Quantitative risk assessment of the likelihood of introducing porcine reproductive and respiratory syndrome virus into New Zealand through the importation of pig meat

S.P. Cobb [1]*, H. Pharo [1], M. Stone [1], H. Groenendaal [2] & F.J. Zagmutt [2]

(1) Ministry for Primary Industries, P.O. Box 2526, Wellington 6140, New Zealand

(2) EpiX Analytics, 1643 Spruce Street, Boulder, Colorado 80302, United States of America

*Corresponding author: stephen.cobb@mpi.govt.nz

Summary

A quantitative model was developed to estimate the likelihood of an incursion of porcine reproductive and respiratory syndrome virus (PRRSV) into New Zealand through the importation of fresh consumer-ready cuts of pig meat. A sensitivity analysis of all the inputs used in this model illustrated the importance of correctly modelling the available 'dose-response' data, and a mechanistic Beta-Poisson model was shown to be the most appropriate method for this in the authors' assessment. The output of this model predicts an average of approximately 1,200 years between PRRSV introductions resulting in primary infections in New Zealand. Given the uncertainties in the model, there is 95% confidence that this time period ranges from 52 to 6,200 years. The values chosen in this model are considered to provide a conservative estimate of the likelihood of introducing PRRSV into New Zealand via the importation of fresh pork.

Keywords

Import risk analysis – Monte Carlo simulation – New Zealand – Pig – Porcine reproductive and respiratory syndrome – Quantitative model – Risk assessment – Swine.

Introduction

In 2001, New Zealand introduced controls on the import of uncooked pig meat from countries with porcine reproductive and respiratory syndrome (PRRS); an infectious disease of pigs. This was in response to research showing that PRRS may be transmitted to uninfected animals by feeding them on meat from the carcasses of pigs which have been experimentally infected with PRRS (1). This decision was made without conducting a full risk analysis and was considered to be a 'provisional measure', permitted under the World Trade Organization (WTO) Sanitary and Phytosanitary Agreement ('SPS Agreement') (2) when decisions must be made without full information being available.

In 2006, a qualitative import risk analysis for PRRS in pig meat concluded that, in the absence of specific control measures, pork might pose a risk of introducing PRRS to New Zealand. However, this risk could be adequately managed by either cooking or curing imports or ensuring that such imports were in the form of consumer-ready cuts (3). This conclusion was based on evidence that PRRS virus (PRRSV) could be found in only a very small proportion (1.2%) of carcasses at slaughter (4), and that virus levels were reduced dramatically by commercial slaughter, processing, and handling. Virus levels continue to fall as the meat is stored, and the virus is rapidly denatured by heating. Therefore, only fresh, uncooked pork would pose any risk.

As an adjunct to this qualitative risk analysis, a quantitative risk assessment was developed to examine the likelihood of PRRSV entry in imported pig meat and the likelihood of pigs in New Zealand being exposed to this virus. This model was then subjected to peer review by an expert working group (EWG), made up of domestic and international experts nominated by key stakeholders. A full report of

the EWG process is publicly available (5) and this publication summarises the final quantitative model that came out of this process.

Model overview

A stochastic model was constructed, using MS Excel (Microsoft Corporation, USA) and ModelRisk 3.0 (Vose Software, Belgium), to estimate the annual frequency with which backyard pigs in New Zealand would be infected with PRRSV through the consumption of fresh raw scraps from the preparation at home of consumer-ready cuts of pork imported from countries where this disease is present. An outline of the model structure and parameters used in this model is shown in Figure 1 and Table I.

After discussions within the EWG, this initial model was expanded to include the likelihood of disease introduction into non-commercial and para-commercial pig herds through feeding with meat scraps collected from the retail and food service sectors. Figure 2 illustrates the major steps in this model, including these additional pathways.

Step 1 – Estimate the annual proportion of PRRSV-infected pig meat entering New Zealand

The annual proportion of PRRSV-infected pig meat entering New Zealand (PCT_PORK_INFECTED) was defined as the product of the following four independent probabilities.

– IMPORT_P is the proportion of pork consumed in New Zealand that is imported. Previous modelling of this parameter used a point estimate of 0.42 (6). It was assumed that relaxing import laws may result in increased pig meat imports. To reflect the uncertainty in this parameter and remain conservative, IMPORT_P was *Uniform (0.42, 0.5)*, allowing for up to 50% of domestically consumed pork to originate from overseas.

– PRRS_POSITIVE_COUNTRY_P is the proportion of pork imported into New Zealand that arrives from PRRSV-positive countries. Statistics New Zealand data (available at www.stats.govt.nz/infoshare) for the period 1997 to 2000 (the period immediately before New Zealand introduced import restrictions due to

PRRSV) showed that between 15.45% and 24.54% of imported pork came from Australia (the only major source of pig meat imports that is PRRSV-free), therefore PRRS_POSITIVE_COUNTRY_P was described by *Uniform (0.7546, 0.8455)*.

– CONSUMER_READY_P is the proportion of imported pork that is intended for direct retail sale as fresh, unprocessed meat. To estimate a value for this figure, the authors used 2009 data from Statistics New Zealand. By weight, 0.95% of imports in 2009 were in a form considered likely to be processed into fresh meat for retail. A further 6.78% were in a form that could be processed into 'fresh' meat for retail after defrosting, although discussion with industry representatives suggested this was unlikely. To reflect this, CONSUMER_READY_P was represented by *Pert (0.0095, 0.02645, 0.0434)*. The maximum value in this Pert distribution reflected the case where all chilled imports and 50% of imported frozen carcasses, half carcasses, hams, shoulders, and cuts of these items were destined for retail as 'fresh' pork, and the midpoint of this distribution was chosen as the most likely value.

– CONTAMINATION_P reflects the proportion of imported pork from countries where PRRSV is present that would be expected to contain an infectious dose of PRRSV. Magar and Larochelle reported that the prevalence of PRRSV-infected animals at slaughter in Canada, based on testing by reverse-transcription polymerase chain reaction (RT-PCR), was 19/1,027 and the proportion of these positive samples that were able to transmit infection in feeding trials was 7/11 (4). By making no prior statement about the values of each proportion, we can estimate the uncertainty about the true value of the proportion by using a *Beta (successes + 1, trials – successes + 1)* distribution, where trials are the number of individuals tested (or exposed in the feeding trial) and successes are the PCR-positive tests. Therefore, CONTAMINATION_P was *Beta (19 + 1, 1,027 – 19 + 1) × Beta (7 + 1, 11 – 7 + 1)*.

Step 2a – Estimate the number of premises that own 'backyard' pigs and feed them potentially infected domestic waste

The total number of households in New Zealand that have backyard pigs and feed them with domestic kitchen scraps that are potentially infected with PRRSV (TOT_NUM_WASTEFEEDERS) was defined in this model by the product of the following four parameters.

– TOT_PIGPREMISES was previously described with the distribution *Pert (7,000, 12,000, 20,000)* (6). The authors' model recognises that there are no better data to support this parameter, although retaining the previously estimated maximum value of 20,000 pig-owning properties in New Zealand maintained the overall conservative bias.

– The proportion of backyard pig keepers that feed waste food to their pigs (WASTEFEED_P) was adopted from a previously published Normal distribution centred on 0.8 (6), but was corrected to avoid values >1. Thus, the revised estimate was *Pert (0.55, 0.8, 1.0)*.

– A study of the behaviour of pig keepers in New Zealand (9) reported that 2/15 para-commercial farms (13%) and 2/18 non-commercial farms (11%) included raw meat in their food waste. To reflect this finding, MEATFEED_P was represented in this model by *Pert (0.11, 0.13, 0.2)*, the maximum value of this distribution reflecting a previously reported point estimate (6).

