

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

REVIEW OF SUBMISSIONS ON:

**REVISED QUANTITATIVE RISK
ASSESSMENTS ON CHICKEN MEAT
FROM THE UNITED STATES;**

**REASSESSMENT OF HEAT TREATMENT
FOR INACTIVATION OF NEWCASTLE
DISEASE VIRUS IN CHICKEN MEAT.**

21 November 2000

MAF Biosecurity Authority

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TREATMENT FOR INACTIVATION OF NEWCASTLE DISEASE VIRUS IN
CHICKEN MEAT**

**Biosecurity Authority
Ministry of Agriculture and Forestry
Wellington
New Zealand**

21 November 2000

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Approved for general release

A handwritten signature in black ink, appearing to read 'Derek Belton', is centered on the page. The signature is written in a cursive, flowing style.

Derek Belton
Director Animal Biosecurity
Biosecurity Authority

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INTRODUCTION

In April 2000 the Ministry of Agriculture and Forestry released for public consultation a revised risk assessment on certain aspects of possible chicken meat imports.¹ That assessment examined the risks of introducing infectious bursal disease (IBD) virus and Newcastle disease (ND) virus in chicken meat imports from the United States.

MAF's original risk analysis² on chicken meat dealt with a very extensive range of diseases and covered imports from any country. It was released for public consultation in March 1999. A review of the submissions received was published in September 1999.³

Information submitted by the United States Department of Agriculture during the consultation period led MAF to revise the original IBD risk assessment. Other information received during the consultation process led to a re-assessment of the heat treatment needed to inactivate ND virus, should it be present in chicken meat.

Thirteen submissions on the revised risk assessment were received by the 1 August deadline, or shortly after, and are reviewed in this document. Submissions were received from:

1. United States Department of Agriculture. Facsimile received from Mr David Young, Agricultural Attaché, US Embassy, Wellington. 15 June 2000.
2. United States Department of Agriculture. Letter of 12 May 2000 from Dr Cristobal Zepeda, Veterinary Epidemiologist, Trade Assessment Team, Fort Collins.
3. United States Department of Agriculture. Email dated 8 June 2000 received from L Ferguson and TJM Myers, USDA Animal and Plant Health Inspection Service.
4. United States Department of Agriculture. Files received by email 2 August 2000 and by facsimile 3 August from Dr Alfonso Torres, Deputy Administrator Veterinary Services.
5. South African Department of Agriculture. Facsimile received 29 June 2000 from Dr Gideon Brückner, Director Veterinary Services.
6. Department of Conservation. Letter of 13 June 2000 from Ms Clare Miller, New Organisms Officer.
7. Kenepuru Branch Rural Women New Zealand. Undated letter from Mrs Maggie Girling, Secretary.
8. Barwell Pacific Ltd, Auckland. Facsimile received 2 June 2000 from Mr Bruce McLeod.

¹ *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.

² *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.

³ *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Review of Submissions. 21 September 1999.

9. William J Wyber, Christchurch. Letter dated 6 July 2000.
10. The Poultry Industry Association of New Zealand (Inc). Letter of 25 July from Mr. R J Diprose, Executive Director.
11. The Poultry Industry Association of New Zealand (Inc). At the request of the Association, material from its letter of 13 June 2000, a submission on import risk analyses for pigeons and budgerigars, was also taken into account in the current review.
12. Commission of the European Community. Health and Consumer Protection Directorate-General. Facsimile of 2 August 2000 from Mr Bernard Van Goethem.
13. Dr Dennis Alexander, Veterinary Laboratories Agency, Weybridge, United Kingdom. Email 17 May 2000 to Howard J Pharo.

It is apparent that some of the people making submissions on the revised risk assessment were not aware of the existence of the original risk analysis or the review of submissions made in response to its publication. It needs to be emphasised, therefore, that the present review of submissions is part of a formal process of risk analysis and consultation, and this document should be read in conjunction with all those that have gone before. These include:

- *Import risk analysis:* chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.
- *Import risk analysis:* chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Review of Submissions. 21 September 1999.
- *Import risk analysis:* chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.

REVIEW OF SUBMISSIONS

Note: The substance of all submissions is reproduced word for word. No submission has been paraphrased.

1. UNITED STATES DEPARTMENT OF AGRICULTURE

Facsimile received from Mr David Young, Agricultural Attaché, US Embassy, Wellington. 15 June 2000.

This facsimile comprised a brief covering note, a page of comments from L Ferguson and TJ Myers (received subsequently as an email, Submission 3, and in a letter from Dr Alfonso Torres included as Submission 4 below) and a single page of comments not submitted through other channels. It is these latter comments which are addressed here.

The U.S. appreciates New Zealand's efforts to revise the above subject import risk analysis. We are also pleased with the positive results of this review which (with similar positive conclusions of the pending New Castle's disease virus review) will have the effect of opening market access for poultry products from the United States to New Zealand, albeit for a limited quantities. We view the proposed quantitative limitation as a confidence-building measure, which will assist in providing more performance data on actual risks. Therefore, reviews at future dates of updated risk profiles would be appropriate based on the most useful data.

MAF comments: The risk estimates resulting from the revised IBD risk assessment were not intended as a "confidence-building measure." The revised risk assessment examined the risks posed by three different commodities imported in three different volumes. Only one commodity, imported at a relatively small volume appeared to pose a risk sufficiently small to be considered acceptable.

MAF is not sure what is meant by "...similar positive conclusions of the pending New Castle's disease virus review" since the document under discussion did contain a section on Newcastle disease and no further work was pending.

The U.S. Department of Agriculture provided technical comments to this risk analysis on June 8, 2000. We trust these have been well received and that the concerns expressed in those technical comments can be readily addressed. In addition to those comments, we have a general comment related to the underlying approach and results of this analysis. We applaud the quantitative approach applied in assessing and evaluating the risks associated with the importation of these products. As you are well aware, the US has supported the appropriate use of quantitative risk assessments in our bilateral discussion, quadrilateral meetings and forums such as the WTO Committee on Sanitary and Phytosanitary Measures. The discipline of quantifying individual

risks and the consequences of such risks is generally not easy, and therefore, a preponderance of assessments undertaken by WTO members appears to be qualitative and not quantitative.

MAF comments: We agree that the discipline of quantifying risks is not easy.

In reviewing New Zealand's current revised risk analysis, we became increasingly aware of possible broader policy issues associated with the distinctions between qualitative and quantitative risk analysis and their practical application. One issue involves quantitative product restrictions based on sanitary and phytosanitary risk profiles. As this has stimulated new thoughts, the U.S. looks forward to discussing these issues with New Zealand, potentially as early as the meeting of the SPS Committee in Geneva next week.

MAF Comments: The preliminary discussions between MAF and the USDA held in the margins of the June 2000 SPS committee meeting did not consider this issue in any detail. Instead, the USDA indicated that any further discussion of such trade policy issues would come at a later date.

2. UNITED STATES DEPARTMENT OF AGRICULTURE

**Letter of 12 May 2000 from Dr Cristobal Zepeda, Veterinary Epidemiologist,
Trade Assessment Team, Fort Collins.**

IBD Risk Assessment [*Submitter's heading*]

Risk is the likelihood of occurrence of an adverse event and the magnitude of the consequences. The risk estimates provided in the assessment are based only on the release and exposure assessments (i.e. the likelihood portion of the risk definition) and are not considering the consequences of a potential IBD introduction. The OIE Code specifies that both direct and indirect consequences should be taken into consideration. The number and type of farms that could be affected, the impact on production, potential trade losses, the cost of control and eradication measures are examples of some of the issues to consider in a consequence assessment. Clearly the risk would be different if the disease would affect a few backyard farms and be self-contained (or detected and stamped-out) than if it would spread into large commercial operations.

MAF comments: It is unclear why USDA considers that a more detailed consequence assessment would assist in the risk analysis process.

*The present values of the New Zealand poultry industry (Gallus gallus only) are:*⁴

- *Chicken meat production, annual wholesale value* \$506 million
- *Commercial eggs* \$150 million
- *Meat industry direct purchases of goods and services* \$290 million
- *Egg industry direct purchases of goods and services* \$ 70 million

There are presently around 3,200 people directly employed in the poultry industry.

New Zealand is presently free from IBD (See Appendix 1). Any reintroduction of this disease is undesirable. As long ago as 1985 it was estimated⁵ that the cost of introduction of IBD would be in excess of \$5.25 million (1985 dollars) per annum for the meat chicken industry alone. This figure is made up of costs due to growth depression, increased mortality, increased coccidiostat usage, cost of control measures, extra processing costs, and increased susceptibility to other diseases. Given the expansion of the broiler industry which has occurred in the past 15 years, the annual cost of IBD to the broiler industry would probably be significantly greater than estimated in 1985.

A further indication of the cost of an IBD introduction to the New Zealand poultry industry may be gauged from the cost of eradicating the vaccinal

⁴ Diprose, RJ, email to Stuart MacDiarmid, 28 September 2000.

⁵ Christensen, N H, 1985: The cost to the meat chicken industry of the introduction of infectious bursal disease to New Zealand. *New Zealand Veterinary Journal* 33: 191-193.

strain of IBD which was introduced through mislabeled vaccine some years ago. Between 1994 and 2000 the New Zealand Poultry Industry spent \$5,572,405 to eradicate the virus.⁶ Industry sources estimate the on-going annual surveillance costs to confirm that the IBD strain has been eradicated will be around \$360,000.

Having demonstrated that the introduction of IBD would cause significant economic damage, it is difficult to see how further refinement of the consequence assessment would clarify issues.

The conclusions of the risk assessment touch on sensitive issues: the concept of imposing limits to the volume of trade based on risk and the definition of the appropriate level of protection. Presumably for this assessment, the threshold for the acceptability of risk is somewhere between 0.006 and 0.06 disease introductions per year. However, without reference to the potential impact of a disease incursion it is a difficult concept to grasp. A current line of thought is to include economics in the determination of the appropriate level of protection by taking into consideration the benefits of trade and the potential costs of disease introduction and its associated likelihood of occurrence. However, this is an idea that has not yet gained wide acceptability, particularly at political levels.

MAF comments: We do not know how to interpret USDA's comment that "A current line of thought is to include economics in the determination of the appropriate level of protection by taking into consideration the benefits of trade..." This "line of thought" is not congruent with MAF's understanding of the SPS Agreement, and if it has "not yet gained wide acceptability, particularly at political levels", MAF is unsure why USDA has raised it in a submission.

A suggestion for the assessment would be to try to predict the expected volume of trade based on economic principles and model the uncertainty surrounding this estimate.

MAF comments: Given all the uncertainty surrounding the assessment of risk, MAF does not believe that any clarity would be introduced by attempting to predict a volume of trade. MAF considers that an appropriate approach is to estimate the risk associated with a range of possible volumes of consumption of imported commodity.

The revised assessment utilises a modified BetaPert distribution to model the age of chickens when infected with the IBD virus. The distribution utilises a weight so that 90% of the area under the curve falls between specified ages. Although we agree with the use of this distribution it is unclear how the weight was determined. In the interest of transparency it would be useful to describe the method used. Further, the parameters m and c used in the distribution are not defined in the text, perhaps it would be clearer to say that the distribution has a form $\text{Beta}(a1, a2) * (\text{max}-\text{min}) + \text{min}$.

⁶ Diprose, RJ, email to Stuart MacDiarmid, 28 September 2000.

MAF comments: USDA considers it is unclear why MAF chose to utilise a distribution in which 90% of the area under the curve falls between specified ages. As stated on page 4 of the revised risk assessment, this value was chosen because USDA had asserted⁷ that “nearly all” IBD infection in broilers occurs between 14 and 28 days of age. MAF interpreted “nearly all” to mean that 90% of the area under the curve of a distribution describing the age at first infection falls between 14 and 28 days of age. MAF had to place a numerical value on USDA's "nearly all" and arbitrarily settled on 90%.

In Section 2.2.1., page 4 of the revised risk assessment we explained how this weighting was achieved using standard solver function in Microsoft Excel.

Newcastle disease risk assessment [*Submitter's heading*]

The assessment comes to the conclusion that, given the current vaccination practices in the US, the risk of introducing vaccinal strains of PMV-1, is negligible. However, given that no information is available on the potential spread of field strains into commercial flocks prior to slaughter, the assessment concludes that mitigation measures such as cooking or sampling flocks prior to slaughter are warranted. If trade from the US is initiated it would be interesting to reassess the risk after enough information has been gathered by the sampling process.

MAF comments: Since publication of the original risk analysis we have become aware of a report of avian PMV-1 having been imported from the spinal cord tissues of frozen poultry carcasses traded internationally.⁸ This report confirms that importation of frozen carcasses could pose a risk with respect to PMV-1.

As in the IBD assessment, no explicit consideration is given to the magnitude of the consequences if these strains would be introduced into New Zealand.

MAF comments: New Zealand has a number of endangered native bird species which could be jeopardised by any new strains of PMV-1. MAF does not consider that any more explicit assessment of the economic consequences of introduction of PMV-1 would clarify issues. This was discussed in some detail in the original risk analysis.⁹

The assessment uses a modified BetaPert distribution to model the age at infection with Newcastle disease virus. The same comments for IBD above apply.

MAF comments: In Section 3.1.3.1., page 17, we explained how this weighting was achieved using standard solver function in Microsoft Excel.

⁷ Email from W Jolly at the New Zealand Embassy in Washington to B O'Neil of MAF, containing forwarded email from L Fergusson of USDA, 9 November 1999.

⁸ Altmann, S, Kaleta, EF, 1994. Isolation of avian PMV-1 from spinal cord tissues of frozen carcasses of Pekin ducks imported from a third country. In *New and Evolving Diseases of Poultry*, editors MS McNulty and JB McFerran, proceedings of a seminar organised by the European Commission, Brussels, 15 to 16 December 1992. Pages 37-45.

⁹ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.*

3. UNITED STATES DEPARTMENT OF AGRICULTURE

Email dated 8 June 2000 received from L Ferguson and TJM Myers, USDA Animal and Plant Health Inspection Service.

This submission was the same text as that included in Submission 1, the 15 June facsimile from US Embassy, Wellington, and in Submission 4 below from Dr Alfonso Torres. Its content and MAF's responses appear below under Submission 4.

4. UNITED STATES DEPARTMENT OF AGRICULTURE

Files received by email, 2 August 2000 and by facsimile 3 August

Covering letter from Dr Alfonso Torres, Deputy Administrator Veterinary Services.

We appreciate the ongoing science-based efforts in this risk analysis.

MAF comments: We are pleased that USDA recognises the effort we have made to base our risk analysis in science.

While these comments are essentially the same as those transmitted electronically last month, you will find some minor grammatical changes. In addition, we have taken this opportunity to clarify our comments concerning our surveillance for avian paramyxovirus-1. These clarifications do not change the essential point made in the electronically submitted comments.

Letter on US surveillance for PMV-1 from Dr Alfonso Torres

The following clarification of our PMV-1 surveillance activities is designed to assure you that PMV-1 viruses that meet the current OIE definition of Newcastle disease are not circulating in commercial U.S. poultry. As you are aware, we cannot provide specific evidence that PMV-1 field strains with an intracerebral pathogenicity index (ICPI) greater than 0.0 do not exist in the United States. However, based on the various factors described in this letter, we believe that we can state that there are no PMV-1 viruses with an ICPI greater than 0.7 circulating in commercial poultry flocks.

*MAF comments: Studies, confirmed by testing in laboratories in other countries, have shown that while some New Zealand PMV-1 isolates may have an ICPI greater than 0.0, none are greater than 0.2 (See Appendix 2 for details). These isolates still have ICPI values considerably less than those with ICPI of 0.7 acknowledged by USDA to be circulating in US poultry flocks. MAF takes the position that while some PMV-1 viruses which are regarded as "lentogenic" may cause few clinical signs in poultry, it is difficult to predict how these viruses would behave if they got into native bird species in this country. Such strains do not occur in New Zealand, and it is MAF's responsibility to keep them out. Furthermore, recent Australian experience strongly suggests that PMV-1 viruses may not be as "stable" in terms of pathotype as was once thought. There is now good evidence that PMV-1 viruses may become virulent by mutation **after** introduction in chickens.¹⁰ Until this is clarified MAF considers a precautionary approach is justified.*

The surveillance system for PMV-1 in the United States is by necessity a passive surveillance system. The U.S. poultry industry consists of over 8 billion chickens, turkeys, and other poultry species that are susceptible to PMV-1 infection, regardless

¹⁰ Alexander, DJ. Newcastle disease. *Poultry Science*. In press.

of their vaccination status. These birds are held either commercially or privately across 50 States with varying animal health laws and regulations. In addition, we have a wide variety of wild avian species susceptible to PMV-1 infection, many of which migrate annually to Central and South American locations where Newcastle disease is endemic.

Based on all of these factors, we believe that an active, nationwide PMV-1 surveillance system would be cost prohibitive and would not necessarily provide the U.S. poultry industry and our trading partners with any greater level of confidence in our PMV-1 status of freedom from mesogenic and velogenic viruses than our current system.

Despite its passive nature, however, our surveillance system has several features which allow us to maintain confidence in our reported status:

1. All birds imported into the United States are quarantined and tested for PMV-1. This has been described to you in previous correspondence.

2. Producers, veterinarians, and laboratories are obligated through a combination of Federal and State requirements to report Newcastle disease. The list of reportable diseases is codified in Title 9, Code of Federal Regulations (9 CFR), section 71.3, which prohibits the interstate movement of diseased animals and poultry. This section lists some diseases as endemic to the United States and also includes a broad definition for “any other communicable foreign disease not known to exist in the United States.” Newcastle disease, with no further specific definition, is included in this list. More specific control requirements, which would be applied in the case of an outbreak of exotic Newcastle disease, appear in 9 CFR part 82 subpart A. Furthermore, individual States have their own lists of reportable diseases, which are in addition to federally reportable diseases.

3. All poultry slaughtered in the United States are subject to inspection on the processing line, including an evaluation for clinical signs and post-mortem lesions. This provides at least one point where every commercial poultry flock is physically examined by a Federal veterinarian for signs of Newcastle disease. Even if one assumes that ante-mortem clinical signs might not be seen if a mild field strain of PMV-1 moves through a vaccinated flock, one would expect to see at least some increase in acute or chronic air sac lesions and airsacculitis condemnations at slaughter, leading to diagnostic laboratory investigation by the producer. The sheer volume of U.S. poultry examined in this manner assures us that the regular circulation of PMV-1 viruses of which you are concerned does not occur.

