

Causal hidden variable model of pathogenic contamination from pig to pork

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Abstract: Risk assessments relating to food safety over more than one step along a production chain are frequently hampered by lack of detailed quantitative data. This study set out to develop a Bayesian hidden variable model to integrate available limited data of the combined occurrence of three bacterial pathogens, *Listeria monocytogenes*, *Yersinia enterocolitica* and *Yersinia pseudotuberculosis*, with causal assumptions along three steps of pork production chain. The pathogen occurrence data were animal specific both on conventional and organic pig farms and at the abattoir, but merely farm specific at meat cutting plants. The model was able to incorporate all data concerning different types of testing at different steps of the chain, and missing data values were dealt with in a straightforward manner. It provides a tool for quantitative risk assessments and for estimating the causal risk mitigation effects by combining external data with the specific follow-up data. Intervention effects are provided with Bayesian credible intervals indicating the uncertainty due to all information sources included in the model. Combined prevalence in Finnish pork was estimated to be 1–11% and it could be reduced to 0–2% if head was removed intact and rectum sealed off.

Key words: Bayesian causal model; food safety; *Listeria*; pork production; risk assessment; *Yersinia*

Received January 2008; revised June 2008; accepted June 2008

1 Introduction

The aim of this paper is to present a hierarchical Bayesian model to study the combined occurrence of three bacterial pathogens causing foodborne infections in humans and the effect of possible options for public health risk reduction. Pork production has been associated with several bacteriological pathogens such as *Salmonella* spp., *Yersinia enterocolitica* and *Listeria monocytogenes* (Jacquet *et al.*, 1995; Buchholz

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et al., 2005; Fredriksson-Ahomaa *et al.*, 2006). These pathogens may colonize live pigs on the farms or they may occur in food via environmental contamination in the abattoir and at the meat processing plant. Since foodborne pathogens in humans can cause not only short-term gastrointestinal symptoms but also long-term health effects and even death (e.g., 20–40% of *L. monocytogenes* infections are fatal, especially in sensitive groups), it is important to know which factors affect on their occurrence on the food chain and how effective different control options are.

Quantitative microbiological risk assessment (QMRA) (McMeekin and Ross, 2002) is a powerful tool to study complex, multistage food production chains. However, typically each pathogen is assessed separately for each specific production chain. Tailored specific models of food pathways can be inflexible to be applied to new situations. Therefore, there is also a need for more generic and versatile models. An example of a generic import risk analysis that is not restricted to specific exporting countries can be found in the Australian Government Department of Agriculture, Fisheries and Forestry Generic Import Risk Analysis for Pig Meat, which is modelling the risk of 26 pathogens in imported pork meat (Banks *et al.*, 2004). In comparison, our model is generic in that it is not pathogen specific so that it can be used to assess one pathogen or the combined pathogen risk, based on pathogen sample data. In practice, several pathogens can coexist in the production chain and even at the plate at the time of consumption and therefore pose a multiple risk. Furthermore, the risk reduction measures may be targeted for one or several different pathogens and it is important to know how they affect the overall risk.

QMRA of food production chains are often hampered by lack of detailed production step-specific data concerning same individual animals or products from the same animals. Commonly, the data may be collected from different studies with different analytical methods and study protocols and it is therefore difficult to assess whether an observed change of prevalence at retail level is due, e.g., to processing contamination or differences in the primary production of animals.

Food chain QMRAs most often use Monte Carlo simulations of assigned distributions in the forward direction of the chain. Although such models can be fairly detailed, each distribution is fitted separately to contextual data as an expert's opinion, authors' opinion or guess (Alban and Stärk, 2005). The models are also typically modular, aiming at mechanistic description of each step of the food production chain (Rosenquist *et al.*, 2003; van der Gaag *et al.*, 2004; Lammerding, 2006).

We present in this paper a QMRA on three different pathogens combined (*Listeria monocytogenes*, *Y. enterocolitica* and *Yersinia pseudotuberculosis*) which is based on a dataset specially collected to study the combined risk at three different steps of pork production. To overcome the limitations of probabilistic estimation in forward simulating mechanistic models, we constructed a hierarchical Bayesian model derived from causal structures assigned on the basis of expert knowledge rather than assigned distributions or assigned point values. The role of inference is for extracting information from the data, within the Bayesian model, and hence to enable the computation of probabilistic predictions over the causal pathway. By conditioning

to all data from all production steps at once, the Bayesian model learns the unknown parameters and variables and produces a posterior distribution describing the remaining uncertainty jointly for all unknowns. The hidden variable model used employs various microbiological results which depend conditionally on the hidden indicator variable describing the general presence of pathogen carriage, together with the probability that this presence is manifested and detected with a specific indicator test of samples taken from the animal. Examples of hidden transition models can be found, e.g., in epidemiological applications (Congdon, 2003; Reilly *et al.*, 2004; Chung *et al.*, 2005), survey analysis (Gajewski *et al.*, 2006) and a risk assessment of broiler production (Ranta and Maijala, 2002). A closely related modeling problem is analysing measurable changes under differing conditions (Clarke, 2005).

2 Data collection

Three groups of pig farms, namely five organic farms, five conventional farms with a similar production and capacity to the organic farms and five conventional large meat producing farms were selected from south-west Finland for the study. From each farm, about 25 finishers (weight over 85 kg) were selected. All samples were gathered between June 2003 and January 2005 and examined for *L. monocytogenes*, *Y. enterocolitica* and *Y. pseudotuberculosis* using conventional microbiological culture and typing methods at the Department of Food and Environmental Hygiene, Faculty of Veterinary Medicine, Helsinki University. Isolation of *L. monocytogenes* was performed according to the ISO standard (Anonymous a, 1996) with the modification that an LMBA (*Listeria Monocytogenes* blood agar) plate was used instead of an Oxford plate. Isolation of *Y. enterocolitica* and *Y. pseudotuberculosis* was performed according to modified Nordic Committee on Food Analysis (NCFA) method (Anonymous b, 1996) and ISO (Anonymous, 2003) standard using CIN (cefsulodin–irgasan–novobiocin) plating after ITC (irgasan, ticarcillin and potassium chlorate) enrichment and cold enrichment of seven and 14 days. The status of pathogen carriage of each pig was measured at two time points by microbiological testing: at the farm from living pigs and after slaughter. While the first measurement was a single faecal sample, the second measurement consisted of several different post-mortem samples per animal taken at the abattoir. These consecutive samples were taken from the same individuals which allowed detailed studies of the change of carriage over time. A third sample was collected from the fresh meat, but these tests could not be linked to the previous individual animals. Instead, the fresh meat samples mainly represent the same farm and were collected after meat cutting from the assortment intended for minced meat production.

The observed combined status of a sample is positive if any of the three pathogens is detected. It is negative only if none of them is detected. Since all the tests were focused on different aspects of pathogen carriage and contamination (faecal, intestinal, pluck set, tonsils, carcass, fresh meat), there is a need to define a comparable variable over the production stages to monitor changes in pathogen risk level.

