A Quantitative Microbial Risk Assessment to Evaluate Zoonotic Risks: Human Salmonellosis through Household Consumption of Fresh Minced Pork Meat in Belgium as Example

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Development of a Quantitative Microbial Risk Assessment for Human Salmonellosis through Household Consumption of Fresh Minced Pork Meat in Belgium

Abstract

A quantitative microbial risk assessment (QMRA) according to the Codex Alimentarius Principles is conducted to evaluate the risk on human salmonellosis through household consumption of fresh minced pork meat in Belgium. The quantitative exposure assessment is carried out by building a modular risk model, called the XXX-model, which covers the pork production from farm to fork. In the XXX-model, the food production pathway is split up in six consecutive modules: (1) primary production, (2) transport & lairage, (3) slaughterhouse, (4) post-processing, (5) distribution & storage and (6) preparation & consumption. All the modules are developed to resemble as closely as possible the Belgian situation making use of the available national data. Several statistical refinements and improved modeling techniques are proposed. The model produces highly realistic results. The baseline predicted number of annual salmonellosis cases is 20513 (st. dev. 9061.45). The risk is estimated higher for the susceptible population (est. 4.713×10^{-5}; st. dev. 1.466×10^{-5}) compared to the normal population (est. 7.704×10^{-6}; st. dev. 5.414×10^{-6}) and is mainly due to undercooking and for a smaller extent to cross contamination in the kitchen via cook's hands.

Keywords: *Salmonella*, fresh minced pork meat, quantitative microbial risk assessment, modular risk model, farm-to-fork

Note to the referees: For reasons of anonymity, we replaced the name of the research consortium/ risk model with XXX.

1 Introduction

Current and emerging threats to human health posed by zoonotic infectious diseases are put high on the agenda of international and national authorities. Zoonoses are infectious diseases caused by pathogens transferred from animals to humans and are often foodborne. One of the eight zoonoses listed in the European Union (EU) Zoonoses Monitoring Directive [1], for which continuous monitoring is mandatory, is salmonellosis. In Belgium, *Salmonella* was the second most common bacterial cause of gastrointestinal diseases with 35.1 reported human cases per 100,000 inhabitants in 2006. The serovar Typhimurium (STM) was responsible for 49.4% of these reported cases [2] and it was the predominant serovar isolated from pork during monitoring in the EU [3] and in Belgium [4], suggesting that consumption of pork is a major risk factor for human STM salmonellosis.

In the EU Regulation on the control of *Salmonella* and other zoonotic agents [5], it is stated that proper and effective measures are to be taken to detect and control these agents at all relevant stages of the food production chain. Therefore, the Belgian Federal Public Service of Health, Food Chain Safety and Environment (FPS) decided to financially support an interdisciplinary Belgian research consortium (XXX-consortium) to develop a methodology to quantitatively assess the risk for human salmonellosis through consumption of fresh minced pork meat. The most important deliverable of the XXX-consortium was the development of a quantitative microbial risk assessment (QMRA) that allows testing mitigation strategies by means of what-if-scenario and cost-benefit analyses. Other deliverables of the consortium were: (a) development of a methodology to objectively describe the quality of the information and assumptions used to build the QMRA; (b) development and refinement of statistical methodology supporting QMRA's and (c) identification of data gaps and collecting new data to be used in the QMRA. This paper discusses the first deliverable whereas the other deliverables are discussed elsewhere (for reasons of anonymity, the references will be provided later).

A (quantitative) microbial risk assessment ((Q)MRA) is a science-based methodology that can be used to investigate health risks following ingestion of foodborne pathogens and can serve as a basis for risk management decisions. Several QMRA's have already been developed to describe the contamination of pork meat with *Salmonella*. Besides the country-specific differences, these models vary in approach and level of detail. In the UK, Hill et al. [6] developed a modular risk model for *Salmonella* Typhimurium in pork, mixed meat products and bacon covering the food pathway from farm to fork. The principal objective of the model of Van der Gaag [7] was

to investigate the economic aspects of *Salmonella* reducing interventions in the Netherlands. In Denmark, Alban & Stärck simulated prevalences throughout the production chain until the final carcass [8]. In Finland, Ranta et al. [9] modelled the *Salmonella* infection route from slaughterhouse to consumption with modules developed using a Bayesian approach.

In this article, a QMRA following the Codex Alimentarius Principles is presented to assess health risks associated with the household consumption of fresh minced pork meat contaminated with *Salmonella* in Belgium. To quantitatively assess exposure, a modular risk model, in which the food production pathway is split up in consecutive modules with the output of one module serving as input of the next module, is developed. In particular, a risk model is built that covers the food pathway from farm to fork comprising the same six modules as the model developed by Hill et al. [6]. These are: (1) primary production, (2) transport & lairage, (3) slaughterhouse, (4) post-processing, (5) distribution & storage and (6) preparation & consumption. Throughout the text, for reasons of convenience, we call the risk model presented in this article the XXX-model. Although the XXX-model and the risk model developed by Hill et al. [6] comprise the same modules, the implementation of the modules is completely different. The XXX-model is developed to closely resemble the Belgian pork production and consumption incorporating as many national data as possible. Hereby, priority is given to Belgian data published in the scientific peer-reviewed international literature, describing the base year 2006. If not available, data from other (recent) years or from other European countries are used. In addition, several statistical refinements and improved modeling techniques have been proposed. In the XXX-model, the pre-harvest stage, where living animals are at focus, is developed using concepts of infectious disease modeling. The harvest and post-harvest stage, where animal food products and the transmission of microbial agents in these products are at focus, are modeled using the Modular Process Risk Model (MPRM) methodology proposed by Nauta [10]. The latter methodology provides a structured approach to build pathogen transmission models, making it especially suitable to model complex and lengthy food pathways. In particular, in a MPRM, changes in prevalence, bacteriological concentration and unit size within each module are modeled by means of six basic processes, two of which are microbial processes (*e.g.* growth and inactivation) and the remaining four are food handling processes (*e.g.* cutting, cross-contamination, mixing and partitioning). For the characteristics of these processes, the reader is referred to Nauta [10].

2 Quantitative Microbial Risk Assessment

According to the Codex Alimentarius Commission [11] a QMRA should include four steps; (1) hazard identification, (2) exposure assessment, (3) hazard characterization and (4) risk characterization. These steps are successively discussed in the subsequent sections.

2.1 Hazard Identification

In Belgium, non-typhoidal salmonellosis and campylobacteriosis are the two most frequently reported foodborne illnesses. Figure 1 gives a graphical representation of the reported *Salmonella* cases in Belgium per 100,000 inhabitants from 1984 to 2006 [12]. This figure also displays the evolution of the *Salmonella* serotypes *S.* Enteritidis (SE) and *S.* Typhimurium (STM). As can be seen, the reported number of Belgian human salmonellosis cases shows an increase until 1999 reaching 154 cases per 100,000 habitants. Since 2000, an overall decline of the incidence of salmonellosis was observed, diminishing up to 35.1 cases per 100,000 inhabitants in 2006. It appears to be largely attributable to a decrease in SE. As can be seen in Figure 1, SE declined drastically from 2004 onwards where as STM remained more or less stable. Although no causal relationship between vaccination of laying hens in Belgium and the decline in human SE infections has been established, the increased vaccination status of flocks has most probably contributed to decreased egg contamination [2].

According to Mead et al. [13], more than 95% of all *Salmonella* infections are foodborne. In the Netherlands, eggs and poultry meat were responsible for 39% and 21% of human salmonellosis cases, respectively, whereas pork was responsible for 25% of the cases and beef for about 10% of the cases [14]. A case-control study of risk factors for salmonellosis in the Netherlands revealed that risk factors for endemic STM infection were occupational exposure to raw meat (Odds Ratio (OR): 3.0, 95% CI: 1.1-7.9), use of proton pump inhibitors (OR: 8.3, 95% CI: 4.3-15.9), playing in a sandbox (for children aged 4-12 years) (OR: 2.4, 95% CI: 1.6-3.7), consumption of undercooked meat (OR: 2.2, 95% CI: 1.1-4.1) and use of antibiotics (OR: 1.9, 95% CI: 1.0-3.4) [15]. Children younger than 5 year are the most affected age group. In Belgium, this age group represents 44% of all cases of salmonellosis reported [12], but sampling of faecal material for culturing is most frequently done by physicians in this age group.

[Figure 1 about here.]

2.2 Exposure Assessment

A quantitative exposure assessment is carried out by building a modular risk model, called the XXX-model, covering the pork meat production from farm to fork. The XXX-model is developed using Matlab (7.1) and Monte Carlo (MC) simulation is used to obtain stochastic estimates of the output variables. The input variables of the different modules in the XXX-model are expressed as distributions to reflect stochastic uncertainty in estimated values, natural variability and epistemological uncertainty [16]. Table 1 gives a detailed overview of the XXX-model, the distributions of the input parameters and their major sources and references. Clarification of all notation used in the text can also be found in this table. Hereby, the convention for index notation is that index i refers to animal, j to herd/batch and k to portion. A schematic overview of the XXX-model is given in Figure 2. The left side of this figure displays the different modules of the model, the middle part gives a flowchart describing the changes in unit size along the pork food pathway and the right side displays which of the MPRM basic processes are modeled in each of the (post)-harvest modules. As can be seen in Figure 2, meat cuts from different animals originating from different herds are joined together in one meat mix, which is subsequently partitioned in servable portions, to be consumed by one person. In the XXX-model, all pigs of which meat cuts will eventually end up in the same meat mix and all portions thereof are monitored simultaneously in one iteration of the model. This results in fast computation and, more importantly, allows correct modeling of the *Salmonella* load of a meat mix by simply taking the sum of *Salmonella* loads of the composing meat cuts. The details on the number and herd origin of the pigs to be monitored simultaneously (*i.e.* composition of a meat mix) are given first. Subsequently, the different modules of the XXX-model being (1) primary production, (2) transport & lairage, (3) slaughterhouse, (4) post-processing, (5) distribution & storage and (6) preparation & consumption are successively discussed.

