

***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

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Approved for general release

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EXECUTIVE SUMMARY

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.

New information received from the USDA since the MAF review of submissions allowed a reassessment of the risk of introduction infectious bursal disease (IBD). Although the revised risk estimates were lower than in the original assessment, they still led to the conclusion that the importation of carcasses and bone-in chicken meat products would require the application of the sanitary measures recommended in the original chicken meat import risk analysis (i.e. flock freedom).

However, the results indicated that providing boneless chicken meat imports do not exceed 1% of current consumption (that is, equivalent to around 500 tonnes of boneless chicken per year), this would pose very little risk of introducing IBD so long as the birds were not vaccinated with live IBD vaccines in the 21 days prior to slaughter and provided the age of the birds at slaughter was not less than 42 days.

Because of the effect that quantity has on risk, should the amount of boneless chicken imported exceed 500 tonnes per year MAF will need to reassess the sanitary measures necessary to protect against IBD, probably requiring demonstration of flock freedom as required for carcasses and bone-in cuts.

Based on information received from the USDA since the MAF review of submissions, a quantitative model was developed to assess the risk of for Newcastle disease (PMV-1) in chicken meat imported from the USA. The model indicated that there is a negligible risk of introducing vaccinal strains of PMV-1 virus, but it was not possible to conclude that the risk of introducing field strains of PMV-1, whose presence in source flocks would be likely to be masked by vaccination, would be similarly negligible. Although the risk of exposure is low, given the severe consequences of introduction, this analysis concluded that assurances are required to ensure that broiler flocks have not been exposed to field strains of PMV-1 within the week prior to slaughter.

For flocks not able to demonstrate freedom, revised standards for cooking chicken meat to inactivate PMV-1 viruses are recommended.

1 INTRODUCTION

In March 1999 MAF released for public consultation its *Import Risk Analysis: Chicken Meat and Chicken Meat Products; Bernard Matthews Foods Ltd Turkey Meat Preparations from the United Kingdom*⁽¹⁾. MAF received 12 submissions on the risk analysis and a review of these submissions was published in September 1999⁽²⁾.

The review of submissions identified three issues that remained outstanding:

- public health risks
- risk of introduction and establishment of infectious bursal disease (IBD)
- risk of introduction and establishment of Newcastle disease (ND)
- time/temperature requirements to inactivate Newcastle disease virus by cooking

As mentioned in the review of submissions, the Ministry of Health is carrying out an assessment of the public health impact of importing raw chicken meat which will be reported on separately.*

This document deals with the remaining three issues.

In the review of submissions, MAF noted that the USDA contended that the risks of introduction and establishment of IBD and ND viruses was not as high as suggested by MAF. In August and September MAF requested further information from the USDA on the following matters^(3, 4):

- current vaccination practices for ND and IBD
- rationale for those practices
- evidence of when infection with these viruses is likely to occur
- what are considered to be the most likely sources of infection in broiler flocks
- age of birds at slaughter

In November 1999, the USDA supplied detailed responses to MAF's questions⁽⁵⁾.

The above new information indicated that some of the inputs used in the IBD simulation model should be revised and that quantitative modelling of the ND risk was possible and appropriate.

Consequently, the IBD risk assessment model for chicken meat from the United States was revised. Further, a ND disease risk assessment applicable to importation of chicken from the United States, and based on the IBD model⁽⁶⁾, was developed. These quantitative assessments, which apply only to the United States, are described in this document. Similar risk assessments for chicken commodities from other countries could be developed if the competent authorities provided information similar to that provided by USDA.

In addition, further consideration has been given to the time/temperature requirements to inactivate ND virus by cooking.

* The original risk analysis also dealt with turkey meat preparations from a single British company, Bernard Matthews Foods Ltd. The risk analysis process concluded that these preparations should be permitted entry subject to specific measures designed to protect animal health. However, Ministry of Health has yet to conclude what sanitary measures are appropriate to manage public health risks.

2 RE-MODELLING THE RISK OF IBD INTRODUCTION

The information supplied by the USDA allowed MAF to revise the estimates for R1 and R2, which were used in the original model⁽⁶⁾ to calculate the risk of introducing IBD virus in imported chicken meat products.

2.1 R1: The probability that the source flock is infected

In the original MAF *Import Risk Analysis*⁽⁶⁾ R1 was modelled as a Pert distribution with a minimum of 30%, a most likely value of 70% and a maximum of 90%, which was based on published reports from Europe and the USA.

After examination of the new information supplied by the USDA, MAF considered it reasonable to accept the USDA position that “less than 5% of US flocks have not been exposed to IBD virus”⁽⁵⁾.

Therefore for remodelling the risk of IBD introduction the distribution for R1 was modified to a uniform distribution with a minimum of 90% and a maximum of 99%.

2.2 R2: The probability that tissues from a chicken will be carrying infection at slaughter

2.2.1 Age at first infection

In the original MAF *Import Risk Analysis*⁽⁶⁾ the age at first infection with IBD virus was considered to be anywhere from 1 day of age up until the maximum age at slaughter. A Uniform (1,49) distribution was used to model this. However, the new information provided by the USDA⁽⁵⁾ supported their contention in their submission on the risk analysis⁽²⁾ 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.

USDA’s justification⁽⁵⁾

- 1) IBD viruses are ubiquitous and persistent in the US broiler industry. Because of the expense and availability of litter, coupled with environmental waste disposal concerns, litter is, at best, replaced annually. As a result broilers are inevitably exposed to IBD viruses in nearly all houses from 1 day of age.
- 2) Effective prevention and control involves a breeder vaccination program, with the passive transfer of maternal immunity to broiler chickens, and a biosecurity program. A broiler vaccination program, using live vaccines, may also be undertaken.
- 3) Approximately 50% of broilers are vaccinated at 1 day of age to cover chicks that did not receive an adequate level of maternal antibody. Vaccination at this age establishes a reservoir of vaccine virus which allows lateral transmission to other chicks when their maternal immunity declines.
- 4) Some broiler flocks receive booster vaccinations between 10 and 21 days of age. The specific timing of boosters is generally on the basis of performance and bursal regression. Booster vaccines are more likely to be used during the winter months,

where cold temperatures and closed houses enhance the survival of the virus. Vaccine induced immunity occurs about 7 days post vaccination.

- 5) Broiler producers conduct routine monitoring of flocks for IBD to help adjust the disease control and vaccination practices. Monitoring programs include challenge tests to evaluate the efficacy of the breeder vaccination program, antigen capture ELISAs at 14 to 28 days of age and examination of bursal tissue between 17 and 24 days of age, when maternal antibodies are waning and the flock is experiencing peak exposure to field viruses. Serologic evaluation of broiler flocks is not conducted routinely. However, serologic data are available from "problem flocks" (i.e., flocks experiencing respiratory or enteric signs, etc.). In these cases IBD antibody titres at slaughter are invariably higher than would be predicted by the persistence of maternal antibody or response to live virus vaccines, confirming that exposure to IBD virus is a regular occurrence.
- 6) The presence or absence of clinical IBD depends upon the severity of field challenge and the success of the vaccination program. In areas where field challenge is present but not severe, a breeder vaccination program provides a titre of maternal antibody sufficient to prevent clinical disease while the chick develops active immunity to the field virus. In these situations, the use of live vaccine in broilers is not justified economically. Conversely, where the field challenge is greater, broiler vaccinations provide competition with, and active protection against, the field viruses. In either case "nearly all" the broilers will have been exposed to field virus by 28 days of age, regardless of whether or not live vaccines have been used.
- 7) The USDA estimates that less than 5% of US broiler flocks have not been exposed to IBD virus. A biosecurity failure could result in exposure to the virus through water, feed, faeces, or mealworms from a neighboring infected flock. This is the only subset of US broiler flocks where exposure and infection could occur at any time during the grow out period, as opposed to the majority of flocks which would be exposed by 28 days of age.
- 8) 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.