3 Animal-specific model

Our probability model of the hidden carriage status describes the probability of combined carriage of pathogens of a live pig at the farm and the transition probability from this initial hidden carriage status to the second hidden carriage status at the abattoir. This model was further extended to describe a third measurement which was taken from meat samples at the cutting plant. However, the third measurement no longer represented individual-specific data but merely a processed sample from the slaughter lot associated with the farm. The model then describes the subpopulation level effect only, that is the probability of a certain meat sample result given the prevalence in certain pig population at the abattoir testing. There were some missing values in the complete dataset. With the hierarchical Bayesian model, these partial data were naturally included in the analysis, hence using all the available data. The full model is shown as a directed acyclic graph (DAG) in Figure 1.

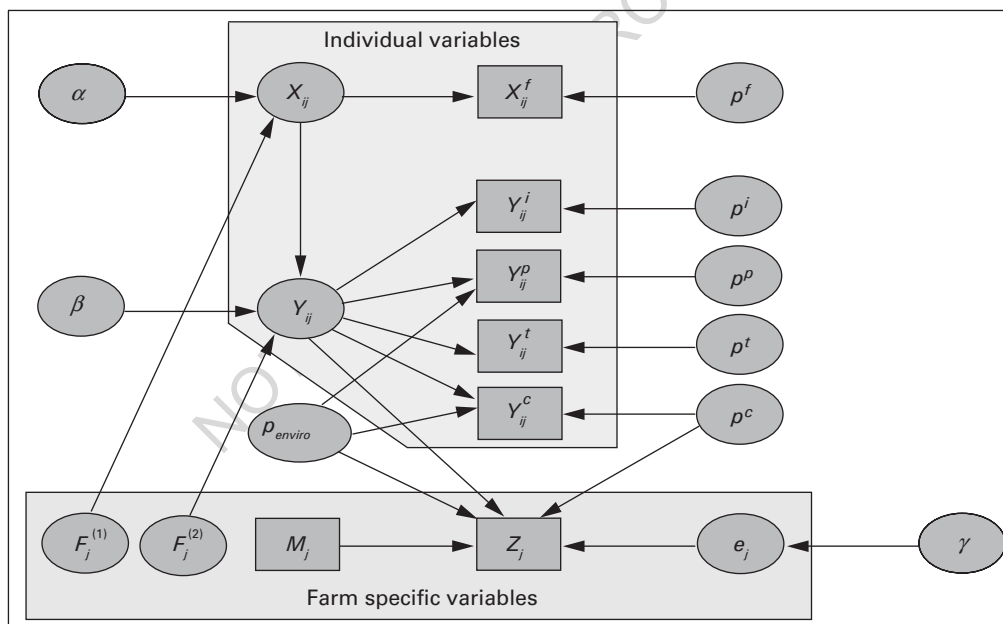


Figure 1 Graphical representation (DAG) of the conditional dependencies in the model. Arrows denote a stochastic dependence. Observed data variables (animal-specific test results $X_{ij}^f, Y_{ij}^i, Y_{ij}^p, Y_{ij}^t, Y_{ij}^c$ and farm-specific meat sample positives Z_j and sample size M_j) are shown in boxes, unknown variables and parameters in ellipsoids. Upper indices: (*f*) faecal, (*i*) intestinal, (*p*) pluck, (*t*) tonsils, (*c*) carcass. Lower indices: *i* = *i*th animal, *j* = *j*th farm. (α, β) = parameters of hidden status model; $F_j^{(i)}$ = farm-specific effect. e_j = farm-abattoir specific cross contamination effect. Covariates (farm types) are not shown explicitly

3.1 Modelling the farm prevalence of hidden carriage

3.1.1 Hidden variable model

As a starting point, selected animals at selected farms were marked and faecal samples collected for pathogen analysis. Each animal, i , at each farm, j , was assumed to be either hidden positive or negative, $X_{ij} = 1$ or 0. By ‘hidden positive’ we mean that at least one of the three pathogens was present, at least to some extent, at least somewhere in the animal, but not necessarily detectable at a specific sampling site. Otherwise, the hidden carriage was defined negative. By definition, this hidden status is not directly observable. Hence, a model of observations is needed, further parameterized given the unknown hidden carriage status. The probability of hidden carriage was set to depend on farm level covariates: type of production (organic farm/small conventional farm/large conventional farm) and general hygiene scores of the farm (low/high). Note that since hygiene scores were designed to identify and count several risk factors, a high hygiene score corresponds to high risk, i.e., a *lower* level of hygiene. These scores were obtained from a previous study based on site visits and questionnaires sent to the selected farms (Siekkinen *et al.*, 2006) and grouped into two categories ‘low’ and ‘high’. In the low-score group, there were two organic, three small conventional and three large conventional farms. In the high-score group, the counts were three, two and two, respectively. The hidden carriage status for the i th pig $i = 1, \dots, n_j$ at farm j can thus be modelled as a four-parameter logit model:

$$\text{logit}(P(X_{ij} = 1)) = \alpha_0 + \alpha_1 \text{organic}_j + \alpha_2 \text{large}_j + \alpha_3 \text{high_score}_j + F_j^{(1)}, \quad (3.1)$$

with a $N(0, \tau)$ prior for the α -parameters, and for the farm-specific effects $F_j^{(1)}$. Here, τ denotes precision, $\tau = 1/\sigma^2$.

Hence, the conventional small farms with low hygiene score were taken as a baseline category and this choice can be made arbitrarily. Term $F_j^{(1)}$ is the farm-specific effect which accounts for the fact that the animal level measurements were clustered into farms.

3.1.2 Faecal sample data at farm

If the unobservable hidden carriage, X_{ij} , is true (i.e., $X_{ij} = 1$), then there is a chance for the i th animal that the carriage will be detected in faecal samples taken at the farm. The conditional probability for the faecal test result (observation X_{ij}^f) is then

$$P(X_{ij}^f | X_{ij}, p^f) = \text{Bernoulli}(X_{ij} p^f). \quad (3.2)$$

The parameter p^f describes ‘expression and detection probability’, that the carriage is found specifically in faecal samples, given that the animal carries any of the pathogens at all. The parameter thus includes also the microbiological test

sensitivity. All isolated strains were confirmed according to international standards, so specificity is assumed to be virtually 100%. The animal may be a hidden positive although not detected in any site-specific test. For parameter p^f , we assume no prior information. Therefore, a non-informative uniform prior distribution, $\text{Uniform}(0, 1)$, was chosen and the parameter will be estimated from the posterior distribution together with other unknowns.