– A previously reported expert estimate suggested that only 10% of swill-feeding premises comply with New Zealand's current waste food regulations, which require that any food waste fed to pigs must be first heated to 100°C for one hour (6). Recognising that less stringent conditions may also inactivate PRRSV, NONCOMPLY_P (the proportion of backyard pig keepers who do not cook waste food sufficiently to inactivate PRRSV) was modelled using *Pert (0.75, 0.85, 0.95)*.

Step 2b – Estimate the number of non-commercial pig herds that feed potentially infected waste from the food service and retail sector

Agribase data (a national spatial farms database of non-urban areas in New Zealand) suggest that a total of 7,000 premises in New Zealand could be classified as either para-commercial or non-commercial pig enterprises. Industry experts involved in the EWG process estimated that 94% of these properties (6,580) should be considered to be non-commercial pig holdings.

Based on the findings reported by Pearson *et al.* (9), 11% of these properties were considered likely to feed their pigs with meat collected from the food service and retail sectors as part of their waste food supply. The model presented here therefore assumed that a total of 724 non-commercial pig herds would eat potentially infected meat from the food service and retail sector.

Step 2c – Estimate the number of para-commercial pig herds that feed potentially infected waste from the food service and retail sector

As described above, industry experts in the EWG estimated that 6% of the 7,000 herds recorded on Agribase would be likely to be para-commercial pig herds (420 herds), with 21% of these feeding on meat from the food service and retail sectors (9). Therefore, it was assumed that a total of 88 para-commercial pig herds would eat potentially infected meat from the food service and retail sector.

Step 3a – Estimate the number of meals consumed per year using purchased pork in households which keep pigs

The total number of meals containing purchased pork consumed per year in households that keep backyard pigs (TOT_NUM_PORKMEALS) was estimated as the product of TOT_NUM_WASTEFEEDERS (as determined above) and the following three parameters.

– PREMISES_FREQ, the number of pork meals consumed annually by each pork-consuming household, was derived from the results of a

marketing survey undertaken by the New Zealand Pork Industry Board (cited in 6). It was reported that 61% of New Zealand households consume pork on at least one occasion every month, with 17% consuming pork once a month, 17% consuming pork twice a month, 11% eating pork three times a month, 7% consuming pork four times a month, and 9% eating pork five or more times a month. Given the large number of non-pork-consuming households, the survey data were fitted to a Zero-inflated BetaGeometric distribution using Maximum Likelihood Estimation (MLE) methods, and a parametric bootstrap was used to model the uncertainty around the mean number of meals/month/family. Since the number of families surveyed was not reported, a small number of families (100) was assumed to have been surveyed and thus used for the size of the bootstrap sample, to assign maximum uncertainty to the parameter estimated (22, 23).

– PORK_CONS_PREMISES, the proportion of households that eat pork at least once per month, was derived from the uncertainty distribution described above.

– PIGPREMISE_BUYPORK_P, the proportion of meals that comprise purchased pork – this parameter was previously modelled as a point value of 0.25, although this approach did not account for individuals that may be totally or partially reliant on home-grown pork for their household consumption (6). To be consistent with other parameters used in the model, a *Beta (10 + 1, 40 – 10 + 1)* distribution was used, assuming that only 40 premises were surveyed and ten answered that they bought pork.

Step 3b – Estimate the number of meals consumed per year by non-commercial herds that contain pig-meat waste from the food service and retail sector

The EWG estimated that a total of 71 tonnes of pork waste would be generated per year in New Zealand and made available to pigs in the non-commercial and para-commercial sectors. The model presented here assumed that this waste food would be distributed evenly between the pigs in the para-commercial and non-commercial sectors in New Zealand that practise waste-feeding. Based on expert opinion,

this model also assumed that waste food is collected on a four-day cycle from the retail and food service sectors.

From these estimates, this model assumed that, each year, pig-meat waste from the retail and food service sectors in New Zealand contributes to a total of 66,047 meals in non-commercial herds, with each meal containing an average of 698 g of pork. Furthermore, the model assumed that this meal is then distributed evenly amongst all the pigs in the non-commercial herd and also assumed an average of 14 individuals in each non-commercial herd (based on an estimate by the EWG).

The model therefore included an expected $66,047 \times 14$ 'feeding events' of non-commercial pigs with fresh meat scraps generated from the retail and food service sectors.

Step 3c – Estimate the number of meals consumed per year by para-commercial herds that contain pig-meat waste from the food service and retail sector

Based on the same estimates reported above, and an assumption of 62 pigs in each para-commercial herd (again, based on an estimate by the EWG), the model presented here assumed that, each year, pig-meat waste from the retail and food service sectors in New Zealand would contribute to a total of 8,048 meals in para-commercial herds, with each meal containing an average of 3,092 g of pork.

Step 4a – Estimate the likelihood that household waste food will result in PRRSV infection

Based on published dose-response data (8), this step of the model contained four elements – the weight of scrap generated, the concentration of virus in the consumed scrap, the consumed dose of virus (the product of scrap weight and viral concentration), and the conditional probability of infection of a pig with PRRSV, given the consumed dose.

Step 4a (i) – Estimating the weight of scrap generated

SCRAP_WEIGHT was calculated as the product of two parameters, the weight of fresh pork prepared for an average household meal (PORK_PER_MEAL) and the proportion of that fresh pork discarded as fresh raw scrap during the preparation of that meal (RAW_MEAT_SCRAP_P).

Neumann *et al*. (6) reported that an average-sized serving of pork was 125 g and estimated that a typical family meal would contain 1,250 g of pork, calculated by dividing the total consumption of fresh pork in New Zealand by the estimated total number of family meals per year. As this parameter is a rate, and assuming independence in the meal sizes, the authors modelled PORK_PER_MEAL using *Gamma (125, 1)* to be maximally conservative, and then multiplied this by 2.6, the average household size in New Zealand (6).

There has been no significant work published on levels of meat waste generated in households in New Zealand. Hence, international literature on food waste was studied to determine a range for RAW_MEAT_SCRAP_P, the proportion of fresh pork in a meal that is discarded as raw scrap.

A total of 169,900 tonnes of pork, in the form of bellies, chops, leg roasts, loin roasts, shoulder roasts, steaks and pork mince, were consumed in the United Kingdom (UK) in 2007 (10). A comprehensive survey of UK household food waste production (11) found that 28,800 tonnes of 'pork portions' were disposed of in 2007, equivalent to 16.95% of total pork consumption. Some 11,600 tonnes (6.83% of purchased pork) were disposed of in a fresh, raw or minimally processed form, while 5,100 tonnes of this were disposed of unopened (likely to have gone beyond its use-by date), suggesting that around 3.8% of all pork purchased in the form of bellies, chops, leg roasts, loin roasts, shoulder roasts, steaks and pork mince in the UK in 2007 was disposed of as fresh raw scraps in garbage.

United States pork industry representatives have estimated that between 0.001% and 0.1% of pork is discarded as trim in restaurants

(12), while Gale (2004) suggested that 1% of all meat is discarded uncooked as catering waste (13).

An informal survey of personnel in an Australian government department, with households ranging from one to six members, revealed that most consumers who were surveyed discarded between 1% and 10% of purchased pig meat (14). This survey was subsequently used in an Australian model to justify Pert *(0.01, 0.05, 0.10)* as the proportion of pig meat purchased by households that is discarded as waste, although this figure included uncooked, cooked and processed pig meat.

As the present model is concerned with waste generation from fresh meat in a 'consumer-ready' form, it is reasonable to suggest that the minimum value for this commodity should be the case in which no waste is generated, a most likely value of 1% scrap generation consistent with Gale (13), and a maximum value of 5% was suggested. RAW_MEAT_SCRAP_P was modelled using *Pert (0, 0.01, 0.05)*.