Composition meat mix

Because of the simultaneous monitoring of all pigs composing one meat mix, the number and herd origin of these pigs need to be determined at the start of every iteration of the model. To this end, the weight of the meat mix is calculated first as the sum of the weights of 5000 portions, $W_{mix} = \sum_{k=1}^{5000} W_{portion\,k}$ (corresponds to ±450kg, which is in agreement with reality in practice). In 9.2% of the cases [17], the meat mix contains pork meat only (pure pork minced meat). In the remaining cases, the meat mix is a mixture of pork meat and beef, calf or lamb (mixed minced meat). The weight of the meat mix corresponding to pork is a fraction of the total weight of meat

mix or $W_{mix\,pork} = k_1 \times W_{mix}$ where k_1 equals one in case of a pure pork meat mix and follows a betapert distribution with minimum value = 0.5, most likely value = 0.7 and maximum value = 0.9, otherwise. Then, a first batch j is randomly selected, of which maximally n_j animals will (partially) end up in the meat mix. The weights of the meat cuts of these n_j animals are sampled and the cumulative sums $W_{I_j} = \sum_{i=1}^{I_j} W_{cut\,ij}$ for $I_j = 1, ..., n_j$ are calculated. Then, the largest value I_j for which $W_{I_j} < W_{mix\,pork}$ is obtained and denoted ${I_j}^*$. If ${I_j}^* < n_j$, the size of the $({I_j}^* + 1)^{th}$ meat cut is adjusted such that $W_{{I_j}^*+1} = W_{mix\,pork}$. If ${I_j}^* = n_j$, a second batch j' is randomly selected and the weights of the meat cuts of the $n_{j'}$ animals are simulated. Now the cumulative sums are calculated as $W_{I_{j'}} = \sum_{i=1}^{n_j} W_{cut\,ij} + \sum_{i=1}^{I_{j'}} W_{cut\,ij'}$ for $I_{j'} = 1, ..., n_{j'}$. Denote the largest value $I_{j'}$ for which $W_{I_{j'}} < W_{mix\,pork}$ as $I^*_{j'}$. Again, if ${I_{j'}}^* < n_{j'}$, the size of the $({I_{j'}}^* + 1)^{th}$ meat cut is adjusted such that $W_{{I_{j'}}^*+1} = W_{mix\,pork}$. If ${I_{j'}}^* = n_{j'}$, a third batch j'' is selected et cetera. This process is repeated until the sum of the weights of sampled meat cuts equals the target weight $W_{mix\,pork}$. During partitioning, a meat mix is divided in K portions. For practical reasons, it is assumed that every meat mix is partitioned in exactly $K = 5000$ portions. As such, each iteration of the model yields exactly the same number of risk estimates.

[Figure 2 about here.]

[Table 1 about here.]

[Table 2 about here.]

[Table 3 about here.]

[Table 4 about here.]

Module 1: Primary Production

In the first module, the *Salmonella* serological status (positive or negative) of the pigs composing a meat mix is simulated. To this end, a density estimate of the within-herd seroprevalence is first obtained using data (base year 2006) from the serological *Salmonella* surveillance programme, organized by the Belgian Federal Agency for the Safety of the Food Chain (FASFC). Within this programme, all Belgian fattening herds are monitored by taking blood samples of 10 to 12 pigs every 3 to 4 months per year. Of these blood samples, the *Salmonella*-specific antibodies are determined by an indirect ELISA and the results are reported as sample-to-positive ratios (SP-ratios), which are then transformed to seroprevalence data S_{ij} (threshold value $\zeta = 0.25$). For more details on the the surveillance programme and the serological data, the reader is referred to Van der Stede et al. [18].

Based on these serological data, within-herd seroprevalences $\bar{s}_j$ are obtained by calculating the serological mean for each herd separately, $\bar{s}_j = \frac{1}{m_j} \sum_{i=1}^{m_j} S_{ij}$ with m_j being the number of samples taken in herd j (ranging from 2 to 41). For the current analysis, only data on 303 herds is used for which additional information on biosecurity is available [19]. The results are graphically represented by a means of a normalized histogram in Figure 3. Both a parametric and non-parametric density are estimated using weighted maximum likelihood (MLw) with the weights w_j being proportional to the herd-specific sample size n_j to account for differences in these sample sizes. The parametric estimate is obtained using the beta likelihood whereas the non-parametric estimate is obtained using P-splines density smoothing as proposed by Eilers & Marx [20]. The P-splines density estimate is preferred to the beta density estimate because of its flexibility and non-asymptotic behavior at 0 and 1.

The obtained density estimate is used to calculate the number of seropositive animals $N_{pos\,j}$ in batch j. Of this batch having size $N_{batch\,j}$, n_j animals will partially end up in the meat mix. To simulate the serological status of these n_j pigs, the hypergeometric distribution is used since the n_j pigs are taken from the batch without replacement.

In addition, for the data shown in Figure 3, information on internal and external biosecurity measures is available from a survey conducted by Ribbens et al. [19]. The serological data [18] and biosecurity data [19] are merged to conduct a *Salmonella* risk factor analysis, being presented elsewhere [21]. The latter analysis indicate that nose contact between pigs of different pens is the most important risk factor. This confirms the results obtained by Hill et al. [22] based on a stochastic transmission model for the dynamics of *Salmonella* infection within typical British

pig farms showing that the most effective *Salmonella* control strategies at primary production are those that reduce between-pen transmission.

[Figure 3 about here.]

Module 2: Transport & Lairage

In the first module, the *Salmonella* serological status at primary production is obtained. However, not the animals being seropositive at primary production but the animals being internally infected (carrier + excreting) and/or externally contaminated at slaughter potentially contaminate the pork carcasses [23]. First, the serological status at primary production is converted to internal infection status after transport & lairage with an animal being assumed to be internally contaminated if the mesenteric lymph node (MLN) sample is positive (carrier pig) and/or if the colon content (CC) is positive (excreting pig). The corresponding conditional probabilities could be derived using data from two Belgian studies [24], [25]. Second, the internal infection status of a pig is converted to external contamination status.

Internal infection

In a first Belgian study, conducted by Nollet et al. [24], the association between the results from serological screening (indirect ELISA, threshold value $\zeta = 0.25$) and the isolation of *Salmonella* from MLN at slaughterhouse is investigated. In this study, 60 Belgian herds were sampled at four different slaughterhouses. An average number of 30.35 animals per herd was screened both serologically and bacteriologically, yielding a total of 1821 observations. We re-analyzed these data to obtain an estimate for the conditional probabilities that an animal is MLN positive given the animal is seropositive (resp. seronegative). However, the data are correlated due to the three-stage sampling procedure used: slaughterhouses were sampled first, then herds within slaughterhouses and finally, animals within herds. Not accounting for this hierarchical structure, as in ordinary logistic regression, might lead to an underestimation of the standard errors and hence to spurious significant results. Therefore, the data are analyzed using Generalized Estimating Equations (GEE). The GEE approach requires only a correct specification of the mean structure (like in ordinary logistic regression) provided one is willing to adopt 'working' assumptions about the correlation structure [26]. An attractive feature of the GEE approach is that it yields consistent estimators for the parameters of the mean structure even if the correlation structure is misspecified. For the analysis of these data, we adopt the independence working

correlation and use the following mean structure with index i referring to animal ($i = 1, ..., n_{j\ell}$), j to herd ($j = 1, ..., n_\ell$) and ℓ to slaughterhouse ($\ell = 1, ..., 4$)

$$g\left(\pi(MLN_{ij\ell} = 1)\right) = \beta_{00}(1 - S_{ij\ell}) + \beta_{10}\ln(\bar{s}_{j\ell})(1 - S_{ij\ell}) + \beta_{01}S_{ij\ell} + \beta_{11}\log(\bar{s}_{j\ell})S_{ij\ell}, \qquad (2.1)$$

where g is the logit link, where MLN and S are binary variables indicating the lymph node bacteriological and serological status of the animal, respectively. Observe that the within-herd seroprevalence $\bar{s}_{j\ell}$ can be considered as proxy for infection pressure. In model (2.1), the natural log-transformation $\ln(\bar{s}_{j\ell})$ is used to constrain the estimated curve to go through the origin reflecting that all animals are expected to be bacteriologically negative in case the herd is free from infection. Parameter estimates and empirical standard errors are given in the left part of Table 2. The estimated curves are graphically displayed in Figure 4 a on top of a bubble plot of the herd-specific proportions of MLN-positive animals given the animals serological status. Not surprisingly, the probability of an animal being lymph node positive increases with increasing infection pressure (∼within-herd seroprevalence) whereas the 95% confidence intervals of the estimated curves for the serological positive and serological negative animals are completely overlapping. These findings support the generally accepted result that serology is a good indication for the presence of *Salmonella* at herd-level only, not at animal-level. This can also be seen in Figure 4b, representing the ratio of the odds of a MLN-positive sample given the sample is serological positive to the odds of a MLN-positive sample given the sample is serological negative as function of the within-herd seroprevalence.

[Figure 4 about here.]

In a second Belgian study, conducted by Botteldoorn et al. [25], mesenteric lymph nodes (MLN) and colon content (CC) samples are taken from 329 animals belonging to 62 different herds at five different slaughterhouses. These data are used to obtain an estimate of the conditional probabilities that an animal is CC positive given the animal is MLN positive (resp. MLN negative). As before, the data are correlated due to the three-stage sampling procedure used. Again, we use a GEE-model adopting the independence working correlation. The mean structure is now given by

$$g\left(\pi(CC_{ij\ell} = 1)\right) = \beta_{00}(1 - MLN_{ij\ell}) + \beta_{01}MLN_{ij\ell} \qquad (2.2)$$

where g is the logit link and where CC and MLN are binary variables indicating the colon content and lymph node bacteriological status of the animal, respectively. The middle part of

Table 2 contains the parameter estimates and empirical standard errors based on which it is calculated that $\hat{\pi}(CC = 1|MLN = 0) = 0.12$ (95% CI:0.056−0.17) and $\hat{\pi}(CC = 1|MLN = 1) = 0.54$ (95% CI:0.40 − 0.68). Clearly, there is a strong association between both bacteriological measurements (OR: 9.21, 95% CI: 3.58-14.84).

Based on the obtained expression for the conditional probabilities, the internal infection status (carrier + excreting) of the pigs can be simulated. From the module 'primary production' simulated values of the serological status of the pigs and the within-herd seroprevalence are obtained. These are combined as in expression (2.1) to calculate the probability that the animal is MLN positive, which is then used to simulate the MLN status of the animals using a Bernoulli distribution. The simulated MLN status is then used as in expression (2.2) to calculate the probability that the animal is CC positive. Again, the Bernoulli distribution is used to simulate the CC status of the animal. Finally, if the MLN status and/or CC status of the animal is positive, the animal is considered internally bacteriological positive and a potential contaminant of pork carcasses. In the study by Nollet et al. [24], serological and bacteriological samples were taken at slaughterhouses. By linking the conditional probabilities derived from this data with simulated values from the within-herd prevalence at primary production, it is assumed that seroconversion does not increase during transport & lairage. This seems reasonable because of the lag time for seroconversion.

[Table 5 about here.]

