THE IMPORTATION INTO NEW ZEALAND OF MEAT AND MEAT PRODUCTS:

A REVIEW OF THE RISKS TO ANIMAL HEALTH

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SUMMARY OF RECOMMENDATIONS

Mammalian meat products

All mammalian meat and meat products except meat-, blood- and bone- meals imported into New Zealand;

either (i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection[Footnote] and

or (ii) In the case of game animals only must originate from animals which have passed veterinary post-mortem inspection and

(iii) Must have been processed in premises under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iv) Must comply with the specific safeguards (listed in section 8.1) for foot and mouth disease, swine vesicular disease, rinderpest, African swine fever, hog cholera, anthrax, hydatids, leptospirosis, Aujeszky's disease, scrapie and BSE, cysticercosis, vesicular exanthema of swine, trichinellosis, tularaemia, viral haemorrhagic disease of rabbits, brucellosis and tuberculosis and

(v) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

Throughout this review it is assumed that before importation is permitted from countries outside the range of our traditional sources of meat products a MAF assessment of the veterinary and inspection services will be carried out. In many instances this assessment will require a visit to the country by a MAF specialist. However, in some cases, acceptance by other major trading partners, once verified officially, may be considered sufficient. For example, if the Australian authorities were to consider that the veterinary and zoosanitary infrastructure of a particular country was sufficiently developed for importation into Australia to be acceptable, then New Zealand MAF should be prepared to adopt a similar stance. See section 3.1.

Meat-, blood- and bone-meals must comply with the specific safeguards (listed in section 8.1) for anthrax, scrapie and BSE.

Poultry meat products

All poultry (avian) meat and meat products imported into New Zealand;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been processed in premises under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iii) Must comply with the specific safeguards (listed in section 8.1) for Newcastle disease and infectious bursal disease and

(iv) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

Recommendations for crocodile meat products

All crocodile meat and meat products imported into New Zealand;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been processed under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iii) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

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1. Introduction

The New Zealand consumer has become more cosmopolitan in his or her tastes in recent years and, as importers try to satisfy demand for specialist meat products, MAF receives requests for permits to import a broad range of meat products such as Hungarian salamis, Italian Parma hams and Asian meat or chicken flavoured instant noodle dishes etc. The popularity of modern convenience foods has led to a demand for the importation of prepared frozen meals containing meat or chicken. Some importers wish to tempt the consumer with unusual or exotic meats while local manufacturers want to be able to import meat-based products for incorporation in locally-manufactured foods. It is for all these reasons that a review of MAF policy on the importation of meats and meat-containing products is necessary.

The intention of the present review is to provide a technical assessment of the animal health risks posed by different types of meat product, from different sources, so as to allow as wide a range of products as possible access to the New Zealand market without jeopardising this country's enviable animal health status.

At present, the following categories of meat products are permitted entry into New Zealand.

(a) Australia, Canada, Republic of Ireland, Europe, United Kingdom, United States of America and Japan.

(i) Cooked meats of animals and their products contained in hermetically sealed tin cans, glass containers or retort pouch packs (aluminium foil type container) which do not need to be kept refrigerated.

(ii) Cooked meat products which have been dehydrated (for example, instant meat and chicken soups, gravy

etc packed either in sealed tinfoil packets, in sealed jars, or as cubes or cakes) or are packed in sealed glass containers, providing they are commercially manufactured and packed.

(b) Other products on an approved meat products list.

Uncooked meat products which have been preserved by chemical means, salting, drying, pasteurisation methods or accelerated freeze-drying are prohibited.

Imported meat products are treated in two separate ways:

(a) Further processing within MAF meat service licensed premises either for the local market or re-export.

(b) Direct use on the domestic market.

Products offered for sale on the domestic market must comply with the Food Act, and this is stated on the import permit. The Food Act does not apply to exports but it is impractical to isolate the product intended for export or to prevent any residue of such product appearing on the domestic market.

Products intended for re-export must have been produced in the country of origin under conditions equivalent to those which apply to production in
New Zealand (Meat Act). This equivalence is not specified for imports for the domestic market but has nevertheless been included in recent import permits. The New Zealand public should not be exposed to product which is inferior nor should the New Zealand producer be disadvantaged by competition which does not conform to the rules. The New Zealand law should be viewed as a minimum standard.

The use of equivalency as part of the import permit is open to challenge. Not all the conditions can be related to the prevention of disease introduction (Animals Act). The Primary Products Bill will remedy this discrepancy. Import permits will then be issued in consideration of the Animals Act and the Primary Products Act.

The approach used in this review has been to examine the potential risks posed by each type of meat or meat-based product and make recommendations. The assessments of risk are qualitative, rather than quantitative. This approach was chosen because in many cases examined the actual quantitative risk elements in each assessment are unknown. For this reason the discussion has been kept at a

qualitative level only. Phrases such as a "relatively high risk" or "virtually without risk" are used. It is hoped that the reader will accept that, in general, to attempt to quantify most of these risks serves little purpose, as any numerical estimate can only be the product of a number of guesses multiplied together. Such a numerical risk factor may have little validity. This is not to say that numerical risk factors cannot be calculated for specific imports from specific countries. Indeed, such risk assessments may well be warranted when assessing specific import proposals (see Appendix I).

2. Types of product

Several preservation methods are used to allow meats and meat-based products to be stored and transported. The intention of all these methods is to reduce the activity of those microorganisms responsible for the deterioration and spoilage of the product. The preservation methods may also act to destroy, or reduce the numbers of, pathogens present in meat which has come from a diseased animal or contaminated carcass. In some instances, however, measures intended to preserve the product may also preserve the pathogen.

Methods of meat preservation are;

(a) Refrigeration and freezing
(b) Thermal processing
(c) Dehydration
(d) Irradiation
(e) Chemicals.

2.1 Refrigeration and freezing

Refrigeration is the most common method of meat preservation. Low temperatures retard microbial growth as well as enzymatic and chemical reactions. Reduction of temperature below -2^{o}C causes meat to freeze.

Carcasses of beef, mutton and pork are chilled (kept below 10^{o}C) by hanging in chilling rooms. Poultry carcasses are usually chilled by immersion in cold water, and this has the potential to spread pathogens from one carcass to a batch of carcasses.

Freezing may inactivate a number of livestock pathogens (such as *Trichinella spiralis* and tapeworm cysts) but in most cases refrigeration and freezing prevent multiplication of pathogens but do not reduce their numbers.

2.2 Thermal processing

Thermal treatment is the most widely used method of killing microbial agents in meat. In general, products may be subjected to a moderate level of heat (such as in many cured products) which extends the refrigerated shelf life of a product, or they may be subject to a more severe (above 100^{o}C) heat treatment (such as canning) which results in a product which can be stored without refrigeration.

The first type of heat treatment may destroy livestock pathogens in meat, or it may only reduce their number. In most cases the second type of heat treatment (e.g. canning) will destroy all pathogens, with the possible exception of *Bacillus anthracis* spores.

2.3 Dehydration

Many meat products are preserved by a process of air drying. Some may be dried over smoke. Others, such as salamis, may be subject to a fermentation stage during their production and the resulting drop in pH, coupled with their partial dehydration, is responsible for their extended shelf-life.

Meat products may be dried by a hot air process or a freeze drying process. The dehydration may have little effect on animal pathogens present in the meat. Indeed, freeze drying (lyophilisation) may actually preserve viruses and bacteria. However, the various curing or heating processes which occur in some products may contribute to rendering them free from livestock pathogens.

2.4 Irradiation

Ionizing radiation may be used to kill microorganisms in or on meat without appreciably raising its temperature. While radiation may be used to destroy virtually all pathogens in meat, irradiated food has yet to gain acceptance in New Zealand.

2.5 Chemicals

Traditional curing agents used in the preservation of meat have been sodium chloride, sodium nitrite or nitrate, sugar and, sometimes, spices. These agents are usually used in combination. The microorganisms mainly responsible for meat spoilage are gram-negative bacteria. The chemicals used as curing agents tend to inhibit these. They have little effect on other microorganisms.

Salt is the most common constituent of curing mixtures. By increasing osmotic pressure and lowering water activity (A_w) of meat, salt inhibits the growth of some microorganisms. It is unlikely, however, to render safe meat taken from viraemic or bacteraemic livestock.

Nitrite exhibits a variable bacteriostatic effect against different strains of microorganism, and the mechanism by which it exerts its bacteriostatic effect differs from one organism to another. Its effect is dependent on pH.

Nitrate has a rather obscure effect but, like salt, will reduce water activity.

Sugar at very high concentrations can be used to preserve foodstuffs. However, at the concentrations commonly used in meat curing it is unlikely to extend any significant antimicrobial effect.

Although some spices contain essential oils which may be bacteriostatic, at the concentrations used in cured meats no antimicrobial effect can be expected.

Woodsmoke has a complex chemical composition. Among the many chemicals which have been identified in woodsmoke, formaldehyde and phenolic compounds appear to be the chief bacteriostatic and bactericidal substances.

Smoking of most meat products is accompanied by varying amounts of heat. In addition to the dehydration which also occurs, the formaldehyde and phenol form a resinous barrier over the surface of the meat.

These days very few meat products are produced in which smoking plays a significant role in preservation. The effects of smoking are largely confined to appearance and flavour. For this reason "liquid smokes" are often used in modern meat product manufacture.

Organic acids are sometimes used for their bacteriostatic and fungistatic properties. They act to lower the pH of a product and in so doing may inactivate some important livestock pathogens (e.g. foot and mouth disease virus). Lactic acid is usually produced in muscle tissue during *rigor mortis*. In some products (fermented sausages such as thuringer, cervelat, Lebanon, bologna, dry summer sausages) a fermentable sugar is added to the sausage mixture and is fermented to lactic acid by starter cultures.

2.6 Inactivation of pathogens

Throughout the literature, and the text of this report, it is stated that heating a product to a certain temperature for a

certain period of time will inactivate , kill or destroy pathogens. However, in many instances, it is not possible to prescribe heat treatments which will result in the inactivation of all virus particles. Heat sterilisation is dependent on the particular organism, the medium, pH, temperature and time of exposure. At a given temperature there is usually a linear relationship (often biphasic) between log infectivity and time. For this reason, survival of infectivity is also dependent on the initial titre, and complete sterility cannot be predicted, only the probability of viruses surviving under the stated conditions. When the probability is remote, sterility is effectively achieved.[1]

The effect of initial titre and the often-biphasic inactivation of pathogens may be responsible for the apparently contradictory results sometimes recorded in different studies.

2.7 The different meats considered

The types of meat and meat product which will be examined in this review are shown in Table 1. The species of animal from which meat or meat products are likely to be derived are listed in Table 2.

TABLE 1: TYPES OF MEAT PRODUCT

Chilled meat
Frozen meat
Offals, chilled and frozen
Cured meats requiring refrigeration
Cooked meats requiring refrigeration (includes canned meats)
Dehydrated meat, dried at ambient temperature
Dehydrated meat, hot-air dried
Dehydrated meat, freeze-dried
Cured meats not requiring refrigeration
Meat extracts and flavourings

TABLE 2: SPECIES OF MEAT CONSIDERED

Species	Meat
Cattle	beef, veal
Sheep	mutton, lamb
Goat	chevon
Pig	pork
Deer	venison
Horse	
Rabbit	
Chicken	
Turkey	
Duck	
Goose	
Buffalo	
Kangaroo	
Crocodile	
Ostrich	

It can be seen that in some instance the meat for which import permit is sought has undergone a minimum of processing and so the safety of the product is very much dependent on the health status of the animal from which it is derived and by the degree of cross contamination which may have occurred

during processing. In other cases, the meat has been subject to one or more of a number of processes which will have a significant effect in making the end product free of animal pathogens.

In the case of meat-flavoured products (e.g.instant noodles from Asia) the meat component is usually limited to extracts which may be prepared from muscle tissue, plasma or other tissues. The methods of preparation are various. The actual amount of meat in any of these products is unspecified but is likely to be very small. Most of these products come in pack sizes of around 100 g. This is relevant, as one factor influencing the risk of an exotic disease being introduced is the dose of pathogen contained in the vehicle. For example, some of the experimental studies on the survival of foot and mouth disease virus in heated tissue suspensions required that calves be injected with 50 ml of a 10% suspension to demonstrate residual infectivity. Even if meat extract-containing noodle soup contained residual FMD virus, the virus would have to be present in a dose sufficient to establish infection by the oral route.

Canned meat and poultry products are also among those which importers wish to bring in but will be considered only superficially in this review, as MAF has recently adopted a policy of free access for canned products which do not require refrigeration.

3. Meat as a vehicle for livestock diseases

Meat and meat products have the potential to serve as vehicles for a number of serious diseases of livestock. This review examines the potential for imported meats to introduce livestock diseases into New Zealand. It is concerned only with pathogens of livestock and does not address the possibility that such products could serve to carry agents harmful to human health. MAF's legal authority is restricted to the zoo-sanitary aspects of importation. MAF has no authority to restrict products because a perceived human health risk and so pathogens and residues which are solely of human health concern are not able to be considered in this review.

For meat or meat product to serve as vehicles for the introduction of animal disease, a number of criteria must be met. These criteria are;

(a) The disease must be present in the country of origin.

(b) The disease must be present in the particular animal slaughtered (or the carcass must have become contaminated during the butchering process).

(c) The pathogen must be present in the edible tissues.

(d) The diseased meat must pass inspection procedures.

(e) The pathogen in the meat must survive storage and processing and be present at an infectious dose.

(f) The pathogen must be able to establish infection by the oral route.

(g) Scraps of the meat product must find their way into a susceptible animal of the appropriate species in New Zealand.

The likelihood of each of these criteria being met will be different for different pathogens, different meats and different countries of origin. However, taken together, the probability that each criterion will be met for any one disease must be considered remote.

One further criterion must be fulfilled before an exotic disease poses a risk to New Zealand's livestock industry. This is;

(g) Should the agent establish infection in a susceptible

host in New Zealand, local conditions must be such that the disease could spread and become endemic.

This latter criterion is particularly apt for those diseases whose transmission is dependent on arthropod vectors which are not present in New Zealand.

3.1 Safeguards against introducing diseases in meat

By examining the risks which different types of meat and meat product pose as potential vehicles for disease, it is possible, as shown below, to formulate safeguards which will permit the importation of products while still preventing the introduction of disease. However, many of the safeguards discussed below require the presence in the exporting country of a veterinary and zoosanitary infrastructure capable of carrying out the safeguards demanded and providing certification which can be relied on. The New Zealand livestock industries must be assured that product imported into this country meets the safeguards necessary to avoid the introduction of unwanted diseases.

An important safeguard against introducing many of the exotic virus diseases is the ultimate pH of the meat. So long as meat is matured to a pH below 6.0, the more important viruses will have been inactivated, even if present in the carcass. However, the ultimate pH of meat is affected significantly by the physiological condition of the animal prior to slaughter.[2] If the animal has been stressed prior to slaughter, ultimate pH may be greater than 6.0. Adequate resting prior to slaughter is important in ensuring that muscle pH falls below 6.0. So, slaughtering practices in the exporting country are an important part of the overall system of safeguards against the introduction of livestock diseases in meat.

As is the case when importations of livestock or germ plasm are proposed from countries whose veterinary and zoosanitary infrastructure is unfamiliar to staff of MAF, it is essential that veterinarians from MAF assess the veterinary and inspection services there. Throughout this review it is assumed that before importation is permitted from countries outside the range of our traditional sources of meat products such an assessment will be carried out. Such an assessment would also cover slaughtering practices. In many instances the assessment will require a visit to the country by a MAF specialist. However, in some cases, acceptance by other major trading partners, once verified officially, may be considered

sufficient. For example, if the Australian authorities were to consider that the veterinary and zoosanitary infrastructure of a particular country was sufficiently developed for importation into Australia to be acceptable, then New Zealand MAF should be prepared to adopt a similar stance. Similarly, European and United States' assessments of the exporting country's infrastructure should be taken into account.

3.2 Audit of safeguards

MAF has no experience with certification from many of the countries from which importers are interested in obtaining specialised meat products. Some spokespersons for New Zealand's livestock industries have questioned whether certification from non-traditional source countries is reliable. Such concerns are shared by veterinarians in MAF's import/export section.

Until experience has provided solid assurances as to the reliability of certification from non-traditional source countries some form of product auditing may need to be developed to ensure that imported meat products have, indeed, been subjected to the safeguards prescribed in the schedule of import conditions and attested to on the accompanying certification. This will require the development of a strategy for sampling imports. It will also require the formulation of a testing regime to which the samples can be subjected.

4. Poultry products

Poultry meat poses a different set of risks to those posed by mammalian meat products.

There is theoretically a more immediate risk that avian diseases could be introduced via infected scraps because the probability that scraps will be scavenged by birds is higher than the probability of scraps finding their way into the livestock food chain. It is the mobility of scavenging birds which heightens the risk from poultry meat products.

Another reason that poultry carcasses may pose greater risks than mammalian meat is that they usually retain the skin and kidneys, and frequently contain scraps of respiratory organs. These tissues may harbour pathogens not found in muscle.

5. Diseases considered

As a starting point in a review such as this it is necessary to decide which diseases of livestock need to be taken into account. The diseases which were given preliminary consideration are shown in Tables 3 to 6. The diseases were the *Office International des Epizooties* (OIE) List A and List B diseases, the Food and Agricultural Organisation of the United Nations (FAO) List C diseases, and a miscellaneous list of "other" diseases included because they are exotic to New Zealand or were considered to warrant discussion.

TABLE 3: DISEASES ON OIE LIST A

Foot and mouth disease
Vesicular stomatitis
Swine vesicular disease
Rinderpest
Peste de petits ruminants
Contagious bovine pleuropneumonia
Lumpy skin disease
Rift Valley fever
Bluetongue
Sheep pox and goat pox
African horse sickness
African swine fever
Hog cholera (classical swine fever)
Teschen disease
Fowl plague
Newcastle disease

TABLE 4: DISEASES ON OIE LIST B

Multiple species diseases
Anthrax
Aujeszky's disease
Echinococcosis/hydatidosis
Heartwater
Leptospirosis
Paratuberculosis
Q fever
Rabies
Screw worm

Cattle diseases
Anaplasmosis
Babesiosis
Bovine brucellosis (*B. abortus*)
Bovine genital campylobacteriosis
Bovine malignant catarrh
Bovine spongiform encephalopathy
Bovine tuberculosis (*Mycobacterium bovis*)
Cysticercosis (*C. bovis*)
Dermatophilosis
Enzootic bovine leucosis
Haemorrhagic septicaemia
Infectious bovine rhinotracheitis
Theileriasis
Trypanosomiasis

Sheep and goat diseases
Brucella ovis infection
Caprine and ovine brucellosis (*B. melitensis*)
Caprine arthritis/encephalitis
Contagious agalactia
Contagious caprine pleuropneumonia
Enzootic abortion of ewes
Pulmonary adenomatosis
Maedi-visna
Nairobi sheep disease
Salmonellosis (*S. abortus ovis*)
Scrapie

Horse diseases
Contagious equine metritis
Dourine
Epizootic lymphangitis
Equine encephalomyelitis (Borna disease)
Equine infectious anaemia
Equine influenza (virus type A)
Equine piroplasmosis (babesiosis)
Equine rhinopneumonitis

Pig diseases
Atrophic rhinitis
Cysticercosis (*C. cellulosae*)
Porcine brucellosis (*B. suis*)
Transmissible gastroenteritis of pigs
Trichinellosis

Poultry diseases
Avian infectious bronchitis
Avian infectious laryngotracheitis
Avian tuberculosis
Duck virus hepatitis
Duck virus enteritis
Fowl cholera
Fowl pox
Fowl typhoid (*S. gallinarum*)
Infectious bursal disease
Marek's disease
Mycoplasmosis (*M. gallisepticum*)
Psittacosis and ornithosis
Pullorum disease (*S. pullorum*)

Lagomorph diseases
Myxomatosis
Tularaemia
Viral haemorrhagic disease

Glanders
Horse pox
Infectious arteritis of horses
Japanese encephalitis
Mange
Salmonellosis (*S. abortus equi*)
Surra (*T. evansi*)
Venezuelan equine encephalomyelitis

TABLE 5: DISEASES ON FAO LIST C

Multiple species diseases
Listeriosis
Toxoplasmosis
Melioidosis
Blackleg
Botulism
Other clostridial infections
Other pasteurelloses
Actinomycosis
Intestinal Salmonella infections
Coccidiosis
Distomatosis (liver fluke)
Filariasis

Cattle diseases
Mucosal disease/bovine virus diarrhoea
Vibrionic dysentery
Warble infestation

Sheep and goat diseases
Contagious pustular dermatitis
Footrot
Contagious ophthalmia
Enterotoxaemia
Caseous lymphadenitis

Horse diseases
Equine coital exanthema
Ulcerative lymphangitis
Strangles

Pig diseases
Swine erysipelas

Poultry diseases
Infectious coryza
Avian encephalomyelitis
Avian spirochaetosis
Avian salmonellosis (excluding fowl typhoid and pullorum disease)
Avian leucosis

TABLE 6: OTHER DISEASES OR PATHOGENS

Vesicular exanthema of swine
Yersiniosis (*Yersinia pseudotuberculosis, Y. enterocolitica)*
Campylobacteriosis (*Campylobacter jejuni, C. coli*)
Sarcocystis species
Porcine epidemic diarrhoea virus
Egg drop syndrome '76
Goose parvovirus hepatitis
Duck astrovirus
Turkey rhinotracheitis
Chicken anaemia agent
Avian nephritis virus
Big liver and spleen disease

The lists of diseases under consideration can immediately be reduced by dropping those for which there is clearly no risk of there being introduced in carcasses, meat, offals or meat products. Reasons for dropping pathogens from the list include;

(a) The pathogen is not present in edible tissues.

(b) Susceptible species are not carnivorous or omnivorous.

(c) Infection does not occur by the oral route.

(d) Transmission of infection requires an arthropod vector.

Having gone through this preliminary screening process, the reviewer is left with the reduced list shown in Table 7.

TABLE 7: DISEASES OF LIVESTOCK WHICH MAY POSSIBLY BE CARRIED IN CARCASSES, MEAT, OFFALS OR MEAT PRODUCTS.

Foot and mouth disease
Swine vesicular disease
Rinderpest and peste de petits ruminants
African swine fever
Hog cholera (classical swine fever)
Anthrax
Aujeszky's disease
Echinococcosis/hydatidosis
Leptospirosis
Q fever
Rabies
Paratuberculosis
Brucellosis (*Brucella abortus, B. melitensis, B. suis*)
Tuberculosis (*Mycobacterium bovis* and *M. avium*)
Cysticercosis (*Cysticercus bovis, C. cellulosae*)
Scrapie and bovine spongiform encephalopathy
African horse sickness
Glanders
Melioidosis
Vesicular exanthema of swine
Atrophic rhinitis
Transmissible gastroenteritis and porcine epidemic diarrhoea virus
Trichinellosis
Listeriosis
Toxoplasmosis
Botulism
Blackleg and other clostridial infections
Salmonella infections
Mucosal disease/bovine virus diarrhoea
Erysipelas
Yersiniosis
Campylobacteriosis
Sarcocystis species
Tularaemia
Viral haemorrhagic disease of rabbits
Fowl plague
Newcastle disease
Duck virus hepatitis
Duck virus enteritis
Fowl cholera
Fowl pox
Fowl typhoid (*Salmonella gallinarum*) and pullorum disease (*S. pullorum*)
Infectious bursal disease
Marek's disease
Avian leucosis
Egg drop syndrome '76
Goose parvovirus hepatitis
Duck astrovirus
Turkey rhinotracheitis
Chicken anaemia agent
Avian nephritis virus

Big liver and spleen disease

For each of the diseases in Table 7, a qualitative examination is undertaken below to assign relative risks to meats and meat products. Note that even those considered to constitute a relatively high risk are, in reality, unlikely to fulfil all the criteria necessary to introduce exotic disease into New Zealand.

Some of the diseases listed in Table 7 are already endemic in New Zealand and are not subject to any control program. It would not be reasonable, therefore, to exclude any product because it could conceivably serve as a vehicle for such a disease. It is not reasonable to demand that an imported product meet more stringent zoo-sanitary standards than a locally-produced product.

6. Diseases which could be introduced through carriage of pathogens in meat

Mammalian diseases are examined first, starting with the most serious (OIE List A) and then those of lesser importance (OIE List B, FAO List C, others). Avian diseases are then examined in a similar order.

6.1 Foot and mouth disease

6.1.1 The disease

Foot and mouth disease (FMD) is one of the most contagious diseases of domestic animals. It is an acute viral (picornavirus) disease which affects a wide variety of domesticated and wild cloven-hoofed animals including cattle, buffaloes, sheep, goats, llamas, camels, swine and deer. Horses are refractory.[3] FMD is characterised by the formation of vesicles and erosions of the mouth, nose, feet and teats, but infections in sheep and goats are often subclinical.[3]

Of the diseases to be considered it is probably the one constituting the greatest threat to New Zealand's economy.

6.1.2 Effects of introduction

The clinical diagnosis of any vesicular disease in New Zealand would lead to an immediate suspension of exports of animals and animal products. This suspension would remain in place until the possibility of the disease being FMD was eliminated by laboratory tests.

If the presence of FMD was confirmed, it is highly probable that overseas markets for all New Zealand livestock, animal products and byproducts would close immediately.[4]

The duration and severity of the trade restrictions imposed, and the fate of product already in transit, would depend on the importing country.[4] The United States, for example, requires that a country be confirmed FMD free for 12 months before trade may resume.

The effect of a FMD outbreak on the New Zealand economy would be devastating.[4] Financial costs would include;

(a) loss of export earnings from most animal, animal product and byproduct exports. (In 1989 these were worth approximately $5,000 million).

(b) Direct costs of control procedures such as quarantine, slaughter and disposal.

(c) Cost of compensation paid for livestock slaughtered.

(d) Costs of storing animal products such as meat and dairy products during the period of no exports.

Should FMD establish in New Zealand's feral animal population, the likelihood of eradicating the disease would be remote. In such a case, the country would be faced with the necessity for an on-going FMD vaccination program and the continued exclusion from the major world markets for animal products.

6.1.3 World distribution

FMD is widespread. It occurs endemically in large areas of South America, Africa and Asia. It has been eradicated from, or never existed in, North America, the British Isles, Australia, Japan and New Zealand. It has been eradicated from Europe, the last outbreak having occurred in July 1988, in Italy.

6.1.4 Meat as a vehicle

In the carcasses of animals infected with FMD the virus is rapidly inactivated in skeletal and heart muscle tissue as a result of the drop in pH which usually accompanies *rigor mortis*.(Footnote) The virus may, however, survive for more

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Rigor mortis is usually, but not always, accompanied by a drop in pH. However, in animals with low muscle glycogen reserves an ultimate pH greater than 6.0 is not uncommon and is the cause of the condition known as dark cutting beef. Such high pH meat is considered desirable for the manufacture of sausages because of its high water-holding capacity.[6]

The ultimate pH of meat may differ between different muscles and between animals which are rested or stressed prior to slaughter. Preslaughter stress may result in an ultimate pH greater than 6.0.[2]

than 80 days in meat which has been frozen too soon after slaughter, i.e. before maturation has taken place.[5] The virus may also persist for long periods in blood clots, bone marrow, lymph nodes and viscera (offal) because in these tissues it is protected from the pH changes which accompany *rigor mortis*.[5]

Many outbreaks of FMD have been traced to waste food being fed to pigs.[3] The 1967 United Kingdom outbreak was attributed to the importation of virus in bone marrow of sheep carcasses from South America. After this experience the United Kingdom changed its importation requirements for fresh meat originating from areas where FMD is endemic. Since 1968 the United Kingdom has only permitted the importation of beef which originates from vaccinated cattle, has been matured for a length of time sufficient to ensure that pH has fallen below 6.0, and has been deboned. The importation of fresh pig or sheep meat from FMD-endemic areas is prohibited. The European Community implemented a similar policy in 1978. Maturing the carcass by allowing it to hang at 2°C for 24 hours prior to deboning is considered satisfactory.

