The influence of *Salmonella* in pigs pre-harvest on *Salmonella* human health costs and risk from pork

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Abstract

Salmonellosis in people is a costly disease, much of it occurring because of food associated exposure. We develop a farm-to-fork model which estimates the pork associated *Salmonella* risk and human health costs. This analysis focuses on the components of the pork production chain up to the point of producing a chilled pork carcass. Sensitivity and scenario analysis show that changes that occur in *Salmonella* status during processing are substantially more important for human health risk and have a higher benefit/cost ratio for application of strategies that control *Salmonella* compared with on-farm strategies.

Keywords: swine, risk assessment, *Salmonella*, pre-harvest, food safety, human health costs

INTRODUCTION

Foodborne disease has drawn increasing attention in the U.S. and *Salmonella* are one of the major foodborne pathogens. Meade et al. (1999) reports that there are more than 38 million cases of illness caused by known foodborne pathogens annually; among these cases, about 14 million are considered to actually be foodborne. They report also that more than 95% percent of non-typhi salmonellosis is attributable to food. Studies from Europe suggest that 90% of human salmonellosis could be attributed to food (Anonymous, 2001). *Salmonella* are more virulent than many pathogens that can cause foodborne illness with relatively high hospitalization and death rankings for non-typhi *Salmonella* compared with other pathogens.

Salmonella contamination on pork carcasses has been documented. Rigney et al. (2004) found that 7.0% of 8,483 cooled market hog carcasses sampled between January 1998 and December 2000 yielded *Salmonella* spp. It has been estimated that 5-25% of the foodborne salmonellosis is from pork (Berends et al., 1998).

Salmonellosis attributable to meat and meat products leads to societal costs (Buzby, et al., 1996). While these societal costs may have declined given what appears may be a gradual reduction of *Salmonella* contamination seen recently in pork carcasses (FSIS, 2003) possibly due to HACCP, *Salmonella* are still seen as a major cost estimated to be $3B for the U.S. (USDA, ERS. 2003). McNamara et al. (2004) estimate the cost of pork associated salmonellosis to be $81.5 million. The magnitude of this estimated burden of disease raises the issue of effective solutions and the need for economic evaluations of methods to decrease this social cost.

The paper evaluates the economic impacts that can be attributed to the prevalence of *Salmonella* pre-harvest in the pork chain, and examines the impact and economic effectiveness that some corresponding mitigation strategies have on the social costs of illness. Specifically, this study investigates the influence on human health risks and social costs of three stages in the pork chain: prevalence at the farm, transportation to the slaughter plant and lairage (holding/resting) at the slaughter plant, and the processing to the point of a cooled pork carcass. We discuss possible mitigation strategies at the farm level and at slaughter/processing. We compare the cost-effectiveness for controlling

Salmonella between these two stages in pork production and conduct sensitivity analyses to assess the corresponding impacts on human health, *Salmonella* risks and societal costs of these risks associated with U.S. pork production.

RELATED LITERATURE

A large literature has emerged in the past decade of factors at the pre-harvest stage of pork production that may contribute to pig prevalence of *Salmonella*. Dickson et al. reviewed the studies on *Salmonella* shedding in pork production focusing generally on U.S. studies. A similar review (Wong and Hald, 2000) with much attention to European practices and studies, investigate pre-harvest and post-harvest control options based on epidemiological, diagnostic and economic research. These two reviews, provide an outline of studies and production practices used in developed countries, and suggest that there is a general consensus that the pre-harvest links in the pork supply chain are an important part of the overall pork associated risks for *Salmonella*. Indeed some studies show a decrease in *Salmonella* prevalence resulting from specific practices, although there seems to be some inconsistencies in the various findings (Wong et al., 2004 ; Kolb et al., 2003; Jensen et al., 2000; Bahnson 2001; Kjarsgaard et al. 2001).

In general, previous studies are of two types. One type is based on specific experiments and is generally focused on demonstrating degree of contamination at specific points or stages of the pork supply chain. This represents the majority of the relevant literature. The second type of study attempts to synthesize data across the entire pork supply chain and examines many stages of the pork chain, modeling the introduction and spread of *Salmonella* in multiple stages. Roberts et al. lay the theoretical foundation for the latter type of study. They emphasize the need to dissect the complex series of events from farm to table into smaller, more manageable chunks and stress the necessity of reflecting the uncertainty and correlation among links in the modeled food chain. They suggest two interrelated approaches: Probabilistic Scenario Analysis and Fault Tree Analysis, to model pathogen disseminations in meat supply chains. Van der Gaag (2003) used a state transition simulation model for the spread of *Salmonella* in the pork supply chain. Their study used transition probabilities that had time dependence to model

Salmonella spread and Monte Carlo simulation to capture the status of individual animals at varying stages under transition probabilities that vary with different strategies or control measures. This approach recognizes implicitly that it is infeasible to conduct real experimentation of the integration of the entire pork chain, but use methods to amalgamate the results of multiple studies.

The prevalence and shedding of *Salmonella* of grow/finish pigs is related to many factors. Influencing factors include contaminated feeds, the use of pelleted feeds, antibiotic use, vaccination, and bio-security measures, to list a few.

Examination of the influence of feeds on *Salmonella* shedding demonstrated that feed can itself be contaminated. Harris et al. (1997) found a *Salmonella* prevalence of 2.9% in feeds and feed ingredients taken from farm environments. McChesney et al. (1995) cited an FDA survey of animal and plant protein processors demonstrating that 57.4% of the animal protein and 36% of the vegetable protein products taken from 124 processors were positive for *Salmonella*. Thus feeds are a clear source of possible *Salmonella* exposure for swine herds. Also, the physical structure of the feed as well as feeding methods influenced *Salmonella* prevalence in pigs. Jorgensen *et al.* (1999) compared the effect of feeding pellets and meal on the prevalence of *Salmonella* in pigs; they found that meal reduced the risk but also reduced the feed conversion of the pigs. When meal was finely ground, the *Salmonella* risk was three times greater than for coarsely ground feed. Hansen *et al*. (2001a) found that dried sugar beet pulp reduced *Salmonella* prevalence significantly and did not affect productivity. Dahl *et al.* (1999) found that adding non-heat treated wheat or barley to pelleted feed reduced the prevalence of *Salmonella* shedding. Van der Wolf *et al.* (1999c) used a mixture of acids including formic and lactic acid and found 17.8% positive pigs among those receiving treatment as compared with 24.7% of the controls. Dahl (1998); van der Wolf *et al.* *(*1998) have shown that liquid feed, especially fermented liquid feed, reduced the risk of *Salmonella* infection in pigs. Perhaps the most definitive study is one published by Wong et al (2004) who use a logistic regression model to evaluate multiple risk factors for *Salmonella*, and demonstrated a substantial decrease in *Salmonella* prevalence for pigs fed meal compared with those fed pelleted diets.