Step 4a (ii) – Estimating the concentration of virus in the consumed scrap

The concentration of virus in a consumed scrap of pork (SCRAP_VIRAL_CONCENTRATION) was calculated as the product of the following six parameters.

PEAK_VIRUS_SLAUGHTER

Given the inclusion of CONTAMINATION_P earlier in the model, it was assumed that meat samples were taken from individuals at the peak of viraemia, which results in an overestimation of the overall risk.

Meta-analytical methods were used to pool estimates to model this parameter. Twenty-seven references were initially proposed during the EWG process (15, 16, 17, 18, 19, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45). The following criteria were used to select the final articles from this initial list.

− Results from studies that report titration of serial dilutions on cell cultures. Reverse-transcription PCR results were excluded since expert opinion determined that these provided little evidence concerning the quantity of infectious virus.

− Results from studies with pigs or sows close to the age of slaughter. The age of slaughter ranges from 150 to 220 days (21–32 weeks). However, to include as much information as possible, the authors used study results from animals over 13 weeks old. The findings from Klinge *et al.* (2009) suggest that there is a significant difference in viraemia depending on age, so it was very important to include this consideration (17).

− Studies that included one of the following peak viraemia data:
   i) individual pig peak viraemia (explicitly or derived from tables and figures), or
   ii) mean and standard deviation of individual pig peak viraemia. Studies reporting only means were excluded as the meta-analysis also required an estimate of the standard deviation.

As a result of further analysis based on these criteria, five studies were considered eligible for inclusion (15, 16, 17, 18, 19). From each of the five eligible studies, the mean peak of PRRSV viraemia in pigs of slaughter age was extracted. Using this, a pooled estimate was calculated as the average of the statistics estimated in the individual studies.

PROP_AFTER_BLEEDING

As noted by the European Food Safety Authority (EFSA) (7), Warriss (1984) concluded that the residual blood content of lean meat is 2 to 9 ml/kg muscle and that there is no evidence that this amount is affected by different methods of slaughter (20). Reflecting these data, the viral titre of meat would be between 0.2% and 0.9% of the viraemic titre. This parameter in the model was represented by *Uniform (0.002, 0.009)* ml blood/g meat.

PROP_AFTER_MATURE

This parameter was also reviewed by EFSA (7). While the decline in pH observed during carcass maturation does not impair the viability of PRRSV, the time period from slaughter to maturation results in some loss of infectivity. This process takes a minimum of 30 hours and the use of fast or intensive cooling results in a quick reduction of the carcass temperature to 7°C. On the basis of this, EFSA estimated that the minimum effect of maturation would be a 90% reduction of the amount of viable virus. However, a more recent publication (21) suggests that (as a worst-case scenario) exposure to 7°C for 30 hours may result in as little as a 14% reduction in viable virus. *Uniform (0.1, 0.86)* was used to represent maximum uncertainty between both findings.

PROP_AFTER_THAW

This parameter represents the effect of the delay between product maturation and its arrival at the point of retail. Like the above parameters, this aspect was considered by EFSA (7), which concluded that thawing after freezing would result in a 90% reduction in the amount of virus present in pig meat. For any meat that was imported chilled (not frozen), one might expect a 90% reduction in virus after 30 hours at 4°C (15). However, more recent studies suggest that this reduction may be less, and for a shipping time of four days (the minimum possible for sea transport from Australia to New Zealand), storage at 4°C may result in as little as a 32% reduction in viable virus (21), resulting in *Uniform (0.1, 0.68)* being used here for both chilled and frozen meat.

PROP_DELAY_ PURCHASE

There is likely to be some delay between the purchase of pork at the point of retail and the generation of raw trim in a household kitchen. Given the shelf life of fresh pork, this is unlikely to exceed four days and, maintaining the conservative bias of this model, it was assumed most likely that fresh pork would be consumed within one day of purchase.

Bloemraad *et al.* (15) suggested that there would be a 90% reduction in the amount of virus after 30 h at 4°C, which would result in a maximum of 99.9% reduction in the amount of viable virus present in pig meat after four days of domestic storage. This could be represented by *Pert (0.001, 0.5, 1.0).* Jacobs *et al*. (21) predicted a PRRSV half-life of 157 h at 4°C, suggesting a distribution of *Pert (0.68, 0.92, 1.0).*

Both of these predicted ranges appear to have equal value. Therefore, to accommodate these, the present model uses a mixture distribution (46), with both these input ranges given equal weighting.PROP_DELAY_DISPOSAL

EFSA (7) concluded that there would be a minimum time of 16 h between the generation of raw scraps in a restaurant kitchen and feeding these to pigs, which – based on the findings of Bloemraad *et al*. (15) that the D-value at 25°C was 21 h – would result in decay of around 32% of the virus. In a domestic environment, waste from a main evening meal would be likely to be fed either immediately after generation of the waste, the following morning, or the following afternoon – i.e. delays of 1 h, 12 h or 21 h. Based on the Bloemraad *et al*. D-value cited above, this parameter could be represented by an empirical *Discrete (0.96, 0.42, 0.1)* distribution, whereas the Jacobs *et al*. (21) publication suggests using a *Discrete (0.96, 0.62, 0.4)* distribution.

To accommodate both of these predicted ranges in the authors' model this value was represented by an empirical *Discrete* distribution using the counts above.

Step 4a (iii) – Calculating the consumed dose of virus (the product of scrap weight and viral concentration)

The product of SCRAP_WEIGHT and SCRAP_VIRAL_CONCENTRATION is the dose of virus present in the disposed-of meat scrap. It was assumed that the scrap generated in a domestic kitchen would be consumed by an individual backyard pig.

Step 4a (iv) – Estimating the probability of infection as a function of the consumed dose, based on the work of Hermann *et al.*

P_SCRAP_INFECTIOUS was modelled in logs and thus is highly influential in the output of the model once the dose has been brought back to actual viral concentration, expressed as a median tissue culture infective dose ($TCID_{50}$).

Curve fits such as the Logistic model or the Probit model provided in the article by Hermann *et al.* (8) are not based on a biological principle, and thus they are just mathematical models fitted to data. Although this type of fit is appropriate to interpolate the doses presented in Hermann *et al.* (8), the lack of a theoretical principle related to dose-response interactions makes them unsuitable for the extrapolation of parameters outside the data observed.

One way to overcome this limitation is to use mechanistic models that attempt to explain the dynamics between infectious agent and host that result in the infection (47). Such mechanistic models are widely used in food-safety risk modelling and are broadly categorised as 'dose-response' models. Haas *et al.* (48) present a comprehensive introduction to the subject and discuss the advantages and disadvantages of the most commonly used dose-response models.

The Hermann *et al.* (8) study provides an accurate estimation of the dose of PRRSV administered to each animal, so it is reasonable to assume that the actual dose is known. Under this assumption, the authors fitted a Beta-Binomial (BB) model to the data using MLE methods.

Both the BB and Beta-Poisson (BP) model use the Beta distribution to model *p* in an identical form. Therefore, the α and ß parameters estimated from either model can be used in the other. Although the fit of both models to the Hermann *et al.* (8) data was nearly identical, the BP model was preferred for this risk assessment as the uncertainty in the expected dose, rather than the actual dose itself, is modelled.

Steps 4b and 4c – Estimate the likelihood that food service and retail waste food will result in PRRSV infection in a non-commercial and para-commercial herd

Each of the assumed 724 non-commercial and 88 para-commercial pig farms was assumed to feed their animals waste from the retail and food service sectors every four days, resulting in a total of 66,047 and 8,048 meals in non-commercial and para-commercial herds, respectively, each year.