6.1.5 Meat products

The drop in pH which usually accompanies *rigor mortis* ensures that FMD virus is inactivated within 48 hours in skeletal muscle at 4°C. However, in lymph nodes, clotted blood and bone marrow FMD virus remains infective for over 4 months at 4°C.[7]

Freeze drying (lyophilisation) of media containing FMD virus will preserve the virus for long periods. Even in products dried at relatively high temperatures the virus may remain active for a significant period. For instance, when dried at 37°C FMD virus in proteinaceous fluid may remain infective up to 112 days when stored at 18°C.[8]

Cooking may inactivate FMD virus in meat and meat products but the conditions under which the cooking takes place can influence the extent to which the virus is inactivated. Minced beef containing FMD virus-infected lymph nodes can be rendered free of virus by retort cooking to an internal temperature of 68.3°C. The same cooking process will not inactivate the virus if an internal temperature of only

63°C is attained.[9]

Higher internal temperatures are required when cooking minced beef in nylon tubes. In this process the virus may survive an internal temperature of 72°C but is inactivated at 79.4°C.[9]

A recent study[10] has confirmed that a core temperature of 79.4°C is sufficient to inactivate FMD virus in minced beef products, so long as they do not contain virus-positive milk or heart muscle. In such products FMD virus may survive 79.4°C and a core temperature of 93°C is required to ensure inactivation of the virus.

FMD virus in blood may survive 55°C for 15 minutes but is inactivated by 55°C for 20 minutes and 60°C for 2 minutes.[8] However, lack of moisture increases the resistance of FMD virus to heat. The virus may remain active in dried tissue products[8] after;

2.5 hours at 70°C
5 minutes at 110°C
3 minutes at 120°C
1 minute at 130°C.

It is clearly important to know whether heating of the infected medium took place before or after desiccation. However, it is probably reasonable to assume that tissue extracts are desiccated after they have been cooked. The cooking of the extract or broth before desiccation must be examined to assess the risks.

In moist tissue products such as broths, soups, extracts etc it is reasonable to work on the basis of FMD virus being destroyed by;

80-100°C for a `short time' or
70°C for 25 minutes.

A `short time' would be 2 to 3 minutes.

High salt concentration *per se* does not inactivate FMD virus in meat products. In products in which the meat has matured before being salt cured the virus does not survive in muscle tissues (because of the pH changes) but it may persist in lymph nodes, large blood clots, bone fragments, fat etc. The mincing and mixing processes used in the manufacture of sausages and similar products reduce the chances of FMD virus survival. Any further heating will

have an additional inactivating effect on virus.[11] However, FMD virus can survive for prolonged periods in high salt concentrations such as those used to preserve sausage casings or hides.[11]

As already mentioned, FMD virus is readily inactivated by lowered pH. At pH 6.0 FMD virus is inactivated at a rate of 90% per minute. At pH 5.0 the rate of inactivation is 90% per second.[12]

Bohm[13] demonstrated that infected intestines could be freed of FMD virus by washing in 0.5% citric or lactic acid for 5 minutes. On the basis of this work, the European Community recommends that sausage casings be treated with 0.5% to 2.0% lactic or citric acid, or a citric acid buffer system (pH 5.3), for 8 to 10 hours.[14]

There is a demand in New Zealand for Italian Parma hams. FMD virus can survive for up to 190 days in salted bacon and up to 89 days in ham fat.[11,15] However, not all types of ham should be regarded as of equal risk. The name "Prosciutto di Parma" (Parma ham) is reserved exclusively for ham having characteristic qualities related to the geographical area of its production (in Italy) and the various phases of preparation from salting to the end of the curing period. The curing period, which begins with the salting, must not be less than 12 months.[15]

In an extensive series of collaborative experiments carried out by workers in the United States and Italy it was demonstrated that the curing process used in the manufacture of Parma hams inactivates FMD virus well within the minimum curing period.[15] In no case was FMD virus recovered from any sample beyond 108 days of curing. Therefore, ham which qualifies for the denomination "Prosciutto di Parma" can be regarded as constituting no risk so far as FMD is concerned.

Another type of product for which there is a demand is salami (e.g. from Hungary and Italy). The Italian production of salami takes place mainly in a few large factories manufacturing to international standards. These plants process selected and controlled raw material under strict and well-defined procedures to ensure a consistent quality product.[16]

Recently, a study was made of the possible persistence of FMD virus in Milano (fine particle size, 3.5-4 mm) and Varzi (coarse particle size, 12 mm) salamis.[16] Meat and fat

from swine slaughtered during the viraemic phase of FMD was held for 72 hours before mincing and mixing with a *Lactobacillus* starter culture and being stuffed into a natural pig casing. Salamis of different sizes were prepared. After a drying and maturing stage the salamis were evaluated by an expert panel to confirm that they were true to type.

The pH of the various salamis ranged from 4.94 to 5.80. Virus isolation was attempted using *in vitro* and *in vivo* methods. In no instance was FMD virus detected in the matured salamis. Even more significant, in no instance was the virus isolated from the minced meat mixture prior to stuffing the sausage casings.[16]

It was clear from this study that FMD virus undergoes rapid inactivation in pigmeat processed for salami manufacture. Inactivation is completed within a week at most and inactivation occurs at the same rate in muscle tissue and fat. It is not affected by particle size nor size of the salamis.[16] Salamis are not likely to serve as vehicles for the spread of FMD virus.

6.1.6 The risk of introduction

Even though the pH changes associated with *rigor mortis* will inactivate the virus in muscle, FMD virus may persist in bones and lymph nodes of infected animals. Realistically though, the only way FMD virus in meat could enter New Zealand livestock is if the infected meat were fed to pigs. In examining this risk, two factors (apart from those discussed above) need to be taken into account. These are;

(a) The concentration (titre) of FMD virus likely to be present in the meat or meat product.

(b) The dose of FMD virus required to infect pigs by the oral route.

FMD virus may be present in the edible tissues of living animals from the time of onset of viraemia until an immune response to the infection has developed.[11] Viraemia usually begins about one day before, or coincident with, the first appearance of vesicular lesions. Immunity is usually well developed by 7 to 10 days from the start of clinical disease. During the early acute phase of disease the titres of virus will be at their greatest. The highest titres are

likely to be found in heart muscle, lymph nodes and glands. For example, Burrows and coworkers[17] found peak titres of $10^{10.0}$ ID_{50}/g in heart muscle, $10^{10.6}$ ID_{50}/g in adrenal tissue and $10^{8.2}$ ID_{50}/g in retropharyngeal lymph nodes.

Peak titres of virus in the blood and liver of infected cattle have been reported as $10^{5.6}$ ID_{50}/ml and $10^{3.6}$ ID_{50}/g respectively.[11] These peaks were recorded on the second day after inoculation. By the fourth day the titre in both tissues had dropped to $10^{1.5}$ ID_{50}/ml or g. The concentration in bone marrow declined to $10^{1.0}$ ID_{50}/g by the sixth day after inoculation.

In the infected pigs the peak concentrations recorded were $10^{7.2}$ ID_{50}/ml of blood and $10^{5.6}$ ID_{50}/g of liver.[18]

A relatively high dose of FMD virus is required to infect animals by the oral route. The minimum dose required to infect pigs by ingestion has been reported as $10^{5.0}$ ID_{50} although a lower dose ($10^{3.9}$ ID_{50}) of a pig adapted strain has been reported as establishing infection following instillation into the mouth.[18]

For a pig to receive $10^{5.0}$ ID_{50} of FMD virus it would need only consume 222 mg of pig liver containing $10^{5.6}$ ID_{50}/g. On the other hand, it would need to consume 100 g of bone marrow containing $10^{4.0}$ ID_{50}/g or 1 kg of liver containing $10^{3.0}$ ID_{50}/g. However, for tissues containing $10^{1.0}$ ID_{50}/g or less, the amount to be eaten would need to be greater than the daily intake.[18] Even so, caution is needed before dismissing the risks posed by such tissues. It may be that there are circumstances where an animal could be infected by doses smaller than those suggested above, perhaps through virus being sniffed rather than ingested.[19] Given the serious effects which would result from an introduction of FMD, precautions should take into account the unusually susceptible host, unusually invasive or pathogenic strains of the virus and environmental conditions unusually favourable to virus survival.[19]

During the early acute phase of clinical disease the virus content of an animal's tissues is likely to be sufficiently high for even small scraps to contain enough to infect pigs fed on them. However, once immunity develops (seven to ten days later) the titres of virus are likely to be very low or undetectable in edible tissues.

Importation of boneless chilled or frozen beef, even from countries in which FMD is present, could be permitted safely, so long as certain conditions could be met. These would be;

(a) Official certification that the animals were free from signs of FMD at the time of slaughter.

(b) Official certification that the animals came from areas or regions free of FMD.

Britain has imported boneless beef from FMD-endemic countries (e.g. Argentina) for many years without introducing FMD. In 1978 the European Community (EC) implemented a similar policy for the importation of boneless beef from countries where FMD is present. The safe importation of beef is thus possible.

(Comment: There is no evidence that boneless beef has ever been the origin of a FMD outbreak. Thirty four primary outbreaks occurred in the EC during the period 1977 to 1987. Eight of these originated from outside the Community and were probably due to imports of meat which had not been deboned. Thirteen of the outbreaks were most probably due to faulty FMD vaccines or laboratory escapes and 13 remain of unknown origin.[14])

As an additional safeguard, checks on the pH of boneless beef should be required.[19] Such testing is simple to carry out and, indeed, is often part of the quality control measures implemented by companies packing boneless cuts. Certification that meat had reached pH 6.0 before freezing would provide good assurances that it was free of FMD virus.

Cooked meat products and meat flavourings pose no FMD risk so long as there are adequate assurances that they have been heat treated to 80-100^{o}C for 2 to 3 minutes or 60-70^{o}C for 25 minutes.

Speciality cured products such as salami can be considered safe, as can Parma hams. Other cured meats such as hams and bacon cannot necessarily be considered free of FMD unless additional safeguards are imposed. Assurances that the product had been subjected to pH or temperature/time conditions suitable to inactivate FMD virus would be required.

Most canning processes for low-acid food (pH above 4.5) involve heat treatment for 3 or more minutes at 121°C. Such canned meats should be considered safe with respect to FMD.

6.1.7 Recommendations for foot and mouth disease

For countries or areas which have not been free from FMD for at least the preceding 12 months, restrictions must apply to meat of all animals which are capable of being infected naturally with FMD virus. Such animals include cattle, sheep, goats, deer, pigs, buffaloes and game animals.

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection(Footnote) and

(b) Chilled or frozen meat;

(i) Only beef may be imported as chilled or frozen meat and

(ii) Must originate from cattle which have been adequately vaccinated against FMD and

(iii) Must be carefully boned out and

(iv) Must have reached pH 6.0 before freezing.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100°C or higher for 2 to 3 minutes or 70°C or higher for 25 minutes.

(d) Dried meat products;

Throughout this review it is assumed that the before importation is permitted from countries outside the range of our traditional sources of meat products a MAF assessment of the veterinary and inspection services will be carried out. See section 3.1.

(i) Must be certified as containing only beef and

(ii) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be certified as containing only beef and

(ii) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

(g) Meat products from wild game animals;

(i) Clearly, the recommendations in (a) above cannot be applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

6.2 Swine vesicular disease

6.2.1 The disease

Swine vesicular disease (SVD) is a contagious viral (picornavirus) disease of swine which is indistinguishable in the field from foot and mouth disease, vesicular stomatitis and vesicular exanthema of swine.

Swine and humans are the only species affected by SVD. Sheep may be experimentally infected, without developing any signs of disease.[5]

6.2.2 Effects of introduction

Should the disease enter New Zealand's susceptible pig population, incidences of 80-90% could be expected.[5] All exports of livestock and livestock products would cease until laboratory confirmation was obtained that the outbreak was not FMD.[4] Once FMD was ruled out, the effects of an SVD outbreak would probably be felt mainly by the pig farming sector. The small export market for pigmeat would be lost for at least 6 months after the disease was stamped out.[5] Because SVD virus may infect sheep[5], some markets for sheep and sheepmeat could be affected.

6.2.3 World distribution

SVD was first recorded in Italy in 1966. Since then outbreaks have been reported in Great Britain, Austria, Belgium, France, Greece, the Netherlands, Switzerland, the German Federal Republic, the German Democratic Republic, Malta, Japan and Hong Kong.[5,20] It almost certainly occurs in China.

Africa, the Americas, Australia (and New Zealand) have remained free of SVD.[20]

There is no evidence that SVD virus exists in any country without manifesting itself clinically.[3] However, strains of SVD which produce mild or subclinical disease *are* known.[21] For this reason, a country which has reported SVD should be required to carry out a serological survey to demonstrate that its pigs are free of SVD virus before it is recognised as being free from the disease and infection.[14]

6.2.4 Meat as a vehicle

All tissues of infected pigs contain the SVD virus and can serve as vehicles for transmission of infection.[5,20]

International transport of infected meat is probably the main route by which SVD virus spread around the world in the 1960s.[3,5]

6.2.5 Meat products

The SVD virus is remarkably stable over a wide pH range (pH 2.0 to 12.0) and is able to survive almost indefinitely in refrigerated or frozen pig meat.[3,5,20] The virus has been shown to persist in the muscle of frozen pig carcasses for at least 11 months.[7]

McKercher and coworkers[22] studied the persistence of the virus in a number of processed pigmeat products. In lactic acid cured smoked salami and pepperoni sausages SVD virus was still detectable after 400 days.

SVD virus has been shown to persist for at least 780 days in salted intestinal casings, but is inactivated after 24 hours in 0.5% citric acid.[7]

"Prosciutto di Parma" (Parma ham) is a speciality product in which the curing period, which begins with the salting, must not be less than 12 months. Even though the preparation of Parma hams does not involve heat treatment, a joint American and Italian study demonstrated that SVD virus is unable to survive the curing period for these products.[15]

The situation is different for hams in which the curing period is shorter than that of "Prosciutto di Parma". The virus in hams is inactivated so long as they are heat treated to an internal temperature of 70^{o}C.[3,15] Lower temperatures may also be satisfactory for inactivating SVD virus in prepared meat products. The virus in the Italian meat product `mortadelle' was inactivated when they were heated to 84^{o}C. However, the internal temperature of the mortadelle only reached 60^{o}C, but this was sufficient to destroy the SVD virus.[23]

I have not been able to locate studies of the inactivation

of SVD virus in broths, soups, extracts etc. However, Herniman and others[24] demonstrated that SVD virus suspended in milk was inactivated in 30 minutes at 56°C and in 2 minutes at 60°C.

6.2.6 The risk of introduction

Tissues of infected pigs contain large amounts of SVD virus before clinical signs are apparent and, due to the mild nature of disease in some cases, it may not be recognised and reported promptly. Contaminated pigmeat may thus find its way into the food chain.[20] However, the time of slaughter of the pigs is critical.[22] The titres of virus in tissues are highest 2-3 days after inoculation and drop rapidly as antibodies develop.[5] The amount of SVD virus in pork products would be very small unless they were prepared from a herd early in an epidemic.

Approximately 7×10^5 ID_{50} of SVD virus is required to produce clinical infection in swine.[22] At their highest, titres of virus in meat are around 10^3 - $10^{4.5}$ ID_{50}/g. A pig would, therefore, need to be fed between 22 and 700 g of infected meat.[22] While one can accept that swine might receive scraps of imported pigmeat products, it is unlikely that quantities approaching 700 g would be fed. However, as pointed out for FMD (section 6.1.6), safeguards need to cover the possibility that, under certain unusual conditions, animals could be infected with doses of virus smaller than those studied under experimental conditions.[19]

Because of the persistence of SVD virus in uncooked pork products, the importation of such products should be contemplated only in cases where there is reliable official certification of country or regional freedom from SVD.

Product heated to an internal temperature greater than 70°C can be considered to pose no risk as a vehicle for SVD virus[5,22,23], thus the temperatures required to ensure freedom from FMD virus would also suffice to protect against the entry of SVD.

6.2.7 Recommendations for swine vesicular disease

For countries or areas which have not been free of SVD(Footnote) for at least the preceding 12 months,

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For any country which has, at some time, experienced SVD, recognition of freedom from SVD virus should be based on a

restrictions must apply to pig meat;

(a) Chilled or frozen meat;

(i) Importation prohibited.

(b) Cooked meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater.

(c) Dried meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater before drying.

(d) Cured meat products such as salamis;

(i) Importation prohibited.

(e) Hams and bacon;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

either (ii) qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 months curing process.

or (ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or

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properly-structured serological survey.[14]

greater.

6.3 Rinderpest and peste de petits ruminants

6.3.1 The diseases

Rinderpest is an acute, highly contagious viral disease (paramyxovirus) of cattle and, secondarily, sheep, goats and all cloven-hoofed animals. Swine may become infected and spread the disease.[3,5,12]

The disease in cattle is characterised by high fever, necrotic stomatitis and gastroenteritis. Mortality in epizootics of rinderpest can be very high; 90% to 100% in naive populations and up to 30% to 50% in enzootic situations.[3,5]

Peste de petits ruminants (PPR) is a similar acute viral disease of sheep and goats caused by a paramyxovirus closely related to, but distinct from, the virus of rinderpest. Explosive outbreaks of PPR occur, with mortalities of around 90%.[3,5]

6.3.2 Effects of introduction

Very high mortalities could be expected should either rinderpest or PPR enter New Zealand. In addition to these direct costs, and the costs of control, there would be effects on overseas trade. Even though the spread of rinderpest through meat is unlikely (see below), major beef markets would probably close down.[4] Export of live ruminants would also cease.

6.3.3 World distribution

Rinderpest is present throughout Africa, the Middle East, the Indian subcontinent and Asia. PPR is restricted to Africa and the Arabian Peninsula at present.

Neither rinderpest nor PPR occur in Europe, the Americas, Australia or New Zealand.

6.3.4 Meat as a vehicle

The transmission of rinderpest is nearly always dependent on close contact between animals. However, under certain

circumstances the virus may persist in meat and infect swine fed on scraps of such meat. Pigs then spread the virus to cattle, sheep, goats and other susceptible ruminants.[5,12] Such indirect spread is, however, unusual.[3]

PPR does not spread in this way. Close contact between infected and susceptible live animals is required.[5]

6.3.5 Meat products

The rinderpest virus is not very hardy.[3] Virus in infected meat is usually inactivated rather rapidly due to the lactic acid-induced drop in pH which accompanies *rigor mortis*[5,7] (c.f. FMD virus). Muscle tissue, lymph nodes and spleen held at 5°C will usually be free of virus within 2-3 days.[3] However, meat frozen before *rigor mortis* has set-in may retain infective virus for several months.[5] Indeed, even in refrigerated meat the virus may persist for some time. The virus has been recovered from carcasses held at 4°C for 30 days and from quarters aged for 24 hours and then held at 4°C for 8 days.[7]

It is conceivable, therefore, that beef, sheepmeat, chevon, venison, buffalo meat or pork could serve as vehicles for rinderpest virus.

Rinderpest virus is relatively heat sensitive, but the sensitivity is influenced markedly by the salt concentration of the medium. Salts reduce the rate of inactivation. A small fraction of virus in tissue culture preparations may survive heating at 56°C for 50-60 minutes and 60°C for 30 minutes.[12]

In salted meat rinderpest virus may persist for several months.[5]

6.3.6 The risk of introduction

Cattle are normally infected with rinderpest by the nasopharyngeal route and, even with high doses of virus, they are unlikely to be infected the oral route.[5,19] They are unlikely to eat meat scraps anyway. Pigs, on the other hand, may be infected quite readily when fed meat scraps contaminated with rinderpest virus.[5,12] Because the signs

of rinderpest are mild in pigs the disease could be overlooked for some time, increasing the chances of infection spreading to cattle, sheep or goats.[5,12]

Despite these possibilities, the probability of introducing rinderpest in meat products is not great.[12] However, because the effects of introducing this disease would be so serious, authorities recommend that the importation of uncooked meat and meat products should be permitted only from countries which have been free from rinderpest for at least the previous 12 months.[3,5,25] Meat should be derived from unvaccinated animals only.[5]

6.3.7 Recommendations for rinderpest and peste de petits ruminants

For countries or areas which have not been free from rinderpest for at least the preceding 12 months, restrictions must apply to the meat of all animals which are capable of being infected naturally with the disease. Such animals include cattle, sheep, goats, deer, buffaloes, pigs and game animals.

(a) All meat and meat products;

(i) Must originate from animals which have not been vaccinated against rinderpest and
(ii) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Importation prohibited.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100°C or higher for 2 to 3 minutes or 70°C for 25 minutes.

(d) Dried meat products;

(i) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

(i) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

(g) Meat products from wild game animals;

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

6.4 African swine fever

6.4.1 The disease

African swine fever (ASF) is probably the most serious viral disease threatening the world's swine-producing industries.[3] It is an acute to chronic disease which affects swine only.[20] Mortalities in outbreaks of ASF may be extremely high; up to 100%.

6.4.2 Effects of introduction

ASF virus is most likely to be introduced into New Zealand in infected pig meat or meat products which are fed to pigs. Subsequent spread would occur mainly by direct contact between infected and susceptible pigs or indirectly by infected blood, excretions or tissues which contain high titres of infectious virus during the acute phase of the disease and for several weeks after.[3,5] Virus may also be spread by biting flies.[26] Airborne transmission occurs over very short distances only[27] and is limited to spread within buildings.

The probability of introduced ASF spreading and establishing would be quite high.

Very high mortalities could be expected, resulting in serious economic hardship to affected pig farmers. It is probable that pork prices would rise on the domestic market, with an increased importation of pigmeat to make up the shortfall.[4]

The small export markets for New Zealand pigmeat would be lost[4] and such restrictions would probably remain in force for at least 12 months after the outbreak was stamped out.

6.4.3 World distribution

ASF is enzootic in parts of Africa, Spain, Portugal, and the island of Sardinia (Italy). Outbreaks of the disease which have occurred in other European countries in recent years (mainland Italy 1983, Belgium 1985, Netherlands 1986) have been stamped out. The disease has never been reported in Asia, on the North American continent or in Australasia.[3,5,28,29]

6.4.4 Meat as a vehicle

The international spread of ASF has invariably been linked to the feeding to pigs of waste food containing scraps of uncooked pigmeat originating in countries where ASF is endemic.[3,5,20]

6.4.5 Meat products

ASF virus is resistant to the pH changes which accompany *rigor mortis*, and it is not inactivated by freezing and thawing. The virus thus may survive for many months in raw unprocessed frozen meat.[3,5,20] ASF virus has been recovered after 150 days from infected meat kept at 4°C, after 104 days from meat kept at -4°C and after 188 days from bone marrow stored at -4°C.

ASF virus is relatively resistant to the pH changes which occur during the lactic acid curing of certain dried and smoked meat products.[5] Uncooked products prepared by curing and smoking, such as salami, pepperoni and salchicón which contain infectious virus immediately after manufacture, may be free of virus after a four month curing period.[7,12,20] However, Wilkinson[30] of the Institute for Animal Health, Pirbright, considers the risks posed by such products to be unacceptably high and recommends that their importation be prohibited from countries not free of ASF.

Brining alone is insufficient to inactivate ASF virus in hams.[7] The virus may be recovered from brined hams after periods of up to six months.[28] However, cooked or canned hams are safe, so long as they have been heated throughout to 70°C.[7,20,31]

Although not cooked, "Prosciutto di Parma" (Parma ham) is rendered free of ASF virus by virtue of its prolonged (12 month) curing process.[15]

Although ASF virus is relatively heat stable, it is inactivated in liquid media by heating at 60°C for 30 minutes.[5,20] Such heat treatment could be sufficient to render safe soups, broths and meat extracts, however such a heat treatment would have no margin of error and would be

very risky.[30]

6.4.6 The risk of introduction

All cooked meat products and certain uncooked products such as Parma hams could be imported safely from countries where ASF is endemic.

However, some authorities advise that the importation of uncooked pigmeat products should be prohibited unless the source country has been free of ASF for at least 12 months.[3,5,25,30]

Because persistent asymptomatic infections are possible in some pigs in endemic areas, it is not sufficient safeguard to require only that meat come from pigs free from signs of ASF at the time of slaughter.[30]

6.4.7 Recommendations for African swine fever

For countries or areas which have not been free from ASF for at least the preceding 12 months, restrictions must apply to all pigmeat products.

(a) All pigmeat and pigmeat products;

  (i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

  (i) Importation prohibited.

(c) Cooked meat products;

  (i) Must be free of bone and

  (ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or higher for 25 minutes.

(d) Dried meat products;

  (i) Must have been subjected to heat treatment

resulting in a core temperature of 70°C or greater for 25 minutes <u>before</u> drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Importation prohibited.

(f) Hams and bacon;

<u>either</u> (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

<u>or</u> (ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

(g) Meat from wild pigs (game meat);

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone <u>and</u>

(ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

6.5 Hog cholera (classical swine fever)

6.5.1 The disease

Hog cholera (HC) is a highly infectious viral disease of pigs characterised by rapid spread, septicaemia, haemorrhage and high mortality. Outbreaks of HC in susceptible populations often result in morbidity and mortality approaching 100%.[4,28] Worldwide, HC is, economically, probably the most important contagious disease of swine.[3,4]

Pigs are the only species affected by HC.[3]

6.5.2 Effects of introduction

On two occasions (1930, 1953) HC has been introduced into New Zealand through the feeding to pigs of ship's garbage. Both outbreaks were contained and stamped out by whole-of-herd slaughter.

Should HC enter New Zealand again, very high mortalities (90-100%) could be expected, because of the fully-susceptible status of New Zealand herds.[5,12] If the disease was not contained in the primary outbreak, the economic effects on the local pig farming industry could be devastating.

Once established, HC would be difficult to eradicate because of a number of factors including persistent subclinically infected carrier animals and maintenance of infection in feral pigs, etc.[5,12]

Little pigmeat is exported from New Zealand and so, apart from some loss of export to Pacific Island nations, the international trade effects of a HC outbreak may not be very great.[4]

Some individual farmers would be seriously affected by the disease. The costs of eradication or control would also have to be borne, either by the taxpayer or the industry itself.

HC would not infect or affect species of livestock other than pigs.[28]

6.5.3 World distribution

HC has occurred in most countries, including two outbreaks in New Zealand.[4,5] In recent years there have been widespread epizootics of HC in Western Europe and in some European countries (eg Italy) the disease is enzootic.

HC is enzootic throughout most countries of Latin America, Asia, and probably Africa.[29]

North America, Australia, the United Kingdom and a number of other countries have eradicated HC and can be considered free of the disease.[29]

6.5.4 Meat as a vehicle

Outbreaks of HC have often been traced to the feeding of garbage from ships or aircraft. About 60% of HC outbreaks in territories previously free of HC have been attributable to importation of contaminated meat products which find their way to pigs via the practice of garbage feeding.[5,12]

Wood and colleagues[32] studied the titres of virus in tissues of pigs experimentally inoculated with HC and slaughtered between 7 and 25 days after infection. Pigs were infected by intranasal inoculation with $10^{6.5}$ $TCID_{50}$/pig. HC virus titres in muscle ranged from $10^{3.4}$ to $10^{4.9}$ $TCID_{50}$/g and titres in lymph nodes ranged from $10^{5.0}$ to $10^{7.5}$ $TCID_{50}$/g. Similar titres were recorded in a wide range of other tissues. It is easy to appreciate that only a few grams of such tissues would be required to infect pigs fed on them.

6.5.5 Meat products

Pig meat, offal and by-products are potential vehicles for the introduction of HC virus. The HC virus is relatively stable and can survive 50°C for 3 days, 37°C for 7 to 15 days and -70°C for years. After lyophilisation (freeze-drying) it can remain viable for years at 6°C.[5,12] It can survive for several years in frozen pork and for months in chilled meat and bone marrow.[25] The virus is, however, susceptible to rapid changes in temperature such as thawing and refreezing.[5]

The HC virus is relatively stable within the pH range 3.0-13.0.[5]

The incubation period of HC is two to ten days and so pigs may be slaughtered before clinical signs develop. Meat from such pigs may contain high titres of virus and the virus may persist in fresh pork, frozen pork, pickled pork and smoked ham.[12] In salted and brined meat (ham) HC virus may survive for 2 to 4 months.[25] However, HC virus does not persist as long as SVD virus in smoked, fermented meat products.[7] While some authorities claim that HC virus may be recovered from some smoked and salted products for as long as 70 to 90 days[5,25], others claim that products such as fully-cured (15 to 30 days) pepperoni, salami and partly cooked hams (internal temperature 69°C) are safe.[7,12,31]

A joint American/Italian study[15] demonstrated that HC virus disappeared from Parma hams within 189 days. As the minimum curing time for Parma hams ("Prosciutto di Parma") is 12 months, they can be considered to present no risk, so far as HC is concerned.