Vaccination is another factor that has significant impacts on *Salmonella* shedding of swine herds. Theoretically, vaccination can be given to pigs of any stage including either nursery or grow/finish pigs, but use is often discussed for breeding animals, especially sows, because of economic considerations. Ghosh (1972), Letellier et al. (1999) and Davies et al (2000) reveal *Salmonella* carriers have frequently been identified in breeding sows, and breeding animals are one of the sources for the introduction and dissemination of *Salmonella* in grow/finish herds. Davies and Wray (1997) showed that vaccination of breeding stock on a farm with an inactivated *S.* Typhimurium/ *S.* Dublin vaccine was associated with a reduction of *Salmonella* from 67% to12% in weaned pigs and from 52% to 5% in adults pigs. In Denmark, Dahl et al. (1997) demonstrated that the use of killed vaccines reduced the clinical impact of *S.* Typhimurium infection in pigs but did not reduce subclinical infection. Lumsden et al. (1991), using a mutant of *S.* typhimurium, found that vaccinated pigs shed *Salmonella* significantly less frequently than nonvaccinated pigs. Kolb et al. (2003) found a decrease of 50-73% in *Salmonella* prevalence in vaccinated hogs compared with controls.

Subtherapeutic antibiotic use has been found to influence *Salmonella* shedding on-farm. Shryock et al. (1998) found tylosin in feeds (100g/ton) reduced the duration of *S.* typhimurium shedding in the feces. However, they found no effect on *Salmonella* prevalence or the quantity of *Salmonella* in the feces. Girard et al. (1976) noticed subtherapeutic levels of oxytetracycline plus neomycin in animal feeds did not increase the number of organisms of *S.* typhimurium in swine, but did tend to reduce the proportion of animals carrying *S*. typhimurium. Evangelisti et al. (1975) evaluated subtherapeutic oxytetracycline in animal feeds to determine their influence on the relative quantity, prevalence, shedding and antibiotic susceptibility of *S.* typhimurium in swine, calves and chickens, when compared with non-medicated controls. Antibiotics were not associated with increased quantity, prevalence, or shedding of *S.* typhimurium in all three animal species as evidenced by colony counts in feces measured on seven separate occasions over a 28 day observation period. Ebner et al. (2000) found the incidence of shedding was reduced in pigs receiving a combined apramycin, oxytetracycline treatment, when compared to control pigs; however, no differences were observed between antibiotic treatments. Thus, while antibiotic feeding may have some degree of

influence, the data are generally obtained from small numbers of experimental animals. Also, a report prepared for the Center for Veterinary Medicine and published on their websight (CVM, 2000) concluded that experimental studies of this sort were not generally useful in evaluating shedding outcomes with respect to behavior in the field and conclude the results from such studies generally do not support the hypothesis that antibiotics added to animal feed substantially affect pathogen load. So the influence of antimicrobial feeding is not sufficiently compelling to warrant further consideration in our analyses.

When considering biosecurity, the number of potential sources of *Salmonella* infection is large. Pests (rodents, wild birds and other wildlife species) have often been implicated as potential sources of *Salmonella* for swine. Davies and Wray (1997) found a wide range of animals, including rats, mice, cats, rodents and birds, carry *Salmonella* and were involved in herd infection. Disinfection of the environment and exclusion of those animal pests should significantly lower *Salmonella* environmental contamination and incidence/prevalence in pigs. Linton et al. (1970) found that uninfected animals, which remained in disinfected pens usually stayed free of *Salmonella* but as the number of pigs per pen increased a higher prevalence of infection was found. Tielen et al. (1997) found that *Salmonella* negative piglets placed in clean accommodations remained free despite serological evidence of *Salmonella* in the sows. Dahl et al. (1997) studied cases in Denmark and found that removal of 10 week old pigs from breeding farms infected with *S.* typhimurium and placement in clean premises appeared to be effective in prevention of infection at market age. Fedorka-Cray et al. (1997) studied pigs weaned at 14-21 days and removed to clean accommodation; piglets remained free of *Salmonella.* Davies and Wray (1997) showed improved disinfections of pens on several farms produced significant reductions in the incidence of positive batches. Proescholdt et al. (1999) compared continuous flow (CF) systems and all-in-all-out (AIAO) systems in the U.S. and found little difference between the 2 systems when tissues collected in the abattoir were examined for *Salmonella.*

Although biosecurity is important in controlling *Salmonella*, it is also quite complex and may be more difficult to quantitatively analyze the impact of each specific biosecurity factor on the overall risk. The resulting influence may well be attributed to

the integration of many factors correlated with each other as a set of umbrella biosecurity measures to be used simultaneously. *Salmonella* may persist in the environment for long periods, and cleaning and disinfection procedures may not be efficient in eliminating environmental contamination (Schwartz (1999). *Salmonella* are hardy, surviving freezing and desiccation, and persisting for years in suitable substrates. Gray and Fedorka-Gray (1995) found that *S.* choleraesuis survives in dry feces for at least thirty months post-shedding. Gebreyes et al. (1999) detected *Salmonella* in drag swabs of floors from barns after cleaning and disinfections, and before pig placement in 82% of the studied cases. Such results suggest that controlling *Salmonella* with biosecurity measures will have a wide range of effectiveness, and poorly predictable outcomes. Thus, biosecurity is not pursued further in the analyses presented here either.

MATERIALS AND METHODS

@Risk Model details.

Following the theoretical approach suggested by Roberts et al. (1995), we have developed an integrated model which uses @Risk and estimates the risk and economic impact of *Salmonella* in humans derived from pork. This model consists of seven modules of the farm-to-fork pork chain in the U.S. These modules estimate pig/carcass prevalence, degree of contamination 1) on-farm pig prevalence (apparent and after adjustment for test sensitivity and specificity), 2) pig prevalence after transport and lairage, 3) carcass prevalence during processing to the point post-carcass chilling, 4) prevalence at fabrication and retail, 5) affects of consumer cooking and consumption, 6) *Salmonella* health consequences, and 7) social costs. We use parameter estimates and data from the scientific literature throughout the model. The model works on a flow basis, with output estimates from one segment of the model generally serving as input estimates for the next segment, and assumes that all of the contamination found at the various

stages originated with the pig on-farm. When possible and appropriate, we treat inputs as distributions rather than point estimates.

The mean prevalence of *Salmonella* in pigs at the farm is assumed to be 0.06 (USDA, APHIS VS CEAH CAHM (1997)); we assume a pert distribution with a mode, and upper bound of 0.06 (Table 1). Adjustments for apparent prevalence were made to derive true prevalence (assumed triangular distribution) using assumed test sensitivity (with a range of 0.325-0.688) and specificity (0.998) (Funk et al., 2000). There are substantial increases (from 2.96-6.84-fold increase) seen in prevalence from the combined effects of transportation and lairage (Hurd et al., 2001; McKean et al., 2001; Proescholdt et al., 1999). We use an average of these studies to model the increase in prevalence as a triangular distribution for market hogs (1.96, 3.9, 5.84), and sows (2.17, 5.0, 7.83).

Between the time of killing the pig and having a cooled pork carcass, there are many considerations. There is the relationship between fecal positive and the degree of contamination that occurs on the carcass; Morgan et al. (1987) show a two fold drop in fecal positive rate compared to carcass positive rate. There is the potential for cross carcass contamination that can occur as the carcass progresses thru various steps in processing (Dickson at al., 2003). Additionally, a large number of particular steps do or can occur in the processing of pork carcasses including scalding, dehairing/polishing, singeing, carcass washing, evisceration, carcass rinse post-evisceration, steam pasteurization, and chilling. Many of these steps are specifically put in place to decrease the degree of carcass contamination. These relationships and steps with the associated effects are not modeled individually. Instead, prevalence results derived from the model

are adjusted downward by the proportional decrease needed to produce carcass prevalence post-chilling suggested by USDA (USDA, 1996, and 2003); this resulted in applying a triangular (0.87, 0.91, 0.96) distributional decrease in prevalence occurring during processing.