3.2 Modelling the abattoir prevalence of hidden carriage

3.2.1 Hidden variable model

Each individual pig was examined again at the abattoir post-mortem by microbiological testing of the intestinal sample, pluck set sample, carcass sample and tonsils sample. Similar to the faecal test model, the conditional probability of these was defined to depend on the hidden carriage status at slaughter, Y_{ij} , which itself depends on the earlier hidden carriage status at the farm, X_{ij} :

$$P(Y_{ij} = 1 \mid X_{ij}) = \begin{cases} p^{(01)}, & \text{if } X_{ij} = 0, \\ p^{(11)}, & \text{if } X_{ij} = 1. \end{cases} \quad (3.3)$$

Notice that $p^{(00)} = 1 - p^{(01)}$ and $p^{(10)} = 1 - p^{(11)}$, so that the probabilities constitute a transition probability matrix which defines a hidden Markov chain model (Karlin and Taylor, 1975; MacDonald and Zucchini, 1997)

$$P = \begin{bmatrix} p^{(00)} & p^{(01)} \\ p^{(10)} & p^{(11)} \end{bmatrix}. \quad (3.4)$$

Again, the model for hidden status, Y_{ij} , was defined as

$$\text{logit}(P(Y_{ij} = 1)) = \beta_0 + \beta_1 \text{organic}_j + \beta_2 \text{large}_j + \beta_3 \text{high_score}_j + \beta_4 1_{\{X_{ij}=1\}} + F_j^{(2)}, \quad (3.5)$$

where β_4 is the effect of earlier hidden positive status at farm. Again, $N(0, \tau)$ prior was set to the β -parameters and the farm effects. The transition probabilities represent everything that occurs between the two hidden states X_{ij} and Y_{ij} . Without additional measurements in between, it is not possible to draw separate estimates describing intermediate steps, e.g., transporting of pigs to the abattoir. Elapsed time between the two measurements is assumed to be relatively constant (roughly a week). Therefore, the exact time was not included in the discrete time transition model.

3.2.2 Sample data at slaughter

If any of the three pathogens was found at a slaughter sample (intestinal, pluck set, tonsils or carcass sample), the overall pathogenic status at that sample was defined positive ($Y_{ij}^{\text{observed}} = 1$). If none of the three pathogens was detected, the overall pathogenic status at that sample was defined negative ($Y_{ij}^{\text{observed}} = 0$). If

some of the three test results were missing, the rest being negative, the status was defined unknown ($Y_{ij}^{observed} = \text{NA}$). Each sample test result was defined to depend conditionally on the hidden carriage status, Y_{ij} , at slaughter, reflecting the causal assumptions:

$$\begin{aligned} Y_{ij}^i &| Y_{ij}, p^i \sim \text{Bernoulli}(Y_{ij} p^i) \\ Y_{ij}^p &| Y_{ij}, p^p, p_{enviro} \sim \text{Bernoulli}(1 - (1 - Y_{ij} p^p)(1 - p_{enviro})) \\ Y_{ij}^t &| Y_{ij}, p^t \sim \text{Bernoulli}(Y_{ij} p^t) \\ Y_{ij}^c &| Y_{ij}, p^c, p_{enviro} \sim \text{Bernoulli}(1 - (1 - Y_{ij} p^c)(1 - p_{enviro})). \end{aligned} \quad (3.6)$$

Note that the subindex i refers to the same animal for both variables X and Y , for a farm j . The parameters p^i , p^p , p^t and p^c each describes the conditional probability that the carriage is expressed and detected in a site-specific test ($i = \text{intestinal}$, $p = \text{pluck set}$, $t = \text{tonsils}$, $c = \text{carcass}$), given that the carriage is present at all in the animal. Parameter p_{enviro} describes the chance of carriage due to the abattoir environment. The essential causal assumption was that the carcass and pluck set positives could occur either due to a contaminated animal itself or due to the environmental cross contamination from the abattoir (Autio *et al.*, 2000; Nesbakken *et al.*, 2003). In contrast, the tonsils and intestinal samples can only be contaminated when the animal itself was previously infected. No prior knowledge was assumed for these parameters and thus a non-informative prior density, Uniform(0, 1), was used for each.

4 Fresh meat prevalence model

4.1 Group specific model

As an end point of the follow-up study of the pork production chain, meat samples from the cutting plant were analyzed. Each meat sample represents the quality of meat from an animal, merely from a specific farm. The meat samples cannot be linked to any individual animals and they may have been cross contaminated during the process of cutting at the cutting plant (Chasseignaux *et al.*, 2001). Reflecting causal assumptions, the conditional probability model was set to depend on population characteristics of the original animals in a slaughter lot and the contamination effect due to abattoir processing. The meat samples Z_{mj} , $m = 1, \dots, M_j$, concerning farm j at the abattoir were observed to be either positive or negative. The number of meat samples (M_j) was zero, 10, 15 or 20 depending on the availability. If any of the three pathogens was found in a single sample, the result was defined as observed positive $Z_{mj} = 1$. If all the three results were negative, the overall result was defined as observed negative ($Z_{mj} = 0$). If some pathogen test results were missing while others

were negative, the observed result was defined as unknown ($Z_{mj} = \text{NA}$). A possible model for the observed number of positives $Z_j = \sum_{m=1}^{M_j} Z_{mj}$ in M_j meat samples originating from farm j is

$$Z_j \sim \text{Binomial}(M_j, \theta_j), \quad (4.1)$$

where

$$\theta_j = \frac{\bar{\mu}_j e_j}{1 - \bar{\mu}_j} \bigg/ \left(\frac{\bar{\mu}_j e_j}{1 - \bar{\mu}_j} + 1 \right) \quad (4.2)$$

and $\bar{\mu}_j$ is the mean of

$$\mu_{ij} = 1 - (1 - Y_{ij} p^c)(1 - p_{\text{enviro}}). \quad (4.3)$$

Here, μ_{ij} is the conditional probability of carcass contamination, given hidden carrier status of the i th animal and the chance of environmental contamination. Parameter e_j measures how the expected prevalence (in odds scale) of the meat samples differs from the expected slaughter carcass prevalence (in odds scale) for a farm j . A reasonable choice for an uninformative prior of e_j could be any density $\pi(e_j)$ over \mathfrak{R}^+ which gives $P(e_j < 1) = \int_0^1 \pi(e_j) \mathrm{d}e_j = 0.5$, that is the probability for any e_j to be less than one equals the probability to be greater than one. For example, an exponential distribution with parameter $\lambda = 1.5936$ has the required property. A uniform density with similar property would be $\text{Uniform}(0, 2)$ which is more restrictive. An alternative way is to construct a hierarchical prior. For example,

$$\begin{aligned} \log(e_j) &\sim N(\gamma, 1)I(-4.60517, 4.60517) \\ \gamma &\sim N(0, 10). \end{aligned} \quad (4.4)$$

As a prior, this mixture density is flexible enough to allow very small and very large values of e_j for different slaughter animal groups, but we used a constraint $(e^{-4.60517}, e^{4.60517}) \approx (0.01, 100)$ to prevent overflow into too large or too small values. The median of the hierarchical prior for e_j is one, but the width of the prior is consistent with the fact that in some groups there was a clear increase from the observed carcass prevalence to the observed prevalence in meat sample—and that in some slaughter groups no positive meat samples were detected, see Figure 2.

