Import Risk Analysis: Hides and skins from specified animals

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Biosecurity New Zealand
Ministry of Agriculture and Forestry
Wellington
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RISK ANALYSIS FOR THE IMPORTATION OF HIDES AND SKINS FROM SPECIFIED ANIMALS INTO NEW ZEALAND

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1 Executive Summary

This document is an analysis of the biosecurity risks posed by imported hides and skins of ruminants, horses, pigs, lamoids and ratites for processing into leather. This trade is in the interests of New Zealand companies operating in a globally competitive, fashion-driven industry where access to raw material at appropriate cost is vital.

This risk analysis was initiated as part of New Zealand's obligation under Annex V of the EU veterinary agreement. However, it considers hides and skins from all countries.

The processes involved in the processing of hides and skins into leather are discussed in relation to the physical and chemical processes which agents on hides and skins would be subjected to.

In this risk analysis, the starting point for the hazard identification is the disease lists of the OIE. When this risk analysis was initiated, OIE listed diseases were in two lists, A and B, and although the OIE list arrangement was changed in 2004, the diseases on the lists are the same and for this risk analysis the original A and B lists have been retained for convenience. The zoonotic agents of Crimean-Congo haemorrhagic fever and West Nile Fever were included because they were added to the new OIE lists in 2005. Besides these listed diseases two important infections of ruminants (bovine viral diarrhoea of cattle and border disease of sheep) have been included. A section on all Enterobacteriacae including *Salmonella* spp. is also included. Specific diseases of ostriches and emus are included as these animals are included in the commodity definition. The section on infectious bovine rhinotracheitis considers all bovine herpes viruses.

At request of the Ministry of Healthi, the transmissible spongiform encephalopathies of deer are included. In addition, several unwanted insects and arachnids are also included, as well as weed seeds and the broad category of 'hitch-hiker pests'.

The epidemiology of each disease is considered, particularly the survival of the agent in the environment and the commodity, and the route of transmission. For each disease a conclusion is reached as to whether or not it is considered a potential hazard in the commodity.

The release assessment then examines the likelihood of potential hazards surviving processing. The exposure assessment considers possible routes of exposure of agents to susceptible animal species in New Zealand, and the consequence assessment considers the possible effects of such exposure. The risk estimation comes to a conclusion for each potential hazard as to whether safeguards are warranted.

[i] Mary Harvey, Ministry of Health, email to HJ Pharo dated 10/5/2000.

The highest risks posed by imported hides and skins are the agents of foot and mouth disease and anthrax. Specific options for managing those risks are discussed.

General risk management options that are appropriate for effectively managing the low or very low risk posed by a number of organisms are discussed. These options include:

- Importation from safe sources.
- Treatment of hides and skins before importation.
- Secure packaging of imported commodities.
- Safe transport to tanneries that are approved transitional facilities.
- Safe disposal of tannery wastes.

2 Introduction

2.1 BACKGROUND

This risk analysis examines biosecurity risks involved in importing hides and skins for processing into leather. Trade in hides and skins is in the interests of New Zealand companies operating in a globally competitive, fashion-driven industry where access to raw material at appropriate cost is vital (1).

2.2 SCOPE

In early 2006, a survey was conducted of tanneries issued permits to import hides and skins in the last three years; 22 questionnaires were sent out and 12 replies were received from companies still processing skins. Relevant information from the questionnaires was:

- Nine tanneries have imported hides and skins during the last 3 years, two tanneries have not and one has ceased to operate in the business.

- Hides and skins were imported from :
  - Australia by 9 importers
  - Vanuatu by 3 importers
  - USA by 2 importers
  - China by 1 importer

- 10 tanneries were discharging waste to municipal sewers and 8 were treating waste before disposal. Two were discharging waste onto agricultural land, one of these was treating the waste before disposal and one was not.

- 9 tanneries were importing wet-salted skins, 4 were importing dry skins and 4 were importing products that had been further processed.

Another importer of leather, the Leather and Shoe Research Association (LASRA), imports samples of pickled or tanned products mainly from Italy, China, Korea and India, but also from other countries. Waste water is disinfected and discharged to a municipal sewer.

Hides and skins of the following animal species are considered in this risk analysis:

- sheep
- cattle
- buffalo
- goats
- pigs
- horses
- deer
- llamas
- alpacas
- emus
- ostriches

The New Zealand leather industry needs to be able to import five forms of processed hides and skins of the above species (1):

- air dried
- wet salted
- pickled
- wet blue (chromium iii) tanned
- wet white tanned

The processes by which the above categories of skins are prepared are briefly described in section 3 of this risk analysis.

The risk analysis focuses primarily on infectious disease agents but also includes some insect parasites and weed seeds. It does not address issues relating to chemical contamination of the environment, poisoning of animals or tannery workers, or any diseases that are not caused by infectious agents.

This risk analysis constitutes New Zealand's obligation with regard to annex V of the EU veterinary agreement, although it is not confined to hides and skins from the EU.

2.3 RELEVANT LEGISLATION

2.3.1 Domestic legislation

2.3.1.1 Biosecurity Act 1993

Under section 22(5) of the Biosecurity Act 1993, MAF is obliged to have regard to a number of matters when considering the effective management of risks posed by imported goods :

> *(a) The likelihood that goods of the kind or description to be specified in the import health standard may bring organisms into New Zealand*
>
> *(b) The nature and possible effect on people, the New Zealand environment, and the New Zealand economy of any organisms that goods of the kind or description specified in the import health standard may bring into New Zealand*
>
> *(c) New Zealand's international obligations*
>
> *(d) Such other matters as the chief technical officer considers relevant*

2.3.1.2 Anthrax Prevention Regulations 1987

Section 5 of the Anthrax prevention regulations 1987 is as follows:

> *If the Minister of Health has reasonable cause to believe that anthrax is likely to be conveyed by:*

(a) any wool, hair, bristle, skin or fur obtained from any animal; or

(b) any article made wholly or partly from wool, hair, bristle, skin or fur obtained from any animal; or

(c) any skin bearing wool, bristle or fur, the Minister of Health may, by notice in the Gazette, prohibit the importation of those goods.

Under section 6 of the same regulations, inspectors of health have powers to inspect, seize, direct return of, direct disinfection of, or destroy any goods imported in contravention of section 5.

Methods of disinfection are specified in the first schedule of the Anthrax Regulations 1987. For hides and skins, the methods are:

(a) immersion in a solution of 1 in 10,000 sodium bisulphate for not less than 5 hours;

(b) immersion in a solution containing 20 ppm free chlorine for not less than 2 hours.

2.3.2 International legislation and international agreements

2.3.2.1 EU Balai Agreement.

Under terms of the Veterinary Agreement that New Zealand has with the EU, the Balai agreement of the European Union (Directive 92/118/EEC) is of relevance. The Balai agreement includes conditions for trade of hides and skins of “ungulates”. The word "ungulates" appears to include “bovine animals, swine, sheep and goats and solipeds” (Directive 64/433/EEC).

Under the Balai agreement the following hides and skins of ungulates are not subject to any trade restrictions in the EU:

- hides or skins having undergone the complete process of tanning
- “wet-blue”
- “pickled pelts”
- “limed hides” (treated with lime and in brine at a pH of 12 to 13 for at least 8 hours).

For intra-community trade of fresh or chilled hides and skins, the same animal health conditions apply as those for fresh meat (Directive 72/461/EEC). That is, the hides and skins must not be sourced from animals which have been imported in the last 21 days, the animals must not be from a property which is under restriction for FMD, and the animals must not have been slaughtered in a slaughterhouse in which FMD has been recorded.

For intra-community trade in "treated hides and skins", each consignment must be accompanied by a commercial document certifying that:

a) the hides and skins have been treated by one of the methods mentioned in the following definition (specifying which one)

b) the consignment has not been in contact with any other animal product or live animals presenting a risk of spreading a “serious transmissible disease”

“Treated hides and skins” are defined as hides and skins which have been:

- dried, or
- dry-salted or wet-salted for at least 14 days prior to despatch, or
- salted for 7 days in sea salt with the addition of sodium carbonate to 2%, or
- dried for 42 days at a temperature of at least 20°C, or
- preserved by a process other than tanning.

2.3.2.2 SPS Agreement and the Terrestrial Animal Health Code

Under article 3 of the SPS agreement, member countries are obliged to base SPS measures either on international standards or on a scientifically valid risk analysis.

The only international standards relevant to the trade in animals and their products are those of the World Organisation for Animal Health (OIE), as presented in the *Terrestrial Animal Health Code* (2) (hereafter referred to as the *Code*).

The *Code* specifically mentions hides and skins in the case of:

Disease	**Article**
Foot and mouth disease	2.2.10.27
Rinderpest	2.2.12.27
Peste des petits ruminants	2.4.9.20
Lumpy skin disease	2.3.14.12
Sheep pox and goat pox	2.4.10.9
Anthrax	2.2.1.5

Article 2.2.10.27 of the *Code* states that when importing from FMD infected countries, *Veterinary Administrations* should require:

> for wool, hair, bristles, raw hides and skins (from domestic or wild ruminants and pigs)
>
> the presentation of an *international veterinary certificate* attesting that:
>
> 1) these products have been processed to ensure the destruction of the FMD virus in conformity with one of the procedures referred to in Article 3.6.2.2, Article 3.6.2.3 and Article 3.6.2.4;
>
> 2) the necessary precautions were taken after collection or processing to avoid contact of the products with any potential source of FMD virus.

> *Veterinary Administrations* can authorise, without restriction, the import or transit through their territory of semi-processed hides and skins (limed hides, pickled pelts, and semi-processed leather - e.g. wet-blue and crust leather), provided that these products have been submitted to the usual chemical and mechanical processes in use in the tanning industry.

Article 3.6.2.4 of the OIE *Code* states:

> **Raw hides and skins**
>
> For the inactivation of viruses present in raw hides and skins for industrial use, the following procedure should be used: salting for at least 28 days in sea salt containing 2% sodium carbonate.

The statement for rinderpest in article 2.2.12.27 is almost identical to that for FMD in article 2.2.10.27, and it also refers to articles 3.6.2.2, 3.6.2.3 and 3.6.2.4 for processes to destroy the rinderpest virus (although these articles are focussed only on FMD virus).

For PPR, article 2.4.9.20 refers to "adequately disinfected" raw hides and skins of small ruminants, but does not specify what would be adequate.

For lumpy skin disease, article 2.3.14.12 states that raw hides and skins from infected countries should be stored for at least 40 days before shipment.

For sheep pox and goat pox, article 2.4.10.9 states that sheep and goat skins from infected countries should be "processed to ensure the destruction of the sheep pox and goat pox virus", but it does not specify what processing is required.

For anthrax, article 2.2.1.5 states that hides and skins from ruminants, equines and pigs should have been from animals that passed pre- and post-mortem inspection and from establishements which were not under quarantine on account of anthrax control.

2.3.2.3 The Risk Analysis Process

MAF Biosecurity New Zealand's risk analysis procedures[i] and the *Code* (2) present the following steps for import risk analysis:

1) Hazard identification

2) Risk assessment
- Entry assessment
- Exposure assessment
- Consequence assessment
- Risk estimation

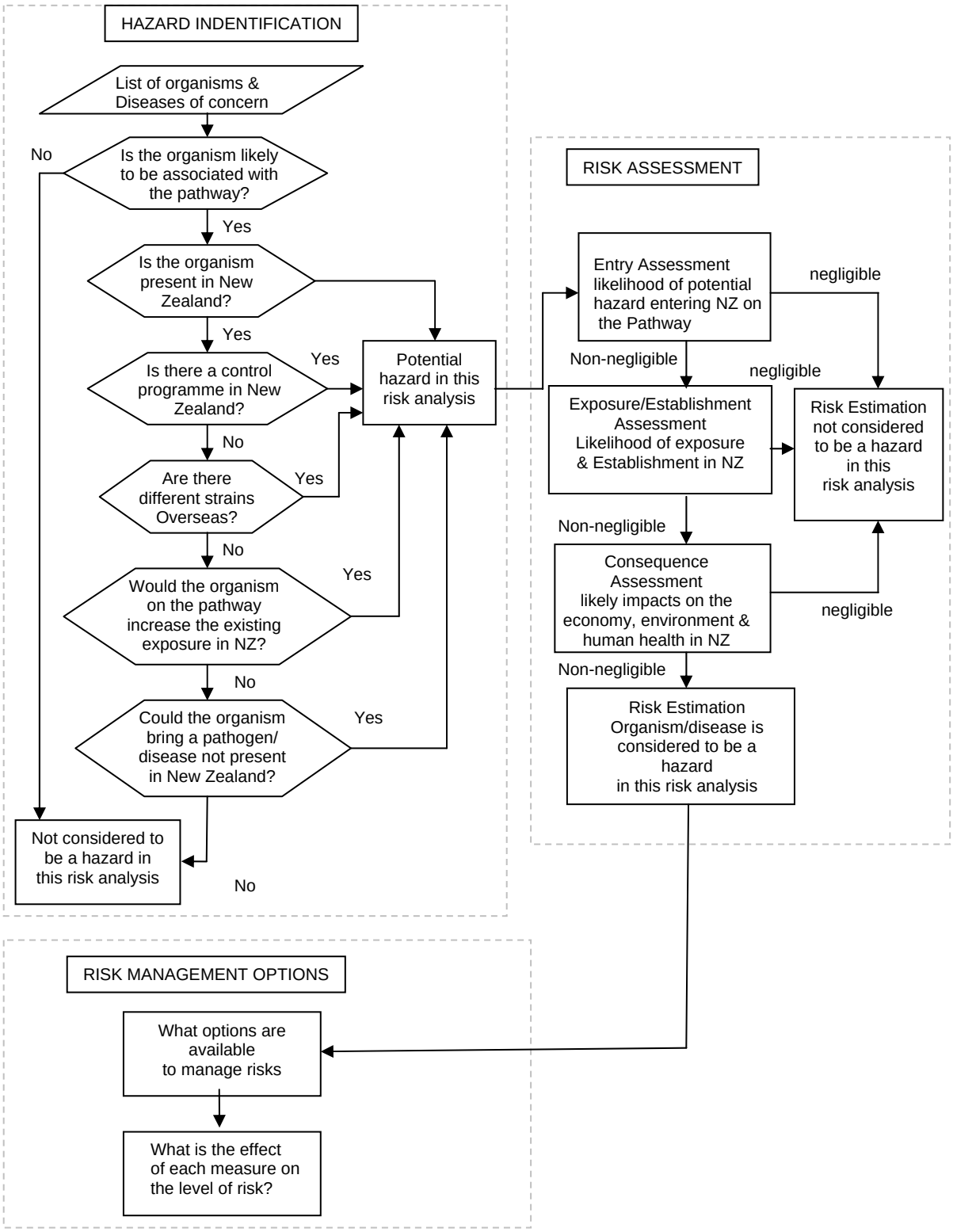
3) Risk management

[i] Biosecurity New Zealand *Risk Analysis Procedures – Version 1*. See: www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf

Prior to carrying out the hazard identification, a preliminary step is to assemble a list of organisms that may be associated with the commodity.

The process is summarised in Figure 1.

Figure 1. Risk analysis process



2.3.3 Organisms that may be associated with the commodity

The primary focus of this risk analysis is the risk to animal and human health, including the risk of zoonotic diseases to persons handling imported hides and skins. Weed seeds and other 'hitch-hiker pests' on imported hides and skins are also included, in so far as the risk they pose to the economy, people and the environment.

2.3.4 Hazard identification

The following aspects must be considered for an organism to be considered a potential hazard:

1) whether the pathway could lead to the introduction of the organism into New Zealand;

AND

2) if the organism requires a vector, whether competent vectors might be present in New Zealand;

AND

A) whether the organism is exotic to New Zealand and likely to be present in an exporting country;

OR

B) if the organism is present in New Zealand,

   a. whether it is 'under official control', which could be by government departments, by national or regional pest management strategies, or by a small-scale programme; **or**

   b. whether more virulent strains are known to exist in other countries, **or**

   c. whether the arrival of the organism in association with the pathway would likely increase the existing exposure to the organism in New Zealand.

For any organism, if the answer to item 1 is "yes" (and the answer to item 2 is "yes" in the cases of organisms requiring a vector), and the answer to either item A or B are "yes", the organism is classified as a potential hazard.

Under this framework, organisms present in New Zealand cannot be considered as hazards unless there is evidence that strains with higher pathogenicity are likely to be associated with the pathway, or the arrival of the organism in association with this pathway would increase the existing exposure to the organism in New Zealand.

Therefore, if risks to human or animal health (or subsequent progeny) posed by the introduction of the organism in association with the pathway, are no different from the

existing risks resulting from the current presence of the organism in New Zealand, mitigating measures should be appropriate to good practice irrespective of the importation.

2.3.5 Risk assessment

Each organism classified as a potential hazard is submitted to "*Risk Assessment*". This comprises four steps.

a) Entry assessment

This is the process of describing the potential for the commodity to introduce organisms into New Zealand's animal and human populations or the environment. In this case, the entry assessment evaluates likelihood that potential hazards could occur in hides and skins and survive transport to this country and the various steps involved in further processing.

b) Exposure assessment

This step describes the possible exposure of susceptible hosts in New Zealand to the potential hazards released from the risk source. In the case of imported hides and skins the analysis considers the likelihood of effective contact with humans, animals or the environment in New Zealand, either directly by contact with the imported commodity prior to processing, or indirectly by contact with waste products generated during processing. The exposure assessment ends at this point without considering the ways by which the organism could be further distributed.

c) Consequence assessment.

This is the process of describing the economic, environmental and health consequence associated with the exposure to the risk agents. In this risk analysis, if it is concluded in the release assessment that an organism or disease agent is a potential hazard in imported hides and skins, then the consequences of its introduction and establishment are assessed.

However it is not necessary to carry out this step in detail when considering the exotic disease agents of international concern with regard to animal trade, as the fact that agents are listed by OIE is enough to signal that the consequences of introduction are unacceptable. In many such cases the impact would be mainly due to the negative effect on exports of animals and animal products that would occur if the agent were introduced into this country, and in other cases it would be due to production losses.

Consequences to human health and the environment are considered where applicable. Any introduction of exotic human pathogens is considered to involve non-negligible risk

For hitch-hiker pests and weed seeds, the consequences may be related to effects on the environment.

d) Risk estimation

This is the step which integrates the results from the release, exposure, and consequence assessments. This step is a summarisation of the preceding three steps and involves a decision as to whether introduction of the commodities constitutes a risk for a particular agent of concern.

It is important to understand that not all of the above steps may be necessary in all risk assessments. The MAF Biosecurity New Zealand and OIE methodologies make it clear that if the likelihood of release is negligible for a certain potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises where the likelihood of release is non-negligible but the exposure assessment concludes that the likelihood of exposure to susceptible species in the importing country is negligible, or where both release and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

2.3.6 Risk management

Risk management is the formulation of options for risk mitigation measures (safeguards) that may be appropriate for effectivley managing the risks posed by the identified hazards.

2.3.7 General considerations

The categories of skins that may be imported into New Zealand include:

- air dried
- wet salted
- pickled
- wet blue (chromium iii) tanned,, but not yet dried
- wet white tanned are hides pre-tanned with alum sulphate. They have been limed and pickled

The processes used to produce various categories of skins are discussed in section 3 of this document.

The processes of tanning and pickling destroy contaminating pathogens that are on or in the skin. For this reason processed leather is traded world-wide without restrictions (section 2.3.2.1). Any hides that have been pickled, limed or tanned are considered to be safe. However, pathogens may survive the milder processes of air drying or salting. Therefore this risk analysis is restricted to the case of air dried or salted skins.

References

(1) Passman T, Director, Leather and Shoe Research Association (LASRA), Letter to HJ Pharo, dated 14/6/2000.

(2) OIE (2005). *Terrestrial Animal Health Code* *http://www.oie.int/eng/normes/MCode/en_chapitre_2.3.8.htm*

3 Processes In Leather Manufacture

The processes used in leather manufacture, up to tannage are as follows:

Flaying – removing the hide or skin from the animal

Fleshing – cutting away unwanted fat and flesh

Curing – (drying, salting) to preserve raw hides or skins during transport or storage

Soaking (wet-salted material) or *soaking back* (dry material) – to restore "cured" hides and skins to a rehydrated condition

Unhairing – removing the hair from hides

De-wooling or fellmongering – recovering the wool from skins

Liming – loosening hair/wool, fat, flesh, etc., removing interfibrillary material and "plumping up" the skin ready for tanning

Lime splitting – the machine cutting of limed hide into layers where this may be done as an alternative to the splitting of tanned hide

Deliming – neutralising the alkali used in liming

Bating – making the hide/skin softer and cleaner

Pickling – bringing the hide/skin to the right acidity for tannage or shipment. Skins may be preserved and traded at this stage as pickled pelts

Degreasing – removing natural grease, mainly from sheep skins and pickled pelts

Tannage – the hides and skins are tanned by a variety of methods

Due to the nature of the above processes, tannery workers must wear protective clothing. This risk assessment is carried out on the assumption that at the end of the day such protective clothing will be removed and workers will shower before leaving the tannery. Further, it is assumed that the tannery will have the protective clothing laundered professionally.

3.1 FLAYING

Following slaughter, the hide is removed from the carcass by a combination of cutting and traction. Hides may also be removed from animals that have died of natural causes.

3.2 FLESHING

Fleshing is carried out to remove residual meat tissue and fat from the hide/skin. This would usually be done prior to wet salting or brining or fellmongering, and prior to or after liming.

3.3 CURING

After removing the hide/skin from the carcass, some form of preservation is necessary to prevent putrefaction except in situations where the hide/skin is to be immediately processed into leather.

- Freezing and freeze-drying. These techniques are considered too expensive for common use. Rapid chilling to 2-3°C by blasting with air at 1°C overnight allows hides to be stored for two weeks in a cold store.

- A number of biocides are available to impede bacterial growth for various periods:

(i) spraying with a 10% solution of biguanidine hydrochloride onto the flesh side, giving a storage life of 4-5 days;
(ii) immersing in or spraying with a 0.08% solution of sodium dimethyl dithiocarbamate, giving preservation for several days;
(iii) immersing in a 0.2% commercial formalin, resulting in preservation for several months.

- Drying is an effective means of protecting against putrefaction, especially in countries with hot and dry climates (India, Africa, Australia, South America). It has the added advantage of considerably reducing weight and transport costs. Dry hides may be ground dried, sun dried, frame dried, or shade dried. To prevent insect attack, such hides in some countries may be treated with naphthalene, sodium silicofluoride, or other insecticide by means of spray, dip or dusting powder.

- Dry salting is the term used to describe the process by which hides are first salted and then hung up to dry.