External contamination

It is well known that mingling with excreting animals during transport to slaughterhouse and subsequent lairage increases the external contamination [27],[28]. The association between internal infection in the intestines (excreting animals) and external contamination of the carcass at animal-level could be assessed using part of the data from a British study conducted by Davies et al. [29]. The data concern large intestine samples and carcass samples at slaughter on 80 pigs. Of the 18 positive intestinal samples and 17 positive carcass samples four samples originate from the same pig (Davies, personal communication). Based on this information, the conditional probability that an animal is externally contaminated given the animal's internal contamination status in the intestines is derived using the following logistic regression model

$$C_{EX\,i} \sim Bernoulli(\pi_i) \tag{2.3}$$

$$g(\pi_i) = \beta_{00}(1 - C_{IN\,i}) + \beta_{01}C_{IN\,i}$$

where g is the logit link and where C_{EX} and C_{IN} are binary variables indicating the external contamination and internal infection status in the intestines, respectively. The parameter estimates and standard errors of this model are given in the right part of Table 2. The corresponding odds ratio is calculated as well, clearly indicating that there is no association between the internal infection and external contamination status at animal level (OR: 1.077, 95% CI: 0.30-3.83).

The association between internal and external contamination at batch-level can not be estimated based on the data at our disposal. This is unfortunate since a positive association between the number of excreting animals in a batch and the probability that an animal is externally contaminated is expected. Therefore, in the XXX-model, it is assumed that the number of externally contaminated animals in a batch is equal to the number of excreting animals in that batch. This assumption is informally supported by the marginal internal and external contamination prevalences found in the study by Davies et al. [29]. Of course, additional data collection and a proper statistical analysis are preferably conducted to establish this relationship. Finally, to reflect the previous finding of no association between internal and external contamination at animal level, the external contamination status of all animals in batch j is obtained as a random permutation of the vector containing the colon content status of these animals.

Module 3: Slaughterhouse

[Figure 5 about here.]

In the slaughterhouse module, changes in status of the external *Salmonella* contamination of the carcasses are modeled at $\ell = 5$ different stages of the slaughter process. These 5 stages were divided into 2 groups: killing, polishing and evisceration being the 'increasing' stages while singeing and chilling being the 'decreasing' stages. Subsequently, the environmental contamination is modeled at those critical 'increasing' points and it was assumed that a carcass being contaminated before such an 'increasing' stage will also be contaminated after this stage. In contrast and for *Salmonella* 'decreasing' stages (singeing and chilling) it was assumed that a carcass not being contaminated before that stage will also not be contaminated after this stage. These assumptions come down to imposing structural zeros in the 2×2-tables on *Salmonella* prevalence for two consecutive stages, as illustrated in Figure 6. In literature [30], [31] singeing and chilling are seen as 'decreasing' steps. Whether or not the decrease during the chilling process is due to the difficult recovery of freeze- or chill-injured cells is not of importance for further

modeling in the XXX model as it results always in lower (decrease) numbers of *Salmonella* spp. on the pig carcasses.

Based on Figure 6 and using basic probability calculations, it is straightforward to derive that, for *Salmonella* decreasing stage ℓ (for which $\pi_\ell \leq \pi_{\ell-1}$),

$$\pi(S_\ell = 1|S_{\ell-1} = 0) = 0 \quad \text{and} \quad \pi(S_\ell = 1|S_{\ell-1} = 1) = \frac{\pi_\ell}{\pi_{\ell-1}}, \tag{2.4}$$

where S_ℓ denotes *Salmonella* status and π_ℓ *Salmonella* prevalence at stage ℓ. For *Salmonella* increasing stage ℓ (for which $\pi_\ell \geq \pi_{\ell-1}$), these conditional probabilities are

$$\pi(S_\ell = 1|S_{\ell-1} = 0) = \frac{\pi_\ell - \pi_{\ell-1}}{1 - \pi_{\ell-1}} \quad \text{and} \quad \pi(S_\ell = 1|S_{\ell-1} = 1) = 1. \tag{2.5}$$

However, as can be seen in Figure 6, it is assumed that cross-contamination during evisceration is only possible for animals being internally contaminated. This translates into the following modification of the left expression in (2.5)

$$\pi(S_\ell = 1|S_{\ell-1} = 0) = \frac{\pi_\ell - \pi_{\ell-1}}{1 - \pi_{\ell-1}} S_{in}, \tag{2.6}$$

where, in this case, index ℓ refers to the evisceration stage and S_{in} to the internal contamination status of the pig after lairage.

The prevalences π_ℓ, $\ell = 1, ..., 5$, which are used in the expressions above, are derived from expert opinion being elicited by Boone et al. [32] during a workshop organized at an international conference on food safety in pork production. At this workshop, experts were asked to give minimum, most likely and maximum values of the prevalence π_ℓ of pig carcasses being externally contaminated after the different stages of the slaughter process, based on which betapert distributions [33] are defined. In addition, experts were asked to answer calibration questions (*i.e.*, questions of which the answers are known by the analyst) which are used to construct weights reflecting the expert's ability [32]. Two experts performed very well and their expert opinion is incorporated in the XXX-model. Denote the betapert distribution fitted to data of expert $m = 1, 2$ with respect to stage ℓ as $bpert_{\ell m}$. These distributions are incorporated in the XXX-model by randomly selecting one expert m during each iteration of the model with the sampling weights proportional to the expert's ability. Recall that for *Salmonella* decreasing (resp. increasing) stages $\pi_\ell \leq \pi_{\ell-1}$ (resp. $\pi_\ell \geq \pi_{\ell-1}$). To ensure that these conditions are not violated for particular MC-samples, conditional random sampling is used to obtain values of the betapert distributions $bpert_{\ell m}$ with $\ell = 1, ..., 5$. In particular, for decreasing stages, random values $x^*_{\ell m}$ are generated from the betapert distributions smaller than the value generated for

the previous stage or $bpert_{\ell m}^{-x^*_{(\ell-1)m}}$. Similarly, for increasing stages, random values $x^*_{\ell m}$ are generated from the betapert distributions larger than the value generated for the previous stage or $bpert_{\ell m}^{+x^*_{(\ell-1)m}}$. Then, these random values are used in the expressions (2.4) to (2.6) to obtain the corresponding conditional probabilities. Starting from the external *Salmonella* status after lairage, the conditional probability of a *Salmonella* positive carcass after killing is calculated which is used to simulate the latter status using a Bernoulli distribution as before. This is repeated for all stages of the slaughter process, eventually resulting in simulated values for the *Salmonella* status of chilled carcasses.

[Figure 6 about here.]

Module 4: Post-processing

In this module, changes in *Salmonella* concentration (or alternatively, in numbers per unit) during post-processing are modeled making use of the Modular Process Risk Model (MPRM) methodology proposed by Nauta [10]. So far, changes in *Salmonella* concentration are not modeled due to lack of relevant data. However, from a Belgian study conducted by Delhalle et al. [34], semi-quantitative data on *Salmonella* concentration in meat cuts could be obtained. To these data, a normal distribution has been fitted [34], which is used as input distribution in the XXX-model. From this distribution, samples above the detection limit are taken in case the *Salmonella* status of the chilled carcass of which the meat cuts are taken is positive and below the detection limit otherwise, as such, making the link between prevalence and concentration.

Microbial growth

Unfortunately, no satisfactory growth model for *Salmonella* in pork exists. Therefore, in the XXX-model (as in [6] and [35]), microbial growth is modeled making use of Oscar's model describing growth of STM on poultry [36]. In particular, Oscar [36] developed response surface models to investigate the effect of temperature (10°C to 40°C) and previous growth natrium chloride (NaCl) (0.5% to 4.5%) on lag time λ (period of assimilation) and specific growth rate μ of STM on cooked chicken breast. Hereby, the minimum growth temperature is 10°C. Furthermore, in the XXX-model, it is assumed that λ equals zero since the time between initial contamination at slaughterhouse and post-processing is sufficiently large for the bacteria to be acclimated on the pork meat. For the same reason, previous growth NaCl is considered to be the same as the current NaCl level of the pork product. An expression for the hourly log growth μ ($\log_{10}$

CFU/hour) is given by

$$\mu = e^{-6.26-0.011NaCl+0.32Temp+0.002NaCl\times Temp-0.0085Temp^2-0.0045NaCl^2}, \tag{2.7}$$

where $NaCl$ is the NaCl level (%) and $Temp$ is the temperature (°C) of the meat [36]. The total log growth ($\log_{10}$ CFU/time) is then given by

$$\Delta = \mu \times Time, \tag{2.8}$$

where $Time$ is exposure time (in hours). The total number of viable counts ($\log_{10}$ CFU) after growth is given by

$$Y_{\ell+1} = Y_\ell + \Delta, \tag{2.9}$$

where Y_ℓ are the viable counts ($\log_{10}$ CFU) before growth. In order to obtain adequate data on temperature of the meat products and exposure time during the different stages of post-processing, a survey [37] has been conducted within the XXX-project. All temperatures obtained are below the minimum *Salmonella* growth temperature of 10°C as assumed in Oscar's growth model [36]. This finding is in line with the European regulation (EC) 853/2004 [38] stating that minced meat and prepared meats must comply immediately after production with the specified maximal temperature of 2°C for minced meat and 4°C for meat preparations. As such, it is assumed that microbial growth is negligible during post-processing.

Food handling processes

Two food handling processes are modeled during post-processing, being mixing and partitioning. Assuming no microbial losses, the numbers of *Salmonella* in a meat mix is simply the sum of the numbers on the composing meat cuts. In the XXX-model, this is straightforward to calculate since all pigs of which meat cuts will end up in the one meat mix are monitored simultaneously. Next, the meat mix is divided into $K = 5000$ servable portions with varying weights $W_{portion}$. Assuming no microbial losses, partitioning implies that each CFU in the meat mix is allocated to one of the K portions. This is implemented by randomly selecting N_{mix} portions from $X_\ell \sim$ multinomial$(1, ..., k, ...K; w_1, ..., w_k, ...w_K)$ with sampling probabilities w_k. Then, the number of *Salmonella* in portion k is obtained by simply counting how often a CFU is allocated to portion k or $N_{portion\,k} = \sum_{\ell=1}^{N_{mix}} I(X_\ell = k)$ with $I = 1$ if $X_\ell = k$ and $I = 0$ otherwise. The sampling probabilities w_k are chosen to reflect both the variability in portion weights and the clustering of CFU's in a meat mix. Evidently, larger portions are expected to contain a higher number of CFU's following partitioning. Furthermore, clustering implies

additional heterogeneity in sampling probabilities. In particular, the sampling probabilities are chosen as

$$w_k = \frac{W_{portion\,k} \times \pi_k}{\sum_{k=1}^{K} W_{portion\,k} \times \pi_k} \tag{2.10}$$

where $W_{portion\,k}$ denotes the weight of portion k and where π_k follows a beta distribution expressing heterogeneity in the sampling probabilities due to clustering. Observe that the sampling probabilities w_k are standardized such that their sum equals one. The beta distribution expressing heterogeneity due to clustering is of the form $\pi_k \sim \text{beta}(b, b(K-1))$, having mean $\mu = 1/K$ and variance $\sigma^2 = (k-1)/(K^2(bK-1))$ [10]. From the expression of the variance, it easily follows that $\sigma^2 \to +\infty$ if $b \to 0$, which represents extreme clustering and that $\sigma^2 \to 0$ if $b \to +\infty$, which represents the absence of clustering.