5 The effect of interventions on slaughter practice

Based on available literature, pathogenic prevalence reduction in resulting pork meat can be achieved by the adoption of more hygienic methods in slaughter (and cutting) process. Specifically, we focus on (i) removal of pig head intact and (ii) sealing off the rectum. The reduction factor of an intervention was included in the model as an exogenous control variable $\kappa \in \mathfrak{R}^+$ describing this causal effect (Lindley, 2002). Its

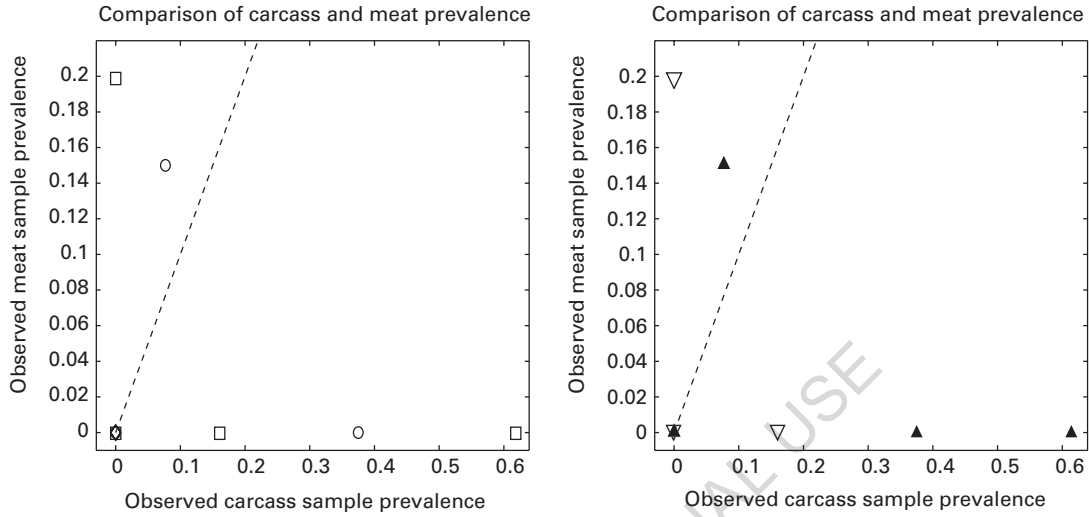


Figure 2 Comparison of combined observed carcass and meat prevalence. Left: Organic farms (\circ), small conventional farms (\diamond) and large conventional farms (\square). Right: Low hygiene score (white inverted triangle), high hygiene score (black triangle)

Note: Some values coincide at origin

value was specified as a prior probability distribution that was chosen as the posterior distribution estimated from data described elsewhere in literature. Such data were available from Denmark, Sweden and Norway, reporting observed results from test group of pigs (with intervention) and from control group (without intervention). Our aim was to use these sources of information in combination with our data, to assess the probable meat prevalence that would result from similar interventions if they were applied in Finnish population, accounting for uncertainty about the treatment effect κ and uncertainty about the Finnish expected carcass prevalence $E(\mu_j)$ for a farm in each farm type category. The expected meat prevalence was then calculated, analogous to equation (4.2), as a function of κ :

$$\theta_j(\kappa) = \frac{E(\mu_j) \bar{e} \kappa}{1 - E(\mu_j)} \bigg/ \left(\frac{E(\mu_j) \bar{e} \kappa}{1 - E(\mu_j)} + 1 \right), \quad (5.1)$$

where $\bar{e} = \frac{1}{15} \sum_{j=1}^{15} e_j$, the average contamination effect over the 15 actual farms in our study and

$$\begin{aligned} E(\mu_j) &= 1 - (1 - \text{logit}^{-1}(\bar{\beta}_j) p^c)(1 - p_{enviro}), \\ \bar{\beta}_j &= \beta_0 + \beta_1 \text{organic}_j + \beta_2 \text{large}_j + \beta_3 \text{high_score}_j + \text{logit}^{-1}(\bar{\alpha}_j) \beta_4, \quad (5.2) \\ \bar{\alpha}_j &= \alpha_0 + \alpha_1 \text{organic}_j + \alpha_2 \text{large}_j + \alpha_3 \text{high_score}_j. \end{aligned}$$

It was acknowledged that the reduction factor can be prevalence dependent, but this dependence was not possible to estimate since there are no published studies of interventions under a range of different levels of baseline prevalence. According to published studies on the effect of removal of pig head (Olsen *et al.*, 2001), the reduction of *Salmonella* in the carcass could be quantified. The pigs in these published reports were studied separately in a test batch (with head removal) and in a control batch (without removal). Prevalence in the two batches could be estimated as posterior distributions $p_{test} \sim \text{Beta}(167, 485)$ and $p_{control} \sim \text{Beta}(187, 322)$, based on the Danish sample data and uniform prior. This provides an estimate of the effect $\kappa_{head} = [p_{test}/(1 - p_{test})]/[p_{control}/(1 - p_{control})]$, resulting to mean 0.60 and 95% confidence interval (CI) [0.46, 0.76].

Another source of information concerns the effect of sealing off the rectum with a plastic bag (Nesbakken *et al.*, 1994). This was studied by using the method for one group of pigs and comparing to a control group (data from Sweden and Norway). Based on a uniform prior and published sample results on *Y. enterocolitica*, the carcass prevalence in test batch and control batch could be quantified as $p_{test} \sim \text{Beta}(2, 120)$ and $p_{control} \sim \text{Beta}(13, 109)$. For $\kappa_{rectum, Y.e.}$, this results to mean 0.15 and 95% CI [0.02, 0.48]. Similarly, the resulting distributions concerning *Listeria innocua* were $p_{test} \sim \text{Beta}(7, 55)$ and $p_{control} \sim \text{Beta}(21, 40)$, resulting to mean 0.26 and 95% CI [0.08, 0.59] for $\kappa_{rectum, L.i.}$. Both these sample data were added up and the resulting distribution was $p_{test} \sim \text{Beta}(8, 174)$ and $p_{control} \sim \text{Beta}(33, 148)$. The mean and 95% CI for κ_{rectum} were then 0.21 and [0.08, 0.43]. The effect of applying both the interventions jointly was computed by multiplying the two factors, $\kappa_{head}\kappa_{rectum}$.

Note that these sources of information were based on *Salmonella*, *Y. enterocolitica* and *L. innocua*. None of them applies exactly for our study on joint prevalence of *Y. enterocolitica*, *Y. pseudotuberculosis* and *L. monocytogenes*. It is a common situation in QMRAs that available data are not exactly ideal for the assessment but they convey information in a similar or closely related setting. For example, the dose–response model of a pathogen for a target population may be lacking (Lindqvist and Westöö, 2000) microbial test characteristics such as sensitivity may be reported for a different setting in another country (Sandberg *et al.*, 2002), or data concerning one pathogen need to be used to estimate the plausible reduction of another (Alban *et al.*, 2002). Meta-analysis (hierarchical modelling) could be used to make a synthesis of different studies, but even the number of relevant studies can also be quite small. Hence, the proposed reduction factors here should be taken indicative of similar reduction in similar pathogens. The estimated $\theta_j(\kappa)$ are shown in Table 1. Indistinguishable genotypes among human and porcine strains, the high prevalence of *Y. enterocolitica* among pigs and the contamination of pork with *Y. enterocolitica* strongly indicate that pigs are the primary source of sporadic human *Y. enterocolitica* infection in Finland (Fredriksson-Ahomaa *et al.*, 2006). In a follow-up discussion (Nesbakken and Skjerve, 1996) stated that the human yersiniosis had reduced after the introduction of the plastic

Table 1 Observed prevalence in meat and predicted expected prevalence $\theta_j(\kappa)$ with and without intervention