- Wet-salting and brining processes aim to achieve a bacteriostatic salt content in the hide. Additives may have been added to the salt to protect against any salt-tolerant bacteria. Such additives are usually based on boric acid, and dichlorophen or naphthalene. Naphthalene also protects against insect infestation. The aim of salting is to reduce and inactivate the bacteria that damage the skin by causing decomposition. It is not primarily aimed at eliminating pathogens. A cured hide should contain less than 50% moisture and the moisture should be at least 85% saturated with salt. In this state the hide will resist decomposition. Some bacteria are killed but some become inactive or dormant and will again become active when the salt is removed by soaking. The numbers and types of bacteria that will survive the soaking process are not well

known and are also affected by additives/preservatives used in the salting and soaking solutions. Salting processes produce an effluent of 5 litres of brine per 25 kg hide. This effluent is not sterile. Generally an imported wet-salted or brined hide will have been cured with about half its weight of salt, together with about 0.5% boric acid and perhaps 0.25% of a secondary additive.

3.4 SOAKING BACK

To restore cured hides and skins to a natural raw condition prior to tanning, they must be soaked in water. "Soaking back" involves putting the hides or skins into an enclosed vessel and soaking them for 16 hours or more (usually overnight) at ambient temperature in water, which will usually contain detergent (e.g. alcohol ethoxylate, a non-ionic surfactant, at a concentration of about 5%) and biocides (e.g. metabisulphite at a level of about 0.5%).

A small amount of alkali is commonly added to aid in hide rehydration and to raise the pH in preparation for unhairing and liming. The amount of water would normally be about 1-2 times the weight of hides, and the pH would usually be between 9 and 10.

Sodium carbonate is commonly used and this reagent delivers pH values of up to 11 in the soaking back stage, depending on requirements.

The salt is largely soaked out of the hides and skins along with some blood and dirt, and the hides and skins become cleaner and softer and increase in weight owing to the absorption of water.

Water used in soaking back would be discharged into the normal sewage system.

In respect of any requirement for disinfection beyond the level afforded by the usual inclusion of a biocide in the soaking, New Zealand processors have recourse to the methods stipulated for disinfection in the First Schedule of the Anthrax Prevention Regulations 1987. These methods stipulate immersion in a 1/10 000 solution of sodium bisulphate for not less than 5 hours or immersion in a solution of free chlorine to a level of 200 ppm for not less than 2 hours

3.5 UNHAIRING AND LIMING

The aim of unhairing/de-wooling and liming is to remove/recover the hair or wool, the epidermis, and to some degree the inter-fibrillary proteins, and in some instances to prepare the hide or skin for removal of loose flesh and fat by the fleshing process and subsequent splitting, where this is done in preference to splitting tanned hide.

The most common method for the recovery of wool from sheepskins is to spray the flesh side of the sheepskin with a viscous "paint" which is a solution of sodium sulphide at concentrations of around 20% thickened in New Zealand with modified starch products and some hydrated lime. The soluble chemicals in the paint penetrate the skin from the flesh side and dissolve the basal young epidermal cells of the

epidermis and the region near the wool root, thus loosening the wool which is removed by hand or more commonly machine pulling after the skin is left standing for periods of between 2 and 16 hours.

The same chemical principle applies in the unhairing of hides, which takes about 2-5 hours, and is carried out in a large rotating drum or inclined processor. The hair may either be completely pulped and discharged together with liquid effluents, or it may be removed in the "hair save" system, in which case it can be screened out of the effluent as a wet solid.

There are many systems of liming, but virtually all systems use sodium sulphide to create a highly alkaline reducing environment. The minimum pH for depilation is 12.5, and this was traditionally achieved by the use of saturated lime. However, modern processes which are aimed to increase the speed of depilation generally involve pH levels of above 13, through the use of aggressive reducing agents such as sodium sulphide at a concentration of about 2%. These conditions are maintained for around 16 hours (usually overnight) at a temperature of 25-27°C; although some pelts (sheepskins from which wool has been removed) may be limed for only 6 hours.

At the end of the liming process the fluid is discharged into a separate waste stream which goes into aeration systems in the presence of a manganese sulphate catalyst in order to convert the potentially toxic sulphide to sulphate or thiosulphate. If this is not done, a slight drop in the pH of the alkaline waste stream will result in the production of hydrogen sulphide gas (H_2S) which is highly toxic. After this detoxifying process, the alkaline waste stream can be safely mixed with acidic waste discharged after pickling to get the target pH allowable.

3.6 DELIMING

After liming, the lime and other free and combined alkali in the skin must be removed or neutralised.

Chemical deliming may be carried out using a range of acidic agents (strong acids such as sulphuric or hydrochloric, which are difficult to control and potentially damaging, are not commonly used, but rather weak acids such as boric or ammonium salts are). Another system in use in this country is the injection of CO_2, which brings the pH down to about 8 without production of ammonia gas. Chemical deliming takes on average 90 minutes, although for heavy ox hides it may require up to 2½ hours. The target pH will vary depending on the process to follow (i.e. if bating is to follow, which is the more usual procedure, the target pH will be higher than if the hides or skins are going straight to pickling).

3.7 BATING

After deliming, a proteolytic enzyme known as 'bate' is added to the liquid to clean up the hide further by removing unwanted inter-fibrillary proteins, resulting in a softer and more stretchy leather. During bating the pH is usually 8.0 to 9.0, and the bating liquid is maintained at about 32-35°C to achieve maximum proteolytic activity.

Peroxide (an oxidising agent) is often included at this stage to remove residual sulphide out of the hide/skin matrix so that the application of acidic pickling agents will not result in the production of H_2S.

3.8 PICKLING

The use of liquors containing acid and salt is referred to as pickling. This can be either a short process in preparation for tanning (this is used for hides in New Zealand, where hides are exported in the 'wet-blue' form) or a longer process for medium-term preservation, which allows the pickled product to be exported (this is done for all pelts in New Zealand, even for those pelts which are to progress straight on to tanning). Pelt pickling includes a fungicide, such as 0.05% 2 (thiocyanomethylthio) benzothiazole.

The pickling process for pelts aims to preserve them for up to 12 months. In that case the pickling liquor pH is 0.9 to 1.0, and this is maintained for around 16 hours (overnight). If tanning is to follow pickling immediately, as is the case for hides in New Zealand, then the pickling pH is around 2.5 – 3.0.

Pickling liquor is usually recycled, at least in part, and the balance is discharged to trade waste.

Following pickling, lamb pelts must be degreased prior to tanning. This involves drumming for 1-2 hours with a solvent such as white spirits or kerosene or more commonly in New Zealand an aqueous surfactant system having usually increased the ability of the pelt to withstand a higher temperature for fat removal by a minor tannage or pre-tannage.

3.9 TANNING

The tanning process converts the protein of the raw hide or skin into a stable material, which will not putrefy. A wide variety of tanning materials are available (mineral, vegetable, aldehyde or synthetic) and the choice depends mainly on the desired properties of the finished leather.

The most common method all over the world is chrome tanning, resulting in "wet-blue" hides and pelts. Probably 90% of the leather produced worldwide is tanned using this method. Chrome tanning begins at a low pH for a few hours to allow the chrome oxide (chromium) to penetrate the hide or skin, and then the pH is raised somewhat to fix it. The whole process takes about 8-10 hours and the finishing pH is 3.5 – 4.0.

Vegetable tanning has in the past focussed largely on extracts of the mimosa plant. For heavy leathers such as belts, bags, harnesses, saddles, and sole leather, wattle is the tanning agent of choice, but the brown colour that is associated with wattle-tanned leather and its lack of light fastness is not always appropriate.

3.10 WOOL SKIN TANNING

For tanning wool sheepskins, there are some differences in the processes used. First and foremost, the highly alkaline liming step is not used, as it would obviously destroy the wool. Instead, soaking back in a mildly alkaline environment with biocides is followed by pickling and then tanning.

Various synthetic tanning agents that are more or less equivalent to vegetable tannages have been developed in the past 50 years, and these are widely used in the white tanning of woolskins, in preference to chrome tanning which imparts a green tinge to the wool. One group of these agents is called 'syntans' and these are used widely. However, for use in woolskins oxazolidine and phosphonium compounds have to a large degree taken over from the syntans as the resulting tanned product is not as bulky. These compounds have an action very similar to formaldehyde, and considerable care has to be taken in the final stages of tanning to remove residual formaldehyde from the product – this is usually done through the use of strong oxidisers such as peroxide to negate the formaldehyde.

The steps in tanning such skins are:

- Sheepskins are imported in salted/bacteriostat form, 1-6 months old
- Overnight soaking with detergent and bactericide and mild alkali
- Trimming
- Fleshing
- Scouring with anionic detergent and alkali
- Fleshing
- Pickling
- Tanning e.g. using syntan for 48 hours at pH 2.8 - 3.3
- Stand to drain for 1 day
- Retannage using aluminium sulphate or chromium for 24 hours at pH 3.6 – 3.8
- Rinsing
- Dry cleaning with white spirits

3.11 CONCLUSION

The least processed form of hides and skins likely to be imported into New Zealand are those that have been cured either by air drying (or dry salting) or by wet salting. The other methods of curing are either too expensive (freezing) or of too short a duration (biocides and short term chemical treatments) to be realistically applied to hides and skins imported into New Zealand.

Curing methods cannot ensure the destruction of all pathogens which may be associated with hides and skins at the time of slaughter. Therefore, at the time of importation hides and skins may carry certain pathogens.

The key steps in terms of inactivation of disease agents are likely to be:

- disinfection on receipt of hides/skins according to the requirements of the Anthrax Prevention Regulations 1987, which is a provision accepted by the industry for use as required.

- liming, in highly alkaline conditions (pH > 12.5) for 6 to 16 hours.

- pickling, in highly acidic conditions (pH < 3) for up to 16 hours.

- tanning, e.g. chrome tanning takes about 8 to10 hours, starting at a pH of about 2.0 to 3.0, and ending at about pH 3.8 to 4.0.

Washing and soaking back would have little effect on pathogens present unless they contained a biocide appropriate to the risk and/or were treated at an alkaline pH. These initial processes without such provisions may generate large volumes of potentially contaminated waste water. Similarly, fleshing following soaking back would have no effect on pathogens but could potentially generate contaminated solid waste unless the hides/skins had been subject to treatment.

Thus the main effluent flows of potential concern, prior to liming, are:

- water from soaking back, containing salt, blood, dirt

- solid wastes from fleshing prior to liming

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4 Hazard Identification

In this risk analysis, the starting point for the hazard identification is the disease lists of the OIE. When this risk analysis was initiated, OIE listed diseases were in two lists, A and B, and although the OIE list arrangement was changed in 2004, the diseases on the lists are the same and for this risk analysis the original A and B lists have been retained for convenience. The zoonotic agents of Crimean-Congo haemorrhagic fever and West Nile Fever were included because they were added to the new OIE lists in 2005. Besides these listed diseases two important infections of ruminants (bovine viral diarrhoea of cattle and border disease of sheep) have been included. A section on all Enterobacteriacae including *Salmonella* spp. is also included. Specific diseases of ostriches and emus are included as these animals are included in the commodity definition. The section on infectious bovine rhinotracheitis considers all bovine herpes viruses. At request of the Ministry of Healthi, the transmissible spongiform encephalopathies of deer are included. In addition, several unwanted insects and arachnids are also included, as well as weed seeds and the broad category of 'hitch-hiker pests'. The organisms of concern are listed in Table 1.

Table 1. Disease agents of possible concern

Name	**Type**
LIST A DISEASES	
Foot and mouth disease	virus
Vesicular stomatitis	virus
Swine vesicular disease	virus
Rinderpest	virus
Peste des petits ruminants	virus
Contagious bovine pleuropneumonia	mycoplasma
Lumpy skin disease	virus
Rift Valley fever	virus
Bluetongue	virus
Sheep pox and goat pox	virus
African horse sickness	virus
African swine fever	virus
Classical swine fever	virus
Highly pathogenic avian influenza	virus
Newcastle disease	virus
NEW OIE-LISTED DISEASES	
Crimean Congo haemorrhagic fever	virus
West Nile fever	virus
LIST B DISEASES **DISEASES OF MULTIPLE SPECIES**	
Bacillus anthracis (anthrax)	bacteria
Aujeszky's disease	virus
Echinococcus granulosus (hydatidosis)	cestode
Erhlichia ruminantium (heartwater)	rickettsia
Leptospira spp. (leptospirosis)	bacteria

[i] Mary Harvey, Ministry of Health, email to HJ Pharo dated 10/5/2000.

Coxiella burnetii (Q fever)	bacteria
Rabies	virus
Mycobacterium paratuberculosis (Johne's disease)	bacteria
Cochliomyia hominivorax (new world screw-worm)	insect
Chrsomyia bezziana (old world screw-worm)	Insect
DISEASES OF CATTLE	
Anaplasma spp. (bovine anaplasmosis)	rickettsia
Babesia spp. (bovine babesiosis)	protozoa
Brucella abortus (bovine brucellosis)	bacteria
Campylobacter fetus subsp. *venerealis* (bovine genital campylobacteriosis)	bacteria
Mycobacterium bovis (bovine tuberculosis)	bacteria
Cysticercus bovis (bovine tapeworm cysts)	cestode
Dermatophilus congolensis (dermatophilosis)	bacteria
Enzootic bovine leukosis	virus
Pasteurella multocida (haemorrhagic septicaemia)	bacteria
IBR/IPV and other bovine herpes virus infections	virus
Theileria spp.(theileriosis)	protozoa
Trichomonas fetus (trichomonosis)	protozoa
Trypanosoma spp. (African typanosomosis)	protozoa
Malignant catarrhal fever	virus
Bovine spongiform encephalopathy	prion
Bovine viral diarrhoea	virus
DISEASES OF SMALL RUMINANTS	
Brucella ovis (ovine epididymitis)	bacteria
Brucella melitensis (caprine and ovine brucellosis)	bacteria
Caprine arthritis/encephalitis	virus
Mycoplasma agalactia (contagious agalactia)	mycoplasma
Mycoplasma capripneumoniae (contagious caprine pleuropneumonia)	mycoplasma
Chlamydophila abortus (enzootic abortion of ewes)	chlamydia
Ovine pulmonary adenomatosis	virus
Nairobi sheep disease	virus
Salmonella spp. And Enterobacteriaceae	bacteria
Scrapie	prion
Maedi-visna	virus
Border disease	virus
DISEASES OF HORSES	
Taylorella equigenitalis (contagious equine metritis)	bacteria
Trypanosoma equiperdum (dourine)	protozoa
Histoplasma capsulatum var farciminosis (epizootic lymphangitis)	fungus
Equine encephalitides	virus
Equine infectious anaemia	virus
Equine influenza	virus
Babesia spp (equine piroplasmosis)	protozoa
Equine rhinopneumonitis	virus
Burkholdria pseudomallei (glanders)	bacteria
Horse pox	virus
Equine viral arteritis	virus
Japanese encephalitis	virus
Trypanosoma evansi (surra)	protozoa

Venezuelan equine encephalomyelitis	virus
DISEASES OF PIGS	
Pasteurella multocida (atrophic rhinitis of pigs)	bacteria
Cysticercus suis (pig tapeworm cysts)	cestode
Brucella suis (porcine brucellosis)	bacteria
Transmissible gastroenteritis	virus
Trichinella spiralis (trichinellosis)	nematode
Enterovirus encephalomyelitis	virus
Porcine reproductive/ respiratory syndrome	virus
MISCELLANEOUS DISEASES	
Chronic wasting disease of deer	prion
Arthropod parasites of skin	mite
Hypoderma bovis and *H lineatum* (warble flies)	insect
'Hitch-hiker pests'	Insects
Weed seeds	Plant

Infectious agents/diseases in table 1 were generally submitted to individual hazard identification and in a few cases closely related organisms (e.g. the *Brucella* spp.) were combined into groups of for this step (see sections 4.2 - 4.75). Hazard identification was carried out using the criteria described in section 4.1. Organisms judged to be potential hazards in the commodity were subjected to a risk assessment in section 5. Finally, in section 6 for risk management options are explored for organisms that were classified as risks in section 5.

4.1 CRITERIA FOR CLASSIFICATION AS POTENTIAL HAZARDS

Organisms in table 1 were submitted to hazard identification and classified as potential hazards if they were:

- disease agents that are exotic to New Zealand; or
- disease agents that occur in New Zealand but for which an eradication programme administered by a Pest Management Strategy under the Biosecurity Act is in place; or
- disease agents that occur in New Zealand but for which there are known sub-species or strains or host associations that do not occur in New Zealand and are potentially harmful; or
- Zoonotic disease agents that are already in New Zealand but, because of the nature of the commodities, are, if imported, likely to significantly increase existing hazards associated with them; or
- Zoonotic disease agents that occur only in well-defined geographically bounded areas of New Zealand.

Additionally, disease agents were only considered to be potential hazards requiring risk assessment if an investigation of the epidemiology of the disease revealed that

there was a potential for them to be present in hides and/or skins and that they could potentially be transmitted directly or indirectly to animals, humans or the environment.

Some disease agents could be present in hides or skins but cannot be transmitted from them to animals. Therefore the following categories of disease agents were not classified as potential hazards and were excluded from further analysis:

- disease agents that are transmitted exclusively by arthropod vectors which will not be found on dead skins.

- disease agents that are only transmitted venereally.

- diseases that are transmitted exclusively by the respiratory route in aerosols or droplets generated by a living animal.

- arthropod pests that are parasites of living animals but do not survive on skins and hides.

4.2 FOOT AND MOUTH DISEASE

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

The respiratory or oral routes are the usual routes 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, especially skin, giving rise to characteristic lesions of FMD. High titres of the virus may be found without lesions in many tissues (1).

FMD virus has been found in skin from all areas of the body. In steer skins, FMD virus has been found to persist for as long as 5 days after cessation of viraemia, at titres of up to $10^{3.6}$ pfu per gram of skin (4). Normal drying and salting of skins has not been shown to be effective in destroying foot and mouth disease virus. The *Code* (article 3.6.2.5) recommends salting for 28 days in sea salt containing 2% sodium carbonate.

Conclusion: As it may be present in the skin of infected animals and may survive salting (without addition of sodium carbonate) and drying, FMD virus is considered to be a potential hazard in the commodity.

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

The natural history of VS, including its endemic, maintenance and epizootic patterns, remains enigmatic (3). However, numerous observations incriminate biting arthropods as vectors. 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 viraemia occurs in VS infections of horses, cattle, and pigs (4). Some authors suggest that viraemia does not occur in mammalian hosts (5). It has been suggested that insects may become infected from non-domestic animal species which have a prolonged high-titre viraemia after infection (6). However a maintenance host of the virus has not been identified. It has also been suggested that direct feeding from skin lesions is the most likely route of insect infection (4), and transmission of the virus from infected to non-infected blackfly that co-feed on the same host has been demonstrated (5).

There is no evidence for persistence and subsequent shedding of VS virus in livestock (6). No virus, viral antigens, or viral RNA was found in experimentally infected pigs beyond 6 days post-infection (7). Immuno-suppression of recovered pigs has not produced recrudescence or virus shedding (4). All available epidemiological evidence suggests that the disease is transmitted by insect vectors

Conclusion: In view of the very low levels of viraemia and the non-contagious nature of this disease, and because it is considered to be an insect-borne disease, vesicular stomatitis virus is not considered to be a potential hazard in the commodity.

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4.4 SWINE VESICULAR DISEASE

Swine vesicular disease (SVD) is a contagious vesicular disease of pigs, caused by a porcine variant of human coxsackievirus B5 of the genus *Enterovirus* in the family *Picornaviridae* (1). Strains of SVD vary in virulence, and infection may be sub-clinical or result in mild or severe disease (2). The main importance of SVD is that it is clinically indistinguishable from foot and mouth disease. It 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. 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).

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 occurs most commonly via skin abrasions (6). 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).

Swine vesicular disease virus is very resistant to environmental factors and disinfectants, and survives over a wide pH range (7). Enteroviruses may survive for months in soil, and for up to 15 days on vegetable matter (8).

Conclusion: The virus is present in excretions and secretions of infected animals. Since it is highly resistant to environmental conditions and there is no specific information about its survival in dried and salted hides it must be assumed that it could survive in them. Therefore SVD virus is considered to be a potential hazard in the commodity.

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4.5 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).

Transmission requires close contact between sick and healthy animals. Natural infection is usually via the upper respiratory tract following inhalation of virus in aerosols, or via the oropharynx as a result of ingestion of material containing virus. The virus has an affinity for lymphoid tissue (particularly T-lymphocytes), and primary replication has not been demonstrated in the invaded epithelium. The incubation period following contact infection is from 8-15 days. The virulence of different strains of virus is related to the ability to infect lymphoid cells and mononuclear phagocytes, as it is these cells that transport the virus to epithelial tissues, especially those of the alimentary tract. During disease the virus is also found in non-lymphoid organs, such as lungs, liver, and kidneys (2).

Virulent strains of rinderpest virus are excreted from infected epithelial tissues for 1-2 days prior to the development of fever, and this continues for 9-10 days after the start of fever. Virus excretion declines as the immune response develops (2). At the height of excretion, 3-6 days after the onset of fever, large amounts of virus are excreted in expired air, nasal and oral secretions, and in urine and faeces.

Recovery from the disease results in lifelong immunity, and it is generally accepted that recovered cattle are free from infection and that there is no carrier state (2).

The virus is fragile and survives for only a few hours outside the host and is therefore unlikely to survive in dried and salted skins (2). In addition, rinderpest has been virtually eliminated from the world. One outbreak of the disease was reported in Kenya in 2003 and it has not been reported since then in any country (3).

Conclusion: As there appears to be no carrier state in recovered animals, the likelihood of the virus being present in hides and skins from clinically normal animals is remote. In addition, the virus is fragile and does not survive outside of the animal and the disease has been virtually eliminated from the world. Therefore, rinderpest virus is not considered to be a potential hazard in the commodity.

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4.6 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 sub-clinical or are insufficiently severe to attract veterinary attention (2). However, in naïve populations mortality can vary from 20-90% (2).

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).

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. 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).

As with rinderpest virus, PPR virus survives for only a short period outside the host (2). It is destroyed by desiccation within 4 days (4).

Conclusion: Since the virus is transmitted predominantly by inhalation and only survives for short periods outside the host, the virus is not considered to be a hazard in the commodity.

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4.7 CONTAGIOUS BOVINE PLEUROPNEUMONIA

Contagious bovine pleuropneumonia (CBPP) is a contagious disease of cattle caused by *Mycoplasma mycoides* subsp. *mycoides* Small Colony (MmmSC) (bovine biotype) (1).

Many recovered animals have pulmonary sequestra which result in a 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. Transmission is by droplet infection and direct contact between susceptible and diseased animals (i.e. either cattle with clinical disease or carriers that are actively excreting the organism) (2).

As it lacks a cell wall, MmmSC is readily inactivated in the environment. Exposure to UV light inactivates the organism within a few minutes. It does not persist in the environment and there is no evidence that it is transmitted on fomites (2).

Conclusion: Since CBPP can be transmitted only by direct contact between infected and susceptible animals, MmmSC is not considered to be a potential hazard in the commodity.