*MAF Comments: It is our understanding that field strains of PMV-1 can circulate without producing **any** signs in vaccinated birds. Indeed, the recent Australian experiences that virulent field strains can circulate for some time without detection in flocks which have, in effect, been “vaccinated” through the circulation of avirulent field strains.*

4. Any flock or bird which presents to a State or university diagnostic laboratory with a history of respiratory signs is evaluated for PMV-1. Our poultry industry and the laboratories which support them are keenly aware of the threat posed by Newcastle

disease. As the most critical points of surveillance, State and university diagnostic laboratories attempt to identify any and all strains of PMV-1 when they occur in poultry or other avian species. These laboratories are diligent in identifying any virus that is either unusual or more pathogenic than a lentogenic virus (as defined by mean death time or chicken pathogenicity tests) and submitting such viruses to the National Veterinary Services Laboratories (NVSL) for further typing or sequencing. Because all such viruses are referred to the NVSL, we are confident that the absence of submissions of mesogenic or velogenic viruses in the past 10 years accurately reflects the absence of these viruses in the commercial poultry population.

MAF Comments: We have been unable to find any published reports documenting these findings.

5. Serology results are monitored for PMV-1. Serological testing for PMV-1 is included in routine diagnostic screens on flocks which present with respiratory signs. Laboratories are familiar with expected serology results because of vaccination practices. Any unusual results found through such serology, e.g., increased titers or rising titers, are investigated further to rule out the presence of pathogenic PMV-1 viruses.

MAF Comments: As vaccination against Newcastle disease is widespread in the US, it is not clear to us how results of serological surveillance for PMV-1 could be interpreted.

We would also like to comment on the possibility of Newcastle vaccines masking the presence of Newcastle disease:

The examples in the literature that you cited typically discuss the problem of intense vaccination programs masking the detection of new cases during an ongoing outbreak. In the absence of an outbreak, the frequency and variety of vaccines used are much reduced, so that vaccination poses little impediment to detection. For example, the routine practice of vaccinating broilers with live lentogenic Newcastle vaccines at hatch and at 14 days of age did little to prevent either the recent outbreak of Newcastle disease in Mexico or its detection. This vaccination regime has been practiced in both the United States and Mexico, and the recent outbreak has led both countries to question the value of such vaccines in the field. Masking has become an issue in Mexico's ongoing surveillance program only when Mexico began using a combination of live and killed virus vaccines in broilers.

MAF Comments: USDA is implying that "masking" is only likely to become an issue where vaccination practices similar to those in Mexico are adopted. Recent Australian experience suggests otherwise.

Currently in the United States, we would only expect vaccination to inhibit our ability to detect a new occurrence of Newcastle disease in poultry kept for a considerable period of time, and hence subjected to several live and killed virus vaccinations, e.g., in table egg layer flocks and breeding stock. However, since replacement pullets are typically grown within a few miles of the older table egg layer or breeder flocks, a regional outbreak would no doubt be readily detected in these younger birds.

In response to the new OIE definition, during fiscal year 1999 the NVSL implemented molecular methods to pathotype PMV-1 viruses. Molecular pathotyping was done for 24 viruses in fiscal year 1999. The viruses came from a variety of sources, including domestic poultry, imported pet birds, live-bird markets, and wild birds. Fourteen domestic viruses—from commercial poultry or other birds—were characterized as lentogenic strains by mean death time in chicken embryos and in the chicken pathogenicity test. They had an amino acid sequence at the fusion peptide cleavage site compatible with low pathogenic strains of PMV-1 and the consensus sequence for these viruses was RQGR/L.

MAF Comments: We have been unable to find published reports of this molecular pathotyping. However, the statement does suggest that PMV-1 strains of low virulence are circulating in US poultry despite vaccination practices.

The remaining 10 viruses—from imported birds in quarantine, smuggled birds, or wild birds, but none from commercial poultry—were characterized as velogenic, viscerotropic velogenic, and pigeon paramyxovirus-like (PPMV-1) viruses. They had fusion peptide cleavage site sequences compatible with virulent Newcastle disease virus and the consensus sequence for these viruses was RQKR/F. With the exception of the PPMV-1 viruses, the sequencing results for this group of viruses were consistent with the characterization results obtained by the mean death time test and the chicken pathogenicity test.

The NVSL is planning to implement the ICPI test on a more routine basis as the new OIE definition is accepted. This test will be used to characterize any of the PMV-1 virus submissions they receive through our surveillance system as described. In addition, they will offer this test to those laboratories who do not have similar capabilities.

Specific response to MAF's revised risk assessment from USDA, Animal and Plant Health Inspection Service

The following text was also received in Submissions 1 and 3.

We appreciate the fact that the original risk analysis was revised based on information provided in our initial comments. We strongly support this type of transparency and science-based risk analysis efforts, and will do our best to ensure that similar efforts will continue to take place relative to any future issues from either side.

We have one significant comment on the revised assessment relative to infectious bursal disease (IBD.) In the initial risk assessment, two classes of products were identified - carcasses vs. boneless and bone-in cuts - based on adherent organ tissue. The revised risk assessment now separate boneless from bone-in cuts based on the age of birds at slaughter. The risk assessment makes the assumption that bone-in cuts will be obtained from birds slaughtered at a younger age than those from which boneless meat is obtained. This might be an accurate assumption, based on current marketing patterns, but we believe that the age of slaughter should be the determining factor rather than the specific cut. While the risk assessment didn't model this assumption, we believe that the risk presented by boneless or bone-in cuts obtained from birds

slaughtered after 42 days of age should be similar. Therefore, the import requirements should at least allow for the possibility of importing bone-in cuts obtained from birds slaughtered after 42 days of age.

MAF comments: We believe that USDA may have partly misunderstood the reasons why the revised risk assessment generated different risk estimates for bone-in and boneless cuts. Part of this difference was attributable to MAF's acceptance and use of information provided by USDA in its submission on the original risk analysis. In the revised risk assessment (Section 2.2.2., page 5), MAF stated that age at slaughter is potentially important as younger birds may pose a greater risk of having active IBD infection at slaughter. Conversely older birds may pose less risk. In the original MAF Import Risk Analysis age at slaughter was considered to be anywhere from 32 to 49 days, but the USDA contended that most broilers in the USA are slaughtered between 42 and 56 days of age. The youngest slaughter age reported for broilers was 35 days of age. Birds used to produce de-boned meat may be slaughtered as late as 63 days of age. The USDA contended that birds slaughtered at 35 days of age would "typically supply parts for the domestic fast food market, and would not be exported" and that "due to shipping costs" ... "older birds with greater muscle mass" ... "are often used to produce de-boned meat" which would comprise the export product.

In the revised risk assessment, MAF chose not to limit the model to considering de-boned chicken only as New Zealand entrepreneurs have expressed interest in importing bone-in products such as wings and drums. Therefore, for remodelling the risk of IBD virus introduction, the slaughter age for bone-in products was modelled as a uniform distribution between 35 and 56 days and for boneless products it was modelled as a uniform distribution between 42 and 63 days.

After consideration of submissions made on the revised risk assessment, and after further investigation of industry practices with respect to age at slaughter, MAF no longer considers it appropriate to model different ages at slaughter for boneless and bone-in products. The values used in the further revision of the IBD risk are presented in Appendix 3 below.

We stated above that we believe that USDA may have partly misunderstood the reasons why the revised risk assessment generated different risk estimates for bone-in and boneless cuts. While part of the reason for the difference in the revised risk assessment (but not in the original model) was attributable to age at slaughter in both risk assessments an important part of the difference was attributable to the probability of the commodity generating scraps. No-one can realistically doubt that bone-in products are more likely to generate scraps than boneless products, and this difference was reflected in both risk assessments, and is repeated below in Appendix 3.

We understand your point concerning surveillance for vaccinal vs. field strains of avian paramyxovirus-1 in US poultry flocks. Our surveillance system for PMV-1 is primarily a passive system. Any identified strains of the virus which are unusual or more pathogenic than what has traditionally been determined as lentogenic are

reported to USDA-APHIS for further characterization. While this passive surveillance demonstrates no evidence that there is a significant presence of any such pathogenic strains, neither do we have specific evidence obtained via active surveillance to demonstrate definitively that these strains do not exist. We trust that this issue can be revisited at a later date as our surveillance systems continue to evolve.

MAF Comments: Because of the potential consequences for New Zealand's domestic and native bird populations, until this issue of the pathogenicity of the field strains of PMV-1 circulating in the US is clarified, specific sanitary measures against PMV-1, as outlined in the original risk analysis, are warranted.

We would like to clarify one point relative to the requirements for cooked product, specifically how the IBD and PMV-1 requirements will be combined. This will most likely be clarified during the development of an import health standard, but this is also a good opportunity to ask the question. We assume that if a product meets the IBD requirements - i.e., a boneless product obtained from birds slaughtered after 42 days of age - then it must only meet the cooking requirements relative to PMV-1. No additional time and temperature requirements relative to IBD would apply in this instance. Please let us know if this assumption is incorrect.

MAF Comments: Imported chicken meat products would have to meet the requirements for IBD and Newcastle disease.

Our final comment is relative to the pending public health risk assessment which is to be completed by the Ministry of Health. We assume that this will also be available for comment. While APHIS would not have the lead authority in public health issues, we would like to monitor this issue and track how it fits into the overall picture concerning poultry meat exports. Please keep us advised of progress on this pending risk assessment.

MAF comments: It is our understanding that the Ministry of Health will make its public health risk analysis available for stakeholder comment. We assume that the US Embassy will monitor progress and report back to the appropriate US agencies.

5. SOUTH AFRICAN DEPARTMENT OF AGRICULTURE

Facsimile received 29 June 2000 from Dr Gideon Brückner, Director Veterinary Services.

The risk analysis is based on information provided by the USDA, but the USDA has no enforcement in place in all instances to guarantee certain provisions regarding vaccination programmes and official records thereof. The basis for the risk assessment place in my opinion is too little emphasis on the human factor, which is a crucial variable in the transmission of diseases in intensive animal production systems.

MAF comments: International trade is based on trust between the Competent Authorities in the trading countries. MAF is expected to accept certification from USDA just as New Zealand exporters expect the Competent Authority in any other country to accept MAF's certification.

Risk of IBD introduction [Submitter's heading]

It is stated that there is very little risk as long as birds are not vaccinated with live IBD vaccines in the 21 days prior to slaughter and provided that the age of the birds at slaughter is not less than 42 days. It is also stated that 'Under Federal Regulations US poultry are not permitted to be slaughtered within 21 days of receiving any live virus vaccine. Poultry veterinarians and producers are expected to abide by these restrictions, but there is no system of enforcement'. IBD vaccine strains are however classified as mild, intermediate and virulent. It is unclear how this aspect has been addressed, as the slaughter of chickens vaccinated with e.g. an intermediate or virulent virus vaccine, within 21 days prior to slaughter, or slaughtered at an age younger than 42 days could lead to the introduction of IBD. There is no system of enforcement and/or official records.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

The risk assessment specifies bone-in as well as bone-out products, but boneless cuts should be skinless as well. The presence of skin has an important influence on risk as many pathogens are carried in feather dust and trapped inside feather follicles during de-feathering and subsequent chilling.

MAF comments: The assertion that "many" pathogens are carried in feather dust and trapped inside feather follicles is not supported by any reference to scientific literature. Nevertheless, the issue of specific pathogens being carried on skin was addressed in the original risk analysis.

The risk assessment is based on certain importation figures. It is stated that the risk assessment would have to be repeated should the importation increase above the current level. A system should thus be in place to monitor the importation figures and

activate the re-evaluation of the risk. The prevention of the possible introduction of an exotic disease or virulent strain should rather be based on the risk of the presence of an agent in a product or product unit, regardless of how small the quantities (qualitative approach), than on a basis of imported products in limited quantities and thus 'diluted' by domestic supplies for general consumption. This renders the most important approach of non-negotiable strict biosecurity control to prevent the spread of diseases, a less effective exercise.

MAF comments: The OIE International Animal Health Code, Article 1.4.2.3. states "Risk increases with increasing volume of commodity imported." Similarly, the more tickets one buys in a lottery, the greater the chance one has of winning. MAF rejects the assertion that risk prevention should be based on the presence of an agent in a product or product unit, regardless of how small the quantities to be imported. Such a position is tantamount to demanding "zero risk". Further, the International Animal Health Code explicitly recognises that the risk of introduction is dependent not merely on the presence of a pathogen in a commodity, but also on the likelihood of susceptible animals in the importing country being exposed to the pathogen.

Newcastle disease risk assessment [Submitter's heading]

It is stated that the meat has to originate from chickens vaccinated at 10-14 days of age with an attenuated vaccine. There is however no guarantee that vaccination will not take place at a later age, as indicated before.

Although vaccination with live vaccines is carried out with lentogenic strains only and it is stated that flocks could be exposed to lentogenic field strains (last outbreak of virulent NCD in the USA in 1971/72), the outbreaks of Newcastle disease in Australia have indicated that lentogenic field strains could possibly re-assort and cause outbreaks. It is therefore essential that all PMV-1 viruses are considered to be important as they pose a possible risk at all times.

MAF comments: New Zealand PMV-1 isolates have ICPI values less than 0.2 while vaccine strains typically have an ICPI greater than 0.4. While some PMV-1 viruses which are regarded as "lentogenic" may cause few clinical signs in poultry, it is difficult to predict how these viruses would behave if they got into native bird species in this country. Such strains do not occur in New Zealand, and it is MAF's responsibility to keep them out. Furthermore, recent Australian experience indicates that PMV-1 viruses may not be as "stable" in terms of pathotype as was once thought; until this is clarified MAF considers a precautionary approach is justified.

The time/temperature requirements to inactivate ND virus are calculated for a whole chicken carcass. It is unclear whether it has been taken into account that the virus could persist in bone marrow for at least 21 days after infection and whether the cooking regime will ensure inactivation of the virus in bone marrow. Chicken bones are commonly discarded at food outlets and swill could pose a serious risk when fed to backyard animals.

MAF comments: No authority is cited in support of the assertion that could persist in bone marrow for at least 21 days. We are not aware of any studies specifically examining the stability/lability of PMV-1 in bone marrow. We have assumed that core temperatures specified in the document under discussion¹¹ will inactivate PMV-1 regardless of the medium.

It is stated that the litter in houses is removed on a yearly basis (once per annum). If one considers that there are at least five broiler production cycles per house per year and the important role that faeces plays in a situation where carriers are shedding viruses (agents), it seems strange that this production practice with its aggravating effects has not been addressed in the risk assessment as a high risk factor.

MAF comments: Although shed cleaning practices certainly influence the likelihood of successive batches of broilers becoming infected during the growing period, this was dealt with in the original risk analysis, when reviewing submissions, and in the revised risk assessment.

¹¹ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.*

6. DEPARTMENT OF CONSERVATION

Letter of 13 June 2000 from Ms Clare Miller, New Organisms Officer.

The risk analysis appears to be a thorough and well researched document. The consideration of volume of trade as a component of risk is valid and important.

The Department supports the conclusions that MAF reached regarding acceptability of the risks relating to different levels of trade. If trade rose above 500 tonnes of boneless meat, we would find the associated risks of disease introduction unacceptable. I have some concern about how MAF will monitor the level of trade in boneless chicken meat, and whether there is any mechanism whereby that trade can be suspended if it exceeds the 500 tonne mark. I would appreciate it if you could provide me with information on these points.

MAF comments: We note that Department of Conservation considers that the risk of IBD introduction would be unacceptable if more than 500 tonnes of boneless chicken product were to be imported. Should importation of chicken meat occur, MAF would implement a permit system to monitor volume of commodity imported. In a briefing to the Minister for Biosecurity¹² MAF stated "...should any trade in boneless chicken cuts originating from US flocks not free of IBD virus take place at a level where we could not be 95% certain that the risk of IBD introduction did not exceed 1 per 100 years..., then sanitary conditions for the trade will need to be reconsidered. New additional conditions would be needed to bring the estimated risk of the trade to an acceptable level."

Obviously, DOC would like to see the risks of introduction of Newcastle disease and IBD reduced as far as possible due to the threat that these diseases could pose to our native avifauna. This risk analysis is a well considered assessment of how the risks can be reduced if trade in chicken products from the USA takes place.

MAF comments: Throughout the risk analysis process MAF has recognised the need to assess the risks to New Zealand's unique native bird species. In the original risk analysis (Section 3.3.9.6., page 90) it was pointed out that Newcastle disease could infect native birds, possibly causing serious mortalities. However, the original risk analysis also made the point (Section 3.3.7.6., page 77), that IBD virus causes disease only in chickens and its introduction would be expected to impact only on the poultry industry.

¹² File reference: AR60-060, Brief No: 99/389, 4 April 2000, Bruce Ross, Director-General of Agriculture to Hon Marian Hobbs, Minister for Biosecurity.

7. KENEPURU BRANCH RURAL WOMEN NEW ZEALAND

Undated letter from Mrs Maggie Girling, Secretary.

We would like to protest the proposed importation of US chicken meat into New Zealand.

Any disease risk, however minimal you consider it, is a matter of concern, particularly when New Zealand is perfectly capable of producing enough chicken meat to meet its needs.

Probably the US product will be cheaper: This is not, we feel, a sufficient reason for imports and such a move will also hit existing home producers.

There have been too many 'accidental' breaches of biosecurity recently. We do not need to tempt fate on purpose.

MAF comments: This submission opposes importation of chicken meat on philosophical grounds and does not raise any technical issues. It is not clear whether the submitters have actually read either the original risk analysis or the revised risk assessment.

8. BARWELL PACIFIC LTD, AUCKLAND

Facsimile received 2 June 2000 from Mr Bruce McLeod.

- Page 4, paragraph 7 states that the USDA estimates that less than 5% of US broiler flocks have not been exposed to I.B.D. virus. If another country's I.B.D. exposure is less than in the US, will that country be regarded as a more favourable country of origin for imported poultry?

MAF comments: The original risk analysis concluded (Section 3.3.7.9.1, page 80), that chicken meat should be sourced only from broiler flocks demonstrated to be free from infection with IBD virus and which have not been vaccinated with live IBD vaccines.

- Page 19, paragraph 3.4, final paragraph. We have asked that Canadian F.I.A. to provide an assurance on this, as we believe it can be done.

MAF comments: The section referred to in the revised risk assessment reads "...MAF considers it is reasonable to conclude that assurances are required to ensure that broiler flocks have not been exposed to field strains [of PMV-1] within the last few weeks prior to slaughter." MAF has received no communication from the Canadian Food Inspection Authority suggesting that it is able to provide this assurance.

- No mention is made in these pages, which conclude (pt 2.4) pages 13 & 14, on inactivation of Avian disease concerns via a thermal process. Starting at page 15 the paper considers the Newcastle's Disease risk. It proposes a thermal process (70°C/30 min, 80°C/5min). Are these two analyses linked to the same thermal process requirement [?]

MAF comments: The revised risk assessment, to which this submission is addressed, dealt with revised quantitative risk assessments on the IBD risks posed by importation of chicken meat from the United States only, and a reassessment of heat treatment required for the inactivation of Newcastle disease virus in chicken meat (from any source). The revised risk assessment did not address the question of heat inactivation of IBD virus. This had been dealt with thoroughly in the original risk analysis where it was concluded (Section 3.3.7.9.2., page 80) "...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 and not vaccinated with live IBD vaccines."

