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5. RISK CHARACTERIZATION OF *SALMONELLA* ENTERITIDIS IN EGGS

5.1 SUMMARY

In risk characterization for *S.* Enteritidis in eggs, the output of exposure assessment was combined with hazard characterization, and the probability that an egg serving results in human illness was demonstrated. Changes in predictive risk upon changes in the flock prevalence and time-temperature scenarios are investigated. Key uncertainties that might have certain influence on the result are also shown. In addition, effects of risk management options are quantitatively compared and evaluated. It should be noted that the risk assessment of *S.* Enteritidis in eggs was intentionally conducted so as not to be representative of any specific country or region. The probability of illness and the

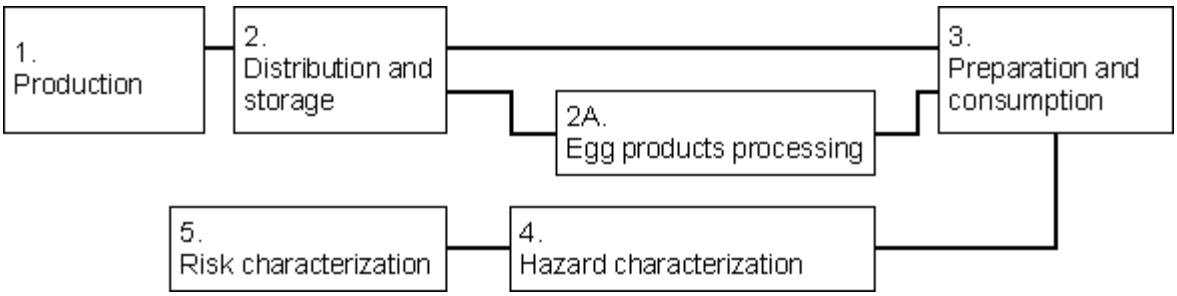
compared effects of possible management options therefore only reflect the data environment used in this assessment.

5.2 RISK ESTIMATION FOR S. ENTERITIDIS IN EGGS

5.2.1 Model overview

The general structure of the *S.* Enteritidis in eggs risk assessment is outlined in Figure 5.1. The exposure assessment model consists of three stages: production; shell egg processing and distribution; and preparation and consumption; and combined with egg products processing if appropriate. This information is combined with the dose-response model from the *Salmonella* hazard characterization to estimate human illnesses resulting from exposures predicted by the exposure assessment to provide the risk characterization. The parameters used for the beta-Poisson dose-response function were described in Hazard Characterization (Table 3.16 in Section 3.5.2). One simulation of the entire model consists of 30 000 iterations, sufficient to generate reasonably consistent results between simulations.

Figure 5.1. Schematic diagram showing the stages of the risk assessment of *Salmonella* Enteritidis in eggs



5.2.2 Results

The final output of the shell egg model is the probability that an egg serving results in human illness. This probability is determined as the weighted average of all egg servings (both contaminated and not contaminated) in a population. Clearly, the risk per serving is variable when we consider individual egg servings (e.g. a serving containing 100 organisms is much more likely to result in illness than a serving containing just 1 organism), but the meaningful measure is the population likelihood of illness. This risk per serving can be interpreted as the likelihood of illness given that a person consumes a randomly selected serving.

Three values for flock prevalence (5%, 25% and 50%) were considered. As explained earlier, three scenarios for egg storage time and temperature were also considered (reduced, baseline and elevated). The combination of these uncertain inputs generates nine different outputs from the model.

The lowest risk of illness is predicted when flock prevalence is 5% and storage times and temperatures are reduced (Table 5.1). In this scenario, the calculated risk is 2 illnesses in 10 million servings (0.00002%). The highest risk is predicted when flock prevalence is 50% and storage times and temperatures are elevated. In this case, the calculated risk is 4.5 illnesses in each million servings (0.00045%).

Table 5.1. Predicted probabilities of illness per egg serving based on different flock prevalence settings and different egg storage time and temperature scenarios.

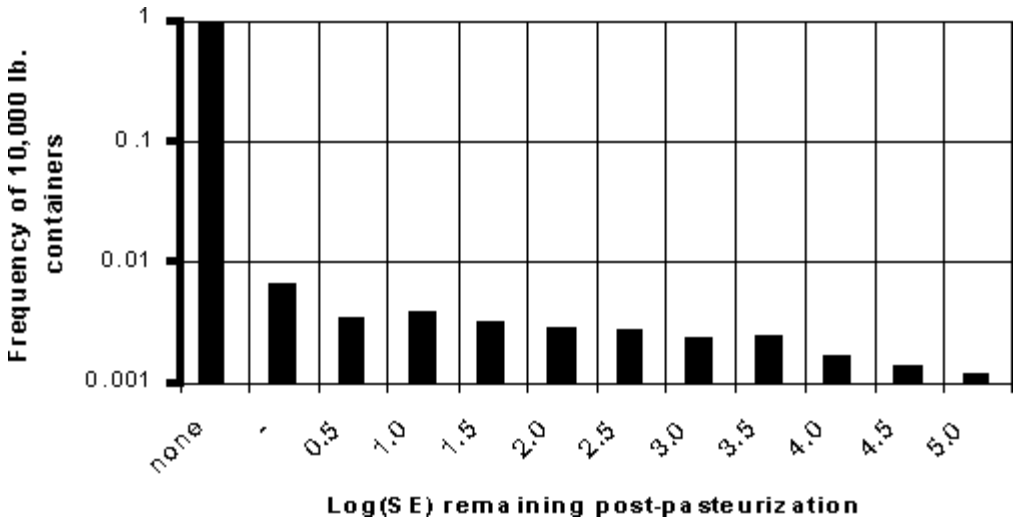
Flock prevalence	Time-temperature scenarios		
	Reduced	Baseline	Elevated
5%	0.00002%	0.00002%	0.00004%
25%	0.00009%	0.00012%	0.00022%
50%	0.00017%	0.00024%	0.00045%

Changes in risk are approximately proportional to changes in the flock prevalence. For example, 5% flock prevalence is one-fifth of 25%. Correspondingly, the risk of illness for scenarios with 5% flock prevalence is one-fifth that of scenarios with 25% flock prevalence. Similarly, doubling flock prevalence from 25% to 50% also doubles the risk of illness if all other inputs are constant.

Under the baseline conditions using data set for this model, for any constant flock prevalence, the risk decreases by almost 25-30% from the baseline time-temperature scenario to the reduced time-temperature scenario. This risk increases by almost 90% between the baseline and elevated time-temperature scenarios. Although the degree of change in risk would be altered from baseline conditions, these simulations show, for example, that changing storage times and temperatures from farm to table results in disproportionately large effects on risk of illness.

The final output of the egg products model is a distribution of the numbers of *S.* Enteritidis remaining in 10 000-lb (~4500 litre) containers of liquid whole egg following pasteurization. The *S.* Enteritidis considered in this output are only those contributed by internally contaminated eggs. This output serves as a proxy for human health risk until the model is extended to consider distribution, storage, preparation - including additional processing - and consumption of egg products. Figure 5.2 shows the output for the 25% flock prevalence, baseline scenario. About 97% of the pasteurized lots are estimated to be *S.* Enteritidis-free, and the average level is about 200 *S.* Enteritidis remaining in each lot.

Figure 5.2. Predicted distribution of *Salmonella* Enteritidis (SE) contributed by internally contaminated eggs remaining in 10 000-lb (~4500 litre) containers of liquid whole egg after pasteurization. This distribution is predicted based on an assumed 25% flock prevalence, and the baseline egg storage times and temperatures in the model. Note that the y-axis is in $\log_{10}$ scale.



5.2.3 Uncertainty

Key uncertainties considered in this analysis relate to within-flock prevalence, frequency of egg contamination from infected hens, frequency of contaminated eggs laid in which the yolk is contaminated, and dose-response parameters.

Within-flock prevalence (FHen_Flock) is a distribution fitted to available data (Table 4.20 and Figure 5.3). Uncertainty regarding the mean of this distribution is estimated by re-sampling from the estimated lognormal distribution with a sample size equivalent to the original data and re-calculating the mean of the simulated data (i.e. bootstrap methods). For simplicity, it was assumed that the standard deviation of this lognormal distribution was constant and equal to 6.96% (Table 4.20). Uncertainty in this curve was calculated by assuming that the uncertainty about the mean was normally distributed. The standard deviation of the mean calculated from 1000 bootstrap replicates was 0.38%. The 5th and 95th confidence bounds are shown in Figure 5.3.

Frequency of egg contamination from infected hens is assumed constant in the model, but its uncertainty is modelled using a beta distribution with inputs from Humphrey et al.(1989). The frequency of yolk-contaminated eggs is constant in the model, but its uncertainty is modelled using a beta distribution reflecting the outcome of Humphrey et al. (1991). Uncertainty regarding the dose-response parameters is modelled as described in the hazard characterization section.

Uncertainty about the probability of illness per serving is shown to increase as the assumed flock prevalence increases (Figure 5.4). For any given flock prevalence, the uncertainty distribution has a constant coefficient of variation (i.e. standard deviation/average). Therefore, as the average probability of illness increases, its uncertainty increases proportionately.

Figure 5.3. Cumulative frequency distributions for within-flock prevalence (FHen_Flock). The curve predicted by available data from infected flocks is shown relative to the best fitting lognormal distribution curve. Upper and lower bound curves are predicted using the 95th and 5th confidence intervals of the mean of the best fitting lognormal distribution.

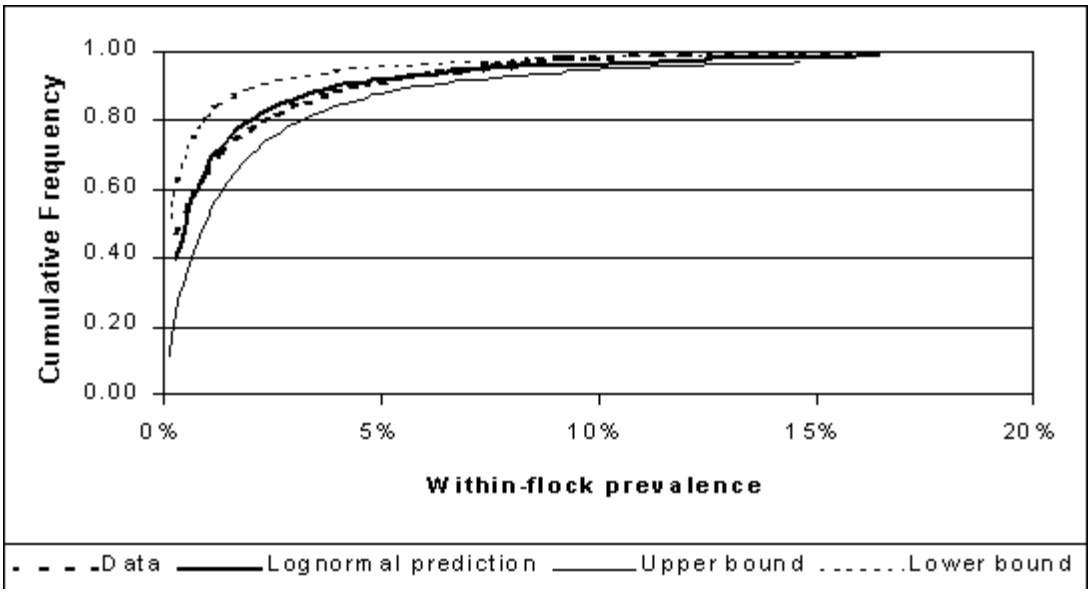
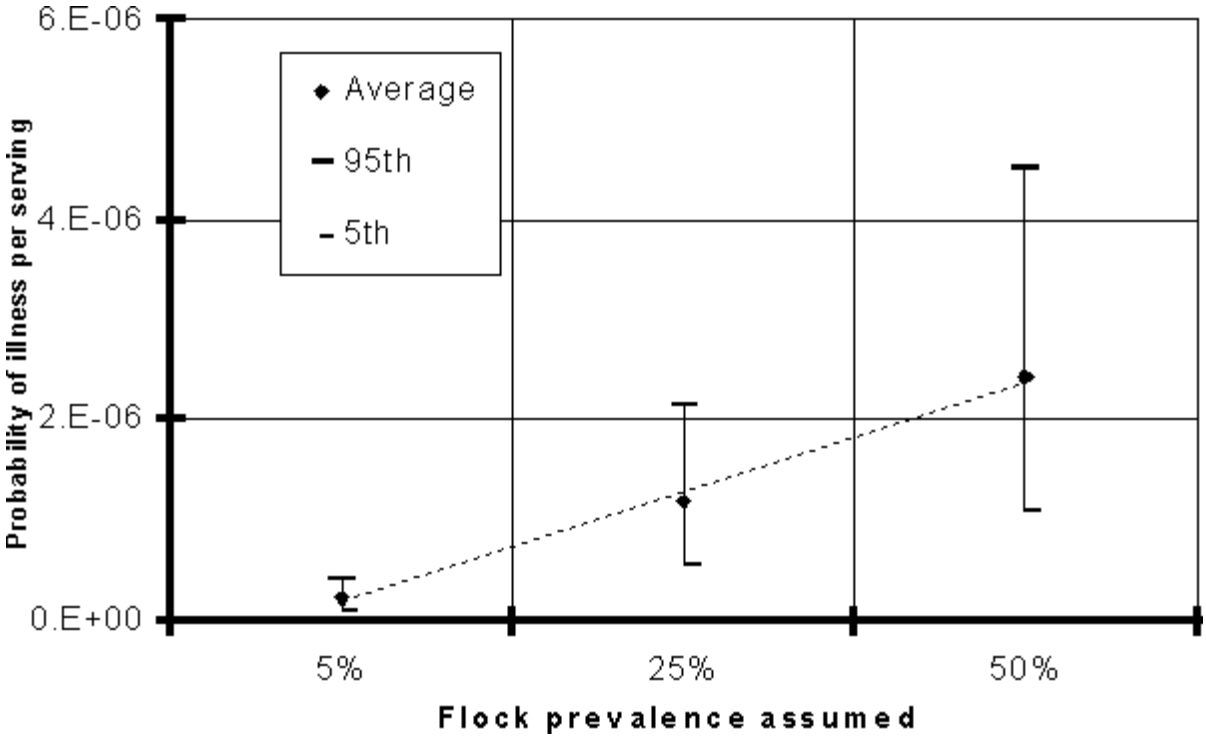


Figure 5.4. Uncertainty in probability of illness for different flock prevalence inputs assuming the baseline egg storage times and temperatures. Error bars represent the 90% confidence intervals for calculated uncertainty distributions.



Uncertainty not considered in this analysis relates to flock prevalence, predictive microbiology equations, time and temperature of storage, and pathway probabilities. Nevertheless, by changing the input values for flock prevalence, storage time and storage temperature, some evidence is

provided regarding the effect of these inputs on risk (i.e. the sensitivity of the predicted risk per serving to these model inputs).

5.2.4 Discussion

The range in risk of illness predicted by this model extends from at least 2 illnesses per 10 million shell egg servings to 45 illnesses per 10 million servings. The scenarios considered represent a diversity of situations that approximate some countries or regions in the world. Nevertheless, no specific country is intentionally reflected in this model's inputs or outputs.

The effect of different flock prevalence levels on per serving risk is straightforward to calculate from this model. Nevertheless, the impact of changing egg storage times and temperatures is not trivial. These effects must be simulated to estimate the result. The model shows that change of 10% (either increase or decrease) in storage times and temperatures result in greater than a 10% change in the predicted risk per serving.

The uncertainty of probability of illness per serving was proportional to the average probability in each scenario considered. That finding suggests that we should be able to simulate scenarios and directly calculate uncertainty based on the average risk predicted by this model.

5.3 RISK MANAGEMENT OPTIONS FOR S. ENTERITIDIS IN EGGS

5.3.1 Estimation of the risk of illness from S. Enteritidis in eggs in the general population at different prevalence and concentration levels of contamination

The model was used to estimate the relative effects of different prevalence and concentration levels of *S.* Enteritidis in contaminated eggs. Prevalence can either be the proportion of flocks containing one or more infected hens (i.e. flock prevalence) or the proportion of infected hens within infected flocks (i.e. within-flock prevalence). The risk associated with different flock prevalence levels was illustrated in Table 5.1. That analysis illustrated that risk was generally proportional to the flock prevalence level. Reducing the proportion of infected flocks is therefore associated with a proportional decline in the likelihood of illness per serving among the population of all servings. One can also examine the risk of illness per serving for different within-flock prevalence levels, as well as for different starting concentrations of *S.* Enteritidis per egg.

To model the effect of within-flock prevalence on risk, the 1st, 50th and 99th percentile values of the within-flock prevalence distribution (0.1%, 0.5% and 22.3%, respectively) were simulated (Figure 5.5). The point of this analysis is to isolate the effect of within-flock prevalence on likelihood of illness by considering within-flock prevalence to be non-variant, but examining three different levels. This analysis also provides insight as to the effect of assuming different *average* within-flock prevalence levels on probability of illness. For these simulations, flock prevalence was assumed to be 25%. In the baseline time-temperature scenario, risk per serving was 6×10^{-8}, 3×10^{-7} and 1×10^{-5} for within-flock prevalence levels of 0.1%, 0.5% and 22.3%, respectively. The results show that risk of illness per serving changes in direct proportion to changes in within-flock prevalence. This effect occurs regardless of the time-temperature scenario considered. Consequently, the risk per serving if all infected flocks had within-flock prevalence levels of 10% (i.e. 10 of every 100 hens are infected) is 100 times the risk per serving when the within-flock prevalence is fixed at 0.1% (i.e. 1 in every 1000 hens is infected). In terms of control, these results

suggest that reducing the proportion of infected hens in flocks provides a direct means of reducing illnesses from contaminated eggs.

Different initial levels of *S.* Enteritidis in eggs at the time of lay were modelled by assuming that all contaminated eggs started with 1, 10 or 100 organisms (Figure 5.6). The baseline egg storage time and temperature scenario was assumed, but flock prevalence was varied. For a flock prevalence of 5%, risk per serving was about 2 per 10 million regardless of whether the initial number of *S.* Enteritidis per egg was 1, 10 or 100. For flock prevalence levels of 25% and 50%, a more detectable change in risk per serving occurs between eggs initially contaminated with 1, 10 or 100 *S.* Enteritidis. For example, at 25% flock prevalence, the risk per serving increases from 8 per 10 million to 10 per 10 million as the number of *S.* Enteritidis in eggs at lay increases from 1 to 100. Nevertheless, for one-log changes in the initial numbers of *S.* Enteritidis, the resulting change in probability of illness is much less than one log.

Figure 5.5. Predicted probability of illness, assuming that within-flock prevalence is either 0.1%, 0.5% or 22.3% (1st, 50th, or 99th percentiles of the lognormal distribution used in the model, respectively). Three egg storage time and temperature scenarios are considered. Flock prevalence is assumed to be 25%.

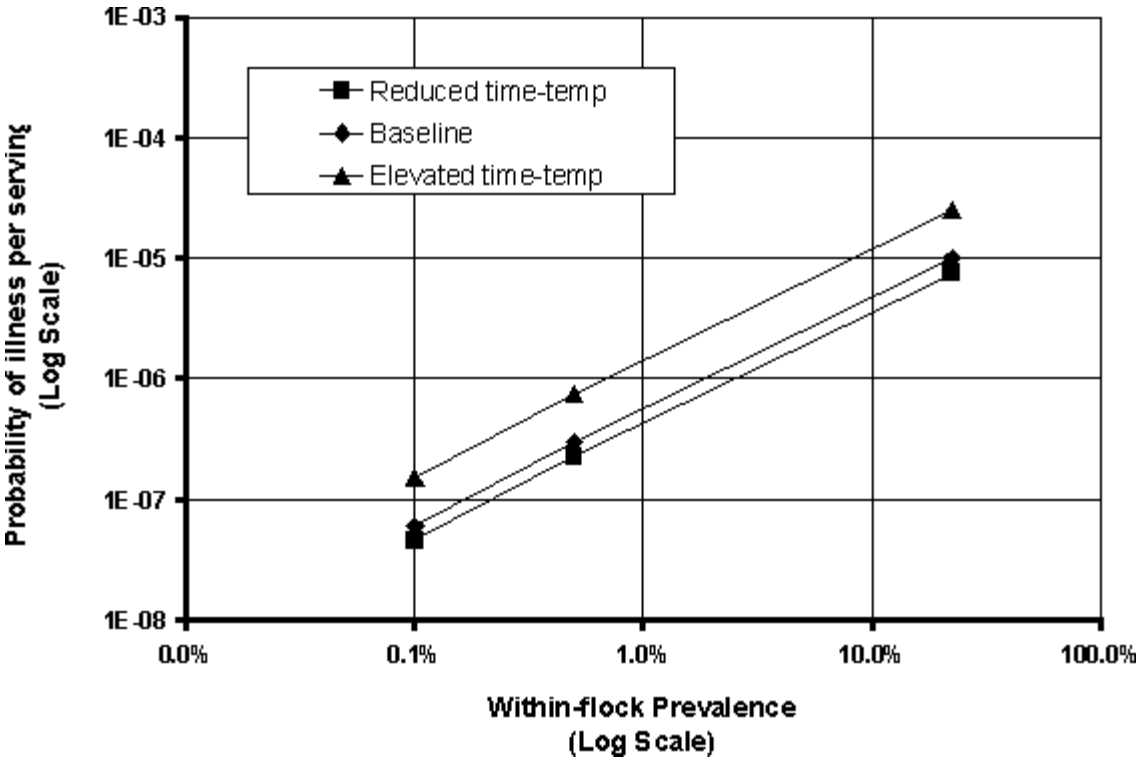
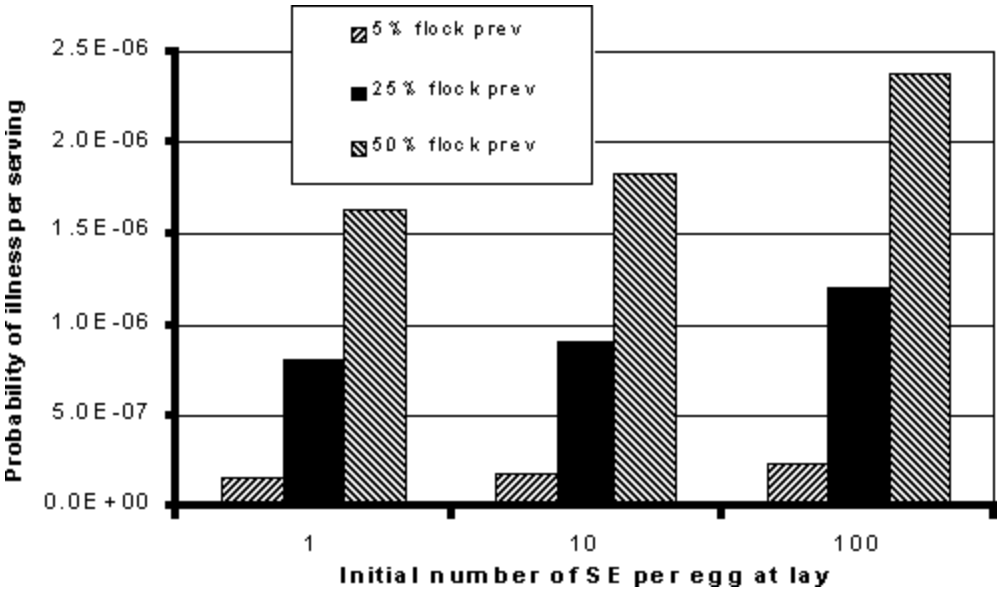


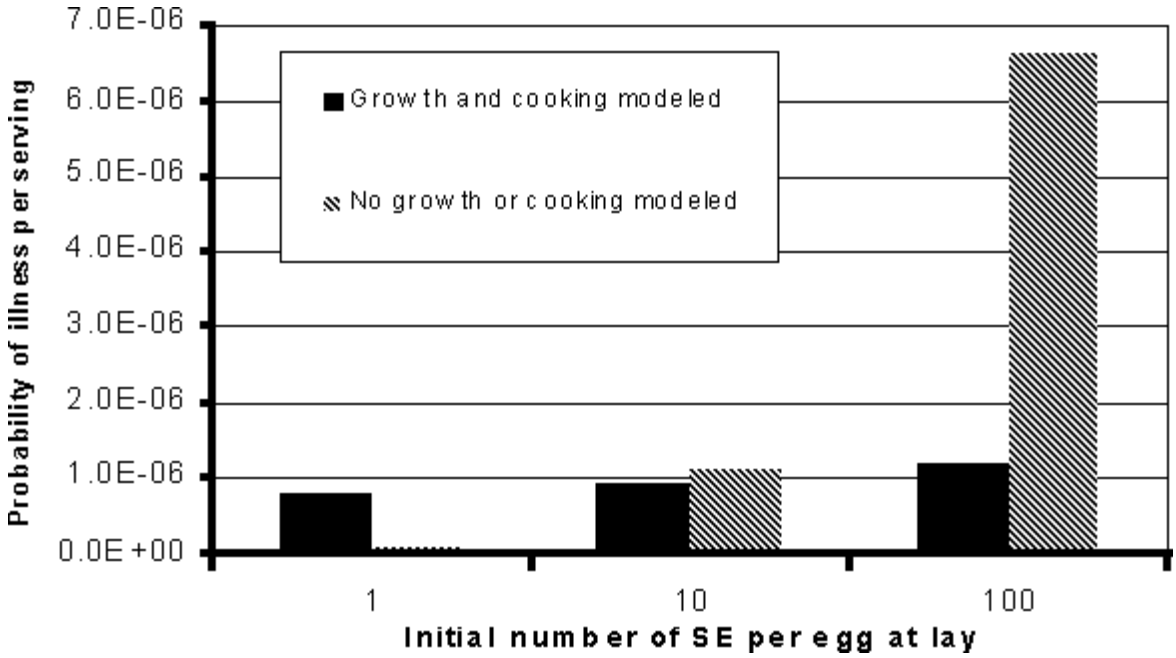
Figure 5.6. Predicted probability of illness per serving, assuming that the number of *Salmonella* Enteritidis (SE) per contaminated egg at lay is 1, 10 or 100. Three flock-prevalence levels are considered. Egg storage times and temperatures are assumed to be the baseline settings.



The dose-response function used in this risk characterization predicts that the probability of illness given an average dose of 1, 10 or 100 organisms is 0.2%, 2.2% or 13%, respectively. If all contaminated eggs were consumed raw immediately after lay, one would expect these probabilities to be appropriate to predict illnesses. The production module predicts that contaminated eggs are produced at a frequency of about 5×10^{-5} (~1 in 20 000) when flock prevalence is 25%. If all contaminated eggs contained just one organism, with no growth or decline before consumption, the predicted risk per serving should be $5 \times 10^{-5} \times 0.002$, or 10^{-7}. Similarly, the risk per serving if all eggs were contaminated with 10 and 100 organisms would be 10^{-6} and $\sim 7 \times 10^{-6}$, respectively.

Figure 5.7 compares these predicted risks - when no growth or cooking is assumed - to the predictions shown in Figure 5.6 for 25% flock prevalence. When just a single *S.* Enteritidis organism is in contaminated eggs, Figure 5.6 implies that allowing growth inside eggs elevates the risk. Yet when contaminated eggs contain 10 or 100 organisms, Figure 5.6 implies that cooking of egg meals substantially reduces the risk. The explanation for these findings is that, regardless of the initial contamination, the combined effect of growth and cooking is to stabilize the risk per serving to nearly one per million; whereas if growth and cooking are not modelled, the risk per serving only depends on a dose-response function that is increasing at an increasing rate across the dose range considered. Therefore, it can be concluded from Figures 5.5 and 5.6 that the model's output is relatively less sensitive to initial numbers of *S.* Enteritidis than other inputs that influence growth and cooking.

Figure 5.7. Predicted risk of illness when the exposure assessment model includes effects of growth and cooking compared with cases when no growth or cooking is modelled, for situations where the initial number of *S.* Enteritidis in contaminated eggs at lay is 1, 10 or 100. Flock prevalence is assumed to be 25% and baseline egg storage times and temperatures are assumed when growth and cooking are modelled.



5.3.2 Estimation of the change in risk likely to occur from reducing the prevalence of infected flocks and destroying breeding or laying flocks, and estimation of the change in risk likely to occur from reducing the prevalence of S. Enteritidis-positive eggs through testing of flocks and diversion of their eggs to pasteurization, and including the effect of pasteurization

As shown previously, risk of illness per serving decreases as the percent of infected flocks (i.e. flock prevalence) decreases. Table 5.2 illustrates the influence of flock prevalence on risk of illness per serving. Because the model includes uncertain inputs, risk per serving is also uncertain and this table summarizes uncertainty as the mean, 5th, and 95th percentile values (rounded to the nearest significant digit) of the predicted distribution.

Table 5.2. Predicted uncertainty in risk of illness per egg serving for different flock prevalence levels.

Flock prevalence	Mean	5th	95th
0.01%	0.00000005%	0.00000002%	0.00000009%
0.10%	0.0000005%	0.0000002%	0.0000009%
5.00%	0.00002%	0.00001%	0.00004%
25.00%	0.0001%	0.0001%	0.0002%
50.00%	0.0002%	0.0001%	0.0005%

We can use the results in Table 5.2 to predict the reduction in risk for a country or region that decides to control infected flocks. For example, consider a country with 5% of its flocks containing one or more infected hens. If such a country were to institute a programme with 98% effectiveness in reducing flock prevalence, then successful implementation of the programme would result in a flock prevalence of about 0.1%. The model predicts, in this case, that the mean risk of illness per egg serving would decrease from 200 per thousand million to 5 per thousand million. Pre-harvest interventions, such as those used in Sweden and other countries, might result in flock prevalence levels of 0.1% or lower.

Although the model predicts that probability of illness per serving is proportional to flock prevalence, the question remains: how can we reduce prevalence of infected flocks? To accomplish this seemingly requires either preventing uninfected flocks from becoming infected, or treating infected flocks to render them uninfected.

Treatment of breeding flocks to render them uninfected has been used in The Netherlands (Edel, 1994). Antibiotic treatment of the flock followed by competitive exclusion culture administration might succeed in eliminating the organism from infected hens, but environmental reservoirs may still exist to re-infect hens once the effects of the antibiotic have worn off. Furthermore, application of this method to commercial flocks may not be feasible or economic.

Preventing uninfected flocks from becoming infected is where most attention is focused in control programmes. Uninfected flocks can become infected via vertical transmission (i.e. infected eggs before hatch result in exposure of a cohort via horizontal transmission following hatching), via feed contamination, or via environmental sources (i.e. carryover infection from previously infected flocks). Control programmes may attempt to eliminate these avenues of exposure by applying one or more actions.

1. Test breeding flocks to detect *S.* Enteritidis infection, followed by destruction of the flock, if infected, to prevent it from infecting commercial flocks consisting of its future offspring.

2. Require heat treatment of feed before its sale (thereby eliminating *S.* Enteritidis and other pathogens).

3. Following depopulation of an infected flock, intense cleaning and disinfecting of poultry environments known to be contaminated. Such an approach must also eliminate potential reservoirs (e.g. rodents).

Most control programmes use all three interventions to preclude *S.* Enteritidis-infected flocks. The control programme in Sweden consists of such an approach (Engvall and Anderson, 1999). The Pennsylvania Egg Quality Assurance Program in the United States of America also used such an approach (Schlosser et al., 1999). However, discerning the efficacy of each intervention is difficult. Ideally, one would like to know what percent of newly infected flocks result from vertical transmission, feed contamination or previously contaminated environments.

Giessen, Ament and Notermans (1994) present a model for determining the relative contribution of risk of infection from vertical, feed-borne (or other outside environmental sources) and carryover environmental contamination. Comparing the model with data collected in The Netherlands, it appears that carryover infection was the dominant contributor to infection risk. Such a conclusion is based on the shape of a cumulative frequency curve for flock infection, which suggests that most flocks are infected soon after placement in commercial facilities. There is also evidence that the prevalence of infected breeder flocks is very low in The Netherlands.

Data from the United States of America *Salmonella* Enteritidis Pilot Project (Schlosser et al., 1999) suggest a fairly constant prevalence of positive samples collected in flocks by age, and that infection did not necessarily increase over time. Nevertheless, these data do not describe the age when infection was introduced. Roughly, 60% of the poultry flocks tested in this project were *S.* Enteritidis-positive. Additional evidence presented shows that 6 of 79 pullet flocks (8%) tested were *S.* Enteritidis-positive. These data suggest that the risk of infection from vertical transmission might be about 8%. Furthermore, there is some suspicion that feed contamination is an important source of *S.* Enteritidis for United States of America poultry flocks.

The data from The Netherlands and the United States of America suggest that the carryover route may account for >80% of the risk of flock infection in countries where *S.* Enteritidis is endemic. If true, then complete control of breeder flocks might only be expected to achieve £20% reduction in the prevalence of *S.* Enteritidis-infected flocks in such countries.

Results of an aggressive monitoring programme for breeder flocks in The Netherlands between 1989 and 1992 have been reported (Edel, 1994). For egg-sector breeding flocks, there is some suggestion that prevalence of infected flocks was reduced by about 50% per year. Effectiveness was less dramatic for meat-sector breeding flocks. This programme involved regular faecal testing of all breeder flocks, as well as regular testing of hatchery samples from day-old chicks. Positive flocks were depopulated until mid-1992, when treatment with enrofloxacin and a competitive exclusion culture was allowed as an alternative to the expense of prematurely depopulating a breeding flock. If a programme with 50% effectiveness in reducing prevalence of infected flocks each year were implemented for 3 years, one might predict that prevalence would fall to 12% (0.5^3) of the prevalence at programme start.

To reduce the risk of carryover infection for commercial flocks, it is thought that aggressive cleaning and disinfection must be completed after an infected flock is depopulated and before another flock is placed to begin a new production cycle. Cleaning and disinfection must also include an effective long-term rodent-control programme. Analysis of efforts in Pennsylvania to reduce the prevalence of infected commercial flocks suggests a decline from 38% to 13% during three years of programme operation (White et al., 1997). This programme routinely screened flocks for evidence of *S.* Enteritidis and required thorough cleaning, disinfection and rodent control once positive flocks

had been depopulated. Another study in Pennsylvania (Schlosser et al., 1999) found 16 of 34 (47%) poultry environments that were initially *S.* Enteritidis-positive were negative for the pathogen following cleaning and disinfection.

Risk characterization of test and diversion programmes depends on the specific testing used in commercial flocks. For example, the Swedish programme collected three pooled samples, each consisting of 30 faecal droppings, during two or more examinations of egg production flocks during each production cycle (Engvall and Anderson, 1999). In The Netherlands, the breeder-flock monitoring programme testing protocol required the collection of 2 pools of 50 caecal droppings each every 4 to 9 weeks of production (Edel, 1994). The *Salmonella* Enteritidis Pilot Project's protocol required collection of swabs from each manure bank and egg belt in a hen house on three occasions in each production cycle (Schlosser et al., 1999).

Regardless of the size or type of sample collected, it would seem that a testing protocol that examines commercial flocks frequently and diverts eggs soon after detection should result in a meaningful reduction in the contaminated shell eggs marketed each year.

To examine the effect of test and diversion with the present model, two protocols were assumed, with either one or three tests administered to the entire population of egg production flocks. The single test would be administered at the beginning of egg production. Under the three-test regime, testing at the beginning of egg production would be followed by a second test four months later, and the third administered just before the flock is depopulated. Each single test consists of 90 faecal samples randomly collected from each flock. A flock is considered positive if one or more samples contained *S.* Enteritidis.