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4.8 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 (2).

It appears that infected animals are 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 a potential 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).

The LSD virus has been recovered from lesions on air-dried hides after a period of 18 days (2).

Conclusion: Since the virus may be present in hides and skins from infected animals, LSD virus is considered to be a potential hazard in the commodity.

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4.9 RIFT VALLEY FEVER

Rift Valley fever (RVF) is a peracute or acute disease of domestic ruminants in Africa, and more recently also Saudi Arabia and Yemen. It is caused by mosquito-borne viruses belonging to the Rift Valley fever complex within the genus *Phlebovirus* in the family *Bunyaviridae* (1).

The disease is most severe in sheep and goats, producing high mortality rates in newborn animals and abortions in pregnant animals. However, many infections with RVF virus are inapparent or mild, especially in adult cattle (2).

RVF is also a zoonosis, and close association with domestic animals is an important risk factor for human infections. Humans become infected with the virus mainly by direct contact with blood and tissues of sick animals or by a mosquito bite (3). It is therefore primarily an occupational hazard for farm, abattoir, and veterinary workers in countries where the virus is present. 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 (4). Mortality in humans is generally low but in an outbreak in Saudi Arabia there were 882 confirmed cases and 124 deaths. The high proportion of deaths reported may have been influenced by under-reporting of mild cases (5).

Although the virus is stable in serum, from which it can be recovered after several months storage at 4°C or after 3 hours at 56°C, under natural conditions it is rapidly inactivated outside its host or vector (2). In carcasses the virus is rapidly inactivated by pH changes following slaughter (4).

Conclusion: Since the Rift Valley fever virus is rapidly inactivated outside its host and is an insect-borne virus, it is not considered to be a potential hazard in the commodity.

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4.10 BLUETONGUE

Bluetongue (BT) is an infection of sheep and other domestic and wild ruminants, caused by viruses (24 serotypes) within 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%. Many infections in sheep are clinically inapparent, even in fully susceptible animals (3).

According to the *Code*, the global BTV distribution is currently between latitudes of approximately 50°N and 35°S but is known to be expanding in the northern hemisphere. However, for the next edition of the code it is expected that the southern limit will be altered to 34°S and New Zealand is entirely below this limit. However, 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 (4).

BTV is largely cell-associated, involving erythrocytes and leukocytes, and only a very small fraction of virus is found free in plasma (5). Replication is primarily in endothelial cells and pericytes of capillaries and small blood vessels (4). Under natural conditions the BT virus does not persist outside its host or vector, and apart from limited transmission via genetic material or across the placenta of viraemic animals, transmission occurs only by competent *Culicoides* midges (4).

Conclusion: Apart from rare transplacental transmission or transmission via genetic material, BTV is transmitted exclusively by *Culicoides* midges, which are not present in New Zealand. Therefore it is not considered to be a potential hazard in the commodity.

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4.11 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 virus belonging to the genus *Capripoxvirus* in the family *Poxviridae* (1). Most strains of the virus are specific to the host species from 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. 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 stable 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. 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). The closely related lumpy skin disease virus has been shown to survive 18 days in dried skins (9).

Conclusion: Since pox viruses may be present in the skin of infected animals, sheep pox and goat pox viruses are considered to be potential hazards in the commodity.

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4.12 AFRICAN HORSE SICKNESS

African horse sickness (AHS) is a peracute, acute, subacute or mild infectious but non-contagious disease of equine animals caused by viruses (9 serotypes) belonging to the genus *Orbivirus* in the family *Reoviridae* (1). AHS virus is transmitted biologically by certain species of *Culicoides* (Diptera: Ceratopogonidae), which are not present in New Zealand (2).

The distribution of AHS virus mirrors the distribution of its main insect vector, *Culicoides 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 (3).

Following the bite of an infected insect, there is a viraemia and dissemination of the AHS virus to all tissues via the blood. The AHS virus is associated with erythrocytes; very little is present in plasma (3). Dogs are susceptible and have been infected by eating infected horse meat (4). Apart from the consumption of horse flesh by dogs, transmission occurs only as a result of biting by competent *Culicoides* midges (3).

Conclusion: Since AHS virus is almost exclusively transmitted by *Culicoides* midges which are not present in New Zealand, it is not considered to be a potential hazard in the commodity.

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4.13 AFRICAN SWINE FEVER

African swine fever (ASF) is a disease of domestic pigs caused by a unique virus that has been classified as the sole member of a new genus, *Asfivirus*, in the family *Asfarviridae* (1). 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). Spain has been free from the disease since 1994 and Portugal since 1999, but Italy is still classed as infected (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). Transmission in domestic pigs probably occurs by the oronasal route. Vertical transmission has never been reliably reported (3).

Inapparent 'carriers' have been recognised but their role in the maintenance and spread of the virus is uncertain. Serological surveys in various infected countries have indicated that between 0.3% and 8% of sera from slaughtered pigs can be positive (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 re-activation by stress for the following period of up to 6 months (3).

The stability of the ASF virus is well-recognised. It has survived in serum at room temperature for 18 months, in blood in a refrigerator for at least 6 years, at 37°C for up to a month, and at 55°C for 30 minutes. Putrefaction does not destroy the virus quickly. ASF virus has persisted in faeces at room temperature for 11 days (3).

Conclusion: Since ASF virus is present in all secretions and excretions of infected animals, it is possible that contamination of the skin may occur. The virus is highly resistant to environmental conditions and is therefore considered to be a potential hazard in the commodity.

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4.14 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. 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).

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. Infected pigs shed large quantities of the virus, especially in saliva (3). Transmission of CSF virus is mainly by the oro-nasal route, through direct contact (4). 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). Therefore infected live animals are an important means of spread.

Pig meat and meat products are also important vehicles for virus spread. The virus survives in fat tissue for several months (3). The movement of infected 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 a high density of pig populations (2).

The virus is usually inactivated in a few days outside the host (4).

Conclusion: Although it is possible that the virus may be present on pig skins through contamination with saliva of infected animals, the very limited survival of the virus outside the host would mean that such contamination is unlikely to be significant as far as the commodity is concerned. However, since CSF virus may survive for months in fat tissue, which may be present on hides and skins, the virus is considered to be a potential hazard in the commodity.

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4.15 HIGHLY PATHOGENIC AVIAN INFLUENZA

Avian influenza is caused by viruses of the genus *Influenzavirus A* in the family *Orthomyxoviridae* (1). All reported outbreaks of highly pathogenic avian influenza (HPAI) have been of the H5 or H7 subtype although many H5 and H7 subtype viruses isolated from birds have been of low virulence (2).

Since the mid-1970s influenza viruses have been isolated from avian species representing most of the major families of birds throughout the world. Migratory waterfowl, particularly ducks and geese, have yielded more avian influenza viruses than any other group of birds, but overt disease does not seem to occur in these birds (3). Outbreaks of avian influenza in ostriches have been reported from South Africa (4) and Zimbabwe (5), caused by viruses which were of low pathogenicity for chickens.

In general, transmission of infection is by close contact or indirectly from infected waterfowl and other wild birds. Infection occurs by ingestion of contaminated material, spread by equipment and humans. There is little evidence of airborne spread over significant distances. Wild birds are probably the most common method by which HPAI is introduced into domestic flocks, but there is increasing evidence that virulent viruses may arise by mutation of viruses of low pathogenicity which may be cycling covertly in domestic flocks. The greatest threat of secondary spread of avian influenza viruses is by mechanical transfer of infective faeces. For all birds, the ingestion of faeces appears to be the most important mode of transmission. Because of the intestinal nature of avian influenza infections in waterfowl, large quantities of virus are excreted in faeces. Virus has been shown to replicate and be excreted by ducks for 30 days, chickens for 36 days and turkeys for 72 days (6).

The survival of AI viruses in the environment is increased in cool and moist conditions. For example, the viruses have been recovered from liquid manure for 105 days after depopulation in wintertime following outbreaks of HPAI (6), and infectivity in faecal material has been retained for 30-35 days at 4°C, and for 7 days at 20°C (5). Transmission by fomites is considered possible (7).

Avian influenza virus could be present in the skins of viraemic birds (ostrich and emu skins) or on the surface of skins contaminated with faeces from subclinically infected birds.

In recent years there has been a global pandemic of avian influenza in poultry caused by an avian influenza H5N1 strain. This outbreak has been responsible for massive mortalities in poultry and has spread from Asia to Europe and Africa (8). The virus also occasionally infects humans that have close contact with poultry. Up to 11 April 2007 there had been 291 confirmed cases in humans and 172 deaths (9).

Conclusion: Avian influenza virus could be present in faeces contaminating ostrich hides, and may survive in faeces for 7 days at 20°C. It is a potentially zoonotic organism. Therefore avian influenza virus is considered to be a potential hazard on the commodity.

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4.16 NEWCASTLE DISEASE

Newcastle disease (ND) is caused by avian paramyxovirus 1 (APMV-1), a member of the genus *Rubulavirus* in the family *Paramyxoviridae* (1).

As APMV-1 infections have been reported in at least 236 species from 27 of the 50 Orders of birds, it seems probable that all birds are susceptible to infection (2). ND has been recorded in ostriches of all ages (3) (4), with birds less than 6 months of age being at greatest risk (5). Field and experimental data indicate that the production system has a major influence on the clinical severity of disease in ostriches. Severe respiratory disease with rapid spread and high mortality is generally seen only in closed chick-rearing units, while ostriches kept outdoors usually contract the disease by the oral route from faeces or water, resulting in nervous signs and a slow spread of infection (6).

Infection with APMV-1 results in large quantities of virus being shed in faeces, and the faecal-oral route is probably the major route of transmission (7). The virus is relatively stable in the environment, especially if protected from UV light. Poultry houses can remain contaminated for months. The virus can survive freezing for extended periods; it has been isolated from poultry carcasses frozen for 2 years, and may survive on poultry meat wrappings for as long as 9 months when stored at -14°C to -20°C (8).

Newcastle disease virus could be present in the skins of viraemic ostriches and emus or on the surface of skins contaminated with faeces from subclinically infected birds.

The greatest risk of spreading APMV-1 virus during an outbreak comes from movement of people and equipment. Other methods of spread of APMV-1 virus have been implicated in various epidemics (8) including:

- movement of live birds, including wild birds, pet/exotic birds, game birds, racing pigeons, and commercial poultry;
- movement of poultry products;
- contaminated poultry feed.

Conclusion: It is possible that contamination of ostrich or emu skins could occur in viraemic birds or as a result of faecal contamination, and since the virus is relatively stable in the environment, APMV-1 virus is considered to be a potential hazard on the commodity.

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4.17 CRIMEAN-CONGO HAEMORRHAGIC FEVER

Crimean-Congo haemorrhagic fever (CCHF) is caused by a member of the *Nairovirus* genus, Crimean-Congo haemorrhagic fever virus (CCHFV) (1).

CCHFV occurs in Africa, Asia, the Middle East and Eastern Europe (2) . Eastern European countries of the EU could therefore be endemically infected with the virus. The virus infects humans and a wide variety of ruminants and other smaller animals such as hares; it can also infect ostriches (2). In humans the virus causes a serious disease but in animals it causes a transient inapparent infection (2).

The principle methods of spread are by tick-bite and by contact with infected blood and meat. People involved in slaughtering animals are at risk (3) and nosocomial infections occurred in a South African hospital (4). The virus has been isolated from at least 30 species of ixodid ticks (2) but not from argasid ticks (5). Transovarial transmission of the virus in ticks has been described in a few species of the genera *Rhipicephalus*, *Hyalomma* and *Dermacentor* but it has been suggested that this does not occur regularly and that transstadial infection following amplification in a mammalian host is the usual method of transmission (2). *Hyalomma* spp. are the principle vectors of the disease and the distribution of the virus mirrors the distribution of these ticks (6).

The virus is relatively labile and does not survive in dried blood, at high temperatures (cooking of meat) or in a low pH environement (less than 6) and in matured meat (7)

<u>*Conclusion:*</u> As the principal vectors (*Hyalomma* spp.) are not found in New Zealand, and the virus is unlikely to survive on imported hides and skins, CCHFV is not considered to be a hazard in the commodity.

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4.18 WEST NILE FEVER

West Nile virus (WNV) is a member of the *Flavivirus* genus (1).

WNV was originally isolated in Uganda in 1937. It is found all over Africa and was found in France in 1962 and has been found in Romania (1996) and Russia (1999) (2). The virus spread to the United States in 1999 and since then has spread throughout the USA (3) and adjoining countries. The disease is seen mainly in humans and in horses and also causes deaths in wild birds. Most cases in humans are asymptomatic but in the epidemic in the USA there have been over 15,000 cases of disease and over 600 deaths (4).

The virus is transmitted by mosquitoes and maintained in a bird mosquito cycle (5). At least 43 species of mosquitoes have been suspected of acting as vectors of the disease (6). The virus can be transmitted from infected mosquitoes to non-infected mosquitoes when they feed together on non-infected hosts (4).

Studies on WNV have shown that virus is inactivated in the presence of detergent-based wash buffer after 30 minutes at 37^{o}C. WNV held at 28oC in a cell-free medium containing fetal calf serum shows a ten-fold decrease in viral titre after 24 hours (7). Viable virus is therefore unlikely to be present in imported hides and skins.

Conclusion: Since the WNV is rapidly inactivated outside its host, it is not considered to be a potential hazard in the commodity.

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4.19 ANTHRAX

Anthrax is a peracute, acute or subacute infectious bacterial disease caused by an aerobic spore-forming bacillus, *Bacillus anthracis* (1). Generally speaking, cattle, sheep, horses, and goats, in that order, are the most susceptible and commonly affected domestic animals (2), and in these species infection is usually by the oral route. The disease is also an important zoonosis. Transmission to humans can be by ingestion or inhalation of material contaminated with spores, or through spores contaminating skin abrasions (1).

Anthrax spores, which are highly resistant to the environment, are never found in the animal body during life. Rather, they are formed when the vegetative form of the anthrax organism is exposed to air, either in bloody discharges from body orifices of affected animals, or when the carcass of an animal that has died from the disease is opened. Anthrax bacilli do not remain alive in most tissues of unopened carcasses for longer than three days at temperatures of 25-30°C or higher, as they are rapidly killed by putrefactive organisms. However, at temperatures of 5-10°C the rate of decomposition of a carcass is reduced, and anthrax bacilli may still be recovered for up to 4 weeks (1).

Ambient temperature has an important effect on spore formation. Sporulation is slow at temperatures below 20°C. In countries with a cold climate, the temperature is unfavourable for sporulation for much of the year and anthrax tends to be self-limiting. However in countries with warm climates which favour the sporulation of *Bacillus anthracis* in body fluids and in pools of blood or serum in the immediate surroundings of an opened carcass, the occurrence of anthrax is closely integrated with a soil phase (1).

Spore survival depends on a number of factors such as the initial number of spores, the climate, topography, and the presence of soil saprophytes, certain chemicals, plant material and anthrax bacteriophages. In soils of high biological activity that contain a great diversity of microbial life, the survival period of spores is probably limited to around 3-4 years. However, anthrax spores may remain viable in soil for 50 years, or even up to 250 years if the soil is dry and has either a very low or a very high pH which adversely affects the biological activity of other microbial organisms (1).

Persistent outbreaks seem to be dependent on soils comprising calcium top soils, a granular alkaline soil, and shallow pans (3). A recent outbreak in Australia was restricted to farms which had areas of poorly drained, swampy, alluvial soils (4). However, it is likely that specific socio-economic conditions are also necessary for its persistence. The disease remains endemic in Africa and Asia where, following sudden death, the value of a carcass as meat for local consumption and as hide, hair, wool, and/or bones for sale greatly outweighs the perceived merits of burying or burning it (5). In temperate regions, infection in animals tends to occur sporadically through the importation of contaminated animal feed, while infections in humans are usually related to handling imported hides and wool (6).

Outbreaks of anthrax in New Zealand were usually traced to the use of imported bonemeal for fertilizer. With the advent of efficient controls on the importation of

bone for fertiliser outbreaks of anthrax became rare and ceased to occur. The last two outbreaks occurred on farms where previous outbreaks had occurred. The last suspected outbreak occurred in 1954 on two farms where anthrax had occurred 51 years previously. The outbreak occurred after old paddocks were ploughed and re-grassed (7). Freedom from the disease for over fifty years is probably due to soil and climate conditions being unsuitable for the production and long-term survival of spores.

Humans may develop localised cutaneous lesions (malignant pustule or malignant carbuncle) from contact of broken skin with infected blood or tissues, acquire a highly fatal haemorrhagic condition (woolsorters' disease or Bradford disease) from spore inhalation when handling contaminated wool or hair, or develop the intestinal form of the disease from eating infected meat (8). They occasionally develop acute meningitis as a complication of bacteraemia or intestinal anthrax from consumption of undercooked meat of animals which have died of anthrax (9). The most common form in humans is cutaneous anthrax which accounts for about 95-98% of human cases (2).

Anthrax in humans in New Zealand occurred in a few farmers who skinned animals which had died of the disease (7). The recent Australian outbreak resulted in one human case of cutaneous anthrax in a knackery worker, presumably from direct contact with infected carcasses (10).

Hides and skins coming from animals which died of the disease are likely to be contaminated with anthrax spores, and cutaneous anthrax in tannery workers has long been recognised as an occupational hazard (11). While the risk of contamination is minimal where hides come from animals that were slaughtered in supervised premises, dry hides of uncertain origin from countries where the disease is endemic are widely regarded as high risk material (11, 12).

<u>*Conclusion:*</u> *Bacillus anthracis* is well-recognised as a potential hazard in the commodity.

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4.20 AUJESZKY'S DISEASE

Aujeszky's disease (AD), also known as pseudorabies, is caused by suid herpesvirus-1 a member of the *Herpesviridae* family (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). Aujeszky's disease was eradicated from New Zealand in 1997 (4).

Pigs are the most important source of infection for all species. Primary sites of replication are the nasopharynx and the tonsil, and the associated lymphatics. The virus then infects nervous tissue and eventually the brain, which may result in nervous signs. Some strains have a tropism for the respiratory and genital tracts (5).

Acutely infected pigs excrete virus in nasal discharges, aerosols, saliva and semen. Close contact is required for virus spread (3). Susceptible pigs are infected by inhalation of virus-containing aerosols or by licking and biting of infected pen-mates (6). Transmission can also occur through contact with contaminated feed and water, and rats may spread the virus between farms. However, herpesviruses are fragile and do not survive well outside the body (6).

Conclusion: Although infected pigs excrete virus in secretions and excretions, the very limited survival of the virus outside the host would mean that such contamination is unlikely to be significant as far as the commodity is concerned. Therefore, Aujeszky's disease virus is not considered to be a potential hazard in the commodity.

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4.21 BOVINE VIRAL DIARRHOEA VIRUS

Bovine viral diarrhoea is caused by a flavivirus of the genus *Pestivirus*. There are two genotypes of bovine pestivirus, BVDV-1 and BVDV-2. In each genotype both cytopathic and non-cytopathic biotypes occur.

Bovine viral diarrhoea virus genotype 1 (BVDV-1) is endemic in New Zealand but genotype 2 (BVDV-2) is exotic.

BVDV-1 has a world-wide distribution, including New Zealand and Australia (1) (2). In New Zealand most animals have been exposed to BVDV-1 and the prevalence of antibodies is around 60% (3). BVDV-2 occurs in North America (4) and was introduced into Italy (5) and the Netherlands (6) in batches of contaminated vaccine. It also occurs in the United Kingdom (7, 8, 9, 10), where it might have been introduced by contaminated embryos. The only isolation of a BVDV-2 strain in New Zealand was from a batch of foetal calf serum imported from the USA (1). The virus was contained in the laboratory and New Zealand remains free from this sub-type of the virus. BVDV-2 has not been described in Australia.

The virus is normally transmitted by direct contact between infected animals and/or possibly by aerosol transmission over short distances (4). The incubation period is usually about 3-7 days (11) and the animals may remain viraemic for 4-15 days after initial infection (4). Duration of viraemia seldom exceeds 10-14 days (11). Antibodies develop 2-4 weeks after infection.

BVDV-1 infection of non-pregnant cattle usually results in mild infection typified by pyrexia and leukopenia from about 3-7 days. Viraemia and nasal excretion of the virus occurs during this period (11). The clinical signs are often so mild that they are not observed and occasionally diarrhoea is seen (4). Since it is widely distributed in most cattle herds, cattle are commonly infected before they become pregnant, resulting in a population of cattle that is largely immune and do not carry the virus. Infection of naïve pregnant animals, particularly during the first trimester, may result in death of the conceptus or full-term or near full-term delivery of immunotolerant persistently infected calves (3, 4, 11, 12). It was suggested that 7% of foetal deaths in Swiss dairy cattle may be caused by infection with BVDV (13). BVDV infection around the time of insemination significantly affected breeding performance (14). BVDV-2 strains that cause a more severe form of the disease following an initial infection were described in the USA (15). In these cases the mortality rate was up to 10% (4) and the disease was characterized by severe leukopenia and haemorrhagic disease (11).

Immunotolerant persistently infected animals may be clinically normal or may fail to thrive and die within a year. They are always infected with non-cytopathic strains of the virus (11). Superinfection of persistently infected animals with a cytopathic BVDV strain results in the development of mucosal disease (4, 11, 16). The cytopathic strain that re-infects the persistent carrier animals usually results from a mutation of the persistent non-cytopathic strain, but may also result from infection with a new extrinsic cytopathic virus (4, 11). Mucosal disease invariably terminates

fatally. In acute cases death occurs within 2-21 days while in chronic cases the animal may survive for up to 18 months (4).

The virus is stable below 10^0C and over a pH range of 3-9. It may survive for 3-7 days at 20^0C and for 3 weeks at 5^0C (4).

Conclusion: BVDV-1 is endemic in New Zealand. However, BVDV-2 virus is exotic and can cause severe disease. Since infected animals are septicaemic in the acute phase of the disease and persistently septicaemic immunotolerant animals occur, BVDV virus could be found in skins and in blood contaminating skins. Therefore BVDV-2 is considered to be a potential hazard in the commodity.

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4.22 BORDER DISEASE

Border disease is caused by a virus that is very closely related to bovine viral diarrhoea virus and is endemic in New Zealand.

Conclusion: Since the virus is endemic in New Zealand it is not considered to be a potential hazard in the commodity.

4.23 ECHINOCOCCOSIS/HYDATIDOSIS

Four species of *Echinococcus* tapeworms that occur in the small intestine of dogs or other carnivores are recognised. Only *Echinococcus granulosus* has ever been reported in New Zealand, and it has been eradicated. New Zealand was declared provisionally free from this parasite in 2002 (1).

The life cycle of *Echinococcus* spp. involves several stages. Eggs passed in the faeces of the definitive host are ingested by intermediate hosts in which the cystic stage develops in offal. Intermediate hosts are usually ruminants and pigs, or occasionally horses (2), and cysts also occasionally develop in humans.

