# *Import risk analysis : chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom.*

**March 1999**

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***Ministry of Agriculture and Forestry***
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***Wellington***
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# *Import risk analysis : chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom.*

**Part one: Risk analysis for the importation of chicken meat and chicken meat products into New Zealand**

**Part two: Risk analysis for the importation of Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom into New Zealand**

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Import Risk Analysis : Chicken Meat

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Animal Health and Welfare Group
MAF Regulatory Authority

Approved for general release

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## EXECUTIVE SUMMARY{tc \l1 "EXECUTIVE SUMMARY}

### Chicken Meat and Chicken Meat Products {tc \l2 "Chicken Meat and Chicken Meat Products }

This risk analysis has examined the epidemiology and physical characteristics of various pathogens considered to be of importance to avian health in New Zealand. The analysis has also examined the potential for certain pathogens of human health significance to be introduced into local poultry flocks.

An examination of the literature demonstrates that while it is theoretically possible for some disease agents to be present in chicken meat products, in reality there are very few pathogens for which specific import safeguards are required.

For some diseases, the risks from imported chicken meat products are no greater than those from locally manufactured chicken products. It is not the policy of the New Zealand government to impose sanitary measures on imports which are more stringent than those applying to locally-traded products.

For all chicken meat products significant risks were considered to be associated with certain serotypes of *Salmonella* and with paramyxovirus 1 (Newcastle disease). Specific measures were formulated to reduce the risk of introducing these pathogens.

Further, this analysis identified another technical issue requiring detailed consideration before a judgement could be made regarding the disease risks posed by importations of the product. The specific issue of concern is the risk of introducing exotic strains of infectious bursal disease (IBD) virus.

It was determined that a quantitative risk analysis was necessary to evaluate the risk of introducing exotic strains of IBD virus into backyard flocks in uncooked broiler chicken meat products. The analysis concluded that the risk of introducing IBD virus into backyard poultry would be high. In fact, the probability of IBD introduction approaches 1 if as few as 1% of the chickens consumed annually in New Zealand were to be imported. The analysis also concluded that conventional cooking times and temperatures could not be relied upon to inactivate IBD virus in chicken meat products. Therefore, it is recommended that the importation of chicken meat products should be permitted only from flocks demonstrated to be free from IBD. However, it is recognised that new information or technologies may become available which will warrant re-evaluation of this conclusion.

In addition to the measures against salmonellae, Newcastle disease and IBD, the risk analysis recommended specific safeguards against the introduction of avian bronchitis virus, the agent of big liver and spleen disease, avian influenza and certain other paramyxoviruses.

**Bernard Matthews Food Ltd Turkey Meat Preparations from the United Kingdom**

Turkeys are susceptible to most of the diseases affecting chickens and, therefore, these diseases have not been re-examined in detail for turkey meat. The same importation recommendations apply for most diseases affecting both species. However, there are some diseases which affect turkeys exclusively and detailed consideration was given to these.

As for chicken meat, IBD virus was identified as the main hazard of concern in BMFL turkey meat preparations. The analysis determined that IBD serotype 1 poses a negligible risk in these specific turkey meat preparations. This is in contrast to the proposal to import chicken meat products. Serotype 1 has *never* been found in turkeys in the United Kingdom, and BMFL turkeys have been monitored specifically for this infection.

The analysis further concluded that there is a small risk only that IBD serotype 2 might be introduced through importation of BMFL turkey meat preparations. Furthermore, it is considered highly improbable that IBD serotype 2 would result in disease in any avian species in New Zealand even if it were introduced. The consequence of the introduction of IBD serotype 2 into this country is considered to be limited to possible interference with serological testing for IBD1 in chickens, which could impose additional costs on the poultry industry should the introduction of new testing procedures be necessary.

The overall recommendation of this risk analysis is that BMFL turkey meat preparations from the United Kingdom should be permitted entry to New Zealand provided that they comply with specific safeguards against various salmonellae, avian influenza, Newcastle disease and certain other paramyxoviruses, and turkey viral hepatitis.

# PART ONE: RISK ANALYSIS FOR THE IMPORTATION OF CHICKEN MEAT{tc \l1 "PART ONE: RISK ANALYSIS FOR THE IMPORTATION OF CHICKEN MEAT} AND CHICKEN MEAT PRODUCTS

## 1. INTRODUCTION{tc \l1 "1. INTRODUCTION}

### 1.1 Background{tc \l2 "1.1 Background}

In recent years, there has been considerable interest in importing chicken meat products into New Zealand. Until now, the only poultry meat products that have been permitted entry are those that have been subjected to a specified heat treatment. This policy has been maintained to ensure that New Zealand continues to be free from several serious avian pathogens considered to have the potential for introduction in chicken meat products.

Requests for access to the New Zealand market for uncooked chicken meat have been received from prospective importers, foreign exporters and government trade officials representing other countries. On the other hand, the Poultry Industry Association of New Zealand has raised their concern that the current time/temperature parameters specified for the importation of cooked chicken meat products may be inadequate to inactivate exotic avian viruses, particularly infectious bursal disease virus.

For these reasons the New Zealand Ministry of Agriculture and Forestry (MAF) has carried out an analysis of the risks of introducing exotic avian pathogens through the importation of chicken meat.

For an exotic pathogen to be introduced through imports of chicken meat and to establish in local poultry flocks, *every one* of the following criteria must be met:

- The disease must be present in the country of origin.

- The disease must be present in the flock of origin. The particular birds slaughtered for meat production must have been harbouring an active infection at the time of slaughter, or their carcasses must have become contaminated subsequent to slaughter.

- The disease agent must remain present and viable in those parts of the bird that are traded.

- The disease agent in the tissues traded must remain viable despite pH changes, freeze/thaw cycles, storage and cooking processes.

- The pathogen must be present in tissues at a titre sufficient to cause infection by the oral route.

- Tissues containing the disease agent at an infectious dose must become accessible to susceptible host animals in New Zealand.

- Infection must establish in the host ingesting the tissues and the *infection must spread beyond the index case.*

## 1.2 Commodities considered in the risk analysis{tc \l2 "1.2 Commodities considered in the risk analysis}

This risk analysis examines the disease risks posed by imported meat and meat products derived from chickens (*Gallus gallus*) that have passed ante-mortem and post-mortem inspection in slaughter and processing plants which operate effective Good Management Practice (GMP) and Hazard Analysis and Critical Control Point (HACCP) programmes.

This risk analysis considers only broiler chickens which are slaughtered at 5 - 7 weeks of age; culled-for-age layer or breeder birds are not considered.

The commodities covered in this risk analysis are :
whole chicken carcasses; uncooked, unskinned, eviscerated, not containing giblets;
bone-in chicken meat products such as wings or legs;
boneless chicken meat products such as breasts, boned-out thighs;
cooked whole chicken carcasses or cooked chicken meat;
reconstituted chicken meat products comprised of chicken meat and skin.

## 1.3 Risk analysis methodology{tc \l2 "1.3 Risk analysis methodology}

This risk analysis consists of the following steps:

1. *Hazard identification*: process of identifying the diseases which might conceivably be introduced in the commodity in question.

2. *Risk assessment:* process by which identified diseases of concern are evaluated in terms of the likelihood that they might be introduced in the particular commodity under consideration and the consequences of such introduction.

3. *Risk management*: the formulation of safeguards which are considered appropriate to minimise or eliminate risks, where necessary.

## 1.4 Proposed safeguards

Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

## 2. HAZARD IDENTIFICATION{tc \l1 "2. HAZARD IDENTIFICATION}: DISEASES OF CONCERN FOR THE COMMODITY

### 2.1 Diseases reported to affect avian species{tc \l2 "2.1 Diseases reported to affect avian species}

A risk analysis for the importation of ratites and ratite products[1] identified a list of avian diseases exotic to New Zealand which were considered to be of regulatory concern. It is not considered necessary to repeat that work, and so the list of avian diseases determined to be of concern in the ratite risk analysis has been used as a starting point for this analysis examining chicken meat (see Table 2.1).

Before embarking on this analysis, the Department of Conservation, the Ministry of Health and the Poultry Industry Association of New Zealand were asked to provide MAF with a list of chicken diseases that they considered should be included. As a result, several agents and disease syndromes that were not covered by the ratite risk analysis are included in this analysis; *Salmonella* Enteritidis phage type 4, *Salmonella* Typhimurium definitive phage type 104, avian polyomavirus and avian intestinal spirochaetosis. These agents and disease syndromes are shown in Table 2.5.

Although MAF's primary concern is animal health, this ministry also has an obligation under section 22 of the Biosecurity Act 1993 to consider human health issues when assessing importation proposals. However, it must be emphasised that this risk analysis does not represent the views or recommendations of the Ministry of Health, which is currently responsible for setting public health standards for imported food.

### 2.2 Diseases reported to infect chickens{tc \l2 "2.2 Diseases reported to infect chickens}

The diseases listed in Table 2.1 were evaluated in order to determine which diseases would be taken into further consideration. All of the disease agents were assessed as to whether or not they had been reported to infect chickens. This resulted in:

- a list of avian diseases which have not been reported to infect chickens and which would NOT be subjected to further consideration. This list is presented in Table 2.2.

- a list of avian diseases which have been reported to infect chickens and which would be subject to further consideration. This list is presented in Table 2.3.

### 2.3 Chicken diseases of concern with the potential for transmission in chicken meat{tc \l2 "2.3 Chicken diseases of concern with the potential for transmission in chicken meat}

The diseases listed in Table 2.3 were evaluated in order to determine which diseases would be taken into further consideration. The agents thought to be capable of survival in or on chicken meat, as well as those agents excreted in the faeces were considered to have the potential for transmission in the commodity; these agents are listed in Table 2.4.

A number of disease agents were not considered to be capable of transmission in chicken meat for various reasons, including:

- the disease is transmitted only by arthropods: aegyptianellosis[2], leucocytozoonosis[3], *Plasmodium* infection[3], *Trypanosoma* infection[3], Alfuy virus[4], equine encephalomyelitis (Eastern and Western)[4], Murray Valley encephalitis[4].

- the disease agent is an external parasite: Argasid ticks[2], Ixodid ticks.[2]

- the pathogen is not found in any part of the edible carcass: verminous encephalitis[2], vesicular stomatitis.[5]

- the agent is non-contagious: zygomycosis.[6]

- the agent is fragile and dies quickly outside the living animal host: infectious coryza[3], *Mycoplasma iowae*[3], lymphoproliferative disease[4], myelocytomatosis[4], reticuloendotheliosis[4], rabies.[7]

This assessment resulted in a list of diseases of concern that are thought to have the potential to be transmitted in chicken meat (Table 2.4).

The diseases in Table 2.5 were also considered in the risk analysis at the request of other organisations or added during the technical review process.

The diseases listed in Tables 2.4 and 2.5 were subjected to a qualitative risk assessment to determine the need for, and type of, safeguards. In some cases quantitative analysis was also carried out.

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## Table 2.1: Avian diseases exotic to New Zealand and of regulatory concern.

| Bacteria | Fungi, Parasites, Protozoa, Other | Viruses | Viruses continued |
|---|---|---|---|
| Aegyptianellosis | Zygomycosis | Alfuy virus | Japanese encephalitis |
| Anthrax | Argasid ticks | Amazon tracheitis | Lymphoproliferative disease |
| Avian chlamydiosis (exotic strains) | Balantidiasis | Astrovirus infection of turkeys | Macaw wasting disease (Proventricular dilatation) |
| Avian spirochaetosis | Filariae | Astrovirus infection of ducks | Marek's disease |
| Infectious coryza | *Haemoproteus* infection | Avian adenovirus type II | Murray Valley encephalitis |
| Intracellular infection in ducks | Hexamitiasis | Avian infectious bronchitis | Myelocytomatosis |
| *Mycoplasma iowae* infection | Ixodid ticks | Avian rhinotracheitis | Newcastle disease |
| *Ornithobacterium rhinotracheale* infection | Leucocytozoonosis | Beak and feather disease | Pacheco's disease |
| Q fever | *Libyostrongylus* infection | Big liver and spleen disease | Papillomas in finches |
| *Salmonella arizonae* | Ostrich tapeworm | Borna disease | Paramyxovirus 2 infection |
| *Salmonella gallinarum* | *Plasmodium* infection | Bunyavirus infection | Paramyxovirus 3 infection |
| *Salmonella pullorum* | Sarcosporidiosis (exotic species) | Cholangio-hepatitis virus | Paramyxovirus 5 infection |
| Tularaemia | *Trypanosoma* infection | Coronaviral enteritis of turkeys | Paramyxovirus 7 infection |
| Turkey coryza | Verminous encephalitis | Crimean-Congo haemorrhagic fever | Paramyxovirus 8 infection |
| | Ostrich fading syndrome | Derzsy's disease of geese | Paramyxovirus 9 infection |
| | Encephalopathy | Duck hepatitis | Quail bronchitis virus infection |
| | | Duck hepatitis B virus infection | Rabies |
| | | Duck virus enteritis | Reticuloendotheliosis |
| | | Equine encephalomyelitis | Rift Valley fever |
| | | Haemorrhagic nephritis and enteritis of geese | Ross River virus infection |
| | | Heron hepatitis B virus | Turkey meningoencephalitis |
| | | Herpesvirus infection of pigeons and wild birds | Turkey viral hepatitis |
| | | Highlands J virus infection | Vesicular stomatitis |
| | | Highly pathogenic avian influenza | Wesselsbron disease |
| | | Infectious bursal disease | |

## Table 2.2: Avian diseases of concern that have not been reported in chickens.

| Bacteria | Fungi, Parasites, Protozoa, Other | Viruses | Viruses continued |
|---|---|---|---|
| Anthrax | Balantidiasis | Amazon tracheitis | Quail bronchitis virus infection |
| Intracellular infection in ducks | Filariae | Astrovirus infection of turkeys | Rift Valley fever |
| | *Haemoproteus* infection | Astrovirus infection of ducks | Ross River virus infection |
| | Hexamitiasis | Beak and feather disease | Turkey meningoencephalitis |
| | *Libyostrongylus* infection | Borna disease | Turkey viral hepatitis |
| | Ostrich tapeworm | Bunyavirus infection | Wesselsbron disease |
| | Ostrich fading syndrome | Cholangio-hepatitis virus | |
| | Encephalopathy | Coronaviral enteritis of turkeys | |
| | | Crimean-Congo haemorrhagic fever | |
| | | Derzsy's disease of geese | |
| | | Duck hepatitis | |
| | | Duck hepatitis B virus infection | |
| | | Duck virus enteritis | |
| | | Haemorrhagic nephritis and enteritis of geese | |
| | | Heron hepatitis B virus | |
| | | Herpesvirus infection of pigeons and wild birds | |
| | | Highlands J virus infection | |
| | | Japanese encephalitis | |
| | | Macaw wasting disease (Proventricular dilatation) | |
| | | Pacheco's disease | |
| | | Papillomas in finches | |
| | | Paramyxovirus 5 infection | |
| | | Paramyxovirus 7 infection | |
| | | Paramyxovirus 8 infection | |
| | | Paramyxovirus 9 infection | |

## Table 2.3: Avian diseases of concern that have been reported in chickens.

| Bacteria | Fungi, Parasites, Protozoa | Viruses |
|---|---|---|
| Aegyptianellosis | Zygomycosis | Alfuy virus infection |
| Avian chlamydiosis | Argasid ticks | Avian adenovirus type II |
| Avian spirochaetosis | Ixodid ticks | Avian infectious bronchitis |
| Infectious coryza | Leucocytozoonosis | Avian rhinotracheitis |
| *Mycoplasma iowae* infection | *Plasmodium* infection | Big liver and spleen disease |
| *Ornithobacterium rhinotracheale* infection | Sarcosporidiosis | Equine encephalomyelitis (Eastern and Western) |
| Q fever | *Trypanosoma* infection | Highly pathogenic avian influenza |
| *Salmonella arizonae* | Verminous encephalitis | Infectious bursal disease (exotic strains) |
| *Salmonella gallinarum* | | Lymphoproliferative disease |
| *Salmonella pullorum* | | Marek's disease |
| Tularaemia | | Murray Valley encephalitis virus infection |
| Turkey coryza | | Myelocytomatosis |
| | | Newcastle disease |
| | | Paramyxovirus 2 infection |
| | | Paramyxovirus 3 infection |
| | | Rabies |
| | | Reticuloendotheliosis |
| | | Vesicular stomatitis |
| | | |

**Table 2.4: Agents that may have the potential for transmission in chicken meat**

| Bacteria | Protozoa | Viruses |
|---|---|---|
| Avian chlamydiosis | Sarcosporidiosis | Avian adenovirus type II |
| Avian spirochaetosis | | Avian infectious bronchitis (exotic strains) |
| *Ornithobacterium rhinotracheale* infection | | Avian rhinotracheitis |
| Q fever | | Big liver and spleen disease |
| *Salmonella arizonae* | | Highly pathogenic avian influenza |
| *Salmonella gallinarum* | | Infectious bursal disease (exotic strains) |
| *Salmonella pullorum* | | Marek's disease |
| Tularaemia | | Newcastle disease |
| Turkey coryza | | Paramyxovirus 2 infection |
| | | Paramyxovirus 3 infection |

**Table 2.5: Agents and syndromes included in the chicken meat risk analysis at the request of other organisations**

| Agent or syndrome | Request by |
|---|---|
| Avian polyomavirus | Department of Conservation |
| *Salmonella* Enteritidis PT 4 | Ministry of Health |
| *Salmonella* Typhimurium DT 104 | Ministry of Health |
| Intestinal spirochaetosis | Poultry Industry Association |

# 3. QUALITATIVE RISK ASSESSMENT{tc \l1 "3. QUALITATIVE RISK ASSESSMENT}

## *3.1. BACTERIAL INFECTIONS*{tc \l2 "*3.1. BACTERIAL INFECTIONS*}

### 3.1.1 AVIAN CHLAMYDIOSIS{tc \l3 "3.1.1 AVIAN CHLAMYDIOSIS}

#### 3.1.1.1 Aetiology

*Chlamydia psittaci* is a rickettsia in the family Chlamydiaceae. Chlamydiae are intracellular parasites and have evolved a specialised form adapted for extracellular survival.[1, 2, 3]

Zoonotic strains of *C. psittaci* are also responsible for ornithosis in birds.[4] Avian chlamydial isolates causing disease in birds and humans are antigenically unrelated to mammalian isolates.[5] Mammalian strains are not thought to cause infections in birds.[6]

#### 3.1.1.2 The disease

In general, chlamydiae are ubiquitous and rarely kill their hosts. Persistent, clinically inapparent infection is a feature of chlamydiosis.[2, 3] Avian chlamydiosis is a contagious systemic disease most likely to be fatal in younger birds. Infection in older birds can be subclinical unless birds are stressed. The incubation period varies considerably from 3 days to 106 days.[6, 7]

Strains of both high and low virulence exist and both spread rapidly through a flock. Studies show that more than 90% of birds may develop antibodies to infection by the time clinical signs are seen.[6]

Chlamydiosis is seen most frequently in psittacine birds but has also been reported in domestic poultry, particularly turkeys and ducks.[8, 9] Chickens are rarely affected, and most infections are inapparent and transient.[2, 6, 10, 11]

#### 3.1.1.3 Physical and chemical stability

Chlamydiae in tissue homogenates are inactivated by a heat treatment of 5 minutes at 560C. The organisms may be preserved at -200C or below, although freezing and subsequent thawing causes a loss of infectivity.[6, 7] The pH range in which chlamydiae can survive is pH 7 - 8,[5] which is outside the normal pH range of chicken meat (the ultimate pH of breast muscle is 5.7 to 5.9).[12]

#### 3.1.1.4 Epidemiology

*C. psittaci* has been demonstrated in over 130 species of birds, including turkeys, ducks, pigeons, geese, pheasants and chickens.[6, 7] Wild birds act as reservoirs of chlamydiae.[6]

The usual site of infection is the intestinal tract.[3] Infection occurs via the respiratory route from airborne faeces or respiratory exudates. Birds are the principal vectors, either inapparently infected carriers or secondarily infected species that serve to amplify spread during migration or

feeding. Egg transmission to chicks occurs rarely from contamination of the shell with infective respiratory exudates or faeces.[3, 6, 7, 13]

Pet birds can also act as reservoirs of *C. psittaci*. Diseased and subclinically infected birds shed chlamydiae intermittently, and are therefore a potential threat to human and animal health.[9] In the United States, 70% of reported human cases resulted from exposure to caged birds.[14]

Humans are not thought to be susceptible to infection caused by ingestion of chlamydiae. Psittacine birds are the main source of infection, but outbreaks have occurred where the sources were other birds including live commercial poultry.[5, 9]

### 3.1.1.5 Occurrence

Avian chlamydiosis occurs world-wide.[6, 15] The disease is present in wild birds in New Zealand, although infection has not been found in native psittacines.[8, 16] There are no reports of *C. psittaci* affecting poultry in this country.

### 3.1.1.6 Effect of introduction

Avian chlamydiosis can cause economic losses to poultry producers through carcass condemnation at slaughter, reduced egg production and costs associated with control.[6, 9]

Avian chlamydiosis presents a significant public health risk. Strains from psittacines are most likely to cause disease in humans, although infected live turkeys, ducks and pigeons can also transmit disease. Chickens are of little importance as public health hazards.[6, 10]

### 3.1.1.7 Risk of introduction in chicken meat

Chlamydiae may be found in blood within 3 days post-infection. Infection may persist in the kidneys and liver for up to 2 months.[3]

As chlamydiae may be excreted in the faeces, there is a possibility of contamination of carcass skin by infectious faeces. However, as they are intracellular parasites, unlike bacteria of public health concern, chlamydiae will not multiply on the carcass surface. Chlamydiae in blood may be distributed to edible parts of the carcass. However, the organisms do not survive in the normal pH range of poultry muscle.

Kidney, respiratory and liver tissues are most likely to harbour chlamydiae, particularly as the organisms can persist in deep-frozen tissues. While respiratory tissue is largely removed from carcasses,[17] and livers are only present in carcasses as giblets, which are specifically excluded in this risk analysis, kidneys are left in carcasses after processing.

As the disease does not spread by the oral route, infection would not establish even if infected raw tissues were consumed by a susceptible host. Therefore the risk of introducing exotic strains of chlamydiae in imported chicken meat products is considered to be negligible.

### 3.1.1.8 Recommendations for risk management

No specific safeguards are required.

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### 3.1.2 BORRELIOSIS{tc \l3 "3.1.2 BORRELIOSIS}

#### 3.1.2.1 Aetiology

*Borrelia anserina* is an anaerobic bacterium.[1, 2] Borreliae are parasites of argasid ticks and lice.[2]

#### 3.1.2.2 The disease

Borreliosis is a tick-borne disease of birds. It is an acute, septicaemic disease with obvious clinical signs. Chicks less than 3 weeks of age are most susceptible while older birds are usually more resistant. Chicks infected at 1-day-old experience a prolonged spirochaetaemia lasting 2-3 weeks compared with 3-5 days in older birds.[1, 3, 4] The incubation period is 3-12 days. Morbidity and mortality are highly variable.[1]

In a 10 year retrospective study carried out in Nigeria, all recorded cases of borelliosis occurred in backyard chicken flocks. There were no cases in commercial flocks. *Argas persicus* ticks were found on all chickens in the affected flocks and in the chicken houses.[5]

#### 3.1.2.3 Physical and chemical stability

*Borrelia anserina* is not resistant outside the host.[1] The organism can survive in carcasses for up to 31 days at 00C and for 3-4 weeks in serum at 40C.[1] It may survive for 1 year in chicken blood stored at -200C.[6]

#### 3.1.2.4 Epidemiology

Chickens, turkeys, pheasants, ducks, geese, grouse, parrots and canaries may be infected, but pigeons, guinea fowl and mammals are resistant to infection.[1, 7, 8]

Borelliosis can be transmitted by any means where blood, excreta or tissues from an infected live or recently dead bird comes into contact with a susceptible bird. Infection can be established by oral, ocular and nasal routes and virulent strains can penetrate unbroken skin. Cannibalism, ingestion of blood or droppings, either directly or indirectly via contaminated feed and water, or use of syringes and needles for multiple birds are all means by which the disease can be transmitted.[1] The incidence of carrier birds appears to be relatively low or non-existent.[1, 9, 10]

The disease is transmissible indirectly via blood-sucking arthropods, particularly the fowl tick, *Argas persicus*. *Argus* ticks act as the principal reservoir of *B. anserina*. Ticks become infective 6-7 days after biting a host and can harbour infection for up to 488 days. As the organism cannot survive for long periods in either the bird or the environment, it relies on the tick for its continued existence.[1]

While other vectors, such as mosquitoes and fowl mites, may play a role in short-term disease transmission, they are unimportant in maintaining infection.[1, 2, 3, 9]

#### 3.1.2.5 Occurrence

Borelliosis occurs worldwide, most frequently in tropical and subtropical countries and in extensive husbandry systems.[2, 8, 11] Occurrence in temperate areas or intensively managed flocks is uncommon.[2] Neither *B. anserina* nor the fowl tick *A. persicus* are found in New Zealand.

### 3.1.2.6 Effect of introduction

Borelliosis causes significant economic losses in areas where it is endemic.[1]

### 3.1.2.7 Risk of introduction in chicken meat

Borreliae are present in blood only during the acute stages of infection,[1, 2] and disappear within 9 days post-infection. Spirochaetes may also be found in liver, kidney and spleen.[8, 10]

The organisms may survive in frozen tissues derived from infected birds. As the disease can be transmitted orally, there is a possibility that uncooked scraps from infected chicken carcasses could transmit spirochaetosis to backyard chickens. However, the organism is reliant on *Argus* species ticks as reservoir hosts. These ticks are not present in New Zealand. Even if infection were introduced, it is unlikely that spirochaetosis would establish in either wild or domestic avian populations.

Because borelliosis is a disease of extensively raised chickens[1] there is little likelihood that meat produced from commercial intensively managed flocks would present a risk. It is also a disease of very young birds so it is unlikely that slaughter-age chickens would be infected with *B. anserina*.

The risk of introduction of borelliosis in imported chicken meat products is considered to be negligible.

### 3.1.2.8 Recommendations for risk management

No specific safeguards are required.

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### 3.1.3 AVIAN INTESTINAL SPIROCHAETOSIS{tc \l3 "3.1.3 AVIAN INTESTINAL SPIROCHAETOSIS}

Note : this disease was considered in this risk analysis at the request of the Poultry Industry Association of New Zealand.

#### 3.1.3.1 Aetiology

An unnamed intestinal spirochaete of chickens has been shown to be mildly pathogenic for young chicks and adult hens. It is related to *Serpulina (Treponema) hyodysenteriae* of swine.(1)

#### 3.1.3.2 The disease

Avian intestinal spirochaetosis (AIS) is a subacute to chronic non-septicaemic disease of broiler breeder and layer flocks, characterised by spirochaetes in the caecum and/or rectum, and variable clinical illness, morbidity and mortality.(2) There can be chronic diarrhoea, with wet greasy droppings, reduction in egg production and quality.(1)

#### 3.1.3.3 Physical and chemical stability

A related species, *Serpulina hyodysenteriae*, survives in faeces for 2 months, especially in waste pits.(2)

#### 3.1.3.4 Epidemiology

AIS is more likely to be associated with free range chickens than battery management although it is seen in caged hens.(1) A survey of 52 broiler, broiler breeder or layer flocks in Queensland, New South Wales, Tasmania, Victoria and South Australia showed 55% of broiler breeder and layer flocks were infected. As with previous findings in Western Australia and Europe, no broiler (meat) flocks were infected.(3) Half the isolates tested belonged to two species of intestinal spirochaetes which are known to be pathogens of poultry (*S. intermedia and S. pilosicoli*).(3)

#### 3.1.3.5 Occurrence

AIS has been identified in Europe, North America, and Australia.(2) Since 1986, AIS has been reported in commercial laying chickens in the Netherlands, England, and the USA. The disease is routinely diagnosed in Europe; a survey showed that 27% of chicken flocks with intestinal disorders were positive for intestinal spirochaetes, while only 4% of flocks without enteric signs were positive.(2) Sporadic cases of AIS also have been identified in domestic turkeys, broilers, and broiler breeders in recent years.(2)

In a survey of zoo animals in the USA, the highest infection rates were found in rheas (32%) and birds of the Anseriformes [ducks, geese swans] (46%).(2)

*S. pilosicoli* has been isolated in pig faeces submitted to Animal Health laboratories in New Zealand. *S. intermedia* has not been identified in New Zealand, but it has only been relatively recently recognised as a new species and surveys have not been carried out.(4)

### 3.1.3.6 Effect of introduction

The introduction of *S. intermedia* into New Zealand poultry flocks, assuming it is not already present, could result in disease. There could be a reduction in egg production by 10%, higher food conversion, and in young birds there could be retarded growth and stunting and delayed onset of lay.(1)

### 3.1.3.7 Risk of introduction in chicken meat

The organs harbouring the pathogen (that is, the intestines) are removed at slaughter. Furthermore, AIS is rarely seen in flocks which do not have intestinal disorders, and has only been seen sporadically in broiler flocks.

It is concluded that the risk of introduction of the AIS agent in imported chicken meat products is negligible.

### 3.1.3.8 Recommendations for risk management

No specific safeguards are required.

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### 3.1.4 *ORNITHOBACTERIUM RHINOTRACHEALE*{tc \l3 "**3.1.4 *ORNITHOBACTERIUM RHINOTRACHEALE***}

#### 3.1.4.1 Aetiology

Gram-negative bacterium, *Ornithobacterium rhinotracheale*.[1]

#### 3.1.4.2 The disease

*O. rhinotracheale* is associated with respiratory disease, decreased growth, and mortality in chickens and turkeys.[1, 2] Although 3-4 week old chickens can be infected, infections are most common in broiler breeders between 24 and 52 weeks of age, especially during peak egg production.[2]

In turkeys, infections have been seen in 2-week-old birds, but most severe lesions are in birds older than 14 weeks, and in breeders. Clinical disease has been reported in 23-week-old turkeys in Germany and 42-week-old turkeys in the USA.[2]

Lesions are most common in the respiratory tract and include pneumonia, pleuritis and airsacculitis.[1, 2]

#### 3.1.4.3 Epidemiology

*O. rhinotracheale* infections occur naturally in chickens and turkeys. There are also reports of the organism being found in other bird species including rooks, chukkar, pheasants, pigeons and a partridge. There is no known public health significance.[2]

Infection by *O. rhinotracheale* can only be spread by the respiratory route. The trachea, lungs and air sacs are the best tissues from which to isolate the organism.[2]

#### 3.1.4.4 Occurrence

*O. rhinotracheale* has been isolated from birds in Europe, Israel, South Africa and the USA, and it is considered that it is probably distributed worldwide.[2] The organism has not been reported in New Zealand.

#### 3.1.4.5 Effect of introduction

Disease in chickens and turkeys due to infection with *O. rhinotracheale* is an emerging problem in countries where it has been diagnosed. Economic losses can be considerable when breeder flocks are infected. A mortality rate of 2-11% may occur in affected chicken and turkey flocks. Further losses may occur because of condemnation of broiler carcasses during processing.[2]

### 3.1.4.6 Risk of introduction in chicken meat

The disease mainly affects older birds, especially breeders.

Clinically healthy broilers which pass ante- and post-mortem examination are unlikely to be carrying the organism in their carcass tissues. Moreover, respiratory tissues are largely removed from chicken carcasses at slaughter.(3)

*O. rhinotracheale* infection has been shown to spread only by the respiratory route.

The risk presented by the importation of chicken meat products into New Zealand is considered to be negligible.

### 3.1.4.7 Recommendations for risk management

No specific safeguards are required.

## References


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### 3.1.5 Q FEVER{tc \l3 "3.1.5 Q FEVER}

#### 3.1.5.1 Aetiology

A rickettsial infection caused by *Coxiella burnetii*.[1]

#### 3.1.5.2 The disease

Infection in animals is usually inapparent.[2] Humans and guinea pigs are the only species which have been shown to develop illness following infection with *C. burnetii*.[1, 2, 3]

Import restrictions are currently imposed on ruminants, ruminant semen and ruminant embryos imported into New Zealand from countries where Q fever is endemic. The most compelling reason to maintain Q fever safeguards is to protect public health in New Zealand. Q fever is a major zoonosis, but presents a minimal threat to domestic animal production. The threat to wildlife is unknown. Chickens are susceptible to infection.[4]

#### 3.1.5.3 Physical and chemical stability

The organism is very resistant to desiccation and to physical and chemical inactivation.[2, 3, 5, 6] Heat treatments for the destruction of the organism in liquids are: 62.80C for 30 minutes, 650C for 15 minutes, 71.70C for 15 seconds, 750C for 8 seconds and 1000C for 7 seconds.[6, 7] *Coxiella* will resist a temperature of 600C for 1 hour.[8]

#### 3.1.5.4 Epidemiology

*C. burnetii* can infect many species, including ticks, rabbits, deer, mice, pigeons, sparrows, cattle, sheep, goats, horses, pigs, dogs, cats, poultry and humans.[3, 6, 8, 9]

The agent is maintained in a wild-life reservoir primarily involving rodents and birds. Infection is transmitted to domestic animals by ticks.[2, 6, 10, 11] Tick-independent cycles of infection can develop in ruminant livestock. However, the virulence of the organism appears to diminish in infection cycles where ticks are not involved.[10] Further sources of infection are tick faeces, contaminated feed, water and litter.[6, 10]

Cattle, sheep and goats are the chief sources of infection for humans.[2, 5, 10, 12] Humans are usually infected by inhalation of the organism. Ingestion is a poor route for infection with this organism.[3] However, humans may be infected by the ingestion of infected milk.[9]

Chickens excrete the organisms in their faeces from the seventh to the 40th day post-infection.[8] It is thought that birds are not directly involved in the transmission of disease to humans.[9] Carnivorous birds probably acquire the infection from infected prey. Granivorous and insectivorous birds feed and roost in close proximity to cattle and probably become infected via the aerosol route.[5]

#### 3.1.5.5 Occurrence

Q fever occurs in virtually all countries[3] although New Zealand is free.[8]

### 3.1.5.6 Effect of introduction

Should Q fever establish in New Zealand, its effects on livestock would probably be minimal. Some clinical signs, such as abortions and infertility, would be expected in a large naive animal population.[4, 7]

Q fever is a zoonosis and occurs particularly among slaughterhouse workers. People working with livestock are also at risk.[2, 7, 13] Clinical disease in humans is relatively infrequent and infection is often asymptomatic. Clinical cases of Q fever seem to occur under unusual circumstances linked with exposure to large infectious doses.[14]

### 3.1.5.7 Risk of introduction in chicken meat

During the bacteraemic phase of the disease, *C. burnetii* is carried to all organ systems. While slaughterhouse workers are 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. Humans may occasionally become infected by eating infected food-stuffs, but this is uncommon.[6, 7, 12, 13]

Ingestion is a poor route for transmission of Q fever.[3] It is only milk that has been recognised as acting as a vehicle for oral infection.[9]

The risk that *C. burnetii* could be introduced to New Zealand in chicken meat products is considered to be negligible.

### 3.1.5.8 Recommendations for risk management

No specific safeguards are required.

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## 3.1.6 AVIAN SALMONELLOSIS{tc \l3 "3.1.6 AVIAN SALMONELLOSIS}

### 3.1.6.1 Aetiology

Modern nomenclature[1, 2] classifies the genus *Salmonella* into only two species: *S. enterica* and *S. bongori*. *Salmonella enterica* is divided into six subspecies, some of which correspond to previous subgenera. These subspecies are:

| | |
|---|---|
| Subspecies I | = subspecies *enterica* |
| Subspecies II | = subspecies *salamae* |
| Subspecies IIIa | = subspecies *arizonae* |
| Subspecies IIIb | = subspecies *diarizonae* |
| Subspecies IV | = subspecies *houtenae* |
| Subspecies VI | = subspecies *indica* |

Strains of salmonellae are classified into serovars. The serovars most commonly causing infections in humans and food animals belong to subspecies 1. The other serovars are common in reptiles, although some serovars of subspecies *arizonae* are associated with disease in poultry and sheep. According to this latest nomenclature *Salmonella typhimurium* is now known as *Salmonella enterica* subspecies *enterica* serovar Typhimurium. For the sake of brevity the older nomenclature will be used in this chapter.

In veterinary literature a distinction is usually made between infections caused by the two non-motile host adapted serovars of *Salmonella pullorum* (pullorum disease) and *Salmonella gallinarum* (fowl typhoid), the arizonae group of salmonellae (arizonosis) and the remainder (paratyphoid infection).[1, 3] Following this convention this chapter is divided into three main sections covering:

1. The two non-motile serotypes, which are generally host-specific for poultry.

   (i) *Salmonella pullorum* which causes pullorum disease, an acute systemic disease of chicks and poults.

   (ii) *Salmonella gallinarum* which causes fowl typhoid, an acute or chronic septicemic disease that most often affects growing or mature chickens and turkeys.[3]

2. The motile *Salmonella* serotypes collectively referred to as paratyphoid salmonellae.[3]

   Although there are over 2,300 serotypes, only about 10% of these have been isolated from poultry. Moreover, an even smaller subset of serotypes account for the vast majority of poultry *Salmonella* isolates. Several serotypes are consistently found at a high incidence. For example in the USA, *S. typhimurium, S. enteritidis, S. heidelberg, S. hadar, S. agona, S. reading, S. saintpaul* and *S. senftenberg* are regularly isolated from chickens.[3] In New Zealand *S. agona, S. infantis* and *S. typhimurium* routinely account for over 96% of isolates from

broilers. *Salmonella enteritidis* has not been isolated from poultry in this country.[4, 5, 6]

This diverse group of serotypes is principally of concern as a cause of foodborne disease in humans. Although infections are common in poultry they seldom cause acute systemic disease except in highly susceptible young birds subjected to stressful conditions.[3] The two most important paratyphoid salmonellae in cases of human infections are *S. enteritidis* and *S. typhimurium*.[7]

The focus of this risk analysis will be on these latter two serotypes because any conclusions and recommendations reached will apply equally to other paratyphoid salmonellae identified as potential hazards.

3. The motile salmonellae of *S. enterica* subspecies *enterica* serovar Arizona[1, 2], previously referred to as subgenus *Salmonella arizonae*. These cause arizonosis and have been of particular economic significance in turkeys.[3]

## Section I: *Salmonella pullorum* and *Salmonella gallinarum*{tc \l4 "*Salmonella pullorum* and *Salmonella gallinarum*}

### I: 3.1.6.2 The disease

These non-motile salmonellae are highly host-adapted and primarily affect chickens and turkeys. Whilst other birds such as quail, pheasants, ducks, peacocks and guinea fowl are susceptible, significant clinical signs are seldom seen in these species.[8]

Occasional infections with *S. pullorum* have been seen in humans following massive exposure through ingesting contaminated foods. Recovery is rapid without treatment. *Salmonella gallinarum* is rarely isolated from humans and like *S. pullorum*, is of little public health significance.[8,9]

### I: 3.1.6.3 Physical and chemical stability

In general, the resistance of these organisms is about the same as that of other members of the paratyphoid group. They may survive for several years in a favourable environment, but they are less resistant to heat and adverse environmental factors than other paratyphoid salmonellae. *Salmonella gallinarum* has been shown to retain viability for up to 43 days when subjected to daily freezing and thawing.[8] Further information on physical and chemical stability for salmonellae in general is provided in section II: 3.1.6.3 under paratyphoid salmonellae.

### I: 3.1.6.4 Epidemiology

Morbidity and mortality are highly variable in chickens and turkeys and are influenced by age, strain susceptibility, nutrition, flock management and characteristics of exposure. Mortality from pullorum disease may vary from 0% to 100%, with the greatest losses occur during the second week after hatching, followed by a rapid decline between the third and fourth week. Mortality

due to fowl typhoid has been recorded as ranging from 10% to 93%. Morbidity is normally higher than mortality.[8]

Birds of any age may be infected with either organism but fail to show grossly discernable lesions. Acute pullorum disease and fowl typhoid are characterised by systemic infections and the causative organisms can be isolated from most body tissues including tendon sheaths and joints such as the hock or wing. In chronically affected birds these organisms may be isolated from reproductive tissue, the peritoneum, various internal organs including the intestines, synovial fluid and the interior of the eye.[8] In older, chronically infected birds, *S. pullorum* is most frequently recovered from the ovaries and only exceptionally from other organs and tissues, including the alimentary tract. In contrast, *S. gallinarum* is most frequently found in the liver and faeces of carrier animals.[10]

The primary role of egg transmission for both of these disease has been recognised for many years, with infection rates as high as 33% of all eggs laid by an infected hen being recorded. Contact transmission from infected chicks or pullets can be important. Faeces from infected birds, contaminated feed, water and litter can all be sources of infection. Various fomites have been implicated and humans, wild birds, mammals and flies may be important mechanical vectors.[8]

Live vaccines have been developed for use against *S. gallinarum* using the 9R strain. This strain can survive in vaccinated birds for many months and may be transmitted transovarially and between birds. Vaccination may reduce flock losses but will not prevent infection with field strains.[10]

Both pullorum disease and fowl typhoid can be eradicated by establishing breeding flocks that are free of these diseases and hatching and rearing their progeny in suitable premises to prevent direct and indirect contact with infected chickens and turkey.[8] These eradication programs have substantially eliminated *S. pullorum* from commercial poultry production in most of the world. *Salmonella gallinarum* has been virtually eliminated from most Western countries but remains a problem in areas of Central and South America, Africa and the Middle East.[9] There have also been a few outbreaks in commercial poultry in Germany and Denmark in recent years.[8]

### I: 3.1.6.5 Occurrence

*Salmonella gallinarum* has never been reported in New Zealand and *S. pullorum* has been eradicated from commercial flocks, with the last case being reported in 1985. Ongoing monitoring has confirmed that commercial flocks have remained free.[11] Although backyard flocks were not directly involved in the eradication program, many are established from layers retired from commercial flocks so that it is considered unlikely that they would be a source of *S. pullorum.*[12] Both diseases still exist in backyard flocks in the USA and experience there has indicated that the usual separation of commercial and non-commercial poultry is effective in preventing transmission of *S. pullorum* or *S. gallinarum* between these populations. Nevertheless, as infected backyard flocks pose some danger to commercial flocks, authorities recommend ongoing testing of commercial flocks. The major economic losses from pullorum disease in the last 20 years in the USA has been the cost of testing breeding flocks[8]. The National Poultry Improvement Plan in the USA details specific criteria for establishing and

maintaining official United States pullorum-typhoid free flocks and hatcheries. The plan is voluntary and is administered by an official state agency co-operating with the USDA.[13]

### I: 3.1.6.6 Effect of introduction

Since both of these salmonellae are highly adapted to chickens and turkeys, and clinical symptoms and mortality are seldom seen in other avian species[8], it is likely there would be little, if any, impact on birds other than poultry in New Zealand. Indeed, there is no evidence that wild birds were ever affected by *S. pullorum* during the many years it was endemic in this country. If either of these salmonellae were to be introduced and become established there could be a significant short term impact until they were eradicated. The impact could include potentially significant effects on production, increased mortality and control and eradication costs.

### I:3.1.6.7 Chicken meat as a vehicle

Even though potentially infected organs such as ovaries and liver are removed at slaughter, it is likely that some affected birds would have infected joints and tendon sheaths. In addition, faecal contamination might be a problem. Consequently, it is possible that carcasses or portions might be infected or contaminated. Breast muscle would be the cut least likely to be infected, but contamination is still possible.

### I: 3.1.6.8 Risk of introduction

Live birds or hatching eggs from endemically infected flocks would be the greatest risk of introducing these diseases into New Zealand. However, it is likely that broilers from endemically infected flocks could be harbouring infection. Raw or inadequately cooked chicken scraps fed to backyard poultry could lead to infection becoming established. For this reason it is concluded that sanitary measures are required that ensure imported chicken meat is free from these salmonellae.