For the within-flock prevalence distribution used in this model, a single test of 90 faecal samples was likely to detect 44% of infected flocks. This was calculated using a discrete approximation to Equation 5.1, where a summation replaces the integral and discrete values of p, the within-flock prevalence. This equation assumes that an infected hen sheds sufficient *S.* Enteritidis in her faeces to be detected using standard laboratory methods.

$$\text{Probability of flock testing positive} = \int_0^1 1-(1-p)^{90} f(p)\,dp \qquad \text{Equation 5.1}$$

If a flock was found positive on a test, its entire egg production was diverted to pasteurization. It was assumed that the egg products industry normally uses 30% of all egg production (consistent with the United States of America industry). Therefore eggs going to breaker plants from flocks other than those mandatorily diverted were adjusted to maintain an overall frequency of 30% (i.e. the percentage of eggs sent to breaker plants from test-negative infected flocks, and non-infected flocks, was reduced proportionally).

Test-positive flocks' premises were assumed to be cleaned and disinfected following flock depopulation. The effectiveness of cleaning and disinfection in preventing re-infection of the subsequent flock was assumed to be 50%. Furthermore, it was assumed that carryover infection was responsible for flocks becoming infected. Consequently, houses that were not effectively cleaned and disinfected resulted in infected flocks when they were repopulated.

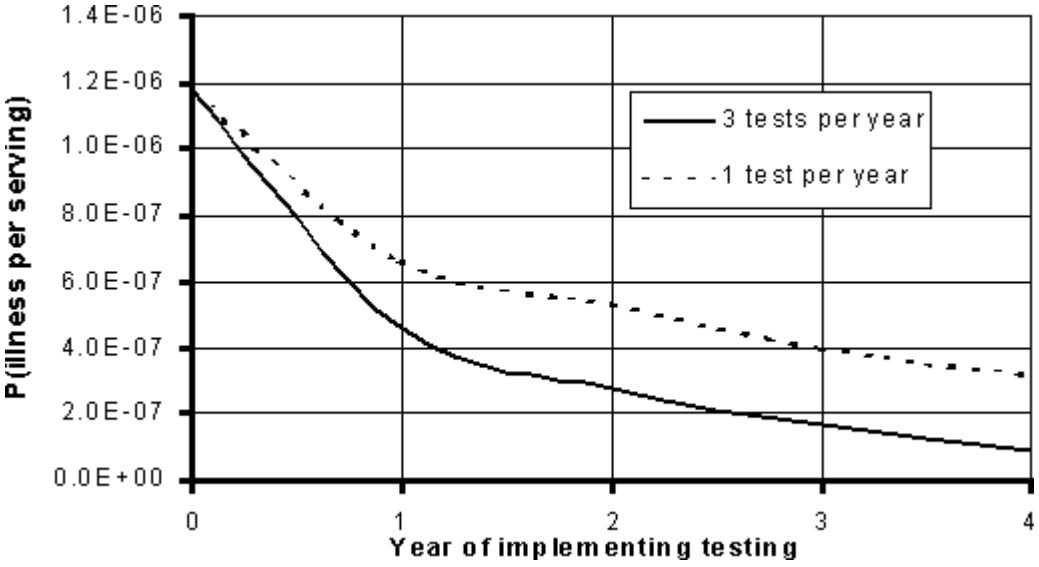
Assuming a starting prevalence of 25% and the baseline egg storage time and temperature scenario, the effectiveness of the two testing protocols was estimated over a four-year period. Probability of illness per shell egg serving in each year was calculated for each protocol (Figure 5.8). Testing three times per year for four years reduced the risk of human illness from shell eggs by more than 90% (i.e. >1 log). Testing once a year for four years reduced risk by over 70%. At the end of the fourth year, the flock prevalences for the one-test and three-test protocols were 7% and 2%, respectively. Therefore, assuming the cost of testing three times per year to be three times greater than the cost of testing once a year (ignoring producer costs or market effects from diversion of eggs), then the flock prevalence results suggest a roughly proportional difference (i.e. 7%/2% » 3) in the protocols. However, the reduction in risk per serving of the one-test protocol is greater than one-third of the three-test protocol. In other words, the one-test protocol achieves a 70% reduction while a testing protocol that is three times more costly achieves a 90% reduction (i.e. a 20% improvement). Such a result is not surprising when we consider that the single (or first) test at the beginning of the year most substantially affects risk. This is because flocks detected on the first test have their eggs diverted for the entire year, while flocks detected on a second test have their eggs diverted for just over half the year. Furthermore, flocks detected on the third test are tested so late in production that diversion of their eggs does not influence the population risk at all.

While egg diversion from positive flocks reduces the public health risk from shell eggs, it might be expected that there is some increased risk from egg products. Mandatory diversion causes more contaminated eggs to be sent to pasteurization. Nevertheless, the average quality of contaminated eggs is improved by diversion in this model.

It was assumed in the model that all diverted eggs were nest run (i.e. stored usually less than 2 days). Without mandatory diversion, 97% of lots were *S.* Enteritidis-free post-pasteurization and the average number of surviving *S.* Enteritidis in a 10 000-lb (~4500 litre) bulk tank was 200 (assuming 25% flock prevalence and the baseline egg storage times and temperatures). If a single test is used to determine which flocks are diverted, there are still 97% of vats that are *S.* Enteritidis-free and they average 140 *S.* Enteritidis per lot. The decrease in the average number of *S.* Enteritidis per lot is due to the increased proportion of nest run eggs that are diverted. Nest run eggs are stored for a shorter period and consequently contribute fewer organisms. If two tests are used, then there are 97% of vats that are *S.* Enteritidis free, and the average is 130 per lot. If three tests are used, there is no additional effect on egg products beyond the second test because the third test occurs just as the flock is going out of production.

Although not a direct measure of public health risk, these results suggest that the risk from egg products decreases as flocks are detected and diverted. However, this effect is conditional on nest run eggs being substantially less contaminated than restricted or graded eggs. Alternative scenarios might result in some increase in risk from diversion.

Figure 5.8. Predicted probability of illness per serving from shell eggs per year after implementing two testing protocols. It is assumed that all flocks in the region are tested each time and that initial flock prevalence is 25%. Baseline egg storage times and temperatures are used for the four years.



5.3.3 Estimation of the change in risk likely to occur from the use of competitive exclusion or vaccinating flocks against S. Enteritidis

The effects of competitive exclusion (CE) treatment are difficult to quantify from field evidence. Sweden and The Netherlands are examples of countries that include the use of CE in their *S.* Enteritidis control programmes. Nevertheless, such treatment is only one component of these programmes and its effect is not clearly separable from other components. CE has been studied in experimental settings for newly hatched chicks. The intent of CE inoculation in chicks is to quickly establish an indigenous intestinal flora to resist *S.* Enteritidis colonization. Efficacy of preventing infection appears to depend on the CE culture used, timing of exposure, dose of exposure, and possibly the addition of lactose (Corrier and Nisbet, 1999). Field evidence of CE efficacy in mature hens comes from the United Kingdom and from The Netherlands. In both countries, antibiotic treatment was applied to flocks known to be infected and the hens were subsequently inoculated with CE cultures. The intent of CE inoculation for hens was to quickly restore intestinal flora - that had been destroyed by the antibiotic treatment - to assist the hens in resisting future *S.* Enteritidis exposures. In the UK, 20 of 22 trials that combined antibiotic and CE treatments succeeded in preventing re-infection of flocks for a 3-month study period (Corrier and Nisbet, 1999). Infection status was determined from cloacal swab samples in treated flocks. In The Netherlands, combining antibiotic and CE treatments resulted in preventing 72% (n = 32) of flocks becoming re-infected. Two such combined treatments protected 93% of flocks from re-infection.

Vaccination for *S.* Enteritidis has been examined extensively in experimental settings, but less so in field trials. Experimentally, several types of vaccines have been evaluated: killed bacterins of various strains, live bacterins of attenuated strains, and surface antigen extracts of various strains. Injected killed bacterins are thought to have limited efficacy in preventing intestinal colonization of hens with *S.* Enteritidis, although such bacterins may, through stimulation of humoral antibody, reduce internal organ (including ovary) infection. Live bacterins - or surface antigen vaccines - may

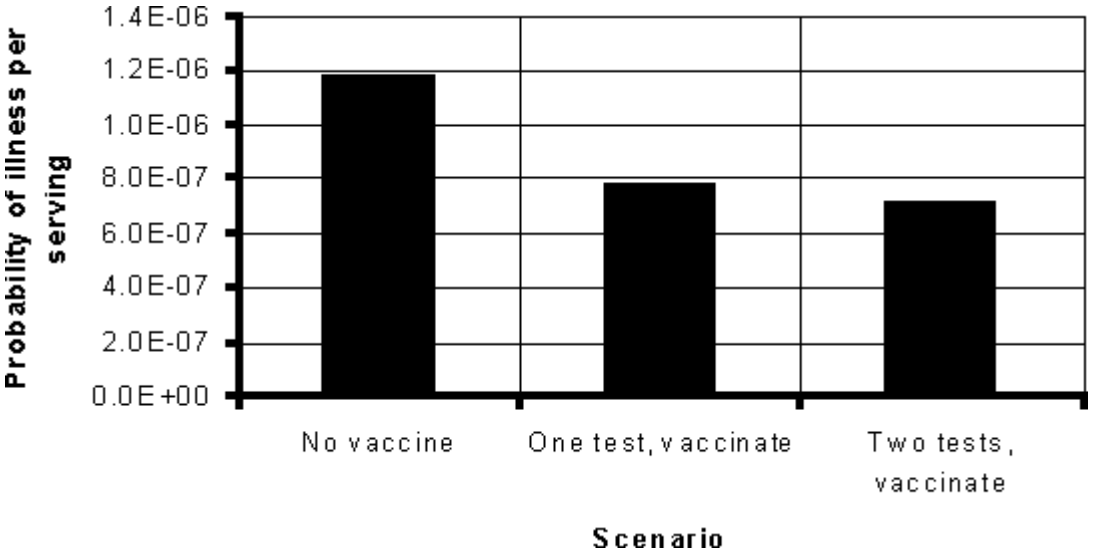
be more effective at modulating intestinal colonization by *S.* Enteritidis because these products may elicit the cell-mediated immune response needed to resist colonization. Nevertheless, most commercially available vaccines are currently of the killed variety.

Evidence concerning the effectiveness of *S.* Enteritidis bacterins in controlling infection has been reported for some Pennsylvania flocks (Schlosser et al., 1999). A total of 19 flocks from two farms used a bacterin to control their *S.* Enteritidis infection and sampling results were compared with 51 flocks that did not use a bacterin. Only a slight difference was noted in environmentally-positive samples collected in vaccinated (12%) and unvaccinated (16%) flocks. Yet, the overall prevalence of *S.* Enteritidis-positive eggs was 0.37 per 10 000 in vaccinated flocks and 1.5 per 10 000 in unvaccinated flocks. These results support the hypothesis that bacterins may not influence risk of colonization, but may reduce systemic invasion of *S.* Enteritidis, with resultant egg contamination. Nevertheless, this analysis did not control for confounding factors (e.g. rodent control, adequacy of cleaning and disinfection) that may have influenced the differences between vaccinated and unvaccinated flocks.

To evaluate the effect of vaccination against *S.* Enteritidis using the present model, it was assumed that flocks would need to be tested to determine their status prior to use of a vaccine. A single test or two tests four months apart, with 90 faecal samples per test, were assumed. The vaccine was assumed to be capable of reducing the frequency of contaminated eggs by approximately 75% (e.g. 0.37 per 10 000 for vaccinated flocks ¸ 1.5 per 10 000 for non-vaccinated flocks).

Assuming 25% flock prevalence and the baseline egg storage time and temperature scenario, the probability of illness per serving for a single test and vaccination protocol is about 70% of a non-vaccination protocol (Figure 5.9). Risk is reduced to 60% of the non-vaccination protocol if two tests are applied.

Figure 5.9. Comparison of predicted probability of illness per serving between three scenarios: when no vaccination is used; when one test is applied at the beginning of production and positive flocks are all vaccinated; and when a second test is applied four months after the first test and additional test-positive flocks are vaccinated. Flock prevalence is assumed to be 25%, and the baseline egg storage time and temperature scenario is used.



Given the efficacy of bacterin use implied by the field evidence, one can assume that universal vaccination might reduce baseline risk to 25% of the risk resulting from a non-vaccinated

population. However, the cost of vaccinating the entire population of laying hens could be high. The scenarios considered here assume that before a flock is vaccinated some testing is done to determine if that flock is infected. Nevertheless, the cost of testing all flocks must be weighed against the cost of vaccination. Also, more field research concerning the true efficacy of vaccination should be conducted before the cost of vaccination is borne by more than a few producers (i.e. if costs are to be paid by the public or shared across the entire industry).

5.3.4 Estimation of the change in risk likely to occur from minimizing the number of S. Enteritidis organisms in eggs through refrigeration of eggs after lay and during distribution, or requiring a specific shelf life for eggs stored at ambient temperatures

Interventions intended to minimize the dose of *S.* Enteritidis in contaminated eggs focus on preventing any growth of the pathogen after the egg is laid. Most evidence suggests that naturally contaminated eggs contain very few *S.* Enteritidis organisms at lay. If eggs are consumed soon after lay, or if eggs are kept refrigerated during storage, then the number of *S.* Enteritidis remains relatively unchanged prior to preparation of egg-containing meals.

Available predictive microbiology models suggest that eggs stored at 10°C will not grow *S.* Enteritidis for an average of 46 days. If most eggs are stored at <10°C and are consumed within 25 days, then interventions intended to improve egg handling will only influence the fraction of eggs that are time and temperature abused.

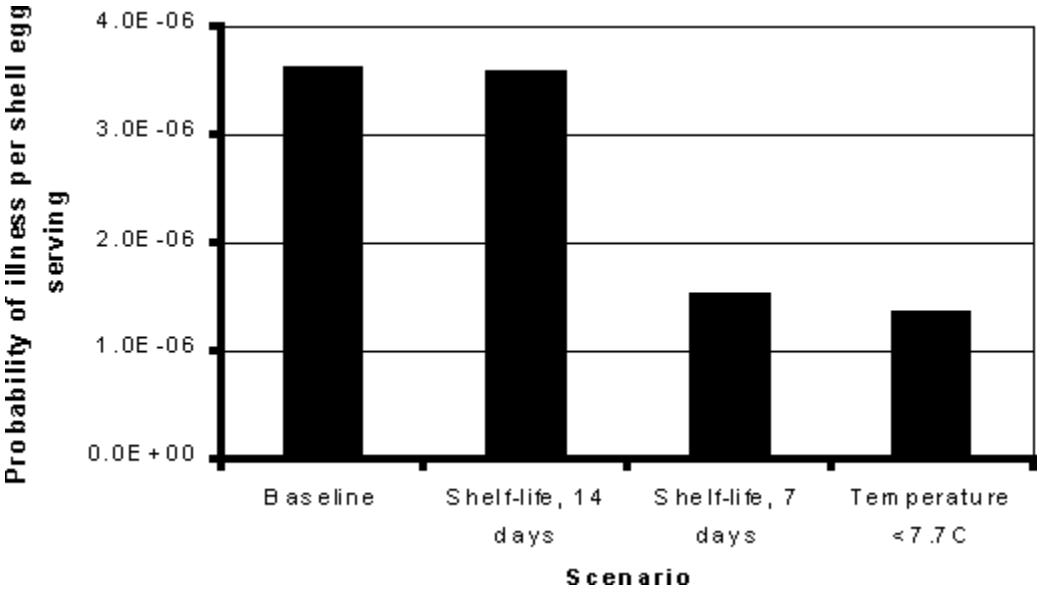
The effect of mandatory retail storage times and temperatures were evaluated using slightly different baseline assumptions (Table 5.3). These hypothetical settings might be typical in a country that does not have egg refrigeration requirements. The effects of time and temperature restrictions were evaluated assuming a flock prevalence of 25%.

Table 5.3. Hypothetical baseline input distributions for egg storage time and temperatures, assuming no egg storage requirements.

Inputs	**Distributions**
Storage temperature before transportation (°C)	*=RiskPert(0,14,35)*
Storage time before transportation (hours)	*=RiskUniform(0,3)*24*
Storage temperature after processing (°C)	*=RiskPert(5,14,30)*
Storage time after processing (hours)	*=RiskUniform(1,5)*24*
Retail storage temperature (°C)	*=RiskPert(0,14,35)*
Retail storage time (hours)	*=RiskPert(1,9.5,21)*24*

NOTES: PERT distribution has parameters RiskPert(minimum, most likely, maximum). Uniform distribution has parameters RiskUniform(minimum, maximum).

Figure 5.10. Probability of illness per serving of shell eggs given mandatory shelf lives of <14 or <7 days at retail, or mandatory retail storage temperature <7.7°C. Egg storage times and temperatures are modelled as for the baseline scenario, except for changes noted in Table 5.3. These changes to baseline egg storage times and temperatures were made to represent a country or region that does not routinely refrigerate eggs. Flock prevalence was assumed to be 25%.



Truncating retail storage time to a maximum of either 14 days or 7 days simulated a shelf-life restriction scenario. Truncating the retail storage temperature to less than 7.7°C simulated a refrigeration requirement. The results are summarized in Figure 5.10.

Restricting shelf life to less than 14 days reduced the predicted risk of illness per serving by a negligible amount (~1%). However, keeping retail storage temperature at no more than 7.7°C reduced risk of illness per serving by about 60%. If the shelf life was reduced to 7 days, risk per serving was also reduced by about 60%.

5.4 DISCUSSION

This model was purposely configured and parameterized to not reflect any specific country or region, although its results might be indicative of many country situations. A generic risk assessment such as this one provides a starting point for countries that have not developed their own risk assessment. It can serve to identify the data needed to conduct a country-specific risk assessment, as well as to provoke thinking concerning policy development and analysis.

Control of prevalence - either the proportion of flocks infected or the proportion of infected hens within flocks - has a direct effect in reducing probability of illness per serving. On the whole, egg storage times and temperatures can disproportionately influence the risk of illness per serving. Numbers of organisms initially in eggs at the time of lay seems less important.

Testing flocks, combined with diversion of eggs from positive flocks, is predicted to reduce public health risk substantially. In the scenarios considered here, diversion of eggs from test-positive flocks also reduced the apparent risk from egg products. Vaccination may reduce risk of illness by about 75%, but is typically less effective because producers would only vaccinate test-positive flocks.

As discussed in the Exposure Assessment for *S.* Enteritidis in Eggs (Chapter 4), biological inputs may be constant between models for different countries or regions, yet little else is likely to be similar. The predictive microbiological inputs, the distribution of within-flock prevalence, and the frequency at which infected hens lay contaminated eggs are examples of biological inputs that might be constant from one country to another (although not necessarily). The effects of uncertainty regarding these biological inputs to the model have been examined. Nevertheless, there are many aspects of uncertainty not fully considered (e.g. alternative statistical distributions were not evaluated for the predictive microbiology equations or within-flock prevalence distributions). Furthermore, many of the inputs are both highly uncertain and variable between countries. For example, times and temperatures of egg storage may vary considerably within and between countries, but it is difficult for any country to precisely know its distributions for storage times and temperatures.

This model introduces two new concepts not included in previous exposure assessments for *S.* Enteritidis in eggs. First, it considers the possibility of eggs being laid with *S.* Enteritidis already inside the yolk. Such eggs defy previous model descriptions of the time and temperature dependence of *S.* Enteritidis growth in eggs. Although predicted to be uncommon, yolk-contaminated eggs can support rapid growth of *S.* Enteritidis in much shorter times than eggs contaminated in the albumen. Second, this model considers the role of *S.* Enteritidis growth in eggs destined for egg products. While most eggs are modelled as being shipped very quickly to egg products plants (i.e. nest run eggs), some eggs can experience moderate or high levels of growth before being broken and pasteurized.

Many of the results generated by this model are contingent on epidemiological assumptions:

- It is assumed that infected hens produce contaminated eggs at a constant frequency that is independent of host, bacterial strain or environmental factors.

- A homogeneous population of layer flocks is assumed (e.g. same size, same basic management and environment). This model also does not consider the effect of moulting practices on egg contamination frequency.

- It is assumed that within-flock prevalence is random and independent of hen age or other host, bacterial strain or environmental factors.

These may be reasonable default assumptions, but more research is needed to determine their appropriateness. Changing these assumptions could generate results that differ from the model, and the model can be adapted to consider such changes.

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6. EXPOSURE ASSESSMENT OF *SALMONELLA* IN BROILER CHICKENS

6.1 SUMMARY

This section considers the development of an exposure assessment of *Salmonella* in broiler chickens. Initially, a general framework and the data requirements for such an assessment are considered. Data collected during the course of this work is then presented, and its usefulness for inclusion within an exposure assessment is discussed. Using appropriate data, an exposure assessment model is then developed. This model is general in nature, rather than being representative of any particular country or region. It is parameterized using two categories of data - country-specific data and general data - and these types are highlighted at the appropriate place in the model description. The output from the model is the probability of exposure by two routes: an undercooked serving of chicken, and cross-contamination resulting from preparation of that serving. For each of these routes, the number of organisms is also an output. These outputs are used to undertake a risk characterization, described in the next Chapter

6.2 REVIEW OF LITERATURE, DATA AND EXISTING MODELS

6.2.1 Introduction

Purpose

This section describes the information available to develop a production-to-consumption exposure assessment of *Salmonella* in poultry, specifically broiler chicken. To date, no complete quantitative exposure assessments have been developed for this pathogen-commodity combination. This discussion considers the way in which such assessments could be developed, focusing on data requirements and possible methodologies. In addition, this report presents summaries of some of the available data and discusses the utility and limitations of existing data. This discussion is followed by a description of the exposure assessment model developed for the current FAO/WHO risk assessment of *Salmonella* in broiler chicken (Section 6.3). The assessment focuses on home preparation and consumption of the product.

Organization

A general model framework for conducting an exposure assessment for this pathogen-commodity combination is outlined. The framework covers the various stages on the production to consumption pathways that can be analysed as individual modules.

Each module identified is discussed in detail with respect to data requirements, possible modelling approaches and data availability. The discussions on data availability are followed by a presentation of data that has been collected for each module, together with an assessment of its use in conducting a full exposure assessment. Some of these data will be country specific, while the remainder will be general and can thus be used for the majority of countries. Collection and presentation of the data serves to illustrate the type of information that is currently available to individual member countries, and simultaneously demonstrates where information is lacking, and thus highlights critical data gaps.

The data summarized in the following sections have been collected from the literature, through the FAO/WHO calls for data, from discussions with *Salmonella* experts (microbiologists, veterinarians and epidemiologists) and other sources. Therefore the database is current up to the point of writing this report, but it is acknowledged that additional information may become available in the future.
Although no complete quantitative exposure assessments, from production to point of consumption, have been developed to date for *Salmonella* in poultry products, there are models that describe segments of poultry production and processing. These are also reviewed, together with a model for *Campylobacter* spp. in fresh broiler products.

6.2.2 Production-to-consumption pathways

Overall model pathway

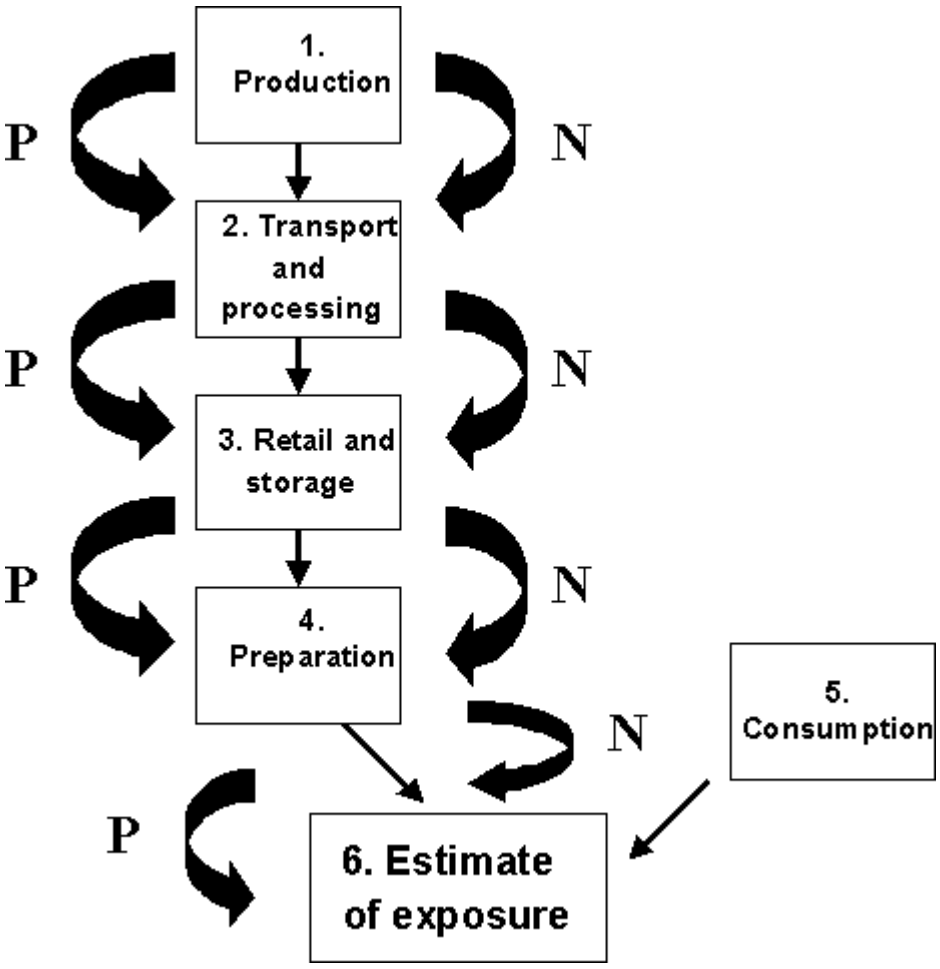
A general aim of microbiological exposure assessment for any pathogen-commodity combination is to provide estimates of the extent of food contamination by the particular pathogen, in terms of both prevalence and numbers of organisms, together with information on commodity consumption patterns for the population of interest. Estimation of these outputs can involve consideration of a number of complex and interrelated processes that relate to all stages of the production-to-consumption pathway. Throughout this pathway, process-specific factors will influence both prevalence and numbers of organisms on the product, and hence final exposure. Such effects will be both inherently variable, due to, for example, differences in production and processing methods, and uncertain because some aspects lack appropriate information.

Given this complexity, it is often necessary to split the overall pathway into a number of distinct modules, each representing a particular stage from production to consumption (Lammerding and Fazil, 2000). Such an approach has previously been used for *S.* Enteritidis in eggs (USDA-FSIS, 1998), *Campylobacter jejuni* in fresh poultry (Fazil et al., unpublished; A.M. Fazil, personal communication) and *Escherichia coli* O157 in ground beef hamburgers (Cassin et al., 1998). The resulting exposure model is then integrated with a dose-response assessment to yield the risk characterization outcomes. This type of an approach has also been described as a Process Risk Model (Cassin et al., 1998).

A modular framework for an exposure assessment of *Salmonella* in fresh broilers is outlined in Figure 6.1. Outputs from one module are used as inputs to the subsequent module. In particular, the variables that are likely to flow from one module to the next are the prevalence of contaminated birds, carcasses or products (*P*) and the probable numbers of organisms per contaminated unit (*N*).

Each module should describe, quantitatively, the changes in prevalence and numbers that occur within that step, attributable to specific factors, including, for example, the extent of cross-contamination, processing effects, the opportunity for temperature abuse, and the organism's ability to survive or grow under the conditions described.

Figure 6.1. Modular pathway to describe the production-to-consumption pathway. Each step describes the changes to prevalence (P) and numbers of *Salmonella* (N) that occur within that specific module.



Individual modules of the overall pathway

The first module shown in Figure 6.1 relates to on-farm production of broilers. Here the aim is to estimate prevalence of *Salmonella*-positive birds (intestinal carriage of *Salmonella*) and the probable number of organisms per bird at the time of transportation for primary processing. This can involve taking into account various epidemiological and farm management factors that may influence these parameters.

Following farm production, the second module of the overall pathway considers transport and processing of broilers. This module models the effects of transport and the sequential processing steps on the prevalence and numbers of organisms. Important considerations are changes because of the type of transport facilities, processing methods and conditions, including changes in prevalence because of cross-contamination between negative and positive birds.

In the third module, the effects of retail distribution and storage in the home of the consumer are modelled. With respect to retail, both transportation and "on-shelf" storage are considered. Similarly, home storage includes transportation from retail source.

Preparation of the broiler chicken product is considered in the fourth module. Changes in prevalence and numbers of *Salmonella* present for the specific product purchased is determined by handling and cooking practices, and may include estimating impacts of cross-contamination. The outputs from this module - the estimated prevalence of contaminated products and number of organisms present in the food at time of consumption - are used in the calculation of exposure.

The amount of chicken consumed during a meal by various members of the population, and over a period, is quantified in the fifth module. This information is combined with the outputs from the previous module - i.e. the predicted likelihood that the pathogen will be in the food, and the predicted numbers of organisms present - to yield an estimate of the total number of *Salmonella* ingested. This information, together with the dose-response (i.e. the likelihood of illness associated with the number of *Salmonella* the consumer ingests), is then used to calculate the risk estimate in the risk characterization.

Data needs

Quantitative modelling of the individual exposure steps requires quantitative information. Data can be collected from a number of sources including, but not limited to:

- national surveillance data;
- epidemiological surveys;
- industrial surveys;
- research publications;
- unpublished research work; and
- government reports.

Often these data are publicly available, appearing, for example, in the published literature. However, other data, such as those collected through industry surveys, are often confidential and thus access becomes difficult. It is vital that confidence be built up between the risk managers, the assessors and those who can provide valuable data for risk assessment. Confidence building requires discussions and meetings (interactive risk communication) to discuss the type of data needed and what the data are being used for (the risk management activity). In addition, discussions provide insight into the data and how they were generated, with regards to sampling strategy, testing methods, etc. Such insight can be important for correct modelling, and thus the final results. Overall, good communications among all parties is essential.

In certain cases, adequate data may not be available. One way of dealing with this is to use expert opinion. Use of expert opinion introduces several considerations, such as how to choose experts, how to avoid biased judgement, how to elicit information and how to combine information from different experts. This area of study has been discussed by Kahneman, Slovic and Tversky (1982) and by Vose (2000).

In risk assessment, and particularly in the development of generic models (i.e. for application in general commodity production, processing, distribution and consumption management decision-making), data often come from many different sources. Two issues arise from this: first, what data to include within the model, and, second, how to combine such information. Determining what data to include involves consideration of applicability, such as whether the data are relevant for a particular country; whether the data are representative of the existing situation; and whether scientifically and statistically sound sampling and testing methods were used in the collection of the data. Furthermore, regardless of the data selection criteria, the rationale and process for selection must be transparent. The importance of transparency is also emphasized for combining data. Thus various methodologies could be used, such as weighting of information, but the assessor must clearly set out the methodology to ensure clarity and reproducibility.

Overall, data collection is probably the most resource-intensive part of modelling exposure and involves many issues that influence the quality of the risk assessment outcome.

Modelling approaches

The modelling approach used for individual stages of the overall pathway will necessarily depend on the data available to quantify input parameters and, in certain cases, the simplifying assumptions made until further data becomes available. Approaches are likely to differ from one exposure module to the next, depending on the parameters being described. Moreover, the risk management question will also determine the overall approach followed.

Static and dynamic approaches

Mathematical models can be described as either static or dynamic in nature. Dynamic models describe a process over time while static models consider the state of a process at one particular point in time. Dynamic models are generally constructed in terms of differential or difference equations that describe the rate of change of model variables over time. This approach has been used for several years to describe the spread of infectious diseases in both humans and animals (see Anderson and May, 1991). In contrast, static models consider the probability of an event happening at a certain time, such as the probability of infection from consumption of a chicken product, or over a period of time, such as the probability of introduction of infection in a year.

To date, most full quantitative risk assessments have been driven by static risk management questions and thus the output estimates of risk can usually also be termed static. However, many of the sub-modules of the assessment may involve dynamic modelling to some extent. In particular, in a microbial exposure assessment, the retail and storage step may involve dynamic modelling of the growth of the organism under conditions of temperature abuse (for an overview of bacterial growth modelling, see McMeekin et al., 1993; Baranyi and Roberts, 1995). Some modules of the pathway may require a combination of static and dynamic modelling; thus, preparation may involve a description of both growth (dynamic component) and cross-contamination (static component).

Uncertainty and variability

Modelling of each stage will have to account for the inherent variability of the specific process. The level of variability may be country or region specific, although it may be possible to generalize. Variability will arise due to causes that include seasonal effects, different procedures followed by different producers, differences in primary processing facilities, characteristics of the distribution

chain, and consumption patterns. Variability cannot be reduced within a model because it describes the natural process.

In addition to variability, it will be necessary to model the uncertainty surrounding these processes. Such uncertainty will relate to the level of knowledge concerning a process and is usually reflective of the amount of available data.

Ideally, risk assessment models will explicitly separate uncertainty and variability; in essence, not separating means that one is neglected, and this can be a critical assumption with regard to further analysis. Various methods for such separation have been proposed (such as Vose, 2000), but, in reality, this often becomes complex. Ideally, factors that may be variable or uncertain, or both, should be identified and their influence on the risk assessment outcome described.

Deterministic versus stochastic models

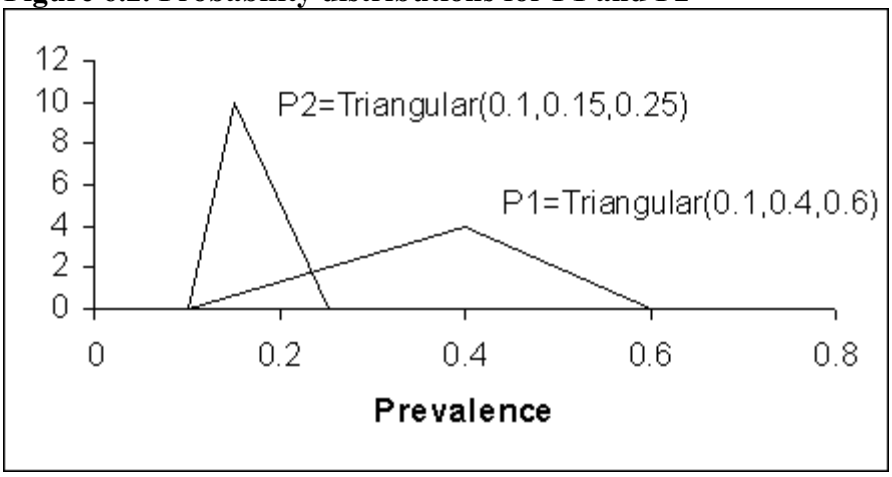
Consideration of variability and uncertainty within exposure assessments leads to discussion of deterministic versus stochastic modelling. Deterministic models use point values (e.g. the mean of a data set) to describe inputs and thus to determine outputs. Stochastic models modify the data inputs to represent variability, uncertainty or both, using probability distributions. Probability distributions describe the relative weightings of each possible value and are characterized by a number of parameters that determine their shape, such as the mean and standard deviation or the most likely, minimum and maximum.

Consider the situation where prevalence of *Salmonella* infection in broilers is unknown in two countries and an expert has provided the following opinion.

	Minimum	**Best estimate**	**Maximum**
Country 1 (P1)	0.1	0.4	0.6
Country 2 (P2)	0.1	0.15	0.25

In this situation, in order to capture the expert's opinion, a triangular distribution could be used to describe the uncertainty about prevalence for each country (Figure 6.2):

Figure 6.2. Probability distributions for P1 and P2



P1 = Triangular(0.1,0.4,0.6)
P2 = Triangular(0.1,0.15,0.25)

Stochastic models are most easily implemented on a computer using Monte-Carlo simulation. The technique of Monte-Carlo simulation involves repetition of the following events a large number of times (iterations):

1. Select a value for each input from its associated probability distribution (selection is determined by the shape of the distribution) to give one combination of input values.

2. Calculate the estimate of exposure for this combination of values.

3. Store the calculated value.

The stored values are then combined to give a probability distribution for the estimate of exposure. There are numerous references in the literature (e.g. Haas, Rose and Gerba, 1999; Vose, 2000) explaining Monte Carlo techniques, and the uses of different probability distributions.

