseases such as enzootic pneumonia in many species, snuffles in rabbits etc. However there is no clinical or laboratory evidence that other serotypes of *P. multocida* are present in New Zealand[(2)].

The diagnosis of HS is based on clinical signs and is confirmed by isolation of the causative organism from infected animals. Serological tests are not normally used for diagnosis[(3)].

Conclusion: Serology is not applicable for diagnosis of this disease

References

(1) Bastianello SS, Nesbit JW. Haemorrhagic septicaemia. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1180-3. Oxford University Press Southern Africa, Capetown, 1994.

(2) Fairley R. Respiratory diseases of New Zealand cattle. Surveillance 23(4), 15-6, 1996.

(3) De Alwis MCL. Haemorrhagic septicaemia. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 331. OIE, Paris, 1996.

B110. Infectious bovine rhinotracheitis / infectious pustular vulvovaginitis

Infectious bovine rhinotracheitis / infectious pustular vulvovaginitis is caused by bovine herpesvirus 1 (BHV-1), which is a member of the genus *Varicellovirus* in the subfamily *Alphaherpesvirinae,* family *Herpesviridae*[1]. BHV-1 is distributed worldwide. Several subtypes of the virus can be distinguished by DNA restriction enzyme analysis. The OIE recognises subtypes 1.1 and 1.2a ("IBR-like") and subtype 1.2b ("IPV-like")[2]. The encephalitic bovine herpesviruses which were previously classified as BHV-1 subtypes 1.3a and 1.3b[3] are now classified as bovine herpesvirus 5 (BHV-5) [1].

A mild respiratory strain of BHV-1 is widespread and prevalent in New Zealand, especially in dairy cattle[4]. Surveys have shown that up to 100% of cattle are seropositive in some areas, but disease is relatively rare. According to laboratory records most clinical cases occur in 2-year-old dairy heifers after they have joined the milking herd in January and February. In these animals there is typically a mild upper respiratory tract disease with recovery over 7-14 days. Outbreaks of respiratory disease in yearlings and conjunctivitis in calves have also been attributed to the virus[5]. Clinical evidence suggests that the BHV-1 subtypes which cause severe respiratory disease and abortion are not present in New Zealand[4].

REA typing carried out in the 1980s found that fourteen New Zealand bovine herpesvirus isolates (6 genital and 8 respiratory) were indistinguishable from Australian respiratory and genital isolates, but were quite distinct from encephalogenetic herpesviruses (now known to be BHV-5) from Australia and elsewhere[6]. Subsequent REA typing placed all the New Zealand viruses in the 1.2b subtype[3]. A BHV-1 virus from bovine semen was REA typed in 1996 and was also found to be subtype 1.2b[7]. Subtypes 1.1 and 1.2a have never been isolated in this country.

Bovine herpesviruses have a tendency to become latent following primary infection. Animals with such latent infections will usually have detectable antibody. Stress may cause reactivation of the infection with shedding of the virus. Carrier cattle are therefore important in the spread of bovine herpesviruses[8].

Vaccines usually prevent the development of clinical signs and reduce shedding of the virus after infection[2]. In the early nineties about 10% of New Zealand dairy herds were vaccinated using an attenuated New Zealand strain of the virus[3]. However, that vaccine is no longer produced, and the use of live vaccines in production animals is no longer permitted. Two killed vaccines are licensed for use in New Zealand.

Serological tests used to detect antibody are the virus neutralisation test and the ELISA[2]. Serological tests cannot distinguish between antibodies resulting from infection and antibodies resulting from vaccination. It is also not currently possible to differentiate which subtype an animal is infected with on the basis of the serological response. That can be done by isolating the virus and typing it using restriction endonuclease analysis[2].

Conclusion: Most cattle with humoral antibody are latently infected. Although one mild subtype of the virus is widespread in New Zealand, the two more pathogenic subtypes are exotic. Seropositive animals may be carriers of the exotic subtypes.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 120, 1995.

(2) Van Oirschot JT. Infectious bovine rhinotracheitis / infectious pustular vulvovaginitis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 281. OIE, Paris, 1996.

(3) Smith GA, Young PL, Reed KC. Emergence of a new bovine herpesvirus 1 strain in Australian feedlots. Archives of Virology, 140, 599-603, 1995.

(4) Horner GW. Infectious bovine rhinotracheitis in New Zealand. Surveillance 17(2), 25-6, 1990.

(5) Fairley R. Respiratory diseases of New Zealand cattle. Surveillance 23(4), 15-6, 1996.

(6) Brake F, Studdert MJ. Molecular epidemiology and pathogenesis of ruminant herpesviruses including bovine, buffalo and caprine herpesviruses 1 and bovine encephalitis herpesvirus. Australian Veterinary Journal 62, 331-4, 1985

(7) Horner G, National Centre for Disease Investigation, Wallaceville, New Zealand. Unpublished.

(8) Barnard BJH, Collett MG. Infectious bovine rhinotracheitis / infectious pustular vulvovaginitis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 932-41. Oxford University Press Southern Africa, Capetown, 1994.

B111. Theileriosis

Theileriosis is caused by tick-transmitted protozoa in the genus *Theileria*. East Coast Fever is a severe non-contagious disease of cattle caused by *Theileria parva parva,* occurring in eastern and central Africa. Related bovine theilerioses caused by other members of the *T. parva* complex include Corridor disease (*T. parva lawrenci*) and Zimbabwe theileriosis (*T. parva bovis*). A similar disease in east Africa is caused by *T. mutans*, although it is usually non-pathogenic. Mediterranean coast fever or tropical theileriosis is caused by *T. annulata* in North Africa, southern Europe and Asia. *T. orientalis* is a relatively benign infection of cattle and Asian buffalo[1].

However, the taxonomy of ovine theileriosis is confused, and there are differences of opinion as to whether *T. orientalis* is a single species or a complex of several species[2]. Benign ovine theileriosis in Africa caused by *T. ovis* or *T. separata. T. hirci* causes malignant ovine theileriosis, a disease of sheep similar to *T. annulata* infection in cattle, which occurs from North Africa through the middle East to India[1].

The distribution of the theilerioses is determined by the distribution of specific tick vectors which are an essential part of the life cycle as a complex development cycle of the disease agent takes place in them. East coast fever is naturally transmitted only by *Rhipicephalus appendiculatus*[3]; this tick is also the main vector for the related bovine theilerioses of the *T. parva* complex[4, 5]. *T. mutans* is transmitted by several species of tick in the genus *Amblyomma*[6]. *T. annulata* is transmitted by two- and three-host ticks of the genus *Hyalomma*[7]. The ovine theilerioses are transmitted by *Rhipicephalus* spp. and *Hyalomma* spp[8].

T. orientalis is mainly confined to Southeast Asia, but it also occurs in Australia, where it is transmitted by *Haemaphysalis longicornis* and *H. bancrofti. T. orientalis* also occurs in the north of New Zealand[9]. It was most probably introduced into New Zealand in symptomless carrier animals and subsequently was able to spread throughout the northern half of the North Island where *H. longicornis* is endemic[10].

Ticks can only be infected if they have fed on cattle with circulating piroplasms. Infective cattle can be clinically ill, recently recovered, or persistent carriers. Calves appear resistant, (although not in the case of tropical theileriosis) and most likely this is due to something other than the colostral antibody passed to them by their immune dams[3].

Apart from tick transmission, artificial transmission of theileriosis between cattle by blood or tissues is only irregularly successful, and accidental transmission through contaminated instruments or mechanical transmission by biting flies has never been proven, and it is not likely to be of any significance[11].

Diagnosis of theileriosis is usually by examination of stained blood and lymph node smears. Serological testing is not commonly used to diagnose theileriosis because of the problems of cross-reactivity among some *Theileria* species[12].

Conclusion: Animals reacting to serological tests are likely to be either infected or vaccinated. Theileriosis is not a contagious disease and it cannot be spread effectively without the presence of specific tick vectors. The absence of competent vectors in New Zealand for the pathogenic species of *Theileria* means that these agents would not become established even if infected animals were imported. The only way that transmission could occur would be through needle sharing, which might possibly result in isolated cases of disease. Nevertheless, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) Lawrence JA, de Vos AJ, Irwin AD. Theilerioses. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 307-8. Oxford University Press Southern Africa, Capetown, 1994.

(2) Kawazu S, Kamio T, Kakuda T, Terada Y, Sugimoto C, Fujisaji K. Phylogenetic relationships of the benign *Theileria* species in cattle and Asian buffalo based on the major piroplasm surface protein (p33/34) gene sequences. International Journal for Parasitology, 29 (4) 613-618, 1999.

(3) Lawrence JA, de Vos AJ, Irwin AD. East Coast Fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 309-25. Oxford University Press Southern Africa, Capetown, 1994.

(4) Lawrence JA, de Vos AJ, Irwin AD. Corridor Disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 326-8. Oxford University Press Southern Africa, Capetown, 1994.

(5) Lawrence JA, de Vos AJ, Irwin AD. Zimbabwe Theileriosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 329-30. Oxford University Press Southern Africa, Capetown, 1994.

(6) Lawrence JA, de Vos AJ, Irwin AD. Theileria mutans infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 336-7. Oxford University Press Southern Africa, Capetown, 1994.

(7) Pipano E. Theileria annulata theileriosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 341-8. Oxford University Press Southern Africa, Capetown, 1994.

(8) Robertson A (ed). Handbook on Animal Diseases in the Tropics. 3rd Edition, Pp 188-90. British Veterinary Association, 1976.

(9) James MP, Saunders BW, Guy LA, Brookbanks EO, Charleston WAG, Uilenberg G. Theileria orientalis, a blood parasite of cattle; first report in New Zealand. New Zealand Veterinary Journal 32, 154-6, 1984.

(10) Thompson J. Theileriasis in New Zealand. Surveillance 18(5), 21-23, 1991.

(11) Robertson A (ed). Handbook on Animal Diseases in the Tropics. 3rd Edition, Pp 180. British Veterinary Association, 1976.

(12) Dolan TT, Morzaria SP, Katende JM. Theileriosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 321. OIE, Paris, 1996.

B112. Trichomonosis

Trichomonosis, caused by the flagellate protozoan *Tritrichomonas foetus*, is a non-febrile, sexually transmitted disease confined to the reproductive tract of the cow and the preputial sac of the bull. It was at one time of major economic importance as a cause of irregular returns to service, early abortions and some cases of pyometra, especially in dairy cattle. With the widespread use of artificial insemination, its significance has declined along with its prevalence in most countries. Although it was last diagnosed in New Zealand in 1975[1], it is still considered to be endemic[2], and it is not under regulatory control.

Diagnosis in the male is by microscopic examination of preputial washings. An agglutination test is available for use on vaginal mucus[3].

Conclusion: The organism is endemic in New Zealand, and is not of regulatory concern, and serological testing is unlikely to be carried out on imported animals for this agent.

Recommendation: *caveat emptor*

References

(1) Food and Agriculture Organization of the United Nations. Animal Health Yearbook 1995. P 160. FAO, Rome, 1997.

(2) Bruere SN. Trichomonas foetus infection in a beef herd. New Zealand Veterinary Journal 30, 15-6, 1981.

(3) Soulsby EJL. Trichomonosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 291. OIE, Paris, 1996.

B113. Trypanosomosis

Trypanosomosis results from infection with parasitic protozoa of the genus *Trypanosoma*. There is considerable variation in the pathogenicity of trypanosomes. The most pathogenic forms for cattle are *T. vivax*, *T. congolense*, *T. evansi* and *T. brucei*. In infections with the more pathogenic forms, there is intermittent fever accompanied by parasitaemia, whereas in the less pathogenic forms there can be high parasitaemia in the absence of clinical signs[1].

T. evansi, which causes the disease surra in many countries, is a non-pathogenic trypanosome which is found in cattle all over the world, and which is spread by biting flies in the family *Tabanidae*[1]. Only one trypanosome is transmitted without any insect vector - *T. equiperdum*, which causes dourine in horses, is transmitted venereally[1]. Surra and dourine are considered separately in this document (see B202 and B215).

In Africa, where the trypanosomosis is of greatest importance, transmission is by bloodsucking flies of the genus *Glossina*, commonly known as "tsetse flies". These flies are restricted to Africa, where they are distributed over 11 million square kilometres between 14 ˚N and 29 ˚S. These limits are determined by climate, often through its effect on vegetation[2]. Tsetse flies require an ambient temperature of 15-35˚C to function and breed successfully, and are restricted to frost-free areas that have an annual rainfall of 650 mm or more. While wild animals coexist with tsetse flies and trypanosomes without problems, large parts of the continent are effectively closed to cattle due to the presence of tsetse flies, and it is only in livestock that are introduced into such areas, particularly *Bos taurus* breeds of cattle, that disease (intermittent fever, anaemia, and loss of condition) occurs[1].

Tsetse flies ingest trypanosomes when they feed on the blood of infected animals. Within the fly, a cycle of development and maturation takes place, which lasts up to 45 days, after which after which the trypanosome is transmitted to vertebrate hosts as the fly feeds. Transmission is either by inoculation of trypanosomes with saliva (salivarian trypanosomes), or by contamination of mucosa or broken skin with trypanosomes in the vector's faeces, voided during the blood meal (stercorarian trypanosomes)[1].

T. vivax is the only species of tsetse-borne trypanosome that has become permanently established outside Africa. It is present in Mauritius and South America; the herd prevalence in Colombia varies considerably between different geographical regions, but it can be up to 95%[3]. This obviously raises questions regarding possible vectors other than tsetse flies[4].

Mechanical transmission of trypanosomes on the mouthparts of biting flies has been accepted as possible in Africa, although trypanosomosis has not shown to be endemic anywhere in Africa in the absence of tsetse[5]. But as trypanosomes survive for only a short time outside the mammalian or tsetse fly host, a fly with contaminated mouthparts would have to feed on a susceptible host within a few minutes of the infective meal in order to transmit the parasite mechanically[1]. There is only a small amount of experimental data and no convincing field data to suggest that any such transmission is of importance[6].

Therefore, the validity of the widely-held view that mechanical transmission by biting flies in the genera *Tabanus*, *Stomoxys* and *Lyperosia* are probably responsible for maintaining *T. vivax* in countries outside Africa[1] is uncertain. It is, after all, an assumption which is based solely on the absence of the tsetse fly[3], and it may be that cyclical transmission in an as yet unidentified vector other than *Glossina* spp. is a more likely explanation[5].

Although the absence of tsetse flies and most of the above biting flies makes it unlikely that trypanosomosis would be able to become established in New Zealand, it may be possible that the stable fly *Stomoxys calcitrans*, which is present in New Zealand[7], could be responsible for limited transmission. Relatively little is known about the distribution, population density, ecology and natural history of *S. calcitrans* in this country, but it appears to be widespread in the dairying areas of the north island where it breeds especially in silage stacks[8]. Therefore the possibility cannot be excluded that this insect may be present in sufficient numbers to allow some transmission of trypanosomes if they were introduced, although it is unlikely that establishment would be possible.

Because of the varying parasitaemia and the lack of specificity of clinical signs, the diagnosis of trypanosomosis can be extremely difficult. A number of serological tests are available, the most precise of which is currently the indirect fluorescent antibody test. An antigen detection ELISA is also available, and this may be of value in detecting latently infected animals[9].

