Table 2. Number of strains tested (N) and resistance proportions (R%) for E. faecalis and E. faecium from pork meat.

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Breakpoint (µg/ml)</th>
<th>E. faecalis</th>
<th>E. faecium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>&gt;16</td>
<td>40</td>
<td>0.0</td>
</tr>
<tr>
<td>Bacitracin</td>
<td>&gt;128</td>
<td>40</td>
<td>15.0</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>&gt;8</td>
<td>40</td>
<td>0.0</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>&gt;4</td>
<td>40</td>
<td>25.0</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>&gt;4</td>
<td>40</td>
<td>15.0</td>
</tr>
<tr>
<td>Flavomycin</td>
<td>&gt;16</td>
<td>40</td>
<td>5.0</td>
</tr>
<tr>
<td>Gentamycin</td>
<td>&gt;500</td>
<td>40</td>
<td>7.5</td>
</tr>
<tr>
<td>Lincomycin</td>
<td>&gt;4</td>
<td>40</td>
<td>97.5</td>
</tr>
<tr>
<td>Pirlimicyn</td>
<td>&gt;2</td>
<td>40</td>
<td>12.5</td>
</tr>
<tr>
<td>Salinomycin</td>
<td>&gt;16</td>
<td>40</td>
<td>7.5</td>
</tr>
<tr>
<td>Strep &gt; 1000</td>
<td>&gt;1000</td>
<td>40</td>
<td>12.5</td>
</tr>
<tr>
<td>Strep &gt; 2000</td>
<td>&gt;2000</td>
<td>40</td>
<td>2.5</td>
</tr>
<tr>
<td>Quinupristin/dalfopristin</td>
<td>&gt;4</td>
<td>40</td>
<td>15.0</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>&gt;16</td>
<td>40</td>
<td>0.0</td>
</tr>
</tbody>
</table>

**Conclusion:** These results confirm that raw meat is contaminated with pathogenic bacteria, mainly *Salmonella* spp., and consequently with these raw products pathogens and resistant strains will enter the kitchen and may lead to food borne diseases and/or transfer of resistant strains or resistance genes to humans. Therefor, the prevention of food borne diseases and resistance transfer is mainly in the hands of those preparing food. These persons should as well be educated on the basic sanitary principles of food preparation as well as be informed on the possible presence of pathogens in raw products, for instance by informative labels.

**References:**

DANMAP 2000 (2001). Consumption of antimicrobial agents and occurrence of antimicrobial resistance in bacteria from food animals, foods and humans in Denmark. ISSN 1600-2032.

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**PREHARVEST INFLUENCE ON SALMONELLAHE HEALTH COSTS AND RISKS FROM PORK**

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**Summary:** The purpose of this study is to understand the implications of the prevalence of salmonellae in pigs preharvest for the food safety risks and human health costs for humans. Mean costs from human cases of salmonellae predicted by the model was 77,183,000 $, with a range of 6,019,000 - 723,777,000 $. Using sensitivity analysis, on-farm apparent prevalence has an important influence on pork-associated human health costs. Tornado graphs are a poor way to assess model variable importance, and should only be used for preliminary analysis with a flow model of this type. Mismatched data sources, and
lack of data for linking prevalence to degree of contamination make risk assessment very difficult, and results are tenuous at this time. Nonetheless, we believe post-harvest handling of pork products during processing and fabrication, cooking, on-farm prevalence, and increases at transport and lairage are all important determinants of pork-associated salmonellae human health costs.

Keywords: Risk assessment, farm-to-fork, human health, pigs

Introduction: Food borne illness in the U.S., particularly illness believed to be associated with eating foods of animal origin, has received increased research and media attention. Of the zoonotic pathogens in pork, salmonellae are some of the most important. The economics of salmonellae mitigation strategies has been minimally researched. The purpose of this study is to understand the implications of the prevalence of salmonellae in pigs preharvest for the food safety risks and costs for humans.