Consideration of the risk management question

Production-to-consumption exposure assessments require considerable time, data and other resources. The inherent uncertainty and variability associated with modelling individual exposure steps in a production-to-consumption exposure pathway increases its complexity. However, this type of an assessment provides the most information for risk managers when implementation of intervention strategies may be considered at any point of the food chain, and, perhaps more importantly, for identifying important information gaps. However, alternative approaches can also be useful, depending on the risk management information needs for decision-making, and the availability of adequate data. For example, the exposure assessment can begin at the point of retail sale of poultry products, using contamination data collected at that point. This approach, in effect, disregards the effects of individual factors occurring prior to retail sale that contributed to the microbiological status of the product. A similar approach has been taken to model exposure to chicken contaminated with fluoroquinolone-resistant *Campylobacter* (CVM, 2001). This methodology is useful when data are limited, or when the complexity of the process and associated uncertainties means that modelling becomes difficult and resource intensive, but it does not facilitate the investigation of specific control measures. In particular, the effects of mitigation at different stages throughout the exposure pathway cannot be quantified. Of course, in certain cases, the investigation of specific control strategies may not be required and thus the importance of the risk question is highlighted.

Defining the correct question is the most important part of any risk assessment. The risk question drives the model and hence the approach followed in any one module. As such, it must be stressed that this report does not present a prescribed formula for model development. Rather, general approaches are presented.

6.2.3 Primary production

The overall aim of the production module is to estimate, first, the prevalence of live broiler chickens contaminated with *Salmonella* at the time of leaving the farm for processing, and, second, the number of *Salmonella* per contaminated bird.

Sources of infection

Ideally, control of *Salmonella* within broiler flocks relies on knowledge of the source of infection. Possible sources include water, feed, litter, farm staff and the environment both inside and outside the broiler house (Mead, 1992). Furthermore, hatcheries are possible sources of infection, as is vertical transmission.

Many studies associated with the production of broilers have investigated factors that increase the prevalence of *Salmonella.* Rose et al. (1999) summarize the literature into five groups of risk factors:

- Inadequate level of hygiene, *Salmonella* contamination of the previous flock, with persistence inside the house.
- Contaminated day-old chicks and contaminated feed.
- The farm structure (>3 houses on the farm).
- Wet and cold seasonal conditions.
- Litter-beetle infestation of the house.

Several of the studies included within this summary focus on broiler-breeder farms rather than broiler chicken production farms. However, it may be assumed that the risk factors identified above are applicable to all poultry flocks. Of the above-listed factors, feed and hatcheries are regarded as principle sources of infection.

An ideal exposure assessment of *Salmonella* in broilers would include the calculation of the probability of infection from a number of possible sources. Such calculation could be based on, for example, the numbers of salmonellae a chicken is exposed to from each source and the subsequent consequences of exposure. Results from epidemiological studies could assist in this type of calculation. Given such a model, possible control strategies could be investigated in a quantitative manner.

In reality, data relating to the numbers of *Salmonella* organisms within feed, litter, etc., and the numbers to which a bird has been exposed, is extremely limited or simply unknown. Due to this limitation, previous microbial exposure assessments have started from the point of estimating the prevalence of contaminated, *Salmonella*-positive birds (Fazil et al., unnpublished; A.M. Fazil, personal communication; Hartnett et al., 2001). Although this approach inhibits the investigation of on-farm control strategies, it is currently the most likely approach that can be used for developing an exposure assessment of *Salmonella* in broilers.

Prevalence of Salmonella-*positive birds*

Prevalence in this document is defined to be the probability of a bird being infected with *Salmonella.* To estimate prevalence, data are required on positive (infected) birds at the point of leaving the farm for slaughter. Such data should be representative of the population of broilers and hence should cover a number of producers, flocks and seasons. Often, this type of information is not available (Hartnett et al., 2001), and, in this case, flock prevalence and within-flock prevalence can be estimated and used to generate an estimate of bird-level prevalence.

Flock prevalence

Flock prevalence is the proportion of flocks containing one or more infected - *Salmonella*-positive - birds. Flock prevalence is a national estimate, hence country-specific data are required. Estimation of flock prevalence requires consideration of the broiler production methods used. Differences in production practices occur not only between countries, but also within countries. For example, within the United Kingdom (and therefore probably in many other industrialized countries), many poultry companies may have their own feed mills, breeder flocks and hatcheries, thus differences between companies may exist. In addition, different breeds of birds may be used, both within a country and worldwide. Further, flock sizes, densities and the conditions under which a bird lives can also vary, such as free-range and organic birds versus mass-produced commercial birds. Many of these factors may influence the *Salmonella* status of a flock.

In addition to production methods, it is possible that climatic conditions may also influence flock prevalence. There is distinct seasonal effect in the outbreak of human *Salmonella* cases, which peak in the summer months. However, Angen et al. (1996) have showed a significant increase in prevalence of *Salmonella* in broiler chickens in Denmark during the autumn months of September-November, and Soerjadi-Liem and Cumming (1984) demonstrated a higher probability of *Salmonella* infection in Australian flocks during the cold and wet season. Climatic effects may in turn produce variation in flock prevalence between different geographical locations of a particular country.

Consequently, it is likely that flock prevalence may vary from region to region, from producer to producer, from season to season, and even from year to year. Testing all poultry before leaving the farm is impractical, and hence, data from sampling a portion of flocks are used to estimate the flock prevalence distribution, and should be defined by the associated uncertainty.

Within-flock prevalence

Within-flock prevalence refers to the proportion of birds within a single flock that are infected with *Salmonella.* Within-flock prevalence of *Salmonella* is very likely to vary from flock to flock for a number of reasons. Factors influencing such variability include the virulence of the *Salmonella* strain, levels of stress within the broiler house, and the occurrence of other avian diseases that may concurrently weaken resistance to *Salmonella.* As with flock prevalence, this variability should be represented within the exposure assessment model.

Ideally, the prevalence of *Salmonella* within flocks would be determined by sampling all broilers within all flocks just before leaving the farm for slaughter, but such comprehensive data collection is impractical. Therefore, as with flock prevalence, sample data could be utilized to obtain an estimate of the distribution for within-flock prevalence, together with a description of its associated uncertainty.

Note that intermittent shedding may affect the detection of *Salmonella* and thus birds and flocks testing negative by cloacal swabbing just prior to slaughter may nevertheless carry external contamination.

Number of* Salmonella *in infected birds

In addition to prevalence of *Salmonella*-positive broiler chickens, the number of organisms per positive bird is also a consideration, so that contamination in the processing environment can be

modelled. Methods for determining the numbers of salmonellae in or on a bird can differ markedly, and a large degree of variability arises from different procedures. Results are reported in different units depending on the methodology, e.g. colony-forming units (CFU) or most probable number (MPN). In general, for risk assessments, CFU is the preferred unit of data, but MPN data can also be formulated such that they can be of use for estimation. In addition, the true number of organisms per bird is likely to vary from bird to bird. Consequently, there will be a large amount of variability in this estimate, and such variability may arise from a number of different sources.

Sampling information

For both prevalence and concentration, other information related to the collection of the data is also important. In particular, the test method used and its associated sensitivity and specificity must be considered. At the farm level, many different sample collection methods are used to determine the *Salmonella* status of individual broiler chickens or of the flock. For example, samples may be faeces, the caeca, cloacal swabs, and various environmental specimens. Other factors that influence results include the basis for the sampling strategy, the statistical validity of the sampling plan, information on farm management, the time of year of data collection, and the age of birds. Consequently, interpretation and combination of data can become difficult.

Summary of available data

Salmonella-*positive flocks and within-flock prevalence*

Tables 6.1 to 6.4 provide a summary of the flock and within-flock prevalence collected for this project. Initial observation of the data indicates that at the time of writing this report, information on *Salmonella* prevalence is missing for countries in a number of regions of the world. In particular, there is no or limited data for African, Asian and South American countries. Many countries within these regions provided some information for the 1995 Animal Health Yearbook (FAO-OIE-WHO, 1995), but information is restricted to details such as when the last case was reported and the level of occurrence. For other countries, no information appears to be have been reported.

For flock prevalence, in Table 6.1, much of the reported prevalence data include details of the numbers of flocks tested and the numbers of positive flocks. In cases where number of flocks tested and numbers positive are not provided, point estimates or ranges for flock prevalence are reported (e.g. studies by Mulder and Schlundt, in press; Hartung, 1999; White, Baker and James, 1997). In some cases (Tables 6.1 and 6.2), different sample materials are used to derive the flock prevalences, which introduces uncertainty. In addition, specificity and sensitivity of the various test protocols are rarely described. Few of the reports include information on how the results relate to the overall population of broiler chicken flocks, hence any variability due to, for example, differences between poultry companies (vertically integrated operations) is difficult to estimate. At the time of preparing this report, only one study (Soerardi-Liem and Cumming, 1984) had considered seasonality by sampling at different times of the year (Table 6.3).

Overall, it appears that flock prevalence is very variable between countries. However, it must be recognized that different sampling methods have been used in the different studies. In particular, in some reports environmental samples such as the litter, water and feed have been tested to determine positive flocks (for example, Lahellec et al., 1986; Jones et al., 1991a; Poppe et al., 1991). In contrast, other studies (such as Jacobs-Reitsma, Bolder and Mulder, 1994; Angen *et al.* 1996) involve direct testing of the broilers by examining the cloaca or caeca. Given these differences, comparison of country data must be undertaken with caution.

For within-flock prevalence, the data presented in Tables 6.3 and 6.4 indicate that there is very limited information relating to within-flock prevalence. In contrast to the flock prevalence data, several of the reported studies have considered variability among flocks, using the same sampling and testing protocols. For example, the data reported by Jacobs-Reitsma, Bolder and Mulder (1991) for the Netherlands show a large amount of variation in within-flock prevalence (a range of 0 to 80% for the caeca samples, and a range of 0 to 100% for the liver samples). Similarly, a wide range in values is reported from the Australian study by Soerjadi-Liem and Cumming (1984) (Table 6.3). As noted for some of the flock prevalence studies, the sample sizes reported in these surveys are small and thus there will be a large amount of uncertainty associated with any derived distributions for within-flock prevalence.

Number of organisms

At present, there are few data for numbers of *Salmonella* within infected broiler chickens (e.g. number per gram of faeces), or the numbers that may be present on feathers, skin, etc., of either birds that are infected, or birds that do not have intestinal carriage of the organisms but are surface contaminated. Most studies simply determine the presence or absence of salmonellae in the material tested. However, one study reported 100-1000 CFU of *Salmonella* per gram of gut content (Huis in 't Veld, Mulder and Snijders, 1994). Humbert (1992) reported that samples of *Salmonella*-positive faeces in the environment contain between 10^2 and 10^4 CFU salmonellae per gram. This small amount of information could be used to derive a distribution for the number of organisms, but there would be large associated uncertainty.

Data gaps

Overall, the following main data gaps have been identified for the primary production module.

- *Salmonella* prevalence information is available for some countries worldwide, but many of these studies give *limited details of study design*.

- Regions for which there is no or limited prevalence data include Africa, Asia and South America.

- No information relating to *sensitivity* or *specificity* of tests used is presented in the studies.

- There are very limited data relating to *numbers of organisms* per *Salmonella*-positive or contaminated bird.

Table 6.1. *Salmonella* flock prevalence data (see also Table 6.2).

Country (and year of sampling if stated)	Sample	No. flocks tested	Percentage of positive flocks	Reference
Australia				
(April-Sept.) 1984	Caeca	7	86	Soerjadi-Liem & Cumming, 1984
(Oct.-March) 1984		13	46	
Austria				
1998	Cloaca	5 029	3.4	EC, 1998
1997		8 698	4.8	
1996		7 412	5.5	
Belgium				
1998	Faeces	122	36.1	EC, 1998
Denmark				
1998	Sock-samples	4 166	6.5	EC, 1998
1997		4 139	12.9	
1996		3 963	7.9	
1996-97	NS[1]	NS	5-10	Mulder and Schlundt, in press
1995	NS	NS	25-30	
1996	Caeca	7 108	16.8	Angen et al., 1996
Finland				
1998	Faeces	2 856	0.7	EC, 1998
1997		2 951	0.7	
1996		2 568	0.9	
France				
	NS	86	69.8	Rose *et al.*, 1999
	Walls, drinkers, litter, feed	180	53.3	Lahellec, Colin and Bennejean, 1986
Germany				
-	NS	58	12.0	Hartung, 1999

1998	NS	455	4.2	EC, 1998
1997	NS	691	5.8	
1996	NS	3 119	4.2	
Ireland				
1998	NS	1 732	20.7	EC, 1998
Italy				
1998	NS	1 093	3.1	EC, 1998
1997	NS	754	1.1	
Japan				
1995-96	Faeces	35	57.1	Murakami et al., 2001
Netherlands				
1998	NS	192	31.8	EC, 1998
1997	NS	63	25.4	
-	Caeca	181	27.0	Jacobs-Reitsma, Bolder and Mulder, 1994
-	NS	NS	Up to 25.0	MSF, 1990
-	Faeces (trucks, crates)	107	67.3	Goren et al., 1988
Norway				
-	NS	2 639	<0.01	ARZN, 1998
Sweden				
1998	Faeces	2 935	0.03	EC, 1998
1997		3 379	0.06	
1996		3 300	0.12	
UK				
-	Litter	3 073	18.5	Anon., 1999

Note: NS = not stated

Table 6.2. Flock prevalence and comparison of different sampling methods

Country	No. of flocks tested	Sample (no. of samples)	% Positive	Reference
Canada	294	Environment	76.9	Poppe et al., 1991
		Litter	75.9	
		Water	21.6[1]	
		Feed	13.4[2]	
Netherlands	141	Caeca	24.1	Goren et al., 1988
	92	Litter	19.6	
	49	Skin	12.0	
USA	267		4.5	
		Dead bird rinse (14)	14.3	Jones et al., 1991a
		Live bird rinse (14)	7.2	
		Faeces (155)	5.2	
		Environment (42)	2.4	
		Litter (14)	0	
		Water (14)	0	
		Feed (14)	0	

NOTES: (1) 63 of 292 flocks. (2) 39 of 290 flocks

Table 6.3. Seasonal flock and within-flock prevalence of *Salmonella* in Australian flocks based on caecal samples (Source: Soeradi-Liem and Cumming, 1984).

Season	No. of birds tested per flock	% positive birds
Autumn-winter (April-Sept.)	50	32
	50	36
	50	34
	50	92
	50	90
	50	40
	50	0
Spring-Summer (Oct.-March)	50	22
	50	12
	50	30
	50	10
	50	4
	50	22
	7 flocks, 50 birds each	0

Table 6.4. Within-flock prevalence and bird prevalence.

Country	No. of birds tested per flock (flocks sampled)	Caeca	Liver	Caeca 5-6 weeks (on-farm)	Skin and feathers 5-6 weeks	Caeca, 5-6 weeks (at processing)	Other	Source
Netherlands	3 399 (1)	14.3						[1]
Netherlands	10 (10)	20	10					[2]
		20	0					
		10	20					
		0	50					
		70	100					
		30	80					
		0	10					
		80	90					
		20	100					
USA	100 (3)	52(1)		15	9	2		[3]
		48(1)		17	5	4		
		66(1)		25	49	11		
Iraq	232 (NS)(2)						1.3(3)	[4]

NOTES: (1) Caecal samples at 3-4 weeks, on farm. (2) Not stated if from one or more flocks, therefore considered as individual bird prevalence. (3) Cloacal swabs.

SOURCES: [1] Goren et al., 1988. [2] Jacobs-Reitsma, Bolder and Mulder, 1991. [3] Corrier et al., 1995. [4] Hadad and Mohammed, 1986.

6.2.4 Transport and processing

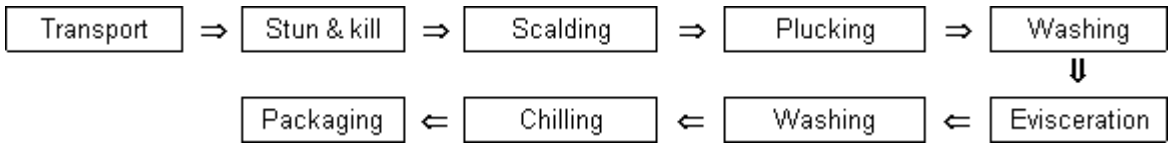
The transport and processing module describes the processing of broiler chickens, from the point of leaving the farm to the time the finished product leaves the slaughterhouse. The outputs of this step should be an estimate of (i) the prevalence of *Salmonella*-contaminated product, and (ii) the numbers of organisms per contaminated product unit.

Transport and processing steps

Overview

There are many different sub-modules within this stage, some of which increase or decrease the level of *Salmonella* contamination. Figure 6.3, from Eley (1996), summarizes the main steps of the process. This discussion focuses on transport, stun-and-kill, scalding, de-feathering (plucking), evisceration and chilling, although the other operations are also briefly mentioned.

Figure 6.3. A flow chart describing transport and processing of raw poultry meat (from Eley,1996)



Many sources give detailed descriptions of the processing of poultry (e.g. Geornaras and von Holy, 1994; ACMSF, 1996). Each stage can potentially increase or decrease the prevalence of *Salmonella* in broilers, or increase or decrease the numbers of organisms on the exterior of the broiler chicken carcass, or a combination. Overall, it is probable that the stages will be similar in all regions of the world, although the changes in microbial load occurring at each step can differ, depending on the facilities, technologies and hygienic practices employed.

Transport

During transportation, birds are often stored in open crates that are placed on top of each other; thus, faeces can drop from an upper crate to a lower crate and cause cross-contamination. The stress of transport associated with factors such as vehicle conditions, length of journey, temperature and road conditions, will increase faecal excretion (and hence *Salmonella* excretion in *Salmonella*-positive birds) and therefore the possibility of cross-contamination is increased (ACMSF, 1996). There is an additional problem if the crates used are not thoroughly cleaned and disinfected between each collection of birds.

Stun and kill

Birds are stunned when their heads are submersed into water within which there is an electrical current. They are then killed by exsanguination. These procedures have not been identified as major cross-contamination steps. A second, more modern technique is using a mixture of gas, which is also unlikely to be a significant cross-contamination step.

Scalding

Scalding facilitates the removal of feathers. Birds are immersed in water, the temperature of which can depend on whether the bird is to be sold fresh or frozen. A scald tank with water that is too hot can cause discoloration of the skin, so broilers to be sold fresh are scalded at a lower temperature of 50-52°C (soft-scald), whereas birds to be sold frozen are scalded at higher temperatures, 56-58 C (hard-scald) (ACMSF, 1996). The temperatures have important implications of *Salmonella.* In particular, some *Salmonella* species may remain viable in the scald tanks for long periods (ICMSF, 1996). As a result, there is potential for cross-contamination.

The addition of chemicals to the scald tank water may reduce the potential for pathogen survival and hence cross-contamination. However, in certain areas of the world (e.g. Europe) regulations may not permit such practices due to the requirements to use only potable water and to demonstrate that no residues remain on the carcass.

There are a number of options for the mechanical system used for scalding, including spray systems, counter-current scald tanks and multi-stage scalding. More information from different areas of the world is required to assess the different systems used.

Plucking or de-feathering

During de-feathering, machinery mechanically removes the feathers from the birds using counter-rotating domes or discs that have rubber fingers mounted on them. De-feathering is regarded as a major site for contamination. In particular aerosol spread of microoganisms may occur as the feathers are removed (ACMSF, 1996). In addition, organisms can sometimes persist in machines due to inadequate cleaning.

Evisceration

Evisceration involves the removal of internal organs. Initially, the intestines remain attached so that they can be inspected. Due to this, the exterior of the bird may be contaminated if the intestines are damaged. Such damage can occur frequently since the machinery used for evisceration is not flexible with respect to the size of the bird. However, newer evisceration machines, which separate the carcass from the offal at the point where the offal becomes exposed, may overcome this problem.

Washing

Washing a carcass (in any form) should decrease the numbers of *Salmonella* residing on the exterior, although many studies have highlighted the attachment of *Salmonella* to the skin of broiler chickens during processing (e.g. Notermans and Kampelmacher, 1974, 1975). Depending on the method of washing, the prevalence of *Salmonella* may increase or decrease. For example, if washing takes place in an immersion tank, although *Salmonella* will be washed off those carcasses contaminated on their exterior, these organisms may then cross-contaminate an initially *Salmonella*-free carcass.

Chilling

The two most common methods of chilling are the immersion chiller and the air chiller. Different countries may use different chilling methods. For example, in the United States of America, immersion chilling is generally used, while in Europe immersion chilling can only be used for frozen poultry products. With immersion chilling, a counterflow current can be used such that a carcass is always moving towards cleaner water. Note that counterflow immersion chilling is a requirement of the EU, but it is not necessarily used in other parts of the world. Chlorine in the form of hypochlorite or chloride dioxide has been shown to reduce levels of cross-contamination within immersion chillers. Addition of chemicals to the chill tank is country dependent and, as with scalding, may depend on regulations. In the United States of America, in 1992, a decision was made to include chlorine in the chill tank (Waldroup et al., 1992).

Portioning and packaging

Portioning and packaging of broiler chicken products can also potentially cause cross-contamination, but it is not considered to be significant. Briefly, a chicken can be portioned either by personnel from the processing plant or by machinery. The usual order of removal is neck skin, wings, breast, backbone, thighs and drumsticks (ACMSF, 1996). Manual handling by workers during inspection for cosmetic defects in de-boned meat, such as chicken breast, can also increase the level of cross-contamination.

Data requirements

Data requirements for modelling transport and processing fall into two categories. First, data are needed to describe how the prevalence of contaminated birds, carcasses and products changes during each sequential step, and, second, data are needed to describe the corresponding changes in numbers of the pathogen per contaminated bird, carcass or product at each stage.

Change in prevalence and numbers during transport and processing will be variable in nature, due to varying conditions, handling practices and temperatures. In addition to variability, it is likely that there will be an extensive amount of uncertainty associated with each step. Therefore, ideally, data to quantify both variability and uncertainty would be useful to characterize these steps.

Many studies that have investigated the effect of processing on *Salmonella* contamination of broiler chicken only consider a single step or a few of the sequential steps. Consequently, if combining data to generate estimates of the magnitude of change, details of the sampling methods and tests used and the associated sensitivity and specificity is important. Several different methods have been employed by various researchers to determine the presence or numbers of *Salmonella*, and samples may range from carcass rinse fluids and carcass swabs, to neck skin, or intestinal contents for direct testing.

Summary of data available

Information collected for pathogen prevalence and concentration changes in and on birds during transport is limited. Studies in the late 1970s by Rigby et al. (1980b) indicated that *Salmonella* could be isolated from debris in live-haul trucks and crates before live poultry was loaded, after unloading, and after washing. In the United States of America, Jones et al. (1991a) reported that debris from 33.3% of live-haul trucks and crates were positive for *Salmonella*, and similar levels were reported by Carraminana et al. (1997) in Spain. However, these data do not provide sufficient quantitative information to use for risk modelling.

Tables 6.5 to 6.13 provide a summary of data collected for individual steps during processing, and give a snapshot of the *Salmonella* situation at the various processing steps. However, they do not monitor change directly. In Table 6.9, some data is included that shows changes that occur during one of the processing steps.

In general, most studies consider prevalence of positive birds or carcasses. Further, the extent of contamination in the surrounding environment is often investigated, such as the knife used for slaughter (Table 6.5), the scald tank water (Table 6.6), the de-featherer (Table 6.7) and the chill water (Table 6.9). Environmental data can be used to give an indication of the extent of cross-contamination and, in theory, could also be used to predict prevalence levels or numbers of organisms at a particular point. Such predictions would require appropriate mathematical techniques and might require a number of assumptions relating to, for example, the rate of transfer of

organisms at different sites. However, the limited amount of available data would mean that any predictions would be very uncertain and thus should be undertaken with caution.

Differences in prevalence resulting from different practices are considered in several studies. In particular, differences between tanks (with and without additives) has been investigated for both scalding (Humphrey and Lanning, 1987) and chilling (Surkiewicz et al., 1969; Lillard, 1980; Campbell et al., 1983; Dougherty, 1974). The studies that look at the addition of chemicals show, in general, a reduction in prevalence (Table 6.10). In addition, variation during a day of processing is investigated for scalding (Abu-Ruwaida et al., 1994), plucking (Rigby et al., 1980a) and chilling (Rigby et al., 1980a). Also in relation to time, variation from day to day and from year to year is investigated for scalding (Abu-Ruwaida et al., 1994), evisceration (Baumgartner et al., 1992) and chilling (Rusul et al., 1996). Finally, plant-to-plant variation is considered for plucking (Chambers et al., 1998) and chilling (Lillard et al., 1990). Few of the studies on individual processing steps consider the number of organisms per bird. In fact, the only results relate to chilling (Surkiewicz et al., 1969; Dougherty, 1974; Waldroup et al., 1992). Although data for prevalence and numbers of organisms are available for individual processing steps, using these data to estimate levels of change requires additional assumptions because the data have been generated from different studies and thus there is no baseline value from which to commence estimation (Table 6.11).

Data relating to changes in prevalence and numbers of organisms are given in Table 6.9 and Table 6.11. Most of this data focuses on changes in prevalence; only one considers changes in numbers (Campbell et al., 1983). Of these studies, Abu-Ruwaida et al. (1994) and Lillard (1990) consider changes throughout the significant stages of processing. Abu-Ruwaida et al. (1994) also consider day-to-day variation, but their results give 100% prevalence at all points and thus would not be suitable for modelling change. The remaining studies in Table 6.11 commence later in processing and thus the problem of no baseline information from which to start, again arises. For example, the investigations by James et al. (1992a, b) commence after defeathering and so the prevalence level at the point of entry into the processing plant is unknown. These studies could, however, be used to look at change from one point to the next.

General conclusions on changes could be made from this data, but much of it is old and thus would require careful consideration within an exposure assessment. In particular, the effect of changes in practices and regulations would have to be investigated. Finally, Table 6.12 presents data on prevalence of *Salmonella* on finished products, at the end of processing. It is evident that it is difficult to combine these data for a risk assessment, as the different studies have used different sample types and analytical methods. Very few studies have quantified the numbers of *Salmonella*, and these are shown in Table 6.13 for whole carcass.

Table 6.5 Data collected at stun and kill processing stage.

Sample	No. tested	% *Salmonella*-Positive	Enumeration (average of positive samples	Reference (Country)
Throat-cutting knife	20	50		Carraminana et al., 1997 (Spain)
Feathers				Kotula and Pandya, 1995 (USA)
Breast	40	75	7.2 "0.2 log CFU/g	
Thigh	40	53	6.5 "0.2 log CFU/g	
Drum	40	55	6.5 " 0.2 log CFU/g	
Skin				Kotula and Pandya, 1995 (USA)
Breast	40	45	6.3 "0.2 log CFU/g	
Thigh	40	30	5.9 "0.2 log CFU/g	
Drum	40	27	5.8 "0.2 log CFU/g	
Foot	40	55	5.8 "0.2 log CFU/g	

Table 6.6 Data collected at scalding processing stage.

Sample	Number tested	% *Salmonella*-positive	Enumeration (average of positive samples)	Reference (Country)
Tank Water	15	100	13.9 "13.4 MPN/100 ml	Humphrey and Lanning, 1987 (UK)
Tank Water + NaOH	15	27	3.0 "2.3 MPN/100 ml	
Tank Water - Entry	4	NS(1)	2.9 log CFU/ml	Abu-Ruwaida et al., 1994 (Kuwait)
Tank Water - Middle	4	NS	2.3 log CFU/ml	
	4	NS	2.1 log CFU/ml	
Tank Water - Exit	4	NS	2.3 log CFU/ml	
	4	NS	2.3 log CFU/ml	
Tank Water	20	75		Carraminana et al., 1997 (Spain)
Carcass, 52°C scald	NS		3.0 log MPN per carcass	Slavik, Jeong-Weon and Walker, 1995.
	NS		3.17 MPN per carcass	
	NS		3.09 MPN per carcass	
Carcass, 56°C	NS		3.16 MPN per carcass	
	NS		3.17 MPN per carcass	
	NS		3.34 MPN per carcass	
Carcass, 60°C	NS		3.50 MPN per carcass	
	NS		3.48 MPN per carcass	
	NS		3.36 MPN per carcass	

Note: (1) NS = not stated

Table 6.7 Data collected at de-feathering processing stage.

Sample	Number tested	% *Salmonella*-positive	Reference (Country)
De-featherer swabs			Rigby *et al.*, 1980a (Canada)
Before start-up	3	33.3	
Coffee break	3	100.0	
End of shift	3	66.7	
Crop swabs	273	2.2	Chambers *et al.*, 1998 (Canada)
(post-de-feathering)	362	5.8	
De-feathered carcass rinse	6	83.3	Fuzihara, Fernades and Franco, 2000 (Brazil)

Table 6.8 Data collected at evisceration processing stage.

Sample	Number tested	% *Salmonella*-positive	Reference (Country)
Carcass swabs			Morris and Wells, 1970 (USA)
Pre-Evisceration	203	23.6	
Post- Evisceration	212	17.9	
Neck skin, 10-g sample(1)			Goren et al., 1988 (Netherlands)
Carcasses	3 099	11.7	
Flocks (25 birds each)	124	62.9	
Neck skin, 50-g sample(1)			Baumgartner et al., 1992 (Switzerland)
Carcasses	485	19.2	
Flocks (5 birds each)	97	47.4	

NOTES: (1) Sampled post-evisceration.

Table 6.9 Data collected at chilling.

Sample	Number tested	% *Salmonella*-positive[1]	Enumeration (average of +ve samples) if available	Reference (Country)
Carcass rinse				Lillard, 1990 (USA)
Pre-chill A[2]	40	13		
Post-chill A	40	28		
Pre-chill B[3]	40	10		
Post-chill B	40	38		
Pre-chill	48	100		Izat et al., 1989 (USA)
Post chill	103	58		
Carcass rinse				Campbell et al., 1983 (USA)
Entry final wash	108	22	1-30 MPN - 17 samples 30-300 MPN - 4 samples >300 MPN - 3 samples	
Entry chill tank	108	6	1-30 MPN - 5 samples 30-300 MPN - 0 samples >300 MPN - 1 samples	
Exit chill tank	215	12	1-30 MPN - 24 samples 30-300 MPN - 1 samples > 300 MPN - 0 samples	
Chill water 1st tank	71	20	< 1.1 MPN/ml - 14 samples)	Campbell et al., 1983 (USA)
Final tank	71	3	>1 MPN/ml - 2 samples	

NOTES: (1) Percentages rounded. (2) Inside/outside bird washer used in facility. (3) Outside bird washer only

Table 6.10 Data collected at chilling processing stage: effects of chlorine addition (Lillard, 1980).

Concentration of ClO_2 (ppm)	Time of day	Number tested	% *Salmonella*-positive[1]	MPN/ml	Number tested	% Positive[1]	MPN/g
0	a.m.	30	43	<0.4-15.8	28	21	< 0.4-48
	p.m.	30	40		28	7	
3	a.m.	24	29	<0.4	24	4	< 0.4
	p.m.	24	21		24	0	
5	a.m.	24	0	0	48	0	< 0.4
	p.m.	24	0		48	2	
20	a.m.	26	15	<0.4	26	4	< 0.4
	p.m.	26	19		26	0	
34	a.m.	22	0	0	22	9	< 0.4
	p.m.	22	0		22	0	

NOTES: (1) Percentages have been rounded.

Table 6.11 Summary of data collected for changes during processing.

Sample and site		No. positive out of no. tested (%)[2]	Reference (Country)
Cloacal and pericloacal swabs, 5 pooled			Carraminana et al., 1997 (Spain)
	Post-picking	11/20 (55%)	
	Post-vent cutting	9/20 (45%)	
	Post-evisceration	12/20 (60%)	
	Post-spray washing	7/10 (70%)	
	Post-air chilling	12/20 (60%)	
	Overall change		
Neck skin			Abu-Ruwaida et al., 1994 (Kuwait)
	Bleed (pre-scald)	11/11 (100%)[1]	

	De-feathering	11/11 (100%)	
Carcass rinse			Dougherty, 1974 (USA)
	Pre-evisceration	39/60 (65%)	
	Final product	28/60 (47%)	
Carcass rinse			Fuzihara, Fernades and Franco, 2000 (Brazil)
	Post-de-feathering	5/6 (83%)	
	Post-evisceration	4/6 (66%)	
	Post-immersion 1	5/6 (83%)	
	Post-immersion 2	5/6 (83%)	
Carcass rinse			Lillard, 1990 (USA)
	Pre-scald	16/84 (19%)	
	Post-scald	10/84 (12%)	
	Post-pick	10/84 (12%)	
	Post-evisceration	12/84 (14%)	
	Pre-chill (after wash)	12/84 (14%)	
	Post-chill	31/84 (37%)	
Carcass rinse			James et al., 1992A (USA)
	Pre-evisceration:	93/160 (58%)	
	Pre-chill	77/160 (48%)	
	Post-chill	114/158 (72%)	
	Post-cut	119/154 (77%)	
Carcass Rinse			James et al., 1992b (USA)
	Pre-evisceration	33/99 (33%)	

	Pre-chill	21/50 (43%)	
	Post-chill	23/50 (46%)	
Carcass Rinse			James et al., 1992c (USA)
	Pre-evisceration:	24/99 (24%)	
	Pre-chill	28/99 (28%)	
	Post-chill	24/49 (49%)	
Carcass Rinse			Jones et al., 1991b
	After chilling	6/57 (11%)	
	At packaging	3/14 (21%)	
Swab - post-scalding		Day 1: 0%	Patrick, Collins and Goodwin, 1973 (USA)
		Day 2: 0%	
		Day 3: 0%	
		Day 4: 4%	
		Day 5: 16%	
Swab - after de-feathering		Day 1: 12.5%	Patrick, Collins and Goodwin, 1973 (USA)
		Day 2: 0%	
		Day 3: 0%	
		Day 4: 4%	
		Day 5: 16%	
Swab - after chilling		Day 1: 19%	Patrick, Collins and Goodwin, 1973 (USA)
		Day 2: 4%	
		Day 3: 8%	
		Day 4: 4%	
		Day 5: 32%	
Carcass rinse/caeca cutting		129/330	McBride et al., 1980

		(39%)	
	Before scalding		
	After inspection	59/330 (18%)	
	After chilling	73/330 (22%)	
Not stated			Rigby et al., 1980b
	Unloading	311/331 (94%)	
	After chilling	11/25 (44%)	

Table 6.12 Prevalence of *Salmonella* on finished carcasses and portions.