	Observed sample prevalence in meat		Posterior 95%CI	
	Low H-score	High H-score		
Organic	NA	0.05 (3/60)		
Small	0 (0/20)	0 (0/15)		
Large	0.067 (3/45)	0 (0/15)		
$\theta_j(\kappa = 1)$	Posterior mean		Posterior 95%CI	
Organic	0.05	0.05	[0.02, 0.10]	[0.02, 0.11]
Small	0.04	0.04	[0.01, 0.08]	[0.02, 0.10]
Large	0.05	0.05	[0.02, 0.11]	[0.02, 0.11]
$\theta_j(\kappa_{head})$	If head removed intact			
Organic	0.03	0.03	[0.01, 0.07]	[0.01, 0.07]
Small	0.02	0.03	[0.01, 0.05]	[0.01, 0.06]
Large	0.03	0.03	[0.01, 0.07]	[0.01, 0.07]
$\theta_j(\kappa_{rectum})$	If rectum sealed off			
Organic	0.01	0.01	[0.00, 0.03]	[0.00, 0.03]
Small	0.01	0.01	[0.00, 0.02]	[0.00, 0.03]
Large	0.01	0.01	[0.00, 0.03]	[0.00, 0.03]
$\theta_j(\kappa_{head\kappa_{rectum}})$	If head removed intact and rectum sealed off			
Organic	0.01	0.01	[0.00, 0.02]	[0.00, 0.02]
Small	0.00	0.01	[0.00, 0.01]	[0.00, 0.02]
Large	0.01	0.01	[0.00, 0.02]	[0.00, 0.02]

bag technique in most slaughterhouses in Norway suggesting practical relevance of the intervention at the slaughterhouse. Comparable results for listeriosis are not available.

6 Results

6.1 Descriptive data analysis

The observed combined prevalence was studied in each category of farms (organic, small conventional and large conventional) and also in groups defined by the hygiene score (low and high). In the faecal samples taken at the farm, the conventional small farms showed smaller observed combined prevalence than large conventional or organic farms, see Figure 3 (left). Also, the farms with lower hygiene score tend to show lower combined prevalence, see Figure 3 (right). The relation between carcass prevalence and the prevalence observed in meat samples was studied by plotting these for each farm type, and also in each hygiene score group (low and high). The descriptive results from carcass to meat samples show a decrease of prevalence towards zero except for two farms, see Figure 2. Note that meat samples were available only from three organic, three small conventional and four large conventional farms. The results represent the outcome of the same culture methods uniformly for all samples over the three specified steps in production chain. If other methods were used, the absolute level of combined pathogen prevalence could be markedly different.

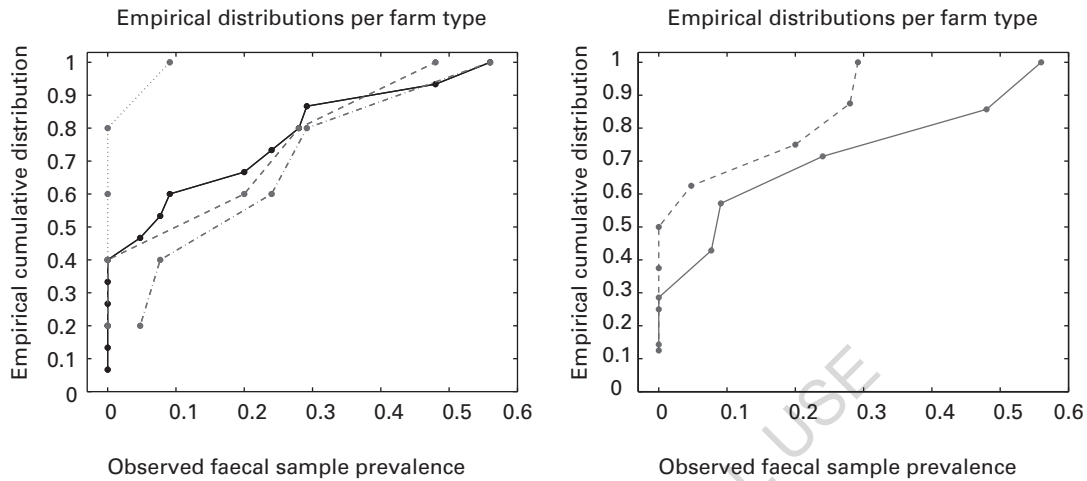


Figure 3 Empirical cumulative distributions of combined observed faecal prevalence. Left: organic (-.-.-), small conventional (-.-.-), large conventional (-.-.-) and all farms (-.-.-). Right: low hygiene score farms (-.-.-) and high hygiene score farms (-.-.-)

6.2 Model fit

A summary statistic (Gelman *et al.*, 2004) was defined using the observed number of positive meat samples z_j for each farm, if available. Thus, we computed

$$T(z, \theta) = \sum_j \frac{(z_j - \theta_j M_j)^2}{\theta_j(1 - \theta_j)M_j} \quad (6.1)$$

at each iteration, since this depends on the unknown parameters θ_j . The reference distribution was obtained by sampling $T(z^{pred}, \theta)$, where z^{pred} and θ were sampled from posterior predictive distribution and posterior distribution, respectively. The Bayesian p -value was computed by averaging over θ with respect to the posterior, to get $P(T(z^{pred}, \theta) > T(z, \theta)) = 0.4523$.

Another model fit diagnostics was also computed. Twenty-five new animals were predicted for a single new farm in each of the six farm categories defined by the production type (organic, small conventional and large conventional) and the hygiene score (low and high), and the results were compared with the observed average prevalence for each of the five samplings (faecal, intestinal, pluck set, tonsils and carcass). When the model predicts well, the corresponding predicted quantities should fall half of the time below/above the observed values. This can be monitored by defining a Bernoulli variable (indicator function) which takes values 0 and 1 correspondingly. Thus, the Bernoulli variable should have mean 1/2 and standard deviation 1/2, and

the mean of all 30 such variables

$$\frac{1}{30} \sum_{j=1}^6 \left[\mathbf{1}_{(\bar{x}_j^{f,pred} > \bar{x}_j^{f,obs})} + \mathbf{1}_{(\bar{x}_j^{i,pred} > \bar{x}_j^{i,obs})} + \mathbf{1}_{(\bar{x}_j^{p,pred} > \bar{x}_j^{p,obs})} + \mathbf{1}_{(\bar{x}_j^{c,pred} > \bar{x}_j^{c,obs})} + \mathbf{1}_{(\bar{x}_j^{t,pred} > \bar{x}_j^{t,obs})} \right] \quad (6.2)$$

should have mean 1/2 and standard deviation $1/(2\sqrt{30}) \approx 0.091$. The posterior predictive mean (0.49) and standard deviation (0.093) were in agreement.

Sensitivity of the model was studied by setting different values for the precision parameter of the $N(0, \tau)$ prior, in the range $\tau \in [0.5, 10]$. Values below 0.5 led to runtime errors. Above this range, the results were qualitatively the same. Value of $\tau=1$ was used as the default model for which the results are discussed.