The effect of heat treatment on HC virus is influenced by the physical medium in which the virus is heated. While heating the virus in tissues to 80°C for 60 seconds may not inactivate it, virus in defibrinated blood (of relevance to soups, broths and extracts) is inactivated by 66°C for 60 minutes, 68°C for 45 minutes or 69°C for 30 minutes.[29]

Much of the available data are unsatisfactory and difficult to interpret.[25] In addition, actual manufacturing processes vary widely between products and countries and so a 120 day curing period should be a minimum requirement for products originating in countries which have experienced HC outbreaks in the previous 12 months.[25] Even a 120 day curing period is considered insufficient safeguard by some experts.[19]

6.5.6 The risk of introduction

The *Office International des Epizooties* (OIE) recommends that a country be regarded as free from HC when the disease has not been recorded for at least 2 years. This period shall be reduced to one year for countries in which stamping out is practised in conjunction with vaccination, or 6 months where stamping out is the sole method of control.[5]

For countries not free of HC, most authorities agree that there should be a prohibition on the importation of fresh or frozen pigmeat.[5,20]

While tinned meat is considered safe[5] there appears to be less agreement on cured meat products. However, from information presented in the previous section (6.5.5) it appears that certain smoked, lactic-cured products such as salami, Parma hams, and other products heated to an internal temperature of 70°C should be considered as presenting no risk as vehicles for HC.

6.5.7 Recommendations for hog cholera

A country may be considered free from HC when;

(i) A policy of vaccination and stamping out is practised and no case has been seen for 1 year.

or (ii) A policy of stamping out alone is practised and no case has been seen for 6 months.

For countries or areas not free from HC, restrictions must apply to all pigmeat products.

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Importation prohibited.

(c) Cooked meat products;

(i) Must be free of bone and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or higher for 25 minutes.

(d) Dried meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C or higher for 25 minutes before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Importation prohibited.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

(g) Meat from wild pigs (game meat);

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

6.6 Anthrax

6.6.1 The disease

Anthrax is an acute, infectious non-contagious bacterial disease of mammals (including humans) caused by the spore-forming bacterium *Bacillus anthracis*.

In its commonest form it is essentially a septicaemia characterised principally by a rapidly fatal course.

While all mammals are susceptible to anthrax, the degree of susceptibility varies. Common domesticated animals may be ranked in order of decreasing susceptibility as; sheep, cattle, goats, deer, buffaloes, horses, pigs, dogs and cats.[5,12,33] The course of the disease is usually rapid in herbivores, but in pigs can be as long as 14 days.[5]

Birds are generally more resistant than mammals, but ducks, geese and ostriches may be affected and carrion-eating birds may become infected and/or spread anthrax after feeding on infected tissue.[5,33,34]

Cold blood animals are relatively resistant (eg crocodiles).[5,33]

6.6.2 Effects of introduction

Anthrax has occurred in New Zealand. The last time was in 1954. However, the disease has never established here. The reasons for this lie in the ecology of *Bacillus anthracis*.

Although the spores may remain viable in the soil for decades, establishment of the disease is dependent on the existence of incubator areas.[33] These are areas where the soil is alkaline and has a high content of organic material. The incubator area must be subject to periodic heavy rainfall alternating with drought, with minimum temperatures of 15.5^{o}C, but preferably higher.[5,12,33]

Anthrax spores survive in the soil of the incubator areas. During the wet period, when temperatures are high, spores germinate and *B. anthracis* undergoes a rapid proliferative phase. When the incubator area dries out, the bacterium sporulates and the resulting large spore population becomes

available to contaminate herbage and infect grazing herbivores.

Presumably it is the lack of incubator areas which has prevented the establishment of anthrax, despite a number of introductions into New Zealand.

The establishment of anthrax in New Zealand, even if it occurred, would probably not be of great significance. Sporadic outbreaks could cause losses for individual farmers, but the effects on overseas trade would be minimal.

6.6.3 World distribution

Anthrax occurs on all continents including Australia. However, the major enzootic zones are in the tropics and subtropics. The disease is most widespread in Asia, Africa and the Indian sub-continent. However, it also occurs frequently in Latin America, the Middle East and Southern Europe. Anthrax has occurred in New Zealand (last case was 1954).

6.6.4 Meat as a vehicle

Most cases of anthrax result from animals ingesting spores present on feedstuffs. However, cases may also occur in pigs, dogs and cats following ingestion of meat from animals dying of anthrax.

In developing countries, cases of anthrax in humans may occur following the ingestion of insufficiently cooked meat derived from animals dying of anthrax.[34]

6.6.5 Meat products

It is highly unlikely that meat from animals dying from anthrax would find its way into products intended for human consumption, at least in developed countries.

However, meat meal, blood meal or bone meal intended for stock food could serve as a vehicle for introducing anthrax. A major outbreak of anthrax in the United States was attributed to the feeding of contaminated bone meal to swine, from which the disease then spread to other livestock.[33]

To ensure bone meal is not contaminated with anthrax spores, it must be heated to 150°C for at least 3 hours.[33]

6.6.6 The risk of introduction

Meat from animals dying from anthrax would be unlikely to find its way into the commercial operations producing meat products intended for export. While pigs are relatively resistant to anthrax and the disease is sometimes not acute in this species,[28] affected animals would be unlikely to pass ante-mortem and post-mortem veterinary inspection.

In the 1979 epidemic of human anthrax which occurred in the city of Sverdlovsk, USSR, and was attributed to the eating of infected meat, the meat originated on the black market and not through commercial abattoirs.[34]

Even in outbreaks of disease attributable to the ingestion of partially-cooked meat from infected animals, the proportion of people who become infected is relatively low.

Pigs, dogs and cats, the animals which one would expect to be the ones infected by contaminated meat, are all relatively resistant to anthrax.
Even if cases of anthrax were to occur as a result of the importation of meat products, they would be isolated.

Contaminated meat meal, blood meal or bone meal may serve as vehicles to infect herds of pigs. However, even if such an event occurred it is improbable that anthrax would establish itself in New Zealand because of our apparent lack of incubator areas.

In my opinion, the probabilities are low that meat products or canned products constitute a significant risk, so far as anthrax is concerned.

6.6.7 Recommendations for anthrax

(a) Meat products intended for human consumption;

It would be desirable for all meat and meat products to originate from animals which have passed veterinary ante-mortem and post-mortem inspection. However, this requirement is not feasible for game animal meat. Such meat (including kangaroo meat) must have passed

veterinary post-mortem inspection and have been processed in premises under the supervision of the veterinary authorities.

(b) Meat, blood or bone meal intended for feeding to livestock;

Importation prohibited (see 6.16.7).

(c) Meat, blood or bone meal intended for use as fertiliser;

Must have been subjected to heat treatment of 150°C for at least 3 hours.

6.7 Aujeszky's disease

6.7.1 The disease

Aujeszky's disease (AD), or pseudorabies, may affect all species of domestic livestock and many species of birds, including domestic poultry. It is a viral disease caused by porcine herpes virus 1.[5] In all species except swine AD is a rapidly fatal disease.

In swine AD causes fatal nervous disease in piglets, pneumonia with retardation in growth rate in fatteners, and abortion and reproductive failure in breeding stock.

The South Island of New Zealand is free of AD, and an eradication program in the North Island commenced in December 1989.

6.7.2 Effects of introduction

AD has been present in New Zealand since at least 1973. In the years since then there have been only about 14 recorded clinical outbreaks of disease and infection has spread to an estimated 100 herds.

AD has been a cause of serious economic loss to very few farmers in New Zealand. Neither is there a significant export market which is, or would be, adversely affected by the presence of AD.

However, the New Zealand pig farming industry, through the Pork Industry Board, has a commitment to an industry-funded AD eradication program. This commitment of resource must be protected, as a re-introduction of AD, or the introduction of more virulent viral strains, would be a serious financial blow to the industry.

6.7.3 World distribution

AD occurs or has occurred in most swine-rearing countries. Australia and Canada are free of the disease and it has been eradicated from Great Britain and Denmark.

6.7.4 Meat as a vehicle

Cases of AD in dogs, cats, farmed mink and ferrets, and wild rats have been attributed to the eating of meat from AD infected swine. Outbreaks in pigs have been attributed to their eating the carcasses of rats dying of the disease.[5,20,28] While it is possible that pigs can be infected via pork scraps[5] the lack of prominence given this possibility by most writers suggests that it is not very probable.[3,5,12,20,28]

German workers Weyhe and Benndorf (cited by Harkness[30]) conducted experiments to determine virus persistence in the tissues of pigs infected artificially with AD. In pigs with clinically inapparent infection AD virus was not detected in carcass meat after 72 hours storage at 1-2°C. However, in pigs which developed clinical manifestations of AD virus was still recoverable from carcass muscle after 30 days storage. These workers concluded that while meat from clinically inapparent cases of AD constituted little risk, carcass and offals from clinically affected pigs constitute a real hazard if fed uncooked to carnivores.

6.7.5 Meat products

Although some authorities claim that the AD virus may persist in carcasses for some weeks or months[5] others have demonstrated that survival in muscle does not exceed 11 to 36 days, depending on temperature.[20] The survival of the virus is affected by the pH and by other conditions at the time of freezing. While it may persist in deep frozen carcasses for up to 6 months[5] freezing may actually inactivate the virus under some conditions.[28] The virus survives best at a pH between 6.0 and 7.0, while the pH of properly slaughtered meat is lower than this. In meat heavily contaminated with AD virus no infectivity was recovered from muscle, bone marrow or lymph nodes after freezing to -18°C for 35 days.[20]

AD virus is relatively heat labile. Harkness[25] cited three studies by Bulgarian workers Dimitrov and Kunev on the survival of AD virus in cured pigmeat products. When ham was prepared from pig meat infected with AD virus, curing for 7 days at 7-8°C in brine containing nitrate and sugar did not reduce the titres of virus significantly. After boiling, when internal meat temperature reached 65°C, virus was isolated from bone marrow on 3 out of 5 occasions. It was shown that to inactivate the virus core temperatures of 70°C or higher must be reached. To ensure that this

internal temperature was reached, hams had to be cooked at 90-95°C for at least 120 minutes.

The same workers also studied the survival of AD virus in sausages. The heat treatment involved in the manufacture of large (5cm diameter) Bulgarian sausage ensures the inactivation of AD virus is achieved. However, the same did not apply to a smaller type of sausage made with pig intestine casing. In the latter, infectious AD virus was still recoverable. After further experiments these Bulgarians were able to recommend that an internal temperature of 60°C would ensure the inactivation of any AD virus in sausage.

6.7.6 The risk of introduction

The risk that meat could serve to introduce Aujeszky's disease is slight. Pigs are viraemic for a short time only, so the probability that meat has come from a viraemic pig is low. In addition, the pH changes associated with normal meat maturation serve to reduce the levels of any virus which may be present. Freezing also serves to inactivate the virus.

Should infected meat be imported, and should it be fed uncooked to animals, Aujeszky's disease could establish only if the meat were fed to swine. Although dogs and cats could become infected, they are dead-end hosts which die without excreting virus.

6.7.7 Recommendations for Aujeszky's disease

All pigmeat and pigmeat products must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and were free from signs of AD at the time of slaughter.

6.8 Echinococcosis/hydatidosis

6.8.1 The disease

Echinococcosis (`hydatids') is a parasitic infestation involving dogs (and some species of wild canids) as primary hosts of the tapeworms *Echinococcus granulosus* or *E. multilocularis* and sheep, goats and other herbivores as the secondary hosts of the cystic larval stages of the same tapeworms. (In Australia, wallabies and kangaroos may act as secondary hosts).

Tapeworm eggs are produced by mature adults in the intestine of the primary host (in New Zealand, this is the dog). Eggs are ingested by the herbivorous secondary host and release larvae which migrate in the body of the secondary host before forming the hydatid cyst.

The lifecycle is completed when the carnivorous primary host (dog) ingests the cystic stage in the tissues of the secondary host.

Echinococcosis is a very serious zoonosis. Humans may become infested by the cystic stages following ingestion of eggs passed in the faeces of infested dogs. (Humans do not become infested by eating cysts.)

The clinical signs of hydatids are those of space-occupying lesions in the affected organs.

6.8.2 The effects of introduction

E. granulosus used to be widespread in New Zealand, but a national eradication program over the last forty years has virtually eradicated the parasite.

Reintroduction of the infestation, at some stage in the future, could undo much of the hard work that has been put into hydatids eradication.

However, to be realistic, re-establishment of hydatids in New Zealand would be a slow process and would require the breakdown of the control measures which have been developed over the years.

Laws governing the feeding of dogs would need to be ignored

on a large scale for the disease to become widespread again.

6.8.3 World distribution

E. granulosus occurs all over the world. The Mediterranean region, the Middle East, the Indian subcontinent, Australia and the southern parts of South America are considered to be endemic areas.[5] *E. multilocularis* occurs endemically in northern regions of Asia and North America.[5]

E. granulosus has been eradicated from Iceland and eradication is imminent in New Zealand, Tasmania and Cyprus.[34]

6.8.4 Meat as a vehicle

The life cycle of *Echinococcus* is dependent on meat serving as a vehicle by which the cystic larval stage (protoscolices) are transmitted to the carnivorous primary host.

Most hydatid cysts are found in offal (liver, lungs etc) and are only rarely located in muscle tissue.

6.8.5 Meat products

The protoscolices in hydatid cysts may remain viable in carcasses for at least three weeks.[35] Freezing to -18^{o}C or -20^{o}C for at least 48 hours kills most protoscolices.[34,36]

Cooking meat adequately destroys the viability of *Echinococcus* protoscolices. The manufacturing processes used in the production of most products (mincing, drying, heating, smoking) should destroy protoscolices, ensuring that such products do not serve as vehicles for hydatids.

6.8.6 The risk of introduction

The importation of live animals constitutes a greater risk than does the importation of meat products.

Because hydatid cysts are rare in muscle tissue, the only real possibility of their entering the country in meat products would be in chilled offals. That is, the risk of introduction is negligible.

6.8.7 Recommendations for echinococcosis/hydatidosis

(i) All meat and meat products must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Offals must be frozen to -18°C or less for at least 48 hours.

6.9 Leptospirosis

6.9.1 The disease

Leptospirosis is a contagious, acute to chronic, often inapparent infection of animals and humans caused by more than 100 serovars of *Leptospira interrogans*. The most common clinical manifestations of leptospirosis include fever, abortions, jaundice and haemoglobinuria.

6.9.2 Effects of introduction

Infection with *Leptospira* is widespread and common in New Zealand.[37] Six serovars have been isolated and each has a preferred maintenance host.[37] The serovars and their hosts are listed below in Table 8.

TABLE 8: LEPTOSPIRES ISOLATED IN NEW ZEALAND

Serovar	Maintenance host	Accidental host
hardjo	cattle	humans
balcanica	possums	
pomona	pigs	humans, cattle, sheep, dogs, cats
tarassovi	pig	dogs?
copenhageni	Norway rat	humans, cattle, dogs
ballum	house mouse, ship rat, hedgehog	humans, cattle

As leptospirosis is already widespread in New Zealand, the effects of introduction of further infections would depend on the serovars introduced. There are a large number of serovars overseas, many of which could cause serious disease in New Zealand livestock and against which currently available vaccines would provide inadequate protection.

However, while individual herds or flocks could be seriously affected by outbreaks of disease attributed to new serovars of *Leptospira*, the effects on trade would be

negligible.

6.9.3 World distribution

Leptospirosis occurs in virtually all countries. Incidence of disease and prevalence of the different serovars depend on the presence of the corresponding maintenance host species and the intensity of animal production.[5]

6.9.4 Meat as a vehicle

Leptospires are fragile organisms which are destroyed rapidly by heating, drying or extremes of pH.[5]

However, it is possible that infection may occasionally be spread to carnivores in slaughter scraps from the urinary system.[5] It has been demonstrated that leptospires in naturally infected pig kidneys may survive for at least 30 days[38] and that a high proportion of pigs' kidneys on sale in New Zealand butchers' shops may contain viable leptospires.[39]

6.9.5 Meat products

The only meat products which are likely to serve as vehicles for leptospires are kidneys. In New Zealand up to 40% of kidneys from young calves and 50% of pigs' kidneys may contain leptospires.[6,39]

6.9.6 The risk of introduction

Kidneys infected with leptospires could introduce leptospirosis in one of two ways.

Firstly, humans handling infected kidneys could become infected via skin abrasions while handling the organs.[39] Such infections are highly unlikely to result in leptospirosis being transmitted to other humans or animals and so are unlikely to lead to the establishment of new serovars in this country.

The second means by which infection could occur would be if

infected kidneys were fed raw to pigs, dogs or cats. The establishment of new serovars outside the index case would depend on that case being able to transmit the new serovar to the required maintenance host. If this did not occur the particular serovar would die out and not establish.

As an example, *Leptospira grippotyphosa* can cause serious disease in cattle, but only rarely does it infect pigs. It requires as a maintenance host the field vole, hamster or marsh mouse.[5] None of these maintenance hosts are present in New Zealand.

6.9.7 Recommendations for leptospirosis

No specific recommendations.

6.10 Q fever

6.10.1 The disease

Q fever is a rickettsial infection caused by *Coxiella burnetii*. The infection in animals is usually inapparent, but clinical disease involving fever, conjunctivitis, arthritis, mastitis, abortions and reproductive disorders is seen occasionally. Cattle, sheep and goats are the species most likely to show clinical signs of Q fever.

Humans are occasionally affected by Q fever. Symptoms include pneumonia and, sometimes, endocarditis. Mortality is rare.

The agent, *Coxiella burnetii*, is maintained in a wild-life reservoir involving especially rodents and birds. Infection is transmitted to domestic animals (particularly sheep and cattle) by ticks.[5]

Some animals which may be infected with Q fever are; ticks, lice, fleas, flies, camels, buffaloes, kangaroos, rabbits, mice, deer, hedgehogs, cattle, sheep, goats, horses, pigs, dogs, cats and poultry.[5]

6.10.2 Effects of introduction

All available evidence suggests that New Zealand is free of Q fever. This is an unusual situation, as *Coxiella burnetii* is present in most other countries.

It is probable that Q fever has been introduced in live animal importations in the past. However, the disease has failed to establish itself, possibly because of the lack of ticks in most parts of the country.

Should Q fever become established in New Zealand, its effects on livestock would probably be minimal. The infection is widespread in Australia, but the Australian attitude is that "it does not cause disease in animals ..."[40]

There would be virtually no effects on trade, should Q fever be introduced into New Zealand. However, Q fever is a zoonosis and the disease in humans can be severe.[34] Outbreaks of human Q fever occur particularly among workers

in slaughterhouses.[34,40] People working with livestock are also at risk.

6.10.3 World distribution

Q fever occurs in virtually all countries except New Zealand.

6.10.4 Meat as a vehicle

During the bacteraemic phase of the disease, *Coxiella burnetii* is carried to all organ systems. In some cattle the agent may persist for months in liver, kidney, muscles, lymph nodes etc.[5]

While slaughterhouse workers are particularly at risk from Q fever, their exposure is usually via aerosols, not meat *per se*. Even so, infection may occur through skin abrasions while handling infected organs.[34,40,41]

Humans may, occasionally, become infected by eating infected food-stuffs but this route of infection is uncommon.[5,34] I have found no reference to meat serving as a vehicle for *Coxiella burnetii* and suspect that this is because it is only milk which serves as a vehicle for oral infection, not meat.

6.10.5 Meat products

Probably because meat is not regarded as a vehicle for *Coxiella burnetii*, no mention of its destruction in meat is given in the standard texts consulted.[5,12,34,41] However, heat treatments for the destruction of the organism in milk or other moist environments are;

62.8°C for 30 minutes[40]
65°C for 15 minutes[5]
71.7°C for 15 seconds[40]
75°C for 8 seconds[5]
100°C for 7 seconds[5]

6.10.6 The risk of introduction

The risk that *Coxiella burnetii* could be introduced into New Zealand through meat products is negligible.

6.10.7 Recommendations for Q fever

No specific safeguards are warranted.

6.11 Rabies

6.11.1 The disease

Rabies is an almost invariably fatal viral (family *Rhabdoviridae*) encephalitis which may affect all warm-blooded animals although infection in birds is uncommon. It is characterised by a unique mode of transmission and a long and variable incubation period. The disease is a very important zoonosis.

6.11.2 Effects of introduction

Rabies is a disease with very major public health significance.

It is difficult to predict how the disease might behave if introduced into New Zealand. The disease is transmitted almost exclusively through the bites of rabid animals, although in bat caves the virus can be transmitted via aerosols.[3,12] In New Zealand the disease might be able to establish in urban dog and cat populations where it would be controllable, even eradicable, with vaccines.

Effects on trade would be negligible.

6.11.3 World distribution

Rabies occurs worldwide, with the exception of Australia, New Zealand, the Pacific Islands, Great Britain, Japan and most of Scandinavia.

6.11.4 Meat as a vehicle

Infection by the oral route must at least be considered for rabies because the virus has been found in a number of tissues outside the nervous system (e.g. adrenal glands, lungs, heart, kidney, intestinal wall etc[22]) and because it is possible to immunise animals by administering oral vaccines in sausages etc.[12]

However, while laboratory animals have been infected by mouth[34], human cases of rabies acquired by ingestion are not known, even when the virus has been detected in the milk of

some rabid cows.[34] The possibility, therefore, of meat serving as a vehicle for rabies cannot be completely excluded[5] but if it does occur, must be extremely rare.[12]

6.11.5 Meat products

Rabies virus is fragile and heat labile.[12] Because none of the authorities consulted consider that meat is likely to serve as a vehicle, it appears that no studies on the survival of the virus in meat have been conducted.

6.11.6 The risk of introduction

The probability that rabies could be introduced in meat or meat products must be considered to be nearly zero. There is a greater risk of rabies being introduced by ships' rats.

6.11.7 Recommendations for rabies

No specific safeguards are warranted.

6.12 Paratuberculosis

6.12.1 The disease

Paratuberculosis, or Johne's disease, is a chronic, mostly fatal infection of cattle, sheep, goats and deer caused by *Mycobacterium paratuberculosis*. Its main signs are a progressive diarrhoea (in cattle) and wasting (in all species).

Pigs can be infected experimentally with *M. paratuberculosis* and should thus be regarded as potential shedders of the organism.[5,12]

6.12.2 Effects of introduction

Johne's disease is very widespread in New Zealand. It occurs at high prevalence in the sheep and cattle populations and has also been reported in goats and deer.

No control programs are in place, nor indeed are such programs feasible with the technology presently available.

Further introductions of *M. paratuberculosis* would be of little significance, unless new strains, with different species affinities or greater virulence were introduced.

Paratuberculosis already causes significant problems when exporting live animals. However, its presence in New Zealand has no effect on other trade.

6.12.3 World distribution

Paratuberculosis occurs all over the world.

6.12.4 Meat as a vehicle

The lesions of paratuberculosis are usually confined to the intestines, regional lymph nodes and liver. They may occasionally occur in spleen, uterus, udder and male reproductive organs.[12]

Theoretically then, infected tissues such as liver or lymph nodes could find their way into the diet of swine which

could then pass infection on to other livestock.

6.12.5 Meat products

M. paratuberculosis is readily destroyed by "moderate heat".[12] I have no other relevant information.

6.12.6 The risk of introduction

The risk that new strains of *M. paratuberculosis* could be introduced in meat is very low indeed. The organism is not usually present in edible tissue, it is not a hardy organism and the animals likely to be fed scraps (i.e. swine) are not very susceptible to infection.

6.12.7 Recommendations for paratuberculosis

No specific safeguards are warranted.

6.13 Brucellosis

6.13.1 The disease

Brucellosis is a chronic infection caused by bacteria of the genus *Brucella*. Of concern are *Br. abortus*, *Br. melitensis* and *Br. suis*.

Br. abortus infects primarily cattle, but may also infect other food animal species (sheep, goats, horses, pigs) and humans.

Br. melitensis is primarily a pathogen of sheep and goats, but may also infect cattle, pigs and humans. Hares may be infected with *Br. melitensis*.

Br. suis infects pigs, but may also infect cattle, horses and humans. It is also found in wild hares in Europe, and can infect rodents.

Dogs may become infected with any of the brucella species listed. Cats are resistant to infection with brucellae and no natural cases of feline brucellosis have ever been recorded.[34]

Brucellosis in cattle, sheep, goats and swine usually causes reproductive problems, such as infertility, abortion etc. In humans, brucellosis is the cause of the debilitating undulant fever.

There is a remote possibility that scavenging birds could serve to spread brucellosis.[42] There have been a few reports of scavenging birds becoming infected with brucellae for short periods and shedding the organisms in their faeces.

6.13.2 Effects of introduction

Br. abortus has probably been eradicated from New Zealand. *Br. suis* and *Br. melitensis* have never occurred here.

When the New Zealand national brucellosis eradication scheme began in 1971, it was estimated that 5% of adult female dairy cattle and 1.5% of adult female beef cattle were infected. These animals were distributed through 36% (some 14,400) of the herds in the country. Brucellosis was

a major cause of abortions in heifers and depressed milk yields.

The reintroduction of bovine brucellosis would undo years of investment and work in brucellosis eradication. It would also adversely affect the access of New Zealand dairy products, livestock and, possibly, meat into important markets overseas.

Br. melitensis is a major cause of reproductive problems in those countries in which it is endemic. It is a more difficult disease to control than is bovine brucellosis. Its introduction into the New Zealand sheep flock would have a serious impact on the local industry and on access of products and livestock to overseas markets.

Br. suis is also more difficult to control that is *Br. abortus*. Its introduction into the New Zealand pig herd would cause significant problems for individual farmers, but would have little trade impact as there is no significant export of pigmeat from this country.

All the brucellae listed are serious zoonoses and are of particular concern to people working with livestock (farmers, veterinarians, slaughterhouse employees etc). They also constitute a threat to the consumers of unpasteurised milk and dairy products and, to a much lesser extent, of meat.

6.13.3 <u>World distribution</u>

Br. abortus is found worldwide, although it has been eradicated from a number of developed countries (Australia, Scandinavia, Great Britain). It is present in many European countries, the United States, most of South America, Asia and Africa.

Br. melitensis occurs in sheep and goat flocks, and less commonly in cattle, throughout southern Europe, the Middle East, Africa, South America etc. Australia is free.

Br. suis is enzootic in North, Central and South America, in South East Asia and occasionally in some countries of western Europe. It occurs sporadically in Australia.

It has not been reported in Great Britain or Canada.

6.13.4 Meat as a vehicle

When animals first become infected with one of the *Brucella* species there is a bacteraemia. Subsequently the organisms localise in certain tissues such as lymph nodes, liver, bone marrow etc.[34]

Human cases of brucellosis have occurred as a result of people eating raw bone marrow or raw meat of animals infected with *Br. suis*.[34] Pigs may become infected by eating the carcasses of hares infected with *Br. suis*.[5] Dogs may be infected by eating reindeer meat from animals infected with *Br. suis*.[12] It is apparent, therefore, that under certain circumstances meat could serve as a vehicle for brucellae.

6.13.5 Meat products

Brucellae are resistant to freezing so could, theoretically, survive for prolonged periods in frozen meat. Experimentally, *Br. abortus* has been shown to survive in a (guinea pig) carcass for up to 44 days when kept under cold conditions.[12]

Brucellae are resistant to pickling and smoke curing, so there is a possibility that some meat products could serve as vehicles. However, this has never been verified.[34] *Br. abortus* has been shown to survive in meat and salted meat for 65 days at 0-20^{o}C.[12]

Brucellae are quite sensitive to heat and are destroyed by cooking or pasteurisation.[5,12]

6.13.6 The risk of introduction

Lymph nodes in carcasses, or offals, could possibly act as vehicles by which brucellae could be introduced into New Zealand.

To establish infection the imported infected meat would need to be fed to either dogs or pigs. Cats are resistant to infection with brucellae[34] and ruminants do not eat meat scraps.

While it is possible, therefore, that a dog or a pig could become infected, the chances of their passing infection on to other animals are not great. Pigs can be infected with

Br. abortus, but are dead-end hosts which do not transmit the disease to other animals.[34] Dogs can be infected with *Br. abortus* or *Br. melitensis*, but transmission of infection to other animals is rare.[34] Dogs may also become infected with *Br. suis* and may, occasionally, transmit this pathogen on to swine.[12]

In all, the probability that meat or meat products could introduce brucellosis into New Zealand is remote.

6.13.7 Recommendations for brucellosis

Brucellosis should be subject to an official control program in the country of origin of the meat. The program should be appropriate to the species of meat animal and the species of *Brucella* (ie *Br. abortus* in cattle, *Br. melitensis* in sheep and goats and *Br. suis* in pigs).No other specific safeguards are warranted.

6.14 Tuberculosis

6.14.1 The disease

Tuberculosis is a chronic, infectious disease caused by *Mycobacterium bovis*, *M. tuberculosis* or *M. avium*.