Increases in prevalence of *Salmonella* during fabrication and at the retail level are assumed based on data from Duffy et al. (2001). The degree of contamination is also affected by cooking by the consumer. Cooking effects and food handling (Woodburn and Raab, 1997; Ralston et al, 2002) are assumed to protect against exposure differently between two distinctive risk groups. The pork consuming public is divided into low and high risk groups (Table 2). The high risk group is assumed to be 20% of the U.S. pork consuming public, and the low risk group, 80% (Gerba et al., 1996; USDA 1998). High risk individuals would include the elderly, children and immunocompromised individuals. Not all consumers eat pork (Miller and Unnevehr, 2001); 7.6 % of the population is assumed not to eat pork.

Human risks and the associated health costs which can be attributed to pork are estimated using literature that documents risks and costs from *Salmonella* infection (Table 2). Specifically, the dose response model outlined by WHO (2002) was used. We used a beta-Poisson function with $\alpha = 0.1324$, and $\beta = 51.45$, with an associated distribution around the curve, and α and β were assumed the same for low and high risk groups (WHO, 2002). Costs for human *Salmonella* cases were assumed to be $482.26, $1032.12, $11,812.19, and $500,923.23 for no visit to a physician, physician visit, hospitalization, and death respectively (Buzby, 1996). We assumed no development of immunity; so exposure by one person 10 times to contaminated pork results in the same

number of cases as exposure by 10 people one time each to contaminated pork. Further elaboration of the later stages of this model is detailed in McNamara et al. (2004) where sensitivity analyses are shown on the effects seen after carcass chilling, thus at retail, cooking and dose-response for the amount of pathogen intake required for human illness.

Sensitivity analyses for the base model. The relative importance of the various elements in the model up to the point of a chilled pork carcass was evaluated using sensitivity analyses. Sensitivity analyses were conducted for on-farm pig apparent prevalence, prevalence increases that occur in transportation and in lairage, and prevalence decreases that occur during slaughter to the point of producing the chilled pork carcass.

Scenarios comparing on-farm with processing control. We evaluate the relative merit of controlling *Salmonella* on-farm vs in the slaughter processing plant by conducting several scenario analyses. One analysis considers the value of vaccination of pigs on-farm. We assume that the cost of the vaccine per pig is $0.852 including product cost, cooler required for shipment and 2nd day air postage expense (Livestock Concepts, Inc.). The total cost of vaccination is then just the cost per pig multiplied by the number of U.S. swine slaughtered each year. The vaccination effect assumed is to decrease prevalence in pigs on-farm by 50% to 73% (Kolb et al., 2003). The benefit calculation is the social costs baseline (no vaccination assumed) minus the social costs if farm prevalence decreases by a uniform distribution of 50-73% (from vaccination). We assume in the baseline model that there is no vaccination of pigs, while the scenario evaluated assumes 100% of swine receive vaccination.

A second on-farm analysis considers the relative value of two basic feedstuffs (meal feeding vs. pelleted feeding). Pelleted feeding has been shown to increase the amount of *Salmonella* in pigs (Wong, LF et al., 2004). The offsetting effect is that pigs fed pelleted feeds have improved productivity and efficiency (Wondra et al,., 1995). We assume the feed conversion ratio (pounds of feed fed to pounds gained by the pig; FCR) of pigs is 2.98, and the average daily gain (pounds gained per pig per day; ADG) is 1.67 (NAHMS 2000 swine survey; Miller et al., 2004). Pigs fed pellets have improved FCR by 7% and improved ADG by 5% (Wondra et al., 1995). Pelleted feed costs $7 more per ton of feed (Harper, 1998). Combined with a simple budgeting method we have used previously to describe the economic impact for a synthetic average U.S. swine farm (Miller et al, 2003), we estimate the economic advantage of pelleted feeding for producers and compare this to the social costs from using pelleted feeds. We assume in the baseline model that all pigs are fed pelleted feed, while the scenario evaluated assumes 100% of swine are fed meal feed.

A third analysis compares the costs and reduction in human cases for the two on-farm scenarios to results predicted by the model from an enhanced carcass rinsing procedure used in slaughter/processing plants. Jensen and Unnevehr, 2000, evaluate the cost-effectiveness of different technologies for pathogen reduction in slaughter/processing plants. They show carcass rinsing with and without sanitizers and applied at various temperatures resulted in varying degrees of reduction in carcass bacterial contamination depending on the specific rinse details. They also calculated the costs per carcass for applying each rinse type. We use a reduction in bacterial contamination (19% to 61%) implied by Jensen and Unnevehr assuming the baseline

technology would be rinsing with plain water (at 25C) to infer the degree of carcass contamination post-chilling. The costs per carcass for applying the different strategies ranged from $0.2659 to as high as $0.19658. The number of cases of human illness controlled by each strategy is then estimated and compared.

Lastly, we estimate the benefit cost/ratios of the on-farm strategies of vaccination and feedtype and the slaughter plant strategy of various carcass rinses.

RESULTS AND DISCUSSION

Baseline Model Results. Our model depends on many different inputs and modifications to these inputs. Figure 1 shows some of the intermediate results from the model including as examples, market hog *Salmonella* on-farm true prevalence, market hog prevalence after transportation and lairage, and finally at the point post-carcass chilling. The model predicts human *Salmonella* cases that are pork associated (mean = 99,430 (20,970 - 245,560 (90% confidence internal; figure 2))) and the associated social costs (mean = $81 Million ($18.8 Million - $197.4 Million (90% confidence internal; figure 1))). There is a broad confidence internal for both human pork-associated salmonellosis cases and the social costs. This confidence interval is large because of the degree of uncertainty on the various parameter estimates that influence the final model results. The pork supply chain incorporates a large degree of complexity at multiple stages even in the extreme simplicity of this model compared with the real world details. Nonetheless, our results suggest that the costs from pork-associated salmonellosis are a very small fraction (less than 3% (81/3,000) of the total costs of human salmonellosis. Berends et al. (1998) suggest that 15% of all cases of salmonellosis in The Netherlands are associated with pork consumption.

Sensitivity Analysis. The sensitivity results presented focus on that portion of the model which leads to a chilled pork carcass. By far, the most important element influencing the model results is the decline in carcass prevalence that occurs during slaughter and processing (Table 3). Here a 10% increase in reduction of prevalence during slaughter and processing decreases the number of human cases by approximately 75% (number of pork-associated cases falls from approximately 100,000 to about 25,000). A similar decline in associated social costs is also seen. Decreasing on-farm *Salmonella* prevalence for market hogs or sows has a much smaller affect than a similar change in prevalence during slaughter and processing (decreasing the number of human cases from 2.3% to 7.6%, respectively).