As noted above, the EWG estimated that, in New Zealand, a total of 71 tonnes of pork waste would be generated per year and made available to pigs in the non-commercial and para-commercial sectors. If we assume that this pork is evenly distributed amongst the recipients in para-commercial and non-commercial herds, then each pig receives 12 g per day through this pathway.

Based on the average herd sizes, the model represented the amount of raw pork waste (in grams) fed to non-commercial herds as *Pert (175, 436, 698)* and the amount fed to para-commercial herds as *Pert (773, 1,933, 3,092)*. The model then divided this four-daily 'dose' of raw pork amongst the recipients, based on the average herd sizes described.

The probability that these scraps collected from the food service and retail sector would contain an infectious dose of PRRSV was then determined, using the same parameters described above (Step 4a), although the distribution used to represent PROP_DELAY_DISPOSAL was replaced with an inactivation curve to account for viral decay, as meat scraps are held in a bucket for up to four days.

Step 5 – Estimating the annual number of primary infections in pigs due to exposure from imported meat

The total number of primary infections per year in New Zealand pigs due to exposure from meat scraps was calculated from the product of the steps described above. For example, the annual frequency of PRRSV introduction into backyard herds though feeding waste generated in household kitchens was the product of

(P_SCRAP_INFECTIOUS) × (TOT_NUM_PORKMEALS) × (PCT_PORK_INFECTED). By adding the results of the three exposure pathways simulated in this model, the total number of annual infections with PRRSV that may occur, due to the relaxation of New Zealand's requirements for imported pork from countries with PRRSV, was estimated.

Results and discussion

The model presented here was developed over a four-year period and was constrained by a need to maintain the model structure initially published by Neumann *et al.* (6). Contributors to the EWG process considered that, although the model generated through this process had limitations and there was room for improvement, the biological conclusions that could be made on the basis of this model were valid and credible.

Figure 3 shows the sensitivity of the model to the inputs discussed above. This illustrates how influential P_SCRAP_INFECTIOUS is in this model and highlights the importance of correctly modelling the data from Hermann *et al*. (8) to inform this parameter. Figure 4 shows a comparison of the fit of Logistic, Probit, BB and BP models to the Hermann *et al*. (8) feeding trial data, and Figure 5 shows the behaviour of the BP, Logistic and Probit models when extrapolating to low doses far beyond those tested in that feeding trial. The BB model was excluded from Figure 5 as doses used to create this plot are smaller than one and the BB model takes only actual (integer) doses.

Figures 4 and 5 show that these models provide a similar fit to the observed data, with the BB/BP and Logistic/Probit model pairs exhibiting nearly identical fits. However, when extrapolating to low doses, the BP model quickly drops to near-zero values, whereas the Logistic model still provides non-zero probabilities of infection for doses that would be equivalent to less than one virus particle.

These results clearly show that the empirical fit provided by the Logistic model is not appropriate to make the low-dose extrapolations that are required in this risk assessment model. This, together with the evidence supporting the use of mechanistic models, supports the

notion that the BP model is the most suitable dose-response model for the purposes of this work.

The model presented here reports a mean of 0.0038 PRRSV primary introductions per year and an average of 1,226 years between outbreaks.

Caution is required to interpret the results of a model of this nature correctly. The model is not intended to provide interpretations such as 'there will be a PRRSV introduction after 1,226 years' or 'there is a 0.0038 probability of at least one PRRSV primary introduction for next year'. However, we can say that, under the current conditions assumed in the model, the authors estimate an average of roughly 1,200 years between PRRSV introductions resulting in outbreaks (due to imported pig meat) in New Zealand, and that, given the uncertainties in this model, the 95% confidence interval ranges roughly from 52 to 6,200 years. It is important to point out that the model estimates the probability of an outbreak in any given year, and not for next year. Furthermore, the 'year' is not a calendar year but essentially 365 days, as the variability from year to year is not modelled.

The results of this model are consistent with the history of pig meat imports in New Zealand. There was a three-and-a-half-year period (the beginning of 1998 to mid-2001) during which pig meat was imported from PRRS-infected countries without any animal health measures against the disease being imposed on imports. Despite this, New Zealand remained free from PRRSV. About 30,000 tonnes of pig meat may have been imported from PRRS-infected countries over that three-and-a-half-year period without implementing any controls on garbage feeding. Similarly, both Sweden and Chile have recently eradicated PRRS and, in both these cases, meat from infected pigs was released without restriction for domestic consumption (49). From this work, it is the authors' belief that the current model provides a conservative estimate of the risk of introduction of PRRSV into New Zealand via the importation of fresh pork.

References

1. Van der Linden I.F.A., van der Linde-Bril E.M., Voermans J.J.M., van Rijn P.A., Pol J.M.A., Martin R. & Steverink P.J.G.M. (2003). – Oral transmission of porcine reproductive and respiratory syndrome virus by muscle of experimentally infected pigs. *Vet. Microbiol.*, **97** (1–2), 45–54.

2. World Trade Organization (WTO) (1994). – Agreement on the application of sanitary and phytosanitary measures, the results of the Uruguay round of the multilateral trade negotiations. WTO, Geneva. Available at: www.wto.org/english/tratop_e/sps_e/spsagr_e.htm (accessed on 27 March 2015).

3. Ministry of Agriculture and Forestry, Biosecurity New Zealand (2006). – Import risk analysis: porcine reproductive and respiratory syndrome (PRRS) virus in pig meat. Ministry of Agriculture and Forestry, Wellington, New Zealand. Available at: www.biosecurity.govt.nz/files/regs/imports/risk/prrs-risk-analysis.pdf (accessed on 9 May 2012).

4. Magar R. & Larochelle R. (2004). – Evaluation of the presence of porcine reproductive and respiratory syndrome virus in pig meat and experimental transmission following oral exposure. *Can. J. Vet. Res.*, **68** (4), 259–266.

5. Stone M., Depner K., Aubry P., Alban L., Wilesmith J., Neumann E., Lawton D., Glass S., Perkins N. & Rowland R. (2010). – Report of the Expert Working Group (EWG) considering the MAF quantitative risk assessment examining the likelihood of introducing PRRS in imported pig meat (Pork EWG). Biosecurity New Zealand, Wellington, New Zealand. Available at:

www.biosecurity.govt.nz/files/ihs/pig-meat-ihs/pork-ewg-report.pdf (accessed on 31 March 2015).

6. Neumann E.J., Morris R.S. & Sujau M. (2007). – Analysis of the risk of introduction and spread of porcine reproductive and respiratory syndrome virus through importation of raw pigmeat into New Zealand. *N.Z. Vet. J.*, **55** (6), 326–336.

7. European Food Safety Authority (EFSA) (2005). – The probability of transmission of porcine reproductive and respiratory syndrome virus (PRRSv) to naive pigs via fresh meat. *EFSA J.*, **239**, 1–85.

8. Hermann J.R., Muñoz-Zanzi C.A., Roof M.B., Burkhardt K. & Zimmerman J.J. (2005). – Probability of porcine reproductive and respiratory syndrome (PRRS) virus infection as a function of exposure route and dose. *Vet. Microbiol.*, **110** (1–2), 7–16.

9. Pearson A.B., Neumann E., Sanson R., Nicol K. & Clement F. (2009). – Pig movements transmission of exotic diseases. Final Report. MAF Biosecurity New Zealand Technical Paper No. 2009/19. Prime Consulting International, Levin, New Zealand. Available at: www.biosecurity.govt.nz/files/pests/salt-freshwater/022009-pig-movements-transmission-of-exotic-diseases.pdf (accessed on 9 May 2012).