Conclusion: Animals which are serologically positive for trypanosomosis are likely to be carriers. The absence of tsetse flies makes it unlikely that trypanosomosis would be able to become established in New Zealand. Some transmission may be possible by the stable fly, but it is unlikely to be sufficient to allow the parasite to become established. Moreover, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) Connor RJ. African animal trypanosomiases. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 167-205. Oxford University Press Southern Africa, Capetown, 1994.

(2) Phelps RJ, Lovemore DF. Vectors: tsetse flies. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 25-52. Oxford University Press Southern Africa, Capetown, 1994.

(3) Wells EA, Ramirez LE, Betancourt A. *Trypanosoma vivax* in Colombia: interpretation of field results. Tropical animal Health and Production 14, 141-50, 1982

(4) Foil L Tabanids as vectors of disease agents. Parasitology Today 5(3), 88-96, 1989.

(5) Wells EA. The importance of mechanical transmission in the epidemiology of Nagana: a review. Tropical Animal Health and Production 4, 74-88, 1972.

(6) Krinsky WL. Animal disease agents transmitted by horse flies and deer flies (Diptera: Tabanidae). Journal of Medical Entomology 13(3) 225-75, 1976.

(7) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(8) Todd DH. The biting fly *Stomoxys calcitrans* (L.) in dairy herds in New Zealand. New Zealand Journal of Agricultural Research 7, 60-79, 1964.

(9) Schlater J. Trypanosomosis (Tsetse-borne). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 660. OIE, Paris, 1996.

B114. Malignant catarrhal fever

Bovine malignant catarrh or malignant catarrhal fever (MCF) is a sporadic but almost invariably fatal viral disease of cattle, buffalo and deer. The causative viruses are unassigned members of the subfamily *Gammaherpesvirinae*, in the family *Herpesviridae*[1].

There are two forms of MCF. In Africa the disease is caused by the alcelaphine herpesvirus-1 (AHV-1), the natural host of which is the wildebeest. This virus causes MCF in cattle and deer in Africa and in a variety of ruminants in zoological collections worldwide. The sheep-associated form of the disease is caused by ovine herpes virus-2 (OHV-2) and is the cause of MCF in most regions of the world[2].

In susceptible species the disease is characterised by profuse muco-purulent nasal and ocular discharges, corneal opacity and nervous signs. The disease occurs worldwide. Neither wildebeest nor sheep show any signs of infection. Deer appear more susceptible than cattle[3].

Most free-living adult wildebeest are persistently infected with AHV-1, but adult wildebeest probably only excrete virus under conditions of severe stress, such as following capture or in zoos. However, about 40% of wildebeest calves excrete non-cell-associated virus in ocular and nasal secretions up to about 3 months of age, which is thought to be responsible for transmission to cattle in Africa[3]. Close contact with wildebeest or sheep is necessary for transmission, which is thought to be by the upper respiratory tract[4].

Cattle appear to be dead end hosts and do not naturally transmit the virus, probably because the levels of virus in nasal secretions are low and cell-associated[3]. There is only circumstantial evidence that deer to deer transmission can occur[4].

Sheep-associated MCF is endemic in New Zealand, and the majority of sheep can be expected to be carriers. The disease in cattle and deer occurs sporadically and is not under regulatory control[5].

The few cattle that survive infection with wildebeest-derived MCF are resistant to reinfection for at least several years if not for life[4].

Diagnosis is usually based on history, clinical signs, and pathology[3]. Serological tests are available but are of limited use in clinically affected animals, as only a small proportion of cattle develop humoral antibody responses late in the course of disease. In addition, serological cross-reactions occur between herpesviruses and even where antibody is produced in measurable quantities, it is present in low concentration. Therefore interpretation of serological results is difficult[3]. PCR techniques are now available, and are more useful in diagnosing infection. Serology may be used to detect subclinically infected wildebeest and domestic sheep. However, antibody to OHV-2 has been detected only by using AHV-1 as the source of antigen, and most sheep would be expected to test positive[2].

Conclusion: Sheep-associated MCF is endemic in New Zealand, and not under regulatory control, so testing of imported animals for OHV-2 is not necessary. Most adult wildebeest, whether serologically positive or not, would probably be carriers of AHV-1. Therefore testing of imported wildebeest seems unnecessary.

Recommendation: Wildebeest, whether serologically positive or not, could not be released from PAQ without an appreciation of the risk of AHV-1 . Such animals should remain in a transitional facility physically separated from cervidae and bovidae. Seropositive cattle would be expected to be encountered very rarely as the disease is almost invariably fatal in this species. However, as cattle are dead-end hosts they are not epidemiologically significant, and therefore seropositive cattle could be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 124, 1995.

(2) Reid HW. Malignant catarrhal fever. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 627. OIE, Paris, 1996.

(3) Barnard BJH, van der Lugt JJ, Mushi EZ. Malignant catarrhal fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 946-57. Oxford University Press Southern Africa, Capetown, 1994.

(4) Plowright W. Malignant catarrhal fever virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 123-50. Elsevier, Amsterdam, 1990.

(5) Horner G. Malignant catarrhal fever in New Zealand. Surveillance 23(2), 16-8, 1996.

B115. Bovine spongiform encephalopathy

BSE is a fatal, non-contagious, predominantly food-borne neurological disease of adult cattle, caused by an unconventional infectious agent.

There is currently no basis for serological tests, as no immunological response has been detected in affected animals[1].

Conclusion: Serology is not applicable for the diagnosis of this disease.

References

(1) Wells GAH, Bradley R, Dawson M, Wilesmith J. Bovine spongiform encephalopathy. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 338. OIE, Paris, 1996.

7. LIST B SHEEP AND GOAT DISEASES

B151. *Brucella ovis* infection

The bacterium *Brucella ovis* produces a clinical or subclinical disease in sheep characterised by genital lesions and infertility in rams and, in rare cases, placentitis in ewes. The organism is excreted in the semen of infected rams, and infection is spread ram to ram by homosexual activity or venereally during coitus when non-infected rams mate with ewes which passively harbour the bacteria[(1)]. The disease is widespread in New Zealand, and a voluntary control scheme is in place[(2)].

The agent is considered to be highly adapted to sheep and is not a zoonosis[(3)]. However, it has recently been recognised as a naturally occurring disease in farmed deer in New Zealand[(4)].

Infection is transient in ewes, but it persists in rams even though they develop a strong antibody response[(1)].

Serological tests available include agar gel immunodiffusion (AGID), complement fixation (CF) and the ELISA. A combination of the AGID and the ELISA seems to give the best results in terms of sensitivity, but the prescribed test for international trade remains the CF test[(3)].

<u>*Conclusion:*</u> A positive serological test is indicative of a carrier animal. However, this disease is endemic and not under regulatory control. Serological testing of imported animals for this agent is not a regulatory requirement.

<u>*Recommendation:*</u> *caveat emptor*

References

(1) Tonder EMV, Herr S, Bishop GC, Bosman PP. *Brucella ovis* infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1067-72. Oxford University Press Southern Africa, Capetown, 1994.

(2) Chief Veterinary Officer. Annual Report 1994. Surveillance 22(3),15, 1995.

(3) Garin-Bastuji B. Ovine epididymitis (*Brucella ovis*). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 343. OIE, Paris, 1996.

(4) Bailey K. Naturally acquired *Brucella ovis* infection in a deer. Surveillance 24(3), 10-1, 1997.

B152. Caprine and ovine brucellosis *(B. melitensis)*

Brucella melitensis (biovars 1, 2, or 3) is the main causative agent of caprine and ovine brucellosis which causes abortion, retained placenta, orchitis, epididymitis and arthritis. The organism is highly pathogenic for humans, and is a serious zoonosis worldwide. Infection of humans is often by ingestion of raw goats' milk[(1)].

Spread between animals is by contact with genital discharges of infected sheep and goats, which may contain large numbers of organisms. Transmission is usually by inhalation. Young animals usually recover spontaneously from infection, but mature animals usually remain carriers for long periods. Congenital latent infection is suspected but has never been proven[(1)].

The rose bengal plate agglutination test and the complement fixation test are usually recommended for screening flocks and individual animals. Vaccinated animals may become persistent reactors[(2)].

Conclusion: Recovered animals are usually long term carriers.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Herr S. Brucella melitensis infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1073-5. Oxford University Press Southern Africa, Capetown, 1994.

(2) Garin-Bastuji B. Caprine and ovine brucellosis (exc luding *Brucella ovis* infection). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 350. OIE, Paris, 1996.

B153. Caprine arthritis / encephalitis

Within the family *Retroviridae*, viruses in the genus *Lentivirus* are divided into five groups, one of which is the caprine/ovine lentivirus group[1]. Although the caprine arthritis-encephalitis (CAE) virus and the maedi-visna virus have long been considered two distinct, albeit very closely related, members of this group, recent nucleic acid sequence data indicates that these two viruses are not as distinct as previously thought, and it has been suggested that it is now more appropriate to consider these as "small ruminant lentiviruses"(SRLVs) rather than as separate viruses[2].

Three biological properties of SRLVs lend themselves to persistent infection. They can sequester themselves in host cells by integrating their proviral DNA into host cell DNA, they replicate preferentially in macrophages, and they do not usually induce virus neutralising antibodies[3].

There is considerable genetic variation between strains of SRLVs. In general terms, the genome of field viruses from either goats and sheep differ from one another by about 1-10% of their nucleotide sequence[4]. Studies of the virus envelope gene have shown that there is about 85% homology between strains from goats[5], versus about 60% homology when comparing strains from goats and sheep[6].

There is some uncertainty regarding the host specificity of SRLVs[7]. Experimentally viruses from either sheep or goats can infect animals of the reciprocal species and many field strains of ovine lentivirus have biological properties similar to those of caprine viruses[8].

It has been suggested that cross-species infections may result in evolution of SRLVs to suit their new host[8]. Some phylogenetic studies have indicated that north American ovine strains may have originated from CAE or a CAE-like virus, possibly by natural cross-species transfer with subsequent adaptation of the virus in its new ovine host[8]. However, other studies have suggested that French and US caprine SRLVs emerged from a much more diverse group containing all SRLVs infectious for sheep[9].

Maedi and visna are Icelandic names denoting the two most common forms of disease in sheep infected with SRLVs, namely maedi (dyspnoea) and visna (wasting). These diseases were first identified in South Africa in 1915, followed by USA and Iceland over the next two decades[7]. There now seems to be a widespread geographical distribution of these diseases in sheep[10]. Most infected sheep show little or no signs of disease, but remain carriers and can transmit the infection to others. Clinical signs are variable, including lymphoproliferative pneumonia, encephalitis, non-suppurative arthritis and lymphocytic mastitis, but they are rarely seen in animals younger than 3-4 years. The course of the disease may be up to a year, but there is no recovery once clinical signs are manifested[7].

CAE is a relatively new disease, and its epidemiology is still being evaluated[11]. This evaluation is complicated by the close serological relationship between CAE virus and maedi-visna virus, together with the observation that arthritis and mastitis may also occur in maedi-visna of sheep[3].
Serological surveys have shown that the CAE virus is widely disseminated in goat herds in North America, Europe and Australasia, and the prevalence in individual herds may be as high as 60-70%, but in the majority of infected herds the prevalence is around 20-40%. However, only a small fraction of infected animals show clinical disease. Three different disease syndromes in different age groups of

goats are recognised. These include rapidly progressive leukoencephalitis and pneumonia in newborn and young goats, chronic arthritis and mastitis in adult goats, and a sporadic slowly progressive pneumonia and encephalitis in adult goats[3].

Transmission of SRLVs is mainly via colostrum or milk[11]. Transmission of may also occur between adult sheep under conditions of close contact, presumably by the transfer of bodily secretions[7].

CAE virus was first isolated in New Zealand in 1981. Infection is considered to be endemic in goats, and it appears to be more common in dairy goats than in goats raised for fibre. A voluntary flock accreditation scheme has been in operation since 1984[12].

Active and passive surveillance of sheep has demonstrated that ovine lentiviruses are not present in New Zealand[13]

A number of serological tests are available for the SRLVs[14], but the different isolates cannot be distinguished serologically by conventional techniques such as immunodiffusion, immunofluorescence or ELISA[7].

Conclusion: Serologically positive animals will be persistent carriers of small ruminant lentiviruses, and these could be new and possibly more virulent strains. Cross-species infections may be possible, and exotic strains might be able to cross species barriers more readily than endemic strains. Serological tests cannot differentiate between viruses from sheep and those from goats.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 202, 1995.

(2) Pasick J. Maedi-visna virus and caprine arthritis-encephalitis virus: distinct species or quasispecies and its implications for laboratory diagnosis. Canadian Journal of Veterinary Research 62(4), 241-4, 1998.

(3) Narayan O, Cork LC. Caprine arthritis-encephalitis virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 441-52. Elsevier, Amsterdam, 1990.

(4) Rosati, S, Kwang J and Keen JE. Genome analysis of North American small ruminant lentiviruses by polymerase chain reaction and restriction enzyme analysis. J. of Vet. Diag. Invest. 7,437-443, 1995.

(5) Knowles DP, Cheevers WP, McGuire TC, Brassfield AL, Harwood WG, Stem TA. Structure and genetic variability of envelope glycoproteins of two antigenic variants of caprine arthritis-encephalitis lentivirus. Journal of Virology, 65, 5744-5750, 1991.

(6) Saltarelli M, Querat G, Konings DAM, Vigne R, Clements JE. Nucleotide sequence and transcriptional analysis of molecular clones of CAEV which generate infectious virus. Virology 179, 347-364, 1990.

(7) Verwoerd DW, Tustin RC, Williamson AL. Maedi-visna. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 792-6. Oxford University Press Southern Africa, Capetown, 1994.

(8) Karr BM, Chebloune Y, Leung K, Narayan O. Genetic characterisation of 2 phenotypically distinct North American ovine lentiviruses and their possible origin from vaprine arthritis -encephalitis virus. Virology 225, 1-10, 1996.

(9) Valas S, Benoit C, Guionaud C, Perrin G, Mamoun RZ. North-American and French caprine arthritis -encephalitis viruses emerge from ovine maedi-visna viruses. Virology, 237(2) 307-318, 1997.

(10) Petursson G, Georgsson G, Palsson PA. Maedi-visna virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 431-40. Elsevier, Amsterdam, 1990.

(11) Verwoerd DW, Tustin RC. Caprine arthritis -encephalitis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 797-9. Oxford University Press Southern Africa, Capetown, 1994.

(12) Chief Veterinary Officer. Annual Report 1994. Surveillance 22(3),16, 1995.

(13) Thornton R, Motha J. A serological survey to confirm New Zealand's freedom from maedi-visna. Surveillance 22(2), 22-24, 1995.

(14) Dawson M, Lysons RE. Caprine arthritis/encephalitis & Maedi-visna. Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 369. OIE, Paris, 1996.

B154. Contagious agalactia

Contagious agalactia is a disease complex of sheep and goats, mainly in Europe, western Asia and North Africa, which manifests as mastitis, arthritis and keratoconjunctivitis. The clinical condition has been known for 170 years[1] but the cause is not completely clear[2]. It was originally associated only with *Mycoplasma agalactiae*, but there are now three other mycoplasmas that have been shown to cause similar diseases, sometimes accompanied by pneumonia - *M. capricolum* subsp. *capricolum*, *M. mycoides* subsp. *mycoides LC*, and *M. putrefacens* can produce a similar clinical picture, particularly in goats[1]. *M. arginini* may also be involved[2]. This confusing picture complicates the diagnosis and international reporting of cases of contagious agalactia.

