

**Title:** Salmonellosis from Pork Products: A Quantitative Risk Assessment – **NPB #07-079**

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### Industry Summary

We developed a farm-to-illness risk assessment to investigate the risk of human salmonellosis from the consumption of fresh pork. The risk assessment explores major issues in both pre-harvest and post-harvest pork safety including on-farm transmission of *Salmonella*, the effect of various slaughter processes on both the prevalence and the level of the pathogen, and the persistence of contamination through distribution, storage, and preparation. The model is a probabilistic simulation capturing the real-world variation that is inherent in pork production systems. The risk assessment tracks the prevalence and level of salmonella contamination on carcasses through grow-out, multiple stages of slaughter, retail and consumer storage and consumer cooking in the home environment. The result is a prediction of the level of salmonella ingested by an individual from the consumption of a serving of pork. The probability that this will result in salmonellosis is finally estimated. Model results estimate that the risk of salmonellosis from the consumption of fresh pork meat products prepared in the home is  $8 \times 10^{-7}$  (mean estimate), which translates to 0.8 illnesses per million servings. Based upon available consumption data, we estimate this would result in 8,120 cases of salmonellosis per year in the US. (Note that the scope of the work included only fresh pork meat products, and does not include RTE or mixed meat products, or those prepared outside the home).

Since the specific values of the inputs on which the model is based (for example prevalence of *Salmonella* in weaners), can be varied to explore the effect of different control measures, scenario analysis permits a comparison of these approaches in terms of their ultimate impact on the risk of illness. Model analyses indicate that key parameters in the pork production chain that have a strong influence upon the risk of illness are:

- the between herd and within herd prevalence at slaughter,
- the change in prevalence that occurs during lairage,
- the amount of gut contents added to a carcass during evisceration if rupture of the viscera occurs.

Of less importance in controlling the risk of illness are:

- The cross contamination that occurs during scald
- Probability of external contamination on pigs at slaughter that are not infected with salmonella.

This project is of interest to stakeholders at all stages of the pork production chain.

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## Scientific Abstract

The risk of illness due to salmonellosis from fresh pork was estimated by modeling aspects of the pork production chain from the grower operation to consumption, in a 'farm-to-illness' risk assessment. Specifically, probabilistic modeling simulated transmission of the pathogen on-farm, the effect of various slaughter processes on both the prevalence and the level of the pathogen, the persistence of contamination through distribution, storage, and preparation, and the dose-response effect upon consumption. Scenario analysis was performed to identify components of the model that can influence the eventual risk of illness. Model results estimate that the risk of salmonellosis from the consumption of pork meat products prepared in the home is  $8 \times 10^{-7}$  (mean estimate), which translates to 0.8 illnesses per million servings. Based upon available consumption data, we estimate this would result in 8,120 cases of salmonellosis per year in the US.

## Introduction

*Salmonella* infection in the US has been estimated to cause 1.4 million illnesses annually, leading to 31,000 hospitalizations and 1,100 deaths (52). Half of all *Salmonella* cases in humans have been attributed to two of the roughly 2,500 recognized serotypes, namely *S. Enteritidis* and *S. Typhimurium* (15). In humans, salmonellosis is characterized by diarrhea, fever, and abdominal cramps which manifest within three days after infection (14). Although the illness typically resolves without medical treatment within a week, in some cases hospitalization is required (14).

The main route to infection of humans with *Salmonella* species is through food. *Salmonella* spp. often cause asymptomatic infections in a variety of animals, including cattle, pigs, chickens and turkeys (67). Consumption of pork was implicated in 15% of salmonellosis cases in the Netherlands in 1998 (4), and 9% of cases in Denmark in 1999 (30). By 2002 control measures had reduced the proportion of salmonellosis from pork in Denmark to below 4% (1).

In the US, pork consumption averages 51 pounds per person, and pork ranks as the third most-consumed meat after beef and chicken. The Continuing Survey of Food Intakes by Individuals (CSFII) indicates that most pork is consumed at home (24). Previous work predicts from 21,000 to 245,000 annual cases of salmonellosis in the US associated with pork consumption (53), or roughly 1.5 to 17.5% of all cases. In another study the proportion of foodborne salmonellosis due to pork in the US was estimated to be between 6 and 9% (Frenzen *et al.*, 1999 *in* (23)).

*Salmonella* can be introduced to the farm environment not only in contaminated feed and water, but also by soil or dust brought in on personnel, rodents, insects and even air currents (22). Once established, the pathogen can be transmitted between pigs through the fecal-oral route and possibly via aerosols (22); (75). Estimates of *Salmonella* prevalence in US pigs from the nursing/weaning to the finishing stage range from 3 to 35% (66).

Measures to control *Salmonella* in the slaughterhouse have been enhanced since 1996 when the Food Safety and Inspection Service published its final rule on Pathogen Reduction and HACCP (Hazard Analysis Critical Control Point) Systems. This rule obliges plants to identify 'critical control points' where the impact on microbial contamination is greatest, and implement some means of addressing the risk at each of these points (56). In pork production, the critical control points include the dehairing step, the pre-evisceration wash, head drop, evisceration, the final trim and wash, and the chilling step (26).

The purpose of this project is to model pork production and consumption in the US, in order to i) estimate the risk of illness associated with pork consumption, and ii) to examine the influence of the

stages of pork production upon the risk, and iii) to identify points within the production and consumption chain at which potential control measures might have the most impact.

## Stated Objectives

The objective is the development of a quantitative risk assessment model that will estimate the probability and level of exposure of consumers to *Salmonella spp.* as a result of consumption of pork meat and products, and the resulting risk of illness and number of cases of salmonellosis. This model will be developed in the software Analytica, and will enable the user to predict the impact of interventions in the farm-to-fork continuum.

## Materials and Methods

The risk assessment is based on a probabilistic simulation of the pork supply chain to arrive at estimations of the risk of illness to human consumers of fresh pork. Various elements of the pork supply chain have been modeled previously to predict the risk of illness or to shed light on potential critical control points (Table 1). These range from simulations of nearly the entire production and consumption chain, as in (33), to simpler models in which the risk of illness is estimated from measured prevalence and level at retail (29). Five published risk assessments of *Salmonella* in pork have been reviewed and compared, with the results summarized in **Error! Reference source not found**. Although the usefulness of four of the risk assessments is limited by their relatively narrow scope, we based our Farm Module on that presented by the Veterinary Laboratories Agency. The techniques used to arrive at values for the modeled parameters (as shown in Table 1) ranged from simply including a correction to values obtained from the literature, to performing a simulation of the process underlying the variable in order to calculate its value.

Table 1: A comparison of risk assessments of *Salmonella* in pork.

Phase of Pork Production-Consumption Modeled?	Hill <i>et al.</i> , 2003	Miller <i>et al.</i> , 2005	Alban <i>et al.</i> , 2002	Giovannini <i>et al.</i> , 2004	Van der Gaag <i>et al.</i> , 2004
On-farm prevalence and level	Yes	Yes			Yes
Prevalence and level during transport/lairage	Yes	Yes			Yes
Prevalence and level during slaughter	Yes				Yes
Prevalence and level at retail	Yes		Yes		
Preparation	Yes	Yes			
Consumption	Yes	Yes		Yes	
Dose-Response	Yes	Yes	Yes	Yes	

Table 2: A comparison of risk assessments of *Salmonella* in pork.

	Hill <i>et al.</i> , 2003	Miller <i>et al.</i> , 2005	Alban <i>et al.</i> , 2002	Giovannini <i>et al.</i> , 2004	Van der Gaag <i>et al.</i> , 2004
Scope	Farm to human illness; 3 categories of product	Farm to human illness and economic cost; 2 products	Carcass to human illness; dry sausage	Retail to human illness; 4 products	Farm to trimmed carcass
Model summary	The prevalence and/or degree of <i>Salmonella</i> contamination was modeled, based on initial prevalence in weaner pigs, and effect of slaughter and processing conditions on growth and survival of the pathogen.	Reported prevalence of <i>Salmonella</i> on-farm, post-slaughter, and at the retail level were inputs into a model that was then used to evaluate the effects (including economic) of interventions within the farm-to-fork continuum.	The prevalence and level of <i>Salmonella</i> contamination in pork meat for sausages was adjusted for methods of Danish sausage production based on <i>E. coli</i> survival data, and results validated with retail sampling. This was combined with consumption data to predict incidence of human illness.	The prevalence and level of <i>Salmonella</i> contamination of pork products was obtained from retail sampling, and consumption was simulated to arrive at a risk of illness.	The prevalence of <i>Salmonella</i> in incoming pigs was the first step in a transmission model that predicted prevalence through growing and slaughter. according to different facility “risk-profiles”.
Modelled elements	- on-farm transmission - prevalence of <i>Salmonella</i> and amount of contamination through slaughter, retail and preparation - human dose-response (74)	- ‘true’ prevalence from reported on-farm prevalence - prevalence following transport and lairage - human dose-response (74)	- prevalence and level of bacteria in retailed Danish sausage - number of human infections based on attack risk from a previous epidemic	- pattern of consumption of contaminated pork product - human dose-response (74)	- initial prevalence based on supply relationships - on-farm transmission - transmission during transport and lairage - prevalence of contaminated carcasses during slaughter

The current risk assessment is constructed as a farm-to-illness pathway model, utilizing distinct mathematical models to describe each component of the farm-illness continuum. The key stages considered are summarized in Figure 1.