Revised model

A modified BetaPert distribution was developed to model the additional information provided by the USDA^(2, 5) that "nearly all" IBD infection in broilers occurs between 14 and 28 days of age. MAF interprets "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

The solver function in Microsoft Excel was used to determine the weight for a modified BetaPert distribution once the 90% a target value is set, as shown in Table 1, and the resulting distribution is plotted in Figure 1.

The output, for use in the spreadsheet model for the variable R2, is in the form of Beta ($a1, a2$)* $m+c$.

Table 1 : Calculations for a modified BetaPert distribution for age of chickens when infected with IBD virus.

| Input values | |
|--------------|------|
| minimum | 1 |
| most likely | 21 |
| maximum | 56 |
| weight | 33.3 |

= cell to change

| Calculated parameters | |
|-----------------------|-------|
| <i>a1</i> | 13.11 |
| <i>a2</i> | 22.19 |
| <i>m</i> | 55 |
| <i>c</i> | 1 |
| Distribution 90% | 21 |

| Area under the curve | |
|----------------------|-------------|
| 1 to 14 days | 0.04 |
| 14 to 28 days | 0.90 |
| 28 to 56 days | 0.06 |

= target cell

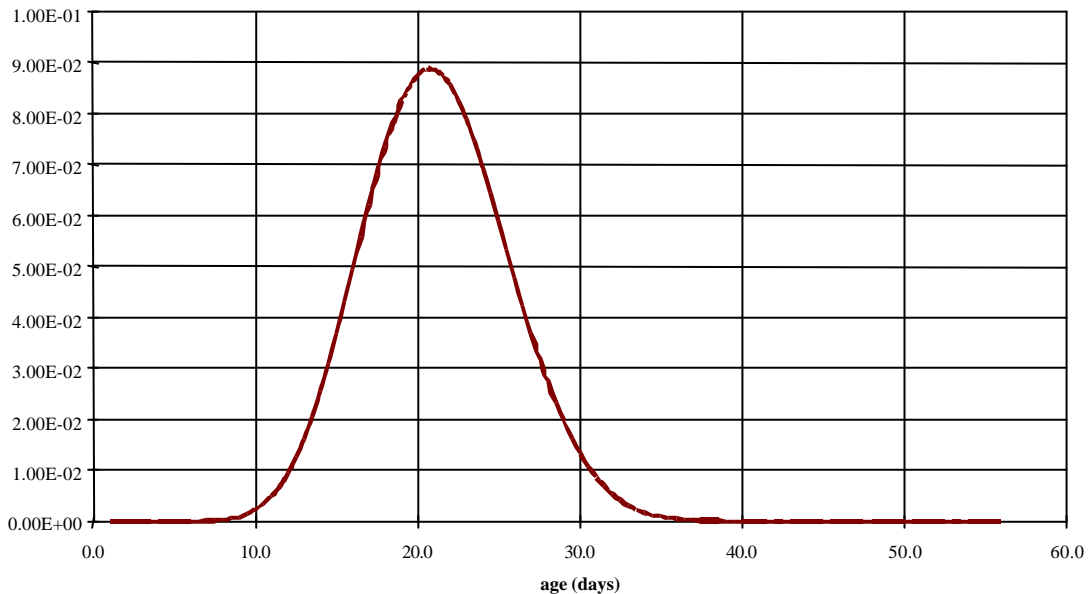


Figure 1: Modified BetaPert distribution for the age at infection with IBD virus.

2.2.2 Age at slaughter

This 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*⁽⁶⁾ this was considered to be anywhere from 32 to 49 days, but the USDA^(2, 5) 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⁽⁵⁾ 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”.

MAF does not wish to limit the model to considering only de-boned chicken as, despite UDSA’s assertion, MAF understands there is also interest in exporting 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.

2.2.3 Results

Figures 2 and 3 compare the results of the original model for $R2^{(6)}$ with the revised results for boneless, bone-in cuts and whole chickens at different ages.

Revising the inputs as outlined results in a reduction, of several orders of magnitude, in the estimate of the risk that boneless and bone-in cuts would be contaminated with IBD virus. Although there is a reduction in the estimated risk associated with whole chickens it is much smaller, and it is still within the same order of magnitude as previously.

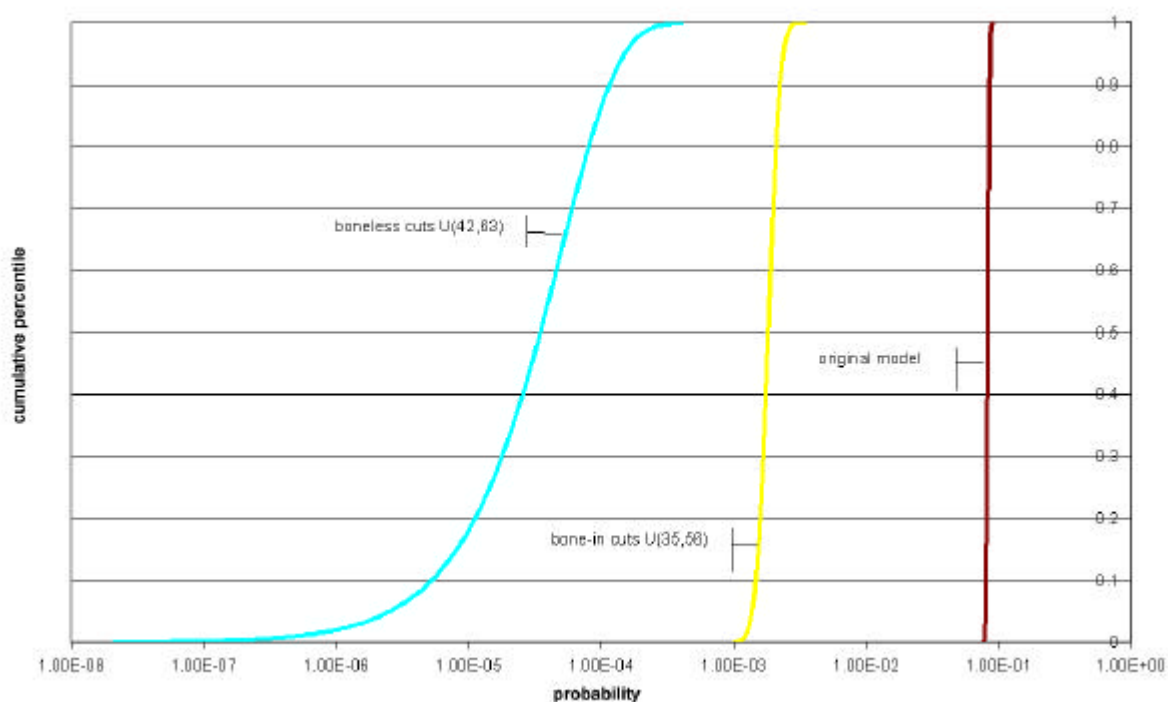


Figure 2: ($R2$) probability that boneless or bone-in cuts will be carrying infection at slaughter.