Clearly, a correct choice for b is of utmost importance. However, the only information we were able to find is expert opinion from Nauta et al. [39]. These authors assessed that clustering in a meat mix is rather profound ($\sim b = 0.15$) because a large batch of meat is not easily mixed well. In the XXX-model, it is reflected that little is known about b by taking random samples $u \sim \text{unif}]0, 1[$, which are transformed as

$$b = \exp\left(\ln\left(\frac{u}{1-u}\right) + \ln(0.15)\right) \tag{2.11}$$

to obtain values for b. From the transformation above, it follows that the minimum value for b reaches 0, the maximum value reaches $+\infty$ and that the median (and at the same time most likely value) equals 0.15.

Module 5: Distribution & Storage

In this module, *Salmonella* growth due to temperature abuse is modeled (1) at retail, (2) during transport from retail to home and (3) during storage at home.

Microbial growth

As before, Oscar's growth model [36] is used to model microbial growth. In order to have adequate data on exposure time and temperature during different stages at retail, a survey [37] has been conducted at the four largest retails in Belgium. Again, all temperatures are found to be below the minimum growth temperature of 10 °C [36], based on which it is assumed that microbial growth is negligible at retail. On the contrary, temperature of the meat product during transport from retail to home is affected by external temperature. From the Belgian Royal Meteorological Institute, the external temperatures are obtained for every day of the

base year 2006 at 10.00AM, 2.00PM and 6.00PM. These temperatures range from -4.8°C to 34.6°C with a yearly average of 13.08°C and are well described by means of a two-component Gaussian mixture, which is used as input distribution in the XXX-model. Remind that Oscar's growth model describes the amount of growth ($\log_{10}$ CFU) per time unit in relation to the salt concentration and the temperature of the product. The salt concentration is assumed to vary uniformly between 1.12% and 1.75% [40]. The temperature of the meat product is assumed to increase as transport time progresses, especially if the external temperature is high. In particular, it is assumed (as in Hill et al. [6]) that the temperature of the meat product increases linearly with transport time and that the temperature at the end of transport is function of the external temperature and product temperature at retail. To simulate *Salmonella* growth during transport, the function given in (2.8) and (2.7) describing Oscar's growth model is to be integrated out over time, for which numerical integration is used. Finally, growth during storage in the fridge at home is modeled with the information on fridge temperature and exposure time obtained from the Belgian Food Consumption Survey [17].

Module 6: Preparation & Consumption

In this module, the process of preparing a meal in households, with the meal partially consisting of a portion of minced pork meat and another food item, is simulated. Hereby, it is assumed that the meat is always cooked whereas the other food item is sometimes consumed raw. Cross-contamination from the meat to another food item via cook's hands and/or carving board as well as microbial inactivation due to cooking are modeled.

Cross-contamination

Cross-contamination to another food item is assumed to happen either via the cook's hands or via the carving board used for manipulating the minced meat. Both routes of cross-contamination are modeled analogously to the WHO model of *Salmonella* in broiler chickens [35], adapted with concepts described by Mylius et al. [41]. The inputs of this module are (a) food handling behavior and (b) transfer probabilities of *Salmonella* CFU's between the meat portion, the cook's hands and board. Food handling behavior of Belgian food preparers was obtained through a Food Consumption Survey held in Belgium in 2004 [17]. Analogously to Mylius et al. [41], it was assumed that other food is handled after meat handling in 50% of the preparations and analogously to Hill et al. [6], it was assumed that a board was used in only 10% of the minced meat preparations. For describing the transfer probabilities involving the cook's hands, models

developed for *Enterobacter aerogenes*, having attachment characteristics similar to *Salmonella*, on chicken meat [42] were applied. For describing transfer probabilities involving a carving board, models developed for *Salmonella* [43] could be used.

The route of cross-contamination via cook's hands is considered first with the resulting number of *Salmonella* on another food item being modeled as

$$N_{X-hand\,k} = N_{stor\,k} \times T_{m,h\,k} \times P_{hand\,k} \times T_{h,o\,k} \times S_{other\,k} \tag{2.12}$$

where the letter T refers to proportions transferred with the first (resp. second) letter in the subscript stating the transfer from (resp. to) an object (m = minced meat portion, h = cook's hands and o = other food item assumed consumed raw), where P_{hand} refers to the proportion of cells that persists on the hands, possibly even after hand washing and where S_{other} indicates whether or not other food is handled after food manipulation. Here it is assumed that transfer of *Salmonella* CFU's in minced meat is the same as on the surface of chicken meat (fillet). This might result in an overestimation of the risk due to cross-contamination as transfer is most likely to happen from the 'surface' of the minced meat. The number of *Salmonella* remaining on the minced pork meat after cross-contamination via hands is simply given by $N_{meat1\,k} = (1 - T_{m,h\,k}) \times N_{stor\,k}$. Secondly, the route of cross-contamination via the carving board is considered with the number of *Salmonella* transferred from the meat after manipulation by hands to the board given by $N_{board1\,k} = N_{meat1\,k} \times T_{m,b\,k}$. Next, the numbers remaining on this board after board manipulation (involving (a) the use of another board or (b) the use of the same board being washed after meat handling or (c) the use of the same board not being washed after meat handling) is given by $N_{board2\,k}$. Then, the number of *Salmonella* transferred from the board to another food item is modeled as

$$N_{X-board\,k} = N_{board2\,k} \times T_{b,o\,k} \times S_{other\,k} \tag{2.13}$$

The total number of *Salmonella* on another food item due to cross-contamination via both routes is simply given by $N_{X\,k} = (N_{X-hand\,k} + N_{X-board\,k}) \times S_{raw\,k}$, where $S_{raw\,k}$ indicates whether the food item is consumed raw. Finally, the number of *Salmonella* that remains on the minced meat portion after food handling is given by $N_{meat1\,k} \times (1 - T_{m,b\,k})$.

Microbial Inactivation

In case of adequate cooking, all microbial organisms die whereas in case of undercooking, some organisms might survive. Unfortunately, assessing the survival of micro-organisms when undercooking takes place is difficult and can only be based on educated guesses as data in this area

are missing. In the XXX-model, the effect of (under)cooking the minced pork meat is modeled based on a log-linear death kinetic model as in [35] and [6]. The assumptions made by Hill et al. [6] are used in the XXX-model as well.

In case of undercooking, only a proportion of the cells in the protected area will survive the cooking process. Denote the temperature to which the protected *Salmonella* cells are exposed as $Temp_{cook\,k}$, the exposure time (in minutes) as $Time_{cook\,k}$ and the total number of cells in the protected area as $N_{protect\,k}$. Then, the log reduction of protected *Salmonella* cells is calculated as

$$\Delta_{protect\,k} = Time_{cook\,k}/D_k, \tag{2.14}$$

where $D_k = 10^{-0.14 \times Temp_{cook\,k} + 8.58}$ [35] is the D-value or decimal reduction time, being the time required at a certain temperature to kill 90% of the bacteria. Then, the number of *Salmonella* on the minced pork meat after cooking equals

$$N_{cook\,k} = \begin{cases} 10^{log_{10}(N_{protect\,k} - \Delta_{protect\,k})} & \text{in case of undercooking} \\ 0 & \text{otherwise.} \end{cases} \tag{2.15}$$

Finally, the total number of *Salmonella* ingested when consuming a meal equals the sum of the number of *Salmonella* remaining on the minced pork meat after cooking and the number transferred to another food item that is consumed raw or,

$$N_{dose\,k} = N_{X\,k} + N_{cook\,k}. \tag{2.16}$$

2.3 Hazard Characterization

Adverse Health Effects

Non-typhoidal *Salmonella* infections are commonly manifested by acute enterocolitis, with sudden onset of diarrhea which can be bloody, abdominal pain, headache, nausea, fever and sometimes vomiting [44]. In most cases, the diarrhea is self-limiting but can evolve to bacteriemia (1.4% of STM cases reported in Belgium in 2006 [12]) or focal infections such as meningitis, septic arthritis, pneumonia, ..., especially in the most susceptible population, being defined as the YOPI (young, old, pregnant, immuno-compromised)-group [45].

In invasive life-threatening infections, the use of antimicrobial drugs is required. Treatment of *Salmonella* bacteremia is generally undertaken with a single bactericidal drug. The resistance

of *Salmonella* to both fluoroquinolones and third-generation cephalosporins have been reported, and these resistance might result in therapeutic problems in the future [45].

Dose-response

A crucial aspect of quantifying microbial risk is the assessment of the dose-response relationship, which is the relationship between the amount of microbial organisms ingested and a specific outcome, like infection, illness or even mortality. Recently, Bollaerts et al. [46] fitted dose-illness models as proposed by Teunis et al. [47] to outbreak data of human salmonellosis [35] using Generalized Linear Mixed Models [26] and modified fractional polynomials [48] of dose. Furthermore, heterogeneity due to differences in host susceptibility (enhanced susceptible versus normal population), serovar type and food-matrix is taken into account and data uncertainty is modeled by means of a two-stage bootstrap procedure. For each of the fifteen unique combinations of serovar type and food-matrix as observed in the outbreak studies summarized by the WHO [35], the estimated dose-illness relationship for the normal and the susceptible population is obtained (unfortunately, no outbreaks with minced pork meat). Exemplary, graphical representations of the dose-illness curves for *S.* Typhimurium are given in Figure 7. Because no dose-illness curve for minced pork meat is available, all different dose-illness curves given in Bollaerts et al. [46] are used in the XXX-model, as such acknowledging the epistemiological uncertainty. In particular, for each simulated dose, one of the fifteen dose-illness curves is randomly selected and the probability of illness for a normal person (resp. susceptible person) is calculated.

[Figure 7 about here.]