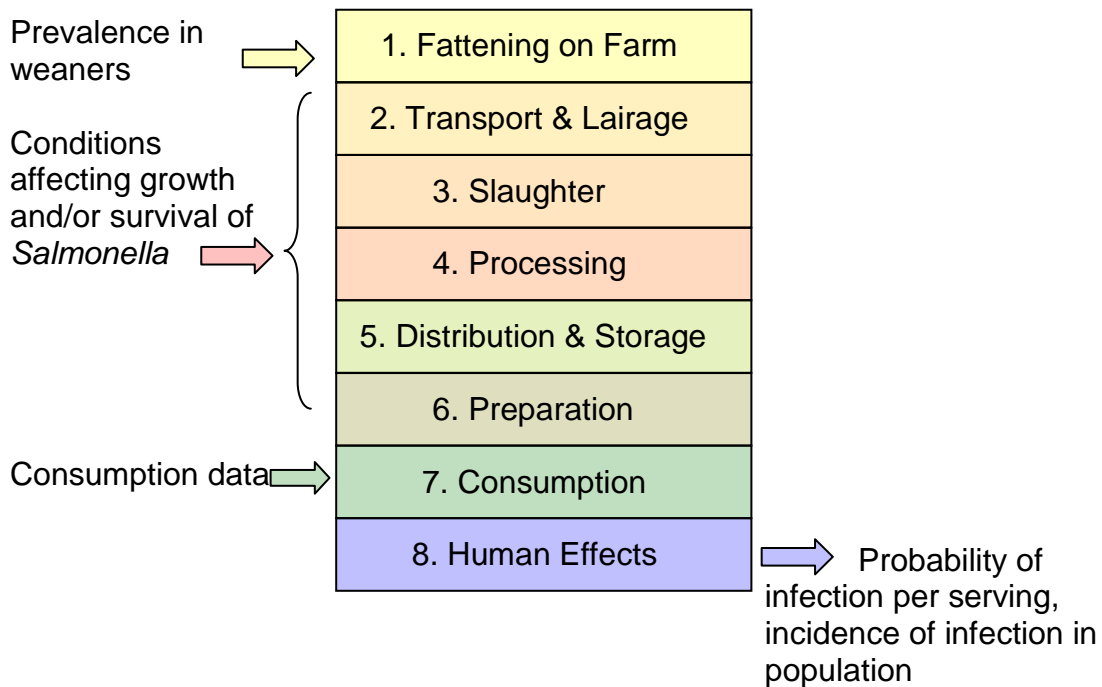


Figure 1: Risk Assessment Model Flow for *Salmonella* in Pork

The modeling begins at the point at which young pigs arrive at the grower/finisher farm where they will grow and fatten for about 4 months (71) before slaughter. This stage of the model is concerned with the rate of transmission of *Salmonella* infection between pigs, and estimates the ultimate prevalence as the pigs leave the facility.

Following the grower/finisher stage the pigs are transported in trucks to the slaughterhouse and spend some time in 'lairage', held in concrete pens in the abattoir, possibly mingled with pigs from other farms (75), while awaiting slaughter. This period is commonly 2-6 hours (34), and rarely more than 8 in the US (23). During this time, the degree of shedding of *Salmonella* in feces of infected pigs often increases due to stress, and lairage has been reported to result in an increase in the prevalence of infection (3);(11);(12);(22).

Pig slaughter involves several steps which may affect the prevalence and level of carcass contamination. Briefly, the pig is stunned, usually with an electric current, and bled. To loosen the hair the carcass is scalded by passing through a hot water bath, then it is de-haired in a rotating drum lined with flails. Since pigs are generally slaughtered with the skin left on, it is important to remove as much of the hair and adherent debris as possible. To remove any remaining hair the carcass is singed in a gas oven, scraped or polished and finally rinsed with a spray wash. The head is removed next and is washed and disassembled separately, and frozen immediately after. The carcass is then eviscerated and the pluck set (tongue, esophagus, trachea, diaphragm, lungs, heart, liver and kidneys) removed and disassembled prior to being washed and frozen. Finally the carcass is split, trimmed and washed a final time before being moved into the cooler for 18 to 24 hours (18). The

stages of slaughter provide opportunities for pathogen inactivation by heat and by antimicrobial compounds in the washes, however there are also opportunities for contamination from other carcasses and from the viscera.

The chilled carcass, trimmings, head and viscera are processed to varying degrees in the production of retail pork products. Products can be cuts of whole pork meat, for example chops, steaks and roasts, or can be prepared by combining meat from different parts of the carcass and even from different carcasses, for example sausages. The product is then stored at retail and by the consumer. Assuming packaging effectively prevents cross-contamination, this stage is associated with added risk of illness only if the 'cold chain' is broken, and temperatures increase enough to allow the growth of *Salmonella* within the packaged meat product, altering the level of the pathogen. Once the pork product is taken out of storage in the home, various factors may come into play including cross-contamination and under-cooking. The latter acts on the extent of contamination already present in the product, and affect the risk of illness when the product is consumed.

**Model Scope** – The model considers the risk (prevalence and level of *Salmonella*) associated with fresh cuts of pork (chops, steak, ribs, fresh ham and roasts), and examines products consumed at home. The risk is estimated for inadequate cooking of products. RTE, mixed meat, and products prepared outside the home are beyond the scope of this assessment.

## Mathematical Description of Model Components

### 1 Farm component

Nursery pigs entering the grower-finisher facility may or may not be carrying *Salmonella*, depending on the prevalence of *Salmonella* at their previous site. In a continuous-management style of grower-finisher operation, the pigs already residing in the facility may also be carrying the bacterium, and represent a source of potential infection to the newly-arriving pigs. The farm component of this risk assessment adopts a published model describing the transmission of *Salmonella* on continuously-managed grower farms in the United Kingdom (UK) (32), which has been adapted to reflect US swine production systems.

In the US, groups of pigs are contained in individual pens in a large building; a typical building consists of 40 pens of growers. The underlying concept of the model is that transmission of *Salmonella* can occur within any given pen, and between pens. The model is designed to consider both the spread of infection, and the infection state, specifically excretor, carrier, or immune. For the purposes of the risk assessment we assume that once a pig is infected (and classed as an excretor) it is considered an infected pig at slaughter. We do not consider reversion to the carrier or immune state under the assumption that these infection states may still pose a risk to consumers. The model is implemented as described by the authors, using the data presented in Table 3, to estimate the prevalence of excretors in a group of pigs at removal from the grower farm for slaughter. From this, we determine the prevalence between groups of pigs at removal from the farm.

The Farm module is designed for grower-finisher operations using confinement-type housing. While confinement is used in just over 53% of U.S. grower-finisher sites, the larger operations favor this style, resulting in 81% of the national herd being finished this way (71).

Table 3: Variables and values used in farm module implementing the model as described by (61).

Variable	Value	Reference
Prevalence of <i>Salmonella</i> in nursery pigs	5 % (fecal)	Sanchez <i>et al.</i> , 2007
Prevalence of <i>Salmonella</i> in existing growers at T <sub>0</sub>	8.5% (fecal)	Sanchez <i>et al.</i> , 2007
Pigs per building	1100	Jackson <i>et al.</i> , 2000
Pigs per pen	25	Brumm <i>et al.</i> , 2005
Time at grower-finisher operation	normal(114.6,1.2)	Swine 2006
Transmission rate within pens	$1.4 \times 10^{-3}$	Hill <i>et al.</i> , 2008
Transmission rate between pens	$9 \times 10^{-4}$	Hill <i>et al.</i> , 2008

## 2 Transport and Lairage

After nearly four months at the grower-finisher facility the pigs are loaded into a trailer and transported to the abattoir, where they and others may commingle in lairage pens as they await slaughter.

Prevalence of *Salmonella* within herds of pigs has been shown to increase over transport and lairage (46);(47);(39);(3);(11);(12);(22);(35);(38);(34). This increase in within-herd prevalence may be mediated by soiling of the lairage environment and subsequent contamination of the pigs' digestive system (36).

In a meta-analysis of *Salmonella* prevalence studies, Sanchez *et al.* concluded that the change in prevalence over lairage ranged from 3- to 7-fold (66). However these include results based on fecal prevalence, which is a measure affected by re-activation of latent infections. Hurd *et al.*, (2003) cited a 39.9% culture prevalence and a 17% seroprevalence at slaughter, which implies up to a roughly 2.5-fold increase of internal contamination over transport and lairage (37).

Soiling of the lairage environment by pigs harbouring *Salmonella* also contributes to external contamination of the waiting pigs. The assignment of external contamination is based upon the infection status of a pig, specifically infected or not infected based upon the prevalence following lairage. The assumption is made that pigs which are infected with *Salmonella* are also externally contaminated. According to Tamplin *et al.*, the prevalence of *Salmonella* externally on carcasses just after bleeding was 73% whereas the prevalence in fecal samples was 33.3% (69). Therefore for pigs which are not infected the probability that they are contaminated on their exterior with *Salmonella* is given by 0.6. Given a pig is contaminated on the exterior, the level of contamination is then assigned. Davies *et al.*, found levels of *Salmonella* on the surface of pigs sampled just before scalding from  $10^2$  to  $10^3$  counts per  $0.1 \text{ m}^2$  (19). This is in line with findings by Berends based on Oosterom 1985 (5). Given this information the surface contamination of a pig is assumed to be Poisson distributed with a mean concentration per  $\text{cm}^2$  of 1680 CFU assuming a pig surface area of  $16,800 \text{ cm}^2$  (43).