- Reading of the paper indicates that MAF will allow uncooked boneless chicken meat at a 500MT level and that they will allow cooked poultry if thermally treated as outlined above to satisfy ND concerns. Is this a correct understanding?

MAF comments: In the revised risk assessment, MAF proposed that an IBD risk no greater than 1 disease introduction per 100 years would be acceptable.

The assessment examined the risks posed by three different commodities imported in three different volumes. Only one commodity, imported at a relatively small volume, appeared to pose an IBD risk sufficiently small to be considered acceptable. However, as discussed below in response to Submission 10, and in Appendix 3, the uncertainty surrounding data used in the revised risk assessment results require a further re-examination of the IBD risks and the conclusions of the revised assessment can not be supported.

On the basis of a reassessment of the heat treatment required to inactivate Newcastle disease virus in chicken meat, the revised risk assessment recommended cooking times of 5 minutes at 80° C or 30 minutes at 70° C.

- The paper is titled “Import Risk Analysis: Chicken Meat and Chicken Meat Products: Bernard Matthews Foods Ltd turkey meat preparation from the United Kingdom.” Nowhere in the MAF paper is the UK noted NOR is there any mention of turkey. Where is the connection to Bernard Matthews?

MAF comments: The original risk analysis¹³ dealt in Part 1 with chicken meat products from any country and in Part 2 with Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. The revised risk assessment¹⁴, to which this submitter is responding, addressed only three issues arising out of the analysis of submissions received in response to that original document. This point was made explicitly in the opening paragraph on page 1 where it was stated "This document, which follows on from the original import risk analysis (March 1999) and the review of submissions (September 1999) presents the results of the following analyses:

- *remodelling the risk of introduction of infectious bursal disease virus in chicken meat products of US origin*
 - *modelling the risk of introduction of Newcastle disease virus in chicken meat products of US origin*
 - *time/temperature requirements to inactivate Newcastle disease virus in chicken meat."*
- The entire analysis is referenced to US data. Where does Turkey and the UK figure in this discussion?

MAF comments: See above.

- Is the paper taking a view on the United States, as a high risk exporter, thus accepting that exporting countries with a better Avian status are therefore covered if complying with US parameters? If not, then is this paper designed to be exclusive to the USA?

MAF comments: See above.

¹³ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.*

¹⁴ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.*

- Please explain 'flock freedom' page 1, paragraph 4.

MAF comments: "Flock freedom" means free from infection with the virus in question, in this case IBD virus. As stated in the original risk analysis "Where it has been concluded that flock freedom is a necessary safeguard for a particular disease, the specific details of testing, monitoring and certification are not prescribed [in the current document], 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."

- The report ascertains that an import of 500MT per annum poses an acceptable quarantine risk but that a quantity in excess of the figure would need to be reviewed. How does MAF propose to regulate imports and issue permits to import in view of the proposed 500MT ceiling? Page 14, paragraph 2.

MAF comments: Should importation of chicken meat occur without specific safeguards for IBD, MAF would implement a permit system to monitor volume of commodity imported. In a briefing to the Minister for Biosecurity¹⁵ MAF stated "...should any trade in boneless chicken cuts originating from US flocks not free of IBD virus take place at a level where we could not be 95% certain that the risk of IBD introduction did not exceed 1 per 100 years..., then sanitary conditions for the trade will need to be reconsidered. New additional conditions would be needed to bring the estimated risk of the trade to an acceptable level."

However, discussed below in response to Submission 10, and in Appendix 3, the great uncertainty surrounding data used in the revised risk assessment results require a further re-examination of the IBD risks and the conclusions of the revised assessment, that certain volumes of chicken meat could safely be imported without specific IBD safeguards, can not be supported, so the issue of restricting imports to a specified volume is no longer relevant .

We submit that Chicken Meat and Chicken Meat products from Canada present less risk than those from the USA.

MAF comments: No evidence is presented to support this assertion.

Our Principal, Northern Goose, Teulon, Manitoba, Canada, is the only EEC and UK approved plant in North America. It is also a USDA approved plant.

Northern Goose have formally requested the C.F.I.A. Canada, to request MAF New Zealand for approval of their products to enter New Zealand, under the same conditions/existing Health Standards as the USA currently enjoys.

¹⁵ Ref: AR60-060, Brief No: 99/389, 4 April 2000, Bruce Ross, Director-General of Agriculture to Hon Marian Hobbs, Minister for Biosecurity.

MAF comments: No communication has been received from either the Canadian Food Inspection Authority or Northern Goose. However, once the specific safeguards required to safeguard New Zealand's biosecurity have been finalised and incorporated into an Import Health Standard, any chicken products which can meet the conditions in the Import Health Standard will be permitted access.

9. WILLIAM J WYBER, CHRISTCHURCH

Letter dated 6 July 2000.

I would submit as follows:-

- a) That the New Zealand Poultry Industry is already sufficiently depressed, in a financial sense, that it is not necessary or desirable to depress the Market further by overloading supplies to the customer;
- b) That with the current price structure, which allows me to buy frozen chick- meat – whole or in portions – at a retail price of an average of \$4.85 per kilogramme this makes it one of the cheapest meats ... the other being the ability to buy fish from a “wholesale market”, unprocessed, at \$3.80 and less ... available to the consumer. Further supplies to a Market which is already patently and adequately supplied is entirely unnecessary, and could be called “hare-brained”;
- c) That the supplies of eggs from the Industry are currently retailing at as low as \$1.65 per dozen for 6’s., amply demonstrates that the Poultry Industry could be classified as a “depressed industry” of already low margins – it is therefore a situation without public/consumer demand – because of a supply-shortage – that importation not be permitted from any source; for it would further damage the Industry which has been severely curtailed and damaged since deregulation in approx. 1982, and the General Public have come to rely upon a market in which they can trust;
- d) That the risk of importation of biohazards and organisms it too great to justify any risk whatsoever – probably jeopardising the good name and reputation of New Zealand’s “Clean and Green Image” and of our produce and most probably become a disincentive, in the culinary sense, to our most valued Tourist Industry. Now you will see why I’m inclined to call the proposal, if that is what it is, “hare-brained”.

MAF comments: This submission opposes importation of chicken meat on philosophical grounds and does not raise any technical issues. It is not clear whether the submitter has actually read either the original risk analysis or the revised risk assessment.

10. THE POULTRY INDUSTRY ASSOCIATION OF NEW ZEALAND (INC)

Letter of 25 July from Mr. R J Diprose, Executive Director.

In addition to this submission, we would also like to refer you to our letter of 24 May 1999 regarding the original import risk analysis and would wish to reconfirm the points made in that letter and the original submission.

MAF comments: The points made by the Poultry Industry of New Zealand in their submission on the original risk analysis were addressed in the review of submissions published 21 September 1999.

You will see in our submission that the Industry does not support the Revised Quantitative Risk Assessments and assumptions made in relation to the risk associated with IBD virus and chicken meat imports from the USA. Our submission clearly illustrates that there is a considerable degree of doubt with the information supplied by the USDA and that there is significant variability as to when IBD virus will infect broilers.

MAF comments: We accept the assertion that there is considerable degree of doubt surrounding the information upon which the revised risk assessment was based. The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

I would respectfully request that once you and the MAF Biosecurity officials involved have reviewed our submission that the Association is given the opportunity to meet with and discuss in detail the issues raised before the import risk analysis is finalised and any public announcements made.

MAF comments: All stakeholders will be given the opportunity to comment on MAF's review of the submissions received in response to the revised risk assessment.

Where we have not made comment we are in general agreement with these sections in the import risk analysis.

We agree that there are four main issues that must be addressed in assessing the risk of introducing exotic avian and public health disease agents. Thus:

- Public health risks – in relation to exotic pathogens
- Risk of introduction and establishment of infectious bursal disease (IBD) in avian species
- Risk of introduction and establishment of Newcastle disease (ND) in avian species
- Time/temperature requirements to inactivate Newcastle disease virus by cooking.

We understand that the Ministry of Health is carrying out a risk assessment of the public health impact of importing raw chicken meat and that they will be issuing two discussion documents for public consultation. Firstly, a discussion document on what the salmonella status is in New Zealand and the trends observed in the last few years together with a

comparison of the New Zealand salmonella status with overseas countries. We understand this document is planned to be issued 15 September 2000 followed by a 60 day public consultation period.

MAF comments: This is also MAF's understanding.

Secondly, a discussion document on the risk assessment of food products that could compromise salmonella status in New Zealand. This document to be issued 15 March 2001 and to be followed with a 60 day consultation period.

MAF comments: This is also MAF's understanding.

We further understand that MAF cannot develop any import health standard for the importation of chicken meat until the Ministry of Health completes its separate assessment of public health risks of importing exotic strains of salmonella.

MAF comments: This is correct.

We note that this MAF revised risk assessment deals with the remaining three issues above following MAF's request for further information from the USDA subsequent to the initial import risk analysis in March 1999.

The Introduction in the revised risk assessment states that the USDA supplied detailed responses to MAF's five questions as listed. We disagree with this statement and refer to reference (5)* – Email from W Jolly 9 November 1999. This reference (5) does not contain detailed data in relation to the total USA broiler chicken production. Also it only contains anecdotal evidence from Dr Lisa Ferguson, USDA. This anecdotal evidence is challenged later in our submission and as a result of this we believe that there is not any scientific grounds on which to revise the original risk assessment in the document March 1999.

Detailed examination of the IBD situation in the USA would confirm that there was no new information to indicate that some of the inputs used in the IBD simulation model in the March 1999 risk analysis should be revised. Thus we will show that the original risk analysis should stand and that there is an unacceptable level of risk for importation of even boneless chicken meat products from USA flocks.

Re-modelling the risk of IBD introduction [*Submitter's heading*]

R1: The probability that the source flock is infected [*Submitter's heading*]

We would ask on what scientific grounds MAF has considered it reasonable to accept the USDA's position that "less than 5% of US flocks have not been exposed to IBD virus", and as a consequence have changed the probability from Pert to a Uniform distribution. The information is an estimate from L Ferguson from the USDA.

In the communication from the New Zealand Veterinary Councillor in the USA, Dr Bill Jolly, New Zealand Embassy, Washington DC, November 1999, he states that Dr Lisa Ferguson

* The reference number (5) cited here is reference (5) in the revised risk assessment: Email from W Jolly at the NZ Embassy in Washington to B O'Neil of MAF, containing forwarded email from L Ferguson of USDA, 9 November 1999.

admits that the USDA has no way of enforcing a restriction that live vaccines may not be used within 21 days prior to slaughter of chickens. He also comments that “while AFIS is no doubt well intentioned in representing the interests of the US poultry industry what became clear to him at the USAHA (US Animal Health Association) meeting was that the US poultry industry is much like the poultry industry world wide in that they very much keep the regulations at arms length. Potentially a great deal more disease incidences and type information remains closeted within the industry’s own laboratories than APIS (Animal and Plant Health Inspection Services) ever becomes aware of”.

Dr Jolly however states that on the basis of intelligence gathered at the USAHA meeting he thinks that USDA’s ability to survey disease status is questionable.

We thus repeat our question to MAF and ask on what basis can they accept their anecdotal evidence from Lisa Ferguson, USDA, and revise the original risk assessment.

MAF comments: The information submitted by USDA was accepted on the basis that MAF assumed that USDA would have access to reliable information on the incidence of IBD infection and vaccination practices in US poultry flocks.

This question also relates to all sections where MAF has accepted and imputed this new anecdotal evidence from the USDA.

R2: The probability that tissues from a chicken will be carrying infection at Slaughter
[Submitter’s heading]

We dispute the statement that “most chickens in the USA are likely to become infected between 14 and 28 days of age with either a field strain or a vaccine strain of IBD virus”.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

The age of infection can be variable between day old and slaughter depending upon maternal antibodies, viral challenge, strain of virus and stresses either present or absent within the flock.

The dynamics of infection onset and spread of IBD virus within a flock will be influenced by the level of maternal antibody titres, the variability of maternal antibody titres, the pathogenicity and invasiveness of the particular strain of virus, genetic makeup of the bird, intercurrent diseases, nutritional factors and environmental conditions.

The revised model’s assumptions do not allow or make reference to the variability that does exist in biological systems which are known to occur within the broiler industry.

We would also ask for MAF’s interpretation of “most” chickens. What mathematical value is attributed to “most”.

MAF comments: As stated on page 4 of the revised risk assessment, MAF had to place a numerical value on USDA's "nearly all" and arbitrarily settled on 90%.

We dispute the claim that “nearly all” the USA broilers will have been exposed (infected) to field virus by 28 days regardless of whether or not live vaccines have been used.

The following information has been received from Dr Bruce Stewart-Brown DVM, Perdue Poultry Company (Perdue Farm, Inc is based in Salisbury, Maryland, Delmarva Peninsula is the fourth largest chicken processing company in the USA currently producing around 11.6 million broiler chickens per week).

Dr Stewart-Brown states that currently at Perdue 70% of their broiler chickens get to 28 days without field infection. However by 35 days over 80% have become infected, by processing virtually 100% have seen and responded to the field virus. Therefore it is obvious that broiler chickens at slaughter can be carrying IBD virus. This documented position with Perdue would also be found to be the normal situation in most broiler companies in the USA that have elected to use bursal derived IBD vaccines in their breeders giving high maternal antibody to the progeny pushing the age of infection beyond 28 days.

We are advised from a number of sources that other major companies in the USA, including Tyson Foods Inc (the largest chicken processing company in the USA currently producing around 45 million broiler chickens per week) are currently relying on maternal antibody titres for protection with their broilers and are currently not vaccinating broiler chickens.

Dr Margaret McKenzie in personal discussions with the major integrators during a visit to Arkansas, USA, in July this year, has been informed that the time of IBD infection in flocks can occur at any age during the growout cycle with both classical and new variant strains. The companies visited did not practice any broiler IBD vaccination and all agree that IBDV challenge can occur at any time and that variants can break through vaccine IBDV immunity at any time.

It is quite clear that the revised risk assessment model is invalid with the inputs used supplied from USDA and thus the real risk has been properly identified in the original risk assessment March 1999.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

The information above is further substantiated with the following references:

¹⁶ Dr Joseph J. Giambrone states: “4) there are differences in efficiency of transmittal of antibody from hen to the progeny between strains of hens and within hens of the same strain: and 5) often broilers are placed in the same house from different age breeder flocks, which have highly variable levels of immunity. Therefore, broiler progeny in the same house will have mosaics of immunity, resulting in a wide coefficient of variation (CV) in mean antibody titer.”

¹⁶ Giambrone Dr JJ, Poultry Science Department, Auburn University, Atlanta, USA, Broiler Vaccination-Additional Protection is Often Needed! Watt Publishing International Poultry Symposium Summit on Infectious Bursal Disease Proceedings, April 30-4, 1995, University of Georgia Continuing Education Centre.

¹⁷ K J Fahey states: “To protect chickens to 34 days of age would require a titre in chickens at one day of age of 12,800.”

Fahey provides data to show that maternal antibody ELISA titres range up to 83,200 in his experimental flocks thus providing protection in excess of 34 days of age to IBD infection.

¹⁸ J B McFerran states: “Maternal antibody is transferred in the egg yolk to the chicks and if titres are high enough, it will protect them for 5 weeks or longer.”

¹⁹ S A Lister states: “The disease seems to be worse where infection occurs just as maternally derived antibody is waning, ie about 35 days.”

²⁰ P J Wyeth states: “The decay of maternally derived antibody is linear so that chicks with low levels become susceptible to field challenge at an earlier age than chicks with high levels.

The difference between the onset of susceptibility and the time when all the flock is susceptible can be up to 20 days.”

²¹ P J Wyeth demonstrated that: “The onset of susceptibility IBD challenge occurred at 25 days of age in one group and 27 days in another. Total susceptibility did not occur until 43 and 45 days of age respectively.”

²² P J Wyeth in comparing the efficiency of four inactive IBD vaccines demonstrated that: “The group A chicks were all resistant to challenge until 30 days old and were not fully susceptible until 44 days old. In groups, B, C and D the onset of susceptibility occurred on days 24, 24 and 23 respectively and in each group all chicks were susceptible to challenge on days 34, 36 and 34 respectively.”

The above evidence confirms that there is significant variability in timing of IBD infection both within and between broiler flocks and that individual broilers within any flock can be carrying IBD virus at any time from day old to slaughter. Therefore we believe that the revised model with 90% of the age of first infection falling between 14 and 21 days is invalid.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

¹⁷ Fahey KJ et al, CSIRO Division of Animal Health, Animal Health Research Laboratory, Private Bag No. 1, PO Parkville, Victoria 3052, Australia, Assessment by ELISA of passively acquired protection against infectious bursal disease virus in chickens, Australian Veterinary Journal, Vol. 64, No. 7 July, 1987.

¹⁸ McFerran JB, Infectious Bursal Disease, Chapter 16, Virus Infections of Birds.

¹⁹ Lister SA, BVet Med, MRCVS, Veterinary Investigation Centre, Government Buildings, Jupiter Road, Norwich NR6 6ST, Short communication Gumboro Disease (Infectious Bursal Disease).

²⁰ Infectious Bursal Disease in Great Britain, pers com PJ Wyeth, Central Veterinary Laboratory, Weybridge, Surrey

²¹ Wyeth PJ, Cullen GA, Central Veterinary Laboratory, New Haw, Weybridge, Surrey, The use of an inactivated infectious bursal disease oil emulsion vaccine in commercial broiler parent chickens, The Veterinary Record, March 3, (1979) 104. 188-193

²² Wyeth PJ, Chettle N, Central Veterinary Laboratory, New Haw, Weybridge, Surrey, Comparison of the efficacy of four inactivated infectious bursal disease oil emulsion vaccines, The Veterinary Record, April 10, 1982, 110, 359-361

It is also acknowledged in the USA that there are new variants of IBD appearing and that broiler chickens will be re-exposed to these variants from vaccine strains after 28 days of age.

MAF comments: We are not sure who it is that “acknowledged” the above. No reference is provided to support the contention that chickens will be re-exposed after 28 days of age.

It should also be noted that industry husbandry practices in the USA are under constant revision and change and there is currently a strong push for improved biosecurity and hygiene on the broiler farms. This means that some companies are now adopting an all in all out production system as practiced in New Zealand. It means there is a total shed and litter cleanout after each run which will lead to a later field challenge of IBD with the broiler chickens. That is well after 28 days of age.

MAF Comments: It is not clear to us why “total shed and litter cleanout after each run” will necessarily result in age at first infection being “well after 28 days of age.”

There is no doubt the US industry is changing from an annual cleanout to one after each run with many variations in between. This is due to a result of requirements under the USDA Mega Regs particularly in relation to salmonella control.