Intermediate hosts can only become infected by ingesting eggs that contain viable embryos (oncospheres). Hides and skins could become contaminated with eggs if the animals were in direct contact with dog faeces, but as eggs of *Echinococcus granulosus* are very sensitive to desiccation, survival in the environment is limited (3). When eggs were stored at a relative humidity of 60 to 80%, oncospheres survived 1 and 2 days respectively (3), and, regardless of humidity, temperatures over 40°C are rapidly lethal (4).

For the parasite to re-establish in New Zealand, a definitive host (dog) would have to become infected by eating the offal of an infested intermediate hosts. Hides and skins will not have hydatid cysts attached to them and will therefore not be infectious for dogs. To become infested, intermediate hosts would have to ingest eggs containing viable onchospheres. The likelihood that dried or wet salted skins and hides will be infested with viable eggs is considered to be negligible.

Conclusion: Since hydatid cyts will not occur on hides and skins, *Echinococcus* is not consdiered to be a hazard in this commodity.

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4.24 HEARTWATER

Heartwater is a non-contagious tick-borne disease of ruminants caused by the rickettsia *Ehrlichia ruminantium* (1). 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 unless treated. However, many infections are inapparent, and animals with such infections act as reservoirs of the organism (1).

As *Ehrlichia ruminantium* is transmitted only by *Amblyomma* spp. ticks, the disease is confined to sub-Saharan Africa and the Caribbean (2). Infected ticks probably carry the organism for life (1). *Amblyomma* ticks are not present in New Zealand (3). Because *Amblyomma* spp occur mainly in warmer climates they are unlikely to be able to establish in New Zealand.

Ehrlichia ruminantium is heat labile and loses viability in 12-38 hours at room temperature (1).

<u>*Conclusion:*</u> *Ehrlichia ruminantium* is transmitted only by exotic ticks and does not survive for significant periods outside hosts or ticks. Therefore, it is not considered to be a potential hazard in the commodity. Exotic ticks are considered separately in this risk analysis.

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4.25 LEPTOSPIROSIS

Leptospirosis is a bacterial disease caused by spirochetes of the genus *Leptospira*. Pathogenic leptospires are now identified in seven species of *Leptospira*, comprised of 198 serovars arranged in 23 serogroups (1).

Leptospirosis is an important zoonosis, and infection in man is usually associated with exposure to the urine of infected animals. Leptospirosis is an occupational hazard of persons in close contact with animals, such as farmers, slaughterhouse workers, veterinarians, etc (2).

The disease in animals has been associated with haemolytic crisis, nephritis, mastitis, abortions, stillbirths, and reproductive failure in cattle and pigs, agalactia in sheep and goats, and ophthalmia in horses. Transmission is usually indirectly by water or mud contaminated with infected urine, particularly when skin is abraded or softened by prolonged immersion in water. Following infection, leptospires enter the bloodstream and localise in the liver, where primary replication takes place. From there the organisms are released into the blood, and the leptospiraemia results in organisms localising in the lungs, brain, kidneys, eyes or the pregnant uterus. Outside the host, leptospires may survive for long periods in wet soil or stagnant water, but drying destroys them quickly and they survive for only 30 minutes in air-dried soil (2).

Conclusion: Leptospires are sensitive to desiccation and will not survive in dried or salted hides. Therefore leptospires are not considered to be a potential hazard in the commodity.

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4.26 Q FEVER

Q fever is a zoonosis caused by an obligate intracellular bacterium, *Coxiella burnetii*, which is not closely related phylogenetically to other Rickettsiales (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.

Infection of humans may occur directly through contact with infected animals or indirectly through contact with contaminated dust, as the organism remains viable for long periods in the dry state (2). Human infections may be subclinical, acute or chronic and may cause influenza-like symptoms, pneumonia, hepatitis, and endocarditis. Spread between humans is rare (1).

Natural hosts include 40 species of hard and soft ticks of 11 genera, and a wide variety of animals and birds (2). 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 transovarially. Infections in mammals and birds similarly persist for long periods, if not for life (2).

Not all species of tick that become infected can transmit infection, as most carry the organism for only a short time after engorging on contaminated blood (3). Natural infections have not been reported in the New Zealand cattle tick, *Haemaphysalis longicornis* (4), and in experimental infections of that tick, transovarial transmission has not been demonstrated (5).

Initial transmission to populations of domestic animals is usually by tick bites or through contact with dried tick faeces. The lungs are the primary site of multiplication of *Coxiella burnetii*, and in the final stages of pregnancy there may be a massive multiplication of the organism in the uterus, foetal tissues and the udder (1). 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 (2).

No evidence of the organism has been found in New Zealand (6), and the small mammals in which infection has commonly been reported abroad (bandicoot, gerbil, porcupine) (5) are not present in this country.

Wool of sheep may be heavily contaminated by *Coxiella burnetii* excreted in the birth fluids and faeces at lambing, and since the organism is highly resistant in the environment, such contamination may persist for long periods (5). High-risk human occupations include all of those in which live and dead cows, does and ewes are handled (2). The fact that humans working with cattle hides in abattoirs frequently become seropositive (5) indicates that *Coxiella burnetii* contamination of hides is not uncommon.

<u>*Conclusion:*</u> *Coxiella burnetii* is highly resistant and can survive for long periods in the environment. It may contaminate wool, hair, and hides and is therefore considered to be a potential hazard in the commodity.

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4.27 RABIES

Rabies is a fatal nervous disease of warm-blooded vertebrates, caused by a *Lyssavirus* in the family *Rhabdoviridae* (1). Transmission is 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 animals are cattle. Sheep, goats, buffalo, horses, and pigs are rarely affected. Rabies is an important zoonosis (2). After infection by a bite the virus rapidly enters the nervous system and thereafter is not found in other tissues until the terminal stages of the disease which may occur weeks, months or years after the infection (2). Therefore skins from animals that are not clinically affected will not be infected with rabies virus (2). Rabies virus is sensitive to sunlight, ultraviolet light, heat, detergents, halogens, lipid solvents, and many disinfectants (2).

Chronic rabies, clinically inapparent infections, and recovery from clinical disease with persistent shedding are extremely rare, but have been described in Ethiopia, West Africa and India. The literature on this form of rabies has been reviewed (2, 3).

Conclusion: Rabies virus is not considered to be a potential hazard in the commodity as it will not be found in the skin of animals that are not in the terminal stage of the disease. In addition the virus does not survive long in the external environment.

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4.28 PARATUBERCULOSIS

Paratuberculosis or Johne's disease is a chronic infectious enteritis of cattle, sheep and goats caused by the bacterium *Mycobacterium avium* subspecies *paratuberculosis* (1). 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.

Cattle are usually infected during the first days or weeks of life after ingesting food or water contaminated with faeces of infected animals. Following oral infection, the bacteria enter the lymphatics through the tonsils and intestinal mucosa, where they are phagocytosed by macrophages. The majority of organisms are contained intracellularly in macrophages, where they are resistant to lysosomal enzymes and are therefore able to multiply. The majority of exposed animals become subclinically infected and shed bacteria intermittently in their faeces throughout their lives (1).

Mycobacterium avium subsp. *paratuberculosis* can survive for long periods in faeces, on pasture and in the presence of salt (2).

<u>*Conclusion:*</u> *M. avium* subsp. *paratuberculosis* could be associated with the commodity. However, since it is endemic in New Zealand, is not under regulatory control, and the import of hides and skins will not result in a significant increase in exposure, it is not considered to be a potential hazard in this risk analysis.

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4.29 SCREWWORM

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).

Wounding is usually a pre-requisite for SWF strike; eggs are laid on the periphery of wounds or body orifices in masses of up to 250 eggs. The eggs hatch within 12-20 hours, and the larvae burrow deep into the wound and feed on blood for 6-7 days, after which they drop off the host and burrow into soil to a depth of 2cm or more to pupate, usually within a week, but pupation may take up to 60 days in adverse conditions (1).

The average life span of adults is 21 days, but they require a supply of water and carbohydrate to survive more than a day or two. The optimal temperature range for the flies is 20-30°C. Flies will not move at temperatures below 10°C, and in the range 10-16°C they will not mate. Long distance spread of the disease is most likely to be by the transport of infested animals (1).

Screw worm larvae are not able to survive outside the host. Drying or salting of hides would destroy any larvae present.

Conclusion: Since screwworm larvae would not survive in dried or salted skins and the parasite could not establish in New Zealand, screwworm is not considered to be a potential hazard in the commodity.

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4.30 ANAPLASMOSIS

Bovine anaplasmosis is a disease caused by *Anaplasma marginale, Anaplasma centrale* or *Anaplasma caudatum*. Most outbreaks of clinical disease are caused by *Anaplasma 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). *Anaplasma centrale* causes mild infections and is used as a vaccine strain. *Anaplasma caudatum* causes occasional cases of anaplasmosis in the USA. *Anaplasma ovis* occurs in small ruminants but is often a mild infection in these species (2).

Anaplasma spp. have a wide distribution in the world, and are 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). Mechanical transmission by biting flies has been described.

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 (4).

New Zealand does not have capable tick vectors (5, 6).

<u>*Conclusion:*</u> *Anaplasma* spp. are carried by exotic arthropod vectors and are not considered to be potential hazards in the commodity.

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4.31 BABESIOSIS

Bovine babesiosis, or redwater as it is commonly known, is a tick-borne disease caused by intra-erythrocytic *Babesia* spp. The parasites and their usual vectors are listed below (1, 2):

.

Babesia bigemina	*Boophilus microplus, Boophilus decoloratus, Boophilus annulatus*.
Babesia bovis	*Boophilus microplus, Boophilus annulatus, Ixodes spp*.?.
Babesia divergens	*Ixodes ricinus*.
Babesia major	*Haemaphysalis punctata*.
Babesia jakimovi	*Ixodes ricinus*.
Babesia ovata	*Haemaphysalis longicornis*.
Babesia occulans	*Hyalomma marginata rufipes*.

The distribution of bovine babesiosis in the world depends entirely on the distribution of its tick vectors (2). 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 (3).

Babesia bigemina occurs in South America, the West Indies, Australia and Africa. *Babesia bovis* occurs in the tropics including South and Central America, Africa, Australia, Asia and southern Europe. *Boophilus microplus* and *Boophilus annulatus* are the major vectors of *Babesia bovis* and *Babesia bigemina* worldwide, although in Africa, *Boophilus decoloratus* is the vector of *Babesia bigemina* (2).

Babesia divergens occurs in north-west Europe, Spain, and Eire where the vectors are *Ixodes persulcatus* and *Dermacentor reticulates* (4). *Babesia divergens* is the principal cause of babesiosis in the United Kingdom where *Ixodes ricinus* is the vector (5).

Only *Haemaphysalis longicornis*, occurs in New Zealand (6) and it is not a known vector of the most important *Babesia* spp. (*Babesia bovis, Babesia bigemina* or *Babesia major*) (1, 6), but is a vector of *Babesia ovata* (1).

<u>*Conclusion:*</u> Since *Babesia* spp. are transmitted only by ticks, they are not considered to be potential hazards in the commodity.

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4.32 BRUCELLA SPECIES

Organisms of the *Brucella* genus are all very similar in their characteristics and can be considered as a group. Each *Brucella* sp. typically infects a particular species of animal but they may also rarely infect animals that are not their normal hosts. The important members of the genus are:

Brucella abortus (cattle)
Brucella melitensis (sheep and goats)
Brucella suis (pigs)
Brucella ovis (sheep and deer)
Brucella canis (dogs)

Brucella canis will not be considered in this risk analysis since it is a pathogen of dogs, and dog skins are not included in the scope of this analysis. *Brucella ovis* is endemic, and there would be no significant increase in exposure associated with this pathway, so is therefore not considered to be a potential hazard here.

Brucella abortus occurred worldwide but has been eradicated from many developed countries, including New Zealand (1). *Brucella suis* and *Brucella melitensis* do not occur in New Zealand.

Typically *Brucella* spp. cause abortions and infections of the genital tract, udder and associated lymph nodes. Sexually mature heifers (and especially pregnant cattle) are more susceptible to infection than immature heifers. Infected animals usually abort only once; most subsequent calves are carried to full term, 90% of infected cows remain chronically infected, sometimes for life, with infection confined to the udder and lymph nodes. Organisms are excreted in very large numbers in uterine discharges and in milk after abortion or calving. Up to 9% of heifers born from seropositive cows may be latently infected but serologically negative until the middle of their first gestation after which antibodies are usually 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 (2).

In other animals the position is broadly similar although arthritis may be a common sign, especially in pigs infected with *Brucella suis*.

Transmission is generally by the oral route when susceptible animals are exposed to the copious, massively infected discharges from infected cattle following abortion or calving (2).

Infections of animal species by *Brucella* spp. that are not the usual pathogens for the particular animal species do occur but are rare e.g. cattle are occasionally infected with *Brucella suis* or *Brucella melitensis.*

In some stages of the disease infected animals are septicaemic and infection of the skin is therefore possible. However, there is nothing in the literature that suggests that the disease is transmitted by hides and skins. It was found that the organism survived for up to 25 days on leather contaminated with infected manure and for up to 17 days

on leather contaminated with culture in skim milk (Parli (1957) cited by Mitscherlich and Marth (3)). The organism is highly resistant to the environment and may survive for months in manure and soil, and days in milk (see Mitscherlich and Marth (3) for a review). Huddleston (1943) found that the organism could survive for 6-8 months in a foetus in the shade and 3-4 months in faeces (cited by Ministry of Agriculture and Fisheries (4)). Growth of *Brucella abortus* was completely inhibited by 4% sodium chloride (Lerche (1960) cited by Mitscherlich and Marth (3)). *Brucella canis* survived desiccation for 0.5-9 days in various media on aluminium sheets, building stone, stone and cement plaster (Weber (1976) cited by Mitscherlich and Marth (3)). It is concluded that *Brucella spp.* might survive several weeks on salted or dried hides and in damp manure contaminating hides for several months.

Brucella abortus, Brucella melitensis and *Brucella suis* are zoonotic organisms that cause serious disease in humans.

Conclusion: Since exotic *Brucella* spp. could be found in skin or in manure, milk or vaginal discharge contaminating skins, and may be able to survive on dried or salted hides for several weeks, or even months, they are considered to be potential hazards in this risk analysis.

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4.33 BOVINE GENITAL CAMPYLOBACTERIOSIS

Bovine genital campylobacteriosis is a venereal disease characterised by infertility, early embryonic death and abortion, caused by the bacterium *Campylobacter fetus* subsp. *venerealis.* *Campylobacter fetus* subsp. *fetus* may be responsible for sporadic abortions in cattle but is more commonly found in the intestinal tract. It is a more common cause of abortion in sheep and is endemic in New Zealand.

Campylobacter fetus subsp. *venerealis* is carried on the prepuce of clinically normal carrier bulls. Bulls older than 3 years usually remain permanently infected (1). Cows and heifers carry the infection in their genital tract and may exhibit infertility or may abort. However, in subsequent years their fertility is not generally affected (2).

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

Conclusion: Since *Campylobacter fetus* subsp. *venerealis* is a venereal disease it is not considered to be a potential hazard in the commodity. In addition, *Campylobacter* spp. are endemic and, as association with this pathway would not result in a significant increase in exposure, are therefore not considered to be potential hazards in this risk analysis.

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4.34 BOVINE TUBERCULOSIS

Bovine tuberculosis is caused by the bacterium *Mycobacterium bovis*. 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).

Transmission between animals is mainly by droplet infection (2). *Mycobacterium bovis* is rapidly inactivated by ultra violet light (3) and survives on pasture for less than a week and, in possum dens, for 14 days but no longer than 28 days (4).

Conclusion: *Mycobacterium bovis* is usually transmitted by droplet infection and in most situations survives for comparatively short periods outside the body. Therefore it is not considered to be a potential hazard in the commodity.

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4.35 BOVINE CYSTICERCOSIS

Bovine cysticercosis is caused by the larval stages of the human tapeworm *Taenia saginata*. Cattle are the intermediate host for this parasite, and *Cysticercus bovis* is the name given to the pea-sized cysts in striated muscles of cattle; these cysts are detectable only by careful post-mortem inspection (1). Infestation of humans, the primary hosts of the tapeworm, arises from eating undercooked beef that is infested with the tapeworm cysts.

Even if there were cysts in muscle attached to skin, the life cycle would not be completed since the skin and attached muscle would not be eaten by humans.

Conclusion: The likelihood of humans eating tapeworm cysts attached to imported hides and skins is considered to be negligible. Therefore this pathogen could not complete its life-cycle and is not considered to be a potential hazard in the commodity.

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4.36 DERMATOPHILOSIS

Dermatophilosis is an exudative, pustular dermatitis, caused by the bacterium *Dermatophilus congolensis*. It affects many species of domestic animals. It survives well in the environment and has been shown to occur in soil collected during the dry season. The infective form of the organism is the motile zoospore, which is released when infected skin becomes wet. The life span of the motile zoospore is only a few hours, but dried spores can survive for long periods (1).

Dermatophilus congolensis is endemic and widespread in New Zealand, and not under any form of regulatory control.

Conclusion: Since *Dermatophilus congolensis* is endemic and widespread in New Zealand it is not considered to be a potential hazard in the commodity.

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4.37 ENZOOTIC BOVINE LEUKOSIS

Enzootic bovine leukosis (EBL) is a disease of adult cattle, and occasionally sheep, caused by the bovine leukemia virus, which belongs to the genus *Deltaretrovirus* in the family *Retroviridae* (1).

Infection with the EBL virus in cattle is lifelong, giving rise to a persistent antibody response. Most infections are subclinical. Up to 30% of serologically positive cattle develop a persistent lymphocytosis and 30% of these develop tumours. High antibody titres develop in sheep, but persistent lymphocytosis has not been observed in this species (2).

A dairy industry-led eradication campaign has resulted in the virtual eradication of the disease from dairy cattle in New Zealand. The herd prevalence has fallen from 7.5% in 1998 to less than 0.02% in 2006 (3). Only one infected dairy cow was detected in 2005, which has been slaughtered. New Zealand has now entered the so-called 'monitoring' phase, in which half of the dairy herds are sampled and checked each year. There is no programme in the beef cattle industry in New Zealand, but testing of beef animals entering dairy herds suggests that the virus is present at a very low level in beef herds (4).

Transmission between animals generally requires the transfer of blood cells, and the most important transmitters are probably humans - iatrogenic transmission at dehorning, blood testing, rectal palpation, and vaccination are the main routes of transmission within herds. Movement of infected animals is the most important route of transmission between herds. Transmission by infected milk and colostrum has also been described (2). Pre and peripartum infections have been reported. 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).

EBL virus is strictly cell-associated (5) and will therefore only survive for a very short period outside the host (6).

Conclusion: The bovine leukemia virus is transmitted only by close contact or iatrogenically and does not survive long outside the host. Therefore it is not considered to be a potential hazard in the commodity.

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4.38 HAEMORRHAGIC SEPTICAEMIA

Haemorrhagic septicaemia (HS) is an acute, highly fatal bacterial septicaemia of cattle and buffaloes caused by *Pasteurella multocida* serotype B or E. The disease occurs almost exclusively in Asia (serotype B) and Africa (serotype E), in countries with a high and seasonal rainfall (1).

Pasteurella multocida is normally maintained as a commensal in the oropharynx of mammals (2), and various stresses (especially the sudden onset of the rainy season, with an associated drop in temperature) appear to be associated with disease outbreaks (1). *Pasteurella multocida* is described as a commensal of the upper respiratory tract or as a primary or secondary pathogen (3). No reports were found of its survival as a free living organism in the environment. Few animals survive an episode of haemorrhagic septicaemia. The likelihood that the organism would be in the skin of healthy animals is therefore remote. There is no evidence to indicate that *Pasteurella* spp. survive outside of their hosts for prolonged periods or that the organism is transmitted by skins or hides.

<u>*Conclusion:*</u> *Pasteurella multocida* is a commensal or opportunistic pathogen that is not known to be transmitted by hides and skins. Therefore it is not considered to be a potential hazard in the commodity.

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4.39 INFECTIOUS BOVINE RHINOTRACHEITIS (IBR/IPV) AND RELATED HERPES VIRUSES

Bovine herpesvirus1 is associated with infectious bovine rhinotracheitis and infectious pustular vulvovaginitis/infectious pustular balanoposthitis (IPV/IPB). Subtypes BHV-1.1 and BHV-1.2 can be identified by restriction endonuclease analysis of DNA (1, 2, 3). Rhinitis and respiratory signs are associated with subtype 1.1, pustular vulvovaginitis and balanoposthitis are associated with subtype 1.2. Strains formerly described as IBRV 1.3 that are associated with encephalitis are now classified as BHV5. Subtype 2 strains can be further classified as BHV-1.2a and BHV-1.2b strains. Some subtype 1.1 and 2a strains are abortifacient as shown by association with clinical cases of abortion and by experimental infection of pregnant heifers (4) . Subtype 2b strains are associated with respiratory and genital infections but not with abortions (4, 5). Under recent reclassification of the herpes viruses BHV3 is considered to be redundant and BHV4 is a bovine orphan virus.

Type	**Syndrome**			
	IBR	**IPV/IPB**	**Abortion**	**Encephalitis**
BHV 1.1	+	-	+	-
BHV 1.2a	+	+	+	-
BHV 1.2b	+	+	-	-
BHV5	-	-	-	+

BHV-1 is distributed worldwide although Norway, Sweden, Finland, Denmark, Austria, Switzerland and the region of Bolzano in Italy are IBR-free and national eradication programs have been implemented in Germany, The Netherlands, Belgium, Hungary, the Czech Republic and Slovakia.

A mild respiratory strain of BHV-1.2b is widespread and prevalent in New Zealand, especially in dairy cattle, but clinical and serological evidence suggests that the BHV-1 subtypes which cause severe respiratory disease and abortion are not present in New Zealand (6, 7). Transmission occurs by the respiratory route in cattle in close contact with each other or by the genital route (1). The virus can persist in the environment for several days under appropriate temperature and humidity conditions (1).

Bovine herpesviruses have a tendency to become latent following primary infection, and stress may cause reactivation and shedding from the respiratory or genital tracts (1, 8). The sites of latency are ganglia and peripheral nerve fibres (1).

Conclusion: BHV-1 is not considered to be a potential hazard in the commodity as it is transmitted by the respiratory or venereal route and is not described as being transmitted on fomites. There is no evidence that it is transmitted on hides or skins. In addition it will only survive a few days in the environment.