10. British Pig Executive (BPEX) (2008). – BPEX Annual Technical Report 2007–2008. Available at: www.bpex.org.uk/article.aspx?ID=295923 (accessed on 9 May 2012).

11. Waste and Resources Action Programme (2008). – Food waste report. The food we waste. Available at: wrap.s3.amazonaws.com/the-food-we-waste.pdf (accessed on 9 May 2012).

12. United States Department of Agriculture (USDA), Animals and Plant Health Inspection Service (APHIS) (2000). – Risk analysis for importation of classical swine fever virus in swine and swine products from the European Union, USDA–APHIS, Washington, DC.

13. Gale P. (2004). – Risks to farm animals from pathogens in composted catering waste containing meat. *Vet. Rec.*, **155** (3), 77–82.

14. Department of Agriculture, Fisheries and Forestry (Australia) (2004). – Generic import risk analysis (IRA) for pig meat. Final import risk analysis report. Australian Government, Department of Agriculture, Fisheries and Forestry. Available at: www.daff.gov.au/__data/assets/pdf_file/0018/18081/2004-01b.pdf (accessed on 9 May 2012).

15. Bloemraad M., de Kluijver E.P., Petersen A., Burkhardt G. & Wensvoort G. (1994). – Porcine reproductive and respiratory syndrome: temperature and pH stability of Lelystad virus and its survival in tissue specimens from viraemic pigs. *Vet. Microbiol.*, **42** (4), 361–371.

16. Costers S., Lefebvre D.J., Goddeeris B., Delputte P.L. & Nauwynck H.J. (2009). – Functional impairment of PRRSV-specific peripheral CD3+CD8 high cells. *Vet. Res.*, **40** (5), 46.

17. Klinge K.L., Vaughn E.M., Roof M.B., Bautista E.M. & Murtaugh M.P. (2009). – Age-dependent resistance to porcine reproductive and respiratory syndrome virus replication in swine. *Virol. J.*, **6** (177), 1–11. Available at: www.virologyj.com/content/pdf/1743-422X-6-177.pdf (accessed on 9 May 2012).

18. Vanhee M., Delputte P.L., Delrue I., Geldhof M.F. & Nauwynck H.J. (2009). – Development of an experimental inactivated PRRSV vaccine that induces virus-neutralizing antibodies. *Vet. Res.*, **40** (6), 63.

19. Wasilk A., Callahan J.D., Christopher-Hennings J., Gay T.A., Fang Y., Dammen M., Reos M.E., Torremorell M., Polson D., Mellencamp M., Nelson E. & Nelson W.M. (2004). – Detection of U.S., Lelystad, and European-like porcine reproductive and respiratory syndrome viruses and relative quantitation in boar semen and serum samples by real-time PCR. *J. Clin. Microbiol.*, **42** (10), 4453–4461.

20. Warriss P.D. (1984). – Exsanguination of animals at slaughter and the residual blood content of meat. *Vet. Rec.*, **115** (12), 292–295.

21. Jacobs A.C., Hermann J.R., Muñoz-Zanzi C., Prickett J.R., Roof M.B., Yoon K.J. & Zimmerman J.J. (2010). – Stability of porcine reproductive and respiratory syndrome virus at ambient temperatures. *J. Vet. Diagn. Invest.*, **22** (2), 257–260.

22. Efron B. (1987). – Better bootstrap confidence intervals: (with discussion). *J. Am. Stat. Assoc.*, **82** (397), 171–200. Available at: www-rohan.sdsu.edu/~babailey/stat672/BCa.pdf.

23. Gzyl H. (1995). – The method of maximum entropy. World Scientific, London.

24. Calzada-Nova G., Husmann R.J., Schnitzlein W. & Zuckermann F.A. (2008). – Effectiveness of a PRRS modified live virus vaccine prepared in a novel porcine alveolar macrophage cell line. *In* Proc. 39th Annual Meeting of the American Association of Swine Veterinarians (AASV), 8–11 March, San Diego, California. AASV, 109–114.

25. Cano J., Dee S.A., Murtaugh M., Polson D., Johnston D., Finnegan C. & Pijoan C. (2006). – Evaluation of PRRSV mass vaccination to reduce the impact of the virus in infected populations: Part 2 – heterologous infection. *In* Proc. 19th International Pig Veterinary Society Congress, 16–19 July, Copenhagen, Denmark, 240.

26. Cuartero L., Pijoan C., Ruiz A., Dee S. & Rossow K. (2000). – Relationship between clinical signs and PRRS viremic titers under field conditions. *In* Proc. 16th International Pig Veterinary Society Congress, 17–21 September, Melbourne, Australia, 607.

27. Duan X., Nauwynck H.J. & Pensaert M.B. (1997). – Virus quantification and identification of cellular targets in the lungs and lymphoid tissues of pigs at different time intervals after inoculation with porcine reproductive and respiratory syndrome virus (PRRSV). *Vet. Microbiol.*, **56** (1–2), 9–19.

28. Geldhof M.F., Vanhee M., Van Doorsselaere J. & Nauwynck H.J. (2010). – Efficacy of an experimental inactivated PRRSV vaccine based on a recently isolated field strain. *In* Proc. 21st International Pig Veterinary Society Congress, 18–21 July, Vancouver, Canada, 238. Available at: http://orbit.dtu.dk/fedora/objects/orbit:63423/datastreams/file_5574380/content (accessed on 2 October 2015).

29. Gozio S. & Van Woensel P. (2006). – The comparative efficacy of porcilis PRRS, a live attenuated PRRS vaccine, and a commercial inactivated PRRS vaccine in PRRS-naive pigs in controlled laboratory conditions. *In* Proc. 19th International Pig Veterinary Society Congress, 16–19 July, Copenhagen, Denmark, 58.

30. Grebennikova T.V., Clouser D.F., Vorwald A.C., Musienko M.I., Mengeling W.L., Lager K.M., Wesley R.D., Biketov S.F., Zaberezhny A.D., Aliper T.I. & Nepoklonov E.A. (2004). – Genomic characterization of virulent, attenuated, and revertant passages of a North American porcine reproductive and respiratory syndrome virus strain. *Virology*, **321** (2), 383–390.

31. Greiner L.L., Stahly T.S. & Stabel T.J. (2000). – Quantitative relationship of systemic virus concentration on growth and immune response in pigs. *J. Anim. Sci.*, **78** (10), 2690–2695.

32. Greiner L.L., Stahly T.S. & Stabel T.J. (2001). – The effect of dietary soy genistein on pig growth and viral replication during a viral challenge. *J. Anim. Sci.*, **79** (5), 1272–1279.

33. Greiner L.L., Stahly T.S. & Stabel T.J. (2001). – The effect of dietary soy daidzein on pig growth and viral replication during a viral challenge. *J. Anim. Sci.*, **79** (12), 3113–3119.

34. Karniychuk U.U., Geldhof M., Vanhee M., Van Doorsselaere J., Saveleva T.A. & Nauwynck H.J. (2010). – Pathogenesis and antigenic characterization of a new East European subtype 3 porcine reproductive and respiratory syndrome virus isolate. *BMC Vet. Res.*, **6** (30), 1–10. Available at: www.biomedcentral.com/content/pdf/1746-6148-6-30.pdf (accessed on 9 May 2012).

35. López O.J., Oliveira M.F., García E.A., Kwon B.J., Doster A. & Osorio F.A. (2007). – Protection against porcine reproductive and respiratory syndrome virus (PRRSV) infection through passive transfer of PRRSV-neutralizing antibodies is dose dependent. *Clin. Vaccine Immunol.*, **14** (3), 269–275.

36. Loving C.L., Brockmeier S.L., Vincent A.L., Lager K.M. & Sacco R.E. (2008). – Differences in clinical disease and immune response of pigs challenged with a high-dose versus low-dose inoculum of porcine reproductive and respiratory syndrome virus. *Viral Immunol.*, **21** (3), 315–325.