### I: 3.1.6.9 Recommendations for risk management

1. Country freedom or a free zone. Vaccination is not practised.[1]

   or

2. Flock of origin freedom[i]

   A flock accreditation program involving both parent and broiler flocks, approved by MAF New Zealand. Vaccination is not permitted.
   or

[1] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

## Section II: Paratyphoid salmonellae{tc \l4 "**Paratyphoid salmonellae}**

### II: 3.1.6.2 The disease

This diverse group of serotypes is mainly of concern as a cause of foodborne disease in humans[3] and contaminated poultry products are major sources of infection.[9] Paratyphoid salmonellae are not host specific and are found nearly ubiquitously in domestic animals, wild animals and humans.[3, 9] The two most important paratyphoid salmonellae in cases of human infection are *S. enteritidis*, which is mainly associated with poultry and eggs, and *S. typhimurium*, found in a broad range of foodstuffs including poultry and eggs.[7]

Although infections are very common in poultry they seldom cause acute systemic disease. They are usually characterised by asymptomatic colonisation of the intestinal tract, sometimes persisting until slaughter and leading ultimately to contamination of the finished carcass.[3, 9, 14] Concerns about the microbial safety of foods have led to the initiation of numerous testing programs to detect paratyphoid salmonellae in poultry and poultry products. Control costs, growth depression and mortality in young chickens and fluctuating consumer demand resulting from food poisoning scares all contribute to significant losses experienced by poultry industries in most developed countries .[3]

#### *Salmonella typhimurium*

Although there are many phage types of *S. typhimurium*, one in particular has attracted considerable attention. It is a multiple antibiotic resistant serotype, known as DT104, and has emerged as a major public health issue of international concern over the last decade.[15, 16, 17] DT104 has been associated with hospitalization rates twice that of other zoonotic foodborne *Salmonella* infections and case-fatality rates that are ten times higher.[7] It also appears to cause higher morbidity and mortality rates among infected animals than other paratyphoid infections.[15]

#### *Salmonella enteritidis*

The recent pattern of human *Salmonella* infections internationally has been dominated by *S. enteritidis*, which is strongly associated with poultry, principally chickens.[14] Three phage types have been reported most commonly. Phage types 8 and 13a have been the most common isolates in the USA[9] and mostly associated with eggs.[18] Phage type 4 (PT4) is common in the UK and much of Europe and has been reported in Central and South America.[14,19] More recently it has become a problem in the egg industry in the USA.[18] In the UK PT4 accounted for 48% of poultry meat associated outbreaks and 90% of egg associated outbreaks between 1989 and 1991.[19] The potential threat of PT4 to both human health and the poultry industry may be greater than that of the other phage types. Although not a problem in the broiler industry in the USA there are fears that it may spill over into broilers as it has in the UK and Europe.[18]

### II: 3.1.6.3 Physical and chemical stability

Salmonellae are relatively resistant to various environmental factors. They grow in liquid media with water activities ($a_w$)[2] between 0.999 and 0.935[20], at temperatures between 8□C and 45□C and in a pH range of 4 to 8. Under experimental conditions growth has been observed after prolonged storage at 5□C.[21] They can survive for considerable periods in frozen product, for example at least 20 days at -2□C in chicken meat and at least 390 days on turkey skin stored at -20□C.[22] There is, however, a reduction in the number of viable cells. They are also able to multiply in an environment with a low level of oxygen or none at all.[21]

Salmonellae die slowly at an $a_w$ value below 0.93. However, the death rate decreases when $a_w$ becomes very low and, for this reason, they can survive over long periods in very dry food, dried faeces and dust.[20]

Salmonellae may survive for long periods in faeces and slurry, for example 140 days in liquid faeces and at least 180 days in sewage sludge.[22] A survey found 10% of cattle slurry samples contained salmonellae, with survival periods ranging from 11-12 weeks.[23] Prolonged survival in water and soil has also been described.[22]

Processes such as salting and smoking have a limited effect on the survival of salmonellae. Several months survival has been observed in brine containing more than 20% salt, especially in commodities with a high protein or fat content. In smoked dry meat products salmonellae may survive for weeks and even months. The relatively high resistance to drying, salting, smoking and freezing explains why these organisms survive in many kinds of food.[21]

Salmonellae are relatively sensitive to beta and gamma irradiation. In many cases they are killed by doses of irradiation of 2-7 kGy, depending on the type of food and its temperature.[21] *Salmonella panama* may be the dose limiting serotype as it has been found that 7.0 kGy was necessary to destroy it on poultry carcasses.[24] In an experiment studying the effects of heat and ionising radiation it was found that treating mechanically deboned chicken meat with gamma radiation (0.9 kGy at 0□C) sensitised *S. typhimurium* to the effects of heat. This effect was not altered by subsequently storing chicken at 5□C for 6 weeks prior to cooking.[25] Another study found that both *S. typhimurium* and *S. enteritidis* were protected against irradiation when deboned chicken was irradiated to an absorbed dose of 1.80 kGy at temperatures below -20□C.[24] In the USA ionizing radiation is an approved additive in fresh, uncooked, packaged poultry products and mechanically separated poultry for the purpose of reducing pathogenic microorganisms. The existing recommendations for poultry are a minimum dose of 1.5 kGy and a maximum of 3.0 kGy although there is a proposal to align this recommendation to that approved for refrigerated and frozen red meat (i.e. 4.5 kGy and 7.0 kGy respectively).[26]

As with other bacteria, the thermal resistance of salmonellae is influenced by a number of factors including the stage of growth and the fat and moisture content of the medium.[22, 27]
The moisture content of various media has an important influence on thermal resistance. As $a_w$ decreases thermal resistance increases.[22] Agents such as salt and sugar may be used to reduce $a_w$ and have been reported to increase the thermal resistance of salmonellae.[28]

[2] Water activity ($a_w$) is a measure of the amount of water available to microorganisms in a given medium. It is expressed as a ratio between the vapour pressure of the medium and pure water.[22]

It is not possible to determine the thermal resistance of salmonellae in one medium and apply this to other media. For example, a significant increase in thermal resistance was reported when salmonellae were heated in chocolate (low $a_w$) as compared to milk (high $a_w$).[29] It was noted that *S. typhimurium* in chocolate was more resistant than *S. senftenberg*. Other studies have reported that *S. senftenberg* is much more resistant than *S. typhimurium*.[21] However, the difference appears to be related to the medium in which these salmonellae were suspended. *Salmonella senftenberg* is much more heat resistant in milk than *S. typhimurium*. This study cautioned against extrapolating heat resistance values obtained under one set of conditions to any other.[29] *Salmonella senftenberg* strain 775W is often described as being more heat resistant than other serotypes and it has been suggested that this strain could be used to test for the effectiveness of heat treatment methods.[21] However, the results from the study described above caution against this suggestion.

A number of authors have concluded that salmonellae are generally quite susceptible to heat[8, 22, 30] and state that they will not survive temperatures above 70□C.[22] As can be seen in Table 3.1.6-1, which lists some experimental thermal inactivation results for salmonellae in various foods, such conclusions may be misleading. These results demonstrate significant variation in heat sensitivities amongst different foods and even within the same food group. An early study by Hussemann and Buyske[30] indicated that *S. typhimurium* could be isolated from inoculated chicken meat heated to a range of temperatures up to and including 90□C for 10 minutes. Although Bayne *et al*[31] were unable to reproduce these results, both experiments were undertaken in somewhat artificial conditions. The chicken meat was heated in small volume pyrex tubes in either a water or oil bath. The study by Schnepf and Barbeau[32] is particularly illuminating and indicates that normal domestic cooking practices cannot always be relied upon. They cooked fresh whole chickens, inoculated with *S. typhimurium*, to an internal temperature of 74□C, 77□C, 79□C or 85□C in a microwave, convection microwave or conventional electric oven. *Salmonella* was not recovered from any of the chickens cooked to an internal temperature of 79□C or 85□C in the convection microwave or conventional oven whereas it was recovered in 82% of the chickens cooked in the microwave oven. Schnepf and Barbeau[32] cited two other studies in which salmonellae were also recovered after microwave cooking: the first in which turkeys were cooked to an internal temperature of 76.6□C; and secondly where chickens were cooked to 85□C. Since large temperature fluctuations occur due to the differential absorption of microwave radiation in various tissues, chicken carcasses and portions may not be heated evenly. In addition, microwave cooked meats are less moist than those cooked in conventional ovens.[32] These results indicate that cooking poultry in a microwave oven cannot be relied upon to kill salmonellae.

There is some variation in recommendations made for cooking poultry by various food authorities. For example the United States Department of Agriculture Food Safety and Inspection Service recommends that poultry breasts and roasts be cooked to an internal temperature of 77□C and whole chickens, thighs or wings be cooked to an internal temperature of 82□C.[35] Health Canada recommends an internal temperature of 85□C[36] and Australian authorities recommend cooking to at least 75□C[37].

Chlorine is not very effective against the salmonellae on the surface of carcasses as it is quickly de-activated on contact with skin. It has been demonstrated experimentally that 50 ppm chlorine is insufficient to decontaminate *Salmonella* infected carcasses.[21] Chlorine is added to the water

used for washing and chilling poultry carcasses in slaughter establishments in many countries.[21] The extent of the reduction in the number of viable salmonellae is dependant on the concentration of chlorine, temperature, the amount of organic matter present and allowing sufficient contact time for inactivation to occur.[21] For example, the following times were required to achieve a 6 log reduction in a solution prepared by soaking a chicken carcass for one hour: at a free chlorine concentration of 25 ppm, 5 minutes at 48□C and 1,250 minutes at 4□C; at a free chlorine concentration of 50 ppm, 48 seconds at 48□C and 286 minutes at 4□C.[38] Immediately after evisceration, poultry carcasses are chilled to a temperature of 4□C to prevent multiplication of bacteria.[21] At this temperate it is unlikely that chlorine will exert any useful effect, even though it has been proposed as a means of reducing such cross contamination as may occur in the spin chiller.

### II: 3.1.6.4 Epidemiology

*Salmonella* infections have been reported in a high percentage of commercial poultry flocks in all areas of the world where appropriate surveys have been undertaken. Often infections by multiple serovars have occurred within single flocks. A large number of serovars have also been isolated from captive and free-living wild birds. Major mortality is not often recorded in free-living wild birds, and infections usually have been limited to small percentages of these wild populations.[9]

Although chickens and turkeys are susceptible to a broad range of *Salmonella* serotypes, the resulting infection process is determined less by the serotype involved than by other factors. These include the age of the birds, the infecting dose, concurrent illnesses such as coccidiosis or IBD, the use of antibiotics and various environmental and management stressors.[3]

Chickens and turkeys can be infected by a very small numbers of salmonellae during the first few weeks of life. Thereafter the infective dose required in otherwise normal birds becomes progressively greater. This apparent age-related resistance to infection is due, at least in part, to the acquisition of protective intestinal microflora. The prevalence of salmonellosis in a flock of chickens colonised by such protective microflora is greatly limited, even in a highly contaminated environment. However, commercial poultry are usually raised in an environment which precludes their early access to the range of bacteria necessary for this protection. If chicks are provided with this microflora a high degree of protection occurs within 32 hours, effectively limiting *Salmonella* infection within a flock to a low prevalence, even under heavy continuous environmental exposure. It may even prevent infection from low levels of exposure.[9]

**Table 3.1.6 - 1:** Experimental thermal inactivation results for salmonellae in various foods.

| Reference | Serotype | Commodity | Results |
|---|---|---|---|
| Hussemann and Buyske, 1954[30] | *S. typhimurium* | breast and leg muscle | 45% of samples survived 75□C for 40 minutes<br>17% of samples survived 80□C for 40 minutes<br>15% of samples survived 85□C for 40 minutes<br>5% of samples survived 90□C for 10 but not 15 minutes |
| Bayne et al, 1965[31] | *S. typhimurium* | breast muscle | survived 55□C for 25 but not 28 minutes<br>did not survive 65□C for 5 minutes |
| | *S. senftenberg* | breast muscle | survived 65□C for 10 but not 15 minutes<br>survived 75□C for 5 but not 8 minutes |
| Schnepf and Barbeau, 1988[32] | *S. typhimurium* | whole chickens | <u>1. microwave</u><br><br>survived in 82% of chickens cooked to reach an internal temperature of 74□C, 77□C, 79□C or 85□C. Average cooking time was 42 minutes.<br><br><u>2. convection microwave oven</u><br><br>survived 40% of chickens cooked to reach an internal temperature of 74□C or 77□C but not at temperatures of 79□C or 85□C. Average cooking time was 46 minutes.<br><br><u>3. conventional electric oven</u><br><br>survived in 33% of chickens cooked to reach an internal temperature of 77□C but not at temperatures of 79□C or 85□C. Average cooking time was 65 minutes. |
| Thayer et al, 1991[25] | *S. typhimurium* | deboned chicken meat | survived heating for 3 minutes at 60□C |
| Palumbo et al, 1995[28] | *S. typhimurium*<br>*S. enteritidis*<br>*S. senftenberg* | liquid egg yolk and egg yolk products | concluded that heating for 3.5 minutes at 61.1□C is adequate for egg yolk; 3.5 minutes at 63.3□C is adequate for yolk with 10% sugar added but that 3.5 minutes at 64.4□C for yolk with 10% or 20% added salt might permit survival |
| Rasmussen et al, 1964[33] | *S. senftenberg* | meat and bone meal | heating for 15 minutes at 65.6□C was not sufficient but 15 minutes at 68.3□C was |
| | *S. brendeney*<br>*S. derby* | meat and bone meal | heating naturally contaminated meat and bone meal for 15 minutes at 76.7□C was not sufficient but 7 minutes at 82.2□C was |
| Himathongkham et al, 1995[34] | *S. enteritidis* | turkey grower feed mash | predicted that a heat treatment of 93□C for 90 seconds in feed with a 15% moisture content would be sufficient |

*Salmonella* infection is usually associated with disease only in very young birds. Clinical disease does not normally occur in mature birds. Birds infected at a young age are more likely to develop a persistent infection and shed salmonellae for greater periods than birds infected at older ages. Persistent shedding of *S. enteritidis* has been shown to last for at least 28 weeks in chicks exposed within 24 hours of hatching.[3]

Paratyphoid salmonellae can usually be isolated from the faeces of experimentally infected adult birds for the first 2 weeks after infection followed by a steady decline in incidence thereafter, although some strains of *S. enteritidis* have been shown to persist for several months. Many serotypes are highly invasive and gut colonisation is usually followed by dissemination to various tissues such as liver, spleen, lung and the ovary.[3]

Paratyphoid salmonellae can be introduced into a poultry flock from many different sources and the wide host range creates an equally large number of reservoirs. Among the most frequently implicated sources of infection are contaminated feed and various animal and insect vectors.[3]

Sewage and slurry contaminated with salmonellae may be important means of introducing and perpetuating infection in a broad range of hosts[23, 39, 40]. Sewage effluents of rural or urban origin, including those from modern treatment facilities, have often been found to be contaminated and have served to contaminate inland and coastal waters. Once a water supply is contaminated, rapid spread of infection may occur. In addition, pasture contamination results when flooding occurs, and there are many reports of clinical cases in adult cattle arising from grazing recently flooded pasture. Further spread from contaminated environments also may take place by wild animals and birds.[23]

Given the current conditions under which poultry are raised, transported, marketed and slaughtered in many countries, as well as existing food processing practices, it may be impossible to obtain salmonellae-free foods.[41] It is widely recognised that faeces are the predominant source of *Salmonella* on the final dressed carcass and that contamination of carcasses and cuts is usually confined to the surfaces, although PT4 has been isolated from aseptically collected muscle samples from chickens purchased at retail outlets.[14] Surveys of meat-type poultry have reported high flock prevalences of *Salmonella.* For example 94% in the Netherlands (1991) and 87% in Canada (1994). However, the actual prevalence within *Salmonella*-infected flocks is often relatively low.[3] Despite low prevalences within flocks, poultry carcasses and cuts are frequently contaminated. Surveys from a number of countries have found chicken broiler carcass and/or product contamination rates of between 29% and 67%.[3, 9] A study conducted by the USDA Food Safety and Inspection Service[15] found that while 20% of broiler carcasses were contaminated with salmonellae, over 95% had three or fewer salmonellae per $cm^2$ of the surface area, indicating that the actual numbers of salmonellae on carcasses leaving processing plants is usually very low. Another study in the Netherlands found between two and 1,400 colony forming units per 100 g of skin on chilled and deep-frozen broiler carcasses[24]. However, the presence of even small numbers of salmonellae in carcass meat and edible offal may lead to heavy contamination of meat products.[28] If not properly cooked, held, cooled and stored, the pathogens present can multiply and cause foodborne illness.[42, 43]

Efforts to reduce or eliminate *Salmonella* contamination of poultry products have been applied both on the farm and in slaughterhouse and processing establishments. Since faeces are the

predominant source of *Salmonella* on the final dressed carcass a great deal of effort is expended on controlling the risks of cross contamination on the slaughter line. HACCP procedures have been developed to identify potential microbiological problems and apply control measures.[9] In the first 9 months following the implementation of mandatory HACCP systems in 1998 in large slaughterhouse establishments and establishments producing raw ground products in the USA, a reduction in chicken carcass contamination rates has occurred, from 20% of chicken carcasses down to 10.7%.[44]

There is increasing agreement that *Salmonella* control may be most successfully applied on farm.[14] Furthermore the chances of eliminating *Salmonella* contamination from poultry meat will be improved if steps are taken to ensure birds entering the slaughter and processing chain are either free from infection or identified as contaminated and treated accordingly. A HACCP system applied across the entire industry, involving feed suppliers, the breeding flock, hatchery and rearing farms, transport, slaughter and processing, provides the most effective means of eliminating salmonellae from poultry meat.[19] There has been considerable success in tackling the problem in Sweden where such an approach has effectively been implemented. The aim is to deliver *Salmonella*-free broilers to slaughterhouses and ultimately *Salmonella*-free food to consumers. Amongst other aspects of the program, broiler flocks have been compulsorily tested 1 to 2 weeks prior to slaughter since 1984. Pooled caecal samples from 10 birds and two lots of 30 pooled faecal samples are cultured. If any *Salmonella* serotypes are detected the flock is not sent for slaughter and processing but destroyed.[45]

### *Salmonella typhimurium* DT104

Consumption of food items contaminated with DT104, such as beef, pork and poultry, and direct contact with infected animals, including farm animals and possibly cats are important risk factors. A study[46] of human infections in the UK showed an independent association between cases of infection and contact with animals, with two of 83 cases reporting contact with sick cats. A review of *Salmonella* isolates from cats from 1991-95 found that 36% were DT104. These findings suggest that cats may play a role in the spread of infection. Further the transmission of *Salmonella* from cats to humans has been reported. Clinically ill cats shed large numbers of salmonellae from the buccal cavity and grooming habits can lead to contamination of the coat. Faecal shedding of DT104 lasts for 14 weeks or longer in cats. Possible sources of infection to cats may include eating scraps of contaminated human food, particularly raw or undercooked meat. Another study[47] in the UK identified a high cat population density as an important risk factor for DT104 infection in cattle.

## II: 3.1.6.5 Occurrence

### *Salmonella typhimurium*

*Salmonella typhimurium* is routinely isolated in New Zealand. For example in 1996-97 it accounted for 50% of all *Salmonella* spp isolates from broilers and 42% of isolates from other species.[4, 5] In 1997, 55.6% of human isolates were *S. typhimurium*. The most prevalent phage types in both humans and animals are 1, 9, 101, 135 and 156. Phage types 101 and 135 accounted for over 70% of *S. typhimurium* isolates in poultry.[48]

A survey conducted in 1997 to assess the antibiotic susceptibility of *Salmonella* confirmed the low prevalence of antibiotic resistance in this country.[49] Fewer than 3% of *S. typhimurium* isolates were resistant. There were no isolates of DT104, which accounted for an average of 0.33% of all human *S. typhimurium* isolates from 1991 to 1998. The proportion of DT104 isolates from humans has remained stable over this period. During the same period there were six bovine isolates, one each in the years 1992 and 1994 and three in 1998.[50] There have been no isolates from other species, including poultry.[50]

The situation in New Zealand is in contrast to the UK and USA. In 1995 in the UK DT104 accounted for 52% of all *Salmonella* isolates from poultry, approximately 70% from cattle, calves and sheep and 36% from swine. The number of human isolates increased nearly 16 times from 1990 to 1996 making DT104 the second most prevalent strain of *Salmonella* isolated. In the US, the number of *S. typhimurium* isolates from humans did not change greatly between 1990 and 1996, but the proportion due to DT104 increased from 9% in 1990, to 33% in 1996. DT104 has been present in cattle in the US since at least 1993 and has been isolated from a wide variety of other animals.[15]

DT104 has also been reported from Canada and many European countries. Although precise information on the incidence of DT104 is not available, several countries have reported increases similar to the United Kingdom. The increase in DT104 occurred at approximately the same time and molecular studies indicate that the organism in these other countries is very similar, if not identical, to the organism in the United Kingdom.[15]

***Salmonella enteritidis***

While phage types 1, 4, 6, 8, 9A, 13 and 23 have been reported in New Zealand's animal and human populations there is no evidence that any of these occur in the poultry industry.[5, 50, 51, 52] Apart from PT9A, which is widespread in animals and humans in this country,[53, 54] the majority of the other phage type isolates are associated with returning overseas travellers or foreign visitors. There have been no isolates of PT4 in animals since 1992 and never from poultry. Prior to that there were three bovine isolations, one each in the years 1988, 1991 and 1992, one ovine isolation in 1991 and one environmental isolation in 1991.[50] It can be concluded that PT4 is, at most, a rare infection in animals in New Zealand and that, perhaps, it has not become established.

PT4 is the predominant phage type in the UK and Europe and has emerged in the egg industry in the western USA.[18] Prior to 1994 it had only been reported in people with foreign sources of infection.[28] It has also been reported in Central and South America.[19, 28]

In the USA phage types 8 and 13a have been found most frequently[28] and are most commonly associated with eggs.[18]

Both the USA and UK have implemented control programs to identify flocks infected with *S. enteritidis*. PT4 was eradicated from Northern Ireland by slaughter and decontamination of poultry feeds.[9] However, it was subsequently re-introduced into both the layer and broiler industries, possibly through contaminated trolleys or feed.[55]

Sweden has so far not been involved in the pandemic spread of *S. enteritidis*. The objective of Sweden's *Salmonella* program is to deliver *Salmonella*-free food to consumers. All broiler flocks are tested before slaughter; layer flocks are tested twice during the production period and before slaughter; imported grandparent stock are derived from free sources; and, all imported meat and meat products are inspected for *Salmonella* to detect a prevalence of contamination above 5%.[45]

### II: 3.1.6.6 Effect of introduction

The most significant effect associated with the establishment of either of *S. enteritidis* PT4 and *S. typhimurium* DT104 would be on human health. New Zealand is one of the few countries that has so far escaped the pandemic spread of these serotypes. Neither would be likely to result in significant disease impacts in the poultry industry. They could, however, result in significant control costs, particularly in the case of *S. enteritidis* PT4 if it were to become established in the poultry breeding and egg laying industry. Since *S. typhimurium* DT104 in particular has a very wide host range it is likely that this serotype could affect a broad range of animals and industries. In fact, it is likely that animals and industries other than poultry, particularly the dairy industry, may be at greater risk of exposure and infection. Once widely established it would be virtually impossible to eradicate.

### II: 3.1.6.7 Chicken meat as a vehicle

Numerous surveys[3, 9, 14, 15] have demonstrated that a significant proportion of chicken carcasses and cuts are contaminated with salmonellae, albeit often with low numbers.[15, 24] However, the presence of even small numbers of salmonellae may lead to heavy contamination if not properly cooled, stored and cooked.[28, 35, 36] Even when HACCP procedures are implemented in slaughterhouses and processing establishments significant contamination rates occur.[44] Given that both *S. enteritidis* PT4 and *S. typhimurium* DT104 are the predominant serotypes associated with poultry in many countries,[15] it is almost certain that chicken meat derived from infected flocks, or from free flocks that are processed at the same time or following chickens from infected flocks, will be contaminated with one or both of these serotypes.

### II: 3.1.6.8 Risk of introduction

It appears highly probable that, despite the best efforts aimed at reducing contamination at slaughter or processing through the application of HACCP procedures, a significant proportion of chicken carcasses or cuts derived from the majority of flocks in many countries will be contaminated with *S. typhimurium* DT104 and/or *S. enteritidis* PT4.[3, 9, 14, 15, 44] Even though the actual contamination rate on carcasses and cuts may be low[15, 24] there are likely to be many exposure opportunities as a result of the volume of imported product and breakdowns in storage and cooking, particularly in the domestic household environment. There are also a number of exposure pathways whereby these salmonellae could infect humans and domestic and wild animal/avian populations in New Zealand. Humans, domestic pets and backyard poultry flocks are the most likely groups to be directly exposed to raw or inadequately cooked or stored chicken meat or scraps. Cats may be particularly important, as they have been shown to excrete *S. typhimurium* DT104 for several months.[46] They are likely to pass on their infection to a number of other species including humans and dairy cattle.[47]

New Zealand has so far avoided the dramatic increase in the levels of *S. typhimurium* DT104 and *S. enteritidis* PT4 reported in many countries throughout the world. Likely reasons may include New Zealand's geographic isolation and a lack of exposure of its human, animal and avian populations to risky commodities. For example New Zealand does not import uncooked poultry meat or eggs or unpasteurised milk. As a result of the probable introduction of these serotypes, the potentially widespread exposure of humans and various animal and avian species and significant risks of establishment, it is concluded that sanitary measures are required that ensure imported chicken meat is free from these salmonellae.

### II: 3.1.6.9 Recommendations for risk management

There are essentially two means of ensuring that chicken carcasses or cuts are not contaminated: ensuring that poultry are free of infection at slaughter or applying a post-slaughter treatment to kill these salmonellae.(20) The two most effective post-slaughter treatment options appear to be either cooking in a convection microwave or conventional electric oven to ensure an internal temperature of 79□C is reached(32) or irradiating fresh or frozen product to ensure a dose of 4.5 kGy or 7.0 kGy respectively is achieved.(26) At the present time it is New Zealand Government policy not to permit food to be irradiated, although the Australia and New Zealand Food Authority is reconsidering this policy.(56) However, until such time as the review is completed sourcing poultry from flocks free from *S. typhimurium* DT104 and *S. enteritidis* PT4 and ensuring that there are no opportunities for cross contamination during transport, slaughter and processing or cooking remain the only two options available:

1. Country Freedom or a free zone[3]

   or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. typhimurium* DT104 and *S. enteritidis* PT4. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of *S. typhimurium* DT104 and *S. enteritidis* PT4 and that there are no opportunities for cross contamination during transport, slaughter and processing.

   or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

## Section III: *Salmonella arizonae*{tc \l4 "*Salmonella arizonae*}

### III: 3.1.6.2 The disease

[3] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

There has been some controversy in the past concerning the nomenclature and classification of this diverse group of bacteria, which consists of over 300 serotypes. *Salmonella arizonae* was originally called *Salmonella arizona* and *Paracolobactrum arizonae*, then *Arizona arizonae*, later *Arizona hinshawi* in the USA and *Salmonella arizonae* in other countries. The current classification is *Salmonella enterica* subspecies IIIa (subspecies *arizonae*).[57] However, for convenience this chapter will refer to these bacteria as *Salmonella arizonae*. There have also been different serotyping schemes which has led to some confusion and apparent errors in reporting.[58, 59] Historically, two serotypes have accounted for nearly all isolates in turkeys and chickens in the USA. Originally they were designated as 7a,7b:1,2,6:- and 7a,7b:1,7,8:- but are now recognised as 18:$Z_4$,$Z_{23}$ and 18:$Z_4$,$Z_{32}$ respectively.[57, 60]

*Salmonella arizonae* is most commonly isolated from turkeys and reptiles, although infections have also been reported in a wide range of mammals, including humans, and other avian species such as chickens.[58] Although chickens are susceptible, infections are rare[9] and *S. arizonae* is not considered to be economically important in this species.[57] *S. arizonae* has been one of the most frequently identified salmonellae in turkeys in the USA and has had considerable economic impact through reduced egg production, hatchability and significant morbidity and mortality in poults.[60] Serious, though uncommon, infections have occurred in humans.[61, 62]

### III: 3.1.6.3 Physical and chemical stability

In general the resistance of *S. arizonae* is similar to that of other salmonellae. It may survive for 5 months in water, 17 months in feed, 6 to 7 months in soil on turkey ranges and 5 to 25 weeks or more on materials and utensils in poultry houses.[60] Further information on physical and chemical stability for salmonellae in general is provided in section II: 3.1.6.3 under paratyphoid salmonellae.

### III: 3.1.6.4 Epidemiology

There has been a dramatic change in the relative proportion of the two most common serotypes reported in turkeys and chickens over the last 40 years. From 1959 to 1967 18:$Z_4$,$Z_{23}$ and 18:$Z_4$,$Z_{32}$ accounted for 37% and 61% of turkey isolates respectively and 49% and 25% of chicken isolates.[63] During the period from 1967 to 1976 all isolates from turkeys were 18:$Z_4$,$Z_{32}$.[57] This trend has continued in more recent times. All isolates reported in either turkeys or chickens in the proceedings from the annual meeting of the United States Animal Health Association in 1997 are 18:$Z_4$,$Z_{32}$.[64] Human isolations of these serotypes have closely followed this trend also.[57] It appears that 18:$Z_4$,$Z_{23}$ is now rare and perhaps of historical significance only. The reasons for this dramatic shift are not obvious although it is likely that host adaptation has occurred which has resulted in a change in transmission to, and incidence of, infection in humans, animals and birds.[57] In addition, there has been a significant change in turkey management with most commercial turkeys now raised indoors.[59, 65, 66]

Infection with either serotype in young turkeys can result in mortality rates of up to 50%.[60] Onset of disease is usually 3 to 5 days after hatching and most deaths occur during the first 3 weeks. Infection in adults does not appear to cause illness but recovery of the organism from intestinal samples, gall bladder, ovary and eggs has been frequently reported.[67] Adult birds

frequently develop chronic infections and shed *S. arizonae* in their faeces for long periods. Transmission through eggs occurs as a result of faecal contamination of the egg shell and transovarial infection.[60, 63, 67]

Infection in turkeys was a significant problem in early 1960s and 1970s in USA[60, 63, 68] but is much less important now as a result of a widespread control program.[68] Coinciding with the decrease in incidence, there has been a significant change to the way turkeys are reared. Most turkey raising has moved from range type premises to rearing indoors in total confinement with strict attention to sanitation and biosecurity.[59, 65, 66]

Although *S. arizonae* is considered to be a rare disease in chickens[9] it has been isolated from chopped chicken meat on sale in Italy [69] and frozen chicken carcasses purchased from retail outlets in USA.[70]

### III: 3.1.6.5 Occurrence

*Salmonella arizonae* has not been isolated for at least 10 years in New Zealand and has never been reported in animals or birds in this country. Although a number of authors report that it occurs worldwide wherever poultry are raised,[60, 63] this may no longer be accurate. Certainly, it is not the case in New Zealand. Such reports are based on observations that *S. arizonae* was introduced into a number of countries, such as the UK,[60] by importing infected hatching eggs or young poults from the USA.[63] However, there do not appear to be any recent publications indicating how widely *S. arizonae* occurs in poultry outside the USA. It is likely that *S. arizonae* was eradicated from the UK in the early 1970s.[67] *Salmonella arizonae* is no longer the problem it once was in turkeys in the USA[60, 66] and it has always been regarded as rare and unimportant in chickens.[9, 60] A recent report from the National Veterinary Services Laboratory in the USA indicated that *S. arizonae* accounted for 4.6% of all salmonellae isolates in turkeys and 0.4% in chickens.[64]

### III: 3.1.6.6 Effect of introduction

The most significant effect associated with the introduction and establishment of *S. arizonae* would be on turkey health and production. Foodborne illness in humans could occur but would probably be uncommon. There would be little, if any, consequences for chickens or other birds. However, animals such as rats and mice could act as carriers and introduce infection onto turkey farms.

### III: 3.1.6.7 Chicken meat as a vehicle

As discussed in section II: 3.1.6.7 a significant proportion of chicken carcasses and cuts are likely to be contaminated with salmonellae despite HACCP procedures. Since *S. arizonae* is shed in the faeces of carrier birds, contamination at slaughter cannot be ruled out. It is likely that chicken meat derived from infected flocks, or from free flocks that are processed at the same time or following chickens from infected flocks, would be contaminated.

### III: 3.1.6.8 Risk of introduction

Live birds or hatching eggs from endemically infected flocks would be the greatest risk of introducing these diseases into New Zealand. However, it is likely that broilers from endemically infected flocks could be harbouring infection. Raw or inadequately cooked chicken scraps fed to backyard poultry, particularly those with turkeys, could lead to infection becoming established. For this reason it is concluded that sanitary measures are required that ensure imported chicken meat is free from *S. arizonae*.

### III: 3.1.6.9 Recommendations for risk management

As discussed in section II: 3.1.6.9 sourcing poultry from free flocks from or cooking are the only two options available to ensure imported chicken meat is free from *S. arizonae*.

1. Country freedom or a free zone.[4]

   or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. arizonae*. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of salmonellae and that there are no opportunities for cross contamination during transport, slaughter and processing.

   or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

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[4] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

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### 3.1.7 TULARAEMIA{tc \l3 "3.1.7 TULARAEMIA}

#### 3.1.7.1 Aetiology

*Francisella tularensis* - a Gram-negative bacterium of the family *Brucellaceae*.[1]

#### 3.1.7.2 The disease

Tularaemia is an infectious septicaemic disease of wild and domestic animals. Highly susceptible host species usually die after an incubation period of 1-25 days. Less susceptible hosts may become chronically infected with *F. tularensis*. Tularaemia is a zoonosis.[2]

#### 3.1.7.3 Physical and chemical stability

*F. tularensis* can persist for up to 4 months in carcasses of animals which have died of the disease and longer in frozen carcasses. It can remain viable in salted meat for up to 31 days. *F. tularensis* is readily destroyed by exposure to heat; it is inactivated by heating to 560C for 10 minutes. Freezing does not destroy *F. tularensis* in infected game meat.[1, 2, 3] *F. tularensis* is resistant to cold and alkaline conditions. Optimal growth takes place at 370C and pH 7.6.[2, 3]

*F. tularensis* has a low resistance to common disinfectants.[2] Chlorine in the concentrations used for water treatment (1.5 mg/l) destroys the organism.[1]

#### 3.1.7.4 Epidemiology

Natural infections with *F. tularensis* have been found in 145 species of vertebrates including lagomorphs, rodents, insectivores, carnivores, ungulates, marsupials, birds, amphibians and fish, and in 111 species of invertebrates.[4] Small rodents and ticks act as the main reservoirs of the organism.[2, 4]

Tularaemia has occurred in at least 25 avian species including chickens, turkeys, waterfowl and wild birds.[1, 2, 5] Turkeys are susceptible, while chickens and other birds are relatively resistant.[2]

*F. tularensis* circulates between biting arthropods and wild mammals, but may also spread directly by respiratory aerosol or ingestion. Affected animals shed organisms in saliva, urine, faeces and milk. Ticks and other insects such as *Culicidae*, *Muscidae*, *Tabanidae*, fleas and lice may spread the disease.[1, 2, 6] Although many biting arthropods can transmit tularaemia, it appears that ticks are essential to maintain the disease in an environment.[1] Livestock ticks are absent from most areas of New Zealand.

Carnivores can become infected by the ingestion of contaminated meat, but high doses are required. Carnivores rarely develop bacteraemia, and only occasionally manifest overt disease.[6] Human infection by ingestion of meat contaminated with *F. tularensis* is relatively common in endemic areas, with undercooked game meat being most commonly implicated.[1, 2, 6]

*F. tularensis* is notoriously invasive. There is evidence that it will penetrate unbroken skin. Infection can be also be acquired through the respiratory tract.[1, 6]

### 3.1.7.5 Occurrence

Foci of tularaemia occur in most countries of western, central and southern Europe. The disease also occurs in parts of Africa, Asia and in North and Central America.[1, 6] It does not occur in New Zealand.

### 3.1.7.6 Effect of introduction

Tularaemia is an important zoonosis and its introduction and establishment in New Zealand would be of public health concern. In countries where it is endemic, tularaemia causes significant epidemics with high mortality in sheep flocks.[6]

The only livestock tick in New Zealand is *Haemaphysalis longicornis*.[7] If the disease could become established in this tick, and if the tick could transmit the disease to sheep, it is possible that outbreaks of disease could result in significant losses. Most birds apart from turkeys appear to be relatively resistant to *F. tularensis*.

### 3.1.7.7 Risk of introduction in chicken meat

Although tularaemia is a sporadic disease of wild birds, it is not known to occur in commercially raised poultry.[5] The risk of imported chicken meat products introducing tularaemia is considered to be negligible.

### 3.1.7.8 Recommendations for risk management

No specific safeguards are required.

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## 3.1.8 BORDETELLOSIS{tc \l3 "3.1.8 BORDETELLOSIS} (TURKEY CORYZA)

### 3.1.8.1 Aetiology

Turkey coryza, or bordetellosis, is caused by *Bordetella avium*, which is a Gram-negative, strictly aerobic bacillus.[1]

### 3.1.8.2 The disease

Turkey coryza is a highly contagious upper respiratory tract disease. Natural infection is usually seen in turkeys 2-6 weeks old, although older turkeys may also develop disease. The incubation period is 7-10 days. Bordetellosis in young turkeys is characterised by high morbidity and low mortality. Infection of adults may result in up to 20% morbidity with no mortality.[1]

*B. avium* is considered to be an opportunist pathogen in chickens.[2] Chickens may be infected with some strains of the organism, but disease tends to be less severe than in turkeys.[1]

### 3.1.8.3 Physical and chemical stability

Survival of *B. avium* is prolonged by low temperatures, low humidities and neutral pH. The organism is killed within 24 hours at 450C.[1, 3] Dust is probably an important source of infection.[3, 4] The bacterium survives longer at pH 7-8 than in more acid or alkaline conditions.[3]

### 3.1.8.4 Epidemiology

Turkeys are the natural host, although isolations have been made from chickens and other birds. It appears that turkey and chicken strains of *B. avium* are similar. There is no evidence that *B. avium* can infect humans.[1]

*B. avium* manifests a tissue tropism for cilia of the respiratory tract epithelium.[5] Numbers of *B. avium* in the trachea peak at 2-3 weeks post-infection and then decrease rapidly from 4-8 weeks.[6]

Bordetellosis is a highly contagious disease readily transmitted to susceptible poults through close contact with infected poults or through exposure to litter or water contaminated by nasal discharges of infected poults. Infection is not transmitted between adjacent cages thus providing evidence against aerosol transmission.[1, 3]

### 3.1.8.5 Occurrence

Bordetellosis is an important disease in major turkey production regions of the United States of America, Canada, Australia and Germany. The aetiology of turkey rhinotracheitis in Great Britain, France, Israel and South Africa may frequently include viruses and other bacteria in addition to *B. avium*.[1]

Bordetellosis has not been reported in New Zealand.

### 3.1.8.6 Effect of introduction

Economic losses to turkey growers would occur as a result of secondary bacterial infections causing impaired growth and mortality. These effects are seen in the United States and probably result in losses of several million dollars annually.(1)

### 3.1.8.7 Risk of introduction in chicken meat

Bordetellosis is a disease of the upper respiratory tracts, and these tissues are largely removed from the carcass at slaughter.(7) Infection of chickens is uncommon.

The likelihood of a chicken carcass being contaminated with *B. avium* is remote.

The risk of introduction of *B. avium* to New Zealand in imported chicken meat products is considered to be negligible.

### 3.1.8.8 Recommendations for risk management

No specific safeguards are required.

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## *3.2. PROTOZOAL INFECTIONS*{tc \l2 "*3.2.PROTOZOAL INFECTIONS*}

### 3.2.1 SARCOSPORIDIOSIS{tc \l3 "3.2.1 SARCOSPORIDIOSIS}

#### 3.2.1.1 Aetiology

The protozoan *Sarcocystis horwathi (S. gallinarum*) causes sarcosporidiosis (sarcocystosis) in chickens.(1) *Sarcocystis* species are two-host parasites. All species are highly host specific, both for the carnivorous definitive host and the intermediate host.(2)

#### 3.2.1.2 The disease

The intestinal phase of the life cycle occurs in a specific carnivorous host and the tissue cyst (sarcocyst) phase is found in the muscle tissue of another specific host which has ingested sporulated sporocysts originating from the faeces of the carnivorous host. Carnivores become infected by ingestion of bradyzoites in muscle tissue of infected intermediate hosts.

The disease is recognised by cysts located in the muscles of mammals, birds and reptiles. Infection with sarcocysts is common but clinical disease is rare. The intestinal phase usually produces no clinical signs in animals.(1, 2) *Sarcocystis* infection is common in free-range chickens with up to 45% of birds being infested. Most infections are subclinical, but occasionally birds develop severe myositis.(3)

#### 3.2.1.3 Physical and chemical stability

Heating to 600C for 20 minutes destroys sarcocysts.(4) Freezing reduces the number of viable sarcocysts in meat.(5)

#### 3.2.1.4 Epidemiology

Natural and experimental infections have been observed in 58 species of birds, including ducks and chickens, but not turkeys. The chicken is thought to be the intermediate host for *S. horwathi* and the dog as the definitive host. The life cycles of *Sarcocystis* species that infect most species of birds remain incompletely known.(1)

Obligatory two-host transmission cycles have been described for many species of *Sarcocystis*. Two vertebrate hosts are required, usually a carnivorous predator or scavenger and the prey or food animal. Sexual reproduction occurs in the predator (definitive host) and asexual reproduction in the prey (intermediate host). The intermediate host becomes infected by the ingestion of sporocysts derived from the faeces of an infected carnivorous host.(1, 6)

Carnivorous hosts may shed sporocysts in faeces for several months. Sporocyst contaminated food or water are common sources of infection for the intermediate host. The definitive host becomes infected when flesh containing sarcocysts from the intermediate host is ingested.(1, 6) Sarcocysts from infected ducks were found to be viable at the end of a 3 year observation period. Thus, intermediate hosts may serve as a source of infection for prolonged periods.(1)

#### 3.2.1.5 Occurrence

*Sarcocystis* species occur commonly worldwide and are already widely distributed throughout New Zealand. Species present in this country include *S. tenella* - sheep and dog, *S. gigantea* - sheep and cat, *S. medusiformis* - sheep and cat, *S. cruzi* - cattle and dog, *S. hirsuta* - cattle and cat, *S. capracanis* - goat and dog.(2) Avian sarcosporidiosis is found throughout the world, but the disease has only been reported six times in intensively raised domestic chickens.(1) *S. horwathi* has never been reported in New Zealand.

### 3.2.1.6 Effect of introduction

The disease is not economically important to the commercial poultry industry.(1)

### 3.2.1.7 Risk of introduction in chicken meat

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

Sarcosporidiosis is not an important disease in intensive broiler production. While sarcosporidiosis infection is common in free-range chickens, the disease is extremely rare in intensively raised domestic chickens.(1, 3) Presumably this is due to the absence of the essential carnivorous host from poultry sheds. The risk of introduction in imported chicken meat products is considered to be negligible.

### 3.2.1.8 Recommendations for risk management

No specific safeguards are required.

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## *3.3 VIRAL INFECTIONS*{tc \l2 "*3.3 VIRAL INFECTIONS*}

### 3.3.1 AVIAN ADENOVIRUS GROUP II SPLENOMEGALY OF CHICKENS{tc \l3 "3.3.1 AVIAN ADENOVIRUS GROUP II SPLENOMEGALY OF CHICKENS}

#### 3.3.1.1 Aetiology

The virus causing avian adenovirus group II splenomegaly (AAS) of chickens is one of three avian adenoviruses which share a common group antigen; the others are turkey haemorrhagic enteritis (THE) virus and the virus of marble spleen disease (MSD) of pheasants.(1) These three viruses have only been classified to source (chickens, turkeys and pheasants respectively) . Antibodies to AAS virus are indistinguishable from those to THE and MSD, but the viruses themselves can be distinguished by restriction endonuclease typing.(1)

#### 3.3.1.2 The disease

Clinical AAS is manifested as splenomegaly of market age or younger broilers, or as splenomegaly with pulmonary congestion and oedema in mature birds, which can result in sudden death without clinical signs, due to asphyxia.(1)

Mortality rates from AAS of up to 9% have been reported in mature chickens,(1) but infection is usually subclinical. However, there is some strain variation in pathogenicity. In the case of the similar condition in turkeys caused by THE virus, infection with strains that cause less than 1% or no mortality induces immunity that prevents infection upon subsequent challenge with more pathogenic strains which would normally produce greater mortality. Such birds remain resistant for life.(1)

#### 3.3.1.3 Physical and chemical stability

Although it is accepted in general that adenoviruses are inactivated in aqueous solution by 56□C for 30 minutes, the avian adenoviruses show more variability and are apparently more heat resistant.(2) Some strains appear to survive 60□C and even 70□C for 30 minutes.(3)

Infectivity of THE virus was destroyed by heating at 70□C for 1 hour.(1) Infectivity was not destroyed by heating at 650C for 1 hour, storage for 6 months at 4□C or 4 years at -40□C, or maintenance at pH 3.0 at 25□C for 30 minutes.(1) The virus is destroyed by chlorination i.e. treatment with 0.0086% sodium hypochlorite, which (assuming a standard 12% availability) corresponds to approximately 10 ppm available chlorine.(1)

#### 3.3.1.4 Epidemiology

Turkeys, chickens and pheasants are the only known natural hosts of group II avian adenoviruses.(1) Antibodies have not been detected in wild birds.(1) MSD isolates will infect turkeys experimentally, and THE isolates will infect pheasants. Similarly, chicken isolates will infect turkeys. In addition, THE isolates have produced spleen swelling and lesions in all avian species where experimental infection has been attempted (golden pheasants, peafowl, chickens, chukars) but deaths have not been seen except in the natural host.(1)

Although the route of infection of natural infections by the AAS virus is not known, transmission of other Group II avian adenoviruses appears to be by the faecal-oral route.[1] Litter from THE virus infected flocks is infectious, and disease often strikes in houses where it has occurred previously.[2]

Unlike the Group I avian adenoviruses, there is no evidence for egg transmission of Group II viruses.[4]

A transient viraemia has been demonstrated in turkeys and chickens infected orally with THE virus.[2] In turkeys the highest titre of THE virus is found in the spleen.[2] Electron micrographs suggest that the THE and MSD viruses replicate in cells of the reticuloendothelial system, primarily in the spleen. These findings are supported by immunodiffusion studies which indicate that viral antigen is concentrated in the spleen, is barely discernible in a small percentage of liver and serum samples, and is not detectable in thymus, bursa of Fabricus, intestinal wall or muscle.[1] Using the ELISA, which is more sensitive, THE viral antigen has been detected in turkeys in small amounts from 2 -6 days post infection in the spleen, liver, intestine, kidney and bone marrow. Peak titres were found in the spleen on day 3 post infection and virus was not detected after day 6.[5]

Group I avian adenoviruses are known to be shed in faeces for up to 2 months.[6] In the absence of specific information on AAS in chickens, it is reasonable to assume that faecal shedding may be of similar duration.

### 3.3.1.5 Occurrence

Group II avian adenoviruses occur all over the world[1]. MSD virus has been reported in USA, Canada, Europe and Australia.[1] Haemorrhagic enteritis has been observed in all countries where turkeys are raised.[1] Serological evidence suggests that infection with the THE virus is widespread in turkeys in the USA, although the clinical syndrome occurs in only a small proportion of infected flocks.[2] One outbreak of THE has been reported in imported turkeys in New Zealand,[7] but it appears that the disease did not become established in this country.

A high prevalence of AAS antibody in mature chickens suggests that most flocks in the USA have been infected.[1] AAS virus infection of chickens has not been reported in this country.

### 3.3.1.6 Effect of introduction

The effect of introduction of AAS in New Zealand poultry flocks is difficult to predict. Infections could be subclinical or outbreaks of mortality might be seen in mature chickens.

### 3.3.1.7 Risk of introduction in chicken meat

There is a limited and short-lived distribution of Group II adenoviruses in the tissues of infected birds, and the vast majority of infectivity is concentrated in the reticuloendothelial tissues, which are removed at slaughter. Infectivity is barely discernable in a small proportion of liver samples, and is not present in muscle.

Therefore it is unlikely that the AAS virus would be found in the tissues of chickens slaughtered and processed for human consumption.

The disease is usually subclinical in chickens and the virus could be excreted in the faeces of slaughter-age birds. However, although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

It is concluded that the risk of this disease being introduced to New Zealand in imported chicken meat products is negligible.

#### 3.3.1.8 Recommendations for risk management

No specific safeguards are required.