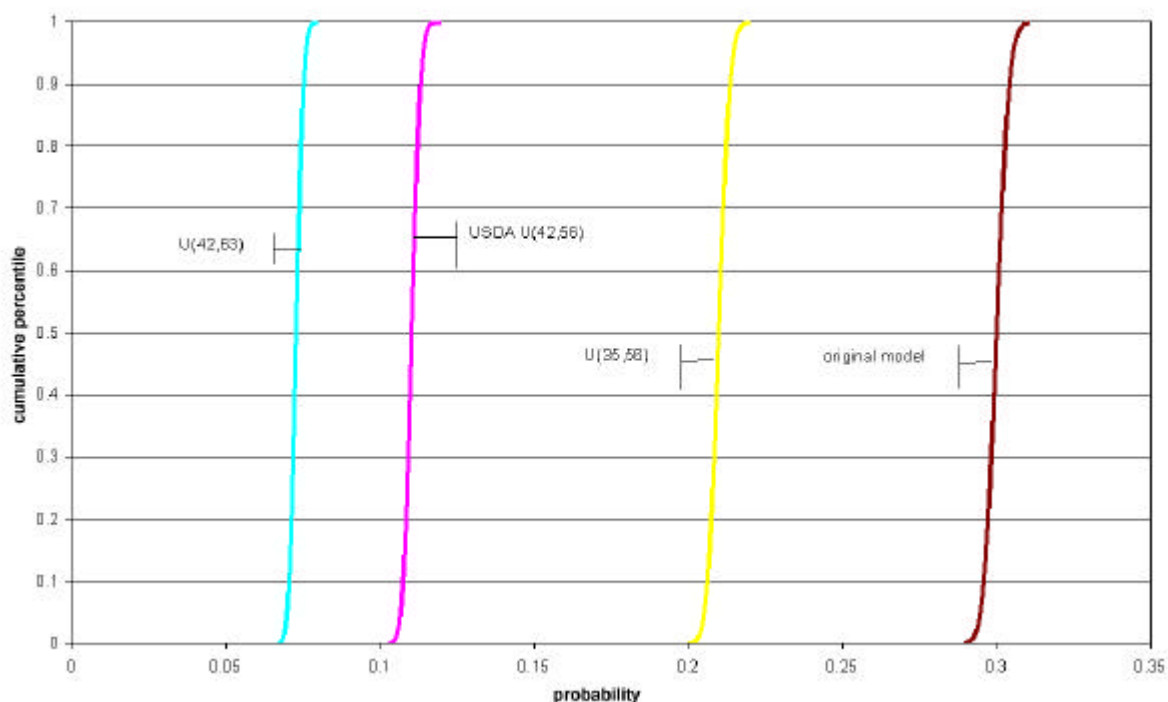


Figure 3: (R2) probability that a chicken carcass will be carrying infection when slaughtered at different ages.

2.3 Model output

Once revised estimates of R2 were obtained for the different chicken commodities, they were entered into the main simulation model⁽⁶⁾ to re-estimate the probability of IBD virus becoming established in backyard flocks in New Zealand.

Table 2 summarises the outputs from 10,000 iterations of the model. It presents the original and revised estimates of the probability of IBD becoming established in backyard flocks if they are fed cooked chicken scraps derived from imported chicken.

Figures 4, 5 and 6 compare the original and revised risk estimates for different quantities of imported boneless chicken product consumed, and Figure 7 is a summary graph of the revised risk estimates for three quantities of chicken consumption.

Figures 8, 9 and 10 compare the original and revised risk estimates for different quantities of imported bone-in chicken product consumed, and Figure 11 summarises the revised risk estimates for such products for three quantities of chicken consumption.

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

| Commodity | Consumption of imported chicken (expressed as a percentage of current chicken consumption) | Mean result | 95th percentile |
|--|--|--------------------------------------|-------------------------------------|
| Whole chicken carcasses (Note: the original and revised results are the same) | 1% | 1 | 1 |
| | 10% | 1 | 1 |
| | 20% | 1 | 1 |
| Bone-in cuts | 0.1% | Original = 0.26 Revised = 0.01 | Original = 0.52 Revised = 0.03 |
| | 1% | Original = 0.85 Revised = 0.098 | Original = .99 Revised = 0.26 |
| | 10% | Original = 0.99 Revised = 0.64 | Original = 1.0 Revised = 0.95 |
| Boneless cuts | 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 |

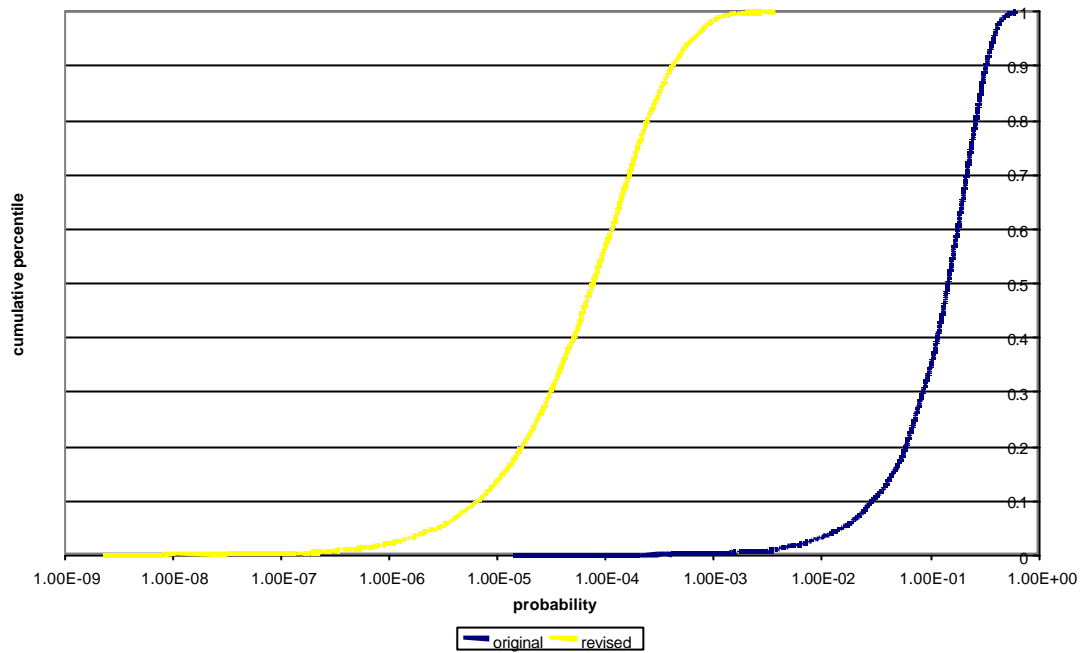


Figure 4: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if boneless cuts imported from the USA resulted in a consumption of imported chicken equivalent to 0.1% of current consumption.