In addition to this the USA broiler industry is expanding into greenfield areas closer to the grain growing belt rather than expansion in the South East where the poultry industry has been concentrated. The outcome of this will be a change in the epidemiology of IBD infection which will result in later challenge from field strain IBD virus.

MAF comments: It is unclear why expanding into “greenfield areas” should necessarily lead to a change in the epidemiology of IBD infection and why such a change, if it did occur, would necessarily result in later challenge with field strains of the virus. No argument is offered to support this assertion, and no authority is cited to support it.

These industry changes were not taken into account with the information provided by Lisa Ferguson from the USDA.

²³ Also, research carried out by J J Giambrone: “Research by Dr David Snyder of the University of Maryland, Dr Daryl Jackwood of Ohio State University and by myself at Auburn University indicates that these viruses are continuing to mutate, and now are predominant in the field.”

This demonstrates that the situation in the USA with regards to IBD infection and disease is not static but continually changing. Therefore the absolute assumptions made by Lisa Ferguson are not valid.

We believe the assumptions in relation to age of slaughter and the model uniform distribution is an over simplification of actual industry practices in the USA. Boneless meat may come from any age of broiler slaughtered particularly from the lower value cuts in the USA such as

²³ Joseph J Giambrone, PH.D, Vineland Laboratories, Monitoring the Immune Status of Breeders Against IBDVs Using Progeny Challenge and Serological Data, www.vinelandlabs.com/pages/pub64.html, 20/07/00

drum and thigh meat. There is an over supply of these low value cuts in the USA market and most of these are disposed in export markets.

MAF comments: The question of age at slaughter is revisited below in Appendix 3.

A further point to consider was highlighted in MAF's original risk assessment in relation to work commissioned on duration of tissue infectivity. Refer reference (5)* MAF Risk Assessment March 1999.

It is an established fact that secondary viraemia will occur after extensive stress such as caging, loading, and transport and holding birds at processing. Viraemia can occur within a few hours of such a stress, certainly at a shorter time than that which will elapse between catching and slaughter in the USA.

MAF comments: We are not aware that this is "an established fact". Given the significance of this assertion we would have expected it to be supported by reference to peer-reviewed scientific literature.

We do not accept the results of the revised model as we have shown above that the inputs into this model are invalid. The assumptions made in model 2 do not cover all the field situations that will be faced in the USA chicken broiler production.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

We do not accept the findings in the model output because we do not accept the model inputs.

We do not accept the conclusion that for US boneless chicken consumption at a level of 1% of current New Zealand consumption presents an acceptable risk in importation. The risks for this has been clearly outlined above. We do not accept the outcome of the revised model, therefore the risk assessments in the conclusion can also not be accepted.

We believe a more accurate assessment was that contained in the findings of the Risk Assessment March 1999.

MAF comments: The revised risk assessment was made, in good faith, using information supplied by USDA. Subsequently, on the basis of our own further investigations and inquiries, and the information contained in these submissions, we re-examine this issue in Appendix 3 below.

We again confirm that we do not accept the assessments made that birds in excess of 42 days of age would produce product with an acceptable level of risk for importation as the findings

* MAF is not certain which reference (5) in the original risk analysis is referred to here, as each section in the original risk analysis had its own bibliography. However, none of the references labelled (5) in the original risk analysis appear pertinent to the point being made here. Perhaps the submitter is referring to reference (6) on page 166; Quality Control Unit (1997). Study Report: Dissemination of infectious bursal disease virus in chickens infected with very virulent strain CS88. Study number CVLS/07/97, Contract number FT0518. Central Veterinary Laboratory, United Kingdom.

presented confirm that broiler chickens in excess of 42 days could be carrying IBD virus and boneless product could come from birds processed at any age.

We would also ask MAF on what grounds have they come to the conclusion that an importation risk of less than one disease per 100 importation years is acceptable while six introductions per 100 importation years is an unacceptable high risk. Who determines and how has this determination been made that one disease introduction per 100 importation years is an acceptable level of risk to New Zealand's avian species.

MAF comments: Current government policy recognises that it is impossible to eliminate all risks in the importation of goods and management of international passenger movements. Therefore its policy is to operate a biosecurity system under the Biosecurity Act 1993 that mitigates biosecurity risks in a consistently effective manner.

Determining risk management measures to apply (and thus the level of protection achieved) in different situations is part of the day-to-day work of departments administering the Biosecurity Act (in this case the Ministry of Agriculture and Forestry or MAF). Under section 22(1) of that Act the Director-General of MAF may, on the recommendation of a chief technical officer (CTO), issue an import health standard (IHS) specifying the requirements to be met for the effective management of risks associated with the importation of risk goods before those goods may be imported. The authority of the Director-General to issue IHSs is delegated to CTOs, and the authority to make recommendations about the issuing of IHSs is delegated to, among other officials, National Managers and National Advisers in the Animal Biosecurity group of MAF. The State Sector Act 1988 provides for such delegations.

When making a recommendation for the issuing of an IHS in accordance with section 22 of the Biosecurity Act (and thus recommending a level of protection appropriate in any given situation), section 22(5) provides the relevant officials must have regard to the following matters:

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

The process followed by MAF in discharging its responsibilities under the Biosecurity Act is consistent with meeting New Zealand's obligations under the World Trade Organization SPS agreement (the agreement on the application of sanitary and phytosanitary measures):

- A systematic analysis of risks is conducted, taking into account relevant international methodology.*
- Biosecurity measures are proposed that are firmly based on the supporting risk analysis.*
- Proposed risk management decisions are compared with those taken previously for similar risks or similar products (situations can be compared if they involve either a risk of entry, establishment or spread of the same or similar disease, or a*

risk of the same or similar associated potential biological and economic consequences).

- *This whole process is well documented and transparent.*

As a result of this process, MAF concluded that if the upper 95th percentile of the annual risk of introducing infectious bursal disease (IBD) was estimated to be 0.006 per year (that is, less than one disease introduction per 100 importation years) the risk was acceptable, and that if the upper 95th percentile of the annual IBD risk was estimated at 0.06 per year (or 6 introductions per 100 importation years) the risk would be unacceptably high.

The risk analysis process is still continuing, as further information is analysed and comments from interested parties assessed. When this current risk analysis process is completed, a recommendation will be made to a CTO (in this case the Director Animal Biosecurity) on whether an IHS for chicken meat from the USA should be issued, and if so with what conditions.

We are also extremely concerned to note correspondence from Dr W Jolly and from L Ferguson that there is a problem relying on USDA certification as a safeguard against introduction of poultry diseases. It is probable that certifying vets in the USA may be signing statements they have no way of verifying and secondly the powerful US poultry industry clearly sees that open reporting is against its interests and dismisses concerns of countries such as New Zealand. It was also noted that USDA's Foreign Agricultural Service (FAS) has consistently misrepresented New Zealand's concerns as a trade policy issue and states that "the FAS would appear to be representing more a technical opinion of the US poultry industry rather than their own technical agency".

One should also note in the document entitled 'US Poultry News' published by the US Poultry and Egg Association 'the pros and cons of a National Health Reporting System (NAHRS)' which states that open and honest reporting of disease status information is against the interests of the USA poultry industry because honest reporting of diseases will interfere with trade.

With these stated positions in the US how can New Zealand accept that any level of "equivalence" will be delivered?

MAF comments: International trade is based on trust between the Competent Authorities in the trading countries. MAF is expected to accept certification from USDA just as New Zealand exporters expect the Competent Authority in any other country to accept MAF's certification.

Newcastle disease risk assessment [Submitter's heading]

The comments we have made in our submission in the IBD risk assessment in relation to the disease surveillance and reporting in the USA, and age of birds at slaughter also pertain to this Newcastle disease risk assessment.

We agree with MAF's assessment that there would be a very real risk that chicken meat imported from the USA would be contaminated for non-vaccinal strains of PMV-1 virus and

that the conclusion that the consequence of introduction of field strains of PMV-1 virus (particularly velogenic strains) into New Zealand is certainly to be severe.

MAF comments: The revised risk assessment did not state that "... that there would be a very real risk that chicken meat imported from the USA would be contaminated for non-vaccinal strains of PMV-1 virus and that the conclusion that the consequence of introduction of field strains of PMV-1 virus (particularly velogenic strains) into New Zealand is certainly to be severe." To clarify, we cite here from the risk estimation made in Section 3.4, page 19, of the revised risk assessment.

*"Given the assumptions made regarding age at vaccination and age at slaughter, the quantitative risk assessment model demonstrated that there is a negligible risk of **vaccinal strains** [emphasis added] of PMV-1 virus being present in tissues of US chickens at the time of slaughter. However, MAF was not able to objectively assess the likelihood that field strains of PMV-1 would be circulating in poultry flocks during the last few weeks prior to slaughter. That is, in the absence of specific information on the issue, the release assessment model did not address the risk that a non-vaccinal strain might enter a US flock close to slaughter, replicate without producing clinical signs, and result in the presence of virus in tissues at the time of slaughter. While it is unlikely that such an introduction would remain undetected in the long term, MAF considers it is likely that it could escape detection in the short to medium term and thus lead to chicken meat being contaminated with a non-vaccinal strain of PMV-1.*

*"While there remains a **small** [emphasis added] risk of PMV-1 being present in US chicken meat products, it must also be recognised that any exposure risk in New Zealand is likely to be **very small** [emphasis added]."*

However, the revised risk assessment did go on to say "MAF considers that the consequence of introduction of field strains of PMV-1 virus (particularly velogenic strains) is almost certain to be severe.

"Therefore MAF considers it is reasonable to conclude that assurances are required to ensure that broiler flocks have not been exposed to field strains within the last few weeks prior to slaughter."

In further support of a cautious approach MAF reiterates that since publication of the original risk analysis we have become aware of a report of avian PMV-1 having been isolated from the spinal cord tissues of frozen poultry carcasses traded internationally.²⁴ This report confirms that importation of frozen carcasses could pose a risk with respect to PMV-1.

We believe that any risk management option aimed at preventing the introduction of PMV-1 virus into New Zealand must include virus isolation from the flock immediately prior to slaughter.

²⁴ Altmann, S, Kaleta, EF, 1994. Isolation of avian PMV-1 from spinal cord tissues of frozen carcasses of Pekin ducks imported from a third country. In *New and Evolving Diseases of Poultry*, editors MS McNulty and JB McFerran, proceedings of a seminar organised by the European Commission, Brussels, 15 to 16 December 1992. Pages 37-45.

MAF comments: In the revised risk assessment we concluded "... assurances are required to ensure that broiler flocks have not been exposed to field strains within the last few weeks prior to slaughter." However, where the risk analysis process concludes that flock freedom is a necessary safeguard for a particular disease, as in the case of infection with PMV-1 virus, the specific details of testing, monitoring and certification are not prescribed in the risk analysis, 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."

We support the MAF recommendation that from flocks not able to demonstrate freedom from PMV-1 virus that only cooked meat be recommended with times and temperatures ie five minutes at 80⁰C or 30 minutes at 70⁰C be considered for importation.

We would draw to MAF's attention that the Industry is extremely concerned to discover (after reviewing documents obtained under the Official Information Act) that the USDA is unable to guarantee the validity of their export certifications, and of their inability to have accurate information in relation to the animal health status in the USA commercial broiler flocks.

MAF comments: International trade is based on trust between the Competent Authorities in the trading countries. MAF is expected to accept certification from USDA just as New Zealand exporters expect the Competent Authority in any other country to accept MAF's certification.

11. THE POULTRY INDUSTRY ASSOCIATION OF NEW ZEALAND (INC)

Extract from letter of 13 June 2000, a submission on import risk analyses for pigeons and budgerigars.

This text is included here, at the request of PIANZ, because of its relevance to the issue of chicken meat imports.

As you are aware from the email from Bill Jolly to yourself dated Tuesday November 9 1999, Lisa Ferguson from the USDA has stated "There have been no isolations of mesogenic or velogenic ND virus in the US for several decades." This statement is called into dispute by the following references:

Panigraphy B, et al (1993) *Avian-Diseases* 37: 1,254-258 states: "In 1991 velogenic viscerotropic Newcastle disease was diagnosed in domestic psittacine birds in 6 states, Illinois, Indiana, Michigan, Texas, California and Nevada. In the first 4 states the disease assumed outbreak proportions".

Foreign-Animal Diseases Report 1987, 15:2,1 states: "In April 1987 exotic Newcastle disease was confirmed in 7 young yellow napped Amazon parrots ... in Maryland".

Foreign-Animal Diseases Report 1988 16, 3 1-3 states: "during investigations of foreign animal diseases in the USA VVNDV was diagnosed in 7 birds in an 8 month period."

Avian Diseases 1983 27 3, 731-744 states: "From October 1973 to September 1987 viruses were isolated from 26.3% of quarantined birds. VVNDV was isolated from 141 lots of 2274. Mesogenic and lentogenic positive birds were allowed entry to the USA".

Dr Daniel King (1996) ZooTechnica states that VVNDV occurred in a range reared turkey flock in 1992 in North Dakota.

MAF comments: We interpret this submission as implying that mesogenic or velogenic strains of PMV-1 are more widespread in the United States than stated in submission from USDA. MAF questions whether this implication can be drawn.

12. COMMISSION OF THE EUROPEAN COMMUNITY HEALTH AND CONSUMER PROTECTION DIRECTORATE-GENERAL

Newcastle disease (ND) [*Submitter's heading*]

The risk analysis was developed regarding the disease situation in the United Kingdom in relation to the risk of importing poultry meat products from one plant. It must be pointed out that in the European Union there are three Member States (Denmark, Finland and Sweden) that have an approved ND non-vaccinating status for Newcastle disease. Annual testing of breeding flocks according to EC criteria is carried out in these countries to maintain this status. To ensure protection for the status of these countries in Intra-Community trade and to facilitate diagnostic investigations for the confirmation of ND the criteria for vaccines used for routine vaccination were harmonised in the European Community. An introduction of ND to Denmark, Finland or Sweden by this trade has not been reported.

MAF comments: The statement that "The risk analysis was developed regarding the disease situation in the United Kingdom in relation to the risk of importing poultry meat products from one plant" is incorrect. The original risk analysis covered chicken meat products from any country. However, as was stated in its opening paragraph, the revised risk assessment did deal with new information provided by USDA in response to the original risk analysis. Nevertheless, once the specific safeguards required to safeguard New Zealand's biosecurity have been finalised and incorporated into an Import Health Standard, any chicken products which can meet the conditions in the Import Health Standard will be permitted access.

That importation of poultry carcasses could pose a risk with respect to PMV-1 is supported by a report which has come to our attention since MAF completed its original risk analysis. Avian PMV-1 has been reported as having been isolated from the spinal cord tissues of frozen poultry carcasses imported into the European Union.²⁵

In point 3.1.2 of the revised risk analysis it is speculated that vaccine virus will show the same distribution and duration of presence in tissues as virulent field viruses in infected animals. This is not a valid assumption as the very reason that the vaccine strains are less virulent is the fact that they are restricted in their replication to specific sites of the host. These sites are, where trypsin-like proteases occur i.e. primarily in the respiratory and intestinal tract, which will not be used for poultry meat exports and poultry meat preparations.

MAF comments: A similar point was made by other submitters responding to the original risk analysis. (The European Commission did not make any technical submission on that risk analysis: See the review of submissions,

²⁵ Altmann, S, Kaleta, EF, 1994. Isolation of avian PMV-1 from spinal cord tissues of frozen carcasses of Pekin ducks imported from a third country. In *New and Evolving Diseases of Poultry*, editors MS McNulty and JB McFerran, proceedings of a seminar organised by the European Commission, Brussels, 15 to 16 December 1992. Pages 37-45.

Section 8, page 20.) For example USDA asserted (Review of submissions, Section 1.8, page 5) that "it is well understood that lentogenic ND viruses are restricted in their tissue distribution to the respiratory and intestinal tracts". USDA cited a chapter by Dennis Alexander in Calnek 10th edition as supporting this contention. USDA went on to assert that "Therefore the risk of finding these [PMV-1] viruses in meat is virtually nil". In response to this submission, MAF pointed out that chapter cited does not, in fact, completely support the contention. On page 550 it is explained that lentogenic viruses can replicate only in tissues such as the respiratory and intestinal tracts, whereas virulent viruses can replicate in a range of tissues and organs. On page 556, under the heading "Samples". The following is stated: "The two main sites of replication of NDV in infected poultry appear to be the respiratory and intestinal tracts." But there is no mention of tissue distribution of the virus as a result of viraemia, let alone differentiation of such tissue distribution by pathotype. It was precisely because of the lack of information on tissue distribution of ND virus that MAF commissioned work on this matter. The trial done in the USA and quoted as reference 20 in the original risk analysis used a mesogenic strain. **In the absence of other information to the contrary, a reasonable precautionary approach is to assume that all PMV-1 viruses have tissue distributions during viraemia similar to the mesogenic strain discussed in the original risk analysis.**

Until scientific studies have been undertaken to clarify the tissue distribution of strains of relatively low virulence, the potential consequences for commercial poultry and endangered native species of PMV-1 introduction require MAF to take a precautionary approach. In the absence of specific information to the contrary, MAF assumes that the tissue distribution of all PMV-1 strains is similar.

In 3.3 information is given about the ND surveillance which has been carried out in New Zealand during the last few years. It is claimed that all isolates have shown intracerebral pathogenicity indexes (ICPI) of 0.0. It is questionable how many of these findings have been confirmed by laboratories outside New Zealand for reference purposes taking into account the biological nature of the test and the likelihood of variation associated with such tests.

MAF comments: Studies, confirmed by testing in laboratories in other countries, have shown that while some New Zealand PMV-1 isolates may have an ICPI greater than 0.0, none are greater than 0.2 (See Appendix 2 for details). These isolates still have ICPI values considerably less than those considered of concern by the European Union. MAF takes the position that while some PMV-1 viruses which are regarded as "lentogenic" may cause few clinical signs in poultry, it is difficult to predict how these viruses would behave if they got into native bird species in this country. Such strains do not occur in New Zealand, and it is MAF's responsibility to keep them out. Furthermore, recent Australian experience strongly suggests that PMV-1 viruses may not be as "stable" in terms of pathotype as was once thought. There is now good evidence that PMV-1 viruses may become virulent by

mutation after introduction in chickens.²⁶ Until this is clarified MAF considers a precautionary approach is justified.

Considering the current EC definition for Newcastle disease, which covers all paramyxoviruses (PMV) 1 strains showing a higher pathogenicity index than 0.7 and the definition as given by the O.I.E. it is unjustified for New Zealand to require a zero risk by asking for country freedom of paramyxoviruses 1 with a higher ICPI than 0.0 or originating from establishments which are located in a zone not infected with strains of PMV 1 with an ICPI greater than 0.0. This is not in line with international standards and poses therefore an unnecessary trade barrier.