Cattle are the principal hosts of *M. bovis*, but goats, swine, deer, humans, dogs, horses and cats may also become infected with this organism. Birds do not become infected.[5,12]

M. avium affects mainly birds (including poultry). Cattle, swine and deer may be infected when exposure is sufficiently great, and the infection is also seen rarely in sheep, goats, horses and cats.[5,12]

M. tuberculosis is primarily a pathogen of humans. Infections are occasionally seen in other animals such as pigs and cattle.

6.14.2 Effects of introduction

Tuberculosis is widespread in cattle herds in New Zealand. It also occurs commonly in deer, less commonly in pigs and rarely in goats and sheep. It is endemic in possums and feral swine, deer and goats in a number of areas.

Tuberculosis is subject to a compulsory control program in the cattle and deer populations.

The presence of tuberculosis in New Zealand will lead to increasing difficulties in gaining access for product and livestock into important overseas markets. The situation is already serious, regardless of any possible introduction of the disease in meat and meat products.

6.14.3 World distribution

Bovine tuberculosis is distributed throughout the world. Most European countries have eradicated the disease, and eradication is close in Australia.

Avian tuberculosis has a worldwide distribution but the disease is no longer of any significance in intensive

poultry production establishments.

6.14.4 Meat as a vehicle

Meat is an unlikely vehicle for tuberculosis, however "alimentary transmission via the intake of raw meat of tuberculosis-infected animals and its unheated products is ... possible."[5]

Swine may contract tuberculosis after being fed improperly cooked or raw offal from tuberculous cattle or poultry.[28] However, the doses required to infect by mouth are very high.[43] For example, as few as 5 bacilli may infect calves by the respiratory route, but oral infection requires several million bacilli.

6.14.5 Meat products

Tubercle bacilli are not found in the muscle of infected cattle or pigs. Although tuberculous animals may have bacteraemic episodes, these occur very infrequently and involve small numbers of bacilli only. Even in animals dying from miliary tuberculosis, the dose of bacilli present in muscle tissue is only 100-200 per gram.[43]

"... The danger to [humans] from tubercle bacilli in or on flesh is very slight indeed if animals are in reasonably good health at the time of slaughter and good hygienic procedures are observed - this is still generally true even when cattle have quite severe lesions of tuberculosis."[43]

M. bovis, *M. avium* and *M. tuberculosis* are destroyed readily by normal cooking. United States Department of Agriculture regulations consider that meat from tuberculous carcasses may be rendered safe for human consumption by cooking at 76.7°C for 30 minutes.[28] In liquid products, such as milk, pasteurisation at 71-74°C for 30 seconds or 85°C for 5-10 seconds will destroy tubercle bacilli.[5]

6.14.6 The risk of introduction

The probability of *M. bovis* or *M. tuberculosis* surviving processing and finding their way into livestock through imported meat products is remote.

6.14.7 Recommendations for tuberculosis

Bovine tuberculosis should be subject to an official control program in the country from which the meat originates. No other specific safeguards other than veterinary post-mortem inspection are necessary.

6.15 Cysticercosis *(C. bovis and C. cellulosae)*

6.15.1 The disease

Cysticercosis is the infestation with the larval stages (cysticerci) of the tapeworms *Taenia saginata* and *T. solium*.

The adult tapeworms are obligatory parasites of humans. The larval stage of *T. saginata* (*Cysticercus bovis*) occurs in muscle, mainly of cattle. *C. bovis* may occur in reindeer and, possibly, buffalo, but African wild ruminants are not hosts to this parasite.[36]

The pig is the main host for the larval stage of *T. solium* (*C. cellulosae*). However, humans may also become infested with *C. cellulosae* in situations of poor personal and environmental hygiene.[36,44]

Humans infested with *T. saginata* or *T. solium* pass gravid tapeworm segments in their faeces. If faeces are not disposed of adequately, then cattle or pigs may become infested with the respective cysticercosis.

Humans become infested by eating meat containing viable cysticerci. Human infestation with *C. cellulosae* is a very serious disease, with high mortality[36,44], owing to the location of cysts in the brain.

6.15.2 Effects of introduction

It is possible that *C. bovis* could establish in New Zealand, if introduced. In Australia, where farming conditions are often similar to those in New Zealand, *T. saginata* is said to be "widespread" in humans and *C. bovis* occurs in all states but not the Northern Territory.[45] *C. bovis* is detected at slaughter in around 0.2% of cattle[33] but it needs to be borne in mind that the sensitivity of routine meat inspection procedures ranges from 0 to 72% for *C. bovis*[46], with around 15% being most likely.[6]

C. bovis already occurs in New Zealand cattle at low prevalence. A higher prevalence could lead to increased meat inspection costs, problems with access to some markets, and reduced value of product because lightly infested carcasses must be frozen before being passed as

fit for human consumption.[46]

In those parts of the world where *C. bovis* infestation is common, the costs to the meat industry may be substantial. For example, in East Africa, where prevalence is high, the annual cost to the meat industry is estimated to be in the order of US$10 million.[44]

While *T. saginata* infestation may be a significant zoonosis, *T. solium* presents much greater dangers to humans, because humans may also become infested with the larval stage, *C. cellulosae*.[36,44] The establishment of *T. solium/C. cellulosae* in New Zealand would have little impact on trade, because New Zealand has no significant export trade in pigmeat. However, the presence of this serious zoonosis would result in increased meat inspection costs which would have to be borne by the New Zealand pig farmers.

6.15.3 World distribution

T. saginata is widely distributed throughout the world. High prevalences (greater than 10%) are recorded in some African countries, eastern Mediterranean countries and in parts of the USSR. There is a moderate prevalence in Europe (5-10%, and increasing), the Indian subcontinent, southern Asia, Japan, the Philippines and much of Latin America. Australia, Canada and the USA have low prevalences.[34,36]

The larval stage of *T. saginata* (*C. bovis*) occurs in most countries, at a prevalence related to the prevalence of human infestation. In European countries it is found in 0.3-4.0% of slaughtered cattle. High prevalences occur in Africa, with rates of 30-80% being recorded in some parts of East Africa.[36]

T. solium infestation is restricted mainly to regions of low socioeconomic development in central and southern Africa, Mexico, Central and South America and southern Asia. In Europe, *T. solium* is rare, with only sporadic cases occurring, mainly in southern Europe. It is also very rare in muslim countries.[34,36,44]

6.15.4 Meat as a vehicle

Meat is the vehicle by which the larval stage (cysticercus)

reaches the definitive host in which the tapeworm develops to complete the parasite's life cycle.

6.15.5 Meat products

Because the sensitivity of meat inspection for cysticerci is low (0-72%)[45], there is always a chance that meat originating from endemic areas could be passed as fit for human consumption yet still contain viable cysticerci. These may survive in carcasses for up to six weeks.[5]

Freezing is an effective means of destroying cysticerci and thus ensuring meat is safe. Recommended freezing regimens include;

C. bovis	-18 to -22°C for 10 days[5]
C. cellulosae	-10°C for 4 days[5]
C. bovis	-10°C for 10 days[44,45]
	-18°C for 5 days[44]

Cooking is also an effective means of killing cysticerci in meat. Recommended regimens include;

C. cellulosae	45 to 50°C for 15 to 20 minutes[5]
C. bovis and *C. cellulosae*	raising to a core temperature of 90°C[36]
C. bovis	56°C for 5 minutes[5]

Salting and smoking are not reliable methods for killing cysticerci.[5]

6.15.6 The risk of introduction

The most probable route by which *T. saginata* or *T. solium* could enter New Zealand is in the intestines of humans. Given that *T. saginata* is relatively common in many countries, including Australia, it is highly likely that it enters New Zealand frequently. The fact that its cystic larval stages are rare in New Zealand cattle indicates that sewage disposal in this country minimises the risk of

cattle becoming exposed to faeces from infested humans. However, the prevalence of *T. saginata/C. bovis* could increase in New Zealand, as it has in Europe.

T. solium is far less common worldwide than *T. saginata*. For this reason it probably enters New Zealand much less frequently. Modern pig farming practices mean that farmed pigs are less likely than cattle to come into contact with human faeces.

Compared to the risk posed by human travellers, meat poses a small risk only. The risk from chilled, uncooked meat products is mainly one of public health. While people could be at risk, New Zealand livestock are unlikely to be threatened to any significant extent.

6.15.7 Recommendations for cysticercosis

All meat must have passed veterinary post-mortem inspection.

Before permits are issued to import beef, buffalo meat or pork, an official statement on the prevalence of cysticercosis in slaughter animals should be obtained from the veterinary authorities in that country. Where prevalence of cysticercosis in slaughtered animals exceeds 5% the importation of chilled meat should not be permitted. Unprocessed meat should be frozen to -18^{o}C.

6.16 Scrapie and bovine spongiform encephalopathy

6.16.1 The diseases

Scrapie is a transmissible, progressive and invariably fatal neurological disease that occurs in most breeds of sheep. It can affect sheep of either sex, particularly those between 2½ and 4½ years of age. Scrapie also occurs in goats, especially when they have been in contact with sheep with the disease.

Scrapie manifests itself as incoordination, ataxia and pruritis. Wasting is also a feature in the later stages of the disease.

Scrapie is caused by a small infectious agent (termed a prion by some researchers), which has many of the biological properties of a virus. For example, it is filterable and exhibits strain variation and mutation. It differs from conventional virus in that;

(a) It is highly resistant to many disinfection procedures, such as heating, ultraviolet and ionizing radiation, and prolonged exposure to formalin.[5,47]

(b) It does not induce humoral or cellular immunity in an infected animal. There is, therefore, no serological test for detection of pre-clinical infection.[5,47]

Bovine spongiform encephalopathy (BSE) is a related progressive, fatal neurological disease of cattle, first described in British cattle in 1986. BSE has resulted from feeding cattle concentrates containing meat and bone meal prepared from scrapie-infected sheep offals.[48]

Scrapie and BSE are members of a group of diseases known as transmissible degenerative encephalopathies. Other diseases in the group include transmissible mink encephalopathy (resulting from feeding scrapie-infected sheep material to mink), chronic wasting disease of deer and elk, and the human diseases Creutzfeldt-Jakob disease, kuru and Gerstmann-Straussler syndrome.

6.16.2 Effects of introduction

The introduction of scrapie into New Zealand would probably

have little effect on the export trade, although export certificates for wool, skins and hides would need to be rewritten, as they state the country is free of scrapie.[4]

The effect that BSE could have on trade cannot be assessed at present. However, since the BSE epidemic in the United Kingdom attained prominence, a number of countries have introduced bans on meat and bone meal and on live cattle over six months of age. Some have also placed restrictions on the importation of British beef.

The financial losses caused by scrapie can be considerable. Losses of 10 to 20% have been recorded in some British flocks.[47]
No country has been able to conduct a successful eradication program against endemic scrapie. Control programs in countries such as Canada and the United States of America have proven to be costly and difficult to implement.

6.16.3 World distribution

Australia and New Zealand are among the few significant sheep rearing countries known to be free of scrapie. Denmark, Finland and Israel may be free.

BSE has been recorded outside the British Isles only three times at the time of writing. The disease was diagnosed in two cattle imported from the United Kingdom into the Sultanate of Oman, one cow imported from the United Kingdom into France, and two cases were confirmed in cows in Switzerland. It is believed that the Swiss cases resulted from the feeding of British meat and bone meal imported into Switzerland *via* France.

6.16.4 Meat as a vehicle

Until the advent of BSE, the transmission of the scrapie agent to other species was regarded as a rare event which had occurred only in mink fed on untreated sheep offals. The present major epizootic of BSE (or "bovine scrapie") in the United Kingdom has shown that the feeding of processed offals can also transmit scrapie infection to other species. However, the epidemiological evidence suggests strongly that the transmission from sheep to cattle occurred at an extremely low level and that recycling of infected material from cattle to cattle, *via* contaminated

feedstuffs, was the major factor responsible for the large scale of the epidemic.[49] This has sent ripples of alarm through all countries which had imported meat and bone meal or live cattle from Great Britain.

The British authorities have banned ban the inclusion of ruminant-derived protein in the diet of ruminants. In addition, there is a ban on the use for human or animal food of six specified offals, including brain and spinal cord. The specified offals are the tissues which, potentially, could have significant titres of infectivity and the ban applies to all cattle over six months of age. However, no steps have been taken to exclude sheep brains or offal from the human food chain as there is no evidence to suggest that scrapie agent is the cause of Creutzfeldt-Jakob disease of humans.

6.16.5 Meat products

The scrapie agent is extraordinarily resistant to procedures which normally inactivate pathogens.[5,47] Although any exposure to temperatures over 100°C will lead to some loss of infectivity, complete destruction of the scrapie agent was not achieved by autoclaving at 121°C for 1 hour or 126°C for 2 hours, or by dry heating at 240°C for 1 minute.[48] Autoclaving at 134-138°C for 18 minutes will inactivate the agent.[48]
Meat and bone meal heated to 150°C for at least 3 hours to render it free of anthrax spores (see 6.6.5) would also be safe so far as the scrapie/BSE agent is concerned.

It has been known for many years that the scrapie agent may occasionally be present at very low titres in muscle tissue.[50] If present in meat, the scrapie agent is likely to remain infective throughout processing and storage. However, because the canning process involves heating under pressure, and is thus tantamount to autoclaving, titres of agent would be substantially reduced in canned meat and meat products.

6.16.6 The risk of introduction

The only way that scrapie/BSE could, realistically, be expected to be introduced in meat products would be in the vehicle of meat and bone meal prepared from infected sheep and cattle and intended for incorporation into rations for ruminants.

While it is recognised that scrapie agent may be present occasionally in muscle tissue, the titres of agent are very low[50] and the dose needed to infect by the oral route is very high.[49]

Meat scraps which do not contain large lymph nodes will not have a sufficient dose of scrapie agent to infect by the oral route and they are unlikely to be eaten by a ruminant.

6.16.7 Recommendations for scrapie and BSE

(a) Meat products intended for human consumption;

No specific safeguards are warranted.

(b) Meat and/or bone meal of ruminant animal origin into for feeding to livestock;

Importation prohibited.

(c) Meat and/or bone meal intended for use as fertiliser;

Must have been heated to 150^{o}C for at least 3 hours.

6.17 African horse sickness

6.17.1 The disease

African horse sickness (AHS) is a highly fatal insect-borne, acute viral disease (reovirus) of Equidae and dogs.[5] After Venezuelan equine encephalitis, AHS is the most important viral disease capable of causing widespread mortality in horses.[3] Four clinical forms of the disease are described;

(a) An abortive form, with spontaneous recovery. Often seen in donkeys and partially immune horses.

(b) A rapidly fatal acute pulmonary form, seen in fully susceptible horses and dogs.

(c) A subacute cardiac form.

(d) A mixed syndrome, with pulmonary and cardiac signs.

Subclinical forms of the disease may occur in partially immune horses in endemic areas.

6.17.2 Effects of introduction

In a fully susceptible horse population, the effects of AHS can be devastating. Mortality up to 95% can be expected.[3]

In attempting to assess the impact of a different exotic disease (equine influenza) on the New Zealand horse industries, O'Neil[51] estimated that an outbreak of an equine disease which necessitated the cancellation of all horse gatherings for a period of four weeks would cost the industries nearly $175 million dollars. The costs of an outbreak of AHS would be considerably greater, because of the high mortality, not a feature of the hypothetical equine influenza outbreak used in O'Neil's assessment.

The presence of AHS in New Zealand would cause major disruption to the export of horses.

6.17.3 World distribution

The entire African continent is considered to be

enzootically infected with AHS.[5] In 1989 AHS epizootics occurred in Spain, Portugal and the Arabian peninsula.

The disease has not occurred in the Americas, Oceania and most of Asia. Outbreaks occurred in India and the Middle East between 1956 and 1961.

6.17.4 Meat as a vehicle

AHS is an insect-borne disease and cannot be transmitted between horses by contact. However, if meat from clinically or subclinically infected horses is fed to dogs, they may become infected and serve to infect vectors before themselves dying from the disease.[5]

6.17.5 Meat products

At pH value below 6.0, that is, at the sort of pH usually found in meat which has undergone *rigor mortis*,(Footnote) the virus of AHS is inactivated quickly. It is also inactivated by temperatures greater than 60°C.[5]

6.17.6 The risk of introduction

While it is known that dogs may be infected by eating meat from horses infected with AHS virus, the role of dogs in the epidemiology of AHS is of questionable significance.

To maintain itself in a country, AHS requires the presence of suitable insect vectors. The major vectors are *Culicoides* species[3,5] which are not found in New Zealand. However, mosquito species and other biting insects may play a role as vectors and so it cannot be ruled out that an epizootic of AHS could occur here, should the virus be introduced.

Despite this, the disease is unlikely to establish in this country. The maintenance of AHS in a country appears to depend on a population of suitable reservoir hosts

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Rigor mortis is usually, but not always, accompanied by a drop in pH. However, in animals with low muscle glycogen reserves an ultimate pH greater than 6.0 is not uncommon.

(probably zebras and elephants).[3,5] In the absence of such a reservoir population the disease is likely to die out, as it has always done following extensive epizootics in North Africa, the Middle East and the Indian subcontinent.[3]

It must be concluded that the risk of introducing AHS into New Zealand via horse meat is extremely remote.

6.17.7 Recommendations for African horse sickness

All horse meat must originate from animals which have passed veterinary ante-mortem and post-mortem inspection.

6.18 Glanders

6.18.1 The disease

Glanders is a bacterial disease of *Equidae* caused by the gram-negative bacillus *Pseudomonas mallei*. Carnivores and humans may be infected with this organism, and infection is frequently fatal.

The disease is usually characterised by ulcerating nodules in the upper respiratory tract, the lungs or the skin. In its acute form, there is a septicaemia followed by death within a few days.

P. mallei is an obligate parasite and has no reservoir outside *Equidae*. It survives outside the host for 1 to 2 months only under favourable conditions.

Dogs and cats may become infected with *P. mallei*, but swine are highly resistant to infection.[33]

6.18.2 Effects of introduction

Should glanders be introduced the effects on the horse industries could be serious. Serious epizootics of the disease have occurred when horses are brought together in large numbers. However, control and eradication of the disease is readily achieved in developed countries.

The presence of glanders in New Zealand would have a major effect on the export of horses. Most of the major export markets would close to horses from this country.

6.18.3 World distribution

Glanders has been eliminated from most developed countries. It remains endemic in much of Asia, North Africa, India and parts of southern Europe.

6.18.4 Meat as a vehicle

Glanders may occur in carnivores fed on meat from infected horses.[5,12]

6.18.5 Meat products

P. mallei does not survive well outside the live animal. It has little resistance to drying, heat or chemicals.[5,12,33] In the absence of specific information to the contrary it can be assumed that there is not a high probability that meat products could serve as vehicles for *P. mallei*.

6.18.6 The risk of introduction

Glanders is normally transmitted by direct or indirect contact between infected and susceptible equids.[5,12] Although carnivores may be infected by eating infected meat the probability of this occurring as a result of pets being fed imported horse meat must be considered remote. (Note: swine are highly resistant to infection.[34]) Even if such infection of pets should occur, it is difficult to see how the disease could then pass to horses.

Should glanders manage to establish itself in the New Zealand horse population it could be eradicated by test and removal measures.[12,33]

6.18.7 Recommendations for glanders

No specific measures are required.

6.19 Melioidosis

6.19.1 The disease

Melioidosis is a bacterial disease caused by the gram-negative bacillus *Pseudomonas pseudomallei*. Infection results in multiple abscesses in a wide variety of tissues and organs and cases are seen in cattle, sheep, goats, swine, horses, dogs and humans, among others. *P. pseudomallei* is normally an inhabitant of moist, clayey soils in tropical areas and has been described as "... an accidental pathogen that can on rare occasions find a niche in the animal body ..."[33]

6.19.2 Effects of introduction

The introduction of melioidosis into New Zealand would probably have little significant impact. It would probably remain localised[46] and cases would be sporadic. No impact on trade would be expected.

6.19.3 World distribution

Melioidosis occurs mainly in Southeast Asia and tropical Australia, with cases occurring less commonly in other tropical countries. Occasional foci of infection may be found outside these areas.[33]

6.19.4 Meat as a vehicle

The reviewer has not found specific statements that melioidosis can be transmitted by meat from infected animals. However, several authors refer to the possibility of ingestion by the oral route, but are referring mainly to the drinking of contaminated water.[33,34,46]

6.19.5 Meat products

P. pseudomallei survives better in the environment than *P. mallei*. In fact, its normal habitat is moist, clayey tropical soils. I have no specific information on its ability to survive in meat products.

6.19.6 The risk of introduction

It is improbable that melioidosis would enter New Zealand in meat, and if it did infect a carnivore, the likelihood of the disease establishing is remote. Animal to animal transmission is considered not to occur.[33,34] It is also unlikely that this bacterium, which is a soil inhabitant, would find soil conditions suitable for its survival except, perhaps, in a few localities in the far north.

6.19.7 Recommendations for melioidosis

No specific measures are required.

6.20 Vesicular exanthema of swine

6.20.1 The disease

Vesicular exanthema (VES) is an acute, febrile, contagious viral disease of swine characterised by formation of vesicles. It may, therefore, resemble foot and mouth disease.[52]

VES is caused by one or more strains of marine calicivirus which cycle naturally between certain small fishes (primary host) and marine mammals (secondary hosts).[53]

The VES virus has been isolated from several species of pinnipeds and whales. Closely related strains have been isolated from dolphins.[53]

Domestic livestock which may be infected by VES virus include dogs, swine, horses, goats and sheep. Closely related strains can affect cattle, mink and other animals.[52,53]

6.20.2 Effects of introduction

As with any vesicular disease, an outbreak of VES would immediately halt all exports of animal products until confirmation was received that it was not foot and mouth disease.[4]

6.20.3 World distribution

Outbreaks of VES have not been seen since the 1950s and, with the exceptions of small outbreaks in Iceland and Hawaii, all were confined to the United States.[52]

However, disease attributed to caliciviruses is seen commonly in marine mammals along the Pacific coast of the United States. Serological evidence also confirms that caliciviruses circulate in marine mammals in Soviet waters.[53] It is possible that marine caliciviruses are even more widespread than the available data suggest.

6.20.4 Meat as a vehicle

Meat is the main route by which the VES virus and the other closely related marine caliciviruses are transmitted to livestock. The outbreaks of VES seen in swine are believed to have resulted from the feeding of garbage containing pork scraps and marine scraps. Seal meat fed to mink has served as a vehicle for a closely related marine calicivirus.[52,53]

Meat contaminated with VES virus may remain infectious for as long as 4 weeks at 7°C.[54]

6.20.5 Meat products

Fresh, frozen, chilled and even some cooked pork products could serve as vehicles for VES virus.

VES virus has similar stability to FMD virus. It is inactivated in 60 minutes at 62°C or in 30 minutes at 64°C.[12]

6.20.6 The risk of introduction

Except for the single Icelandic outbreak, attributed to the feeding of garbage from the US airforce base at Keflavik, VES has not been seen outside the United States. In addition, no case has been recorded in over 30 years.

There is virtually no risk of VES being introduced into New Zealand in pork, because of the extreme rarity of the disease in pigs. It is also unlikely to be imported in fish meal, as the processing would destroy the virus if present in the source material.

VES is unlikely to be imported in whale meat. The importation of whale meat requires a permit from the Department of Conservation (Marine Mammals Protection Act 1978, section 4, ss 2). In the present political climate, I was told, the Department would be unlikely to issue a permit for the importation of whale meat.

The likelihood of importing VES in meat is extremely low. However, the presence of marine caliciviruses in fish, shellfish and marine mammals in at least some parts of the Pacific rim means that there is always the potential for a VES outbreak to occur locally.

Such an outbreak could conceivably occur in cases where local pigs become infected by eating, say, waste from a fish processing factory or meat from beached marine mammals.

6.20.7 Recommendations for vesicular exanthema of swine

In the unlikely event that a country could not be certified as free from VES, restrictions should be imposed on the importation of pigmeat. These restrictions should be closely similar to those required for FMD and would include;

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Must be carefully boned out and

(ii) Must have reached pH 6.0 before freezing.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100°C or higher for 2 to 3 minutes or 70°C or higher for 25 minutes.

(d) Dried meat products;

(i) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

6.21 Atrophic rhinitis

6.21.1 The disease

Progressive atrophic rhinitis of pigs is a contagious disease attributed to infection with toxigenic strains of *Pasteurella multocida*. Growth of the bacterium in the nasal cavities of infected pigs results in destruction of the bone of the nasal conchae which, in turn, can lead to facial distortion, reduced growth rates and serious economic loss.

6.21.2 Effects of introduction

Atrophic rhinitis is a disease of major economic importance. Its presence in New Zealand herds could result in serious losses.

Apart from an adverse effect on exports of live pigs, the presence of atrophic rhinitis would have little effect on New Zealand's export trade.

6.21.3 World distribution

New Zealand is, perhaps, the only swine-rearing country free from progressive atrophic rhinitis. Surveys have failed to detect the toxigenic strains of *P. multocida* which are an essential cause of the disease.

6.21.4 Meat as a vehicle

It is highly unlikely that meat could serve as a vehicle for the strains of *P. multocida* necessary for the development of progressive atrophic rhinitis. However, it is just conceivable that the organism could survive in the nasal cavity of pigs' heads, if these were imported attached to chilled carcasses.

6.21.5 Meat products

Pasteurellae may survive in carcasses for an "extended period".[12]

6.21.6 The risk of introduction

While toxigenic strains of *P. multocida* capable of causing progressive atrophic rhinitis could, conceivably, be imported in the nasal cavities of chilled pig carcasses, it is highly improbable that they could find their way, by the necessary airborne route, into the nasal cavities of local pigs.

The risks, therefore, must be considered negligible.

6.21.7 Recommendations for atrophic rhinitis

No specific measures are necessary.

6.22 Transmissible gastroenteritis and porcine epidemic diarrhoea virus

6.22.1 The diseases

Transmissible gastroenteritis (TGE) is a highly contagious disease of swine caused by a coronavirus. The disease is characterised by vomiting and profuse diarrhoea. Mortality approaching 100% is seen in young piglets when the disease first enters a herd. Mortality is low in swine over 5 weeks of age.

Porcine epidemic diarrhoea (PED) is a disease similar to TGE but which affects pigs of all age groups. Mortality in young piglets can be as high as 50%. In weaned pigs the disease manifests as diarrhoea and weight loss, but most animals recover. PED is caused by a coronavirus serologically unrelated to TGE virus.

6.22.2 Effects of introduction

When TGE first enters a herd its effects can be dramatic. Mortality among young piglets commonly approaches 100%. Once established enzootically in a herd, disease in young piglets is less common, but mortalities sometimes as high as 50% are seen amongst weaners. Economic losses can be substantial. It has been estimated that in one year (1977) TGE cost French producers US$10 million.[20]

TGE is not amenable to eradication, and vaccines are relatively ineffective in controlling the disease. It may be spread between herds by transiently-infected dogs, cats, starlings and flies.[5,20]

6.22.3 World distribution

TGE is present in many European countries, Canada, USA, Central and South America, Taiwan, Korea, China, and the Philippines.[5,20]

Surveys for TGE have revealed seroprevalences such as; France 20% of herds and 17% of swine, Canada 19% of herds and 8% of swine, German Federal Republic 21% of swine, the Netherlands 17% of swine, USA 50-54% of herds and 31-54% of swine.[20,28] In Iowa, TGE virus was isolated from 1.5% of

pigs at slaughter.[20]

PED is less widespread than TGE. Surveys have detected its presence in Belgium, England, the German Federal Republic, France, the Netherlands, Bulgaria, Taiwan and China. Evidence for its presence was not revealed by surveys in Sweden, Northern Ireland, USA, Australia and Hungary.

6.22.4 Meat as a vehicle

The main route of transmission of TGE and PED is faecal-oral.[5,20] However, it is recognised that TGE may be introduced into herds in uncooked blood and slaughter scraps.[5] At the peak of viraemia, some tissues, such as kidney, may contain titres of TGE virus as high as 10^6 $TCID_{50}$/g.[5] Piglets may be infected by a dose as low as 10-100 virus particles, while older pigs (5-6 months) require a dose 10,000 times greater.[5]

Recent Australian experiments[55] have demonstrated that it is possible for meat of pigs slaughtered during the viraemic phase of TGE to transmit infection to other pigs. However, the amount of meat fed to susceptible pigs was large (1 kg) and the experimental conditions were rather artificial. These findings are supported by an unpublished North American study[55] in which piglets developed TGE after being dosed orally with homogenised muscle tissue collected from infected pigs.

These studies constitute sufficient evidence to demonstrate that, under certain conditions, frozen pork could serve as a vehicle for TGE virus.