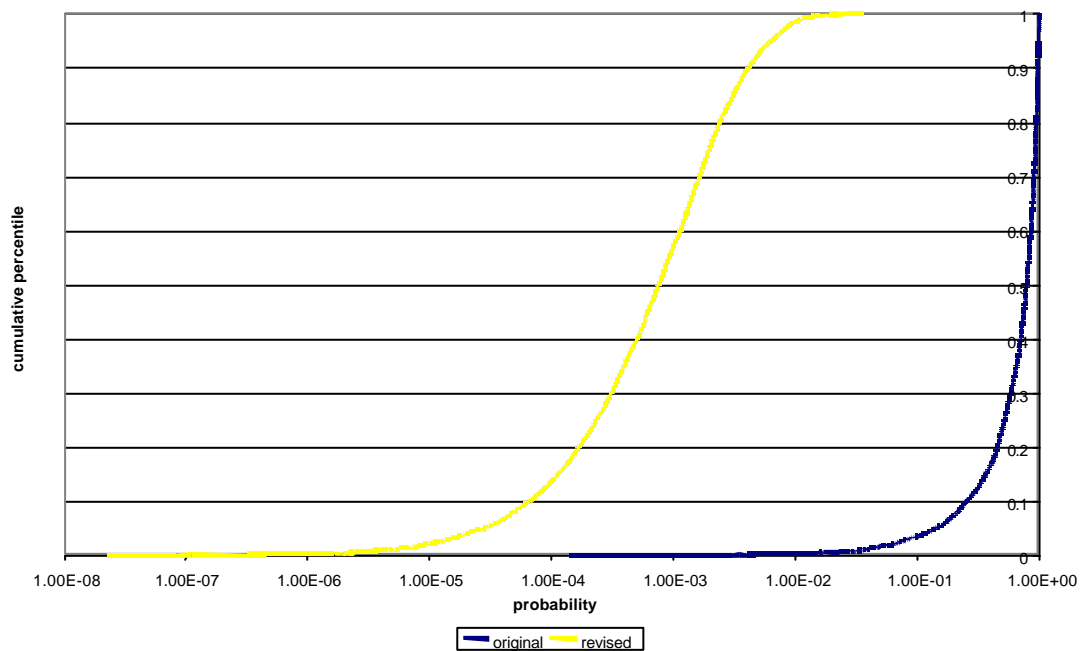


Figure 5: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if boneless cuts imported from the USA resulted in a consumption of imported chicken equivalent to 1% of current consumption.

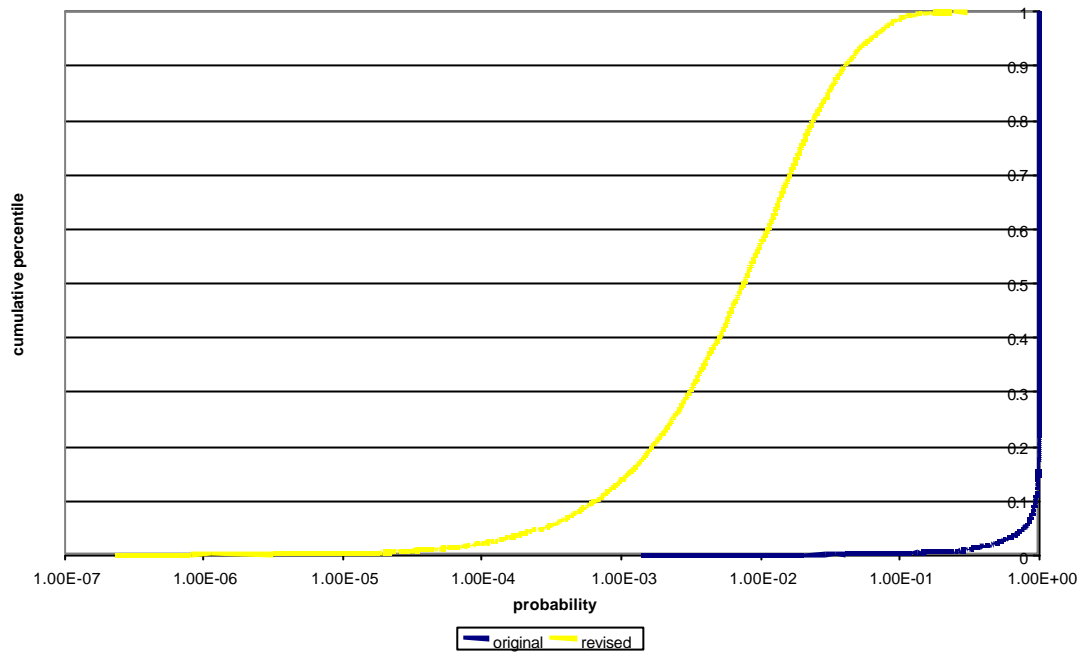


Figure 6: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if boneless cuts imported from the USA resulted in a consumption of imported chicken equivalent to 10% of current consumption.

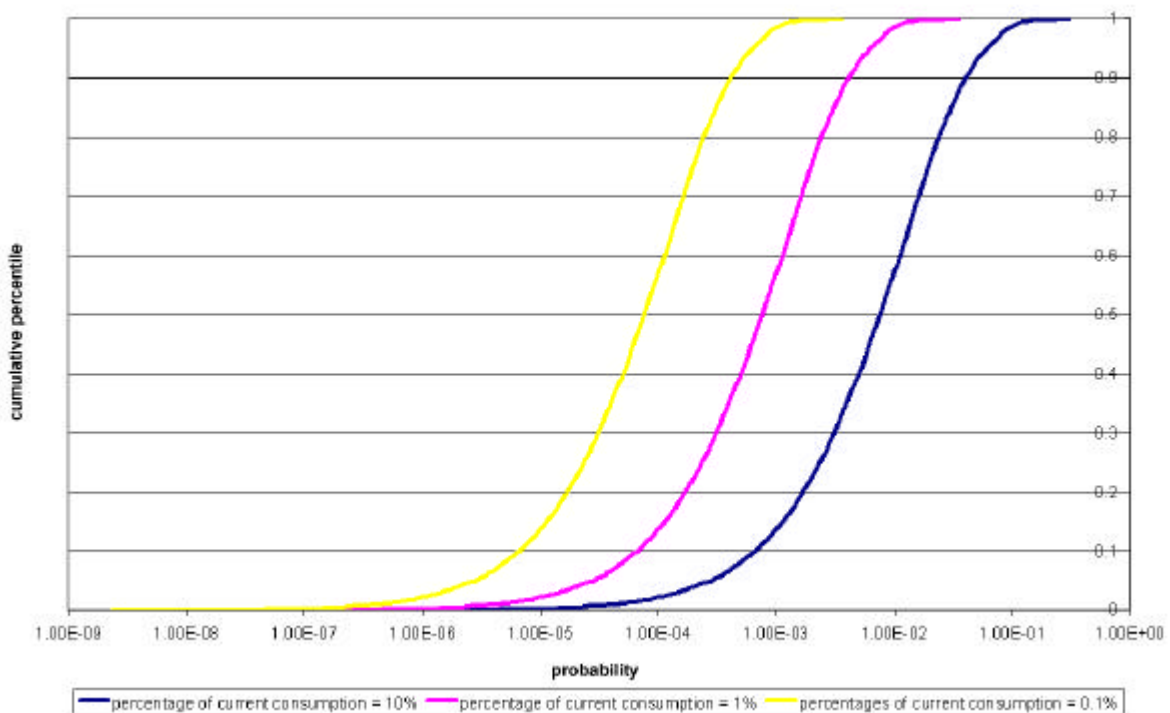


Figure 7: Revised probability that at least one backyard flock becomes infected per year if boneless cuts are imported from the USA in quantities equivalent to three different percentages of current consumption.