MAF comments: See above. New Zealand may adopt measures providing greater biosecurity than provided by international standards where justified. New Zealand's unique native bird species, and the extremely low pathogenicity of local PMV-1 isolates justify the adoption of more stringent safeguards.

As mentioned above the harmonised rules for the use of live attenuated vaccines restrict the use of strains to those that show an ICPI lower than 0.4. Pre-export examinations in third countries for the detection of the presence of ND virus therefore require freedom of ND viruses showing an ICPI higher than 0.4. This requirement should also be considered as offering sufficient protection for exports to NZL.

MAF comments: See above.

In point 4 of the document MAF New Zealand has reassessed the time/temperature requirements to inactivate ND virus. Cooking times of 5 minutes at 80°C for 30 minutes at 70° Celsius to inactivate PMV 1 in chicken meat are recommended. In the original document a study by Dennis Alexander from the Community Reference laboratory for Newcastle disease in Weybridge was cited, in which the duration of heat treatment would be considerably shorter. The calculated D values which would give a 9 log drop in titre after 12.3 minutes at 70° Celsius are more realistic than the values taken from the inactivation data presented by NZL, which suggests the re-examination of the data is required.

MAF Comments: In Appendix 3 of the original risk analysis²⁷ we explained the basis for our interpretation of the raw data and subsequent recommendations. We do not believe a re-examination of the data is required.

Infectious bursal disease (IBD) [Submitter's heading]

The work that has been carried out to assess the heat resistance of the virus has to be acknowledged and the EC can agree on the conclusion that heat inactivation of IBD virus does not seem feasible for poultry meat products. In regard to future trade the specific details for the testing and the monitoring requirements to demonstrate flock freedom of infectious bursal could cause serious restrictions to trade from EC countries to New Zealand.

²⁶ Alexander, DJ. Newcastle disease. *Poultry Science*. In press.

²⁷ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.*

MAF comments: We regret that the necessary safeguards might be seen as causing serious restrictions to trade, but MAF has the responsibility of protecting New Zealand from the introduction of IBD. This drawn-out and exhaustive risk analysis process has demonstrated that the only way that chicken meat can be imported safely is to require demonstration that source flocks are free from infection with IBD virus. There is too much uncertainty for MAF to do less than adopt a precautionary approach. The threat posed by viable IBD virus in internationally-traded poultry products has been recognised by other authorities.²⁸

Concerning the occurrence of infectious bursal disease in New Zealand it is stated in the original document that the viruses isolated in New Zealand have been identified as “relatively avirulent”, although causing bursal damage and immunosuppression. It is furthermore assumed that the “elimination of the infections from farms in NZL appears possible”. Therefore it is highly questionable if the imposition of such stringent requirements as envisaged in the risk analysis are justified.

MAF comments: At the time the original risk analysis was published it appeared likely that the avirulent IBD strain which had been introduced into some New Zealand broiler flocks (probably via a mislabelled batch of imported vaccine) would be eliminated. Two years later, this elimination appears to have been attained. Recent testing figures are shown in Appendix 1 and ongoing surveillance is in place to confirm these results. MAF considers that stringent safeguards are warranted to preserve this hard-won freedom from IBD.

Given that so-called “very virulent IBD virus” (vvIBDV) is widespread in Europe and is still considered by experts to represent a considerable threat to the poultry industry²⁹ MAF considers that stringent safeguards are warranted to avoid introducing vvIBDV into New Zealand.

Conclusions [Submitter’s heading]

In view of the different disease recommendations given in the document it still not transparent what import conditions and veterinary certification would exactly be required for the importation of poultry and poultry meat products to New Zealand. The main issues that would need reconsideration from an European standpoint are:

- NZL requirements for the Newcastle disease status of the exporting country
- Further evidence the ND viruses isolated in NZL have an ICPI not greater than 0.0. including findings in wild birds
- The fact that the three Member States Denmark, Finland and Sweden have an approved status as ND free and ND non-vaccinating countries
- The re-calculation of the D value for 70° Celsius for the inactivation of ND virus
- Clarification on the IBD status of NZL poultry flocks

²⁸ For example, “The interstate and international commerce of poultry products, which could contain viable IBDV, threatens the poultry industry.” Shane et al 1994, cited in Mandeville, WF, Cook, FK, Jackwood, DJ . Heat lability of five strains of infectious bursal disease virus. *Poultry Science* 79: 838-842, 2000.

²⁹ Van den Berg, TP. Acute infectious bursal disease in poultry: A review. *Avian Pathology* 29, 175-194. 2000.

- Details of testing and monitoring to demonstrate freedom of infection with IBD in the flocks of origin in the exporting country

MAF comments: These points have been addressed above.

13. DR DENNIS ALEXANDER

Dr Dennis Alexander, Veterinary Laboratories Agency, Weybridge, United Kingdom. Email 17 May 2000 to Howard J Pharo.

As far as my knowledge of IBD and maths go that section seems to be about right. I have one or two comments concerning ND:

3.1.1 1st paragraph 1st sentence. This statement just isn't true. Mixson, M.A. and Pearson, J.E. (1992): Velogenic neurotropic Newcastle disease (VNND) in cormorants and commercial turkeys, FY 1992. In Proceedings of the 96th annual meeting of the United States Animal Health Association, Louisville, Kentucky, 1992: pp 357-360. describes the spread of virus from cormorants to turkeys. You see in the USA, the notifiable disease is "velogenic viscerotropic ND" so if they describe it a neurotropic velogenic it's not counted. As you know my opinion is that there is no real difference. It would be pertinent to ask them how many viruses have been isolated from poultry in the USA that over the last 30 that would come within the OIE definition adopted last year.

MAF comments: The sentence Dr Alexander refers to reads "The last outbreak of velogenic ND in poultry in the USA was in California in 1971-72." The issue has been raised in other submissions, as outlined above.

3.1.2 paragraph 4. I don't think this is a "reasonable" speculation. All the molecular biological evidence suggests that ND viruses of low virulence are restricted to replication in places where there are trypsin-like enzymes i.e. the respiratory and intestinal tracts. Even if this resulted in a viraemia no replication should take place in muscles. I think should replace reasonable with "safe".

MAF comments: The paragraph to which the submitter refers reads "The titres of vaccine strain virus in various tissues have apparently not been studied. In the absence of specific data, it is reasonable to speculate that the distribution and duration of ND virus in the tissues of vaccinated chickens is likely to be similar to that of the field isolates studied by Alexander⁽¹⁹⁾ and Lukert⁽²⁰⁾."

The same point was made in Submission 12. Nevertheless, as we responded above, there appear to be no data on tissue distribution of the virus as a result of viraemia, let alone differentiation of such tissue distribution by pathotype. Because of this lack of data MAF commissioned research. The study, reported as reference 20 in the original risk analysis, used a mesogenic strain. In the absence of data to the contrary, as opposed to assertion, a reasonable precautionary approach is to assume that all PMV-1 viruses have tissue distributions during viraemia similar to the mesogenic strain discussed in the original risk analysis.

3.3 I see you still claim NZ isolates only have ICPI values of 0.0. Perhaps you should send your isolates to an independent reference laboratory to see what values they get!

MAF comments: Studies, confirmed by testing in laboratories in other countries, have shown that while some New Zealand PMV-1 isolates may have an ICPI greater than 0.0, none are greater than 0.2 (See Appendix 2 for details). These isolates still have

*ICPI values considerably less than those considered of concern by the European Union. MAF takes the position that while some PMV-1 viruses which are regarded as "lentogenic" may cause few clinical signs in poultry, it is difficult to predict how these viruses would behave if they got into native bird species. Such strains do not occur in New Zealand, and it is MAF's responsibility to keep them out. Furthermore, recent Australian experience strongly suggests that PMV-1 viruses may not be as "stable" in terms of pathotype as was once thought. There is now good evidence that PMV-1 viruses may become virulent by mutation **after** introduction in chickens.³⁰ Until this is clarified MAF considers a precautionary approach is justified.*

I think the proposed times in 4.2 are more realistic than the previous assessment, although, as you know I disagreed with the interpretation of our data.

MAF comments: The cooking time/temperature regimens which the submitter considers "more realistic" to inactivate PMV-1 are :

- 5 minutes at 80°C or
- 30 minutes at 70°C.

³⁰ Alexander, DJ. Newcastle disease. *Poultry Science*. In press.

Conclusions

PMV-1 (Newcastle disease): uncooked chicken meat:

Some of the submissions appeared to argue that New Zealand should be prepared to accept the introduction of PMV-1 strains of ICPI up to 0.7. New Zealand isolates of PMV-1 have ICPI values less than 0.2 while vaccine strains typically have an ICPI greater than 0.4 (See Appendix 2 for details). While some PMV-1 viruses which are regarded as "lentogenic" may cause few clinical signs in poultry, it is difficult to predict how these viruses would behave if they got into native bird species in this country. Such strains do not occur in New Zealand, and it is MAF's responsibility to keep them out. Furthermore, recent Australian experience indicates that PMV-1 viruses may not be as "stable" in terms of pathotype as was once thought; until this is clarified MAF considers a precautionary approach is justified

That is, 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. The recommended 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 *International Animal Health Code*] attesting that the entire consignment comes from birds which have been kept in an *establishment* [defined by the OIE *Code*] free from infection with strains of PMV-1 with ICPI greater than 0.2.

In its *Review of Submissions*³¹ received in response to the original risk analysis, MAF accepted that prescribing country or zone freedom from PMV-1 having an ICPI less than that of vaccine strains is probably unrealistic. However, MAF went on to recognise that adequate safeguards against PMV-1 could be provided by virological sampling and testing five days prior to slaughter, following the protocols laid down in the European Union decision 95/117/EC. This EU decision specifies: "The test should be regarded as negative if no haemagglutination activity is detected and no virus is isolated." This would satisfy New Zealand MAF. However, the EU decision goes on to prescribe the measures to determine the origin of the virus should any be isolated, and implies that virus of vaccine origin is acceptable. New Zealand would not accept the presence of **any** PMV-1 virus with an ICPI >0.2.

PMV-1: cooked chicken meat:

The current review of submissions did not convince MAF that the recommendations made in the *Revised Risk Assessment*³² need further revision. MAF considers that heat treatment of chicken meat for PMV-1 viruses should aim to achieve a final titre of not higher than $-9 \log_{10}$ CID₅₀/g (that is 10^{-9} chicken infectious doses per gram of tissue).

³¹ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Review of Submissions. 21 September 1999.*

³² *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.*

Therefore, cooking times recommended to inactivate PMV-1 in chicken meat remain the same as in the *Revised Risk Assessment*, namely:

- 5 minutes at 80°C or
- 30 minutes at 70°C.

Infectious bursal disease:

The original quantitative assessment of IBD risk³³ was revised³⁴ on the basis of submissions made by USDA³⁵. The significant revision was to the estimates for the age at which chickens first become infected with IBD virus, whether vaccinal strains or field strains.

A key variable in the risk assessment model is the probability of there being active IBD virus infection present in chickens at the time of slaughter. In the original *Risk Analysis*, MAF considered that the age at which broilers are slaughtered to be anywhere between 32 and 49 days of age. However, in its submission on that analysis, USDA asserted that "most" broilers in the United States are slaughtered between 42 and 56 days of age, with the youngest slaughter age being reported as 35 days. They also stated that birds used to produce de-boned meat may be slaughtered as late as 63 days of age. These values were used in the *Revised Risk Assessment*.

However, on the basis of the submissions examined in this document, further exploration of common industry practices, and the slaughter age data obtained by the New Zealand Embassy, MAF considers a different range of slaughter ages more appropriately reflects the situation in the United States. This new range of ages was incorporated into the revised quantitative assessment described in Appendices 3 and 4.

On the basis of the evidence discussed in this document, MAF no longer considers it tenable to assert that most US chickens become infected with IBD virus by 21 days of age. Because of the uncertainty surrounding the issue, MAF considers that the best that can be said is that chickens become infected with IBD virus sometime between hatch and slaughter, with most birds probably becoming infected sometime after maternal immunity has waned. For this reason, in calculating the probability that a chicken is infected with IBD virus at the time of slaughter, the risk assessment uses a uniform distribution of 1 to 57 days as the input for age when chickens become infected. The lack of credible data precludes attempts to achieve greater precision.

The quantitative assessment of the risk that IBD virus might be introduced through importation of US boneless chicken meat was revised for a second time. The inputs used in this second revision are described in Appendix 3 and the model itself is described again in Appendix 4. The results of this further revision are shown in Table 6 of Appendix 3. The conclusion of this second revision is that, under the assumptions used, if boneless chicken meat products from the United States were to be imported into New Zealand, even in

³³ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.*

³⁴ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.*

³⁵ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Review of Submissions. 21 September 1999.*

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 0.34 if as few as 0.1% of the chicken carcass equivalents consumed in New Zealand were to be imported.

Because of this high risk, the following safeguard is recommended to insure that IBD is not introduced into New Zealand:

Meat products, uncooked or cooked, must be sourced from broiler flocks demonstrated to be free from infection with IBD virus³⁶ and not vaccinated with live IBD vaccines.

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.

Appendix 1: COUNTRY-FREEDOM PLAN FOR INFECTIOUS BURSAL DISEASE: A PRODUCER-LED NATIONAL DISEASE CONTROL PROGRAMME³⁷

In 1993 a mild strain of IBD virus was introduced into New Zealand. Since then the poultry industry has eliminated infection from known positive farms, and is introducing a programme to confirm eradication and to obtain international recognition that New Zealand is free from IBD virus.

Virulent strains of IBD are not present in New Zealand. In late 1993 a poultry-processing plant reported lesions suggestive of IBD infection. Serological testing of blood samples taken from birds in another shed on the property of origin showed that many samples had high titres, suggesting recent infection. MAF initiated an exotic disease investigation and an initial containment response. IBD virus was isolated from samples of bursa of Fabricius, and tests carried out at an OIE reference laboratory for IBD (Central Veterinary Laboratory, Weybridge, UK) showed that the virus was a non-pathogenic strain of IBD type 1⁽¹⁾. Because this was a mild strain and a number of farms appeared to be infected, government and industry decided at that time not to adopt an eradication strategy.

The poultry industry then undertook a nationwide serological survey of all commercial breeding, meat (broiler) and layer flocks, which within a year identified 46 infected farms. Investigation revealed that all positive flocks had originated from one hatchery during a single month, and all had been vaccinated with a single batch of a Marek's disease vaccine. It was suspected that the vaccine had been incorrectly labelled, and was in fact a combination IBD and Marek's disease vaccine.

Although no clinical signs of IBD were observed, the bursal lesions that were consistently observed in infected broilers suggested that there may have been potential for production losses, at least in some broiler flocks⁽²⁾. With a view to enhancing the longer term interests of New Zealand producers (especially the potential to export birds and product), the poultry industry resolved to eradicate the IBD virus.

Epidemiological issues

An understanding of the various host, agent and environmental factors, and their interactions, is a prerequisite to successful disease control. The host range for IBD is wide, and includes poultry, turkeys and ducks. There is little evidence for spread via insect vectors. Recovered birds do not become carriers of virus, and vertical transmission does not occur.

Epidemiologically, the most important feature of the IBD virus is its ability to survive for long periods outside the host, including in extreme environments. For example, it is resistant to many solvents, acid conditions and heat (surviving 56°C for 5 hours). Survival has been recorded in poultry houses for more than 100 days and in contaminated feed, water and faeces for 50 days. There is evidence that the virus can survive on poultry products, including packaging. The primary method of spread is horizontal transmission, either direct or indirect.

Also significant to the epidemiology are certain modern poultry management practices, especially the high stocking density of birds in sheds. If the virus is introduced into a fully susceptible flock housed at high density, large numbers of birds are likely to become infected within a short period and the shed becomes contaminated with large amounts of virus. This, together with the virus's ability to survive, make it easy to see why, despite biosecurity measures around a poultry farm, infection almost

³⁷ Ryan, T, Diprose, R, Leong, R. *Surveillance*, In press.

invariably spreads from shed to shed. If equipment is shared between farms, infection can spread just as easily from farm to farm.

Control programme

The control programme, which began in 1994, was based on identification of infected poultry flocks by serological testing of representative samples of birds. Since 1994, all breeding flocks have been sampled every 2 months and all broiler flocks at slaughter. Layer flocks were tested every 6 months for the first 4 years until all tested free, and all pullet replacement layer flocks are tested before pullets are placed in laying sheds.

A protocol to decontaminate the infected farms involved strict control over movement of personnel, vehicles and equipment; shed and farm clean-out and sanitation; and stand-down periods. Some owners of infected flocks were compensated when properties were destocked for prolonged periods.

The year-by-year results are shown graphically in Figure 1, and the numbers of flocks and farms tested are listed in Table 1 (broiler) and Table 2 (layer). The last seropositive flock, detected in January 1999, was a 45-week-old layer flock known to have been infected since pullet stage. Since then there has been no evidence of infection in any of the several layer flocks that have been placed on the property, and currently no properties have evidence of infection with IBD virus.

The poultry industry is now seeking international recognition of this improved disease status. It has developed and is implementing a 'Country Freedom Quality Plan' for IBD, which is an amalgamation of a traditional national disease control scheme with a modern quality management approach. This will enable the industry to continue with the final eradication of the virus, and allow independent parties to audit and verify the activities.

The industry-freedom strategy and quality plan

The disease control strategy for achieving industry freedom is based on a national system of passive surveillance for IBD infection coupled with an active testing programme on commercial poultry, turkey and duck farms.

Infection with the introduced IBD virus consistently results in detectable gross lesions of the bursa of Fabricius. A national extension campaign will support surveillance by ensuring that managers of poultry farms and staff at processing plants can recognise the gross pathology of IBD infection.

The objective of the testing programme is to 'accredit' all farms as free from IBD virus. A random sample of birds from each shed will be tested for IBD using an ELISA. The sample size will be set after issues surrounding the sensitivity of the IBD tests have been resolved. The tested birds must have been in the shed for a minimum of 28 days to ensure that, if the virus is present, sufficient time has elapsed for it to spread within the shed. Field data show that 50% or more of birds would have seroconverted in this period. Despite the manufacturer's claim of very high test specificity, in some flocks in New Zealand false-positives occur commonly. To deal with this, the scheme rules allow for some re-testing of flocks and for additional screening with the virus neutralisation test. After three negative tests of all sheds the farm will be declared 'accredited-free'. Thereafter an annual test of all farms will continue until country freedom from IBD is achieved.

The industry freedom strategy is supported by a quality plan that was developed using principles similar to those in modern HACCP (Hazard Analysis Critical Control Point) food safety procedures. In consultation with poultry industry veterinarians, the critical points in the freedom strategy were identified and ranked, and methods to control, measure, verify and validate the key items were established.