A 10% change in on-farm prevalence for market hogs has a much larger influence on human salmonellosis than a similar change for sows (Table 3). This is to be expected given that the number of market hogs slaughtered in the U.S. is roughly 31 times the number of sows slaughtered. So even though the assumed on-farm prevalence for sows is higher, and the weight of sows slaughtered is higher, market hog prevalence changes are still considerably more important than changes in sow prevalence. Also, given that sows are processed differently (assumed entire carcass is ground, and that ground product has increased risk for *Salmonella* (Table 2)), there was some expectation that the influence from sow prevalence might be more substantial than the model demonstrates. Our results suggest that the most important place to conduct further on-farm research, at least for the purpose of decreasing overall social costs, would be control of *Salmonella* in market hogs.

The influence of transportation and lairage changes on human salmonellosis has about the same affect (although somewhat less; Table 3) as changes in on-farm market hog prevalence. This is to be expected given that the market hog prevalence serves as an input to the changes that occur in transportation and lairage. So a 10% increase in market hog prevalence on the farm should result in approximately the same affect as a 10% increase in prevalence from transportation and lairage since all subsequent effects in the model are then the same. If this is indeed a reflection of real world affects, then our results suggest that there is comparable value to be gained from research in influences on prevalence on-farm compared to influences on the increase in prevalence due to transportation and lairage. But the costs of such research may be different and also the ability to control risk factors in these two segments of the pork-chain may well be different. Producers have more control over production practices and have much less control over marketing influences like distance/time traveled to market or time animals spend on the truck and in lairage after arriving at the slaughter plant. Similarly, slaughter plants have much less control over production practices and more control over marketing distances (e.g. decisions related to plant locations distributed around the U.S.), the coordination for truck arrival and unloading, and the amount of time animals spend in lairage at the plant prior to slaughter. Additionally, there are other considerations relating to meat quality that encourage packers to have a minimum holding time of at least a few hours for pigs after arrival at the slaughter plant (Dickson et al (2002), Hurd et al (2002)). Such considerations should influence future decisions about funding allocations for on-farm vs transportation and lairage research.

Relative merit of controlling *Salmonella* on-farm compared with control at processing. To understand the relative merit of on-farm control compared with processing we use Tables 4 and 5. Vaccination has the potential to decrease human salmonellosis costs and cases by over 70% (Table 4). However, the benefit/cost ratio from a social perspective (benefits obtained socially from decreased human cases, compared with costs incurred by swine producers to use the vaccine) is less than one (Table 5). We assumed that no animals were vaccinated in the baseline case, and that 100% of animals (both market hogs and sows) were vaccinated in the scenario. However, it is very likely that at least some (currently unknown) portion of animals were vaccinated for *Salmonella*. Whatever the proportion of vaccination that occurred during the late 90's, the baseline estimates for social cost already incorporates this effect. The social costs if all pigs were vaccinated will be more than assumed. So the numerator (of the benefit/cost ratio) would decline. Also, the denominator (cost of the strategy) would need a similar adjustment and would decline. While it seems likely that the benefit/cost ratio will not change substantially (certainly not by an order of magnitude), the exact influence is uncertain. Further data are needed to know more definitely the benefit/cost ratio from this strategy.

We see similar large potential gains possibly achievable from meal feeding which might decrease human salmonellosis cases by over 60% (Table 4). But here, the benefit/cost ratio from a social perspective is even worse than for vaccination being 0.09 (i.e. approaching zero). Additionally, we know that some proportion of the U.S. swine herd does already use meal feeding. Therefore the effect from switching to meal feeding may similarly overstate the potential benefit. We assumed that no animals were receiving

meal feed in the baseline case, and that 100% of animals were fed meal feed in the scenario analysis. This calculation has similar difficulties as those mentioned for vaccination, and further data are needed to understand the proportion of hogs already receiving meal feed compared to pellets. Again, it seems likely that the benefit/cost ratio will not change substantially, the exact influence is uncertain. Further data are needed to know more definitely the benefit/cost ratio from this strategy. However, it seems likely that meal feeding of pigs to control human salmonellosis is an even lower ranked strategy from a social benefit-cost perspective.

In contrast, we see the strategies employed at processing which uses carcass rinsing at varying water temperatures (both with and without sanitizer) generally not only lower costs and cases from *Salmonella* for humans (Table 4), but also have benefit/cost ratios greater than one (Table 5). The strategies which only employ increasing the temperature of the carcass rinse water, actually have higher benefit/cost ratios than those which increase the temperature and also add a sanitizer. Further information data on the amount of various rinsing strategies currently employed would similarly enhance these results.

There is intuitive appeal to these results generally. The closer to the consumer that a control strategy can be employed, the more likely there will be a direct and major influence on human salmonellosis. Also, the strategies used in plants have much lower per carcass/pig costs (costs for rinses were all under $0.20 per carcass) than on-farm strategies (costs for vaccination were about $0.85 per hog and costs for feedstuff changes were $5.79 per hog). So the per carcass and overall social costs are lower for processing resulting in this be a lower cost and more efficient place to apply control. Additionally,

the processor has a more direct ability to channel pork which has been handled to enhance pork safety to niche markets (e.g. hospitals or specific product labels) where they might charge sufficiently more to cover the costs for producing this enhanced safety product. A producer has little opportunity to do this. The social benefit/cost ratio doesn't suggest this is a reasonable strategy in any case.

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Table 1. *Constants and parameter inputs employed in the farm-to-fork model*

Constant/ parameter inputs	Value	*Sources*
Number of sows	3,005,400	USDA, NASS (2001)
Number of gilts/barrows	93,114,900	USDA, NASS (2001)
Lower bound of sensitivity of fecal sample	0.326	Funk et al. (2000)
Upper bound of sensitivity of fecal sample	0.688	Funk et al. (2000)
Specificity of fecal samples	0.998	Baggesen et al. (1996)
Carcass weight of sows	309	USDA, NASS (2001)
Carcass weight of gilts and barrows	191	USDA, NASS (2001)
Ratio of pork to carcass	0.76	Hog carcass breakdown http://www.tysonfoodsinc.com
Ratio of trimmings to pork	0.13	Hog carcass breakdown http://www.tysonfoodsinc.com
Portion of sow pork going to ground pork	1	Assumed
Survival rate of Salmonella organisms for low risk population	0.00000010	USDA , FSIS (1998)
Survival rate of Salmonella organism for high risk population	0.00000100	USDA , FSIS (1998)
Total population in the U.S.A	287,151,740	US census bureau (2000)
High risk population in the U.S.A	57,430,348	Gerba, C.P. et al. (1996)
Low risk population in the U.S.A	229,721,392	Gerba, C.P. et al. (1996)
Treatment costs of salmonellosis with no physican visits ($)	374	Buzby et al. (1996)
Costs of salmonellosis with physican visit ($)	794	Buzby et al. (1996)
Costs of hospitalized patients ($)	9,087	Buzby et al. (1996)
Costs of death ($)	385,355	Buzby et al. (1996)
Costs adjusted by price index of 2000	260.8/201.4	http://www.bls.gov/cpi/home.htm