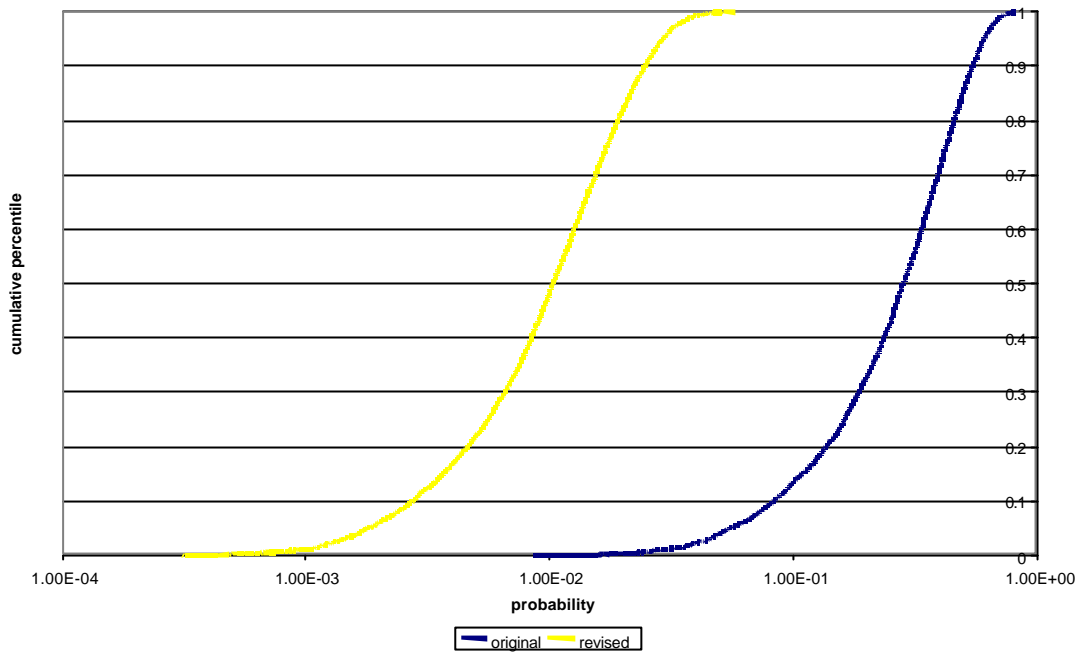


Figure 8: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if bone-in cuts imported from the USA resulted in a consumption of imported chicken equivalent to 0.1% of current consumption.

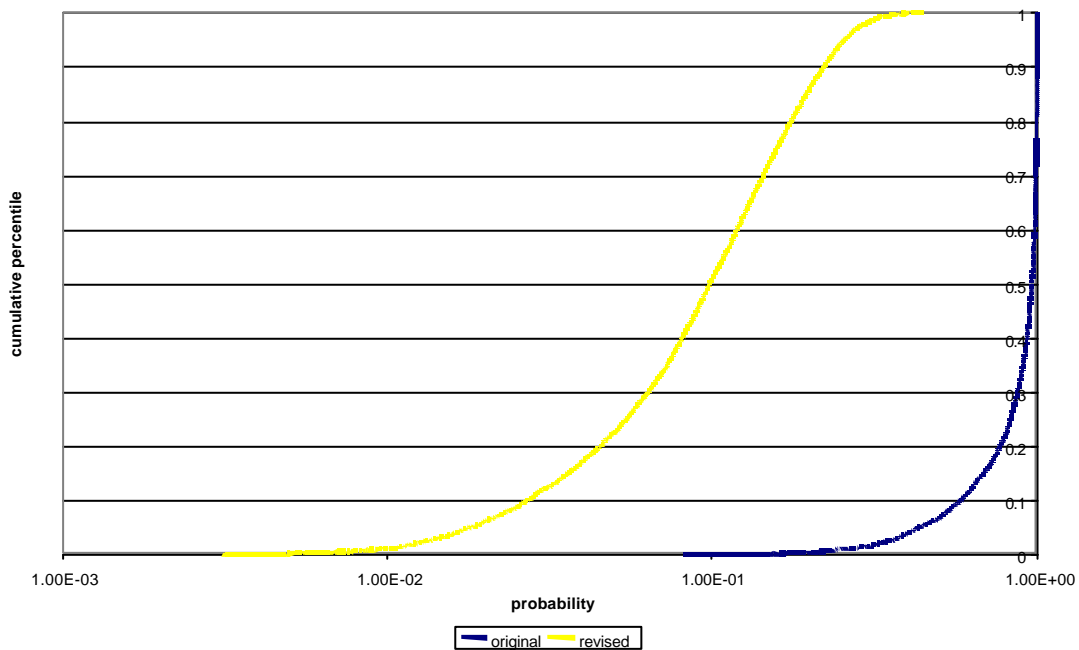


Figure 9: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if bone-in cuts imported from the USA resulted in a consumption of imported chicken equivalent to 1% of current consumption.

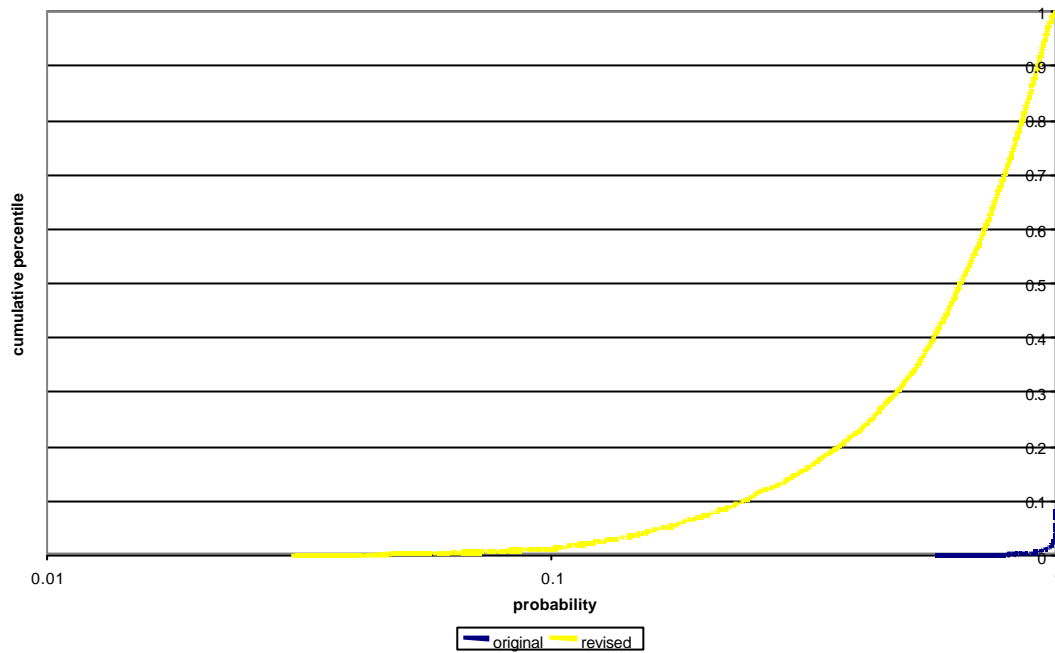


Figure 10: Comparison of the original and revised estimates of the probability that at least one backyard flock becomes infected per year if bone-in cuts imported from the USA resulted in a consumption of imported chicken equivalent to 10% of current consumption.