Of the above mycoplasmas, only *M. arginini* is seen in New Zealand. Together with *M. ovipneumoniae* and *Acholeplasma laidlawii*, it is commonly recognised in the New Zealand sheep and goat populations[3, 4].

Mycoplasma infections tend to be chronic in both flocks and in individuals, and prolonged symptomless shedding of mycoplasmas may occur, especially in the milk. Transmission is mainly by ingestion or by direct entry into the teat[5].

Detection of serum antibodies by complement fixation, growth inhibition or ELISA is useful in diagnosis and epidemiological studies, but it does not replace identification of the causal organism for specific diagnosis[1].

Conclusion: *Mycoplasma* spp. carriers can be detected by serology, but there are significant complexities surrounding the aetiology and diagnosis.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Lefèvre PC. Contagious agalactia. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 363. OIE, Paris, 1996.

(2) Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth Edition. P 914. Balliere Tindall, London, 1994.

(3) Belton D. Mycoplasmas of sheep and goats in New Zealand. Surveillance 17(2), 18-19, 1990.

(4) Belton D. Abattoir surveillance of mycoplasmas in the lungs and udders of New Zealand goats. Surveillance 23(1), 21, 1996.

(5) Timoney JF, Gillespie JH, Scott FW, Barlough JE. Hagan and Brunner's Microbiology and Infectious Diseases of Domestic Animals. Eighth Edition. Pp 304-305. Cornell University Press, 1988.

B155. Contagious caprine pleuropneumonia

Contagious caprine pleuropneumonia (CCPP) is a serious contagious disease of goats caused by *Mycoplasma capricolum* subsp. *capripneumoniae* (strain F38)[1]. CCPP seems to be restricted to North Africa, the Mediterranean, and southern Asia[2].

The diagnosis of outbreaks of respiratory disease in goats in general, and of CCPP in particular, is complicated. The F38 strain is closely related to three other Mycoplasmas: *M. mycoides* subsp. *mycoides*, *M. mycoides* subsp. *capri*, and *M. capricolum* subsp. *capricolum*, all of which may confuse the diagnosis of CCPP both because the diseases they produce can resemble the classical disease and because they share several serological and biochemical characteristics with F38[1].

Pleuropneumonia has never been diagnosed in goats in New Zealand, and surveys for mycoplasmas have never isolated strain F38[3, 4].

Transmission is by inhalation of infectious aerosols. Outbreaks of the disease often occur after heavy rains and after cold spells. Latently infected animals are often responsible for spreading the disease between herds and regions[5].

Definitive diagnosis requires isolation of the organism. The complement fixation test is the most commonly used serological test. It is less sensitive than the indirect haemagglutination test. An ELISA is also being developed[1].

Conclusion: *Mycoplasma* spp. carriers can be detected by serology, but there remain significant complexities surrounding the aetiology and diagnosis.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Rurangirwa FR. Contagious caprine pleuropneumonia. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 374. OIE, Paris, 1996.

(2) Thiaucourt F, Bölske G. Contagious caprine pneumonia and other pulmonary mycoplasmoses of sheep and goats. Rev. sci. tech. Off. int. Epiz. 15(4), 1397-1414, 1996.

(3) Belton D. Mycoplasmas of sheep and goats in New Zealand. Surveillance 17(2), 18-19, 1990.

(4) Belton D. Abattoir surveillance of mycoplasmas in the lungs and udders of New Zealand goats. Surveillance 23(10, 21, 1996.

(5) Mare CJ. Contagious caprine pneumonia. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1495-7. Oxford University Press Southern Africa, Capetown, 1994.

B156. Enzootic abortion of ewes

Enzootic abortion of ewes (ovine chlamydiosis) is caused by a sheep-specific strain of *Chlamydia psittaci* . It is a common cause of abortion in Europe on intensively managed sheep farms. New Zealand is free of ovine chlamydiosis[(1)]. Following abortion, ewes remain infected and continue to excrete the organism seasonally in uterine discharges and faeces. Transmission is mainly by ingestion, through ewes grazing contaminated lambing fields[(2)].

Diagnosis depends on the identification of large numbers of organisms in the products of abortion or the vaginal excretions of freshly aborted ewes[(3)].

A killed vaccine is available; it does not prevent or eliminate infection, but it does reduce the incidence of abortion. A complement fixation test is available, but there are problems with lack of sensitivity and specificity, and the test does not distinguish between responses to vaccination and infection[(3)].

Conclusion: Carriers exist, and serological testing is imperfect.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Thornton R. Chlamydial abortion in sheep. Surveillance 24(2), 18-9, 1997.

(2) Blaha, T (ed). Applied Veterinary Epidemiology. Pp 166-71. Elsevier, Amsterdam, 1989.

(3) Aitken ID. Enzootic abortion of ewes (ovine chlamydiosis). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 384. OIE, Paris, 1996.

B157. Pulmonary adenomatosis

Pulmonary adenomatosis or jaagsiekte ("driving disease") is a contagious neoplasm which affects the lungs of mature sheep, and rarely goats. It is caused by virus which belongs to the genus *Type D Retroviruses* in the family *Retroviridae*[1].

Jaagsiekte occurs worldwide, but not in Australia or New Zealand[2].

The disease has a protracted course but is invariably fatal. Annual mortality in infected flocks varies from less than 1% to as high as 25%[3]. The disease is often closely associated with *Lentivirus* infections[4].

Transmission is by droplet infection, and outbreaks occur when infected sheep are introduced into clean flocks[3].

Diagnosis is by clinical and postmortem signs; there is no method to detect subclinically infected sheep[3].

Conclusion: Serology is not applicable for diagnosis of this disease.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 199-200, 1995.

(2) Hosie BD. Pulmonary adenomatosis and maedi: exotic pneumonias of sheep. Surveillance 18(1), 27-28, 1991.

(3) Verwoerd DW, Tustin RC, Williamson AL. Jaagsiekte. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 783-91. Oxford University Press Southern Africa, Capetown, 1994.

(4) Verwoerd DW. Jaagsiekte (ovine pulmonary adenomatosis) virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 453-63. Elsevier, Amsterdam, 1990.

B158. Nairobi sheep disease

Nairobi sheep disease (NSD) is a tick-transmitted viral disease of small ruminants, especially sheep, caused by a virus belonging to the genus *Nairovirus* in the family *Bunyaviridae*[1]. The disease is severe, characterised by fever, haemorrhagic gastroenteritis, abortion and a high mortality (up to 90%). It occurs mainly in East and Central Africa, but may extend as far north as Ethiopia and Somalia, and as far south as Botswana[2]. An apparently identical virus, Ganjam virus, causes a similar disease of sheep in India[3].

NSD is not contagious and it can be transmitted only by specific ticks. *Rhipicephalus appendiculatus* is by far the most efficient vector, and the only one in which transovarial transmission has been demonstrated, but other species of *Rhipicephalus* and *Amblyomma* ticks can occasionally act as vectors[3]. None of these ticks are present in New Zealand[4].

In an area endemic for NSD most sheep and goats carry antibody for the virus, and only incidental losses are observed. Outbreaks of NSD may arise either as a result of the movement of susceptible animals into endemic areas or the incursion of infected ticks into NSD-free flocks or areas. The latter situation may occur in years with excessive or prolonged rains which result in vegetation and microclimatic changes favourable for an extension in the range of *R. appendiculatus*[2].

The disease results in a short-lived viraemia, and recovered animals do not carry the virus. The incubation period is 4-6 days after tick attachment. Fever lasts 1-7 days, and is accompanied by viraemia, which disappears within 24 hours of the temperature returning to normal. Recovered animals are immune for life[2].

Effective vaccines have been produced experimentally, but there is little demand for their use in the field. Antibodies can be demonstrated by complement fixation, virus neutralisation, agar gel immunodiffusion, indirect immunofluorescence, and indirect haemagglutination[5].

Conclusion: There is no carrier state in recovered animals.

Recommendation: Seropositive animals could be safely released from PAQ, and their offspring would not pose a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 310, 1995.

(2) Terpstra C. Nairobi sheep disease. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 718-22. Oxford University Press Southern Africa, Capetown, 1994.

(3) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. Pp 169-72. Australian Government Publishing Service, Canberra, 1995.

(4) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

(5) Davie FG. Nairobi sheep disease. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 669. OIE, Paris, 1996.

B159. Salmonellosis (*S. abortus ovis*)

Salmonella abortus ovis is one of four salmonellas incriminated as causes of abortion in sheep. It is endemic in parts of Europe, and abortions due to *S. dublin* and *S. typhimurium* occur endemically in parts of England as well as Europe[(1)].

In New Zealand, outbreaks of abortion have occurred periodically in New Zealand since the winter of 1997, caused by *S. brandenburg*[(2)], and isolated cases of *S. typhimurium* abortion occur periodically.

Sheep which have recovered from clinical disease may become subclinical carriers and excrete organisms in their faeces intermittently. Some sheep which do not abort can also become carriers. Infection is predominantly by the oral route[(3)].

Although experimentally serological testing has been used to measure flock immunity against *S. abortus ovis*[(4)], diagnosis of infections in individual animals is by bacterial culture[(1)].

Conclusion: Serology is not applicable for the diagnosis of this disease.

References

(1) Aiello S (ed). The Merck Veterinary Manual. Eighth Edition. P 994. Merck & Co, Whitehouse Station, 1998.

(2) Bailey KM. Sheep abortion associated with *Salmonella* Brandenburg. Surveillance 24(4), 10-11, 1997.

(3) Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth Edition. P 747. Balliere Tindall, London, 1994.

(4) Sanchis R, Abadie G, Pardon P. Subcutaneous and conjunctival vaccination with a live attenuated strain of *Salmonella* Abortusovis: effect of gestation on serological response of ewes. Veterinary Research 26, 110-5, 1995.

B160. Scrapie

Scrapie is a transmissible spongiform encephalopathy of sheep and goats. It has been recognised in Great Britain and Western Europe for 250 years, predominantly in sheep. Although it is generally accepted that scrapie is an infectious, contagious disease, the means of natural transmission are not understood[(1)]. There appears to be a genetic influence on susceptibility and incubation period, and there is evidence for a dose-response relationship[(2)]. Infectivity is associated with an abnormal isoform of a host-encoded cellular glycoprotein PrP^{C}. The abnormal form, PrP^{SC}, which is protease resistant, may be the scrapie agent or may be somehow coupled to the agent, but its presence is specific to the diseased state[(1)].

The principal method of postmortem confirmation of clinical scrapie remains histopathological examination of the brain. Scrapie infection is not known to elicit any specific immune response and there is no basis for establishing a diagnosis by detecting specific antibodies[(3)].

Conclusion: Serology is not applicable for the diagnosis of this disease.

References

(1) Detwiler LA. Scrapie. Rev. sci. tech. Off. int. Epiz., 11 (2), 491-537, 1992.

(2) Hoinville LJ. A review of the epidemiology of scrapie in sheep. Rev. sci. tech. Off. int. Epiz., 15 (3), 827-852, 1996.

(3) Wells GAH. Scrapie. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 673-7. OIE, Paris, 1996.

B161. Maedi-Visna

Retroviruses in the *Lentivirus* genus are divided into five groups, one of which is the caprine/ovine lentivirus group[1]. Although the caprine arthritis-encephalitis (CAE) virus and the maedi-visna virus have long been considered two distinct, albeit very closely related, members of this group, recent nucleic acid sequence data indicates that these two viruses are not as distinct as previously thought, and it has been suggested that it is now more appropriate to consider these as "small ruminant lentiviruses"(SRLVs) rather than as separate viruses[2].

For this reason, in this document the consideration of maedi-visna is included in the section on CAE (B153).

Conclusion: Serologically positive animals are likely to be persistent carriers of small ruminant lentiviruses. Cross-species infections may be possible, and serological tests cannot differentiate between viruses from sheep and those from goats.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 202, 1995.

(2) Pasick J. Maedi-visna virus and caprine arthritis-encephalitis virus: distinct species or quasispecies and its implications for laboratory diagnosis. Canadian Journal of Veterinary Research 62(4), 241-4, 1998.

8. LIST B HORSE DISEASES

B201. Contagious equine metritis

Contagious equine metritis is an inflammation of the endometrium of mares caused by *Taylorella equigenitalis* infection, usually resulting in temporary infertility. Recovery is uneventful, but recovered mares can carry the infection for years without showing signs. Transmission is most commonly by sexual contact with carrier stallions, which do not show signs but carry the organism on external genitalia[(1)].

Diagnosis is by bacterial culture. Serum antibody does not persist significantly beyond clinical disease, and is absent in carrier mares and stallions. Therefore serological tests are not able to detect infection reliably[(2)].

Conclusion: Serology is not relevant for the diagnosis of this disease.

References

(1) Henton MM. *Taylorella equigenitalis* infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1510-3. Oxford University Press Southern Africa, Capetown, 1994.

(2) Chanter N. Contagious equine metritis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 389. OIE, Paris, 1996.

B202. Dourine

Dourine is a chronic infectious disease of horses, mules and donkeys, caused by the venereally transmitted protozoan parasite *Trypanosoma equiperdum*. The disease is characterised by oedema of the external genitalia and ventral abdomen. It differs from other trypanosomes in that it does not require an arthropod vector[(1)].

As identification of the agent is rarely possible, diagnosis is based on clinical signs together with serological evidence. The CF test is used to detect specific humoral antibodies which are present whether clinical signs exist or not. Non-inflected animals, especially donkeys, often yield inconsistent results. The indirect fluorescent antibody test is used to confirm infection or resolve inconclusive CF test results[(2)]. An ELISA is also available, and although it seems not to be completely validated[(2)] it may be preferable to the CF test in terms of antigen stability[(1)].

Conclusion: Serological tests can detect clinical cases and carriers.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Barrowman P, Stoltsz WH, van der Lugt JJ, Williamson CC. Dourine. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 206-12. Oxford University Press Southern Africa, Capetown, 1994.

(2) Donnelly J. Dourine. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 394. OIE, Paris, 1996.

B203. Epizootic lymphangitis

Epizootic lymphangitis is a contagious chronic systemic fungal disease of horses, mules and donkeys characterised by spreading ulcerating dermal nodules, conjunctivitis or pneumonia. It is caused by a dimorphic saprophytic soil fungus, *Histoplasma farciminosum*[(1)]. The fungus is yeast-like in its parasitic phase in horses, but exists as a mycelium in its saprophytic soil phase. The organism retains its virulent form in the soil for 15 days[(2)].

Transmission is by contamination of traumatised skin (grooming, harness equipment), biting flies (of the *Musca* or *Stomoxys* genera), or inhalation. The clinical form of the disease seems to vary with the route of entry; not all clinical cases present obvious lymphangitis[(1)]. Typical epizootic lymphangitis runs a chronic course for as long as a year. There is considerable loss of condition and animals are unable to work. Most animals eventually develop a solid immunity and recover. Cell-mediated immunity seems to be important in resistance to infection[(3)].

Antibodies to *H. farciminosum* develop at or before the onset of clinical signs. Assays reported for antibody detection include fluorescent antibody, ELISA, and haemagglutination inhibition. In addition, a skin hypersensitivity test has been described[(1)]. It is not known how long recovered animals are seropositive or whether such animals would be carriers of the agent.