Table 2. *Distributional assumptions employed in the farm-to-fork model*

Name	Distribution	Parameter / value	Sources
Apparent prevalence on the farm (sows)	Pert	Pert(0.039,0.078,0.078)	USDA (1997), Davies et al. (1998)
Apparent prevalence on the farm gilts and barrows (G/B)	Pert	Pert (0.03,0.06,0.06)	USDA (1997); Proesholdt (1999)
Prevalence on the farm (sows)	Triangular	Triang (0.05,0.24,0.24)	Funk et al. (2001); Smith(1995)
Prevalence on the farm gilts and barrows (G/B)	Triangular	Triang (0.04,0.18,0.18)	Funk et al. (2001); Smith(1995)
Prevalence increase in transport and lairage (sows)	Triangular	Triang(2.17,5,7.83)	Larsen et al. (2003)
Prevalence increase in transport and lairage (G/B)	Triangular	Triang(1.96, 3.9,5.84)	Hurd et al. (2001); McKean (2001): Proesholdt (1999)
Prevalence reduction in slaughtering (sows)	Triangular	Triang(0.87,0.911,0.96)	USDA (2003) progress report
Prevalence reduction in slaughtering (G/B)	Triangular	Triang(0.87,0.911,0.96)	USDA (2003) progress report
Impacts of fabrication and retail on ground pork	Triangular	Triang(0.073,0.099,0.125)	Duffy et al. (2001)
Impacts of fabrication and retail on pork cuts	Triangular	Triang(0.083, 0.093,0.103)	Duffy et al. (2001)
CFU/ounce in ground pork	Triangular	Triang(15,716,1418)	FSIS (1998) http://www.fsis.usda.gov/haccp/lethality.pdf
Amount of ground pork per serving for low risk population	Normal	Normal(3,0.9 trunc(0.1,6))	USDA (1998)
Amount of ground pork per serving for high risk population	Normal	Normal(1.5,0.6) trunc(0.1,6)	USDA (1998)
CFU/ounce in pork cuts	Triangular	Triang(15, 2828, 5642)	FSIS (1998), Duffy et al. (2001)
Amount of pork cuts per serving for low risk population	Normal	Normal(3,0.9 trunc(0.1,6))	USDA 1994-1996 1998
Amount of pork cuts per serving for high risk population	Normal	Normal(1.5,0.6) trunc(0.1,6)	USDA 1994-1996 1998
Dose-response relationship	Beta-Poisson	Beta-Poisson(51.45,.0.1324)	WHO (2000) http://www.WHO.INT/FSF/Micro/Ra_Salmonella_report.pdf
Exposure adjustment for non-pork-eating group	Normal	Normal(0.924, 0.03, trunc(0,1))	Miller et al. (2001)
Cooking effect for low risk population	Normal	Normal(0.15,0.03,trunc(0,1))	Gerba et al. (1996)
Cooking effect for high risk population	Normal	Normal(0.2, 0.03, trunc(0,1))	Gerba et al. (1996)
No physican visit patients of low risk population	Triangular	Triangular(0.934, 0.95, 0.96)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Physican visit patients of low risk population	Triangular	Triangular(0.0364, 0.048, 0.0629)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Hospitalized patients of low risk population	Triangular	Triangular(0.00204, 0.00349,0.00596)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Death of low risk population	Triangular	Triangular(0.000127, 0.000254, 0.000553)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
No physican visit patients of high risk population	Triangular	Triangular(0.9, 0.93, 0.95)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Physicans visit patients of high risk population	Triangular	Triangular(0.0437, 0.0699, 0.0911)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Hospitalized patients of high risk population	Triangular	Triangular(0.00324, 0.00643, 0.0166)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/
Death of high risk population	Triangular	Triangular(0.000248, 0.000783, 0.00387)	USDA 1998 http://www.fsis.usda.gov/ophs/risk/

Table 3. *Sensitivity analysis for changes in 1) on-farm Salmonella apparent prevalence in market hogs; 2) on-farm Salmonella apparent prevalence in sows; 3) prevalence increases during transportation and lairage; 4) prevalence changes from pre-to-post slaughter*

			Human salmonellosis			Social costs		
Input variable	Change	Hog/carcass prevalence	0.05	Mean	0.95	0.05	Mean	0.95
	-0.1	0.0495	13,300	91,693	244,239	17,543,410	75,310,740	180,204,900
Hog prevalence [a]	0	0.055	14,567	99,212	261,522	19,114,180	81,692,700	195,945,900
	0.1	0.0605	15,915	106,738	282,080	20,705,730	87,658,510	208,979,600
	-0.1	0.0648	14,212	97,146	255,007	18,504,110	79,948,300	191,933,400
Sow prevalence [a]	0	0.072	14,452	99,432	260,841	18,864,420	81,836,200	195,940,000
	0.1	0.0792	14,647	101,085	266,160	19,292,300	83,201,970	199,980,700
Prevalence increase	-0.1	0.54	13,416	92,207	244,491	17,430,990	75,735,560	182,366,600
in transport & lairage	0	0.6	14,731	99,851	264,370	18,860,720	82,015,970	195,770,900
	0.1	0.66	15,816	107,071	283,875	20,283,130	87,927,880	210,508,900
Prevalence reduction	-0.1	0.057	46,218	211,705	512,092	41,164,330	173,773,500	410,114,300
in slaughter	0	0.052	21,170	99,624	245,399	18,731,870	81,805,790	196,397,500
	0.1	0.047	5,580	25,568	63,339	5,037,695	21,135,220	51,394,730