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4.40 THEILERIOSIS

Theileriosis is caused by tick-transmitted protozoa of the genus *Theileria* (1). East Coast Fever is a severe non-contagious disease of cattle caused by *Theileria parva parva*, occurring in eastern and central Africa (2). Related bovine theilerioses caused by other members of the *Theileria parva* complex include corridor disease (*Theileria parva lawrenci*) (3) and Zimbabwe theileriosis (*Theileria parva bovis*) (4). *Theileria mutans* is usually non-pathogenic (5). Mediterranean coast fever or tropical theileriosis is caused by *Theileria annulata* in North Africa, southern Europe and Asia (6). *Theileria orientalis* is a relatively benign infection of cattle and Asian buffalo. The nomenclature of this species is somewhat confused and *Theileria sergenti* and *Theileria buffeli* are synonyms (7).

The distribution of the theilerioses is determined by the distribution of specific tick vectors which are essential for the completion of a complex life-cycle. East coast fever is naturally transmitted only by *Rhipicephalus appendiculatus* (2); this tick is also the main vector for the related bovine theilerioses of the *Theileria parva* complex (3, 4). *Theileria mutans* is transmitted by several species of tick in the genus *Amblyomma* (5). *Theileria annulata* is transmitted by two- and three-host ticks of the genus *Hyalomma* (6).

Theileria orientalis is mainly confined to Southeast Asia, but it also occurs in Australia, where it is transmitted by *Haemaphysalis longicornis* and *Haemaphysalis bancrofti*. *Theileria orientalis* also occurs in the north of New Zealand (8). It was most probably introduced into New Zealand in subclinically infected carrier animals and subsequently was able to spread throughout the northern half of the North Island where *Haemaphysalis longicornis* is endemic (9).

Benign ovine theileriosis in Africa is caused by *Theileria ovis* or *Theileria separata* (*Theileria recondita*). *Theileria lestoquardi* (*Theilei hirci*) causes malignant ovine theileriosis, a disease of sheep similar to *Theileria annulata* infection in cattle, which occurs from North Africa and southern Europe through the Middle East to India. The vectors or probable vectors of the ovine *Theileria* spp. are:

Theileria lestoquardi –	*Hyalomma anaticolicum, Hyalomma* spp.
Theleria seperata –	*Rhipicephalus evertsi evertsi, Rhipcephalus evertsi mimeticus*
Theileria ovis –	probably *Rhipicephalus bursa*

All the Theileria spp. are tick-borne diseases and no other natural methods of transmission are known to occur. They can be transmitted by injection of blood although transmission by this method may be erratic at least for *Theileria parva parva* (2).

<u>*Conclusion:*</u> Since *Theileria* spp. are transmitted only by ticks, they are not considered to be potential hazards in the commodity.

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4.41 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 abortion and infertility especially in dairy cattle (1). With the widespread use of artificial insemination, its significance has declined along with its prevalence in countries where artificial insemination is practiced in dairy cattle. It was diagnosed in a beef herd in 1981 (2), and one case was reported in 2005 (3). It is not under regulatory control.

Conclusion: Because it is a venereally transmitted pathogen, not known to be transmitted by skins and hides, and is endemic, *Tritrichomonas foetus* is not considered to be a potential hazard in the commodity

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4.42 TRYPANOSOMOSIS

Trypanosomosis results from infection with parasitic protozoa of the genus *Trypanosoma* (1). In Africa, where trypanosomosis is of greatest importance, transmission is by blood-sucking flies of the genus *Glossina* (tsetse flies). These flies infest an area of 10 million square kilometres that are frost-free and have an annual rainfall of over 650mm. These limits are determined by climate, often through its effect on vegetation (1).

The trypanosomes which cause tsetse transmitted trypanosomosis in cattle, goats, sheep, pigs, horses, and donkeys include: *Trypanosoma congolense*, *Trypanosoma simiae*, *Trypanosoma vivax*, *Trypanosoma brucei*, and *Trypanosoma suis*. In infections with the more pathogenic forms, there is intermittent fever, anaemia and loss of condition accompanied by parasitaemia, whereas in the less pathogenic forms there can be high parasitaemia in the absence of clinical signs (1).

Trypanosoma evansi, which causes the disease surra in many countries of Asia, Near and Far East, North Africa and South America, is transmitted mechanically by biting flies such as tabanids (1). *Trypanosoma theileri* is a non-pathogenic trypanosome with a world-wide distribution, which is also transmitted by biting flies such as tabanids. Other non-pathogenic trypanosomes are also known. Only *Trypanosoma equiperdum* is transmitted without any insect vector. It causes the disease dourine in horses and is transmitted venereally (1). Surra and dourine are considered separately in this document.

In Africa, wild animals coexist with tsetse flies and trypanosomes without problems but large parts of the continent are effectively closed to cattle due to the presence of tsetse flies. The disease occurs only in tsetse fly infested areas or the marginal areas bordering them. *Bos taurus* breeds of cattle are particularly susceptible (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 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).

Trypanosoma vivax is the only species of tsetse-borne trypanosome that has become permanently established outside Africa. It is present in Central and South America where it is believed to be transmitted by biting flies (2).

<u>*Conclusion:*</u> *Trypanosoma* spp. are not considered to be potential hazards in the commodity as they are all insect transmitted parasites, except for *Trypanosoma equiperdum* which is transmitted venereally.

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4.43 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).

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. A sheep-associated form of the disease is caused by ovine herpes virus-2 (OHV-2) and is the cause of MCF in most other 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 to be 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. Wildebeest calves are thought to be mainly 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 disease in cattle and deer occurs sporadically and is not under regulatory control (5).

Conclusion: MCF viruses are transmitted by contact through infection of the upper respiratory tract. There is no evidence that the disease is transmitted on fomites including skins and hides. Wildebeest malignant catarrhal fever is not transmitted by cattle and could not establish in New Zealand as there are no wildebeest. Wildebeest skins are beyond the scope of this risk analysis. The sheep associated virus is endemic in New Zealand. Therefore, these viruses are not considered to be potential hazards in the commodity.

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4.44 BOVINE SPONGIFORM ENCEPHALOPATHY

BSE is a fatal neurological disease of adult cattle, generally believed to be caused by an unconventional infectious agent. The disease was first recognised in the UK in 1986, and retrospective investigation indicates that the first date of clinical infection was likely to have been about April 1985 (1). The majority of BSE-infected animals are dairy cattle which were exposed to infectivity in calfhood. There is very strong evidence that infected feed is the major factor of transmission of the disease, and that the disease was spread directly as a result of the practice of feeding concentrate feeds containing ruminant-derived meat and bone meal. Although maternal transmission may occur, its occurrence is very low and it will not significantly influence the incidence of BSE (2). Therefore, the only route of transmission of any significance is thought to be the oral route.

BSE has been transmitted from natural cases by the feeding (or by injection) of brain, cervical and terminal spinal cord, retina and distal ileum. In experimental infections, infectivity has been detected in the distal ileum of challenged calves, where it is presumably associated with the lympho-reticular tissues of the Peyer's patches. No infectivity has been found in any other tissue, including skin (1, 2).

The Code states that when authorising import or transit of skins and hides Veterinary Administrations should not require any BSE related conditions (3).

Conclusion: Since the BSE agent does not occur in skin, and is transmitted orally it is not considered to be a potential hazard in the commodity.

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4.45 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 more appropriate to consider these as "small ruminant lentiviruses" (SRLVs) rather than as separate viruses (2). There is a close serological relationship between CAE virus and maedi-visna virus, and arthritis and mastitis may also occur in maedi-visna of sheep (3, 4). However, in New Zealand CAE has remained confined to goats and infection of sheep with either virus has not been reported.

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 (4).

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 (5).

Three biological properties of SRLVs lend themselves to persistent infection. They can sequester themselves in host cells by integrating their pro-viral DNA into host cell DNA, they replicate preferentially in macrophages, and they do not usually induce virus neutralising antibodies (4).

Transmission of SRLVs is mainly via colostrum or milk (3). Transmission may also occur between adult sheep under conditions of close contact, presumably by the transfer of bodily secretions (6).

Small ruminant lentiviruses are cell-associated and therefore quite fragile; they survive for only a short period outside the host (4).

Conclusion: Small ruminant lentiviruses are fragile cell associated viruses and are therefore not considered to be potential hazards in the commodity. In addition, CAE is endemic in New Zealand.

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4.46 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 - *Mycoplasma capricolum* subsp. *capricolum*, *Mycoplasma mycoides* subsp. *mycoides* (Large Colony) (LC), and *Mycoplasma putrefaciens* can produce a similar clinical picture, particularly in goats (1). *Mycoplasma 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 *Mycoplasma arginini* and *Mycoplasma mycoides* subsp. *mycoides* LC are seen in New Zealand (3, 4, 5). *Mycoplasma ovipneumoniae* and *Acholeplasma laidlawii* also occur in New Zealand (3, 4).

Mycoplasma infections tend to be chronic in both flocks and in individuals, and prolonged subclinical shedding of mycoplasmas may occur, especially in the milk. Transmission of contagious agalactia is mainly by ingestion or by direct entry into the teat (6).

Mycoplasma spp. lack a cell wall and are frail organisms that do not survive long in the environment. A review of 6 articles in which *Mycoplasma agalactia* was submitted to desiccation in a variety of different conditions showed that the organism survived between 0 and 9 days and that survival was considerably shorter when also exposed to light (7).

<u>*Conclusion:*</u> *Mycoplasma agalactia* is a fragile organism that does not survive for long in the environment, would not survive on dried or salted hides, and is not considered to be a potential hazard in the commodity.

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4.47 CONTAGIOUS CAPRINE PLEUROPNEUMONIA

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

CCPP 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). The organism is very fragile and does not survive long in the external environment (5).

Conclusion: Since the organism does not survive in the external environment it is not considered to be a potential hazard in the commodity.

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4.48 ENZOOTIC ABORTION OF EWES

Enzootic abortion of ewes (ovine chlamydiosis) is caused by an organism previously considered to be a sheep-specific strain of *Chlamydia psittacii* (1) which has been recently renamed as *Chlamydophila abortus* (2). It is one of the most important causes of ovine abortion in most sheep-rearing countries of the world (3), although it is not present in New Zealand (1). The environment becomes contaminated with *Chlamydophila* by both diseased and carrier animals shedding the organism in faeces and discharges from the genital and respiratory tracts. The faecal-oral route is probably the most common means of transmission (4). The organism in the body occurs in two forms, the reticulate bodies and the condensed form known as elementary bodies. The elementary body form of the organism is adapted to survival in the environment (4).

Ovine chlamydiosis is a zoonosis; pregnant women working with sheep have become infected and aborted as a result (3).

Conclusion: Ovine chlamydiosis is resistant to the environment and faeces and genital tract discharges could contaminate hides and skins. Therefore the organism is considered to be a potential hazard in the commodity.

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4.49 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 a virus belonging to the genus *Betaretrovirus* in the family *Retroviridae* (1). Jaagsiekte occurs worldwide, but not in Australia or New Zealand (2).

Transmission is by droplet infection, and outbreaks occur when infected sheep are introduced into clean flocks. The disease has a protracted course but is invariably fatal (3).

Retroviruses are cell-associated and therefore quite fragile; they survive for only a short period outside the host (4).

Conclusion: Since the organism is fragile and does not survive in the environment it is not considered to be a potential hazard in the commodity.

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4.50 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 (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 trans-ovarial 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 antibodies 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 micro-climatic changes favourable for an extension in the range of *Rhipicephalus 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 have lifelong immunity (2).

The virus remains viable for only 1.5 hours at 37^0C (2).

<u>*Conclusion:*</u> Nairobi sheep disease virus is only transmitted by ticks and does not survive in the environment. Therefore it is not considered to be a potential hazard in the commodity.

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4.51 SALMONELLOSIS AND OTHER ENTEROBACTERIAL INFECTIONS

The Enterobacteriaceae include 28 genera and more than 80 well-defined species. They include a variety of pathogens and many non-pathogenic organisms. In this risk analysis *Salmonella* will be regarded as the typical representatives of the group. It is assumed that all members of the group will have broadly similar characteristics with regard to survival on skins and hides. Because of its availability some data relating to *Escherichia coli* will be used. *Escherichia coli* are normal inhabitants of the gut of all animals, but some strains (with genetic components encoding for virulence factors) are important pathogens.

The *Salmonella* genus contains about 2,500 serotypes. Important serotypes include *Salmonella* Abortus ovis and *Salmonella* Dublin. *Salmonella* Abortus ovis is one of the salmonellas incriminated as a cause of abortion in sheep. It is endemic in parts of Europe, and abortions due to *Salmonella* Dublin and *Salmonella* Typhimurium occur endemically in many countries (1). *Salmonella* Dublin is a major cause of enteric salmonellosis in cattle and is also a pathogen of sheep.

Outbreaks of ovine abortion caused by *Salmonella* Brandenburg (2) have occurred periodically in New Zealand since the winter of 1997, and cases of *Salmonella* Typhimurium abortion occur from time to time, but the definitive phage type DT 104 has only occurred rarely in humans (3, 4) and once in dogs (5). Many serotypes of *Salmonella* have been identified in New Zealand, but equally a large number have not been described. Although it is most important to exclude *Salmonella* Dublin, *Salmonella* Abortus ovis and *Salmonella* Typhimurium DT 104 it would be desirable to exclude all exotic *Salmonellas*.

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 (6).

Mitscherlich and Marth (1984) have reviewed the survival of *Salmonellas* in the environment (7). The organism may survive in the environment for many months, especially in faeces, and is resistant to desiccation and salting (7).

<u>*Conclusion:*</u> Exotic *Salmonella* spp. and other Enterobacteriaceae are considered to be potential hazards because they can survive for long periods in the environment and infected faeces could contaminate hides and skins.

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4.52 SCRAPIE

Scrapie is a transmissible spongiform encephalopathy of sheep and goats. It has been recognised in Great Britain and Western Europe for around 250 years, predominantly in sheep. Although it is generally accepted that scrapie is an infectious, contagious disease, the means of natural transmission are not well understood (1). There is a genetic influence associated with susceptibility that also influences the length of the 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, is generally believed to be the scrapie agent (1). The course of the disease is progressive, with death usually occurring 1-6 months after the initial development of clinical signs (1, 3).

The most likely route of infection in natural scrapie is the oral route, although other routes which have been shown to be effective experimentally are scarified skin and the conjunctiva (1). Mechanisms for horizontal transmission remain the subject of speculation. Many studies have shown that offspring of infected dams have a significantly increased risk of developing scrapie (2), and this has given rise to the use of the term "maternal transmission". However, precisely how and when transmission occurs (*in utero* and/or *post partum*) remains unknown (1). Very few studies have addressed this question, and the results are inconclusive (2).

Studies of the distribution of the scrapie agent in tissues and organs of sheep clinically affected with naturally acquired scrapie have indicated that the agent is confined to tissues of the central nervous system, reproductive tract, and reticulo-endothelial system (2, 4). Tissues with no detectable infectivity are heart, kidney, mammary gland, salivary glands, seminal vesicle, skeletal muscle, testis, and thyroid (5, 6). In addition, infectivity has not been detected in faeces, urine, milk, colostrum, clotted blood, semen, or saliva of infected sheep or goats (2).

Infectivity has been found in the placentae of ewes with clinical scrapie (7, 8) and also of clinically normal ewes with scrapie-consistent microscopic lesions in the brain and/or detectable PrP^{Sc} in the brain (9). So far, foetal membranes and placenta are the only tissues ordinarily associated with living sheep and naturally shed to the environment that have yielded infectious agent, which suggests that placenta may play a significant role in the spread of the disease (1, 2).

How soon scrapie infectivity begins to accumulate in the placenta is not known, but two ewes which shed infected placentas did not die of scrapie until more than 470 days later, suggesting that the agent may begin to accumulate in the placenta quite early after infection of the animal. Moreover, infection of a placenta in a given pregnancy may not be associated with infection of the placenta in subsequent pregnancies (9).

It is unclear how these findings are related to natural transmission. It has been suggested for some time that so-called "cannibalistic" behaviour by sheep at lambing (when sheep eat or nibble the placenta of other sheep) could be an important route of transmission (7). However, the importance of indirect transmission through

environmental contamination is difficult to assess as it is not known whether scrapie infectivity persists in the environment. It is known, however, that the scrapie agent is highly resistant to heating, disinfectants and UV light (10), and its survival in soil for 3 years has been demonstrated experimentally (2). Moreover, as infectivity has been found in decomposing animals, it has been suggested that sheep could die and contaminate the area where they have decayed, or that lambing areas could become contaminated as a result of the agent being present in placentae of infected animals (9). However, few attempts have been made to isolate the agent from potentially contaminated environmental sources, and all of them have been negative (2).

PrP^{c} is a normal cellular protein, and the abnormal isoform of that protein, PrP^{Sc}, is closely involved in the disease process. The nature of the ovine placenta together with what is known about the scrapie agent suggests that infectivity is unlikely to be associated either with the foetal part of the placenta or with the foetal fluids, and it is concluded that placental infectivity is most likely associated with maternal placental tissues.

Titres of the scrapie agent detected in tissues prior to the development of clinical signs are generally low, but there appears to be a sudden rise of titre in several tissues around the time of development of clinical signs (11). It follows that the placenta might contaminate a small area of the perineum of sheep at lambing, but this would probably occur only in sheep which were either showing clinical signs of scrapie at the time of lambing or were in the late preclinical stage of scrapie at the time of lambing, and such contamination would not be of significance in terms of transmission potential. The likelihood that hides or skins of sheep or goats that were not showing clinical signs of scrapie and were not slaughtered at lambing time or shortly thereafter, would be contaminated with the scrapie agent is considered to be low. Skin is not a tissue in which the scrapie agent has been detected and there is nothing in the literature that suggests that scrapie has been transmitted in hides or skins.

The Code states that regardless of the scrapie status of the exporting country Veterinary Administrations should authorise without exception transit through their territory of hides and skins originating from sheep and goats (12).

Conclusion: Since scrapie agent is confined to nervous tissue, reproductive tract and lympho-reticular tissues and the OIE recommends free trade in hides and skins from scrapie infected countries, this agent is not considered to be a potential hazard in the commodity.

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4.53 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).

'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 (3). There now seems to be a widespread geographical distribution of these diseases in sheep (4). 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 (3).

Active and passive surveillance of sheep has demonstrated that ovine lentiviruses are not present in New Zealand (5).

Three biological properties of SRLVs lend themselves to persistent infection. They can sequester themselves in host cells by integrating their pro-viral DNA into host cell DNA, they replicate preferentially in macrophages, and they do not usually induce virus neutralising antibodies (6).

Transmission of SRLVs is mainly via colostrum or milk (7). Transmission may also occur between adult sheep under conditions of close contact, presumably by the transfer of bodily secretions (3).

Small ruminant lentiviruses are cell-associated and therefore quite fragile; they survive for only a short period outside the host (6).

Conclusion: Since small ruminant lentiviruses are not associated with skin, are fragile, and do not survive long outside the host, they are not considered to be potential hazards in the commodity.

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4.54 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 in the clitoral sinuses and vagina 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). It can also be transmitted indirectly on equipment for washing and bandaging mares. The organism is fragile and does not survive for long in the environment and was shown to survive for only 40 minutes when dried on metal surfaces (2).

<u>*Conclusion:*</u> *Taylorella equigenitalis* is generally transmitted venereally and does not survive long in the environment and is therefore not considered to be a potential hazard in the commodity.

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4.55 DOURINE

Dourine is a chronic infectious disease of horses, mules and donkeys characterised by oedema of the external genitalia and ventral abdomen. It is caused by the protozoan parasite *Trypanosoma equiperdum*, which differs from other trypanosomes in that it is transmitted venereally rather than through an arthropod vector (1).

Conclusion: *Trypanosoma equiperdum* is transmitted venereally and therefore could not be transmitted by hides and skins and is not considered to be a potential hazard in the commodity.

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4.56 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 thermally dimorphic saprophytic soil fungus, *Histoplasma capsulatum* var. *farciminosum* (1, 2). 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 (3).

Transmission is by contamination of traumatised skin (wounds, 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 (4).

The disease is endemic in parts of Africa (especially North Africa), the Middle East, and Asia. However, it is now rarely reported. Historically the disease was more common when large numbers of horses were stabled together for cavalry or other transportation needs (1, 2).

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. The distribution of the fungus is generally restricted to Africa and Asia but large outbreaks occurred in horses during the First World War. The lack of apparent establishment of this saprophytic soil fungus in New Zealand may be a result of the absence of a suitable habitat including soil type and climatic conditions.

It should be noted that *Histoplasma capsulatum* var. *farciminosum* is distinct from *H. capsulatum* var. *capsulatum*. Inhalation of *H. capsulatum* var. *capsulatum* spores in dust generally associated with bird or bat droppings is associated with histoplasmosis in humans (2). Whilst exudates from equine *H. capsulatum* var. *farciminosum* infections have been used to experimentally infect rabbits, mice, and guinea pigs (2), no reports have been located which confirmed human infection with this organism.

<u>*Conclusion:*</u> Since it is a saprophytic soil fungus that does not appear to have established in New Zealand despite ample opportunity for this to occur, *Histoplasma capsulatum* var. *farciminosum* is not considered to be a potential hazard in the commodity.

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4.57 EQUINE ENCEPHALITIDES

Eastern, Western and Venezuelan equine encephalomyelitis (EEE, WEE and VEE respectively) are diseases caused by members of the genus *Alphavirus* in the family *Togaviridae* (1). The diseases are confined to the Americas, where they are mainly seen in horses. Another closely related but distinct virus, Highlands J virus, also occurs in the USA. The viruses primarily cause disease in horses but also occasionally cause serious human disease, and EEE and WEE viruses have caused outbreaks of disease in poultry and various species of farmed birds (2). In the past the viruses have caused major epidemics in horses such as the 1947 outbreak of EEE in which 14,000 horses and 12,000 mules died in Louisiana and Texas, and the 1969-71 epidemic of VEE in northern South America, Central America, Mexico, and Texas in which hundreds of thousands of horses died. Since the introduction of inactivated vaccines, outbreaks of this magnitude have not been seen (2).

The epidemiology of EEE, WEE and VEE viruses is complex, involving normal cycles between wild animals and mosquitoes in specific environments with spill over into man and horses only under certain conditions (2, 3). EEE and WEE are maintained in birds, primarily passerines, and carried by mosquitoes. The cycle is maintined by ornithophilic mosquitoes but periodically, when high levels of infection and high concentrations of mosquitoes are involved, other mosquitoes that feed on a broader range of hosts transmit the disease to horses and man. Other maintenance cycles such as a jackrabbit/*Aedes* cycle for EEE in California have also been described. VEE is believed to be maintained in a cycle involving mosquitoes and forest rodents. In addition, at least 100 species of birds have also been shown to be infected with VEE virus or to have antibody to the virus and at least 41 species of mosquitoes from 11 genera have been shown to be naturally infected (2). Knowledge of the natural maintenance and transmission cycles of all the encephalitis viruses is still incomplete, but it is clear that the viruses are all mosquito-borne.

EEE and WEE occur sporadically in horses and humans in the USA 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 (2). Horses infected with VEE virus may have high viraemias which are sufficient to infect mosquitoes, but the duration of viraemia is no longer than five days (2).