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## 3.3.2 AVIAN INFECTIOUS BRONCHITIS{tc \l3 "3.3.2 AVIAN INFECTIOUS BRONCHITIS}

### 3.3.2.1 Aetiology

Avian infectious bronchitis (IB) is caused by a coronavirus. Although antigenic and biological variation among IB strains is well reported, there is no agreed definitive classification system.(1)

### 3.3.2.2 The disease

IB is usually defined as an acute contagious disease of chickens characterised by respiratory signs.(1) However, although sudden outbreaks with high morbidity and mortality may be a feature of infections of young chicks with certain strains of the virus, infections of older birds are more commonly asymptomatic or may result in various clinical signs reflecting involvement with the respiratory, renal or reproductive systems(2).

Mortalities of 20-30% have been reported; these are almost certainly due to mixed infections. Mortality usually occurs when fully susceptible chicks are infected in their first few days of life. Infection of birds more than a few weeks old generally produces no mortality except from nephritis.(3)

Economic losses may be high due to poor weight gain and feed efficiency, and the virus is often a component of mixed infections producing airsacculitis that may result in condemnations of broilers at processing.(2)

Extensive use is made of live and inactivated vaccines, but this does not completely prevent IB infections.(1)

### 3.3.2.3 Physical and chemical stability

Thermoinactivation of IB virus at 56□C shows a typical diphasic curve, with approximately 98% of virus particles being heat sensitive, and the remainder more resistant. There is considerable variation between strains in the time required to inactivate the heat sensitive virions at this temperature. For some strains inactivation was complete after 10 minutes, while other strains were not inactivated even after 60 minutes.(4)

The IB virus survives for several months at -20□C, and indefinitely at -30□C.(3)

IB viruses are considered to be sensitive to common disinfectants.(2)

### 3.3.2.4 Epidemiology

The domestic chicken was long regarded as the only natural host for the IB virus,(2) but it is now accepted that farmed pheasants are also a natural host, at least in the UK.(5)

Respiratory, faecal-oral and mechanical transmission may occur.(6) Spread within poultry houses during outbreaks of respiratory disease is mainly via infected droplets shed from the respiratory

tract during the acute phase of infection.[2] Virus was isolated consistently from the trachea, lungs, kidneys, and bursae of chickens from 24 hours post infection to 7 days post-infection.[2] The virus can be found in respiratory tissues of recovered birds up to 7 weeks post-infection.[3]

Virus also infects the oviduct, and eggs have been found to contain the virus for approximately 50 days post-infection,[3] But egg transmission appears to be exceptional.[6]

The fact that the kidney is a frequent site of virus replication suggests that this organ might be the site of virus persistence, particularly as at least with some strains there is no inflammatory response.[3]

Many strains of IB virus are also excreted in the faeces. IB viruses have been shown to be present in the intestine for up to 28 days and the virus can establish persistent infection in some chickens, with shedding of the virus in faeces for several months.[7] The virus was isolated from tracheal and cloacal swabs collected at the point of lay (19 weeks of age) from hens that had been virus-negative for several weeks following recovery from inoculation at 1 day of age.[2]

### 3.3.2.5 Occurrence

IB viruses are distributed world-wide, but there is considerable strain variation in virulence and tissue tropism.[2, 3] Several serotypes have been identified in the USA. Many other serotypes, distinct from those in North America, have been isolated in Europe and Australia.[2]

Serological testing indicates that IB viruses are widespread in New Zealand poultry. The respiratory effects of all New Zealand field strains appear to be mild, but severe uraemia has been seen in chicks; the effects on egg production have not been studied in this country.[8]

Four serotypes of IB viruses have been distinguished in New Zealand,[9] and an attenuated vaccine has been prepared from a serotype A virus.[10]

The strains present in this country are unrelated to either field or vaccine strains present in Europe or North America.[11] Genetic sequencing has suggested that the domestic strains of IB virus are closely related (98% base-pair homology) to the Vic-S strain from Australia.[12]

Vaccination against IB in this country is carried out in broiler breeders and some layer flocks with the live New Zealand "A" strain vaccine at 6-8 weeks, followed by a killed Massachussets-derived vaccine at about 16 weeks of age. Vaccination of broiler flocks is not practised.[13]

### 3.3.2.6 Effect of introduction

IB viruses can potentially cause considerable losses including mortality and reduced growth rate in chicks, nephritis, increased feed conversion ratios, long-term reproductive problems, reduced egg production and egg quality in laying hens, disruption of breeding programmes, and condemnation of broilers during processing. Annual losses of up to 15-20% of total income have been estimated. IB viruses may also act synergistically with other infections such as *M. gallisepticum* and certain serotypes of *E. coli*, so that economic losses could be greater, perhaps

up to 25% of annual income. Even in the best managed poultry industries, IB probably reduces income by about 5%.[3]

The effects of introducing new strains of IB virus into New Zealand would be restricted to chicken flocks. Depending on the strain concerned, there could be significant effects in such flocks.

### 3.3.2.7 Risk of introduction in chicken meat

IB viruses are found in tissues of the respiratory, digestive, reproductive and urinary systems. Prolonged faecal shedding may occur. The virus survives freezing for several months.

Intestines and reproductive tissues are removed during slaughter, but it has been claimed that approximately 10% of carcasses would contain some lung tissue, and approximately 0.2% of carcasses would contain remnants of trachea.[14]

As kidney tissue remains in carcasses, it is concluded that there is a possibility that the IB virus may be present in carcass tissues.

The virus could be excreted in the faeces of slaughter-age birds. However, although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

As IB viruses can be readily transmitted by the oral route, viable virus present in scraps fed to backyard poultry might result in establishment of infections in those flocks.

The virus survives freezing for long periods, so frozen poultry products would be equally likely as fresh products to harbour the virus.

### 3.3.2.8 Recommendations for risk management

For whole chicken carcasses, the broiler flocks should not have been immunised against IB with live vaccines and the birds should have showed no clinical signs of IB prior to slaughter.

For boneless or bone-in chicken meat cuts (which lack organ remnants such as kidney), no specific safeguards are required.

For cooked chicken meat products, the chicken meat products must be subjected to heat treatment resulting in a core temperature of at least 600C for 5 minutes or 1000C for 1 minute.

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### 3.3.3 AVIAN POLYOMAVIRUS{tc \l3 "3.3.3 AVIAN POLYOMAVIRUS}

Note : this disease was considered in this risk analysis at the request of the Department of Conservation.

#### 3.3.3.1 Aetiology

Genus Polyomavirus, family Papovaviridae.

#### 3.3.3.2 The disease

Avian polyomavirus is the cause of budgerigar fledgling disease (BFD), which occurs in many species of caged psittacines. The virus is best known for its role in psittacine nestling mortality, particularly affecting budgerigars, and is being described with increasing frequency in several species of newborn parrots. There are no reports of polyoma-induced disease in free-ranging psittacine birds.(1)

The virus also commonly infects finches, and gallinaceous birds appear to be susceptible to infection.(1)

#### 3.3.3.3 Physical and chemical stability

Polyomaviruses are resistant to severe environmental conditions.(1) A BFD virus survived five cycles of freezing to -70□C for 15 minutes and thawing at 37□C for 2 minutes. The virus also survived heating at 560C for 2 hours.(2)

#### 3.3.3.4 Epidemiology

Avian polyomaviruses have been reported to infect a wide variety of birds, primarily budgerigars, other psittacines and finches.(1)

The evidence for natural infections in birds other than psittacines and finches is very limited.

A virus that morphologically resembled a polyomavirus was recovered from the intestinal contents of asymptomatic turkeys, but the recovered virus did not cause any disease in experimentally infected birds.(1) A polyomavirus-like agent was identified in the faeces of an ostrich in the USA.(1)

A polyomavirus with similarities to BFD virus was recovered from the drinking water and faeces associated with a chicken layer replacement farm suffering from infectious bursal disease in Germany.(3) However, it was not determined if the virus in this poultry house originated from chickens or was a contaminant from another source.(1)

Polyomavirus inclusions were observed by electron microscopy in caecal epithelial cells of a chicken in the USA. DNA analysis indicated that it was different from polyomaviruses observed in psittacines.(4)

Polyomavirus-specific antibodies have been demonstrated in clinically healthy broiler chickens from central Europe[3] and the USA.[1] Virus neutralising antibodies were detected in two golden pheasants and a Lady Amhurst pheasant that had been naturally exposed to affected psittacine birds, while a potentially exposed Bantam chicken and two Toco toucans remained seronegative.[1] Inclusion bodies suggestive of polyomavirus have been described from Australia in a kakariki, a peaceful dove, a brown pigeon, and a canary.[1]

Broilers and SPF chickens have been shown to develop VN antibodies following experimental infection with avian polyomavirus.[1] However, the effect of BFD virus on experimentally infected chickens varied dramatically with the age of exposure. Chicken embryos infected at 10 days of age died 10 days later, and had gross and histologic lesions characteristic of the disease. By contrast, chicken embryos infected at 11 and 12 days of age remained normal, devloped precipitating antibodies that could be detected 2 weeks after hatching, and did not develop gross or microscopic changes suggestive of infection. Two-week to 4-month-old broilers and SPF chickens inoculated with avian polyoma virus by the intramuscular or intravenous routes developed VN antibodies, suggesting that they had become infected. Some experimental chickens developed a transient diarrhoea, but otherwise remained clinically normal. None of the experimentally infected birds developed gross or histologic changes suggestive of a polyomavirus infection. In two infectivity trials, several chickens used as in contact controls seroconverted, suggesting that transmission of the virus had occurred between experimentally infected and seronegative birds. However, chickens administered avian polyomavirus by the oral route did not develop VN antibodies, suggesting that they had not been infected.[1]

The BFD virus is transmitted both horizontally and vertically in budgerigars, but in other birds probably only horizontally. Horizontal transmission is via the respiratory route. Clinically normal psittacine birds are thought to act as carriers. Persistently infected budgerigars and clinically affected birds of other species have been shown to shed virus in faeces. Birds may shed virus intermittently or continuously over months to years. Transmission of polyomavirus occurs by the inhalation of infected faecal dust and feather dander. Persistent infections are not known to occur in non-psittacine species.[1]

### 3.3.3.5 Occurrence

Avian polyomaviruses have been described throughout the world. Lesions associated with the BFD virus have been seen in caged psittacine birds in the United States, Canada, Japan, Europe, South Africa and Australia.[1] In Australia, infections are considered particularly common in lovebirds.[1]

The BFD virus has not been isolated in New Zealand. However, tests capable of identifying its presence are not available here. Furthermore, given that the virus has been reported in Australian birds, large numbers of which have been imported into this country legally and illegally for many years, it is likely that a thorough survey of psittacine birds in New Zealand would detect the presence of avian polyomavirus.[5]

### 3.3.3.6 Effect of introduction

Introduction of the disease could have serious consequences for caged psittacine birds in the form of high mortality rates in hatchlings. As the disease has never been identified in free-ranging psittacines, it is by no means certain that it would have any effect on native psittacines even if it were introduced or already present in this country.

### 3.3.3.7 Risk of introduction in chicken meat

Polyomaviruses infections in chickens appear to be very rare and the likelihood of an infected bird being slaughtered for export to New Zealand is remote.

DNA analysis of a polyomavirus found in caecal epithelial cells of a chicken indicated that it was different frompolyomviruses of psittacines.

Moreover, polyomaviruses are transmitted by the inhalation of virus-laden dust, not by ingestion. Therefore, meat is not considered to act as a vehicle for transmission of this agent.

It is concluded that the risk of introduction of avian polyomavirus in imported chicken meat products is negligible.

### 3.3.3.8 Recommendations for risk management

No specific safeguards are required.

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### 3.3.4 AVIAN RHINOTRACHEITIS{tc \l3 "3.3.4 AVIAN RHINOTRACHEITIS}

#### 3.3.4.1 Aetiology

Rhinotracheitis in turkeys and chickens is caused by the turkey rhinotracheitis (TRT) virus, which is a member of the genus *Pneumovirus*, in the family Paramyxoviridae.[1] Pneumoviruses from different geographical locations may show marked antigenic differences between strains,[2] although some chicken and turkey isolates appear to be closely related.[3]

#### 3.3.4.2 The disease

The disease caused by TRT virus in chickens is known as "swollen head syndrome" (SHS); it is characterised by the development of subcutaneous oedematous swelling of the head and neck region of broilers or broiler breeders. However, as the TRT virus is rarely isolated from chickens,[4] some authorities consider that the establishment of a firm association between SHS and the TRT virus has not been confirmed.[5] Nevertheless, while the disease is thought to be initiated by infection with the virus, the clinical signs are the result of a facial cellulitis caused by secondary infection with bacteria such as *Escherichia coli*.[2, 6] If chickens survive the acute stage of infection, the disease may progress to a secondary coliform septicaemia and death.[6] Usually fewer than 4% of the flock is affected and mortality ranges from 1% to 20%.[2, 5, 6, 7] Egg production may be depressed in layers. Broiler chickens are generally affected at 4-6 weeks of age, while layers may be affected from peak of lay onwards (30 to 52 weeks of age).[2, 4, 7]

There is considerable variation in reported clinical signs in turkeys, and this is probably due to the secondary adventitious organisms that frequently appear as a problem with TRT. In adult breeding birds disease signs may be slight, but egg production in layers may be reduced by as much as 70%. In some adult turkey flocks, there may be seroconversion without any clinical signs. When disease is seen, morbidity in birds of all ages is usually described as "very high" or "100%". Reported mortality varies from 0.4% to 90%, but is usually highest in young poults, in which the disease is characterised by rapidly spreading upper respiratory tract signs, and often very high morbidity.[2, 7]

#### 3.3.4.3 Physical and chemical stability

The virus is stable over the pH range 3.0 - 9.0. It is sensitive to lipid solvents and is inactivated by heating to 560C for 30 minutes.[8]

#### 3.3.4.4 Epidemiology

Experimental infection with TRT virus has been attempted in turkeys, chickens, ducks, geese, pheasants, guinea fowl and pigeons. Virus was only recoverable from turkeys and chickens, although seroconversion was seen in pheasants and guinea fowl.[4]

The remarkable characteristic of TRT was its rapid spread. In Great Britain it radiated to most parts of England and Wales within 9 weeks of the first report on the East Coast.[7] It is not clear how the virus spreads between flocks, but movement of affected or recovered birds, contaminated water, movement of personnel and equipment have been implicated. However,

only spread by direct contact has been confirmed.[2, 4, 5, 7, 9] Susceptible birds become infected via the respiratory tract,[5] and the disease has been transmitted by inoculation with materials taken from the respiratory tract of affected birds.[2, 7] There have been no reports of spread by wild birds.[4]

TRT is greatly exacerbated by poor management practices such as inadequate ventilation, overstocking, poor litter conditions, poor general hygiene, and mixed age groups.[2] Debeaking or vaccination with live Newcastle disease vaccine, if done at a critical time, might also increase the incidence and severity of clinical signs and mortality.[2]

The epidemiological relationship between TRT and SHS are not clear. Although in Great Britain SHS has been reported in chicken housed in close proximity to turkeys with TRT, there is no evidence to indicate which species was first affected upon introduction of the virus.[7]

### 3.3.4.5 Occurrence

Avian rhinotracheitis was first seen in Great Britain in June 1985. Since then the virus has been isolated from chickens and/or turkeys in France, Italy, Spain, The Netherlands, Germany, Hungary, Taiwan, South Africa and Israel.[7] In addition, antibodies have been demonstrated in chickens and/or turkeys in Austria and Greece. SHS appears to have been prevalent in South Africa for a number of years.[7] Recent reports of the virus in the far east and North America suggest that it is distributed worldwide.[10]

An extensive serological study of Australian turkey flocks in 1990 failed to find any birds seropositive to the TRT virus.[4] The clinical syndrome has never been reported in New Zealand, and there is limited serological evidence to suggest that the TRT virus is not present in this country.[9]

### 3.3.4.6 Effect of introduction

Economic losses may occur through poor weight gains, higher food consumption, mortality due to secondary infections, increased condemnations and/or downgrading at slaughter and increased medication and production costs. Mortality rates are generally 2-5% but may reach 70% where secondary bacterial infections occur. The disease is strongly influenced by poor management practices.[2]

### 3.3.4.7 Risk of introduction in chicken meat

It appears that viraemia is transient and of a low titre.[11] The virus was not isolated from blood collected between 1 and 7 days post-infection, or from liver, spleen or kidney between days 1 and 12 post-infection. Viral replication occurs principally in the upper respiratory tract (nasal tissue). High titres of virus are recovered from the upper respiratory tract at 3 and 5 days post-infection; thereafter, the amount of virus recovered falls quickly and little virus is recovered from 7 days post-infection. In turkeys, low titres of virus can be shed from the respiratory tract up to 14 days after inoculation and the virus can be detected in the trachea up to 7 days post-inoculation. The virus has been isolated from the lung of infected turkey poults up to 9 days post-infection.[2, 3, 11]

Only very small amounts of virus may be recovered from the trachea, air sacs, kidney, ovary and spleen.(3)

Broiler chickens are generally affected at 4-6 weeks of age and so could be infected at the time of slaughter.

There is a transient, low-level viraemia and a limited and short-lived distribution of virus in the tissues of infected birds.

The virus is primarily found in the upper respiratory tract, which is removed from all but around 0.2% of chicken carcasses at slaughter.(12) The temperature achieved during cooking would be expected to destroy the virus.

Furthermore, natural transmission has only been confirmed by direct contact, and experimental transmission has been possible only by the respiratory route, so it is considered that chicken meat products are unlikely to act as a vehicle for the introduction of the virus.

It is concluded that the risk of introduction of TRT virus in imported chicken meat products is negligible.

### 3.3.4.8 Recommendations for risk management

No specific safeguards are required.

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### 3.3.5 BIG LIVER AND SPLEEN DISEASE{tc \l3 "3.3.5 BIG LIVER AND SPLEEN DISEASE}

#### 3.3.5.1 Aetiology

Field observations and laboratory transmission studies indicate that big liver and spleen disease is infectious. The causative agent is presumed to be a virus. Attempts to cultivate the disease agent have been unsuccessful.[1, 2, 3]

#### 3.3.5.2 The disease

The disease 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. Clinical signs in affected birds include depression and anorexia. The disease has been recognised almost exclusively in adult flocks in production between 24 and 58 weeks of age. The drop in egg production lasts for 3-4 weeks and then returns to normal over a further 3 weeks. An increase in hen mortality rate of between 0.1% and 1% per week may be seen. The severity of the egg production drop may vary from 5-20% on a hen per day basis.[2, 3, 4, 5] The disease is most prevalent in adult broiler breeder flocks but has been seen in one layer breeder and some broiler flocks that reach sexual maturity. Antibodies have been detected in one flock of commercial layers. Antibodies have not been recognised in sexually immature birds.[2]

#### 3.3.5.3 Epidemiology

Evidence of natural infection has only been found in chickens older than 24 weeks.[2, 6]

The disease agent spreads rapidly through chickens housed in the same shed and usually affects all sheds on a farm within 3-10 weeks, despite quarantine precautions. People and equipment are thought to be important for mechanical spread of disease. Airborne spread or insect vectors have not been ruled out. Thorough end-of-batch hygiene eliminates big liver and spleen disease from isolated farms.[2]

Young birds exposed by natural routes do not develop signs of disease or seroconvert until after sexual maturity. Big liver and spleen disease has been reproduced in adult birds by parenteral or oral inoculation with faeces, buffy coat, spleen or liver material taken from birds in the acute stage of the disease. Transmission is also possible by direct contact.[2, 3, 4]

After oral administration, antigen may be detected between 2 and 4 weeks post-infection, and antibody between 3 and 6 weeks. The liver probably contains the highest concentration of the disease agent, as it has been shown as having the highest infectivity. It is thought that excretion of the agent may occur when birds develop antigenaemia.[4]

#### 3.3.5.4 Occurrence

The disease has only been seen in Australia, although serological reactions have been recorded in flocks in the United Kingdom and the United States.[1, 2, 3, 5] It has not been reported in New Zealand.

### 3.3.5.5 Effect of introduction

The disease is considered by some to be the most economically significant disease of sexually mature broiler breeders in Australia, because of loss of egg production in affected flocks.[2, 5, 7]

### 3.3.5.6 Risk of introduction in chicken meat

The lack of understanding of the causative agent and how it is transmitted makes the formulation of specific safeguards difficult.

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 big liver and spleen antigen has been confirmed in these tissues.[1, 3]

Transmission is possible by the oral inoculation of spleen or liver, but these tissues are removed during processing. The agent can infect birds at a young age and appears to remain dormant in tissues for long periods of time.

### 3.3.5.7 Recommendations for risk management

For all commodities under consideration, chicken meat products must be certified to have been derived from birds whose parent flocks did not show clinical signs of big liver and spleen disease in the previous 6 months.

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## 3.3.6 HIGHLY PATHOGENIC AVIAN INFLUENZA{tc \l3 "**3.3.6 HIGHLY PATHOGENIC AVIAN INFLUENZA}**

### 3.3.6.1 Aetiology

Avian influenza is caused by viruses of the genus *Influenzavirus A, B* in the family Orthomyxoviridae. Only type A viruses are found in birds. Many species of bird have been shown to be susceptible to infection with influenza A viruses, but the overwhelming majority of isolates are of low pathogenicity for chickens and turkeys. Influenza A viruses are classified into subtypes on the basis of antigenic variation of their haemagglutinin (H) and neuraminidase (N) surface proteins, [1] against which the greatest selection pressure is exerted by the host's immune response.[2] All reported outbreaks of highly pathogenic avian influenza (HPAI) have been of the H5 or H7 subtype although many H5 and H7 subtype viruses isolated from birds have been of low virulence.[1]

### 3.3.6.2 The disease

The disease caused by influenza A viruses in chickens and turkeys has historically been called "fowl plague".[2] It implies the involvement of a virulent virus and is used to describe a disease of chickens with clinical signs such as excessive lacrimation, respiratory distress, sinusitis, oedema of the head and face, cyanosis of the unfeathered skin, and diarrhoea. Sudden death may be the only sign, especially in the case of infections with very highly virulent influenza viruses. These signs vary enormously depending on the host, age of the bird, presence of other organisms and environmental conditions.[1] In general, however, more clinical signs are seen the longer the birds survive the HPAI infection.[2]

Highly pathogenic strains of avian influenza virus are now defined by the OIE as able to produce mortality in six or more of eight susceptible 4-8-week-old chickens within 10 days of intravenous inoculation.[1] The European Commission has a similar definition using the intravenous pathogenicity index, or IVPI.[3] There are a large number of viruses classified as low to moderately pathogenic.[4]

In outbreaks of HPAI in turkeys and chickens, morbidity and mortality can reach 100%.[5]

Type A influenza viruses can be zoonotic under natural conditions. The sporadic spread of these viruses to people is usually associated with occupational exposure, and human-to-human transmission is very limited.[6]

### 3.3.6.3 Physical and chemical stability

Influenza viruses are most stable at pH 7-8.[4] They are not very stable below pH 6. The ultimate pH of poultry muscle is between 5.7 and 5.9.[7]

Infectivity is retained for several weeks at 4□C, for months at -200C, and for years at -40□C .[8] Survival in the environment is increased in cool and moist conditions. For example, the viruses have been recovered from liquid manure for 105 days after depopulation in wintertime following

outbreaks of HPAI.[4] Infectivity in faecal material was retained for 30-35 days at 4□C and for 7 days at 20□C.[4]

Thermoinactivation rates differ among strains,[8] but two different strains were inactivated by heating for 15 minutes at 560C or for 5 minutes at 600C.[8] At temperatures above 600C, infectivity is destroyed very quickly.[9]

### 3.3.6.4 Epidemiology

The frequency of antigenic variation among the influenza viruses is high and occurs in two ways, drift and shift. Antigenic drift involves minor antigenic changes in the H and/or N antigens, apparently caused by single point mutations which alter the structure of surface proteins. Avian influenza viruses show less antigenic drift than mammalian viruses, for reasons which are unknown. Antigenic shift involves major antigenic changes in the H and/or N antigens. The segmented nature of the viral genome (eight segments of RNA) allows segments to reassort when a cell is infected with two different influenza viruses, potentially yielding 256 genetically different progeny viruses. Genetic reassortment has been demonstrated to occur when ducks are infected with two antigenically distinct viruses, and it is not surprising that viruses with almost every combination of antigenic subtypes have been recovered from ducks in nature. Reassortment is suggested as the mechanism by which "new" pandemic strains arise.[4]

Since the mid-1970s influenza viruses have been isolated from avian species representing most of the major families of birds throughout the world. Migratory waterfowl, particularly ducks and geese, have yielded more avian influenza viruses than any other group of birds, but overt disease does not seem to occur in these birds.[2] A theory which is gaining support is that all H5 and H7 viruses are of low virulence in their natural reservoir, waterfowl, but they mutate to HPAI *after* introduction into poultry flocks.[10] It is possible that the virulence shift may occur some months after introduction, after repeated cycles of replication in large numbers of chickens.[11, 12] Although evidence for this theory is still limited, it may mean that cause and effect relationships between specific avian influenza viruses in waterfowl and outbreaks in poultry will be impossible to prove.

Because of the intestinal nature of avian influenza infections in waterfowl, large quantities of virus are excreted in faeces, and ducks have been shown to excrete the virus for as long as 30 days.[4] One study showed that 60% of juvenile ducks sampled on a lake in Alberta, Canada prior to migration were excreting virus, and such quantities of virus were present that isolation from untreated lake water was possible.[4] However, once the birds began their migration the isolation rate dropped markedly, and the lake water did not retain infectious virus after the birds left, suggesting that the viruses may survive for only short periods in the environment.[4]

Ducks, geese, pigeons, and pheasants appear to be refractory to even the most pathogenic influenza viruses for chickens and turkeys. Quail may be affected by some HPAI viruses. There has been only one report of disease in wild birds associated with HPAI virus, that is, among South African terns in 1961.[2]

The methods of transmission of avian influenza viruses are poorly understood.[2] The viruses replicate in the respiratory and intestinal tracts of infected birds, and transmission appears to be

by respiratory aerosols or through faeces, either directly or in contaminated water or food.[5] In view of the relatively slow and inefficient spread observed in both natural and experimental infections, especially with HPAI viruses, the faecal/oral route appears to be the main route of spread. There is little evidence for airborne spread over significant distances.[5] Wild birds are probably the most common means by which HPAI is introduced into domestic flocks, and for all birds, the ingestion of infective faeces appears to be the most important mode of transmission.[13] In experiments carried out almost 70 years ago, it was shown that transmission of infection was not possible through the feeding of muscle tissue of viraemic birds.[14]

None of the avian influenza viruses are considered to be endemic in turkeys or chickens. It is significant that the majority of outbreaks in the USA and the United Kingdom have occurred in turkey flocks situated on the migratory route of waterfowl.[5, 13]

Outbreaks of HPAI have always been much more common in turkey flocks than chicken flocks, presumably because turkeys were traditionally raised outdoors, where they were able to come into direct contact with migrating waterfowl, whereas intensive chickens production has always been predominantly in totally enclosed houses.[4] However, over the past two decades there has been a steady move toward total housing production systems for meat turkeys.[15] In the USA at least 80% of meat birds are now reared indoors,[16] and in North Carolina, the most intensive turkey production area of the USA, fewer than 1% of meat turkey flocks are now raised under range conditions. [17] A similar trend has occurred in Great Britain.[18] This may explain why over the past 20 years reports of severe disease outbreaks involving highly pathogenic influenza A viruses have become less common.[4]

### 3.3.6.5 Occurrence

Avian influenza viruses in their natural reservoir, waterfowl, appear to have a worldwide distribution.[4] Outbreaks of HPAI were reported in Hong Kong,[19] Australia and Italy during 1997.[20]

Periodic surveys of wild ducks in New Zealand have resulted in the isolation of several strains of avian influenza A virus, although the infection rate has always been low in comparison to reports in waterfowl in other parts of the world.[21] Until recently, none of the avian influenza strains isolated had the H5 or H7 antigens which are a feature of all the virulent strains. However, a survey of 346 wild ducks from five sites in New Zealand which was carried out in early 1997 resulted in the isolation of six avian influenza viruses,[22] two of which were H5N2.[23] Nevertheless, no outbreaks of avian influenza have been reported in this country.

### 3.3.6.6 Effect of introduction

Losses to the poultry industry could be significant if highly pathogenic strains of avian influenza were introduced to New Zealand. Depending on the strain of virus introduced, flocks of chickens, turkeys or ducks could be affected. Individual poultry producers could be severely affected and the costs of control procedures could be high.[24] An outbreak of HPAI would affect exports of poultry products and hatching eggs.[25]

While native bird species could be infected with HPAI viruses, it is unlikely that they would be affected clinically. However, the potential for infected wild ducks to act as a reservoir of the virus and to spread HPAI to domestic poultry would be of concern.

### 3.3.6.7 Risk of introduction in chicken meat

Avian influenza viruses appear to be natural infections of waterbirds, which cause no disease in the natural hosts but which are periodically introduced into turkey and chicken flocks, causing dramatic disease characterised by extremely high mortality. There is no evidence that HPAI viruses become endemic in poultry.[2, 4]

Several criteria would need to be met for chicken meat to introduce HPAI into poultry flocks in New Zealand. Firstly, the virus would have to be present in the tissues or on the surface of a clinically normal bird at slaughter. This could occur only by slaughtering a bird that was either viraemic or was shedding HPIA virus in the faeces after recovery from disease. It is considered that in view of the rapid course of disease, the high mortality and the dramatic clinical signs of HPAI, the probability of either a viraemic or a recovered bird being slaughtered for human consumption is remote, at least with respect to commercial broiler operations.

Secondly, the virus would have to survive during storage and shipping of the chicken meat products to New Zealand. On the one hand, influenza viruses are not very stable at the ultimate pH of chicken meat. On the other hand, they can survive chilling and freezing for long periods, so shipping of chicken meat products to this country might result in introduction of influenza virus if it were present in those products at the time of packing.

Thirdly, the virus would have to infect poultry in this country. The main route of infection is the oral route, so infectious material would have to be fed to poultry, which would be more likely in the case of flocks of backyard chickens than for commercial poultry flocks. As influenza viruses survive for very short periods at temperatures of 60□C, cooking would readily destroy any surface contamination of the virus, and cooking times which resulted in a core temperature of greater than 60□C would destroy any virus within tissues. Therefore, for contaminated imported chicken meat products to pose any risk of transmission of HPAI virus infection to local poultry, raw scraps of the imported carcass tissue would have to be fed.

However, there is no evidence that transmission of HPAI viruses is possible in meat from clinically healthy chickens,[2, 4] and experiments carried out nearly 70 years ago showed that transmission was not possible by the feeding of meat from viraemic birds.[14]

Although the likelihood of imported chicken meat products being a vehicle for the introduction of HPAI viruses is considered to be remote, the impact of any such introduction into New Zealand's poultry flocks is likely to be severe. Therefore it is concluded that safeguards are justified.

### 3.3.6.8 Recommendations for risk management

#### *3.3.6.8.1 Fresh chicken meat*

For fresh chicken meat, MAF considers that the safeguards recommended by the Office International des Epizooties, as detailed in the OIE International Animal Health Code(26) (OIE Code) are appropriate :

> When importing fresh chicken meat from HPAI free countries[5], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment of meat comes from birds which have been kept in an HPAI-free country since they were hatched or for at least the past 21 days.
>
> When importing fresh chicken meat from countries or zones considered infected with HPAI[6], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Health Code] attesting that the entire consignment of meat comes from birds which have been kept in an establishment free[7] from HPAI and not situated in an HPAI *infected zone* [defined by the OIE Code].
>
> In addition, the chicken meat products must come from birds which have not been vaccinated for avian influenza.

*3.3.6.8.2 Cooked chicken meat*

The OIE Code does not recommend safeguards for cooked chicken meat. The current MAF requirements for cooked chicken meat, based on an earlier review,(7) are as follows:

> The meat products must have been subject to heat treatment resulting in a core temperature of at least 600C for 30 minutes or 1000C for 1 minute.

However, 5 minutes at 60□C is sufficient to inactivate influenza viruses. Therefore, for chicken meat from countries which do not meet the standards in 3.3.6.8.1, the chicken meat products must be subjected to a heat treatment resulting in a core temperature of at least 600C for 5 minutes or 1000C for 1 minute.

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5 A country may be considered free from HPAI when it can be shown that HPAI has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against HPAI.

6 A HPAI infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

7 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

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### 3.3.7 INFECTIOUS BURSAL DISEASE{tc \l3 "3.3.7 INFECTIOUS BURSAL DISEASE}

#### 3.3.7.1 Aetiology

Infectious bursal disease (IBD) is caused by the IBD virus which is a member of the *Avibirnavirus* genus in the family Birnaviridae.[1] IBD serotype 1 (IBD1) viruses mainly infect fowl, and antibodies are widely distributed. There are many strains of IBD1. Some turkeys have antibody to IBD1, and it has been isolated from ducks.[2]

IBD serotype 2 (IBD2) viruses are widely distributed in turkeys, and antibodies are common in chickens and ducks.[2] However, IBD2 has not been shown to cause disease in any avian species.[2, 3] IBD2 infection of turkeys is dealt with in detail in section 9.2.6.

#### 3.3.7.2 The disease

Serotype 1 viruses cause disease only in chickens.[2]

IBD is an acute, highly contagious viral infection of young chickens. The clinical signs of the disease are related to immunosuppression caused by damage to the bursa of Fabricius, and the earlier in life a chick is infected the greater the bursal damage and the greater the immunosuppression.[2] Under commercial conditions chicks are usually infected only when maternal antibody levels begin to wane at 2-3 weeks of age. The incubation period is 2-3 days.[2] Disease has been recorded in birds between 10 days and 20 weeks of age, a time period which corresponds well with the development of the bursa.[2]

In acute outbreaks, clinical signs include diarrhoea, anorexia, prostration.[2, 3] In fully susceptible flocks the disease appears suddenly and there is a high morbidity rate, usually approaching 100%.[3] Mortality is often nil but may be as high as 20-30%, usually beginning on the third day post-infection and peaking over the next 5-7 days.[3]

Many outbreaks are less severe and in some cases the only sign may be impaired weight gain, which results in poor economic returns.[2] Mild strains of the virus do not produce clinical signs unless the degree of immunosuppression is sufficient to predispose the infected birds to secondary infection.[2]

Around 1987 a highly virulent form of IBD was recognised in Belgium and the Netherlands and this has subsequently spread widely.[2] Highly virulent viruses cause high mortality; 10-15 % is common,[2] but sometimes it can be as high as 90% or 100%.[3]

Vaccination is commonly practised. It is usually based on hyper-immunisation of breeder hens by the repeated administration of inactivated vaccines. Maternal antibody is transferred in egg yolk to the chicks, normally providing protection at least for the first 2-3 weeks of life. If titres are high enough, chicks may be protected for 5 weeks or even longer.[2] If vaccination of broilers is practised, they may be vaccinated once or twice, using live vaccines. The first vaccination is

usually either in ovo (3 days before hatching) or at day 1, and the second vaccination is usually between 10 days and 2 weeks of age.[4]

Many different live vaccines are available for use in broilers, including mild, "intermediate" and "hot" strains. The more virulent vaccine strains are generally used in the United States, and these have recently been used with some success in Europe to counter the new highly virulent viruses. Vaccine viruses may cause considerable damage by themselves, including bursal atrophy, reduced weight gain, immunosuppression and death in chicks without maternal antibody.[2, 3, 5]. Birds may shed "hot" vaccine strains for prolonged periods,[2, 5] and spread to other flocks can occur.[2]

### 3.3.7.3 Physical and chemical stability

The IBD virus is very stable. It resists freezing and thawing and is stable at pH 2 or greater.[3]

The thermostability of the IBD virus has long been recognised, and several documents have examined the quarantine implications of this.[6, 7]

Reports from the late 1960s indicated that the virus was still viable after 90 minutes at 60□C, but was inactivated by heating for 30 minutes at 70□C.[3] A report from the 1980s indicated that in tissue culture fluids the virus survived heating for 10 minutes at 72□C but did not survive heating for 1 minute at 82□C.[6] A vaccine strain added to a nugget meat mixture was inactivated by heating for 5 minutes at 75□C or for 1 minute at 80□C.[6]

These early studies were usually based on one-off batch testing with very low starting titres of virus, such that after the first phase of biphasic heat inactivation, no virus was detectable.[8]

Experiments commissioned in 1988 by the New Zealand Poultry Industry Association[6] on the thermostability of the IBD virus, using the 52/70 strain in bursal homogenates, showed that there was a rapid fall in infectivity within a very short time, followed by a more gradual decline of more heat-resistant virus particles. The initial fall in infectivity was about two logs (99% of the initial material). In the second phase, the approximate times taken to reduce the infectivity by 1 $\log_{10}$ were 19 minutes at 700C, 11 minutes at 750C, and 3 minutes at 800C. This work confirmed the heat resistance of the IBD virus, and it was concluded that reducing the probability of remaining infectivity to 0.1($\log 10^{-1}$) required heating for 50 minutes at 700C, or 9 minutes at 800C. It was further concluded that to reduce the probability of remaining infectivity to 0.001 ($\log 10^{-3}$), heating for 90 minutes at 700C or for 14 minutes at 800C was required.[9]

The New Zealand heat treatment requirements for imported poultry meat, which are based on these 1988 experiments, are 50 minutes at 700C, 9 minutes at 800C, or 1 minute at 1000C.[10]

However, a deficiency of this early work on IBD virus thermostability, from a biosecurity decision-making point of view, was that the thermostability of IBD virus in chicken meat as distinct from other suspension media or dilution media was not studied.[7]

In 1997, the Central Veterinary Laboratory in the United Kingdom carried out further research into the heat inactivation of IBD virus (CS88 strain) on behalf of the Australian Chief Veterinary

Officer.[11, 12] It was demonstrated that IBD virus in tissue homogenates survived high temperatures for an unexpectedly long time; chicken meat heated for 15 minutes at 800C still contained IBD virus at a titre of $10^{2.68}$ $CID_{50}$/g, that is 478 chick infectious doses (50%) per gram.

Several differences between the 1988[9] and 1997[11, 12] studies may have contributed towards the different results. The earlier experiment was conducted using the 52/70 virus strain, and it is possible that the CS88 strain used in the 1997 study is more heat resistant as well as being more virulent. In addition, the 1988 work was undertaken on a clarified suspension of the virus while the 1997 study used an unclarified suspension of infected tissues, a medium possibly more reflective of the condition under which chicken products are cooked. It was observed in the 1997 study that the suspension coagulated after heating for 60 minutes at 700C or 15 minutes at 800C, and this may have had some protective effect on the virus. Moreover, the titre of virus in the homogenate used in the 1997 study ($10^{5.50}$ $CID_{50}$/0.1 ml) was more that $10^{2.2}$ times higher than the 1988 study.

The Australian Quarantine and Inspection Service used the results of the 1997 study to formulate their requirements for the importation of cooked chicken meat into Australia.[13] The Australian conditions include heating to achieve a 6 D (million fold) reduction in the titre of virus in meat, namely 165 minutes at 74□C, 158 minutes at 75□C, 152 minutes at 76□C, 145 minutes at 77□C, 138 minutes at 78□C, 132 minutes at 79□C, or 125 minutes at 80□C.[14]

A recent study directly examined the effect of cooking on the survival of IBD virus on poultry meat products. Four strains of IBD1 virus and one strain of IBD2 virus were seeded onto poultry products which were then cooked under typical industry conditions. No appreciable differences in thermostability were observed between the strains tested. It was shown that while cooking inactivated most of the virus, some infectious virus was still recoverable from drumsticks and chicken patties cooked to internal temperatures of 71□C and 73.8□C respectively.[15]

### 3.3.7.4 Epidemiology

IBD serotype 1 viruses mainly infect fowl, but they have also been isolated from turkeys and ducks.[3] Antibody to IBD1 has been found in geese, shearwaters, terns, common noddy, silver gulls, ducks and penguins.[2, 16]

The IBD virus is transmitted by the faecal-oral route. Chickens are highly susceptible to oral infection. During the acute phase of infection, birds excrete large amounts of the virus in their faeces for up to 2 weeks following infection. The virus is highly contagious and spreads rapidly by direct contact and by contamination of food, water and litter. Aerial spread is not important and there is no evidence for vertical transmission. Wild birds, rodents, humans or fomites may be responsible for mechanical transmission between flocks.[2, 3, 17]

### 3.3.7.5 Occurrence

IBD virus is distributed world-wide.[2, 3]

Serological evidence of IBD in broiler chickens was detected in New Zealand in 1993. Cross neutralisation tests confirmed that the isolates belonged to serotype 1. Pathogenicity tests demonstrated that the isolates did not cause clinical signs in SPF chickens. Thus it was concluded that the New Zealand isolates were apathogenic. However, the isolates did produce bursal damage and immunosuppression.[18] The virus has been identified as a relatively avirulent, possibly vaccinal, strain that is less contagious than classical strains. Given attention to flock hygiene and repopulation policies, it appears possible to eliminate infection from farms which are currently infected.[19]

### 3.3.7.6 Effect of introduction

As IBD virus causes disease only in chickens, its introduction would be expected to impact only on the poultry industry.

The effect on the poultry industry would depend on the virulence of the introduced strain and the extent to which it spread before being detected.[17] The highly virulent form of the disease which was recognised in 1987 in Europe can cause exceptionally high mortalities (10-15 % is common, and figures even above 65% have been recorded).[2] Highly pathogenic IBD in Asia frequently causes a 30% flock mortality as well as other losses including deaths and downgrading due to secondary bacterial and viral infections following immunosuppression.[11]

If an intermediate strain of IBD were to become endemic, the clinical signs might be less obvious than with the acute disease, but increased losses would be expected from a variety of other diseases due to the immunosuppressive effects of IBD virus.[20] A difference in economic returns between infected and uninfected flocks of 11-14% was demonstrated in Northern Ireland.[21] Even vaccine strains may cause considerable damage, including bursal atrophy, reduced weight gain, immunosuppression and death in chicks without maternal antibody.[2, 3].

It might be difficult to restrict the spread of IBD viruses, although New Zealand producers appear to have been successful in controlling the strain introduced in 1993.[19]

### 3.3.7.7 Chicken meat as a vehicle

Following oral infection, the virus is rapidly carried by macrophages and lymphoid cells of the gut to the liver, and from there via the bloodstream to the bursa, where there is massive virus replication in macrophages by 11 hours post-infection. This leads to a second and pronounced viraemia and secondary replication in other organs.[2] Large amounts of the virus are shed in the faeces.[2, 3]

In 1997, the Central Veterinary Laboratory in the United Kingdom investigated the dissemination of IBD virus (strain CS88) in the tissues of infected chickens.[11, 12] The virus was found in liver, kidney, faeces, bursa and blood samples from 24-96 hours post-infection. It was detected in muscle homogenates at 48, 72 and 96 hours post-infection. The titres of IBD virus in muscle tissue were significantly lower than the titres that would be expected in the bursa. Previous studies had shown that it was possible for the titre of virus in the bursa to be more than $10^6$ per

gram, whereas in the 1997 study the virus titre per gram of muscle tissue peaked at less than $10^{1.5}$ (at 3 days post-infection). It was concluded that the CS88 strain of IBD virus is widely disseminated throughout tissues and organs of infected chickens for at least 96 hours post-infection.[12]

MAF-commissioned trials carried out recently in the USA have also investigated the persistence of IBD virus in various tissues of infected chickens. Pooled samples of muscle, liver, kidney, spleen, lungs and bursa harvested from chickens at 4, 7, 9, 11, 16, 18 and 21 days post-infection were infectious to 3-week-old chickens when inoculated by the oral route. The experiment was repeated and infected chickens were held for 3, 4 and 5 weeks post-infection. Pooled samples collected from these chickens were found to be infectious at 3 and 4 weeks post-infection, but not at 5 weeks. This work indicated that the IBD virus persists in the tissues of recovered birds for at least 4 weeks after infection.[22]

A further experiment was carried out in the USA to examine the duration of persistence of IBD virus in different tissues of chickens. Samples of breast muscle, liver, lung, kidney, spleen, and bursa were taken from chickens killed at weekly intervals for 5 weeks following infection with IBD virus. Breast muscle was not infective at any sampling in this study, but liver, lung, kidney, spleen and bursa contained infectious virus at 7 days post-infection. At 14 days post-infection, only bursal tissue remained infective, and it remained infective until 28 days but was negative at 35 days. This experiment confirmed that the bursal tissue is the site of persistence of IBD virus.[23]

In summary, IBD virus has been detected in muscle tissue at 2, 3 and 4 days post-infection,[12] but not at 7 days post-infection.[23] Since neither of these experiments have looked for the virus on days 5 and 6 post-infection, it is reasonable to be cautious and to conclude that the virus may be present in muscle from 2 - 6 days post-infection.

All chicken carcasses include kidney tissue, and it has been stated that in all birds some fragments of the bursa of Fabricius will remain after processing, and in 10-30% of birds the whole of the bursa may be left in the carcass.[24]

Chickens usually become infected with IBD virus at 3-6 weeks of age and are slaughtered at 5-7 weeks of age.

As the virus is present in faeces of infected birds, some faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, but unlike bacteria of public health concern viruses will not multiply on the carcass surface.

As the virus may be present in liver tissue of infected chickens for at least 2 weeks and in bursal tissue for at least 4 weeks, and as at least fragments of bursal tissue may be present in chicken carcasses after processing, it can be concluded that chicken carcasses could serve as a vehicle for the introduction of IBD virus. Bone-in and boneless chicken meat cuts would be less likely to harbour the virus.

### 3.3.7.8 Risk of introduction

There are two possible routes of introduction of IBD virus with imported poultry. As the virus is excreted in faeces, faecal contamination during processing of infected birds might result in the virus being carried on the carcass surface. However, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

More importantly, as the virus may be present in the tissues of infected or recovered birds for as long as 4 weeks after infection, any bird killed during that period could harbour the virus. As the virus does not persist in muscle tissue for longer than 6 days, the risk of introducing the virus in imported bone-in or boneless cuts of chicken meat would be less than the risk posed by the importation of whole chicken carcasses.

As the virus is known to be extremely resistant to inactivation by freezing or cooking, it is considered that scraps from infected imported chicken meat could act as a source of IBD virus for chickens.