## 3 Slaughter component

To examine the influence of the stages of pork production on the risk of illness a model describing the influence of the slaughter process upon the *Salmonella* contamination level of carcasses is required.

The slaughter component of the risk assessment considers 5 key stages of slaughter, specifically scalding of the carcass, singe and dehairing, pre- and post-evisceration washes, evisceration, and chilling.

The slaughter process begins with stun and exsanguination. Following this, the carcasses pass through a scald tank, and typically spend about 6 minutes in the hot (~58.8°C) water (22). While the purpose of the scald is to loosen hair for removal it also results in a reduction of the *Salmonella* contaminating the surface of the pig. Recovery of *Salmonella* on pigs following scald range from 0% (21), to prevalences of 1% (60) and 5.7% (20). A decimal reduction value (D-value) for *Salmonella* of greater than 9 is expected to result from typical scald conditions (Humphrey, 1981 in (22)), and Pearce *et al.* (59) recovered no *Salmonella* from scald tank water (average temperature 61°C). However, Swanenburg *et al.* (68) report recovery of *Salmonella* from scald tank water samples. In addition, the prevalence of contaminated carcasses has been reported to increase when scald water temperature falls below 61°C (Davies *et al.*, 1999 in (31) *et al.*, 2003) indicating cross-contamination amongst carcasses entering the same scald tank. Cross-contamination during scald processes has also been demonstrated for other species of livestock. Mulder *et al.* demonstrated that a marker organism could be detected on >200 carcasses following the scald of the seed carcass when examining poultry, and demonstrating that if the organism of interest can survive in the conditions of the scald tank there is opportunity for carcasses to be cross-contaminated (55). We therefore include explicit modeling of cross-contamination at this step by considering the exchange of organisms from the carcass to the scald water and conversely from the scald water to the carcass.

Following scald the hair loosened in the scald step is removed in a dehairing machine, and any remaining hair is singed and in some cases polished or scraped off. This combination of steps has been reported to result in a level of *Salmonella* that could not be detected ((60); (8), Gerats, 1990 in (6), (27)). However, others have been able to recover *Salmonella* after this step (for example (20)). Counts of *Salmonella* on carcasses through these stages have not been reported, and the effect of these processes on other bacteria is variable (see for example (62); Gerats, 1990 in (6); (19); (64); (9)) ; (73); (59).

The slaughter process includes two steps in which an antimicrobial wash is applied to the carcass: prior to evisceration and at the end of the slaughter line just before the carcass is sent to the cooler. In the US a 2% lactic acid solution is typically used for this purpose in larger plants, and overall 53% of slaughter plants use such an organic acid rinse (13).

The pre-evisceration wash is followed by head drop during which the head is severed and taken away for further processing. Next evisceration is performed, in which the carcass is opened ventrally and the viscera and pluck set (tongue, esophagus, trachea, diaphragm, lungs, heart, liver and kidneys) are cut loose from their attachments and also sent for further processing.

Evisceration involves manipulation of the carcass and viscera as the incision is made and features the risk that a rupture may result in contamination of the carcass with the contents of the cecum, colon, or the lymph glands in the region, any of which may contain *Salmonella*. *S. typhimurium* has been isolated from tissues of pigs as early as 2-3 hours after exposure to a contaminated environment, such as a lairage pen ((12); (35)). Furthermore, pigs can harbour the pathogen for up to 36 weeks (Wood and Rose, 1992 in (75)). An industry survey (2005) found that in fewer than half of pork slaughter plants, workers sanitize their hands or gloves following each unit of product ((13)). Similarly, Taormina and Dorsa (70), observing knife decontamination practices in 2 large US slaughter plants, reported that up to 5 carcasses were processed after a single decontamination (a 1s dip in 83°C water), and that less than 50% immersion of the knife was more common than complete

immersion. As a result, any material that is released onto the carcass in a poorly performed evisceration has a chance of contaminating not only the same carcass, but also subsequent carcasses.

Contamination may also result from the environment of the slaughterhouse. In two of four visits to a French facility (and none in three to another), Giovannacci et al. (28) recovered from the evisceration environment *Salmonella* serotypes that had not been isolated from either pigs or environmental samples at previous stages of slaughter. In one of the two cases one of the novel serotypes was also recovered from slaughtered carcasses prior to cutting.

The slaughter model examines changes in prevalence and level of *Salmonella* on carcasses as they move through the stages of slaughter. Note that only groups of pigs containing at least 1 infected pig are considered. The assumption is made that groups that are free of *Salmonella* (based on the group prevalence on removal from the farm), remain free of *Salmonella* and do not pose a risk of infection.

### 3.1 Scald

The number of *Salmonella* on a carcass following scald is therefore dependent upon the level of contamination in the scald water, which is in turn dependent upon the contamination level of carcasses scalded previously. The scald model is set up for 100 carcasses, and assumes each carcass is in the scald tank for an average of 6 minutes. Each of the 100 carcasses is assigned an infection state based upon the within-herd prevalence of pigs at slaughter with each carcass considered as a Bernoulli trial of either infected or not infected. The initial level on each carcass is randomly assigned using the infection state as a conditional factor as previously described. The carcass enters the tank and organisms are transferred from the carcass to the scald tank. In addition to organisms washed-off into the scald water the carcass may pick-up *Salmonella* by cross-contamination if there are surviving organisms in the scald water from previously-processed carcasses. To determine this, the survival of the changing population of *Salmonella* in the scald tank is tracked throughout the scald of the 100 carcasses. Carcasses that have no contamination when they enter the scald tank may become contaminated if surviving organisms are present in the scald water.

The number of *Salmonella* contaminating a carcass scalded at time  $t$ ,  $N_{sc_t}$ , is given by the following set of equations:

$$\begin{aligned}
 N_{sc_t} &= N_c - NT_t^+ + Ng_t \\
 Ng_t &\sim Bin \left( NT_t, P_g \right) \\
 NT_t &= Bin \left( NT_{t-1}, P_s \right) + NT_t^+ - Ng_t \\
 NT_t^+ &\sim Bin \left( N_c, P_w \right)
 \end{aligned}$$

Here,  $N_c$  is the initial contamination level of a carcass prior to scald,  $NT_t$  is the number of *Salmonella* in the scald tank at time  $t$ ,  $NT_t^+$  is the number transferred to the scald tank from the carcass,  $Ng_t$  is the number added to a carcass as a result of cross-contamination from the scald water to the carcass,  $P_g$  is the probability that *Salmonella* are transferred from the scald water to a carcass during the scald process,  $P_s$  is the probability that *Salmonella* will survive in the scald tank and  $P_w$  is the probability that *Salmonella* will be washed off the carcass into the scald water.

The probability that *Salmonella* will survive in a timestep  $t$  is given by  $P_S = t/10^{\text{Log}D}$  where  $\text{Log}D = -0.1759 T + 10.643$  ( $R^2 = 0.98$ ) fitted using least squares linear regression to examine data on D-values tested in scald water (from (7)). A typical scald tank in the US uses 58.8°C water (22); this temperature is adopted here. The duration of timestep  $t$  in minutes is given by 60/plant capacity. The typical plant capacity in the US is 685 pigs per hour (34). No data could be identified to estimate  $P_w$  in the context of the scald of pigs. Therefore, the assumption is made that scald tank immersion has a similar effect to spray washing. Spray washing produces between a 1.36 and 3.51 log reduction (25) (17). We therefore assume uniform distribution using these as limits, i.e. Uniform( $10^{1.36}$ ,  $10^{3.51}$ ).

### 3.2 Dehair and Singe

Following scald, the impact of dehair and singe is considered. To capture the variation in the reports on the effect of dehair and singe we consider the impact of dehair and singe to be one of two types, either highly effective (in terms of the ability to reduce *Salmonella* levels) or of reduced efficiency. The probability that dehair and singe will be highly effective is assumed to be Triangular(0.9,0.95,0.99) based on the estimated effectiveness reported by (1), and the log removal achieved given a highly effective dehair and singe is assumed to be 7 logs. If for a particular carcass the steps are categorized as reduced efficiency, then the log reduction for *Salmonella* is assumed to be Uniform( 2.67, 3.12) based on (17).

### 3.3 Pre-evisceration Wash

Washing has been demonstrated to reduce carcass contamination, but not eliminate it. Van Netten et al. (72) succeeded in reducing levels of *S. typhimurium* from inoculated pig carcasses to below detection with a 60 second 2% lactic acid rinse when the initial contamination was 1 log CFU/cm<sup>2</sup>, but not when initial contamination was twice that. Clayton (17) achieved a 2.77 log reduction in levels of *S. typhimurium* inoculated onto pig half-carcasses with a 2% lactic acid spray, and Fabrizio and Cutter (25) demonstrated a 1.79 log reduction in levels of the pathogen on inoculated pork bellies treated with a 15s spray of 2% lactic acid. The log reduction from washing is described as an empirical probability distribution using available data (17) (25), assuming all data points have equal weight.