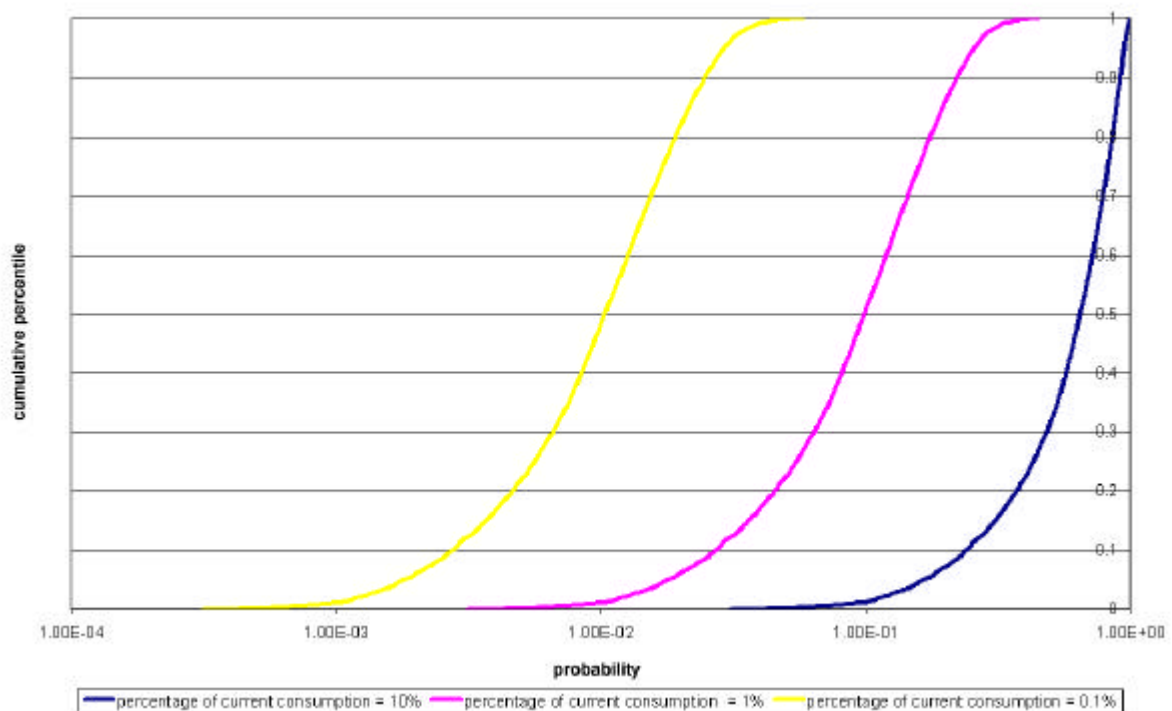


Figure 11: Revised probability that at least one backyard flock becomes infected per year if bone-in cuts are imported from the USA in quantities equivalent to three different percentages of current consumption.

2.4 Conclusions

The new information provided by USDA resulted, in all cases, in reduced estimates of the likelihood of IBD virus being present in tissues of broiler chickens at the time of slaughter.

Nevertheless, when this revised estimate was used in the reassessment of the risk of IBD becoming established in backyard flocks, the risk estimate for whole chicken carcasses remained unchanged. However, lower risk estimates were generated for bone-in and boneless cuts.

The risk estimate for bone-in cuts remained relatively high, even when the new information was used. Even with a consumption of US chicken equivalent to 0.1% of current New Zealand consumption, the upper 95th percentile of the estimated risk of IBD being introduced was 0.03 per year (that is, a risk of three introductions per 100 importation years). MAF considers this to be an unacceptably high risk.

The likelihood of IBD being introduced in boneless cuts was estimated to be considerably lower, provided birds were not slaughtered earlier than 42 days and were not vaccinated with live IBD vaccines in the 21 days prior to slaughter. As was shown in Figure 2, this is strongly influenced by the greater age at slaughter of the birds processed for boneless cuts. If the amount of US boneless chicken consumed was 0.1% of current New Zealand consumption[▲] (that is, around 50 tonnes) the 95th percentile of the risk estimate was 0.0006 per year (that is, less than 1 disease introduction per 1,000 importation years). MAF considers this risk to be acceptable.

If the amount of US boneless cuts consumed in New Zealand each year was equivalent to 1% of current consumption (that is, around 500 tonnes per year) the 95th percentile of the annual risk of IBD introduction was estimated to be 0.006 per year (that is, less than 1 disease introduction per 100 importation years). MAF considers this risk to be acceptable.

However, if US boneless cuts consumed were equivalent to 10% of current annual New Zealand consumption, the upper 95th percentile of the annual IBD risk was estimated to be 0.06 per year, or 6 introductions per 100 importation years. MAF considers this to be an unacceptably high risk.

Assuming that the quantity of boneless chicken meat imported from the United States, under an Import Health Standard developed from this risk analysis process, would be unlikely to exceed 500 tonnes per year, MAF concludes that the following sanitary measures are appropriate for IBD:

- a) Boneless meat must be certified to have come from birds which were not vaccinated with live IBD vaccines in the 21 days prior to slaughter

[▲] The quantitative model used the 1998 PIANZ figure of 63 million birds for total consumption of broilers in New Zealand. An amount equivalent to 0.1% of current consumption was calculated as 63 million x 0.001 = 63 thousand birds, which according to PIANZ would yield approximately 52 tonnes of boneless meat. On the same basis, an amount equivalent to 1% would comprise approximately 520 tonnes of boneless meat.

- b) Boneless meat must be certified to have come from birds which were at least 42 days old at slaughter.
- c) Other chicken meat products (carcasses and bone-in cuts) must be certified as originating from flocks demonstrated to be free from infection with IBD virus.

Because of the effect that quantity has on risk, should the risk of introducing IBD rise above that represented by the annual importation of 500 tonnes of boneless chicken MAF will need to reassess the sanitary measures necessary to protect against that disease, probably requiring demonstration of flock freedom as required for carcasses and bone-in cuts.

3 NEWCASTLE DISEASE RISK ASSESSMENT

3.1 Release assessment

The release assessment considers the probability that tissues from a chicken will be carrying the agent at slaughter. This depends on the prevalence of infection, the age at which chickens become infected, the duration of infection, and the age at slaughter.

3.1.1 Newcastle disease in the USA

The last outbreak of velogenic ND in poultry in the USA was in California in 1971-72. There was also an outbreak in 1988 in cormorants kept at a non-commercial facility in California. This latter outbreak did not spread and the outbreak was quickly contained, but the source of ND virus could not be determined. The only sources that could not be fully explored were traffic of people or undocumented movement of birds^(7, 8, 9).

USDA has indicated⁽⁵⁾ that there is no routine surveillance for ND in the USA. However, broiler flocks showing respiratory signs are often serologically tested for ND antibody, usually by the producers, who report to the USDA that titres are generally low, indicating that exposure to ND virus is the result of vaccination rather than natural infection. In addition, passive monitoring results in “opportunistic evaluation” of flocks or individual birds presented to university or state laboratories, a system which has identified “pathogenic ND” in non-commercial poultry and other avian species (wild birds, backyard chickens or smuggled pet birds). However, in commercial broilers this monitoring has only resulted in the isolation of lentogenic strains in recent years⁽⁵⁾.