6.22.5 Meat products

The TGE virus is fairly heat labile, being inactivated rapidly at temperatures above 37°C.[20,54] It is stable when frozen[20] and may survive for months at 4-5°C.[5]

It is relatively pH stable and is not inactivated by lactic fermentation of meat products or by the pH changes occurring in meat.[5] Putrefaction of infected tissues destroys the virus.[28]

6.22.6 The risk of introduction

There is little likelihood of TGE (or PED) being introduced in meat. Infected pigs have a brief viraemia only[5] and virus is recovered from few normal-appearing pigs at slaughter (1.5% in one study[20], 0.8% in another[55]).

The quantity of infected meat necessary to transmit infection was approximately 1 kg in the Australian study[55], an unrealistically large quantity.

A number of interest groups in Australia have calculated the probability that pigmeat from Canada could serve to introduce TGE into Australia. Estimates ranged from an annual risk of 1 in 3.3 million to 1 in 15,500.[55] The most carefully calculated estimate produced a figure of 1 in 81,300 per year. It is unlikely that the risk to New Zealand would be significantly greater and, indeed, the risk would probably be smaller given that the volume of pork imported into New Zealand would be smaller (smaller human population) and the number of pig herds which might be fed garbage is fewer than in Australia.

6.22.7 Recommendations for transmissible gastroenteritis

No specific safeguards are warranted.

6.23 Trichinellosis

6.23.1 The disease

Trichinellosis (trichinosis) is the infestation with the nematode parasite *Trichinella spiralis*, of which there are a number of subtypes. Trichinellosis may affect all species of carnivores and omnivores, but is especially common in pigs, rats, cats and humans. In the latter species it is a serious zoonosis.

Infestation with *T. spiralis* is usually without clinical signs in pigs. In humans infestation is often symptomless, but serious disease is seen when the ingested dose of encysted larvae is high. Signs of infestation include fever, myalgia, facial oedema (especially of the eyelids), exanthema and functional disorders of the respiratory, pharyngeal and masticatory muscles.

In massive infestations death may occur as a result of circulatory failure and/or pneumonia. Mortality in human outbreaks of trichinellosis ranges as high as 40%.

The importance of trichinellosis lies exclusively in the danger posed to humans.[5]

The adult *T. spiralis* live in the small intestine. Females produce larvae (up to 1,000 per female) and these penetrate the intestinal wall and find their way into the circulatory system which carries them to striated muscles in which they prefer to lodge in the most active muscle groups. Encysted in muscle fibres, the larvae (or trichinae) may remain viable for years.

Infestation is spread to another host when encysted trichinae in muscle are eaten by carnivores or omnivores. Occasionally, infestation is spread via larvae passed in faeces of an infested host (especially pig or rat).[5,34,54]

Pigs usually become infested by being fed garbage containing meat scraps. Spread is promoted by poor hygiene and management practices, and for this reason the prevalence of trichinellosis in swine can be reduced by good, modern management and the cooking of any garbage fed to pigs. The infestation is rare in grain-fed pigs.

6.23.2 The effects of introduction

T. spiralis is already present in New Zealand. There is a reservoir of infestation in wildlife (rats etc) from which there have been rare instances of spill-over into domestic pigs.[56]

Modern pig management practices reduce the likelihood of the prevalence of trichinellosis in domestic swine increasing significantly. However, any increase, or even a single dramatic case, could jeopardise the small export trade in pigmeat to Australia.

6.23.3 World distribution

Trichinellosis occurs all over the world, but local prevalences vary significantly. It does not occur in Australia.[34,54]

Infestation with *T. spiralis* is uncommon in western Europe and Scandinavia. It is a problem in Spain (prevalence in swine 0.002% to 0.43%). It is relatively common in eastern Europe; Hungary, 1-2.5% of humans at necropsy, Romania, 1 per 1,000 swine, Bulgaria, 1 per 1,000 swine, Poland, 15 per 10,000, Soviet Union, 2.4 per 10,000 etc.[57]

Trichinellosis is uncommon in most of Asia and may be absent from some countries (such as the Philippines).[57]

In Latin America some countries may have a high prevalence (e.g. Uruguay, Mexico, Chile) and some countries may be free of the infestation (e.g. Brazil).[57]

Trichinellosis occurs in the United States and Canada and human infestations are frequently associated with the eating of undercooked meat of game animals, often wild pigs or bears.[57]

Trichinellosis occurs in Africa, with varying prevalence.

6.23.4 Meat as a vehicle

Meat is the usual vehicle for spreading *T. spiralis*. Pigs are usually infested by the practice of feeding them garbage containing meat scraps, although they may contract the parasite from eating rodent carcasses or faeces (of rodents or pigs).[34,54,57]

Humans usually become infested with *T. spiralis* after eating infested pigmeat.[34,57] However, infestations have also been acquired via bear meat (and other game meat), marine mammal meat and horse meat.[5,36,56] Horses are presumed to have become infested following contamination of their feed with rat faeces.

6.23.5 Meat products

The encysted larvae of *T. spiralis* are very resistant to physical and chemical factors. They remain viable in rotten meat for up to 4 months.[5,34] *T. spiralis* cysts can survive desiccation, salting and smoking.[34,36,56] Because of the low sensitivity of trichinoscopic examination of meat, outbreaks have occurred following consumption of pork which has passed inspection.[34,57]

Viable trichinae have been found in Nham, a fermented pork product from Thailand.[57]

Freezing will destroy encysted larvae of *T. spiralis*. Freezing regimens recommended include;

-15°C for 20 days[34,57]
-23°C for 10 days[57]
-25°C for 20 days, if > 15 cm thick[36]
-25°C for 10 days, if < 15 cm thick[36]
-30°C for 6 days[34,57]
-35°C for 40 minutes.[57]

The varieties of *T. spiralis* which infest arctic mammals withstand much lower temperatures than do the varieties infesting swine. Temperatures as low as -32°C may not be sufficient to kill cysts in polar bear, whale, walrus etc meat.[34,57]

Cooking will also destroy encysted trichinae. Regimens recommended by various authorities include;

(a) for swill intended for pigs, 100°C for 30 minutes[34,57]
(b) roasting, 77°C "more than sufficient"[34,57]
(c) core temperature of 60°C.[54,57]

A number of countries (the Netherlands, Sweden, Denmark, Canada) screen pigs for the presence of *T. spiralis* using an ELISA.[34,57] Such screening gives far greater assurances than those provided by trichinoscopy.

6.23.6 The risk of introduction

Cooked meat products are unlikely to constitute any risk, so far as *T. spiralis* is concerned.

Frozen meat, so long as it has been frozen to the specifications recommended above, is also likely to be safe.

Hams, sausages and other salted, pickled, smoked or cured products must be assessed on their country of origin and the meat inspection practices in that country. Products from North America and western Europe should constitute little risk. However, Spain and some eastern European countries (Hungary, Romania, Poland and, possibly, the Soviet Union) may be sources of product containing *T. spiralis*.

Game meat products should be regarded as relatively high risk, but again this would depend on the country of origin and the species of meat.

6.23.7 Recommendations for trichinellosis

For countries in which trichinellosis occurs at low prevalence (or is absent), such as North America, western Europe and Australia, no specific safeguards against trichinellosis are required.

For countries of unknown states, or where trichinellosis occurs at a higher prevalence, such as Spain and eastern Europe, additional safeguards need to be imposed. These should include;

(a) All pigmeat and pigmeat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

either (ii) Must originate from a herd of origin which can be

officially certified as having been free of trichinellosis for at least 3 years[5] and

(iii) Must have been subjected to trichinoscopic examination with negative results.[5]

or (iv) Must have been frozen according to a regimen recognised by OIE as being capable of destroying all trichinae.[5]

or (v) Must have been subject to heat treatment resulting in a core temperature of 60°C.

6.24 Listeriosis

6.24.1 The disease

Listeriosis is a bacterial disease caused by *Listeria monocytogenes*. Most species appear susceptible, including cattle, sheep, goats, pigs, horses, poultry and humans.

The usual route by which animals become infected with *L. monocytogenes* is by ingestion. Disease syndromes due to *L. monocytogenes* include meningoencephalitis, abortions and septicaemia.

L. monocytogenes is ubiquitous in nature and in animal and human faeces. It survives for years in soil, milk, silage and faeces. It is commonly found in tissues such as tonsils and lymph nodes of normal, healthy animals.[12]

6.24.2 Effects of introduction

L. monocytogenes is widespread in New Zealand.[58]

6.24.3 World distribution

L. monocytogenes is ubiquitous.[12,58]

6.24.4 Meat as a vehicle

Meat, poultry and their products are not uncommonly contaminated with *L. monocytogenes*.[58] Meat and poultry products have been implicated in the transmission of listeriosis to humans.[58]

Livestock are usually infected by ingestion of feedstuffs contaminated with soil or faeces.[12,28]

6.24.5 Meat products

A major factor in the survival of *L. monocytogenes* is pH. At pH 5.0 or above the organism multiples, below pH 5.0 survival is poor.[12]

It is able to tolerate high levels of salt, nitrates,

smoking and may survive standard pasteurisation, especially if contamination is high.[58]

Fresh meats and poultry are commonly contaminated with *L. monocytogenes*. In New Zealand between 20% and 68% of local retail meat products may be contaminated.[58]

Delicatessen products, dried meat products, cooked sliced meats may all be contaminated with *Listeria*. Common food preservatives such as salt, nitrates or smoke will not guarantee products free of *Listeria*, although they will reduce the risk of contamination.[58]
Any one of the following treatment regimens may be considered suitable to render meat products free of *L. monocytogenes* contamination;[58]

(a) Heat treatment in a hermetically sealed container.

(b) Treatment resulting in a water activity (A_w) less than 0.91 and pH less than 5.2.

(c) Treatment resulting in A_w less than 0.85.

(d) A pH less than 4.5.

6.24.6 The risk of introduction

It is possible that *L. monocytogenes* could be introduced into New Zealand in imported meat products. However, given the very high contamination rates in local products[58] no special significance should be attached to imported meat products, so far as listeriosis is concerned.

6.24.7 Recommendations for listeriosis

No specific safeguards are warranted.

6.25 Toxoplasmosis

6.25.1 The disease

Toxoplasmosis is the disease caused by infection with the protozoan parasite *Toxoplasma gondii*. This protozoan has been recorded in approximately 200 species of animals, including birds, reptiles and humans.

Infection of humans is very common, clinical disease is uncommon. Serious clinical disease is seen in humans who are immunocompromised or who have been infected *in utero*.

Disease in most species of animals is uncommon, although toxoplasmosis is a common cause of abortion in sheep.

The sexual stage of the parasite's life cycle occurs in the intestinal epithelium of the definitive host; the domestic cat or other *Felidae*.

Oocysts are excreted in cat faeces. After undergoing sporulation these become infective for the large number of animals capable of acting as secondary hosts. Sporulated oocysts infect secondary hosts by the oral route. The organisms enter the blood stream and are distributed to a range of tissues in which they become encysted.

The life cycle is completed when tissue containing the encysted parasites (bradyzoites) is ingested by cats.

Secondary hosts may also become infected via encysted bradyzoites in tissues.

6.25.2 Effects of introduction

The "introduction" of *T. gondii* into this country would be without consequences. Already the majority of cats become infected at an early age. At any one time approximately 1% are excreting oocysts.[59]

Inapparent infection of intermediate hosts with tissue cysts is very high. In New Zealand, 60-70% of meat animals are infected and approximately 50% of middle-aged humans.[59] It is difficult to see what effect any further importation might have.

6.25.3 World distribution

Toxoplasmosis occurs worldwide, being one of the most widespread zoonoses. In Europe *T. gondii* cysts are found in more than 50% of sheepmeat and pigmeat.[34]

6.25.4 Meat as a vehicle

Intermediate hosts become infected with *T. gondii* either by ingestion of oocysts from cat faeces or by ingestion of tissue cysts in raw or undercooked meat.[34,46,59]

Humans can also acquire toxoplasma infection from handling raw meat.[34,46]

6.25.5 Meat products

Adequate cooking (core temperature of 65°C) will destroy *T. gondii* tissue cysts.[46]

Freezing, either -15°C for 3 days or -20°C for 2 days, will significantly reduce the number of viable tissue cysts in meat.[34]

Smoking or salt curing will destroy *T. gondii* tissue cysts.[46]

6.25.6 The risk of introduction

There is a high probability that any meat, imported or locally produced, will contain tissue cysts of *T. gondii*.

6.25.7 Recommendations for toxoplasmosis

No specific safeguards are warranted.

6.26 Botulism

6.26.1 The disease

Botulism is a severe, often fatal, food poisoning of mammals (including humans) and birds caused by heat labile neurotoxins produced by *Clostridium botulinum*. Signs of illness are seen within one to 72 hours of ingestion of toxin in foods, feed-stuffs, carcasses, plant material etc.

Less commonly, the production of neurotoxin may also occur in the intestinal contents when conditions favour the proliferation of ingested *C. botulinum* spores, or in wounds contaminated by the spores.

The reservoir of *C. botulinum* is in the upper layers of soil rich in organic matter. The organism is also found in invertebrates and in the intestinal contents of fish, other cold-blooded animals and, less frequently, mammals and birds.

Under appropriate conditions, in suitable media, *C. botulinum* will proliferate and produce toxins. The ingestion of the toxins results in disease.

There are six or seven different types of *C. botulinum*, classified according to the type of toxin produced.

6.26.2 Effects of introduction

Type C *C. botulinum* is already established in New Zealand, as demonstrated by outbreaks of botulism in waterfowl and, less commonly, other animals, such as dogs.[60]

As botulism is a serious zoonosis, the importation of product containing the toxins could have serious effect on people consuming the product. Mortality can be as high as 15%-60% of cases.[34]

6.26.3 World distribution

C. botulinum is found worldwide. It occurs most frequently in tropical and subtropical areas. The different subtypes (based on serologic and toxigenic differences) have different geographic distributions. Types C and D, which

are most commonly involved in botulism of animals, occur most commonly in the tropics and subtropics. Types E and F are better adapted to lower temperatures. Type C is found in New Zealand and a single human case of Type A botulism has been recorded.[60]

The spores of *C. botulinum* are distributed far more widely than the occurrence of botulism would suggest. This is so because a combination of circumstances is necessary for the occurrence of disease.[12] These necessary circumstances are;

(a) Contamination of a suitable substrate by *C. botulinum*.

(b) Proliferation of the bacterium and production of its toxin.

(c) Survival of the toxin in the face of autolysis or processing of the substrate.

(d) Ingestion of the toxin.

6.26.4 Meat as a vehicle

Any food, whether of vegetable or animal origin, can give rise to botulism if conditions favour the multiplication of *C. botulinum*.[34] Foods become contaminated from the environment or gut contents.

6.26.5 Meat products

C. botulinum was discovered in 1897 following a fatal outbreak of illness in people who had consumed imperfectly smoked ham.[12] Human outbreaks have been attributed to fish and fish products, beef, pork, mutton, venison and poultry.[34]

C. botulinum is a strict anaerobe. Under unfavourable conditions it produces spores which are extraordinarily resistant to heat, acids, alkalis and disinfectants. Types A and F can survive 105°C wet heat for 120 minutes.[5] Spores remain viable in decomposing carcasses for more than 6 months.[5]

The bacterium survives in a pH range 4.0 to 9.0. It survives in salt at a concentration up to 5.3%. The toxins are stable in carcasses for months, even when dried, but

are reliably denatured by boiling.[5]

The main requirements for proliferation of *C. botulinum* in food are anaerobic conditions and a pH above 4.5.[34] Home canned foods are usually responsible for disease, although incorrectly sterilised or preserved commercial products are sometimes the cause. Regulation and inspection of industrial bottling, canning and food preserving processing plants provide safeguards against botulism.[34]

Autoclaving at 120°C kills spores of *C. botulinum* and commercial canning should attain such temperatures for at least 3 minutes.

Adequate salting, pickling, smoking, drying or heating should ensure products are safe.[33]

6.26.6 The risk of introduction

There is a minor risk that improperly processed food products (meat or vegetable) could contain the botulinum toxin and result in limited outbreaks of human botulism.

There is little likelihood that imported meat products, except perhaps meat meals, could serve to introduce new types of *C. botulinum* into the New Zealand environment. However, with the exception of Type E, all strains of *C. botulinum* are strongly proteolytic and contaminated food is likely to show obvious signs of spoilage. If such contaminated, spoiled food product were to be disposed of into a favourable environment, it is conceivable that the bacterium could establish itself. It should be remembered, however, that the environmental requirements for different types of *C. botulinum* are rather specific.

6.26.7 Recommendations for botulism

No specific safeguards are required.

6.27 Blackleg and other clostridial infections

6.27.1 The diseases

Blackleg is a non-communicable gangrenous myositis caused by *Clostridium chauvoei*. It occurs usually in cattle, less commonly in sheep and rarely[5], if at all[61], in goats. It has also been recorded in deer[61] and buffaloes.[5]

The reservoir of *C. chauvoei* is in the soil. In cattle, spores are ingested in the feed, enter the bloodstream and are carried to muscles where they lodge until predisposing factors, such as bruising, trigger their proliferation, with resulting disease.

In sheep *C. chauvoei* occurs mostly as a wound infection.

Malignant oedema is a gangrenous wound infection, seen mainly in sheep and to a lesser extent in cattle, caused by several species of the genus *Clostridium*. Most commonly involved is *C. septicum* while *C. novyi*, *C. chauvoei* and *C. perfringens* type A are involved sometimes.[61]

6.27.2 Effects of introduction

The clostridial species responsible for blackleg and malignant oedema are widespread within New Zealand already. Disease attributable to them is common.

6.27.3 World distribution

Blackleg and malignant oedema occur worldwide.

6.27.4 Meat as a vehicle

It is possible that meat could contain dormant spores of *C. chauvoei*. However, such meat is unlikely to serve as a vehicle to introduce disease as humans, dogs, cats and pigs (the species likely to eat meat) are resistant to infection with *C. chauvoei*.

As the only other meat likely to carry these species of *Clostridium* would be from clinical cases of malignant oedema, meat can be considered a highly unlikely vehicle.

Affected meat would not pass inspection, nor would it be acceptable to consumers. The clostridial wound infections are sometimes referred to as `gas gangrene'. The presence of such gangrene is unlikely to pass unnoticed.

6.27.5 Meat products

Not relevant, for the reasons outlined in the previous section.

6.27.6 The risk of introduction

There is no risk of these clostridial diseases being introduced in meat or meat products.

6.27.7 Recommendations for blackleg and other clostridial infections

No specific measures are warranted for these diseases.

6.28 *Salmonella* infections

6.28.1 The disease

Salmonellosis is a bacterial infection of many wild and domestic animals as well as humans. It is caused by the numerous species (serotypes, serovars) of the genus *Salmonella*. All *Salmonella* species are pathogenic, with their virulence varying considerably between species.

As a zoonosis, salmonellosis is seldom acquired directly, it is usually a food-borne infection.[5]

Animals or humans fall ill within a few hours to a few days of ingesting salmonellae. The main manifestation of the disease is a profuse diarrhoea or dysentery. Pneumonia, hepatitis and other manifestations may be seen. Pregnant animals may abort and death may occur. Disease in animals is usually precipitated by stress.

Chronic, asymptomatic carriers of salmonellae occur. Between 1 and 5% of New Zealand cattle are probably asymptomatic carriers of salmonellae.[59]

Salmonellae are gram-negative rods. They are subdivided into four subgenera, only one of which is epidemiologically/epizootiologically important.[5] Within each subgenus there is a further subdivision into serotypes (serovars).

It is important to distinguish between *Salmonella* species which are highly adapted to either one or several host species. Highly adapted species are;

(a) *S. typhi* and *S. paratyphi* B to humans.

(b) *S. gallinarum-pullorum*(Footnote) to chickens.

(c) *S. abortus ovis* to sheep.

In some areas of the world, including parts of Europe, *Salmonella pullorum* and *S. gallinarum* are considered to be the same species.[62] They are listed as a single species in *Bergey's Manual.* Some European writers use the name *S. gallinarum-pullorum.*

(d) *S. abortus equi* to horses.

(e) *S. cholerae suis* to swine.

(f) *S. dublin* to cattle.

These are `host specific' salmonellae. The degree of specificity varies. For example, *S. gallinarum-pullorum*, *S. abortus ovis* and *S. abortus equi* are found only in their host species.[5] *S. dublin*, on the other hand, is occasionally detected in pigs and sheep and may infect humans. The other species of *Salmonella* (e.g. *S. typhimurium*, *S. enteritidis* etc) are adapted to many species.

6.28.2 Effects of introduction

Salmonellae are ubiquitous organisms capable of infecting virtually all vertebrates. They may also survive in the environment , under favourable conditions, for relatively long periods. The salmonelloses of animals are generally a food-chain problem associated with feeding livestock on contaminated feedstuffs. The prevalence in animals tends to reflect the extent to which the feedstuffs they eat are contaminated.[59]

Salmonellae are already widespread and common in New Zealand. As already mentioned, between 1 and 5% of sheep and cattle are inapparent carriers. New strains could spread rapidly and widely throughout the livestock populations if they were associated with widely-used concentrate feeds.

Human salmonellosis is the second most common disease notified to the Department of Health, exceeded only by campylobacteriosis, another food borne zoonosis. The introduction of further strains would exacerbate this problem.

6.28.3 World distribution

Salmonellae are common worldwide, although the prevalence of different species shows marked regional differences.

S. typhimurium is the most common species worldwide.

6.28.4 Meat as a vehicle

Almost any food-stuffs, whether vegetable or animal origin, may serve as a vehicle for salmonellae.

The most common vehicles for human infection are poultry, pork, beef, eggs, milk and their products.[34] In the United States beef is the most common source of human salmonellosis, while in the United Kingdom poultry is responsible for more than 50% of outbreaks while beef accounts for only 2%.[63] In northwestern European countries poultry and pork are the most common sources.[63]

6.28.5 Meat products

Compared with other gram-negative rods, salmonellae are relatively resistant to various environmental factors. They grow at temperatures between 8 and 45°C, and water activities (A_w) above 0.94, and in a pH range 4.0 to 8.0. Salmonellae are also able to grow in an environment with little or no oxygen.[63]

Salmonellae are resistant to freezing. They are sensitive to heat and will not survive temperatures above 70°C.[63] With the exception of some heat-resistant strains of *S. senftenberg*, salmonellae are destroyed by 56°C for 10 to 20 minutes.[12] Heat resistance may be greater at low water activities or in foods with high fat content. Some recommended[63] heat regimens include;

(a) Slurry; 70°C for 30 minutes.

(b) Milk; 71.2°C for 15 seconds.

(c) Milk with added fat and sugar; 74.4°C for 15 seconds.

(d) Ice-cream mixes; 79.4°C for 25 seconds.

(e) Shredded coconut; 80°C for 30 minutes (steam).

(These treatments are effective in eliminating most non-spore forming bacteria, including salmonellae,

campylobacters, staphylococci, pathogenic streptococci, brucellae etc).

Salmonellae are resistant to drying and may survive years in certain dried foods. Meat-meal, bone-meal, dried blood and fish-meal are all vehicles for salmonellae.[5] In some instances the salmonellae contaminated the meat from intestinal contents and survived processing. However, it is more common for the product to have been recontaminated after manufacture. Only good manufacturing processes, subject to inspection, can safeguard against this sort of contamination.

Salting and smoking have a limited effect on the survival of salmonellae. Salmonellae have been shown to survive for several months in brine containing more than 20% salt, especially in products with a high protein or fat content, such as certain salted sausages.[63]

In smoked dry meat products salmonellae may easily survive for weeks or months.[63]

Gamma irradiation of foods of animal origin, using doses of up to 7 kGy has been shown to reduce significantly the number of contaminating salmonellae (and other foodborne pathogens).[63] Packaging prior to treatment is an extra advantage as it prevents recontamination.

Adjustment of pH below 4.0 using suitable acids (e.g. lactic acid) has a definite bactericidal effect on salmonellae in meat and poultry products.[63]

6.28.6 The risk of introduction

Given the conditions under which livestock and poultry are currently raised, transported, marketed and slaughtered, as well as existing food processing practices, it is impossible to obtain salmonellae-free foods of animal origin. For the present, control is based on protecting humans from infection and reducing the prevalence of infection in animals. Veterinary meat and poultry inspection are important for consumer protection.[34]

Imported meat products may cause cases of human salmonellosis. However, the risks may be no greater than with locally-produced goods. New species of *Salmonella* may be introduced in meat products (or, indeed, any food products) but new species are just as likely, if not more

so, to be introduced in the intestines of humans.

6.28.7 Recommendations for *Salmonella* infections

Meat products should be processed according to good manufacturing practices, from inspected meat certified as fit for human consumption.

6.29 Mucosal disease/bovine virus diarrhoea

6.29.1 The disease

Bovine virus diarrhoea (BVD) and mucosal disease are clinically dissimilar disease syndromes caused by the same pestivirus. There is a very close relationship between BVD virus, hog cholera (HC) virus and hairy shaker disease virus of sheep. In fact, one authority considers that speciation within the genus *Pestivirus* is not warranted and the group is better regarded as a multidimensional continuum.[64] For this reason, and because HC virus is commonly spread via infected meat[5,12], it is prudent to examine the possibility that BVD virus could also be spread in meat scraps.

BVD virus is readily transmitted to pigs via contact with infected cattle.[65]

6.29.2 The effects of introduction

If BVD virus were to be introduced into New Zealand in meat, it would most likely show up in pigs. Clinical disease resembling HC would occur,[65] and this could lead to an exotic disease emergency.

The last outbreak of HC in Australia, eradicated in 1963, was probably not due to an exotic `HC' virus, but rather to bovine pestivirus entering the local pig population.[64]

However, to keep a perspective on this risk, the majority of adult cattle in New Zealand (60%) are infected with BVD virus at some stage in their lives[66] and local strains of pestivirus are capable of evolving into more pathogenic strains.[64]

Further introductions of BVD virus in meat products are unlikely to have any effect on the local situation.

6.29.3 World distribution

BVD virus infection is very common in cattle throughout the world. In New Zealand and Australia 60% of cattle are likely to be infected during their lifetime.[66]

In Australia up to 50% of breeding pigs may have serological evidence of prior infection with BVD virus. In the German Federal Republic 42% of breeding pigs may have antibodies against BVD, while 15-20% of pigs in the Netherlands may be seropositive. Similar seroprevalences have been recorded in Ireland and the United States.[65]

5.29.4 Meat as a vehicle

In the absence of any specific information one could assume that, as with HC virus, meat could serve as a vehicle for BVD virus.

6.29.5 Meat products

Given the close similarity between BVD and HC viruses one could assume that BVD virus, if present in meat, would be susceptible to the same influences outlined in the section on HC virus.

6.29.6 The risk of introduction

While there is a possibility that BVD virus could find its way into pigs through the medium of imported meat products, this risk must be orders of magnitude smaller than the risk of pigs being infected from locally-derived pestivirus, either in beef or sheepmeat of local origin. Of even greater probability is the spread of pestivirus from ruminants to pigs by contact.

6.29.7 Recommendations for mucosal disease/bovine virus diarrhoea

No special attention need be given to meat or meat products as a potential vehicle for BVD virus.

6.30 Erysipelas

6.30.1 The disease

Erysipelas is a disease of swine, sheep, turkeys and a variety of other animals caused by the bacterium *Erysipelothrix rhusiopathiae*. In animals the disease is manifested as an acute septicaemia, chronic proliferative lesions, or arthritis. In humans infection with *E. rhusiopathiae* is known as erysipeloid.

E. rhusiopathiae has been isolated from many species of wild and domestic animals (swine, sheep, cattle, horses, dogs, cats[12,28]) and birds (turkeys, ducks, chickens, geese[62]), as well as reptiles (crocodiles[34]), amphibians and fish.[28]

It has been estimated that 30-50% of healthy pigs harbour *E. rhusiopathiae* in their tonsils.[28,34]

In livestock the majority of infections occur by the oral route[11] although humans are very resistant to infection by ingestion[34] and, although infection by that route is possible, nearly all human infections with *E. rhusiopathiae* are acquired through wounds and abrasions.

6.30.2 Effects of introduction

E. rhusiopathiae is widespread and well-established in New Zealand where it causes polyarthritis in lambs and erysipelas in swine and turkeys.

6.30.3 World distribution

Erysipelas occurs worldwide and is of economic importance throughout Europe, Asia, Australia and North and South America.