### 3.4 Evisceration

During evisceration the population of carcasses can be segregated into those which were contaminated prior to evisceration, and those that were not contaminated. During evisceration there are three possible outcomes – 1) there is no change in contamination compared to prior to evisceration, 2) the viscera is ruptured and if the carcass is categorized as infected the exterior becomes contaminated with *Salmonella* from the gut as described below, and 3) the carcass becomes contaminated by cross-contamination from the equipment where outcome 2 has occurred previously. Here, outcomes 2 and 3 are not mutually exclusive, whereas outcome 1 requires that neither outcome 2 nor 3 occur. To simplify the modeling approach, at this point the model pathway is segregated into these categories and recombined later in the model according to the associated weight (i.e likelihood of occurrence) of each of the three outcomes. The modeling approach can be summarized in Table 4. Each carcass is randomly assigned to one of these outcomes based upon the probability of occurrence, and the appropriate number of organisms is added to the carcass on this basis.

Table 4. Calculation of the change in contamination level as a result of evisceration.  $P_R$  is the probability of rupture occurring during the evisceration of carcass,  $P_{CC}$  is the probability of cross-contamination of a carcass from the equipment,  $N_{rup}$  is the number of *Salmonella* added to a carcass if rupture occurs given a carcass is infected, and  $N_{CC}$  is the number of *Salmonella* added if cross-contamination occurs. Note that for carcasses that are not infected with *Salmonella*  $N_{rup} = 0$ .

EVISCERATION OUTCOME	PROBABILITY OF OCCURRENCE	NUMBER OF ORGANISMS ADDED
Rupture of intestines	$P_R$	$N_{rup}$
Cross-contamination	$P_{CC}$	$N_{CC}$
Rupture and cross-contamination	$P_R P_{CC}$	$N_{rup} + N_{CC}$
Neither rupture nor cross-contamination occurs	$1 - P_R - P_{CC} + P_R P_{CC}$	0

One of the most significant potential sources of equipment contamination that can lead to subsequent cross-contamination of proceeding carcasses is the rupture of the intestines during evisceration (22). McDowell *et al.* (50), reported a significant association between *Salmonella* in intestines of pigs at slaughter and the occurrence of *Salmonella*-positive carcasses post-evisceration.

The probability that cross-contamination occurs for a given carcass is determined by considering the probability of viscera rupture in infected carcasses that were processed prior to the carcass being considered. Considering the same group of 100 carcasses examined during the scald process, we first determine how many of those carcasses were infected. We then introduce the concept of a cross-contamination threshold group. This is the number of carcasses, prior to the carcass of interest, over which cross-contamination could be expected to persist. That is, if a specific carcass results in contamination of the equipment during evisceration, in how many carcasses following this one might we expect to see cross-contamination from the original eviscerated carcass. This value must be equal to or less than the size of the group being considered (in this case 100 carcasses). It is assumed that the threshold group is 100 carcasses (the same size as the group of carcasses being monitored). The probability that cross-contamination will occur as a result of the evisceration of previous carcasses is given by  $P_{CC} = P_{cl} (1 - P_{rup})^{N+_{TG}}$ , where  $P_{cl}$  is the probability that the equipment is adequately cleaned before the current carcass is eviscerated,  $P_{rup}$  is the probability that the intestines will rupture during evisceration, and  $N+_{TG}$  is the number of infected carcasses in the threshold group given by  $N+_{TG} \sim Hypergeometric(N_{TG}, N+, N-)$ , and where  $N_{TG}$  is the size of the threshold group (assumed to be 100 carcasses),  $N$  is the total size of the group from which the threshold group originates (assumed to be 100 carcasses), and  $N+$  is the number of infected carcasses in  $N$ .

The probability of viscera rupture has been measured at 4.5% in a study of 1600 pigs undergoing slaughter (54). Careful evisceration has been shown to be associated with lower rates of carcass contamination compared to standard techniques (Oosterham, Childers in (6); see also (10);(20)), yet line speeds and a high rate of labor turnover (a turnover of 100% in some plants (49)) conspire to discourage this. The probability of cross-contamination during evisceration is based upon data from a US industry survey (13) examining how often equipment was cleaned (34.5%).

### 3.5 Post evisceration wash

Post-evisceration washing is assumed to have the same impact as the pre-evisceration wash.

### 3.6 Chilling

Chilling reduces the level of *Salmonella* on a carcass. The log reductions for chilling are described using an empirical distribution of the data presented by (16). Whilst carcasses may come into contact during chilling we do not consider the cross-contamination as it has been reported that *Salmonella* prevalence is reduced over the chill step (65), particularly in large plants (18).

## 4 Processing

Following chilling the carcass is portioned. A typical 265 lb live pig is assumed to yield about 88 lbs of lean meat. The distribution of serving sizes for pork in the US has been previously described by a normal(3,0.9) distribution truncated at 0.1 and 6 ounces at the lower and upper tails respectively (53). Using this information, the concentration per gram of pork is determined and subsequently the number per contaminated serving determined (assuming a Poisson distribution).

## 5 Distribution and Storage

**Storage of the products** – During storage of products we are particularly interested in predicting any growth or decline that may occur in contaminated products.

For the purposes of this risk assessment growth is modeled as a three-stage process: the lag phase, where the organism appears to be acclimatizing to the conditions and preparing for growth (and the population size does not change), the growth phase, and finally the stationary phase during which the number of organisms is sufficiently high as to be self-limiting, so again the density of organisms does not change. The model is parameterized by just three parameters — growth rate; lag time; and maximum density. The lag period for *Salmonella* in pork is characterized using available data (41). Using these data, the duration of the lag period,  $L(T)$ , is given by  $L(T) = 9572 \cdot 10^{-2.4196 T}$ , with an associated  $R^2$  of 0.96. To evaluate the variation in growth rate with temperature, the model adopted in the risk assessment is a variation of the square root of growth rate that is quadratic with temperature, up to a maximum temperature. Whilst a linear variation of square root of growth rate is a well-recognized model for the growth of bacterial populations (51), the linear assumption fails at high enough temperatures; Curves are more desirable to describe the overall shape (63), but insufficient data are available to parameterize such a model for *Salmonella* in pork. Using least squares regression, the growth rate  $k(T)$  is given by:

$$\sqrt{k(T)} = \begin{cases} -0.04972 + 0.0051 T + 0.00036 T^2 & 6.5 < T < 48 \\ 0 & \text{otherwise} \end{cases}$$

where  $T$  is the temperature (°C). The model fit has an associated  $R^2$  of 0.85. The lower limit of temperature for growth is assumed to be the intercept of the predicted growth rate at  $k = 0$ . The maximum growth temperature for *Salmonella* spp. in poultry has been reported to be 48°C (58). Data are not available to characterize this parameter for pork, therefore 48°C is adopted here and the maximum temperature for growth is set to 48°C.

Baseline retail storage temperatures are based on the Audits International (2) data on fresh meat product temperatures immediately upon removal from retail display cabinets. The storage time in retail cabinets is not known. WHO/FAO (74) assumed a uniform distribution from 1 to 7 days for retail storage time (but applied a negative correlation between time and temperature). For this assessment, the baseline has been set as a Triangular(1,2,7) days distribution. The temperatures of fresh meat products purchased by consumers were measured immediately after removal from retail cabinets, and on placement in consumer refrigerators (2). In these data there is a correlation between these temperatures (Pearson correlation coefficient  $\rho = 0.58$ ), and between the final temperature and temperature rise ( $\rho = 0.58$ ).

Temperature rise ranged from  $-10\text{ }^{\circ}\text{C}$  to  $40\text{ }^{\circ}\text{C}$ , and could be adequately represented by the probabilistic sum of two normal distributions, one with mean  $3.483\text{ }^{\circ}\text{C}$ , standard deviation  $2.134\text{ }^{\circ}\text{C}$ , and weight 0.866; the second with mean  $5.353\text{ }^{\circ}\text{C}$ , standard deviation  $5.223\text{ }^{\circ}\text{C}$ , and weight 0.134. The transport temperature in the model is taken to equal the temperature rise (with baseline distribution just described) added to the retail storage temperature (in an uncorrelated manner). This approach may slightly overestimate transport temperature, since the temperature presumably rises during transport (and the measurements were taken at the end of transport).

The transport time of meats from retail store to consumer refrigerator was measured by the Audits International survey (2). Those data indicate that the distribution of the transport time can be well-represented by a normal distribution with mean 1.01 (hours) and standard deviation 0.1539 (hours) to the power of 2.5.

The temperature in consumers' refrigerators was measured in the Audits International survey by measuring the temperature of a semi-solid dairy product after 24 hours in the refrigerator. The empirical distribution of 939 measurements of refrigerator temperatures was fitted to the probabilistic sum of two normal distributions, the first with mean  $3.818\text{ }^{\circ}\text{C}$ , standard deviation  $2.170\text{ }^{\circ}\text{C}$ , and weight 0.887; the second with mean  $5.405\text{ }^{\circ}\text{C}$ , standard deviation  $4.815\text{ }^{\circ}\text{C}$ , and weight 0.113. This fit to the empirical data is used in the model. The storage time for raw meat products in consumer refrigerators is not known. The FAO/WHO (74) risk assessment for *Salmonella* in broiler chickens suggested a minimum time of zero, a mode of 2 days, and a maximum of 5 days, based on adherence to "use by" dates. In line with the FAO/WHO risk assessment we use a triangular(0, 2, 5) days distribution to describe storage of raw meat in the refrigerator.