Country & year of sampling if known	**Sample**	**Number sampled**	**Percentage positive**	**Data source**
Argentina	Carcass surface swab	96	31.3	Terisotto et al., 1990
Argentina	Carcass rinse	86	2.3	Argentina - Call for data by FAO/WHO
1994-98	Carcass rinse	39	15.4	
Austria	NS[1]	1342	3.7	EC, 1998
Austria	NS	124	2.4	EC, 1998
Austria - 1998	Skin samples	1207	22.2	EC, 1998
Austria - 1997		80	62.5	
Austria - 1996		3485	20.9	
Belgium	NS	127	28.4	EC, 1998
Brazil	25 g of meat+skin	60[2]	42.0	Fuzihara, Fernandes and Franco, 2000
Canada - 1985-86	Carcass rinse	205 (46)[3]	80.5 (89.2)[4]	Lammerding et al., 1988
1984-85		180 (47)[3]	80.6 (76.6)[4]	
1983-84		140 (41)[3]	70.0 (68.3)[4]	

Denmark	Neck skin	4985	11.1	EC, 1998
Finland - 1998	NS	384	0.52	EC, 1998
1997		611	3.1	
Ireland - 1998	NS	2 695	16.6	EC, 1998
1997		2 218	22.6	
1996		1 632	22.2	
Malaysia	Carcass rinse - Plant A [5]	12	91.7	Rusul et al., 1996
		12	75	
		20	75	
Malaysia	Carcass rinse - Plant B [5]	20	30	Rusul et al., 1996
		20	0	
		20	55	
Netherlands - 1997	Neck skin	NS	53.4	EC, 1998
1998		NS	41-50	
Netherlands	10 g fillet[6]	10 10 10 10 10 10 10	0 1 90 80 10 80 60	EC, 1998
Norway - 1998	Neck skin	7 112	0.0	ARZN, 1998
1997		7 591	0.0	
Portugal	Swabs of surface and abdominal cavities	300	57	Machado and Bernardo, 1990
Sweden - 1998	NS	1 138	0.0	EC, 1998
1997		723	0.0	
1996		581	0.0	
Sweden	Neck skin	4 010	0.02	EC 1998
Thailand	Chicken meat [7]	353	181 (51%)	Jerngklinchan et al., 1994

USA		NS	3-4%	Lillard, 1989a
USA	Cloacal swabs, giblets, whole carcasses and parts	247	4.0%	Harris et al., 1986
USDA-FSIS	Carcass rinse	1 297	20% [8]	USDA-FSIS, 1996
			11.6% (MPN)	
USA	Carcass rinse[6]	14	21.4	
Venezuela		45	49	Rengel and Mendoza, 1984

NOTES: (1) NS = Not stated. (2) Sampled from 60 individual small poultry slaughterhouses (<200 birds per day). (3) Number of lots sampled, with 5 carcasses per lot. (4) Percentage of lots positive; one or more positive carcasses. (5) Samples not specified - some pre-chill, others post-chill;. (6) Sampled prior to packaging. (7) 25 g sample of raw chicken muscle. (8) Recovered using enrichment media.

Table 6.13 Numbers of *Salmonella* on finished carcasses.

Number of samples	%	MPN per carcass[1]	Source
136	79.5	< 1	Surkiewicz et al., 1969
28	16.4	1- 30	
1	0.6	30 -300	
6	3.5	> 300	
112	25.9	0.108 ±0.279	Waldroup *et al,* 1992
112	32.1	0.172 ±0.363	
112	77.3	0.736 ±0.672	
112	38.2	0.188 ±0.259	
112	30.4	0.085 ±0.226	
109	41.9	< 12	USDA-FSIS, 1996
118	45.4	12 - 120	
24	9.2	121 - 1200	
6	2.3	1201 - 12000	
3	1.2	>12000	
99	60.7	< 12	CFIA, 2000
60	36.8	12 - 120	
2	1.3	121 - 1200	
1	0.6	1201 - 12000	
1	0.6	>12000	

Notes: (1) MPN per carcass calculated from reported values (MPN per millilitre rinse fluid) × 400 ml total rinse fluid for USDA-FSIS and CFIA results.

Data gaps

The main data gaps for processing are:

- There is limited public information on the *processing practices* followed by different countries of the world (for example, scalding or chilling methods, including addition of chemicals).
- *Quantitative data* (i.e. numbers of organisms) are limited for several processing steps.
- Many studies are old, so *more recent information* on changes in prevalence and numbers would be beneficial.

6.2.5 Retail, distribution and storage

The aim of the retail, distribution and storage module is to estimate the change in numbers of *Salmonella* on broilers after processing and before preparation and consumption by the consumer.
Retail, distribution and storage steps

When considering distribution and storage of broilers, it is assumed that the broilers are already dressed, chilled or frozen, and ready for supply. Storage can mean storage at the processing plant prior to distribution, storage at the retail outlet or central distribution centre, and storage in the home.

The distribution and storage of processed broilers can influence the bacterial load on the meat. If broiler chickens are not packaged individually, cross-contamination can occur, increasing the prevalence of salmonellae within a batch. These bacteria can also multiply as a function of the temperature, the nutrient conditions, moisture content and pH of their environment. Hence there are several variables that can influence the contamination of an individual broiler by the time it is cooked in the home, including:

- The prevalence and numbers of salmonellae on finished broiler chickens.
- The conditions of storage, including:

- storage temperature;
- relative humidity and broiler moisture;
- muscle pH;
- whether pre-packed or unpacked; and
- storage density.

- The conditions of distribution, especially

- external temperature during:

loading,
transport, and
delivery.

Data requirements and models available

There are several variables that may influence the prevalence and level of salmonellae on broiler chickens during retail, distribution and storage. For a general risk assessment framework, it is important to recognize the potential consequences of these variables in the production-to-consumer food chain. Factors such as likely temperature abuse conditions at any one stage can be utilized to model potential growth. For this, it is necessary to use predictive models that estimate the likely outcome of changes in the environmental conditions that the *Salmonella* experience. Data requirements for this purpose can be split into two main areas: choice of suitable predictive models, and the measurement of environmental changes during the retail, distribution and storage chain. In addition, studies that provide data on prevalence or numbers of organisms at retail are important in validating predictive modelling of the food chain.

Microbiological models can differ in mathematical complexity, but a complex model may not necessarily be the best choice to answer a particular risk management question (van Gerwen, 2000). The need for an accurate prediction needs to be offset by a consideration of whether the model is easy to use, whether it is robust and precise, and whether it has been validated against independent data. For example, if the objective of a risk assessment exercise is to demonstrate the most significant risk factors in a process, a simple model may have advantages over a complex model. However, if an accurate prediction of bacterial numbers is necessary, a more complex and accurate model may be preferable. In the choice of a suitable model, one must also consider the quality of the data that is going to be used to generate a prediction. If the temperature data on a process are poor, it may not be appropriate to use a complex model for the predictions. Often this can lead to a misinterpretation of the accuracy of the final prediction. The most appropriate model would be the simplest model possible for a given purpose and the given data quality, providing that it is validated and precise. A good model should also be subjected to an analysis method that quantifies the accuracy and bias of its predictions (Buchanan and Cygnarowicz, 1990). Ideally, a model should be both accurate and unbiased.

Models used in risk assessment must adequately reflect reality (Ross, Baranyi and McMeekin, 1999; Ross, Dalgaard and Tienungoon, 2000). Before predictive models are used in exposure assessment, their appropriateness to that exposure assessment and overall reliability should be assessed.

It is always possible to create a model that perfectly describes the data, simply by having a sufficiently complex model (Zwietering et al., 1991), but such models lack generality and would be *less* useful for predicting responses in other situations.

Two complementary measures of model performance can be used to assess the 'validity' of models (Ross, Baranyi and McMeekin, 1999; Ross, Dalgaard and Tienungoon, 2000). These measures have the advantage of being readily interpretable. The 'bias factor' (B_f) is a multiplicative factor by which the model, on average, over- or under-predicts the response time. Thus, a bias factor of 1.1 indicates that the prediction response exceeds the observed, on average, by 10%. Conversely, a bias factor less than unity indicates that a growth time model would, in general, over-predict risk, but a bias factor of 0.5 indicates a poor model that is overly conservative because it predicts generation times, on average, half of that actually observed. Perfect agreement between predictions and observations would lead to a bias factor of 1.

The 'accuracy factor' (A_f) is also a simple multiplicative factor indicating the *spread* of observations about the model's predictions. An accuracy factor of two, for example, indicates that the prediction, on average, differs by a factor of 2 from the observed value, i.e. either half as large or twice as large. The bias and accuracy factors can equally well be used for any time-based response, including lag time, time to an *n*-fold increase, death rate and D value. Modifications to the factors were proposed by Baranyi, Pin and Ross (1999). As discussed above, typically, the accuracy factor will increase by 0.10-0.15 for every variable in the model. Thus, an acceptable model that predicts the effect of temperature, pH and water activity on growth rate could be expected to have A_f = 1.3-1.5. Satisfactory B_f limits are more difficult to specify because limits of acceptability are related to the specific application of the model. Armas, Wynn and Sutherland (1996) considered that B_f values in the range 0.6-3.99 were acceptable for the growth rates of pathogens and spoilage organisms when compared with independently published data. te Giffel and Zwietering (1999) assessed the performance of many models for *Listeria monocytogenes* against seven datasets and found bias factors of 2-4, which they considered to be acceptable, allowing predictions of the order of magnitude of changes to be made. Other workers have adopted higher standards. Dalgaard (2000) suggested that B_f values for successful validations of seafood *spoilage* models should be in the range

0.8 to 1.3. Ross (1999) considered that, for pathogens, less tolerance should be allowed for B_f >1 because that corresponds to under-predictions of the extent of growth and could lead to unsafe predictions. That author recommended that for models describing *pathogen* growth rate, B_f in the range 0.9 to 1.05 could be considered good; be considered acceptable in the range 0.7 to 0.9 or 1.06 to 1.15; and be considered unacceptable if <~0.7 or >1.15.

In another approach to assessing model performance, the group of researchers involved in the development of the predictive modelling program Food MicroModelä proposed that validation could be split into two components: first, the model's mathematical performance ($error_1$), and second, its ability to reflect reality in foodstuffs ($error_2$) (Anon., 1998). They found that the error of a single microbiological concentration record was about 0.1-0.3 $\log_{10}$ CFU/ml. Therefore, this could be considered the standard error obtained by fitting the model. If, during comparison of the predicted data with the measured data used to generate the primary model, the standard error was greater than 0.3-0.4 $\log_{10}$ CFU/ml, then the authors suggested that the curve should only be used with caution for any secondary modelling stage. They went on to suggest that when a quadratic response surface was fitted to predicted kinetic parameters from the primary model to create the secondary model, the statistical tests should include a measure of goodness of fit. They suggested that the aim of a good model would be to achieve a standard error of no greater than 15-20%. Other suggested statistical tests were measures of parsimony (e.g. t-test), errors of prediction (e.g. least squares) and measures of robustness (e.g. bootstrap methods). The ability of a model to reflect reality in foodstuffs ($error_2$) is often assessed by conducting a review of the literature for measured data describing the kinetic parameter for prediction by the model. These data must not be the data used to generate the model. Ross (1999) suggested that validation data could be subdivided into sets that reflected the level of experimental control. Hence, data generated in a highly controlled broth system would be separated from data generated in a less controlled foodstuff. In this way, he argued that the performance of the model would not be undermined by evaluation against poor quality data or unrepresentative data. For examples of the limitations and difficulties of using validation data from the literature, see McClure et al., (1997); Sutherland and Bayliss, (1994); Sutherland, Bayliss and Roberts (1994); Sutherland, Bayliss and Braxton (1995); and Walls et al. (1996). The multiplicative factors of bias and accuracy discussed previously could be equally applied to quantification of both $error_1$ and $error_2$.

The selection of a model for a microbiological phenomenon must go further than the mathematics. It is all too easy to forget that a model is only as good as the data on which it is based. Bacteria are biological cells and as such the methodology used to enumerate their numbers greatly affects the count obtained. For this reason the predictive model should be based on replicate data using recognized enumeration methods. The use of resuscitation procedures for enumeration is particularly important when the organism has been growing near its physiological limits. Here, bacteria are often in a state of environmental stress and recovery is necessary to prevent the artificial depression of bacterial numbers. The method used to generate the data must be free from experimental artefacts that might artificially increase or decrease the bacterial count.

Growth

Bacteria multiply by a simple process of cell division, known as binary fission. A single bacterial cell reaches a stage in its growth when it undergoes a process that results in the single cell dividing into two daughter cells. The growth of bacterial populations therefore follows a predictable cycle that involves a period of assimilation - called the lag phase; a period of exponential growth - called the exponential phase; and a period of growth deceleration and stasis - called the stationary phase. Growth curves are often described kinetically by three variables: initial cell number (N_0), lag time

(l) and specific growth rate (m), which can also be used to determine the generation time or doubling time of the population. Note that this simple description does not take the stationary phase into account. Prediction of the stationary phase is not always necessary for risk assessment, although a maximum population density parameter is often useful as an endpoint for the prediction of the exponential phase of growth. The values of these variables change with environmental conditions, including temperature, pH, water activity (a_w), nutrient state and the presence and concentration of preservatives. Studies of the growth of bacteria can generate different types of data. Kinetic data, involving the enumeration of bacteria during the growth cycle, describe the shape of the population growth curve in response to a specific set of growth conditions. Probabilistic data, involving measurement of simple growth or no-growth characteristics of the bacterial population, describe whether or not the bacteria will grow under certain growth conditions.

Growth Models

Microbiologists recognize that not all equations that are applied to bacterial processes can be considered models. A kinetic model should have a sound physiological basis (Baranyi and Roberts, 1995). This distinction has not always been made in the literature, and the word "model" has been invariably used to describe empirically-based curve fitting exercises.

Growth models increase in complexity from primary models that describe a population response, e.g. growth rate and lag time, to secondary models that describe the effect of environmental factors on the primary response, e.g. temperature and pH.

For the growth process of bacteria, an example of a simple primary model is shown in Equation 6.1.

$$N = N_0 \cdot \exp(m(t-l)) \quad \text{Equation 6.1}$$

Where N = number of bacteria; N_0 = initial number of bacteria; m = specific growth rate; and l = lag time.

This type of model could be applied to growth data to determine the primary kinetic parameters for specific growth rate and lag time for the given set of environmental growth conditions under which the data was generated.

There are several primary models that have been used routinely to describe the growth of bacteria. Examples are the Gompertz equation (Gibson, Bratchell and Roberts, 1988; Garthright, 1991), which is an empirical sigmoidal function; the Baranyi model (Baranyi and Roberts, 1994), which is a differential equation; and the three-phase linear model (Buchanan, Whiting and Damart, 1997), which is a simplification of the growth curve into three linear components.

Secondary growth models based on primary models have been created by replacing the term for specific growth rate and the term for lag time with a function that described the change of these response variables with respect to environmental factors such as temperature, water activity and pH. Examples are the non-linear Arrhenius model - where the square root model relates the square root of the growth rate to growth temperature (Ratkowsky et al., 1982) - and the response surface model. In the case of the simple model example in Equation 6.1, an example secondary model can be used to describe the growth of a bacterial population when temperature changes (Equation 6.2).

$N = N_0 \cdot \exp(\mu_{TEMP} m(t - \mu_{TEMP} l))$ Equation 6.2

Where N = number of bacteria; $N_0 \cdot$ = initial number of bacteria; m = specific growth rate; l = lag time; and μ_{TEMP} = mathematical function for the effect of temperature, such as a quadratic equation.

This type of model could be applied to growth data at different temperatures and would allow the calculation of the number of bacteria after a given growth period when temperature changes during that growth period. Secondary models developed from primary models are more useful than primary models alone for the quantification of risk, providing that the environmental factors influencing growth can be measured dynamically.

Growth Models for Salmonella *in Chicken Meat*

An ideal growth model for *Salmonella* should take into account the general issues raised previously about model selection, but, in addition, it should be tailored for the product under study. The ideal growth model would aim to encompass the variable limits for temperature, pH and a_w shown in Table 6.14, for which *Salmonella* are estimated to grow.

In the case of *Salmonella* in broilers, the model either should have been developed using data describing *Salmonella* growth in chicken meat, or at least be validated against real product data.

Table 6.14. Limits for growth of *Salmonella* (ICMSF, 1996)

Conditions	**Minimum**	**Optimum**	**Maximum**
Temperature (°C)	5.2	35-43	46.2
pH	3.8	7-7.5	9.5
Water activity (a_w)	0.94	0.99	>0.99

Table 6.15. Growth models for *Salmonella*

Salmonella serotype	Growth medium	Temp. range (°C)	pH range	Other conditions	Primary model	Secondary model	Reference
Typhimurium	Milk	10-30	4-7	a_w 0.9-0.98. Glucose as humectant	Non-linear Arrhenius	Quadratic response	Broughall and Brown, 1984
Typhimurium	Laboratory media	19-37	5-7	Salt conc. 0-5%		Quadratic response	Thayer et al., 1987
Mixed Stanley, (Infantis and Thompson)	Laboratory media	10-30	5.6-6.8	Salt conc. 0.5-4.5%	Gompertz	Quadratic response	Gibson, Bratchell and Roberts, 1988
Typhimurium	Laboratory media	15-40	5.2-7.4	Previous growth pH 5.7-8.6	2 phase linear	Quadratic response	Oscar, 1999a
Typhimurium	Cooked ground chicken breast	16-34		Previous growth temp. 16-34°C	2 phase linear	Quadratic response	Oscar, 1999b
Typhimurium	Cooked ground chicken breast	10-40		Previous growth salt 0.5-4.5%	2 phase linear	Quadratic response	Oscar, 1999c

Published growth models for *Salmonella* predict growth as a function of temperature, pH, water activity (a_w) and previous growth conditions. Table 6.15 summarizes the basis of several models.

The models of Broughall and Brown (1984) and Thayer et al. (1987) do not appear to have been validated by the authors. Validation is included for the other four models. Gibson, Bratchell and Roberts (1988) validated their model against growth data generated using pork slurry and data published in the literature. The model predictions were in good agreement with the observed data. The greatest variance was found at the extremes of the model, with low temperature or high salt concentration. This model has the advantage of being based on a considerable quantity of experimental observations and covers a wide selection of environmental growth conditions. However, the authors did not validate the work against observed data in chicken meat. The work reported by Oscar (1999a, b and c) concluded that previous growth temperature, pH and salt concentration had little effect on the estimates of specific growth rate and lag time for *Salmonella* Typhimurium. The author also demonstrated that it was possible to develop models in a food matrix including chicken meat, and hence these are useful for the purposes of this exposure assessment.

Survival

Under stress conditions, bacteria will either remain in a state of extended lag or may die slowly. Studies on the survival of *Salmonella* under stress conditions are limited. The number of *S.* Enteritidis was shown to remain constant during the storage of chicken breast at 3°C under a range of modified atmospheres over a 12-day study period (Nychas and Tassou, 1996). However, growth of enterobacteriaceae, including *Salmonella*, on naturally-contaminated chicken meat occurred at 2°C after 3 days in 30% CO_2, and after 5 days in 70% CO_2, with numbers increasing by 3 log cycles after 15 and 23 days, respectively (Sawaya et al, 1995). These investigators noted that *Salmonella* composed about 12% of the total enterobacteriaceae microflora, and the proportion remained constant throughout storage. It is possible that *Salmonella* growth is enhanced by the presence of competitive microflora. Hall and Slade (1981) carried out an extensive study of the effect of frozen storage on *Salmonella* in meat. In chicken substrate, the numbers of *S.* Typhimurium declined by 99.99% (4 log cycles) at -15°C over 168 days, and by 99.4% (2-3 log cycles) at -25°C over 336 days. Survival data for *Salmonella* have been summarized by ICMSF (1996).

Model selection for exposure assessment model

When considering broiler meat as a media for growth and survival of *Salmonella*, several factors can be simplified. At the surface of the meat, water activity might vary as a function of air moisture, chilling conditions and packaging method, but generally falls between a_w 0.98 and 0.99. The pH varies among muscle types, but is between pH 5.7 and 5.9 for breast meat and pH 6.4-6.7 for leg meat. The skin averages pH 6.6 for 25-week-old chickens (ICMSF, 1996). Poultry meat is also a rich source of nutrients such as protein, carbohydrate and fat, with essential minerals and vitamins. Consequently, it can be assumed that the growth of *Salmonella* will not be limited by the lack of available nutrients and hence the growth rate will be optimal for a given temperature within the pH and a_w limits of the poultry meat.

For the purposes of a simple exposure assessment model, the change in environmental conditions could be considered solely as a change in external temperature and chicken carcass temperature. It can be assumed that the pH of a broiler chicken will be pH 6.0 and that the water activity will be 0.99. Some appropriate models that could be used to predict changes in growth rate during retail, distribution and storage are:

- For temperatures between 10°C and 30°C, the growth model of Gibson, Bratchell and Roberts, 1988.
- For temperatures between 16°C and 34°C, the growth model of Oscar, 1999b.
- For temperatures between 4°C and 9°C, the survival model of Whiting, 1993.
- There are no appropriate models for temperatures below 4°C.

For the purposes of the current exposure assessment, the model developed by Oscar (1999b) was selected. The model was developed in chicken meat slurry and therefore took account of the interactions between the bacteria and the food matrix. In addition, the model was simple and easily applied. The author also assessed the accuracy and bias of the model by measuring the relative error of predictions against:

(i) the data used to generate the model; and

(ii) new data measured using the same strain and experimental conditions, but at intermediate temperatures not used in the data set used to develop the original model.

Median relative errors for lag time were given as 0.9% and -3% for comparisons (i) and (ii), respectively, and the median relative errors for growth rate were given as 0.3% and 6.8% for comparison (i) and (ii), respectively. The predictions for either parameter were unbiased. The accuracy of the model was deemed to be within accepted guidelines, as discussed above.

Temperature data characterizing retail, distribution and storage

Providing that suitable secondary kinetic models are available, it is necessary to examine the change in the environmental conditions with time during the retail, distribution and storage chain. The most common studies involve the use of temperature probes to measure the changes in product temperature during a process. For broiler chickens, the measurement of external surface and deep muscle temperatures may be used to characterize the growth or survival of *Salmonella* at these locations. Sampling can be used to measure pH and water activity changes with time, but these types of study are rarely conducted. Alternatively, thermodynamic models can be used to predict the temperature of a product given the external temperature and time. To ensure the predictions are consistent with measured data, caution must be exercised when using this approach.

Temperatures in the retail, distribution and storage chain tend to become less controlled from processor to consumer. Temperature and time studies of storage at the processing plant, distribution to the retailer and storage at the retailer often remain the unpublished property of the broiler industry or retailers. Few studies, if published, carry detailed data. Temperature and time studies of transport and storage by the consumer tend to be carried out by food safety organizations and are also largely unpublished. This presents problems for risk assessment unless access to these data can be arranged. Even with access to data in commercial organizations, it is often unlikely that data will be released that characterizes poor practice.

Data requirements and the data available

Growth modelling

Calibrated equipment should always be used for measuring time and temperature profiles of processes. Studies can be of a single step, such as storage at the retail stage, or be of multiple steps. In both cases, it is important to measure the environmental temperature, the external product temperature and the internal product temperature. Profiles should be measured in more than one product and, in the case of multi-step measurements, careful notes on the start and end times of the individual steps must be kept. It is important, where possible, to follow the same product throughout a multi-step process so that measurements from one step to the other can be related. Wherever possible, data should be analysed statistically to determine the within-step and step-to-step variability. If continuous measurement is not possible using a temperature data logger, then as many real-time measurements as possible should be made using a temperature probe.

Few thermal profile data for retail, storage and distribution were provided by FAO/WHO member countries as a result of the call for data. No actual data were found in the literature, although profiles were shown in graphic form in some studies. As an example, time and temperature data were kindly provided on whole broilers by Christina Farnan (Carton Group, Cavan, Republic of Ireland). These data are summarized in Tables 6.16 and 6.17.

When carrying out a quantitative exposure assessment, it is important to access national data. Data should be requested from national broiler processors and retailers.

Table 6.16. Summary of chilled chain data from Carton Group.

Location of product (probed chicken in box of 5 carcasses)	**Trial 1: 1000-g broilers**			**Trial 2: 2300-g broiler**		
	Time (minutes)	Average temperature (°C)		Time (minutes)	Average temperature (°C)	
		surface	muscle		surface	muscle
Primary chill	0	-	36	0	-	41
Packing hall	43	-	7.0	80	-	10.2
Boxed	55	-	7.0	85	-	10.2
Blast chill	57	-	7.0	100	-	10.2
Storage chill	75	1.1	2.0	155	5.0	6.2
Dispatch lorry	717	1.1	1.1	230	4.0	4.0
Depart plant	755	1.1	1.1	315	3.0	2.4
Arrival at retailer	945	1.7	1.1	500	3.0	0.7
Storage at retailer (back chill)	968	2.3	1.1	505	3.0	0.7
Storage at retailer	>48 hours	Max. 3.7	Max. 3.3	N/A	N/A	N/A

SOURCE: Data supplied by Christina Farnan, Carton Group, Cavan, Republic of Ireland.

Table 6.17. Summary of frozen chain data from Carton Group.

Location of product (probed chicken in box of 5 carcasses)	**Trial 2: 2300-g broiler**		
	Time (minutes)	Average temperature (°C)	
		surface	muscle
Boxed	0	19.5	2.8
Into blast freezer	1	19.5	2.8
Out of blast freezer	3925	-34.7	-32.8
Into cold store	3930	-33.9	-32.8
Depart plant	4140	-32.1	-32.3
Arrive central distribution	4180	-32.0	-31.6

SOURCE: Data supplied by Christina Farnan, Carton Group, Cavan, Republic of Ireland.

Transport and storage temperatures during consumer handling of products can vary greatly. In the United States of America, a study was carried out in 1999 to quantify this process (Audits International, 1999). This work is a good template for carrying out similar research in other nations.

Data were generated on retail backroom storage temperature, display case temperature, transit temperature, ambient temperature in the home, home temperature and home temperature after 24 hours. Tables 6.18 and 6.19 summarize the data. These example data were not generated in chicken but may be used as a guide.

These data can be useful to estimate growth or survival, or both, in a deterministic assessment, or as a basis for probability distributions for time and temperature in stochastic modelling.

Table 6.18. Summary of consumer transport and storage study on chilled products including meat

Location	Average time (minutes)	Average temperature (°C)	Maximum time (minutes)	Maximum temperature (°C)
Retail backroom cold store air	N/A	3.3	N/A	15.5
Product in retail backroom cold store	N/A	3.3	N/A	16.6
Product in retail display refrigerator	N/A	4.0	N/A	14.4
Product from retail to home	65	10.3	>120	(max. 36.6 at home)
Product in home refrigerator (after 24 h)	N/A	4.0	N/A	21.1
Home ambient temp	N/A	~27.0	N/A	>40.5

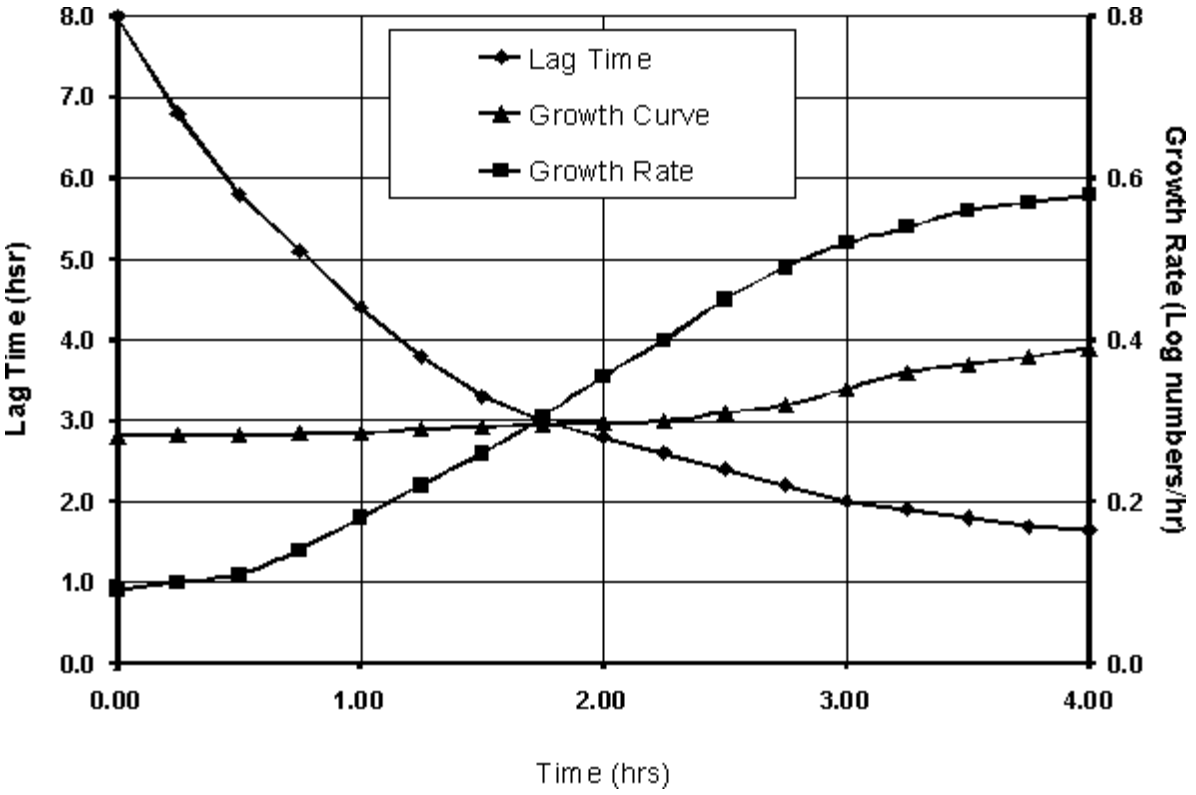
NOTES: N/A = Not available. SOURCE: Audits International, 1999.

Table 6.19: Summary of consumer transport and storage study on frozen dairy products

Location	Average time (minutes)	Average temperature (°C)	Maximum time (minutes)	Maximum temperature (°C)
Product in retail display freezer	N/A	-12.9	N/A	6.6
Product from retail to home	51	-8.4	>120	20
Product in home refrigerator (after 24 h)	N/A	-15.9	N/A	8.9
Home ambient temp	N/A	~27.0	N/A	>40.5

NOTES: N/A =Not available. SOURCE: Audits International, 1999.

Figure 6.4. Relationship of lag time and growth rate with increasing temperature as a function of time.

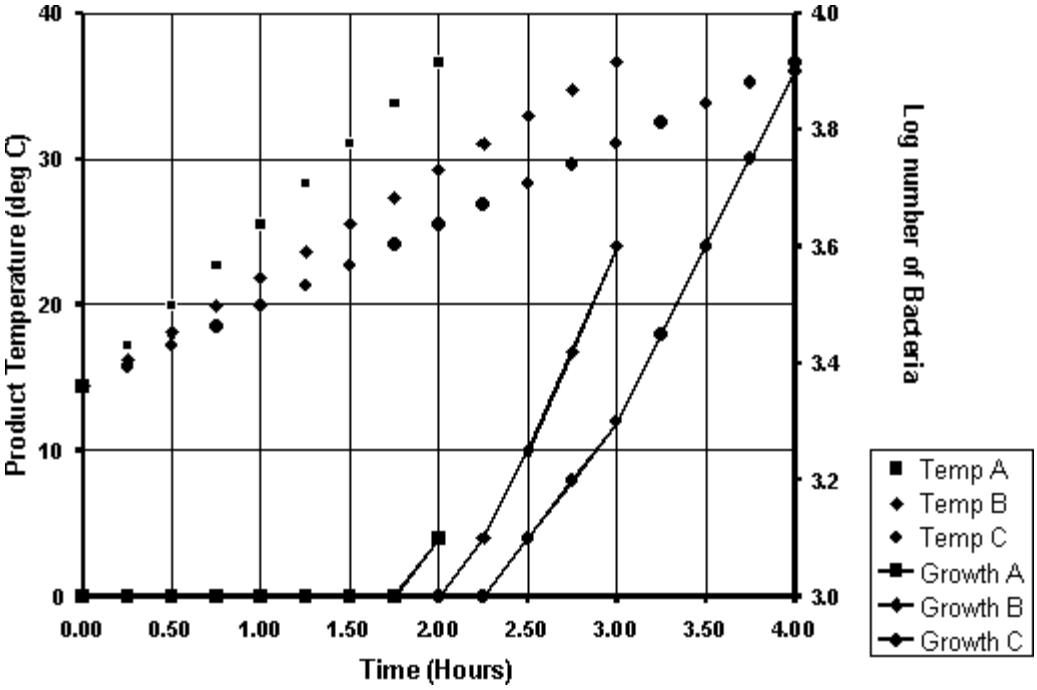


To illustrate a deterministic approach, the data in Table 6.19 can be used to demonstrate the predicted effect on the growth of *Salmonella* in a product during transport from the retail store to the consumer's home. For this example, let the number of salmonellae on the product be 1000 CFU at the start and assume that the temperature increases linearly over the transport period. It is also assumed that the growth of the organism starts at the beginning of the transport period rather than in the store. The Oscar growth model (1999b) can be used to calculate the predicted growth pattern. The model calculates the lag time and specific growth rate for salmonellae as a function of time and temperature. The organism cannot grow until the elapsed time exceeds the lag period. As temperature increases, the lag period decreases and the specific growth rate increases. This is shown in Figure 6.4. Until the elapsed time is equal to the lag period the numbers of bacteria are fixed at the starting number (in this case 1000 CFU). Figure 6.4 shows that after 2.5 hours the lag period has been exceeded and the organism is allowed to grow at a rate set by the specific growth rate.

To calculate the relationship shown in Figure 6.4, the steps followed were:

- The thermal profile was divided into equal time and temperature blocks of 15 minutes.
- For each block, the model was used to calculate the lag time and specific growth rate.
- The growth curve was fixed at the starting cell number until the elapsed time was greater than the lag period (2.5 hours).
- After completion of the lag period, the growth at each time and temperature block was calculated by dividing the specific growth rate by the growth period.
- The increases in bacterial numbers predicted at each time and temperature block were summed to give the final increase in numbers after completion of the thermal profile.

Figure 6.5. The predicted effect on the growth of *Salmonella* of temperature increase during consumer transport of product to home.



Data in Table 6.18 suggest that in a worst case scenario, a product at 14.4°C in the store could reach 36.6°C during transport over a period greater than 2 hours. Using the same approach, the effect of journey time on the growth of salmonellae can be demonstrated. Figure 6.5 shows the predicted consequences of a journey that results in a product at 14.4°C reaching 36.6°C over a 2-, 3- or 4-hour journey time.

The Oscar model (1999b) has a temperature range of 16°C to 34°C and calculations were only performed within this temperature range. It must be emphasized that predictive models should not be extrapolated beyond their boundaries.

Retail level prevalence and concentration data

Data on concentration and prevalence at the retail level could be useful as a starting point for an exposure assessment. Tables 6.20a, 6.20b and 6.20c summarize the data reported and collected to date. It is important to note, however, that study design details are lacking and the future collation of such details should be recommended.

Table 6.20a. Reported prevalence of *Salmonella* in poultry at retail.

Type of Product	Number sampled	Percentage positive	Reference (Country), and year of sampling, if reported
Fresh or frozen poultry (NS)[1], domestic and imported	322	7.8	Kutsar, 2000 (Estonia), FAO/WHO call for data. No year.
Imported frozen	151	7.3	Al Busaidy, 2000 (Sultanate of Oman), FAO/WHO call for data. No year.
Broiler chicken and hens	1186	17.3	BgVV, 2000 (Germany) - 1999
Supermarket, frozen	52	2.0	Wilson, Wilson and Weatherup, 1996 (Northern Ireland, UK). No year.
Supermarket, chilled	58	5.0	
Butcher, frozen	6	0.0	
Butcher, chilled	24	25.0	
Giblets, skin and carcass samples			ACMSF, 1996 (UK)
Chilled	281	33.0	- 1994
Frozen	281	41.0	- 1994
Chilled	143	41.0	- 1990
Frozen	143	54.0	- 1990
Chilled	103	54.0	- 1987
Frozen	101	64.0	- 1987
Frozen	100	79.0	- 1979/80
Poultry products (NS)			EC, 1998
	1931	17.5	Austria - 1998
	286	10.6	Denmark - 1998
	404	5.7	- 1997
	462	9.5	- 1996

	114	0.88	Finland - 1998
	100	3.0	- 1996
	1207	22.2	Germany - 1998
	3062	22.2	- 1997
	3979	27.2	- 1996
	198	5.6	Greece - 1998
	69	0	- 1997
	51	47.1	Ireland - 1998
	104	14.4	Italy - 1997
	1010	20.2	Netherlands - 1998
	1314	29.2	- 1997
	1196	32.8	- 1996
	31	0	Northern Ireland (UK) - 1998
	314	12.1	- 1996
	73	34.3	Portugal - 1998
	34	23.5	- 1997
	562	36.8	UK - 1996
Poultry breast meat			Boonmar et al., 1998 (Bangkok, Thailand). No year.
5 traditional open markets	50	80	
5 supermarkets	50	64	
Carcasses, at distribution centre for large food chain [Positive if >1CFU/100 cm^2 or/25g]	123	24.4	Uyttendaele et al., 1998 (Belgium) 1996
	131	17.6	- 1995
	114	27.2	- 1994
	81	19.7	- 1993

Chicken portions [Positive if >1CFU/100 cm^2 or/25g]	153	49.0	- 1996
	117	39.3	- 1995
	112	41.1	- 1994
	101	35.0	- 1993
Carcasses, retail markets. [Positive if >1 CFU/100 cm^2 or/25 g]	133	33.8	Uyttendaele, de Troy and Debevere, 1999 (Belgium, France, Italy, the Netherlands, UK). No year.
Chicken products	41	82.9	
Chicken portions	225	51.1	
Carcasses, cuts, processed			
with skin	183	47.0	
without skin	182	34.6	
Carcasses, cuts, processed	279	54.0	Belgium. No year.
	434	33.6	France. No year.
	13	30.8	Italy. No year.
	2	0.0	Netherlands. No year.
	44	47.7	UK. No year.
Wet market - carcasses	445	35.5	Rusul et al., 1996 (Malaysia). No year.
- intestinal content	54	11.0	
Open Market - chicken meat	164	87.0	Jerngklinchan et al., 1994 (Thailand). No year.
gizzard	14	86.0	
liver	94	91.0	
heart	8	88.0	
Supermarket - chicken meat	188	77.0	
gizzard	31	77.0	

liver	36	28.0	
heart	38	87.0	
Chicken meat, supermarkets	41	7.3	Swaminathan, Link and Ayers, 1978 (USA). No year.
Chicken meat	283	10.6	ARZN, 1998 (Denmark). No year.
Products (drumsticks, wings, livers, fillets, etc.)	81	54	de Boer and Hahn, 1990 (the Netherlands). No year.
Products (drumsticks, wings, livers, fillets, etc.)	822	33.3	Mulder and Schlundt, in press (the Netherlands) - 1995
	907	32.5	- 1994
	840	32.1	- 1993

NOTES: NS = not stated.