As IBD virus is transmitted orally, it is considered that for the virus to become established in New Zealand, it would be necessary to feed the virus to poultry in this country. For imported chicken meat products to be the source of that virus would require the importation of chicken meat products which were carrying the virus, and there would have to be a route by which the imported chicken meat products could transmit the virus to poultry flocks in this country. It is considered that the most likely route would involve scraps of imported chicken meat in household garbage. For household garbage to contain viable IBD virus originating from infected chicken meat being imported into New Zealand, the virus would have to survive on the chicken meat products, parts of which would have to be thrown out as garbage, and the garbage would have to be fed to a poultry flock. Poultry with the greatest chance of coming into contact with household garbage are those in backyard flocks.

If infection were to become established in backyard chickens it would be able to spread to other flocks, and therefore infected backyard poultry flocks could act as sources of IBD virus for commercial flocks.

To investigate the likelihood of the introduction and establishment of IBD in backyard flocks, two different quantitative risk assessment models were developed to assess the risk posed by IBD virus carried in chicken carcasses, bone-in cuts of chicken meat or boneless chicken meat.

The first model is presented in Appendix 1.

The model indicates that even if only 0.1% of the current annual broiler consumption (measured in terms of carcasses or carcass equivalents) were imported from countries with endemic IBD, even in the form of boneless cuts, without appropriate safeguards it is virtually certain that IBD would become established in backyard chicken flocks, as the highly heat-resistant virus is likely to survive cooking and any chicken scraps in household garbage which is fed to backyard poultry would contain enough viable virus to result in infection.

Because of the importance of these assumptions, a further model was developed to utilise the heat inactivation data generated by the Quality Control Unit at CVL in the UK in 1997[11], and to express infectivity of cooked chicken meat scraps in terms of $CID_{50}$ per gram of tissue. This second model is presented in Appendix 2. The conclusions reinforce those reached in the model presented in Appendix 1; that is, that cooking cannot be considered a reliable safeguard against IBD.

### 3.3.7.9 Recommendations for risk management

*3.3.7.9.1 Uncooked chicken meat:*

Meat products must be sourced from broiler flocks demonstrated to be free from infection with IBD virus[8] and not vaccinated with live IBD vaccines.

3.3.7.9.2 Cooked chicken meat:

The modelling of the heat inactivation of the IBD virus, presented in Appendix 2, indicates that realistic cooking times cannot be relied on as a safeguard against IBD virus, so meat products must be sourced from broiler flocks demonstrated to be free from infection with IBD virus[i] and not vaccinated with live IBD vaccines.

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### 3.3.8 MAREK'S DISEASE{tc \l3 "3.3.8 MAREK'S DISEASE}

#### 3.3.8.1 Aetiology

Marek's disease (MD) is caused by gallid herpesvirus 2, an unassigned virus in the family Herpesviridae.[1] There is considerable variation in virulence of MD viruses, ranging from almost avirulent to very virulent.[2]

#### 3.3.8.2 The disease

MD is a neoplastic lymphoproliferative disease of chickens characterised by mononuclear cell infiltration and the development of lymphomas, principally in the peripheral nerves and visceral organs.[3] The disease takes a chronic course (classic form) or an acute course (acute form) and mainly affects 6-20 week old birds. The incubation period varies considerably, from 2-20 weeks. Before the introduction of vaccination, mortalities were 10% - 15% (classic form), 20 - 30% (acute form) and up to 70% in some outbreaks.[4]

The classical form is characterised by peripheral nerve enlargement and paralysis. In the acute form, there are multiple and diffuse lymphomatous tumours in visceral organs. In general, the more virulent the virus and the more susceptible the chicken, the more likely that infection will result in the acute form. In addition, there is a third less common form of the disease which results from acute viral encephalitis and is termed transient paralysis.[3] Virulent MD isolates are highly pathogenic and oncogenic for HVT-vaccinated and genetically resistant chickens. Virulent MD viruses also cause immunosuppression in susceptible chickens.[2]

#### 3.3.8.3 Physical and chemical stability

When present in skin epithelial cells, the virus is relatively resistant to environmental factors. The virus survives extended freezing. Cell-free preparations of MD virus are inactivated by 560C for 30 minutes and 600C for 10 minutes.[2, 3]

The virus is not likely to be adversely affected by the pH changes associated with *rigor mortis*.[2] Cell-free preparations are inactivated when treated for 10 minutes at pH 3 or 11, but are relatively stable at pH 7.0.[2, 3]

#### 3.3.8.4 Epidemiology

Under natural conditions MD infection occurs almost exclusively in chickens. MD infections may also occur in quail and turkeys.[2, 3]

MD is highly contagious and the virus spreads by direct or indirect contact. The agent is excreted by apparently normal infected chickens during the incubation period and after the development of clinical signs. The agent can be excreted as early as 2 weeks post-infection. Infection occurs via the respiratory route.[2, 3, 5]

Infectious virus is associated with feathers, dander and faeces. Infection probably persists indefinitely in infected birds; some chickens were found to shed the virus from skin for 76 weeks. Chickens that survived exposure to MD virus at 2-3 weeks of age were shown to excrete the agent at 16 and 24 months of age, and the virus was demonstrated in the blood of such birds at 24 months of age.[2, 3]

Vaccines are available for the control of MD. None of the commercially available vaccines prevent superinfection with virulent virus, but viraemia and virus shedding from feather follicles are reduced. Vaccinated chickens exposed to virulent MD virus become chronically infected with both the vaccine virus and the challenge virus.[6]

### 3.3.8.5 Occurrence

MD occurs world-wide,[2, 3, 7] including New Zealand. Very virulent MD virus strains were first identified in the late 1970s, mainly in HVT-vaccinated flocks with high MD losses, and these now appear to be the dominant type in many countries.[2, 7] Virulence studies have not been done on New Zealand strains of MD virus,[8] but virulent strains do occur, necessitating vaccination with HVT vaccines.[9] However, the so-called very virulent form of the disease has not been reported in this country.

### 3.3.8.6 Effect of introduction

Economic effects of MD infection include mortality, poor feed conversion, reduced productivity, carcass condemnation and the costs associated with vaccination.[3, 4] The introduction of very virulent strains of MD into New Zealand would result in more clinical disease and, perhaps, reduced efficacy of vaccines.

### 3.3.8.7 Risk of introduction in chicken meat

The MD virus replicates in many tissues including skin epithelial cells in feather follicles.[2] The infectious agent is present in tumours, nerves, whole blood and many other organs of chickens affected with MD.[2, 3]

As MD is almost universal in chickens, slaughter-age birds are likely to be infected. The virus is likely to be present in skin at the time of slaughter and would survive in frozen chicken meat, as it is not affected by freezing.

Commercial poultry flocks are vaccinated against MD, but vaccinated chickens may harbor and excrete virulent strains, although the amount of virus excreted is less than that shed by non-vaccinated chickens.

Although it is likely that the skin of imported carcasses could contain MD virus, the route of infection is respiratory by inhalation of infectious dust. Therefore, meat is not considered a vehicle for transmission of the virus.

The risk of introduction of exotic strains of MD virus in imported chicken meat products is considered to be negligible.

### 3.3.8.8 Recommendations for risk management

No specific safeguards are required.

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### 3.3.9 NEWCASTLE DISEASE / PARAMYXOVIRUS 2 / PARAMYXOVIRUS 3 {tc \l3 "3.3.9 NEWCASTLE DISEASE / PARAMYXOVIRUS 2 / PARAMYXOVIRUS 3 }

#### 3.3.9.1 Aetiology

Newcastle disease (ND) is caused by a member of the genus *Rubulavirus* in the family Paramyxoviridae. There are nine avian paramyxoviruses, all within this genus.(1) Newcastle disease is caused by avian paramyxovirus 1 (PMV-1). In addition to Newcastle disease, this risk analysis considers avian paramyxovirus 2 and avian paramyxovirus 3 (PMV-2 and PMV-3).

#### 3.3.9.2 The disease

All birds appear susceptible to infection with ND viruses, but the pathogenicity varies greatly with the host. Chickens are highly susceptible, but ducks and geese may be infected and show few or no clinical signs, even with strains lethal for chickens.(2)

Little is known about the pathogenicity of avian paramyxoviruses for non-commercial avian species, as the amount of investigation into the disease potential of these viruses has been directly related to the perceived economic importance of the avian species concerned. Thus, although disease caused by PMV-1 is most commonly reported in poultry, it appears that there is a complete spectrum of virulence of PMV-1 viruses for most species of birds, from inapparent infection to 100% mortality.(3) There have been numerous reports of severe clinical disease and high mortalities caused by PMV-1 in a number of species of caged psittacines and passerines.(4)

It is very difficult to predict how a PMV-1 virus from one species of bird will behave in another species. Chickens may either be unaffected or may die when infected experimentally with a PMV-1 virus which causes severe disease in parrots, and likewise a strain which is 100% fatal to young chickens may cause few-to-no deaths in pigeons or psittacine birds.(4)

In chickens, the pathogenicity of ND is determined chiefly by the strain of the virus, although dose, route of administration, age of the chicken, and environmental conditions all have an effect.(2) In general, the younger the chicken, the more virulent the disease. With virulent viruses in the field, young chickens may experience sudden deaths without obvious clinical signs, while in older birds the disease may be more protracted with characteristic clinical signs; listlessness, increased respiration, weakness, prostration and death.(2)

The neurotropic velogenic form of the disease has been reported mainly from the USA. In chickens it is characterised by sudden onset of severe respiratory signs, followed a day or two later by neurological signs.(2)

Mesogenic strains usually cause respiratory disease in field infections. In adult birds there may be a marked drop in egg production that may last for several weeks. Nervous signs may occur uncommonly, but mortality is usually low.(2)

The clinical signs produced by specific viruses in other hosts may differ widely from those seen in chickens. In general, turkeys are as susceptible as chickens to infection with ND virus, but

clinical signs are usually less severe. Although readily infected, ducks and geese are usually regarded as resistant even to the strains of ND virus most virulent for chickens. However, outbreaks of severe disease in ducks infected with ND virus have been described. Outbreaks of virulent ND have been reported in most game bird species and the disease appears similar to that in chickens.[2]

One of the most characteristic properties of different strains of ND is the great variation in pathogenicity for chickens. Strains of ND virus have been grouped into five pathotypes based on the clinical signs seen in chickens; viscerotropic velogenic, neurotropic velogenic, mesogenic, lentogenic and asymptomatic enteric.[5] However these groupings are rarely clear-cut, and even in infections of SPF chickens, considerable overlapping is seen. Several pathogenicity indices have been devised to classify PMV-1 viruses isolated from chickens. The mean death time in eggs (MDT) has been used to classify strains as velogenic, mesogenic, and lentogenic. However, the intravenous pathogenicity index (IVPI) and the intracerebral pathogenicity index (ICPI) are considered more objective. As viruses capable of producing quite severe disease may have IVPI values of 0, the ICPI test is used most often for such assessments.[5] Generally, lentogenic vaccines have an ICPI of up to 0.4. Asymptomatic enteric viruses are usually slightly lower, mesogenic vaccines are usually around 1.4 and velogenic viruses are 1.7 upwards.[6]

PMV-2 has been associated with mild respiratory or inapparent diseases in chickens and turkeys. Unlike ND virus, PMV-2 infections have been reported to be more severe in turkeys than in chickens, and there has been a report of severe respiratory disease, sinusitis, elevated mortality, and low egg production in turkey flocks infected with PMV-2 complicated by the presence of other organisms.[2]

Natural infections of domestic poultry with PMV-3 have been restricted to turkeys. Clinical signs are usually egg production problems, occasionally preceded by mild respiratory disease.[2]

Humans are susceptible to all pathotypes of ND virus, including lentogenic vaccine strains. Infections may occur in laboratory personnel, poultry slaughterhouse workers, and vaccinators applying live vaccines. Symptomless infections are more frequent than disease, and when clinical signs are seen, they are usually restricted to a mild conjunctivitis. Systemic manifestations are rare, and most patients recover fully in a week.[7]

### 3.3.9.3 Physical and chemical stability

As avian paramyxoviruses have a lipoprotein envelope, they lose infectivity at high or low pH, and their heat lability rapidly increases at temperatures above 40□C.[3]

ND virus is relatively stable at pH values between 3 and 11,[8] so is unlikely to be affected by pH changes accompanying *rigor mortis*. It can survive freezing for extended periods. It has been isolated from poultry carcasses frozen for 2 years, and it may survive on poultry meat wrappings for as long as 9 months when stored at -140C to -200C.[9]

It is widely accepted that the virus is relatively sensitive to thermal inactivation, and is likely to be destroyed by cooking. Various heat treatments have been reported to be effective: 560C for

between 5 minutes and 6 hours, 600C for 7 minutes, 600C for 30 minutes, 700C for 50 seconds and 1000C for 1 minute.[8, 10]

Recent studies have confirmed the heat sensitivity of ND virus in homogenised chicken meat, and the following heat treatments have been reported to be effective in reducing the titre of the virus in poultry meat by 1 $\log_{10}$: 650C for 120 seconds, 700C for 82 seconds, 740C for 40 seconds and 800C for 29 seconds.[11]

### 3.3.9.4 Epidemiology

As ND virus infections have been reported in at least 236 species from 27 of the 50 Orders of birds, it seems probable that all birds are susceptible to infection.[12] However, it is only in the last 20 or 30 years that ND viruses which are not pathogenic for chickens or any other species of birds have been described. These viruses were mostly discovered as a result of avian influenza surveillance studies.[2]

Infection appears to take place either by inhalation or ingestion. Although conclusive experimental proof is lacking, it is generally accepted that ND virus is primarily transmitted by fine aerosols or large droplets that are inhaled by susceptible birds. In naturally occurring infections, large and small droplets containing virus will be liberated from infected birds as a result of replication in the respiratory tract or as a result of dust and other particles, including faeces. Inhalation of these virus-laden particles results in infection. During the course of infection of most birds with ND virus, large amounts of virus are excreted in the faeces. Ingestion of faeces is likely to be the main method of bird-to-bird spread for avirulent enteric ND virus.[2]

A number of methods of spread of ND virus have been implicated in various epidemics:[9]

- movement of live birds, including wild birds, pet/exotic birds, game birds, racing pigeons,
- commercial poultry;
- other animals;
- movement of people and equipment;
- movement of poultry products;
- airborne spread;
- contaminated poultry feed;
- water;
- vaccines.

There is little information on the spread of avian paramyxoviruses apart from ND virus. It is assumed that the methods of spread of PMV-2 and PMV-3 would be similar to that of PMV-1.[2]

Although vaccination may protect birds from the more serious consequences of ND virus infection, vaccinated chickens often become infected with virulent ND virus and shed the virus, although to a much reduced extent. The virus may be present in the faeces, tissues and organs (including muscle) even in well-vaccinated apparently healthy chickens.[9, 13]

The duration of virus excretion from vaccinated birds varies considerably; vaccinated flocks have shed virulent field virus for more than 4 months. In experiments involving turkeys, very few isolations of virus were made following challenge with virulent virus.[9, 13] Vaccine virus may spread laterally to susceptible flocks. Chickens vaccinated with mesogenic strains eliminate the virus in their faeces for up to 19 days. Lentogenic strains have been transmitted from vaccinated chickens to susceptible chickens by direct contact.[2, 12] Mesogenic vaccine virus was shed irregularly in the yolk of eggs for at least 1 month following vaccination, the greatest extent of vaccine shedding occurred during the first 9 days of vaccination.[9]

Mesogenic live vaccines tend to be used only where virulent ND is widespread.[12] La Sota vaccine has been shown to be present in reproductive organs after vaccination.[2]

### 3.3.9.5 Occurrence

Vaccination of poultry throughout the world makes assessment of the geographical distribution of ND difficult. The less virulent strains of ND virus probably occur worldwide in waterfowl and wild birds. There are very few poultry-keeping countries which have not reported ND in recent times, and there are frequent epidemics of the disease throughout Africa, Asia and Central and South America.[2] Sporadic epidemics occur in Europe despite vaccination programmes. In recent years, a series of outbreaks has affected birds in all the countries of the European Union, and the frequency of reported outbreaks has markedly increased since 1991.[13] About 40% of the outbreaks in Belgium, The Netherlands, Germany, and France have occurred in backyard or hobby poultry, which are able to be moved around Europe with relative ease.[13]

In 1998 an outbreak of highly virulent ND occurred in NSW, Australia. This was the first such outbreak in 60 years, and was apparently due to mutation of a lentogenic strain from wild birds.[14]

Survey results indicate that paramyxoviruses are not present in feral pigeons or native birds in New Zealand.[15] ND virus strains isolated from poultry in this country have all been of the pathotype asymptomatic enteric, with ICPI values of 0.0.[16] Lentogenic, mesogenic and velogenic strains have not been detected in this country.

The natural distribution of PMV-2 and PMV-3 is unclear, as many of the isolations of PMV-2, and to a lesser extent PMV-3, have been from imported cage birds in quarantine in England.[17]

PMV-2 is widespread in poultry flocks in many countries, more commonly in turkeys than in chickens.[2] Apart from turkeys and chickens, PMV-2 appears to be common in wild passerines, and has been isolated from caged passerines and psittacines in a number of countries.[2, 3]

PMV-3 has been found in turkey flocks in North America and Europe.[2] Besides turkeys, PMV-3 infection has been reported in caged passerines and psittacines in Europe and Asia. There have been no reports of isolation of PMV-3 from wild birds or of natural infections of chickens with this virus.[2, 3]

### 3.3.9.6 Effect of introduction

The effect of introduction of ND virus would depend on the strain introduced. The introduction of a velogenic strain could cause high mortalities which would severely affect individual poultry farmers.[18] Poultry products could become more expensive, and the small but expanding export trade in poultry products and genetic material would probably be affected.

ND could be difficult to contain if introduced. The recent European experience suggests that outbreaks in backyard or hobby poultry flocks would become common.[13] It is also possible that ND could infect native birds, possibly causing serious mortalities. The introduction of velogenic ND virus would result in considerable cost to the poultry industry in the implementation of disease control/eradication programmes.

However, even the introduction into New Zealand of mesogenic or lentogenic vaccine strains of ND virus, which have a considerable range of virulence,[2] could result in impaired productivity of commercial poultry and perhaps necessitate control measures.

The introduction of PMV-2 or PMV-3 into commercial poultry flocks might cause economic losses to poultry farmers.

#### 3.3.9.7 Chicken meat as a vehicle of ND virus

Recent studies in the United Kingdom showed that virus titres in muscle and faeces were about $10^4$ $EID_{50}$ (50% egg infectious doses)/g. The oral infectious dose of ND virus was also studied, and for ND virus Herts 33/56, $10^4$ $EID_{50}$ were required to establish infection in 3-week-old chickens when given orally.[19]

Experiments carried out in the United States involved the inoculation of 3-week-old chickens with a mesogenic strain of ND. Samples of muscle, liver, spleen, lung, kidney and bursa were collected and tested. Spleen, lung and bursa were the tissues with the most virus with the highest titres found in lung tissue. The virus was detected in muscle only at 4 days post-infection and was not detected in any tissues after 10 days post-infection. Tissue pools of muscle, liver, spleen, lung, kidney and bursa collected at 2, 4, 7 and 9 days post-infection were infectious for 3-week-old birds.[20]

The results of the above experiments confirm previous reports[3] that poultry meat is a suitable vehicle for the spread of ND and that poultry can be infected by the ingestion of contaminated meat scraps.

#### 3.3.9.8 Risk of introduction

Given the stability of ND virus in poultry meat, there is a relatively high risk that it could be introduced in uncooked poultry meat products. Should infected meat be imported, the virus

would probably be spread to backyard poultry in meat scraps. Intermediaries such as wild birds or insects could spread the virus to commercial poultry and possibly to native birds.

Vaccinated chickens may become super-infected with virulent ND virus and become virus shedders. The virus may be present in the tissues of vaccinated apparently healthy chickens. Vaccine strains are likely to be more virulent than ND strains occurring in New Zealand (ICPI = 0.0) and may spread to susceptible birds. Chickens vaccinated with mesogenic vaccine strains excrete the virus in faeces and it has been shown that tissues derived from chickens inoculated with mesogenic strains may transmit the virus.

As it is difficult to predict the pathogenicity of strains of PMV-1 in avian species other than that from which a particular strain is isolated, it is not possible to exclude the possibility that lentogenic or even asymptomatic strains which may be present in chicken flocks could have severe effects if introduced into native bird populations.

A quantitative risk analysis was carried out to re-assess recent experimental results on heat inactivation of ND virus and to compare its heat sensitivity with a known heat tolerant virus such as IBD. The analysis uses the predicted fall of titre after certain time/temperature treatments in an infectious dose model to assess the risk of introduction and establishment of ND in a backyard flock fed chicken scraps.

The model is presented in Appendix 3.

Given the assumptions in this model, in particular the initial viral titre in a chicken carcass, the current MAF time/temperature recommendations provide an adequate level of insurance against the risks associated with ND in imported cooked chicken meat. Detailed results of the model in terms of predicted cooking times to achieve a target titre are given in Appendix 3, Table 5.

Isolations of PMV-2 from domestic poultry have been rare. However, this virus is thought to spread in the same way as ND virus, so it is likely that poultry meat could pose a risk of introducing PMV-2 to New Zealand. As natural infections of domestic poultry with PMV-3 have been restricted to turkeys, chicken meat products are not considered to be a vehicle for the introduction of PMV-3 virus.

The importation of chicken meat is considered to pose a risk of introducing exotic strains of PMV-1 and PMV-2 to New Zealand.

Safeguards are considered necessary for these viruses.

### 3.3.9.9 Recommendations for risk management

*3.3.9.9.1 PMV-1 : Newcastle disease - uncooked chicken meat:*

A major difficulty in the formulation of safeguards against ND is the lack of an internationally agreed definition of what consitutes "Newcastle disease." The definition of ND given in the International Animal Health Code[21] is as follows : "ND is a disease of birds caused by strains of avian paramyxovirus type 1, significantly more virulent than lentogenic strains." A more precise definition is given in the European Community Directive 92/66/EEC definition of ND for which control measures should be imposed : "an infection of poultry caused by an avian strain of the paramyxovirus 1 with an ICPI in day old chicks greater than 0.7." This definition includes all highly virulent (velogenic) and moderately virulent (mesogenic) viruses and may include some of the lentogenic vaccines licensed in the EU.[6] It is also based on infection of birds and not on the presence of disease signs or mortality.

MAF considers it appropriate that safeguards for PMV-1 should aim to prevent the introduction of any strains of the virus which are more pathogenic than the strains already in this country. That is, safeguards should prevent the introduction of any strains of PMV-1 virus with an ICPI greater than 0.0. The required safeguards are as follows:

> When importing fresh/frozen chicken meat products, the consignment must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment comes from birds:[9]
>
> 1. which have not been vaccinated for PMV-1 ; and
>
> either
>
> 2. which, since hatching or for at least the past 21 days, have been kept in a country which is free[10] from infection with strains of PMV-1 with ICPI greater than 0.0;
>
> or

9 If chickens from infected flocks are likely to be slaughtered or processed in the same slaughter establishment, then chickens destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

10 A country may be considered free from PMV-1 (ICPI > 0.0) when it can be shown that PMV-1 (ICPI > 0.0) has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against PMV-1 (ICPI > 0.0).

3. which have been kept in an *establishment* [defined by the OIE Code] free from infection[11] with strains of PMV-1 with ICPI greater than 0.0 and not situated in a zone[12] which is infected with strains of PMV-1 with ICPI greater than 0.0.

*3.3.9.9.2 PMV-2 - uncooked chicken meat:*

The broiler flocks were kept in establishments that have remained free from evidence of PMV-2 infection[i] for the 21 days prior to slaughter.

*3.3.9.9.3 PMV-3:*

No specific safeguards are required.

*3.3.9.9.4 PMV-1 and PMV-2 - cooked chicken meat:*

The chicken meat product has been cooked to achieve a core temperature of 70 □C for 50 minutes, or 80 □C for 9 minutes.

## References


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11 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

12 A PMV-1 (ICPI > 0.0) infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

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# 4. SUMMARY OF RECOMMENDATIONS : CHICKEN MEAT AND CHICKEN MEAT PRODUCTS{tc \l1 "4. SUMMARY OF RECOMMENDATIONS : CHICKEN MEAT AND CHICKEN MEAT PRODUCTS}

To summarise, the following recommended safeguards are suggested for the importation of chicken meat and chicken meat products into New Zealand:

## 4.1 General recommendations for ALL importations

4.1.1 Chicken meat products to be derived from birds slaughtered in an abattoir approved by the competent authority.

4.1.2 Chicken meat products to be derived from birds which passed ante-mortem and post-mortem inspection procedures.

4.1.3 Chicken meat products to be certified as fit for human consumption.

4.1.4 Chicken meat products to be derived from broiler birds only, rather than culled breeders or layers, and giblets shall not be included.

4.1.5 HACCP programs to be implemented at all points in slaughter and processing

## 4.2 Specific recommendations

### *4.2.1 Salmonellae*

If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

#### *4.2.1.1 Salmonella pullorum and Salmonella gallinarum*

1. Country freedom or a free zone. Vaccination is not practised.[i]

   or

2. Flock of origin freedom[13]

[13] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

A flock accreditation program involving both parent and broiler flocks, approved by MAF New Zealand. Vaccination is not permitted.

or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*4.2.1.2 S. enteritidis* PT4 and *S. typhimurium* DT104

1. Country Freedom or a free zone[14]

or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. enteritidis* PT4 and *S. typhimurium* DT104 . The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of *S. enteritidis* PT4 and *S. typhimurium* DT104 and that there are no opportunities for cross contamination during transport, slaughter and processing.

or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*4.2.1.3 Salmonella arizonae*.

1. Country freedom or a free zone.[i]

or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. arizonae*. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of salmonellae and that there are no opportunities for cross contamination during transport, slaughter and processing.

or

[14] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*4.2.2 Avian infectious bronchitis*

For whole chicken carcasses, the broiler flocks should not have been immunised against IB with live vaccines and the birds should have showed no clinical signs of IB prior to slaughter.

For cooked chicken meat products, the chicken meat products must be subjected to heat treatment resulting in a core temperature of at least 600C for 5 minutes or 1000C for 1 minute.

*4.2.3 Big liver and spleen disease*

For all commodities under consideration, chicken meat products must be certified to have been derived from birds whose parent flocks did not show clinical signs of big liver and spleen disease in the previous 6 months.

*4.2.4 Highly pathogenic avian influenza (HPAI):*

*4.2.4.1 Fresh chicken meat*

For fresh chicken meat, MAF considers that the safeguards recommended by the Office International des Epizooties, as detailed in the OIE International Animal Health Code (OIE Code) are appropriate :

> When importing fresh chicken meat from HPAI free countries[15], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment of meat comes from birds which have been kept in an HPAI-free country since they were hatched or for at least the past 21 days.
>
> When importing fresh chicken meat from countries or zones considered infected with HPAI[16], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Health Code] attesting that the entire consignment of meat comes from birds which have been kept in an establishment free[17] from HPAI and not situated in an HPAI *infected zone* [defined by the OIE Code].

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15 A country may be considered free from HPAI when it can be shown that HPAI has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against HPAI.

16 A HPAI infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

17 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

In addition, the chicken meat products must come from birds which have not been vaccinated for avian influenza.

#### *4.2.4.2 Cooked chicken meat*

For chicken meat products from countries which do not meet the standards in 4.2.4.1, the chicken meat products must be subjected to heat treatment resulting in a core temperature of at least 600C for 5 minutes or 1000C for 1 minute.

### *4.2.5 Infectious bursal disease (IBD):*

Meat products must be sourced from broiler flocks demonstrated to be free from infection with IBD virus[18] and not vaccinated with live IBD vaccines.

### *4.2.6 Newcastle disease (ND):*

#### *4.2.6.1 PMV-1 - uncooked chicken meat*

When importing fresh/frozen chicken meat products, the consignment must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment comes from birds:[19]

1. which have not been vaccinated for PMV-1 ; and

   either

2. which, since hatching or for at least the past 21 days, have been kept in a country which is free from infection[20] with strains of PMV-1 with ICPI greater than 0.0;

   or

---

18 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

19 If chickens from infected flocks are likely to be slaughtered or processed in the same slaughter establishment, then chickens destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

20 A country may be considered free from PMV-1 (ICPI > 0.0) when it can be shown that PMV-1 (ICPI > 0.0) has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against PMV-1 (ICPI > 0.0).

3. which have been kept in an *establishment* [defined by the OIE Code] free from infection[21] with strains of PMV-1 with ICPI greater than 0.0 and not situated in a zone[22] which is infected with strains of PMV-1 with ICPI greater than 0.0.

*4.2.6.2 PMV-2 - uncooked chicken meat:*

The broiler flocks were kept in establishments that have remained free from evidence of PMV-2 infection[i] for the 21 days prior to slaughter.

*4.2.6.3 PMV-1 and PMV-2 - cooked chicken meat:*

The chicken meat product has been cooked to achieve a core temperature of 70 □C for 50 minutes, or 80 □C for 9 minutes.

21 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

22 A PMV-1 (ICPI > 0.0) infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

# PART TWO: RISK ANALYSIS FOR THE IMPORTATION OF BERNARD MATTHEWS TURKEY MEAT PREPARATIONS FROM THE UNITED KINGDOM{tc \l1 "PART TWO: RISK ANALYSIS FOR THE IMPORTATION OF BERNARD MATTHEWS TURKEY MEAT PREPARATIONS FROM THE UNITED KINGDOM}

## 5. INTRODUCTION{tc \l1 "5. INTRODUCTION}

Bernard Matthews Foods Limited (BMFL), a British company, first requested access to the New Zealand market for a range of turkey meat preparations in 1994. At that time, the products did not meet existing importation policies for poultry meat with respect to infectious bursal disease (IBD).

The company still wished to export the products and so it was decided that safeguards proposed by the United Kingdom Ministry of Agriculture, Fisheries and Food (UK MAFF) would be incorporated into a draft import health standard. The proposal was discussed with the Agricultural Security Consultative Committee (ASCC) and the ASCC Technical Subcommittee (Avian) and released for public consultation during 1996. Objections were raised by main stakeholders during the consultation phase and many technically-based questions were raised which needed to be addressed. It was determined that further technical assessment was necessary in order to provide sufficient information to enable the New Zealand Ministry of Agriculture and Forestry (MAF) to make a decision on whether or not to permit the importation of these products.

The products are manufactured in accordance with European Union Council Directive 71/118/EEC. Meat preparations are defined in Council Directive 94/65/EEC as "meat which has had food-stuffs, seasonings or additives added to it, or meat which has undergone a treatment insufficient to modify the internal cellular structure of the meat and thus to cause the characteristics of the fresh meat to disappear".

The range of products covered by this risk analysis includes uncooked deboned turkey meat roasts and crumbed, flash-fried deboned products. The preparations include edible tissues only, that is muscle, fat and skin and are derived from turkeys that have passed ante-mortem and post-mortem inspection. The processing premises are approved for export to the European Union, the United States of America, Canada and South Africa.

BMFL slaughter establishments are used exclusively for the slaughter of BMFL's own turkeys. The preparations are derived from turkeys slaughtered at 8, 12 and 23 weeks of age. It is proposed that turkeys intended for the New Zealand market will be derived from four nominated turkey houses and processed "first of the day" to avoid cross-contamination.

This risk analysis is intended to be read in conjunction with the more detailed generic risk analysis for the importation of chicken meat in Part One of this document.

It must be emphasised that this risk analysis is specific and examines the disease risks posed by the importation of BMFL turkey meat preparations from the United Kingdom only.

Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time the particular trade is negotiated.

# 6. HAZARD IDENTIFICATION: DISEASES OF CONCERN FOR THE COMMODITY{tc \l1 "6. HAZARD IDENTIFICATION: DISEASES OF CONCERN FOR THE COMMODITY}

## 6.1 Diseases reported to affect avian species{tc \l2 "6.1 Diseases reported to affect avian species}

Part One of this document includes a list of 86 diseases of concern. The diseases listed in Table 2.1 were used as a starting point to assess the potential risks of introducing exotic avian pathogens in the vehicle of BMFL turkey meat preparations from the United Kingdom.

Before embarking on this analysis, the Department of Conservation, the Ministry of Health and the Poultry Industry Association of New Zealand were asked to provide MAF with a list of turkey diseases that they considered should be included. As a result, several agents and disease syndromes that were not covered by the ratite risk analysis are included in this analysis; *Salmonella* Enteritidis phage type 4, *Salmonella* Typhimurium definitive phage type 104, avian polyomavirus and spiking mortality of turkeys. These agents and disease syndromes are shown in Table 6.4

## 6.2 Diseases reported to infect turkeys{tc \l2 "6.2 Diseases reported to infect turkeys}

The diseases listed in Table 2.1 were evaluated in order to determine which diseases would be taken into further consideration. The disease agents were assessed as to whether or not they had been reported to infect turkeys. This resulted in:

- a list of avian diseases which have not been reported to infect turkeys and which would NOT be subjected to further consideration. This list is presented in Table 6.1.

- a list of avian diseases which have been reported to infect turkeys and which would be subject to further consideration. This list is presented in Table 6.2.

## 6.3 Turkey diseases of concern with the potential for transmission in turkey meat{tc \l2 "6.3 Turkey diseases of concern with the potential for transmission in turkey meat}

The diseases listed in Table 6.2 were evaluated in order to determine which diseases would be taken into further consideration. All of the disease agents were assessed as to whether or not they have the potential to be transmitted in turkey meat.

The diseases were examined with regard to their mode of transmission. All disease agents thought to be capable of survival in or on turkey meat, as well as those agents excreted in the faeces are considered to have the potential for transmission in turkey meat. Disease agents that were not considered to be capable of transmission in turkey meat for various reasons, including:

- the disease is transmitted only by arthropods: aegyptianellosis[1], *Haemoproteus* infection[1], leucocytozoonosis[2], *Plasmodium* infection[2], *Trypanosoma* infection[2] turkey meningoencephalitis[1].

- the disease agent is an external parasite: Argasid ticks[1], Ixodid ticks[1]
- the pathogen is not found in any part of the edible carcass: verminous encephalitis[1], vesicular stomatitis[3]

- the agent is non-contagious: zygomycosis[4]

- the agent is fragile and dies quickly outside the living animal host: *Mycoplasma iowae*[2], hexamitiasis[2], lymphoproliferative disease[5], reticuloendotheliosis.[5]

This resulted in a list of diseases of concern that are thought to have the potential to be transmitted in turkey meat. This list is presented in Table 6.3.

The diseases listed in Tables 6.3 and 6.4 were subjected to qualitative risk assessment to determine the need for, and type of, safeguards. In some cases quantitative analysis was also carried out.

## References


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(5) McFerran J B, McNulty M S (eds) (1993) *Virus Infections of Birds*. Elsevier, Amsterdam.

## Table 6.1: Avian diseases that have not been reported in turkeys.

| Bacteria | Fungi, Parasites, Protozoa, Other | Viruses | Viruses continued |
|---|---|---|---|
| Anthrax | Balantidiasis | Alfuy virus | Myelocytomatosis |
| Infectious coryza | Filariae | Amazon tracheitis | Pacheco's disease |
| Intracellular infection in ducks | *Libyostrongylus* infection | Astrovirus infection of ducks | Papillomas in finches |
| | Ostrich tapeworm | Avian infectious bronchitis | Paramyxovirus 5 infection |
| | Sarcosporidiosis (exotic species) | Avian polyomavirus | Paramyxovirus 8 infection |
| | Ostrich fading syndrome | Beak and feather disease | Paramyxovirus 9 infection |
| | Encephalopathy | Big liver and spleen disease | Parvovirus infection of chickens |
| | | Borna disease | Quail bronchitis virus infection |
| | | Bunyavirus infection | Rabies |
| | | Cholangio-hepatitis virus | Rift Valley fever |
| | | Crimean-Congo haemorrhagic fever | Ross River virus infection |
| | | Derzsy's disease of geese | Wesselsbron disease |
| | | Duck hepatitis | |
| | | Duck hepatitis B virus infection | |
| | | Duck virus enteritis | |
| | | Equine encephalomyelitis | |
| | | Haemorrhagic nephritis and enteritis of geese | |
| | | Heron hepatitis B virus | |
| | | Herpesvirus infection of pigeons and wild birds | |
| | | Highlands J virus infection | |
| | | Japanese encephalitis | |
| | | Macaw wasting disease (Proventricular dilatation) | |
| | | Murray Valley encephalitis | |

## Table 6.2: Avian diseases that have been reported in turkeys.

| Bacteria | Fungi, Parasites, Protozoa, Other | Viruses |
|---|---|---|
| Aegyptianellosis | Zygomycosis | Astrovirus infection of turkeys |
| Avian chlamydiosis (exotic strains) | Argasid ticks | Avian adenovirus type II |
| Avian spirochaetosis | *Haemoproteus* infection (exotic species) | Avian rhinotracheitis |
| *Mycoplasma iowae* infection | Hexamitiasis | Coronaviral enteritis of turkeys |
| *Ornithobacterium rhinotracheale* infection | Ixodid ticks | Highly pathogenic avian influenza |
| Q fever | Leucocytozoonosis (exotic species) | Infectious bursal disease (exotic strains) |
| *Salmonella arizonae* | *Plasmodium* infection (exotic species) | Lymphoproliferative disease |
| *Salmonella gallinarum* | *Trypanosoma* infection | Marek's disease |
| *Salmonella Pullorum* | Verminous encephalitis | Newcastle disease |
| Tularaemia | | Paramyxovirus 2 infection |
| Turkey coryza | | Paramyxovirus 3 infection |
| | | Paramyxovirus 7 infection |
| | | Reticuloendotheliosis |
| | | Turkey meningoencephalitis |
| | | Turkey viral hepatitis |
| | | Vesicular stomatitis |

**Table 6.3: Agents that may have the potential for transmission in turkey meat.**

| Bacteria | Viruses |
|---|---|
| Avian chlamydiosis | Astrovirus infection of turkeys |
| Avian spirochaetosis | Avian adenovirus type II |
| *Ornithobacterium rhinotracheale* infection | Avian rhinotracheitis |
| Q fever | Coronaviral enteritis of turkeys |
| *Salmonella arizonae* | Highly pathogenic avian influenza |
| *Salmonella gallinarum* | Infectious bursal disease (exotic strains) |
| *Salmonella pullorum* | Marek's disease (exotic strains) |
| Tularaemia | Newcastle disease |
| Turkey coryza | Paramyxovirus 2 infection |
| | Paramyxovirus 3 infection |
| | Paramyxovirus 7 infection |
| | Turkey viral hepatitis |

**Table 6.4: Agents and syndromes included in the turkey meat risk analysis at the request of other organisations**

| Agent or syndrome | Request by |
|---|---|
| Avian polyomavirus | Department of Conservation |
| *Salmonella* Enteritidis PT 4 | Ministry of Health |
| *Salmonella* Typhimurium DT 104 | Ministry of Health |
| Spiking mortality of turkeys | Poultry Industry Association |

# 7. QUALITATIVE RISK ASSESSMENT{tc \l1 "7. QUALITATIVE RISK ASSESSMENT}

## *7.1 BACTERIAL INFECTIONS*{tc \l2 "*7.1 BACTERIAL INFECTIONS*}

### 7.1.1 AVIAN CHLAMYDIOSIS{tc \l3 "7.1.1 AVIAN CHLAMYDIOSIS}

This disease has been assessed in Part One of this document (see section 3.1.1). The risk of introducing chlamydiae in imported BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

### 7.1.2 AVIAN SPIROCHAETOSIS{tc \l3 "7.1.2 AVIAN SPIROCHAETOSIS}

Avian spirochaetosis has been assessed in Part One of this document (see sections 3.1.2 and 3.1.3). Borrelliosis has never been reported in the United Kingdom,(1) and intestinal spirochaetosis is rarely reported in birds other than laying hens.(2)

Furthermore, the organs harbouring the pathogen (that is, intestines) are not included in BMFL turkey meat preparations.

The risk of introduction of avian spirochaetosis in BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

## Reference


(1) FAO-OIE-WHO (1997) *Animal Health Yearbook: 1995*. Food and Agriculture Organization of the United Nations, Rome.

(2) Swayne DE (1997) Avian intestinal spirochetosis. In : Calnek BW (ed) *Diseases of Poultry*. Tenth Edition. Pp 325-32.

### 7.1.3 *ORNITHOBACTERIUM RHINOTRACHEALE*{tc \l3 "**7.1.3 *ORNITHOBACTERIUM RHINOTRACHEALE*}**

The disease caused by this organism has been assessed in Part One of this document (see section 3.1.4).

The risk presented by the importation of BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

### 7.1.4 Q FEVER{tc \l3 "7.1.4 Q FEVER}

This disease has been assessed in Part One of this document (see section 3.1.5).

The risk that *C. burnetii* could be introduced in BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

### 7.1.5 AVIAN SALMONELLOSIS{tc \l3 "7.1.5 AVIAN SALMONELLOSIS}

Avian salmonellosis caused by *Salmonella arizonae, S. pullorum, S. gallinarum,* and paratyphoid salmonellae has been assessed in Part One of this document (see section 3.1.6).

The risk and effect of introduction of these organisms in turkey meat products is considered to be similar to the risk presented by chicken meat products.

Safeguards are considered necessary for all salmonellae of concern.

#### 7.1.5.1 Recommendations for risk management

If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

*7.1.5.1.1 Salmonella pullorum and Salmonella gallinarum*

1. Country freedom or a free zone. Vaccination is not practised.[23]

   or

2. Flock of origin freedom[i]

   A flock accreditation program involving both parent and broiler flocks, approved by MAF New Zealand. Vaccination is not permitted.

   or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*7.1.5.1.2 S. enteritidis* PT4 and *S. typhimurium* DT104

1. Country Freedom or a free zone[i]

   or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. typhimurium* DT104 and *S. enteritidis* PT4. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry

[23] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

destined for export to New Zealand are free of *S. typhimurium* DT104 and *S. enteritidis* PT4 and that there are no opportunities for cross contamination during transport, slaughter and processing.

or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*7.1.5.1.3 Salmonella arizonae*.

1. Country freedom or a free zone.[24]

or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. arizonae*. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of salmonellae and that there are no opportunities for cross contamination during transport, slaughter and processing.

or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

[24] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

### 7.1.6 TULARAEMIA{tc \l3 "**7.1.6 TULARAEMIA}**

This disease has been assessed in Part One of this document (see section 3.1.7).

Tularaemia has never been reported in the United Kingdom.(1)

The risk of introducing tularaemia by importing BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

## Reference

(1) FAO-OIE-WHO (1997) *Animal Health Yearbook: 1995*. Food and Agriculture Organization of the United Nations, Rome.

### 7.1.7 TURKEY CORYZA{tc \l3 "7.1.7 TURKEY CORYZA}

This disease has been assessed in Part One of this document (see section 3.1.8).

The risk of introduction of *Bordetella avium* in the vehicle of imported BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

## *7.2 VIRAL INFECTIONS*{tc \l2 "*7.2 VIRAL INFECTIONS*}

### 7.2.1 ASTROVIRUS INFECTION OF TURKEYS{tc \l3 "7.2.1 ASTROVIRUS INFECTION OF TURKEYS}

#### 7.2.1.1 Aetiology

Astroviruses have not yet been classified taxonomically. Astroviruses isolated from turkeys have been shown to be antigenically distinct from astroviruses isolated from ducks.[1]

#### 7.2.1.2 The disease

Astroviruses have been identified as the cause of duck viral hepatitis type II (duck astrovirus hepatitis)[1, 2] and have also been associated with enteric disease in turkey poults.[1, 3]

Astrovirus infections of turkeys occur commonly in the first 4 weeks of life and are rare in older turkeys. Astrovirus infections in commercial turkeys have been associated with a syndrome characterised by diarrhoea, listlessness, nervousness, reduced growth and increased mortality in poults.[1, 3] Clinical signs of the disease usually last 10-14 days. Morbidity ranges from mild to moderate with only slight mortality.[3] The precise role of astroviruses in this syndrome is still to be defined.[1, 3]

#### 7.2.1.3 Physical and chemical stability

Duck astrovirus is stable at pH 3.0, which is below the ultimate pH of poultry meat (pH 6-7). It can withstand heat at 600C for 5 minutes, but is inactivated by 600C for 10 minutes.[4]

#### 7.2.1.4 Epidemiology

Astroviruses have been reported to cause disease in humans, pigs, lambs, calves, turkey poults and young ducks.[1] Turkeys appear to be the only avian species naturally infected by astroviruses, apart from the antigenically distinct astrovirus which affects ducks only.[1, 2, 3]

Astroviruses may be detected in the intestinal contents and faeces of affected turkeys. Spread probably occurs by ingestion of infective virus in the faeces of affected birds.[1, 2, 3] Oral inoculation of poults with astrovirus resulted in the production of watery droppings and frothy yellow-brown droppings from 3 to at least 13 days post-inoculation. There is no evidence for vertical transmission in turkeys or any other species.[1]

Astroviruses can be detected in the intestinal contents prior to the onset of clinical signs and shedding of astroviruses into the intestinal tract wanes before clinical signs abate. Therefore, poults in the later stages of disease may display clinical signs but may not have detectable levels of astrovirus present in their intestinal tract. Experimentally infected poults cease astrovirus shedding by 14 days post-infection. Whether convalescent birds are protected from further infection has not been determined. However, this appears to be true in naturally infected turkeys since the viruses are rarely detected in turkeys beyond 5 weeks of age.[3]

### 7.2.1.5 Occurrence

Astrovirus infection of turkeys has been reported in the United Kingdom and United States of America.[1, 2, 3]

### 7.2.1.6 Effect of introduction

Where the astrovirus occurs, it is usually the most prevalent virus infection other than rotavirus in 1-5 week old poults with enteric disease. Growth reduction in affected poults is likely to cause significant economic losses.[3]

### 7.2.1.7 Risk of introduction in turkey meat

The astrovirus which affects ducks is present in high titres in the livers of infected birds.[1, 2] It is assumed that the same might be the case for turkey astrovirus. There is no evidence for a viraemia.[2] Astroviruses are excreted in the faeces of affected turkeys and birds can be infected by ingestion of virus in faeces.

Although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface. Furthermore, astrovirus infections of turkeys occur in the first few weeks of life and the virus is rarely detected in turkeys beyond 5 weeks of age. BMFL turkeys are slaughtered at or after 8 weeks of age.

In addition, astroviruses appear to be highly host-specific, turkeys are the only avian species shown to be naturally infected by the turkey astrovirus.