Togaviruses are sensitive to drying, heat, and acidic conditions (pH 3) (3).

Conclusion: The encephalitides viruses do not occur outside of the Americas and are transmitted by mosquitoes, therefore equine encephalitides viruses are not considered to be potential hazards in the commodity.

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4.58 EQUINE INFECTIOUS ANAEMIA

Equine infectious anaemia (EIA), colloquially known as "swamp fever", is a viral disease of horses and other Equidae, caused by the sole equine virus 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). Subclinically infected animals and animals which recover from clinical disease remain carriers (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 transmitted only by the transfer of contaminated blood. It 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). Although horse flies are not present in New Zealand, the stable fly *Stomoxys calcitrans,* is present and widespread (6).

EIA virus, like other lentiviruses, is cell-associated and therefore quite fragile; it survives for only a short period outside the host (2).

Conclusion: Equine infectious anaemia virus is a fragile, cell-associated virus that will not survive in the external environment and is only transmitted by insects. Therefore, it is not considered to be a potential hazard in the commodity.

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4.59 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 *Influenza virus 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 without maternal antibody (2).

The virus can be isolated from naso-pharyngeal secretions of infected animals for up to 10 days (2). The frequent and harsh cough which is seen in infected horses 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 (2).

Many factors influence the epidemiology of the disease, but most important in recent years has been the transport of horses by air (2).

The virus does not survive for long in the environment. Influenza viruses survived for 1-2 days on hard porous surfaces and 8-12 hours on cloth, paper and tissues (3, 4).

Conclusion: Equine influenza is a contagious disease transmitted by aerosols between horses. Equine influenza viruses do not survive for long in the environment and are therefore not considered to be potential hazards in the commodity.

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4.60 EQUINE PIROPLASMOSIS

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 *Babesia caballi*. It has recently been suggested that *Babesia equi* is in fact a *Theileria* (1, 2), but the name *Babesia equi* will be retained in this risk analysis. The disease can be characterised by fever, progressive anaemia, jaundice, redwater and abortion (1).

As is the case with babesiosis in other species, the world distribution of equine babesiosis depends on the distribution of its specific tick vectors. Twelve species of ixodid ticks in the genera *Dermacentor*, *Rhipicephalus* and *Hyalomma* have been identified as trans-stadial vectors of *Babesia equi* and *Babesia caballi*, and eight of those are also able to transmit *Babesia caballi* trans-ovarially (2). None of these ticks are present in New Zealand (3). There is no evidence of mechanical transmission of equine babesiosis by biting insects (1).

Babesia 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 Andalusian horses 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* (4). Quarantine of infected properties was lifted after 6 months, by which time it was established that there was no natural transmission. It is now considered that *Babesia equi* is almost certainly exotic to Australia (5).

<u>*Conclusion:*</u> *Babesia equi* and *Babesia caballi* are tick borne infections and are not considered to be potential hazards in the commodity.

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4.61 EQUINE VIRAL RHINOPNEUMONITIS AND OTHER EQUID HERPES VIRUSES

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, equid herpesvirus 1 and equid herpesvirus 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).

The natural route of infection of EHV1 and EHV4 is via the upper respiratory tract (6). The virus is maintained in latently infected horses.

EHV2 and EHV5 also infect the respiratory tracts of horses but generally cause mild or subclinical infections (7). EHV3 is a venereally transmitted virus that causes superficial and self-limiting lesions on the skin of the external genitalia of mares and stallions (8). These viruses occur in New Zealand.

Conclusion: EHV1, EHV2, EHV3, EHV4, and EHV5 occur in New Zealand but the abortion and meningoencephalitis syndrome associated with EHV1 and EHV4 have not been seen. The viruses are transmitted by the respiratory tract or venereally. Therefore equine herpes viruses are not considered to be potential hazards in the commodity.

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4.62 GLANDERS

Glanders is a contagious and fatal disease of horses, donkeys, and mules, and is caused by infection with the bacterium *Burkholderia mallei* (previously named *Pfeifferella, Loefflerella, Malleomyces, Pseudomonas* or *Actinobacillus mallei*) (1).

The disease causes nodules and ulcers in the upper respiratory tract. The skin form called "farcy" is characterised by enlarged lymphatics beaded with nodular abscesses and ulcers which discharge pus (1). It is also an important zoonosis - 95% of untreated human cases are fatal. Glanders is now a rare disease but has been reported in recent times in Lithuania, Mongolia, Ethiopia, Eritrea, Sudan and Brazil (2). Acute glanders is more common in donkeys and mules; and is characterised by high fever, respiratory signs, and death in a few days. In horses, glanders generally runs a chronic course; affected animals may survive for years (3).

Work cited by Mitscherlich and Marth indicates that the organism can survive for 14-40 days in naturally infected putrid material and up to 90 days when desiccated on silk threads (3).

Conclusion: Since the organism may be present in the skin of animals with farcy, and may survive in the environment, *Burkholderia mallei* is considered to be a potential hazard in the commodity.

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4.63 HORSE POX

Poxviruses are unimportant as causes of viral disease in equines. Currently there is only one such disease known, Uasin Gishu disease, an infection described in horses in Kenya. Uasin Gishu is caused by a virus tentatively classified as a member of the genus *Orthopoxvirus* in the family *Poxviridae* (1). Infected horses show typical pox lesions which may appear and disappear intermittently for years (2).

In Europe, before vaccination campaigns against smallpox (vaccinia) were discontinued, horses were quite frequently accidentally infected with vaccinia virus from recently vaccinated humans. Horse pox and vaccinia may be caused by the same virus. The disease in horses took two forms - pox lesions in the mouth and on the lips, or "grease" lesions on the lower legs. Since smallpox vaccination has stopped, the condition in horses has become rare (3). All forms of horse pox are rare and of minimal importance.

There is no chapter on horse pox currently available in the *Code*.

Conclusion: Horse pox is not considered to be a potential hazard in the commodity as it causes a rare and unimportant disease that may no longer exist.

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4.64 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, and abortion (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 naso-pharyngeal secretions from horses with acute respiratory disease. 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). Infection usually causes mild or sub-clinical disease. Fever and a wide variety of other signs have been described in clinical cases.

Infected stallions that are acutely infected may be sub-fertile for up to 6-8 weeks but recover with no loss of fertility. They may shed the virus in semen continuously for years and perhaps for life. Venereal transmission occurs when persistently infected stallions are mated to mares by natural or artificial insemination (2). Mares do not become persistently infected; the virus has been isolated from the reproductive tract only up to a month after infection (2).

EVA virus appears to be distributed worldwide (2, 3). The infection occurs in New Zealand and is controlled by a scheme previously administered by MAF (4) and now by the Equine Health Association. Currently there are two known shedder stallions in the country (5). The virus remains viable for only 2-3 days at 37°C (2).

Conclusion: There is nothing to indicate that the virus would be present in the skin of infected horses and the virus does not survive long in the environment. Therefore, equine viral arteritis is not considered to be a potential hazard in the commodity.

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4.65 JAPANESE ENCEPHALITIS

Japanese encephalitis (JE) virus is a member of the *Flavivirus* genus in the family *Flaviviridae* (1). The primary hosts are ardeid birds (herons and egrets) (2). It is spread by mosquitoes and is endemic throughout much of Asia, particularly southeast Asia and Japan (3). Infection of humans and horses may cause severe and often fatal encephalitis. Pigs are the primary amplifying host (2). Although clinical signs are not common in pigs in endemic areas, in immunologically naive sows there can be significant rates of abortion (4). Inapparent infections also occur in goats, sheep, cattle, and dogs, and they have been reported in cats, rodents, bats, snakes, and frogs (5).

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 (4).

Although 28 mosquito species have exhibited vector competence for JE virus in field and laboratory studies, only a few species found in endemic areas occur in sufficient abundance, have long enough flight ranges, exhibit sufficient longevity, and have the breadth of host feeding preferences to become natural vectors (4). In endemic areas of Asia the primary vectors belong to the *Culex vishnui* group, mainly *Culex tritaeniorhynchus*. However, other mosquitoes are important vectors locally, including *Culex vishnui*, *Culex fuscocephala*, *Culex gelidus*, and *Culex annulus*. The type of available larval habitat determines which species will predominate - both *Culex tritaeniorhynchus* and *Culex vishnui* breed predominantly in rice paddies and are therefore the most important rural vectors (3). At temperatures below 20^{o}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^{o}C (6).

Two introduced mosquitoes that are established in New Zealand (*Culex quinquefasciatus* and *Aedes notoscriptus*) (7) are competent vectors of Japanese encephalitis (8).

In horses, clinical signs appear 8-10 days post infection, by which time viraemia may have passed (2). Viraemia in horses appears 1-4 days after infection and lasts for 2-6 days (9), ending with the development of antibodies (3). The level of viraemia in horses is very low in comparison to pigs and birds (9), and horses are considered dead-end hosts for the virus as they do not develop viraemias of sufficient titre to infect mosquitoes (4, 10).

Conclusion: Japanese encephalitis virus is not considered to be a potential hazard in the commodity as it is an insect-borne virus that will not be transmitted in hides and skins.

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4.66 SURRA (*TRYPANOSOMA 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, *Trypanosoma 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). Surra is spread to new areas by the movement of infected animals (1). It is not transmitted directly between animals and since biting flies do not feed on skins and hides it could not be transmitted from them.

Conclusion: *Trypanosoma evansi* is transmitted mechanically by biting insects and will not survive on dried and salted skins, therefore it is not considered to be a potential hazard in the commodity.

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4.67 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 *Bordatella bronchiseptica* alone causes a less severe form of disease in which non-progressive turbinate bone atrophy occurs, without significant snout changes (1).

Surveys of chopper pigs in New Zealand have shown that *Pasteurella multocida* and *Bordetella bronchiseptica* are widespread, and that mild turbinate atrophy and nasal septum deviation does occur. However, toxigenic strains of *Pasteurella multocida* serotype D have not been found, and the severe form of the disease has never been reported in New Zealand (2).

Pasteurella multocida transmission is by the respiratory route. *Pasteurella multocida* is a commensal organism in the naso-pharynx or acts as a primary or secondary pathogen in various animal species (3). It has not been described as a free living organism in the environment. There is nothing in the literature to suggest that it is found in the skin or transmitted by hides or skins.

Conclusion: Since it is transmitted by the respiratory route and there is no evidence that it can be transmitted by hides or skins, *Pasteurella multocida* is not considered to be a potential hazard in the commodity.

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4.68 CYSTICERCOSIS (*CYSTICERCUS CELLULOSAE*)

The cysts of porcine cysticercosis are 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 cyst occur in New Zealand (2).

Even if there were cysts in muscle attached to skin the life cycle would not be completed unless a person ate the attached muscle. The likelihood of this occurring is considered to be negligible.

Conclusion: The likelihood of the lifecycle being completed in man is considered to be negligible. Therefore *Cysticercus cellulosae* is not considered to be a potential hazard in the commodity.

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4.69 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*, order *Nidovirales* (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 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).

TGE virus is highly photosensitive and does not survive well at room temperature. At 37°C all infectivity is lost in 4 days (3).

Conclusion: TGE virus is not considered to be a potential hazard in the commodity as it does not survive for long in the environment.

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4.70 TRICHINELLOSIS

Trichinella spiralis is a nematode parasite of many animals, particularly pigs, and also occurs in man. The adult stage lives a few weeks in the small intestine of a large number of mammal species. After mating in the intestine the male worms die and the females penetrate the gut wall and find their way into lymph spaces where they give birth to live larvae which pass from the lymph into the bloodstream and are disseminated throughout the body. Larvae that find their way into muscle tissue develop into cysts that can remain viable for years. Transmission occurs through the eating of meat containing encapsulated infective larval cysts, following which the ingested larvae develop into adult worms in the gut of the host (1). Pigs are infected mainly by feeding scraps of meat particularly in garbage.

The nematode may be found in humans, pigs, rats, bears and many other flesh-eating mammals, even in horses that eat fodder containing dead infected rodents (2). It is mainly important because of its public health significance. Humans that 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). In modern piggeries where pigs are meal-fed infestation is rare.

Seven other species of *Trichinella* are recognised (2) but these are of minor importance as parasites of domestic animals and man, and do not need to be considered separately in this risk analysis.

<u>*Conclusion:*</u> *Trichinella* cysts occur only in muscle. For the life cycle to be completed remnants of muscle attached to skin would have to be eaten by a host animal. The likelihood that *Trichinella* cysts would occur in muscle attached to skins, survive salting or drying, and be eaten by a suitable host is considered to be negligible. Therefore, *Trichinella spiralis* is not considered to be a potential hazard in the commodity.

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4.71 ENTEROVIRUS ENCEPHALOMYELITIS (TESCHEN-TALFEN DISEASE)

Porcine enteroviruses belong to the genus *Enterovirus* in the family *Picornaviridae* (1), and are ubiquitous in pig populations throughout the world. There are 13 serotypes of porcine enteroviruses (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, non-pathogenic variants of the virus circulate in pig populations (2). Several serotypes may produce sporadic outbreaks of encephalomyelitis - at least types 2, 3, 4, 5, 6, 8, 12 and 13 (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 a mild strain (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 in their faeces (4). 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). Enteroviruses are relatively resistant to inactivation, so transmission by fomites is a possibility (5).

Conclusion: Virulent strains of enteroviruses should be excluded from New Zealand. Since enteroviruses are resistant to the external environment, and hides and skins could be contaminated by faeces, porcine enteroviruses are considered to be potential hazards in the commodity.

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4.72 PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME

Porcine reproductive and respiratory syndrome (PRRS) is a disease of pigs caused by a member of the genus *Arterivirus*, in the family *Arteriviridae*, order *Nidovirales* (1).

The disease is characterised by reproductive failure including abortions, stillbirths, the birth of weak piglets 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 (2). New Zealand is free from PRRS virus (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 oro-pharyngeal scrapings at different times following infection (4). Infected aerosols have been considered to be the most likely source of the virus for susceptible pigs (2). Other possibilities include semen transmission, and in certain conditions, very limited airborne spread may be possible (4).

Infected pigs can remain viraemic for 4-6 weeks after detectable antibody has been formed, and can transmit the virus to other pigs (2). Field experience indicates that recovered pigs are not responsible for herd breakdowns (5). 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 sero-negative contacts (6). Therefore it is generally considered that the virus cannot be recovered from infected pigs more than about 3 months after infection (7).

Arteriviruses do not survive well in the environment. A closely related virus, equine viral arteritis virus, survived for only 2-3 days at 37°C (8). The half life of PRRS virus was 20 hours at 21^{o}C (9). The virus is relatively unstable and does not survive well at temperatures above 20°C (10).

Conclusion: PRRS virus does not survive long in the external environment and therefore it is not considered to be a potential hazard in the commodity.

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4.73 ARTHROPOD PARASITES FOUND ON SKIN

A large number of insects and arachnid parasites are found on skin. The most important are:

- *Ticks*: There are at least 690 species of ixodid (hard) ticks and 200 argasid (soft) ticks. Ticks are probably the most important vectors of infectious diseases including viral, protozoal and bacterial diseases and tick toxicoses (1). They also cause production losses due to irritation and loss of blood. Only one species of tick (*Haemaphysalis longicornis*) occurs on livestock in New Zealand and there are no important tick-borne diseases carried by this tick in New Zealand. For this reason it is important not to introduce new species of ticks.

  Ticks are blood sucking parasites and are unlikely to stay attached to skins of dead hosts which do not provide a source of blood although some ticks can survive for long periods while looking for a host. The likelihood that they will remain on skins for significant periods, especially when these are dried or salted and unable to provide a blood meal, is considered to be negligible.

- *Mange Mites*: Mange mites are parasites of skin. They burrow into the superficial layers of the skin causing damage and irritation resulting in typical lesions which are generally distinctive for each species of mange mite. Some of the common mange mites are listed below

  Demodex spp. (sheep, goats, cattle, pigs)
  Psorergates spp. (sheep)
  Sarcoptes spp. (cattle sheep, goats, dogs, man)
  Psoroptes spp. (cattle, sheep, horses)
  Chorioptes spp. (horses, cattle, goats, sheep)

  Mange mites are transmitted by contact between animals. They do not survive for long periods outside host animals. Sarcoptic mange mites do not survive for more than a few days away from their host and, in optimal laboratory conditions, for 3 weeks (2, 3). Psoroptic mites survive about 10 days away from the host and, under optimal conditions, up to 3 weeks (4, 5). *Demodex* mites survive for several days off the host and, in skin kept moist and cool, for 3 weeks (6). Completion of the life cycle of mange mites varies from about 10 days to 3 weeks. One publication suggests that *Sarcoptes scabei* can survive off the host for up to a month when protected by straw, soil, hair etc but that exposure to drying wind destroys them within 48 hours (7). Another worker found that they survived 1-3 weeks at 10-15^{o}C at a relative humidity of 97% but at a relative humidity of 25% and a temperature of 20-25^{o}C they survived for only 2 days (8). No reference could be found concerning the effects of salting and drying but these processes are likely to greatly reduce the survival time of mites.

- *Lice*: Lice occur commonly on most species of animals and cause irritation, resulting in rubbing, scratching, and damage to hides and skins. They are not generally associated with transmission of diseases. Lice that occur commonly on livestock include:

  Bovicola spp.
  Linognathus spp.
  Haematopinus spp.
  Solenopotes spp.

  Adult lice attach their eggs to hairs on the host and these take about 17 days to hatch and the whole life cycle lasts about 43 days (9). They can survive off the host for 3 days on pasture (9). They are spread by contact between animals or on saddle blankets, grooming brushes etc. Adult or nymphal forms will not survive on skins of dead animals, and eggs on skins will hatch within 17 days and only survive a few days after hatching. Lice survived for up to10 days on shearer's moccasins (10). Salting and drying are likely to shorten the survival period. Therefore, the likelihood that lice will survive on skins imported into New Zealand is negligible.

<u>*Conclusion:*</u> Ticks are not likely to remain on hides and skins of dead animals for long. Mites and lice are unlikely to remain viable on skins for longer than a few weeks. Salting and drying are likely to greatly reduce the survival times of parasites on hides and skins. Therefore they are not considered to be potential risks in the commodities.

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4.74 WEEDS AND WEED SEEDS

Weeds and weed seeds could be found attached to the hair or wool on hides. Some plants can replicate asexually and are able to be grown from cuttings, and could grow from pieces of plants introduced on hides or skins. However, large seed heads and pieces of plant material would be easily visible and removed from skins and hides before they are exported but single small seeds would not be obvious.

Seeds are specifically adapted to survive unfavourable environmental conditions and most will at least survive from one growing season to another. Many will survive for several years and germinate when favourable conditions occur. Most seeds are highly resistant to dehydration, particularly those from plants adapted to survival in deserts or hot dry climates, and most seeds retain viability well in dry conditions. Some are specifically adapted to remain viable in water. *Mimosa glomerata* seeds survived 221 years in the herbarium of the Museum National d'Histoire Naturelle in Paris. *Lupinus arcticus* seeds frozen in a lemming's burrow that was dated as 10,000 years old germinated within 48 hours when placed in favourable conditions (1). Some seeds are adapted to environments subjected to periodic fires and survive or are activated by fires. Others are adapted to be dispersed by water including those that are adapted to salt water.

Seeds could also be present in manure contaminating hides. Weed seeds can survive passage through digestive systems and viable seeds can be passed out in faeces (2). This is a recognised method of dispersal of weed seeds. Composting of manure so that high temperatures are reached in the process reduces weed seed numbers but cannot be relied upon to completely eliminate weed seeds (2). However, manure on hides is not likely to have come from compost pits but directly from animals and seeds in that manure are likely to survive dehydration on the hides.

During the process of dry-salting, skins will be manually handled and cleaned (at least wiped free from contaminating material such as pieces of plants, seed heads and large pieces of manure). Dry-salting results in the extraction of water from the skin and formation of a saturated solution of salt. This is likely to damage and destroy many of the less resistant seeds but it cannot be relied upon to destroy all weed seeds. Similarly immersion of skins in very high concentrations of salt in wet-salting is likely to clean the skins of most contaminating material such as manure and soil and destroy less resistant seeds but cannot be relied on to destroy all seeds. Drying of skins without salting and the normal cleaning of skins in this process is also likely to reduce the number of weed seeds on hides. However, no information could be found on the number or types of seeds that survive salting and drying. Intuitively it is assumed that most but not all weed seeds would be removed or destroyed by these processes.

Conclusion: It is concluded that some resistant weed seeds could survive on dried and salted skins and weed seeds are considered to be potential hazards in the commodities.

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4.75 WARBLE FLIES

Hypoderma lineatum and *Hypoderma bovis* occur in the USA and Canada (1). Warble flies have been eradicated from Great Britain (2) and many European countries, and are absent from Australia (3) and New Zealand (4). *Hypoderma lineatum* occurs in countries in the northern hemisphere, mainly in the region of 25-60 degrees north, with a southern limit of the Punjab of India, Northern Mexico and Hawaii (5). *Hypoderma bovis* has a slightly more northerly distribution.

Warble fly infestations of cattle cause serious economic losses due to production losses and damage to hides. In 1986 the cost of warble fly in cattle was estimated at 35 million pounds in Great Britain and $85 million in Italy (6).

The adult flies lay their eggs on the hairs of animals and the larva hatch and penetrate the skin of the animals and then migrate to the oesophageal region or the spinal canal where they remain dormant for the winter. They then migrate to the sub-cutaneous tissues on the back where they develop into warbles and cut a breathing hole in the skin of the animal. Finally, after developing for about 30 days, third-stage instars emerge through the breathing hole in the skin and pupate in the soil. After about 36 days they emerge as adult flies. The whole life cycle takes a full year (2). Warble fly larvae imported from the northern hemisphere would not be synchronised to the seasons of the southern hemisphere and would therefore be unlikely to survive. This is probably why warbles have never established in the southern hemisphere.

<u>*Conclusion*</u>: Hides originating from the southern hemisphere will not contain warbles. Cattle hides from the northern hemisphere could contain mature warbles when they are slaughtered. The warble fly larvae would have to leave the skins during or shortly after skinning and mature in the soil if they are to complete their life cycle. In this case they would no longer be in the skin and would not be imported into New Zealand. If they were retained as warbles in the subcutaneous layers of the skin at the time of slaughter they would be unlikely to survive drying or curing. Therefore warbles are not considered to be a hazard in the commodity.

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4.76 HITCH-HIKER PESTS

Hitch-hiker pests are any organisms that may become attached to, or found on, imported skins and hides. This is a large and ill-defined category and could include a wide range of undesirable organisms. Contamination by manure, soil, secretions or excretions of animals, and any parasites or organisms contained in manure or body secretions can be considered to be hitch hikers. Several cases relating to manure and body excretions have been discussed, and there are separate sections on weed seeds and skin parasites. However, for many other potential hitch hikers there is no greater likelihood of being imported on skins and hides than on other objects such as people's shoes, camping gear, clothes etc. Detailed risk analysis for these random hitch-hikers is not possible since they are near-infinite in number and will appear randomly and erratically. It will be up to border staff to remain vigilant and to impose suitable treatments such as fumigation or forbid importation depending on the particular case. Only hitch hikers that may occur specifically on hides and skins are discussed here.