2.4 Risk Characterization and Baseline Results

Annual Cases

In 2006, the total population size in Belgium was $N = 10\,511\,382$, of which 24.0% is considered enhanced susceptible. The latter fraction is estimated as the percentage Belgian inhabitants belonging to the YOPI group: young (0-5 year, 5.52%), old (> 65 year, 17.21%), pregnant (0.86%) and immuno-compromised (0.43%) [49]. From the Belgian Food Consumption Survey [17], it is obtained that the number of servings of a fresh minced (pure and mixed) pork meat per person year is normally distributed as $N(115.28, 13.70)$. All this information is combined

to estimate the yearly number of human salmonellosis cases in Belgium within the enhanced susceptible population as

$$N_{sus\ cases} = N \times 24\% \times n_{servings} \times \overline{\pi}_{sus\ risk}, \tag{2.17}$$

and within the normal population as

$$N_{nl\ cases} = N \times 76\% \times n_{servings} \times \overline{\pi}_{nl\ risk}, \tag{2.18}$$

with $\overline{\pi}_{sus\ risk}$ (resp. $\overline{\pi}_{nl\ risk}$) being the average risk of illness following consumption of *Salmonella* contaminated minced pork meat for the enhanced susceptible population (resp. normal population) as estimated by the XXX-model. The average risk is calculated based on $R = 1000$ iterations of the model (with each iteration producing $K = 5000$ risk estimates). This is repeated $B = 500$ times in order to obtain a distribution of the estimated number of the annual cases within the enhanced susceptible and the normal population. The results are summarized in Table 3 and a graphical representation of the simulated distributions of the risk and annual cases for the normal and enhanced susceptible population is given in Figure 8. As expected, the risk is higher for the enhanced susceptible population (est. 4.713×10^{-5}; 90%CI: 2.750×10^{-5}-7.563×10^{-5}) compared to that of the normal population (est. 7.704×10^{-6}; 90%CI: 2.251×10^{-6}-1.822×10^{-5}). The same holds for the predicted number of annual cases within the enhanced susceptible [est. 13517; 90% CI: 7887-21691] and normal population [est. 6996; 90% CI: 2045-16555], however the difference between both groups is smaller because the normal population is larger in size than the enhanced susceptible population. The total number of annual cases attributed to fresh minced pork meat is estimated as 20513 and the corresponding 90% percentile interval as (90%CI:9932−38246).

[Table 6 about here.]

[Figure 8 about here.]

Exposure assessment

Baseline predictions of the *Salmonella* prevalence and concentration per gram ($\log_{10}$CFU/g) or alternatively, numbers per unit ($\log_{10}$CFU) at various stages of the food chain are calculated based on $R = 1000$ iterations of the XXX-model. The results are summarized in Table 4. In particular, prevalence and, for post-processing onwards, summary statistics of the concentration/numbers (mean, variance, 5% and 95% quantile) are given. The concentration/numbers

statistics are calculated based on the contaminated units only. Each time, the number of units based on which these statistics are calculated is given as well.

Baseline results show that 31.3% of the pigs at primary production are predicted to be seropositive for *Salmonella* on an ELISA-test using a threshold value of $\zeta = 0.25$. After transport and lairage, 35.3% of the pigs are internally infected in the lymph nodes or the intestines and 20.6% of the pigs are externally contaminated. At the end of the slaughter process, the model predicts 4.27% of the chilled carcasses being contaminated with *Salmonella*. As reflected in the results for post-processing, a dilution of *Salmonella* cells takes place when mixing meat cuts, implying a higher prevalence but lower concentration for minced meat [25.1%;-0.583 $\log_{10}$CFU/g] compared to meat cuts [4.27%;-0.346 $\log_{10}$CFU/g]. The results for distribution & storage suggest that growth, although limited, primarily happens during storage at home. Finally, the results for preparation & consumption indicate that the final risk for consumers is mainly due to undercooking [0.0122% ;1.082 $\log_{10}$CFU/unit] and for a smaller extent to cross contamination in the kitchen [0.00043%; 1.041 $\log_{10}$CFU/unit], mostly via cook's hands.

[Table 7 about here.]

2.5 Validation

The XXX-model is validated using external information, showing that the model produces realistic results. In order to validate the predicted number of annual cases, public health surveillance statistics reported by the Belgian national reference center for *Salmonella* and *Shigella* (NRRS) are used. In particular, the NRRS reported 3693 cases of human salmonellosis in Belgium in 2006, of which 1826 were serotyped STM [12]. However, this number represents only a portion of the true number of illness cases due to underreporting. The amount of underreporting varies strongly between countries because of differences in national surveillance systems. In England, it is estimated that the true number of salmonellosis cases is 3.8 times higher than the number of reported cases whereas this is estimated to be 13.4 in the Netherlands and as high as 38.6 in the United States [50]. In addition, human salmonellosis is attributable to different food sources (*e.g.* pork, beef, broilers, eggs). The proportion of human salmonellosis attributable to pork was estimated to be between 9% and 15% in Denmark [51] and around 21% in the Netherlands [52]. However, it is questionable whether the latter results carry over geographically and historically, especially because in Belgium a strong decline in SE has been observed since 2004, most likely due to the vaccination of laying hens, rendering pork a relatively more

important source of salmonellosis. To acknowledge this epistemological uncertainty, the annual number of human salmonellosis cases is approximated in two different ways. First, the total number of reported salmonellosis cases is multiplied with a factor to correct for underreporting and one to correct for source attribution. Using the available correction factors given above, the estimated number of annual cases attributable to pork ranges from 1263 ($= 3693 \times 3.8 \times 0.09$) to 29935 ($= 3693 \times 38.6 \times 0.21$). Second, since STM is the predominant serovar isolated from pork, the number of reported STM salmonellosis cases is multiplied with a factor that corrects for underreporting only, yielding a range from 6938.8 ($= 1826 \times 3.8$) to 70484 ($= 1826 \times 38.6$). The rationale behind the latter approach is that the resulting errors (*i.e.* the underestimation since other serovars are isolated from pork as well and the overestimation since STM is isolated from other food sources as well) cancel out. The obvious advantage is that a factor correcting for source attribution is not needed. Taking both ranges together, the number of annual human salmonellosis cases attributable to pork is estimated to vary between 1263 and 70484. Finally, since minced pork meat implies a much higher risk for salmonellosis (*e.g.* [53]) and is twice as much consumed than pork flesh [17], at least two third of the pork attributed cases are due to consumption of minced pork products (this lower bound follows from the assumption that minced pork meat and pork flesh are equally risky). As such, it can be concluded that the total number of annual cases attributed to minced pork meat as estimated by the XXX-model (90% CI:[9932-38246]) is well within the range of the number of annual cases attributed to pork meat as estimated based on the health surveillance statistics ([1263-70484]).

Some of the exposure assessment results could be validated as well. In the XXX-model, the percentage pigs contaminated in the intestines at slaughter is estimated as 20.6%. In an American study [54] and a Belgian study conducted in 2000 [55], this number is found to be 21.2% (s.e.:2.5%) and 45% (s.e.:11%), respectively. In that same Belgian study [55], the percentage of pigs contaminated in the lymph nodes is found to be 40% (s.e.:10%), which is, taking into account the statistical uncertainty, not contradicting the XXX-model prediction of 21%, especially since important *Salmonella* reducing efforts are taken in the primary production between 2000 and 2008, which are likely to have reduced the prevalence of carrier pigs. For chilled carcasses at the end of slaughter, a prevalence of 7.1% (s.e.: 2.07%) is obtained by the Belgian official monitoring of the Federal Food Agency in 2006 [56] and of 18.1% (s.e.: 2.04%) by the European Food Safety Authority survey conducted in 2007 [57], whereas the XXX produces an estimate of 4.27%. Finally, the prevalence of contaminated minced meat portions could be validated using a Belgian study conducted in 2001 [58] and data from the Belgian Federal

Food Agency of 2006 [56]. The first study obtained a prevalence of 13% (s.e.:4.5%) whereas the federal food agency obtained a prevalence of 2.6% (s.e.:1.75%). The XXX-model produces a prevalence estimate of contaminated minced meat portions of 12.2%, which is in line with results above. Hence, based on the data available for validation, the XXX-model could not be falsified. Taking into consideration the historical and geographical differences, the variability in results between different empirical data sets as well as the statistical uncertainty, it can be concluded that XXX-model produces very realistic results.

3 Discussion

In this manuscript, a QMRA is described to assess the risk of human salmonellosis through consumption of fresh minced pork meat in Belgium. A new risk assessment model, briefly called the XXX-model, is developed. The model is implemented in Matlab, a programming language allowing fast computation using vectorization and flexible programming, which are not feasible in a spreadsheat based programming environment. The XXX-model is a modular 'farm to fork' risk model closely resembling the Belgian situation. In comparison to existing models, several statistical refinements and improved modeling techniques are proposed. In brief, at primary production, the within-herd seroprevalence is used to account for the huge differences in *Salmonella* infection levels between Belgian farms. During transport & lairage, transition probabilities are calculated to obtain both the internal and external contamination status of the pigs at lairage. The effect of different steps of the slaughter process are modeled using transition probabilities as well. At the beginning of post-processing, the contamination status of meat cuts is linked with data on *Salmonella* concentrations. At post-processing, the process of mixing is easily (and correctly) modeled since all pigs ending up in the same meat mix are monitored simultaneously. Subsequently, the process of partitioning is modeled using fast computation without looping accounting for both varying portion sizes and CFU clustering. At distribution & storage, *Salmonella* growth is modeled taking into account changing temperatures during transport from retail to consumer's home using numerical integration. Finally, at preparation & consumption, cross-contamination via cook's hands and carving board is modeled as well as the effect of (under)cooking.

The results of the XXX-model are validated, based on which it is concluded that the model produces very realistic results. This makes the XXX-model a powerful tool to assess the effectiveness of mitigation strategies or, ultimately, use the model as part of a cost-benefit analysis.

However, the model has limitations. Although the model is already complicated, it remains a gross simplification of reality, possibly neglecting important unknown aspects of the pork food chain. However, this feature is inherent to model-based risk assessments, for which it is hard or even impossible to quantify the tradeoff between model complexity and accuracy. Furthermore, the validity of a risk model relies on the quality of the data used and of the assumptions taken. Therefore, the data used and the assumptions taken in the XXX model are evaluated with respect to their quality and impact on the risk model results, as is extensively discussed in [32] and in [59], respectively. In addition, for some input variables, empirical data were non-existent and expert opinion or assumptions published elsewhere are used instead. This is not an intrinsically bad practice as long as the uncertainty associated with their use is acknowledged and incorporated into the model. Otherwise, GIGO models (garbage in-garbage out) are readily obtained producing precise outputs by arbitrarily restricting the input space [60]. Over and above acknowledging uncertainty, a proper sensitivity analysis is considered indispensable for model-based risk assessment. Therefore, the XXX-model is currently subject to an elaborated quantitative sensitivity analysis.

comments on earlier versions of the risk model and manuscript.

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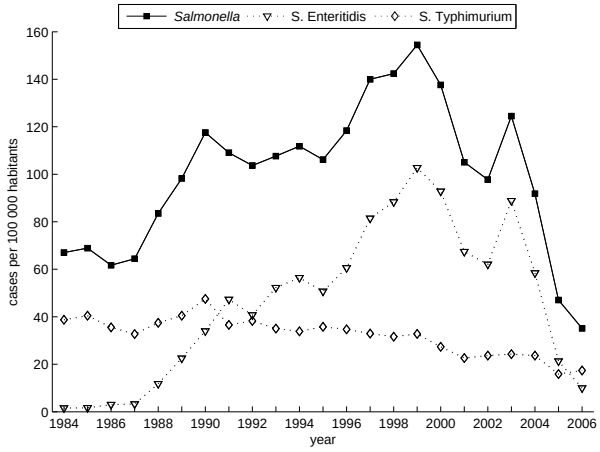


Figure 1: *Salmonella* reported cases in Belgium per 100,000 inhabitants and the distribution of the serotypes *S.* Enteritidis and *S.* Typhimurium from 1984 to 2006 [12].