The risk of astroviruses being introduced into poultry flocks in New Zealand through BMFL turkey meat preparations is considered to be negligible.

### 7.2.1.8 Recommendations for risk management

No specific safeguards are required.

## References


(1) McNulty MS, McFerran JB (1996). Astroviruses. In : Jordan FTW, Pattison M (eds) *Poultry Diseases.* Fourth Edition. Pp 226-8. Saunders, London.

(2) McNulty MS (1993) Astrovirus infection of turkeys. In: McFerran J B, McNulty M S (eds) *Virus Infections of Birds*. Pp 509-11. Elsevier, Amsterdam.

(3) Reynolds DL (1997) Astrovirus infections. In : Calnek BW (ed) *Diseases of Poultry*. Tenth Edition. Pp 701-5.

(4) Porterfield J S (1989) *Andrewes' Viruses of Vertebrates*. Fifth Edition. Bailliere Tindall, London.

## 7.2.2 TURKEY HAEMORRHAGIC ENTERITIS{tc \l3 "7.2.2 TURKEY HAEMORRHAGIC ENTERITIS}

### 7.2.2.1 Aetiology

Avian adenovirus group II splenomegaly of chickens has been assessed in Part One of this document (see section 3.3.1). A related group II avian adenovirus causes turkey haemorrhagic enteritis (THE).(1)

### 7.2.2.2 The disease

THE is an acute disease of 4-12-week-old turkeys, characterised by depression, bloody droppings, and sudden death. Mortality in field outbreaks has varied from over 60% to less than 0.1%. In naturally infected flocks, all signs of disease usually subside within 6-10 days after the first observation of bloody droppings.(1) Outbreaks may be precipitated by overcrowding, chilling, or a low plane of nutrition.(2)

### 7.2.2.3 Physical and chemical stability

Infectivity of THE virus has been shown to be destroyed by heating at 70□C for 1 hour.(1) However, infectivity was not destroyed by heating at 650C for 1 hour, storage for 6 months at 4 □C or 4 years at -40 □C, or maintenance at pH 3.0 at 25 □C for 30 minutes.(1) Chlorination (treatment with 0.0086% sodium hypochlorite) has been shown to destroy the THE virus.(1) This chlorine concentration corresponds to approximately 10 ppm available chlorine.

### 7.2.2.4 Epidemiology

The virus is widespread in turkey flocks in the United Kingdom without showing signs of disease. Most flocks develop antibody between 8 and 19 weeks of age.(2) The fact that the clinical syndrome occurs in only a fraction of infected flocks suggests either that most THE strains are avirulent or that the THE viruses are avirulent alone but may predispose birds to infection by some other organism.(3)

THE isolates have produced spleen swelling and lesions in all avian species where infection has been attempted (golden pheasants, peafowl, chickens, chukars) but clinical disease has been seen only in turkeys.(1)

Transmission of Group II avian adenoviruses appears to be by the faecal-oral route.(1) Litter from THE virus infected flocks is infectious, and disease often strikes in houses where it has occurred previously.(3) Unlike the group I avian adenoviruses, there is no evidence for egg transmission of Group II viruses.(2)

In turkeys infected with THE virus the highest virus titre is found in the spleen.(3) Electron micrographs suggest that THE viruses are replicated in cells of the reticuloendothelial system, primarily in the spleen. These findings are supported by immunodiffusion studies which indicate that viral antigen is concentrated in the spleen, is barely discernible in a small percentage of liver

and serum samples, and is not detectable in thymus, bursa of Fabricus, intestinal wall or muscle.[1] Using the ELISA, which is more sensitive, THE viral antigen has been detected in turkeys in small amounts from 2 -6 days post infection in the spleen, liver, intestine, kidney and bone marrow. Peak titres were found in the spleen on day 3 post infection and virus was not detected after day 6.[4]

Group I avian adenoviruses are known to be shed in faeces for up to 2 months.[5] In the absence of specific information on THE, this analysis assumes that faecal shedding may be of similar duration. However, since latency is a feature of adenovirus infections,[3] it is assumed that reactivation and faecal shedding of the THE virus can occur periodically through the life of the bird.

#### 7.2.2.5 Occurrence

The THE virus is widely distributed in turkey flocks in the UK.[2] One outbreak of THE has been reported in imported turkeys in New Zealand,[6] but it appears that the virus has not become established.

#### 7.2.2.6 Effect of introduction

The effect of introduction of THE into New Zealand poultry flocks is difficult to predict. Chickens could become infected, but would not be expected to show any clinical signs. If the virus did find its way into turkey flocks, based on the UK experience it might be expected that most turkey flocks would seroconvert without showing clinical signs. If turkey flocks were to become infected with a pathogenic strain, there could be outbreaks of acute mortality in turkeys.

#### 7.2.2.7 Risk of introduction in turkey meat

There is a limited and short-lived distribution of Group II adenoviruses in the tissues of infected birds, and the vast majority of infectivity is concentrated in the reticuloendothelial tissues, which are removed at slaughter. Infectivity is barely discernable in a small proportion of liver samples, and is not present in muscle.

Therefore it is unlikely that the THE virus would be found in the tissues of turkeys slaughtered and processed for human consumption.

Infection is usually subclinical and the virus could be excreted in the faeces of slaughter-age birds. However, although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

It is concluded that the risk that this disease could be introduced to New Zealand in imported BMFL turkey meat products is negligible.

### 7.2.2.8 Recommendations for risk management

No specific safeguards are required.

## References


(1) Pierson FW, Domermuth CH. Hemorrhagic enteritis, marble spleen disease and related infections. In: Calnek BW (ed) (1997) *Diseases of Poultry*. Tenth Edition. Pp 624-33. Iowa State University Press.

(2) McNulty MS, McFerran JB (1996). Adenoviruses. In : Jordan FTW, Pattison M (eds) *Poultry Diseases*. Fourth Edition. Pp 204-17. Saunders, London.

(3) McCracken RM, Adair BM (1993) Avian adenoviruses. In: McFerran J B, McNulty M S (eds) *Virus Infections of Birds*. Pp 123-44. Elsevier, Amsterdam.

(4) Silim A, Thorsen J (1981) *Haemorrhagic Enteritis: Virus Distribution and Sequential Development of Antibody in Turkeys*. Avian Diseases. 25: 444-453.

(5) McFerran J B, Adair B M (1977) *Avian Adenoviruses: A Review*. Avian Pathology. 6: 189-217.

(6) Howell J (1991) *Viral diseases and the New Zealand poultry industry*. Surveillance. 19(2): 15-17

### 7.2.3 AVIAN INFECTIOUS BRONCHITIS

This disease has been assessed in Part One of this document (see section 3.3.2).

Farmed pheasants are now considered to be a natural host for this disease, at least in the UK.(1)

However, as discussed in section 3.3.2, poultry meat products which do not contain organ tissues (such as kidney) are unlikely to harbour the IB virus.

It is considered that the risk of introduction of *IB virus* in BMFL turkey meat preparations is negligible.

No specific safeguards are required.

## Reference

(1) Alexander DJ, Central Veterinary Laboratory, Weybridge, UK. Personal communication with H Pharo, March 1999.

### 7.3.3 AVIAN POLYOMAVIRUS

Note : this disease was considered in this risk analysis at the request of the Department of Conservation.

This disease has been assessed in Part One of this document (see section 3.3.3).

A virus that morphologically resembled a polyomavirus was recovered from the intestinal contents of asymptomatic turkeys, but the recovered virus did not cause any disease in experimentally infected birds.(1)

It is considered that the risk of introduction of polyomavirus in BMFL turkey meat preparations is negligible.

No specific safeguards are required.

## Reference

(1) Ritchie B R (1995) *Avian Viruses: Function and Control*. Pp 136-70. Wingers Publishing, Florida, USA.

### 7.2.4 TURKEY RHINOTRACHEITIS{tc \l3 "7.2.4 TURKEY RHINOTRACHEITIS}

Avian rhinotracheitis has been assessed in Part One of this document (see section 3.3.4).

Turkey rhinotracheitis is widespread in the United Kingdom, and turkeys may be vaccinated with either inactivated or attenuated live vaccines.

It is considered that there is a negligible risk of introducing this disease in BMFL turkey meat preparations derived from clinically healthy birds.

No specific safeguards are required.

## 7.2.5 CORONAVIRAL ENTERITIS OF TURKEYS{tc \l3 "7.2.5 CORONAVIRAL ENTERITIS OF TURKEYS}

### 7.2.5.1 Aetiology

The disease is caused by a coronavirus.[1]

### 7.2.5.2 The disease

Coronaviral enteritis of turkeys (CET) is an acute highly infectious disease affecting turkeys of all ages characterised by inappetence, weight loss, diarrhoea and depression. A rapid drop in egg production is seen in infected breeder hens.[1, 2]

Under natural conditions, the disease spreads rapidly through a flock and between flocks on the same farm.[1] The incubation period varies from 1-5 days. Clinical signs usually develop within 48 hours and the clinical course often lasts for 10-14 days. Morbidity approaching 100% is typical, with weight loss depending on the degree that birds go off feed and water. Experimentally, mortality in young poults ranges from 50-100%, while in older birds (6-8 weeks), mortality may reach 5-50%. Birds that have recovered from the disease are resistant to subsequent challenge, but remain carriers for life.[2]

### 7.2.5.3 Physical and chemical stability

Coronaviruses in general are readily inactivated by ultraviolet light, disinfectants, heat,[3] lipid solvents, non-ionic detergents and oxidizing agents.[4] As with other enveloped viruses, chloroform treatment (10 minutes at 4 □C) inactivates the CET virus.[2]

Most strains of infectious bronchitis virus, another coronavirus of poultry, are inactivated at a temperature of 56 □C in 15 minutes,[5] but very little work has been carried out on the heat lability of the CET virus.

In 1969, at which time there were no *in vitro* methods for cultivating the CET virus, it was reported that filtrates prepared from suspensions of intestinal contents of infected turkey poults were rendered non-infective by exposure to pH 3.0 or by heating at 50 □C for 1 hour.[6] However, in 1974 after systems had been developed for growing the virus in embryonated turkey eggs, two of the authors of the 1969 report reported that the CET virus was, in fact, not inactivated either by pH 3.0 or by heating for 50 □C for 1 hour.[7] Despite this obvious contradiction, recent textbooks on poultry diseases[1, 2] quote the latter work in concluding that the CET virus is "moderately resistant to temperature", and since 1974 there have been no reports published on the effect of incubation of the virus at different temperatures for different times.

Freezing has little effect on the virus. Faeces of infected turkeys remain infectious for months under freezing environmental conditions, and the virus remains viable in intestinal tissues and contents for several years at -20 □C or below.[1]

### 7.2.5.4 Epidemiology

Turkeys are the only known natural host. Chickens, pheasants, seagulls and quail are refractory to infection.[2]

The CET virus spreads through the ingestion of water, feed and litter contaminated with infectious faeces.[1] However, as is the case with coronavirus infections of ruminants[8] and pigs,[9] the duration of shedding of infective virus in recovered turkeys is unclear. The virus has been shown to replicate only in enterocytes of the gut and in unidentified cells in the bursa of Fabricus. Electron microscope studies have shown that the virus can be found in the intestinal epithelium only 24 - 96 hours post-infection.[1] The disease signs are considered to be caused by changes in the cellular physiology of intestinal epithelium and consequent malabsorption of nutrients.[1] Nevertheless, it appears to be generally accepted[1, 2] that the virus may be shed in the droppings of recovered turkeys for several months, although there do not appear to be any published reports to substantiate this important point.

Infection is readily transmitted by intestinal material inoculated orally, and cell-free filtrates of the bursa were pathogenic for adult turkeys, but suspensions of heart, liver, spleen, kidney and pancreas from infected turkeys did not cause the disease when administered orally to 1-day-old poults.[2] This suggests that there is no viraemia and no distribution of the virus to tissues apart from the gut and the reticuloendothelial system.

There is no evidence that CET virus is egg-transmitted.[1]

### 7.2.5.5 Occurrence

Coronaviral enteritis has been reported in several states in the United States, Canada, and Australia, but not in turkey-raising areas of Europe.[1, 2] It has not been reported in New Zealand.

### 7.2.5.6 Effect of introduction

The virus has the potential to cause considerable economic losses, encompassing high mortality rates in young turkey poults, weight loss and reduced egg production in laying hens.[1, 2]

### 7.2.5.7 Risk of introduction in turkey meat

There appears to be no viraemia, and no distribution of the virus to tissues apart from the intestines, which are removed at slaughter, and the bursa, which is not used in the manufacture of BMFL turkey meat products.

Moreover, CET virus has never been reported in the United Kingdom or Europe.

The risk of introducing coronaviral enteritis in BMFL turkey meat preparations from the United Kingdom is considered to be negligible.

### 7.2.5.8 Recommendations for risk management

No specific safeguards are required.

## References


(1) Naqi SA (1993) Coronaviral enteritis of turkeys. In: McFerran J B, McNulty M S (eds) *Virus Infections of Birds*. Pp 277-81. Elsevier, Amsterdam.

(2) Nagaraja KV, Pomeroy BS (1997) Coronaviral enteritis of turkeys (bluecomb disease). In: Calnek BW (ed) *Diseases of Poultry*. Tenth Edition. Pp 686-92. Iowa State University Press.

(3) Porterfield JS (ed) (1989) *Andrewes' Viruses of Vertebrates*. Fifth Edition. P 43. Ballière Tindall, London.

(4) Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD. *Virus Taxonomy : classification and nomenclature of viruses.* P 407. Springer-Verlag, Wien, 1995.

(5) McMartin DA (1993) Infectious bronchitis. In: McFerran J B, McNulty M S (eds) *Virus Infections of Birds*. P 249. Elsevier, Amsterdam.

(6) Deshmukh DR, Larsen CT, Dutta SK, Pomeroy BS (1969) Characterization of pathogenic filtrates and viruses isolated from turkeys with bluecomb. *American Journal of Veterinary Research* 30: 1019-25

(7) Deshmukh DR, Pomeroy BS (1974) Physicochemical characterisation of a bluecomb coronavirus of tuekeys. *American Journal of Veterinary Research* 35: 1549-1552

(8) Mebus CA (1990) Neonatal calf diarrhoea virus. In: Dinter Z, Morein B (eds). *Virus Infections of Ruminants*. P 297. Elsevier, Amsterdam.

(9) Bohl EH (1989) Transmissible gastroenteritis virus (classical enteric variant). In: Pensaert MB (ed). *Virus Infections of Porcines*. P 142. Elsevier, Amsterdam.

### 7.2.6 HIGHLY PATHOGENIC AVIAN INFLUENZA (HPAI){tc \l3 "**7.2.6 HIGHLY PATHOGENIC AVIAN INFLUENZA (HPAI)}**

This disease has been assessed in Part One of this document (see section 3.3.6).

As turkey production in the UK has moved away from the old "pole barn" system, which allowed contact with small birds through open sides, towards fully indoor systems similar to chickens, HPAI has become less common.(1) HPAI last occurred in the United Kingdom in 1991.(2)

Because of the extra processing involved in their production, it is considered that the risk of introducing HPAI in BMFL turkey meat preparations is even less than the low risk identified for chicken meat products in section 3.3.6.7 of this risk analysis.

Nevertheless, in view of the potentially catastrophic impact of HPAI should it be introduced into this country, safeguards are considered to be justified.

#### 7.2.6.1 Recommendations for risk management

The following safeguards for turkey meat products are based on those for chicken meat, as detailed in the OIE International Animal Health Code (OIE Code):

> When importing turkey meat from HPAI free countries[25], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment of meat comes from birds which have been kept in an HPAI-free country since they were hatched or for at least the past 21 days.
>
> When importing turkey meat from countries or zones considered infected with HPAI[26], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Health Code] attesting that the entire consignment of meat comes from birds which have been kept in an establishment free[27] from HPAI and not situated in an HPAI *infected zone* [defined by the OIE Code].
>
> In addition, the turkey meat products must come from birds which have not been vaccinated for avian influenza.

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25 A country may be considered free from HPAI when it can be shown that HPAI has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against HPAI.

26 A HPAI infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

27 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

## References

(1) Alexander DJ, Central Veterinary Laboratory, Weybridge, UK. Personal communication with SC MacDiarmid, January 1999.

(2) Alexander DJ, Central Veterinary Laboratory, Weybridge, UK. Personal communication with H Pharo, March 1999.

### 7.2.7 INFECTIOUS BURSAL DISEASE{tc \l3 "7.2.7 INFECTIOUS BURSAL DISEASE}

This disease has been assessed in Part One of this document (see section 3.3.7).

#### 7.2.7.1 Aetiology

Infectious bursal disease (IBD) virus was first identified in 1962, and until 1978 only a single serotype was recognised. In that year a second serotype was identified in turkeys.[1]

Serotype 1 (IBD1) occurs predominantly in chickens.[1] It is rarely reported in turkeys.[2] Viral replication in the bursa of 3-6- week-old poults has been demonstrated experimentally for 5 days after IBD1 infection and seroconversion, and mild lymphocytic degeneration of bursae, spleens and caecal tonsils of infected poults has also been seen.[3] However, infection of turkeys with IBD1 does not cause clinical signs.[1]

Serotype 2 (IBD2) is widespread in turkeys[2] and chickens.[4, 5]

Both serotypes have also been reported in ducks.[1]

#### 7.2.7.2 IBD serotype 2 infection

Turkeys

Although IBD2 has not been shown to cause clinical disease in any avian species,[1, 6] it has been suggested that infection of poults at a young age may cause immunosuppression which might result in an increased susceptibility to respiratory disease.[6] However, there is little evidence to support this suggestion.[7]

In 1978 it was speculated that there could be an association between natural IBD infections in young turkeys and concurrent respiratory problems.[8] Turkeys in problem flocks were reported to have antibodies to IBD, but no virus was isolated. The reported postmortem picture was complex and dramatic: tracheitis, pulmonary oedema, swollen livers and spleens and a "drastic reduction" in bursal size. It was postulated that the bursal lesions might have been caused by IBD.

In 1980 it was reported that poults recovering from rhinotracheitis had high titres of antibodies to IBD virus.[9] Several IBD vaccines were applied to breeder turkeys (apparently both chicken and turkey strains of IBD virus used), and improved survivability of poults from vaccinated birds was reported. The authors were of the opinion that immunosuppression by the turkey IBD virus might predispose poults to other diseases such as adenoviruses, NDV, or *Alcaligenes faecalis*. However, the results reported for the four vaccinated flocks and 14 unvaccinated flocks (from four farms) do not support any inferences or conclusions regarding poult survivability.

Moreover, 2 years later a study found that infection of poults with IBD2 did not predispose to alcaligenes rhinotracheitis (*Alcaligenes faecalis*).[10] It was reported that no gross or histological

lesions were observed (apart from those due to alcaligenes rhinotracheitis) in any of the poults exposed to IBD virus.

The conclusion that IBD2 infection of turkeys causes neither disease nor immunosuppression has been supported by several further studies. In 1983 it was demonstrated that a chicken isolate of IBD caused immunosuppression in poults, but a turkey strain did not,[11] and a 1984 study found that two isolates of IBD2 had no effect on bursa weight in poults.[12]

However, a few reports have continued to suggest that IBD virus infection of turkeys may cause immunosuppression. Two studies reported mild transient suppression of cellular immunity in poults without causing any demonstrable disease.[13, 14] A single report has indicated that mild degeneration of the bursa, and suppression of both humoral and cellular immune responses may occur in poults infected with IBD2, but only in poults infected at 1 day of age.[15] However, the significance of any such mild and transient immunosuppression is unclear. Although it may persist for 3 weeks after the disappearance of the virus, this immunosuppression has not been correlated to any tissue or cellular changes.[14]

The significance of a report of haemorrhage in thigh muscle of poults at 3 and 7 days post-infection[14] is also unclear.

Chickens

IBD2 virus is widespread in chickens. Antibodies to IBD2 were found in 35 of 75 chicken flocks (47%) surveyed in Ohio,[4] and 39 out of 46 chicken flocks (85%) surveyed in England.[5]

Although there is still some debate as to whether infection of chickens with IBD2 can be expected to result in detectable tissue changes, no clinical disease caused by this serotype has been reported in chickens. A 1985 study reported lack of clinical signs, and neither gross nor microscopic lesions in chickens inoculated with IBD2.[16] Using the same strain of IBD2, a study carried out in 1986 found mild histological lesions in the bursa, spleen and Harderian gland following infection of 1-day-old SPF chickens.[17] However, a further study in 1988 reported that five isolates of IBD2 (including the strain used in the two studies mentioned above) were nonpathogenic in chickens.[18]

Ducks

Rising antibody titres to IBD2 have been reported during an outbreak of severe respiratory disease in 3-week-old ducklings on a large farm, and histological examination of bursae showed lesions similar to that produced by IBD1 virus in chickens.[1]

### 7.2.7.3 Effect of introduction

The effects of IBD1 introduction have been discussed in Part One of this document (see section 3.3.7.6).

New Zealand is free of IBD2.[19, 20]

IBD2 is non-pathogenic in chickens[1, 6] and there is no evidence that the mild and transient suppression of cellular immunity that has been demonstrated in turkey poults infected with this serotype at 1 day of age is of any significance.[7]

Although the susceptibility of other avian species to IBD2 infection has not been fully elucidated, it has not been shown to cause disease in any other birds. Moreover, a study in Nigeria, where IBD was endemic in free-range chickens, found no antibodies to IBD in guinea fowl, barbets, sunbirds and bulbuls, while only a small proportion of weavers and finches tested were seropositive without showing any clinical signs.[6] Therefore, it is highly improbable that IBD2 would result in disease in any avian species in New Zealand.

The Poultry Industry Association of New Zealand (PIANZ) has raised concerns that the introduction of IBD2 would interfere with IBD1 testing and eradication. The agar gel precipitation test (AGP) and the ELISA which are currently used in New Zealand do not differentiate between serotypes 1 and 2,[21] so that if IBD2 were present in this country, new tests would need to be implemented, and this would impose additional costs on the industry.[22] This position is consistent with what is presented in the OIE Manual of Standards.[23]

During the course of this risk analysis MAF has contacted a number of international experts in the field of IBD virology, in an attempt to evaluate the above concerns of PIANZ. These experts included the heads of three OIE reference laboratories in France,[24] Great Britain,[25] and the United States of America.[26] In addition, two researchers in the field of poultry virology in the USA were consulted.[27, 28] The result of this consultation was not clear cut. In general, the laboratories in Europe considered that the AGP and ELISA could be readily used to distinguish between the two IBD serotypes, whereas the American laboratories disagreed, and considered that the only way to do that reliably was by using the serum neutralisation test. The full details of this consultation are provided in Appendix 8.

#### 7.2.7.4 Risk of introduction

There are two potential mechanisms by which imported turkey products might be contaminated with IBD viruses : faecal contamination of carcasses during slaughter, and the presence of the virus in tissues that are used to make the turkey products. Although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

<u>IBD1</u>

Serological surveys of turkey flocks indicate that natural infection with IBD1 is rare in turkeys in the USA.[2] No turkey flocks in the United Kingdom have been found to be positive to IBD1.[5]

In the case of BMFL's own turkey flocks, extensive surveillance testing carried out for IBD1, involving 4,940 blood tests on 65 flocks over 28 months, did not yield any positive results.[29]

It is concluded that the risk of introduction of IBD1 in imported BMFL turkey meat preparations from the United Kingdom is negligible.

IBD2

A survey in the United Kingdom showed that 29 out of 32 (90%) of turkey flocks were seropositive to IBD2,[5] and another survey revealed that all of 42 turkey flocks tested were positive to IBD2.[30]

Considering the widespread infection of IBD2, most poults could be expected to be protected by maternal antibody, which in the case of chickens has a half life of 3-5 days.[1] The effect of maternal immunity is that infections with vaccinal or other strains of the virus are seldom possible prior to around 14 days of age.[6] A survey of turkey flocks in England found that antibodies were first detectable at 36 to 57 days of age.[24] Therefore, as neutralising antibodies in poults infected with IBD2 virus have been shown to be detectable 9-12 days after infection,[3, 12, 31] it can be concluded that infection of poults in England takes place from 4 to 7 weeks of age. However, there is no reason to suspect that poults could not become infected any time up to the age of slaughter.

In the absence of specific information on the shedding of IBD2 by infected poults, this analysis assumes that it is of similar duration as IBD1 in chickens. That is, shedding begins 1 day after infection and continues for 14-16 days.[6]

Thus, if infection of poults takes place at 4-7 weeks of age and shedding continues for up to 16 days, turkeys slaughtered at 8 weeks of age and older may be shedding IBD virus in faeces.

However, inspections of the BMFL manufacturing plant (Appendix 5) and a previous risk analysis (Appendix 6) support the conclusion that any risk of cross-contamination during processing is negligible. Although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface. Therefore, faecal contamination of carcasses resulting in IBD viruses being carried on carcass skin is not considered to present a significant risk.

IBD2 has been shown to persist in the tissues (bursa, spleen and thymus) of infected turkeys for at least 7 days post-infection.[14] In the absence of information to the contrary, this analysis assumes that the persistence of IBD2 in tissues of infected birds is similar to that of IBD1 in chickens. Section 3.3.7.7 of Part One of this document outlines available information on the tissue distribution and persistence of IBD1 virus in chickens muscle tissue.

In summary, IBD1 virus has been detected in chicken muscle tissue at 2, 3 and 4 days post-infection,[32] but not at 7 days post-infection.[33] Since neither of these experiments have looked for the virus on days 5 and 6 post-infection, it is reasonable to be cautious and to conclude that the virus may be present in chicken muscle from 2 - 6 days post-infection, and that it may be present in turkey muscle tissue for a similar time period.

There is a distinct risk, therefore, meat from turkeys slaughtered at 8 weeks of age and possibly 12 weeks of age could harbour IBD2 virus in their muscle tissue. It is assumed that IBD2 is similar to IBD1 in that it is not destroyed by freezing or thawing[34, 35] and is likely to survive cooking[36] of turkey meat.

The turkey meat preparations are manufactured only from muscle and skin (see Appendix 5). No viscera or bursal tissue, which are the sources of the highest titres of IBD2 virus, are included in the products. Because BMFL turkey meat preparations contain only edible tissues, the volume of scraps generated will be much less than that generated from whole chicken carcasses.

Because of the apparent high prevalence of IBD2 infection in turkeys in the UK it was considered prudent to conduct a quantitative assessment for that virus, given that scraps derived from these BMFL turkey meat preparations might find their way into the feed of backyard chickens and establish infection.

#### 7.2.7.5 Assessing the risk of IBD serotype 2 introduction

A Monte Carlo model was constructed to assess the risk of backyard poultry flocks becoming infected with IBD2 virus should BMFL turkey meat preparations be imported from the United Kingdom.

The model is presented in Appendix 4. It is similar in structure to the model developed to assess the risk of introduction of IBD1 in imported chicken meat, which is presented in Appendix 1.

The results of the model indicate that even if BMFL turkey meat preparations were to be imported into New Zealand at an annual volume equivalent to 50% of the current annual consumption of turkey meat, the risk of introducing IBD2 virus into backyard poultry would not be high. At that level of importation we can be 95% confident that there would be fewer than two disease introductions per hundred years.

As discussed in Section 7.2.7.3, IBD2 does not cause disease in any avian species. The effect of its introduction into this country would be limited to some degree of interference with serological testing for IBD1 in chickens.

MAF considers that this combination of low probability of introduction and limited consequence if introduced does not justify the imposition of safeguards against IBD2 introduction in BMFL turkey meat preparations.

Since there is good evidence that turkeys flocks in the UK in general, and BMFL turkeys in particular, are not infected with IBD1, in the case of BMFL turkey meat preparations no further safeguards against IBD viruses are justified.

#### 7.2.7.6 Recommendations for risk management

No specific safeguards are required.

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### 7.2.8 MAREK'S DISEASE{tc \l3 "7.2.8 MAREK'S DISEASE}

This disease has been assessed in Part One of this document (see section 3.3.8).

The risk of introduction of exotic strains of MD virus in imported BMFL turkey meat preparations is considered to be negligible.

No specific safeguards are required.

### 7.2.9 NEWCASTLE DISEASE, PARAMYXOVIRUS 2, PARAMYXOVIRUS 3, AND PARAMYXOVIRUS 7 {tc \l3 "7.2.9 NEWCASTLE DISEASE, PARAMYXOVIRUS 2, PARAMYXOVIRUS 3, AND PARAMYXOVIRUS 7 }

#### 7.2.9.1 Newcastle disease, paramyxovirus-2 and paramyxovirus-3

Newcastle disease has been assessed in Part One of this document (see section 3.3.9).

Avian paramyxovirus-2 (PMV-2)

PMV-2 is widespread in poultry flocks in many countries, more commonly in turkeys than in chickens. PMV-2 infections have been reported to be more severe in turkeys than in chickens, and there has been a report of severe respiratory disease, sinusitis, elevated mortality, and low egg production in turkey flocks infected with PMV-2 complicated by the presence of other organisms.(1)

Avian paramyxovirus-3 (PMV-3)

Natural infections of domestic poultry with PMV-3 have been restricted to turkeys. PMV-3 viruses can be divided into two antigenically distinct groups. Turkey isolates from the USA, Germany, France and Great Britain fall into one group, and those isolated from imported exotic psittacines fall into another group.(1)

Clinical signs in turkeys are usually egg production problems, occasionally preceded by mild respiratory disease. Reports have indicated that egg production usually declined rapidly and a high proportion of eggs that were layed were white-shelled, although hatchability and fertility were rarely affected.(1)

PMV-3 has been found in turkey flocks in North America and Europe. Besides turkeys, PMV-3 infection has been reported in caged passerines and psittacines in Europe and Asia. There have been no reports of isolation of PMV-3 from wild birds or of natural infections of chickens with this virus.(1, 2)

Safeguards are considered to be necessary.

#### 7.2.9.2 Avian paramyxovirus-7

In the United Kingdom, avian paramyxovirus-7 (PMV-7) has been isolated from collared doves,(3) but it has not been reported from turkeys.(4)

In the United States, PMV-7 was found to be the primary pathogen in natural outbreaks of respiratory disease with elevated mortality. The isolate caused mild respiratory disease in turkeys infected experimentally.(5)

Attempts to transmit PMV-7 by contact to chickens have been unsuccessful.(2)

Safeguards are considered to be necessary.

### 7.2.9.3 Recommendations for risk management

#### *7.2.9.3.1 Newcastle disease*

A major difficulty in the formulation of safeguards against ND is the lack of an internationally agreed definition of what consitutes "Newcastle disease." The definition of ND given in the International Animal Health Code[(6)] is as follows : "ND is a disease of birds caused by strains of avian paramyxovirus type 1, significantly more virulent than lentogenic strains." A more precise definition is given in the European Community Directive 92/66/EEC definition of ND for which control measures should be imposed : "an infection of poultry caused by an avian strain of the paramyxovirus 1 with an ICPI in day old chicks greater than 0.7." This definition includes all highly virulent (velogenic) and moderately virulent (mesogenic) viruses and may include some of the lentogenic vaccines licensed in the EU.[(7)] It is also based on infection of birds and not on the presence of disease signs or mortality.

MAF considers it appropriate that safeguards for PMV-1 should aim to prevent the introduction of any strains of the virus which are more pathogenic than the strains already in this country. That is, safeguards should prevent the introduction of any strains of PMV-1 virus with an ICPI greater than 0.0. The required safeguards are as follows:

> The turkey meat preparations should be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment comes from birds:[28]
>
> 1. which have not been vaccinated for PMV-1 ; and
>
> either
>
> 2. which, since hatching or for at least the past 21 days, have been kept in a country which is free[29] from infection with strains of PMV-1 with ICPI greater than 0.0;
>
> or

[28] If birds from infected flocks are likely to be slaughtered or processed in the same slaughter establishment, then birds destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

[29] A country may be considered free from PMV-1 (ICPI > 0.0) when it can be shown that PMV-1 (ICPI > 0.0) has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against PMV-1 (ICPI > 0.0).

3. which have been kept in an *establishment* [defined by the OIE Code] free from infection[30] with strains of PMV-1 with ICPI greater than 0.0 and not situated in a zone[31] which is infected with strains of PMV-1 with ICPI greater than 0.0.

*7.2.9.3.2 PMV-2, PMV-3, PMV-7*

The turkey flocks were kept in establishments that have remained free from evidence of PMV-2, PMV-3 and PMV-7 infection[i] for the 21 days prior to slaughter.

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30 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

31 A PMV-1 (ICPI > 0.0) infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

## 7.2.10 TURKEY VIRAL HEPATITIS{tc \l3 "7.2.10 TURKEY VIRAL HEPATITIS}

### 7.2.10.1 Aetiology

The aetiological agent of turkey viral hepatitis (TVH) has not been characterised but enterovirus-like particles have been seen in livers from experimentally infected turkeys. The agent is probably a picornavirus.[1]

Entero-like viruses have also been isolated from turkey poults with enteric problems. It is likely that enteric disease in young turkey poults is caused by a number of viruses, including entero-like viruses.[2]

### 7.2.10.2 The disease

TVH is an acute, highly contagious typically subclinical disease of turkeys that produces lesions of the liver and frequently the pancreas.[1, 2, 3] Outbreaks are usually seen in turkeys under 6 weeks of age. Depression, anorexia and increased mortality are the main signs.[2]

Infection is thought to result in disease only when other stressor agents are present.[2, 3] Morbidity is variable, but up to 100% has been reported by some flock owners.[1, 3] Mortality of up to 25% has been recorded but is usually very low. Mortality in birds over 6 weeks of age has not been reported.[2, 3]

Outbreaks of TVH are comparatively rare, the disease is only a minor problem and the importance of subclinical infection is unknown.[1]

Experimental oral inoculation of turkey poults with turkey entero-like viruses resulted in depression, ruffled feathers, watery droppings and reduced weight gains.[2]

### 7.2.10.3 Physical and chemical stability

The agent survives for 6 hours at 600C and 14 - 16 hours at 560C. It retains its viability for 1 hour at pH 2.0, but not at pH 12.[1, 3]

### 7.2.10.4 Epidemiology

Infection has been recognised only in turkeys. Ducks, chickens, pheasants and quail are refractory to infection.[1, 3] There is no evidence for infection of wild birds, other wildlife or humans. [1] Transmission occurs readily by direct or indirect contact by the faecal-oral route. It is not known whether egg transmission occurs or whether long-term carriers exist.[1, 2, 3]

The virus has been consistently isolated from the liver and faeces of infected birds and less frequently from bile, blood and kidneys during the first 28 days post-infection, after which the virus disappears.[1, 3] Lesions are found in the liver from 2-10 days post-infection.[1]

### 7.2.10.5 Occurrence

The disease has been recognised in the United States, the United Kingdom and Italy.[1, 2, 3] Entero-like viruses have also been isolated from turkey poults with enteric problems in the United States and France.[2] The disease has not been reported in New Zealand.

#### 7.2.10.6 Effect of introduction

In affected flocks, liver condemnation at slaughter varies between 30-90%.[1] There is some evidence that infected breeder flocks may exhibit decreased production, fertility and hatchability.

#### 7.2.10.7 Risk of introduction in turkey meat

Infections are usually seen in turkeys under 6 weeks of age and the virus is only sometimes found in tissues such as liver and kidney for up to 28 days post-infection.

There may be a risk of carcass contamination with the virus following the slaughter of birds shedding the virus in faeces. However, although faecal contamination during slaughter might result in limited contamination of the skin of an infected bird at slaughter, unlike bacteria of public health concern, viruses will not multiply on the carcass surface.

Turkeys slaughtered at above 10 weeks of age which pass post-mortem inspection would be unlikely to be carrying infection. Furthermore, the tissues in which infectivity is found are not used in the manufacture of BMFL turkey meat preparations.

It is concluded that although the risk of introduction of the agent of TVH in imported BMFL turkey meat preparations is probably low, until the aetiology of TVH is further clarified, it is considered reasonable that safeguards should be applied to minimise the risk of introduction of the agent.

#### 7.2.10.8 Recommendations for risk management

The turkey flocks from which birds are sourced for the manufacture of BMFL turkey meat products should have no history of unusually high liver condemnations at slaughter.

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### 7.2.11 SPIKING MORTALITY OF TURKEYS{tc \l3 "7.2.11 SPIKING MORTALITY OF TURKEYS}

Note : this disease was considered in this risk analysis at the request of the Poultry Industry Association of New Zealand.

#### 7.2.11.1 Aetiology

The precise aetiology of spiking mortality in turkeys (SMT) has not been clarified.[1] A variety of enteropathogenic viruses (including rotaviruses, astroviruses, enteroviruses and coronaviruses[2]) have been identified in affected flocks, but none has been found capable of reproducing the disease or has been consistently associated with the disease.[1] A coronavirus is presently considered to be the most likely initiating agent, but it appears that secondary agents are also involved, especially certain strains of *Escherichia coli*, *Salmonella* or *Campylobacter*.[1] Climatic and other stresses are also required for the clinical syndrome to become apparent.[1]

#### 7.2.11.2 The disease

SMT is the more severe of two clinical forms of poult enteritis-mortality syndrome (PEMS). Mortality in flocks with SMT is equal to or greater than 9% occurring between 7 and 28 days of age, including at least 3 consecutive days with mortality equal to or greater than 1%. Losses in excess of 50% have occurred. A less severe form of PEMS has been recognised, in which mortality exceeds 2% during the 7-28 day period, but does not equal or exceed 1% for 3 consecutive days. Mild or inapparent infections are thought to occur.[1]

Poults that recover from clinical disease remain permanently underweight; recovered male turkeys can weigh as much as 2-4 kg less than the breed standard when marketed at 20 weeks of age.[1]

#### 7.2.11.3 Epidemiology

Transmission of the disease is by the faecal-oral route. Transmission by blood or extra-intestinal tissues has not been possible. There do not appear to be long-term carriers; sentinels placed in a breeder flock that had been affected previously did not contract the disease.[1]

SMT is restricted to North Carolina, which is the main turkey farming area in the USA, producing some 60 million birds per year. The population density of turkeys is very high in that state; an area of 10 miles by 10 miles typically contains from 15 to 25 turkey farms, varying in size from 8,000 to 68,000 birds per farm. In very dense areas there may be 40 farms in such an area.[3] SMT has a definite seasonal pattern, occurring only when temperatures and humidity levels are high from late spring to early autumn. Within the area that SMT occurs, outbreaks are clustered in specific localities, apparently related to density of farms and birds.[1]

PEMS and SMT occur only in turkeys.[1]

#### 7.2.11.4 Occurrence

The clinical syndromes of SMT and PEMS are restricted to the southeastern USA.(1)

### 7.2.11.5 Effect of introduction

SMT is a multifactorial disease, involving one or more viruses and several bacteria. The disease occurs only in the southwestern USA, in specific climatic and production conditions and is triggered by stress.

The climatic conditions and density of turkey populations that occur in New Zealand suggests that the clinical syndrome would be unlikely to be seen in this country even if the various infectious agents were present.

### 7.2.11.6 Risk of introduction in turkey meat

SMT has not been reported outside the southwestern USA. Slaughter-age turkeys do not carry the infectious agent even if they experienced the clinical condition as poults.

The risk of introduction of SMT in BMFL turkey meat preparations from the United Kingdom is considered to be negligible.

### 7.2.11.7 Recommendations for risk management

No specific safeguards are required.

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# 8. SUMMARY OF RECOMMENDATIONS : BMFL TURKEY MEAT PREPARATIONS FROM THE UNITED KINGDOM{tc \l1 "8. SUMMARY OF RECOMMENDATIONS : BMFL TURKEY MEAT PREPARATIONS FROM THE UNITED KINGDOM}

To summarise, the following recommended safeguards are suggested for the importation of Bernard Matthews Foods Limited (BMFL) turkey meat preparations into New Zealand from the United Kingdom:

## 8.1 General recommendations

8.1.1 Turkey meat preparations to be derived from birds slaughtered in an abattoir approved by the competent authority.

8.1.2 Turkey meat preparations to be derived from birds which passed ante-mortem and post-mortem inspection procedures.

8.1.3 Turkey meat preparations to be certified as fit for human consumption.

8.1.4 Turkey meat preparations to be derived from broiler birds only, and giblets shall not be included

8.1.5 HACCP programs to be implemented at all points in slaughter and processing

## 8.2 Specific recommendations

### *8.2.1 Salmonella*

#### *8.2.1.1 Salmonella pullorum and Salmonella gallinarum*

1. Country freedom or a free zone. Vaccination is not practised.[32]

   or

2. Flock of origin freedom[i]

   A flock accreditation program involving both parent and broiler flocks, approved by MAF New Zealand. Vaccination is not permitted.

   or

[32] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*8.2.1.2 S. enteritidis* PT4 and *S. typhimurium* DT104

1. Country Freedom or a free zone[i]

   or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. typhimurium* DT104 and *S. enteritidis* PT4. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of *S. typhimurium* DT104 and *S. enteritidis* PT4 and that there are no opportunities for cross contamination during transport, slaughter and processing.

   or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

*8.2.1.3 Salmonella arizonae*.

1. Country freedom or a free zone.[33]

   or

2. A HACCP program approved by MAF New Zealand that ensures the final product is free of *S. arizonae*. The HACCP program must ensure that the breeding flock, hatchery and rearing farms of poultry destined for export to New Zealand are free of salmonellae and that there are no opportunities for cross contamination during transport, slaughter and processing.

   or

3. Heat treatment by cooking to ensure a minimum internal temperature of 79□C is reached. Cooking in a microwave oven is not permitted.

[33] If poultry from infected flocks are likely to be slaughtered or processed in the same establishment then poultry destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

### *8.2.2 Highly pathogenic avian influenza (HPAI)*

When importing turkey meat from HPAI free countries[34], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment of meat comes from birds which have been kept in an HPAI-free country since they were hatched or for at least the past 21 days.

When importing turkey meat from countries or zones considered infected with HPAI[35], the meat must be accompanied by an *international sanitary certificate* [defined by the OIE Health Code] attesting that the entire consignment of meat comes from birds which have been kept in an establishment free[36] from HPAI and not situated in an HPAI *infected zone* [defined by the OIE Code].

In addition, the turkey meat products must come from birds which have not been vaccinated for avian influenza.

### *8.2.3 Newcastle disease (ND) and other paramyxoviruses:*

#### *8.2.3.1 Newcastle disease*

The turkey meat preparations should be accompanied by an *international sanitary certificate* [defined by the OIE Code] attesting that the entire consignment comes from birds:[37]

1. which have not been vaccinated for PMV-1 ; and

   either

---

34 A country may be considered free from HPAI when it can be shown that HPAI has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against HPAI.

35 A HPAI infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

36 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

37 If birds from infected flocks are likely to be slaughtered or processed in the same slaughter establishment, then birds destined for export to New Zealand must be slaughtered and processed first of the day to effectively manage the risks associated with cross contamination.

2. which, since hatching or for at least the past 21 days, have been kept in a country which is free[38] from infection with strains of PMV-1 with ICPI greater than 0.0;

or

3. which have been kept in an *establishment* [defined by the OIE Code] free from infection[39] with strains of PMV-1 with ICPI greater than 0.0 and not situated in a zone[40] which is infected with strains of PMV-1 with ICPI greater than 0.0.

*8.2.3.2 PMV-2, PMV-3, PMV-7*

The turkey flocks were kept in establishments that have remained free from evidence of PMV-2, PMV-3 and PMV-7 infection[ii] for the 21 days prior to slaughter.

### *8.2.4 Turkey viral hepatitis*

The turkey flocks from which birds are sourced for the manufacture of BMFL turkey meat products should have no history of unusually high liver condemnations at slaughter.

## 9. ACKNOWLEDGMENTS{tc \l1 "9. ACKNOWLEDGMENTS}


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38 A country may be considered free from PMV-1 (ICPI > 0.0) when it can be shown that PMV-1 (ICPI > 0.0) has not been present for at least the past 3 years. This period shall be 6 months after the slaughter of the last affected animal for countries in which a *stamping out policy* [defined by the OIE Code] is practised with or without vaccination against PMV-1 (ICPI > 0.0).

39 Where it has been concluded that flock of origin freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed, as there are often many possible ways that this might be achieved. Specific details would be formulated according to the detailed proposals being considered at the time a particular trade is negotiated.

40 A PMV-1 (ICPI > 0.0) infected zone shall be considered as such until at least 21 days have elapsed after the confirmation of the last *case* [defined by the OIE Code] and the completion of a *stamping out policy* [defined by the OIE Code] and *disinfection* [defined by the OIE Code] procedures, or until 6 months have elapsed after the clinical recovery or death of the last affected animal if a *stamping out policy* was not practised.

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## Appendix 1: Quantitative assessment of the risk of introduction of IBD virus in imported chicken meat products and its establishment in backyard flocks

### *Introduction*

For IBD to become established in poultry flocks in New Zealand as a result of importing the virus in chicken meat products, a number of criteria would have to be met.

- Infected chicken meat products would have to be imported;
- These products would have to be fed to poultry;
- Infection would have to establish in the flock.

Initially, we considered it unlikely that commercial poultry in this country would be fed any imported chicken meat products. However, it appears that a small number of commercial free-range egg producer flocks are fed on table waste both from domestic and commercial sources.(1) Furthermore, the feeding of kitchen waste to backyard poultry flocks is a common practice. If such kitchen waste contained scraps of infected imported chicken meat, then it is possible that IBD infection could became established in backyard poultry or free-range egg producer flocks. If that were to occur, the risk of infection also becoming established in other commercial layer and broiler flocks would be increased significantly.

Focussing on backyard flocks, the above criteria may be refined to:

- Infected chicken meat products are imported;
- Imported infected chicken meat products are purchased for consumption in a household where backyard chickens are kept;
- Raw or cooked scraps of the imported chicken meat products are disposed of in kitchen scraps;
- Kitchen scraps containing infected chicken meat scraps are fed to backyard chickens;
- Infection may result in the backyard flock, if birds of the right age are present.

To examine the above scenario, a Monte Carlo model was constructed using the software packages Excel[41] and @Risk[42]. The structure of the model is shown in Figure 1.