The 'Country Freedom Programme (or Pathway)' shown in Figure 2 was adapted from similar plans developed by the Office International des Epizooties (OIE) for rinderpest⁽³⁾ and contagious bovine pleuropneumonia⁽⁴⁾. It includes an intermediate step of 'Provisional Freedom from IBD' before 'Freedom from IBD'.

A two-stage eradication campaign was adopted because of the differences between the commercial and non-commercial poultry sectors in New Zealand. The commercial sector will lead with a traditional testing scheme covering all farms. Active surveillance of the farms will continue after all have achieved 'IBD virus accredited-freedom', both to confirm their status and to detect spillover from any reservoirs of virus from the non-commercial sector and wild birds. For the purpose of achieving international recognition of country-freedom from IBD virus, provision has also been made for a scientifically sound survey of the non-commercial sector.

Natural infections with IBD virus are restricted to chickens, turkeys and ducks. In most countries where IBD is present, the clinical disease is controlled by vaccination. IBD has not been eradicated from any country, so internationally accepted criteria for establishing freedom do not exist. Whereas the establishment of freedom in commercial poultry flocks appears to be feasible, the non-commercial poultry sector may be more complex to address. In New Zealand there are marked differences between the modern commercial poultry sector and the rest of the poultry population. Some groups in the non-commercial sector are accessible and could easily be included in the programme, for example special breeds kept by fanciers. Others, such as back-yard layer flocks, are spread widely throughout rural New Zealand, and their inclusion will be more difficult.

Legal issues

An important requirement of national eradication campaigns is a legal framework to support reporting of suspect cases, to allow field investigations, to impose quarantine, and to ensure compliance with testing. For industry-initiated campaigns (in contrast to government schemes) this can pose problems. This has been resolved as follows.

1. As the mild IBD virus strain that was introduced in 1993 is not a notifiable organism under the Biosecurity Act 1993, suspected or confirmed infection with this agent does not have to be reported to the MAF Director of Animal Biosecurity. However, exotic strains of IBD, both mild and severe, are notifiable. In practice it is not possible to distinguish clinically between the endemic and exotic low virulence strains, and therefore all suspect cases of IBD have to be reported to MAF.
2. The commercial poultry sector is highly integrated, with birds of superior genetic merit coming from a small number of specialist international companies. There is, in effect, a small gateway into the industry. The right to enter a property to test for IBD virus, and if infection is found to enforce 'good disease control practice' (such as quarantine, disposal of infected birds, disinfection), has been obtained by way of a legal contract between the owners of commercial flocks and the primary breeding companies, rather than by reliance on the statutory powers that are available for dealing with notifiable organisms.

Conclusions

Given the success of the IBD control efforts to date, the poultry industry is confident that the virus has been eradicated from all commercial properties and that within a short period all will achieve accredited-free status. Issues involving the non-commercial poultry population will then have to be addressed.

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Figure 1: Number of IBD seropositive meat and layer farms detected during each year over the period 1993 to 1999

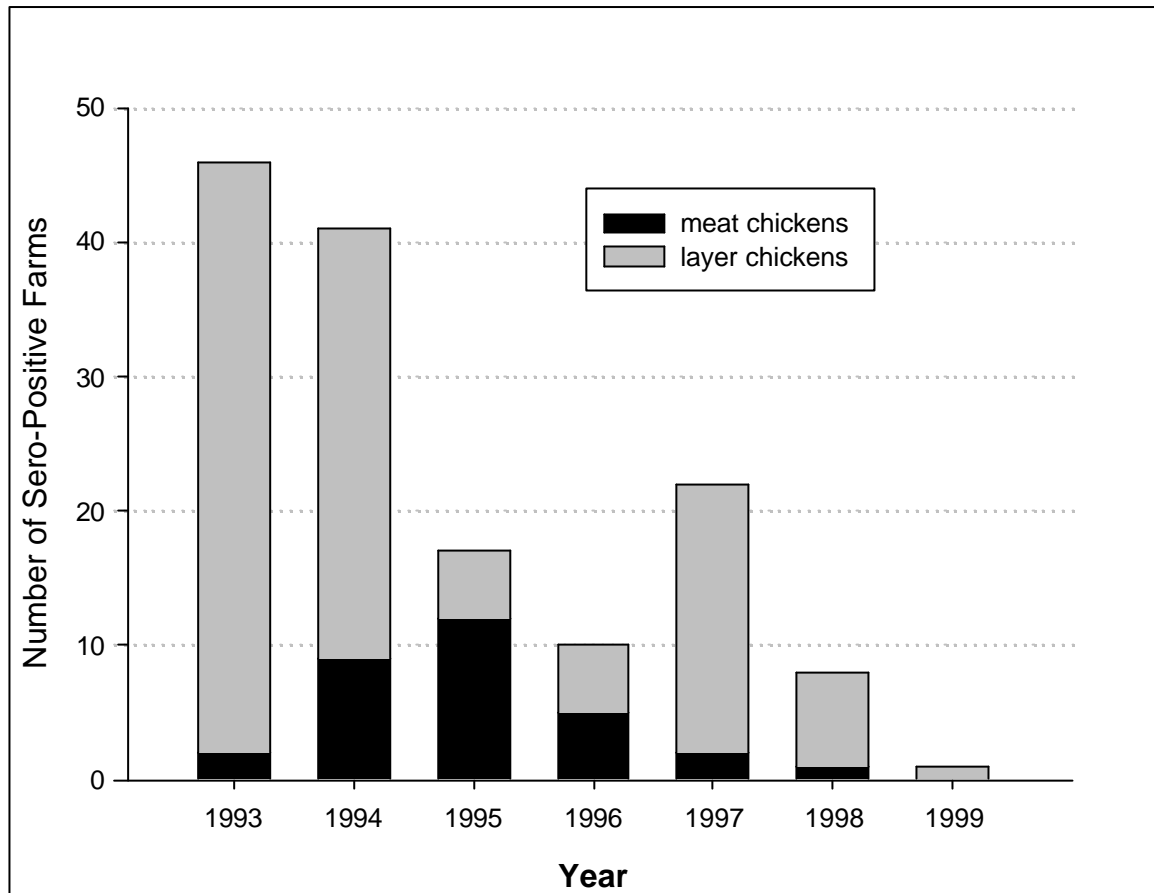


Figure 2: The pathway leading to ‘Declaration of country freedom from IBD’. Adapted from OIE pathways developed for rinderpest and bovine contagious pleuropneumonia

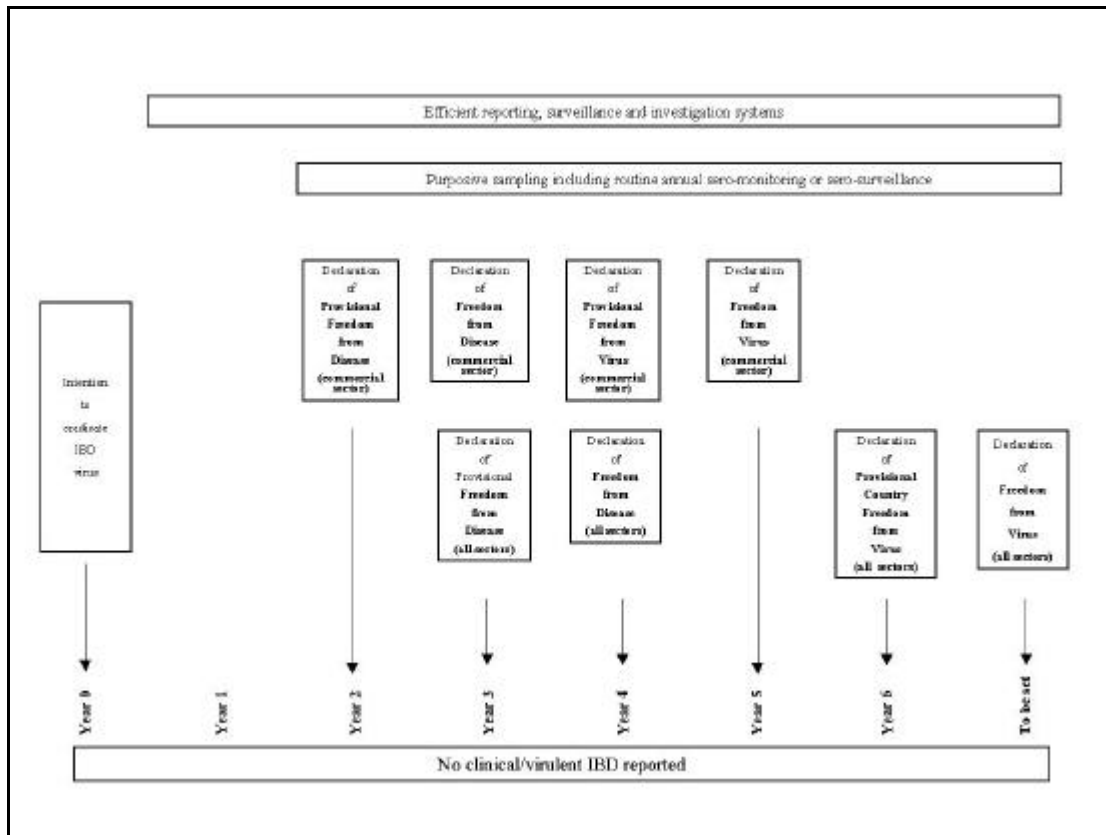


Table 1: Serological testing of commercial broiler flocks by year

Year	Flocks Tested	Number positive	
		Flocks	Farms
1993	707	4	2
1994	2570	26	9
1995	2200	26	12
1996	707	9	5
1997	789	6	2
1998	1023	3	1
1999	863	0	0

Table 2: Serological testing of commercial layer flocks by year

Year	Flocks Tested	Number positive	
		Flocks	Farms
1993	n.a.	n.a.	44
1994	n.a.	n.a.	32
1995	166	7	5
1996	140	7	5
1997	265	47	20
1998	304	13	7
1999	422	1	1

n.a.: data not available

Appendix 2: NEW ZEALAND NEWCASTLE DISEASE STATUS³⁸

New Zealand has never had an outbreak of Newcastle disease. Avian paramyxovirus type 1 has been isolated from several avian species, but the intracerebral pathogenicity index of the isolates is less than 0.2 and the amino acid sequence of the fusion protein cleavage site is typical of viruses of low virulence.

The first serological evidence of avian paramyxoviruses in New Zealand poultry flocks was in 1966⁽¹⁾, the year that the V4 strain of avian paramyxovirus type 1 (APMV-1) was first isolated from chickens in Queensland, Australia⁽²⁾.

MAF's Animal Health Laboratories commenced passive surveillance for avian paramyxoviruses by attempting to isolate the virus from chickens submitted for investigations of respiratory disease or reduced egg production. Between 1972 and 1977, no paramyxoviruses were detected in the approximately 800 chickens submitted to the Ruakura and Wallaceville laboratories⁽³⁾.

However, in 1972 and 1973 serological evidence of APMV-1 infection was detected in poultry including 6- to 8-week-old broilers hatched from imported eggs and two of 37 commercial flocks in the Christchurch area⁽³⁾. Attempts to isolate APMV-1 from 460 imported eggs that failed to hatch were unsuccessful⁽³⁾.

Between 1975 and 1978 no paramyxoviruses were isolated from any of the 252 birds tested in a survey of seabirds at several locations in the southern half of the South Island. The survey included 54 red-billed gulls (*Larus novaehollandiae*), 58 black-backed gulls (*Larus dominicanus*), 40 white-fronted terns (*Sterna striata*), and 100 sooty shearwaters (*Puffinus griseus*)⁽⁴⁾. However, pre-export testing of pheasants (*Phasianus colchicus*) in 1973, and of peafowl (*Pavo cristatus*) in 1976, revealed serological evidence of APMV-1 in 75 of 220, and 4 of 6 birds, respectively⁽³⁾.

The first isolation of an APMV-1 virus in New Zealand was from wild birds in 1976. Samples were taken in March that year from 87 clinically normal mallard ducks (*Anas platyrhynchos*) from a wildlife refuge 30 km north of Dunedin. Six isolates were made from tracheal and/or cloacal swabs⁽⁴⁾, of which three isolates are still available (51/76, 131/76 and 132/76).

The first reported isolations of APMV-1 viruses from poultry were in February 1978 during investigations into ill-thrift, respiratory problems and low egg production in four South Island flocks⁽¹⁾. Other causes were found for the clinical syndromes, and it was concluded that in each case the viruses were incidental findings⁽¹⁾. In 1980 these four viruses, together with four isolates from the mallard ducks sampled in 1976⁽⁴⁾ and one virus isolated from a red-

³⁸ Pharo, H. Stanislawek, W. Thompson, J. *Surveillance*. In press.

breasted musk parrot (*Prosopiea tabuensis*) imported illegally from Fiji⁽⁵⁾, were classified by a number of methods. It was concluded that all nine viruses were avirulent⁽⁵⁾.

PASSIVE SURVEILLANCE IN POULTRY

The New Zealand poultry industry undertakes routine serological testing of commercial breeder flocks for APMV-1. Samples are taken from eight to ten birds at approximately 10-weekly intervals throughout the life of the flock. Broilers and commercial layer flocks are tested occasionally (Catherwood E, Poultry Industry Association of New Zealand, personal communication). The serological testing for APMV-1 antibodies is performed either by the poultry industry laboratories or by the Ministry of Agriculture and Forestry's Animal Health Reference Laboratory (formerly the Central Animal Health Laboratory) at the National Centre for Disease Investigation.

The results of the poultry surveillance from 1997 to 1999 are summarised in Table 1. The figures listed in the tables were published in *Surveillance*⁽⁶⁾⁽⁷⁾⁽⁸⁾. The positive sera have come from flocks either in Taranaki or West Auckland, and the virus does not usually carry over into the young flock following clean-out (Catherwood E, Poultry Industry Association of New Zealand, personal communication). However, the epidemiology of APMV-1 infection in these poultry flocks has not been elucidated, and there has sometimes been repeated detection of antibodies in successive flocks on the same site⁽⁹⁾.

RECENT SURVEYS IN WILD AND CAGED BIRDS

In recent years MAF has periodically carried out surveys of non-commercial avian species for paramyxoviruses. Surveys of wild ducks were carried out in 1989⁽¹⁰⁾, 1990⁽¹¹⁾, and 1997⁽¹²⁾, of feral pigeons and four species of wild and captive native birds in 1993⁽¹³⁾, and of caged and wild birds from 1997 to 1999⁽¹⁴⁾.

The results of these surveys are presented in Table 2. No APMV-1 viruses were isolated in the 1989 and 1990 duck surveys, possibly because they involved adult ducks shot in the duck-shooting season, which begins in May and goes through to around the end of July⁽¹¹⁾. The 1997 wild duck survey involved cage trapping earlier in the year when there was a high ratio of juvenile to adult ducks (Stanislawek WL, unpublished report, 1998), and this resulted in the isolation of 10 APMV-1 viruses from tracheal and cloacal swabs. At the same time, serological reactions to APMV-1 were found in 76% of ducks⁽¹²⁾.

The 1993 survey showed no evidence of APMV-1 in feral pigeons or four species of native birds⁽¹³⁾. Further survey work from December 1997 to April 1999 showed a low prevalence of antibody titres to APMV-1 in caged and wild birds (11/231 and 9/522, respectively), but no paramyxoviruses were isolated from any of the 291 cloacal swabs from these birds⁽¹⁴⁾.

NEW DEFINITION FOR NEWCASTLE DISEASE

Until recently the OIE definition of Newcastle disease was "a disease of birds caused by strains of avian paramyxovirus type 1, significantly more virulent than lentogenic strains"⁽¹⁵⁾.

This definition rested on the grouping of Newcastle disease viruses into five pathotypes on the basis of clinical signs seen in infected chickens⁽¹⁶⁾. Since there has never been any evidence of pathogenic strains of APMV-1 in New Zealand, this country has always been considered free of Newcastle disease.

However, the lack of objectivity in assigning viruses to the pathotype groups has caused difficulties in international trade, and led to the development of other tests to distinguish between strains. The most widely used tests are the intracerebral pathogenicity index (ICPI) in day-old chicks and the intravenous pathogenicity index (IVPI) in 6-week-old chickens⁽¹⁶⁾.

More recently, sequencing studies of the fusion protein of paramyxoviruses have elucidated a molecular basis for pathogenicity⁽¹⁷⁾. It appears that the amino acid sequence at the cleavage site of the virus fusion protein is a key determinant for infectivity and pathogenicity. Strains of the virus with multiple basic amino acids at the F₀ cleavage site are virulent for their hosts, whereas strains with a single basic residue are avirulent⁽¹⁸⁾. The most common amino acid sequences at the fusion protein cleavage site for strains of low virulence are ¹¹²G/E-K/R-Q-G/E-R-L¹¹⁷⁽¹⁹⁾.

Thus, at the 67th General Session of the International Committee of the OIE the definition of Newcastle disease was changed as follows⁽²⁰⁾:

“Newcastle disease is defined as an infection of birds caused by a virus of avian paramyxovirus serotype 1 (APMV-1) that meets one of the following criteria for virulence:

- a) The virus has an intracerebral pathogenicity index (ICPI) in day-old chicks (*Gallus gallus*) of 0.7 or greater.

or

- b) Multiple basic amino acids have been demonstrated in the virus (either directly or by deduction) at the C-terminus of the F2 protein and phenylalanine at residue 117, which is the N-terminus of the F1 protein. The term ‘multiple basic amino acids’ refers to at least three arginine or lysine residues between residues 113 and 116. Failure to demonstrate the characteristic pattern of amino acid residues as described above would require characterisation of the isolated virus by an ICPI test.”

(In this definition, amino acid residues are numbered from the N-terminus of the amino acid sequence deduced from the nucleotide sequence of the F₀ gene, 113-116 corresponds to residues -4 to -1 from the cleavage site.)

The new definition of Newcastle disease has implications for international trade. Firstly, the classification of APMV-1 isolates into pathotype groups is no longer adequate. Secondly, an infection of any bird species with a virus fitting the new definition would affect the status of the country concerned, as disease in poultry is no longer the only focus.

PATHOGENICITY OF ISOLATES

Of the 17 APMV-1 viruses isolated in New Zealand, four are from chickens, one from a parrot, and 12 from wild mallard ducks. In view of the new definition, it became necessary to characterise these 17 isolates using biological and molecular approaches. The results are summarised in Table 3.

To assess the ICPI, day-old specific pathogen free chickens were inoculated intracerebrally with the viruses and scored according to the clinical signs observed over the next 8 days, following the method outlined in the *OIE Manual of Standards*⁽¹⁶⁾. When NCDI carried out the ICPI determination, none of the viruses produced clinical signs in the test chickens, meaning the ICPI value for all isolates was 0.0 (Stanislawek WL, unpublished report, 1996). The isolates were sent to one of the OIE Reference Laboratories for Newcastle disease, the Central Veterinary Laboratory (CVL) in the UK, to get a second ICPI estimate. In early 2000, CVL reported that 10 of the New Zealand isolates had been confirmed as having ICPI values of 0.0, and seven had non-zero ICPI values ranging from 0.02 to 0.16 (Stanislawek WL, unpublished data).