6.30.4 Meat as a vehicle

E. rhusiopathiae can persist in frozen meat, chilled meat, decaying carcasses, dried blood, fish meal, etc.[28]

6.30.5 Meat products

E. rhusiopathiae is remarkably resistant to salting, pickling and smoking and can persist for several months in cured and smoked hams.[28] It is also very resistant to drying.[12,62]

It is destroyed by heat and the following regimens have been reported;

(a) 70°C for 5-10 minutes[62]
(b) 56°C for 10 minutes[28]

The organism is also destroyed by gamma irradiation.[28]

6.30.6 The risk of introduction

While it is possible that some meat products imported into New Zealand could be contaminated with *E. rhusiopathiae*, the organism is already so common in livestock here that such importation would be of no relevance or significance. Humans are very resistant to infection by ingestion.[34]

6.30.7 Recommendations for erysipelas

No specific measures should be required.

6.31 Yersiniosis

6.31.1 The disease

Infections with *Yersinia pseudotuberculosis* or *Y. enterocolitica* may affect a large range of domestic and wild animals and birds, and humans.

Y. enterocolitica is a gram negative coccobacillus of which there are five biotypes and some 34 serotypes. In humans infection with *Y. enterocolitica* results in a disease manifest mainly as an acute enteritis. In animals asymptomatic carriage of *Y. enterocolitica* is common, but stress-precipitated outbreaks of enteric disease are seen, especially in deer, cattle and goats.

The serotypes of *Y. enterocolitica* isolated from most species of animals differ from those isolated from humans.[34] Important exceptions are swine, dogs and cats which are often infected with the serotypes responsible for human disease. Most animal strains of *Y. enterocolitica* do not produce human disease and the serotypes isolated from food often do not correspond to the types affecting humans.[34]

The route of infection is by ingestion.

Y. pseudotuberculosis, a gram negative coccobacillus, is a relatively common cause of stress-precipitated disease in laboratory animals. Sporadic human cases occur and a great many domestic and wild mammals and birds, as well as reptiles,are susceptible to infection.

In humans *Y. pseudotuberculosis* may cause a mesenteric lymphadenitis, resulting in acute abdominal pain easily mistaken for appendicitis. It is also a rare cause of abortions and orchitis in sheep, pneumonia and abortion in cattle and gastroenteritis in swine. In turkeys *Y. pseudotuberculosis* may cause enteric disease. Rabbits and hares are often infected.

Asymptomatic carriage in swine is common (4-6% of apparently healthy pigs) and the serotypes which predominate in humans also predominate in animals.[34] The route of transmission is faecal-oral, often via contaminated food.

6.31.2 Effects of introduction

Both species of *Yersinia* are widespread in New Zealand. Asymptomatic carriage of *Yersinia* species has been demonstrated in up to 30% of clinically normal cattle, lambs and deer.[59]

6.31.3 World distribution

Y. enterocolitica and *Y. pseudotuberculosis* are ubiquitous worldwide. Human disease has been confirmed in more than 30 countries on five continents.
As many as 17% of apparently healthy swine may carry *Y. enterocolitica*. In Belgium a survey isolated the organism from 62% of pork tongues. In Belgium and Denmark intestinal carriage has been demonstrated in 3-5% of swine. Swine are the probable reservoir of the zoonotic strains of *Y. enterocolitica*.

Y. pseudotuberculosis also occurs worldwide and is common in wild rodents, birds and soil.

6.31.4 Meat as a vehicle

Meat may serve as a vehicle for both these species of *Yersinia*. Beef, chicken and pork, both chilled and vacuum packed, have been blamed as sources for human cases of *Y. enterocolitica* infection.[41] Human cases of *Y. pseudotuberculosis* infection appear commonly to be associated with consumption of food contaminated by human carriers, rather than animals.[12]

6.31.5 Meat products

Both species of *Yersinia* are facultative anaerobes, both can grow at temperatures as low as 4°C and in a pH range 6.0 to 8.0.[34,41] So, refrigeration and vacuum packaging do not rule out the possibility of meat products serving to carry *Yersinia*.

If contamination of a product is high, Y. enterocolitica may survive pasteurisation.[34] However, in meat, a temperature of 60°C for `a few minutes' is considered sufficient to destroy it.[41]

6.31.6 The risk of introduction

Either of the *Yersinia* species could be introduced in meat products. However, given the ubiquitous nature and high prevalence of these organisms in New Zealand, there is probably no greater risk from imported products than from locally-produced meats.

6.31.7 Recommendations for yersiniosis

No specific safeguards are warranted.

6.32 Campylobacteriosis

6.32.1 The disease

Two of the species of *Campylobacter* are commonly identified as food-borne, zoonotic causes of enteritis in humans. These species are *C. jejuni* and *C. coli*.

C. jejuni is one of the major causes of bacterial enteritis in humans, especially in developed countries.[34] It is also very common and widespread as an inapparent intestinal infection in a wide variety of livestock and wildlife.[59] *C. jejuni* has been implicated in cases of enteritis disease in cattle, dogs, cats, foals, in hepatitis in chickens, abortions in sheep and mink, and in bovine mastitis.[12]

C. coli is a less common cause of enteritis in humans. It is a common inapparent infection of pigs, but is less common in other species although it has been isolated from a wide spectrum of domesticated and wild animals and birds. It is essentially an inapparent infection in animals.[59]

6.32.2 Effects of introduction

Both species are widespread in New Zealand, being the most common reported cause of enteritis in humans. In infected flocks of broiler chickens, prevalence of *C. jejuni* infection approaches 100%.[59] In pigs over six weeks of age, the prevalence of *C. coli* approaches 100%.[59]

6.32.3 World distribution

C. jejuni and *C. coli* are distributed worldwide.

6.32.4 Meat as a vehicle

Both *C. jejuni* and *C. coli* are transmitted by the oral route. Both are quite pathogenic for human, having low infectious doses. As few as 500 *C. jejuni* can cause enteritis in humans.[12,59]

Both are food borne infections. Incompletely cooked meat and, especially, poultry are important vehicles of infection.[12,59,67]

6.32.5 Meat products

Undercooked poultry, hamburger and pork have all been incriminated in outbreaks of human campylobacteriosis.[67] Cattle, sheep, and pig carcasses may show high rates of contamination in the slaughterhouse, immediately after slaughter, but by the time the meat has reached retail outlets contamination rates are low.[68]

Poultry carcasses are very frequently contaminated and bacterial counts are high; 10^5 campylobacters per gram of carcass is common.[68] Poultry constitutes the biggest potential source of foodborne infection for humans, and poultry carcasses frequently have a high contamination rate at the point of sale.[67]

However, despite high initial contamination rates in meat, campylobacters do not survive well in meat, and growth is usually poor. Normal meat pH (5.5-5.8) adversely affects the viability of *C. jejuni*. It grows on normal meat only at high temperatures (30°C).[67] So, storage of meat with normal pH values at either room, chilling or freezing temperatures results in rapid destruction of *C. jejuni*.[67]

C. jejuni dies off rapidly at temperatures between 15 and 25°C.[68] It is intolerant of temperatures above 50°C and at 60°C the number of contaminating organisms is reduced ten-fold in less than a minute.[67]

Generally, *C. jejuni* survival is adversely affected by high salt concentrations[67], although it is not clear whether it can survive for extended periods in the salt concentrations commonly used in food processing.[68]

Sodium nitrite destroys campylobacters in meat products, especially in combination with freezing.[68] *C. jejuni* is also sensitive to drying.[67,68]

6.32.6 The risk of introduction

Because of the way campylobacter contamination of meat products decreases with storage, there is a greater risk from locally produced meats than from imported ones.

6.32.7 Recommendations for campylobacteriosis

No special measures are warranted.

6.33 *Sarcocystis* species

6.33.1 The disease

Sarcocystosis is an infection with protozoa of the genus *Sarcocystis*. *Sarcocystis* species are two-host parasites with life cycles broadly similar to *Toxoplasma*. All species of *Sarcocystis* are highly host specific, both for carnivorous and intermediate host.[69]

The intestinal phase of the life cycle occurs in a specific carnivorous host and the tissue cyst (sarcocyst) phase is found in the muscles of another specific host which has ingested sporocysts originating from the carnivorous host.

Infections with sarcocysts are common but clinical disease is rare. Syndromes which have been reported include myositis, abortion (cattle, pigs, goats, sheep) and severe generalised infections. The intestinal phase usually produces no clinical signs in animals, but some species infecting humans may do so.[69]

Carnivores become infected by ingestion of bradyzoites in the muscle of infected intermediate hosts. The intermediate host acquires infection by ingestion of sporulated sporocysts from the faeces of specific carnivorous hosts.

6.33.2 The effects of introduction

Sarcocystis species are already widely distributed throughout New Zealand, with most herds and flocks being infected.[69]

Species present in New Zealand[69] include;

(a) *S. tenella* - sheep and dog.

(b) *S. gigantea* - sheep and cat.

(c) *S. medusiformis* - sheep and cat.

(d) *S. cruzi* - cattle and dog.

(e) *S. hirsuta* - cattle and cat.

(f) *S. capracanis* - goat and dog.

Other species have been observed in horses and pigs, but specific identities have not been established.

In New Zealand, approximately 8% and 0.3% of mutton carcasses are detained and condemned respectively for *Sarcocystis* infection. In 1981 it was estimated that this represented an annual loss of $1,700,000.[70]

6.33.3 World distribution

Sarcocystis species occur commonly worldwide. In Australia, in addition to the species listed in the previous section, sarcocysts have been identified in buffaloes, chickens and ducks.

Worldwide, intestinal infection with *S. hominis* (*S. bovihominis*) and *S. suihominis* occurs in 6-10% of humans.[34]

6.33.4 Meat as a vehicle

Meat containing sarcocysts is the natural vehicle by which *Sarcocystis* species complete their life cycle.

6.33.5 Meat products

Heating to 60°C for 20 minutes destroys sarcocysts.[70] Freezing meat reduces the number of viable sarcocysts.[34]

6.33.6 The risk of introduction

Given the high prevalence of sarcocystosis in meat animals worldwide, it is likely that imported meat would contain sarcocysts. However, to keep this risk in proportion, it should be remembered that herd prevalence in New Zealand already approaches 100%.[69] Nevertheless, imports of pork and beef could possibly introduce the two zoonotic species although these are more likely to arrive in the intestines

of humans.

6.33.7 Recommendations for *Sarcocystis* species

Heavy infections with sarcocysts are normally picked up at routine meat inspection and are either trimmed away or the carcass is condemned. Freezing reduces the viability of sarcocysts during storage and transport.

No specific measures are required for *Sarcocystis* infections.

6.34 Tularaemia

6.34.1 The disease

Tularaemia is an infectious septicaemic disease of wild rodents and lagomorphs and also domestic animals and birds. It is caused by *Francisella tularensis*, a gram-negative bacterium of the family *Brucellaceae*.

F. tularensis can infect over 100 species of wild and domestic animals and humans.[34] In the wild hares are the most frequently infected animals[5,] but infection is also seen in rabbits, deer, pigs, cattle, buffaloes, goats, sheep, horses, dogs and cats.[5,34,41] Infection circulates naturally between biting arthropods and wild mammals, but may also spread directly by respiratory aerosol or ingestion.[5,41] Insects such as *Culicidae*, *Muscidae*, *Tabanidae*, fleas, lice and bugs may spread the infection.

Carnivores are infected by ingestion of contaminated meat, but high doses are required, they rarely develop bacteraemia, and only occasionally manifest overt disease.[34]

Highly susceptible host species usually die after an incubation period of 1-25 days. Less susceptible hosts may become chronically infected with *F. tularensis*. In humans the disease usually manifests as a subacute disease with lymphadenitis and fever. Occasionally pneumonia or enteritis are seen.[5]

6.34.2 Effects of introduction

Tularaemia is an important zoonosis and so its introduction and establishment here would be of concern from a human health point of view.

In North America and the USSR, tularaemia causes significant epizootics with high mortality in sheep flocks.[12,34]

The establishment of tularaemia in New Zealand would have little significant effect on trade.

6.34.3 World distribution

Foci of tularaemia occur in most countries of western, central and southern Europe. The disease also occurs in Africa north of the Sahara, parts of Asia north of the 35th parallel and in North and Central America.

6.34.4 Meat as a vehicle

Human infection by ingestion of meat contaminated with *F. tularensis* is relatively common in endemic areas.[41] Game meat is the most common source of infection by ingestion. The meat of rabbits fed to mink and foxes on fur farms has been responsible for high mortality outbreaks of tularaemia.[41]

6.34.5 Meat products

F. tularensis can persist in carcasses for up to 4 months.[5] It can remain viable in salted meat for up to 31 days.[41] Although ingestion of rare meat (mainly rabbit, game) is responsible for many human infections, *F. tularensis* is readily destroyed by exposure to heat; 56^{o}C for 10 minutes.[41]

Freezing does not destroy *F. tularensis* in infected game meat.[41]

6.34.6 The risk of introduction

It is possible that *F. tularensis* could be introduced into New Zealand in imported meat. Relatively large quantities of infected meat fed to pigs, dogs or cats could allow the organism to multiply but whether it could spread to other animals is open to question. These animals seldom show clinical disease and seldom develop bacteraemia. Although many biting arthropods can transmit tularaemia, it appears that ticks may be essential to maintain the disease in an environment.[41]

To avoid the small risk of introducing *F. tularensis* in imported meats there are two reasonable safeguards which could be adopted;

(a) Either prohibit the importation of game meat

(b) Or permit importation of game meat only from countries or regions free of tularaemia.

OIE recognises a country or region as free of tularaemia when no cases have been seen for two years and bacteriological or serological surveys of previously infected areas have produced negative results.[5]

6.34.7 Recommendations for tularaemia

Prohibit the importation of uncooked wild rabbit and hare meat products from countries or areas not demonstrated free of *F. tularensis*.

6.35 Viral haemorrhagic disease of rabbits

6.35.1 The disease

Viral haemorrhagic disease (VHD) of rabbits is a recently-recognised syndrome causing devastating losses in rabbit farms in many countries around the world.

The disease was first reported in China in 1984. The virus has not been adequately characterised yet. It has been variously suggested that the virus may be a picornavirus[71,72], a calicivirus[72,73] or a parvovirus[19,73].

Rabbits appear to be the only species affected. Animals over two months of age are usually affected and suckling rabbits seem to be resistant.[71] After an incubation period of perhaps 48-72 hours animals show signs of an acute haemorrhagic syndrome characterised by respiratory disease and epistaxis.[71,72,73] The attack rate is between 30 and 80% and mortality in affected rabbits approaches 100%.[72,74]

6.35.2 Effects of introduction

The effects of VHD have been devastating on rabbit farming overseas and OIE has added it to its List B diseases. However, it is likely that many would welcome the introduction of this disease into New Zealand, even though it could devastate any rabbit farming operations.

6.35.3 World distribution

The disease first appeared in China. Since then it has been identified in several Asian countries, most eastern and western European countries and in Mexico.

6.35.4 Meat as a vehicle

It is believed that VHD has been introduced into a number of countries in rabbit meat.[19,71,73,74]

6.35.5 Meat products

The virus of VHD appears relatively hardy.[73] It is not

inactivated by pH 3.0 or 50°C[72], nor by freezing.[74]

6.35.6 The risk of introduction

One must assume that where rabbit carcasses are imported from countries where VHD is endemic, there is a possibility that the disease could be introduced. How the virus might get from imported carcasses to local rabbits has not been explained fully. However, in the case of the Mexican outbreak, it appears that introduction occurred when the quality of imported Chinese rabbit carcasses was compared with local product at a meeting of rabbit farmers. It is assumed that Mexican farmers then unwittingly carried the virus back to their own premises on hands and clothing.[19] Once introduced the virus can be spread by aerosol, contact, fomites, insects and rodents.[74]

6.35.7 Recommendations for viral haemorrhagic disease

If the unintentional introduction of VHD is to be avoided, importation of uncooked rabbit meat should only be permitted from countries or areas certified as free from VHD.

Rabbit meat must originate from farmed animals (ie not wild animals) that have passed veterinary ante-mortem and post-mortem inspection.

6.36 Highly pathogenic avian influenza

6.36.1 The disease

Highly pathogenic avian influenza (formerly known as "fowl plague") is an acute, highly contagious, fatal viral disease of chickens and turkeys caused by virulent strains of avian influenza virus. Other avian species are susceptible and may serve to spread the virus between flocks. In fact, wild birds are probably the most common means by which avian influenza is introduced into domestic flocks.

There is also evidence that pigs may become infected with avian influenza viruses, then spread these to poultry. H1N1 strains of influenza virus seem to pass from pigs to poultry and *vice versa*.[1]

While only a few strains of avian influenza virus are serious pathogens, there is a constant recombining of viruses and it is possible that any introduced virus could recombine with the local strains.However, it is not possible for two apathogenic viruses to recombine to produce a pathogenic one. One would have to be pathogenic to start with.[1]

6.36.2 Effects of introduction

An outbreak of highly pathogenic avian influenza would not have a major effect on trade[4] as there is little export of product. Some hatching eggs are exported to Pacific Island countries, but this is not a major trade.

A number of export health certificates for dairy products, wool, skins and hides certify freedom from highly pathogenic avian influenza and so would need to be revised in the event of an outbreak.[4]

A number of effects on the domestic economy would be seen. These would include;

(a) Individual poultry producers could be severely affected.

(b) The consumer could be faced with increased prices for poultry products which would be imported to make up a

shortfall in local production.

(c) The costs of control procedures could be high and would have to be borne by the industry or the taxpayer.

(d) Increased importation of product to meet local shortfalls could increase the risk of importing other important diseases absent from New Zealand (eg infectious bursal disease, Newcastle disease).

6.36.3 World distribution

Avian influenza viruses appear to have a worldwide distribution. However, outbreaks of highly pathogenic avian influenza (`fowl plague') have occurred very infrequently in the last thirty years.[5] Extensive epizootics were responsible for serious losses in the USA in 1983[5], there was an outbreak in the Republic of Ireland in 1983-84[1] and an outbreak occurred on a single property in Victoria, Australia in 1985[54]

Wild waterfowl (eg ducks) are a major natural reservoir of avian influenza viruses, and strains have been isolated from wild ducks in New Zealand.[75] This country is probably unusual in having so few strains of avian influenza virus. There appear to be no strains of having the H5 or H7 antigens which are a feature of all the virulent strains.[5]

6.36.4 Meat as a vehicle

The normal route of transmission of avian influenza viruses is probably by the faecal-oral route or respiratory route, although precise mechanisms and conditions have not been defined.[62]

The virus may be isolated from the respiratory tract of birds, and respiratory tissue is commonly left in poultry carcasses. The virus survives freezing for prolonged periods.[62]

Work carried out over 60 years ago demonstrated that avian influenza virus is able to survive for 287 days in flesh and 303 days in bone marrow stored at chilling temperatures.[76] The researcher who demonstrated this was

not able to infect chickens by feeding them with fresh or chilled contaminated muscle, but found that feeding them contaminated blood could infect poultry.[76]

The avian influenza viruses are also present in the faeces of infected birds and so faecal contamination of carcasses could be a means by which meat could service as a vehicle.

6.36.5 Meat products

Although different strains of avian influenza virus differ in their susceptibility to thermal inactivation, it appears that the virus is not very heat stable. For example, heat treatments in the order of 56°C for 15 minutes and 60°C for 5 minutes appear suitable to inactivate avian influenza virus.[62] At temperatures above 60°C the infectivity is destroyed very quickly.[5]

Avian influenza viruses are not very stable at pH below 6.0.[5] The ultimate pH value of poultry muscle is between 5.7 and 5.9.

6.36.6 The risk of introduction

Authorities consulted do not appear to consider that poultry meat products are likely to constitute a risk as vehicles of avian influenza viruses. However, some consider it is advisable to permit importation only from countries free from highly pathogenic avian influenza.[1,5]

Given the widespread distribution of avian influenza viruses in wild birds,[62] there is a greater probability that new strains of avian influenza will enter the New Zealand domestic poultry flock through contact with wild birds.

6.36.7 Recommendations for avian influenza

For countries or areas which cannot be certified as free from highly pathogenic avian influenza ("fowl plague"), restrictions must apply to meat from all avian species;

(a) All meat of avian species;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from highly pathogenic avian influenza ("fowl plague") and inspected regularly by the veterinary authorities who should also be able to certify that no case of highly pathogenic avian influenza ("fowl plague") has occurred within a 10 kilometre radius within the last 2 months.[5]

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute before drying.

6.37 Newcastle disease

6.37.1 The disease

Newcastle disease (ND) is a highly contagious and destructive viral disease affecting chickens, turkeys and numerous species of wild and captive birds. Ducks may be subclinically infected.

ND is probably the most serious disease of chickens throughout the world. New Zealand is free from it.

6.37.2 Effects of introduction

There are four distinguishable forms of ND seen in chickens;

(a) Velogenic viscerotropic ND is an acute lethal infection of all ages of chicken, with mortality approaching 100%.

(b) Velogenic neurotropic and pneumotropic strains of ND cause an acute pneumo-encephalitis with mortalities of 10-50% in older birds and up to 90% in younger birds.

(c) Mesogenic ND is a milder form causing low mortality.

(d) Lentogenic strains of ND virus cause a very mild or inapparent respiratory infection and mortality is rare. Lentogenic strains of ND virus are present in New Zealand.

The effects of introduction of ND into New Zealand would depend on the strains introduced. If velogenic strains were introduced mortalities could be devastating. Individual poultry farmers could be severely affected and consumers could be forced to pay higher prices for chicken and eggs as products were imported to make up the shortfall in local production. The costs of control of ND would be high and would have to be borne by the taxpayer or industry.

An outbreak of ND would have little effect on trade, as New Zealand exports very little poultry product.[4]

ND may spread up to 8 kilometres by airborne spread[5] and so

could be difficult to contain should it be introduced.

It is also likely that ND could enter New Zealand's native bird population, possibly causing serious mortalities.

6.37.3 World distribution

The less virulent strains of ND virus probably occur worldwide. The velogenic strains have not established themselves in Australia or New Zealand. In Britain, outbreaks of ND have occurred when pigeons infected with avian paramyxovirus 1 have contaminated poultry feed.[12]

6.37.4 Meat as a vehicle

ND virus normally spreads by the aerosol route within flocks and by fomites between flocks. Flies may also transmit the virus.[5,62]

Poultry meat (carcasses and offal) have served as vehicles for the spread of ND. The virus has been isolated from the skin and bone marrow of chickens, ducks, geese and turkeys traded internationally.[78]

The ND virus may remain viable on the skin of carcasses for 300 days or more and in bone marrow and lungs for 190 days.[34] The virus can survive in frozen muscle for at least 6 months[3] and probably for years.[5]

Poultry may be infected by feeding on contaminated meat scraps.[3,5,62,78]

6.37.5 Meat products

The ND virus is relatively stable. It has been demonstrated that in tropical countries with temperatures of around 40°C and relatively humidity of 20-30% the virus persists for at least 4 weeks in carcasses.[5] However, adequate cooking is likely to destroy the ND virus. The following heat treatments have been reported as inactivating strains of ND virus;

56°C for between 5 minutes and 6 hours.[5,62]
60°C for 7 minutes.[79]
60°C for 30 minutes.[5,62]
70°C for 50 seconds.[79]
100°C for 1 minute.[5,62]

6.37.6 The risk of introduction

Given the stability of ND virus in poultry meat there is a relatively high risk that ND could be introduced into New Zealand in uncooked poultry meat products.

The risk is compounded by the fact that, should infected meat be imported, virus could be spread to domestic poultry by intermediaries such as scavenging wild birds or flies. Once an outbreak of disease occurred in a poultry unit airborne spread could result in the virus being carried on the wind to other flocks up to 8 kilometres away.

6.37.7 Recommendations for Newcastle disease

For countries or areas which cannot be certified as free from ND, restrictions must apply to meat from all avian species;

(a) All meat of avian species;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from ND and inspected regularly by the veterinary authorities who should also be able to certify that no case of ND has occurred within a 10 kilometre radius within the last 2 months.[5]

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute

before drying.

6.38 Duck virus hepatitis

6.38.1 The disease

Duck virus hepatitis (DVH, duck hepatitis type 1) is a fatal, rapidly spreading infection of young ducklings characterised primarily by high mortality and hepatitis. It is caused by an enterovirus.

Naturally occurring disease is seen only in ducks although geese may be infected naturally and Muscovy ducks experimentally.[5] Chickens and turkeys may become infected[62] and, although they do not show clinical disease, may pass infection on to ducks. Rats may also serve as reservoirs of DVH virus.[5,62]

6.38.2 Effects of introduction

The introduction of DVH could have serious consequences for New Zealand's small duck farming industry. While morbidity and mortality vary considerably with age, mortality in ducklings may reach 100%.[5] It is an extremely contagious disease. Native species would be threatened.

There would be virtually no significant effect on exports.

6.38.3 World distribution

DVH has occurred in most countries with intensive duck farming. It does not occur in Australia[54] or New Zealand.

6.38.4 Meat as a vehicle

The DVH virus is resistant to adverse environmental conditions and survives freezing. The virus is shed in the faeces of recovered ducks and faecal contamination could result in the virus being carried on carcasses.

Infection can be transmitted by the oral route[5,62] although authorities consulted do not appear to consider meat to be a vehicle.

6.38.5 Meat products

The temperature and pH stability of DVH virus have been reviewed and redetermined by Davis[80] who noted a number of discrepancies between the results produced by different studies. For example, while one study showed DVH virus to withstand 56°C for 18 hours, another reported loss of infectivity after only 105 minutes at the same temperature.

Similarly, while some studies showed DVH virus to be inactivated at 4°C after 300 t0 700 days, in other experiments the virus was apparently inactivated in as few as 7 to 12 days at that temperature.[80]
These discrepant results led Davis[80] to re-examine the stability of DVH virus using a more precise assay technique which had recently become available. He was able to demonstrate that while DVH virus is relatively stable at temperatures below 45°C it is rapidly inactivated at temperatures above this. At 50°C DVH virus has a half-life of 48 minutes. At 56°C the half-life 1s only 1.26 minutes.

Normal cooking will destroy the DVH virus. Heating to 62°C for 30 minutes will inactivate DVH virus.[62]

Davis[80] demonstrated that DVH virus is relatively stable within the range of pH values likely to occur[77] in meat.

DVH virus has a relatively high stability in the environment, surviving up to 37 days in moist faeces.[62] It is possible, therefore, that virus could survive in uncooked product which was contaminated by infected faeces.

6.38.6 Risk of introduction

The risks of DVH being introduced in uncooked duck meat are very slight. Product produced from healthy birds under hygienic conditions should be considered safe.

6.38.7 Recommendations for duck virus hepatitis

No special safeguards required.

6.39 Duck virus enteritis (Duck plague)

6.39.1 The disease

Duck virus enteritis (DVE) is an acute contagious disease of ducks, geese and swans characterised by ocular and nasal discharges, diarrhoea, extensive vascular damage with tissue haemorrhages and high mortality. It is caused by a herpes virus. It does not affect chickens, turkeys or other species of birds or mammals.

6.39.2 Effects of introduction

Morbidity and mortality can reach 100% in outbreaks of DVE.[5] Consequently, the introduction of this highly contagious disease could have serious consequences for New Zealand's small duck-farming industry.

Native species, such as the already-endangered blue duck, would be put at serious risk.

There would be no significant effect on export trade.

6.39.3 World distribution

DVE has never been diagnosed in New Zealand or Australia. It occurs in several countries of Europe, in North America and in Asia.

6.39.4 Meat as a vehicle

The normal route of transmission of DVE virus is faecal-oral, via contaminated water.[5,62] As there is a viraemic phase,[62] it is conceivable that virus could be found in carcasses of birds slaughtered at the appropriate time. Faecal contamination of carcasses could also result in a carcass carrying the virus. However, on balance, it does not seem very probable that meat would serve as a vehicle for DVE virus.

6.39.5 Meat products

The DVE virus is not particularly stable.[5,62] It is

inactivated by drying (9 days at 22°C) and at pH below 6.0[62] (The ultimate pH of poultry muscle is usually between 5.7 and 5.9[77] but may sometimes be greater than 6.0[6]).

The virus would be inactivated readily by normal cooking procedures (56°C for 10 minutes).[62] At 50°C DVE virus is inactivated in 90 to 120 minutes.[62]

6.39.6 The risk of introduction

There is little risk that DVE virus would be introduced in meat or meat products. Authorities consulted do not appear to consider the possibility worthy of mention.

6.39.7 Recommendations for duck virus enteritis

No specific safeguards required.