Materials and Methods: We are developing an integrated model which uses @Risk and estimates the risk and economic impact of salmonellae 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, 2) after transport and lairage, 3) during carcass processing to the point post-carcass chilling, 4) at fabrication and retail, and estimate human risk/costs of 5) consumer cooking and consumption, 6) salmonellae health consequences, and 7) economics of these health consequences. 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 salmonellae in pigs at the farm uses USDA, APHIS VS CEAH CAHM (1997). Adjustments for apparent prevalence are made to derive true prevalence using sensitivity and specificity (Funk et al., 2000). We assume prevalence increases from the combined effects of transportation and lairage estimated by averaging scientific study results (from Hurd et al., 2001; McKean et al., 2001; Proescholdt et al., 1999). We assume a 1/1 correspondence between fecal positive and carcass surface positive. This is substantially more than 2/1 found by Morgan et al., 1987.

There are a large number of steps in the processing of pork carcasses that have been modeled including scalding, dehairing/singeing, carcass washing, evisceration, carcass rinse post-evisceration, steam pasteurization, and chilling. We compare results derived from the model with various published estimates (USDA, 1996, and 2003) of carcass prevalence at the point of post-chilling.

Increases in prevalence of salmonellae that occur during fabrication and at the retail level are assumed based on data from Duffy (2001). The degree of contamination is also affected by cooking by the consumer. Cooking effects and food handling care are assumed to protect against exposure differently between the two risk groups. 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 are estimated which can be attributed to pork using literature that documents risks and costs from salmonellae infection. Specifically, the dose response model outlined by WHO (2002) was used. This model uses a beta-Poisson function with $a = 0.1324$, and $B = 51.45$, with an associated distribution around the curve, and $a$ and $B$ are assumed the same for low and high risk groups (WHO, 2002). Costs for human salmonellae 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 assume 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.

The relative importance of the various elements within the model are ranked using a tornado graph in @Risk. Sensitivity analyses are also conducted.

Results: Mean costs from human cases of salmonellae predicted by the model was 77,183,000 $, with a range of 6,019,000 - 723,777,000 $. Preliminary results from the tornado graph of total costs suggested that farm prevalence is one of the lesser important variables influencing costs from salmonellae in humans. Similarly, the influence of transportation and lairage was of lesser importance; preharvest influence was driven by the assumptions relative to expansion of prevalence (directly related to farm prevalence and time) spent in transportation and lairage. The impact of preharvest prevalence on human risks and costs was influenced by the degree of sanitation in slaughter and processing and the impact of cooking assumed. The errors (mistakes compared to correct ideal process and cooking) which can occur in processing (from fecal contamination) and home preparation (from cross contamination) and cooking (inadequate temperature) appeared to be more important influences on the human health costs of salmonellae than are farm prevalence, at least for the levels of prevalence seen in U.S. pig farms based on the tornado graph.

However, the tornado graph for a flow model of this type is of little use in ranking the importance of various variables. Tornado analysis suggests that the numbers of salmonellosis cases in humans in the two risk groups are the two most important variables. These variables are closest to the calculations of total costs in terms of basic model flow. Thus the tornado graph approach fails to recognize from a biological perspective that the reason these cases occur is because of inputs that occur earlier in the flow of salmonellae in the farm-to-fork chain.

This suggests that sensitivity analysis using this model is a much more appropriate approach in evaluating the influence of model variables on the human health costs. Using sensitivity analysis, on-farm apparent prevalence has an important influence on pork-associated human health costs. An increase in apparent prevalence from roughly 0.06 to 0.09 increases human health costs by 62%. Thus, the estimates for on-farm prevalence are important using this model.

Comparison of USDA estimates of prevalence post-chilling and what is suggested by the model which examines various steps in processing and fabrication resulting in log increases and decreases of surface organisms currently do not give comparable results.