Table 6.20b. Prevalence and concentration.

Sample	Country	Year of Sampling	No. positive/No. sampled	Numbers on positive carcasses	Reference
Frozen thawed carcasses	USA		2/12 (16.7%)	0.23 MPN/m)	Izat, Kopek and McGinnis, 1991; Izat et al., 1991
			3/12 (25%)	0.06 MPN/m)	
			3/12 (25%)	0.09 MPN/m)	
			3/12 (25%)	0.07 MPN/m)	
			6/12 (50%)	0.34 MPN/m)	
			4/12 (33.3%)	0.05 MPN/ml	
Carcasses, after chill(1)	Canada	1997-98	163/774 (21.1%)	<0.03MPN/ml: 99	CFIA, 2000
			C.I. 18 -24	0.03 - 0.30: 60	
				0.301 - 3.0: 2	
				3.0 1 - 30.0: 1	
				>30.0: 1	
Carcass rinse, after chill(2)	USA	1994-95	260/1297	Per cm^2	USDA-FSIS, 1998
Carcass rinse, after chill	USA	[1992]	29/112 (25.9%)		Waldrop et al., 1992

Notes: (1) Immersion, no chlorine. (2) Immersion, unspecified level of chlorine present in chill water.

Table 6.20c. Numbers of *Salmonella* on whole carcasses at retail.

Type of product	Number of samples	%	MPN(1)	Direct count/10 cm^2
Fresh	40	89	0 - 10	<100
	4	9	11 - 100	
	0	0	101 - 1100	
	1	2	> 1100	
Frozen	30	68	0 - 10	
	10	23	11 - 100	
	2	4	101 - 1100	
	1	2	> 1100	
	1	2	No MPN	

Notes: (1) MPN = Most probable number per carcass. Source: Dufrenne et al., 2001.

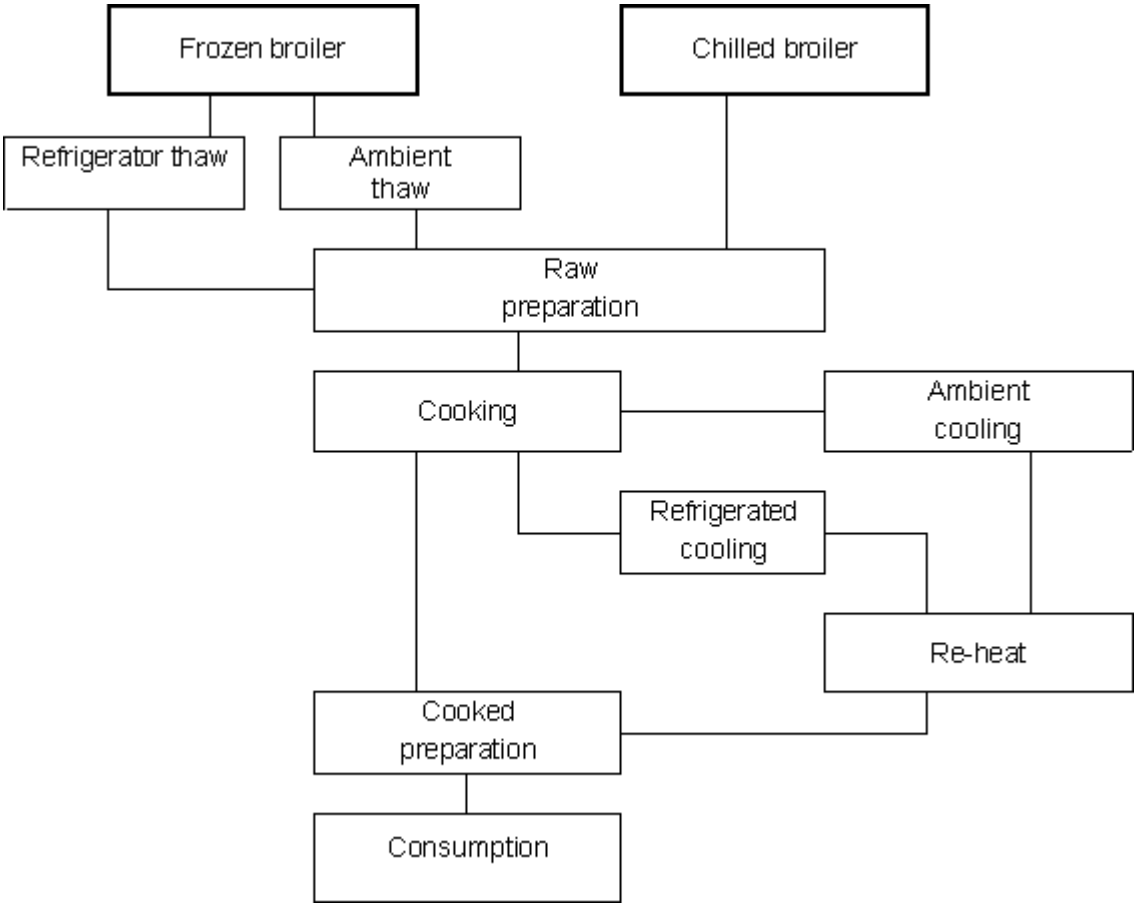
6.2.6 Preparation

The aim of the preparation module is to estimate the numbers of salmonellae in broiler chicken meat prior to consumption.

Preparation steps

The preparation process begins at the point the chilled or frozen broiler chicken, whole or portions, is removed from the refrigerator or freezer, respectively. Frozen whole broilers and portions must be thawed, but then preparation steps for both frozen and chilled whole broiler are essentially the same. Figure 6.6 summarizes common preparation steps. In the following module description, the case of whole broilers is considered. However, a similar approach can be applied to chicken portioned, provided that time and temperature data are available to characterize the storage, thawing, preparation and cooking pathways.

Figure 6.6. Preparation pathways



Thawing

Retailers of frozen poultry recommend that a frozen broiler chicken should be thawed overnight in a refrigerator. This is to maintain the surface of the broiler at a low enough temperature to prevent the growth of bacteria. However, in reality, broilers are often thawed outside a refrigerator or in an oven or microwave. If thawed at ambient temperature, the surface of the broiler can approach moderate ambient temperatures and because thawing often requires several hours there is potential for bacteria to grow on the surface. Thawing a frozen broiler in a heated oven takes a shorter period but surface

temperatures are higher and bacteria grow more quickly. Thawing a broiler in a microwave creates an uneven heating pattern that again raises temperature and growth rate. This is offset by the reduced time-scale, although uneven thawing can leave frozen areas of the meat that can prevent adequate cooking. The thawing process also causes drip loss and this contaminated fluid can be an additional hazard during raw preparation.

Raw Preparation

Raw preparation involves a considerable amount of handling and hence cross-contamination is a serious hazard. Bacteria present on the meat can be transferred to hands, cloths, utensils and surfaces during the process. These items then become a source of contamination for ready-to-eat food in the kitchen. The preparation of the broiler chicken will also influence the cooking step. For example, a stuffed bird may take longer to cook than one where the cavity is open.

Cooking

Cooking is a critical step in the process. Thorough cooking should kill all the bacteria on and in a broiler. However, low cooking temperatures or short cooking times can result in undercooked meat with potentially live bacteria. The probability that bacteria will survive in these circumstances depends on the degree of undercooking and the bacterial load on the raw broiler. If cooked correctly, the meat should be free from bacteria at the point of consumption.

Cooling and re-heating

It is not uncommon for cooked meat portions, or even the whole cooked broiler chicken, to be cooled, stored, then eaten later, either re-heated or not. If carried out correctly, this process should not be hazardous. However, if the cooked meat is not cooled in a refrigerator but left to cool at ambient temperature, then any bacteria that survive cooking or are transferred by cross-contamination can grow, often quickly. If the meat is not re-heated then there is no opportunity to reduce the bacterial load. If the meat is re-heated thoroughly, then these bacteria will again be killed and the product should be safe. Obviously any less than thorough re-heating, as with cooking, may fail to kill all the bacteria. If the product is cooled quickly to refrigerator temperatures and stored chilled, then the product should be safer than cooling at ambient temperatures. There are numerous documented cases of food poisoning attributable to poorly controlled cook and chill processes.

Meal preparation

Preparation of the cooked broiler can involve processes like carving and dressing. The main hazard here is the potential to contaminate the meat with bacteria. Cross-contamination caused by poor hygienic practices may introduce bacteria onto a product that should be free from them following a correct cooking process.

Data requirements, models available and data

General hygienic practice studies

Table 6.21 indicates research into general food safety practices in the home. These studies give an indication of how many consumers may handle food in an unsafe manner. The studies selected for Table 6.21 are a cross-section of the types of studies that have been conducted. Data from this type

of work can be used in an exposure assessment to evaluate the probability of unsafe practices occurring in the home.

Table 6.21. General quantitative surveys of hygiene in the home.

Study	Subject numbers	Data type	Comments
Worsfold and Griffith, 1995	NA(1)	Riskscores	Model for assessing food safety behaviour
Altekruse et al., 1995	1620	% respondents to food safety questions	Telephone survey
Scott, 1996	NA	Various	Review article
Worsfold and Griffith, 1997a	108	% subjects displaying unhygienic practices	Direct observation *in situ*
Jay, Komar and Govenlock, 1999	40	% subjects displaying unhygienic practices	Direct observation via video
Schutze et al, 1999	NA	*Salmonella* serotype, culture sources %	Investigation follow-up after salmonellosis diagnosis.

Notes: (1) NA = not applicable.

Thawing

For an exposure assessment model, any changes in the number of salmonellae during the thawing process can be predicted using the survival and growth models discussed in Section 6.2.5, provided that adequate data are available to describe the temperature changes.

Studies on the thawing of broilers are often carried out by broiler chicken processors and retailers for the development of safe thawing instructions. These data can often be obtained by risk assessors on application to the company. Unlike freezing and chilling where the warmest part would be the deep muscle, the reverse is true of the thawing process. It is important therefore to measure the thermal profile at the surface of the broiler as well as in the deep muscle (Table 6.22). Unfortunately, these measurements are rarely taken. Such is the emphasis for developing thermal profiles for cooking where the coldest spot is measured (the geometric centre), that workers often use the same approach to measure thawing. In these studies the emphasis is on whether thawing is complete, which is essential for the subsequent cooking process. However, few data in the literature are available to describe the surface temperature where *Salmonella*, if present, can begin to multiply. It is possible to use thermodynamic models for thermal diffusivity to calculate a surface temperature given air temperature (Brown et al., 1998).

Table 6.22. Example of data on thawing of a 2300-g raw, frozen broiler chicken carcass.

Process step	Deep muscle temp. (°C)	Surface temp. (°C)
Start thaw in packaging at ambient temperature	-17.3	-16.0
After 24 hours in packaging	-1.9	1.4
After 29 hours (with 5 hours in ambient conditions, removed from packaging)	0	11.3

SOURCE: Provided by Christina Farnan, Carton Group, Cavan, Republic of Ireland.

Bryan and McKinley (1974) studied the preparation process for whole frozen turkey and produced detailed time and temperature profiles for all processes, including thawing. However, they reported only deep muscle temperatures and the air temperature. For a 20-lb [9 kg] turkey, they found that after 40 hours thawing in a refrigerator at ~4°C the deep muscle temperature was only -2.8°C. At ambient temperature (~24°C), the deep muscle temperature was 0°C after 9 hours and 10°C after 18 hours. The surface temperature in this latter case was 10°C after 5 hours and 16.6°C after 22 hours. This demonstrates that the surface temperatures can be relatively high by the time the turkey is thawed. For broilers where weights are lower, the thawing time would be reduced, but the surface temperatures after similar periods are likely to be the same or slightly higher, due to the reduced mass of the bird.

It is important to validate any predictions of growth during thawing and at least one suitable study is reported in the literature. Data on the growth of *Salmonella* following thawing was generated in minced chicken substrate (White and Hall, 1984). Such data could be used to develop a model for frozen storage, periods of freeze-thaw and thawing, but this type of model development is outside of the scope of the current exposure assessment. The White and Hall data show that the numbers of *S.* Typhimurium decreased during frozen storage by approximately 99% after 168 days of storage, but by only 90% for *S.* Hadar in a similar period. They also showed that the numbers of *S.* Typhimurium increased by 1.8 log cycles after 24 hours thawing at 20°C and by 2.93 log cycles after the same period at 27 C. *S.* Hadar grew by 2.87 log cycles after 24 hours thawing at 20°C and by 5.4 log cycles after the same period at 27°C. These data on thawing can be used to validate the growth models selected, given the thawing profiles reported.

Preparation handling of raw chicken

Handling which is typically carried out at ambient temperatures can transfer bacteria via cross-contamination of the hands and food preparation environment and especially if prolonged, this is another factor that may lead to growth of salmonellae.

There are few data available in the literature on time and temperature studies during preparation. Data on the time taken to prepare poultry and the temperature changes were reported by Garey and Simko (1987).

Several studies of cross-contamination have been conducted, but these consider general contamination of the food environment rather than the contamination attributable to a specific

process such as preparation of chicken (Scott and Bloomfield, 1990; Josephson, Rubino and Pepper, 1997). Others have quantified the incidence of cross-contamination due to specific processes (Humphrey, Martin and Whitehead, 1994; Cogan, Bloomfield and Humphrey, 1999). However, few have quantified the numbers of bacteria that are transferred during cross-contamination. Cross-contamination resulting from the preparation of broilers has been studied (de Wit, Broekhuizen and Kampelmacher, 1979). In an elegant study, they used naladixic-acid-resistant *E. coli* K12 as a marker organism to artificially contaminate broilers. The spread of this organism during preparation was studied. The cross-contamination rates show that the more direct the contact between broiler and item, the greater the percentage of positive samples from that item. Washing reduces the incidence of cross-contamination, but not completely. In the preparation process, other surfaces, such as water taps and spice jars, also become contaminated, but to a lesser extent, indicating indirect contamination from hands.

For a quantitative exposure assessment model, these data could be used to calculate the probability of cross-contamination by direct and indirect means, which would be more practical than separate calculations for surfaces, utensils and hands.

A measure of the probability of cross-contamination is not sufficient for an exposure model without an idea of the quantity of bacteria involved. Zhao et al. (1998) developed a model system to enumerate bacteria transferred during common food preparation practices. They found that chicken meat and skin inoculated with 10^6 CFU bacteria transferred 10^5 CFU to a chopping board and hands, and then 10^3-10^4 CFU to vegetables subsequently chopped on the unclean board. Disinfection of the chopping board and hand washing reduced the numbers of bacteria by 1-2.8 logs and reduced the incidence of cross-contamination of the cut vegetables (52%: no bacteria; 33%: 10-50 bacteria; 5%: 100-200 bacteria).

These data can be used as the basis to estimate the numbers of bacteria transferred to a food by cross-contamination. From the work of Zhao et al. (1998), it appears that bacteria transfer at a rate of approximately 10% between items, e.g. between raw chicken and the chopping board. Direct cross-contamination involves two steps, e.g. raw meat to chopping board, and then to another food item. Hence, the direct cross-contamination bacterial load for the second food item should be a maximum point estimate of 1% of the numbers of salmonellae on the broiler chicken. Indirect cross-contamination involves a minimum of three steps, e.g. broiler chicken to hands, to plate, and then to another food item. Hence, for indirect cross-contamination, the bacterial load transferred to another food item would be a maximum of 0.1% of the salmonellae on the broiler.

Cross-contamination can also occur from inadequate hand washing. Studies on hand washing have shown that numbers of bacteria on the hands influences the number of samples that are contaminated through finger contact (Pether and Gilbert, 1971). Reviews of hand washing practices are available in the literature (Snyder, 1999; Restaino and Wind, 1990; Reybrouck, 1986).

Cooking and thermal death models

Bacteria die when subjected to the elevated temperatures found during cooking. It is widely accepted by microbiologists that bacteria die in a predictable, logarithmic way. This is referred to as first-order inactivation kinetics. The physiological assumption is that there is only one heat target per cell that is responsible for the death of the whole cell. Classically, the death of bacteria has been described by the Arrhenius equation that was developed for first-order chemical reaction kinetics (Equation 6.3):

$$K = A \cdot e^{\left(\frac{E_a}{RT}\right)} \quad \text{and} \quad \log_{10} N = \log_{10} N_0 - \frac{(K \cdot time)}{2.303}$$ Equation 6.3

where: E_a = activation energy (J mol^{-1}); A = pre-exponential factor, R = gas constant (8.31 JK^{-1} mol^{-1}), N_0 = initial cell number, N = cell number after time at T, and T = absolute temperature (Kelvin).

However, deviations from the first-order death kinetic model have been observed. Shoulders and tails to the survivor curves are reported. This area has been reviewed extensively (Clark, 1933; Withell, 1942; Rhan, 1945; Cerf, 1977; Casolari, 1994). Several models that have characterized non-linear thermal death curves have used a log-logistic function to describe the data (Cole et al., 1993; Little et al., 1994; Ellison et al., 1994; Duffy et al., 1995; Anderson et al., 1996; Blackburn et al., 1997).

The equation for the log-logistic curve, with a shoulder and a tail, is shown in equation 6.4.

$$\log_{10} N = \alpha + \frac{(\varpi - \alpha)}{1 + e^{\frac{4\sigma(\tau - \log_{10} time)}{\varpi - \alpha}}}$$ Equation 6.4

where N = cell number after time at the temperature studied, a = upper asymptote of the curve, w = lower asymptote of the curve, s = maximum inactivation rate, and t = time to the point of maximum inactivation rate.

For the present exposure assessment, the traditional log-linear-death kinetic model will be considered, for simplicity. Many investigators do not show the inactivation data for their studies and merely quote D-values (i.e. time for a 90% reduction in the numbers of bacteria at a given temperature). Generally, these workers will use regression analysis of data showing $\log_{10}$ bacteria numbers vs heating time. The equation of the regression line can be used to calculate a D-value over 1 log cycle reduction in the numbers of bacteria. When D-values are calculated for a number of different temperatures, a relationship between the D-value and the temperature can be calculated. Data expressed as the reciprocal of the D-value vs temperature of the D-value can be analysed by regression to give a straight-line equation. This equation can be used to calculate a z-value, which is the temperature change required to bring about a 90% change in D-value. Hence, if the z-value = 10°C and a D-value at 70°C = 1min, then by applying the z-value to the D-value we can see that the D-value at 80°C = 0.1 minute and the D-value at 60°C = 10 minutes. Therefore, with a D-value at a given temperature and a z-value for a bacterium in a given heating medium it is possible to calculate the reduction in the numbers of that bacterium at any other temperature.

Secondary models can be constructed that relate the change in D-value to parameters such as pH and water activity. A model describing the death of *S.* Enteritidis was developed by Blackburn et al. (1997). This model is comprehensive, covering the effects of temperature, pH and salt on survival. In addition, the model validated well against D-value data derived in whole foods. Unfortunately, this model is incorporated into the Food MicroModel™ software, which is proprietary.

An alternative approach that has been used in other exposure assessments is to take published D-values for *Salmonella* in foodstuffs, analyse the data, and determine an average D-value and z-value

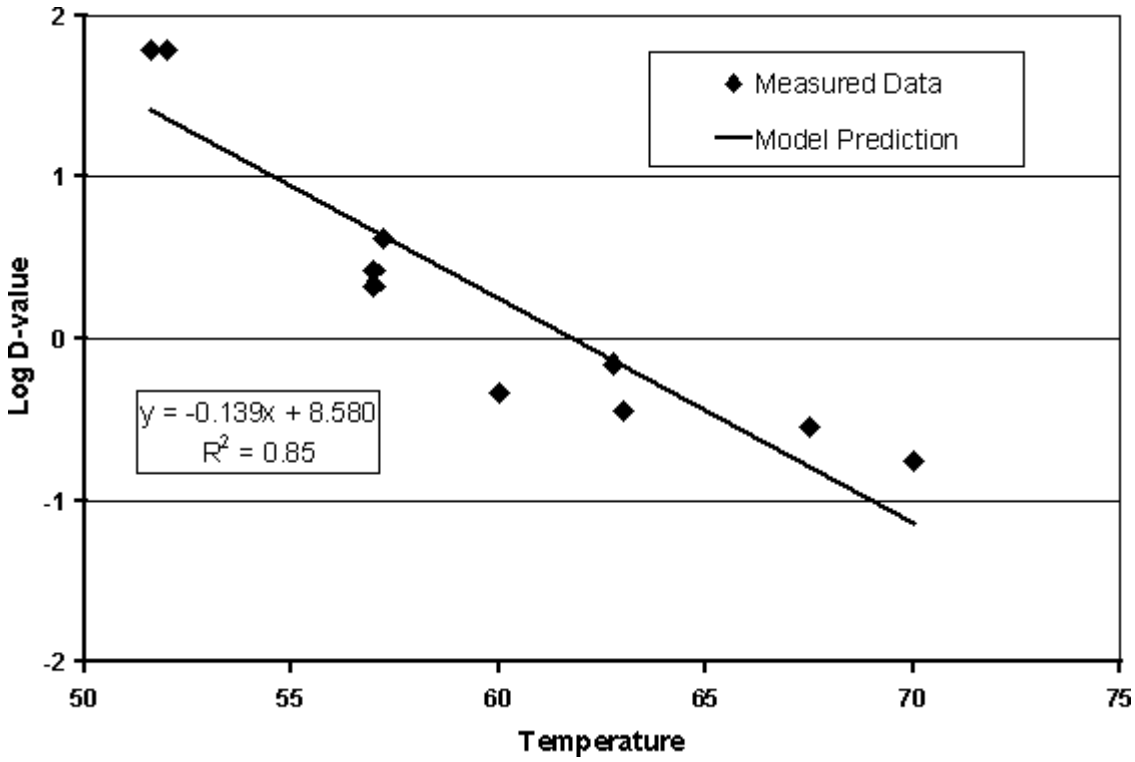
using the method described earlier (Buchanan and Whiting, 1997). Table 6.23 shows data used in this exposure assessment model for the calculation of an average D-value and *z*-value (Figure 6.7).

Table 6.23. Data on the inactivation of *Salmonella*

Serotype	**Meat**	**D-value (minutes)**	**Temperature (°C)**	**Reference**
Salmonella	Chicken	0.176	70	Murphy et al., 1999
Salmonella	Chicken	0.286	67.5	Murphy et al., 1999
S. Typhimurium	Ground Beef	0.36	63	Goodfellow and Brown, 1978
Salmonella	Ground Beef	0.7	62.76	Goodfellow and Brown, 1978
S. Thompson	Minced Beef	0.46	60	Mackey and Derrick, 1987
Salmonella	Ground Beef	4.2	57.2	Goodfellow and Brown, 1978
S. Typhimurium	Ground Beef	2.13	57	Goodfellow and Brown, 1978
S. Typhimurium	Ground Beef	2.67	57	Goodfellow and Brown, 1978
S. Typhimurium	Skin macerate(1)	61.72	52	Humphrey, 1981
Salmonella	Ground Beef	62	51.6	Goodfellow and Brown, 1978

NOTES: (1) from chicken neck.

Figure 6.7. Plot of D-values from Table 6.23 with linear regression model used to subsequently calculate D- and z-values.



The D-value can be calculated using Equation 6.5:

$$D_{value} = 10^{(-0.139 \cdot Temp) + 8.580}$$ Equation 6.5

and the *z*-value is the reciprocal of the slope of the line, Equation 6.6:

$$z_{value} = \frac{1}{0.139} = 7.19$$ Equation 6.6

To utilize the linear model for the thermal death of salmonellae in an exposure assessment model, it is necessary to measure the time and temperature profile for the cooking step. For conventional conduction-limited cooking (i.e. oven roasting, boiling, steaming), measurements are normally taken at the coldest spot, which is the deep muscle tissue of a broiler chicken carcass. However, this does not give information about the temperature at the surface of the carcass, where salmonellae may also be located.

For microwave cooking, where the thermal profile may be uneven, measurements must be taken in a number of places. An exposure assessment should account for differences in cooking methodology and the heterogeneity of temperature that this may cause. Models for microwave cooking are very complex and often require the use of thermodynamic modelling techniques to generate the time and temperature distributions.

Many studies reported in the literature do not contain the thermal profiles. Some report end-product temperatures and cooking time (Baker, Poon and Vadehra, 1983; Schnepf and Barbeau, 1989). Table 6.24 shows some publications where appropriate data are given.

Example data have also been supplied by a manufacturer of cooked chicken products (personal communication). Data for cooking of chicken drumsticks are summarized in Table 6.25.

Table 6.24. Studies on thermal profiles for cooking of poultry.

Study	Item cooked	Cooking method	Parameters measured
Bryan, 1971	Whole turkey	Boiling and steaming	Deep muscle, surface and external temperature
Bryan and McKinley, 1974	Whole turkey	Oven roast	Deep muscle, surface and oven temperature
Lyon et al., 1975	Chicken thighs	Boiling	Internal temperature
Ibarra et al., 1999	Chicken breast	Oven roast	Infrared surface and internal temperature
Chen and Marks, 1997	Chicken patties	Oven roast	Surface, interior and oven temperature
Chang, Carpenter and Toledo, 1998	Whole turkey	Oven roast	Various points

Table 6.25. Example thermal profile data on roasting chicken drumsticks.

Time (minutes)	Time of the temperature block (minutes)	External temp. (°C) (mean of 2 measurements)	Internal temp. (°C) (mean of 6 measurements)
0	5	12.6	14.9
5	5	13.0	14.2
10	5	136.6	13.7
15	5	161.2	27.8
20	5	150.0	43.2
25	5	150.7	56.2
30	5	164.0	68.6
35	5	166.1	78.0
40	5	168.6	85.8
45	5	166.3	83.7
50	5	176.3	93.3
55	5	161.4	94.9
60	5	49.2	82.1

To calculate the lethal effect of the process shown in Table 6.25, the following approach can be applied:

1. Break the profile up into time and temperature blocks as shown in Table 6.25.

2. Using Equation 6.5, calculate a D-value at a suitable reference temperature within the range of the profile.

3. Use Equation 6.7 to calculate the equivalent process time at the reference temperature for each time and temperature block:

$$Etime_{T_{ref}} = \frac{\left(10^{\frac{(T \cdot T_{ref})}{z}}\right)}{time}$$

Equation 6.7

where: *Etime* = equivalent time at the reference temperature; T_{ref} = reference temperature; T = temperature (°C) of the time and temperature block; z = temperature change resulting in a 90% change in D-value, calculated from Equation 6.6; and time = time period of the time and temperature block (in minutes).

4. Use Equation 6.8 to calculate the equivalent reduction in log numbers of bacteria for each time and temperature block.

$$\log red = \frac{Etime_{T_{ref}}}{D_{T_{ref}}}$$

Equation 6.8

where: Log *red* = reduction in log numbers of bacteria; *Etime* = equivalent time at the reference temperature; T_{ref} = reference temperature; and D = D-value.

5. Subtract each reduction from the starting log number of bacteria to determine the number of bacteria surviving the process.

Figure 6.8 shows the application of this approach to the data given in Table 6.25, using the model generated in Equations 6.7 and 6.8 and an assumed starting number of salmonellae of 10 million.

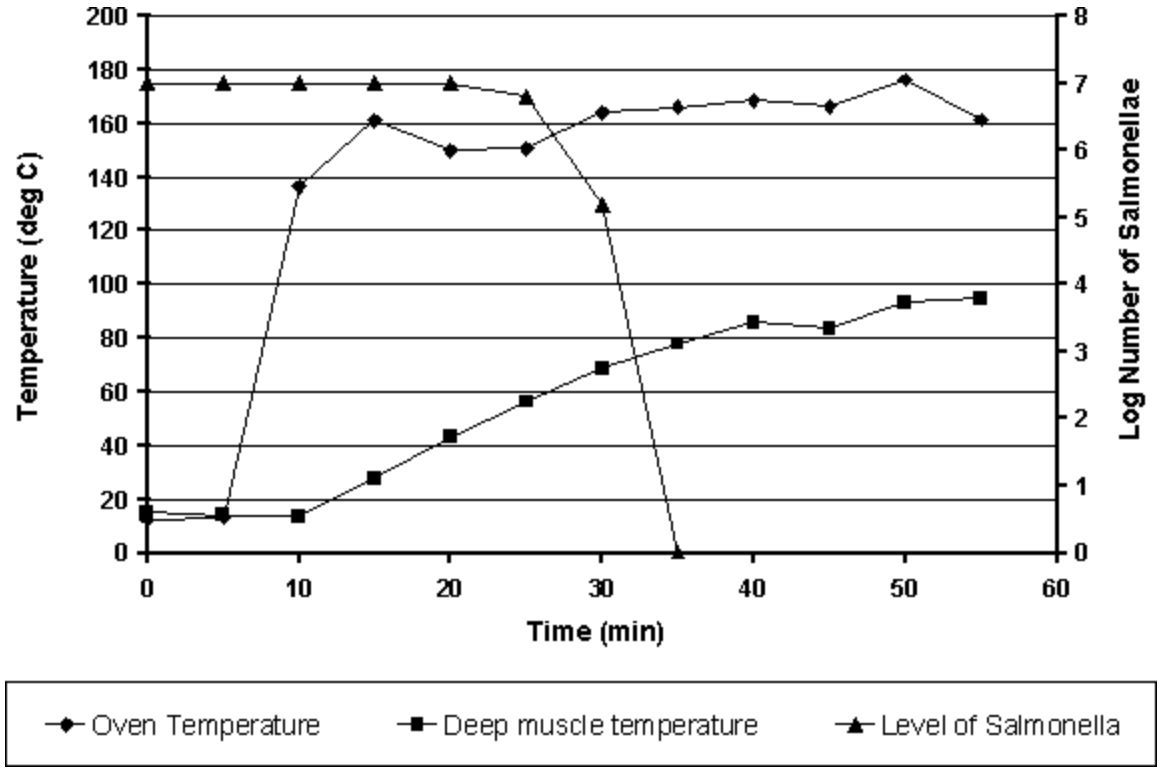
Cooling and re-heating

Providing suitable time and temperature profiles are available, the growth and thermal death models can be used to predict the numbers of salmonellae that may be present after a process. Published time and temperature profiles can be found (Bryan, 1971; Bryan and McKinley, 1974) but, as with all such profiles, data are scarce.

Meal preparation

Meal preparation can involve re-contamination of the cooked chicken from salmonellae present on hands, utensils and surfaces. This can be accounted for in the exposure assessment in a similar manner to the modelling of the raw preparation step. An assumption based on data can be made for the probability of cross-contamination and the numbers of salmonellae transferred (see above: *Preparation handling of raw chicken*).

Figure 6.8. Cooking temperature profile for a chicken drumstick and the predicted reduction in the number of salmonellae in the deep muscle tissue.



6.2.7 Consumption

The aim of the consumption module is to quantify the frequency with which broiler meat is consumed in the form specified in the preparation module, and to quantify the portion size.

Consumption studies

One aim of an exposure assessment model is to provide quantitative data to input into the dose-response model. To do this, it is necessary for the exposure assessment to predict the likelihood of human exposure to a bacterial pathogen, and the numbers of the pathogen to which a person may be exposed. So far in this report, tools have been described that enable a quantitative prediction of the number of salmonellae on ready-to-eat broiler chicken meat, and the likelihood that the chicken meat will be contaminated with salmonellae. However, to become exposed to the bacteria, a person must consume broiler meat. Therefore, the number of bacteria that enter the person's body also depends on the amount of the meat they eat and possibly the frequency of consumption. The final stage in the exposure assessment model is a determination of consumption patterns for broiler chicken meat.

Food consumption patterns vary from country to country, by demographic group, and by age group. Therefore, ideally, countries should determine their own national consumption patterns. Additionally, consumption studies are often undertaken for purposes other than exposure assessment, e.g. nutrition studies. The design of these studies is not necessarily appropriate for determination of exposure to microorganisms from consumption of a product.

Data requirements and available data

Data required for a consumption module would relate to the products specified for the risk assessment, and in the exposure assessment. In this work, consumption data are required for a single serving from whole or portioned broiler chicken, prepared in the home according to the methods used in the previous module.

Commodity consumption data have been compiled and published by WHO (1998). For countries without national studies, this work is a good reference. Unfortunately, because of its general nature, it gives consumption data for chicken meat from all sources on a regional basis. The consumption of chicken meat per day per capita was reported as follows:

- Middle Eastern diet 30.5 g
- Far Eastern diet 11.5 g
- African diet 5.5 g
- Latin American diet 25.3 g
- European diet 44.0 g

These data include meat from whole cooked broilers, but in addition also include servings of cooked minced chicken preparations, pre-prepared commercial meals, and other sources outside the scope of the present exposure assessment model.

A more detailed breakdown of food consumption can often be gained from national nutrition surveys. For example, in Australia, a national survey conducted in 1995 (McLennon and Podger, 1995) classified consumption of whole muscle poultry meat *inter alia* by age group, sex and socioeconomic group. Table 6.26 summarizes the relevant data.

Table 6.26. Mean daily intake of poultry muscle meat per person in Australia.

Male age group (years)									
2-3	**4-7**	**8-11**	**12-15**	**16-18**	**19-24**	**25-44**	**45-64**	**65+**	**19+**
11.3 g	19.2 g	26.8 g	48.4 g	51.4 g	73.1 g	66.7 g	62.6 g	45.4 g	63.3 g
Female age group (years)									
2-3	**4-7**	**8-11**	**12-15**	**16-18**	**19-24**	**25-44**	**45-64**	**65+**	**19+**
8.8 g	12.5 g	23.6 g	29.4 g	32.3 g	33.7 g	31.5 g	34.2 g	29.7 g	32.2 g
SEIFA quintile of relative socioeconomic disadvantage(1), 19 years +									
1st		**2nd**		**3rd**		**4th**		**5th**	
47.3		48.1		45.6		47.1		48.9	

NOTES: (1) Based on the characteristics of an area where the person lives. People in the first quintile live in the most disadvantaged areas, whereas people in the fifth quintile live in the least disadvantaged areas. SOURCE: McLennon and Podger, 1995.

These data allow exposure predictions to be targeted to vulnerable groups, such as the very young and the elderly. The study also showed that, in Australia, the consumption of poultry muscle meat was not influenced to any great degree by socioeconomic group.