The disease is endemic in parts of Africa (especially North Africa), the middle East and Asia. However, it is now rarely reported; it used to be more common historically when large numbers of horses were stabled together for cavalry or other transportation needs[(1)].

Although the disease has not been reported in New Zealand, it is difficult to imagine that opportunities for the introduction of this saprophytic soil fungus would not have occurred historically. It is also not clear whether the fungus could become established in New Zealand if introduced, but the fact that the disease is confined to countries with hot climates suggests that it probably could not. Nevertheless, as *Stomoxys calcitrans* is present in this country[(4)], some caution may be warranted.

Conclusion: Serological tests may be used for detecting animals which have been infected. It is not known how long recovered animals are seropositive or whether such animals would be carriers of the agent. Spread by *Stomoxys calcitrans* may be possible.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Brown C. Epizootic lymphangitis. Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 457-8. OIE, Paris, 1996.

(2) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. Pp 321-3. Australian Government Publishing Service, Canberra, 1995.

(3) Timoney JF, Gillespie JH, Scott FW, Barlough JE. Hagan and Brunner's Microbiology and Infectious Diseases of Domestic Animals. Eighth Edition. Pp 405-6. Cornell University Press, 1988.
(4) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

B204. Equine encephalomyelitis

Eastern, Western and Venezuelan equine encephalomyelitis (EEE, WEE and VEE respectively) are diseases caused by members of the genus *Alphavirus* in the family *Togaviridae*. The diseases are confined to the Americas, where they are mainly seen in horses. The viruses also cause serious human disease, and EEE and WEE viruses have caused outbreaks of disease in poultry and various species of farmed birds[(1)].

The epidemiology of EEE, WEE and VEE viruses is complex, involving normal cycles between wild animals and mosquitoes in specific environments with spillover into man and horses only under certain conditions[(1)].

The primary vector of EEE in the USA is the ornithophilic mosquito *Culiseta (Climacura) melanura*, which frequents wetlands in the southern and southeastern USA, particularly those with peat-muck soil dominated with hardwood trees. These wet and murky habitats and hardwood root systems favour oviposition by *C. (C.) melanura* as its larvae require water with a high content of organic matter and protection from sunlight for development. Female *C. (C.) melanura* feed almost exclusively on birds, especially passerines, which maintain the cycle of infection by the development of high viraemias. Because *C. (C.) melanura* is highly ornithophilic, other vectors are probably responsible for effecting the escape of the EEE virus from the bird/mosquito cycle. *Coquillettidia perturbans* is suspected of fulfilling this role, as it feeds on mammals (including humans and horses) and birds equally. Cases of EEE tend to occur within 8 km of swampy habitats, which is within the flight range of this mosquito[(1)]. In tropical areas of the Americas the primary vectors include *Culex taeniopus*, *Cx. nigripalpus*, and *Aedes taeniorhynchus*. In addition, *Cx. panacossa* and *Cx. dunni* seem to be involved in endemic maintenance of the EEE virus[(2)].

The primary vectors of WEE are irrigation ditch mosquitoes, particularly *Cx. tarsalis*, which in western North America maintains WEE virus in a cycle with birds, in particular nestling passerines. An abundance of *Cx. tarsalis* is favoured by a rapid increase in temperature following a cold, wet, spring, resulting in the rapid melting of snow and flooding of rivers. These mosquitoes also have a predilection for irrigated lands as breeding sites. Other ornithophilic mosquitoes become infected as the summer progresses, and the infection spills over to other types of birds, mammals and reptiles, which in turn allows other vectors with different host preferences (including horses and humans) to become involved. In other parts of the Americas there are other cycles, such as a jackrabbit / *Aedes* mosquito cycle in California, and in South America the cycle has not been identified[(1)].

EEE and WEE disease occurs sporadically in horses and humans from mid-summer to late autumn, but as humans and horses generally do not develop a high enough viraemia to re-infect mosquitoes, they are regarded as "dead-end" hosts. Disease in horses is characterised by fever, anorexia, and severe depression. EEE virus infection in horses is often fatal, while WEE virus can cause a subclinical or mild disease with less than 30% mortality[(1)].

Many different subtypes and varieties of VEE virus exist, each of which seems to have a restricted geographical location in the tropical rainforests and coastal swamps of Northern South America. The

natural ecology of the virus is unknown, but it is assumed that there is a natural "endemic" cycle between mosquitoes and forest rodents. A wide variety of blood sucking insects have been found to be naturally infected with VEE virus, including at least 41 species from 11 genera of mosquitoes, particularly rodent-feeding species of the genus *Culex*. Up to 114 species of birds have been either virologically or serologically associated with virus transmission. Shore birds in general, and herons in particular, appear to be capable of serving as amplifier hosts. Other animals such as bats, marsupials, rats, sloths, rabbits, racoons and non-human primates may also be involved. Epidemics in horses occur irregularly, and the triggering mechanism is unknown. However, it is believed that "epizootic viruses" arise periodically from minor variants present in the "enzootic virus" populations[(1)].

The primary vectors for EEE, WEE and VEE viruses do not exist in New Zealand[(3)]. Although it is not known whether any of the 15 species of mosquitoes present in New Zealand could be competent vectors for these viruses, in view of the highly specific insect vectors, hosts and ecosystems that are involved in the maintenance of these viruses in North and South America, it is considered very unlikely that the virus could become established in New Zealand even if viraemic animals were introduced. Moreover, horses are dead-end hosts for EEE and WEE viruses, due to the low viraemias produced, and although horses infected with VEE virus may have high viraemias which are sufficient to infect mosquitoes, the duration of viraemia is no longer than five days[(1)].

A long-lasting sterile immunity results from both inapparent infection and recovery from clinical disease. Safe and immunogenic vaccines are available for EEE and WEE[(4)].

Antibody can be identified by plaque reduction neutralisation, haemagglutination inhibition, and complement fixation tests or IgM capture ELISA[(4)].

Conclusion: Seropositive reactions would be expected in vaccinated or recovered animals, neither of which will be virus carriers.

Recommendation: Seropositive animals could be safely released from PAQ in New Zealand, and the offspring of such animals would not constitute a biosecurity risk.

References

(1) Thomson GR. Equine encephalitides caused by alphaviruses. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 636-41. Oxford University Press Southern Africa, Capetown, 1994.

(2) Calisher CH, Walton TE. Japanese, Western, Eastern and Venezuelan Encephalitides. In : Studdert MJ (ed). Virus Infections of Equines. Pp 141-55. Elsevier, Amsterdam, 1996.

(3) Belkin JN. Mosquito Studies VII. The culicidae of New Zealand. Contributions of the American Entomological Institute Vol 3, No. 1, 1968.

(4) Pearson JE, Walton TE. Equine encephalomyelitis (Eastern and Western). In : Office Intemational des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 400-5. OIE, Paris, 1996.

B205. Equine infectious anaemia

Equine infectious anaemia (EIA), colloquially known as "swamp fever", is an viral disease of horses and other Equidae, caused by the sole member of the equine lentivirus group of the *Lentivirus* genus in the family *Retroviridae*[1].

The clinical signs of EIA are highly variable. Clinical manifestations have been arbitrarily defined as acute and chronic. In acute disease, the signs may include pyrexia and depression, but anaemia is not a common feature. If the animal survives the acute episode, it is classified as a chronic case, characterised by intermittent bouts of fever, anaemia, and progressive weight loss. Ninety percent of these bouts occur in the first year after infection, following which the animal becomes an inapparent carrier. However, some infections either result in no clinical signs or they show signs so mild that they are not noticed by their owners[2]. Asymptomatically infected animals and animals which recover from clinical disease remain carriers for life[3]. Such animals are the only virus reservoirs[2].

The virus is distributed worldwide[4], but is not present in New Zealand[5].

EIA virus is most frequently transmitted between horses in close proximity through the mechanical transmission of blood by large blood-sucking insects, such as horse flies (*Tabanus spp*. and *Hybomitra spp*.), deer flies (*Chrysops spp.*), and stable flies (*Stomoxys calcitrans*)[2]. For this reason the disease is particularly prevalent in low-lying, humid and swampy areas, particularly in summer when horse flies abound[3]. At such times biting rates by horse flies may be up to 1000 bites per hour[6].

Iatrogenic transmission via instruments and other equipment contaminated by infected blood may be locally important in endemic areas[3]; the virus can survive in blood-contaminated hypodermic needles for up to 4 days[2].

Although horse flies are not present in New Zealand, the stable fly *Stomoxys calcitrans*, is present[7].

There is no vaccine[8].

The agar gel immunodiffusion test is the most commonly used serological technique. Several ELISAs are available, and they are slightly more sensitive than the AGID[8].

Conclusion: Recovered animals are carriers for life, and transmission by stable flies is possible.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 202, 1995.

(2) Cook RF, Issel CJ, Montelaro RC. Equine Infectious Anaemia. In : Studdert MJ (ed). Virus Infections of Equines. Pp 297-323. Elsevier, Amsterdam, 1996.

(3) Verwoerd DW, Tustin RC. Equine infectious anaemia. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 800-2. Oxford University Press Southern Africa, Capetown, 1994.

(4) Ishii S. Equine infectious anaemia or swamp fever. Advances in Veterinary Science and Comparative Medicine 8, 263-98, 1963.

(5) Horner GW. Infectious disease status of New Zealand horses. Surveillance 16(2), 14-6, 1989.

(6) Cordes T, Issel C. Equine Infectious Anaemia - a status report on its control. United States Department of Agriculture Animal and Plant Health Inspection Service

(7) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(8) Coggins L. Equine infectious anaemia. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 406. OIE, Paris, 1996.

B206. Equine influenza

Equine influenza is an acute and highly contagious respiratory disease of horses caused by infection with type A influenza viruses which are members of the genus *Influenzavirus A* in the family *Orthomyxoviridae*[1]. Equine influenza occurs all over the world except for Australia and New Zealand. The disease is mainly seen in horses, but other members of the Equidae family are also susceptible. Coughing and fever are the most common clinical signs. Mortality rates are low in uncomplicated cases, except for young foals which have no maternal antibody[2].

Subtypes of influenza virus are defined by the antigenic character of haemagglutinin and neuraminidase. Only 2 combinations have been identified so far in horses : H7N7 and H3N8. These are known as subtype 1 and subtype 2 respectively[3]. Antigenic drift occurs within these subtypes. Minor antigenic drift has been detected within subtype 1, but more extensive drift has been detected within subtype 2, to the extent that variants have been identified. Major or subtype changes occur as a result of recombinational events with other influenza viruses and are called antigenic shifts. Antigenic shift gives rise to new viruses which can result in pandemics in susceptible populations[4].

Influenza viruses in horses affect the upper and lower respiratory tract epithelium, causing a frequent and harsh cough. This seems to enable the spread of the virus in aerosols over distances of up to around 30 metres. Infection is believed to be transmitted almost exclusively between infected horses, but indirect spread by fomites (people and contaminated vehicles) may also be involved[2].

Many factors influence the epidemiology of the disease, but most important in recent years has been the development of rapid transport of horses by air[2].

The virus can be isolated from nasopharyngeal secretions during the acute phase of disease. In immunologically naive horses virus excretion may persist for 7-10 days, but infected animals are rarely infectious for longer than 10 days[2]. In partially immune horses virus shedding is transient[4]. Long term carriers have been postulated, but this has not been proven[2].

Immunity is short lived. Clinical immunity lasts less than one year, and immunity to infection may be even shorter, allowing reinfection to occur without clinical signs within months[2].

Vaccination is widely used, particularly for horses travelling for competition or breeding purposes. Vaccines are usually inactivated whole virus vaccines containing at least one representative of each subtype. Many vaccines include 2 strains of subtype 2[2]. Complex vaccination protocols have been developed, particularly since the 1979 epidemic in Europe, which was largely attributed to vaccine breakdown[5].

Vaccine efficacy has been studied extensively. There is a clear relationship between the haemagglutinin content of vaccines, the antibody level to haemagglutinin in vaccinated horses, and protection against challenge. Repeated vaccination usually results in durable antibody responses eventually, but this can be highly variable among individual horses. More than 5% of horses may fail to produce an adequate

antibody response after two doses of vaccine, and even after three doses a small number of individuals fail to respond[2]. Further, the efficacy of vaccination is influenced by the exposure dose of virus[4].

The frequency of vaccination of mares determines the level and duration of maternal immunity provided to foals, which in turn affects the response of foals to primary immunisation, since maternally-derived antibody may inhibit the foal's antibody response[2].

Excretion of the virus by clinically normal inadequately vaccinated horses has been identified as a significant contributor to the spread of infection[4].

ELISA techniques for direct detection of the virus in nasal secretions are available[4].

Serological tests for diagnosis must be carried out on paired sera. Antibody titres are determined by haemagglutination inhibition or single radial haemolysis[3].

Conclusion: If serological testing of horses were carried out, a positive serological test would indicate either vaccination or prior infection. Introduction of the virus in seropositive animals could occur with the importation of clinically normal vaccinated horses which are excreting the virus, and it may also be possible with importation of recovered animals which are long term carriers or which have become reinfected without showing clinical signs.

Recommendation: Seropositive animals with a certified history of vaccination could be released from PAQ in New Zealand with relative safety, but it is not possible to be certain that seropositive animals would not introduce the virus.

References

(1) International Committee on the Taxonomy of Viruses. ICTV taxomonic proposals approved in Jerusalem meeting in 1996. http://www.ncbi.nlm.nih.gov/ICTV/Taxpro96.html

(2) Mumford JA. Equine influenza. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 854-9. Oxford University Press Southern Africa, Capetown, 1994.

(3) Mumford JA. Equine influenza. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 409. OIE, Paris, 1996.

(4) Hannant D, Mumford JA. Equine Influenza. In : Studdert MJ (ed). Virus Infections of Equines. Pp 285-93. Elsevier, Amsterdam, 1996.

(5) Plateau E, Jacquet A, Cheyroux M. A study of the serological response of horses to influenza vaccination: comparison of protocols and types of vaccines. In : Powell DG (ed). Equine infectious diseases V; proceedings of the 5th international conference. University Press of Kentucky, Lexington, 1988.

B207. Equine piroplasmosis (Babesiosis)

Equine babesiosis is an acute, subacute or chronic tick-borne protozoal disease of horses, mules, donkeys and zebras caused by the intra-erythrocytic protozoa *Babesia equi* and *B. caballi*. The disease can be characterised by fever, progressive anaemia, jaundice, redwater and abortion[1].
Equine babesiosis is widespread in Europe and former USSR, is probably indigenous in Asia, and occurs throughout Africa and Central and Southern America. It was introduced into Florida in 1958 from Cuba, but did not spread to other parts of the USA[1].

B. equi has been introduced to Australia on a number of occasions from two different sources; in quarter-horses from Texas during the 1950s and 1960s, and in Andalusian horses from Spain on three occasions in the 1970s. Although infection spread from the quarter-horses by iatrogenic transmission, there was no spread from the Andalusians despite opportunities for tick transmission; these horses were kept together with susceptible horses in the presence of *Boophilus microplus*, and heavy burdens of *Haemaphysalis longicornis* and *Ixodes holocyclus*[2]. Quarantine of infected properties was lifted after 6 months, by which time it was established that there was no natural transmission. It was thought that the infection would die out in time, and it is now considered that *B. equi* is almost certainly exotic to Australia[3].