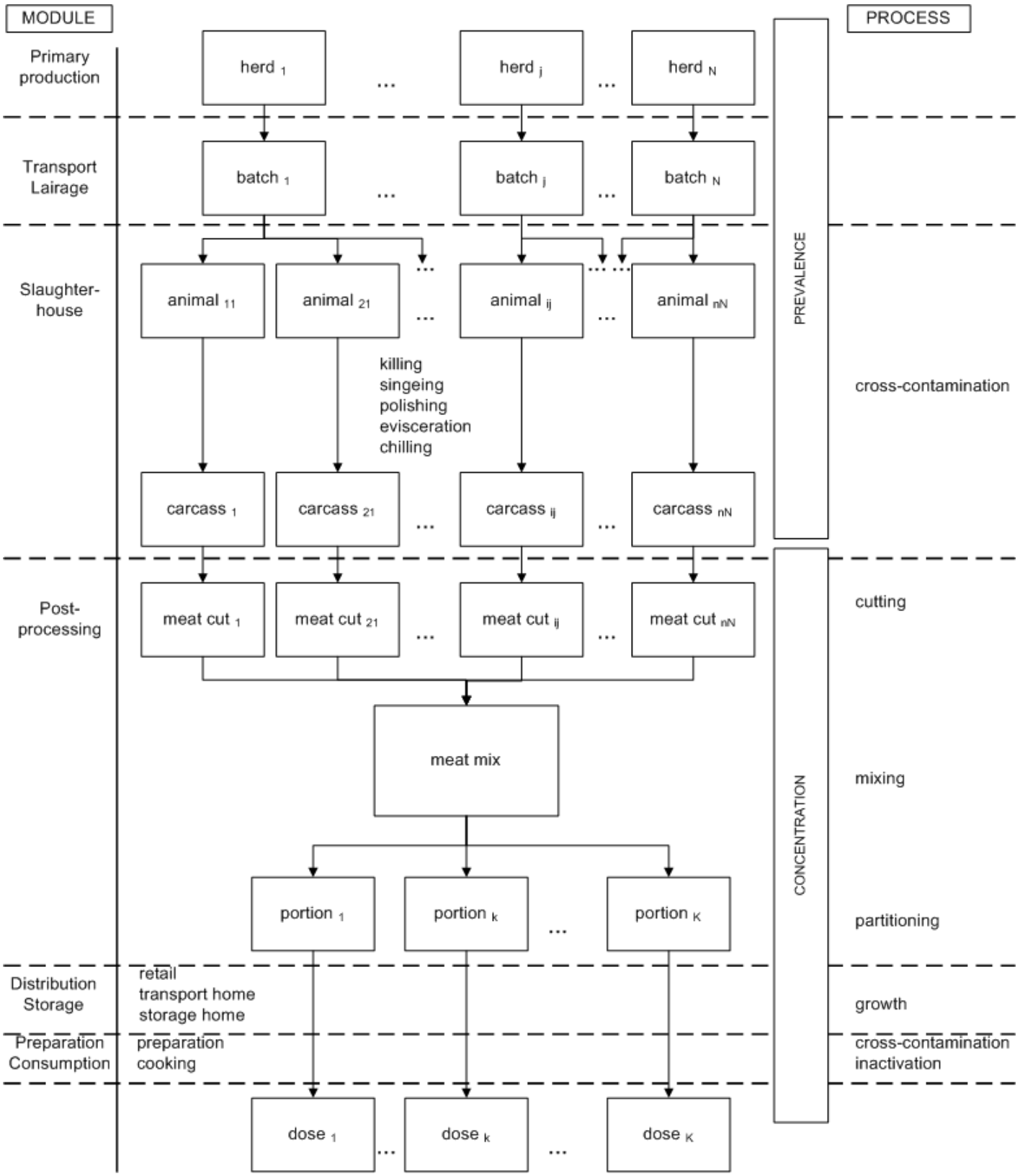


Figure 2: Schematic representation of the XXX-model.

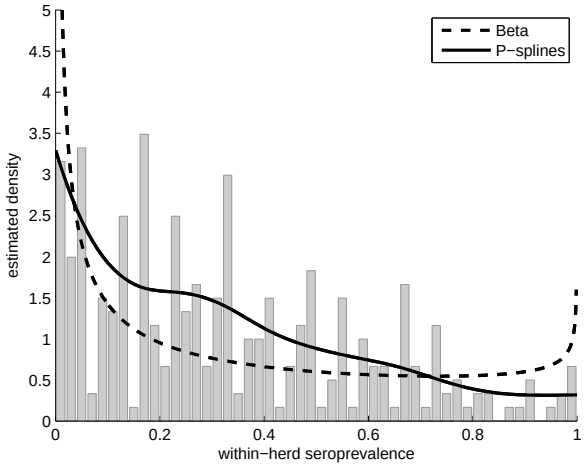
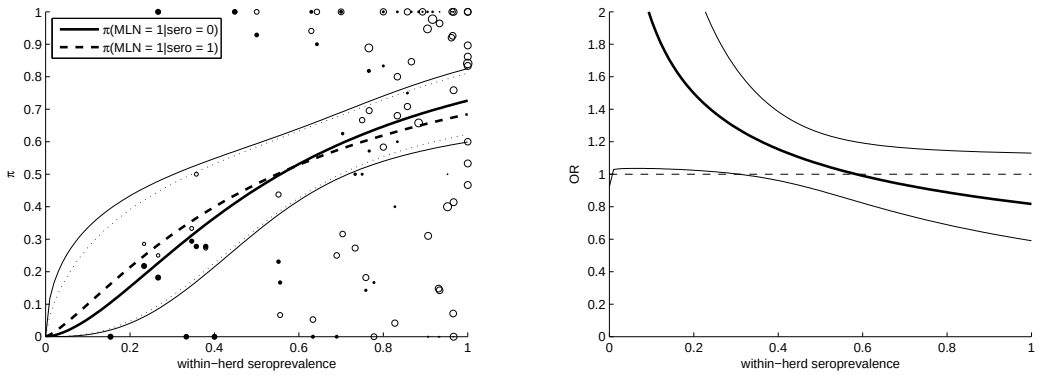


Figure 3: Parametric and non-parametric density estimate of the within-herd seroprevalence in pigs at primary production in 2006.



(a) probability (b) odds ratio

Figure 4: (a) Bubble plot of herd-specific proportions (black colored bubbles refer to serologically positive animals, white colored bubbles to serologically negative animals) with the area of the bubbles reflecting the number of observations based on which the proportions are calculated and estimated probability of an animal being mesenteric lymph node positive as a function of within-herd seroprevalence and serological status of the animal + 95% confidence intervals and (b) corresponding odds ratio + 95% confidence intervals

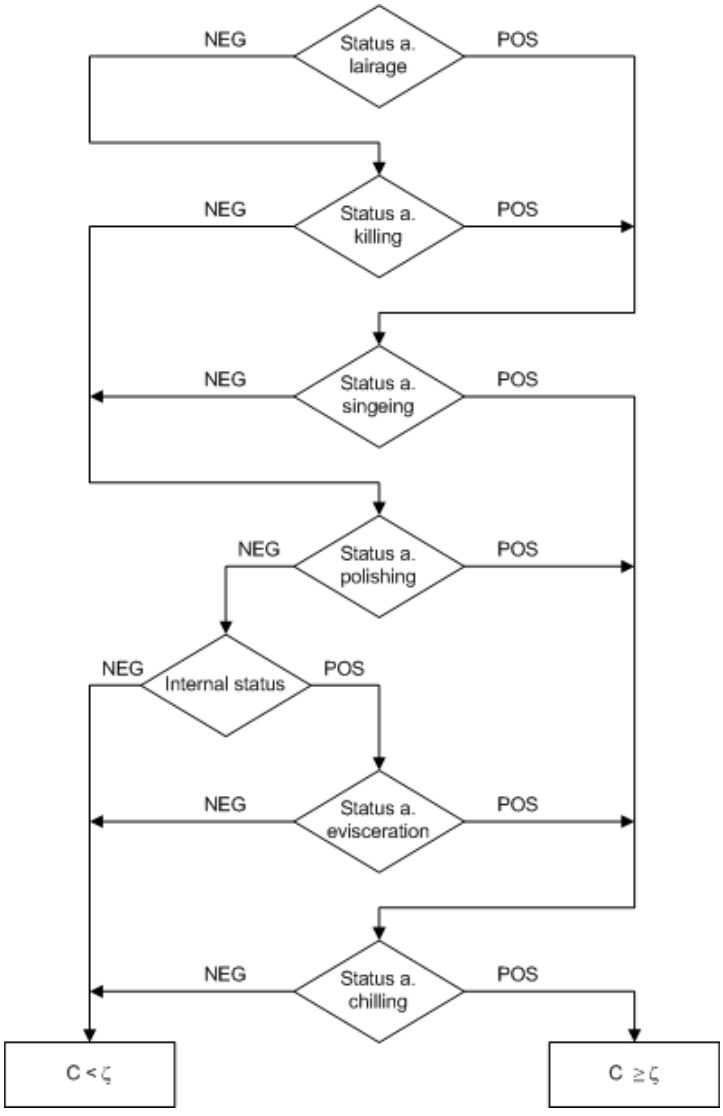
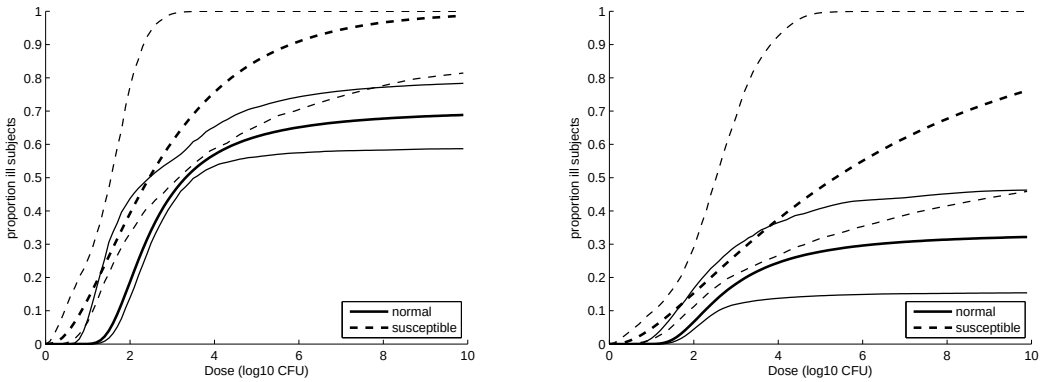