The single drawback to these data is that the only value reported is the mean daily intake. Reporting the standard deviations of the mean values would allow estimation of the distribution range of size of meals consumed.

In Ireland, the Irish Universities Nutrition Alliance (IUNA) have recently completed a food consumption survey. The primary aim of the survey was to establish a database of the habitual food and drink consumption of Irish adults between the ages of 18 and 64. The Republic of Ireland section of the database contains entries for 958 subjects, but as the data were collected *per eating occasion*, there are 159 091 entries in the database. The total food consumption for each subject must be taken to represent his or her habitual weekly intake of a given food. The IUNA database was searched for meals where chicken was casseroled, grilled, stir-fried, deep-fried or roasted. Prepared chicken dishes - chicken Kiev, chicken vindaloo, etc. - were excluded.

Of the 159 091 eating occasions entered in the database, 1289 referred to chicken muscle. In real terms, 633 subjects out of a possible 958 consumed chicken muscle at least once per week (66%). Of those consuming at least once a week, it was found that the chicken muscle was consumed on average 2.04 times per week (maximum 7 times; minimum once).

It is worth noting that consumption data is very country specific as consumption patterns may be very different in different parts of the world. Thus, any national exposure assessment should use data specific for that country rather than data from any other country.

6.2.8 Review of models available

Overview

To date, no full exposure assessments of *Salmonella* in broiler chicken products have been presented, i.e. an exposure assessment that includes all the steps outlined in Figure 6.1 for the production-to-consumption pathway. However, exposure models have been developed for subsections of this pathway. Oscar (1997) considers levels of exposure throughout processing (Module 2 in Figure 6.1) while Oscar (1998) and Oscar (in press) developed models to describe exposure from the point of packaging to the point of consumption (Modules 3 and 4 in Figure 6.1). Brown et al. (1998) consider changes in the numbers of organisms on contaminated raw chicken products following cooking (Module 4 in Figure 6.1).

In contrast, a full exposure assessment has been described for *Campylobacter jejuni* in fresh poultry (Fazil et al., unpublished; A.M. Fazil, personal communication). Although there are key differences between *Salmonella* and *Campylobacter jejuni*, this model can be used as a basis for review.

These models are summarized here with respect to the objectives of the work, and the various methodologies used. It is noted that several of the models consider, to some extent, dose-response and hazard characterization as well as exposure assessment. In such cases, only the exposure assessment part is reviewed. Following each summary, the methodologies are discussed with respect to a full exposure assessment of *Salmonella* in broiler chicken products.

The models of Oscar (1997), Oscar (1998) and Oscar (in press)

The model of Oscar (1997) is essentially a demonstration tool to illustrate the use of simulation modelling in food safety decision-making. Consequently, real data are not used within the model and hence results do not represent actual estimates of exposure.

The demonstration model considers the prevalence of *Salmonella*-positive broiler chicken carcasses and the number of organisms per contaminated carcass following each sequential step of processing, as outlined in Figure 6.3. Each step is characterized by two quantitative parameters, the prevalence and the extent of a specific pathogen event. Pathogen events correspond to either an increase or reduction in numbers of organisms per carcass, depending on the step-specific factors. In the model, increases reflect only cross-contamination, hence bacterial growth is not included.

A similar pathogen event approach is used in subsequent models (Oscar, 1998; Oscar, in press). These models commence at packaging of raw chicken and describe changes in the prevalence of *Salmonella*-positive products and the numbers of organisms per positive product until the point of consumption. Pathogen events again refer to either an increase or decrease in pathogen load, but these subsequent models also consider bacterial growth.

These three models provide simple assessments of exposure. The underlying methodology involves simulation of a random chicken product through various exposure steps.

In general terms, the framework presented in the Oscar models could be used as a basis for development of a full exposure assessment of *Salmonella* in broiler products. Indeed, in the first instance, it may be possible to combine the processing model (Oscar, 1997) with either of the packaging-to-consumption models (Oscar, 1998; Oscar, in press). However, there are important points that would need further consideration before such use.

First, the model framework describes the inherent variability of the sequential exposure steps. In particular, the probabilities (p_i) represent the randomness associated with whether or not the particular pathogen events will occur while the distributions for changes in numbers of organisms describe all possible magnitudes of change. However, it is possible that for *Salmonella* we may not know the exact values for p_i and all possible magnitudes of change. Therefore it is likely that there will be uncertainty associated with these parameters. As is, the model framework does not account for such uncertainty and thus may produce inaccurate estimates of exposure.

The second feature of this framework that should be addressed focuses on the notion of cross-contamination. The model framework for packaging through to consumption does not include cross-contamination to other products or the environment. However, during preparation, for example, such cross-contamination might be very important. Consequently, this approach could underestimate exposure. For the processing model (Oscar, 1997), the methodology used to account for cross-contamination is not explicitly stated, hence it cannot be determined whether or not this would be appropriate in a full exposure assessment.

A final point worth considering is the representation of growth and survival within the packaging-to-consumption models. Growth and reduction due to temperature abuse and cooking, respectively, are not given as time-dependent processes within the models. Rather, the overall change following a period of abuse or cooking is modelled. Although this gives a mechanism for estimating changes in exposure, the effect of different temperature profiles and product specific parameters cannot be investigated. Consequently, investigation of control strategies would be difficult.

In summary, these provide a basis for the development of a full exposure assessment, but issues concerning uncertainty, cross-contamination, growth and decline would have to be addressed before further use.

The model of Brown* et al. *(1998)

This model considers the prevalence of *Salmonella* on raw chicken portions and the numbers of organism per contaminated portion. Prevalence is estimated by a point value while a probability distribution is used to describe the variability in the numbers of organisms per contaminated portion. Given the initial level of pathogen on the raw product, the final level of exposure is then determined by modelling the effects of cooking.

The approach used within this model is deterministic in nature. In particular, point values are used for model parameters such as prevalence of contaminated chicken portions, and the heat transfer coefficient. Estimates of exposure are determined by integration over all parameters that are inherently variable, more specifically time, microbial distribution, and measurement of depth into the product. Although this approach accounts for inherent variability, it does not incorporate uncertainty in parameter values. As a result, it does not facilitate the derivation of confidence intervals for estimates of exposure. The authors present several suggestions for including uncertainty that could be incorporated in a full exposure assessment.

There are two main exposure steps in this model: first, the level of raw chicken contamination and, second, the effect of cooking. Cross-contamination within the kitchen, prior to cooking, is not considered. As discussed previously, cross-contamination within the kitchen could be a very important pathway for exposure to *Salmonella* from raw poultry and thus should be included within a full exposure assessment.

Overall, the framework presented in this model will be very useful for the development of Module 4 (Preparation) (see Figure 6.1) of any full exposure assessment. The framework could also be further enhanced by including uncertainty in model parameters and attempting to model cross-contamination in the kitchen.

The model of Fazil et al. (unpublished)

This assessment is still in-progress (A.M. Fazil, personal communication.). The review presented here considers the information that was available at the time of this review. It is expected that this model will be refined in the future, thus the comments made here may require appropriate modification.

The preliminary model provides a full exposure assessment for *Campylobacter jejuni* in fresh chicken. All stages from on-farm production to consumption are considered. At each stage, the 'fate' of *C. jejuni* on chickens is estimated with particular reference to surface contamination and the numbers of organisms per contaminated unit (carcass). In this way, changes in prevalence and numbers are described and a final estimate of exposure is derived. As this model considers fresh chicken products, the framework presented provides a basis for the development of an exposure model for *Salmonella* in the same commodity.

In a similar manner to the model pathway outlined in Figure 6.1, the exposure assessment commences with estimation of farm-level parameters. More specifically, the number of organisms on the skin and feathers of birds is calculated. Estimation is undertaken by determining the number of organisms excreted in the faeces and then assuming that a proportion of these contaminate the external parts of the bird. Consequently, it is assumed that feather, skin, etc., (i.e. surface) contamination arises directly from the birds. Given that within-flock prevalence of *Campylobacter* is generally very high (Hartnett et al., 2001), this would appear to be a valid assumption. However, for *Salmonella,* within-flock prevalence is much more variable and it may be more appropriate to consider other sources of contamination.

From the initial concentration of organisms on the exterior of birds at the farm level, changes in numbers during transport and subsequent processing are modelled. The modelling approach considers each step in turn and determines the magnitude of change in terms of either a log increase or decrease, depending on the particular step. The magnitude of change is estimated from several data sets that provide this specific type of information, hence particular reasons for change, such as cross-contamination or wash-off, are accounted for. If equivalent data were available for *Salmonella* spp, a similar modelling approach could be used. It is of course important to point out one key difference between *Salmonella* and *Campylobacter*, that is that conditions during processing that may be favourable for the growth of *Salmonella* would probably not result in multiplication of *Campylobacter.*

As changes in concentration are modelled, changes in prevalence of contaminated birds, carcasses or products from farm to the end of processing are also described. The initial prevalence estimate relates to prevalence of contaminated birds on entry into the processing plant, and this estimate essentially describes the probability that any random bird is contaminated. During processing, changes in prevalence have been modelled by initially ranking the different stages according to the extent to which cross-contamination is likely to occur. Based on this ranking, a cross-contamination factor is then assigned to each step. For each step, the resulting prevalence is a function of the prevalence at the start of the step and the cross-contamination factor. Given the generality of the

cross-contamination factor approach, it is likely that a similar methodology could be used to model changes in *Salmonella* prevalence during processing.

Following processing, the time between processing and preparation of the chicken in the home is considered. This period covers both storage and transit. It is assumed that the chicken product remains at refrigerated temperatures and reduction in the number of organisms per day is calculated. This approach, which essentially models survival, is appropriate for *Campylobacter*. However, for organisms such as *Salmonella*, growth during storage and transit may be important, depending on whether or not temperature abuse occurs. Consequently, growth as well as survival would have to be considered.

The final step of the exposure assessment models consumer handling and preparation. It is assumed that exposure to *C. jejuni* occurs via two independent routes: consumption of undercooked chicken and through the raw chicken fluids that may be subsequently ingested through cross-contamination. The models presented for these steps could be adapted for a *Salmonella* exposure assessment by incorporating species-specific data.

The *Campylobacter* exposure assessment is a stochastic model. The stochastic component of the model framework describes the variability in changes in prevalence and numbers of organisms throughout the sequential exposure steps. However, as yet, the uncertainty associated with these distributions of change is not accounted for. Given the limited quantitative information relating to changes in prevalence and numbers, inclusion of uncertainty will be important for an exposure assessment of *Salmonella.*

Overall, the general framework on which the preliminary model of Fazil and collaborators is based could provide a basis for the development of a *Salmonella* exposure assessment. However, other factors would also have to be included, particularly growth during storage and transport and uncertainty associated with probability distributions to describe magnitudes of change.

6.2.9 Recommendations

To date, no full exposure assessments have been undertaken for *Salmonella* in broilers. This present report has considered:

- What is required for undertaking such assessments.
- What information is available.
- How the available information meets the requirements.

The following recommendations for directing future work can be made.

(i) Reporting of prevalence at different steps of the full exposure pathway should be encouraged in all regions of the world.

(ii) Reported data should give full details of study methodology, including sampling site, sampling time, how the sample relates to the overall population, and microbiological methods.

(iii) Determination of quantitative data should be encouraged, and, if it becomes available, then full exposure assessments could be developed to investigate mitigation strategies (e.g. use of chlorine in chill water) or to compare alternative practices (e.g. air chilling versus immersion chilling).

(iv) Cross-contamination during processing and handling operations should be studied quantitatively and methodologies for modelling this process should be developed. Cross-contamination during these stages is a critical factor, which is often associated with outbreaks.

(v) At the national level, the collection of consumption data should be promoted. The design of these studies should accommodate the data requirements for exposure assessments. These requirements include population variability, portion size and frequency of consumption.

(vi) In predictive microbiology, the area of survival has been less well studied than growth or death. There are few predictive models that describe survival at chill and frozen temperatures. Further development of these models is essential.

6.3 EXPOSURE ASSESSMENT MODEL, MODEL PARAMETERS AND ASSUMPTIONS

6.3.1 Introduction

Previous sections examined the data and models available to generate a production-to-consumption risk model. Although there is a substantial amount of literature relating to *Salmonella* in poultry-rearing operations and during processing, the existing data have severe limitations for usefulness in quantitative (or semi-quantitative) risk assessment. Very few investigations have enumerated *Salmonella* either on-farm or at processing, or measured how the populations change, for example in a specific stage during processing. Evidence suggests that numbers of *Salmonella* on poultry carcasses during processing are generally low, at the limits of detection using current enumerative methods, and even then, the commonly used MPN method is very labour and cost intensive. Hence, for practicality, only detect/non-detect (prevalence) investigations are commonly carried out. This results, therefore, in a critical data gap because without enumeration data, risk cannot be estimated. In addition, for both prevalence and the few enumerative investigations, there is a wide diversity in conditions of sampling (sample type, site, size, unit, etc.) and of laboratory testing methods, as well as other confounding factors introduced by the original purposes of the studies and their experimental design. Accommodating these variations and assessing the validity, sensitivity and specificity in each individual report would probably be an exercise in futility. Furthermore, when temporal (if considering data from the early 1980s together with more recent information) and geographical factors are considered, a comprehensive risk model would not be very informative. However, the foregoing sections provide guidelines for the type of information and approach that might be used to develop a production-to-consumption risk model that could be applied to data that represent an individual processing operation, country or region.

Given the lack of use of enumeration data for stages prior to processing, the Exposure Assessment model for purposes of this risk assessment therefore begins at the end of commercial processing, with survey data for contamination levels on chilled broiler carcasses. The subsequent changes in contamination due to storage, handling and preparation were modelled based on information that was presented in detail in the previous section. The construct of the exposure assessment model is summarized in the following model description, and the parameters are shown in Section 6.4

6.3.2 Model overview

The exposure assessment considered fresh, whole broilers that are purchased at retail, then prepared and consumed in the home. The exposure model was analysed using Monte-Carlo simulation facilitated by @RISK software (ÓPalisade). Each iteration of the model tracks a randomly selected

broiler carcass from the time of exit from processing, through storage, preparation and cooking, to consumption. Thus, each run represents a random serving of cooking chicken and the exposures (including cross-contamination) that arise as a result of preparing this serving.

At the start of each iteration, the carcass is assigned to either the *Contaminated* or *Not Contaminated* state according to the prevalence of contaminated carcasses. If the carcass is contaminated, the number of *Salmonella* assumed to be present is selected from the range of values specified by a custom distribution of reported data. If the carcass it is not contaminated, the concentration of organisms is set to zero and this value is held constant for the remainder of the model. For contaminated carcasses, following the start-up step of the model the changes in the level of contamination through storage, preparation (including cross-contamination) and cooking are modelled.

Changes in the level of contamination during the various stages from chilling to consumption occur as a result of a number of variable processes, including storage times and temperatures, practices during preparation, and cooking times and temperatures. This variability is described by probability distributions derived from published and unpublished data and, where necessary, expert opinion.

The model is defined in terms of a number of parameters that describe the processes of broiler carcass distribution and storage, preparation, cooking and consumption. Many of these parameters can be considered general in that they can be used to describe the situation in many countries, such as cooking temperatures and duration of storage. In contrast, some parameters are country specific, such as prevalence of contaminated carcasses exiting processing, and thus to obtain results for individual countries, country-specific data must be input. In addition to the scope for generalization, the model parameters can be modified to determine their influence on the final estimates of exposure.

6.3.3 Processing

Prevalence of* Salmonella-*contaminated carcasses

Prevalence immediately after primary processing was set as a "user" input to the model. In the reports available, prevalence can vary widely from lot to lot, among different processing operations, and among regions and countries, particularly if national standards have been established or *Salmonella* control programmes operate. Thus, if using this model to describe the situation in a specific country, the likely ranges of local prevalence should be used to generate the risk estimate. Reductions to this value can then demonstrate the effect of prevalence reduction strategies, no matter how they are implemented, on the risk of illness. This would be an important preliminary investigation, prior to determining the best options for reduction, because an idea of the magnitude of the benefit can be realized.

For the purposes of this assessment, a baseline model was first developed, using as the initial input a fixed prevalence level of 20% *Salmonella*-contaminated carcasses after chilling. The predicted relative change in risk associated with higher or reduced levels of prevalence were modelled for comparison, using fixed values from 1 to 90% contaminated carcasses, and the results compared with the baseline risk estimate.

Numbers of **Salmonella** *on contaminated carcasses*

Few studies report on concentrations of *Salmonella* on broilers. Five studies reporting pathogen numbers at the end of processing (chilling) were summarized in Tables 6.9, 6.10, 6.12 and 6.13 (Surkiewicz et al., 1969; Dougherty, 1974; Lillard, 1980; USDA-FSIS, 1996; Campbell et al., 1983). Since then, data from Canada (CFIA, 2000), shown in Table 6.13, has been made available. All of these studies report MPN values rather than $\log_{10}$ values and all consider immersion chilling. Some of these studies have characteristics that mean that they are of limited use for inclusion within this exposure assessment. The studies of Surkiewicz et al. (1969) and Campbell et al. (1983) report combined distributions of MPNs from carcasses randomly selected from a number of processing plants. These processing plants differ in their practices relating to the use of chlorine. As chlorine has been reported as having an influence on counts of pathogenic organisms on carcasses (Waldroup et al. 1992), the combined distributions would only be representative if, at a national level, chlorine were used in the same proportion of plants in which it was used in these studies. In addition, these studies are old (published in 1969 and 1983, respectively) and practices affecting concentrations are likely to have changed. Thus, the distributions may not be representative of the current situation. For these reasons, it was decided not to include these studies in the example.

The results reported by Dougherty (1974) and Lillard (1980) give only the mean MPN values, with no information about the distributions of the data. These are therefore of limited use for describing the inherent variability of this parameter. Further, the studies are again old, and may not be representative of current practices. As a consequence, it was decided to exclude them from the example. It is noted that, in the future, more details about unreported original data might be obtainable by contacting the investigators.

National chicken broiler baseline surveys have been conducted in the United States of America in 1994-95 (USDA-FSIS, 1996) and in Canada in 1997-98 (CFIA, 2000) (Table 6.13). These surveys employed statistically based sampling plans, and the same sample collection and laboratory procedures. In the USDA study, carcasses were collected from federally inspected processing plants responsible for approximately 99% of all chickens slaughtered in the United States of America. Similarly, the processing plants from which carcasses were sampled in Canada were federally registered and produced 99.9% of broilers. Both studies report MPN distributions for levels of *Salmonella* on chilled carcasses.

Although the USDA and CFIA studies are similar in nature, and both reported similar prevalence of *Salmonella* on chilled carcasses (20% and 21.1%, respectively, by qualitative enrichment of carcass rinse samples), the resulting MPN distributions cannot be combined. This is because practices relating to the use of chlorine differed between the two countries at the time the baseline surveys were conducted. In the United States of America, the addition of chlorine at levels sufficient to maintain 1-5 ppm free chlorine in the overflow was the norm, while in Canada this was not general practice. However, in isolation, the two studies provide good data sets for characterizing the concentration on carcasses after chilling; they are recent, representative and all sampling methods are clearly described. Of course, neither study reports *Salmonella* concentrations prior to chilling, therefore careful consideration would have to be given if incorporating either data set into a specific processing model.

For the baseline risk model in this assessment, the levels of contamination on chilled broiler carcasses in Canada were used as inputs. This can probably be considered a general data input rather than a country-specific one.

Estimating numbers of **Salmonella** *on contaminated carcasses*

Carcass rinses (400 ml) were obtained for 774 broiler carcasses (CFIA, 2000). From each rinse fluid, a sample was tested for the presence or absence of salmonellae using a qualitative enrichment method. Of these, 163 tested positive. Positive rinse fluids were tested by the MPN method, and the MPN per millilitre calculated. The frequency of positive carcasses in five ranges was recorded. These data are shown in Table 6.27. The MPN per carcass was calculated by making two assumptions: first, all organisms on the carcass would be recovered during the shaking procedure, and, second, these organisms would be uniformly distributed within the rinse fluid. Based on these assumptions, the estimated MPN/carcass is equal to 400×MPN/ml (Table 6.28).

Table 6.27. Canadian national baseline data for *Salmonella* on chicken broiler carcasses

Range (MPN/ml)	Range (MPN/carcass)	Frequency
<0.03[(1)]	<12	99
0.03-0.3	12-120	60
0.301-3.0	121-1200	2
3.01-30	1201-12 000	1
>30.0[(2)]	>12 000	1

NOTES: (1) Positive by qualitative method, negative by quantitative MPN method. (2) Maximum reported value was 110 MPN/ml. SOURCE: CFIA, 2000.

The distribution for the 163 positive carcasses in Table 6.27 gives a description of the variability in the MPN/carcass. However, as the data is from a sample of carcasses, there will be uncertainty concerning the true variability. The cumulative distribution (Table 6.28) set the minimum value as 1 MPN/carcass and the maximum equal to 110% of the maximum observed MPN, i.e. 110% of 110 MPN/ml = 121 × 400 ml.

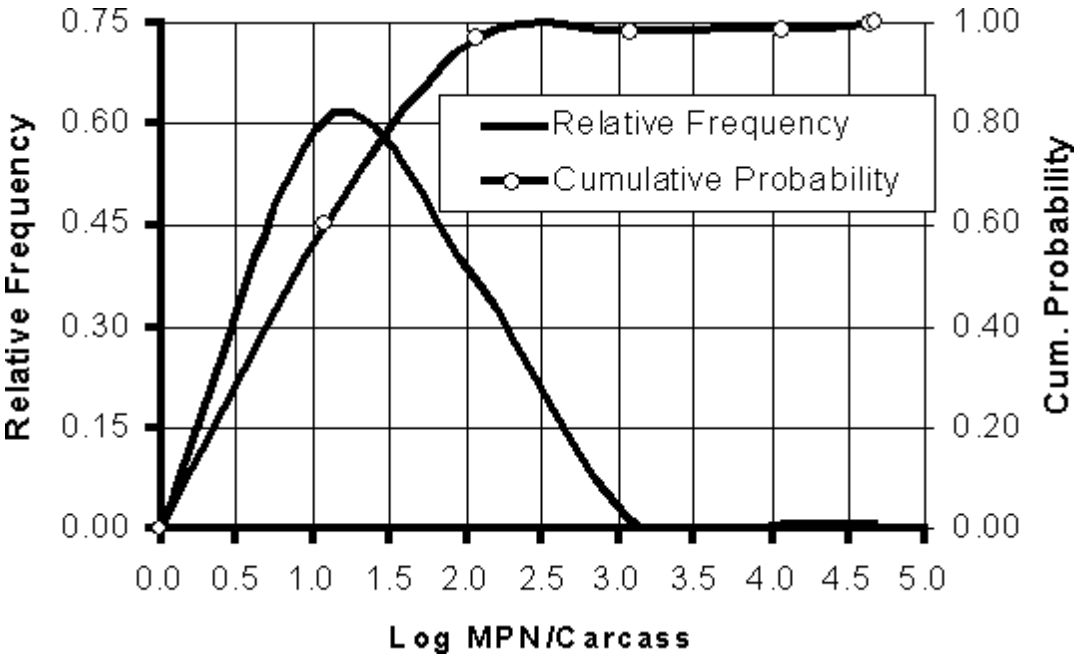
The resulting distribution for log MPN/carcass is shown in Figure 6.9. These distributions were used to characterize the variability in the numbers of *Salmonella* on contaminated carcasses at the end of processing.

The assumptions concerning the calculation of MPN/carcass from the data reported in the Canadian study require thoughtful consideration. In particular, there is uncertainty and variability relating to the MPN method, which has not been accounted for here. Further, it is likely that the carcass rinse method will not recover all organisms from the carcass. Indeed, it has been reported that on successive carcass rinses of the same bird, aerobic bacteria and enterobacteriaceae can still be recovered after 40 rinses (Lillard 1988, 1989b). These issues should be addressed in future refinements of the exposure assessment.

Table 6.28. Cumulative distribution for carcass concentration, with assumed minimum and maximum concentrations.

MPN/carcass	Log_{10} MPN/carcass	Cumulative probability
1	0.00	0.00
12	1.08	0.60
120	2.08	0.97
1200	3.08	0.98
12 000	4.08	0.99
44 000	4.64	0.99
48 400	4.68	1.00

Figure 6.9. Relative frequency and cumulative distribution of Log_{10}MPN/carcass



6.3.4 Distribution and storage

After processing and packaging, poultry carcasses are distributed to retail stores. It was assumed that between processing plant and retail there would be no change in the prevalence of contaminated carcasses or in numbers of *Salmonella* on those carcasses. The latter was based on assuming controlled refrigerated transportation conditions (see below for growth at <10°C). Although in the current exercise it was assumed that transportation was well controlled, this needs to be determined on a case-by-case basis. For that reason, a module was created, although not simulated, to illustrate

how this step might be potentially modelled and is summarized in Section 6.2.5, together with the other modules.

Three opportunities for *Salmonella* to multiply on the raw chicken were considered, from the time it enters the retail chain to the point at which the consumer prepares the chicken for cooking. These were (i) during retail storage and display, (ii) in transport from retail to the home, and (iii) during storage in the home. Survival and growth models currently available for estimating population changes during these stages were reviewed in Section 6.2.5. There are no suitable models to estimate survival and die-off for salmonellae in or on broilers, and therefore for the purposes of this risk assessment it was assumed that the salmonellae either grow given suitable conditions, or the population remains static on poultry, but does not decrease.

Several growth models for salmonellae were evaluated for their relevance and 'ease of use' for this assessment. The growth model selected was developed by Oscar (1999b) for *S.* Typhimurium (Equation 6.9).

$$LGR = \exp\left(\begin{array}{c} -6.225 - [0.0114 \times NaCl] + [0.3234 \times Temp] + \\ [0.002 \times \{NaCl \times Temp\}] - [0.0085 \times NaCl^2] - [0.0045 \times Temp^2] \end{array}\right)$$

Equation 6.9

The equation parameters were developed using ground chicken breast meat as the growth medium (rather than laboratory media), and the model has a wider growth temperature range than others (10°C-40°C). The author also validated the model satisfactorily. The growth model takes account of the influences of temperature and salt concentration (including any previous exposure to NaCl, as in the case of pre-culturing inocula in the laboratory) on the growth of *S.* Typhimurium. The author's opinion was sought on the likely water activity of broiler meat used in the development of the model and a value of 0.99 or 1.9% salt was advised. Therefore, the salt concentration parameters were both fixed at 1.9%, and the external temperature remained a variable that determined the growth rate. A final assumption was that there was no lag phase in the growth phases modelled. This is reasonable given that salmonellae on or in broiler meat would have had ample time to adapt to their environmental conditions prior to retail delivery, and it would be unlikely that the cells would experience significant lag time before commencing growth once storage temperatures rose. It has been emphasized elsewhere that predictive models can only be used for interpolation within their boundaries. The growth model has a lower temperature bound of 10°C and hence it was assumed that there was no growth below this. The upper temperature bound (40°C) was assumed not to be exceeded under normal storage conditions. The lower temperature bound assumption may underestimate some growth at <10°C.

Section 6.2.5 of the Exposure Assessment discussed the modelling of non-isothermal temperature profiles. It was noted that time-temperature storage (retail display, home, etc.) profiles are generally not available for raw poultry. Therefore, in this assessment, the observations of Ross (1999) were used, namely that microbial growth during isothermal temperature conditions could be reasonably predicted using the average temperature of the isothermal profile. Hence, any growth of salmonellae in broilers during storage was based on distributions around the reported average storage temperatures.

Note that while the growth model can be considered general, the time and temperature profiles used within it must be country specific.

Retail storage

A study in the United States of America (Audits International, 1999) reported survey data on the variability of average retail storage temperatures. These may or may not reflect similar conditions in other countries, but in the model these values can be readily replaced with other, more representative, temperatures if appropriate. Temperatures were recorded for 975 fresh meat products. The overall average temperature recorded was 4°C with a standard deviation of 2.8°C. The maximum temperature reported was 10°C and the minimum was -7.2°C. For this exposure assessment model, the variability in retail storage temperatures was represented by a truncated Normal distribution using these data. Hence, during the simulation, values could not be selected which were above the maximum or below the minimum recorded temperatures. Therefore, as 10°C represents the lower temperature bound of the growth model, growth was achieved only when an average retail storage temperature of 10°C or above was selected at random during the Monte Carlo simulation.

The growth model calculated a specific growth ***rate*** for *S.* Typhimurium at the average storage temperature. The ***extent*** of growth was determined by the length of storage time. Advice from retailers in Ireland was sought to estimate the minimum and maximum length of time that fresh chicken broilers were kept at retail. It would be preferable to obtain this information in a much more structured manner, or through a commissioned study, but, as a first step, this may be appropriate. The minimum value was estimated as 2 days and the maximum as 7 days. It was assumed that all values were equally likely and therefore the retail storage duration was represented by a uniform distribution. However, a correlation factor of -0.75% was used to ensure that, in the simulation, combinations of high storage temperatures and long storage times were unlikely (resulting in detectable spoilage and the product discarded before consumption, as would be the case in reality). The specific growth rate calculated by the growth model was multiplied by the storage time in days to give a value for the $\log_{10}$ increase in numbers of salmonellae.

Transport from retail to home

Data describing the variability of temperatures for foods during transport from the retail store to the home have been collected in the United States of America (Audits International, 1999). Variability in transport times from store to home was also measured during this study.

Given the parameters of the growth model used in this assessment, a product temperature of 10°C must be exceeded before a specific growth rate is calculated for *Salmonella.* Therefore it was assumed that if the external temperature that was experienced during transport to the home were below 10°C, no growth would occur. Hence, an estimate of the external temperature during transport was important to determine microbial growth. For the purposes of this assessment, typical northern European temperatures were applied, with the temperature variability represented using a PERT distribution centred on the most likely temperature value. These were a minimum of 0°C, a maximum of 24°C, and with a most likely value of 13°C.

The United States of America study reported changes in product temperature during transport from the retail store to the home for 975 fresh meat products. The overall average was 3.72°C with a standard deviation of 2.82°C. The maximum temperature change was defined as the difference between the external (ambient) air temperature and the minimum growth temperature of the model (10°C). The minimum temperature change was taken as 0°C (no change). This variability was represented by a truncated Normal distribution in a similar way to that described previously for retail storage.

The maximum product temperature during transport was calculated as the retail temperature of the product plus the temperature change if any change occurred. The average product temperature was calculated as the mean of the maximum product temperature plus the retail product temperature. This average temperature was used to calculate the specific growth rate for salmonellae in the growth model.

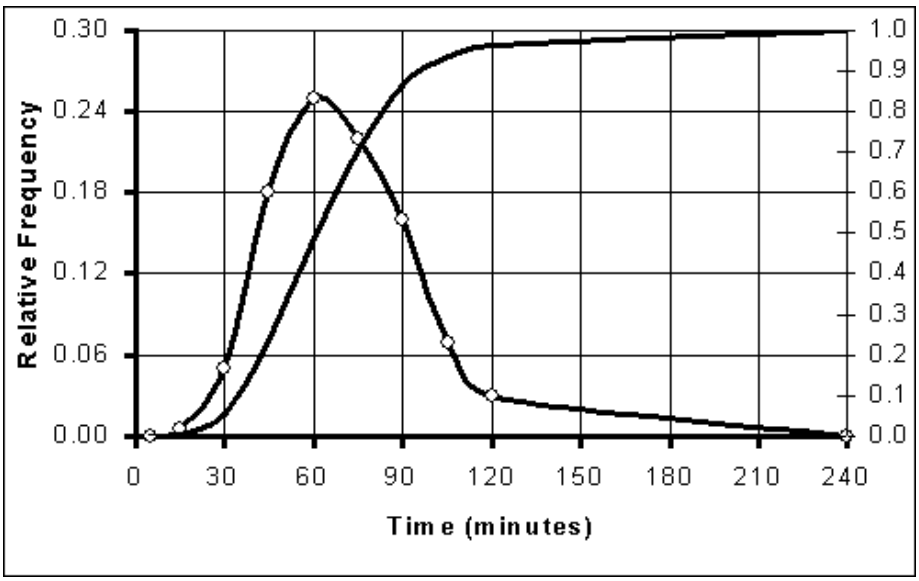
The data for transport time were reported as the frequency of measurements in 15-minute time intervals. A cumulative distribution was fitted to these values and used to represent the variability in these data (Table 6.29 and Figure 6.10). The increase in $\log_{10}$ numbers of salmonellae in or on a simulated broiler was calculated by multiplying the specific growth rate by the transport time.

Table 6.29. Transportation time from retail to home.

Time (minutes)	Frequency	Cumulative
-	0.000	0.000
15	0.005	0.005
30	0.050	0.055
45	0.180	0.235
60	0.250	0.485
75	0.220	0.705
90	0.160	0.865
105	0.070	0.935
120	0.030	0.965
240	0.035	1.000

SOURCE: Data from Audits International, 1999.

Figure 6.10. Probability distribution for transportation time from retail to home.



Home storage

Data on the variability of product temperatures during domestic refrigerated storage are available for the United States of America (Audits International, 1999). Temperatures during domestic refrigerated storage were recorded for unspecified food products, being chilled at an average of 4°C with a standard deviation of 2.65°C, with a maximum temperature of 21.1°C and a minimum of -6.1°C. The variability of reported home storage temperatures was represented by a truncated Normal distribution using these temperatures. Values could not be selected during the simulation above the maximum or below the minimum temperatures recorded. Again, because 10°C represented the lower temperature bound of the growth model, pathogen growth occurred only when a home storage temperature of 10°C or above was randomly selected during the Monte Carlo simulation.

The specific growth rates for *S.* Typhimurium were calculated for the average home storage temperatures and the extent of growth was determined by the length of storage time in the home. Based on adherence to the "use-by" date, expert opinion estimated the minimum value would be no storage time (immediate use), the maximum would be 5 days, with a most likely value of 2 days in the refrigerator. The variability in storage time was represented with a PERT distribution centred on the most likely value of 2 days. A correlation factor of -0.75% was used to ensure that combinations of high temperature and long storage time were unlikely (detectable spoilage and discard of product). The specific growth rate calculated by the growth model was multiplied by the storage time to give a value for the $\log_{10}$ increase in numbers of salmonellae.

6.3.5 Preparation and consumption

Cross-contamination

Cross-contamination of foods during the handling and preparation of raw meats is a recognized hazard in the home. If this parameter were ignored in a risk assessment model, it is likely that the risk would be underestimated. To estimate the risk of illness attributable to the cross-contamination of other foods during preparation of raw poultry, it is necessary to have information about the likelihood that cross-contamination will occur, and what is the likely number of bacteria transferred from raw to a ready-to-eat food. Section 6.2.6 described investigations that have studied these aspects. Estimates of the probability of cross-contamination were available from observational studies of food preparation behaviours (Worsfold and Griffith, 1997b). Estimates for the proportion of bacteria transferred from a raw food to hands or cutting board, and subsequently to other foods, were obtained from studies by Zhao et al. (1998). For the present risk assessment, two pathways of potential cross-contamination were modelled: from raw poultry via hands, and from raw poultry to cutting boards to other foods.

The probability of a person not washing their hands after handling raw poultry was estimated to be 0.6 (Worsfold and Griffith, 1997b). The proportion of salmonellae transferred from the raw broiler to the hands was estimated to be 10% (Zhao et al., 1998). It was assumed that if salmonellae were present, the number of salmonellae on the broiler and the proportion transferred would determine the numbers transferred. If hands were then washed, no further cross-contamination occurred. Hand washing was described by a Binomial distribution with a probability based on the values returned from the uncertainty model, as described previously. Based on this, if the model returned that hands were not washed, then salmonellae would be transferred to other foods. The numbers of salmonellae contaminating the other food was then calculated to be a function of the number of organisms on the hands and the proportion transferred.

Cross-contamination via cutting boards was simulated in the same way as cross-contamination from hands. However, here the probability estimate for the board being used for other food was 0.6 (Worsfold and Griffith, 1997b).