## 6 Preparation

To efficiently simulate the impact of cooking, a critical temperature is defined which is the temperature above which it is assumed that complete cooking occurs and no *Salmonella* survive. This temperature has been defined as a final cooking temperature of  $60^{\circ}\text{C}$ . Only those temperatures below a final temperature of  $60^{\circ}\text{C}$  are simulated. For products cooked to a final temperature below the critical temperature the 'protected areas' approach is adopted to describe the impact of cooking. Used previously in microbial risk assessments, (74), the protected areas approach assumes that a proportion of organisms are in some manner sheltered from the full cooking temperature at which the product is prepared. This may be because of the presence of bone, fat globules or simply the symmetry of the product.

This model was originally developed for *Campylobacter jejuni*. *C. jejuni* is very sensitive to elevated temperatures, with D values reported of 1, 0.35 and 0.26 minutes at  $55^{\circ}\text{C}$ ,  $58^{\circ}\text{C}$  and  $60^{\circ}\text{C}$

respectively (44). The sensitivity of *C. jejuni* to thermal effects suggests that organisms exposed to the heat of cooking without significant thermal protection are unlikely to survive cooking. As a result, those cells that are present on the surface of meat and poultry products are likely to be inactivated with even moderate heat, unless the product is grossly undercooked. Thus, it may only be those cells that are in part of the product affording some level of protection from direct heat that will survive. These areas may include visceral cavities, crevices, and areas around joints or in cut and bruised tissues. *Salmonella* is somewhat less heat-sensitive than *C. jejuni* but is still likely to be destroyed on the surface of a product during adequate cooking. D-values for *Salmonella* in pork products of 10 minutes at 58°C down to 0.5 minutes at 65°C have been reported, with the sensitivity of *Salmonella* rapidly increasing with temperature (42). According to survey data (Audits International) approximately 80% of individuals cook meat products to a final internal temperature of 60°C or higher, probably eliminating the vast majority of surface contamination. It is therefore reasonable to assume the protected areas approach can be applied to *Salmonella*. Final cooking temperatures are assumed to be as measured by Audits International (1999). It is further assumed that if the final cooking temperature exceeds a critical temperature (60°C), then all *Salmonella* not in protected areas are killed. There are three stages to the model:

1) Estimate the proportion of *Salmonella* that is in the protected areas: for final cooking temperatures above the critical temperature (60°C) it is first assumed that between 0.1 and 5% of the population of contaminating *Salmonella* is associated with protected areas, whereas below that temperature, all *Salmonella* are assumed to be effectively protected.

2) Estimate the temperature to which they are exposed: it is assumed that during cooking the temperature in the protected areas can be represented by data reported by Audits International (1999).

3) Estimate the duration of exposure. It is assumed that this duration ranges from 0.5 to 1.5 minutes, with a mode of 1 minute.

These assumptions are used because there are no data indicating what the true time and temperature combinations are for *Salmonella* in protected areas.

The D-value is then used to calculate the reduction in the population in the protected areas. It is assumed that all *Salmonella* not (effectively) in the protected areas are destroyed by cooking.

The cooking temperature  $T$  is selected from the distribution just described. The corresponding logarithmic death rate  $d_s(T)$  for *Salmonella* is estimated. The cooking time  $t_c$  is selected from the distribution of times for exposure of protected organisms. The probability  $p_c$  for an individual organism to survive cooking is then computed as

$$p_c = f_p 10^{-d_s T t_c}$$

where  $f_p$  is the probability for an organism to be in a protected area (unity for  $T$  less than the critical temperature). The number (distribution)  $N_{is}$  of organisms on a serving and assumed to be ingested after cooking is then  $N_{is} \sim B(N_c, p_c)$ .

## 7 Human Effects: Estimation of Illness

To predict and assess the impact of exposure to defined levels of pathogens such as *Salmonella*, probability models are currently being widely used. These models are specified by a dose-response relationship for each pathogen thus assuming that risk depends upon the number micro-organisms ingested. The dose-response relationship for *Salmonella* has been investigated by several researchers, and a number of candidate models have been proposed (see for example (48)). These models have been reviewed, and a model based upon available epidemiological outbreak data

developed (74). This model is adopted here. To describe the probability of developing illness,  $P_{ill}$ , a Beta-Poisson dose-response model was found to provide the best fit to the data. The model has the form  $P_{ill} = 1 - \left(1 + \frac{D}{\beta}\right)^{-\alpha}$  where  $D$  is the dose of *Salmonella* ingested and  $\alpha$  and  $\beta$  are parameters of the model. Using outbreak data to fit the model, the expected values of the best fit  $\alpha$  and  $\beta$  are 0.1324 and 51.45 respectively (74). These parameter values are adopted in this assessment.

## Results

**Risk of illness** - The model estimates that the mean risk per serving for fresh pork meat products, consumed in the home setting is  $8.01 \times 10^{-7}$ . This translates to 0.8 cases per million servings at the mean estimate. The model predicts the serving-by-serving variation; this results in a variation in risk at the individual serving level for contaminated servings as shown in Figure 2. It can be seen there are essentially two populations of products at consumption, those with a high level of contamination that has persisted through cooking, and those with a lower level of contamination. These two populations can be seen in the distribution of the level of contamination following evisceration (Figure 3). Here carcasses either have a low level of contamination, or a high level due to the addition of contamination following rupture of the viscera. The mean estimate of a risk per serving of  $8.01 \times 10^{-7}$  from inadequate cooking is in line with risk estimates from other risk assessments, for example a risk of salmonellosis per serving of  $6.75 \times 10^{-7}$  for pork meat products due to inadequate cooking was estimated in a risk assessment developed to consider the UK pork production system (33).

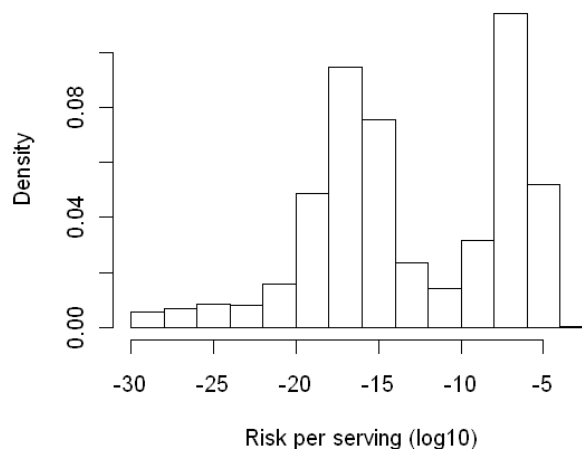


Figure 2: The distribution of the risk per serving (on the  $\log_{10}$  scale) for servings contaminated with at least 1 CFU *Salmonella* at consumption as predicted by model.

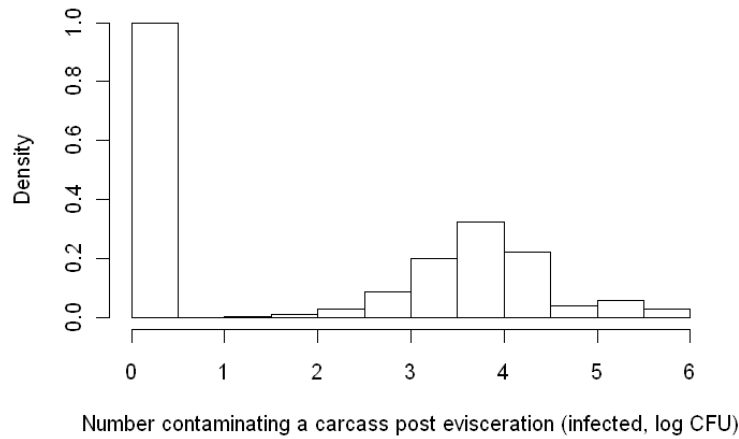


Figure 3: The distribution of the level of contamination ( $\log_{10}$  CFU) post evisceration (figure shows the level for carcasses that originate from pigs that were infected with *Salmonella*).

**Number of cases per year** – National pork board (57) reported that annually there are 116 eating occasions of pork per capita. This includes all forms of pork products (for example mixed meat and RTE). To estimate the number of illnesses per year it is assumed there are 116 eating occasion of pork, and that of these 38% are pork meat products (steaks, chops, ...) and of these the 81% are in the home (57). This results in  $1 \times 10^{10}$  eating occasions of pork per year. This translates to 8,120 cases of salmonellosis (mean estimate) per year from the consumption of fresh pork meat products in the home, where ingestion of *Salmonella* is a result of inadequate cooking of the product.

## Discussion

The model described here is essentially a model describing the variability that occurs during the production of pork products. It has numerous inputs, some based upon assumptions, other based upon scientific data or evidence. All of the inputs will be associated with some degree of uncertainty. Here we explore a selection of the components of the model with particular reference to the impact upon the risk of illness per million servings. These investigations could equally be considered to be explorations of values because they are uncertain in the model, or exploration of the importance of components of the model in terms of the impact upon risk, and therefore used to inform risk mitigation strategies. All results are based upon simulations of 30k samples. This is assumed sufficient to reach stable estimates to show the interrelationships between parameters in the model, based upon examination of the stability of the model.

**Impact of prevalence at slaughter** - There are two estimates of prevalence that feed into the slaughter components of the risk assessment, these are the between herd prevalence and the within herd prevalence. Both of these factors influence the risk of human illness. The relationship between these factors and the risk of salmonellosis is shown in Figure 4, Figure 5, and Table 5.