As is the case with babesiosis in other species, the world distribution of equine babesiosis depends on the distribution of its specific tick vectors[4]. Twelve species of Ixodid ticks in the genera *Dermacentor*, *Rhipicephalus* and *Hyalomma* have been identified as trans-stadial vectors of *B. equi* and *B. caballi*, and eight of those are also able to transmit *B. caballi* transovarially[5]. None of these ticks are present in New Zealand[6].

Ticks in the genus *Haemaphysalis* have not been reported as vectors[2, 7]. The only species of *Babesia* known to be transmitted by *Haemaphysalis longicornis*, which is present in this country, is *B. ovata*, which occurs as a symptomless infection of cattle in Japan[7].

There is no evidence of mechanical transmission of equine babesiosis by biting insects[1].

Infected animals may remain carriers for long periods and will act as sources of infection for vector ticks. After being infected with *B. caballi*, animals may remain carriers of the parasite for up to 4 years, while the carrier state is probably lifelong after infection by *B. equi*[1]. Prenatal infection of foals may occur[3].

No vaccine is available[1].

Infections in carrier animals may be detected by serological tests for specific antibodies. The complement fixation test is the primary test used for the importation of horses internationally. Other serological tests are the indirect fluorescent antibody, and the ELISA[5].

Conclusion: Recovered animals commonly remain carriers for long periods, and animals reacting to serological tests are likely to be infected. However, babesiosis is not a contagious disease and it can only become established where competent vectors are present. The absence of competent vectors in New Zealand means that equine babesiosis would not become established even if infected animals were imported. The only way that any transmission could occur in this country would be through needle sharing, which might possibly result in isolated cases of disease. However, with regard to vector-borne diseases on OIE lists A and B, it is the policy of the Ministry of Agriculture and Forestry that animals which are viraemic or parasitaemic will not be imported or released from post-arrival quarantine, regardless of whether or not competent vectors exist in New Zealand.

Recommendation: The above policy makes it impossible to allow seropositive animals to be released from PAQ in New Zealand, regardless of the biosecurity risk.

References

(1) de Waal DT, van Heerden J. Equine babesiosis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 295-304. Oxford University Press Southern Africa, Capetown, 1994.

(2) Callow LL. Equine babesiosis in Australia. In : Animal Health in Australia, Volume 5: protozoal and rickettsial diseases. Pp165-7. Australian Government Publishing Service, Canberra, 1984.

(3) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. P 377. Australian Government Publishing Service, Canberra, 1995.

(4) Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth Edition. P 746. Balliere Tindall, London, 1994.

(5) de Waal DT. Equine piroplasmosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 420. OIE, Paris, 1996.

(6) McKenna PB. The tick fauna of New Zealand. Surveillance 23(4), 27, 1996.

(7) Kuttler KL. Worldwide impact of babesiosis. In : Ristic M (ed). Babesiosis of Domestic Animals and Man. Pp 1-22. CRC Press, Boca Raton, 1988.

B208. Equine viral rhinopneumonitis

The OIE uses the name equine rhinopneumonitis as a collective term for a number of highly contagious clinical disease entities of *Equidae* which may occur as a result of infection by either of two closely related viruses, equine herpesvirus-1 and -4 (EHV1 and EHV4)[1]. They are members of the genus *Varicellovirus* in the subfamily *Alphaherpesvirinae*, family *Herpesviridae*[2]. These viruses are distributed worldwide, and can cause upper respiratory tract infections, abortions and neurological dysfunction[1].

EHV4 (previously known as subtype 2 or the R strain of EHV1) is endemic and widespread in New Zealand - serological evidence indicates that 80-90% of horses have been exposed. This virus causes sporadic abortions and respiratory disease which is most serious in foals 3-6 months of age[3, 4].

EHV1 (previously known as subtype 1 or the A strain of EHV1) causes abortion storms, neonatal deaths, rhinopneumonitis and meningoencephalomyelitis[5]. Serological studies indicate that 70% of horses in New Zealand have been exposed to EHV1[4], but abortion is rarely reported in this country and neurological disease has not been reported[3]. In New Zealand most horses seem to seroconvert early in life without showing clinical signs[4].

DNA subtyping of EHV1 variants suggests that the "B" variant is the most common cause of abortion in the USA, and the same variant has been isolated from abortion outbreaks in Australia[6]. DNA typing of a number of EHV1 samples collected in New Zealand from 1975-1988 identified the "P" variant but not the "B" variant[7].

Immunity resulting from natural infection of the respiratory tract is of short duration, and asymptomatic reinfection can occur within 3-4 months. Immunity against abortion is more durable, but unpredictable[6].

As with other herpesviruses, it is assumed that if a horse has antibodies to EHV1 or EHV4 it is latently infected and will carry the virus for life[6].

Inactivated vaccines are used widely in New Zealand, at least on stud farms. Passive immunity of foals is obtained from colostrum of immune dams[6].

The available serological tests include complement fixation, virus neutralisation, and ELISA[1]. Antibodies to EHV1 and EHV4 can be differentiated by two recently developed ELISAs[6, 8].

Conclusion: EHV1 and EHV 4 are endemic in New Zealand but it is assumed on the basis of clinical syndromes that occur here and abroad that there are exotic strains of these viruses in other countries. Serological testing cannot distinguish between strains of these viruses. Therefore, the only way to exclude exotic strains would be to exclude all seropositive animals. This would be difficult to justify.

Recommendation: *caveat emptor*

References

(1) Allen GP. Equine rhinopneumonitis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 426. OIE, Paris, 1996.

(2) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 120, 1995.

(3) Horner GW. Infectious disease status of New Zealand horses. Surveillance 16(2), 14-6, 1989.

(4) Donald J. Equine herpesviruses in New Zealand. Surveillance 25(3), 3-5, 1998.

(5) Mumford JA. Equine herpesvirus 1 and 4 infections. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 911-25. Oxford University Press Southern Africa, Capetown, 1994.

(6) Crabb BS, Studdert MJ. Equine rhinopneumonitis (equine herpesvirus 4) and equine abortion (equine herpesvirus 1). In : Studdert MJ (ed). Virus Infections of Equines. Pp 11-37. Elsevier, Amsterdam, 1996.

(7) Studdert MJ, Crabb BS, Ficorilli N. The molecular epidemiology of equine herpesvirus 1 (equine abortion virus) in Australasia 1975 to 1989. Australian Veterinary Journal 69, 104-11, 1992.

(8) Crabb BS, MacPherson CM, Reubel GH, Browning GF, Studdert MJ, Drummer HE, A type specific serological test to distinguish antibodies to equine herpesviruses 4 and 1. Arch. Virol. 140, 245-58, 1995.

B209. Glanders

Glanders is a contagious and fatal disease of horses, donkeys, and mules, and is caused by infection with the bacterium *Pseudomonas mallei* (previously named *Pfeifferella*, *Loefflerella*, *Malleomyces* or *Actinobacillus mallei*, and recently renamed *Burkholderia mallei* although it is still not widely known by this name)[(1)].

The disease causes nodules and ulcers in the upper respiratory tract. The skin form is called "farcy". It is also an important zoonosis - 95% of human cases are fatal. Glanders persists in many countries - eastern Europe, Asia, Africa, Myanmar, Mongolia, China. Acute glanders is more common in donkeys and mules; high fever, respiratory signs, death in a few days. In horses, glanders generally runs a chronic course; affected animals may survive for several years[(204)].

The mallein test is a sensitive and specific test for clinical glanders. The complement fixation test and ELISA are the most accurate and reliable serological tests[(1)].

Conclusion: Chronically infected horses would be detected by serological tests

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Whitwell KE, Hickman J. Glanders. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 434. OIE, Paris, 1996.

B210. Horse pox

Poxviruses are unimportant as causes of viral disease in equines. Currently there is only one such disease known, Uasin Gishu disease, which is an infection of African wildlife, occasionally transferred to horses. It is caused by an *Orthopoxvirus* ; infected horses show typical pox lesions intermittently for years[(1)].

In Europe, before vaccination campaigns against smallpox were discontinued, horses were quite frequently accidentally infected with vaccinia virus from recently vaccinated humans. The disease in horses took two forms - pox lesions in the mouth and on the lips, or lesions on the lower legs. Since smallpox vaccination stopped, the condition in horses has become rare[(2)].

Little is known about the epidemiology of infections causing horsepox, but transmission is probably by contact[(2)].

Diagnosis of pox virus infections is based on detection of virus particles in the skin by electron microscopy[(1)].

Conclusion: Serological diagnosis is not applicable for this disease.

References

(1) Fenner F. Poxvirus infections. In : Studdert MJ (ed). Virus Infections of Equines. Pp 5-8. Elsevier, Amsterdam, 1996.

(2) Munz E, Dumbell K. Horsepox. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 631-2. Oxford University Press Southern Africa, Capetown, 1994.

B211. Equine viral arteritis

Equine viral arteritis (EVA) is caused by a member of the genus *Arterivirus*, in the family *Arteriviridae*, order *Nidovirales*[1]. The virus has a limited host range, affecting only horses, donkeys, mules and perhaps zebras[2].

While most infections with EVA virus are subclinical, it may cause disease of varying severity, including acute respiratory disease, subcutaneous oedema, or abortion with or without clinical signs[3].

Transmission of EVA virus occurs through the respiratory, venereal or transplacental routes. The virus can be spread readily via the respiratory route by direct contact with infectious nasopharyngeal secretions from horses with acute respiratory symptoms. This is probably the primary means of spread of the virus to large numbers of animals[2]. In experimental infections, the virus is recoverable from the nasopharynx for up to 14 days[3]. Other forms of horizontal spread are via blood, faeces, lacrimal fluid, urine and vaginal secretions. Transplacental infection in late pregnancy can result in the birth of congenitally infected foals[2].

Infected stallions may shed the virus in semen continuously for years and perhaps for life, with no obvious negative effect on fertility, and venereal transmission occurs when persistently infected stallions are mated to mares by natural or artificial insemination[2]. More than 95% of mares served by shedder stallions will seroconvert within 3-5 weeks of mating, usually without clinical signs[3]. There is a close relationship between the induction of neutralising antibodies and the development of a protective immune response and the appearance of neutralising antibodies roughly coincides with elimination of the virus from the circulation. Mares do not appear to become persistently infected; the virus has been isolated from the reproductive tract only up to a month after infection[2].

The EVA virus appears to be distributed worldwide[2, 3]. The infection is endemic in New Zealand, where known shedder stallions continue to be used for breeding under an industry-based protocol[4].

Modified live and inactivated vaccines are available but they are not used widely in New Zealand[5]. Vaccination with the live vaccine provides clinical protection against EVA for more than a year, but it neither prevents infection nor limited replication of the challenge virus in vaccinated animals. Vaccination with the killed vaccine may not prevent infection and the development of viraemia, although it does protect pregnant mares from abortion. Both vaccines prevent stallions from becoming persistently infected[2].

Because of the existence of carrier stallions, and because most infections are subclinical and clinical signs are so variable, the diagnosis of infection with EVA virus is dependent on laboratory tests. A variety of serological tests have been used for the detection of antibody, including neutralisation, complement fixation, indirect fluorescent antibody, agar gel immunodiffusion, and the ELISA[3]. The test in widest use currently is a microneutralisation test; an ELISA offering similar specificity and sensitivity is not yet validated[6].

Conclusion: Positive serological reactions may be due to infection or vaccination. Seropositive males may be persistently infected.

Recommendation: Unvaccinated seropositive females could be safely released from PAQ if certification could show that they had not been mated in the previous month. Unvaccinated seropositive males animals could not be released from PAQ unless they were demonstrated by virus isolation or test mating protocols not to be shedders. For vaccinated animals, certification should prove that an appropriate vaccination protocol has been followed.

References

(1) International Committee on the Taxonomy of Viruses. ICTV taxomonic proposals approved in Jerusalem meeting in 1996. http://www.ncbi.nlm.nih.gov/ICTV/Taxpro96.html

(2) de Vries AAF, Rottier PJM, Glaser AL, Horzinek MC. Equine viral arteritis. In : Studdert MJ (ed). Virus Infections of Equines. Pp 171-200. Elsevier, Amsterdam, 1996.

(3) Mumford JA. Equine viral arteritis infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 658-62. Oxford University Press Southern Africa, Capetown, 1994.

(4) Ricketts W. Equine viral arteritis. Surveillance 25(3), 12-3, 1997.

(5) McKenzie J. Compulsory control program for equine viral arteritis. Surveillance 17(1), 19-20, 1990.

(6) Timoney PJ. Equine viral arteritis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 440. OIE, Paris, 1996.

B212. Japanese encephalitis

Japanese encephalitis (JE) virus is a member of the *Flavivirus* genus in the family *Flaviviridae*. It is spread by mosquitoes and is endemic throughout much of Asia, particularly southeast Asia and Japan[1]. Infection of humans and horses may cause a severe and often fatal encephalitis. Pigs are the primary amplifying host. Although clinical signs are not common in pigs in endemic areas, in immunologically naive sows there can be significant rates of abortion[2]. Inapparent infections also occur in goats, sheep, cattle and dogs, and they have been reported in cats, rodents, bats, snakes and frogs[3].

The main reservoir for the virus is water birds, particularly the Nankeen night heron, *Nycticorax caledonicus*[3]. This bird is common and widely distributed throughout the rice growing areas of Asia and Southeast Asia, and also occurs in coastal areas and river systems of the Pacific islands and Australia. In New Zealand up to 1994 this bird was an occasional vagrant from Australia, but it now appears that small numbers of them may be breeding along the Wanganui river. However only 9 birds were seen there in 1994[4].

The factors required for outbreaks of disease in humans and horses are the convergence in time and place of the virus, reservoir and amplifying hosts, susceptible humans or horses, and an abundance of suitable competent mosquito vectors[2].

The efficiency of domestic animal species as amplifying hosts for JE virus is inversely related to their longevity and directly related to their attractiveness to vectors, susceptibility to infection, and ability to sustain a viraemia of sufficient titre to infect biting mosquitoes over several days. In Asia cattle and buffaloes are long-lived, and their economic value as draft animals means that they are kept for many years. This, together with their short viraemias mean that they are not important as virus multipliers. Pigs, by contrast, rarely live longer than a year. Their value is as food, and they do not survive well during hot dry seasons. Consequently, many households obtain weanling piglets at the beginning of the rainy season. As colostrum-derived antibody levels wane, the piglets become susceptible to infection. Infected pigs develop viraemias which persist for about a week, serving as important sources of virus for the many mosquitoes which feed on them during this time. Solid immunity then develops, terminating the viraemias and ending the ability of these pigs to serve as amplifying hosts. However, the pigs are commonly slaughtered at the end of the cool season and their places are taken by new susceptible pigs in time for the next rainy season[2].

Flaviviruses exhibit a high degree of specificity in their ability to infect and be transmitted by individual insect species, or even strains of individual species. Vector competence is under genetic control, with the susceptibility of the midgut epithelium being the primary determinant. In a susceptible insect, a sufficient concentration of the virus must be ingested to exceed the midgut infection threshold[5]. Moreover, although 28 mosquito species have exhibited vector competence for JE virus in field and laboratory studies, only a few species found in endemic areas develop sufficient abundance, have long enough flight ranges, exhibit sufficient longevity, and have the breadth of host feeding preferences to become natural vectors[2].