According to the USDA⁽⁵⁾ “virtually all (100%) US broilers” are vaccinated with a live attenuated lentogenic Newcastle disease (ND) vaccine in the hatchery at 1 day of age. This is often followed by a second vaccination 10 to 14 days later. As a result the USDA⁽⁵⁾ claims that “all broilers in the US will be infected with attenuated live lentogenic vaccine strains at 14 days of age at the latest”. Vaccination is used as an insurance against virulent ND virus⁽⁵⁾, which caused devastating outbreaks in California and Florida in the 1970s^(5, 10, 11).

The outbreak of velogenic ND in Southern California in 1971-72 was preceded by a period when vaccination had not been widely practised, as there had been little problem with ND for several years⁽¹¹⁾. While vaccination was widely employed during this outbreak as a control measure, its application complicated eradication efforts. Surveillance initially relied on reports of sickness in chickens. However, it became apparent that although vaccination reduced losses, it masked clinical signs and did not prevent a flock from becoming infected. As a result, reservoirs of infection were established in apparently normal flocks and major changes in the approach to surveillance were required⁽¹¹⁾. Programmes were implemented involving the use of SPF sentinel chickens and the collection of dead chickens once a week for necropsy. Most new cases of ND were detected as a result of these intensified surveillance efforts⁽¹¹⁾. There have been several other reports of clinical disease being masked in vaccinated chickens experimentally challenged with velogenic ND^(12, 13, 14).

Parede and Young⁽¹²⁾ suggested that where vaccination is practised, or where lentogenic strains circulate, the introduction of velogenic ND virus could be masked. Recent experiences in Australia confirm that virulent ND virus can circulate in chicken flocks in the absence of overt clinical disease^(15, 16, 17, 18). In the Australian situation it appears likely that

the chickens were at least partially immunised against ND with a lentogenic field virus prior to infection with the virulent strain.

3.1.2 Duration of infection and distribution of ND virus in tissues

There is only a limited amount of information on the duration of infection and distribution ND virus in various tissues. Alexander⁽¹⁹⁾ determined the viral titres in a range of tissues and organs from 6-week old chickens experimentally infected ND virus Herts 33/56 strain, which is highly pathogenic. The highest titres were observed in all tissues examined at day 4 post infection. ND virus was isolated from muscle from days 2 to 4 and from a heart/kidney/spleen pool from days 1 to 4. The experiment could only be conducted over a 4 day period as all the chicks died, so subsequent trends in viral titres could not be determined. Alexander⁽¹⁹⁾ noted that little has been published on the titres of ND virus in tissues.

Lukert⁽²⁰⁾ inoculated 3-week old chickens with a mesogenic (Kansas-Manhattan) strain of ND virus. He was able to isolate ND virus from muscle tissue only on day 4 post infection. Muscle samples were negative by day 7. ND virus was isolated from liver, spleen, lung, kidney and bursal samples up until day 10 after which no further isolations were made. Failure to isolate ND virus after day 10 corresponded with peak titres of HI antibody being reached.

Although vaccinated chickens remain susceptible to infection with velogenic ND, the number of chickens shedding virus and the duration of virus excretion is inversely related to their immune status at the time of challenge⁽¹³⁾. Parede and Young⁽¹²⁾ isolated virus from various organs of chickens vaccinated with Hitchner B1 and experimentally challenged with velogenic ND for up to 22 days. Guittet et al⁽²¹⁾ isolated velogenic ND virus from various organs, including muscle, of chickens vaccinated with Hitchner B1 strain from 2 to 3 days post inoculation until the experiment ended on day 6.

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⁾.

During the course of infection of most birds with ND virus, large amounts of virus are excreted in the faeces. Therefore viruses may spread laterally within a flock by the faecal-oral route, and for lentogenic viruses which produce no respiratory signs, this is likely to be the most important form of spread⁽²¹⁾. For mesogenic vaccine strains the duration of shedding in faeces may be less than 12 days⁽²²⁾ or up to 19 days⁽²³⁾.

3.1.3 Model assumptions

3.1.3.1 Age at infection

As discussed in section 3.1.1, lentogenic vaccine strains are periodically isolated from commercial poultry in the USA. But as the USDA has not been able to provide surveillance data to allow the modelling of the circulation of field strains (lentogenic, mesogenic, or velogenic) an important assumption in the model is that any infection in broilers will be due to lentogenic vaccine strains.

Further, it is assumed that between 80% and 90% of broilers will be successfully vaccinated when an attenuated lentogenic vaccine is administered between 10 and 14 days of age. The remaining 10% to 20% of the flock will be susceptible to lateral transmission of the vaccine virus shed from birds that are successfully vaccinated.

A modified BetaPert distribution was developed to model this information. The solver function in Microsoft Excel was used to determine the weight for a modified BetaPert distribution once a target value was set (Table 3). The resulting distributions for the successful vaccination of 80% to 90% of broilers between 10 and 14 days of age are plotted in Figure 12. The output, for use in the spreadsheet model for the variable R2, is in the form of $Beta(a1, a2) * m + c$.

Table 3: Calculations for a modified BetaPert distribution for age of chickens when infected with ND virus.

| Input values | |
|--------------|------|
| minimum | 10 |
| most likely | 12 |
| maximum | 63 |
| weight | 70.9 |

= cell to change

| Calculated parameters | |
|-----------------------|-------|
| <i>a1</i> | 3.68 |
| <i>a2</i> | 69.25 |
| <i>m</i> | 53 |
| <i>c</i> | 10 |
| Distribution 90% | 12 |

| Area under the curve | |
|----------------------|------|
| 1 to 14 days | 0.90 |
| 15 to 63 days | 0.10 |

= target cell

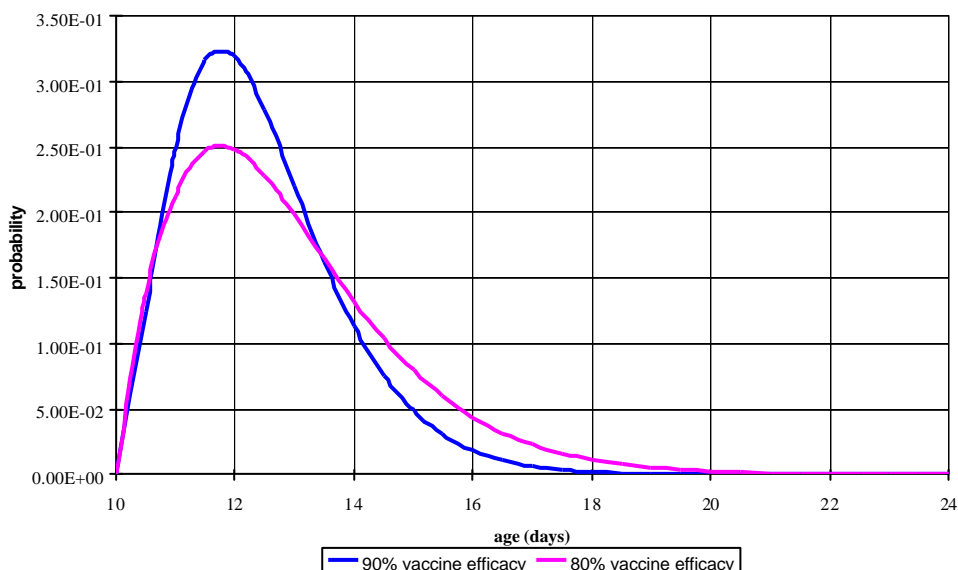


Figure 12: Modified BetaPert distribution for the age at infection with ND virus.