[a] Apparent on-farm prevalence

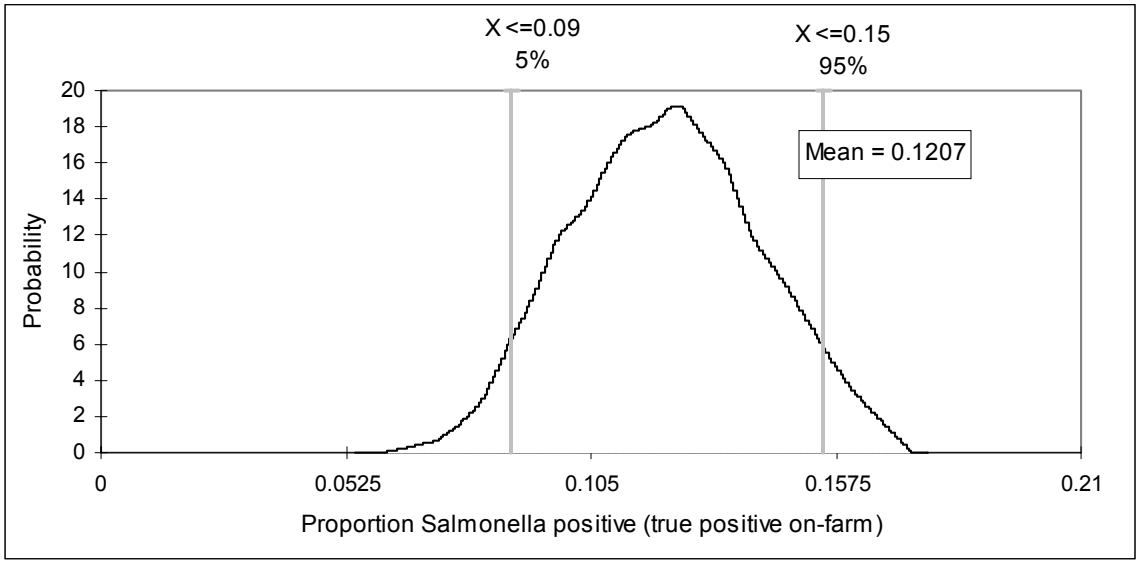
Table 4. *Model results for scenarios*

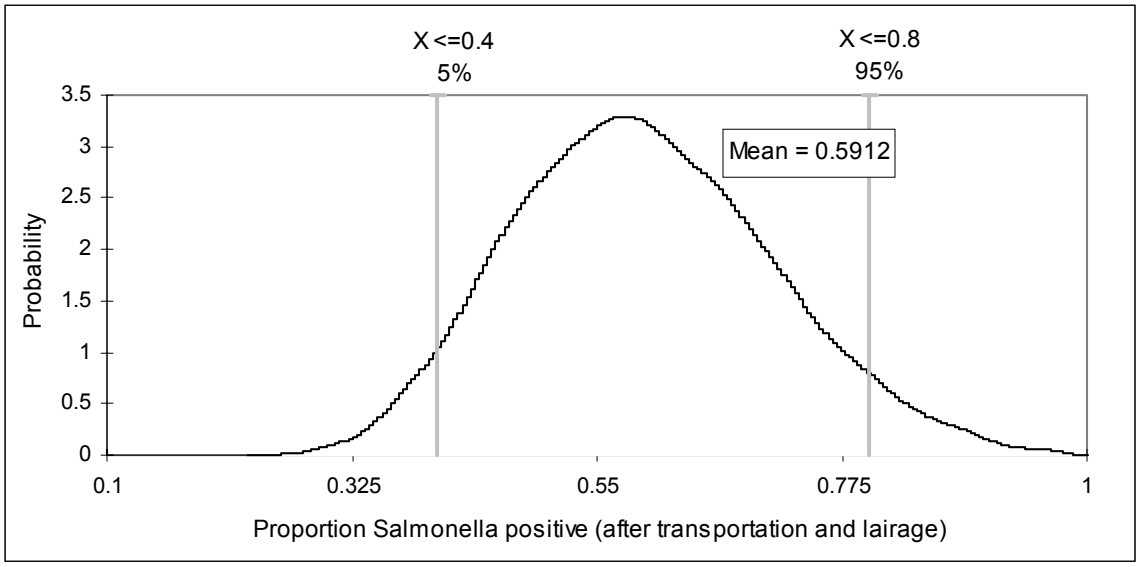
	Human salmonellosis cases			Social costs ($)		
Strategy	5%	Mean	95%	5%	Mean	95%
Baseline	20,967	99,431	245,567	18,758,030	81,528,490	197,445,600
Vaccination	6,199	29,006	75,047	5,461,334	23,845,840	60,057,180
Meal feed	7,848	38,073	95,949	7,106,082	31,348,140	77,583,860
Processing -Rinses						
1 Carcass pasteurization	7,257	34,609	87,790	6,453,287	28,486,540	69,979,420
2. Water rinse (25C) and sanitizer	14,741	70,171	175,585	13,176,230	57,621,210	139,507,000
3. Water rinse (55C)	16,836	79,403	196,402	14,992,200	65,200,560	156,837,200
4. Water rinse (55C) and sanitizer	14,741	70,171	175,585	13,176,230	57,621,210	139,507,000
5. Water rinse (65C)	13,795	65,687	162,882	12,298,550	53,949,230	130,453,700
6. Water rinse (65C) and sanitizer	12,229	58,549	146,690	10,862,760	48,085,550	116,664,700

Table 5. B*enefit cost analyses for scenarios*

Strategy	Social costs ($)	Social Benefits($)	Benefit/cost ratio
Vaccination	81,894,496	57,682,650	0.70
Meal feed	557,445,958	50,180,350	0.09
Processing -Rinses			
1 Carcass pasteurization	14,955,357	53,041,950	3.55
2. Water rinse (25C) and sanitizer	13,511,631	23,907,280	1.77
3. Water rinse (55C)	5,247,207	16,327,930	3.11
4. Water rinse (55C) and sanitizer	16,202,999	23,907,280	1.48
5. Water rinse (65C)	7,939,537	27,579,260	3.47
6. Water rinse (65C) and sanitizer	16,300,080	33,442,940	2.05

Figure 1. *Market hog/carcass prevalence - Model results*





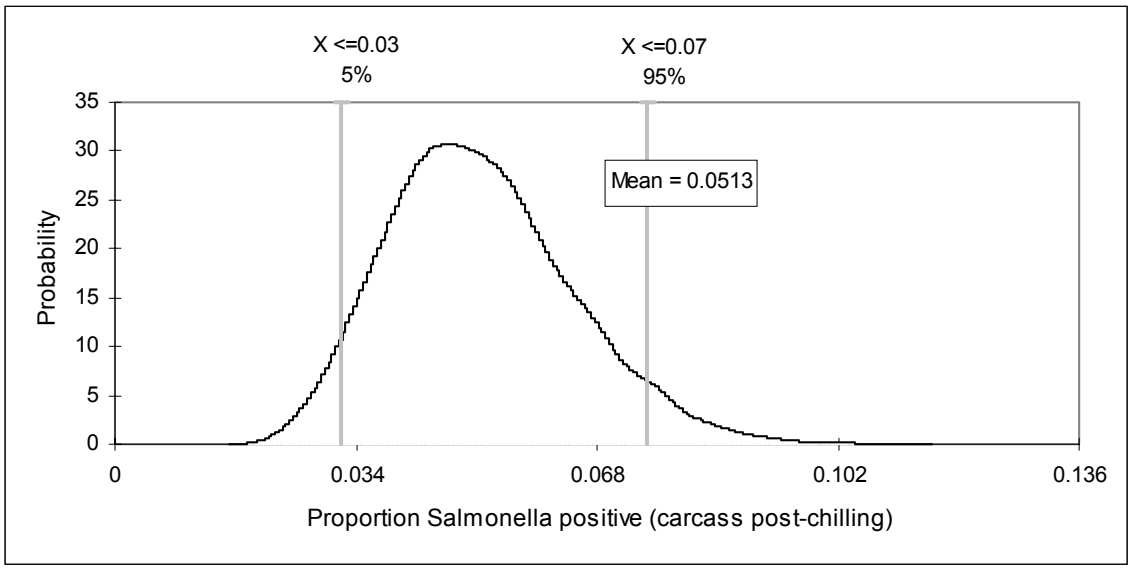
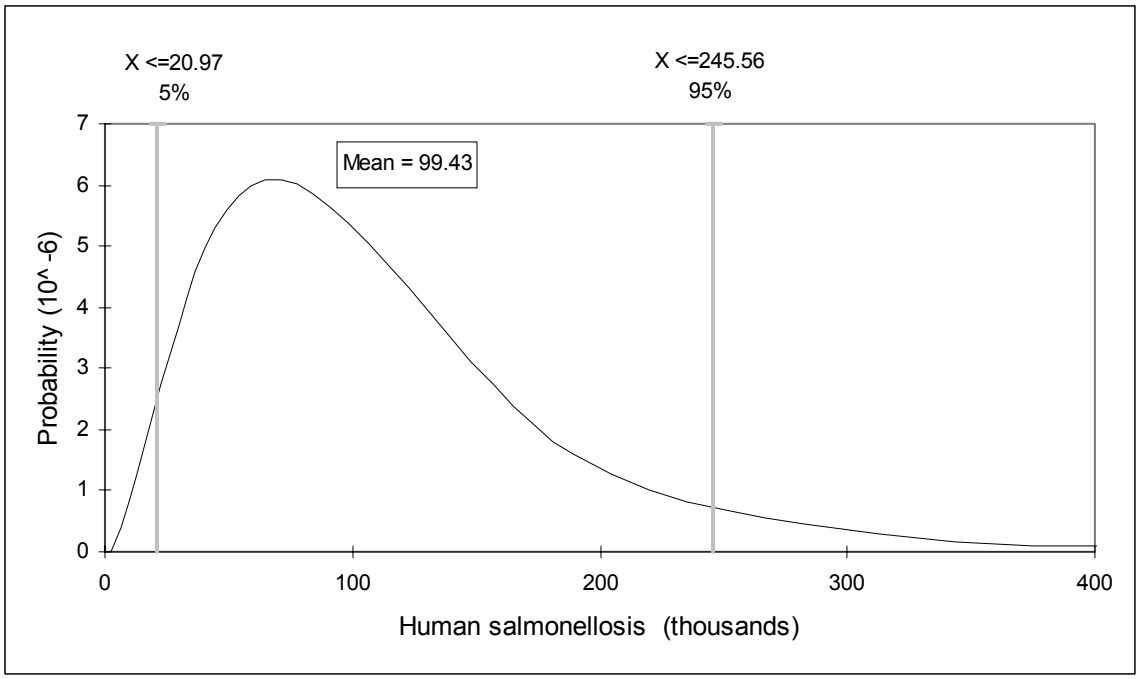