6.3 Estimates from the model

The results were computed using WinBUGS version 1.4 (Lunn *et al.*, 2000). These results were obtained from an MCMC sample of 40 000 that were collected by taking every 10th iteration from the original sample of 400 000. Convergence was checked by visual plots of the MCMC paths and by calculating the modified Gelman–Rubin convergence statistics in WinBUGS. Based on the estimated effects $\alpha_0, \dots, \alpha_3$, there is modest evidence of increased pathogenic risk at farms that are organic. For the organic farm effect, the posterior probability of exceeding zero was the highest $P(\alpha_1 > 0 \mid \text{data}) = 0.95$, but barely significant. At the abattoir level, the estimated effects, β_0, \dots, β_4 , show no clear significant evidence of higher or lower risk. For the large conventional farm effect, the posterior probability of exceeding zero was the highest $P(\beta_2 > 0 \mid \text{data}) = 0.97$, indicating some evidence of risk, though. However, the highest point estimate (posterior mean) was for β_4 showing a tendency for increased risk of being hidden positive at slaughter if the same animal was hidden positive at farm. Hence, the probability $P(\beta_4 > 0 \mid \text{data}) = 0.99$. Posterior estimates for the baseline parameters α_0, β_0 , the effects for organic α_1, β_1 , large farm α_2, β_2 , high hygiene score α_3, β_3 and the effect of previous hidden positive status β_4 are shown in Table 2. There were only two to three farms in each of the six categories, hence the uncertainty about farm type-specific parameters was fairly large.

The farm-specific effects F_j are justified because the animal level measurements are clustered into farms. If these were not included in the model, the results suggested an increased risk associated with organic and conventional large farms (Table 2). The result indicates that variation between farms may be more important than the farm type as such. This was also seen as variation between the estimated F_j . However, it should be noted that the number of farms was not large and therefore the inclusion of farm-specific effects tends to absorb the farm type effect. Yet, their inclusion is needed due to the clustering of the animals within farms. For more conclusive results, the number of farms should be increased. (Although, large proportion, 5/19, of organic farms in Finland were included already in the dataset; 5/6 in the geographical study area.) The identifiability of farm effects $F_j^{(1)}, F_j^{(2)}$ and farm type effects α, β was

Table 2 Posterior estimates of parameters, with and without farm effects and using different prior precision. Default model has $\tau = 1$

	$F_j^{(1)} = F_j^{(2)} = 0, \tau = 1$		$\tau = 1$		$\tau = 0.5$		$\tau = 10$	
	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
α_0	-2.09	[-3.14, -1.00]	-2.17	[-3.24, -1.10]	-2.52	[-3.94, -1.13]	-0.49	[-1.10, 0.15]
α_1	1.80	[0.87, 2.87]	1.02	[-0.18, 2.23]	1.38	[-0.26, 3.04]	0.37	[-0.14, 0.90]
α_2	1.57	[0.67, 2.57]	0.65	[-0.53, 1.84]	0.87	[-0.74, 2.51]	0.21	[-0.31, 0.73]
α_3	0.70	[-0.08, 1.58]	0.30	[-0.79, 1.38]	0.42	[-1.05, 1.90]	0.17	[-0.33, 0.69]
β_0	-0.04	[-0.60, 0.50]	0.23	[-0.67, 1.15]	0.20	[-1.04, 1.45]	0.30	[-0.09, 0.69]
β_1	1.03	[0.04, 2.08]	0.87	[-0.35, 2.11]	1.00	[-0.68, 2.71]	0.48	[-0.01, 0.96]
β_2	1.18	[0.26, 2.14]	1.12	[-0.06, 2.32]	1.32	[-0.31, 2.95]	0.56	[0.09, 1.04]
β_3	0.36	[-0.40, 1.12]	0.58	[-0.52, 1.68]	0.69	[-0.81, 2.21]	0.33	[-0.12, 0.78]
β_4	1.88	[0.57, 3.30]	1.53	[0.26, 2.94]	1.99	[0.33, 3.93]	0.58	[0.05, 1.12]

explored by scatter plots of the two-dimensional marginal posterior distributions for each pair $(F_j^{(1)}, \alpha_k)$ and $(F_j^{(2)}, \beta_k)$, $j = 1, \dots, 15$, $k = 0, \dots, 3$, showing identifiability but the order of magnitude of one-dimensional marginal standard deviations was similar. The latter parameters explain farm and farm type effects on the results at abattoir, after the sampling at farm, although the previous state of a pig appears to be the best predictor. Moreover, the hygiene score was weakly positively associated with pathogenic risk ($P(\alpha_3 > 0 \mid \text{data}) = 0.71$, $P(\beta_3 > 0 \mid \text{data}) = 0.85$). This may be due to the fact that each score is a sum of many variables and this can average out the possible effect that could be associated with some specific explanatory variables.

A number of interesting conditional posterior probabilities can be calculated from the model. For example, to study the causal effect of hidden carriage, it may be of interest to ask how likely it is that an animal that was hidden positive at farm will be hidden negative at the slaughter stage, and how this depends on the farm type? Different transition (posterior predictive) probabilities are shown in Table 3 for different farm types. It can be seen that small conventional farms have smaller transition probabilities $P(0 \rightarrow 1)$, and higher transition probabilities $P(1 \rightarrow 0)$ compared to other farm types. Also, high hygiene score tends to increase the probability $P(0 \rightarrow 1)$ and decrease the probability $P(1 \rightarrow 0)$. Note that the probabilities are derived from the joint posterior density, and as such

Table 3 Transition probabilities (posterior predictive) for hidden carriage. (Prior predictive: 0.5 each)

	$X = 0 \rightarrow Y = 1$		$X = 1 \rightarrow Y = 0$	
	Low H-score	High H-score	Low H-score	High H-score
Organic	0.73	0.82	0.09	0.05
Small	0.55	0.68	0.17	0.11
Large	0.78	0.85	0.07	0.04

they are single numbers (posterior means). For example, for a small conventional farm: $P(0 \rightarrow 1 \mid \text{data, small conv. farm}) = \int_{-\infty}^{\infty} \text{logit}^{-1}(\beta_0)\pi(\beta_0 \mid \text{data})d\beta_0$, where π denotes the posterior density. In other words, this gives the posterior predictive value. Similarly, the model can provide posterior probabilities addressing any specific farm or animal included in this study. Such functionality could have purpose in making prognosis for a specific farm for which we have partial sample data.

Given that the hidden status at farm, X_{ij} , for an animal is true, the posterior probability that the contamination is detected in faeces was 0.68. Moreover, given that the hidden status at slaughter, Y_{ij} , is true, the posterior probability that the contamination is detected in tonsils was 0.78. It is somewhat less probable to detect contamination in intestinal (0.21), pluck set (0.20) or carcass sample (0.11), given the hidden status is true. The average cross contamination factor of the odds, $\sum_{j=1}^{15} e_j/15$, had posterior mean 0.43 and 95% CI [0.14,1.00] which clearly shows a decrease from the average carcass contamination to the meat contamination (observed odds ratios, when computable and finite, ranged from 0 to 2.1, with average 0.53, see Figure 2). The posterior probability of environmental contamination from abattoir was 0.02. Individual parameters, such as p_{faecal} , may not be accurately estimated because the same observed sample prevalence at each farm can be equally well explained by a large hidden carriage $X_j = \sum_{i=1}^{26} X_{ij}/26$ together with a small chance to detect at faecal tests p_{faecal} , or vice versa. This is exemplified in Figure 4 showing how the two-dimensional joint marginal posterior density is informative.