### *Commodities Considered*

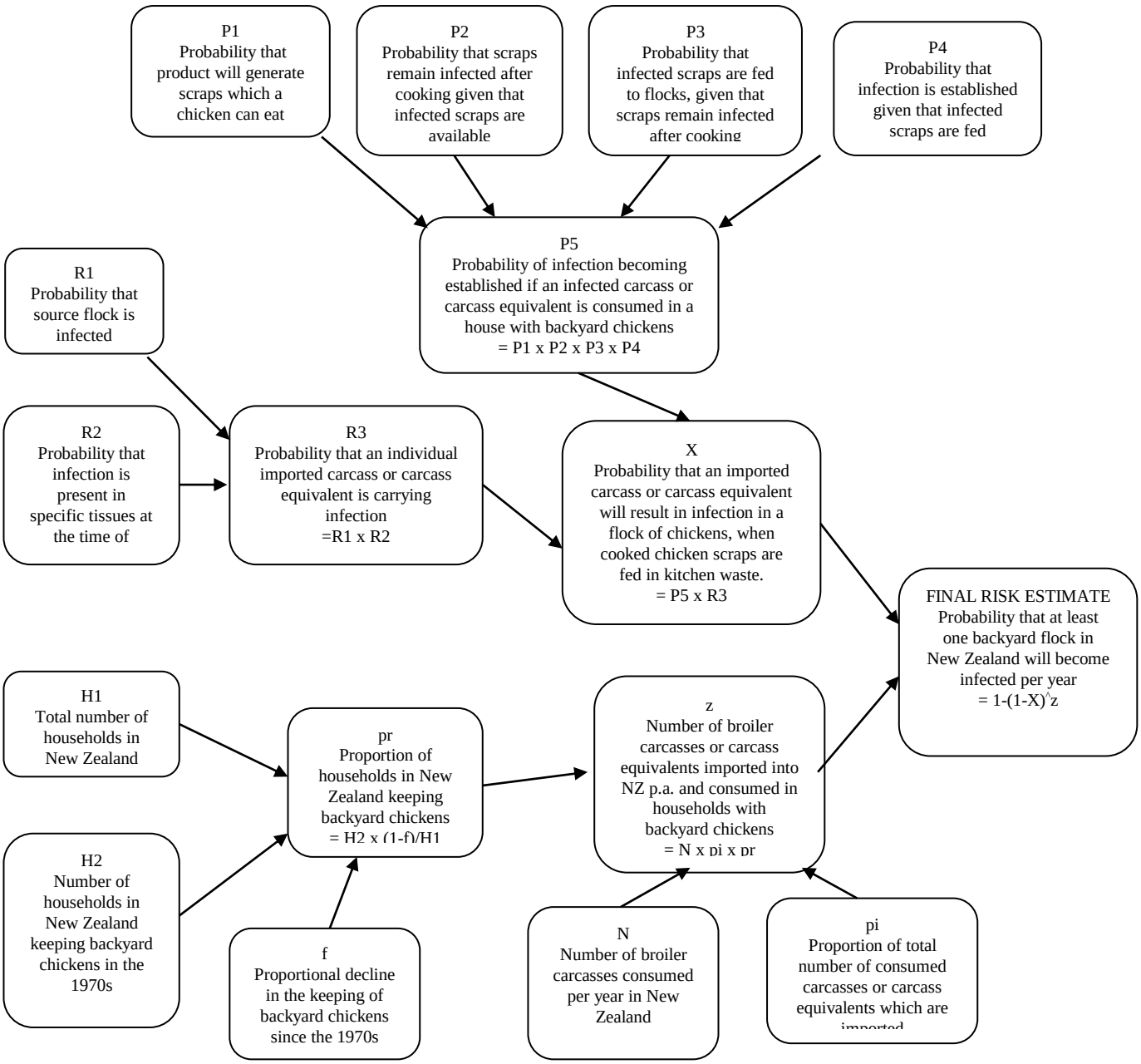
The model considered the following chicken meat products:

- Whole chicken carcasses (no giblets)
- Bone-in chicken cuts (wings, legs, drums)
- Boneless chicken cuts

[41] Microsoft Corporation, USA.

[42] Palisade Corporation, NY, USA

**Figure 1: Structure of model to assess the risk of introduction of IBD virus in imported chicken meat and its establishment in backyard poultry flocks**



### *Model Scenario and Method*

The model focusses primarily on the risk of backyard poultry flocks becoming infected with a virulent field strain of IBD virus should various chicken meat products be imported from either the United States or the European Union.

However, the assessment also applies to the risks from vaccinal strains of IBD virus, as the emergence of very virulent strains has meant that there is widespread use of "hot" and "intermediate" live vaccine strains which can cause significant bursal damage in immunologically naive chickens. Vaccinal strains were considered to constitute a risk despite the experience with IBD virus in New Zealand, which suggests that meat may not act as a vehicle for non-virulent or vaccinal strains. The Poultry Industry Association of New Zealand believes that the IBD virus strain present in some flocks does not appear to be transmitted in poultry meat. Following the identification of IBD in this country, the industry began a campaign to eradicate the disease. The industry has almost eradicated the infection without at any time restricting the distribution and sale of whole, raw chicken carcasses from infected flocks.

Backyard poultry flocks are a relatively heterogeneous group. Most are kept for egg laying and "lifestyle" purposes. These flocks consist mainly of hens, many of which have been purchased from cage layer flocks at the end of their first laying period. Such flocks often consist solely of adult birds. The layers in such flocks would seldom be handled by humans and would have very little contact with other similar flocks and as such there would be limited risk of transmission of diseases between such flocks. At the other extreme are the breeders of fancy poultry. Such flocks contain birds of multiple ages kept in close proximity to one another. They are often handled by their owners, are taken to shows where hundreds of birds are brought together, and traded between breeders. These characteristics make the fancy poultry sector potentially more important for the transmission of introduced pathogens than the backyard layer sector.[(2)]

The magnitude of the risk posed by the importation of chicken meat products obviously depends on the quantity imported; for each unit imported there is a risk of disease introduction, and the annual risk is determined by the number of units imported. This is a binomial process, which is reflected in the structure of the model.

The unit of importation considered in the model is a chicken carcass, or in the case of bone-in or boneless cuts, a chicken carcass equivalent.

To evaluate the effect of volume of trade, a range of imported volumes for each commodity was considered in the model.

### *Release Assessment : probability of infection in imported chicken meat products*

The release assessment estimates the probability that an imported chicken carcass or carcass

equivalent will be infected with IBD virus.

This probability is shown in Figure 1 as *R3*, and is a function of;

- Probability that the source flock is infected, *R1*;
- Probability that infection is present in specific tissues of the birds at the age of slaughter, *R2*.

The variables used in the Monte Carlo simulation model were as follows;

*R1* Probability that the source flock is infected.
Figures are those estimated for Europe and are based on published reports.[3, 4] The figures for the United States are similar.[5]

| | |
|---|---|
| Minimum | 0.3 |
| Most likely | 0.7 |
| Maximum | 0.9 |

The distribution used to model this is PERT (0.3, 0.7, 0.9)

*R2* Probability of active infection when slaughtered.
In estimating this probability, the following need to be considered:

- The age of chickens at slaughter; around 5-7 weeks of age; most likely at 37 days of age but as young as 32 days and as old as 49 days,
- The age at which chickens become infected; any time between 1 and 49 days of age,
- Duration of tissue infectivity. A study commissioned in 1997 by the Chief Veterinary Officer of Australia showed that IBD virus (strain CS88) was recoverable from a range of tissues, including muscle, bone marrow, bursa and liver/kidney of infected chickens[6]. This study, together with further experimental work commissioned by MAF,[7] leads to the conclusion that while IBD virus is recoverable from muscle tissue for only 2 - 6 days post-infection, it is recoverable from organs for 1 - 28 days post-infection.

The probability of active infection in different tissues at the time of slaughter is modelled by the following beta distributions:

∃ muscle: Beta (1669, 18332)

∃ organs: Beta (6004, 13998)

(See *Note I* for method of calculation and explanation.)

*R3* The probability that an imported carcass is infected,
*R3= R1 x R2*

***Exposure Assessment : Probability of imported chicken meat products causing infections in poultry flocks***

The fact that an imported commodity may contain an infective agent does not mean that the agent will necessarily come into contact with a susceptible host in New Zealand. The exposure assessment estimates the probability that, given the importation of chicken meat products which are infected with IBD virus, infection will be able to establish in poultry flocks.

The model is based on the assumption that for IBD to become established in New Zealand poultry flocks as a result of importing infected chicken meat products, scraps of these infected imported chicken meat products would have to be fed to poultry flocks in this country.

This probability is shown in Figure 1 as *P5*, and is a function of;

- Probability that the chicken meat products will generate scraps which a chicken can eat, *P1;*
- Probability that scraps remain infected after cooking, given that infected scraps are available, *P2;*
- Probability that infected scraps are fed to flocks, given that scraps remain infected after cooking, *P3;*
- Probability that infection is established given that infected scraps are fed, *P4*.

The simulation model used the following data for these variables;

*P1* Probability that the chicken meat products will generate scraps which a chicken can eat. (Skin, meat clinging to bone, organ scraps; see *Note II* for details.)

It was considered that whole carcasses will always generate some edible scraps, and therefore the value used in the model was :

*P1* = 1

While it is recognised that some edible scraps will be generated from bone-in cuts, it is considered that the overwhelming proportion (at least 98%) of waste will be in a form that is inedible for chickens. Therefore the distribution for this probability used in the model for bone-in cuts was :

*P1* = Uniform (0.001, 0.02)

It was considered that there would be an even lower probability of generating edible scraps from boneless cuts; not greater than 1%. The distribution for this probability used in the model for boneless cuts was :

*P1* = Uniform (0, 0.01)

*P2* Probability that infected scraps remain infected after cooking. (See *Note III* for data on which these estimates are based.)

| | |
|---|---|
| Minimum | 0.5 |
| Most likely | 0.8 |
| Maximum | 1 |

The distribution for *P2* used in the model is PERT (0.5, 0.8, 1.0)

*P3* Probability that infected scraps of imported chicken meat products are fed to backyard flocks given that scraps remain infected after cooking. (This is a guess, but it is likely that all or most kitchen scraps are fed to the chickens in those households which keep backyard flocks. Indeed, kitchen scraps from more than one household may be fed to a single backyard flock. Large volumes of table scraps may be fed to poultry flocks kept by institutions such as prisons and boarding schools.)

| | |
|---|---|
| Minimum | 0.1 |
| Most likely | 0.9 |
| Maximum | 1.0 |

The distribution for *P3* used in the model is PERT (0.1, 0.9, 1.0)

*P4* Probability that infection is established in a backyard flock that is fed infected scraps. These estimates are based on the widespread distribution of IBD virus in the tissues comprising a carcass,[6] the titres of virus reported in *Note III*, and what is known about the age structure of backyard poultry flocks.[7]

It is guessed that 60% of backyard poultry flocks are comprised of old layer hens which would not be susceptible to IBD infection, and 10% of flocks would be layers established from point of lay pullets, which would also not be susceptible. That leaves approximately 30% of backyard flocks where there are birds of mixed age which would include susceptible age groups. Therefore the following estimates for this variable were used in the model:

| | |
|---|---|
| Minimum | 0.25 |
| Most likely | 0.5 |
| Maximum | 0.75 |

The distribution for *P4* used in the model is PERT (0.25, 0.5, 0.75)

*P5* Probability of infection establishing if infected chicken meat products are consumed in a household which keeps backyard poultry.

*P5= P1 x P2 x P3 x P4*

The variables *R3* and *P5* are combined as follows :

*X* Probability that imported infected chicken meat products will result in IBD infection in a flock of backyard chickens, given that kitchen waste containing scraps of chicken meat products is fed to backyard chickens. This probability can be thought of as the risk per carcass or carcass equivalent imported.

As shown in Figure 1, this is the product of the probability that an imported carcass is infected, *R3*, and the probability of infection establishing if infected chicken meat products are consumed in a household which keeps backyard poultry, *P5*.

$X = P5 \times R3$

***Final Risk Estimate***

Given the estimate *X*, the annual risk of disease introduction and establishment in backyard poultry flocks in New Zealand depends on how many carcasses (or carcass equivalents) are imported per year and consumed in households where backyard poultry flocks are kept, *z*.

This is a function of :

- The number of broiler carcasses consumed per year in New Zealand, *N*;
- The proportion of broiler consumption which would be likely to consist of imported birds, or in other words the market penetration, *pi*;
- The proportion of households in this country which keep backyard poultry, *pr*.

The simulation model used the following data to estimate number of broiler carcasses likely to be imported per year :

*N* Broilers consumed each year in New Zealand[8],
$N = 6.30 \times 10^7$

*pi* Market penetration, the proportion of consumed carcasses which are imported.

It is not possible to predict with any confidence what volume of imported chicken meat products might be consumed in New Zealand if importation were to be permitted.

For example, it is known that currently there are more than 63 million broilers consumed per year in New Zealand, and assuming that importation of chicken meat products would not result in a change in total consumption of poultry meat in this country, even if only 1% of the local broiler market were captured, 630,000 carcasses would be imported in a year.

To model the effects of different assumptions regarding market penetration the Monte Carlo model carried out three simulations for each commodity. The values used for market penetration in these simulations are shown in Table 1.

Table 1: Values used in the model for market penetration by different commodities.

| Commodity | Market Penetration Estimates | | |
|---|---|---|---|
| | Low | Medium | High |
| Whole carcasses | 1% | 10% | 20% |
| Bone-in cuts | 0.1% | 1% | 10% |
| Boneless cuts | 0.1% | 1% | 10% |

Assuming that imported chickens would just as likely be consumed in households that keep backyard chickens as in households which do not, i.e. consumption is uniform, the probability that an imported chicken would be consumed in a household where backyard poultry are kept equals the proportion of New Zealand households which currently keep backyard poultry, *pr*, which is a function of :

- The total number of households in New Zealand, *H1*.
- The number of households which were known to keep backyard poultry when last surveyed, *H2*.
- The proportional decline in the keeping of backyard chickens since the 1970s, *f*.

*H1* Number of households in New Zealand[9],
*H1* = $1.21 \times 10^6$

*H2* Last figure for households keeping backyard poultry flocks, 1970s[10],
*H2* = $7.00 \times 10^4$

*f* Proportional reduction in the practice of backyard poultry keeping since the '70s.
No information is available on this matter, so it is considered a reasonable guess that the number of households which keep backyard poultry flocks today is between 40% and 60% of the number of households which kept them in the 1970s.
*f* = Uniform (0.4, 0.6)

*pr* Proportion of households currently keeping backyard poultry.;
*pr* = [H2 x (1-*f* )] / H1

Therefore,

*z* Number of carcasses or carcass equivalents imported into New Zealand per year and consumed in households which keep backyard poultry;

$z = N \times pi \times pr$

***Final Risk Estimate***

The probability of ***no*** disease introduction per year can be calculated as :

$(1-X)^z$

and the probability that ***at least one*** backyard flock becomes infected per year is :

$1-(1-X)^z$

***Risk Assessment Results***

The key result of interest is the probability that ***at least one*** backyard flock would become infected per year, $1-(1-X)^z$.

The model was run for three different commodities; whole chickens, chicken meat cuts containing bone but no organs, and boneless cuts. For each of these commodities, three levels of market penetration were modelled.

For each of the above scenarios 10,000 iterations of the model were run. This allows the results to be reported in terms of the percent of iterations that had a result above or below a certain value. The most common way to report the result is in terms of the $95^{th}$ percentile of iterations. In other words, in 95% of iterations the result was less than the quoted figure.

The $95^{th}$ percentile results for final risk estimate of the probability that ***at least one*** backyard flock would become infected per year, $1-(1-X)^z$ , are shown in Table 2.

Table 2: Summary of model results

| Commodity | Market Penetration | Mean Result | 95th Percentile Result |
|---|---|---|---|
| Whole chicken carcasses | 1% | 1 | 1 |
| | 10% | 1 | 1 |
| | 20% | 1 | 1 |
| Bone-in cuts | 0.1% | 0.26 | 0.52 |
| | 1% | 0.85 | 0.99 |
| | 10% | 0.99 | 1 |
| Boneless cuts | 0.1% | 0.13 | 0.31 |
| | 1% | 0.68 | 0.97 |
| | 10% | 0.96 | 1 |

***Sensitivity Analysis***

The sensitivity analysis for chicken carcasses showed that the market penetration, *pi*, was the highest ranked variable in terms of the effect on the outcome. However, the results in Table 2 show that changing *pi* from 1% to 10% and 20% did not appreciably affect the final result, as even at the lowest level, the probability of at least one introduction was 1.

The sensitivity analysis for cuts of chicken meat (both bone-in and boneless) showed that the three most important variables affecting the outcome were related to the exposure assessment:

- *P1*, probability that the commodity will generate scraps which can be eaten by a chicken
- *P3*, probability that scraps are fed to poultry given that the household has a backyard flock
- *P4*, probability that feeding scraps will result in infection becoming established.

The assumptions made in assigning distributions to these three variables are clearly explained in the in the previous discussion on the exposure assessment. It is considered that no unreasonable assumptions have been made.

***Conclusion***

Under the assumptions used, if broiler chickens from the the European Union and/or the United States were to be imported into New Zealand, even in relatively small volumes, the risk of introducing a virulent field strain, or a "hot" or "intermediate" vaccine strain, of IBD virus into backyard poultry would be high. Indeed, the probability of IBD introduction and establishment approaches 1 if as few as 0.1% of the chicken carcasses or carcass equivalents consumed in New Zealand were to be imported.

*Key assumptions*

- The prevalence of IBD infection in infected flocks, including "hot" or "intermediate" vaccine strains, is likely to be close to 100%.
- The use of live vaccine, either "intermediate" or hot "strains", is very common in European and American broiler industries.
- Households which keep backyard chickens are just as likely to purchase chicken meat products as other households.
- Imported chicken meat products would be distributed uniformly over New Zealand.

*Caveats*

. The risk assessment was based on data from experiments using highly pathogenic strains of IBD virus.[6, 11] Therefore, some caution is necessary when considering the risk of introduction of IBD virus strains of low or medium pathogenicity, including vaccine strains. Experience in New Zealand suggests that the unrestricted sale of broilers from flocks infected with a strain of low pathogenicity may not result in spread of the virus.

. While the prevalence of IBD infection in European and United States flocks may be relatively high, it is unlikely that *highly-pathogenic strains* are prevalent at the rates used as inputs into the model.

*Note I: Probability that tissues of chickens will be carrying infection at slaughter, R2*

The probability that, at the time of slaughter, different tissues of a chicken from an infected flock will be carrying virus was modelled from the following data :

- Chickens are slaughtered at around 5-7 weeks of age; most likely at 37 days of age but as young as 32 days and as old as 49 days,
- Chickens become infected between 1 and 49 days of age,
- Virus is recoverable from muscle tissue for 2 - 6 days post-infection and from organs for 1 - 28 days post-infection.[6, 7]

A Monte Carlo model was constructed on the following assumptions for chicken meat products containing chicken meat only or also containing organs such as kidney, liver, fragments of bursa of Fabricius etc;

| | Chicken muscle | Chicken organs |
|---|---|---|
| A1, age of chicken at slaughter, in days | PERT (32, 37, 49) | PERT (32, 37, 49) |
| A2, age of chicken at first infection, in days | Uniform (1, 49) | Uniform (1, 49) |
| D, duration of tissue infectivity, in days | Uniform (2, 6) | Uniform (1, 28) |

At each of 20,000 iterations the model asked the question "Is the tissue infected at time of slaughter?" It used the algorithm;

*If A1 is greater than A2, use (if A1 is less than A2+D, use 1, else use 0), else use 0.*

An answer of 1 meant that the chicken meat product was infected, an answer of 0 meant that the product was not infected. That is, an answer of 1 was returned on each occasion when the time of slaughter was ***after*** the tissue became infected but ***before*** virus was eliminated.

The mean output of the model provided the probability that the chicken tissue concerned was infected at the time of slaughter. Since the simulation is an approximation only, the confidence interval for the true probability was calculated using;

Beta(k x mean+1, k x (1-mean)+1)

where k is the number of iterations (20,000) and mean is the mean output of the model.

The model output for the two tissues, and the resulting beta distributions used for modelling R2 in the main model were :

| | Chicken muscle | Chicken organs |
|---|---|---|
| Number of iterations, k | 20000 | 20000 |
| Mean output of the sub-model | 0.08344 | 0.30015 |
| Beta(k x mean+1, k x (1-mean)+1) | Beta (1669, 18332) | Beta (6004, 13998) |

*Note II: Composition of chicken meat products*

The following data on the composition of a broiler chicken carcass are based on figures provided by Mr R Diprose, Poultry Industry Association of New Zealand, 9 December 1997.

| Tissue | Weight in grams | Percentage |
|---|---|---|
| Carcass + giblets | 1604 | 100% |
| Muscle, meat and skin | 840 | 52.4% |
| Bone | 630 | 39.3% |
| Separable fat | 30 | 1.9% |
| Heart, liver, neck, gizzard | 104 | 6.4% |

Mr Diprose also provided the following estimates for percentage of tissues in different portions of raw chicken;

| | Lean | Skin | Separable fat | Bone |
|---|---|---|---|---|
| Breasts | 63% | 11% | 4% | 22% |
| Wings | 41% | 23% | 3% | 31% |
| Thighs | 47% | 16% | 11% | 24% |
| Drumsticks | 57% | 10% | 2% | 30% |

Although intestines and reproductive tissues are removed during slaughter, MAF has been advised by Dr L With that in all birds at least some fragments of the bursa of Fabricius will remain after processing, and that in 10-30% of birds the whole of the bursa may be left in the carcass.[(12)]

This, information, together with subsequent relevant information provided by Dr With,[(13)] is shown in the following table :

*Likelihood of organ tissues being present in chicken carcasses after processing*

| Tissue | Proportion of processed chicken carcasses in which some tissue is present |
|---|---|
| Kidney | 100% |
| Bursa (remnants) | 100% |
| Bursa (intact) | 10-30% |
| Lung | 10% |
| Trachea | 0.2% |
| Liver | 0% |
| Spleen | 0% |

*Note III: Probability that IBD virus will survive cooking, P2*

In 1991, MAF completed a review of the risks to animal health of importing meat and meat products.[14] The review concluded that for poultry meat to be considered safe as far as IBD was concerned, it was necessary to cook the meat for 50 minutes at 70□C, or 9 minutes at 80□C or 1 minute at 100□C.

In 1997 further research into the dissemination of IBD virus through the tissues of chickens and the heat inactivation of the virus was carried out by the Central Veterinary Laboratory, United Kingdom, on behalf of the Australian Chief Veterinary Officer.[6, 11]

The dissemination study demonstrated that IBD virus CS88 was present in muscle, bone marrow, bursa, liver/kidney, blood, spleen and faeces of infected chickens.[6]

The heat inactivation study[11] demonstrated that IBD virus in tissue homogenates survived high temperatures for an unexpectedly long time. For example;

| Temperature | Time (minutes) | Titre ($CID_{50}$) |
|---|---|---|
| 800C | 90 | $<10^{0.83}$ |
| 800C | 30 | $<10^{2.17}$ |
| 800C | 15 | $10^{2.68}$ |
| 800C | 5 | $10^{4.16}$ |
| 740C | 90 | $10^{0.5}$ |
| 740C | 30 | $10^{2.63}$ |
| 740C | 15 | $10^{3.68}$ |
| 740C | 5 | $10^{4.17}$ |
| 700C | 210 | $10^{2.3}$ |
| 700C | 240 | $10^{2.17}$ |
| 700C | 270 | $10^{2.17}$ |
| 700C | 300 | $10^{1.3}$ |
| 700C | 300 | $10^{1.38}$ |

The report on the study of the heat inactivation of IBD virus in tissue homogenates,[11] states that "The virus was unexpectedly resistant to prolonged heating at high temperatures. A previous experiment[15] demonstrated that IBDV was inactivated by heating at 700C for 60 minutes, 750C for 45 minutes and 800C for 10 minutes."

The report continues :[11]

“The earlier work was undertaken on a clarified aqueous suspension of the virus, while this study used an unclarified suspension of infected tissues. After 60 minutes at 700C and 15 minutes at 800C the particulate matter in the suspension seemed to become coagulated, which may have protected the virus to at least some extent. Moreover, the titre of virus in the homogenate used in this study was more than 1 x $10^{2.2}$ higher than in the previous. Also, that experiment was conducted using the 52/70 strain of virus which has a lower virulence than the CS88 strain used in this study.”

These time/temperature parameters need to be related to the sort of cooking times that imported poultry is likely to be subjected to. It is unlikely that domestic cooking will subject chicken to temperatures sufficiently high, for sufficiently long enough, to inactivate IBD virus.

Kentucky Fried Chicken (KFC), the major fast food outlet for cooked chicken, cooks its chicken so as to ensure that the temperature at the bone reaches 850C.[16] Two cooking methods are used :

. Pan frying at 1600C for 12.5 minutes, the largest piece being 180 g, including bone.

. Pressure cooking at 1710C for 14 minutes.

The holding cabinet temperature is 820C and the minimum temperature of chicken as it goes over the counter is 600C.[16]

There is some variation in recommendations made by various food authorities for cooking poultry, for example the United States Department of Agriculture Food Safety & Inspection Service recommends that poultry breasts and roasts be cooked to an internal temperature of 77□C and whole chickens, thighs or wings be cooked to an internal temperature of 82□C;[17] Health Canada recommends an internal temperature of 85□C;[18] and Australian authorities recommend cooking to at least 75□C[19].

Given that the 1997 United Kingdom study[11] showed that chicken which had been heated to 800C for 15 minutes still contained IBD virus at a titre of $10^{2.68}$ $CID_{50}$/g, that is 478 chick infectious dose 50% per gram, there is a very high probability that IBD virus would survive at infectious titres in domestically cooked chicken, especially in deep tissues.

It must also be kept in mind that at least some chicken scraps will be thrown away raw.

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**Appendix 2: Quantitative risk analysis of cooked chicken meat products with specific reference to recent studies on the infectious dose and heat inactivation of IBD virus.**

## 1. Heat inactivation studies

An experiment[1] undertaken in 1997 on behalf of the Commonwealth of Australia to determine the heat inactivation characteristics of infectious bursal disease (IBD) virus in homogenised chicken meat found that the strain of IBD virus used (IBD 1, strain CS88) was considerably more heat resistant than anticipated from previous experimental work undertaken in 1988 with IBD 1, strain 52/70.[2] The results are presented in Table 1. An Australian Scientific Working Group[3] considered that the most likely explanation for the difference was the different nature of the suspension media. In the 1988 experiment the medium used was a clarified aqueous bursal suspension whilst in 1997 an unclarified suspension of homogenised bursa, muscle, skin and fat was used. It was observed in the 1997 experiment that the particulate matter in the unclarified medium coagulated upon heating and it was postulated that this may have afforded some protection to the virus. Other important differences between these experiments included the initial viral titres and the virulence of the strains. Whilst strain CS88 is much more virulent than 52/70, comparative heat stability data is lacking. The Australian Scientific Working Group[3] concluded that the suspension medium used in the 1997 experiment is more typical of chicken meat products.

In the discussion of the 1997 experiment[1] it was suggested that it is useful to determine the D values (the time taken to reduce infectivity by 1 $\log_{10}$) at each temperature. A D value allows the calculation of the time needed at a particular temperature to reduce the probability of remaining infectivity to a given level, provided a starting titre is known or assumed. Where heat inactivation curves are bi-phasic, as some in this experiment were, the D value can be calculated from the second (shallowest) part of the curve. This was done by plotting a best fitting straight line by eye.

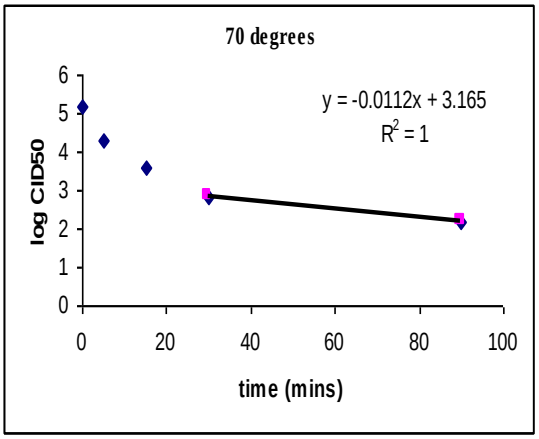
**Table 1:** Experimental data on the heat inactivation of IBD.[1,2] Titres of two strains of IBD 1 (52/70 and CS88) are expressed as $\log_{10}$ mean chicken infectious doses per 0.1 ml of inoculum ($CID_{50}$/0.1ml).

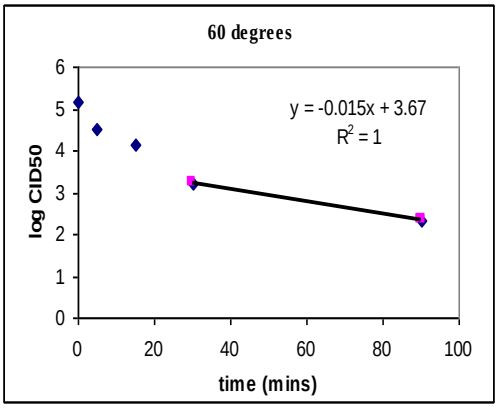
| **Time (min)** | **60 □C** CS88 (1997) | **70 □C** 52/70 (1988) | **70 □C** CS88 (1997) | **74 □C** CS88 (1997) | **80 □C** 52/70 (1988) | **80 □C** CS88 (1997) |
|---|---|---|---|---|---|---|
| 0 | 5.17 | 3.68 | 5.17 | 5.17 | 3.5 | 5.17 |
| 0.5 | | | | | 2.81 | |
| 1 | | | | | 1.67 | |
| 2 | | | | | 1.38 | |
| 5 | 4.5 | 1.37 | 4.32 | 4.17 | 0.38 | 4.16 |
| 10 | | 1.17 | | | | |
| 15 | 4.16 | | 3.57 | 3.68 | | 2.68 |
| 20 | | 0.38 | | | | |
| 30 | 3.22 | | 2.83 | 2.63 | | 2.17 |
| 60 | | 0.083 | | | | |

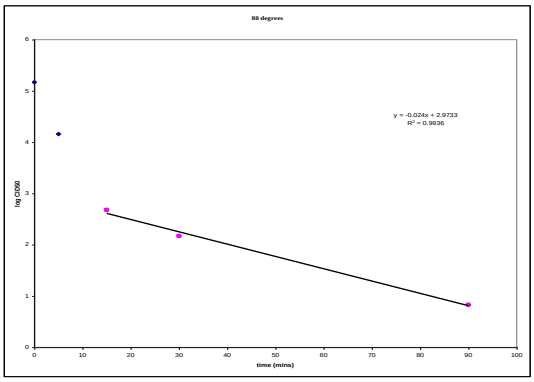
| | | | | | |
|---|---|---|---|---|---|
| 90 | 2.32 | | 2.16 | 0.5 | 0.83 |

There are a number of options available to plot a best fitting line. For example, by eye, as was done in the 1997 experiment[1], using a function-free model, or standard techniques such as linear regression. With any of these techniques the data may be transformed to enhance the "fit", e.g. by taking the square root, plotting on a log scale etc. Table 1 shows that the number of data points for each trial are limited so fitting lines must be done with caution. Two approaches were explored in an attempt to predict the time required to reduce infectivity. The first was based on the 1997 study's[1] approach by fitting a trend line using linear regression on the untransformed data from the shallowest part of the curve (Figure 1). The D value, referred to above, can be directly calculated as it is equal to 1 divided by the slope (see Table 2 for the results). The time can then be directly estimated by simply multiplying the D value by the chosen target, for example 6D. The second approach involves a square root transformation of time to enhance the fit of all data points to a straight line (Figure 2) .

**Figure 1** Linear regression on the untransformed data from the shallowest part of the curve.



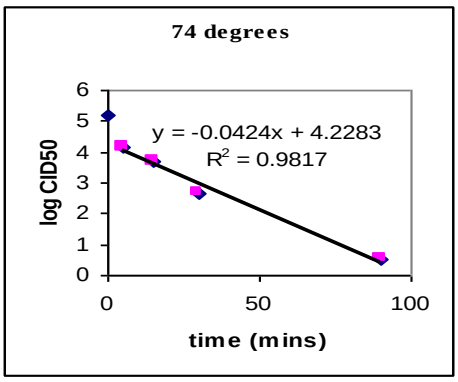
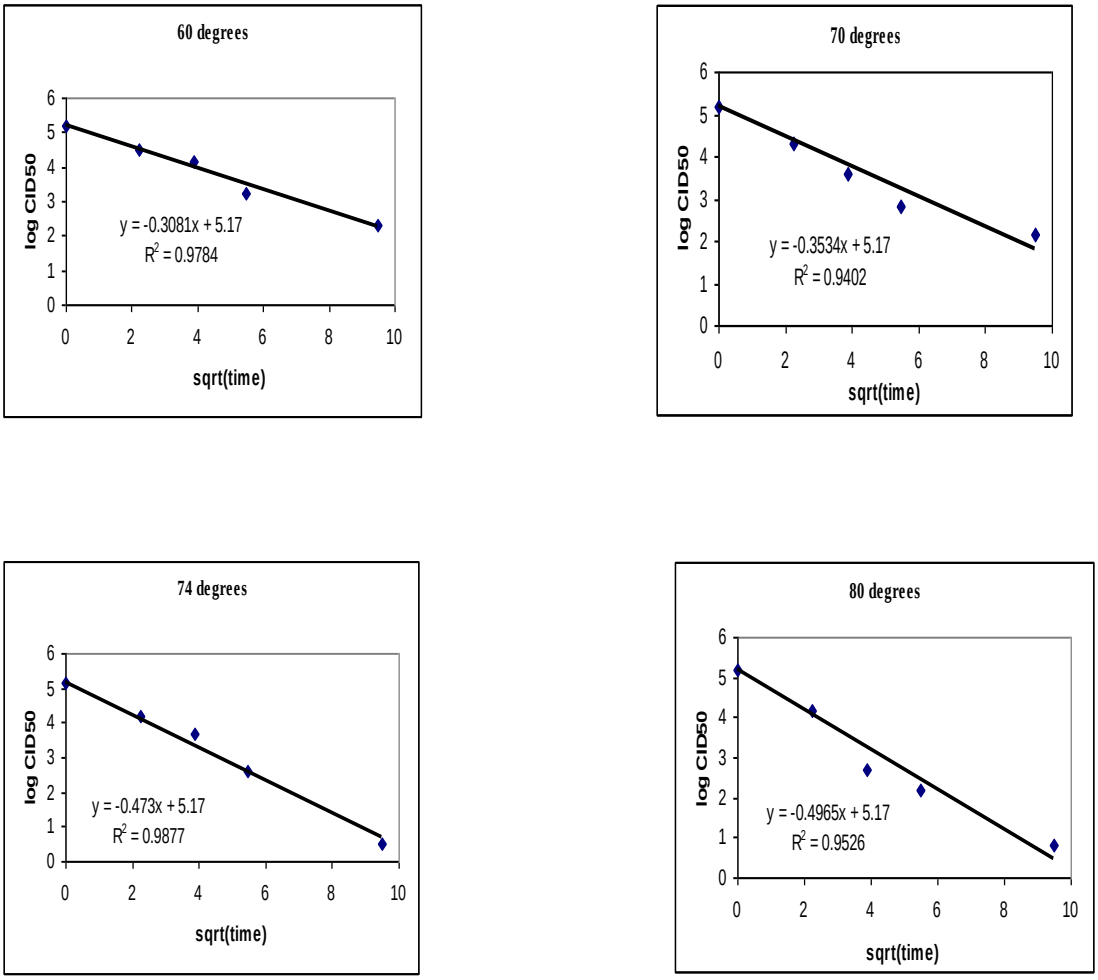


**Figure 2:** Linear regression on transformed data (square root of time).




There appear to be two significant constraints in the 1997 experiment[1] that result in a limited number of data points for each trial. The duration of each heat treatment was not long enough, perhaps because of a much greater thermostability encountered in this experiment than anticipated from earlier studies, and the small number of chickens used for each titration to determine the infectivity remaining after treatment. As Figure 3 demonstrates, once the titre falls below 1 $CID_{50}$ there is a rapid reduction in the probability that at least one chicken will become infected if only five are challenged, as in the 1997 experiment.[1] By using more chickens there is a greater probability of detecting lower viral titres. Therefore, extending the data set allows trends to be determined and predictions to be made with more confidence.

**Table 2:** D values for heat inactivation of two strains of IBD.[1,2] Strain 52/70 was tested in a clarified aqueous bursal homogenate whilst strain CS88 was suspended in an unclarified homogenate consisting of bursa, muscle, skin and fat. D is the time taken to reduce the infectivity by 90% (1 $\log_{10}$ at the specified temperature).

| | **52/70** | **CS88** |
|---|---|---|
| temperature (Celsius) | D (min) | D (min) |
| 60 | - | 45 |
| 70 | 18.8 | 63 |
| 74 | - | 25 |
| 80 | 3 | 42 |

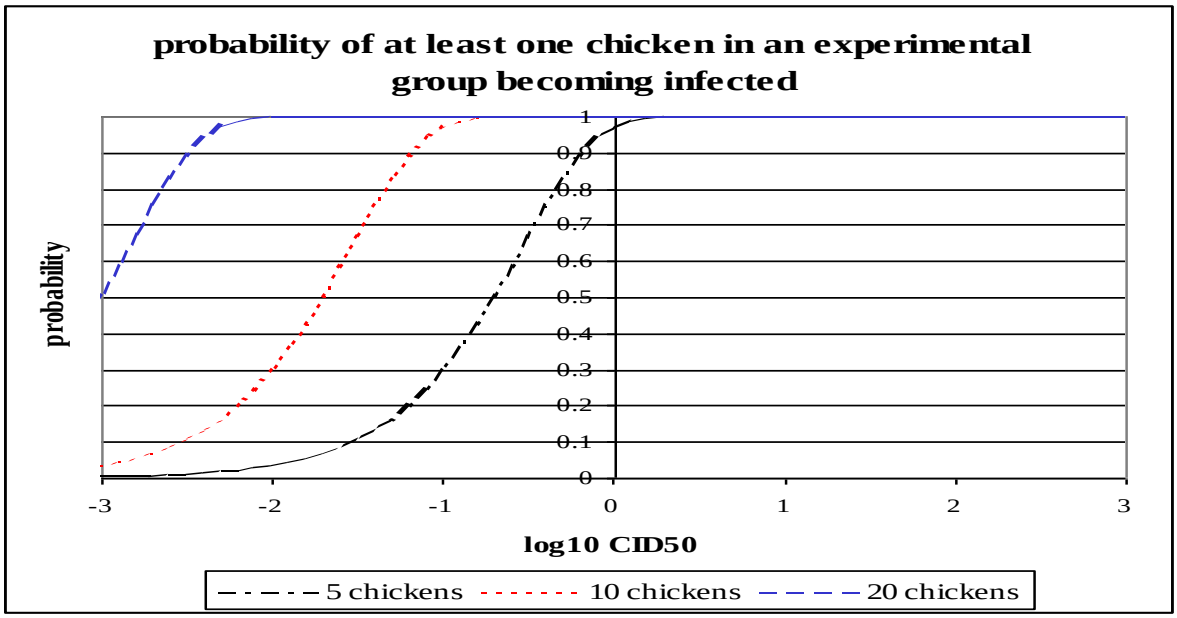



**Figure 3:** The influence of the number of chickens used in titration studies to determine infectivity.

## 2. Viral titre in chicken meat

It is important to establish whether IBD virus is likely to be present in chicken meat and, if so, the likely range of titres. Although little information on titres is available, there are some relevant epidemiological observations and experimental findings:

(i) The disease is highly contagious. Faeces are the main source of the virus in natural infection which is usually via the oral route.[4]

(ii) Commercial flocks are unlikely to be kept free from IBD virus by hygienic measures. The resistance of IBD virus makes it difficult, if not impossible, to

disinfect buildings, particularly poultry houses with earth floors. As a result IBD infection may reoccur in successive batches of birds.[4, 5]

(iii) Although all ages of birds are susceptible to infection, clinical disease is usually not seen in birds less than 3 weeks of age.[4] Under commercial conditions infections usually occur when maternal antibody levels are waning at 2-3 weeks of age.[5]

(iv) Vaccination programs, using both live and killed vaccines are carried out by most producers. Live vaccines are produced from fully or partially attenuated strains of virus, known as 'mild', 'intermediate' or 'intermediate plus' ('hot'), respectively. These live vaccine strains may be shed for prolonged periods.[4, 5] Mild vaccines are used in parent stock to produce a primary response prior to vaccination near to point of lay when inactivated vaccines are used to confer maternal immunity on offspring. Intermediate vaccines are used to protect broiler chickens and commercial layer replacements. They are often administered at 1 day of age to protect any chickens that have no, or minimal, levels of maternal immunity. This establishes a reservoir of vaccine virus within the flock which allows lateral transmission to other chickens when their maternal immunity decays.[6] Some live vaccines may cause reduced weight gain, illness, immunosuppression and even death in young chickens without antibody. They may also spread to other flocks and cause illness.[4]

(v) Previous studies have demonstrated that virus can be recovered from various tissues for up to 11 days post infection (for example the bursa, thymus, spleen, kidney, and caecal tonsils) and is shed in faeces for up to 16 days.[4, 7] The bursa, which is regarded as the target organ,[10] and spleen have been found to have higher titres than other organs.[4, 8] As an example McFerran and McNulty[4] reported titres up to 8 $\log_{10}$ $EID_{50}$ in the bursa, 7 $\log_{10}$ $EID_{50}$ in the spleen and 6 $\log_{10}$ $EID_{50}$ in the thymus and liver in chickens dying 3-4 days post infection.

An experiment in 1997 conducted for the Commonwealth of Australia followed the viral titre in muscle tissue for the first 96 hours post infection.[9] The titre peaked at 1.17 $\log_{10}$ $CID_{50}$/g on day 3 and had declined to 1 $CID_{50}$/g by the fourth day. Unfortunately, the experiment could only be run over 4 days as all the chicks died. While viral titres in other tissues were not determined, all five chicks in each group inoculated with various tissues from infected birds (faeces, spleen, bone marrow, bursa, blood and liver/kidney) were positive for IBD virus at the end of the 4 day study period.

Although a number of authors[4, 5, 8,10] have reported that various tissues are infected with IBD virus there has been little quantification of titres. Apart from the 1997 study[9] of muscle tissue most studies have involved either visceral or lymphatic tissue.[4, 5, 8,10]

While no evidence has been found of a carrier state[5], recent work by Lukert[11] using the virulent Edgar strain, demonstrated infectivity in pooled tissue samples of muscle, liver, kidney, spleen, lungs and bursa for up to 4 but not 5 weeks. Subsequent work by Lukert[12] clarified the status of the various tissues. He

demonstrated that muscle tissue was not infective at 7 days, that liver, lung and kidney had lost infectivity by day 14 and that bursa retained its infectivity for 28 days but not for 35 days. This was much longer than expected from previous studies, although a number, including the 1997 study,[9] studied infected chickens for short periods of time only. Likely explanations include the short duration of the experiments[4] combined with high mortality rates.[9, 10] For instance Kaufer and Weiss[10] used a highly virulent Cu-1 strain to infect both bursectomised and non-bursectomised 4-week old chickens. All of the non-bursectomised chickens serving as positive controls died between 3 and 4 days.

(vi) A study by Jackwood[13] found that IBD virus was found in chickens arriving at a processing plant in the USA.

(vii) Chickens for human consumption are usually slaughtered at 5 to 7 weeks of age. As a result it is reasonable to conclude that chickens sourced from populations where IBD is endemic are likely to be infected either with a natural or vaccinal strain of IBD virus. Unfortunately neither the likely range of prevalences of infection or viral titres associated with different stages of infection within a chicken carcass are known. The 1997 study[9] reported that the viral titre in muscle tissue was 0.83 $\log_{10}$ $CID_{50}$/g on day 2 post infection, peaking at 1.17 $\log_{10}$ $CID_{50}$/g on day 3 and declining to 0 $\log_{10}$ $CID_{50}$/g by day 4. Sources of IBD virus in a chicken carcass would include the bursa, muscle tissue and kidney. Visceral organs such as the spleen, intestines and liver would be removed at slaughter. As over half of a chicken carcass is composed of muscle tissue it would be reasonable to model the likely range of viral titres in a chicken with an active infection using the titres estimated by for muscle.[9] An @Risk function[14], Pert(0,0.83,1.17), where 0, 0.83 and 1.17 represent the $\log_{10}$ $CID_{50}$/g respectively was used in the model outlined in Section 5.

## 3. Estimating the time required to inactivate IBD virus

There are a number of approaches available to determine the time required to inactivate IBD virus. A convention, that appears to have its origins in public health protocols, is based on a 6D reduction. This is the time required to achieve a reduction in viral titre of 6 $\log_{10}$ units, or, in other words, a million-fold reduction. An alternative approach is to calculate the time required to reduce the titre in the product under consideration to a target titre, such as -6 $\log_{10}$. The key assumption in such cases is that the rate of heat inactivation is independent of the initial titre. Table 3 compares the results of the these two approaches with the current MAF time-temperature requirements for IBD virus.

There are two apparent anomalies in these results. For Solution 1 the predicted time for 74 □C is less than for 80 □C, whilst for Solution 2 the predicted time for 70 □C is longer than 60 □C and the time for 74 □C is shorter than 80 □C. Further work is needed to clarify these inconsistencies. However, regardless of these apparent anomalies or the approach adopted, it is apparent that the results from the 1997 study[9] demonstrate that IBD virus is relatively heat tolerant. A comparison with similar studies for Newcastle disease virus[15], outlined in Appendix 3, and

summarised in Table 4, further emphasises just how heat tolerant IBD virus is.