Figure 2. *Model Results of human salmonellosis cases and associated social costs*



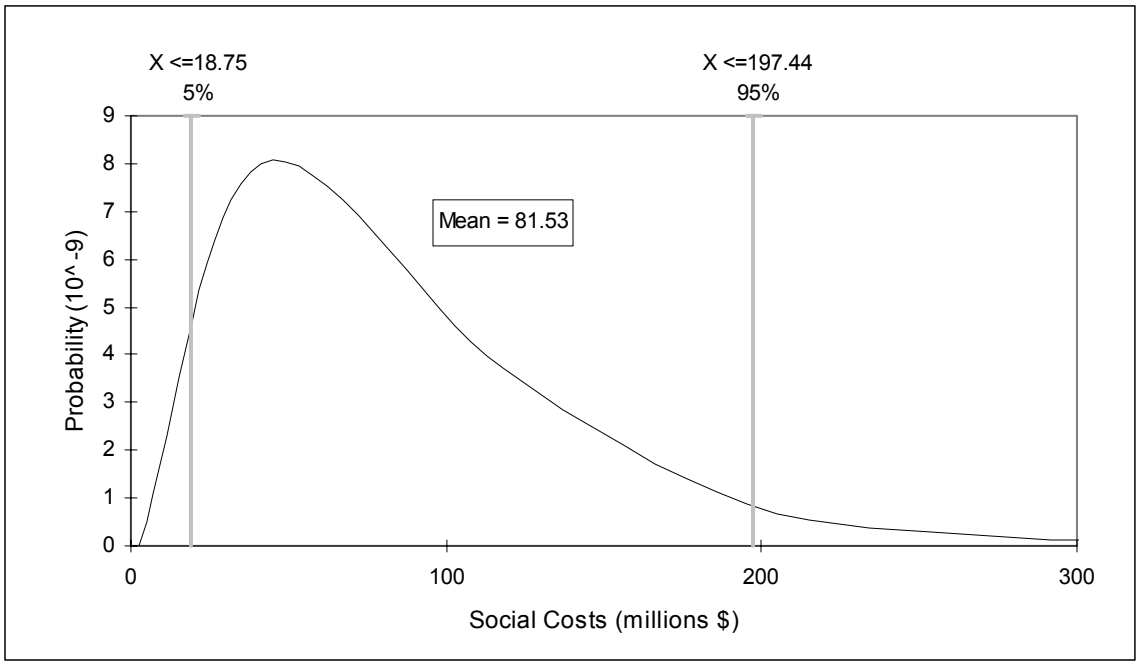
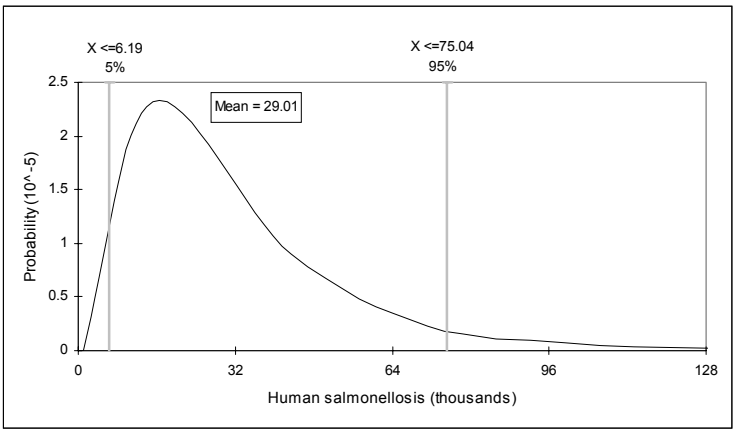
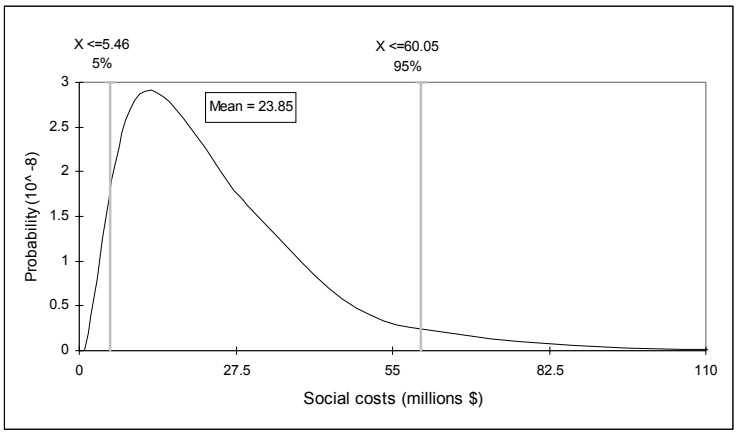
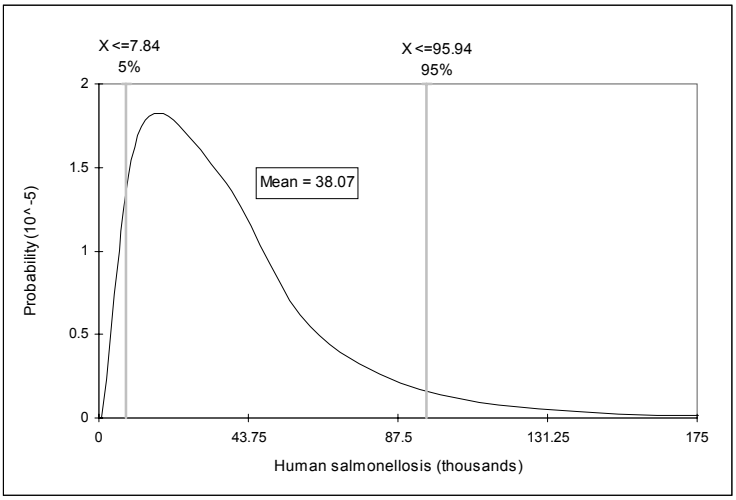