The combined pathogen predictive prevalence in fresh meat was estimated to be approximately 0.05 with no clear differences between farm types (Table 1). The removal of head intact could result to a combined approximate pathogen prevalence of 0.03 in meat, and the sealing of rectum could result to an approximate prevalence of 0.01. If both interventions were applied, this could result to a prevalence of 0.01, or even lower in small conventional farms with low hygiene risk score. This indicates that the pathogen prevalence in fresh meat is low, and with these interventions on slaughter procedures the chance of high prevalence could be further reduced. There were modest differences in prevalence between farm types early in production chain, but less so in produced fresh meat.

7 Discussion

Small conventional pig farms showed lower combined pathogenic prevalence than other farm types when this was measured as underlying hidden presence of *L. monocytogenes*, *Y. enterocolitica* or *Y. pseudotuberculosis* in the pig. The farms were also grouped according to overall hygiene scores, based on a questionnaire and a farm visit. Farms with lower hygiene score (i.e., good hygiene) were modestly associated with lower overall pathogenic prevalence. This indicates that, if further

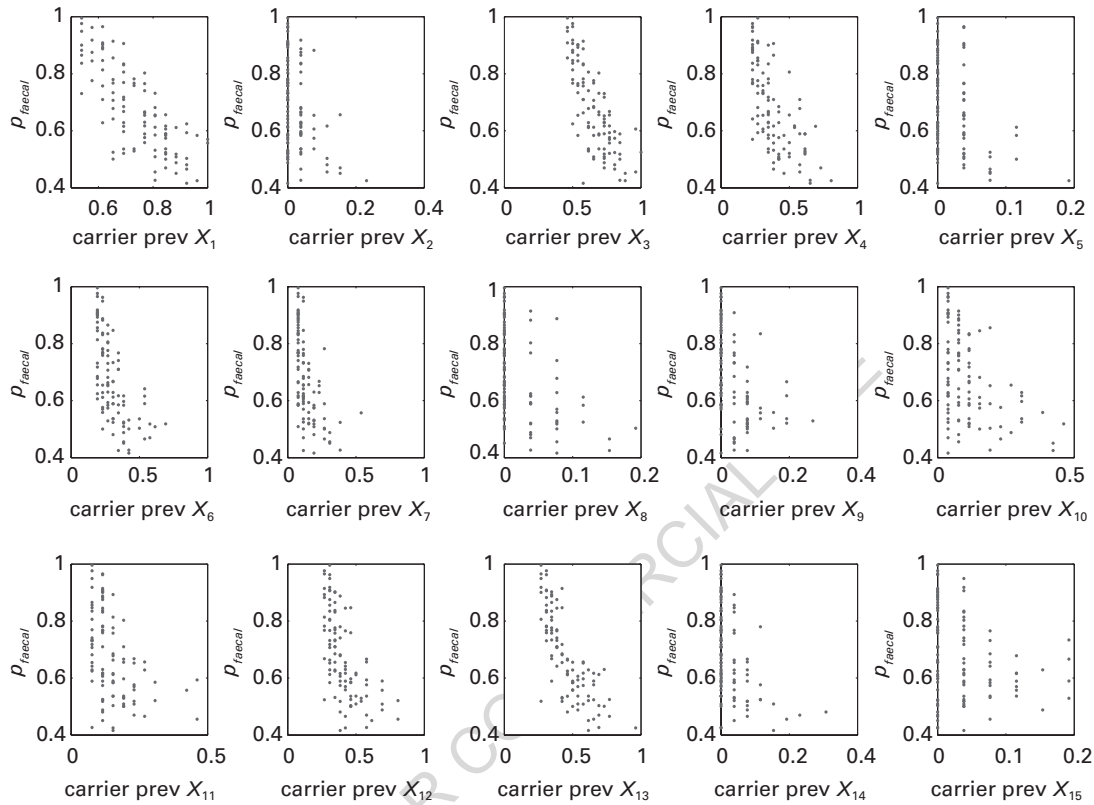


Figure 4 Posterior marginal densities (MCMC samples) of hidden carriage prevalence $X_j = \sum X_{ij}/26$ and p_{faecal} as scatter plots for each farm

developed, the scoring method might be a useful tool for classifying the farms with respect to overall pathogenic carriage, but key variables should be identified for more informative scoring. However, if the pig was hidden carrier at the farm, there was also increased risk of hidden carriage at the abattoir. Contamination prevalence in pork meat was estimated to be lower than the expected carcass prevalence, indicating a minor cross contamination effect at the abattoir and cutting plant, i.e., good slaughtering hygiene, which may still be reduced by more hygienic techniques at slaughter and cutting, such as removal of the head intact and sealing off the rectum.

The model was based on a dataset presenting the combined pathogen status with three pathogens to describe the overall risk. A sample positive occurred if any of the three pathogens was detected. The same model could well be applied to each of the pathogens separately. Therefore, extensions to multivariate models could also be possible in further research.

The model utilized Bayesian analysis with hidden variables. Therefore, it was possible to include all data even though some observations were missing. Another major advantage is that the model aims to provide a unified basis for comparing underlying prevalence changes in contamination over production chain even though all measurements were targeted on different parts of the animal (ante-mortem, post-mortem), with no repetition of exactly the same measurements. Third, the model provides a method for information synthesis of all data at three different points of the production chain. All prior distributions could be chosen as uninformative, in contrast to informative probabilities describing expert knowledge which can be very problematic in practice (Berger, 2006; Goldstein, 2006). This concerns especially quantities that are not directly, if at all, observable and which may be interlinked. Therefore, the method also suggests a way to establish a sound empirical and testable basis for a scientific risk assessment, instead of resorting to many arbitrary uncertainty distributions based on inherently vague expert opinions. Hence, the efforts may be better spent on eliciting expert knowledge concerning a concise model structure that conveys information about the assumed or known causalities and the structure of uncertainties in the given problem. The estimated parameter values then rely on the actual data employed in a holistic probabilistic manner captured by the hierarchical Bayesian model. Finally, hypothetical and uncertain intervention effects can be quantified by two steps: First, an informative distribution for that effect is elicited from literature, external data or expert opinions. Second, the obtained distribution is combined with the posterior density of the model to produce an altered distribution, hence combining both uncertainties. The WinBUGS code of the model is available from the authors, as well as a WinBUGS script for running in batch mode, and a Matlab code for retrieval of results. These are also available from the journal web archive.

Acknowledgements

This study was supported by research funding from the Ministry of Agriculture and Forestry, Finland. We also express our gratitude to the abattoirs and farms for their kind co-operation in data collection, and to Hardy Christensen (Danish Meat Research Institute, Roskilde, Denmark) for original data concerning reduction of *Salmonella* on swine carcasses. We thank the anonymous referee for constructive comments that helped to improve the paper.