**Table 3:** Time-temperature parameters for IBD virus and predicted final viral titres.

| **Temperature** | **60 □C** | **70 □C** | **74 □C** | **80 □C** |
|---|---|---|---|---|
| **SOLUTION 1**<br>**(square root transformation of time)** | | | | |
| **(i) 6D reduction** | 375 min | 343 min | 147 min | 166 min |
| **(ii) Target (-6 log10 $CID_{50}$/g)** | 474 min | 434 min | 186 min | 211 min |
| **(iii) MAF recommended times** | n/a | 50 min<br>-1.54 $\log_{10}$ $CID_{50}$ | n/a | 9 min<br>-0.65 $\log_{10}$ $CID_{50}$ |
| **SOLUTION 2**<br>**(calculated from the 2nd part of curve)** | | | | |
| **(i) 6D reduction** | 273 min | 375 min | 150 min | 250 min |
| **(ii) Target (-6 log10 $CID_{50}$/g)** | 307 min | 422 min | 169 min | 281 min |
| **(iii) MAF recommended times** | **n/a** | **50 min**<br>-0.05 $\log_{10}$ $CID_{50}$ | **n/a** | **9 min**<br>0.5 $\log_{10}$ $CID_{50}$ |

**Table 4:** Time-temperature parameters for IBD (strain CS88) and NDV (strain Herts 33/56) adapted from two studies.[1,15]

| **Temperature** | **70 □C** | **74 □C** | **80 □C** |
|---|---|---|---|
| **Target (-6 log10 $CID_{50}$/g)** | | | |
| IBD (solution 1) | 434 min | 186 min | 211 min |
| IBD (solution 2) | 422 min | 169 min | 281 min |
| NDV | 24 min | 5 min | 4 min |

## 4. Infectious dose

Viral titres are usually reported as the number of infectious doses required to infect half the exposed eggs, animals or birds, that is a probability of 0.5 that an egg, animal or bird will become infected when challenged with one infectious dose. They are usually reported on a log scale, for example 3.5 $\log_{10}$ $ID_{50}$/g, and the route of challenge noted.
Sutmoller and Vose[16] discussed the probability of an animal becoming infected when exposed to

low levels of bacterial or viral contamination, perhaps even less than the minimum infectious dose of the pathogen as determined experimentally. They concluded that if enough animals are exposed to low levels of contamination there is a chance that at least one will become infected and initiate an outbreak. Their fundamental assumption is that each infectious unit has a non-zero probability of independently infecting an animal. They outline a model that can be used to calculate the probability of at least one animal becoming infected when exposed to a contaminated product:

(a) If (X) viruses have a 50% probability of starting an infection then that means at least one virus is able to infect a cell and initiate infection;

(b) If an assumption is made that each virus has same probability (p) of independently infecting a cell and initiating an infection in an animal then

$$0.5 = 1-(1-p)^{n} \qquad \text{Equation 1}$$

$$p = 1-(1-0.5)^{(1/n)} \qquad \text{Equation 2}$$

where n = the number of viruses;

(c) If the probability that the number of viruses in the contaminated product that will initiate infection in an animal is represented as:

$$q = 1-(1-p)^{VT} \qquad \text{Equation 3}$$

where VT = the number of viruses in the contaminated product

then the probability (P) of infecting at least one animal if a number of animals are challenged is:

$$P = 1-(1-q)^{A} \qquad \text{Equation 4}$$

where A = number of animals challenged.

Unfortunately the actual number of viruses that constitute an infectious dose is usually unknown and the approach outlined by Sutmoller and Vose[16] cannot be applied directly. However, by reworking their formulae it can be shown that the calculation of the probability that an animal will become infected is independent of the actual viral titre in an infectious dose. The implicit assumption is that each infectious dose is made up of a significant number of viruses (at least several orders of magnitude), each of which is capable of independently initiating an infection.

As long as we have some measure of an infectious dose we can calculate the probability directly[17]:

let d = the dose for which (100*h)% of animals become infected
let h = the proportion of animals infected at the reported dose (d), for example 0.5
let n = the number of organisms in the dose (d)

then the probability (p) of infection from a single organism, as determined above, is:

$$p = 1-(1-h)^{(1/n)} \qquad \text{Equation 5}$$

if we have a challenge dose of virus, measured as (f) times the infectious dose (d) [f*d] then the number of viruses in this challenge dose = f*n
and the probability of initiating an infection from the challenge dose is:

$$P1 = 1-(1-p)^{f*n} \qquad \text{Equation 6}$$

substituting $(1-h)^{(1/n)}$ from equation 5 for (1-p) in equation 6 then

$$P1 = 1-(1-h)^{[(1/n)*f*n]} = 1-(1-h)^{(f)} \qquad \text{Equation 7}$$

Thus the probability of (f) infectious doses initiating infection in an animal is:

$$P1 = 1-(1-0.5)^{X} \qquad \text{Equation 8}$$

where $X = ID_{50}$ (the number of infectious doses)

If a number of animals are challenged with this infected or contaminated material then the probability that at least one animal will become infected is:

$$P2 = 1-(1-P1)^{n} \qquad \text{Equation 9}$$

where n is the number of animals or birds that are challenged.

Equations 8 and 9 can be combined as:

$$P2 = 1-(1-0.5)^{X*n} \qquad \text{Equation 10}$$

*<u>Hypothetical Example</u>*

*It has been reported that chicken meat contaminated with disease X virus cooked for 27 minutes at 70 □C will have a final viral titre of $10^{-3}$ $CID_{50}$/g. If 15 chickens are each fed 10 g of cooked chicken meat what is the probability that at least one chicken will become infected? [Note: it is assumed that there is a uniform spread on infectivity in the chicken meat.]*

*10 g of cooked chicken meat will contain $10^{-2}$ chicken infectious doses ($CID_{50}$)*

$$10^{-3}\ CID_{50}/g \times 10\ g = 10^{-2}\ CID$$

*Equation 10: $P3 = 1-(1-.5)^{10-2*15} = 1-(1-0.5)^{1.5E-1} = 9.9E-2$*

*Therefore the probability that at least one chicken in a group of 15 will become infected is 0.099 or approximately 1 in 10.*

## 5. Probability of infecting backyard or fancy poultry

Appendix 1 estimated the probability of IBD being introduced in imported chicken meat products and establishing in backyard flocks. The same chicken meat products are considered in this model, namely, whole chicken carcasses, bone-in chicken cuts (wings, legs, drums) and boneless chicken cuts. In addition to backyard flocks, fancy poultry are also included as there are likely to be some significant differences in the exposure scenarios between these two populations. The model consists of:

1 a release assessment, which calculates the probability that an imported chicken carcass is infected;

2 an exposure assessment, which calculates the probability that at least one chicken in a backyard or fancy poultry flock fed infected chicken scraps becomes infected;

3 a risk assessment, which calculates the final probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year.

### 5.1 Release assessment

The same input distributions as used in Appendix 1 are used to calculate the probability that an imported chicken carcass is infected:

**(R1)** the probability that the source flock is infected

$$R1 = PERT(0.3,0.7,0.9)^{(14)}$$

**(R2)** the probability of an active infection at slaughter

for a chicken carcass:

$$R2 = Beta(20000*0.30015+1,20000-(20000*0.30015)+1)^{(14)}$$

for bone in-chicken cuts or boneless chicken cuts:

$$R2 = Beta(20000*0.05095+1,20000-(20000*0.05095)+1)^{(14)}$$

**(R3)** the probability that an imported chicken carcass is infected

$$R3 = R1*R2$$

### 5.2 Exposure assessment

In the IBD model outlined in Appendix 1, the probability that chicken scraps remain infected after cooking and that feeding infected chicken scraps would lead to infection was estimated indirectly. In this model, an infectious dose model incorporating experimentally derived heat inactivation data[1] is used to calculate the probability of a least one chicken in either a backyard or fancy poultry flock becoming infected. The likely viral titre of virus to survive heat inactivation is based on the predictions of solution 1, Table 3.

The exposure assessment consists of:

**(P1)** The probability of backyard or fancy poultry flock being fed scraps

for backyard poultry: P1 = PERT(0.1,0.9,1)[14] This is the same as P3 in Appendix 1.

for fancy poultry: P1 = PERT(0.5,0.1,0.2)[14]

Advice from the Poultry Industry Association of New Zealand (PIANZ) and the Taranaki Poultry and Pigeon Club indicates that backyard flocks are much more likely to be fed kitchen scraps than fancy poultry.

**(P2)** The probability of a backyard or fancy poultry flock having chickens of a susceptible age. Chickens older than 6 months are considered to be refractory to infection with IBD.

for backyard poultry: P2 = PERT(0.01,0.05,0.1)[14]

for fancy poultry: P2 = 1

Advice from PIANZ and the Taranaki Poultry and Pigeon Club indicates that most backyard flocks consist of spent layer hens or point of lay pullets, with only a few having chickens younger than 6 months of age. In contrast most, if not all, fancy poultry flocks breed chicks.

**(P3)** The probability of at least one susceptible chicken in a backyard or fancy poultry flock becoming infected. An infectious dose model, divided into the following sections is used to calculate P3:

**Section 1** Provides the predicted viral titre from the heat inactivation model:

**(PVT)** predicted viral titre ($\log_{10}$ $CID_{50}$)

**Section 2** Calculates the likely amount of edible chicken scraps fed to backyard poultry:

**(W)** weight of a chicken carcass (g).

A Normal(1500,45)[14] distribution is used with an average carcass weight of 1500 g and a standard deviation of 45 g. These values were supplied by PIANZ.

**(S)** proportion disposed of as edible scraps.

From information supplied by PIANZ an average chicken carcass consists of approximately 870 g of edible tissue (muscle, meat, skin and fat). Bones are considered to be inedible. It is considered likely that the following proportions of edible tissue will be disposed of as scraps: whole chicken carcasses, 0.05 to 0.1; bone-in cuts, 0.03 to 0.05; boneless cuts 0.005 to 0.01.

for whole chicken carcasses:

S = (870)*Uniform(0.05,0.1)/1500

for bone-in chicken cuts:

S = (870)*Uniform(0.03,0.05)/1500

for boneless chicken cuts:

S = (870)*Uniform(0.005,0.01)/1500

**(E)** weight of edible scraps generated per carcass (g).

E = W*S

**(F)** number of chickens in a backyard or fancy poultry flock

for backyard poultry:

F = PERT(5,15,30)[14]

where 5, 15 and 30 represent the minimum, most likely and maximum values respectively. As there is no statistical information on the likely numbers of chickens in a backyard flock an educated guess was made based on estimates provided by various staff within MAF, PIANZ and the Taranaki Poultry and Pigeon Club.

for fancy poultry:

From information supplied by the Taranaki Poultry and Pigeon Club, fancy poultry flocks are likely to consist of one to four breeds, with two hens and one rooster per breed producing ten to 20 chicks per year.

F = round(Uniform(1,4)*Uniform(10,20)+ 3*(Uniform(1,4),0)

**(C)** amount of scraps available for each chicken.

C = E÷F

An assumption was made that all the scraps would be eaten and that each chicken in a backyard or fancy poultry flock would consume an equal proportion of scraps.

**Section 3** Calculates the likely viral titre ($\log_{10}$ $CID_{50}$) in the chicken scraps:

**(M)** likely number of $\log_{10}$ $CID_{50}$'s available for each chicken.

$M = C*10^{PVT}$

**Section 4** Calculates the probability of initiating infection in a backyard or fancy poultry flock:

**(Pc)** the probability that a viral titre of (M) in the scraps will initiate infection in a susceptible chicken

$Pc = 1-(1-0.5)^{(M)}$

**(A)** the proportion of a backyard or fancy poultry flock consisting of susceptible age chickens:

A = round(Uniform(1,4)*Uniform(10,20),0)/F

where the same values are used as for the calculation of (F), the number of chickens in a fancy poultry flock, to estimate the proportion of birds less than 6 months of age. This estimate is also used for those backyard poultry flocks that may breed or rear their own chicks.

**(SC)** number of susceptible age chickens in a backyard or fancy poultry flock

SC = round(F*A,0)

**(P3)** the probability of at least one susceptible chicken in a

backyard or fancy poultry flock will becoming infected. A separate calculation is made for backyard and fancy poultry.

$P3 = 1-(1-Pc)^F$

**(P4)** calculates the probability of feeding scraps to susceptible chickens and initiating infection in a backyard or fancy poultry flock. A separate calculation is made for backyard and fancy poultry.

P4 = P1*P2*P3

## 5.3 Risk assessment

The same input distributions as used in Appendix 1 are used to calculate the final probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year:

**(N)** the number of broiler consumed per year in New Zealand

N = 63,000,000

**(pi)** the proportion of broiler carcasses or carcass equivalents that are imported

for whole chicken carcasses:

pi = Simtable({0.01,0.1,0.2}), where the values in the simulation table represent 3 different levels of market penetration

for bone-in cuts and boneless cuts:

pi = Simtable({0.001,0.01,0.1}), where the values in the simulation table represent 3 different levels of market penetration

**(pr)** the proportion of households with chickens

for backyard poultry:

pr = ((7.00E+4*(1-Uniform(0.4,0.6))-400)/1.21E+6)

for fancy poultry:

pr = 400/1.21E+6

where 400 represents the number of households with fancy poultry. This is based on information supplied by the Taranaki Poultry and Pigeon Club.

**(z)** the number of imported birds consumed per year in households with backyard chickens or fancy poultry. A separate calculation is made for backyard and fancy poultry.

$$z = N*pi*pr$$

**(P5)** the probability of at least one flock becoming infected each year. A separate calculation is made for backyard and fancy poultry.

$$P5 = 1-(1-R3*P4)^{z}$$

**(P6)** the combined probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year

$$X = P5_{BYF}+P5_{FPF} - P5_{BYF}*P5_{FPF}$$

where BYF = backyard flock and FPF = fancy poultry flock

## 6. Simulation results

Table 5 shows the results generated by the model for various heat treatment options after 10,000 iterations.

## 7. Conclusions

IBD virus is unusually resistant to thermal inactivation. In most, if not all households, chicken meat is unlikely to be cooked for longer than 45 to 60 minutes. While whole chickens may be cooked in an oven for such times it is very unlikely that bone-in or boneless cuts would be cooked for as long. They would most probably be cooked for considerably shorter periods, perhaps for only 10 to 15 minutes. In most domestic environments the internal cooking temperatures reached are likely to be in the range of 70□C to 80□C and it is unlikely that these temperatures would be maintained for more than several minutes, even in an oven.

If chicken meat were cooked to an internal temperature of 80□C for as long as 1.5 hours there is still a significant risk that sufficient virus will remain viable. The model predicts that, even with boneless cuts at a market penetration of 0.1%, it is likely that at least one backyard or fancy poultry flock will become infected each year. The 95th percentile for this scenario is 0.02 and rises to 0.16 and 0.83 with increasing market penetration. To achieve a significant reduction in viral titre that would mitigate against the risk of IBD becoming established, unrealistically long cooking times would be required.

The predictions for prolonged cooking times to inactivate IBD virus contrast markedly with Newcastle disease virus (NDV). A similar model for NDV, outlined in Appendix 3, predicts that at the current MAF recommendation of cooking chicken at 80□C for 9 minutes, the titre will

have fallen to -20 $\log_{10}$ $CID_{50}$/g. This essentially indicates that ND virus has been inactivated. To achieve a reasonable risk reduction for IBD of say, 1 in 5,000 (2.00E-04) with the commodity least likely to generate scraps, the cooking time would need to be approximately 3 to 3.5 hours at 80□C. Such times are obviously unrealistic and serve to demonstrate that heat inactivation cannot be relied upon as a sanitary measure for IBD virus.

**Table 5:** The 95th percentiles results for (P6), the combined probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year. These results indicate that in 95% of iterations of the model the probability of at least one backyard or fancy poultry flock becoming infected each year is less than the value shown.

| Commodity | Market Penetration | 50 min @ 70□C | 9 min @ 80□C | Target titre (-6) | 6D reduction |
|---|---|---|---|---|---|
| Whole chicken carcasses | 1% | 1 | 1 | 0.01 | 0.08 |
| | 10% | 1 | 1 | 0.1 | 0.56 |
| | 20% | 1 | 1 | 0.195 | 0.81 |
| Bone-in cuts | 0.1% | 0.94 | 0.99 | 9.50E-05 | 7.40E-04 |
| | 1% | 1 | 1 | 9.60E-04 | 7.40E-03 |
| | 10% | 1 | 1 | 9.50E-03 | 7.20E-02 |
| Boneless cuts | 0.1% | 0.62 | 0.96 | 1.80E-05 | 1.40E-04 |
| | 1% | 0.99 | 1 | 1.80E-04 | 1.40E-03 |
| | 10% | 1 | 1 | 1.80E-03 | 1.40E-02 |

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**Appendix 3: Quantitative risk analysis of cooked chicken meat products with specific reference to recent studies on the infectious dose and heat inactivation of Newcastle disease virus.**

## 1. Heat inactivation studies

In 1997 experimental work was carried out by Alexander[1] on behalf of the Commonwealth of Australia to determine the heat inactivation characteristics of Newcastle disease (ND) virus in homogenised chicken meat. He found that the virus was more more heat resistant than anticipated from previous experimental work carried out in liquid whole eggs. The results are presented in Table 1. Alexander[1] suggested that it would be useful to know the D values (the time taken to reduce infectivity by 1 $\log_{10}$) at each temperature. The D value allows the calculation of the time needed at a particular temperature to reduce the probability of infectivity remaining to an acceptable level, provided a starting titre is known or assumed. Alexander[1] recommended that where heat inactivation curves are biphasic, as some in his study were, the D value be calculated from the second or shallowest part of the curve. He plotted a best fitting straight line by eye.

**Table 1:** Experimental data from Alexander.[1] Titres of ND virus are expressed as $\log_{10}$ mean egg infectious doses per 0.1 ml of inoculum ($EID_{50}$/0.1ml). (A) = infective allantoic fluid. (a) = no virus detected.

| Time (sec) | 60 □C | 65 □C | 65 □C (A) | 70 □C | 74 □C | 80 □C | 80 □C (A) |
|---|---|---|---|---|---|---|---|
| 0 | 6.7 | 7.1 | 6.3 | 7.3 | 7.3 | 6.7 | 7.1 |
| 20 | | | | | | 3.1 | 3.3 |
| 30 | | 6.9 | 2.1 | | | | |
| 40 | | | | | | 2.9 | - |
| 60 | 5.3 | 6.1 | 1.9 | 4.1 | 4.1 | 2.5 | - |
| 80 | | | | | | | - |
| 90 | | | | | 4.7 | | |
| 100 | | | | | | | - |
| 120 | 2.7 | 4.9 | 1.7 | 2.7 | 4.7 | -0.1 | - |
| 180 | 2.7 | | 0.7 | | 2.9 | - | |
| 240 | | 2.5 | 0.7 | 1.7 | 1.7 | - | |
| 300 | | | | 1.3 | -0.3 | | |
| 360 | 3.3 | 2.5 | -[a] | | | | |
| 480 | 1.1 | 1.5 | | - | | | |
| 540 | | | | 0.5 | | | |
| 600 | 2.7 | | | | | | |

There are a number of options available to plot a best fitting line, for example by eye, as Alexander[1] did, using a function free model, or standard techniques such as linear regression. With any of these techniques the data may be transformed to enhance the "fit", e.g. by taking the square root, plotting on a log scale etc. Table 1 shows that the number of data points for each trial are limited so fitting lines must be done with caution. Figure 1 is a series plots for the

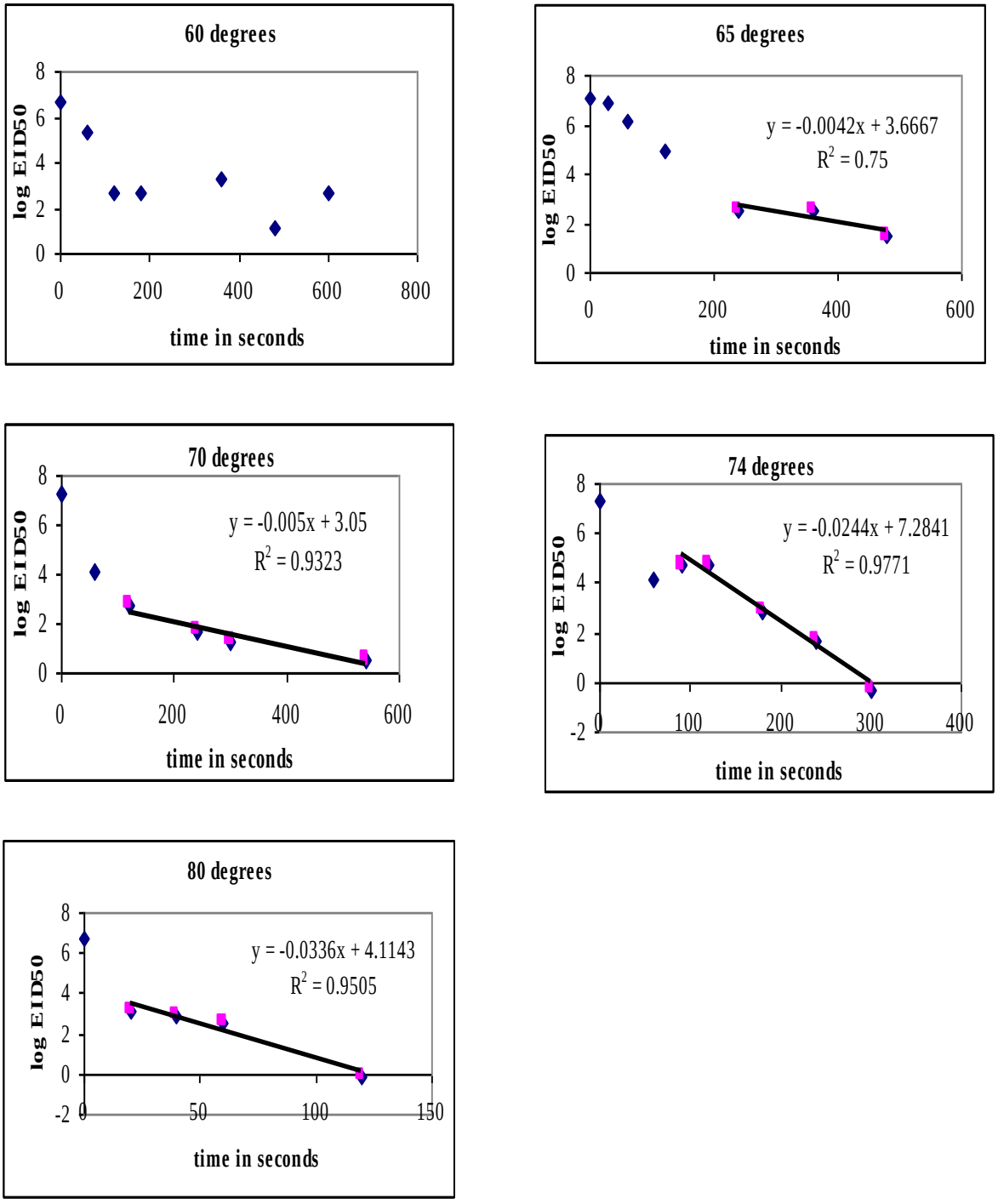


**Figure 1:** Heat inactivation of ND virus in chicken meat homogenate at various temperatures. A trend line was fitted to the shallowest part of the biphasic curve.

different temperatures from Alexander's[1] experiment. After exploring a number of different approaches, the technique adopted for this model was to fit a trend line using linear regression on the untransformed data from the shallowest part of the curve. The output provided an estimate of the mean of the slope and the associated standard error of the mean. The central limit theorem was applied and the mean and standard error used in the software @Risk[2] function, Normal(mean,standard deviation), allowing a distribution of likely slopes to be determined. Since the D value, referred to above, is equal to one divided by the slope, a distribution of D can be modelled.

Table 2 compares the D values calculated by Alexander[1] and those calculated by fitting a trend line using linear regression. There are significant differences in the estimates of D for the 65 □C and 70 □C experiments. Alexander[1] appears to have used the last two data points in the 65 □C experiment to obtain a D value of 120 seconds. However, MAF considers that the last three data points provide a more appropriate estimate as there appears to be a distinct trend towards a biphasic curve involving the last three points. The value of 82 seconds reported by Alexander[1] for the 70 □C experiment does not appear to be supported by the data unless the second to fifth of the six data points are used. There appears to be a distinct trend emerging from the third data point. For this reason, the last four points were used in the linear regression model.

**Table 2:** D values for heat inactivation of ND virus in chicken meat homogenate at various temperatures. D is the time taken to reduce the infectivity by 90% (1 $\log_{10}$ at the specified temperature).

| | **Alexander[1]** | **Linear regression (expected values)** |
|---|---|---|
| temperature | D (seconds) | D (seconds) |
| 60 | not done | not done |
| 65 | 120 | 238 |
| 70 | 82 | 200 |
| 74 | 40 | 40 |
| 80 | 29 | 30 |

## 2. Viral titre in chicken meat

Alexander[3] determined that for the ND virus Herts 33/56 strain, which is highly pathogenic, 4 $\log_{10}$ $EID_{50}$ are required to establish infection in 3-week old chickens when given by the oral route. He also determined the viral titres in a range of tissues and organs from 6-week old experimentally infected chickens. The highest titres reached at day 4 were 6 $\log_{10}$ $EID_{50}$/g in the heart/kidney/spleen pool, 4 $\log_{10}$ $EID_{50}$/g in breast muscle and faeces and 4.2 $\log_{10}$ $EID_{50}$/g in leg muscle. The experiment was only able to be conducted over a 4 day period as all the chicks died, so subsequent trends in viral titres could not be determined. Alexander[3] noted that little has been published on the titres of ND virus in tissues. He considered that the level of infectivity recorded was lower than expected and postulated that factors such as age, partial immunity, less virulent virus or some other factor which may prolong the life of the infected bird may result in higher titres.

The likely titres of vaccine strain in various tissues seem not to have been studied. It may be reasonable to speculate that chickens vaccinated with a live vaccine are likely to have a similar range of titres in tissues.

Although muscle tissue has a lower titre than other tissues, it may be reasonable to assume, for the purpose of modelling, that the likely range of titres in a chicken carcass or cut covers the range seen in all tissues. This assumption is supported by Alexander's observation[3] that he expected a higher level of infectivity in the tissues examined in his. Since Alexander[3] reported that 4 $\log_{10}$ $EID_{50}$ are required to establish infection, the titre in a chicken with an active infection at slaughter was modelled using a Pert(1,1.05,1.5)[2] distribution where the values 1, 1.05, and 1.5 represent the minimum, most likely and maximum $\log_{10}$ $CID_{50}$/g values respectively.

## 3. Estimating the time required to inactivate ND virus.

There are a number of approaches available to determine the time required to inactivate ND virus. A convention that appears to have its origins in public health protocols is based on a 6D reduction. This is the time required to achieve a reduction in titre of 6 $\log_{10}$ units. An alternative approach is to calculate the time required to reduce the titre to a target such as -6 $\log_{10}$. The key assumption in such cases is that the rate of heat inactivation is independent of the initial titre. Table 3 compares the times and predicted titres of these two approaches with the current MAF time-temperature requirements for ND virus.

**Table 3:** The predicted time, expressed as the expected values and the 95th percentile after 10,000 iterations, to reduce the viral titre for ND virus at various temperatures.

| **Temperature** | **65 □C** | **70 □C** | **74 □C** | **80 □C** |
|---|---|---|---|---|
| **6D reduction** | 23.8 min | 20 min | 4 min | 3 min |
| (i) predicted titre = -4.9 ($\log_{10}$ $CID_{50}$/g) | | | | |
| (ii) in 95% of iterations the predicted time is less than | 147 min | 29.8 min | 4.6 min | 4.8 min |
| **Target (-6 log10 $CID_{50}$/g)** | 28.2 min | 23.7 min | 4.7 min | 3.5 min |
| (i) in 95% of iterations the predicted time is less than | 174 min | 35 min | 5.5 min | 3.5 min |
| **MAF recommendation** | not recommended | 50 min | not recommended | 9 min |
| (i) predicted titre (expected value) | | -16.6 $\log_{10}$ $CID_{50}$ | | -19.7 $\log_{10}$ $CID_{50}$ |
| (ii) in 95% of iterations the predicted titre is less than | | -11.7 $\log_{10}$ $CID_{50}$ | | -14.9 $\log_{10}$ $CID_{50}$ |

## 4. Infectious dose

A method which can be used to calculate the probability of at least one animal or bird becoming infected when ingesting a contaminated product is outlined in section 4 of Appendix 2. The same approach is applied in this model.

## 5. Probability of infecting backyard or fancy poultry

The same model described in section 5 of Appendix 2 is used to estimate the probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year. The only difference is in the probabilities used in the release assessment. The probability that the source flock is infected, (R1), and that chickens have an active infection at time of slaughter, (R2), are each assumed to be one in this model.

**(R1)** the probability that the source flock is infected.

$R1 = 1$

**(R2)** the probability of an active infection at slaughter.

$R2 = 1$

This assumption is based on the possibility that all chicken flocks are infected with a strain of ND virus having an intracerebral pathogenicity index (ICPI) greater than 0.0, either through natural infection or vaccination. The strains of ND virus isolated so far in New Zealand have all had an ICPI of 0.0.[4] Even a strain, such as La Sota (ICPI = 0.4), if introduced into New Zealand, could result in impaired productivity and perhaps necessitate vaccination with a strain such as V4 to minimise its impact in the poultry industry (see section 3.3.9).

## 6. Simulation results

Table 4 shows the results generated by the model after 10,000 iterations whilst table 5 shows the cooking times required to achieve a chosen target titre for the various temperatures in Alexander's[1] experiment and the risk estimates for whole chicken carcasses.

## 7. Conclusions

ND virus is relatively sensitive to thermal inactivation. With minimal cooking times a significant reduction in viral titre ($\log_{10}$ $CID_{50}$) can be achieved. For example, by cooking at 80□C for 5 minutes the viral titre is predicted to fall to less than -9 $\log_{10}$ $CID_{50}$. Even under the conservative assumption that all birds from the source flocks are experiencing an active infection, either as a result of natural infection or vaccination, there is only a remote chance of an outbreak of Newcastle disease occurring in a backyard of fancy poultry flock.

Although a million fold (6D) reduction in titre may appear to provide adequate security, this simulation model suggests that caution is warranted. It predicts that at least one outbreak of Newcastle disease in a backyard or fancy poultry flock is quite probable for most of the commodities examined (Table 4). As an example, the predicted cooking time at 80□C to achieve

a 6D reduction is 3 minutes. The viral titre falls to -4.9 $\log_{10}$ $CID_{50}$/g. By cooking chicken for an extra 2 minutes the titre falls dramatically to less than -9 $\log_{10}$ $CID_{50}$/g and, as can be seen in Table 5, the risk has declined significantly.

The important parameter to focus on is the final or target titre. We can then work backwards, taking into account the likely number of chickens that may be exposed and the initial titre. From this information we can determine the appropriate time-temperature parameters to achieve the desired target. As an example of this approach cooking times associated with various temperatures can be selected from Table 5 for whole chicken carcasses to achieve the desired reduction in risk. These time-temperature parameters form the basis for appropriate sanitary standards for Newcastle disease.

**Table 4:** The 95th percentiles results for (P6), the combined probability of at least one backyard or fancy poultry flock becoming infected in New Zealand each year. These results indicates that in 95% of iterations of the model the probability of at least one backyard or fancy poultry flock becoming infected each year is less than the value shown.

| Commodity | Market Penetration | 50 min @ 70□C | 9 min @ 80□C | Target titre (-6) | 6D reduction |
|---|---|---|---|---|---|
| Whole chicken carcasses | 1% | 6.13E-08 | 2.91E-11 | 0.05 | 0.51 |
| | 10% | 6.13E-07 | 2.92E-10 | 0.4 | 0.99 |
| | 20% | 1.23E-06 | 5.84E-10 | 0.64 | 0.99 |
| Bone-in cuts | 0.1% | 3.29E-09 | 1.14E-12 | 2.67E-03 | 3.73E-02 |
| | 1% | 3.29E-08 | 1.14E-11 | 2.64E-02 | 0.32 |
| | 10% | 3.29E-07 | 1.14E-10 | 0.23 | 0.98 |
| Boneless cuts | 0.1% | 5.94E-10 | 0 | 5.24E-04 | 7.21E-03 |
| | 1% | 5.94E-09 | 0 | 5.19E-03 | 6.98E-02 |
| | 10% | 5.94E-08 | 0 | 5.07E-02 | 0.52 |

**Table 5:** Predicted cooking times (expected values) to achieve a target viral titre for ND virus in a whole chicken carcass. The risk estimate indicates that in 95% of iterations the probability of at least one backyard or poultry flock becoming infected each year is less than the value shown.

| target titre ($log_{10}$ $CID_{50}$/g) | 65 □C | 70 □C | 74 □C | 80 □C | risk estimate | | |
|---|---|---|---|---|---|---|---|
| -6 | 28.2 min | 23.7 min | 4.7 min | 3.5 min | 0.51 | 0.99 | 0.99 |
| -7 | 32.2 min | 27.1 min | 5.4 min | 4.0 min | 5.13E-03 | 5.02E-02 | 9.79E-02 |
| -8 | 36.2 min | 30.4 min | 6.1 min | 4.5 min | 5.16E-04 | 5.15E-03 | 1.02E-02 |
| -9 | 40.1 min | 33.7 min | 6.7 min | 5.0 min | 5.21E-05 | 5.20E-04 | 1.04E03 |
| -10 | 44.1 min | 37.1 min | 7.4 min | 5.5 min | 5.17E-06 | 5.18E-05 | 1.03E-04 |

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**Appendix 4: Quantitative assessment of the risk of backyard poultry flocks becoming infected with IBD virus serotype 2 should BMFL turkey meat preparations be imported from the United Kingdom.**

***Introduction***

A Monte Carlo model was constructed using the software packages Excel[43] and @Risk[44] to assess the risk of backyard poultry flocks becoming infected with IBD virus serotype 2 should BMFL turkey meat preparations be imported from the United Kingdom.

The model is almost identical to that developed for assessing the risk of IDB introduction and establishment as a result of the importation of chicken meat, differing only in a number of the assumptions made. (The structure of the model is shown in Figure 1 of Appendix 1 of this document.)

The final output of the model is the probability of IDB serotype 2 occurring in at least one backyard poultry flock per year.

To evaluate the effect of volume of trade, several levels of market penetration were considered in the model.

***Release Assessment : probability of infection in imported turkey meat products***

The release assessment estimates the probability that BMFL turkey meat products will be infected with IBD2 virus.

This probability is *R3*, which is a function of;

- Probability that the source turkey flock is infected, *R1*;
- Probability that infection is present in specific tissues of the turkeys at the age of slaughter, *R2*.

The variables used in the Monte Carlo simulation model were as follows;

*R1* Probability that the source flock is infected.
A 1985 serological survey in England found IBD2 antibody in 29 out of 32 turkey flocks.(1)

The distribution used to model this was Beta(r+1, n-r+1) where r is the number of "successes", 29 and n is the number of "trials", 32.
That is, the model used Beta(30, 4).

[43] Microsoft Corporation, USA.

[44] Palisade Corporation, NY, USA

*R2* Probability of active infection when slaughtered.
This is calculated in a sub-model using the method described in *Note I.*
The key data used for this calculation included:

- Age at slaughter.
  The value used in the sub-model was 84 days (12 weeks).

- Age at which turkeys become infected, in days.
  This distribution used to model this in the sub-model was :
  PERT (1, Uniform (28,49), 84)

- Duration of infectivity in turkey muscle tissue, in days.
  This distribution used to model this in the sub-model was :
  Uniform(2, 6)

The probability of active infection in muscle tissue at the time of slaughter is modelled in the main model by the following beta distribution:
Beta (20, 19982)

(Full details of the method of calculation of the values to be used in the distribution of *R2* are shown in *Note I* of this Appendix.)

*R3* The probability that an imported carcass is infected,
*R3= R1 x R2*

***Exposure Assessment : Probability of imported turkey meat products causing infections in poultry flocks***

The fact that an imported commodity may contain an infective agent does not mean that the agent will necessarily come into contact with a susceptible host in New Zealand. The exposure assessment estimates the probability that, given the importation of BMFL turkey meat preparations which are infected with IBD2 virus, infection will be able to establish in poultry flocks.

The model is based on the assumption that for IBD2 to become established in New Zealand poultry flocks as a result of importing infected BMFL turkey meat preparations, scraps of these infected turkey meat products would have to be fed to poultry flocks in this country.

This probability is *P5*, which is a function of;

- Probability that the BMFL turkey meat preparations will generate scraps which a chicken can eat, *P1;*

- Probability that scraps remain infected after cooking, given that infected scraps are available, *P2;*
- Probability that infected scraps are fed to flocks, given that scraps remain infected after cooking, *P3;*
- Probability that infection is established given that infected scraps are fed, *P4*.

The simulation model used the following data for these variables;

*P1* Probability that the BMFL turkey meat preparations will generate scraps which a chicken can eat.

It was considered that the probability of generating edible scraps from BMFL turkey meat preparations was similar to that for boneless cuts of chicken meat, that is, not greater than 1%. The distribution for this probability used in the model was :

*P1* = Uniform (0, 0.01)

*P2* Probability that infected scraps remain infected after cooking. (See *Note III* in Appendix 1 for data on which these estimates are based.)

| | |
|---|---|
| Minimum | 0.5 |
| Most likely | 0.8 |
| Maximum | 1 |

The distribution for *P2* used in the model is PERT (0.5, 0.8, 1.0)

*P3* Probability that infected scraps of imported chicken meat products are fed to backyard flocks given that scraps remain infected after cooking. (This is a guess, but it is likely that all or most kitchen scraps are fed to the chickens in those households which keep backyard flocks. Indeed, kitchen scraps from more than one household may be fed to a single backyard flock. Large volumes of table scraps may be fed to poultry flocks kept by institutions such as boarding schools.)

| | |
|---|---|
| Minimum | 0.1 |
| Most likely | 0.9 |
| Maximum | 1.0 |

The distribution for *P3* used in the model is PERT (0.1, 0.9, 1.0)

*P4* Probability that infection is established in a backyard flock that is fed infected scraps. These estimates are the same as used in Appendix 1 for chicken meat products.

It is guessed that 60% of backyard poultry flocks are comprised of old layer hens which would not be susceptible to IBD infection, and 10% of flocks would be layers

established from point of lay pullets, which would also not be susceptible. That leaves approximately 30% of backyard flocks where there are birds of mixed age which would include susceptible age groups. Therefore the following estimates for this variable were used in the model:

| | |
|---|---|
| Minimum | 0.25 |
| Most likely | 0.5 |
| Maximum | 0.75 |

The distribution for *P4* used in the model is PERT (0.25, 0.55, 0.75)

*P5* Probability of infection establishing if infected chicken meat products are consumed in a household which keeps backyard poultry.

$P5 = P1 \times P2 \times P3 \times P4$

The variables *R3* and *P5* are combined as follows :

*X* Probability that BMFL turkey meat preparations will result in IBD infection in a flock of backyard chickens, given that kitchen waste containing scraps of turkey meat products is fed to backyard chickens. This probability can be thought of as the risk per turkey carcass equivalent imported. It is the product of the probability that a unit of imported product is infected, *R3*, and the probability of infection establishing if infected chicken meat products are consumed in a household which keeps backyard poultry, *P5*.

$X = P5 \times R3$

***Final Risk Estimate***

Given the estimate *X*, the annual risk of disease introduction and establishment in backyard poultry flocks in New Zealand depends on the volume of BMFL turkey meat preparations that are imported per year and consumed in households where backyard poultry flocks are kept, *z*.

This is a function of :

- The number of turkey carcasses consumed per year in New Zealand, *N*;
- The proportion of turkey consumption which would be likely to consist of imported BMFL turkey meat preparations, or in other words the market penetration, *pi*;
- The proportion of households in this country which keep backyard poultry, *pr*.

The simulation model used the following data to model the number of turkey carcasses likely to be imported per year :

*N* Turkeys consumed each year in New Zealand[(2)],
*N* = Uniform (350,000, 400,000)

*pi* Market penetration, the proportion of consumed carcasses which are imported.

BMFL estimate that they will export to New Zealand the carcass equivalent of about 20% of the current turkey consumption.[(3)] To assess the sensitivity of the model output to this variable three levels of market penetration were considered; 10%, 20% and 50%.

Assuming that imported turkey meat preparations would just as likely be consumed in households that keep backyard chickens as in households which do not, i.e. consumption is uniform, the probability that an imported chicken would be consumed in a household where backyard poultry are kept equals the proportion of New Zealand households which currently keep backyard poultry, *pr*, which is a function of :

- The total number of households in New Zealand, *H1*.
- The number of households which were known to keep backyard poultry when last surveyed, *H2*.
- The proportional decline in the keeping of backyard chickens since the 1970s, *f*.

*H1* Number of households in New Zealand[(4)],
*H1* = $1.21 \times 10^6$

*H2* Last figure for households keeping backyard poultry flocks, 1970s[(5)],
*H2* = $7.00 \times 10^4$

*f* Proportional reduction in the practice of backyard poultry keeping since the ‘70s. No information is available on this matter, so it is considered a reasonable guess that the number of households which keep backyard poultry flocks today is between 40% and 60% of the number of households which kept them in the 1970s.

$f$ = Uniform (0.4, 0.6)

*pr* Proportion of households currently keeping backyard poultry.;
$pr$ = [H2 x (1-$f$ )] / H1

Therefore,

*z* Number of turkey carcass equivalents imported into New Zealand per year and consumed in households which keep backyard poultry;

$z = N \times pi \times pr$

***Final Risk Estimate***

The probability of ***no*** disease introduction per year can be calculated as :
$(1-X)^z$

and the probability that ***at least one*** backyard flock becomes infected per year is :
$1-(1-X)^z$

***Risk Assessment Results***

The key result of interest is the probability that ***at least one*** backyard flock would become infected with IBD serotype 2 per year, $1-(1-X)^z$.

Three levels of market penetration were modelled, and for each scenario 10,000 iterations of the model were run. This allows the results to be reported in terms of the percent of iterations that had a result above or below a certain value. The most common way to report the result is in terms of the 95$^{th}$ percentile of iterations. In other words, in 95% of iterations the result was less than the quoted figure.

The 95$^{th}$ percentile results for final risk estimate of the probability that ***at least one*** backyard flock would become infected per year, $1-(1-X)^z$ , are shown in Table 1.

Table 1: Summary of model results

| Market Penetration | Mean Result | 95th Percentile Result |
|---|---|---|
| 10% | $1.46 \times 10^{-3}$ | $3.47 \times 10^{-3}$ |
| 20% | $2.92 \times 10^{-3}$ | $6.94 \times 10^{-3}$ |
| 50% | $7.29 \times 10^{-3}$ | $1.73 \times 10^{-2}$ |

***Sensitivity Analysis***

The sensitivity analysis for cuts of chicken meat (both bone-in and boneless) showed that the three most important variables affecting the outcome were :

- *P1*, probability that the commodity will generate scraps which can be eaten by a chicken
- *R2*, probability that infectivity is present in turkey meat at the time of slaughter
- *P4*, probability that feeding scraps will result in infection becoming established.

The assumptions made in assigning distributions to *P1* and *P4* are explained in the previous discussion on the exposure assessment. It is considered that no unreasonable assumptions have been made.

The data used for the calculation of the distribution of *R2* is derived from published reports and the method of calculation, outlined in *Note I*, is logical and scientifically valid.

***Conclusion***

Under the assumptions used, if BMFL turkey meat preparations were to be imported into New Zealand, even in relatively large volumes, the risk of introducing IBD serotype 2 into backyard poultry flocks would be considerably less than the risk of introduction of IBD serotype 1 in chicken meat products (see Appendix 1). We can be 95% confident that with a 10% market penetration the risk is less than four disease introductions per thousand years. For 50% market penetration we can be 95% confident that the risk is less than two disease introductions per hundred years.

*Key assumptions*

- The prevalence of IBD2 infection in infected turkey flocks is likely to be close to 100%.
- Households which keep backyard chickens are just as likely to purchase turkey meat products as other households.
- Imported turkey meat products would be distributed uniformly over New Zealand.

*Caveats*

- The assessment is based on the assumption that IBD serotype 2 virus is as widely distributed throughout the tissues as the highly pathogenic CS88 strain of serotype 1. However, this assumption may be unwarranted as experience in New Zealand indicates that the unrestricted sale of chickens from flocks infected with IBD serotype 1 of low pathogenicity has not resulted in spread of the virus.

*Note I: Probability that turkey muscle tissue will be carrying infection at slaughter, R2*

The probability that, at the time of slaughter, muscle tissue of a turkey from an infected flock will be carrying IBD2 virus was modelled from the following data :

- Age at slaughter.

  Turkeys are slaughtered at 12 weeks of age. (See Appendix 5 for details of the production and processing.)

- Age at which turkeys become infected.

  Although turkeys, like chickens, mainly become infected after 1 week of age, after maternal antibody has waned,[6, 7] for the purposes of this model it is assumed that turkeys may become infected as early as 1 day of age. Given that VN antibody is detectable 9-12 days post-infection,[8, 9, 10] the surveys of turkeys in England[11] suggest that most birds become infected between the age of 4 and 7 weeks, but it recognised that turkeys may be infected at any age up to slaughter at 12 weeks.

- Duration of infectivity in turkey muscle tissue.

  A study commissioned in 1997 by the Chief Veterinary Officer of Australia showed that IBD virus (strain CS88) was recoverable from a range of tissues, including muscle, bone marrow, bursa and liver/kidney of infected chickens[12]. This study, together with further experimental work commissioned by MAF,[13] leads to the conclusion that IBD virus is recoverable from muscle tissue for only 2 - 6 days post-infection.

A Monte Carlo model was constructed using the following values/distributions :

| | |
|---|---|
| A1, age of turkey at slaughter, in days | 84 |
| A2, age of turkey at infection, in days | PERT (1, Uniform (28,49), 84) |
| D, duration of infectivity in muscle tissue, in days | Uniform (2, 6) |

At each of 20,000 iterations the model asked the question "Is the turkey muscle tissue infected at time of slaughter?" It used the algorithm;

*If A1 is greater than A2, use (if A1 is less than A2+D, use 1, else use 0), else use 0.*

An answer of 1 meant that the turkey meat was infected, an answer of 0 meant that it was not infected. That is, an answer of 1 was returned on each occasion when the time of slaughter was ***after*** the tissue became infected but ***before*** virus was eliminated.

The mean output of the model provided the probability that the turkey meat was infected at the time of slaughter. Since the simulation is an approximation only, the confidence interval

for the true probability was calculated using;

Beta(k x mean+1, k x (1-mean)+1)

where k is the number of iterations (20,000) and mean is the mean output of the model.