Hitch hikers that are more likely to occur on hides and skins than on other products are confined to those insects that are attracted to skins and hides, such as beetles that infest and feed on hides and skins. Also hides that have been inadequately salted (or a patch of a skin that has been inadequately salted) may become decomposed and attractive to flies and blowflies. Such skins may become fly-blown and covered with fly larvae, or, at a later stage, the larvae could already have developed into pupae or even adult flies. Other insects such as cockroaches could be attracted to hides and skins as a food source.

Conclusion: Since hitch hiker pests of various kinds could be present on skins and hides they are considered to be potential hazards.

4.77 CHRONIC WASTING DISEASE

Chronic wasting disease (CWD) is a transmissible, progressive, fatal spongiform encephalopathy of deer. It was first recognised clinically in captive deer in 1967, but it was not until 1979 that it was classified as a TSE (1).

CWD is predominantly restricted to north-eastern Colorado and south-eastern Wyoming, where it occurs in both captive and wild deer populations (2, 3). However, it has now been found in at least 13 states in the USA and in Canada. Since 1996, surveillance has detected infected animals on more than 25 elk farms in Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, South Dakota, and Alberta, Canada, and the Republic of Korea (3).

Clinical signs include a progressive loss of body condition, abnormal behaviour, ptyalism, and polydypsia/polyuria. The disease occurs in white-tailed deer, mule deer, black-tailed deer and elk (2).

The origin of CWD in farmed deer has not been determined. The disease shares many epidemiological, clinical and pathological features with scrapie of sheep and goats and with bovine spongiform encephalopathy (BSE). However, in contrast to BSE, horizontal transmission appears to occur quite frequently in the case of CWD, and the incubation period may be as short as 24 months (1). There is no evidence for transmission by feed (3).

Other ruminant species, including wild ruminants and domestic cattle, sheep, and goats, have been housed in wildlife facilities in direct or indirect contact with CWD-affected deer and elk. No cases of CWD or other TSEs have been detected in these other ruminant species (2).

The agent has been demonstrated in brain, pituitary gland, spinal cord, eyes, tonsil, lymphoid tissue, spleen, pancreas, and peripheral nerves, but has not been found in dorsal root ganglia, salivary glands, thymus, liver, kidney, urinary and reproductive tract, cardiac and skeletal muscle, respiratory organs, thyroid and adrenal glands, and skin (2).

Conclusion: Since the agent is not found in skin or muscle, the CWD agent is not considered to be a potential hazard in the commodity.

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5 Risk Assessment

The disease agents that are considered to be potential hazards and that consequently require risk assessments are shown in Table 2.

Table 2. Disease agents considered to be potential hazards in the commodity

Disease Name	Organism
Foot and mouth disease	virus
Swine vesicular disease	virus
Lumpy skin disease and sheep and goat pox (Pox viruses)	virus
African swine fever	virus
Classical swine fever	virus
Bovine viral diarrhoea (type 2)	virus
Highly pathogenic avian influenza	virus
Newcastle disease	virus
Bacillus anthracis (anthrax)	bacteria
Coxiella burnetii (Q fever)	bacteria
Brucella abortus, Brucella melitensis and *Brucella suis* (Brucellosis)	bacteria
Chlamydophila abortus (Enzootic abortion of ewes)	Chlamydia
Enterobacteriaceae including exotic *Salmonella* spp.	bacteria
Burkholderia mallei (glanders)	bacteria
Enterovirus encephalomyelitis	virus
Hitch hikers	various
Weed seeds	various

5.1 GENERAL CONSIDERATIONS

5.1.1 Entry assessment

The likelihood that imported hides and skins could be contaminated with infectious agents depends on their country of origin and the care taken to ensure that they are not derived from diseased animals.

5.1.2 Exposure assessment

Infectious agents in imported skins and hides could be transferred to and infect susceptible animals, or contaminate the environment by the following pathways:

- During unloading at the port of entry or transportation to the tannery where they will be processed, hides and skins, or parts of hides or skins, dust and dirt from skins, or liquid from them could be dropped or released and contaminate soil, water, or fomites in the environment.

- People involved in unloading the commodities could become contaminated by contact with the commodities and later transfer the infectious agents to domestic animals they have contact with. Zoonotic agents could infect workers directly.

- Trucks used for transporting the hides and skins could become contaminated and later the same trucks could be used for transporting animals or animal feed. Animals could then be infected or animal rations could be contaminated resulting in subsequent infection of animals. Zoonotic agents could infect personnel associated with the transport industry.

- Once unloaded at the tannery, organisms could contaminate workers and be transferred to animals outside the tannery premises. Some disease agents could be zoonotic and infect workers.

- Liquid or solid waste, generated during processing of the skins, could be discharged into the environment outside of the tannery premises and lead to contamination of the environment, animals and possibly people.

- Packaging materials used to contain the commodities could be contaminated with disease agents and, if these are disposed of outside the tannery premises, could contaminate the external environment.

The likelihood of transmission of infectious agents from hides and skins to animals, humans, or the environment depends on the methods used for transportation and processing of the commodities and the design of the tannery premises.

The most likely pathway for dispersal of infectious agents from tanneries is through the discharge of liquid wastes or disposal of solid waste. The likelihood of dispersal of disease agents in this manner will be reduced or eliminated if suitable procedures are followed. Safe methods of disposal of wastes such as discharging wastes only to municipal sewers, or to secure soak pits, and the destruction of solid wastes by burning or rendering will reduce or eliminate the risks. Similarly, the use of suitable disinfectants in soaking back water will also be effective. These options are discussed in the risk management section.

5.1.3 Consequence assessment

The consequences of dispersal of infectious agents from imported skins would depend on the agent involved. Individual disease agents are discussed in subsequent sections of this risk analysis.

5.1.4 Risk assessment

Since release of infectious agents is possible and pathways of exposure exist, and the consequences of introducing some agents could be severe, risk is considered to be non-negligible. The risk relating to each agent is considered below.

5.2 FOOT AND MOUTH DISEASE

The hazard identification concluded that FMD virus could potentially be associated with hides and skins of animals that were infected at the time of death.

5.2.1 Release assessment

Agent survival

FMD virus is sensitive to both acid and alkaline conditions. It is most stable at pH 7.4-7.6, but all strains are rapidly inactivated below pH 4 and above pH 11 (1). There is some strain variation at intermediate values, but the major determinant is temperature. The virus will retain infectivity at pH 6.7-9.5 at 4°C or lower, but the pH range narrows as the temperature rises (1).

The effect of temperature on viral infectivity is influenced by the suspending medium; organic matter provides some protection against inactivation (1). Suspensions of FMD virus will remain infective for 8-10 weeks at ambient temperatures of 22°C, and for up to 10 days at 37°C. Above this temperature, inactivation is more rapid (1).

There is a critical relative humidity range of 55-60%, below which virus survival is poor (2). Sunlight has little effect on the virus (3); environmental inactivation is related more to the effects of desiccation and temperature than to sunlight *per se* (1).

FMD virus may survive on inanimate objects for long periods, depending on the temperature and weather conditions (4).

Picornaviruses are insensitive to ether, chloroform, and non-ionic detergents (5). However, sodium hydroxide, formalin (1-2%), or sodium carbonate (4%) will destroy FMD virus (9).

Effect of hides and skins processing

FMD virus may survive in salt-cured hides stored at 1-7^{0}C for up to 352 days, on hair at ambient temperature for 4-6 weeks and in dried hides for 8 days (6).

Therefore, although FMD virus would be unlikely to be present in dried or dry-salted hides and skins, it could remain viable in fresh or wet-salted hides and skins.

The addition of 2% sodium carbonate to salt has been shown to inactivate all virus on heavily contaminated salted ox hides, provided they are stored for 4 weeks (7). Based on this finding, the *Code* recommends, as a safeguard against FMD virus, the treating of hides by salting for at least 28 days in sea salt containing 2% sodium carbonate. In a simple bench trial to assess this treatment, LASRA confirmed that the addition of sodium carbonate raises the pH in the salt to at least pH 9.5 (8), which is lethal to the FMD virus at normal ambient temperatures.

Soaking back would inactivate any FMD virus present if the pH were held above 10. Otherwise this step would have little effect on the virus, in which case these initial processes could generate large volumes of potentially contaminated waste water. Similarly, fleshing following soaking back would have no effect on any virus present, but could potentially generate contaminated solid waste.

The pH levels of liming (pH 12.5-13), and pickling and tanning (pH <3) would result in rapid inactivation of any FMD virus present at that stage.

5.2.2 Exposure assessment

The processes used in tanneries, the design of the premises, and the arrangements for disposals of wastes will determine whether FMD virus could be released from a tannery to the environment.

If the pH at the soaking back stage is at least 10 and solid waste and packaging is disposed of by incineration, there is no potential route of exposure for FMD virus from infected hides and skins to susceptible animal species in New Zealand. However, if these conditions are not met, the following contaminated waste materials could be generated through soaking back and fleshing, and could constitute a potential route of exposure:

- Waste water
- Solid waste

5.2.3 Consequence assessment

Any outbreak of FMD in New Zealand would result in extremely serious economic losses (10). The possible effects of an FMD outbreak have been frequently documented and do not need to be repeated.

5.2.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards can be justified for hides and skins of ruminants and pigs that have not undergone pickling or tanning.

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5.3 SWINE VESICULAR DISEASE

The hazard identification concluded that SVD virus has the potential to be associated with hides and skins of animals that were infected at the time of death.

5.3.1 Release assessment

Agent survival

Enteroviruses are insensitive to ether, chloroform, non-ionic detergents (1), alcohol (70%), 5% Lysol, 1% quarternary ammonium compounds or similar laboratory disinfectants (2). Treatment with 0.3% formaldehyde or free residue chlorine at a level of 0.3 to 0.5 ppm causes rapid inactivation, but the presence of extraneous organic matter protects the virus from inactivation (2). Enteroviruses are thermolabile; they are destroyed rapidly at a temperature of 50°C (2). They are rapidly inactivated by UV light and by drying (2). SVD virus is relatively stable over a pH range of 2-12 (3). In another investigation, variable results were found with a range of disinfectants. Alkali in the form of sodium hydroxide or sodium metasilicate was effective at high concentrations and pH above 12. Acids were generally not effective unless the pH was less than 2. Sodium hypochlorite (0.5%) was effective but its effectiveness was greatly reduced in the presence of pig faeces. Sodium carbonate at 4% and a pH of 10.8 was ineffective (4).

Effect of hides and skins processing

Although the SVD virus would be unlikely to be present in or on dried or dry-salted hides and skins, it could remain viable in wet-salted hides and skins.

Soaking back would have little effect on the virus if present, even if the pH of the soaking back solution were maintained at pH 10. However, these initial processes could generate large volumes of potentially contaminated waste water. Similarly, fleshing following soaking back would have no effect on the virus but could potentially generate contaminated solid waste.

The pH levels of liming (pH 12.5-13), or of pickling and tanning (pH <3) would destroy the SVD virus.

5.3.2 Exposure assessment

The potential routes of exposure for SVD virus from infected hides and skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

Since the virus only infects pigs, transfer from released soaking back water is unlikely to occur unless an open range pig farm is in the immediate vicinity of the tannery.

5.3.3 Consequence assessment

If SVD were to become established in New Zealand there could be sporadic cases of disease, and problems of differentiating the disease from foot and mouth disease. While diagnostic problems were being resolved there could be serious losses to the animal industries due to disruption of trade. Although the disease could theoretically become established in feral pigs this is considered unlikely as it has not been described as a problem in wild or feral pig populations. Rare cases of SVD have been described in laboratory workers working with the virus (5), others developed antibody but did not become ill. Therefore, the consequences for humans are likely to be minor.

5.3.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents and the organism is resistant to many disinfectants, safeguards are justified for hides and skins of pigs that have not undergone pickling or tanning.

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5.4 LUMPY SKIN DISEASE AND SHEEP AND GOAT POX

The hazard identification concluded that pox viruses (lumpy skin disease and sheep and goat pox) could be associated with cattle hides and sheep and goat skins if the animals were infected at the time of death.

5.4.1 Release assessment

Agent survival

Pox viruses are generally sensitive to common detergents, formaldehyde, oxidizing agents, and temperatures greater than 40°C. The virion surface membrane is removed by non-ionic detergents and sulfhydryl reagents (1). Virions are relatively stable in dry conditions at room temperature (1).

In animals suffering from lumpy skin disease the virus may remain infective in dried skin lesions for 33 days (2), and in the case of sheep and goat pox, the virus may persist in dried pox scabs of recovering animals for several months (3) and in dried scabs in sheep pens for up to 6 months.

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent would destroy virus in or on hides and skins.

As a safeguard against LSD virus, the *Code* recommends storing hides for at least 40 days prior to shipment, while for sheep and goat skins the recommendation is processing to ensure destruction of the virus (methods not specified).

5.4.2 Exposure assessment

Provided the water used for soaking back contains a suitable viricidal detergent, virus on or in hides would be of potential concern only up to this stage. Liquid effluent from soaking back would not be of concern, nor would any subsequent solid or liquid wastes. If a suitable detergent or sanitising agent was not included in the water used for soaking back, the virus could survive in liquid waste and solid waste. However the likelihood of transmission from soaking back water to a susceptible animal is extremely low.

5.4.3 Consequence assessment

If ruminant pox viruses were to become established in New Zealand there would be serious economic losses, due to production losses and mortalities, and restrictions to trade in live animals, hair, wool, skins, semen, and embryos. Sheep and goat pox and lumpy skin disease viruses are not known to be zoonotic. Sheep and goat pox could infect feral goats and thar but this is considered to be unlikely since close contact

between domestic and feral goats and sheep is uncommon. Humans are not susceptible to the virus.

5.4.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants that have not undergone pickling or tanning.

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5.5 AFRICAN SWINE FEVER

The hazard identification concluded that ASF virus could be associated with skins of pigs that were infected at the time of death.

5.5.1 Release assessment

Agent survival

The ASF virus is sensitive to ether, chloroform, and deoxycholate and is inactivated at 60°C within 30 minutes, but survives for years at 20°C or 4°C (1). Infectivity is destroyed by some disinfectants (1% formaldehyde in 6 days, 2% Sodium hydroxide in 1 day). Paraphenylphenolic disinfectants are very effective (1). The virus is stable over a pH range from 4-10 but, in a suitable medium such as serum, it may remain viable for up to 3 days at a higher or lower pH (2). The intact virus is very sensitive to lipid solvents and detergents, and also to oxidising agents such as hypochlorite (3).

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent would destroy much of the virus associated with hides and skins, and remaining infectivity would be destroyed by unhairing, liming, pickling, and tanning.

5.5.2 Exposure assessment

The potential routes of exposure for ASF virus from infected hides and skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

However, unless the tannery is adjacent to a pig farm where pigs are contained in a free range situation the transfer of the virus to pigs is unlikely to occur.

5.5.3 Consequence assessment

If ASF virus were to become established in New Zealand there would be serious economic losses due to mortalities and production losses, trade restrictions imposed on meat, live animals, semen, and embryos. If the disease became established in feral pigs it could cause mortalities in them and they could become a source of infection for farmed pigs. The virus is not a zoonotic organism.

5.5.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents safeguards are justified for hides and skins of pigs that have not undergone pickling or tanning prior to importation.

References

(1) Fauquet CM, Mayo MA, Maniloff J, Desselberger U and Ball LA (2005) *Virus Taxonomy. Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam, Boston, Heidelberg, London, New York, Oxford, Paris, San Diego, San Francisco, Singapore, Sydney, Tokyo.

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(3) Penrith M-L, Thomson, GR and Bastos ADS (2004) African swine fever. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*, Second Editon, Volume 2, pp.1088-119. Oxford University Press Southern Africa, Cape Town.

5.6 CLASSICAL SWINE FEVER AND BOVINE VIRAL DIARRHOEA TYPE 2

The hazard identification concluded that the CSF and BVDV-2 viruses could be associated with skins.

5.6.1 Release assessment

Agent survival

Pestiviruses are rapidly inactivated by solvents and detergents (1). Although the pestiviruses are stable over a relatively broad pH range, their infectivity is quickly lost below pH 4.0 and above pH 11.0 (2). In the environment, viruses are inactivated in a few days (2).

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent at pH 10 would destroy most pestiviruses associated with hides and skins, and any remaining infectivity would be destroyed by unhairing, liming, pickling, and tanning.

5.6.2 Exposure assessment

The potential routes of exposure for exotic pestiviruses from infected hides and skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.6.3 Consequence assessment

If CSF virus were to become established in New Zealand there would be serious economic losses to the pig industry. These would be caused by production losses and mortalities, and restrictions to trade in live animals meat, semen, and embryos. If the disease became established in feral pigs it could cause mortalities in them and they could become a source of infection for farmed pigs. The virus does not infect humans.

Infection of cattle with the exotic BVDV-2 would have severe consequences for the New Zealand cattle industry. The virus is highly infectious and could spread rapidly through the susceptible cattle population. The information currently available indicates that the endemic infection of the population with a comparatively harmless BVDV-1 strain may not provide immunity against BVDV-2. BVDV-2 causes more damaging effects than BVDV-1 and would probably cause significant economic losses for the cattle industry.

5.6.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants and pigs that have not undergone pickling or tanning.

References

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

5.7 HIGHLY PATHOGENIC AVIAN INFLUENZA

The hazard identification concluded that avian influenza viruses could be associated with skins of ostriches and emus that were infected at the time of death.

5.7.1 Release assessment

Agent survival

Orthomyxoviruses are very sensitive to heat, lipid solvents, non-ionic detergents, formaldehyde, irradiation or oxidizing agents (1). They are easily inactivated by drying, and by extremes of pH (2).

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent and at pH 10 would destroy most virus associated with skins, and remaining infectivity would be destroyed by liming, pickling and tanning.

5.7.2 Exposure assessment

The potential routes of exposure for avian influenza viruses from infected skins to susceptible birds in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

Since the importation of untanned emu and ostrich skins is likely to be comparatively rare, the likelihood of transmission to birds is considered to be very low.

5.7.3 Consequence assessment

If avian influenza viruses were to become established in New Zealand there would be serious economic losses to the poultry industry. Some strains of avian influenza virus are zoonotic and could cause human disease. Some strains of the virus could cause mortalities in feral birds and indigenous wild birds. Humans are susceptible to some strains of avian influenza virus and severe disease and death may occur (3).

5.7.4 *Risk estimation*

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for ostrich and emu hides and skins that have not undergone pickling or tanning.

References

(1) Fauquet CM, Mayo MA, Maniloff J, Desselberger U and Ball LA (2005) *Virus Taxonomy. Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam, Boston, Heidelberg, London, New York, Oxford, Paris, San Diego, San Francisco, Singapore, Sydney, Tokyo.

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(3) WHO (2007). Cumulative number of confirmed human case of avian influenza A/(H5N1) reported to WHO http://www.who.int/csr/disease/avian_influenza/country/cases_table_2007_04_11/en/index.html

5.8 NEWCASTLE DISEASE

The hazard identification concluded that avian paramyxoviruses could be associated with skins of birds that were infected at the time of death.

5.8.1 Release assessment

Agent survival

The virus is very sensitive to heat, lipid solvents, ionic and non-ionic detergents, formaldehyde, and oxidizing agents (1).

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent would destroy most virus associated with skins, and remaining infectivity would be destroyed by liming, pickling, and tanning.

5.8.2 Exposure assessment

The potential routes of exposure for avian paramyxoviruses from infected skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

Since the importation of untanned emu and ostrich skins is likely to be comparatively rare, the likelihood of transmission to birds is considered to be very low.

5.8.3 Consequence assessment

If Newcastle disease virus were to become established in New Zealand there would be serious economic losses to the poultry industry. It would also constitute a threat to feral wild birds and indigenous native birds. Although laboratory accidents have resulted in cases of conjunctivitis in humans, cases of human infection are rare and of little consequence.

5.8.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for ostrich and emu hides and skins that have not undergone pickling or tanning.

References

(1) Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (2005) *Virus Taxonomy. Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam, Boston, Heidelberg, London, New York, Oxford, Paris, San Diego, San Francisco, Singapore, Sydney, Tokyo.

5.9 ANTHRAX

The hazard identification concluded that anthrax could be associated with hides and skins of animals that were infected at the time of death. The UK Committee of Enquiry into Anthrax concluded in 1959 that dry and dry-salted hides and skins were far more likely to carry anthrax spores than wet-salted materials, because wet-salted materials came from animals killed in abattoirs while sun-dried and dry-salted materials may include some hides from animals which had died of anthrax (1).

5.9.1 Release assessment

Agent survival

The spores of *Bacillus anthracis* can be inactivated only by very harsh physical and chemical conditions.

Spores are heat resistant and withstand exposure to alcohols, phenols, quaternary ammonium compounds, ionic or non-ionic detergents, acids and alkalis (2). Most commercial disinfectants are ineffective against anthrax spores. For various applications 2-10% formaldehyde, 3% hydrogen peroxide, 1% peracetic acid or high concentrations of hypochlorite are recommended (3, 4). For disinfection of hides and skins 5-10 % formaldehyde is recommended for 10 hours; an alternative of 3% peracetic acid can also be used but is more expensive (4). In the First Schedule of the Anthrax Prevention Regulations 1987, the recommended method for disinfection is immersion in a 1/10,000 solution of sodium bisulphate for not less than 5 hours or immersion in a solution of free chlorine to a level of 200 ppm for not less than 2 hours. However, no data could be found that indicates that sodium metabisulphate is an effective biocide for anthrax spores.

For disinfection of water 5-10% formaldehyde for 10 hours is recommended (5). Problems associated with discharging high concentrations of formalin into the environment can be avoided if the waste is retained until the formalin has been degraded. The half life of formalin in water has variously been quoted as being 2-20 days (5) or 36 hours (6). It depends on the bacterial flora present and on levels of oxidising agents in the water. Therefore if a 5% solution of formalin is allowed to stand for 10 days before discharge, the concentration of formalin still in the waste water will be very low.

Anthrax spores are more easily inactivated by moist heat than by dry heat. Inactivation by dry heat at 140°C may require up to three hours (2), whereas Murray found that spores exposed to moist heat survive for 15-45 minutes at 90°C, for 10-25 minutes at 95°C, and 2-15 minutes at 100°C (7). Sterne, found that autoclaving at 120°C destroys spores in 10 minutes (7).

Gamma irradiation has been used for the decontamination of wool and a dose of 40kGy is recommended (4).

Effect of hides and skins processing

Soaking back would have little effect on anthrax spores if present, and large volumes of potentially contaminated waste water could be produced. Similarly, fleshing following soaking back would have no effect on spores but could potentially generate contaminated solid waste.