References

- Alban L, Olsen A-M, Nielsen B, Sørensen R, *et al.* (2002) Qualitative and quantitative risk assessment for human salmonellosis due to multi-resistant *Salmonella* Typhimurium DT104 from consumption of Danish dry-cured pork sausages. *Preventive Veterinary Medicine*, 52, 251–65.

- Alban L and Stärk KDC (2005) Where should the effort be put to reduce the *Salmonella* prevalence in the slaughtered swine carcass effectively? *Preventive Veterinary Medicine*, **68**, 63–79.
- Anonymous, a (1996) *ISO 11290-1*. Microbiology of food and animal feeding stuffs horizontal method for the detection and enumeration of *Listeria monocytogenes*. Part 1: Detection method. Geneva: International Organisation for Standardisation.
- Anonymous, b (1996) *NCFA Method No. 117*. *Yersinia enterocolitica*. Detection in foods, 3rd edition. Nordic Committee on Food Analysis.
- Anonymous (2003) *ISO 10273*. Microbiology of food and animal feeding stuffs—horizontal method for the detection of presumptive pathogenic *Yersinia enterocolitica*. Geneva: International Organisation for Standardisation.
- Autio T, Säteri T, Fredriksson-Ahomaa M, Rahkio M, et al. (2000) *Listeria monocytogenes* contamination pattern in pig slaughterhouses. *Journal of Food Protection*, **63**, 1438–442.
- Banks D, Martin R, Doyle K, Cutler R, et al. (2004) Risk analysis panel on Generic Import Risk Analysis (IRA) for pig meat. http://www.daff.gov.au/_data/assets/pdf_file/0014/11930/2004-01a.pdf.
- Berger J (2006) The case for objective bayesian analysis. *Bayesian Analysis*, **1**, 385–402.
- Buchholz U, Brodhun B, Brockmann SO, Dreweck CM, et al. (2005) An outbreak of *Salmonella* München in Germany associated with raw pork meat. *Journal of Food Protection*, **68**, 273–76.
- Chasseignaux E, Toquin M-T, Ragimbeau C, Salvat G, et al. (2001) Molecular epidemiology of *Listeria monocytogenes* isolates collected from the environment, raw meat and raw products in two poultry- and pork-processing plants. *Journal of Applied Microbiology*, **91**, 888–99.
- Chung H, Park Y and Lanza ST (2005) Latent transition analysis with covariates: pubertal timing and substance use behaviours in adolescent females. *Statistics in Medicine*, **24**, 2895–910.
- Clarke PS (2005) Analysing change based on two measures taken under different conditions. *Statistics in Medicine*, **24**, 3401–415.
- Congdon P (2003) *Applied Bayesian modelling*. Chichester, UK: John Wiley & Sons Ltd.
- Fredriksson-Ahomaa M, Stolle A, Siitonen A and Korkeala H (2006) Sporadic human *Yersinia enterocolitica* infections caused by bioserotype 4/O:3 originate mainly from pigs. *Journal of Medical Microbiology*, **55**, 747–49.
- Gajewski BJ, Thompson S, Dunton N, Becker A, et al. (2006) Inter-rater reliability of nursing home surveys: a Bayesian latent class approach. *Statistics in Medicine*, **25**, 325–44.
- Gelman A, Carlin JB, Stern HS and Rubin DB (2004) *Bayesian data analysis*, 2nd edition. Boca Raton: Chapman & Hall/CRC Press.
- Goldstein M (2006) Subjective Bayesian analysis: principles and practice. *Bayesian Analysis*, **1**, 403–20.
- Jacquet C, Catimel B, Brosch R, Buchrieser C, et al. (1995) Investigations related to the epidemic strain involved in the French listeriosis outbreak in 1992. *Applied and Environmental Microbiology*, **61**, 2242–246.
- Karlin S and Taylor HM (1975). *A first course in stochastic processes*, 2nd edition. San Diego: Academic Press, Inc.
- Lammerding AM (2006). Modeling and risk assessment for *Salmonella* in meat and poultry. *Journal of AOAC International*, **89**, 543–52.
- Lindley DV (2002). Seeing and doing: the concept of causation. *International Statistical Review*, **70**, 191–214.
- Lindqvist R and Westöö A (2000) Quantitative risk assessment for *Listeria monocytogenes* in smoked or gravad salmon and rainbow

- trout in Sweden. *International Journal of Food Microbiology*, **58**, 181–96.
- Lunn DJ, Thomas A, Best N and Spiegelhalter D (2000) WinBUGS—a Bayesian modelling framework: concepts, structure, and extensibility. *Statistics and Computing*, **10**, 325–37.
- MacDonald IL and Zucchini W (1997) *Hidden markov models and other models for discrete-valued time series*. London: Chapman & Hall.
- McMeekin TA and Ross T (2002). Predictive microbiology: providing a knowledge-based framework for change management. *International Journal of Food Microbiology*, **78**, 133–53.
- Nesbakken T, Eckner K, Høidal HK and Røtterud O-J (2003) Occurrence of *Yersinia enterocolitica* and *Campylobacter* spp. in slaughter pigs and consequences for meat inspection, slaughtering, and dressing procedures. *International Journal of Food Microbiology*, **80**, 231–40.
- Nesbakken T, Nerbrink E, Røtterud O-J and Borch E (1994) Reduction of *Yersinia enterocolitica* and *Listeria* spp. on pig carcasses by enclosure of the rectum during slaughter. *International Journal of Food Microbiology*, **23**, 197–208.
- Nesbakken T and Skjerve E (1996). Interruption of microbial cycles in farm animals from farm to table. *Meat Science*, **43**(suppl), S47–S57.
- Olsen A-M, Jensen T, Dahl J and Christensen H (2001) Reduction in level of Salmonella on swine carcasses after slaughter without splitting the head. In Peter J. van der Wely, ‘Salinpork 2001, Proceedings of the 4th International Symposium on the epidemiology and control of Salmonella and other food borne pathogens in pork’, Leipzig, Germany, pp. 124–26.
- Ranta J and Maijala R (2002) A probabilistic transmission model of Salmonella in the primary broiler production chain. *Risk Analysis*, **22**, 47–58.
- Reilly M, Salim A, Lawlor E, Smith O, *et al.* (2004) Modelling infectious disease transmission with complex exposure pattern and sparse outcome data. *Statistics in Medicine*, **23**, 3013–32.
- Rosenquist H, Nielsen NL, Sommer HM, Nørrung B, *et al.* (2003) Quantitative risk assessment of human campylobacteriosis associated with thermophilic *Campylobacter* species in chickens. *International Journal of Food Microbiology*, **83**, 87–103.
- Sandberg M, Hopp P, Jarp J and Skjerve E (2002) An evaluation of the Norwegian *Salmonella* surveillance and control program in live pig and pork. *International Journal of Food Microbiology*, **72**, 1–11.
- Siekkinen K-M, Nuotio L, Ranta J, Laukkanen R, *et al.* (2006) Assessing hygiene proficiency on organic and conventional pig farms regarding pork safety: a pilot study in Finland. *Livestock Science*, **104**, 193–202.
- van der Gaag M A, Vos F, Saatkamp HW, van Boven M, *et al.* (2004) A state-transition simulation model for the spread of Salmonella in the pork supply chain. *European Journal of Operational Research*, **156**, 782–98.

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