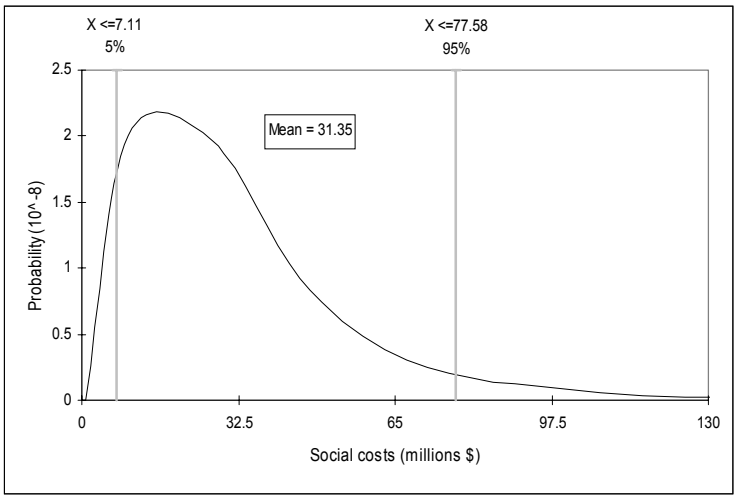
Figure 3. *Scenario model results of human salmonellosis cases and associated social costs*

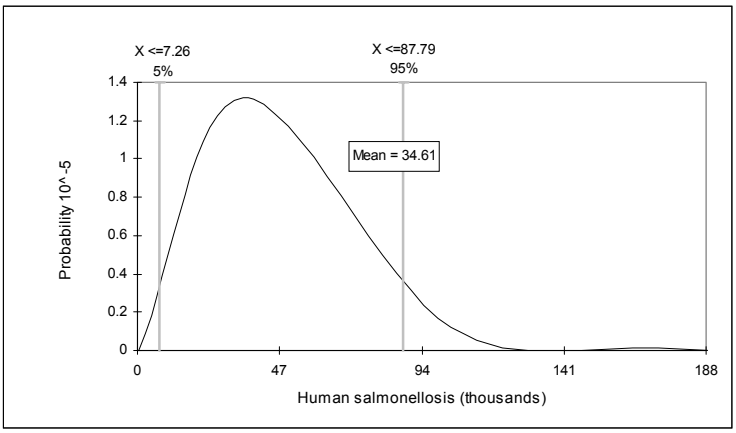
Top two graphs is vaccination results, second two graphs is feed type results, third two graphs is for carcass pasteurization, and bottom two graphs are results from rinsing with water (65C) and sanitizer added; the left hand graphs demonstrate human *Salmonella* cases, and the right hand graphs demonstrate social costs

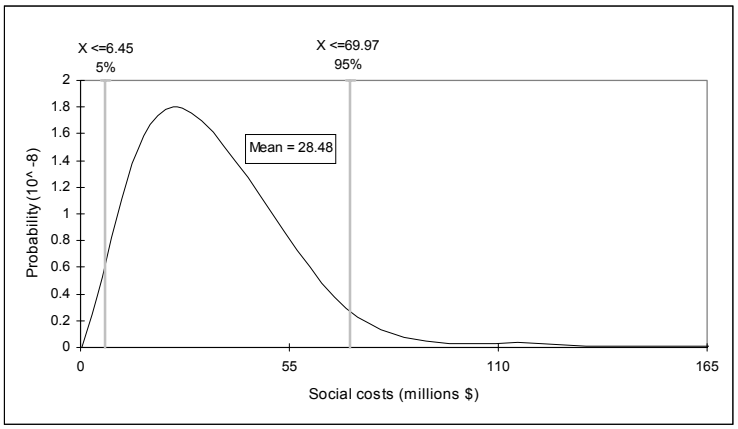

X <=6.19
5%
X <=75.04
95%
Mean = 29.01
Probability (10^-5)
Human salmonellosis (thousands)

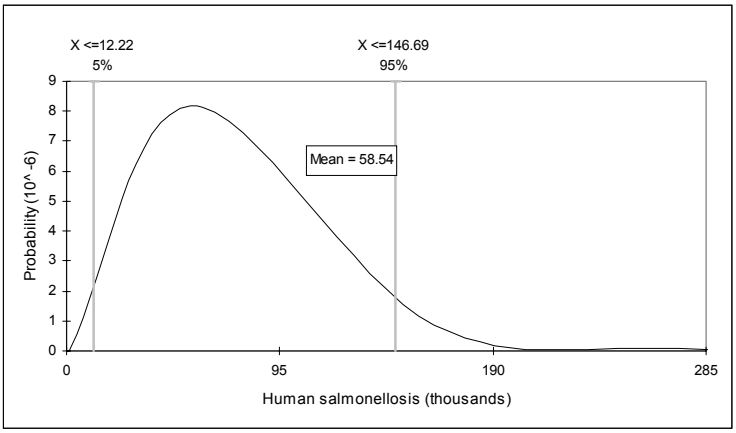

X <=5.46
5%
X <=60.05
95%
Mean = 23.85
Probability (10^-8)
Social costs (millions $)

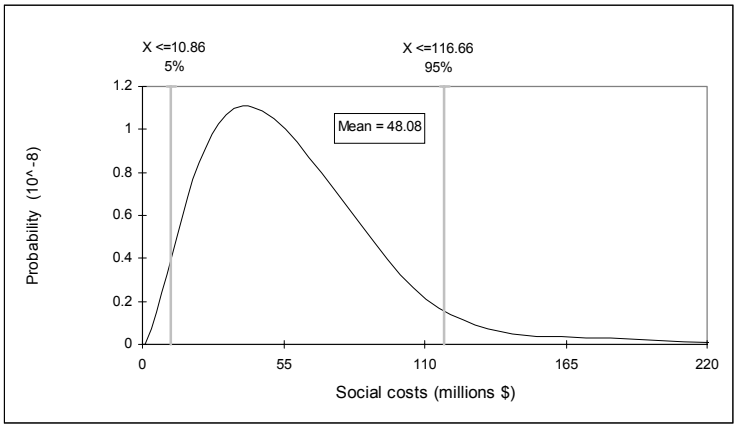

X <=7.84
5%
X <=95.94
95%
Mean = 38.07
Probability (10^-5)
Human salmonellosis (thousands)


X <=7.11
5%
X <=77.58
95%
Mean = 31.35
Probability (10^-8)
Social costs (millions $)


X <=7.26
5%
X <=87.79
95%
Mean = 34.61
Probability 10^-5
Human salmonellosis (thousands)


X <=6.45
5%
X <=69.97
95%
Mean = 28.48
Probability (10^-8)
Social costs (millions $)


X <=12.22
5%
X <=146.69
95%
Mean = 58.54
Probability (10^-6)
Human salmonellosis (thousands)


X <=10.86
5%
X <=116.66
95%
Mean = 48.08
Probability (10^-8)
Social costs (millions $)