37. Martelli P., Cordioli P., Alborali L.G., Gozio S., De Angelis E., Ferrari L., Lombardi G. & Borghetti P. (2007). – Protection and immune response in pigs intradermally vaccinated against porcine reproductive and respiratory syndrome (PRRS) and subsequently exposed to a heterologous European (Italian cluster) field strain. *Vaccine*, **25** (17), 3400–3408.

38. Martin R.G. & Steverink P.J. (2002). – Oral transmission of PRRS virus via the feeding of infected muscle to pigs. *In* Proc. 17th International Pig Veterinary Society Congress, 2–5 June, Ames, Iowa, 45.

39. Mondaca-Fernández E., Meyns T., Rovira A. & Morrison R.B. (2008). – Experimental quantification of PRRSV transmission. *In* Proc. Allen D. Leman Swine Conference, 20–23 September, Saint Paul, Minnesota, 17.

40. Mulupuri P., Zimmerman J.J., Hermann J., Johnson C.R., Cano J.P., Yu W., Dee S.A. & Murtaugh M.P. (2008). – Antigen-specific B-cell responses to porcine reproductive and respiratory syndrome virus infection. *J. Virol.*, **82** (1), 358–370.

41. Opriessnig T., Baker R.B. & Halbur P.G. (2007). – Use of an experimental model to test the efficacy of planned exposure to live porcine reproductive and respiratory syndrome virus. *Clin. Vaccine Immunol.*, **14** (12), 1572–1577.

42. Osorio F.A., Galeota J.A., Nelson E., Brodersen B., Doster A., Wills R., Zuckermann F. & Laegreid W.W. (2002). – Passive transfer of virus-specific antibodies confers protection against reproductive failure induced by a virulent strain of porcine reproductive and respiratory syndrome virus and establishes sterilizing immunity. *Virology*, **302** (1), 9–20.

43. Petry D.B., Holl J.W., Weber J.S., Doster A.R., Osorio F.A. & Johnson R.K. (2005). – Biological responses to porcine respiratory and reproductive syndrome virus in pigs of two genetic populations. *J. Anim. Sci.*, **83** (7), 1494–1502.

44. Reiner G., Willems H., Pesch S. & Ohlinger V.F. (2010). – Variation in resistance to the porcine reproductive and respiratory syndrome virus (PRRSV) in Pietrain and Miniature pigs. *J. Anim. Breed. Genet.*, **127** (2), 100–106. doi:10.1111/j.1439-0388.2009.00818.x.

45. Shin J.H. & Molitor T.W. (2002). – Assessment of porcine reproductive and respiratory syndrome virus RNA load in sera and tissues during acute infection. *J. Vet. Sci.*, **3** (2), 75–86.

46. Vose D. (2008). – 14.3.4. Incorporating differences in expert opinions. *In* Risk analysis: a quantitative guide, 3rd Ed. John Wiley & Sons Ltd, Chichester, United Kingdom, 410–412.

47. World Health Organization (WHO) (2009). – Risk characterization of microbiological hazards in food: guidelines, Microbiological Risk Assessment Series 17. WHO, Geneva. Available at: www.who.int/foodsafety/publications/micro/MRA17.pdf (accessed on 9 May 2012).

48. Haas C.N., Rose J.N. & Gerba C.P. (1999). – Quantitative microbial risk assessment. Wiley & Sons, New York.

49. Pharo H. & Cobb S.P. (2011). – The spread of pathogens through trade in pig meat: overview and recent developments. *In* The spread of pathogens through international trade in animals and animal products (S. MacDiarmid, ed.). *Rev. Sci. Tech. Off. Int. Epiz.*, **30** (1), 139–148. Available at:

http://web.oie.int/boutique/index.php?page=ficprod&id_produit=945 &fichrech=1&lang=en (accessed on 28 September 2015).

_____

Table I

Summary of data used to populate the inputs used in the model

Step of model	Input	Value assigned	Source
PCT_PORK_INFECTED	IMPORT_P	*Uniform (0.42, 0.5)*	(6)
Proportion of all pork consumed in NZ that contains infectious PRRSV	PRRS_POSITIVE_COUNTRY_P	*Uniform (0.7546, 0.8455)*	Import data
	CONSUMER_READY_P	*Pert (0.0095, 0.02645, 0.0434)*	Import data
	CONTAMINATION_P	*Beta (19 + 1, 1027 – 19 + 1) x Beta (7 + 1, 11 – 7 + 1)*	(4)
TOT_NUM_WASTEFEEDERS	TOT_PIGPREMISES	*Pert (7,000, 12,000, 20,000)*	(6)
Total number of NZ pig-owning premises that feed raw meat waste to their pigs	WASTEFEED_P	*Pert (0.55, 0.8, 1.0)*	(6)
	MEATFEED_P	*Pert (0.11, 0.13, 0.2)*	(6, 9)
	NONCOMPLY_P	*Pert (0.75, 0.85, 0.95)*	(6)
TOT_NUM_PORKMEALS	PORK_CONS_PREMISES	Distribution fitted to survey data	(6)
Number of meals consumed per year using purchased pork in households which keep pigs	PREMISES_FREQ		
	PIGPREMISE_BUYPORK_P	*Beta (10 + 1, 40 – 10 + 1)*	(6)
	TOT_NUM_WASTEFEEDERS	Defined above	
SCRAP_WEIGHT	PORK_PER_MEAL	*Gamma (125,1)*	(6)
Weight of pork scrap generated from meal preparation	RAW_MEAT_SCRAP_P	*Pert (0, 0.01, 0.05)*	(10, 11, 12, 13, 14)
SCRAP_VIRAL_CONCENTRATION	PEAK_VIRUS_SLAUGHTER	Meta-analysis of published studies	(15, 16, 17, 18, 19)
Concentration of virus in scrap when consumed	PROP_AFTER_BLEEDING	*Uniform (0.002, 0.009)*	(7, 20)
	PROP_AFTER_MATURE	*Uniform (0.1, 0.86)*	(7, 21)
	PROP_AFTER_THAW	*Uniform (0.1, 0.68)*	(7, 15, 21)
	PROP_DELAY_PURCHASE	*Pert (0.001, 0.5, 1.0)* and *Pert (0.68, 0.92, 1.0)*	(15, 21)
	PROP_DELAY_DISPOSAL	*Discrete (0.96, 0.96, 0.62, 0.42, 0.4, 0.1)*	(7, 15, 21)
DOSE of virus consumed by pig	SCRAP_WEIGHT	Defined above	
	SCRAP_VIRAL_CONCENTRATION	Defined above	
P_SCRAP_INFECTIOUS	Dose/response curve for consumed DOSE	Beta-Poisson model fitted to published data	(10)
Probability that scrap will contain an infectious dose of PRRSV			
Estimate the expected annual number of PRRSV-infected premises	PCT_PORK_INFECTED	Defined above	
	TOT_NUM_PORKMEALS	Defined above	
	P_SCRAP_INFECTIOUS	Defined above	