The fusion protein gene cleavage site of the 17 APMV-1 isolates was initially determined at NCDI and confirmed at another OIE Reference Laboratory, the Australian Animal Health Laboratory, using RT-PCR techniques (Stanislawek WL, unpublished data). The deduced amino acid sequences of the F2/F1 cleavage site for 16 of the New Zealand isolates was ¹¹²GKQGRL¹¹⁷. For one duck isolate the sequence was ¹¹²ERQGRL¹¹⁷. Both sequences are typical for viruses of low virulence⁽¹⁹⁾.

RISK OF MIGRATORY BIRDS INTRODUCING VIRUSES

APMV-1 viruses have frequently been isolated from aquatic birds, and although most isolates have been of low virulence for chickens, it has been suggested that under certain circumstances migratory birds might play a role in the spread of Newcastle disease⁽²¹⁾. The most significant outbreaks of Newcastle disease in wild birds have been reported in double-crested cormorants (*Phalacrocorax auritus*) in North America in the 1990s. The wide geographical spread of the outbreaks suggested that adult birds acquired infection before migration to their breeding sites, with subsequent spread to nestlings. At the same time there was an outbreak of Newcastle disease in a flock of range turkeys near an affected cormorant colony, and the turkey and cormorant isolates were closely related⁽²²⁾.

The outbreaks of Newcastle disease that occurred in UK in 1997 provided further evidence of migratory bird involvement. Genetic analysis indicated close similarities between viruses isolated from domestic chickens and turkeys in UK and viruses present in Scandinavia at the same time⁽²³⁾, and it was suggested that unusual migratory patterns of waterbirds may have been responsible for the virus introduction. In Northern Ireland, four of the first five cases in the 1997 epidemic occurred within 3 km of major waterways, although a survey of resident

and migratory waterfowl and waders from 14 nearby overwintering sites failed to isolate any APMV-1 viruses⁽²⁴⁾.

In New Zealand, banding studies show that wild ducks are not migratory, although they do disperse widely throughout the country⁽²⁵⁾. Further, there is no evidence that Palaearctic ducks reach New Zealand⁽²⁶⁾, so although ducks might play a role in spreading endemic APMV-1 viruses within this country, the risk of them introducing new strains appears to be minimal.

Apart from seabirds, most birds migrating to this country are shorebirds of the family *Scolopacidae* (sandpipers and allies) in the order *Charadriiformes* which breed in the Arctic regions of Europe, Asia and North America, and migrate south for the boreal winter. The most numerous species that migrate to New Zealand are the bar-tailed godwit (*Limosa lapponica*), lesser knot (*Calidris canutus*), ruddy turnstone (*Arenaria interpres*), curlew sandpiper (*Calidris ferruginea*), red-necked stint (*Calidris ruficollis*) and pacific golden plover (*Pluvialis fulva*)⁽²⁵⁾. Although an APMV-1 virus was isolated from one species of sandpiper of the genus *Calidris* in Western Australia in 1979-80⁽²⁷⁾, a review of APMV-1 in wild birds did not mention any of the species that migrate to New Zealand⁽²⁸⁾. MAF's 1997 survey of wild birds included 27 waders: 26 lesser knots and one bar-tailed godwit. Six of the lesser knots were positive for APMV-1 antibodies, which suggests that it is not uncommon for these birds to become infected with the viruses, but none was isolated from any of the birds in this study⁽¹⁴⁾.

For migratory waders to be shedding APMV-1 virus when they arrive in New Zealand, they would have to become infected either prior to migration, and continue shedding throughout migration, or somewhere along the migratory route. The migratory routes are usually not completely understood, but breeding occurs at low latitudes in the Arctic, and migration typically involves long non-stop flights over the west Pacific Ocean between only a few staging areas, during which they live off body reserves. For example, the sub-population of lesser knot that migrates to New Zealand breeds on the Chukotsky Peninsula of eastern Siberia, and begins to head south in late August. About a month later the first birds arrive in New Zealand, and migration is complete by December. Evidence from radar studies at Guam suggests that the lesser knots fly non-stop from Siberia to staging areas in southeast Irian Jaya and the Gulf of Carpentaria. The peak time for passing through the Gulf is September and October, with numbers dropping off by December. From there they either head for southeast Australia or direct to New Zealand. Some birds come to New Zealand via southeast Australia, but most come direct⁽²⁹⁾. Therefore the opportunity to pick up virus infections during migration is probably limited, but it could possibly happen at staging grounds such as in the Gulf of Carpentaria where large numbers of birds from different flight groups congregate.

However, even if they were shedding virus on arriving in New Zealand, waders would have little opportunity to pass APMV-1 viruses directly to domestic poultry. But if an indirect exposure pathway did exist, and if new viruses were introduced into poultry flocks, the consequences could be severe, as there is now good evidence that APMV-1 viruses of low virulence may mutate to become highly virulent *after* their introduction into chickens. Results from viruses isolated from Newcastle disease outbreaks in Ireland and Australia during the 1990s suggest that this may be how some virulent viruses emerge, and that perhaps as few as two point mutations may be required⁽³⁰⁾.

CONCLUSION

New Zealand has never had an outbreak of Newcastle disease. Serological surveillance indicates that APMV-1 viruses are widespread in many species of wild bird and are periodically present in commercial poultry. Pathogenicity testing and fusion protein genotyping of the 17 APMV-1 viruses isolated in this country have confirmed that all isolates are avirulent. That is, none fits the definition of Newcastle disease virus adopted in 1999 by the OIE, and under the old terminology they would be classified as asymptomatic enteric strains.

Little is known about the epidemiology of New Zealand's endemic APMV-1 viruses in wild birds and commercial poultry, including potential transmission pathways within and between these species. Further studies are required to identify reservoir hosts and to explain how poultry flocks become infected. There is a theoretical possibility that migratory waders could introduce exotic APMV-1 viruses. In view of the potentially serious consequences, further surveillance of migratory waders is justified to determine the risk.

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Table 1: Serological surveillance for APMV-1 in poultry flocks			
Year	Number of sera tested	Number of sera positive	Reference
1997	8,372	202	6
1998	8,113	12	7
1999	9,892	281	8

Table 2: Surveys for avian paramyxoviruses in New Zealand native, feral, and caged birds from 1989 onwards

Year	Bird species surveyed	TIME OF YEAR	Samples taken	Location	Findings	REFERENCE
1989	Ducks, wild, shot by hunters Mallard ducks (<i>Anas platyrhynchos</i>)	May-July	277 faecal samples 45 cloacal swabs 19 tracheal swabs 3 pond water samples	16 areas (12 North Island, 4 South Island)	no APMV isolated	10
1990	Ducks, wild, shot by hunters Mainly mallard ducks (<i>Anas platyrhynchos</i>); also a few paradise shell ducks (<i>Tadorna variegata</i>), and grey ducks (<i>Anas castanea</i>)	May-July	85 faecal samples 52 cloacal swabs 66 tracheal swabs 5 pond water samples	16 areas (same as 1989 survey)	one isolation of APMV-4	11
1993	Pigeons 54 feral pigeons (<i>Columbia livia</i>) Native birds 40 kaka (<i>Nestor meridionalis</i>) 12 kea (<i>Nestor notabilis</i>) 7 weka (<i>Gallirallus australis</i>) 2 New Zealand pigeons (<i>Hemiphaga novaeseelandiae</i>)	April-August	54 feral pigeons: • 53 serum samples • 54 tracheal swabs • 54 cloacal swabs • 54 faecal samples • 54 intestinal tissue samples 61 native birds: • 55 serum samples • 61 cloacal swabs • 61 faecal samples	Feral pigeons: • Auckland • Wellington • Christchurch Native birds: • Kapiti Is. • Little Barrier Is. • Auckland Zoo • Wellington Zoo • Mt Bruce • Peacock Springs	no APMV isolated, no antibody to APMV	13
1997	Ducks 346 mallard ducks (<i>Anas platyrhynchos</i>), mostly juveniles, trapped in wire mesh traps	January-March	315 serum samples 321 tracheal swabs 321 cloacal swabs	North Island: • Bay of Plenty • Feilding • Carterton South Island: • Temuka • Invercargill	Sera: 76% positive for APMV-1 Swabs: 33 viruses isolated from 28 ducks (10 tracheal, 23 cloacal), of which 10 viruses were APMV-1	12, ^a

[Continues next page...]

[...Table 2 continues from previous page.]

1997-1999	231 caged birds of 24 species in 4 orders: 126 Psittaciformes 51 Passeriformes 35 Galliformes 19 Columbiformes	December 1997 to April 1999	231 serum samples 116 cloacal swabs	14 sites • 9 North Island • 5 South Island	Sera: 11 positive for APMV-1 Swabs: no APMV isolated	14
1997-1999	522 wild birds of 24 species in 9 orders: 456 Passeriformes 31 Charadriiformes 17 Psittaciformes 6 Columbiformes 4 Gruiformes 3 Falconiformes 1 Anseriformes 1 Galliformes 1 Coraciiformes	December 1997 to February 1999	522 serum samples 155 cloacal swabs	13 sites • 7 North Island • 6 South Island	Sera: 9 positive for APMV-1 Swabs: no APMV isolated	14

^a Stanislawek WL, unpublished report, 1998

Table 3: Classification of New Zealand isolates of APMV-1 by ICPI and fusion protein amino acid sequence

PMV-1 isolate	Species of origin	Isolation		ICPI ^a		Amino acid sequence at F0 cleavage site, from position 112 to 117 ^a
		Date	Reference	Maximum	Number of replicates	
51/76	Duck	1976	4	0.11 ^b	2	GKQGRL
131/76	Duck	1976	4	0.02 ^b	2	GKQGRL
132/76	Duck	1976	4	0.14 ^b	2	GKQGRL
78/3528	Parrot	1976-78	5	0.04 ^b	3 ^c	GKQGRL
79/7579	Chicken	1976-78	5	0.00	3 ^c	GKQGRL
8038	Chicken	1976-78	5	0.00	3 ^c	GKQGRL
8043	Chicken	1995	a	0.00	3 ^c	GKQGRL
1/97	Duck	1997	12	0.00	3 ^d	ERQGRL
2/97	Duck	1997	12	0.00	2	GKQGRL
3/97	Duck	1997	12	0.00	2	GKQGRL
4/97	Duck	1997	12	0.00	2	GKQGRL
5/97	Duck	1997	12	0.00	3 ^d	GKQGRL
6/97	Duck	1997	12	0.00	2	GKQGRL
7/97	Duck	1997	12	0.00	2	GKQGRL
8/97	Duck	1997	12	0.10 ^b	2	GKQGRL
9/97	Duck	1997	12	0.10 ^b	2	GKQGRL
10/97	Duck	1997	12	0.16 ^b	2	GKQGRL

^a Stanislawek WL, unpublished data

^b The ICPI value shown was determined at CVL; when tested at NCDI the ICPI value was 0.0

^c Two ICPI replicates at NCDI, one done at CVL

^d One ICPI replicate done at NCDI, two done at CVL

APPENDIX 3: Second revision of quantitative IBD risk assessment

The original quantitative assessment of IBD risk³⁹ was revised⁴⁰ on the basis of submissions made by USDA⁴¹. The significant revision was to the estimates of the age at which chickens first become infected with IBD virus, whether vaccinal strains or field strains.

A key variable in the risk assessment model is R2, the probability of there being active IBD virus infection present in chickens at the time of slaughter. The various estimates used to calculate this variable in the two earlier risk assessments are compared below in Table 1. Only the inputs for boneless products are reproduced here, as in both models the lowest risk of introduction was associated with these products, because the probability of boneless products generating scraps is less than for carcasses and bone-in products.

Table 1: Inputs used to estimate R2, the probability of active IBD infection at the time of slaughter (for boneless products).

	Original risk assessment	Revised risk assessment
Age at slaughter, days	BetaPERT 32,37,49	Uniform 42,63
Age at first infection, days	Uniform 1,49	Modified BetaPERT 1,21,56
Duration of muscle infectivity, days	Uniform 2,6	Uniform 2,6

The results of the original and revised risk assessments are shown below in Table 2.

³⁹ *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.

⁴⁰ *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.

⁴¹ *Import risk analysis*: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Review of Submissions. 21 September 1999.

Table 2: Summary of model results: probability of establishment of IBD in backyard flocks fed cooked scraps derived from imported boneless chicken products, given three levels of consumption of imported product.

Consumption of imported chicken (expressed as a percentage of current chicken consumption)	Mean result	95th percentile
0.1%	Original = 0.13 Revised = 0.00008	Original = 0.31 Revised = 0.0006
1%	Original = 0.68 Revised = 0.0008	Original = 0.97 Revised = 0.006
10%	Original = 0.96 Revised = 0.008	Original = 1.0 Revised = 0.06

Age at slaughter

In the original risk analysis, MAF considered that the age at which broilers are slaughtered is anywhere between 32 and 49 days of age. However, in its submission on that analysis, USDA asserted that "most" broilers in the United States are slaughtered between 42 and 56 days of age, with the youngest slaughter age being reported as 35 days. They also stated that birds used to produce de-boned meat may be slaughtered as late as 63 days of age.

These figures do not reflect current industry practice in New Zealand (See Table 3).

Table 3: Age at which chickens are slaughtered in New Zealand.⁴²

Company A		Company B		Company c	
				30-32 days	6%
				33-35 days	45%
				37-39 days	19%
35-42 days	70%	35-44 days	58%	40-42 days	21%
42-48 days	30%	44-48 days	42%	43-46 days	9%

While practice in the United States might be a little different from industry practice in this country, with perhaps a very small number of broilers being slaughtered as old as 63 days, we doubt that birds as old as that contribute significantly to the production of chicken meat.

⁴² RJ Diprose, Poultry Industry Association of New Zealand, emails to SC MacDiarmid, 4 and 8 September 2000. These three companies produce around 93% of all the broilers processed in New Zealand, approximately 67 million.

Clearly, modern industry practice worldwide is for the bulk of birds to be slaughtered in the younger age ranges.

One source⁴³, citing the US National Chicken Council, implies that the mean slaughter age of US broiler chickens is 47 days. Additional information on ages at which chickens are slaughtered in the US was obtained by the New Zealand Embassy in Washington DC.⁴⁴ USDA figures for the period January to June 2000 are summarised in Table 4.

Table 4: Annualised broiler performance for different bird weight. USDA Agri Stats Special Report #32. January to June 2000.

	<3.60 lb.	3.60-4.40 lb.	4.40-5.20 lb.	5.20-6.00 lb.	>6.00 lb.
Average weight (lb.)	3.49	3.92	4.83	5.54	6.65
Age (days)	40.13	41.11	46.65	50.93	57.01
Percentage birds in class	24%		19%	33%	24%

In the revised risk assessment MAF used different age at slaughter inputs for boneless and bone-in products. However, on the basis of the most recent USDA submission (Submission 4 above), further exploration of common industry practices, and the slaughter age data obtained by the New Zealand Embassy, MAF considers that an appropriate input for age at slaughter is described by Histogram⁴⁵ (40,57,0.24,0.19, 0.33, 0.24) where 40 and 57 represent the minimum and maximum ages at slaughter and subsequent values are the weights reflecting the probability of occurrence of a value within an age class.

Age at which chickens become infected

The age at which chickens become infected with IBD virus is a key factor in calculating R2, the probability of active infection at the time of slaughter.

On the basis of the evidence submitted by the Poultry Industry Association of New Zealand (Submission 10 above), a re-examination of the original risk assessment, and new evidence, MAF considers that the original estimates for age at first infection are more appropriate than those used in the revised assessment. In particular:

- Dr Stewart-Brown, Perdue Poultry Company, is cited (Submission 10 above) as stating that 70% of Perdue's broiler chickens get to 28 days without field infection. This is not congruent with the assertion about age at first infection made by USDA in its submission on the original risk assessment. This suggests that the "most likely" value used in the modified BetaPERT distribution in the revised risk assessment is inappropriately low, thus leading to an underestimation of risk.

⁴³ <http://www.tyson.com/investorrel/factbook/efficiency.asp>

⁴⁴ Facsimile from Jason Frost, New Zealand Embassy, Washington DC, to Stuart MacDiarmid, 13 September 2000. "Annualized broiler performance for different bird weight. Agri Stats Special Report #32. January-June 2000."

⁴⁵ Histogram distribution function. @Risk, Palisade Corporation, NY, USA.

- Tyson Foods, another major US poultry producer, does not, “as a rule”, vaccinate broilers against IBD.⁴⁶ Instead, broilers are provided with maternally-derived immunity induced by vaccination of layers.
- Seaboard Farms, another US poultry producer, aims to start its broiler chicks with a high, consistent maternal antibody level to protect them from field strains of IBD virus until the immune system becomes functional.⁴⁷ However, chicks are also vaccinated at a day old and on some farms booster vaccination is applied on the basis of performance and bursal regression. This implies that broilers are protected by maternal antibodies for a variable length of time and that vaccination of broilers is sometimes necessary in the face of exposure at some time after maternal antibodies have waned.
- Dr Margaret McKenzie asserts (Submission 10 above) that infection with IBD can occur at any age during the growout cycle in US broiler flocks.
- The Poultry Industry Association of New Zealand's submission (Submission 10 above) cites a number of sources supporting the contention that it is not appropriate to take 21 days as the "most likely" age by which chickens have become infected with IBD virus.
- That chickens may become infected close to slaughter is demonstrated by the observation that researchers in the United States have been able to detect IBD virus in samples collected from chickens at the time of slaughter in at least one slaughterhouse.⁴⁸

On the basis of the evidence, MAF no longer considers it tenable to assert that most US chickens become infected with IBD virus by 21 days of age. Because of the uncertainty surrounding the issue, MAF considers that the best that can be said is that chickens become infected with IBD virus sometime between hatch and slaughter, with most birds probably becoming infected sometime after maternal immunity has waned. For this reason, in calculating R2, the probability that a chicken is infected with IBD virus at the time of slaughter, the risk assessment uses a uniform distribution of 1 to 57 days as the input for age when chickens become infected. The lack of credible data precludes attempts to achieve greater precision.

⁴⁶ L. Fussell, Director of Veterinarian Service, Tyson Foods. Vaccination strategies: Tyson Foods. Proceedings International Poultry Symposium. Summit on Infectious Bursal Disease. 3-4 April 1995, University of Georgia. Watt Publishing. 15-16.

⁴⁷ B L McMurray, Director of Veterinary Services, Seaboard Farms. 1995. Vaccination strategies: Reflections from the field. Proceedings International Poultry Symposium. Summit on Infectious Bursal Disease. 3-4 April 1995, University of Georgia. Watt Publishing. 35-37.