In mainland Japan and Okinawa, the Philippines, the Korean peninsula, China, Taiwan, the Indochina peninsula (except Malaysia), Indonesia, Sri Lanka, India and Nepal, the primary vectors belong to the *Culex vishnui* group, mainly *Cx. tritaeniorhynchus*. However, other mosquitoes are important vectors locally. These include *Cx. vishnui* in India, Thailand and Taiwan, *Cx. fuscocephala* in Malaysia, Thailand and Taiwan, *Cx. gelidus* in Indonesia, Thailand and Vietnam, and *Cx. annulus* in Taiwan. The type of available larval habitat determines which species will predominate. Both *Cx. tritaeniorhynchus* and *Cx. vishnui* breed predominantly in rice paddies and are therefore the most important rural vectors[2].

None of these mosquitoes are present in New Zealand[6].

The time required for the development of arboviruses within the vector (i.e., the extrinsic incubation period, EIP) is a critical parameter in the epidemiology of arboviral transmission, because it determines how long an infected vector must survive before horizontal transmission can occur. Increasing temperatures shorten the EIP by synergistically enhancing the rate of viral replication and mosquito metabolism[7]. Mean daily temperatures between 25˚ - 30˚C are optimal for JE virus transmission, although limited transmission may occur over a wider range of ambient temperatures in endemic areas (between 20˚ - 30˚C)[2]. It has long been recognised that the transmission rate of mosquitoes infected with JE virus at temperatures below 20˚C is limited. For example, in one experiment the transmission rate in mosquitoes incubated at 28˚C exceeded 50% on day 9 and reached 100% on day 14, while at 20˚C the first virus secretion appeared on day 20 and the rate never reached 100% over the observation period of 35 days[8]. At temperatures below 20˚C the rate of viral replication in mosquitoes becomes so slow that mosquitoes will generally not transmit the virus during their lifespan. Multiplication of the virus stops completely when mosquitoes are held at 10˚C[8].

In northern temperate areas of Asia, outbreaks of Japanese encephalitis occur in late summer and autumn. Infection builds up in water birds in early spring, then spreads to pigs in late spring and early summer. Infection only spills over into humans and horses when mosquito populations expand past a certain threshold level, because *Cx. tritaeniorhynchus* preferentially feed on pigs rather than humans and horses[9].

However in tropical regions nearer to the equator the average 24 hour minimum temperature is more constant, around 28˚C, and in such areas the virus circulates more or less continuously between mosquitoes, birds and pigs. Sporadic cases or small outbreaks occur in humans and horses, especially during the monsoon[3].

In Papua New Guineau, where the virus has been present for at least 8 years, the vector is *Cx. annulirostris*, an opportunistic species which occurs in large numbers during the wet season. Mosquitoes blown from Papua New Guinea seem to have been responsible for outbreaks in the Torres Strait islands since 1995, and in Northern Australia in 1998, but there is as yet no evidence that the virus has become established on the Australian mainland[10].

There are direct correlations between vector density and occurrence of epidemics, and between the rate of seropositivity in pigs and occurrence of epidemics in rural residents, including horses[1]. This is due

in part to the low infection rate of mosquitoes. Minimum infection rates of 1:200 in adult *Cx. tritaeniorhynchus* have been demonstrated, while rates for *Cx. vishnui* and *Cx. gelidus* are usually not higher than 1:550[2]. In a 1995 survey in the Torres Strait Islands, the virus was isolated from only 8 of 2,871 *Cx. annulirostris* caught[10].

Thus, transmission requires high vector densities and high biting rates, which are usually achieved only in rice growing areas of Asia. For example, in an endemic area in Sarawak, on average 228 mosquitoes were collected around 2 pigs used as bait in the two hours after sunset and the hour before sunrise[9]. During an outbreak of Japanese encephalitis in Bangkok, up to 5728 mosquitoes per two trap nights were caught using CO_2-baited light traps[11].

A formalin-inactivated bivalent vaccine is available and is widely used in horses in Japan[1].

In horses, clinical signs appear 8-10 days post infection, by which time viraemia may have passed[1]. Viraemia in horses appears 1-4 days after infection and lasts for 2-6 days[12], ending with the development of antibodies[1]. The level of viraemia in horses is very low in comparison to pigs and birds[12] , and horses are considered dead-end hosts for the virus as they do not develop viraemias of sufficient titre to infect mosquitoes[2, 5].

Virus neutralisation, haemagglutination inhibition and complement fixation can be used to assay populations and individual horses. These tests can also be used to determine the degree of antibody production in vaccinated horses[13]. IgM capture ELISA assays indicate recent infections[1].

In view of the highly complex natural history of this disease, which involves species of birds and high densities of pigs and insects in climates and ecosystems which are not found in New Zealand, it could be argued that even if the virus were introduced into New Zealand it is extremely unlikely that any transmission would occur. Climatographs, plotted on a monthly basis[14], show that the hottest part of New Zealand is the far North in the month of February. The mean daily temperature in that month is 20˚C (the mean daily minimum and the mean daily maximum during that month is 17˚C and 29˚C respectively). But summer is also the driest time of the year in New Zealand; the mean February rainfall in Northland is less than 100 mm. Thus, even if a suitable vector did exist in New Zealand, the time of the year with the most suitable temperature for the development of the virus in vectors would be least suitable for the build-up of high vector densities. In addition, amplification of the virus requires a high density of seronegative pigs, which would probably only be found in areas where intensive pig production is practised, and this is limited in most parts of New Zealand, especially in the far north.

Conclusion: Animals which are serologically positive will have been vaccinated or will have recovered from infection with Japanese encephalitis virus. Neither will be virus carriers.

Recommendation: Seropositive animals could be safely released from PAQ, and the offspring of such animals would not pose a biosecurity risk.

References

(1) Calisher CH, Walton TE. Japanese, Western, Eastern and Venezuelan Encephalitides. In : Studdert MJ (ed). Virus Infections of Equines. Pp 141-55. Elsevier, Amsterdam, 1996.

(2) Hoke CH, Gingrich JB. Japanese encephalitis. In : Beran GW (ed). Handbook of Zoonoses. Second Edition. Section B: Viral. Pp 59-69. CRC Press, Boca Raton, 1994.

(3) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. Pp 140-4. Australian Government Publishing Service, Canberra, 1995.

(4) Heather B, Robertson H. Field Guide to the birds of New Zealand. Pp 254-5. Viking, Auckland, 1996.

(5) Monath TP, Heinz FX. Flaviviruses. In : Fields BN, Knipe DM, Howley PM (eds). Fields Virology. Third Edition. Pp 961-1034. Lippincott-Raven, Philadelphia, 1996.

(6) Belkin JN. Mosquito Studies VII. The culicidae of New Zealand. Contributions of the American Entomological Institute Vol 3, No. 1, 1968.

(7) Reisen WK, Meyer RP, Presser SB, Hardy JL. Effect of temperature on the transmission of western equine encephalomyelitis and St. Louis encephalitis viruses by *Culex tarsalis* (Diptera: Culicidae). Journal of Medical Entomology 30(1), 151-160, 1993.

(8) Takahashi M. The effects of environmental and physiological conditions of *Culex tritaeniorhynchus* on the pattern of transmission of JE virus. Journal of Medical Entomology 13(3), 275-84, 1976.

(9) Bendell PJE. Japanese encephalitis in Sarawak: studies on mosquito behaviour in a Land Dyak village. Transactions of the Royal Society of Tropical Medicine and Hygiene 64(4), 497-502, 1970.

(10) Russell RC. Vectors vs humans in Australia - who is on top down under? An update on vector-borne disease and research on vectors in Australia. Journal of Vector Ecology 23(1), 1-46, 1998.

(11) Gingrich JB, Nisalak A, Latendresse JR, Sattabonkott J, Hoke CH, Pomsditt J, Chantalakana C, Satayaphantan C, Uechiewcharnkit K, Innis BL. Japanese encephalitis virus in Bangkok: factors influencing vector infection in three suburban communities. Journal of Medical Entomology 29, 436-44, 1992.

(12) Burke DS, Leake CJ. Japanese encephalitis. In : In: Monath TP (ed). The Arboviruses: Epidemiology & Ecology. Volume III. Pp 63-92. CRC Press, Boca Raton, Florida, 1988.

(13) Takashima I. Japanese encephalitis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 461. OIE, Paris, 1996.

(14) Gerlach JC. Climatographs of New Zealand. Ruakura Agricultural Research Centre Research Bulletin 74-1, 1974.

B213. Horse mange

The most severe mange of horses is sarcoptic mange, caused by *Sarcoptes scabei* var. *equi*. Psoroptic mange caused by *Psoroptes ovis [equi]* is rare. Chorioptic mange (leg mange) in horses is caused by *Chorioptes bovis*. Demodectic mange is rare in horses[(1)].

Serological tests are not available for mange[(2)].

Conclusion: Serology is not applicable for diagnosis of this disease.

References

(1) Aiello S (ed). The Merck Veterinary Manual. Eighth Edition. P667. Merck & Co, Whitehouse Station, 1998.

(2) Soulsby EJL. Mange. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 449. OIE, Paris, 1996.

B214. Salmonellosis (*S. abortus equi*)

Salmonella abortus equi is a specific disease of Equidae characterised by abortion in females, testicular lesions in males and septicaemia in the newborn. Natural infection is probably by ingestion of feedstuffs contaminated by uterine discharges of carriers or mares which have recently aborted. Diagnosis is made by isolation of the causative bacterium[(1)].

The disease was common in many countries in the early part of this century, but is now rare. It was withdrawn from List B at the 61st General Session of the OIE in May 1993.

Conclusion: Serological diagnosis is not relevant for this disease.

References

(1) Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth Edition. P 746. Balliere Tindall, London, 1994.

B215. Surra (*T. evansi*)

Surra is a disease of many species of animal caused by the protozoan parasite *Trypanosoma evansi*. Surra has a wide host range and is distributed within a wide range of vegetation and climate types. It is present in northern Africa, the Middle East, some areas of the former Soviet Union, the Indian subcontinent, China, South-East Asia and South America[1].

Unlike the tsetse-transmitted trypanosomes, *T. evansi* does not have an intermediate host[2]. It is transmitted mechanically by blood sucking flies particularly of the genera *Tabanus*, *Stomoxys*, *Atylotus* and *Lyperosia*[1]. Of these only the stable fly, *Stomoxys calcitrans*, is present in this country[3], but it is possible that this would be enough to allow the parasite to be transmitted in some areas.

Surra is spread to new areas by the movement of infected animals[1]. Diagnosis is by examination of blood smears. A range of antibody detection tests have been developed, including complement fixation, indirect haemagglutination, precipitation, indirect fluorescent antibody, card agglutination and ELISA. All these systems require further evaluation and standardisation[4].

Conclusion: Animals which are serologically positive for surra are likely to be carriers, and it may be that transmission would be possible in New Zealand by biting flies.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Geering WA, Forman, AJ, Nunn MJ. Exotic Diseases of Animals: a field guide for Australian Veterinarians. Pp 380-4. Australian Government Publishing Service, Canberra, 1995.

(2) Connor RJ. African animal trypanosomiases. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 167-205. Oxford University Press Southern Africa, Capetown, 1994.

(3) Tenquist DJ, Charleston WAG. An annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand 11(3), 257-85, 1981.

(4) Kageruka P, Van Meirvenne N. Surra (*Trypanosoma evansi*). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. Pp 686-93. OIE, Paris, 1996.

B216. Venezuelan equine encephalomyelitis

This disease is included in section B204 “Equine encephalomyelitis”

9. LIST B PIG DISEASES

B251. Atrophic rhinitis of swine

Atrophic rhinitis is an infectious disease of pigs characterised by purulent nasal discharge combined with shortening and twisting of the snout. A severe progressive form of the disease, is caused by infection with toxigenic strains of *Pasteurella multocida* serotype D alone or in combination with *Bordetella bronchiseptica* and perhaps other components of the nasal flora. Infections with *P. multocida* alone can cause a less severe form, with mild or moderate nonprogressive turbinate bone atrophy, generally without significant snout changes[(1)].

Increased severity of these infections is associated with intensive production, overstocking, and poor management, housing and environment; elevated concentrations of atmospheric ammonia, often found in weaner and fattening houses, experimentally reproduced moderate turbinate damage. Reduced productivity has generally been associated with the severe progressive form of atrophic rhinitis and therefore by implication with toxigenic *P. multocida*. However, it is not completely clear whether there is a cause and effect relationship between infection with these bacteria and reduced productivity. Although the view that it is causal is widely held, another view is that the major factors influencing the severity of the disease are managemental and environmental rather than microbiological[(1)].

Surveys of chopper pigs in New Zealand have shown that *P. multocida* and *B. bronchiseptica* are widespread, and that mild turbinate atrophy and nasal septum deviation does occur. However, toxigenic strains of *P. multocida* serotype D have not been found, and the severe form of the disease has never been reported in New Zealand[(2)].

Diagnosis is based on clinical and postmortem signs, and agent isolation. Detection of antibodies to *P. multocida* and *B. bronchiseptica* is of no value for identifying diseased or carrier animals, as non-toxigenic strains of *P. multocida* share cross-reactive antigens with toxigenic strains, and *B. bronchiseptica* can be isolated with ease from pigs in most herds[(1)].

Conclusion: Serological testing is not applicable to this disease.

References

(1) Chanter N, Barnard S, Jones PW, Storm P. Atrophic rhinitis of pigs. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 465. OIE, Paris, 1996.

(2) Gardner DE, Young GW. Atrophic rhinitis of pigs - the New Zealand situation. Surveillance 15(5), 13-4, 1988.

B252. Cysticercosis (*C. cellulosae*)

Porcine cysticercosis is caused by the larval stages of the human tapeworm *Taenia solium*. *Cysticercus cellulosae* is the name given to the cysts which occur in pigs chiefly in the muscles of the heart, tongue, and neck. Porcine cysticercosis is an issue of greater public health significance than bovine cysticercosis, as neurocysticercosis can be serious in humans, through auto-infection[1].

Neither the tapeworm nor the cysticercosis have been detected in New Zealand[2].

Cysts can sometimes be detected ante-mortem by palpation of the tongue, but usually are detectable only by postmortem inspection. Serological tests are not available[1].

Conclusion: Serological diagnosis is not applicable for this parasite.

References

(1) Acha PN, Szyfres B. Zoonoses and Communicable Diseases Common to Man and Animals. Second Edition. Pp 749-60. Pan American Health Organisation, Washington, 1987

(2) Fairley R. Infectious agents and parasites of New Zealand pigs transmissible to humans. Surveillance 23(1), 17-8, 1996.

B253. Porcine brucellosis *(B. suis)*

Brucella suis infection of pigs causes an acute or chronic disease that results in abortions, stillbirths, sterility of sows, orchitis in boars, and mortalities in piglets. Of three biovars affecting pigs, two are highly pathogenic for humans[(1)].

Infection is generally introduced into a herd by live pigs, and transmission is mostly coital but also by the ingestion of contaminated feed. Persistent granulomatous lesions develop in affected organs, from which the organism may be shed for years[(2)].

No serological test is reliable in routine diagnosis of individual pigs, but the buffered brucella antigen tests (BBAT) are used for the identification of infected herds[(1)].

Conclusion: BBAT is unlikely to be used as an individual animal test, but if it were used, a positive reaction would indicate animals that were highly likely to be carriers.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Alton GC, Angus RD. Porcine brucellosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 471. OIE, Paris, 1996.

(2) Bishop GC, Bosman PP. Brucella suis infection. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 1076-7. Oxford University Press Southern Africa, Capetown, 1994.