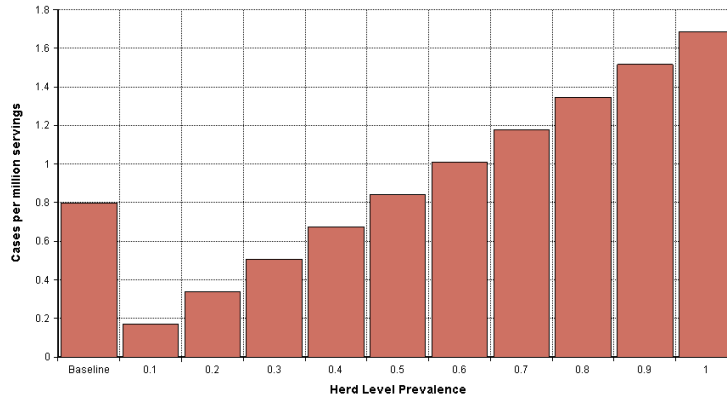


Figure 4: The impact of herd level prevalence upon the estimated number of cases per million servings.

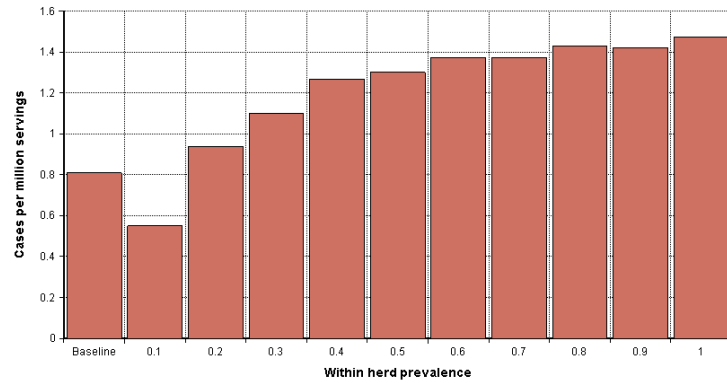


Figure 5: The impact of the within herd prevalence upon the estimate of the number of cases per million servings.

Table 5: The combined impact of varying the between and within herd level prevalence upon the number of cases per million servings.

Herd Prevalence	Within Herd Prevalence										
	Baseline	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
Baseline	0.80	0.56	0.92	1.08	1.27	1.23	1.26	1.44	1.38	1.45	1.49
0.1	0.17	0.12	0.20	0.23	0.28	0.27	0.27	0.31	0.30	0.31	0.32
0.2	0.34	0.24	0.39	0.46	0.54	0.52	0.54	0.61	0.59	0.62	0.63
0.3	0.51	0.36	0.59	0.68	0.81	0.78	0.80	0.92	0.88	0.92	0.95
0.4	0.67	0.47	0.78	0.91	1.08	1.04	1.07	1.22	1.17	1.23	1.26
0.5	0.84	0.59	0.97	1.14	1.34	1.30	1.33	1.52	1.46	1.53	1.58
0.6	1.01	0.71	1.17	1.36	1.61	1.56	1.60	1.82	1.75	1.84	1.89
0.7	1.18	0.83	1.36	1.59	1.88	1.82	1.86	2.13	2.04	2.14	2.20
0.8	1.35	0.95	1.56	1.82	2.14	2.07	2.13	2.43	2.33	2.45	2.52
0.9	1.52	1.06	1.75	2.04	2.41	2.33	2.39	2.73	2.62	2.75	2.83
1	1.68	1.18	1.94	2.27	2.68	2.59	2.66	3.03	2.91	3.06	3.15

**Impact of the probability a carcass that is not infected is contaminated** – The probability that a pig is contaminated on the exterior given it is not infected with *Salmonella* was assumed to be 0.6. To investigate if this is an important component of the model this value was varied. Figure 6 shows the cases per million servings. It can be seen that this factor is not critical in the estimation of illness in the current model framework, with variation in model results being within the bounds of inherent simulation variation (varies between ~0.79 and 0.81).

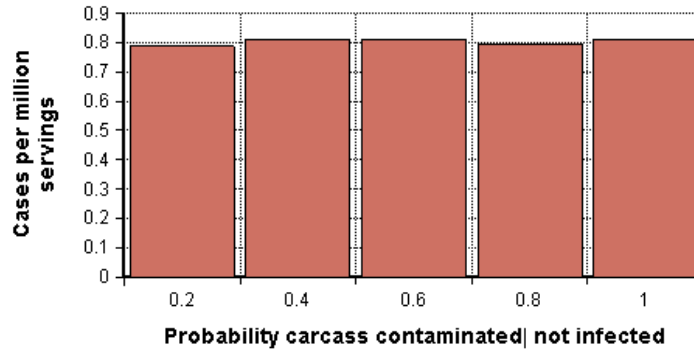


Figure 6: The impact of varying the probability that a carcass is contaminated given it is not infected with *Salmonella*.

**Increase in prevalence during lairage** – During lairage there is evidence that the prevalence within herds increases. Data suggest that this increase is between 1 and 2.5 fold, however, other more qualitative information suggests that it could be as high as 7-fold. To test the importance of this factor values up to 7-fold were investigated. Results are shown in Figure 7.

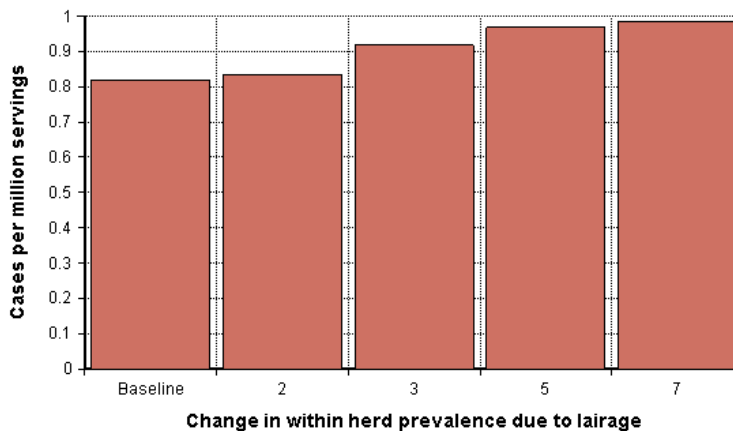


Figure 7: The relationship between the change in within herd prevalence during lairage and the estimate of the number of cases per million servings.

**Cross contamination during scald** - One of the unknowns here is the proportion of *Salmonella* that will be transferred to a carcass as it passes through potentially contaminated water during the scald process. Analysis of the model shows that there is no discernable relationship between this parameter and the risk of illness (values tested ranged from 0.01 to 0.4; values greater than 0.4 are not considered to be biologically plausible by the authors)

**Impact of the amount of feces added to a carcass if rupture occurs during evisceration** – The amount of contamination (and in turn the amount of *Salmonella*) added to the carcass should rupture of the viscera occur is unknown. The impact of this estimate on the risk of human illness is shown in Figure 8.

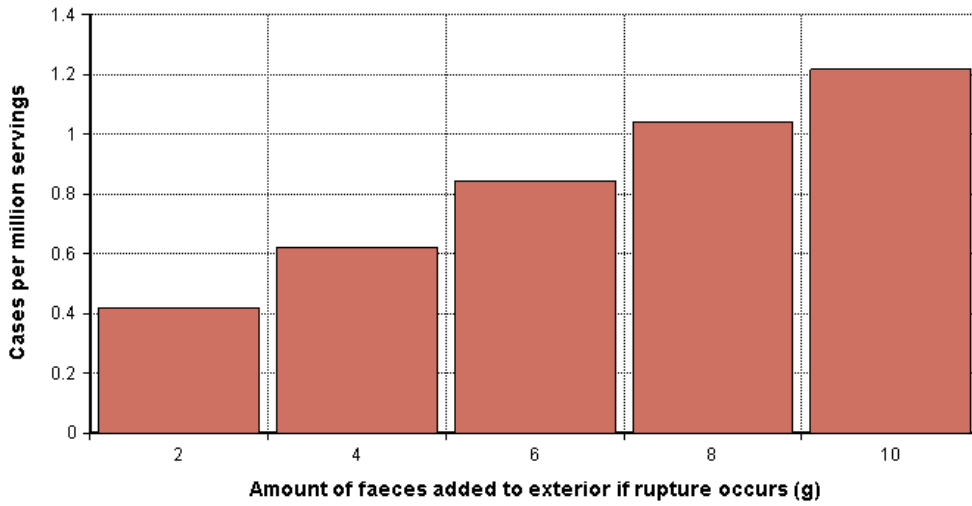


Figure 8: The effect of varying the amount of faeces added to the exterior of the carcass should rupture of the viscera occur.

**Consumer cooking** - The underlying hypothesis of the model component to describe consumer cooking is that a proportion of the organisms are in areas that are not subject to the full heat of the cooking process (“protected areas”), and therefore they survive and persist to ingestion. However, the proportion of organisms that are in such areas is unknown for pork products and this may vary greatly by product and method of cooking. Exploring the impact of this proportion shows that this is highly influential upon risk (Figure 9). This is the last opportunity to mitigate risk prior to consumption and therefore the larger the proportion in protected areas, the greater the risk of illness.