⁴⁸ "...we were able to detect IBDV in chickens after they arrived at a processing plant in the U.S." Daral J. Jackwood, Ph.D., Food Animal Health Research Program, The Ohio State University/OARDC. Email to S C MacDiarmid, 21 August 1998.

"We tested bursa from chickens at the time of slaughter and were able to detect IBDV. The samples were from one slaughter plant in the USA and I do not know if the virus detected was vaccine or pathogenic wild type." Daral J. Jackwood, Ph.D., Food Animal Health Research Program, The Ohio State University/OARDC. Email to H J Pharo, 28 August 1999.

Duration of IBD infectivity in muscle tissue

No evidence was submitted or found through further research which would suggest that any change is needed to the estimate used in either of the earlier risk assessment. Consequently, MAF uses a uniform distribution of 2 to 6 days when re-assessing the IBD risk from boneless chicken meat product.

Re-assessment of IBD risk from boneless chicken meat products

The quantitative assessment of the risk that IBD virus might be introduced through importation of US boneless chicken meat was revised for a second time. The inputs used in this second revision are shown below in Table 5.

Table 5: Inputs used in the second revised assessment to estimate R2, the probability of active IBD infection at the time of slaughter (for boneless products).

Age at slaughter, days	Histogram (40,57,0.24,0.19, 0.33, 0.24)
Age at first infection, days	Uniform 1,56
Duration of muscle infectivity, days	Uniform 2,6

In Table 6, the results of the second revision are compared with those of the original assessment and the first revision.

Table 6: Summary of model results: probability of establishment of IBD in backyard flocks fed cooked scraps derived from imported boneless chicken products, given three levels of consumption of imported product.

Consumption of imported chicken (expressed as a percentage of current chicken consumption)	Mean result	95 th percentile
0.1%	Original = 0.13 First revised = 0.00008 Second revised = 0.15	Original = 0.31 First revised = 0.0006 Second revised = 0.34
1%	Original = 0.68 First revised = 0.0008 Second revised = 0.68	Original = 0.97 First revised = 0.006 Second revised = 0.98
10%	Original = 0.96 First revised = 0.008 Second revised = 0.96	Original = 1.0 First revised = 0.06 Second revised = 1.0

APPENDIX 4: QUANTITATIVE ASSESSMENT OF THE RISK OF INTRODUCTION OF IBD VIRUS IN IMPORTED BONELESS CHICKEN MEAT PRODUCTS AND ITS ESTABLISHMENT IN BACKYARD FLOCKS: ORIGINAL MODEL WITH REVISED INPUTS.

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.⁴⁹ 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 become 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.

Focusing 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 Excel⁵⁰ and @Risk⁵¹. The structure of the model is shown in Figure 1.

Commodities Considered

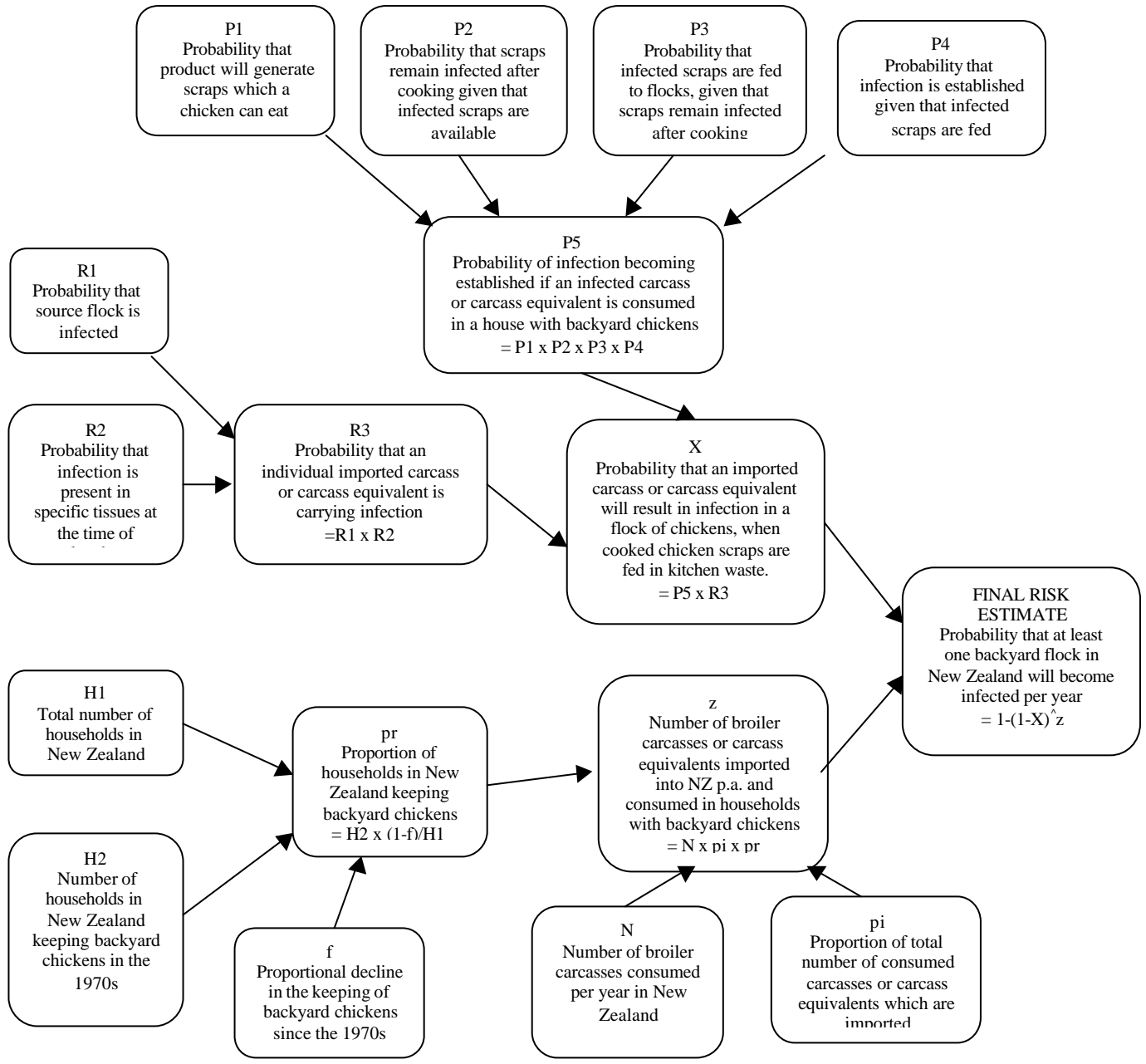
This revision examines only the risk posed by boneless cuts which earlier assessments demonstrated posed the lowest risk of introducing IBD.

⁴⁹ Diprose R, Poultry Industry Association of New Zealand, Personal communications with SC MacDiarmid, June and August 1998.

⁵⁰ Microsoft Corporation, USA.

⁵¹ 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 focuses primarily on the risk of backyard poultry flocks becoming infected with a virulent field strain of IBD virus should boneless chicken meat products be imported from the United States. 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.

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 are seldom handled by humans and have little contact with other similar flocks. 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.⁵²

The magnitude of the risk posed by the importation of chicken meat products obviously depends on the quantity imported. It is a truism that risk is proportional to volume of trade.⁵³ 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 equivalent.

As risk always increases with volume of commodity imported, the model also considered the effect of three levels of consumption of imported chicken.

Release Assessment: probability of infection in imported chicken meat products

The release assessment estimates the probability that an imported chicken 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;

⁵² N Christensen, Avivet, Christchurch, New Zealand, Personal communication with SC MacDiarmid, January 1999.

⁵³ “Risk increases with increasing volume of commodity imported.” *International Animal Health Code*. Article 1.3.2.3. Office International des Epizooties, Paris, 2000.

R1 Probability that the source flock is infected.

In the original *Risk Analysis*⁵⁴ R1 was modelled as a Pert distribution with a minimum of 30%, a most likely value of 70% and a maximum of 90%. This was based on published reports. After examination supplied by the USDA MAF considered it reasonable to accept the USDA's position that "less than 5% of US flocks have not been exposed to IBD virus." Therefore, the first revision⁵⁵ modified the distribution for R1 to a uniform distribution with a minimum of 90% and a maximum of 99%. This same uniform distribution has been retained for the second (current) revision. These values are shown below in Table 1.

Table 1: Values used for probability that source flock is infected with field or vaccine strains of IBD virus.

Original assessment	First revision	Second [current] revision
Pert (0.3, 0.7, 0.9)	Uniform (0.9, 0.99)	Uniform (0.9, 0.99)

R2 Probability of active infection when slaughtered.

In estimating this probability, the following need to be considered:

- The age of chickens at slaughter;
- The age at which chickens become infected;
- Duration of tissue infectivity.

The probability of active infection in muscle at the time of slaughter is modelled by the following beta distribution:

- Beta (1669, 18332)

(See *Note I* for method of calculation and explanation.)

R3 The probability that an imported carcass is infected,
 $R3 = R1 \times R2$

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.

⁵⁴ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. March 1999.*

⁵⁵ *Import risk analysis: chicken meat and chicken meat products; Bernard Matthews Foods Ltd turkey meat preparations from the United Kingdom. Revised quantitative risk assessments on chicken meat from the United States; Reassessment of heat treatment for inactivation of Newcastle disease virus in chicken meat. 7 April 2000.*

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.

It was considered that the probability of boneless chicken meat products generating scraps would be low, not greater than 1%, but not zero. The distribution for this probability used in the model for boneless cuts was:

$P1 = \text{Uniform}(0, 0.01)$

$P2$ Probability that infected scraps remain infected after cooking. (See *Note II* 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,⁵⁶ the titres of virus reported in *Note II*, and what is known about the age structure of backyard poultry flocks.

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.

$$P5 = P1 \times P2 \times P3 \times P4$$

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 equivalent imported.

As shown in Figure 1, this is the product of the probability that an imported carcass equivalent 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*.

⁵⁶ Quality Control Unit (1997) Study Report: dissemination of infectious bursal disease virus in chickens infected with the very virulent strain CS88. Study number CVLS/07/97, Contract number FT0518. Central Veterinary Laboratory, United Kingdom.

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 carcass equivalents, 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 equivalents likely to be imported per year:

N Broilers consumed each year in New Zealand,⁵⁷

$$N = 6.30 \times 10^7$$

pi The proportion of consumed carcasses equivalents 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. Assuming that importation of chicken meat products would not result in a change in total consumption of poultry meat in this country, if imported chicken were consumed at a volume of only 1% of current local consumption, this would be equivalent to 630,000 carcass equivalents in a year.

To model the effects of different assumptions regarding volumes of consumption the Monte Carlo model carried out three simulations for boneless chicken product. The values used for volume of consumption in these simulations are shown in Table 2.

Table 2: Values used in the model for volumes of imported boneless chicken meat consumed.

	Estimates of Imported Commodity Consumed		
	Low	Medium	High
Boneless cuts	0.1%	1%	10%

Assuming that imported chicken meat would just as likely be consumed in households that keep backyard chickens as in households which do not (that is, consumption is uniform) the probability that imported chicken meat 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:

⁵⁷ Diprose R, Poultry Industry Association of New Zealand, Personal communication with SC MacDiarmid, 11 December 1997.

- 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,⁵⁸

$$H1 = 1.21 \times 10^6$$

$H2$ Last figure for households keeping backyard poultry flocks, 1970s,⁵⁹

$$H2 = 7.00 \times 10^4$$

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.

$$f = \text{Uniform}(0.4, 0.6)$$

pr Proportion of households currently keeping backyard poultry.;

$$pr = [H2 \times (1-f)] / H1$$

Therefore,

z Number of 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 volumes of imported boneless cuts consumed.

⁵⁸ New Zealand Official Yearbook. 97th edition. Statistics New Zealand, Auckland, 587 pages, 1994.

⁵⁹ The New Zealand People 1971: Vol 12 : general report on the census of population and dwellings. Wellington, Department of Statistics, 164 pages, 1977.

For each volumes of imported product consumed 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 95th percentile of iterations. In other words, in 95% of iterations the result was less than the quoted figure.

The 95th 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 3.

Table 3: Summary of revised model results for boneless chicken products.

Volume of Current Consumption	Mean Result	95 th Percentile Result
0.1%	0.15	0.34
1%	0.68	0.98
10%	0.96	1

Conclusion

Under the assumptions used, if boneless chicken meat products from 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 0.34 if as few as 0.1% of the chicken carcass equivalents consumed in New Zealand were to be imported.

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 between 40 and 57 days of age (see above, Table 5, Appendix 3).
- Chickens become infected between 1 and 57 days of age,
- Virus is recoverable from muscle tissue for 2-6 days post-infection.^{60 61}

A Monte Carlo model was constructed on the following assumptions for products containing chicken meat only.

	Chicken muscle
A1, age of chicken at slaughter, in days	Histogram (40, 57, 0.24, 0.19, 0.33, 0.24)
A2, age of chicken at first infection, in days	Uniform (1, 57)
D, duration of tissue infectivity, in days	Uniform (2, 6)

The likelihood of IBD virus being present in processed boneless products may actually be higher than one might assume on the basis of the data for chicken muscle. For example, a recent US publication⁶² suggested that the amount of virus present in products such as processed chicken patties could be higher than in cuts such as drumsticks "...due to contamination with **IBDV-infected bursa tissue that remains in the carcass.**" [Emphasis added.]

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 1 (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;

⁶⁰ Quality Control Unit (1997) Study Report: dissemination of infectious bursal disease virus in chickens infected with the very virulent strain CS88. Study number CVLS/07/97, Contract number FT0518. Central Veterinary Laboratory, United Kingdom.

⁶¹ Lukert P (1999). Persistence of IBD in infected SPF chickens. Final report to Ministry of Agriculture and Forestry, January 1999.

⁶² Mandeville, WF, Cook, FK, Jackwood, DJ. Heat lability of five strains of infectious bursal disease virus.. Poultry Science 79: 838-842, 2000.

$\text{Beta}(k \times \text{mean} + 1, k \times (1 - \text{mean}) + 1)$

where k is the number of iterations (20,000) and mean is the mean output of the model.

The model output for chicken muscle, and the resulting beta distribution used for modelling R_2 in the main model were :

	Chicken muscle
Number of iterations, k	20000
Mean output of the sub-model	0.0741
$\text{Beta}(k \times \text{mean} + 1, k \times (1 - \text{mean}) + 1)$	Beta (1483, 18519)

Note II: 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.⁶³ 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.^{64 65}

The dissemination study demonstrated that IBD virus CS88 was present in muscle, bone marrow, bursa, liver/kidney, blood, spleen and faeces of infected chickens.

The heat inactivation study demonstrated that IBD virus in tissue homogenates survived high temperatures for an unexpectedly long time. For example;

Temperature	Time (minutes)	Titre (CID ₅₀)
80°C	90	<10 ^{0.83}
80°C	30	<10 ^{2.17}
80°C	15	10 ^{2.68}
80°C	5	10 ^{4.16}
74°C	90	10 ^{0.5}
74°C	30	10 ^{2.63}
74°C	15	10 ^{3.68}
74°C	5	10 ^{4.17}
70°C	210	10 ^{2.3}
70°C	240	10 ^{2.17}
70°C	270	10 ^{2.17}
70°C	300	10 ^{1.3}
70°C	300	10 ^{1.38}

⁶³ MacDiarmid S C (1991) *The Importation into New Zealand of Meat and Meat Products: A review of the risks to animal health*. MAF, New Zealand.

⁶⁴ Quality Control Unit (1997) Study Report: dissemination of infectious bursal disease virus in chickens infected with the very virulent strain CS88. Study number CVLS/07/97, Contract number FT0518. Central Veterinary Laboratory, United Kingdom.

⁶⁵ Quality Control Unit (1997) Study Report: heat inactivation of infectious bursal disease virus strain CS88. Study number CVLS/06/97, Contract number FT0517. Central Veterinary Laboratory, United Kingdom.

The report on the study of the heat inactivation of IBD virus in tissue homogenates,⁶⁶ states that “The virus was unexpectedly resistant to prolonged heating at high temperatures. A previous experiment⁶⁷ demonstrated that IBDV was inactivated by heating at 70°C for 60 minutes, 75°C for 45 minutes and 80°C for 10 minutes.” (The results of a recently published study “compare well” with this earlier one.⁶⁸)

The report continues:

“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 70°C and 15 minutes at 80°C 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 $1 \times 10^{2.2}$ 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 85°C.⁶⁹ Two cooking methods are used :

- Pan frying at 160°C for 12.5 minutes, the largest piece being 180 g, including bone.
- Pressure cooking at 171°C for 14 minutes.

The holding cabinet temperature is 82°C and the minimum temperature of chicken as it goes over the counter is 60°C.

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;⁷⁰

⁶⁶ Quality Control Unit (1997) Study Report: heat inactivation of infectious bursal disease virus strain CS88. Study number CVLS/06/97, Contract number FT0517. Central Veterinary Laboratory, United Kingdom.

⁶⁷ Alexander D J, Chettle NJ (1998) Heat inactivation of serotype 1 infectious bursal disease virus. *Avian Pathology* 27, 97-9.

⁶⁸ Mandeville, WF, Cook, FK, Jackwood, DJ. Heat lability of five strains of infectious bursal disease virus. *Poultry Science* 79: 838-842, 2000.

⁶⁹ Parker R, Logistics Manager, Kentucky Fried Chicken Head Office, Auckland. Telephone conversation with Martin van Ginkel, November 1997.

⁷⁰ Anon, (1997) [Technical Series] Use a Meat Thermometer. Technical Information from FSIS. United States Department of Agriculture Food Safety and Inspection Service. (<http://www.fsis.usda.gov/OA/pubs/cithermo.htm>)

Health Canada recommends an internal temperature of 85 °C;⁷¹ and Australian authorities recommend cooking to at least 75 °C⁷².

Given that the 1997 United Kingdom study⁷³ showed that chicken which had been heated to 80°C for 15 minutes still contained IBD virus at a titre of 10^{2.68} CID₅₀/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.

⁷¹ Anon, (1998) [Technical Series] Let's Talk Turkey. Health Canada Publications, Ottawa, Ontario, Canada. (<http://www.hc-sc.gc.ca/main/hc/web/datahpb/datafood/english/iyh/turkey.htm>)

⁷² Anon, (1999) [Technical Series] Cooking and Reheating. The Australian Food Safety Campaign Group's Food Safety Tips. ([Http://www.safefood.net.au/foodf/food_august/tips/cook.html](http://www.safefood.net.au/foodf/food_august/tips/cook.html))

⁷³ Quality Control Unit (1997) Study Report: heat inactivation of infectious bursal disease virus strain CS88. Study number CVLS/06/97, Contract number FT0517. Central Veterinary Laboratory, United Kingdom.