The model output for turkey meat, and the resulting beta distribution used for modelling R2 in the main model were :

| | |
|---|---|
| Number of iterations, k | 20000 |
| Mean output of the sub-model | 0.000950 |
| Beta(k x mean+1, k x (1-mean)+1) | Beta (20, 19982) |

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**Appendix 5: Extract from: overseas travel report, 25 September to 18 October 1996, Paris and London. Stuart C MacDiarmid, National Manager (Agricultural Security)**

## 9. Bernard Matthews turkey meat preparations{tc \l1 "**9. Bernard Matthews turkey meat preparations}**

Following the meeting of the OIE Ad hoc Group on Transmissible Spongiform Encephalopathies I returned to the United Kingdom to inspect a turkey slaughterhouse and processing factory belonging to Bernard Matthews Foods Ltd.

Bernard Matthews Foods Ltd is seeking access to New Zealand for a range of turkey meat preparations. [45] However, their application has been vigorously opposed by the Poultry Industry Association of New Zealand which argues that Bernard Matthews turkey products constitute an unacceptable disease risk to New Zealand consumers, poultry flocks and native birds. At the instruction of the Chief Veterinary Officer I visited the plant from which Bernard Matthews Foods Ltd wishes to export product to New Zealand. The aim of my visit was to study processes so as to be able to advise the Chief Veterinary Officer on aspects of the claims made by the Poultry Industry Association of New Zealand.

In preparation for my visit, the Chief Veterinary Officer had written to his British counterpart asking that arrangements be made for me to visit the Bernard Matthews plant to study the following process;

- Live bird pickup/transport
- Holding
- Slaughter
- Chilling/aging
- Boning
- Preparation
- Forming/slicing
- Coating
- Flash-frying
- Freezing
- Storage/transport

On the afternoon of Sunday 13 October, I met Mr John Harris, Meat Hygiene Adviser with the Ministry of Agriculture, Fisheries and Food. Mr Harris is based at Tolworth and is responsible for auditing the activities of the Meat Hygiene Service[46] in the region in which the particular Bernard Matthews plant is located. Mr Harris collected me from my hotel for the three hour drive to Holton near Halesworth in Suffolk where the Bernard Matthews plant is located. We stayed overnight nearby so as to be at the plant by 8:00am.

[45] "Meat preparations" is the definition used in EC Directive 94/65/EC of 14 December 1994 for products manufactured from meat with additional ingredients. In the case of the Bernard Matthews products these additional ingredients are those in the batter in which several are coated.

[46] The Meat Hygiene Service (MHS) veterinarian is called the Official Veterinary Surgeon (OVS).

## 9.1 Bernard Matthews Food Ltd, Holton.{tc \l2 "**9.1 Bernard Matthews Food Ltd, Holton.}**

On Monday 14 October, in the company of Mr John Harris of the Meat Hygiene Service of MAFF, I visited the Bernard Matthews Foods Ltd turkey slaughterhouse and processing plant at Holton, Suffolk. The visit began with a meeting with Mr E. (Ted) Wright, Director of Special Projects for Bernard Matthews Foods Ltd, Mr Tony Blowers, Senior Production Manager and Mr Ken Thomas the Meat Hygiene Service Official Veterinary Surgeon (OVS) in charge of inspection at the plant.

### 9.1.1 Background{tc \l3 "**9.1.1 Background}**

Mr Wright provided background information about the company's operations. He described Bernard Matthews Foods Ltd's involvement in New Zealand which began in 1984 when Advanced Foods of New Zealand was set up to produce Bernard Matthews lamb roasts for sale in the United Kingdom. The plant at Waipukurau became the largest deboning plant in New Zealand and in 1994 Bernard Matthews purchased Advanced Foods. The plant now exports lamb products (roasts and racks) to the UK, US, Japan, France, Germany etc. Bernard Matthews Foods Ltd is a major exporter of finished lamb products from New Zealand, processing around 1.2 million lambs annually.

Bernard Matthews himself has been awarded the Queen's Service Medal for services to the New Zealand meat industry.

The company also purchases New Zealand fish for manufacture into products.

Mr Wright explained that he was making the point that Bernard Matthews Foods Ltd is a company with a significant involvement in New Zealand. The company wants access to New Zealand for its turkey preparations as part of its business in this country.

### 9.1.2 Bernard Matthews turkey business{tc \l3 "**9.1.2 Bernard Matthews turkey business}**

Bernard Matthews Foods Ltd produces 16 million turkeys per year in the United Kingdom, with a £400 million turnover. There are never fewer than 6 million turkeys in the company's flocks at any one time.

- Bernard Matthews purchases 1 day old turkey poults from British United Turkeys.
- The company's breeder/grower farms raise the poults to 28 weeks.
- A selection is made from these for shape. Those that fail to meet the desirable criteria for shape are processed for meat.
- 4 to 5 thousand hens and 200 stags are sent to breeder farms. These lay through to 52 weeks. In that time they lay 400,000 eggs, with a hatchability of between 85% and 95%.

- At the end of 52 weeks these breeding birds are sold to another processing company. They are not slaughtered by Bernard Matthews Foods Ltd. That is, the company's culled for age breeding stock is not processed into the products which Bernard Matthews wishes to export to New Zealand.

- The eggs are sent to two hatcheries.

- All hens are slaughtered by 12 weeks. 4 to 5 million are sold per year as whole birds within the UK.

- Second grade birds are deboned.

- Some stags are sold whole at 14 to 16 weeks at weights up to 12 kg. Some stags are grown to 22 weeks (15 to 17 kg).

- The slaughterhouse at Holton slaughters 18,000 turkeys per day, 5 days per week.

- The Holton slaughterhouse is UK registered (UK 5049), European Union registered (EWP 13) and USDA approved.

- The plant at Holton produces a range of fresh products and meat preparations. It is inspected regularly by the USDA.

- The plant is also licensed by the authorities in South Africa and Canada.

- More importantly, the Bernard Matthews Foods Ltd plant at Holton is approved by the major British supermarket chains.

- The Bernard Matthews Foods Ltd turkey slaughterhouse at Holton has an on-site MHS presence; The OVS and MHS inspectors. The plant is audited by MAFF.

### 9.1.3 The turkey farms

On the larger units the company has up to 32 sheds each with 6,000 birds. However, the current policy is to reduce the size of units to avoid disease risks. Mr Wright emphasised that the company is *not* experiencing disease problems in its flocks. The measures are reduce *potential* risks.

When birds are shipped out at 22 weeks the sheds are all cleaned. Approximately 250 tonnes of litter is removed from each shed. About 50% of this litter goes to power stations to be burned as fuel. The rest is spread on farm land as manure.

The sheds are cleaned, washed, sealed and fumigated before being re-littered and restocked.

Even on the biggest farms there is *always* a clean break between destocking and restocking. This

break is at least two days. To reduce their own disease risks the company takes steps to insure that the trucks carrying the poults for restocking do not pass the trucks carrying birds to slaughter. That is, complete separation is insured between poults coming in and finished birds going to slaughter.

In the United Kingdom, the company slaughters around 60,000 turkeys per day. The Holton plant is only one of those operated by Bernard Matthews Foods Ltd.

### 9.1.4 Health testing, certification requirements{tc \l3 "9.1.4 Health testing, certification requirements}

The health certification with which the company anticipates having to comply is that designed by the New Zealand Ministry of Agriculture.

The New Zealand-prescribed serological testing for infectious bursal disease (IBD) requires a test at an early age (8 weeks) and a second test at 20 weeks. Testing is carried out on birds in two sheds per farm.

Bernard Matthews Foods Ltd has been testing the flocks from which product destined for New Zealand would be sourced. They have already built up a history of negative test results. Testing is carried out at the Central Veterinary Laboratory, Weybridge and copies of all test results are sent to MAFF at Tolworth. I examined test reports at the Holton plant and at Tolworth.

### 9.1.5 Batch isolation{tc \l3 "9.1.5 Batch isolation}

Submissions by the Poultry Industry Association of New Zealand have claimed that Bernard Matthews Foods Ltd would be unwilling or unable to slaughter and process turkeys in separate batches for the New Zealand market.

However, the company already does this for a number of markets. The USDA require that product for entry to the US must be processed separately. Although not a major market (four containers of turkey products in 1995), the company is able and willing to comply.

The turkeys and resulting product are maintained in isolated, bar-coded lots to avoid cross-contamination between batches. Bar-coded lots are tracked and controlled by a computer system.

For the US market, the company slaughters first thing each morning (as they were doing on the morning of my visit) and the product is kept separate throughout manufacture, storage and despatch.

The same procedures apply for turkey products and preparations destined for Sweden and Finland. Again, a batch of turkeys is killed first thing in the morning and is then “sealed” and controlled separately throughout processing.

To meet a New Zealand requirement that product be from specifically tested batches of turkeys, slaughtered first thing each day and then kept separate throughout the process, Bernard Matthews Foods Ltd would *not* be introducing new practices. The company would merely be doing what it

already does for number of other small but sensitive markets.

### 9.1.6 Public health concerns{tc \l3 "9.1.6 Public health concerns}

The Poultry Industry Association of New Zealand has expressed concerns to the New Zealand Ministry of Health that Bernard Matthews Foods Ltd's turkey meat preparations would be a public health risk. Salmonellosis has been raised as a specific concern.

The company exports turkey meat preparations to Sweden and Finland, both countries which have salmonellosis eradication programs operating. Both countries have a zero tolerance for *Salmonellae* in turkey meat preparations and Bernard Matthews Foods Ltd must comply with Swedish and Finnish *Salmonella* requirements to maintain access to those markets.

The British supermarket chains which retail the company's turkey meat preparations also have stringent requirements to protect the consumer.

Bernard Matthews Foods Ltd have contracts to provide their turkey meat preparations to *all* British schools, *all* British hospitals and *all* British prisons. As Mr Wright pointed out, 5 million people a day in the UK eat the company's turkey meat preparations, and that is 5 million potential customer complaints if the products are not safe.

### 9.1.7 The inspection hierarchy{tc \l3 "9.1.7 The inspection hierarchy}

Inspection at the Bernard Matthews turkey slaughterhouse and processing plant at Holton is under the direct control and supervision of the OVS of the MHS.

Under the veterinarian there are three Poultry Meat Inspectors employed by the MHS.

Also under the veterinarian's control during meat inspection are nine Poultry Inspection Assistants employed by the company.

### 9.1.8 Inspection of the slaughter plant{tc \l3 "9.1.8 Inspection of the slaughter plant}

After the meeting at which the issues were discussed, I inspected the plant in the company of Mr Wright, Mr Thomas (OVS) and Mr Harris (MAFF veterinarian).

- Turkeys arrive by truck. No journey greater than 60 miles. Birds in modular crates designed for ease of cleaning. Crates and trucks washed with high-power hoses once emptied of birds.

- Lairage where birds rested prior to slaughter.

- Hang-on bay where birds put onto the chain.

- Electrical stunning, cut both sides of throat, bleed out in enclosed corridor.

- Feet scraped clean, tail feathers pulled *before* scalding to reduce faecal contamination of scalding bath. The birds pass through a series of three separate scalding tanks at 56° C.

The flow rate through the scalding tanks is around 8$m^3$/hour. The US standard is > 1 US gallon/bird. This works out at 30 birds/hour through a tank where water flows at 8.2$m^3$/hour. Scald time is 4 minutes.

- The company kills for the US market first thing in the day and slows the chain down to meet US requirements.

- Plucking. I was struck how well-plucked the birds were.

- *Inspection*. The first inspection follows plucking. There are two inspectors at this point, one MHS inspector and one company PIA. These inspectors are changed every 15 minutes to keep them alert.

- Feet removed.

- Slit neck skin from back.

- Vent cutter, suction to empty cloaca.

[Spray washing along the chain from this point]

- Heads off.

- Evisceration.

- *Inspection*. Two company PIAs doing internal inspection, including inserting hand. The PIAs under the direct supervision of a MHS inspector. Inspectors changed every 15 minutes.

- Recording all condemnations and reason per load. (A "load" is approximately 800 birds). At this point there is a detain rail where birds are either condemned or salvaged.

- Livers harvested.

- Crops out.

[Several trimmers and people cleaning all along the chain at this point. Very labour intensive].

- Necks off.

- Suction device to remove last traces of lungs, etc.

- Inside and outside washer. Flow rate recorded and monitored.

- *Inspection*. More trimming and inspection by a PIA with MHS inspector in the background.

- Final company check of inside of carcass.

- Quality control. (Introduced for the US market). Records feathers, hairs, viscera, contamination, blemishes etc. Each half hour 20 birds are selected at random for this check. They are scored to an American system and every batch must pass to be eligible for the UK market. This is a very detailed inspection.

- *Spin chillers*. These are in three banks in parallel. Each bank comprises three tanks in series. Birds are chilled in water at around 1° C. Contra-flow.

- Birds pass through the spin chillers at the rate of 20 birds/minute (1,200 per hour). So, each *bank* is handling 400 birds/hour. Birds enter at 37° C, leave with a core temperature of 20° C. They are then further cooled by storage overnight in slush ice in steel boxes to get the temperatures down to 4° C.

- Chlorine levels in the spin chillers is monitored. The maximum permissible level is 20 ppm but levels are usually between 10 and 15 ppm.

- Water enters the spin chillers at the output end at 1° C. Birds are in the spin chillers for 16 minutes. The water flows through the spin chillers at a rate of between 7 and 8 litres/bird for the chillers as a whole. In the last tank the rate is 3 to 4 litres/bird, with a minimum of 2 litres/bird.

- Total volume of the spin chillers is 7.5 thousand gallons per tank (six tanks).

- To summarise;
  - 9 chillers in total
    - 3 rows of 3 chillers
    - $99m^3$ per row (total volume)
    - Total volume of all chillers is $297m^3$
    - Chilled water usage per week is $2000m^3$
    - 5 days killing per week.

- Batches, of birds held in steel boxes, labelled and bar coded.

- Tank washing.

- Birds into ice tanks by weight. Held overnight. Temperature in room around 2° C.

- Into the cutting room.

[Batches are kept apart. For US market, slaughtered first of the day between 6.00 and 8.00. Minimal handling to increase shelf life. Computer sorts by weight. Also pack for other brands, eg. Sainsbury's].

- Dispatch

- ∃ Trays and bins washed
- ∃ Blast freezer
- ∃ Mechanically recovered meat
- ∃ Sausages etc. This may be used fresh or frozen.
- ∃ Dry goods store.
  - ∃ Rodent bait stations
  - ∃ Major effort to exclude birds.
- ∃ Cold store, 0.26$^{o}$ C.

### 9.1.9 Inspection of the manufacturing plant{tc \l3 "**9.1.9 Inspection of the manufacturing plant}**

For an inspection of the manufacturing plant, on an adjacent site (EWP 138), we were joined by Mr Nick Gray, the Production Manager.

Meat from the previous plant (the slaughter plant) is manufactured here into turkey meat preparations such as "Crispy Turkey Burgers" and "Dinosaurs". These are turkey meat which is minced, shaped, battered, crumbed, flash fried and frozen.

The turkey meat preparations are made from fresh and/or frozen meat. The meat is at a temperature of around 3 to 4$^{o}$ C during this processing. The different products are reconstituted from a mixture of white meat, red drumstick meat and skin combined to give the right colour and consistency. The final product is raw inside.

Each pallet of skin, white meat, drumstick meat etc is bar coded to enable tracing back to the farm of origin. Separate lot numbers are allocated for each day's kill and are either internal (company) codes or codes for external agencies (such as USDA or New Zealand MAF). The company is thus able to trace the different raw materials back to the farms of origin.

So, Bernard Matthews Foods Ltd kills turkeys to produce raw materials specifically for turkey meat preparations destined for specific markets (such as the US, Sweden, Finland or New Zealand).

The standard for weight is within 1% of stated weight of final product.

### 9.1.10 Final meeting at the Holton plant{tc \l3 "**9.1.10 Final meeting at the Holton plant}**

Following the inspection tour we met to discuss matters arising.

Mr Blowers briefly outlined the company's application of HACCP during manufacture.

It was claimed that viraemic birds would probably not pass the inspections, being condemned as "septicaemic".

I examined a large number of Central Veterinary Laboratory reports give results of serological testing for IBD since March 1996 when the company started testing birds from two sheds each on a number of farms, in anticipation of gaining access to the New Zealand market. Birds were being tested at 8 weeks and 20 weeks.

If processing for the New Zealand market the company would kill two or three lorry loads of birds first thing in the morning. This batch would be run through a single bank of spin chillers (as is currently done for the small US market). There would be what was described as a "positive cut-off", at which point the workers would stop for tea so that there was a gap between birds destined for the special market and those intended for the UK. This special slaughtering would be carried out between 6.00 and 8.00.

Birds killed for the special market (say New Zealand) would be specifically labelled and the OVS would thus able to certify that raw materials and products are kept separate.

For export to New Zealand the OVS would become a Licensed Veterinary Inspector (LVI) of MAFF (versus an OVS of MHS for British and EU requirements).

I discussed the questioned of catching and transporting turkeys from the farms to the plant. The catchers rarely go to more than one farm per day. They must shower and change clothes if they do.

This is company policy to reduce the company's disease risk.

The company knows which two sheds per farm will be producing for the New Zealand market. They are already serotesting these. The OVS will know the sources producing for New Zealand and will thus be able to certify that New Zealand requirements are being met. The OVS has the testing results, as does MAFF Tolworth (John Harris).

New Zealand's testing requirements must be clarified. In early negotiations it was proposed that testing be done at 8 and 20 weeks for birds destined for manufacture. However, when it was proposed that access might be sought for small, whole birds, slaughtered at a younger age, a 6 weeks test was proposed. This 6 week test has now been incorporated (by MAF?) into the proposed certification, meaning that birds are required to be tested three times, at 6, 8 and 20 weeks.

It makes no sense to require the two early tests. One at 6 *or* 8 weeks should be sufficient, followed by one at 20 weeks.

Finally, it was agreed that Bernard Matthews Foods Ltd is seeking access for turkey meat preparations. That is, raw products reconstituted from turkey meat, some of which might be battered, crumbed and flash-fired, but would still be raw inside.

## 10. MAFF, Tolworth{tc \l1 "**10. MAFF, Tolworth}**

The following day, Tuesday 15 October, I visited the British Ministry of Agriculture, Fisheries and Food at Tolworth. I met with Mr Jim Scudamore (Assistant Chief Veterinary Officer), David Pritchard, Allen Wellwood and John Harris. The main purpose of the meeting was to recapitulate the visit to the Holton plant of Bernard Matthew's Foods Ltd, but other import/export matters were discussed.

### 10.1 Bernard Matthews Foods Ltd turkey preparations{tc \l2 "**10.1 Bernard Matthews Foods Ltd turkey preparations}**

Points raised by Mr Scudamore;

- MAFF does not know the New Zealand IBD situation.
- New Zealand MAF does not know officially the IBD status of New Zealand, having had no involvement with IBD surveillance or control since the disease was confirmed as endemic.
- There is no official IBD control program in New Zealand.
- Questioned the IBD testing carried out in New Zealand. Not under official supervision.
- Agar gel immunodiffusion test would not pick up IBD serotype II if present.
- The presence of IBD serotype II would not interfere with any test program based on AGID testing.
- IBD serotype II has not been recorded as causing disease.
- No survey for IBD serotype II has been done in the UK since 1985. Such surveys would be expensive and of little value.
- What evidence is there that IBD serotype II is absent from New Zealand?

MAFF will be sending a technical response to the New Zealand Chief Veterinary Officer addressing points of concern raised by the CVO on behalf of the Poultry Industry Association of New Zealand.

**Appendix 6: Analysis of the probability of cross-contamination of turkey carcasses with IBD during processing at a Bernard Matthews Foods Ltd. processing plant.**

*A Report by : **David Vose, DVRAS, 16 Mill Street, Wincanton, Somerset BA9 9AP, United Kingdom***

## 1. Introduction

This report is the response of a verbal brief and subsequent communications from Stuart MacDiarmid of New Zealand's Ministry of Agriculture, Regulatory Authority. The brief was to assess the probability of cross-contamination of Infectious Bursal Disease (IBD) in turkeys during processing. The purpose of this work is to provide an input to a larger model looking at the probability of IBD entering New Zealand through the importation of Bernard Matthews Foods Ltd (BMFL) turkey products.

## 2. History of this analysis

The initial brief requested that I look at the probability of cross-contamination during chilling in the spin-chillers. The cross-contamination I was asked to consider was from infected turkeys of one batch contaminating another, tested negative batch bound for New Zealand. It is easy to see why the spin chillers might appear to be a point in the processing where contamination would be most likely to occur: turkey carcasses are swirled around in a large mass of water for some 20 minutes. However, after some initial communication with Mr. Ted Wright at BMFL, it became apparent that the spin chillers, like all other equipment on the plant, is drained, cleaned and sanitised at the end of each day *by law*.

I visited the BMFL plant on 23 January 1997 and was given a very thorough tour of the facilities by Mr. Ted Wright (Special Projects Director (Processing)), reviewed the methods of processing and talked to some of the staff. At all times, I was extremely impressed with the openness shown by the staff, the attention to detail and the safety and quality aspects of the way this plant is run. During this visit, I requested various pieces of information from BMFL that might help me with an analysis either directly, or indirectly through comparisons. BMFL has provided all the information I requested. I also wrote to, spoke and met with Mr. Peter Wyeth at Central Veterinary Laboratory (CVL) in the UK. CVL have run numerous tests to check for IBD in BMFL flocks. Mr. Wyeth also provided me with information and estimates where possible.

## 3. Synopsis of the problem

*Cross-contamination* of IBD between batches due to processing at a slaughterhouse requires the following steps in order to occur:

1. IBD infected turkeys are processed at the plant;

2. Infective material adheres to the machinery and/or work surfaces during the slaughtering

and processing;

3. IBD virus survives any cleaning and sanitising at the plant before the next, uninfected batch is processed: Cleaning and sanitising occurs both during the processing of the birds and at the end of a batch;

4. Infective material is transferred to one or more carcasses in the second, uninfected batch during processing;

5. IBD virus survives the remainder of the processing that the carcass it adheres to has to go through, including storage.

In my discussions with Mr. Ted Wright, he has assured me that BMFL would adhere to the following operational procedure when processing turkeys bound for New Zealand:

1. Turkey batches bound for New Zealand would be tested for IBD and only sent to slaughter if they tested negative;

2. Batches of New Zealand bound turkeys would be the first to be processed in a day's operation;

3. All machinery would be cleaned and sanitised at the end of the previous day's processing (see below). It is a legal requirement that this cleaning and sanitising take place at least once a day and it is operationally more efficient that this should occur at the end of the day. BMFL is not, therefore, doing New Zealand any particular favours, neither is it incurring BMFL any additional costs;

4. Spin chillers are drained at the end of each day;

5. Throughout the processing and overnight chilling, the New Zealand bound turkey carcasses and products would be kept physically removed from all other products. The Holton plant is highly mechanised and computer controlled. It is the most modern in Europe and it would appear that such separation would cause them no operational difficulties whatsoever.

In this report, "cross-contamination" will at all times refer to the movement of IBD virus (IBDV) from an infected batch of turkeys processed at the Holton plant to another, uninfected batch processed later at the same plant, as a result of transfer of IBD infected material via the plant's machinery.

## 4. Qualitative analysis of the risk

*Cleaning and sanitising*

BMFL use the following procedures in the cleaning and sanitising of their facility:

1. People are employed to walk around with hoses and remove all large items of debris, as well as general waste, whilst the plant is being operated.

2. At the same time, equipment is sprayed continuously by strategically located nozzles with 25ppm chlorinated water. Chlorines have been shown to be the most active disinfectants against IBDV (Meulemans et al 1982).

3. At the end of a batch run, the equipment is hosed down.

4. At the end of the day, all equipment is drained. It is then thoroughly hosed down and sprayed with a degreasing foam (Shuremousse). This is left on the equipment for several hours, then washed off (see Appendix A for technical information on this foam). Although Shuremousse has no known virucidal activity against IBDV, the alcohol component is likely, at least, to be effective (Peter Wyeth, pers. Comm. - dated 26 February 1997, attached). I inspected the machinery and was impressed at how free from grease the surfaces were.

5. The equipment is then sprayed with a disinfectant (Divosan QC). This is based on a quaternary ammonium compound and will have no effect on IBDV (Meulemans et al 1982).

6. Inspectors from the UK Meat Hygiene Service check to see if the equipment is clean and sanitised before commencing the next run.

*Possible points of contamination*

Excrement on the feet could contain virus. However, feet are scrubbed clean and scalded at the beginning of the processing. Tail feathers are pulled out with extremely high efficiency and the carcass scalded and other feathers removed. The machines effecting the two types of feather removal could harbour excrement and thus pass on infection from one batch to the next. However, the birds are washed a short time after feather removal with chlorinated water. This washing would, in all probability, remove any excrement that had just been transferred, via the feather pluckers, to an uninfected carcass thus minimising this risk.

I noticed at an inspection point just after the throat slitting that each bird was hitting a waste bin with their heads as they went past. Whilst this is theoretically a mutual point of contact between birds of different batches and therefore a potential point of cross-contamination, the heads and necks were removed soon after hitting the waste bins and there seems to be no logical route by which any infection could transfer to the meat. The bins were also regularly changed and washed, so cross-contamination between batches seems very unlikely.

Other potential points of cross-contamination, as I observed the process, were: the conveyor belt used after the birds' feet are removed; the neck slitter; the vent cutter and the spin chillers. The neck slitter and vent cutter are both obvious points where contamination could occur between batches. The neck, being at the lowest point in terms of gravity, may accumulate virus particles from run-off from washing. The vent cutters are boring directly into the areas that would hold the greatest concentration of infection, should a bird be infected. Both pieces of equipment are sprayed liberally with chlorinated water.

The spin chillers also present a possible path for the cross-contamination between batches. Although a large volume of chlorinated water passes through the spin chillers, the water in the baths is quite bloodied, suggesting that significant concentrations of blood and other material from the carcasses may accumulate in the baths. However, as noted before, this batch is drained at the end of the day so the likelihood of cross-contamination *between batches* is related only to whether the baths are free of IBDV after cleaning and sanitising.

## 5. Quantitative analysis of the risk

It has proven very difficult to produce any thorough quantitative analysis of the probability of the existence of viable IBD virus particles on New Zealand bound turkey products. This is because of the very sparse numerical data available that would be relevant to such a quantitative analysis. For example, nobody I have spoken to has a good quantitative feel for the effectiveness of chlorinated water on reducing the survivability of IBD virus in situ, only that it is the most effective. There is little or no observational or experimental data available on the general survivability of IBD virus, its concentration in tissue, the effect of the alcohol in the sanitising foam, the amount of turkey by-product remaining on the equipment after cleaning and sanitising, etc. I have attempted to maximise use of the available information, but this inevitably will lead to wide confidence intervals unless more quantitative information becomes available. It has also proven impossible to estimate the degree of cross-contamination between batches by making comparisons with other disease agents (for example *Salmonella*) where data has been collected because of the way that this data is collated at the Holton plant.

The probability $P_{XI}$ that a New Zealand bound bird is cross-infected from a previous batch can be estimated by the following formula, assuming that no birds in the previous batches are tested for IBD:

$$P_{XI} = \sum_{i=1}^{\infty} p.q^i \text{ which reduces to } P_{XI} = \frac{pq}{(1-q)}$$

where $p$ is the flock prevalence and $q$ is the probability of cross-contamination in the plant from the batch processed immediately prior to the batch in question. This formula assumes that $q$ is essentially the probability that the virus will remain in situ and survive the cleaning and disinfecting at the plant from one batch to the next. Thus, $q^2$ is the probability it would survive two batches worth of cleaning, etc. Thus, the above formula for $P_{XI}$ calculates the probability that a batch of turkeys that was processed $i$ batches ago is infected (probability $p$) and that the virus survives the $i$ cleaning and disinfecting routines in order to infect the batch in question. This probability is then summed for all values of $i$ from 1 to infinity. The formula is a slight approximation in that it only works for low values of $q$, but the error is minimal.

Mr. Wyeth notes (pers. Comm. dated 26 February 1997, attached) that of 32 flocks tested for IBDV type 1, none were infected. This leads us to a *maximum* estimate of prevalence of Beta(1,33). Mr. Wyeth's maximum value for prevalence (0.5%) is lower than one could assume from 32 negative tests and is probably reflective of the extra information he is able to provide in terms of the epidemiology of the virus. If we conservatively take the maxima of Mr. Wyeth's estimates for $p$ and $q$ (0.5% and 1% respectively), $P_{XI}$ works out to be -4.3log10. Using Uniform(0,0.5%) and Uniform(0,1%) for $p$ and $q$ respectively gives a mean of -4.9log10. Uniform distributions can be justified in this situation as simply applying the principle of maximum entropy47.

It is worth noting that where $q$ is small in the above equation, the equation further reduces to:

$$P_{XI} \approx pq$$

i.e., it is only the batch processed immediately prior to the New Zealand bound batch that presents any (relatively) significant risk.

## 6. Risk management

The risk of cross-contamination between batches is taken very seriously by BMFL in the operation of their Holton plant. Chlorinated water is used liberally to spray the equipment and carcasses during the plant's operation. Meulemans et al (1982) demonstrated that, in their experiments, chlorine based disinfectants are the most effective in the reduction of IBDV. Carcasses are stored in bins of chilled, chlorinated water overnight before deboning. Degreasing foam is used to clean all equipment each night and New Zealand bound batches would be the first to run off the freshly cleaned production line. The pieces of processing equipment that have raised most concern are the spin chillers. However, these have smooth, easily cleaned surfaces, they are filled with chlorinated water during operation, and they are drained each evening and subjected to the same thorough cleaning with degreasing foam and pressure hoses as all the other equipment.

| ***Month*** | ***Fresh Portions*** | | ***Whole birds*** | |
|---|---|---|---|---|
| | Total tested | % positive | Total tested | % positive |
| June | 83 | 1 | 20 | 15 |
| July | 136 | 2 | 23 | 9 |
| Aug | 133 | 5 | 21 | 5 |
| Sept | 94 | 0 | 15 | 0 |
| Oct | 125 | 0 | 22 | 0 |
| Nov | 118 | 3 | 19 | 11 |

47 The principle of maximum entropy is to be maximally non-committal in assigning probability distributions to a variable given the available evidence. So, for example, knowing only that $p$ is less than 0.5%, the Uniform(0,0.5%) distribution says that any value for $p$ is possible between 0 and 0.5%, but we cannot be more precise than that and must therefore allow each value within that range to be as likely as any other: i.e. we are assigning the maximum uncertainty (and therefore producing the most conservative estimate) given our knowledge.

Table 1: Salmonella incidence (1996) of turkeys processed at Holton plant

A good indication of the degree of hygiene control that is obtained *during* processing (i.e. not even including the night cleaning) can be determined from looking at the incidence of *Salmonella* in the whole bird pre-processing and the incidence in the resultant fresh portions. Table 1 shows the data provided to me for 1996 by BMFL. It is unfortunate that the information is not available on a batch by batch basis as this might have helped estimate cross-contamination between batches. Nonetheless, it shows that, far from *Salmonella* becoming more prevalent in the final product, it actually decreases in prevalence from that observed in the whole birds. One could justifiably conclude that the constant spraying during processing with, and subsequent chilling in, chlorinated water is reducing the prevalence of *Salmonella*. . Given the efficiency of chlorines in controlling IBDV, one might also expect that there would be a pattern of reduction of prevalence for IBDV during the processing, should it initially be present, similar to that observed with *Salmonella*.

*Possible risk reduction strategy*

It is difficult to imagine any more practical steps that can be taken in the operation of the slaughter house to further reduce the risk of cross-contamination between batches. However, one strategy that may be worth considering is the testing of batches that are processed immediately prior to the batch bound for New Zealand. Although the sensitivity of the agar gel diffusion test in detecting IBDV is unknown, Mr. Peter Wyeth estimates it to be around 99% (telephone conversation). For a batch of turkeys processed prior to a New Zealand bound batch to cross-contaminate the New Zealand bound batch, it must: a) be infected (with probability = flock prevalence $p$); b) escape detection with probability approximately equal to $(1-xs)^n$, where $x$ is the within flock prevalence, $s$ is the agar gel diffusion test sensitivity and $n$ is the number of birds selected for testing; and c) then cross-contaminate the next batch with probability $q$.

IBD is apparently extremely virulent and, if one bird within a flock became infected, almost all would also become infected within five days (Mr. Wyeth, telephone conversation). So $x$ in our equation above would be close to unity. Conservatively setting the test sensitivity $s$ to 80% and varying $x$ and $n$ in the equation above, one arrives at probabilities of an infected flock escaping detection as shown in Table 2:

| x | 80% | 90% | 99% |
|---|---|---|---|
| n | Probability of escaping detection (log10) | | |
| 1 | -0.4 | -0.6 | -0.7 |
| 2 | -0.9 | -1.1 | -1.4 |
| 3 | -1.3 | -1.7 | -2.0 |
| 4 | -1.8 | -2.2 | -2.7 |
| 5 | -2.2 | -2.8 | -3.4 |
| 6 | -2.7 | -3.3 | -4.1 |
| 7 | -3.1 | -3.9 | -4.8 |
| 8 | -3.5 | -4.4 | -5.5 |
| 9 | -4.0 | -5.0 | -6.1 |
| 10 | -4.4 | -5.5 | -6.8 |

**Table 2**: Probability of an agar gel test failing to detect an infected flock with prevalence $x$

when *n* birds are selected for testing, assuming test sensitivity *s* of 80%.

Table 2 demonstrates that, in applying this extra testing restriction, one would expect to be able to decrease the risk of cross-contamination by several orders of magnitude.

## 7. Conclusion

It is estimated that there is at most approximately a 1:20,000 probability (i.e. -4.3log10 or $5x10^{-5}$) that a batch will become cross-contaminated by a previous batch. This probability could possibly be further reduced by certain risk reduction measures. For example, the introduction of testing for batches slaughtered immediately prior to the New Zealand bound batch of turkeys could easily reduce this probability by two to four orders of magnitude.

*Postscript comment*

It is evident that every precaution has been taken to prevent the cross-contamination between batches at the BMFL Holton plant and that these precautions are strongly enforced. Intuitively, one can deduce that the probability of cross-contamination must be very small. Given the modern nature of the Holton plant (it is apparently the most modern in Europe) and the extremely efficient and methodical way it is run, there may be some profit in making comparisons with the record of the ability or otherwise of other plants to prevent the cross-contamination between batches for IBD and other, similar virus agents. One might reasonably assume that the Holton plant will perform at least as well as any other plant of its type, certainly as well as or better than any other plant of similar function in Europe.

## 8. References

Meulemans G, Halen P (1982) Efficacy of some disinfectants against infectious bursal disease virus and avian reovirus. *Veterinary Record* 111: 412-413.

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## Appendix 7: The persistence of IBD in infected SPF chickens

**RETYPED DOCUMENT**

### PERSISTENCE OF IBD IN INFECTED SPF CHICKENS

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In a previous work concerning the persistence of IBD virus following infection with a virulent strain of the virus found that infectious virus was present in pools of tissues (muscle, liver, spleen, kidney, lung and bursa) for 4 weeks following infection. Assays of the various tissues in the laboratory in eggs and cell culture did not detect virus but 3-week-old chickens were infected.

This experiment was designed to repeat the experiments and assay the virus using 1-day-old chickens. Thirty 3-week-old SPF chickens were infected with a bursal homogenate containing 70,000 EID50 of Edgar strain IBD virus. Three of the chickens died within 4 days. Four chickens were killed at weekly intervals for 5 weeks and samples of breast muscle, liver, lung, kidney, spleen and bursa collected. One gram of tissue from each organ was placed in 9 ml of sterile PBS and homogenized. Samples were frozen at +70 C until used for assay. All chickens were bled prior to killing to determine their serologic response by virus neutralisation (VN).

The assays were performed in 1-day-old SPF chicks. The criteria for infection was seroconversion by the VN test and atrophy of the bursa at 10 days postinfection. This strain of IBD does not produce clinical disease in chicks under 2 weeks of age. Four or five chicks were inoculated with 0.1 ml of tissue homogenate per os. Since the tissue homogenate was made as a 10% suspension the undiluted homogenate contained .01 gm in a volume of .01 ml inoculum. From the 10% tissue suspension a series of ten-fold dilutions were made and inoculated into either four or five day-old chicks in isolators and held for 10 days. They were then bled, killed and examined for bursal atrophy.

RESULTS

The breast muscle tissue was never infective but liver, lung, kidney, spleen and bursa contained infectious virus at 7 days. At 14 days the liver, kidney, lung and spleen were negative and remained so for the time of the experiment. The bursal tissue remained infectious for 28 days but was negative at 35 days. This was similar to the results of the previous experiments and documents that the bursal tissue is the site of persistence of IBD virus for 4 weeks postinfection. Table 1 summarizes the results and gives the virus titers from each tissue at the various time intervals of their collection.

Table 1 - Summary of the assays of tissues collected at 1-, 2-, 3-, 4- and 5 weeks postinfection with varying 10-fold dilutions of a 10% suspension of each tissue. A volume of 0.1 ml of each dilution was inoculated into 4 or 5 1-day-old SPF chicks and the reciprocal of the highest dilution that was infective is the titer of the virus in the tissue and is expressed as the chick infective dose (CID)/gm of tissue. Rationale: A volume of 0.1 ml of a 10% suspension of tissue would contain 0.01 gm of tissue, thus infectivity from an undiluted inoculum of the 10% suspension would represent 100 CID/gm of tissue and virus quantities less than that might not be detected in such an assay.

Infectivity of tissue expressed as CID/gm

| No. days postinfection | Muscle | Liver | Lung | Kidney | Spleen | Bursa |
|---|---|---|---|---|---|---|
| 7 | $<10^2$ | $10^3$ | $10^3$ | $10^3$ | $10^3$ | $10^7$ |
| 14 | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $10^5$ |
| 21 | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $10^2$ |
| 28 | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $10^2$ |
| 35 | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ | $<10^2$ |

The nature of the virus that persists in the bursa is of interest and why the virus can not be reisolated using cell culture or embryonating eggs would indicate that virus-antibody complexes exist in the tissue and the chicken is able to dissociate these complexes or the complexes allow infection of macropage via the Fc receptors. Regardless of what the explanation the virus persists longer than was anticipated and was a very repeatable situation since the same results have been achieved in every experiment. It also points out that the chicken is the most sensitive host for IBD virus.

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## Appendix 8: The ability of serological tests to differentiation between infections caused by IBD virus serotype 1 and IBD virus serotype 2.

### Introduction

The risk analysis concludes that there is a small risk of introduction of IBD virus serotype 2 in processed turkey meat products, and that this virus could become established in chicken flocks in New Zealand, initially through the feeding of kitchen scraps to backyard chickens. However, the risk analysis notes that the impact of the introduction and establishment of IBD2 would be insignificant as the agent has not been shown to cause disease in any avian species.

Local poultry industry groups have raised concerns that the introduction of IBD2 would interfere with IBD1 testing and eradication from New Zealand chicken flocks. The agar gel precipitation test and the ELISA which are currently used in New Zealand do not differentiate between serotypes 1 and 2 of the virus. The industry argues, therefore, that if IBD2 were present in this country the virus neutralisation test would need to be introduced in order to differentiate between the two serotypes.

The position taken by the poultry industry is consistent with what is presented on page 543 of the OIE Manual of Standards.

Several opinions on this position were received in the technical review process :

**Professor Simon Shane**
**Louisiana State University**
**Baton Rouge**
**USA**

"In the USA, state and regional laboratories and the integrators implement effective programs of monitoring for IBD antibodies using group specific ELISA, applying commercial test kits with automated sample handling and processing, despite the occurrence of endemic type 2 IBDV in turkeys".

**Dr Peter Wyeth**
**OIE Reference Laboratory for IBD**
**Central Veterinary Laboratory**
**Weybridge**
**UK**

"We routinely use IDEXX Flockchek IBDV ELISA kits for testing sera for type 1 antibodies and we also frequently use and agar gel difusion test using our own antigen prepared from the 1/68 (Cheville) strain. The IDEXX kit uses strain D78 (type 1) as antigen. Neither of these tests detects antibodies to type 2 IBDV. The only test we use for detecting type 2 is the serum neutralisation test."

Dr Wyeth provided MAF with experimental results of both the AGID and the ELISA to substantiate his position.

**Professor Daryl Jackwood**
**Ohio State University**
**Wooster**
**USA**

"My experience is with the ELISA. I do not use the agar-gel precipitin assay because of its poor sensitivity. The ELISAs that are currently on the market, (IDEXX and KPL) detect antibodies to both serotype 1 and 2 viruses. The VP3 proteins of IBDV serotypes 1 and 2 have similar amino acid sequences and thus cross reacting antigens. This cross reaction was demonstrated by Ismail and Saif (Avian Diseases, 34:1002-1004, 1990). Both IDEXX and KPL have recently marketed new ELISAs for IBDV. The KPL test uses whole virus produced in chicks, and the IDEXX test uses IBDV antigens produced using genetic engineering. It is my understanding that both new assays contain VP3 antigens and thus I would expect a cross reaction with serotype 2 antibodies".

"If only VP2 protein is used as antigen in the ELISA, the assay becomes specific for serotype 1 viruses (Jackwood et al., Clinical and Diagnostic Laboratory Immunology, 3:456-463, 1996). One explanation for the discrepancy could be the sensitivity of the ELISA. The reaction of the ELISA with antibodies to serotype 1 viruses (homologous reaction) is stronger than the reaction with serotype 2 antibodies (heterologous reaction). If you set a high baseline for positive reactions it is possible that you will not detect the heterologous reaction. The problem with this is that you will also miss low titres to the homologous serotype 1 viruses."

**OIE Reference Laboratories**

The above information was sent to two further OIE reference laboratories for IBD, Professor Mo Saif at Ohio State University, and Dr. Nicolas Eteradossi in France, asking for their comments on the apparent discrepancy. They responded with the following comments:

**Professor Mo Saif**
**OIE Reference Laboratory for IBD**
**Ohio State University**
**Wooster**
**USA**

"None of the current commercial ELISA kits available in the USA will differentiate between antibodies to serotypes 1 & 2. I doubt that even the ELISA kits that use baculovirus generated VP2 would differentiate between antibodies to serotypes 1 & 2. In earlier studies in my laboratory, we were not able to differentiate between serotypes 1 & 2 using western blot assays performed with polyclonal, monoclonal, or specific polyclonal antibodies against

VP2 or VP3 (see Ture et al Avian Dis. 37:647). In addition, my colleague Dr. Jackwood indicated that his experience with the VP2 based ELISA to detect serotype 2 antibodies is limited to one sample he tested which was frozen for a long time. Please keep in mind that the VP2 from serotype 1 is reactive with serotype 2 antibodies although in general we would like to think that the VP3 is group specific and VP2 is serotype specific.

It is true that the USA commercial ELISA kits are used to routinely monitor serotype 1 antibodies, although we know that we are detecting serotypes 1 & 2 antibodies and both serotypes are widespread in commercial flocks.

The comments of the researcher from the UK are interesting but I wonder if they intentionally attempted to test serotype 2 antibodies alone with the ELISA or AGPT.

My final advice considering our state of knowledge and technology is to use the VN test. It is the only test that is proven to differentiate between serotype 1 & 2 antibodies. "

**Dr Nicolas Eteradossi**
**OIE Reference Laboratory for IBD**
**CNEVA Ploufragan**
**France**

"Two of the advices you refer to are much similar to our own experience. The first American researcher whom you quoted mentioned the use of type 1 ELISA antigens, in spite of circulation of type 2 IBDVs in turkeys.

This seems rather similar to the French epidemiological situation. Indeed, commercial ELISAs (Idexx, KPL or Guildhay) are routinely used in France for the evaluation of anti IBDV type 1 immunity, so as to adapt the vaccination schedules depending on the level of passively transmitted antibodies in broilers for example. Such a monitoring, which we implemented in conventional broiler and pullet flocks in Ploufragan, is applied with good results, although type 2 viruses are likely to be prevalent in the field (type 2 neutralizing antibodies may be found in turkey flocks).

In addition, you quote Dr Peter Wyeth, who does not detect type 2 antibodies in AGP or Idexx ELISA, and uses only the neutralization test for type 2 antibodies detection. This is also consistent with our own experience. Indeed we made some experimental intramuscular inoculation of live type 2 IBDV (strain TY89, kindly provided by Dr McNulty, Belfast) to SPF chickens from Ploufragan. The chickens seroconverted, as demonstrated by mean virus neutralizing antibody (VN) titers to the TY89 virus that reached 7.3 to 11.3 log2 after 4 to 6 weeks. None of this serum was found AGP positive with our home made IBDV AGP antigen which is derived from bursa-propagated strain Faragher 52/70 (type 1). The same sera were tested with Idexx and KPL ELISAs (type 1 antigens).

Low positive results were obtained with both ELISAs (maximum Idexx titre = 1266, maximum KPL titre = 1692, ELISA and KPL results significantly correlated), but these titres did not correlate with the VN titres, and it is not clear if the ELISA results were IBDV

specific (the chickens had received an intramuscular injection of cell propagated virus, and the sera produced some unspecific positive results with another home made non-IBDV cell-derived AGP antigen).

We also performed some serological testing in conventional turkey flocks. Although neutralizing antibodies were detected in some individual sera up to a titre of 11.3 log2, none of the VN positive sera tested positive with our type 1 AGP antigen. The same sera all proved negative with the KPL IBD-ELISA (all values were lower than the kit reference negative), however this kit includes an anti-chicken conjugate and not an anti-turkey one.

In view of these results, cross reactivity between IBDV serotypes seems to be low in the AGP test and ELISA that we have used so far. As does Dr P. Wyeth, we hence rely on the neutralization test for the detection of type 2 antibodies. It should be pointed out , however, that both our type 1 and type 2 reference antisera cross-react in an indirect immunofluorescence assay (antigens = chicken embryo fibroblasts infected by the TY89 or the Lukert (type 1) strains of IBDV). This confirms that type 1 and type 2 IBDVs indeed share some cross reactive group antigen(s)."

**Conclusions**

MAF's conclusions from the above are as follows :

1. If birds have been infected with IBD type 2 then the most sensitive test to detect antibody is the serum neutralization test.

2. While the ELISA and gel diffusion tests are less sensitive in detecting antibody to type 2 virus, it is possible to get positive reactions in birds that have been infected with type 2 virus even when type 1 virus is used to produce the antigen for the test.

In the New Zealand situation, if low level reactions were found in the gel diffusion test or the ELISA it would not be possible to say with confidence whether these were due to infection with IBD type 1 or with IBD type 2.

With the present state of tests it would be necessary to use the serum neutralization test to differentiate the specificity of the antibodies.