B254. Transmissible gastroenteritis of pigs

Transmissible gastroenteritis (TGE) is a highly contagious enteric disease of pigs caused by a member of the genus *Coronavirus* in the family *Coronaviridae*[(1)]. The disease is characterised by diarrhoea and dehydration, which is particularly serious in young piglets; mortality in newborn pigs is often 100%, but it declines with age and is very low in pigs aged over 5 weeks[(2)].

Pigs are the only animals for which TGE virus is pathogenic, and probably the only animal significant in its epidemiology, although a number of other animals species have been experimentally infected without showing signs of disease. Transmission of the classic enteric virus is by direct contact with infected pigs or indirectly through contact with their contaminated faeces. Infected pigs excrete TGE virus in their faeces for up to14 days[(2)]. The existence of long term carrier pigs has been suggested, and in isolated cases infection may persist in respiratory tissues for 100 days or more, although not known whether such animals can transmit the virus[(3)]. The virus appears to become endemic only in herds where susceptible animals are continually available as a result of a continuous farrowing schedule or the addition of feeder pigs[(3)].

Since 1984 a distinct respiratory variant of TGE (porcine respiratory coronavirus, PRCV) has spread throughout many parts of the world. PRCV is probably a deletion mutant of the TGE virus, and while it does not appear to be a primary pathogen, it has complicated the serological diagnosis of TGE. Since the emergence of PRCV and its rapid spread throughout Europe, a large part of the pig population has become immune to PRCV, and hence to TGE as well, with the result that TGE outbreaks in Europe are now rare[(2)]. Neither TGE nor PRCV occur in New Zealand[(4)].

The most widely used serological tests are virus neutralisation and ELISAs. TGE and PRCV antibodies can only be differentiated by ELISA. Passive haemagglutination and haemagglutination inhibition tests have also been developed for TGE[(5)].

Conclusion: Recovered pigs may carry the virus for extended periods.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 409, 1995.

(2) Pensaert MB, Callebaut P. Transmissible gastroenteritis. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 866-9. Oxford University Press Southern Africa, Capetown, 1994.

(3) Bohl EH, Pensaert MB. Transmissible gastroenteritis virus (classical enteric variant and respiratory variant). In : Pensaert MB (ed). Virus Infections of Porcines. Pp 139-65. Elsevier, Amsterdam, 1989.

(4) Motha J, Staerk K, Thompson J. New Zealand is free from PRRS, TGE and PRCV. Surveillance 24(1), 10-1, 1997.

(5) Paton DJ, Saif LJ, Levings RL. Transmissible gastroenteritis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. 3rd Edition. P 488. OIE, Paris, 1996.

B255. Trichinellosis

Trichinella spiralis is a small filiform nematode, the adult stage of which lives a few weeks in the small intestine of a large number of mammal species. The larval stage forms a cyst in the muscle tissue of these hosts, where it can remain viable for years. When meat containing encapsulated infective larvae is eaten, the ingested larvae develop into adult worms in the gut, and female worms produce large numbers of larvae which migrate from the gut into the lymphatic vessels. The larvae thus transported into the bloodstream which results in them being spread to all organs of the body, but larvae which end up in organs and tissues apart from skeletal muscle die in a few days[(1)].

The nematode may be found in humans, pigs, rats, bears and many other flesh-eating mammals - even horses that eat fodder containing dead infected rodents[(2)]. It is mainly important because of its public health significance. Humans who ingest large numbers of larvae in infested meat (usually pork) can develop disease initially characterised by gastroenteritis, nausea, abdominal pain and diarrhoea. The larval invasion phase causes muscular pain, fever, and possibly respiratory or neurological symptoms. In epidemics the mortality may be up to 35%, but is usually less than 1%. Only a small proportion of infections result in any clinical signs[(1)].

Trichinellosis occurs in pigs worldwide, but is now very rare in New Zealand[(3)].

Diagnostic tests for trichinellosis in pigs fall into two categories[(2)]:
a) detection of encysted first-stage larvae in muscle tissue
b) detection of antibodies

The ELISA is the best method for ante-mortem diagnosis of trichinellosis[(1)].

Conclusion: Seropositive pigs would probably be carriers of larvae in skeletal muscles.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Acha PN, Szyfres B. Zoonoses and Communicable Diseases Common to Man and Animals. Second Edition. Pp 820-34. Pan American Health Organisation, Washington, 1987

(2) Gamble HR. Trichinellosis. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 477. OIE, Paris, 1996.

(3) Buncic S. A case of a pig infested with *Trichinella spiralis*. Surveillance 24(3), 8, 1997.

B256. Enterovirus encephalomyelitis (previously Teschen/Talfan diseases)

Porcine enteroviruses belonging to the genus *Enterovirus* in the family *Picornaviridae*[1], are ubiquitous in pig populations throughout the world. A serological classification using the virus neutralisation test defines 13 serotypes of porcine enterovirus. Many strains are non-pathogenic. Others cause reproductive disorders in sows (stillbirths, mummification, embryonic death, infertility). Several serotypes may produce sporadic outbreaks of encephalomyelitis - at least types 2, 3, 4, 5, 6, 8, 12 and 13[2].

A severe form of encephalomyelitis in pigs of all ages is caused by porcine enterovirus type 1 (PEV-1). PEV-1 encephalomyelitis was first diagnosed in Czechoslovakia in 1929, in a town named Teschen. The disease caused serious losses in Europe in the 1940s and 1950s. It is now rare in Europe, although serological evidence suggests that in some countries apathogenic variants of the virus circulate in pig populations[2].

Porcine enteroviruses are widespread in New Zealand pigs. Brain lesions consistent with PEV-1 infection have been observed occasionally in regional laboratories in New Zealand, and have been assumed to be due to the mild strain of that serotype (i.e. Talfan disease, rather than Teschen). PEV-1 has been isolated from pigs in New Zealand, although not from brain, and PEV-6 has been isolated from a mild case of porcine encephalomyelitis although inoculation of material into piglets did not reproduce the disease[3].

Transmission of porcine enteroviruses is mainly by the faecal-oral route. Colostral antibody protects suckling pigs so that infection is most frequent in the post-weaning period. Infected pigs excrete the virus for several weeks, the highest levels of excretion occurring soon after infection[4]. Pigs that have recovered from disease, or those with inapparent infections, produce specific antibodies, detectable by the VN test and the ELISA. The seroprevalence of PEV-1 may exceed 60% in many healthy pig populations[2]. Virus neutralising antibodies are found in serum 6 days post infection and in the intestinal contents 14 days post-infection. The serum response persists for a long period[5]. Although viraemia is not detectable after the development of serum neutralising antibodies, intestinal replication and faecal excretion of the virus persists for up to 8 weeks[5].

Conclusion: Porcine enteroviruses are widespread in New Zealand. Recovered animals usually have high antibody levels but may nevertheless shed the virus in faeces for several months. Seropositive animals may be shedders of a more virulent strain of the virus than those already present in New Zealand.

Recommendation: Seropositive animals could not be safely released from PAQ.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 332, 1995.

(2) Mádr V. Enterovirus encephalomyelitis (previously Teschen/Talfan diseases). In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 481. OIE, Paris, 1996.

(3) Fairley R. Infectious diseases of pigs in New Zealand. Surveillance 24(2), 5-7, 1997.

(4) Thomson GR. Teschen, Talfan and reproductive disorders of pigs caused by porcine enteroviruses. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 2, Pp 813-6. Oxford University Press Southern Africa, Capetown, 1994.

(5) Derbyshire JB. Porcine enterovirus (polioencephalomyelitis). In : Pensaert MB (ed). Virus Infections of Porcines. Pp 225-33. Elsevier, Amsterdam, 1989.

B257. Porcine reproductive and respiratory syndrome

Porcine reproductive and respiratory syndrome (PRRS) is a disease of pigs caused by the swine infertility and respiratory syndrome virus, which is a member of the genus *Arterivirus*, in the family *Arteriviridae*, order *Nidovirales*[1].

New Zealand is free of PRRS virus[2].

The disease is characterised by reproductive failure (abortions, stillbirths, and the birth of weak pigs which often die soon after birth, delayed return to service) and increased death rates in weaned pigs. In older pigs there is respiratory disease, sometimes complicated by secondary infections[3].

Transmission appears to occur by close contact with infected animals, but there is limited understanding of the processes involved. The virus has been identified from serum, semen, saliva, faeces, urine, nasal swabs and oropharyngeal scrapings at different times following infection[4]. Although infected aerosols have been considered to be the most likely source of the virus for susceptible pigs[3], transmission by this route has been difficult to achieve experimentally[4]. Semen transmission is possible, as infected boars shed the virus in their semen for at least 3 weeks after infection[3]. Transmission to females by artificial insemination with undiluted semen has been demonstrated[4]. Limited airborne spread appears to be possible in certain conditions[4], and spread by birds, fomites and in slurry may also be possible[5].

In herds where PRRS virus persists, sows do not suffer repeated reproductive losses, suggesting that some form of protective immunity does develop[6]. In such herds the virus is perpetuated by a cycle of transmission from dams to piglets either in utero or postpartum, or by naive animals coming into contact with infected animals in later stages of production[4]. Antibodies to the virus can be detected as early as 7-14 days after infection, and antibody levels reach maximal titres around 1-2 months post infection and then decline to low or undetectable levels over around 4-6 months[4]. Neutralising antibodies develop more slowly and appear to persist longer (a year or more)[4].

Infected pigs can remain viraemic for 4-6 weeks after detectable antibody has been formed, and can transmit the virus to other pigs[3]. This co-existence of virus and antibody in serum implies that early antibodies have limited protective value[6].

Field experience is that recovered pigs are not responsible for herd breakdowns[6]. Piglets in infected herds generally become infected at around 10 weeks of age, but by 6 months of age they are not able to transmit the virus to susceptible seronegative contacts[7]. Therefore it is generally considered that the virus cannot be recovered from infected pigs more than about 3 months after infection[5]. However, the virus can be isolated from the oropharynx of recovered animals for long periods; in one pig it was isolated on day 157 post-infection, 134 days after the last isolation from serum[8]. Exactly how long animals remain potentially infectious is unknown[4].

Attenuated and inactivated vaccines are available[5]. Vaccine efficacy has not been widely studied, but two modified live vaccines are in widespread use in North America[4]. Clinical signs of the reproductive

form of PRRS may be prevented by vaccination of sows 4 weeks prior to breeding[3]. However, there is probably only limited cross-protection between different strains of PRRS virus[5].

Serological tests for the detection of antibody include indirect immunofluorescence, serum neutralisation, the immunoperoxidase monolayer assay, the indirect fluorescent antibody test, and the ELISA[3].

Conclusion: Animals which are seropositive will have been either vaccinated or infected. Infected animals may carry the virus for more than 5 months after infection.

Recommendation: Seropositive animals could not be safely released from PAQ in New Zealand.

References

(1) International Committee on the Taxonomy of Viruses. ICTV taxomonic proposals approved in Jerusalem meeting in 1996. http://www.ncbi.nlm.nih.gov/ICTV/Taxpro96.html

(2) Motha J, Staerk K, Thompson J. New Zealand is free from PRRS, TGE and PRCV. Surveillance 24(1), 10-1, 1997.

(3) Wensvoort G, Ludemann LR. Porcine reproductive and respiratory syndrome. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 694. OIE, Paris, 1996.

(4) Benfield DA, Collins JE, Dee SA, Halbur PG, Joo HS, Lager KM, Mengeling WL, Murtaugh MP , Rossow KD, Stevenson GW, Zimmerman JJ. Porcine Reproductive and Respiratory Syndrome. In : Straw BE, D'Allaire S, Mengeling WL, Taylor DJ (eds.) Diseases of swine. 8th Edition. Pp 201-32. Iowa State University Press, Ames, Iowa, 1999.

(5) Meredith MJ. Porcine Reproductive and Respiratory Syndrome. Pig Disease Information Centre, University of Cambridge. ISBN 0-9520409-7-2. 1. European Edition, August 1995.

(6) Done SH, Paton DJ, White MEC. Porcine reproductive and respiratory syndrome: a review, with emphasis on pathological, virological and diagnostic aspects. British Veterinary Journal 152, 153-73, 1996.

(7) Paton DJ, Drew TW. Serological monitoring of PRRS transmission: a case study. Veterinary Record 136, 297-8, 1995.

(8) Wills RW, Zimmerman JJ, Yoon KJ, Mcginley MJ, Hill HT, Platt KB, Christopherhennings J, Nelson EA. Porcine reproductive and respiratory syndrome virus - a persistent infection. Veterinary Microbiology 55(1-4), 231-40, 1997.

10. UNLISTED DISEASES

10.1 Akabane and related Simbu-group viruses

Akabane virus is a member of the Simbu group of the genus *Bunyavirus* in the family *Bunyaviridae*. Viruses in the Simbu group cause congenital defects, principally arthrogryposis and hydranencephaly and abortion in cattle, sheep and possibly goats[(1)].

Most of Africa, Asia and Australia may be regarded as endemic for Akabane virus, and in all probability many of its antigenic relatives. Most of the American continent , Papua New Guinea and the island countries of the Pacific are free of the virus[(1)].

There is a large amount of circumstantial evidence suggesting that the virus is transmitted only by infected insects[(1)].

Akabane virus has been isolated in Japan from the mosquitoes *Aedes vexans* and *Culex tritaeniorhynchus* and from the biting midge *Culicoides oxystoma*. It has been isolated from *Anopheles funestus* mosquitoes and in Kenya and from the biting midge *Culicoides brevitarsis* in Australia[(2)]. These vectors do not exist in New Zealand[(3, 4)].

Viraemia in naturally infected cattle lasts 3-4 days, disappearing with the development of neutralising antibody[(1)]. A high prevalence of neutralising antibody has been demonstrated in a wide range of animals in countries where the virus is endemic[(2)].

The virus neutralisation test is the most specific of serological tests available. Other tests available include AGID and ELISA[(1)].

Conclusion: As with other bunyaviruses, infection leads to a short viraemia in affected animals and there is no long term carrier state. The vectors for these viruses are not present in New Zealand.

Recommendation: Seropositive animals could be safely released from PAQ, and the offspring of such animals would not pose a biosecurity risk.

References

(1) St George TD, Standfast HA. Akabane and related Simbu-group viruses. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 681-7. Oxford University Press Southern Africa, Capetown, 1994.

(2) Inaba Y, Matumoto M. Akabane virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 467-80. Elsevier, Amsterdam, 1990.

(3) Belkin JN. Mosquito Studies VII. The culicidae of New Zealand. Contributions of the American Entomological Institute Vol 3, No. 1, 1968.

(4) Motha J, Hansen M, Irwin G. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance 24(4), 18-9, 1997.

10.2 Ephemeral fever

Bovine ephemeral fever (BEF) is an arthropod-borne disease of cattle and water buffalo, caused by a virus of the genus *Ephemerovirus* in the family *Rhabdoviridae*. The disease is characterised by short duration, fever, stiffness, and disinclination to move, and it almost invariably results in complete recovery. It is essentially a summer and autumn disease in subtropical and temperate areas of Africa, Asia, and Australia, disappearing with the first frosts. In tropical areas it is linked to the rainy season[1].