6.40 Fowl cholera

6.40.1 The disease

Fowl cholera is an acute or chronic generalised or local infectious disease of domestic poultry and wild birds caused by strains of *Pasteurella multocida*. Mortality may be very high or it may be insignificant.[62] The course of an outbreak is influenced strongly by environmental factors[5] and strain of *P. multocida*.[62]

Manifestation of *P. multocida* infection include sudden death, respiratory difficulties, loss of appetite, diarrhoea, emaciation, pneumonia etc. In more chronic disease, swelling and suppuration of the wattles is seen. Losses in turkeys can be particularly severe.[62,81]

An asymptomatic carrier state is common.[62,81] Other animals may also carry strains of *P. multocida* which are capable of causing disease in poultry.[62]

6.40.2 Effects of introduction

Avian *P. multocida* infection already occurs widely in New Zealand, although clinical disease is uncommon. Significant losses in turkeys are seen occasionally.

6.40.3 World distribution

The organism, *P. multocida*, is distributed throughout the world. However, clinical disease is uncommon in developed countries. Disease is more likely to occur where rearing conditions and hygiene are poor.[5]

6.40.4 Meat as a vehicle

P. multocida can gain entry to the body through the mucous membranes of the pharynx, although experimental attempts to infect birds through the oesophagus, proventriculus and crop have failed.[81] Despite this failure, some authorities consider that avian pasteurellosis can be spread orally by contaminated waste and offal from poultry slaughterhouses.[5]

6.40.5 Meat products

P. multocida may remain viable in a carcass for 2 weeks at room temperature and for up to 2 months at refrigeration temperatures.[81] However, the organism is easily destroyed by drying and heat, being killed in 15 minutes at 56°C or 10 minutes at 60°C.[62]

6.40.6 The risk of introduction

Avian pasteurellosis already occurs in New Zealand. Importations of poultry meat products are unlikely to result in further outbreaks of disease.

6.40.7 Conclusions for fowl cholera

No specific safeguards required.

6.41 Fowl pox

6.41.1 The disease

Poxviruses affecting birds have been classified as belonging to a special genus, *Avipoxvirus*. Within the genus species are designated according to their principal host, e.g. fowlpox, turkeypox, pigeonpox etc. Avipoxviruses have been isolated from some 60 species of wild and domestic birds and all are related to some degree although their host preferences vary. Ducks are the only domestic species in which pox does not occur naturally.[81]

Fowl pox is a relatively slowly spreading disease of chickens and turkeys. The most common manifestations are cutaneous and mucosal pox exanthemas, but septicaemic forms are seen occasionally.[5]

Morbidity during an outbreak of fowl pox varies from a few birds only to 100%. The mortality is usually low but in young birds may reach as high as 50%.[81]

6.41.2 Effects of introduction

Avian pox infections already occur New Zealand-wide and, although outbreaks of disease are sporadic, within flock incidence can reach 100%.

6.41.3 World distribution

Fowl pox occurs worldwide. Disease is seen most in countries where poultry production is intensive.

6.41.4 Meat as a vehicle

Avian pox is transmitted by contact between virus and abraded skin. The virus is quite stable and can survive for long periods in epithelial cells.[5] Because poultry carcasses are usually traded with skin on, they could, in theory, serve as vehicles for pox viruses.

6.41.5 Meat products

Only products containing skin or fragments of viscera could possibly serve as vehicles for fowl pox virus. The virus is resistant to desiccation and in epithelial cells can survive complete drying for several weeks.[5]

Avian pox viruses are sensitive to heat. Inactivation occurs following heating at 50°C for 30 minutes or 60°C for 8 minutes,[62] so even moderate cooking will render products safe.

6.41.6 The risk of introduction

Besides the fact that avian pox viruses are already widespread in New Zealand, imports of poultry meat products pose very little further risk.

6.41.7 Conclusions for fowl pox

No specific safeguards are required other than certification that the poultry were in good health at the time of slaughter.

6.42 Infectious bursal disease (Gumboro disease)

6.42.1 The disease

Infectious bursal disease (IBD) is an acute contagious virus (*Birnavirus*) disease characterised by immunosuppression, anorexia, depression and, occasionally, nephrosis. Natural infection is seen predominantly in chickens but has also been recorded in turkeys and ducks. Morbidity in chickens may be as high as 30-100%, mortality 20-30%.[5,62]

6.42.2 Effects of introduction

New Zealand is in the fortunate position of being one of the few countries free from IBD. In 1985 Christensen[82] estimated that the cost of introduction of IBD would be in excess of $5.25 million per annum for the meat chicken industry alone. This figure is made up of costs due to growth depression, increased mortality, increased coccidiostat usage, cost of control measures, extra processing costs, and increased susceptibility to other diseases. These estimated costs are shown in Table 8.

A small export trade in day old chicks would probably be lost following the introduction of IBD.[82] This trade amounted to $52,000 and $72,000 in 1983 and 1982 respectively.

Once established, IBD cannot be eradicated from a country.[5]

TABLE 8: ESTIMATED ANNUAL COSTS (NZ$,000S) OF INTRODUCING INFECTIOUS BURSAL DISEASE TO NEW ZEALAND[82]

Growth depression		3,000
Increased mortality		260
Increased coccidiostat usage		60
Control measures	470	
Extra processing costs		1,420
TOTAL		5,210

6.42.3 World distribution

IBD has been reported in most poultry producing countries. In Australia between 45% and 94% of flocks are infected with IBD.[81]

6.42.4 Meat as a vehicle

The IBD virus is very stable and has been shown to resist freezing and thawing. It is stable at pH above 2.0[62] so is not affected by the pH change which accompanies *rigor mortis*.

The probability of the virus surviving in or on the carcasses of poultry from endemic areas is high. Infection by the oral route occurs,[62] so meat could serve as a vehicle by which IBD was introduced.

6.42.5 Meat products

The virus could escape destruction in lightly cooked chicken meat. It has been shown to survive 56°C for up to 5 hours[5] and 60°C for 30 minutes.[62] Over the last two years we have received conflicting advice from Australian and British experts on the heat treatment required to inactivate IBD virus.[83]

The heat treatments recommended by the Australians include;

70°C for 30 minutes.[5,62,83]
75°C for 5 minutes.[83]
82°C for 1 minute.[83]

However, work carried out by Alexander [1,83] indicates that these treatments are inadequate. In Alexander's experiments 0.1 ml aliquots of infected bursa homogenates were subjected to different heat treatments. Heating for 30 minutes at 70°C resulted in a reduction in IBD virus titre from $10^{3.7}$ to $10^{0.08}$/ 0.1 ml. That is, 1.2 CID_{50} (50% chick infectious doses) remained per 0.1 ml. At 75°C 10^4 CID_{50} remained after 5 minutes, i.e. about 40 CID_{50} per 0.1 ml of homogenate. On the basis of these figures and others recorded following treatments at 70°C and 80°C, Alexander concluded that to reduce to 0.1 the probability of infectivity remaining, the following heat treatments are required;

70°C for 50 minutes.
80°C for 9 minutes.

He further recommends;

100°C for 1 minute.

Alexander points out that this latter treatment is the same as that recommended (6.37.7) for Newcastle disease virus, which is more heat-labile than IBD virus.

It might be argued that Alexander's bursa homogenates contained very high titres of virus, and that bursal tissue is unlikely to be included in chicken meat products. If one were to extrapolate from Alexander's graph for inactivation of IBD virus at 80°C, it would appear that from a starting titre of 10^2 CID_{50} approximately 4 minutes should be sufficient to reduce to 0.1 the probability of infectivity remaining. This figure is comparable to figures produced by the Australians. However, until more information is available to resolve the apparent conflict between the British and Australian advice, a conservative approach should be taken to ensure that IBD, a recognised heat-resistant virus, is excluded from New Zealand.

6.42.6 Risk of introduction

Because IBD is widespread in most poultry producing countries, and because the virus is very stable, IBD must be considered one of the poultry diseases most likely to enter New Zealand if unregulated importation of poultry meat is permitted.

6.42.7 Recommendations for infectious bursal disease

For countries or areas which cannot be certified as free from IBD, restrictions must apply to meat and meat products of chickens, turkeys and ducks;

(a) All poultry meat and poultry meat products;

(i) Must originate from birds which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from IBD and inspected regularly by the veterinary authorities.

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C for 50 minutes, 80°C for 9 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C for 50 minutes, 80°C for 9 minutes or 100°C for 1 minute before drying.

6.43 Very virulent Marek's disease

Although Marek's disease is present in New Zealand the Poultry Industry Association of New Zealand (Inc) raised the concern, in May 1986, that importation of poultry meat for human consumption could introduce into this country some of the very virulent strains of the disease which have emerged in the United States.[84]

6.43.1 The disease

Marek's disease (MD) is a viral (herpesvirus) lymphoproliferative disease of domestic chickens affecting nerves, visceral organs and other tissues. Strains have emerged in recent years in North America, Europe and Australia which are more virulent than the classical strains.[84,85] These strains are called very virulent Marek's disease (vvMD).

6.43.2 Effects of introduction

Marek's disease occurs commonly throughout New Zealand. Infection is considered universal, with an average within flock prevalence of around 15%.[86]

Control is based on vaccination. The introduction of vvMD would result in more clinical disease and reduced efficacy of vaccines.[84,85]

6.43.3 World distribution

Marek's disease occurs in all poultry producing countries and vvMD strains have been isolated in North America, Europe and Australia.

6.43.4 Meat as a vehicle

The virus of MD replicates in many tissues including the skin epithelial cells at the site of the feather follicles.[62] Virus at these sites is relatively resistant to environmental factors.

MD virus can survive in feathers for several months at 25^{o}C and up to ten years at 4^{o}C.[5,62] The virus survives extended

freezing and several freeze-thaw cycles. It is not adversely affected by the pH changes associated with *rigor mortis*.[62]

The major route of infection is respiratory, rather than oral

6.43.5 Meat products

Normal cooking destroys the infectivity of MD virus. Cell-free preparations are inactivated by 56°C for 30 minutes and 60°C for 10 minutes.[62]

6.43.6 Risk of introduction

The risks of introducing strains of vvMD in meat products are slight. Transmission of MD virus via meat scraps would depend on those scraps finding their way into the feed of chickens. Indirect transmission via scavenging wild birds or rats is not likely because these are refractory to infection.[62]

6.43.7 Recommendations for Marek's disease

No special measures are warranted.

6.44 Avian leucosis

6.44.1 The disease

Lymphoid leucosis (leucosis/sarcoma) is a retrovirus-induced tumour which originates in the bursa of Fabricius before spreading to other organs. Clinically, disease manifests as emaciation, pallor of the comb, abdominal distension caused by tumours and, eventually, death. Subclinical disease causes production losses in both meat and layer birds. Chickens are the natural hosts although virus has been isolated from pheasants. Several different retroviruses are involved in the leucosis/sarcoma complex of tumours.

6.44.2 Effects of introduction

Leucosis is already present New Zealand-wide and occurs to a significant extent in layer flocks. Clinical disease is not common, but losses as high as 20% may occur on rare occasions.[86]

6.44.3 World distribution

Avian leucosis viruses have been found in every country in which they have been sought.

6.44.4 Meat as a vehicle

I have not found any suggestion that avian leucosis viruses can be transmitted by the oral route in meat. However, congenitally infected birds may have high titres of virus in blood and tissues.[62]

6.44.5 Meat products

The avian leucosis viruses are fragile and do not survive long outside the living bird. The half-life of various leucosis/sarcoma viruses at 37°C varies from 100 to 540 minutes (average around 260 minutes). Inactivation is rapid at high temperatures, with Rous sarcoma virus having a half-life of 8.7 minutes at 50°C and 0.7 minutes at 60°C.[62]

Even at subzero temperatures the avian leucosis viruses do not remain active for long. At -15°C the half-life of avian myeloblastosis virus is less than a week. Virus is inactivated by freezing and thawing.[62]

Below pH 5.0 inactivation of avian leucosis viruses is rapid.[62]

6.44.6 The risk of introduction

The lability of the avian leucosis viruses makes them improbable candidates for introduction in meat products. In addition, there is no evidence that they may establish infection by the oral route.

6.44.7 Conclusions for avian leucosis

No specific safeguards are warranted.

6.45 Egg drop syndrome '76

6.45.1 The disease

Egg drop syndrome '76 (EDS76) is a virus (adenovirus) disease which is characterised in laying chickens by a drop in production and the production of soft-shelled and shell-less eggs in apparently healthy birds. The virus may infect a range of other domestic poultry, such as ducks, geese, Muscovy ducks and swans. A variety of wild birds may also be infected.[62]

The main means of transmission of EDS76 virus is vertically (ie through the egg).

6.45.2 Effects of introduction

There is serological evidence which suggests that EDS76 virus is already present in New Zealand.[86] In clinical outbreaks of the disease in other countries egg production can be reduced by between 30 and 50%[62,86] and this reduction in egg production may last for 4 to 10 weeks.[62]

It is unclear whether or not clinical EDS76 occurs in New Zealand.[86]

6.45.3 World distribution

EDS76 was first recorded in the Netherlands but has since become a major cause of egg loss throughout the world. The virus probably originated as a duck adenovirus which found its way into chickens in contaminated vaccine.[62]

The disease syndrome has been reported from many countries in Europe, America and Australia.[81]

6.45.4 Meat as a vehicle

Although there is a viraemia in infected chickens, this is short-lived and after a week or so detection of the virus in tissues is difficult.[62]

The virus is a resistant one, and is found in the faeces.[62] So faecal contamination could result in carcasses carrying

the virus. However, I am not aware of any reports proposing this as a risk.

6.45.5 Meat products

EDS76 virus is relatively heat stable. It is inactivated by heating to 60°C for 30 minutes, but survives 56°C for 3 hours.[62] It is stable at the pH normally found in poultry meat.[62]

6.45.6 Risk of introduction

The transient nature of the viraemia, and the fact that EDS76 spreads laterally only with difficulty[62] indicate that there is little risk of this disease finding its way into New Zealand in poultry meat products. As already mentioned, I am unaware of any authority proposing meat as a vehicle for this disease.

6.45.7 Recommendations for egg drop syndrome '76

No specific safeguards are warranted.

6.46 Goose parvovirus

6.46.1 The disease

Goose parvovirus causes an acute disease (goose viral hepatitis, Derzsy's disease) characterised by high mortality in young goslings and Muscovy ducklings. Ducks, chickens and turkeys are refractory to infection with goose parvovirus.[87,88,89] In young goslings, depending on their maternal antibody level, mortality varies from 7-100% in a flock.[87]

6.46.2 Effects of introduction

Very high mortalities (90-100%) could be expected amongst young goslings and Muscovy ducklings in outbreaks following the initial introduction of goose parvovirus. The effects on these minor poultry industries could be devastating. Chicken, turkey and duck farming operations would not be affected.

There would be no significant effect on exports.

6.46.3 World distribution

Goose parvovirus is widespread throughout the world but has not been reported from the United States. A disease with similar clinical and post mortem features has been reported from Canada, but parvovirus was not isolated. It has not been recorded in New Zealand.

6.46.4 Meat as a vehicle

Parvovirus is recoverable from heart muscle, liver and bursa of Fabricius of goslings which have died of goose viral hepatitis.[89,90] However, geese which have survived the disease as goslings are immune and do not continue to shed virus as adults.[91] Therefore, meat from clinically healthy birds is most unlikely to serve as a vehicle for goose parvovirus.

6.46.5 Meat products

Parvoviruses are resistant to many environmental factors. [1,9] Goose parvovirus is stable over the pH range 3.0 to 9.0[3,12,89] so would not be inactivated by normal post-mortem pH drop.

Goose parvovirus is not likely to be inactivated by heat treatments of 65°C for 30 minutes[5] or 56-60°C for 60 minutes.[3,12]

6.46.6 The risk of introduction

Goose parvovirus causes disease in young birds. By the time surviving birds have reached adulthood they will be immune and free of virus. Meat and meat products from such birds would not serve to introduce goose parvovirus into New Zealand.

In addition, the limited host range of this virus (geese and Muscovies only) mean that scraps of imported goose meat are unlikely to come into contact with a susceptible host.

6.46.7 Recommendations for goose parvovirus

No specific precautions are warranted.

6.47 Duck astrovirus hepatitis

6.47.1 The disease

A disease of young ducks which is clinically, macroscopically and histologically similar to duck hepatitis type 1 has been reported as being caused by an astrovirus. The names duck hepatitis type 2 or duck astrovirus have been proposed.[92,93,94]

Affected ducklings die in good condition within 1 to 2 hours of appearing sick.[93] Losses ranging from 10 to 70% have been reported.[92,93,94] Survivors recovered with no subsequent stunting of growth.

6.47.2 Effects of introduction

Duck astrovirus could cause serious losses among young ducks.

6.47.3 World distribution

Duck astrovirus has been reported from the United Kingdom only. The disease apparently occurs there infrequently.

6.47.4 Meat as a vehicle

Wild birds are the probable source of infection in outbreaks of duck astrovirus.[94] Spread is probably by infective virus in faeces of affected birds.

The virus is present at high titre in livers of infected birds[92,93,94] and probably in the gut.

Duck astrovirus is stable at pH 3.0[95], well below the ultimate pH of poultry meat.

6.47.5 Meat products

Duck astrovirus can withstand heat at 60°C for 5 minutes but is inactivated by 60°C for 10 minutes.[95]

6.47.6 The risk of introduction

The risk of duck astrovirus being imported in meat products is probably slight, as with duck virus hepatitis (6.38.8).

6.47.7 Recommendations for duck astrovirus

As for duck virus hepatitis, no specific safeguards are warranted.

6.48 Turkey rhinotracheitis

6.48.1 The disease

Turkey rhinotracheitis (TRT), or turkey coryza as it is sometimes known, is an acute, rapidly spreading respiratory disease of turkeys which may affect turkeys of any age, but typically occurs in birds between 1 and 5 weeks old. The causative agent is a pneumovirus, a member of the family *Paramyxoviridae*.[96,97]

The onset of TRT, once it enters a flock, is rapid, with morbidity approaching 100% within 24 hours.[96,97] Mortality is variable, ranging from as low as 0.4% to as high as 90% of the flock.[97] It is usually highest in young poults.

Clinical signs include submaxillary oedema, coughing, frothy ocular discharge, sticky exudate from the nares, distension of infraorbital sinuses and drop in feed consumption. In laying birds there may be a rapid drop in egg production of up to 70%.[96,97]

In chickens, the same pneumovirus causes swollen head syndrome (SHS), manifest as swelling of the periorbital and infraorbital sinuses, torticollis, cerebral disorientation and depression. Usually fewer than 4% of the flock is affected. Broiler chickens may show severe respiratory disease, and egg production may be depressed in layers.[97]

Turkeys and chickens are known natural hosts. Guinea fowl and pheasants may become infected. Pigeons, geese and ducks appear to be refractory to infection.[97]

The methods by which TRT spreads are unclear but movement of infected birds, contaminated water, fomites and people have been implicated. Only contact spread has been confirmed.[97]

6.48.2 Effects of introduction

Morbidity and mortality may sometimes be high. TRT is greatly exacerbated by poor management practices and eradication is difficult.[97]

6.48.3 World distribution

There is evidence that TRT and/or SHS is present in many countries; Great Britain, France, Italy, South Africa, Israel, Germany, the Netherlands, Spain, Greece and North America.[96,97] A limited survey failed to find antibodies against TRT virus in a number of Australian poultry flocks.[98]

6.48.4 Meat as a vehicle

There are no reports which suggest that meat could serve as a vehicle for TRT virus. The distribution of virus in the tissues of infected turkeys has been studied.[99]

No virus was isolated from blood collected between 1 and 7 days after experimental inoculation, nor from lung, air sac, ovary, liver, spleen, kidney or hypothalamus between days 1 and 12 post infection.[99]

Virus was detected in the epithelium of the turbinates and trachea up to 7 days post inoculation.[99] Virus has also been isolated from the lung of infected poults.[97] Virus was demonstrated in the epithelium of the reproductive tract up to 9 days after inoculation.[99]

It appears that any viraemia is transient and of low titre.[99] It appears unlikely that meat would serve as a vehicle for TRT virus.

6.48.5 Meat products

The pH changes associated with *rigor mortis* will not affect any TRT virus present in tissues; TRT virus is stable over the range pH 3.0 to 9.0.[100]

TRT virus is inactivated by heating to 56^{o}C for 30 minutes.[100]

6.48.6 The risk of introduction

Given the transient and/or low titre viraemia associated with TRT, and the limited and short-lived distribution of virus in the tissues of infected birds, there is little risk of introducing this disease in meat harvested from clinically healthy flocks.

6.48.7 Recommendations for turkey rhinotracheitis

No specific safeguards are warranted.

6.49 Chicken anaemia agent

6.49.1 The disease

Chicken anaemia agent (CAA) is possibly a parvovirus and is responsible for an infectious anaemia syndrome of chickens.[96,101] In experimentally infected day old chicks, CAA causes an anaemia and pancytopaenia commencing within 8 to 20 days post-inoculation and reaching a maximum around 14 to 16 days. There is almost complete atrophy of bone marrow, thymus and bursa of Fabricius. Mortality, when CAA is the sole infecting agent, is rather low, but severely enhanced pathogenicity is seen when CAA infects chickens concurrently with a number of other viruses.[96,101,102]

Age resistance against the clinical manifestations of CAA infection develop during the first 7 days of life and is complete by 14 days.[101]

CAA is transmitted by contact and vertically.[96,101]

6.49.2 Effects of introduction

Clinical manifestations of infection with CAA are far less common than serological evidence of infection.[96,101,102] Maternally derived immunity usually protects young chicks until age resistance develops.[101,102]

CAA is present in New Zealand, is probably widespread, and has probably been here for several years.[103] New Zealand's freedom from infectious bursal disease is probably the main reason why CAA causes so few problems, as the interactive and additive effects of these two pathogens can be responsible for serious manifestations of disease.[96,103]

6.49.3 World distribution

CAA infections of commercial and specific pathogen-free chicken flocks are common throughout the world including Japan, USA, Sweden, Germany, Northern Ireland, the United Kingdom and Australia.[104] It is present in New Zealand.[103]

6.49.4 Meat as a vehicle

CAA spreads by contact and vertically.[96,101] The agent is present in a wide range of tissues following experimental infection of chickens. In chicks inoculated at a day old virus is recoverable from brain, thymus, liver, spleen, kidney, bursa of Fabricius, bone marrow, rectal contents and blood for 21 to 28 days.[105] It is recoverable sporadically from brain and rectal contents up to 7 weeks.[105] Chicks inoculated with CAA at an older age have lower titres of virus in fewer tissues and eliminate virus sooner.

Given the presence of CAA in bone marrow, it is theoretically possible that the agent could be introduced in the carcasses of broilers (which are usually slaughtered at around 40 to 42 weeks of age).

6.49.5 Meat products

CAA is able to withstand pH 3.0[106], so would be unaffected by the pH changes associated with *rigor mortis*.

CAA is also fairly heat stable, having been shown to retain infectivity after the following heat treatments;

60°C for 60 minutes[104]
70°C for 5 minutes[107]
70°C for 60 minutes[106]
80°C for 15 minutes[106].

CAA is partially inactivated by 80°C for 30 minutes and is inactivated by 100°C for 15 minutes.[108]

6.49.6 The risk of introduction

It is clear that CAA, if present in broiler carcasses, could be expected to retain its infectivity. Depending on the age at which chickens became infected, varying titres of virus could be expected to be present in a range of tissues which could be imported as part of a carcass.

6.49.7 Recommendations for chicken anaemia agent

As CAA is already present in New Zealand, and has probably been here for a long time and become widespread, no specific safeguards against its presence in imported

poultry meat are warranted.

6.50 Avian nephritis virus

6.50.1 The disease

Avian nephritis virus (ANV) is an enterovirus first isolated from rectal swabs from a clinically-normally week-old broiler chicken. Although ANV has caused interstitial nephritis in experimentally infected specific pathogen free (SPF) chicks up to 4 weeks old, and mortalities 8 to 10 days after inoculation of day old chicks[96,109] it does not appear to be involved with clinical disease under field conditions. For example, surveys have shown infection to be very widespread in commercial and SPF chicken and turkey flocks in Japan, Northern Ireland, England and Europe[96,109,110,111,112] without apparently causing problems.

It is probable that the virus has been introduced into flocks around the world as a viral contaminant of vaccines.[110]

Infection occurs readily by a number of routes including orally, vertically and by contact.[110,113,114] There is an increasing age resistance to infection.[114]

6.50.2 Effects of introduction

Serological evidence for the presence of ANV has already been detected in New Zealand.[103] Although the infectivity of ANV is high, the pathogenicity is low[111,114] and it is obvious that the virus can become very widely disseminated in a population without producing disease problems.[110,112]

6.50.3 World distribution

ANV has probably been spread around the world as a vaccine contaminant.[110] It is widespread throughout chicken and turkey flocks in many countries and, in Ireland, has been detected in many importations of chickens from Europe held in quarantine.[110]

6.50.4 Meat as a vehicle

It is likely that meat could serve as a vehicle for ANV. Chickens are readily infected by the oral route.[109,114] Viral titres are very high in many tissues[109,114],at least for several days after inoculation. For example, in the tissues most likely to be imported in broiler carcasses, viral titres as high as $10^{7.2}$ to $10^{10.5}$ PFU/g in kidneys[109,113] and $10^{3.9}$ PFU/g in lungs[113] have been measured. The oral chick-infective-dose 50% (CID_{50}) is between $10^{0.9}$ and $10^{1.7}$ PFU.[114] This means that chickens could become infected by

eating tissue fragments as small as a few micrograms to a few milligrams.

As ANV is stable at pH 3.0[115] it is unlikely to be in affected by the pH changes which accompany *rigor mortis*. Neither is infectivity of ANV-infected tissues reduced by repeated freezing and thawing.[115]

6.50.5 Meat products

The related enterovirus of duck virus hepatitis is inactivated by 60°C for 30 minutes.[62]

6.50.6 The risk of introduction

ANV is already present in New Zealand and further introductions in poultry, poultry meat and vaccines are not unlikely.

6.50.7 Recommendations for avian nephritis virus

No specific safeguards are reasonable or warranted.

6.51 Big liver and spleen disease

6.51.1 The disease

Big liver and spleen disease (BLS) is a syndrome which has been recognised in commercial broiler breeder hens in Australia since 1980. It is characterised by enlargement of the liver and spleen, a sudden drop in egg production and increased mortality.[116,117] The disease appears to be infectious and the causative agent is presumed to be a virus although all attempts to isolate a virus have failed. It is possible that the aetiology of the disease is complex, with more than one causal agent/factor involved.[118]

BLS has been recognised exclusively in adult flocks in production, with disease signs being recorded as early as 24 weeks of age and as late as 58 weeks of age. The sudden drop in egg production lasts for 3 to 4 weeks and then returns to near normal after another 3 weeks. An increase in hen mortality rate of between 0.1% and 1% per weeks may be seen.[116]

6.51.2 Effects of introduction

BLS is considered by some to be the most economically significant disease of sexually mature broiler breeders in Australia.[116]

6.51.3 World distribution

The disease has been seen only in Australia[116,117] although serological reactions to the rather crude gel diffusion test have been recorded in flocks in Northern Ireland and Great Britain.[118]

6.51.4 Meat as a vehicle

The means by which BLS spreads has not been elucidated, but it appears to be infectious. Many attempts to transmit BLS experimentally have been unsuccessful, but some inoculation attempts have succeeded in transmitting the disease.[119]

Electron microscopic examination of ultra-thin sections of liver, spleen and kidney of affected birds has failed to demonstrate the presence of any virus-like particles, even when the presence of BLS antigen has been confirmed in these tissues.[117,118]

There is no evidence, therefore, to suggest that meat may serve as a vehicle for BLS.

6.51.5 Meat products

No information is available.

6.51.6 The risk of introduction

BLS is seen only in older breeder hens, not the class of bird used for meat production. No virus has yet been demonstrated in tissues and the means by which the disease spreads naturally has not been elucidated. The aetiology may be complex and many attempts to transmit BLS have been unsuccessful.
On the basis of available information it does not seem very likely that BLS could be introduced in meat or meat products.

6.51.7 Recommendations for big liver and spleen disease

No specific safeguards are warranted.

7. Unusual meats

MAF receives inquiries from time to time from people wishing to import many unusual types of meat and meat product. No attempt has been made to cover all possibilities in this review.

For example, consideration has not been given to meat of marine mammals as it is unlikely that the Department of Conservation would issue the necessary permits for importation of whale meat etc.

The unusual meats for which permits have most commonly been sought are buffalo, kangaroo, ostrich and crocodile.

7.1 Buffalo

Asian buffaloes (*Bubalus bubalus*) are members of the family *Bovidae* and, so far as the diseases identified as significant in this review are concerned, may be considered to be covered by the assessments made for diseases of cattle.[120]

Foot and mouth disease, rinderpest, anthrax, hydatids and cysticercosis are diseases of cattle which the review has identified as warranting attention when assessing a proposal to import meat products. All should be considered in assessing any proposal to import buffalo meat.