Cooking module

Preparing the food for consumption was modelled following an approach described by Fazil et al. (unpublished) and A.M. Fazil (personal communication) in a risk assessment for *Campylobacter* in poultry. Briefly, adequate cooking will destroy salmonellae and therefore it is only the broilers that are inadequately cooked that may still contain salmonellae at time of consumption (for the purposes of this module alone, post-cooking contamination is not considered). However, even with undercooking, it was assumed that salmonellae present on the external surfaces of the carcass will be inactivated, and that only some proportion of the total number - those more protected from heat penetration -would survive. The survival of the 'protected' bacteria will then depend on their heat resistance, and the length of time at some final temperature. The work of Fazil et al. (unpublished) and A.M. Fazil (personal communication) modelled this scenario based on published data for thermal profiles during cooking and on expert opinion, which were included in the example model for *Salmonella* in broilers.

The input variables in this module are sources of uncertainty in the example model. Table 6.30 shows the variables and their associated probabilities (Fazil et al., unpublished; A.M. Fazil, personal communication).

Table 6.30. Variables used to describe cooking of broilers.

Variable	**Probabilities**		
	Minimum	Most Likely	Maximum
Proportion of broilers not adequately cooked	0.05	0.1	0.15
Proportion of salmonellae in protected areas	0.1	0.16	0.2
Temperature exposure of protected bacteria (°C)	60	64	65
Time exposure of protected bacteria (minutes)	0.5	1	1.5

SOURCE: Fazil et al., unpublished.

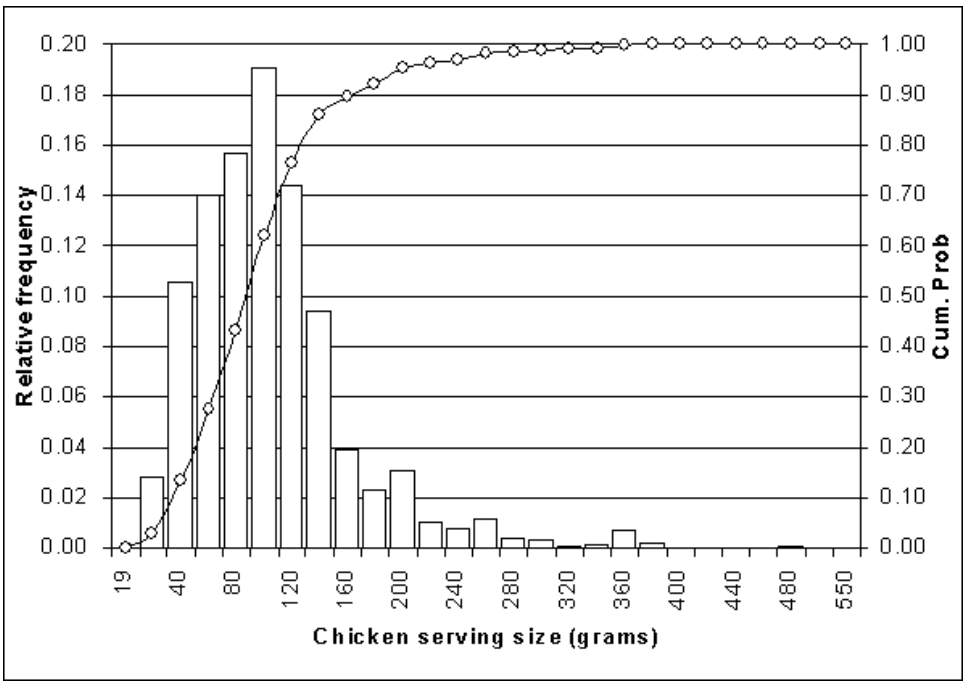
The probability that a randomly selected broiler would be undercooked was determined by a Binomial distribution. If the simulation determined that the broiler was adequately cooked, the broiler was considered *Salmonella*-negative. If the simulation determined that the broiler was inadequately cooked, then the number of salmonellae surviving was calculated as described previously in this section. Having determined the number of salmonellae in protected areas and the time and temperature they may experience, a D-value was used to calculate the numbers of salmonellae surviving. The D-value applied was dependent on temperature and was developed and described in Equation 6.5 (see Section 6.2.6).

Consumption

Section 6.2.7 discussed the consumption data requirements for microbial risk assessment. For the purposes of this assessment, consumption data collected in Ireland by IUNA were used to estimate the range of amounts of chicken in a single serving that might be consumed by individuals 18 to 64 years of age. Note, however, that use of this model in a national setting will require country-specific data to be used. These data are shown as frequency and cumulative distributions in Figure 6.11. The amounts consumed were for meals consisting of whole portions of chicken meat; recipes in which the chicken was present as an ingredient were not considered. For chicken on the bone, the intake was calculated by correcting the weight to reflect the edible portion. The consumption database showed that over the 7 days of recording for 1379 subjects, 65.5% of subjects (903) consumed chicken on 1695 eating occasions. For the purposes of this assessment, the risk estimations were based on one serving of chicken every two weeks (specifically as whole portions, prepared from fresh whole carcasses in the home).

The cumulative frequency distribution was used during simulation to randomly generate serving weights for broiler meat. A United Kingdom retailer supplied data on the likely weights of broilers. Minimum weight was estimated to be 1.1 kg, maximum weight was estimated to be 2.5 kg, with a most likely value of 1.5 kg. These data were fitted with a PERT distribution, which was used during the simulation to randomly generate a broiler weight. Expert opinion from a United Kingdom producer estimated that 30% of the weight of a chicken was edible meat. Therefore the broiler weight was reduced by 70% to generate an edible meat weight. Finally, the edible meat weight was divided by the serving size to calculate the number of servings per broiler.

Figure 6.11. Frequency distribution for the consumption of chicken meat per eating occasion.



6.3.6 Calculation of the number of salmonellae consumed

The outcomes of exposure assessment are probability of ingestion and number of salmonellae ingested in a serving. The number of organisms ingested from undercooked poultry was calculated by dividing the number of organisms on a broiler by the number of servings from the broiler. The ingestion of *Salmonella* from raw poultry via a cross-contamination event was treated as a separate pathway with a separate risk estimate. By running the model through Monte Carlo simulation, distributions were generated of the number of salmonellae that a consumer might be exposed to per serving of cooked broiler meat, and per cross-contamination exposure event.

6.4 MODEL DESCRIPTION AND PARAMETERS

Table 6.31. End of processing.

Description	Variable	Unit	Distribution or Equation		
Prevalence	Prev			Min	Max
			Fixed value	0	1
Concentration	Conc	MPN/bird	Cumulative		

Table 6.32. Transport from processing plant to retail [not simulated in current model]

Description	Variable	Unit	Distribution or Equation			
Transport temperature	T_pr	degree C		Min	Max	
			Uniform			
Transport time	t_pr	hours		Min	Max	CF
			Correlated uniform			-0.75
Minimum growth temperature	Tmin_pr	degree C	Constant	10		
Salt concentration	Slt_pr	%	Constant	1.9		
Log growth per hour	LGR_pr	log/hr	= EXP(-6.2251-(0.0114*Slt_pr) +(0.3234*T_pr) +(0.002*(Slt_pr*T_pr)) -(0.0085*(Slt_pr*Slt_pr)) -(0.0045*T_pr*T_pr)))			
Total log growth at retail	LG_pr	log	= IF(T_pr<Tmin_pr,0,t_pr*LGR_pr)			

Table 6.33. Storage at retail

Description	Variable	Unit	Distribution or Equation				
Retail temperature	Rtl_Temp	degree C		Mean	SD	Min	Max
			Truncated Normal	4	2.8	-7.2	10
Retail time	Rtl_Time	days		Mean	Max	CF	
			Correlated Uniform	2	7	-0.75	
Minimum growth temperature	MGT	degree C	Constant	10			
Salt concentration	NaCl	%	Constant	1.9			
Log growth per hour	LogSGR_Rtl	log/hr	=EXP(-6.2251 -(0.0114*NaCl) +(0.3234*Rtl_Temp) +(0.002*(NaCl*Rtl_Temp)) -(0.0085*(NaCl*NaCl)) -(0.0045*(Rtl_Temp*Rtl_Temp)))				
Total Log growth at retail	Rtl_growth	log	=IF(Rtl_Temp<MGT.0.Rtl_Time*24*LogSGR_Rtl)				

Table 6.34. Transport from retail to home

Description	Variable	Unit	Distribution or Equation				
Ambient temperature during transport	Trans_Temp	degree C	Pert	Min	ML	Max	
				0	13	24	
Maximum change in temperature during transport	TransMax	degree C	= Trans_Temp -Rtl_Temp				
Potential change in temperature during transport	Trans_DTemp1	degree C	Truncated Normal	Mean	SD	Min	Max
				3.72	2.82	0	TransMax
Change in temperature during transport	Trans_Dtemp2	degree C	=IF(Trans_Temp -Rtl_Temp<=0,0,Trans_DTemp1				

Chicken temperature after transport	Post_Trans_Temp	degree C	=Rtl_Temp +Trans_DTemp2				
Average transport temperature	Avg_Trans_ Temp	degree C	=Average(Rtl_Temp, Post_Trans_Temp)				
Transport time	Trans_Time	Minutes	Correlated Cumulative	Min	Max	CF	
				5	240	-0.75	
Log growth per hour	LogSGR_Trans	log/hr	=EXP(-6.2251 -(0.0114*NaCl) +(0.3234*Avg_Trans_Temp) +(0.002*(NaCL*Avg_Trans_Temp (0.0085*(NaCL*NaCL)) -(0.0045*(Avg_Trans_Temp* Avg_Trans_Temp)))				
Total log growth during transport	Trans_growth	log	=IF(Avg_Trans_Temp<MGT,0,Trans_Time/60*LogSGR_Trans)				

Table 6.35. Storage at home

Description	**Variable**	**Unit**	**Distribution or Equation**				
Home storage temperature	Home_Temp	degree C		Mean	SD	Min	Max
			Truncated Normal	4	2.65	-6.1	21.1
Home storage time	Home_Time	days		Min	ML	Max	CF
			Correlated PERT	0	2	5	-0.75
Log growth per hour	LogSGR_Home	log/hr	=EXP(-6.2251 -(0.0114*NaCL) +(0.3234*Home_Temp) +(0.002*(NaCl*Home_Temp)) -(0.0085*(NaCl*NaCL)) -(0.0045*(Home_Temp*Home_Temp)))				
Total log growth in home	Home_growth	log	+IF(Home_Temp<MGT,0,Home_Time*24*LogSGR_Home)				
Total log growth in storage, transport and home	Growth	log	Rtl_growth + Trans-growth + Home_growth				

Table 6.36. Cross-contamination during preparation

Description	Variable	Unit	Distribution or equation			
Number of organisms on bird	Num	cells	=IF(Conc=0,0,10^Conc)			
Chickens Þ Hands						
Transfer from chicken to hands?	XCH	-	=IF(Num=0,0,1)			
Proportion transferred from chicken	Pop_CH	proportion	Pert	Min	ML	Max
				0	0.1	0.15
Number on hands	Num_H	cell	=IF(XCH=0,0,Num*Prop_CH)			
Number left on chicken	Num_C1	cell	=Num -Num_H			
Hands Þ Other food						
Probability that hands are not washed	HW_Prob	-	Beta	alpha	beta	
				64	46	
Hands not washed?	HW	-	=binomial(1,HW_Prob)			
Proportion transferred from hands	Prop_HF	-	Pert	Min	ML	Max
				0.00	0.10	0.15
Number on other foods via hands	Num_OF1	-	=IF(HW=0,0,Num_H*Prop_HF)			
Chickens Þ Board						
Transfer from chicken to board	XCB	-	=IF(Num=0,0,1)			
Proportion transferred from chicken to board	Prop_CB	proportion		Min	ML	Max
			Pert	0	0.1	0.15
Number on board	Num_B	cell	=IF(XCB=0,0,Num*Prop_CB)			
Number left on chicken		cell	=NUm_C1 -Num_B			
Board Þ Other food						
Probability that board is used for other foods	Brd_use_Prob	-	Beta	alpha	beta	
				66	44	
Boards used for other food?	Brd_use	-	=binomial(1,Brd_use_Prob)			
Proportion transferred from board	Prop_BF	-	Pert	Min	ML	Max
				0.00	0.10	0.15
Number on other foods from chicken via board	Num_OF2	-	=IF(Brd_use=0,0,Num_B*Prop_BF)			
Number ingested via-cross-contamination	Num_XC	cell	=Num_OF1 +Num_OF2			
Ingestion via cross-contamination?	-	-	+IF(Num_XC=0,0,1)			

Table 6.37. Cooking

Description	Variable	Unit	Distribution or equation			
Probability of inadequate cooking	Prob_AC	-		Min	ML	Max
			Pert	0.05	0.10	0.15
Adequately cooked?	AC	-	=binomial(1,1-Prod_AC)			
Proportion o0f cells in areas that permit a chance of survival	Prop_Prot			Min	ML	Max
			Pert	0.10	0.16	0.20
Log number of cells with chance of survival	Num_Prot	log cells	=IF(Conc=0,0,LOG10(10^Conc*Prop_Prot))			
Exposure time at exposure temperature for cells in "protected area"	Time_Prot	minutes		Min	ML	Max
			Pert	0.50	1.00	1.50
Exposure temperature during cooking in "protected areas"	Temp_Prot	degree C		Min	ML	Max
			Pert	60	64	65
D-value (at this temperature)	D_Prot	minutes	=10^(-0.139*Temp_Prot +8.58)			
log reduction in "protected area"	Prto_LR	log	=IF(AC=1,"death",Time_Prot/D_Prot)			

Table 6.38. Consumption.

Description	Variable	Unit	Distribution or equation			
Weight of a broiler carcass	Broiler_WT	gram		Min	ML	Max
			Pert	1100	1500	2500
Proportion of edible meat	Prop_edible	-	Fixed. 0.3			
Weight of edible meat	Edible_WT	gram	=Broiler_WT*Prop_Edible			
Serving size	Serve_size	gram		Min	Max	
			Cumulative	19	550	
Number of servings per broiler	Num_Serve	-	=IF(Edible_WT<Serve_Size,1, ROUND(Edible_WT/Serve_Size,0)			

6.5 REFERENCES CITED IN CHAPTER 6

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7. RISK CHARACTERIZATION OF *SALMONELLA* IN BROILERS

7.1 SUMMARY

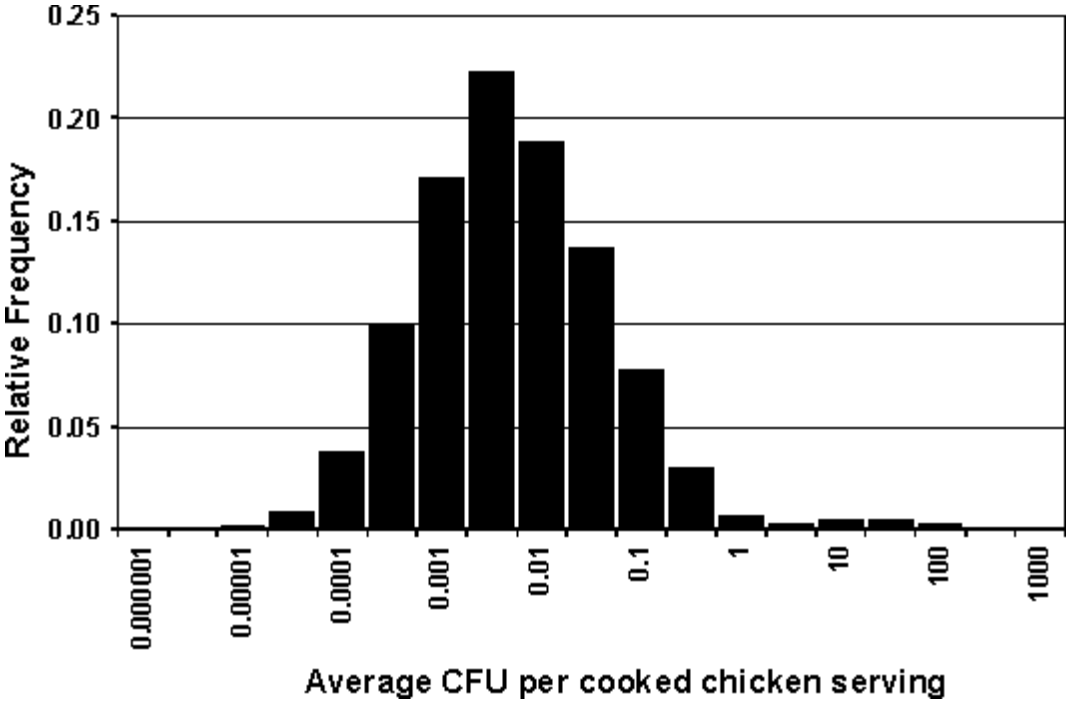
In this section, the results from the exposure assessment are used within the hazard characterization to estimate two quantities: the risk per serving and the risk from cross-contamination as a result of preparing that serving. As before, for the exposure assessment, the risk characterization is not specific to any country and thus comparison with surveillance data is not appropriate. Following calculation of the baseline model, the effect of a number of mitigation strategies is investigated.

7.2 RISK ESTIMATION

7.2.1 Results

The risk estimate for probability of illness was first simulated using a set prevalence for the presence of *Salmonella* in chilled, raw broiler chickens. At a prevalence of 20% contaminated carcasses, and based on the other model parameters, including the probability that the product will be undercooked, approximately 2% of the broilers prepared for consumption in the home could potentially contain viable cells of *Salmonella.* Figure 7.1 shows the distribution of average doses (colony-forming units, CFUs) per serving for contaminated chicken that is subsequently undercooked.

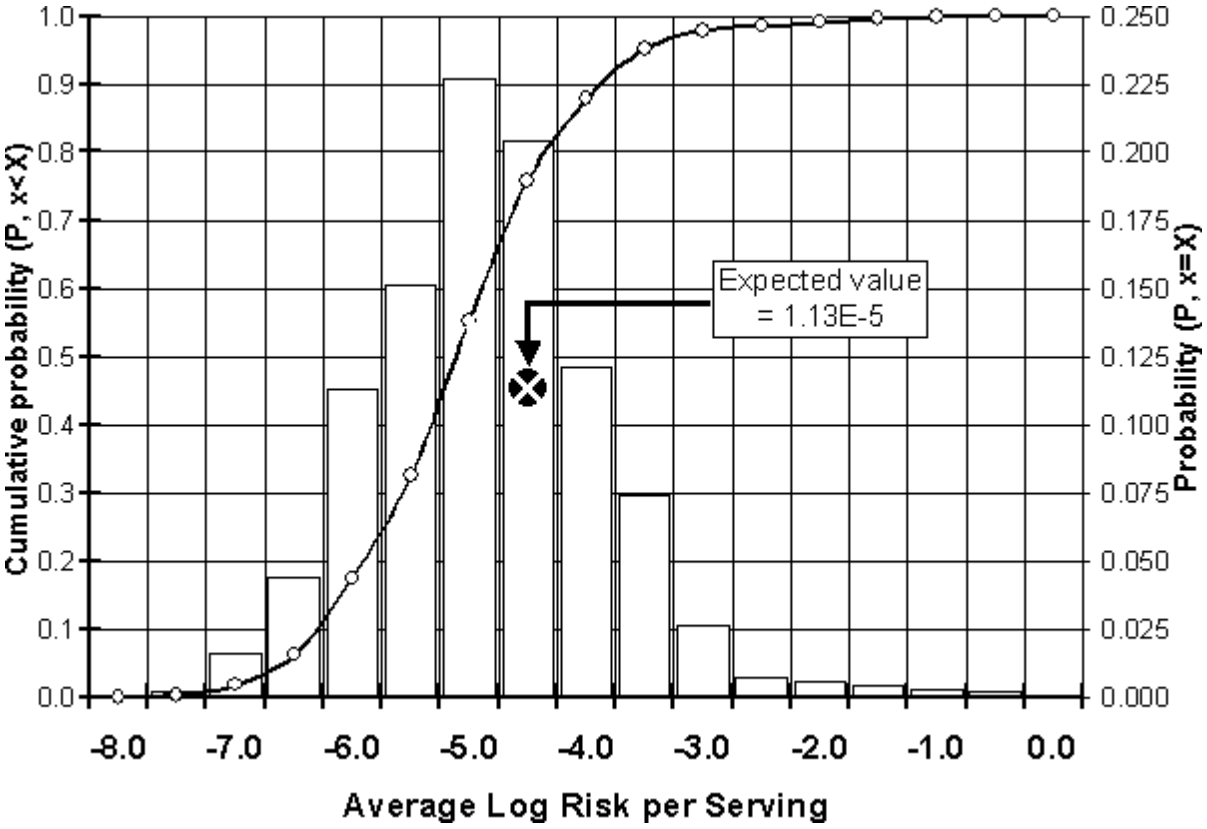
Figure 7.1. Average dose (CFU *Salmonella*) per serving in meals prepared from contaminated broilers.



Note that in Figure 7.1 the interpretation of values of less than 1 CFU per serving is 1 CFU per multiple servings, e.g. an average dose of 0.01 cells per serving translates to one in 100 servings contains a single cell.

Assuming a 20% prevalence of contaminated broilers, the estimated frequency and cumulative distribution of average risk per serving are shown in Figure 7.2. The expected risk per serving is 1.13E-5, or 1.13 illnesses per 100 000 servings. This value represents the average risk for all individuals in the population that consume servings of chicken that are stored, transported and prepared in the manner described in the model, and also accounts for the probabilities that the serving was from a chicken contaminated with *Salmonella*, and that the meal was undercooked. It should be recognized that some individuals consuming a serving on certain occasions would experience a much higher risk than others who may be consuming servings with no salmonellosis risk at all, since the serving is free of the pathogen.

Figure 7.2. Distribution of average risk per serving.



The expected risk per serving can be extended to the expected risk over multiple servings, such as meals consumed in a year. If it is assumed that the risk posed by one exposure (serving) is statistically independent from any other exposure (serving), then the overall risk following a series of exposures can be estimated using Equation 7.1:

$$P_A = 1 - \prod_{j=1}^{i} \left(1 - P_{Dj}\right)$$

Equation 7.1

where P_A is the risk of infection following a series of exposures (annual risk) and P_{Di} is the risk of infection per exposure (daily or serving risk). In order to estimate the annual risk of infection, two pieces of information are required: the risk of infection per serving, and the number of servings consumed in a year. The calculation of annual risk based on the estimated average per serving risk and the assumptions for this baseline scenario are illustrated in Table 7.1.

Table 7.1. Calculation of expected annual risk.

Prevalence of contaminated carcasses	20%
Expected risk per serving	1.13E-05
Number of servings in year	26
Annual expected risk	2.94E-04
Rate of illness per 100 000	29.38
Illustrative calculation for annual expected number of illnesses for a country or region with this annual expected risk:	
Population	20 000 000
Proportion of population that eats chicken	0.75
Potentially exposed population	15 000 000
Expected number of cases in the year	4406

The assumption inherent in the calculation above for the expected annual risk is that each of the servings consumed during the year has the same expected risk per serving and that the risk from each exposure is independent of every other exposure. The number of servings used to estimate the annual risk is assumed to be 26 meals, or once every 2 weeks. For illustration, a population risk for 20 million individuals was assumed to be under consideration, with 75% of that population eating chicken. In this example, the total expected number of salmonellosis cases arising from the model assumptions is estimated to be 4400, equivalent to a rate of 29 cases per 100 000 population. Obviously, these statistics need to be tailored for a specific country or region.

In addition to estimating the risk per serving based on consumption of undercooked poultry, the assessment also modelled the risk from cross-contamination. The sequence and nature of events that need to occur in order for the bacteria on raw chicken to be disseminated and ingested via other pathways is complex and difficult to model completely. There is a lack of information to adequately describe cross-contamination, but it is acknowledged that this is an important route for food-borne illness. The following estimates offer an approximation for the magnitude of the problem, although not all potential pathways were modelled that could result in exposure and illness.

In the baseline scenario, the expected risk from cross-contamination (transfer from raw chicken to hands to non-cooked foods, or from raw chicken to cutting board to non-cooked foods) was estimated to be 6.8E-4, or 6.8 illnesses per 10 000 exposures to contaminated material. This is more than an order of magnitude higher than the expected risk from a serving. This estimate is a function of two factors (conditional probabilities) in the current model: first, the expected risk when the event occurs, and, second, the expected probability that the event occurs.

The expected probability that the event occurs is driven by the prevalence of contamination **and** the probability of undercooking in the case of consumption, versus the prevalence of contamination **and**

the probability of not washing hands or not washing cutting boards in the case of cross-contamination. Given the assumptions made in the model, the expected risk from this cross-contamination pathway is equivalent to approximately 60 chicken consumption exposures. Although the frequency with which people do or do not wash their hands can be debated, the ultimate risk from cross-contamination could in fact be even higher than that estimated here since there are multiple cross-contamination opportunities that exist in the home preparation environment.

7.2.2 Validation of model results

Validation of results derived from microbiological risk assessments (MRAs) is often difficult, primarily due to the large uncertainties that are commonly associated with predictions. Surveillance data can be used for this purpose, but such use should account for sensitivity of detection and reporting methods. Downstream validation can also be used. In this case, intermediate results can be compared with data not used for model development. For example, predictions for the prevalence of contaminated products at the point of retail can be validated using data from retail surveys. The recognized problems associated with validation strengthen the fact that other outputs from risk assessment, for example the identification of data gaps and the ranking of control strategies, are often more useful than the predicted values.

The model developed here does not estimate the risk for a specific country and therefore it was not possible to attempt to validate the predicted results.

7.2.3 Impact of uncertain parameters on risk estimates

In generating the model, some of the input parameters were modelled as variable while others inputs were considered uncertain, so uncertainty and variability were not explicitly separated. Variability is a property of the phenomenon and the variations that are described are a reflection of what could be expected in nature. Uncertainty is driven by the lack of knowledge about the nature and behaviour of a phenomenon. Inputs that are derived from large representative data sets generated by scientifically sound methods are less uncertain than inputs that are based on sparse data, small sample sizes, or poor scientific methods, or a combination. Good data sets can be regarded as representing the actual variability of phenomenon. In contrast, uncertainty arises when assumptions must be made to generate a distribution around a single data point that is reported in the scientific literature (e.g. when only a mean value is available), or when little or no data exist. Although it is recommended that uncertainty and variability should be explicitly separated within the MRA, this would lead to a complex model. For this reason, the effect of uncertainty was investigated by considering the uncertain parameters in a separate analysis.

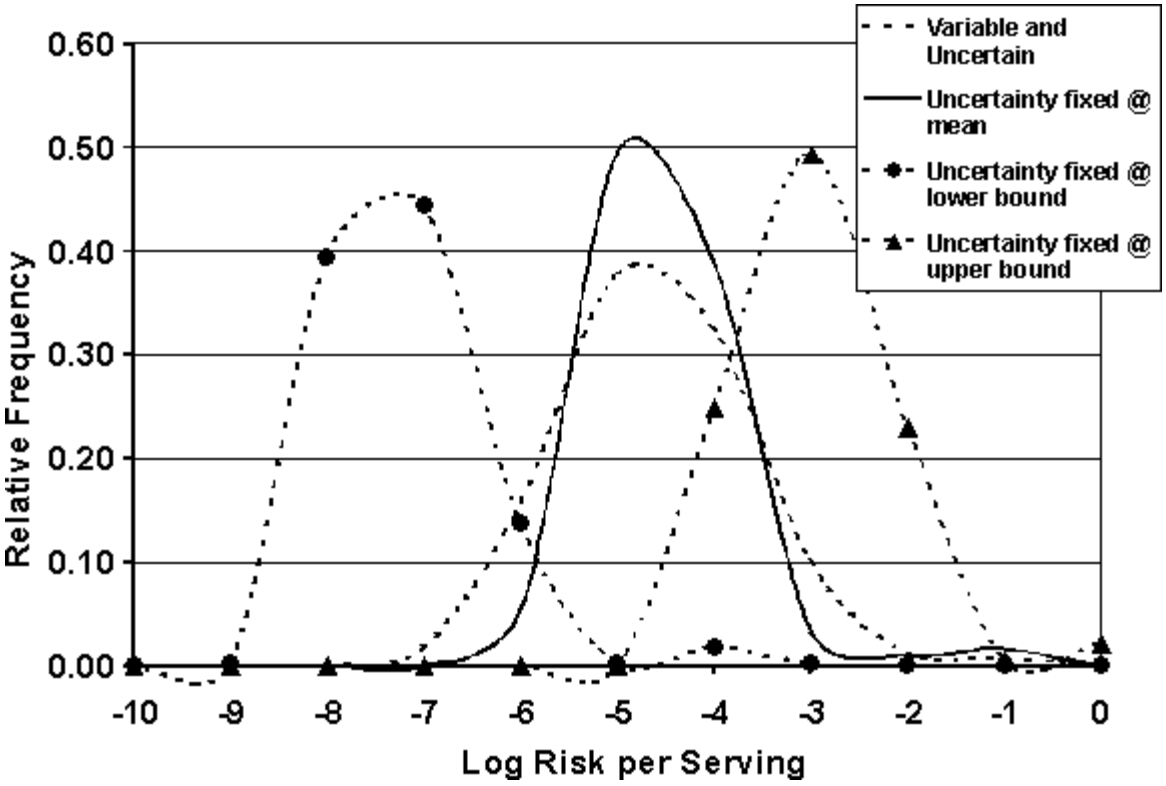
Several of the parameters in the cooking module were considered uncertain and are listed in Table 7.2. The impact of uncertainty in these parameters was investigated in order to evaluate their influence on the risk estimate. To do this, the model was re-simulated using a fixed single value for each of the uncertain parameters while allowing the other parameters of the model to vary within their defined distributions. Three simulations were performed: in the first, the parameters listed in Table 7.2 were set at their mean value. The fixed values used for the second simulation were those that would generate a "worst case" scenario, i.e. the maximum value for probability that the chicken was undercooked, the maximum value for proportion of cells in a protected region, the minimum heat exposure time, and the minimum value for the temperature reached in a protected region (0.15, 0.2, 0.5 minutes and 60°C, respectively). It is recognized that such a scenario may not occur in reality, but it gives an upper bound to the range of possible values. The third simulation used the values that would give a "best case" scenario, i.e. minimum value for probability undercooked, etc. This approach allowed the extremes in the risk distribution, driven by the uncertain parameters, to

be highlighted. The results of performing the analysis on the uncertain parameters influencing consumption risk are shown in Figures 7.3 and 7.4.

Table 7.2. Uncertain parameters in the cooking module.

Consumption relationship	Mean	Min.	Max.
Probability not adequately cooked	0.1000	0.0500	0.1500
Proportion in protected area	0.1567	0.1000	0.2000
Exposure time to cooking temperature of cells in protected areas	1.00	1.50	0.50
Cooking temperature reached in protected areas.	63.50	65.00	60.00

Figure 7.3. Effects of uncertain parameters on per serving risk distribution.

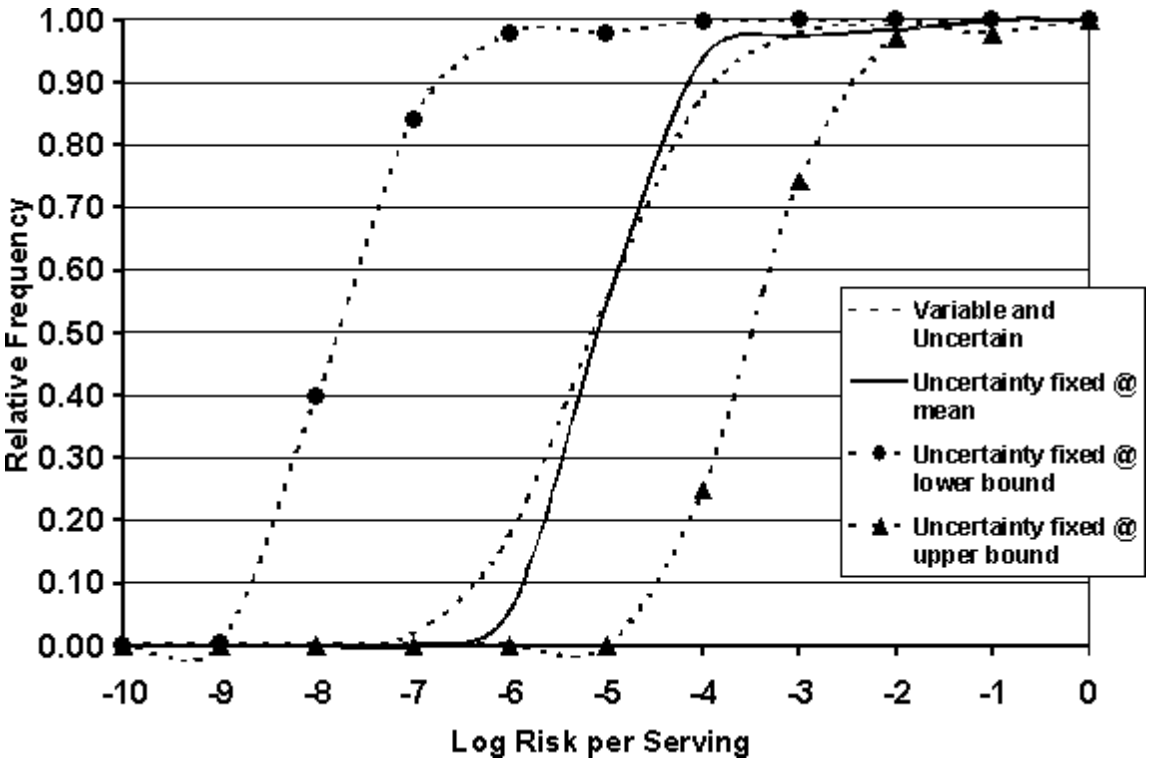


When the uncertain parameters were fixed at their mean values (Uncertainty fixed @ mean) and compared with the risk distribution generated by the model when all parameters were allowed to vary (Variable and Uncertain), it appears that within the range of uncertainty that was assumed to define the parameters, the impact of variation is not very large. The resulting risk distributions are similar and the tails of the currently defined uncertainty distributions do not have a dramatic impact on the overall risk uncertainty distribution. In other words, the range and shape of the distributions defining uncertainty do not influence the risk uncertainty significantly. Alternatively, if the assumptions made were incorrect and the uncertain parameters actually spanned a different range, e.g. if the true values are centred nearer to the min. or max. values rather than at the value assumed to be the mean, the distribution of risk would approach the extreme distributions shown. In these

situations, the expected risk would be dramatically different. It should be noted that the extreme risk distributions shown in Figures 7.3 and 7.4 are truly bounds on the uncertainty range since the worst case or best case scenario has been compounded through the model. For example, the worst case scenario was defined by assuming that all of the uncertain parameters would simultaneously take on the values that give the worst outcome.

A complete quantitative uncertainty analysis of the model and all input parameters was beyond the scope of this work. This type of analysis is time consuming and not necessarily more informative for the purposes of this document. Many of the inputs are generic approximations in order to provide a representative risk scenario. Nevertheless, it is important to recognize these two characteristics - uncertainty and variability - in the probability distributions used in quantitative risk assessments. It is also readily recognizable that several input parameters in this model are *both* variable and uncertain, and, if the individual parameters are important in determining the magnitude of the risk estimate, it may be necessary to separate the uncertainty and variability in the quantitative analysis in order to understand their impacts and arrive at proper risk estimations (Nauta, 2000).

Figure 7.4. Effects of uncertain parameters on per serving cumulative risk distribution.



7.3 RISK MANAGEMENT OPTIONS USING ALTERNATIVE ASSUMPTIONS

7.3.1 Reducing prevalence

A change in the prevalence of contaminated raw product affects the risk to the consumer by altering the frequency of exposure to risk events, i.e. exposure to the pathogen. The change in risk as a result of a change in the prevalence of *Salmonella*-contaminated broilers was estimated by simulating the model using a range of initial prevalence levels. Seven different prevalence levels were investigated: 0.05%, 1%, 5%, 10%, 20%, 50% and 90%. If the prevalence of contaminated chickens leaving processing is altered, through some management practice either at the farm level or at the processing

level, the expected risk per serving is altered. The magnitude of the changes in risk per serving and risk per cross-contamination event as a result of changes in prevalence are summarized in Table 7.3.