BEF virus has been isolated from *Culicoides imicola* and *C. coarctatus* in Africa, and *C. brevitarsis* in Australia. It has also been isolated from a mixed pool of Culicine mosquitoes and from the mosquito *Anopheles bancroftii*. Even with this range of insect species as potential vectors, it is considered that there must be other species involved, as the areas of the world in which BEF occurs extend beyond the distribution of the insect species implicated so far. The whole of Africa, Asia south of the former USSR, and Australasia (but excluding Papua New Guinea and New Zealand) may be regarded as falling within the infected zone even if clinical disease has not been formally reported there. The limits of occurrence in countries that are partially free, such as Japan, China, and Australia, are determined by climate through its effects on vectors[1].

Culicoides spp. do not exist in New Zealand[2], and none of the above mosquito species are present in this country[3].

The overwintering mechanisms of the infection are unknown. Serological monitoring of sentinel cattle in an endemic area in Australia indicated that the virus was unlikely to overwinter in cattle. Viraemia in experimentally infected cattle usually lasts about 4-5 days, commencing the day before fever and terminating 1-2 days after clinical recovery. Very occasionally viraemia may persist as long as 13 days after infection, but there is no long-term carrier state of BEF virus in cattle. One attack of BEF generally confers lifelong immunity[1].

Serological tests available include virus neutralisation and a blocking ELISA[1].

Conclusion: Infection of cattle with BEF virus results in a viraemia which does not last longer than 2 weeks. Recovered animals are immune for life, and there is no long term carrier state. The vectors for this virus are not present in New Zealand.

Recommendation: Seropositive animals could be safely released from PAQ, and the offspring of such animals would not pose a biosecurity risk.

References

(1) St George TD. Bovine ephemeral fever. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 553-62. Oxford University Press Southern Africa, Capetown, 1994.

(2) Motha J, Hansen M, Irwin G. Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. Surveillance 24(4), 18-9, 1997.

(3) Belkin JN. Mosquito Studies VII. The culicidae of New Zealand. Contributions of the American Entomological Institute Vol 3, No. 1, 1968.

10.3 Palyam group orbiviruses

The Palyam serogroup contains 11 arthropod-borne viruses of the genus *Orbivirus* in the family *Reoviridae*. These viruses occur in Africa, Asia and Australia, and they appear to be associated with abortion and teratology in cattle and possibly other ruminants[(1)].

The majority of isolations of Palyam serogroup viruses have come from *Culicoides* midges, although 2 viruses have been isolated from ixodid ticks and 3 viruses from mosquitoes in India. Transmission of the virus by midges has not been demonstrated, but it is generally accepted that *Culicoides* spp. are responsible for transmission. Apart from sheep and cattle, no vertebrate hosts for the Palyam serogroup viruses are known, although antibodies have been found in Asian buffalo in Australia and in goats and humans in South Africa[(1)].

As with other orbiviruses there is no indication that recovered animals carry the virus. Furthermore, the absence of *Culicoides* spp. in New Zealand means that Palyam serogroup viruses could not become established even if viraemic animals were to be imported.

Conclusion: There is no evidence for a permanent carrier state in recovered animals.

Recommendation: Seropositive animals could be safely released from PAQ and the offspring of such animals would not pose a biosecurity risk.

References

(1) Swanepoel R. Palyam serogroup orbivirus infections. In : Coetzer JAW, Thomson GR, Tustin RC (eds). Infectious Diseases of Livestock with Special Reference to Southern Africa. Volume 1, Pp 480-3. Oxford University Press Southern Africa, Capetown, 1994.

10.4 Bovine virus diarrhoea and mucosal disease

Bovine virus diarrhoea (BVD) and mucosal disease (MD) are pathogenically distinct diseases of cattle which are caused by the bovine diarrhoea virus which is the type species of the *Pestivirus* genus in the family *Flaviviridae*[1]. There are three members of the *Pestivirus* genus, the other two being border disease virus and classical swine fever (hog cholera) virus, but although the three viruses are named after the animal species from which they were first isolated, they all may infect and cause disease in other animal species[2].

Genotyping of BVD viruses has revealed two distinct groups of bovine pestiviruses, which are known as BVD-1 and BVD-2[3]. BVD-2 is as different from BVD-1 as it is from other pestiviruses[4], and it is now to be designated as a new (yet to be re-named) species in the *Pestivirus* genus[5]. In addition to this genomic variation, there are two biotypes of BVD virus, cytopathogenic and non-cytopathogenic, which can be distinguished by their effect in cell culture[6].

Infection with *Pestiviruses* produce inapparent infections, acute or persistent subclinical infections, acute fatal disease, foetal death or congenital abnormalities, and a wasting disease[7].

A seroprevalence of about 70-90% in most cattle populations shows that most cattle contract a BVD virus infection during their lifetime. This rate of seroconversion is not correlated with clinical disease, suggesting that most postnatal infections occur unnoticed. However, distinct clinical lesions are also reported. These include a short febrile period, leucopenia, salivation, nasal discharge, coughing and/or sometimes diarrhoea. A rare haemorrhagic syndrome in calves has been reported in North America and Europe. BVD virus also appears to be one component of a complex aetiology in postnatal infections causing pronounced respiratory tract disease[2].

The BVD virus spreads by contact between infected and susceptible animals. The main routes of natural transmission are the oral and nasal routes[8]. Infection of pregnant susceptible animals results in transplacental infection of the foetus. Infections in the first stages of gestation (up to about day 150) result in abortions, stillbirths, and teratogenic effects or the birth of persistently infected calves, whereas infections after 150 days of gestation usually results in the birth of seropositive clinically normal calves[2]. Persistently viraemic animals may remain healthy and be unrecognised clinically, and they constitute the major source of BVD virus for infecting other animals[8].

MD is a sporadic form of disease caused by BVD virus, characterised by low morbidity and high case fatality, which can only arise in animals persistently infected with non-cytopathogenic BVD virus. In these animals the appearance of a cytopathogenic virus is regarded as causative[2]. Until recently it was considered that the cytopathogenic virus was the result of a postnatal superinfection of an animal which had been infected in utero with a non-cytopathogenic virus[8]. However molecular evidence now suggests rather that the cytopathogenic virus arises from a mutation in the non-cytopathogenic virus[6]. Furthermore, while both biotypes can be isolated from persistently infected animals dying from MD, the cytopathogenic biotype cannot be recovered from animals other than these[9].

BVD virus is widespread in New Zealand cattle, with about 60% of animals having antibodies against it[10]. Genotyping of BVD isolates collected in New Zealand from 1967 to 1997 showed that all were BVD type 1[11]. On the basis of these findings, and as there has been no disease detected in New Zealand cattle that resembles type 2 disease as seen elsewhere in the world, it is believed that BVD type 2 does not occur in New Zealand[12].

Infected animals seroconvert within 14 to 28 days after infection, and high levels of serum antibodies are maintained for at least 3 years without being further infected with BVD virus[13]. In experimental infections the virus can be detected in blood 2-4 days after oral or nasal inoculation, and during the period of viraemia large quantities of virus are excreted in saliva, nasal discharges, and to a lesser extent in urine and faeces (if at all, in the absence of diarrhoea). Infectivity can be found in buffy coat cells for up to 11 days after the onset of demonstrable antibody formation, suggesting that the virus associated with leukocytes is not accessible to neutralising antibodies as in blood plasma infectivity disappears as soon as neutralising antibodies appear[7].

It is generally considered that serologically positive nonviraemic cattle may be traded safely, as latent infections are not known to occur in recovered animals, but it is important to avoid the trade of viraemic animals[8].

Acute infection with BVD virus is best confirmed by demonstrating seroconversion using sequential paired samples from several animals in the group. The ELISA and the virus neutralisation test are most widely used[8].

Persistently viraemic healthy animals resulting from congenital infection cannot be identified by serological testing; that requires the isolation of noncytopathogenic virus in cell cultures from blood or serum. Persistence of the virus should be confirmed by resampling after an interval of at least 3 weeks. These animals usually have no or low levels of antibodies to BVD virus[8].

A number of BVD vaccines are available, both modified live and killed. The main objective of vaccination is to prevent transplacental infections and therefore to avoid the birth of persistently infected viraemic calves, but the few vaccines tested are not highly effective in this regard. Vaccination is also used to prevent acute postnatal infections. There are also widespread concerns about the safety of modified live vaccines, as it is thought that their use may lead to recombination of vaccine strains with wild type viruses and the emergence of new, possibly more pathogenic, strains[14].

Three inactivated vaccines are registered for use in New Zealand. Modified live vaccines are not permitted in this country.

Conclusion: There is no evidence for a permanent carrier state in recovered animals. New Zealand is free of BVD type 2 virus. It is generally considered that serologically positive nonviraemic cattle are not able to transmit BVD viruses.

Recommendation: Seropositive, nonviraemic animals could be safely released from PAQ in New Zealand, and the offspring of such animals would not constitute a biosecurity risk.

References

(1) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds). Virus Taxonomy : classification and nomenclature of viruses. Sixth report of the International Committee on Taxonomy of Viruses. Archives of Virology , Supplement 10, 423-4, 1995.

(2) Thiel HJ, Moennig V. The molecular basis of pathogenesis of bovine viral diarrhoea virus infection. OIE Veterinary Biotechnology Newsletter 4, 143-52, 1994.

(3) Ridpath JF, Bolin SR, Dubovic EJ. Segregation of bovine viral diarrhoea virus into genogroups. Virology 205, 66-74, 1994.

(4) Edwards S, Paton D. Antigenic differences among pestiviruses. Veterinary Clinics of North America: Food Animal Practice 11(3), 563-78.

(5) International Committee on Taxonomy of Viruses. Taxonomic proposals approved in Strasburg meeting in 1997; veterbrate viruses. http://www.ncbi.nlm.nih.gov/ICTV/Taxpro97.html

(6) Deregt D, Loewen KG. Bovine viral diarrhoea virus: Biotypes and disease. Canadian Veterinary Journal 36, 371-8, 1995.

(7) Liess B. Bovine viral diarrhoea virus. In : Dinter Z, Morein B (eds). Virus Infections of Ruminants. Pp 247-66. Elsevier, Amsterdam, 1990

(8) Brownlie J, Edwards S. Bovine viral diarrhoea. In : Office International des Epizooties. OIE Manual of Standards for Diagnostic Tests and Vaccines. Third Edition. P 651. OIE, Paris, 1996.

(9) Nettleton PF, Entrican G. Ruminant pestiviruses. British Veterinary Journal 151, 615-41, 1995.

(10) Littlejohns IR, Horner GW. Incidence, epidemiology and control of bovine pestivirus infections and disease in Australia and New Zealand. Rev. sci. tech. Off. int. Epiz. 9, 195-205, 1990.

(11) Vilcek S, Bjorklund HV, Horner GW, Meers J, Belak S. Genetic typing of pestiviruses from New Zealand. New Zealand Veterinary Journal 46, 35-7, 1998.

(12) Anon. Wallaceville animal health laboratory : report for 1997. Surveillance 25(1), 18-20, 1998

(13) Fredriksen B, Sandvik T, Løken T, Ødegaard SA. Level and duration of serum antibodies in cattle infected experimentally and naturally with bovine virus diarrhoea virus. Veterinary Record 144, 111-4, 1999.

(14) van Oirschot JT, Bruschke, van Rijn PA. Vaccination of cattle against bovine viral diarrhoea. Veterinary Microbiology, 64, 169-183, 1999.

ACKNOWLEDGMENTS

The author thanks MAF colleagues Stuart MacDiarmid, Noel Murray and Joanne Thompson, who assisted in a number of ways, including critical review of several drafts of this risk analysis.

A number of experts offered invaluable advice including general and detailed technical review, in some cases related to individual diseases and in other cases related to groups of diseases. Many people also assisted in finding specific publications. The author gratefully acknowledges the inputs from;

John Anderson, Institute for Animal Health, Pirbright, UK - rinderpest
Charles Calisher, CDC, Fort Collins, USA - Japanese encephalitis
Trevor Crosby, Landcare, Auckland, NZ - insect populations in NZ
Peter Daniels, AAHL, Victoria, Australia - Japanese encephalitis
Bert de Vos, Tick Fever Research Centre, Queensland, Australia - anaplasmosis, theileriosis
Stan Fenwick, Massey University, NZ - *Salmonella abortus ovis*, and general review
Lane Foil, Louisiana State University, USA - surra, equine infectious anaemia, and other diseases transmitted by biting flies
Geoff Gard, BRS, Canberra, Australia - bluetongue, akabane, palyam viruses, rift valley fever
Allen Heath, Agresearch, Wallaceville, NZ - mosquito vectors for arboviruses in NZ
Gary Horner, NCDI, Wallaceville, NZ - bovine pestiviruses, porcine enteroviruses, bovine and equine herpesviruses
Dirk Houwers, University of Utrecht, Netherlands - small ruminant lentiviruses, porcine reproductive and respiratory syndrome, Q fever, general review
Tom Ksiazek, Centers for Disease Control and Prevention, Atlanta, USA - Rift valley fever
David Kemp, CSIRO, Longpocket, Australia - ticks
Paul Kitching, Institute for Animal Health, Pirbright, UK - list A diseases
Don Knowles, Washington State University, USA - babesiosis, lentiviruses
Will Laegreid, Meat Animal Research Center, USDA, Nebraska, USA - African horsesickness, equine babesiosis
Colin Leake, University of London, UK - Japanese encephalitis
Tony Luckins, University of Edinburgh, Scotland - trypanosomosis
James MacLachlan, University of California at Davis, USA - *Culicoides*, bluetongue, African horsesickness and other arboviruses
Ronald Magar, Saint-Hyacinthe, Québec, Canada - porcine reproductive and respiratory syndrome
Joanne Meers, Massey University, NZ - equine herpesviruses
Philip Mellor, Institute for Animal Health, Pirbright, UK - bluetongue, African horsesickness and Culicoides in Europe
Julian Motha, NCDI, Wallaceville, NZ - small ruminant lentiviruses
Joseph O'Keef, Agresearch, Wallaceville, NZ - equine rhinopneumonitis
Eileen Ostlund, NVSL, APHIS, USDA, Ames, Iowa, USA - Eastern, Western and Venezuelan equine encephalomyelitis
Ian Parsonson, Veterinary Consultant, Ocean Grove, Victoria, Australia - virus diseases
James E. Pearson, NVSL, APHIS, USDA, Ames, Iowa, USA - Eastern, Western and Venezuelan equine encephalomyelitis
Clarence J Peters, Centers for Disease Control and Prevention, Atlanta, USA - Rift valley fever

Simon Reid, James Cook University of North Queensland, Australia - mechanical transmission of trypanosomes
Robert Sanson, Agriquality, Palmerston North, NZ - foot and mouth disease
Phil Sirvid, Te Papa, Wellington, NZ - insect populations in NZ and Australasia
Peter Van den Bossche, Regional Tsetse and Trypanosomiasis Control Programme, Harare, Zimbabwe - mechanical transmission of trypanosomes
Colin Wilks, VIAS, Victoria, Australia - general review
Nora Wineland and others at CEAH, APHIS, USDA, Fort Collins, USA - virus diseases
Jim C. Williams, Pasteur Merieux Connaught, Pennsylvania, USA - Q fever

In addition, a number of MAF colleagues provided technical comments on specific parts of the document. The author acknowledges in this regard John Bassett, Mark Bosson, Jim Edwards, Mirzet Sabirovic, Ron Thornton and Bob Worthington.

Special thanks are due to Marion Riddell in the MAF Information Bureau, without whose considerable help in locating reference material this risk analysis would not have been possible.