7.2 Kangaroo

Kangaroos may be affected by a range of the infectious diseases which occur in mammals. Many of the pathogens examined in this review can affect kangaroos; e.g., *Erysipelothrix rhusiopathiae*, *Salmonella* species, *Campylobacter* species, *Francisella tularensis*, *Brucella abortus*, *Mycobacterium bovis*, *M. avium*, *Leptospira* species, *Coxiella burnetii*, *Bacillus anthracis* and *Echinococcus granulosus* (hydatids).[121]

As Australia is the only country from which one could reasonably expect to import kangaroo meat, *F. tularensis* may be ruled out, as tularaemia does not occur in Australia.

In fact, hydatids (*E. granulosus*) and anthrax are really the only significant cause for concern among the diseases which could possibly be introduced in kangaroo meat.

As kangaroos are likely to be shot in the wild (game animals), ante-mortem veterinary inspection is not feasible. For this reason reliance must be placed on post-mortem veterinary inspection. Meat must be processed in

premises under supervision of the veterinary authorities and must be passed as sound and fit for human consumption. Kangaroo meat imported into New Zealand must comply with the requirements of the Food Regulations (1984).

7.3 Ostrich

Of the diseases considered in this review, ostriches are susceptible to *Mycobacterium avium* infection, salmonellosis, pasteurellosis, anthrax and Newcastle disease.[122,123] The importation of ostrich meat products should be covered by the same safeguards recommended for other poultry meat.

7.4 Crocodile

There do not appear to be any significant pathogens affecting crocodiles which could be introduced into New Zealand livestock. The most common infectious agents associated with diseases of crocodile are gram negative bacteria.[121,124,125] The most common isolates appear to be *Aeromonas hydrophila* and *Pseudomonas reptilivora*,[124] which are unlikely to infect mammals, poultry or humans.

While crocodiles may be afflicted by tuberculosis, it is caused by species of *Mycobacteria* adapted to reptiles, frogs, turtles, toads etc[121,124], rather than *M. bovis*.

Salmonella species are commonly isolated from turtles[124] and, one presumes that they are similarly common in crocodiles. *Salmonella arizona* and *S. waycross* were isolated from captive crocodiles in Papua New Guinea.[125] Various leptospires have been isolated from crocodiles, including *L. pomona*, *L. ballum*, *L. icterohaemorrhagiae* and *L. tarrasovi*. *Erysipelothrix rhusiopathiae* also infects crocodiles.[124]

Edwardsiella tarda has been isolated from the gut of alligators[124] and an *Edwardsiella* species has been isolated from the lung of a captive crocodile.[125] *E. tarda* is a significant pathogen of warm-water fish (ornamentals, red sea bream, tilapia) but has not been recorded as causing disease in salmonids. It probably occurs in New Zealand; it has been isolated in Tasmania and other parts of Australia, but has never been associated with diseases there.

The reason that *E. tarda* does not cause problems in salmonids is that they do not live in waters warm enough for the survival of *E. tarda*.

Crocodiles are affected by a number of viral diseases, but there are no reports of any of the viruses of livestock,

which are discussed in this report, being isolated.

It is possible that crocodile meat could be infested with the tissue stages of the cestode parasite *Spirometra erinacei.* This parasite is relatively common in Australia[34] (the most likely source of crocodile meat) and infestation with the tissue stages is known as 'sparganosis'.

Sparganosis is a zoonotic disease caused by infestation with first and second stage larvae of *Spirometra erinacei* and related cestodes.[34,44,46]

The life-cycle of this parasite begins when free-swimming larvae hatch from eggs passed in the faeces of the carnivorous definitive host (cats, dogs, dingoes, foxes etc.). Ingested by the first intermediate host, a copepod, the parasite develops further before ingestion by a secondary host in which it develops into a pleurocercoid or 'sparganum'. Second intermediate hosts include many species of amphibians, reptiles, mammals and birds, but not fish.[34] (Recorded examples include frogs, lizards, snakes, alligators, feral swine and humans.)

Completion of the life-cycle occurs when tissues of an infested secondary intermediate host are eaten by a definitive host. Humans may become infested by eating raw or improperly-cooked meat of infested intermediate hosts.

Human sparganosis is uncommon in Australia. It usually manifests as subcutaneous nodules of variable size.

In meat the spargana are seen as opaque, white, wrinkled, ribbon-like structures between muscles and under serous membranes. Spargana in meat survive chilling at 5°C for at least 2 weeks. They do not survive freezing.[46]

Sparganosis has not been detected in farmed crocodiles in Australia and so crocodile meat from that country is unlikely to pose a significant threat, so far as this infestation is concerned. Australian meat inspection procedures are likely to pick up this infestation. This contention is supported by the fact that during the period 1971-1972 one slaughterhouse in New South Wales condemned 100% of feral pigs captured and slaughtered for human consumption because of infestation with spargana.

Only crocodile meat originating from animals which have passed veterinary ante-mortem and post-mortem inspection should be imported.

Crocodile meat must be processed in premises under supervision of the veterinary authorities in the exporting country and must be passed as sound and fit for human

consumption. It must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

8. Conclusion and summary of recommendations

The review has shown that although it is theoretically possible for meat to serve as a vehicle for a large number of pathogens, in reality there are relatively few for which specific safeguards need be applied. The diseases for which such specific safeguards are warranted are shown in Table 9. In addition, specific recommendations are made for brucellosis and tuberculosis because, even though I believe that the risk of these diseases actually being introduced in meat are extremely small, they are very serious zoonoses subject to eradication programs in all developed countries. Official control programs for these two diseases are so common that their absence is regarded by some countries as a barrier to trade. In Table 10, the meat products of concern are shown for each disease agent.

TABLE 9: DISEASES WHICH MIGHT REASONABLY BE EXPECTED TO BE INTRODUCED VIA MEAT UNLESS SPECIFIED SAFEGUARDS ARE IMPOSED

	FMD	SVD	Rinderpest	ASF	HC	Anthrax	Hydatids	Lepto.	Cysticerc.	BSE	VES	Trichinosis	Tularaemia	VHD	ND	AI	IBD
Cattle	+	-	+	-	-	+	+	+/-	+	+/-	+/-	-	+	-	-	-	-
Sheep	+	+/-	+	-	-	+	+	+/-	-	+/-	+/-	-	+	-	-	-	-
Goat	+	-	+	-	-	+	+	+/-	-	+/-	+/-	-	+	-	-	-	-
Pig	+	+	+	+	+	+	+	+/-	+	-	+	+	+	-	-	-	-
Deer	+	-	+	-	-	+	+	+/-	-	-	?	-	+	-	-	-	-
Horse	-	-	-	-	-	+	+/-	+/-	-	-	+/-	+/-	+	-	-	-	-
Rabbit	-	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-
Buffalo	+	-	+	-	-	+	+	+/-	+	-	?	-	+	-	-	-	-
Kangaroo	-	-	-	-	-	+	+	-	-	-	-	-	+*	-	-	-	-
Chicken	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+/-	+
Turkey	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+/-	+
Duck	-	-	-	-	-	+/-	-	-	-	-	-	-	-	-	+	+/-	+
Goose	-	-	-	-	-	+/-	-	-	-	-	-	-	-	-	+	+/-	?
Ostrich	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	+/-	?
Crocodile	-	-	-	-	-	-	-	-	-	-	?	-	-	-	-	-	-

* While kangaroos may be infected with *F. tularensis* the only country from which kangaroo meat is likely to be imported is Australia, and tularaemia does not occur there.

Key: FMD, foot and mouth disease; SVD, swine vesicular disease; ASF, African swine fever; HC, hog cholera; BSE, scrapie and/or BSE; VES, vesicular exanthema of swine; VHD, viral haemorrhagic disease; ND, Newcastle disease; AI, highly pathogenic avian influenza; IBD, infectious bursal disease.

TABLE 10: TYPES OF MEAT PRODUCTS WHICH MIGHT SERVE AS VEHICLES FOR SPECIFIED DISEASES UNLESS SAFEGUARDS ARE IMPOSED

	FMD	SVD	Rinderpest	ASF	HC	Anthrax	Hydatids	Lepto.	Cysticerc.	BSE	VES	Trichinosis	Tularaemia	VHD	ND	AI	IBD
Chilled	+	+	+/-	+	+	+/-	+/-	-	+	-	+	+	+	+	+	+	+
Frozen	+	+	+/-	+	+	+/-	-	-	-	-	+	-	+	+	+	+	+
Offals	+	+	+/-	+	+	+/-	+	+/-	-	-	+	-	+	+	+	+	+
Cooked	-	-	-	-	-	+/-	-	-	-	-	-	-	-	-	-	-	-
Dried	+/-	+/-	+/-	+/-	+/-	+/-	-	-	+/-	-	+/-	+	+/-	?	+	-	+
Cured	+/-	+/-	+/-	+/-	+/-	+/-	-	-	+/-	-	+/-	+	+/-	?	?	-	+
Meal	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-

Key: FMD, foot and mouth disease; SVD, swine vesicular disease; ASF, African swine fever; HC, hog cholera; BSE, scrapie and/or BSE; VES, vesicular exanthema of swine; VHD, viral haemorrhagic disease; ND, Newcastle disease; AI, highly pathogenic avian influenza; IBD, infectious bursal disease.

8.1 Compiled safeguards

The specific safeguards identified in the report comprise;

8.1.1 Recommendations for foot and mouth disease

For countries or areas which have not been free from FMD for at least the preceding 12 months, restrictions must apply to meat of all animals which are capable of being infected naturally with FMD virus. Such animals include cattle, sheep, goats, deer, pigs, buffaloes and game animals.

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Only beef may be imported as chilled or frozen meat and

(ii) Must originate from cattle which have been adequately vaccinated against FMD and

(iii) Must be carefully boned out and

(iv) Must have reached pH 6.0 before freezing.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100^{o}C or higher for 2 to 3 minutes or 70^{o}C or higher for 25 minutes.

(d) Dried meat products;

(i) Must be certified as containing only beef and

(ii) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be certified as containing only beef and

(ii) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

either (i) Must qualify for official certification as

"Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

(g) Meat products from wild game animals;

(i) Clearly, the recommendations in (a) above cannot be applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

8.1.2 Recommendations for swine vesicular disease

For countries or areas which have not been free(Footnote) of SVD for at least the preceding 12 months, restrictions must apply to pig meat;

(a) Chilled or frozen meat;

(i) Importation prohibited.

(b) Cooked meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater.

(c) Dried meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater before drying.

For any country which has, at some time, experienced SVD, recognition of freedom from SVD virus should be based on a properly-structured serological survey.[14]

(d) Cured meat products such as salamis;

(i) Importation prohibited.

(e) Hams and bacon;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

either (ii) Qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 months curing process.

or (iii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater.

8.1.3 Recommendations for rinderpest

For countries or areas which have not been free from rinderpest for at least the preceding 12 months, restrictions must apply to the meat of all animals which are capable of being infected naturally with the disease. Such animals include cattle, sheep, goats, deer, buffaloes, pigs and game animals.

(a) All meat and meat products;

(i) Must originate from animals which have not been vaccinated against rinderpest and

(ii) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Importation prohibited.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100°C or higher for 2 to 3 minutes or 70°C for 25 minutes.

(d) Dried meat products;

(i) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

(i) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

(g) Meat products from wild game animals;

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

8.1.4 Recommendations for African swine fever

For countries or areas which have not been free from ASF for at least the preceding 12 months, restrictions must apply to all pigmeat products.

(a) All pigmeat and pigmeat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Importation prohibited.

(c) Cooked meat products;

(i) Must be free of bone and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or higher for 25 minutes.

(d) Dried meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater for 25 minutes before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Importation prohibited.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

(g) Meat from wild pigs (game meat);

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

8.1.5 Recommendations for hog cholera

A country may be considered free from HC when;

(i) A policy of vaccination and stamping out is practised and no case has been seen for 1 year.

or (ii) A policy of stamping out alone is practised and no case has been seen for 6 months.

For countries or areas not free from HC, restrictions must apply to all pigmeat products.

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Importation prohibited.

(c) Cooked meat products;

(i) Must be free of bone and

(ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or higher for 25 minutes.

(d) Dried meat products;

(i) Must have been subjected to heat treatment

resulting in a core temperature of 70°C or higher for 25 minutes before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Importation prohibited.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

(g) Meat from wild pigs (game meat);

(i) The recommendations in (a) above are not applicable. However, the meat must be free of bone and

(ii) Must have been subjected to a heat treatment resulting in a core temperature of 70°C or greater for 25 minutes.

8.1.6 Recommendations for anthrax

(a) Meat products intended for human consumption;

It would be desirable for all meat and meat products to originate from animals which have passed veterinary ante-mortem and post-mortem inspection. However, this requirement is not feasible for game animal meat. Such meat (including kangaroo meat) must have passed veterinary post-mortem inspection and have been processed in premises under the supervision of the veterinary authorities.

(b) Meat, blood or bone meal intended for feeding to livestock;

Importation prohibited (see 6.16.7).

(c) Meat, blood or bone meal intended for use as fertiliser;

Must have been subjected to heat treatment of 150°C for at least 3 hours.

8.1.7 Recommendations for Aujeszky's disease

All pigmeat and pigmeat products must originate from animals which have passed veterinary ante-mortem and post-mortem

inspection and were free from signs of AD at the time of slaughter.

8.1.8 Recommendations for echinococcosis/hydatidosis

(i) All meat and meat products must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) offals must be frozen to -18°C or less.

8.1.9 Recommendations for brucellosis

Brucellosis should be subject to an official control program in the country of origin of the meat. The program should be appropriate to the species of meat animal and the species of *Brucella* (i.e. *Br. abortus* in cattle, *Br. melitensis* in sheep and goats and *Br. suis* in pigs). No other specific safeguards are warranted.

8.1.10 Recommendations for tuberculosis

Bovine tuberculosis should be subject to an official control program in the country from which the meat originates. No other specific safeguards other than veterinary post-mortem inspection are necessary.

8.1.11 Recommendations for cysticercosis

All meat must have passed veterinary post-mortem inspection. Before permits are issued to import beef, buffalo meat or pork an official statement on the prevalence of cysticercosis in slaughter animals should be obtained from the veterinary authorities in that country. Where prevalence of cysticercosis in slaughter animals exceeds 5% the importation of chilled meat should not be permitted. Unprocessed meat should be frozen to -18°C.

8.1.12 Recommendations for scrapie and BSE

(a) Meat products intended for human consumption;

No specific safeguards are warranted.

(b) Meat and/or bone meal of ruminant animal origin into for feeding to livestock;

Importation prohibited.

(c) Meat and/or bone meal intended for use as fertiliser;

Must have been heated to 150°C for at least 3 hours.

8.1.13 Recommendations for vesicular exanthema of swine

In the unlikely event that a country could not be certified as free from VES, restrictions should be imposed on the importation of pigmeat. These restrictions should be closely similar to those required for FMD and would include;

(a) All meat and meat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen meat;

(i) Must be carefully boned out and

(ii) Must have reached pH 6.0 before freezing.

(c) Cooked meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of either 80-100°C or higher for 2 to 3 minutes or 70°C or higher for 25 minutes.

(d) Dried meat products;

(i) Must have reached pH 6.0 before drying.

(e) Cured meat products such as salamis not requiring refrigeration;

(i) Must be produced by lactic curing to pH 6.0 or lower.

(f) Hams and bacon;

either (i) Must qualify for official certification as "Prosciutto di Parma" (Parma ham) or have undergone an equivalent 12 month curing process.

or (ii) Must have been subjected to a heat treatment resulting in a core temperature of either 80-100°C or greater for 2 to 3 minutes or 70°C or greater for 25 minutes.

8.1.14 Recommendations for trichinellosis

For countries in which trichinellosis occurs at low prevalence (or is absent), such as North America, western Europe and Australia, no specific safeguards against trichinellosis are required.

For countries of unknown states, or where trichinellosis occurs at a higher prevalence, such as Spain and eastern Europe, additional safeguards need to be imposed. These should include;

(a) All pigmeat and pigmeat products;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

either (ii) Must originate from a herd of origin which can be officially certified as having been free of trichinellosis for at least 3 years and

(iii) Must have been subjected to trichinoscopic examination with negative results.

or (iv) Must have been frozen according to a regimen recognised by OIE as being capable of destroying all trichinae.

or (v) Must have been subject to heat treatment resulting in a core temperature of 60°C.

8.1.15 Recommendations for tularaemia

Prohibit the importation of uncooked wild rabbit and hare meat products from countries or areas not demonstrated free of *F. tularensis*.

8.1.16 Recommendations for viral haemorrhagic disease

If the unintentional introduction of VHD is to be avoided, importation of rabbits should only be permitted from countries or areas certified as free from VHD.

Rabbit meat must originate from farmed animals (i.e. not wild animals) that have passed veterinary ante-mortem and post-mortem inspection.

8.1.17 Recommendations for avian influenza

For countries or areas which cannot be certified as free from highly pathogenic avian influenza ("fowl plague"), restrictions must apply to meat from all avian species;

(a) All meat of avian species;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from highly pathogenic avian influenza ("fowl plague") and inspected regularly by the veterinary authorities who should also be able to certify that no case of highly pathogenic avian influenza ("fowl plague") has occurred within a 10 kilometre radius within the last 2 months.

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute before drying.

8.1.18 Recommendations for Newcastle disease

For countries or areas which cannot be certified as free from ND, restrictions must apply to meat from all avian species;

(a) All meat of avian species;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from ND and inspected regularly by the veterinary authorities who should also be able to certify that no case of ND has occurred within a 10 kilometre radius within the last 2 months.

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 60°C or greater for 30 minutes or 100°C for 1 minute before drying.

8.1.19 Recommendations for infectious bursal disease

For countries or areas which cannot be certified as free from IBD, restrictions must apply to meat and meat products of chickens, turkeys and ducks;

(a) All poultry meat and poultry meat products;

(i) Must originate from birds which have passed veterinary ante-mortem and post-mortem inspection and

(b) Chilled or frozen poultry meat;

(i) Must originate from establishments certified as free from IBD and inspected regularly by the veterinary authorities.

(c) Cooked poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C for 50 minutes, 80°C for 9 minutes or 100°C for 1 minute.

(d) Dried poultry meat products;

(i) Must have been subjected to heat treatment resulting in a core temperature of 70°C for 50 minutes, 80°C for 9 minutes or 100°C for 1 minute before drying.

8.2 Recommendations for mammalian meat products

All mammalian meat and meat products except meat-, blood- and bone-meals imported into New Zealand;

either (i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection[Footnote] and

or (ii) In the case of game animals only must originate from animals which have passed veterinary post-mortem inspection and

(iii) Must have been processed in premises under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iv) Must comply with the specific safeguards (listed in section 8.1) for foot and mouth disease, swine vesicular disease, rinderpest, African swine fever, hog cholera, anthrax, hydatids, leptospirosis, Aujeszky's disease, scrapie and BSE, cysticercosis, vesicular exanthema of swine, trichinellosis, tularaemia and viral haemorrhagic disease of rabbits and

(v) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

Meat-, blood- and bone-meals must comply with the specific safeguards (listed in section 8.1) for anthrax, scrapie and BSE.

8.3 Recommendations for poultry meat products

All poultry (avian) meat and meat products imported into New

Throughout this review it is assumed that before importation is permitted from countries outside the range of our traditional sources of meat products a MAF assessment of the veterinary and inspection services will be carried out. In many instances this assessment will require a visit to the country by a MAF specialist. However, in some cases, acceptance by other major trading partners, once verified officially, may be considered sufficient. For example, if the Australian authorities were to consider that the veterinary and zoosanitary infrastructure of a particular country was sufficiently developed for importation into Australia to be acceptable, then New Zealand MAF should be prepared to adopt a similar stance. See section 3.1 above.

Zealand;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been processed in premises under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iii) Must comply with the specific safeguards (listed in section 8.1) for Newcastle disease and infectious bursal disease and

(iv) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

8.4 Recommendations for crocodile meat products

All crocodile meat and meat products imported into New Zealand;

(i) Must originate from animals which have passed veterinary ante-mortem and post-mortem inspection and

(ii) Must have been processed under the supervision of the veterinary authorities and passed as sound and fit for human consumption and

(iii) Must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

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Appendix I: The Australian risk assessments for TGE in Canadian pigmeat

There has been considerable debate in Australia over the magnitude of the risk that the disease transmissible gastroenteritis (TGE, see section 6.22) could be introduced into Australia in the vehicle of Canadian pigmeat. Estimates from different organisations or interest groups ranged from a low as 1 in 3.3 million to as high as 1 in 15,500.

The method used to calculate the risk was developed first by Dr Tony Forman at the Australian Animal Health Laboratory. His method was refined by a working group comprising Dr Jim Hone, Dr Graeme Garner and Mr Rob Cannon of the Bureau of Rural Resources. This group also carried out a sensitivity analysis of their assessment to test how changes in the basic assumptions would affect the estimation of risk. It is this working group's method which I reproduce here.

The probability (**T**) of TGE being introduced is related to the probability (**p**) that a piece of pigmeat contains the virus and the number of occasions (**n**) that raw pigmeat is fed to pigs. The number of occasions that feeding infected pigmeat causes infection in pigs follows a binomial distribution and the chance of introduction of infection is;

$$T = 1 - (1 - p)^n$$

When **T** is less than 1 in 2,500 this estimate can be approximated as;

$$T = p * n$$

This simplifies the interpretation of the estimate of **T**. A change in **p** or **n** will alter **T** proportionately.

It is further assumed that;

$$p = i * v * s$$

where, **i** is the probability that a Canadian pig was infected with TGE virus at the time of slaughter.

v is the proportion of infected pigmeat still containing infective virus when marketed.

s is the proportion of imported meat amongst all the fresh meat sold.

The working group assumed that 1 in 500,000 (**i** = 0.000002) Canadian pigs are infected with TGE virus at the time of slaughter. This is

considered to be a conservative estimate. The group assumed further that the decay of virus in meat between Canada and Australia would leave 10% (**v** = 0.1) of meat still containing infective virus. As imported pork was considered likely to account for 5% of the market share (as is the case in New Zealand) the working group assumed that all this would go into the fresh pork trade resulting in about 10% (**s** = 0.1) of all fresh meat originating in Canada.

From these assumptions the group calculated;

p = 0.000002 * 0.1 * 0.1
= 0.00000002
[= 2 * 10^{-8} in standard risk assessment notation]
= 1 in 50,000,000

The number of occasions that raw pork is fed to pigs is estimated by;

n = h * m * f * t

where: **h** is the number of small pig herds in Australia
m is the number of unprocessed pork meals eaten per year
f is the proportion of pig farmers who feed scraps to pigs
t is the proportion of occasions on which the scraps contain raw pork.

The working group assumed that only the owners of small herds would feed scraps to their pigs. They assumed that there are 6,147 small pig herds in Australia (**h** = 6,147). They assumed that Australian pig farmers eat 20 meals prepared from unprocessed pork per year (**m** = 20), that 5% of herds are fed scraps (**f** = 0.05) and that 10% of the time these scraps contain raw pork (**t** = 0.1).

The assumptions are based on the following;

(a) There are 6,147 herds with fewer than 200 pigs. This size was chosen as being representative of herds smaller than the national average herd size of 225 pigs.

(b) The estimate of 20 meals per year was derived from the consumption of a family of 4 which was estimated to eat 28 kg of fresh pork annually.

This figure of 28 kg is estimated from the national average per capita consumption of 17 kg of pork per year. The 17 kg comprises 7 kg of fresh pork and 10 kg of processed pork.

(c) If a producer feeds scraps to pigs it is assumed that this occurs at each meal from which scraps are available. However, there is little waste from pork at the precooking stage, so **t** is assumed to be low (10%).

From these assumptions one can calculate;

n = 6,147 * 20 * 0.05 * 0.1
= 614.7

Therefore, the probability that TGE virus will be fed to an Australian pig in any one year is;

T = $(2 * 10^{-8}) * 614.7$
= 0.0000123
[= $1.23 * 10^{-5}$ in standard notation]
= 1 in 81,340

If one assumes that feeding a piece of infected raw meat would cause infection in a pig, then the probability of infected meat being fed is the probability of infection occurring. [This is not a valid assumption, in my opinion. The quantity of meat needed to establish infection in these experiments approached 1 kg per piglet, a mass of meat far in excess of that likely to be fed to a single pig via garbage.]
The sensitivity of the estimate of **T** to changes in **p** or **n** is tabulated below (Table 11).

TABLE 11: THE PROBABILITY (T) OF INTRODUCING TGE WITH DIFFERENT VALUES OF p OR n

	Probability (**p**) that a package of meat contains TGE virus		
	$1 * 10^{-7}$	**$2 * 10^{-8}$**	$5 * 10^{-9}$
Exposure (**n**)			
200	$2.0 * 10^{-5}$	$4.0 * 10^{-6}$	$2.5 * 10^{-6}$
500	$5.0 * 10^{-5}$	$1.0 * 10^{-5}$	$2.5 * 10^{-6}$
615	$6.2 * 10^{-5}$	**$1.2 * 10^{-5}$**	$3.1 * 10^{-6}$
1000	$1.0 * 10^{-4}$	$2.0 * 10^{-5}$	$5.0 * 10^{-6}$
2000	$2.0 * 10^{-4}$	$4.0 * 10^{-5}$	$1.0 * 10^{-5}$

Appendix II: Proposed importation of salami from Hungary

An importer wishes to import salami from Hungary. The salami is made from pork. In drawing up conditions covering the entry of this product one needs to incorporate the relevant specific safeguards in section 8.1 and the general safeguards in section 8.2.

Working through section 8.1 then;

1. Foot and mouth disease. At the time of writing, Hungary had been free of FMD since 1973. Vaccination of pigs is not practised.

2. Swine vesicular disease. Never occurred in Hungary.

3. Rinderpest. Last occurred in 1881.

4. African swine fever. Never occurred in Hungary.

5. Hog cholera. Hungary experienced a single outbreak of HC in August 1990. The outbreak was rapidly contained by stamping out and movement control within a 30 kilometre radius of the infected herd.

   However, to avoid any risk of importing the HC virus in Hungarian salami, official certification must show that it has been prepared from meat from pigs slaughtered at least 6 months after the last case of HC was stamped out.

6. Anthrax. This disease occurs at low sporadic incidence in Hungary. However, pigs are relatively resistant and infection is uncommon in this species. Anthrax was not recorded in pigs in Hungary in 1989. Ante-mortem and post-mortem veterinary inspection provide sufficient safeguards.

7. Hydatids. Hungarian slaughterhouse inspection detects hydatids in pigs. In 1988 (most recent OIE statistics) 22,013 pigs were infested at slaughter out of a total population of over 8.2 million pigs. That is, an incidence of 0.27%.

   This is a low incidence, and as salami is made from muscle, in which hydatid cysts are rare, no additional specific safeguards are required.

8. Leptospirosis. Not relevant to salami.

9. Brucellosis. This disease has not been detected in Hungarian pigs since 1985. It is subject to a national control program.

10. Tuberculosis. Subject to a national control program, tuberculosis has not occurred in Hungary since 1980.

11. Cysticercosis. The occurrence of *C. cellulosae* in Hungary is rare. An official statement of incidence from the Chief Veterinary and Food Control Officer, Dr Ferenc Simor, reported that between 1961 and 1982 the number of cases detected at slaughter ranged from 0 to 7 annually. Since 1982 the number of cases detected each year has been;

1983	23
1984	13
1985	42
1986	0
1987	0
1988	0
1989	4

Even the peak figure in 1985 equates to fewer than 0.0003% of pigs inspected at slaughter. This incidence is well below that which should cause concern.

12. Vesicular exanthema. This disease has not occurred outside the United States and Iceland.

13. Trichinellosis. An official letter from Dr Ferenc Simor, the Chief Veterinary and Food Control Officer, states that trichinellosis has not been detected in Hungary since 1975, despite on-going abattoir surveillance.

14. Tularaemia. Not relevant.

A protocol to cover the importation of salami from Hungary should require;

(a) The meat must originate from pigs which have passed veterinary ante-mortem and post-mortem inspection.

(b) The salami must have been manufactured and processed in premises under the supervision of the veterinary authorities and passed as sound and fit for human consumption.

(c) Official certification that Hungary has for the last 12 months been free of foot and mouth disease, swine vesicular disease, rinderpest, African swine fever and vesicular exanthema of swine.

(d) The salami has been lactic cured to pH 6.0 or lower for a minimum period of 30 days.

(e) The salami must comply with the requirements of the New Zealand Department of Health's Food Regulations (1984).

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