PRRSV: porcine reproductive and respiratory syndrome virus

NZ: New Zealand

Fig. 1

Overview of model structure and parameters

The previous model structure (6) has been retained where possible and amendments made consistent with alternative assessments (7), to include available dose-response data (8)
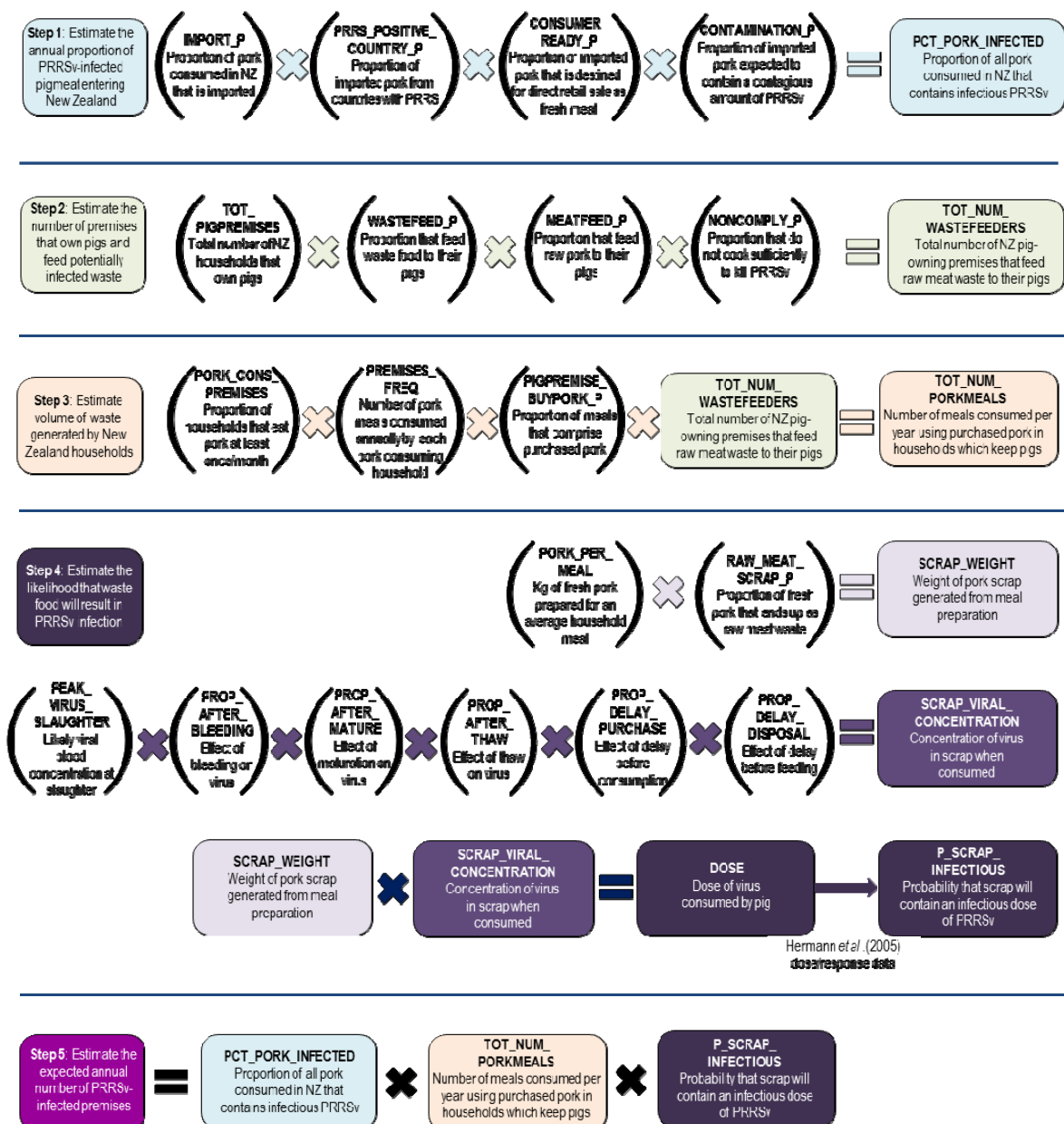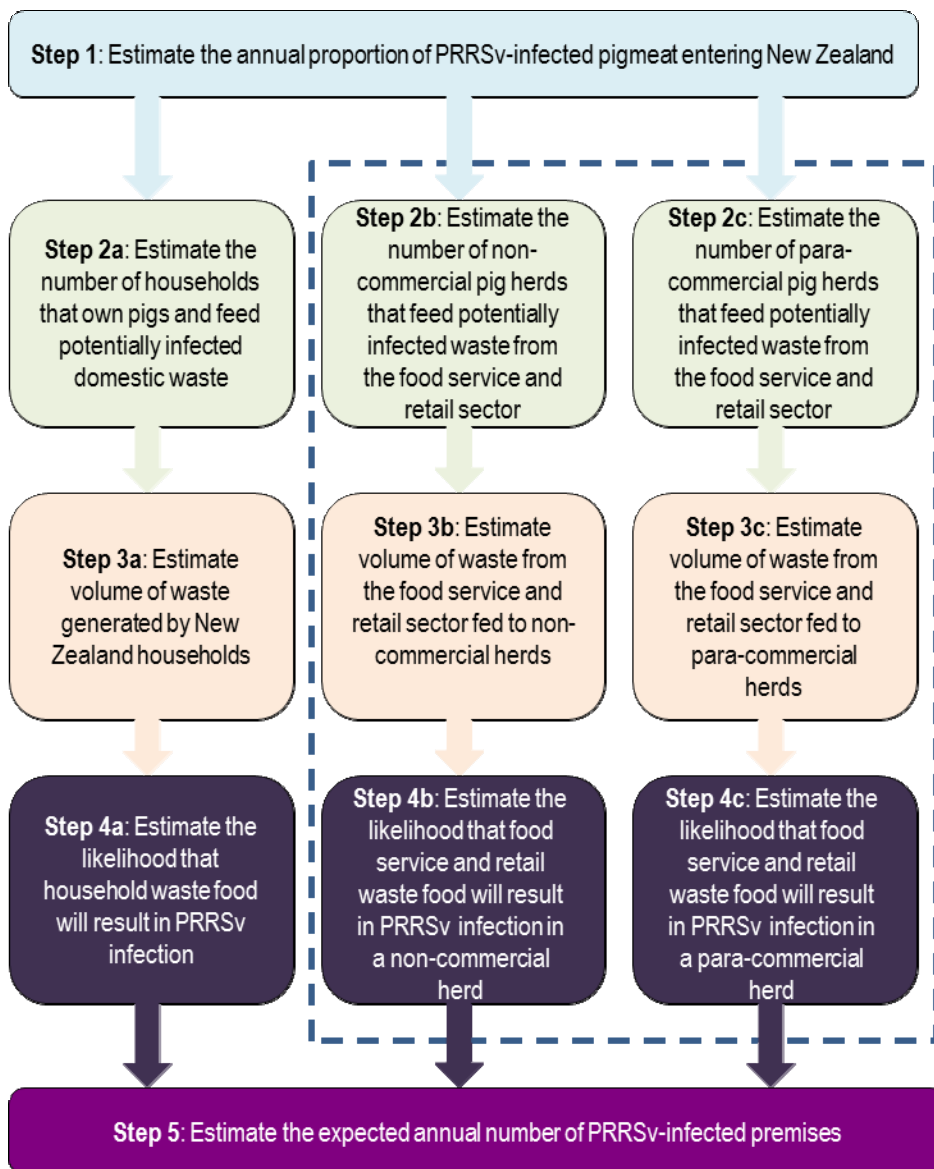


Fig. 1

Fig. 2

Major steps of the model

Dashed box outlines the additional pathways introduced as a result of the Expert Working Group process

Step 1: Estimate the annual proportion of PRRSv-infected pigmeat entering New Zealand

Step 2a: Estimate the number of households that own pigs and feed potentially infected domestic waste

Step 2b: Estimate the number of non-commercial pig herds that feed potentially infected waste from the food service and retail sector

Step 2c: Estimate the number of para-commercial pig herds that feed potentially infected waste from the food service and retail sector