Figure 5: Schematic representation of the XXX module 'slaughterhouse'.

		stage ℓ		
		0	1	
stage $\ell-1$	0	$1-\pi_{\ell-1}$	**0**	$1-\pi_{\ell-1}$
	1	$\pi_{\ell-1}-\pi_{\ell}$	π_{ℓ}	$\pi_{\ell-1}$
		$1-\pi_{\ell}$	π_{ℓ}	

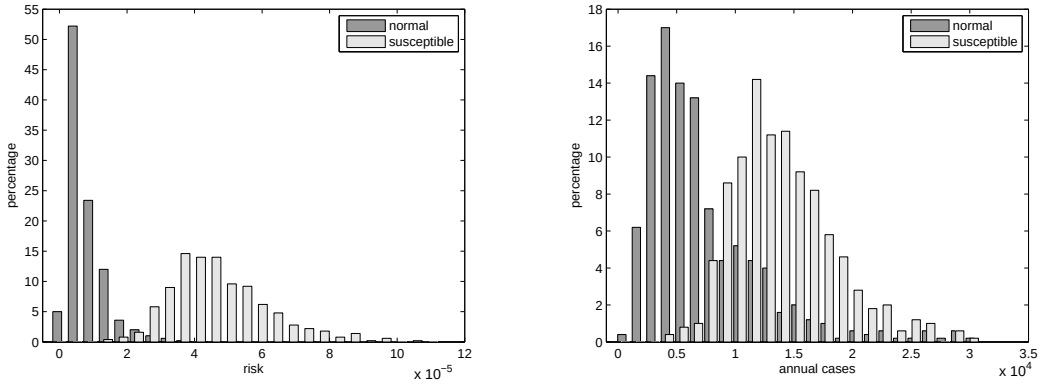
		stage ℓ		
		0	1	
stage $\ell-1$	0	1-π_{ℓ}	$\pi_{\ell}-\pi_{\ell-1}$	$1-\pi_{\ell-1}$
	1	**0**	$\pi_{\ell-1}$	$\pi_{\ell-1}$
		$1-\pi_{\ell}$	π_{ℓ}	

Figure 6: 2×2-tables on *Salmonella* prevalence used to derive conditional probabilities for *Salmonella* decreasing stages (left) and increasing stages (right) of the slaughter process.



(a) ice cream (b) water

Figure 7: Dose-illness curves for *S.* Typhimurium in two different food-matrices + 90% confidence intervals.



(a) predicted risk

(b) predicted number of annual cases

Figure 8: Baseline results of the XXX-model: normalized histogram of the predicted risk for salmonellosis due to consumption of fresh minced pork meat for the normal and enhanced susceptible population and of the predicted number of annual cases.

List of Tables

Table 1: Detailed summary of the XXX-model, the distributions of the input parameters and main sources.

composition meat mix	$W_{portion\,k}{}^*$	Weight of a portion minced pork meat (g)	$\sim$N$(93, 14.83^2)$	Devriese et al. [17]
	W_{mix}	Weight of a meat mix (kg)	$=\frac{1}{1000}\sum_{k=1}^{5000} W_{portion\,k}$	
	S_{pure}	Meat mix contains pork only (no=0, yes=1)	$\sim$bern(π) with π^* $\sim$beta$(101, 889)$	Devriese et al. [17]
	k_1	Portion pork meat in meat mix	$= S_{pure} \times 1 + (1 - S_{pure}) \times k$ with k^* $\sim$unif(0.16,0.9)	expert opinion
	$W_{mix\,pork}$	Weight pork meat in meat mix	$= k_1 \times W_{mix}$	
	$N_{batch\,j}{}^*$	Number of animals in batch at slaughterhouse	$\sim$bpert$(10, 100, 220)$	expert opinion
	n_j	Maximal number of animals in batch of which meat cuts will end up in meat mix	$\sim$discrete$\{1, 2, ...N_{batch\,j}\}$	maximal variability
	$W_{carcass\,ij}{}^*$	Weight of pig carcasses (kg)	$\sim$N$(93.79, 12.21^2)$	Belgian FPS Economy [61]
	$k_{2\,ij}{}^*$	Portion of carcass ending up in meat mix	$\sim$bpert$(0.15, 0.20, 0.30)$	expert opinion
	$W_{cut\,ij}$	Weight of meat cuts (kg)	$= k_{2\,ij} \times W_{carcass\,ij}$	
primary production	$P_{sero\,j}{}^*$	Within-herd seroprevalence	$\sim$ weighted P-splines density	FASFC-data [18]
	$N_{pos\,j}$	Number of seropositive animals in batch	$=$round$(N_{batch\,j} \times P_{sero\,j})$	
	$S_{sero\,ij}$	Serological status of pig	$\sim$hypergeo$(N_{batch\,j}, n_j, N_{pos\,j})$	
transport + lairage	$S_{MLN\,ij}$	MLN (mesenteric lymph node) status of pig after lairage	$\sim$bern(π_{ij}) with $g(\pi_{ij}) = f(P_{sero\,ij}, S_{sero\,ij})$	expression (2.1)
	$S_{CC\,ij}$	CC (colon content) status of pig after lairage	$\sim$bern(π_{ij}) with $g(\pi_{ij}) = f(S_{MLN\,ij})$	expression (2.2)
	$S_{in\,ij}$	Status of pig after lairage (internal contamination)	$= 1 - (1 - S_{MLN\,ij})(1 - S_{CC\,ij})$	
	$S_{ex\,ij}$	Status of pig after lairage (external contamination)	$=S'_{CC\,j}[i]$ with $S'_{CC\,j}$ a permutation on $\{S_{CC\,1j}, ..., S_{CC\,ij}, ...S_{CC\,n_jj}\}$	
slaughter-house	$S_{kill\,ij}$	Status of carcass after killing (external contamination)	$\sim$bern(π_{ij}) with $\pi = f(S_{ex\,ij}, expert^{*\dagger})$	expression (2.5)
	$S_{singe\,ij}$	Status of carcass after singeing (external contamination)	$\sim$bern(π_{ij}) with $\pi = f(S_{kill\,ij}, expert^{*\dagger})$	expression (2.4)
	$S_{polish\,ij}$	Status of carcass after polishing (external contamination)	$\sim$bern(π_{ij}) with $\pi = f(S_{singe\,ij}, expert^{*\dagger})$	expression (2.5)
	$S_{evis\,ij}$	Status of carcass after evisceration (external contamination)	$\sim$bern(π_{ij}) with $\pi = f(S_{polish\,ij}, S_{in\,ij}, expert^{*\dagger})$	expressions (2.5), (2.6)
	$S_{chill\,ij}$	Status of carcass after chilling (external contamination)	$\sim$bern(π_{ij}) with $\pi = f(S_{evis\,ij}, expert^{*\dagger})$	expression (2.4)

* input variables, † probabilities derived from expert opinion, see Boone et al. [62]

Table 1: Detailed summary of the XXX-model, the distributions of the input parameters and main sources (continued 1).

post-processing	$C^*_{<\lambda}$	Concentration ($\log_{10}$ CFU/g) on meat cut below detection limit	$\sim$N$(-2.64, 1.76^2)\|(, -1.04)^\diamond$	Delhalle et al. [34]
	$C^*_{\geq\lambda}$	Concentration ($\log_{10}$ CFU/g) on meat cut above detection limit	$\sim$N$(-2.64, 1.76^2)\|(-1.04,)^\diamond$	Delhalle et al. [34]
	$C_{cut\,ij}$	Concentration ($\log_{10}$ CFU/g) on meat cut	$=(1 - S_{chill\,ij}) \times C_{<\lambda} + S_{chill\,ij} \times C_{\geq\lambda}$	
	$N_{cut\,ij}$	Numbers on meat cut (CFU)	$=W_{cut\,ij} \times 10^{C_{cut\,ij}}$	
	N_{mix}	Numbers in meat mix (CFU)	$=\sum_{j=1}^{N} \sum_{i=1}^{n_j} N_{cut\,ij}$	
	X_ℓ	random allocation of CFU's, $\ell = 1, ...N_{mix}$, to portions	$\sim$discrete$\{1, ...K; w_1, ...w_K\}$ with $w_k = \frac{W_{portion\,k} \times \pi_k}{\sum_{k=1}^{K} W_{portion\,k} \times \pi_k}$ with $\pi_k \sim$beta$(b^*, b^*(K-1))$ with $b^* = \exp\left(\ln\left(\frac{u}{1-u}\right) + \ln(0.15)\right)$ with $u \sim$discrete$]0, 1[$	expression (2.11)
	$N_{portion\,k}$	Numbers in portion (CFU)	$= \sum_{\ell=1}^{N_{mix}} I(X_\ell = k)$ with $I = 1$ if $X_\ell = k$ and $I = 0$ otherwise	
distribution + storage	$Temp_{meat\,k}{}^*$	Temperature (°C) of portion at retail	$\sim$N$(3.14, 7.78)\|(-2, 15)^\diamond$	Consumer's magazine [63]
	$Temp_{ex}{}^*$	External temperature (°C)	$\sim f = \pi f_1 + (1-\pi) f_2$ with $\pi = 0.64, f_1 \sim N(6.7, 17.9)$ and $f_2 \sim N(20.1, 33.0)$	RMI
	$\Delta_{max\,k}$	Maximal possible change in temperature (°C)	$=Temp_{ex} - Temp_{meat\,k}$	assumption Hill et al. [6]
	S_k	Maximal change larger than 0 (no=0, yes=1)	$= I_{(\Delta_{max\,k} > 0)}$	
	Δ_k	Change in temperature (°C)	$\sim$N$(3.72, 2.82)\|(0, \Delta_{max\,k})^\diamond \times S_k$	Hill et al. [6]
	$Temp_{end\,k}$	Temperature (°C) of portion at end of transport	$= Temp_{meat\,k} + \Delta_k$	
	$Time_{tr\,k}{}^*$	Transport time (in 15 minutes)	$\sim$discrete$(v; w)$ with $v = [1, 2, 3, 4, 5, 6, 7, 8, 16]$ $w = [0.005, 0.05, 0.18, 0.25, ...$ $0.22, 0.16, 0.07, 0.03, 0.035]$	Hill et al. [6]
	$NaCl^*$	Salt concentration minced meat (%)	$\sim$unif$(1.12, 1.75)$	Consumer's magazine [40]

* input variables, $^\diamond$ truncated distribution with lower bound min (resp. upper bound max) in $|(min, max)$

Table 1: Detailed summary of the XXX-model, the distributions of the input parameters and main sources (continued 2).