3.1.3.2 Duration of tissue infectivity

Based on the experimental findings outlined in 3.1.2, the duration of tissue infection with ND virus (in days) was modelled as Uniform(1,10) for whole chicken carcasses, which will have lung, kidney and bursal tissue present, and as Uniform(2,6) for boneless and bone-in cuts.

3.1.3.3 Age at slaughter

The age of birds at slaughter is discussed in Section 2.2.2 with respect to IBD. The same considerations are relevant for ND.

3.1.3.4 Modelling the release assessment

A simple algorithm was used in a spreadsheet model to determine if a broiler has an active infection at slaughter:

If the age at slaughter is greater than the age at infection then determine if the age at slaughter is less than or equal to the age at infection plus the duration of tissue infectivity. If it is, then the chicken has an active infection at the time of slaughter, otherwise it does not.

This model was simulated with @Risk* by running 100,000 iterations using Latin-hypercube sampling. None of the iterations resulted in a chicken having an active infection at the time of slaughter. That is, the results of the model indicate that broiler flocks in the USA, vaccinated between 10 and 14 days of age with an attenuated vaccine, pose a negligible risk of introducing vaccinal strains of ND virus (PMV-1) into New Zealand in chicken meat products derived from broilers slaughtered from 35 days of age.

3.2 Exposure assessment

In contrast to IBD, which is a heat-resistant virus, the only risk of imported chicken meat causing an outbreak of Newcastle disease in New Zealand would be that posed by **raw** scraps.

It is difficult to generalise about the probability of raw scraps being generated by different poultry products, as there are many and varied practices for preparation of food using poultry meat. For example, while it could be argued that scraps are unlikely to be generated from whole carcasses if it is assumed that the carcass is roasted, it could just as well be argued that certain people might purchase whole carcasses (for example because they may be cheaper), and then in preparation for cooking, dismember them for a certain recipe, thereby generating raw scraps in the form of muscle and organ scraps on the frame. Similarly, although most drums, wings and thighs could be expected to be cooked whole, some consumers might remove some or all of the skin or even bone prior to cooking. And while skinless, boneless breast meat might not need trimming prior to cooking, it cannot be ruled out that some consumers would do so for individual reasons.

Thus, plausible scenarios can be imagined for each commodity whereby raw muscle scraps could be generated.

* Palisade

3.3 Consequence assessment

In Appendix 1 of the review of submissions⁽²⁾ MAF presented surveillance information which indicates that none of the New Zealand isolates of PMV-1 has an intracerebral pathogenicity index (ICPI) of greater than 0.0, meaning that none of these viruses cause any signs of disease when injected into chickens. As discussed in the original MAF *Import Risk Analysis*⁽²⁵⁾, the immunologically naive status of the avian population within New Zealand means that mesogenic, and particularly velogenic, strains of PMV-1 virus would be almost certain to cause devastating outbreaks of disease if introduced into this country. In addition, MAF takes the position that some lentogenic strains (i.e. ICPI<0.7) may cause some clinical disease in poultry, and as it is difficult to predict how such viruses would behave if they were introduced into populations of endangered native birds in this country, MAF is obliged to be vigilant against **all** PMV-1 viruses.

3.4 Risk estimation

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 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 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. PMV-1 virus is heat labile and, as confirmed in the original risk analysis, even relatively light cooking will render products safe. The exposure risk is therefore dependent on the generation of uncooked scraps, and these finding their way into susceptible hosts. Depending on the method of preparation prior to cooking it could be argued that the probability of discarding raw scraps from some poultry meat products (particularly skin-off boneless breast) would be low.

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.

3.5 Risk management

The objective of risk management measures would be to demonstrate that the flock of origin was either free from PMV-1 viruses at the time of slaughter or that any PMV-1 viruses present had ICPI values not greater than 0.0 (that is, the ICPI of the strain present in New Zealand).

Risk management options include surveillance programmes involving SPF sentinel chickens, collecting dead chickens once a week for necropsy or, as practised by the European Union, taking a minimum of 60 cloacal samples from each flock in the week prior to slaughter for virus isolation⁽²⁶⁾.

4 TIME/TEMPERATURE REQUIREMENTS TO INACTIVATE ND VIRUS

4.1 Work in original import risk analysis

In response to submissions, this section reassesses the time/temperature regimens required to inactivate ND virus.

In Appendix 3 of the original MAF *Import Risk Analysis*⁽²⁷⁾ a model was developed to estimate the time required to inactivate ND virus at different temperatures, and the risk of an outbreak of disease in backyard poultry. The main assumptions made were:

- source flock is assumed to be infected
- birds are assumed to be viraemic at slaughter
- initial titre of virus in meat was modelled as BetaPert(1.0, 1.05, 1.5) \log_{10} CID₅₀/g
- rate of heat inactivation of virus is independent of the initial titre
- each infectious dose is made up of several orders of magnitudes of viruses, each of which is capable of independently initiating an infection

The results of that model were shown in Appendix 3 on page 191, and are reproduced below in Table 4.

Table 4: Predicted cooking times (expected values) to achieve a target viral titre for ND virus in a whole chicken carcass, and the resulting risk of a disease outbreak in backyard flocks, at three levels of consumption.

| Target titre (\log_{10} CID ₅₀ /g) | 65 °C | 70 °C | 74 °C | 80 °C | risk estimates ^a at three levels of consumption ^b | | |
|---|----------|----------|---------|---------|---|----------|----------|
| | | | | | 1% | 10% | 20% |
| -6 | 28.2 min | 23.7 min | 4.7 min | 3.5 min | 0.51 | 0.99 | 0.99 |
| -7 | 32.2 min | 27.1 min | 5.4 min | 4.0 min | 5.13E-03 | 5.02E-02 | 9.79E-02 |
| -8 | 36.2 min | 30.4 min | 6.1 min | 4.5 min | 5.16E-04 | 5.15E-03 | 1.02E-02 |
| -9 | 40.1 min | 33.7 min | 6.7 min | 5.0 min | 5.21E-05 | 5.20E-04 | 1.04E-03 |
| -10 | 44.1 min | 37.1 min | 7.4 min | 5.5 min | 5.17E-06 | 5.18E-05 | 1.03E-04 |

Notes:

^a The risk estimate indicates that in 95% of iterations the probability of at least one backyard or poultry flock becoming infected each year is less than the value shown.

^b The levels of consumption are modelled as a percentage of the current total consumption of broilers in New Zealand (63 million birds per year at the time of the risk analysis).

However, several submissions suggested that the cooking times recommended for ND viruses were extreme.

4.2 Reassessment of conditions

In the original model, MAF simply evaluated the effectiveness of the current cooking requirements for imported chicken meat and did not recommend any new conditions.

From Table 4 it can be seen that if the target titre of virus is 10^{-6} , then at a temperature of 80°C a cooking time of $3\frac{1}{2}$ minutes is adequate. However, with that titre of virus in imported chicken meat, the model predicts that there would be outbreaks of disease in backyard chickens in this country every 6 importation months, even if the consumption of imported chicken was only 1%.

MAF considers that heat treatment of chicken meat for PMV-1 viruses should aim to achieve a final titre of not higher than $-9 \log_{10} \text{CID}_{50}/\text{g}$ (that is 10^{-9} chicken infectious doses per gram of tissue). Under such a cooking regime the risk of ND introduction, if imported poultry meat were consumed at a rate equivalent to 20% of current consumption, would be one outbreak per 1000 importation years.

Therefore, recommended cooking times for chicken meat to inactivate PMV-1 are :

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

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