The UK Committee of Enquiry into Anthrax considered that the major risk of anthrax comes from handling infected hides and skins up to the liming stage, as only a very small proportion of anthrax cases in tannery workers were seen in persons working at later stages of processing. Dry hides can be decontaminated before processing by fumigation with formaldehyde or ethylene oxide (1, 4). The de-hairing stage, which involves liming with a mixture of sodium sulphide and calcium hydroxide, exposes the skins to a high pH, which is likely to kill any spores present (4).

For rendering of solid waste, the temperature must be held at 100-150^{o}C for at least 10 minutes (4).

The likelihood of release of anthrax spores in tannery wastes depends on the source of the hides and the procedures followed for the processing of hides, and the treatment of the waste materials generated.

Anthrax bacilli will only be found in animals that are suffering from anthrax. In cattle and sheep the incubation period is short and the disease course is short, usually often lasting a few hours. In animals in which sub-acute infections occur (pig and horse) the animals would be showing clinical signs of disease and would not be slaughtered. Therefore animals that are healthy and subjected to ante-mortem and post-mortem inspection before slaughter will not be a risk.

5.9.2 Exposure assessment

It is recognised that tannery sites and tannery effluents could be contaminated with spores if infected hides and skins are processed. It is thought that sporadic outbreaks on farms in Great Britain were related to flooding of rivers in areas near disused tanneries where infected imported hides had been processed three decades previously (8). Outbreaks of anthrax have been associated with effluent discharge from tanneries in Italy, Germany, England and Canada (4, 8, 9, 10, 11).

The potential routes of exposure for anthrax from infected hides and skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.9.3 Consequence assessment

If anthrax were to become established in New Zealand it could cause losses in production, mortalities in stock, and sporadic cases of anthrax in humans and in feral animals.

5.9.4 Risk estimation

Since anthrax spores have often been associated with hides and skins and the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants and pigs that have not undergone pickling or tanning.

References

(1) Ministry of Labour (1959) Report of the committee of inquiry on anthrax, p.43-7. HMSO, London.

(2) Böhm R (1990) Resistance, survival, sterilization and disinfection of spores of Bacillus anthracis. In: Turnbull PCB (ed). *Proceedings of the International Workshop on Anthrax*. Winchester, England, April 11-13, 1989. *Salisbury Medical Bulletin* 68, (supp), pp.99-101.

(3) De Vos V and Turnbull PCB (2004) Anthrax. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Vol 3, pp.1788-818. Oxford University Press Southern Africa, Cape Town.

(4) Turnbull PCB (1998) *WHO Guidelines for the Surveillance and Control of Anthrax in Humans and Animals*, Third edition. http://www.who.int/csr/resources/publications/anthrax/whoemczdi986text.pdf

(5) Department for the Environment and Heritage. Commonwealth of Australia. (2005) *Formaldehyde (methyl aldehyde) fact sheet*. http://www.npi.gov.au/database/substance-info/profiles/45.html

(6) Katz SE (1995) *Environmental impact assessment for the use of formalin in the control of external parasites of fish.* http://www.fda.gov/cvm/FOI/140-989EA.pdf

(7) Mitscherlich E and Marth EH (1984) *Microbial Survival in the Environment*, p.11. Springer-Verlag, Berlin, Heidelberg.

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(9) Perone A, Gelosa L (1982) [Findings on the spread of anthrax spores in the provincial territory of Milan with tanneries], *Giornale di batteriologia, virologia ed immunologia*, 75(7-12), pp.322-36.

(10) Moynihan WA (1963) Anthrax in Canada. *Canadian Veterinary Journal*, 4, pp.283-7.

(11) Zettl K and Kauker E, (1959) Incidence of anthrax and blackleg in the German Federal Republic. *Berl-Munch-tierarztl-Wschr* 72, pp.426-9.

5.10 Q FEVER

The hazard identification concluded that *Coxiella burnetii* could be associated with hides and skins of animals that were infected at the time of death.

5.10.1 Release assessment

Agent survival

Coxiella burnetii spores are highly resistant to elevated temperatures, osmotic shock, ultraviolet light and chemical disinfectants (1). Formalin (5%), Lysol (5%), sodium hypochlorite (0,5%) and roccal (2%) were not effective. Formalin fumigation gave variable results and appeared to be more effective if used at a high humidity and in a small space. Ethylene oxide fumigation was effective (1). Another author found 5% Lysol to be the most effective disinfectant (2). According to Maurin and Raoult, "they are resistant to environmental conditions such as desiccation, low or high pH chemical products such as ammonium chloride, disinfectants such as 0.5% sodium hypochlorite, and UV irradiation. Only exposure to high concentrations of formalin (i.e. $\geq$5%) for a prolonged period of time (at least 24-48 h) may allow killing of *Coxiella Burnetii"* (3).

There is nothing in the literature that suggests that Q fever outbreaks in animals have been caused by transmission from tannery effluents. In a British report on ill health in agriculture, the occurrence of Q fever was not reported as a disease associated with tannery workers (4). Therefore, the likelihood of release is low but not negligible.

Effect of hides and skins processing

Soaking back in the presence of 5% non-ionic detergent is unlikely to destroy *Coxiella burnetii* associated with hides and skins, but it would be inactivated by liming, pickling, and tanning.

5.10.2 Exposure assessment

Exposure of humans would most likely be through the respiratory or oral routes by means of exposure to dust while processing sheep skins (especially wool skins) and cattle hides. The potential routes of exposure for *Coxiella burnetii* from infected skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

However, no evidence was found that indicates that Q fever infections are associated with tannery workers. Therefore, the likelihood of exposure is very low but not negligible.

5.10.3 Consequence assessment

Q fever in imported hides and skins would primarily be a public health issue but, if the agent of Q fever were to become established in New Zealand livestock, there could be sporadic minor incidents of disease in livestock, such as sporadic abortions.

5.10.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants and pigs that have not undergone pickling or tanning.

References

(1) Scott GH and Williams JC (1990) Susceptibility of *Coxiella burnetii* to chemical disinfectants. *Annals of the New York Academy of Sciences*, 590, pp.291-296.

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(4) Cowie HA, Soutar CA, RA Graveling, TJ Cattermole, JW Cherrie, MK Graham and RM Mulholland (2005) Institute of Occupational Medicine for the Health and Safety Executive, Research report 370, *Baseline incidence of ill health in agriculture in Great Britain*. Her Majesty's Stationary Office, London. http://www.hse.gov.uk/research/rrpdf/rr370.pdf

5.11 BRUCELLOSIS

The hazard identification concluded that *Brucella* spp. could be associated with skins of cattle, sheep and goats.

5.11.1 Release assessment

Agent survival

Brucella spp. grow at pH between 5 and 8 (1). *Brucella* spp. can survive for 1-2 months in dry soil, at room temperature for 2-3 months in wet soil, and 3-4 months in faeces (2). It survived on hairless hide for up to 5 days and on hairy hide for up to 8 days (Parli cited by Mitscherlich and Marth (3)). Therefore, *Brucella* spp. could survive in faeces and reproductive tract discharges on hides and skins.

Effect of hides and skins processing

Brucella abortus is susceptible to heat, desiccation, UV light, and the usual disinfectants (4). Salting in sea salt containing 2% sodium carbonate would inactivate *Brucella* spp. They would also be inactivated by biocides and alkali added at soaking back.

5.11.2 Exposure assessment

Exposure of humans would most likely be through the respiratory or oral routes after exposure to dust while processing sheep and goat skins. If biocides and alkali were not added at the soaking back stage, the organism would not be inactivated, in which case the potential routes of exposure for *Brucella melitensis* from infected skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.11.3 Consequence assessment

The introduction and establishment of *Brucella abortus*, *Brucella melitensis* or *Brucella suis* into New Zealand would have a serious impact on the animal industries concerned. Production losses, costs of control programmes, and loss of international market access could occur. *Brucella* spp. cause serious disease in humans and sporadic cases could be expected to occur if the organism became established in animal species. *Brucella suis* could become established in feral pigs. *Brucella abortus* could infect deer but it does not cause significant disease problems in deer. *Brucella melitensis* could infect feral goats and thar.

5.11.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants and pigs that have not undergone pickling or tanning.

References

(1) Mitscherlich E and Marth EH (1984) *Microbial Survival in the Environment*, p.564. Springer-Verlag, Berlin, Heidelberg.

(2) Ministry of Agriculture and Fisheries (1977) In Elliot REW, Christiansen KH (eds), *Brucellosis: A Veterinarians Guide to the Literature*. Ministry of Agriculture and Fisheries, Wellington.

(3) Mitscherlich E and Marth EH (1984) *Microbial Survival in the Environment*, p.57. Springer-Verlag, Berlin, Heidelberg.

(4) Animal Health Australia (2005) *Ausvetplan. Disease strategy. Bovine brucellosis*. Version 3. http://www.animalhealthaustralia.com.au/aahc/index.cfm?E9711767-B85D-D391-45FC-CDBC07BD1CD4#sum

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5.12 ENZOOTIC ABORTION OF EWES

The hazard identification concluded that *Chlamydophila abortus* could be associated with skins of sheep and goats.

5.12.1 Release assessment

Agent survival

The organism occurs in two forms - the elementary body (condensed form) and the reticulate body. The elementary bodies are more resistant and survive longer in the environment than reticulate bodies. The closely related *Chlamydophia psittaci* grows at pH 6.5 - 7.5 (Moulder and Weiss cited by Mitscherlich and Marth (1)), survived desiccation for at least 20 days (Diehl cited by Mitscherlich and Marth (1)), and remained viable in litter from turkey farms for 60-240 days (Meyer cited by Mitscherlich and Marth (1)). Another author states that chlamydiae survive between pH 7 and 8 and they gradually lose viability at pHs outside this range (2). Therefore, it can be assumed that they will be inactivated at pH >10. The organism is sensitive to ethanol, formalin, phenol, quaternary ammonium salts, chlorine, iodine, and permanganate (2).

Effect of hides and skins processing

Salting in sea salt containing 2% sodium carbonate would inactivate *Chlamydophila abortus*. The detergent, biocides, and alkali added at soaking back would inactivate any bacteria present at that stage.

5.12.2 Exposure assessment

If biocides and alkali were not added at the soaking back stage, the organism would not be inactivated, in which case the potential routes of exposure for the organism from infected skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.12.3 *Consequence assessment*

The introduction and establishment of *Chlamydophila abortus* into New Zealand would have a serious impact on the sheep industry in terms of production losses, cost of control, and on international market access. It could also cause sporadic cases of abortion in women.

5.12.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of ruminants that have not undergone pickling or tanning.

References

(1) Mitscherlich E and Marth EH (1984) *Microbial Survival in the Environment*, p.565. Springer-Verlag, Berlin, Heidelberg.

(2) Eugster AK (1980) Chlamydiosis. In Steele HH, Stoenner H, Kaplan W, Torten M (eds), *Handbook series in Zoonoses. Section A Bacterial, rickettsial and mycotic diseases Volume II*, pp.357-417. CRC press Boca Raton.

5.13 ENTEROBACTERIACEAE

The hazard identification concluded that *Salmonella* spp. and other Enterobacteriaceae could be associated with skins of sheep, as a result of faecal contamination.

5.13.1 Release assessment

Agent survival

Salmonella spp. grow within a pH range of 4.5-9.0 (optimum is 6.5-7.5) (1). However, many of these organisms show surprising resistance to acidic and basic conditions and show the ability to become more resistant to changes in pH and other harmful environmental conditions if first exposed to mildly acid or alkaline conditions. *Escherichia coli* and *Shigella flexneri* survive pH levels as low as 2 and 2.5, and *Salmonella* Typhimurium survived pH 3.0 (2, 3). Treatment of manure contaminated with *Escherichia coli* O157:H7 and *Salmonella* Typhimurium DT104 with high concentrations of carbonate (>30mM) at pH adjusted to 9.5 reduced the counts of both bacteria by more than 5 logs per ml after 6 hours (4). Formalin at 0.6% concentration reduced the numbers of seven species of Enterobacteriaceae by 7 logs within 7 hours (5).

Effect of hides and skins processing

Acid-adapted organisms were resistant to 8% sodium chloride and significant numbers survived after 18 days at 25^0C and were 10^4 times more resistant than control cells (3). These findings probably indicate that wet-salting of non-acid-adapted organisms will probably inactivate most Enterobacteriaceae but adaptation to survival in a harsh environment may make them resistant. Since high pH and carbonate is effective in killing *Escherichia coli* and *Salmonella* Typhimurium, salting in sea salt containing 2% sodium carbonate would inactivate Enterobacteriaceae. The detergent, biocides and alkali added at soaking back would inactivate any bacteria present at that stage.

5.13.2 Exposure assessment

If biocides and alkali were not added at the soaking back stage, the organism would not be inactivated, in which case the potential routes of exposure for the organism from infected skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.13.3 Consequence assessment

The introduction and establishment of unwanted *Salmonella* spp. into New Zealand would have a serious impact on animal industries in terms of production losses and cost of control and treatment. Introduction of exotic species (such as *Salmonella* Typhimurium DT104) or species with transferrable plasmids encoding novel antimicrobial resistance mechanisms could have serious consequences for human health.

5.13.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents, safeguards are justified for hides and skins of all species of animals that have not undergone pickling or tanning.

References

(1) Anonymous (2004) *Salmonella* spp. infections. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Second Edition, Volume 2, p.1578. Oxford University Press Southern Africa, Cape Town.

(2) Lin J, Lee IS, Frey J, Slonczewski JL and Foster JW (1995) Comparative analysis of extreme acid survival in *Salmonella Flexneri*, and *Escherichia coli*. *Journal of Bacteriology* 177(14), pp.4097-104.

(3) Tosun H and Aktug Gonul S (2003) Acid adaption protects *Salmonella typhimurium* from environmental stress. *Turkish Journal of Biology*, 27, pp.31-6.

(4) Park GW and Diaz-Gonzalez F (2003) Utilization of carbonate and ammonia-based treatments to eliminate *Escherichia coli* O157H7 and *Salmonmella* Typhimurium DT 104 from cattle manure. *Journal of Applied Microbiology* 94, p.675.

(5) Farmer JJ (1975) Formalinized bacterial antigens as a potential infection hazard. *Journal of Clinical Microbiology* 2(4), pp.359-60.

5.14 GLANDERS

The hazard identification concluded that glanders (*Burkholderia mallei*) could be associated with horse hides.

5.14.1 Release assessment

Agent survival

Burkholderia (formerly *Pseudomonas*) *mallei* grows in media of pH 6.5-7.5 (1). Outside the body, the organism has little resistance to external factors and is destroyed by direct sunlight within a day. It may survive approximately one month in clean water and one month in contaminated stables. The agent is killed by most common disinfectants including phenol, chlorine, and formalin(2).

Effect of hides and skins processing

Salting in sea salt containing 2% sodium carbonate would inactivate *Burkholderia mallei*. The detergent, biocides, and alkali added at soaking back would inactivate bacteria present at that stage.

5.14.2 Exposure assessment

The organism can remain viable in tap water for a month (2). If biocides and alkali were not added at the soaking back stage, the organism would not be inactivated, in which case the potential routes of exposure for the organism from infected skins to susceptible animal species in New Zealand would be:

- Waste water
- Solid waste

5.14.3 Consequence assessment

The introduction and establishment of *Burkholderia mallei* would have a serious impact on the horse industry. It is a zoonotic organism and could cause serious, potentially fatal, disease in humans.

5.14.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents safeguards are justified for hides and skins of horses that have not undergone pickling or tanning.

References

(1) Mitscherlich E and Marth EH (1984) *Microbial Survival in the Environment*, p.569. Springer-Verlag, Berlin, Heidelberg.

(2) Van der Lugt JJ and Bishop, GC (2004) Glanders. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Second Edition, Volume 3, pp.1500-4. Oxford University Press Southern Africa, Cape Town.

5.15 PORCINE ENTEROVIRUSES

The hazard identification concluded that porcine enteroviruses could be associated with pig skins.

5.15.1 Release assessment

Agent survival

Porcine enteroviruses are relatively stable over a pH range of 2.8 and 9.5, but are rapidly inactivated at more extreme values (1). Enteroviruses are rapidly inactivated by UV light and drying (2).

Effect of hides and skins processing

Although porcine enteroviruses are unlikely to be present in or on dried or dry-salted hides and skins, they could remain viable in wet-salted hides and skins unless the OIE-recommended addition of 2% sodium carbonate is carried out.

Soaking back would inactivate the virus if the pH of the soaking back solution exceeded 9.5.

The pH levels of liming (pH 12.5-13), pickling and tanning (pH <3) would destroy porcine enteroviruses.

5.15.2 Exposure assessment

If the pH of the soaking back liquid did not exceed 9.5, the porcine enteroviruses would not be inactivated and thus, the potential routes of exposure from infected pig skins to susceptible animal species in New Zealand would be the following waste materials generated through soaking back and fleshing:

- Waste water
- Solid waste

5.15.3 Consequence assessment

The introduction and establishment of pathogenic exotic porcine enteroviruses into New Zealand would have a moderate impact on the pig industry in terms of production losses and cost of control.

5.15.4 Risk estimation

Since the soaking back procedures may not include the use of suitable germicidal reagents safeguards are justified for pig skins that have not undergone pickling or tanning.

References

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(2) Melnick JL (1996) Enteroviruses: Polioviruses, Coxsachieviruses, Echoviruses, and Newer Enteroviruses. In: Fields BN, Knipe DM, Howley PM (eds). *Fields Virology*. Third Edition, pp.657-8. Lippincott-Raven, Philadelphia.

5.16 WEED SEEDS

The hazard identification concluded that weed seeds could be associated with hides and skins.

5.16.1 Release assessment

The category is too broad to make specific comments. However, some weed seeds that are highly resistant to the environment and the physical and chemical treatments normally used on hides and skins could be introduced on the commodities.

5.16.2 Exposure assessment

Weed seeds introduced on hides and skins could be transmitted to the environment in solid and liquid wastes generated during the processing of the commodities. If released into the environment, the seeds could germinate and subsequently reproduce in suitable environments.

5.16.3 Consequence assessment

The consequences would depend on the species of seed introduced. The seeds introduced could be harmful to agricultural crops or to the environment. The effects could vary from negligible to causing severe economic and environmental impacts. Direct effects on human health are unlikely.

5.16.4 Conclusion

Since weed seeds could be introduced and could have significant effects on agriculture and the environment, the implementation of risk management measures can be justified.

5.17 HITCH-HIKER PESTS

The hazard identification concluded that hitch-hiker pests are hazards to be considered in this risk analysis.

5.17.1 Release assessment

Hitch-hiker pests constitute an extremely large number of unknown hazards. Many are likely to be detectable on careful inspection but some could be virtually impossible to detect. Interception records since 1989 have recorded only one arachnid associated with imported hides and skins, *Ixeuticus martius*, a species which is known to be present throughout New Zealand. Therefore, the likelihood of a hitch-hiker pest being released is considered to be low but non-negligible.

5.17.2 Exposure assessment

There are many possible pathways by which hitch-hiker pests could escape from a transitional facility and become established in the external environment. These include waste water release to the environment, wind transport of dust containing fungal or bacterial spores, and mechanical transfer of organisms in mud, dried blood etc by tannery workers and on vehicles leaving the tannery. Live insects could walk or fly away from a facility. Animals or plants in the environment could be exposed to organisms that have escaped from the transitional facility.

5.17.3 Consequence assessment

The consequences would depend on the organism concerned and could vary from negligible to severe. Specific effects on the environment and human health would depend on the pest introduced.

5.17.4 Conclusion

Since hitch-hiker pests could be introduced on the commodity and could cause significant harmful effects, implementation of risk management measures can be justified.

5.18 RISK MANAGEMENT

5.18.1 Risk evaluation

Since pickled, limed and tanned hides do not pose any biosecurity risk, the options discussed here are restricted to importation of dried and salted hides and skins.

For all disease agents the potential routes of exposure are waste water and solid wastes generated during processing. Inactivating relevant organisms in waste products or disposing of them safely will prevent the introduction of harmful organisms.

The two diseases of greatest concern anthrax and foot and mouth disease. It may be appropriate to adopt additional measures to manage the risks posed by these two agents.

5.18.2 Risk management options

The likelihood of hides and skins carrying infectious agents of concern (especially anthrax) would be significantly reduced if the were derived only from animals that were slaughtered in an officially registered abattoir where the animals had been subjected to ante-mortem and post-mortem inspection and found to be free from any signs of infectious disease and certified as suitable for human consumption.

When importing skins from countries that are not free from foot and mouth disease, the veterinary authority of the exporting country might be able to certify that the animals from which the hides or skins were derived did not come from infected herds or flocks. Alternatively, hides and skins from such countries could be preserved by salting with salt containing 2% sodium carbonate for at least 4 weeks in order to inactivate the virus.

The likelihood of organisms being present on imported with hides and skins would be reduced if they were accompanied by documentation certifying that they were clean, and not visibly contaminated with plant material, seeds, soil, dirt, or arthropods pests.

The likelihood of any organisms escaping from the imported hides and skins prior to processing would be minimised by imported hides and skins being securely packaged in impermeable wrapping material.

Visual inspection of shipments by MAF Inspectors at the point of importation would allow an assessment of packaging adequacy to be made and any flaws in the packaging to be addressed by repackaging, reshipping or destruction. At the same time, inspectors could implement appropriate treatments for any hitch-hiker pests found as a result of that inspection.

The likelihood of any organisms being released from the imported commodities could be managed by having the hides transported directly to a tannery/processing plant that

is a transitional facility approved under the MAF Standard 154.02.18 and has been nominated on the permit to import.

The risk of release of organisms from the transitional facility prior to processing could be managed by clearly marking the imported hides and skins and storing them separately from non-imported ones. To further increase security, storage prior to processing could be in a facility that is maintained free from rodents and cannot be accessed by birds.

Import health standards for the importation of the commodities could specify, where applicable, how waste products generated during the processing of imported hides and skins, including the packaging they are contained in, must be disposed of. Options for the disposal of the wastes generated when processing imported hides and skins include:

- Discharge of liquid wastes into municipal sewage systems that do no dispose of sewage onto agricultural land or into rivers; **or**
- Discharge of liquid waste into a securely fenced and isolated soak-pit or by another method approved by MAF; **or**
- Treatment of liquid wastes by a MAF approved method, before discharge; **and**
- Rendering or incineration of solid wastes and packaging materials.

A suitable disinfectant for treating liquid wastes would be 5% formaldehyde (12.5% formalin) which is effective for anthrax spores if used for at least 10 hours (1). Five percent formalin (2% formaldehyde) is effective for Q-fever if used for 24-48 hours (2). All other disease agents that are considered to be potential hazards are more sensitive to disinfectants than these two organisms. To minimise environmental contamination from waste water containing formalin, waste water could be left to stand until the formaldehyde has degraded to a low level before being discharged.

References

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