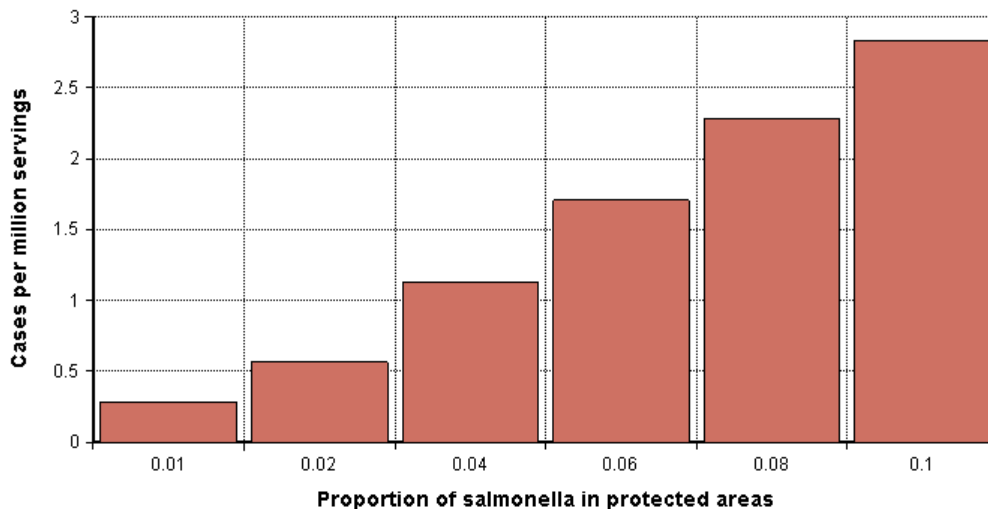


Figure 9: The impact of the assumption of the proportion of salmonella in protected areas during consumer cooking.

## Data gaps and limitations of the work.

A key outcome of any risk assessment is the identification of data and knowledge gaps that can be used to inform future research initiatives. In addition, there are a number of limitations associated with any risk assessment model that are a result of the necessity of attempting to represent real-world situations in closed system models. The data gaps and limitations associated with this work include:

### Rearing

- In the US the continuous management system presented by (61) applies to only 26% of farms, with most of the remaining farms (64%) utilizing variations on an all-in-all-out system (71). Continuous management systems were selected as a mathematical model that has been peer reviewed and is available in the scientific literature is available for inclusion, thus extending the degree of detail of the pork production system that could be represented in the risk assessment within the time and budget constraints of the project. Extending the model to include other production systems is recommended in future work on this risk assessment.

### Transport and Lairage

- Data are lacking for the probability of external contamination as pigs enter slaughter

### Slaughter

- No data available for cross-contamination during scald in pigs
- No data available for amount of material contaminating carcass in the event of a rupture
- Infection status of pigs (excretor, carrier, immune etc) was not explicitly considered.

### Processing

- We did not examine risk of other fresh products such as mixed meats and offal.
- We did not consider the increased risk of cross-contamination during cutting of carcasses, depending on standard of hygiene in plants in question
- We did not consider the effect of modified atmosphere packaging on the risk of illness, or the effect of irradiation and other preservation methods (up to but not including those which render the product RTE)

### Preparation

- Cross-contamination could be included, this would ideally require more detailed consumption data to determine what proportion of pork servings would entail a risk of cross-contamination
- Future work could consider pork eaten outside the home – a growing segment and one which has the potential for widespread outbreaks

### Human Effects

- This model could be refined by identifying high-risk and low-risk groups; however specific applicable dose-response (and consumption data) would have to be available to achieve this.

## 8 Acknowledgements

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## Reference List

- (1) Alban L, Stark KD. Where should the effort be put to reduce the Salmonella prevalence in the slaughtered swine carcass effectively? *Prev Vet Med.* 2005;68:63-79.
- (2) Audits International and Food and Drug Administration. Food temperature evaluation. 99.
- (3) Berends BR, Urlings HA, Snijders JM, Van Knapen F. Identification and quantification of risk factors in animal management and transport regarding Salmonella spp. in pigs. *Int J Food Microbiol.* 1996;30:37-53.
- (4) Berends BR, Van Knapen F, Mossel DA, Burt SA, Snijders JM. Impact on human health of Salmonella spp. on pork in The Netherlands and the anticipated effects of some currently proposed control strategies. *Int J Food Microbiol.* 1998;44:219-29.
- (5) Berends BR, Van Knapen F, Snijders JM, Mossel DA. Identification and quantification of risk factors regarding Salmonella spp. on pork carcasses. *Int J Food Microbiol.* 1997;36:199-206.
- (6) Berends BR, Van Knapen F, Snijders JM, Mossel DA. Identification and quantification of risk factors regarding Salmonella spp. on pork carcasses. *Int J Food Microbiol.* 1997;36:199-206.
- (7) Bolton DJ, Pearce R, Sheridan JJ, McDowell DA, Blair IS. Decontamination of pork carcasses during scalding and the prevention of Salmonella cross-contamination. *J Appl Microbiol.* 2003;94:1036-42.
- (8) Bolton DJ, Pearce RA, Sheridan JJ, Blair IS, McDowell DA, Harrington D. Washing and chilling as critical control points in pork slaughter hazard analysis and critical control point (HACCP) systems. *J Appl Microbiol.* 2002;92:893-902.
- (9) Bolton DJ, Pearce RA, Sheridan JJ, Blair IS, McDowell DA, Harrington D. Washing and chilling as critical control points in pork slaughter hazard analysis and critical control point (HACCP) systems. *J Appl Microbiol.* 2002;92:893-902.
- (10) Borch E, Nesbakken T, Christensen H. Hazard identification in swine slaughter with respect to foodborne bacteria. *Int J Food Microbiol.* 1996;30:9-25.
- (11) Boughton C, Egan J, Kelly G, Markey B, Leonard N. Quantitative examination of Salmonella spp. in the lairage environment of a pig abattoir. *Foodborne Pathog Dis.* 2007;4:26-32.
- (12) Boughton C, Egan J, Kelly G, Markey B, Leonard N. Rapid infection of pigs following exposure to environments contaminated with different levels of Salmonella typhimurium. *Foodborne Pathog Dis.* 2007;4:33-40.
- (13) Cates, S. C., Viator, C. L., Karns, S. A., and Siegel, P. H. Survey of meat and Poultry Slaughter and Processing Plants. 2005.
- (14) CDC (Centers for Disease Control and Prevention). Salmonellosis: General Information. 2008. 2008.
- (15) CDC (Centers for Disease Control and Prevention). Salmonellosis: Technical Information. 2008. 2008.

- (16) Chang VP, Mills EW, Cutter CN. Reduction of bacteria on pork carcasses associated with chilling method. *J Food Prot.* 2003;66:1019-24.
- (17) Clayton NC. The Efficacy of Various Salmonella Intervention Methods Applied to Pork Carcasses During Slaughter. 2002. University of Kentucky.
- (18) Cutter, C. N. Effects of commercial chilling methods for reducing bacteria on pork carcasses. 2003.
- (19) Davies RH, McLaren IM, Bedford S. Observations on the distribution of salmonella in a pig abattoir. *The Veterinary Record.* 1999;145:655-61.
- (20) Davies RH, McLaren IM, Bedford S. Observations on the distribution of salmonella in a pig abattoir. *The Veterinary Record.* 1999;145:655-61.
- (21) Dickson, J. Microbiological Profiles of Hog Carcasses During Processing. 99.
- (22) Dickson JS, Hurd HS. and Rostagno MH. FACTSHEET: Salmonella in the Pork Production Chain. 2002. website pork.org, National Pork Board.
- (23) Dickson JS, Hurd HS. and Rostagno MH. FACTSHEET: Salmonella in the Pork Production Chain. 2002. website pork.org, National Pork Board.
- (24) Continuing Survey of Food Intake by Individuals. Economic Research Service, U. S. Department of Agriculture.
- (25) Fabrizio KA, Cutter CN. Comparison of electrolyzed oxidizing water with other antimicrobial interventions to reduce pathogens on fresh pork. *Meat Science.* 2004;68:463-68.
- (26) FSIS (Food Safety and Inspection Service). Generic HACCP Model for Pork Slaughter. 99. USDA (United States Department of Agriculture).
- (27) Gill CO, Bryant J. The contamination of pork with spoilage bacteria during commercial dressing, chilling and cutting of pig carcasses. *International Journal of Food Microbiology.* 1992;16:51-62.
- (28) Giovannacci I, Queguiner S, Ragimbeau C, Salvat G, Vendeuvre JL, Carlier V et al. Tracing of Salmonella spp. in two pork slaughter and cutting plants using serotyping and macrorestriction genotyping. *J Appl Microbiol.* 2001;90:131-47.
- (29) Giovannini A, Prencipe V, Conte A, Marino L, Petrini A, Pomilio F et al. Quantitative risk assessment of Salmonella spp. infection for the consumer of pork products in an Italian region. *Food Control.* 2004;15:139-44.
- (30) Hald T, Vose D, Wegener HC, Koupeev T. A Bayesian approach to quantify the contribution of animal-food sources to human salmonellosis. *Risk Anal.* 2004;24:255-69.
- (31) Hald T, Wingstrand A, Swanenburg M, von Altrock A, Thorberg BM. The occurrence and epidemiology of Salmonella in European pig slaughterhouses. *Epidemiol Infect.* 2003;131:1187-203.
- (32) Hill AA, Snary EL, Arnold ME, Alban L, Cook AJ. Dynamics of Salmonella transmission on a

British pig grower-finisher farm: a stochastic model. *Epidemiol Infect.* 2008;136:320-33.