	$Temp_{k\,\ell}$	Temperature (°C) of portion after transport time ℓ, $\ell \in [0, Time_{tr\,k}]$	$= Temp_{meat\,k} + \frac{Time_{k\,l}}{Time_{tr\,k}}(Temp_{end\,k} - Temp_{meat\,k})$	assumption Hill et al. [6], linear increase
	$\Delta_{tr\,k}$	Total log growth during transport integrated out over transport time	$=\int \Delta_{(\mu_{k\,\ell}, Time_{k\,\ell})} dTime_{k\,\ell}$ with $\mu_{k\,\ell} = f(NaCl, Temp_{k\,\ell})$	Oscar [36], expressions (2.7),(2.8)
	$N_{trans\,k}$	Numbers on portion (CFU) after transport to home	$=10^{(log_{10} N_{portion\,k} + \Delta_{tr\,k})}$	
	$Temp_{st\,k}{}^*$	Temperature (°C) of portion during storage at home	$\sim$N$(7, 2.97^2)$	Devriese et al. [17]
	$Time_{st\,k}{}^*$	Time (hours) of storage at home	$\sim$bpert$(0, 2, 5)$	Devriese et al. [17]
	$\Delta_{st\,k}$	Total log growth during storage	$=\mu_k \times Time_{st\,k}$ with $\mu_k = f(NaCl, Temp_{st\,k})$	Oscar [36], expressions (2.7),(2.8)
	$N_{stor\,k}$	Numbers on portion (CFU) after storage at home	$=10^{(log_{10} N_{trans\,k} + \Delta_{st\,k})}$	
preparation+ consumption	$S_{other\,k}$	Handling meat before other food (no=0, yes=1)	$\sim$bern(π_K) with $\pi_k^* \sim$ unif$[0.5 - 0.1, 0.5 + 0.1]$	assumption Mylius et al. [41] + uncertainty
	$T_{m,h\,k}{}^*$	Proportion transferred from meat to hand	$\sim$beta$(1.78, 41.10)$	Montville et al. [42]
	$\pi_{h\,k}{}^*$	Probability that hands are not washed after manipulating meat	$\sim$beta$(2027, 2588)$	Devriese et al. [17]
	$P_{hand\,k}$	Proportion persisting on hands after (not) washing	$\sim$discrete$(1, \kappa^*; \pi_{h\,k}, 1 - \pi_{h\,k})$ with $\kappa^* \sim beta(0.24, 6.67)$	Chen et al. [64]
	$T_{h,o\,k}{}^*$	Proportion transferred from hand to other food	$\sim$beta$(0.6, 2.3)$	Montville et al. [42]
	$N_{X-hand\,k}$	Numbers on other food due to cross-contamination via hands	$=N_{stor\,k} \times T_{m,h\,k} \times P_{hand\,k} \times T_{h,o\,k} \times S_{other\,k}$	expression (2.13)
	$N_{meat1\,k}$	Numbers remaining on portion after cross-contamination via hands	$=(1 - T_{m,h\,k}) \times N_{stor\,k}$	
	$S_{b\,k}$	Using carving board (no=0, yes=1)	$\sim$bern(π_k) with $\pi_k^* \sim$ unif$[0.1 - 0.05, 0.1 + 0.05]$	assumption Hill et al. [6] + uncertainty
	$T_{m,b\,k}$	Proportion transferred from meat to board	$\frac{1}{100} 10^{\kappa}$ with $\kappa^* \sim$N$(0.171, 0.16^2)$	Kusumaningrum et al. [43]
	$N_{board1\,k}$	Numbers on board after meat manipulation	$=N_{meat1\,k} \times T_{m,b\,k}$	

* input variables

Table 1: Detailed summary of the XXX-model, the distributions of the input parameters and main sources (continued 3).

$\pi_{b1\,k}{}^*$	Probability that other board is used	$\sim$beta$(2820, 159)$	Devriese et al. [17]
$\pi_{b2\,k}{}^*$	Probability that same board is used and washed	$\sim$beta$(2913, 66)$	Devriese et al. [17]
$\pi_{b3\,k}$	Probability that same board is used and not washed	$=1-\pi_{b0\,k}-\pi_{b1\,k}$	Devriese et al. [17]
$N_{board2\,k}$	Numbers remaining on board after board manipulation: (1) other board, (2) same board washed, (3) same board not washed	$\sim$discrete$(0, \kappa_k, N_{board1\,k}; \pi_{0\,k}, \pi_{1\,k}, \pi_{2\,k})$ with $\kappa_k = 10^{(log_{10} N_{board1\,k} - \Delta)}$ with $\Delta^* \sim$bpert$(1, 4.5, 7)$	Cogan et al. [65]
$T_{b,o\,k}$	Proportion transferred from board to other food	$\frac{1}{100}10^{\kappa}$ with $\kappa^* \sim \mathrm{N}(1.46, 0.3^2)$	Kusumaningrum et al. [43]
$N_{X-board\,k}$	Numbers on other food due to cross-contamination via board	$= N_{board2\,k} \times T_{b,o\,k} \times S_{other\,k}$	expression (2.13)
$S_{raw\,k}$	Consuming other food raw (no=0, yes=1)	$\sim$bern(π_k) with $\pi_k^* \sim beta(239, 3492)$	Devriese et al. [17]
$N_{X\,k}$	Numbers on other food	$= (N_{X-hand\,k} + N_{X-board\,k}) \times S_{raw\,k}$	
$N_{meat2\,k}$	Numbers remaining on portion after food handling	$=N_{meat1\,k} \times (1 - T_{m,b\,k})$	
$P_{protect\,k}{}^*$	Proportion protected area	$\sim$unif$(0, 0.1)$	assumption Hill et al. [6]
$N_{protect\,k}$	Numbers in the protected area	$=P_{protect\,k} \times N_{meat2\,k}$	
$\pi_{u\,k}{}^*$	Probability of undercooking	$\sim$bpert$(0.05, 0.10, 0.2)$	assumption Hill et al. [6]
$S_{u\,k}$	Undercooking (no=0, yes=1)	$\sim$bern$(\pi_{u\,k})$	
$Temp_{cook\,k}{}^*$	Exposure temperature (°C) of protected area in case of undercooking	$\sim$bpert$(60, 65, 70)$	assumption Hill et al. [6]
$Time_{cook\,k}{}^*$	Exposure time (minutes) of protected area in case of undercooking	$\sim$bpert$(0.5, 1, 1.5)$	assumption Hill et al. [6]
$N_{cook\,k}$	Numbers on portion after cooking	$=10^{(log_{10} N_{protect\,k} - \Delta_{protect\,k})} \times S_{u\,k}$ with $\Delta_{protect\,k} = Time_{cook\,k}/D_k$ with $D_k = 10^{-0.14 Temp_{cook\,k} + 8.58}$	Hill et al. [6]
$N_{dose\,k}$	Numbers ingested when consuming meal	$=N_{X\,k} + N_{cook\,k}$	

* input variables.

Table 2: Parameter estimates and standard errors for the models on internal and external contamination after transport & lairage. (data Nollet et al.: association serology-mesenteric lymph node samples; data Botteldoorn et al.: association mesenteric lymph node samples-colon content samples; data Davies et al.: association intestine samples-carcass samples)

	Nollet et al. [24]		Botteldoorn et al. [25]		Davies et al. [29]	
parameter	estimate	empirical s.e.	estimate	empirical s.e.	estimate	s.e.
β_{00}	0.978	0.293	-1.964	0.238	-1.252	0.303
β_{01}	0.776	0.243	-0.257	0.274	-1.178	0.572
β_{10}	1.669	0.561				
β_{11}	1.292	0.475				

Table 3: Baseline results of the XXX-model: summary statistics of the predicted risk of salmonellosis due to consumption of fresh minced pork meat for the normal and susceptible population and of the predicted number of annual cases.

	n	mean	st.dev.	$q_{.05}$	$q_{.95}$
$\overline{\pi}_{nl}$	500	7.704×10^{-6}	5.414×10^{-6}	2.251×10^{-6}	1.822×10^{-5}
$\overline{\pi}_{sus}$	500	4.713×10^{-5}	1.466×10^{-5}	2.750×10^{-5}	7.563×10^{-5}
N_{nl}	500	6996	4916.90	2045	16555
N_{sus}	500	13517	4204.99	7887	21691
N_{tot}	500	20513	9061.45	9932	38246

Table 4: Baseline results of the XXX-model: predicted *Salmonella* prevalence and summary statistics of the *Salmonella* concentration, calculated based on contaminated units only, at different stages of the pork meat pathway. [S = status with $S = 1$ if unit is contaminated and $S = 0$ otherwise; C = concentration per gram; N = numbers per unit].

		EXPOSURE ASSESSMENT:								
			prevalence			$\log_{10}$CFU				
		n	mean	s.e.$_{(\text{mean})}$	n	mean	s.e.$_{(\text{mean})}$	std. dev.	$q_{.05}$	$q_{.95}$
primary production	S_{sero}	18423	0.313	0.00301						
transport & lairage	S_{MLN}	18423	0.207	0.00352						
	S_{CC}	18423	0.206	0.00328						
	S_{in}	18423	0.353	0.00352						
	S_{ex}	18423	0.206	0.00298						
slaughterhouse	S_{kill}	18423	0.497	0.00368						
	S_{singe}	18423	0.0226	0.0011						
	S_{polish}	18423	0.0759	0.0020						
	S_{evis}	18423	0.105	0.00226						
	S_{chill}	18423	0.0427	0.00149						
post-processing	C_{cut}	18423	0.0427	0.00149	787	-0.346	0.0308	0.865	-1.329	1.416
	C_{mix}	1000	0.251	0.0137	251	-0.583	0.0462	0.732	-1.368	0.933
	$C_{portion}$	5×10^6	0.122	0.00015	609917	-0.558	0.00092	0.718	-1.352	0.831
	$N_{portion}$	5×10^6	0.122	0.00015	609917	1.405	0.00092	0.722	0602	2.796
distribution & storage	N_{trans}	5×10^6	0.122	0.00015	610002	1.406	0.00092	0.722	0.602	2.796
	N_{stor}	5×10^6	0.123	0.00015	615591	1.407	0.00092	0.723	0.602	2.799
preparation & consumption	N_{X-hand}	5×10^6	0.000669	0.000012	3347	1.101	0.0088	0.507	0.602	2.127
	$N_{X-board}$	5×10^6	0.0000002	0.0000002	1	0.954	0	0	0.954	0.954
	N_X	5×10^6	0.000043	0.0000029	215	1.041	0.031	0.458	0.602	2.193
	N_{cook}	5×10^6	0.000122	0.0000049	610	1.082	0.0189	0.466	0.602	1.978
	N_{dose}	5×10^6	0.000165	0.0000057	824	1.0723	0.0162	0.464	0.602	2.029