Step 3a: Estimate volume of waste generated by New Zealand households

Step 3b: Estimate volume of waste from the food service and retail sector fed to non-commercial herds

Step 3c: Estimate volume of waste from the food service and retail sector fed to para-commercial herds

Step 4a: Estimate the likelihood that household waste food will result in PRRSv infection

Step 4b: Estimate the likelihood that food service and retail waste food will result in PRRSv infection in a non-commercial herd

Step 4c: Estimate the likelihood that food service and retail waste food will result in PRRSv infection in a para-commercial herd

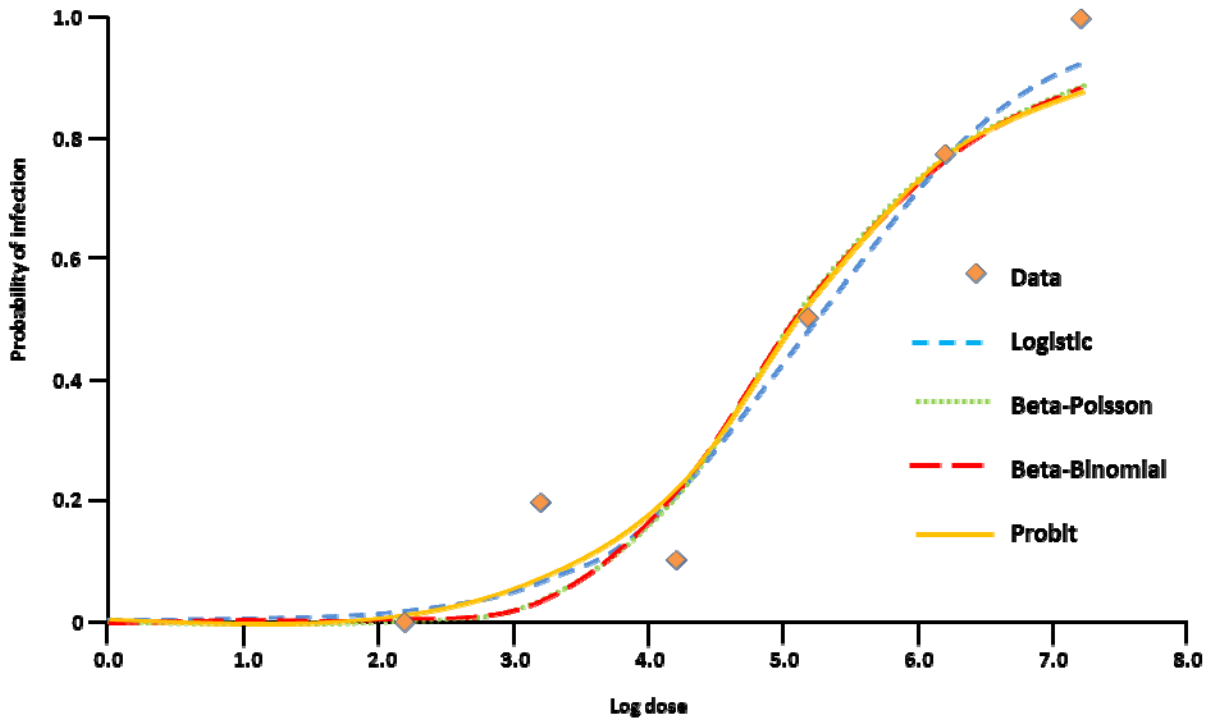**Step 5**: Estimate the expected annual number of PRRSv-infected premises

Fig. 2

Fig. 3

Tornado plot sensitivity analysis showing the influence of all model inputs on the overall output (the number of expected years between outbreaks)
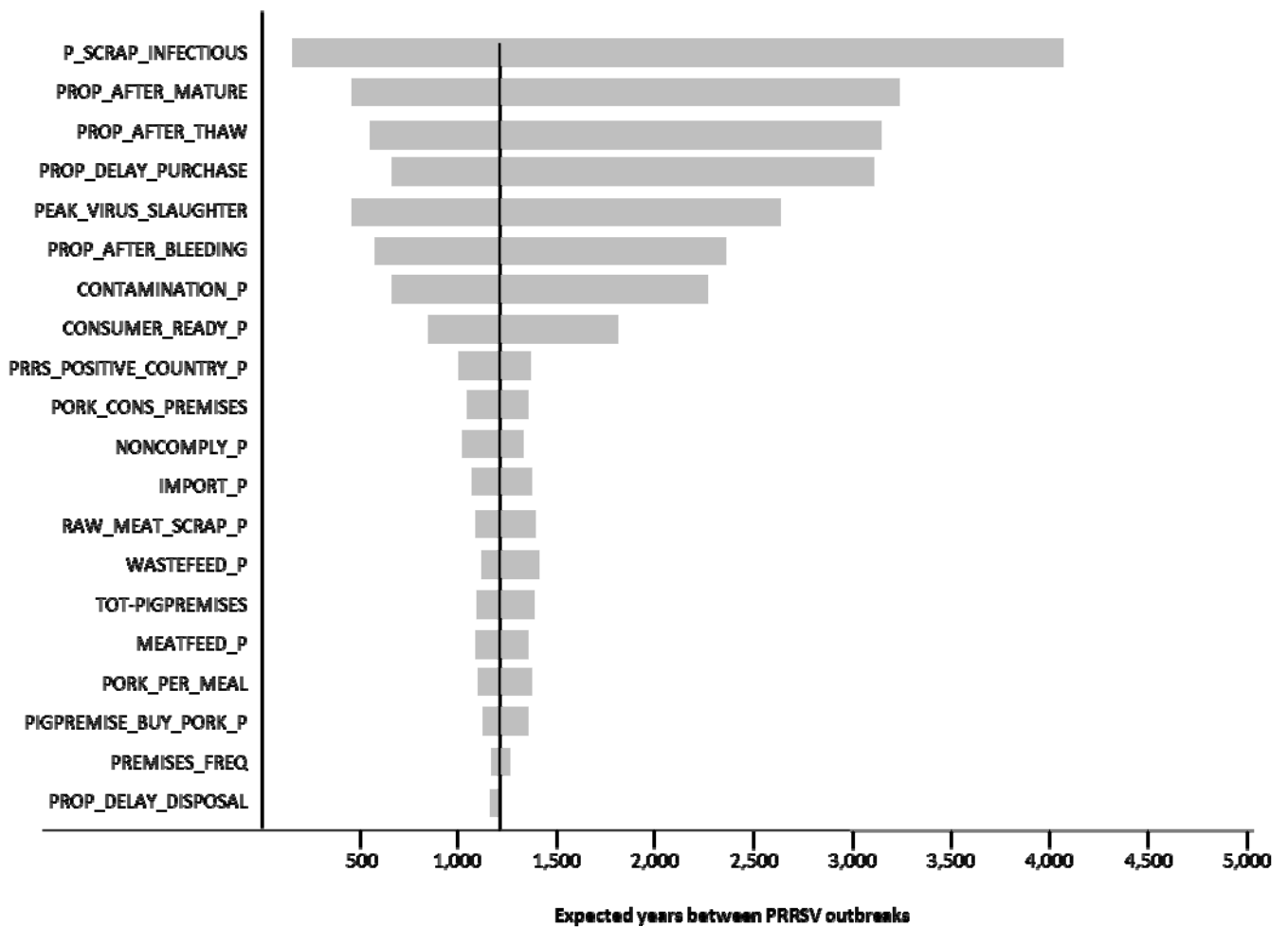
Fig. 4

Fit of four different 'dose-response' models to the feeding trial data reported by Hermann *et al.* (8)

Fig. 4

Fig 5

Extrapolation to low doses using three different 'dose-response' models, fitted to the feeding trial data reported by Hermann *et al.* (8)