- (33) Hill, Andrew, England, Tracey, Snary, Emma, Cook, Alasdair, Kelly, Louise, Evans, Sarah, and Wooldridge, Marion. A 'Farm-To-Consumption' Risk Assessment for *Salmonella* Typhimurium In Pigs. 2003. Department of Risk Research, Veterinary Laboratories Agency Weybridge.
- (34) Hurd HS, Gailey JK, McKean JD, Griffith RW. Variable abattoir conditions affect *Salmonella* enterica prevalence and meat quality in swine and pork. *Foodborne Pathog Dis.* 2005;2:77-81.
- (35) Hurd HS, Gailey JK, McKean JD, Rostagno MH. Rapid infection in market-weight swine following exposure to a *Salmonella* typhimurium-contaminated environment. *Am J Vet Res.* 2001;62:1194-7.
- (36) Hurd HS, Gailey JK, McKean JD, Rostagno MH. Rapid infection in market-weight swine following exposure to a *Salmonella* typhimurium-contaminated environment. *Am J Vet Res.* 2001;62:1194-7.
- (37) Hurd HS, McKean JD, Griffith RD, Rostagno MH. Estimation of the *Salmonella* enterica prevalence in finishing swine. *Epidemiol Infect.* 2004;132:127-35.
- (38) Hurd HS, McKean JD, Griffith RW, Wesley IV, Rostagno MH. *Salmonella* enterica infections in market swine with and without transport and holding. *Appl Environ Microbiol.* 2002;68:2376-81.
- (39) Hurd HS, McKean JD, Griffith RW, Wesley IV, Rostagno MH. *Salmonella* enterica infections in market swine with and without transport and holding. *Appl Environ Microbiol.* 2002;68:2376-81.
- (40) Hurd HS, McKean JD, Wesley IV, Karriker LA. The effect of lairage on *Salmonella* isolation from market swine. *J Food Prot.* 2001;64:939-44.
- (41) Ingham SC, Fanslau MA, Burnham GM, Ingham BH, Norback JP, Schaffner DW. Predicting pathogen growth during short-term temperature abuse of raw pork, beef, and poultry products: use of an isothermal-based predictive tool. *J Food Prot.* 2007;70:1446-56.
- (42) Juneja VK, Eblen BS. Heat inactivation of *Salmonella* typhimurium DT104 in beef as affected by fat content. *Lett Appl Microbiol.* 2000;30:461-7.
- (43) Kelley KW, Curtis SE, Marzan GT, Karara HM, Anderson CR. Body Surface Area of Female Swine. *Journal of Animal Science.* 1973;36:927-30.
- (44) Koidis P, Doyle MP. Survival of *Campylobacter jejuni* in the presence of bisulfite and different atmospheres. *Eur J Clin Microbiol.* 1983;2:384-8.
- (45) Korsak N, Jacob B, Groven B, Etienne G, China B, Ghafir Y et al. *Salmonella* contamination of pigs and pork in an integrated pig production system. *J Food Prot.* 2003;66:1126-33.
- (46) Larsen ST, Hurd HS, McKean JD, Griffith RW, Wesley IV. Effect of short-term lairage on the prevalence of *Salmonella* enterica in cull sows. *J Food Prot.* 2004;67:1489-93.

- (47) Larsen ST, Hurd HS, McKean JD, Griffith RW, Wesley IV. Effect of short-term lairage on the prevalence of *Salmonella enterica* in cull sows. *J Food Prot.* 2004;67:1489-93.
- (48) Latimer HK, Jaykus LA, Morales RA, Cowen P, Crawford-Brown D. A weighted composite dose-response model for human salmonellosis. *Risk Anal.* 2001;21:295-305.
- (49) MacDonald, J. M., Ollinger, M. E., Nelson, K. E., and Handy, C. R. Consolidation in U.S. Meatpacking. *Agricultural Economic Report No. 785.* 2000. Washington, D.C.
- (50) McDowell SW, Porter R, Madden R, Cooper B, Neill SD. *Salmonella* in slaughter pigs in Northern Ireland: prevalence and use of statistical modelling to investigate sample and abattoir effects. *Int J Food Microbiol.* 2007;118:116-25.
- (51) McMeekin TA, Olley JN, Ross T, Ratkowsky DA. *Predictive Microbiology: theory and application.* Taunton, Somerset, England: Research Studies Press, Ltd.; 1993.
- (52) Mead PS, Slutsker L, Dietz V, McCaig LF, Bresee JS, Shapiro C et al. Food-related illness and death in the United States. *Emerg Infect Dis.* 1999;5:607-25.
- (53) Miller GY, Liu X, McNamara PE, Barber DA. Influence of *Salmonella* in pigs preharvest and during pork processing on human health costs and risks from pork. *J Food Prot.* 2005;68:1788-98.
- (54) Miller MF, Carr MA, Bawcom DB, Ramsey CB, Thompson LD. Microbiology of Pork Carcasses from Pigs with Differing Origins and Feed Withdrawal Times. *Journal of Food Protection.* 1997;60:242-45.
- (55) Mulder RWA, Dorresteyn LWJ, Van Der Broek J. Cross-Contamination During the Scalding and Plucking of Broilers. *British Poultry Science.* 1978;19:61-70.
- (56) National Pork Board. Quick Facts: The Pork Industry at a Glance. 2007. 2008.
- (57) National Pork Board. Consumer Eating Trends. 2008.
- (58) Oscar TP et al. Response surface models for effects of temperature and previous temperature on lag time and specific growth rate of *Salmonella* Typhimurium on cooked ground chicken breast. *Journal of food protection.* 1999;62:1111-14.
- (59) Pearce RA, Bolton DJ, Sheridan JJ, McDowell DA, Blair IS, Harrington D. Studies to determine the critical control points in pork slaughter hazard analysis and critical control point systems. *Int J Food Microbiol.* 2004;90:331-9.
- (60) Pearce RA, Bolton DJ, Sheridan JJ, McDowell DA, Blair IS, Harrington D. Studies to determine the critical control points in pork slaughter hazard analysis and critical control point systems. *Int J Food Microbiol.* 2004;90:331-9.
- (61) Placeholder. Placeholder- see note.
- (62) Rahkio M, Korkeala H. Effect of Pre-scalding Brushing on Contamination Level of Pork Carcasses during the Slaughtering Process. *Meat Science.* 1992;32:173-83.
- (63) Ratkowsky DA, Olley J, McMeekin TA, Ball A. Relationship between temperature and growth

rate of bacterial cultures. *J Bacteriol.* 1982;149:1-5.

- (64) Rivas T, Vizcaino JA, Herrera FJ. Microbial contamination of carcasses and equipment from an Iberian pig slaughterhouse. *J Food Prot.* 2000;63:1670-5.
- (65) Saide-Albornoz JJ, Knipe CL, Murano EA, Beran GW. Contamination of Pork Carcasses during Slaughter, Fabrication, and Chilled Storage. *Journal of Food Protection.* 1995;58:993-97.
- (66) Sanchez J, Dohoo IR, Christensen J, Rajic A. Factors influencing the prevalence of *Salmonella* spp. in swine farms: a meta-analysis approach. *Prev Vet Med.* 2007;81:148-77.
- (67) Schlosser W, Hogue A, Ebel E, Rose B, Umholtz R, Ferris K et al. Analysis of *Salmonella* serotypes from selected carcasses and raw ground products sampled prior to implementation of the Pathogen Reduction; Hazard Analysis and Critical Control Point Final Rule in the US. *Int J Food Microbiol.* 2000; 58:107-11.
- (68) Swanenburg M, Urlings HA, Snijders JM, Keuzenkamp DA, van Knapen F. *Salmonella* in slaughter pigs: prevalence, serotypes and critical control points during slaughter in two slaughterhouses. *Int J Food Microbiol.* 2001;70:243-54.
- (69) Tamplin ML, Feder I, Palumbo SA, Oser A, Yoder L, Luchansky JB. *Salmonella* spp. and *Escherichia coli* biotype I on swine carcasses processed under the hazard analysis and critical control point-based inspection models project. *J Food Prot.* 2001;64:1305-8.
- (70) Taormina PJ, Dorsa WJ. Evaluation of hot-water and sanitizer dip treatments of knives contaminated with bacteria and meat residue. *J Food Prot.* 2007;70:648-54.
- (71) USDA. Swine 2006, Part I: Reference of Swine Health and Management Practices in the United States, 2006 . 2007.
- (72) van Netten P, Mossel DA, Huis In 't Veld J. Lactic acid decontamination of fresh pork carcasses: a pilot plant study. *Int J Food Microbiol.* 1995;25:1-9.
- (73) Warriner K, Aldsworth TG, Kaur S, Dodd CE. Cross-contamination of carcasses and equipment during pork processing. *J Appl Microbiol.* 2002;93:169-77.
- (74) WHO/FAO. Risk assessments of *Salmonella* in eggs and broiler chickens. Geneva, Switzerland: 2002.
- (75) Wray, C. Review of Research into *Salmonella* Infections in Pigs. 2001. Meat and Livestock Commission.