Discussion: There are a number of very large problems and heroic assumptions that were made in this salmonellae risk assessment for pork. First, there are limited or no data that allows for translation between prevalence estimates and quantitative estimates of the amount of organism contamination (e.g. CFU/g, or CFU/cm²). Much of the literature collected on processing reports log reductions in surface organisms; but the vast majority of the literature prior to processing reports data on a pig or carcass prevalence basis, with the proportion positive being the reported data. This mismatch of various information sources creates substantial problems. Second, the data relating fecal positive status to surface positive status is very limited. Thus, we assume a one-to-one correspondence which potentially over estimates surface prevalence. Third, to determine human illness, an estimate of the amount of human exposure is needed. This requires translation from prevalence into a quantitative degree of exposure. Fourth, even though we believe that quantitative risk assessment models should include environmental influences, feedback loops and other elements that reflect likely circumstances (Barber et al., 2003), we assume a simple straight flow model with all risk derived originally from pigs. Finally, almost all data are limited and extremely important assumptions that influence model output to varying degrees must be made at every stage of the analysis.
There is disagreement between large and knowledgeable groups of scientists about very basic points important to a salmonellae risk assessment in pork. USDA (1998) argues strongly for a difference between high and low risk groups of people in terms of susceptibility to salmonellae infection, and they propose different betas. WHO (2002) argue that "...it is not possible to conclude that some segments of the population are more susceptible to ... Salmonella...". However, WHO (2002) does suggest that severity of illness can be different for different risks groups.

There may indeed be differences by serotype in dose-response, severity of illness, etc., for humans (Schlosser et al., 2000). Different serotypes of salmonellae predominate in pigs and humans. This model considers all salmonellae as a group and makes no distinctions based on serotype. Additionally, there are important differences in microbiological identification of salmonellae in each of the studies referenced; such differences also have important implications (Maddox, 2003) for the model. These implications are unknown and not explicitly considered.

There is a large body of literature needed to support this risk assessment. We have 323 references in our database that supports details in the model. Only a small fraction of some of the more important studies and references is listed in this paper.

Despite model deficiencies and lack of data, food safety risk assessment models still yield some insights into process control, evaluation, and data collection priorities (Roberts et al., 1999). We hope this is also the case with this model.

In conclusion, post-harvest handling of pork products during processing and fabrication, cooking, on-farm prevalence, and increases at transport and lairage are all important determinants of pork-associated salmonellae human health costs.

References:
PERFORMANCE OF ANTI-SALMONELLA LACTIC ACID BACTERIA IN THE PORCINE INTESTINE

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Summary: Of five anti-Salmonella porcine cultures administered to pigs at 10^10 cfu/day, two Lactobacillus murinus strains demonstrated superior survival during gastrointestinal transit. Both were detected at 10^7-10^8 cfu/g faeces which was higher (P<0.05) than Pediococcus pentosaceus DPC6006 (~10^5 cfu/g). One Lb. murinus strain was also excreted at higher numbers (P<0.05) than either Lb. salivarius DPC6005 or Lb. pentosus DPC6004 (both ~10^6 cfu/g). The Lb. murinus strains persisted in both the faeces and the caecum for at least 9 days post-administration. Animals fed a combination of all five strains at 10^10 cfu/day excreted ~10^7 cfu/g of the administered strains, which was higher (P<0.05) than only P. pentosaceus DPC6006. Randomly amplified polymorphic DNA (RAPD) PCR analysis revealed that both Lb. murinus strains predominated in the faeces of these animals during administration, while post-administration, both Lb. murinus strains and Lb. pentosus DPC6004 were recovered from the faeces and the caecum while P. pentosaceus DPC6006 was only detected in the caecum. After 21 days of culture administration, faecal Enterobacteriaceae counts were reduced in pigs fed Lb. salivarius DPC6005, P. pentosaceus DPC6006, Lb. pentosus DPC6004 and the culture mix, though not significantly. Overall, the porcine intestinal isolates offer potential as probiotics for enteropathogen reduction in pigs; possibly as a combination due to strain variation.

Keywords: probiotic, pigs, gastrointestinal tract, Lactobacillus, Pediococcus

Introduction: Probiotics aimed at restoration and maintenance of a healthy gut microflora offer potential as a means of controlling enteric pathogen carriage in pigs. Competitive exclusion cultures are particularly effective in pigs (Nisbet, 2002). However, uncertainty regarding their exact composition has led to concerns that they may result in pathogen transmission and could also hinder regulatory approval. Alternatively, defined probiotic strains, most commonly lactic acid bacteria, Bacillus or yeast, can be used as feed additives, either individually or as mixtures. However, commercial animal probiotic products do not always contain the strains or species listed on the label at an adequate dose or may lack evidence of a probiotic effect (Weese, 2002), highlighting the need for proper...