Table 7.3. Change in prevalence impact on risk.

	Prevalence						
	0.05%	**1.0%**	**5.0%**	**10.0%**	**20.0%**	**50.0%**	**90.0%**
Consumption							
Expected risk per serving	2.81E-08	5.63E-07	2.81E-06	5.63E-06	1.13E-05	2.81E-05	5.07E-05
Number of servings	26	26	26	26	26	26	26
Annual expected risk	7.32E-07	1.46E-05	7.32E-05	1.46E-04	2.93E-04	7.31E-04	1.32E-03
Rate of illness per 100 000	0.07	1.46	7.32	14.63	29.26	73.14	131.61
Calculation of expected number of cases in the year based on assumed population size and exposed population							
Population			20 000 000				
Proportion of population that eats chicken			0.75				
Potentially exposed population			15 000 000				
Expected number of cases in the year	11	219	1 097	2 195	4 389	10 970	19 741
Cross-contamination							
Expected risk per event	1.70E-06	3.41E-05	1.70E-04	3.41E-04	6.81E-04	1.70E-03	3.07E-03

A reduction of 50% in the number of cases of salmonellosis was estimated if a 20% contamination rate at the retail level was reduced to 10% contamination. The relationship between prevalence and expected risk is largely a linear one, specifically a percentage change in prevalence, assuming everything else remains constant, can be expected to reduce the expected risk by the same percentage.

The effectiveness of specific mitigations, either on-farm or treatments during processing, were not evaluated in the present risk model because of lack of representative data to analyse changes in either or both prevalence and level of contamination that might be attributable to a specific intervention. See Section 7.3.4 for a summary of poultry processing treatments. However, the influence of reducing prevalence can be interpreted, although with a high degree of uncertainty given our current state of knowledge, in the context of chlorine addition to the chill tanks during

processing. There is little evidence that the addition of chlorine at levels of 50 ppm or less actually decreases the numbers of the pathogen attached to the skin of poultry carcasses. However, available data suggest that chlorine prevents an increase in the prevalence of contaminated carcasses, i.e. a reduction in cross-contamination (Table 7.4), although one study observed a substantial reduction in prevalence. The factor listed in the last column of the table is a ratio of the prevalence after chilling to the prevalence before chilling. A ratio greater than 1 indicates an increase in prevalence of contaminated carcasses.

Table 7.4. Experimental data for effects of chlorine on *Salmonella* prevalence after immersion chill tank.

Ref.	Amount	Prevalence before chilling			Prevalence after chilling			Ratio[1]
		Total	Positive	Prevalence	Total	Positive	Prevalence	
With Chlorine								
[1]	20-50 ppm (tank)	48	48	100%	103	60	58%	0.58
[2]	4-9 ppm (overflow)	50	21	42%	50	23	46%	1.10
[3]	1-5 ppm (overflow)?	90	18	20%	90	17	19%	0.94
[4]	15-50 ppm (tank)	48	4	8%	96	7	7%	0.88
								0.87
Without Chlorine								
[5]	-	160	77	48%	158	114	72%	1.50
[6]	-	99	28	28%	49	24	49%	1.73
[7]	-	40	5	13%	40	11	28%	2.20
[7]	-	40	4	10%	40	15	38%	3.75
[7]	-	84	12	14%	84	31	37%	2.58
[8]	-	60	2	3%	120	18	15%	4.50
								2.71

NOTES: (1) Ratio of prevalence after chilling to prevalence before chilling. A ratio >1 indicates an increase in prevalence of contaminated carcasses.
DATA SOURCES: [1] Izat et al., 1989. [2] James et al., 1992a. [3] Cason et al., 1997. [4] Campbell 1983. [5] James et al., 1992a. [6] James et al., 1992a. [7] Lillard, 1980. [8] Campbell, 1983.

7.3.2 Reduction in numbers of *Salmonella* on contaminated carcasses

The effect was assessed of reducing the numbers of *Salmonella* on poultry carcasses without changing the prevalence of contaminated carcasses. The values of the cumulative concentration distribution used in the baseline scenario were reduced by 50% (approximately 0.3 logCFU per carcass; Figure 7.5). The model was run using the reduced level of conta-mination while maintaining the prevalence at 20% and with no changes in any of the other parameters. Figure 7.6 shows a comparison of the per serving risk estimates for the modified simulation against the original data.

Figure 7.5. Original and post-intervention concentration distributions.

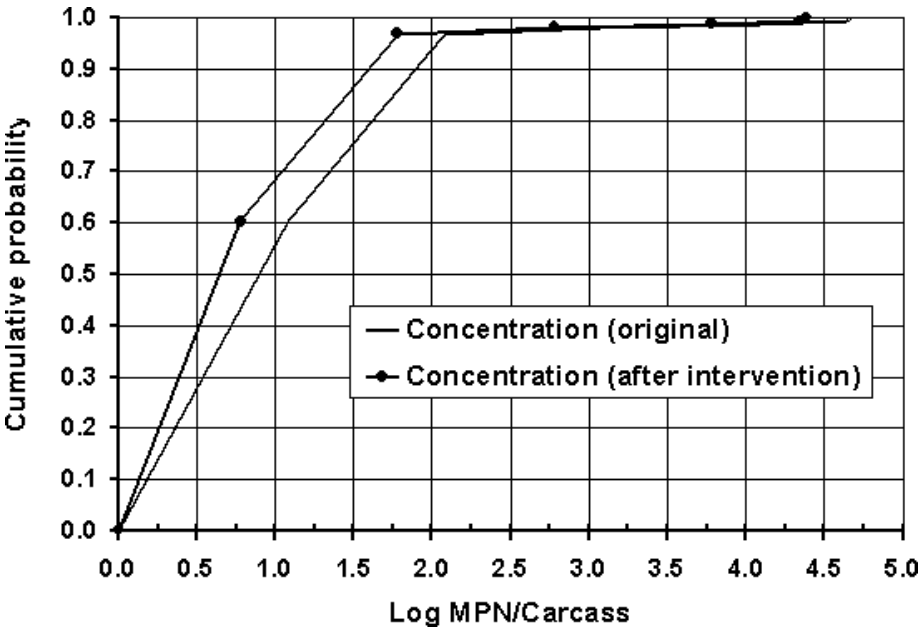
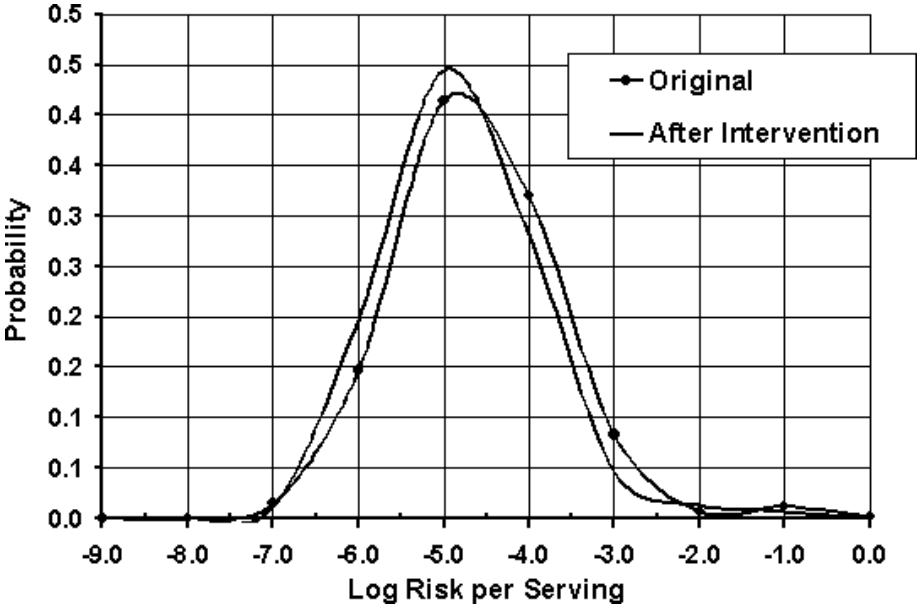


Figure 7.6. Risk per serving distribution before and after concentration-changing intervention.



Unlike a change in prevalence, a change in concentration of the pathogen does not necessarily have a linear relationship with the risk outcome. The distribution of risk shown in Figure 7.6, similar to the distribution of risk per serving shown earlier, is the risk per serving when contaminated. The servings were estimated to be contaminated and potentially undercooked approximately 2% of the time. That statistic remains unchanged if the level of contamination is reduced.

The expected risk per serving, which incorporates the prevalence of contaminated servings and the probability of undercooking, was estimated to be 1.13E-5 (1.13 illnesses per 100 000 servings) in the original case, and 4.28E-6 (4.28 per 1 000 000 servings) in the situation when the level of contamination is reduced. The expected risk per serving is therefore reduced by approximately 62%. A summary of the results is shown in Table 7.5.

Table 7.5. Risk summary before and after intervention to change concentration.

	Original	**After Intervention**
Prevalence	20%	20%
Expected risk per serving	1.13E-05	4.28E-06
Number of servings in year	26	26
Annual expected risk	2.94E-04	1.11E-04
Rate of illness per 100 000	29	11
Illustrative calculation for annual expected number of illnesses for a country/region with this annual expected risk		
Population	20 000 000	20 000 000
Proportion of population that eats chicken	0.75	0.75
Potentially exposed population	15 000 000	15 000
Expected number of cases in the year	4406	000 1670

The risk from cross-contamination events is also affected when the level of contamination is reduced.

7.3.3 Change in consumer behaviour and the impact on risk

Finally, a change in consumer practices can have an impact on risk. The consumer represents the final intervention in mitigating the risk. However, the effectiveness of strategies aimed at changing consumer behaviour is difficult to anticipate, and difficult to measure. For purposes of this assessment, the potential impact on risk by modifying food preparation practices was investigated by running the simulation assuming that a strategy is implemented which changes consumer behaviour. The assumed changes were as follows:

- probability that product is not adequately cooked:

(OLD): Min = 5%, Most likely = 10%, Max = 15%

(NEW): Min = 0%, Most likely = 5%, Max = 10%

- exposure time (minutes):

(OLD): Min = 0.5, Most likely = 1.0, Max = 1.5

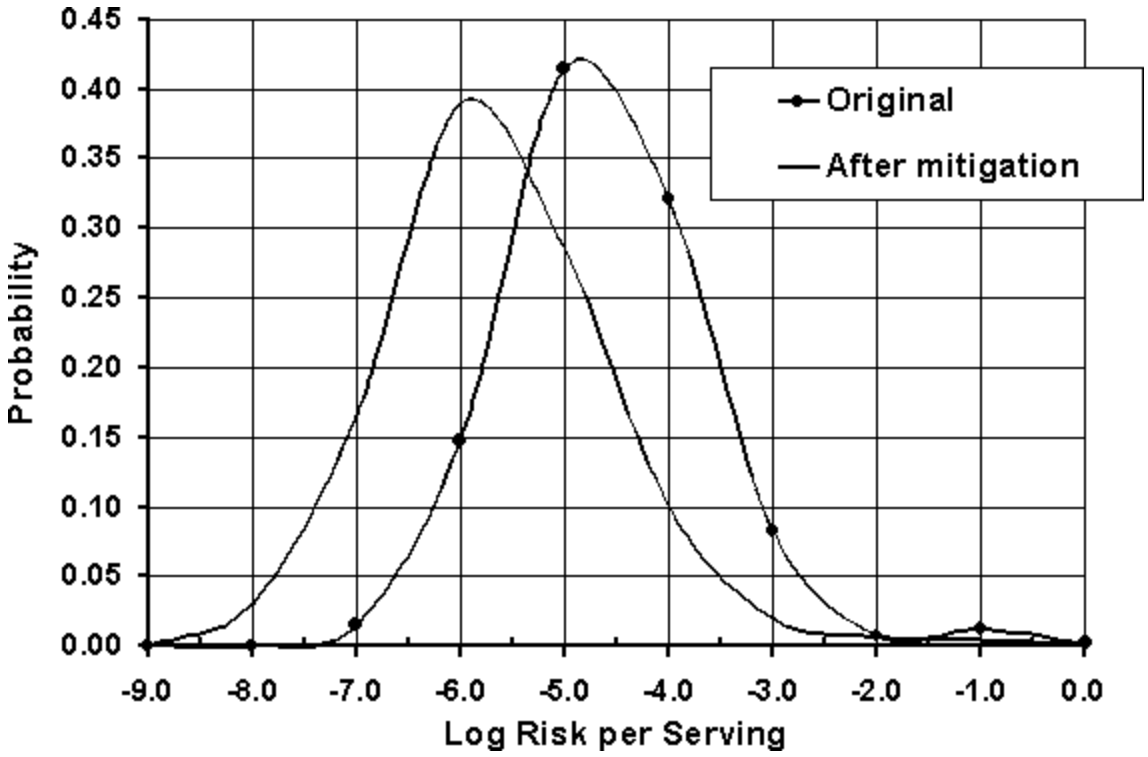
(NEW): Min = 1.0, Most likely = 1.5, Max = 2.0

The changes are thus assumed to reduce the probability of the consumer not adequately cooking their food, and, for those that do tend to undercook, the degree of undercooking is less.

If the simulation model is re-run with these assumptions, the expected risk is reduced to 2.22E-6 (2.22 illnesses per 1 000 000 servings) from 1.13E-5 (1.13 illnesses per 100 000 servings). As a result, the changes in consumer practices reduce the expected risk per serving by almost 80%. The changes in consumer practices have an impact on the frequency with which a potentially contaminated product remains contaminated prior to consumption (probability of undercooking) and reduces the risk when the potentially contaminated product reaches the consumer as well (longer cooking time). The distribution of risk per serving before and after the intervention is shown in Figure 7.7.

It is important to note that the mitigation strategy to alter cooking practices does not address the cross-contamination risk. In the baseline scenario, the expected risk per cross-contamination event was shown to be much larger than the risk from consumption. As a result, the strategy to change the consumers cooking practices needs to be tempered by the fact that cross-contamination may in fact be the predominant source of risk and the nature of cross-contamination in the home is still a highly uncertain phenomenon.

Figure 7.7. Risk distribution per serving before and after consumer behaviour altering intervention.



7.3.4 Intervention methods for controlling *Salmonella* on poultry

SUBSTRATE	CONTROL LEVEL	REDUCTION REPORTED	CONDITIONS	REFERENCE
CHEMICAL TREATMENT - Acetic acid				
Broiler carcasses	0.6%	Significant reduction: 96% of controls positive while treated carcasses were only 8% positive	Used with air injection in a 10-minute pre-chill at 10°C	Dickens and Whittemore, 1994
Chicken carcasses	0.6%	Reduction of 0.34 $\log_{10}$, darkened the carcasses and caused the feather follicles to protrude	1 hour static ice slush in chill tank	Dickens and Whittemore, 1995
Chicken carcasses	0.6%	Reduction of 0.62 $\log_{10}$, darkened the carcasses and caused the feather follicles to protrude	1 hour static ice slush with air injection	Dickens and Whittemore, 1995

Chicken carcasses	0.6%	Reduction of 1.16 log_{10}, darkened the carcasses and caused the feather follicles to protrude	1 hour with paddle chiller	Dickens and Whittemore, 1995
Chicken breast skin	5%	2.5 log_{10} reduction on loosely *attached S.*Typhimurium populations	Chiller for 60 minutes, 0°C	Tamblyn and Conner, 1997
Chicken breast skin	5%	2.0 log_{10} reduction on firmly attached *S.* Typhimurium populations	Scalder for 2 minutes, 50°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Calcium hypochlorite				
Chicken carcasses	20 ppm available chlorine	No reduction	15 minutes, 25°C	Nassar et al., 1997
Chicken carcasses	50 ppm available chlorine	No reduction	15 minutes, 25°C	Nassar et al., 1997
Chicken carcasses	100 ppm available chlorine	3/10 inoculated (10^4CFU) carcasses negative after treatment, but yellow appearance and strong chlorine smell	15 minutes, 25°C	Nassar et al., 1997
Chicken carcasses	200 ppm available chlorine	7/10 inoculated (10^4CFU) carcasses negative after treatment, but yellow appearance and strong chlorine smell	15 minutes, 25°C	Nassar et al., 1997
CHEMICAL TREATMENT - Sodium hypochlorite				
Chicken carcasses	200 ppm available chlorine	99.9% reduction in *Salmonella* count; did not affect odour or flavour of the cooked meat	15 minutes, 25°C	Morrison and Fleet, 1985
Chicken breast skin	400 ppm	2.3 log_{10} reduction on loosely *attached S.* Typhimurium populations and 1.3 log_{10} reduction on firmly	Chiller for 60 minutes, 0°C	Tamblyn, Conner and Bilgili, 1997

		attached *S.* Typhimurium populations		
Chicken breast skin	800 ppm	2.5 log_{10} reduction on loosely *attached* *S.* Typhimurium populations and 1.9 log_{10} reduction on firmly attached *S.* Typhimurium populations	Chiller for 60 minutes, 0°C	Tamblyn, Conner and Bilgili, 1997
CHEMICAL TREATMENT - Lactic acid				
Chicken carcasses	0.75%	4/10 inoculated (10^4CFU) carcasses negative after treatment, discoloration, slimy skin and tears in skin	pH 2.78, 15 minutes, 25°C	Nassar et al., 1997
Chicken carcasses	0.75%	5/10 inoculated (10^4CFU) carcasses negative after treatment, slimy skin and tears in skin	pH 2.68, 15 minutes, 25°C	Nassar et al., 1997
Chicken skin	1%	"Significant reduction"	Inoculated skin washed by agitating solution for 30 minutes at 25°C	Hwang and Beauchat, 1995
Chicken carcasses	1.0%	10/10 inoculated (10^4CFU) carcasses negative after treatment, slimy skin and tears in skin	pH 2.47, 15 minutes, 25°C	Nassar et al., 1997
Chicken skin	1%	2.2 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 seconds at 206 kPa and 20°C.	Xiong et al., 1998
Chicken skin	2%	2.2 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 sec at 206 kPa and 20°C.	Xiong et al., 1998
Chicken breast skins	o4%	2 log reduction of firmly attached cells, but bleaching and off odour	Scalder for 2 minutes at 50°C	Tamblyn and Conner, 1997

Chicken breast skins	6%	2 log reduction of loosely attached cells, but bleaching and off odour	Chiller for 60 minutes, 0°C; scalder for 2 minutes at 50°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Mandelic acid				
Chicken breast skins	6%	2 log reduction of loosely attached cells, but bleaching and off odour	Chiller for 60 minutes, 0°C; scalder for 2 minutes at 50°C	Tamblyn and Conner, 1997
Chicken breast skins	4%	2 log reduction of firmly attached cells, but bleaching and off odour	Chiller for 60 minutes, 0°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Malic acid				
Chicken breast skins	4%	2 log reduction of both loosely and firmly attached cells, but bleaching and off odour	Chiller for 60 minutes, 0°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Propionic acid				
Chicken breast skins	4%	2 log reduction of firmly attached cells, but bleaching and off odour	Chiller for 60 minutes, 0°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Tartaric acid				
Chicken breast skins	6%	2 log reduction of firmly attached cells, but bleaching and off odour	Scalder for 2 minutes at 50°C	Tamblyn and Conner, 1997
CHEMICAL TREATMENT - Peroxidase catalysed chemical dip (PC)				
Broilers	0.1 M citric acid + 0.1 M sodium citrate, ratio 1:1.5	"Significant reduction"	pH 5.0, 30 minutes	Bianchi et al. 1994
CHEMICAL TREATMENT - Hydrogen peroxide				
Chicken carcasses	2%	3/10 (10^4CFU) carcasses negative after treatment, bleaching, bloating and brown spots on skin	pH 4.40, 15 minutes, 25°C	Nassar et al. 1997
Chicken carcasses	3%	7/10 (10^4CFU) carcasses negative	pH 4.77, 15 minutes, 25°C	Nassar et al. 1997

		after treatment, bleaching, bloating and brown spots on skin		
CHEMICAL TREATMENT - Sodium metabisulphite				
Chicken breast skin	1%	No reduction.	Three application methods: chiller for 60 minutes, 0°C; scalder for 2 minutes, 50°C; dip for 15 seconds, 23°C	Tamblyn, Conner and Bilgili, 1997
CHEMICAL TREATMENT - NaOH				
Chicken skin	0.05%	"Significant reduction"	Inoculated skin washed by agitating solution for 30 minutes at 25°C	Hwang and Beauchat, 1995
CHEMICAL TREATMENT - AvGard® (TSP)				
Broiler carcasses	100 g/kg w/w	Greater than 2 log reduction	Immersion tank for 15 seconds	Coppen, Fenner and Salvat, 1998
CHEMICAL TREATMENT - Trisodium phosphate (TSP)				
Post-chill chicken carcasses	10%	Significant reduction (ca 1.6-1.8 logs) at both 1 and 6 days post-treatment. Although 50°C-TSP gave 0.4 log greater reduction than 10°C, the difference was not significant.	Dipped in solution at 10°C or 50°C for 15 seconds.	Kim et al., 1994
Chicken skin	1%	"Significant reduction"	Inoculated skin washed by agitating solution for 30 minutes at 25°C	Hwang and Beauchat, 1995
Chicken skin	1% plus 5% Tween 80	Reduction improved from the use of 1% TSP alone	Inoculated skin washed by agitating solution for 30 minutes at 25°C	Hwang and Beauchat, 1995
Chicken skin	5%	2.1 $\log_{10}$ reduction	Pre-chill spray to inoculated chicken skin for 30 seconds at 206 kPa + 20°C.	Xiong et al., 1998

Chicken breast skin	8%	1.6 log_{10} reduction on loosely *attached S.* Typhimurium populations and 1.8 log_{10} reduction on firmly attached *S.* Typhimurium populations	Chiller for 60 minutes, 0°C	Tamblyn, Conner and Bilgili, 1997
Chicken breast skin	8%	1.8 log_{10} reduction on firmly attached *S.* Typhimurium populations	Dip for 15 seconds, 23°C	Tamblyn, Conner and Bilgili, 1997
Chicken breast skin	8%	1.5 log_{10} reduction on loosely *attached S.* Typhimurium populations	Scalder for 2 minutes, 50°C	Tamblyn, Conner and Bilgili, 1997
Chicken carcasses	10%	Salmonellae not detected on 25-g neck skin sample	pH 12, 15 seconds, 20°C	Whyte et al., 2001
Chicken skin	10%	2.2 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 second at 206 kPa and 20°C.	Xiong et al., 1998
CHEMICAL TREATMENT - Cetylpyridinium chloride (CPC)				
Chicken skin	0.1%	CPC spraying reduced numbers by 0.9 to 1.7 log units (87 to 98%). Generally, 50°C spraying showed greater reduction than 15°C, but the difference was not always significant.	Solution sprayed against inoculated skin samples at 15°C or 50°C for 1 minute, at 138 kPa.	Kim and Slavik, 1996
Chicken skin	0.1%	Reduction ranged from 1.0 to 1.6 log units (90 to 97.5%). Longer immersion times were more effective. Based on amount of CPC used, immersion appears more cost effective than spraying CPC.	Immersion of inoculated skin surface at room temperature for either 1 minute, 1 minute + 2 minutes holding without CPC, or 3 minutes	Kim and Slavik, 1996
Chicken skin	0.1%	1.5 log_{10} reduction	Pre-chill spray to inoculated chicken	Xiong et al., 1998

			skin for 30 seconds at 206 kPa and 20 C.	1998
Chicken skin	0.5%	1.9 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 seconds at 206 kPa and 20°C.	Xiong et al., 1998
CHEMICAL TREATMENT - Grapefruit seed extract (DF-100)				
Chicken skin	0.1%	1.6 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 seconds at 206 kPa and 20°C.	Xiong et al., 1998
Chicken skin	0.5%	1.8 log_{10} reduction	Pre-chill spray to inoculated chicken skin for 30 seconds at 206 kPa and 20°C.	Xiong et al., 1998
SCALD TREATMENTS				
Chicken carcasses	Scald temperatures of 52 C, 56°C and 60°C	Carcasses scalded at 52°C or 56°C showed ~0.3 to 0.5 log reduction greater than those at 60°C	52°C for 2.0 minutes, 56°C for 1.5 minutes and 60°C for 1.0 minute	Slavik, Kim and Walker, 1995
Chicken carcasses	Countercurrent scalding and postscald spray	Changes contributed to substantial improvement in the bacterial quality of carcasses, but additional interventions in the chilling process (such as chlorination of chill water) are necessary	240 ml of 60°C water was sprayed on each carcass at a pressure of 40 lbs/sq inch (psi)	James et al., 1992b
CHILL WATER IMMERSION TANK TREATMENTS				
Chicken carcasses	Chill water without chlorination	Prevalence increased during immersion chilling	I hour in typical drag-through chiller	James et al., 1992a
Chicken carcasses	Chill water with 25 ppm chlorination	Prevalence remained constant with chlorination	I hour in typical drag-through chiller	James et al., 1992a
RADIATION - Cobalt 60				
Chicken carcasses	3 k gray	5/10 inoculated (10^4CFU) carcasses	57 minutes/kGy of radiation	Nassar et al. 1997

carcasses		negative after treatment, no effect on colour, appearance or smell	radiation	1997
Chicken carcasses	4 k gray	8/10 inoculated (10^4CFU) carcasses negative after treatment, no effect on colour, appearance or smell	57 minutes/kGy of radiation	Nassar et al. 1997
Chicken carcasses	7 k gray	10/10 inoculated (10^4CFU) carcasses negative after treatment, no effect on colour, appearance or smell	57 minutes/kGy of radiation	Nassar et al. 1997
RADIATION - Gamma radiation				
Mechanically de-boned chicken meat (MDCM)	3.0 kGy	Reduction of 6.38 logs	Cesium-137 gamma radiation source, irradiated in air, at +20°C	Thayer and Boyd, 1991
RADIATION - Ultraviolet				
Halved broiler breast with skin on	2 000 lWs/cm^2	80.5% reduction	2 cm^{-2} skin pieces were inoculated with 50 ll of solution containing $7x10^5$ CFU/ml and the UV intensity was kept constant at 81.7 lWs/cm^2 while the treatment times were 20, 40, 60, 90 and 120 seconds.	Summer et al., 1996
Halved broiler breast with skin on	82 560 to 86 400 lWs/cm^2	61% reduction compared with untreated halves. No negative effect on colour or increased rancidity of the meat	Halves were inoculated 5 minutes prior to exposure at a wavelength of 253.7 nm	Wallner-Pendleton et al., 1994
AIR SCRUBBING				
Broiler carcasses	Diffused air, 158.6 kPa in tap water	Water only: 32/40 positive. Air scrubbed: 9/40 positive.	30 minutes	Dickens and Cox, 1992
LINE SPEED				

Broiler carcasses	Processed at 70, 80, and 90 birds per minute	Prevalence did not change significantly with processing line speeds.		Brewer et al., 1995

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8. DATA GAPS AND FUTURE RESEARCH NEEDS

One of the important outcomes of the risk assessment work was the compilation of a wealth of information on *Salmonella* in eggs and broiler chickens. The organization of these data in the structured risk assessment format has enabled the identification of the key gaps that exist in the data. This can provide guidance for future research work and help ensure that it is targeted towards generating and collecting the most useful and relevant data. These data and research needs are outlined below.

8.1 HAZARD CHARACTERIZATION

In order to improve the hazard characterization, additional outbreak and epidemiological data are needed. More specifically, these data should indicate cell number in the implicated food, amount of food consumed, accurate estimates of the size of ill and exposed populations, and accurate characterization of the population, including age profiles, medical status, sex and other potential susceptibility factors.

The impact of the food matrix was not incorporated into the hazard characterization due to the limitations of available data. So that these issues can be more completely addressed in future work, there is a need for characterization and quantification of the impact of the food matrix effects and also host-pathogen interactions and virulence factors and their effect on the probability of infection or illness, or both. Quantitative information to facilitate estimating the probability of developing sequelae following illness is also required.

As this is a developing science, the optimal models have not yet been developed. Therefore, new dose-response models that improve the ability to estimate the probability of illness would be useful.

8.2 EXPOSURE ASSESSMENT OF S. ENTERITIDIS IN EGGS

Data relating to the biology of *S.* Enteritidis in eggs is needed. This need is seemingly universal in its application to previous and future exposure assessments.

Additional studies on the numbers, and factors that influence the survival and growth of *S.* Enteritidis in naturally (yolk) contaminated intact shell eggs are needed, as information is currently available for only 63 intact shell eggs. Enumeration data of *S.* Enteritidis in raw liquid egg are also required. Additional data concerning the numbers of *S.* Enteritidis in raw liquid egg before pasteurization would assist in reliably predicting the effectiveness of such a regulatory standard concerning egg products.

More data on the prevalence of *S.* Enteritidis in breeder and pullet flocks and the environment, as well as in feedstuffs, is needed to adequately assess the benefit of pre-harvest interventions. In particular, associations between the occurrence of *S.* Enteritidis in these pre-harvest steps and its occurrence in commercial layers should be quantified.

Better data on time and temperature, specifically in relation to egg storage, and to preparation and cooking, would serve to build confidence in the modelling results. The importance of time and temperature distributions in predicting growth of *S.* Enteritidis in eggs, combined with the lack of reliable data to describe these distributions, highlights the need for these data. Furthermore, new studies are needed on the relationship between cooking time, cooking method and cooking temperature and the death of *S.* Enteritidis.

More studies are needed on the survival and growth of *S.* Enteritidis in eggs, particularly as a function of egg composition and the attributes of infecting strains of organism (e.g. heat sensitivity).

8.3 EXPOSURE ASSESSMENT OF *SALMONELLA* IN BROILER CHICKENS

As indicated earlier in the document, the lack of good quality data, prior to the end of processing in particular, limited the scope of this exposure assessment. In relation to primary production, the information available was mainly prevalence data, but for some regions of the world - including Africa, Asia and South America - even that was limited. In addition, information on study design, specificity or sensitivity of the analytical methodologies used was lacking. Very few quantitative data were available. A similar situation was observed for the processing stage. In addition, data tended to be old, and knowledge of processing practices was not readily available. In order to address these deficiencies, the areas where data collection and research efforts need to focus are identified below.

- Data on prevalence for many regions of the world regarding *Salmonella* in broilers during production and at slaughter, and on carcasses post-processing, together with information on study design.

- Microbial ecology studies to determine sources and numbers of the pathogen.

- Studies on the correlation between within-flock prevalence levels and the number of *Salmonella* cells shed in faeces or on birds.

- Precise estimates of the numbers of organisms per bird for all stages of the exposure pathway and improvements in the sensitivity and availability of cost-effective methods to enumerate small populations of *Salmonella.*

- Between-bird (bird-to-bird) cross-contamination data suitable for modelling this phenomenon at the pre-harvest, transport and processing stages.

- Data on the survival of *Salmonella* under chilling and freezing conditions. This information will improve the predictive microbiology component of exposure assessments relevant to international trade in poultry products.

- Specific consumption data and information about food preparation practices for most geographical locations, preferably presented as portion size and frequency of consumption rather than average consumption per day.

- Information on the distribution of time and temperature for storage and cooking in domestic kitchens in a variety of national environments.

- Data on the magnitude of cross-contamination in the domestic kitchen and the pathways for cross-contamination.

If an attempt were made to extend the risk assessment to assess more fully pre-slaughter interventions, then more data would be required on the prevalence of *Salmonella* in feed and replacement stock, and on fasting prior to slaughter. Data on the effect of scalding, de-feathering, evisceration, washing and chilling processes, as well as other decontamination treatments, are needed to effectively model the benefits of control interventions at the levels of processing.

9. THE APPLICATION OF MICROBIOLOGICAL RISK ASSESSMENT

Quantitative microbiological risk assessment is intended to answer specific questions of importance to public health. For microbiological risk assessment to deliver benefits it needs to be purposefully incorporated into the decision making process. This implies a change in the way nations approach food safety and public health decisions. The novelty of microbiological risk assessment is that it quantifies the hazard throughout the food production chain and directly links this to the probability of food-borne disease. The risk assessments of *Salmonella* in eggs and broiler chickens present an example of the potential of this approach.

The increased use of microbiological risk assessment will result in new capacity building needs. The exercise of producing this risk assessments has been a learning experience and since it is comprehensive, it can also provide a basis for future training efforts and applied research. These risk assessments are a resource that can be used by many parties including the Codex Alimentarius and national authorities. Ensuring their applicability and utility to all regions and countries is a priority for future work in FAO and WHO.

An important prerequisite for microbiological risk assessment is the need for an interdisciplinary approach. There is a dual need to develop the capacity for microbiological risk assessment skills and expertise within all the relevant disciplines (microbiology, modelling, epidemiology, etc.) and to ensure that these disciplines become effectively integrated into the risk assessment process. Transparency must be maintained throughout the risk assessment process from the initial stages of building the risk assessment team, to data collection and analysis.

This exercise in conducting risk assessment at the international level has underlined the need for data to be acquired from all regions and for the development of countries' capacities to conduct risk assessments. The development of these capacities requires an infrastructure for the surveillance of food-borne disease and the monitoring of microbial hazards in foods throughout the food-chain and the effect of processing and other factors on the micro-organism. It also requires human resources with the technical skills needed to conduct microbiological risk assessment.

There is a considerable amount of useful information made available through these risk assessments for both risk assessors and risk managers. The concepts presented are generic, and may be directly adaptable or considered as stand-alone modules. For those planning to undertake a quantitative microbiological risk assessment the models developed can be used as a template for undertaking risk assessment for these pathogen-commodity combinations at regional or national levels. The data used in the models, however, must reflect the food item, raw material, manufacture, retail conditions, and consumption habits as well as the characteristics of the population within the region under consideration.

These *Salmonella* risk assessments provide information that may be useful in determining the impact that intervention strategies have on reducing cases of salmonellosis from contaminated eggs and poultry. This information is of particular interest to the Codex Alimentarius in their work on the elaboration of standards, guidelines and related texts. Furthermore, in undertaking this work a number of lessons were learned with regard to making optimal use of risk assessment as a decision support tool. In order to meet the needs of risk managers, the risk assessment must be clearly focussed. This can be achieved by adequate planning, good communication and a strong interface between the risk assessors and the risk managers. To ensure that risk assessment contributes to management decisions that can be successfully implemented, there needs to be communication from the outset with other relevant stakeholders such as the food industry and consumers.

In conclusion, the risk assessments provide an example of a format for organising the available information in a readable way, and connecting pathogen contamination problems in food with human health outcomes. They provide scientific advice and analysis that may be useful for establishing regulatory policies for control of foodborne disease in different countries. In addition, the risk assessment process has identified important data gaps, and includes recommendations for future research, which can be used to allocate resources to priority areas.

These are the first microbiological risk assessments to be undertaken at the international level. During the course of the work it was recognized that MRA is still a developing science, yet, every effort has been made to provide a valuable and unique resource for those undertaking risk assessments and addressing the problems associated with *Salmonella* in eggs and broiler chickens.

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This volume contains monographs on risk assessment of *Salmonella* in eggs and broiler chickens that have been prepared and reviewed by an international team of scientists. During their preparation input was received from several international fora including expert consultations and Codex Alimentarius committee meetings as well as via public and peer review.

The monographs in this volume comprise data and methodology relevant to the four steps of risk assessment - hazard identification, exposure assessment, hazard characterization and risk characterization - of *Salmonella* in eggs and broiler chickens. They include information on the efficacy of some of the possible risk management options for controlling these pathogens in eggs and broiler chickens.

This volume and others in the Microbiological Risk Assessment series contain information that is useful to risk managers such as the Codex Alimentarius, governments and food regulatory agencies, scientists, food producers and industries and other people or institutions working in the area of microbiological hazards in foods, their impact on human health and food trade and their control.