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International Journal of Food Microbiology 93 (2004) 231–247
INTERNATIONAL JOURNAL OF Food Microbiology
www.elsevier.com/locate/ijfoodmicro

A quantitative risk assessment model for *Salmonella* and whole chickens☆

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Received 15 March 2003; received in revised form 25 October 2003; accepted 1 December 2003

Abstract

Existing data and predictive models were used to define the input settings of a previously developed but modified quantitative risk assessment model (QRAM) for *Salmonella* and whole chickens. The QRAM was constructed in an Excel spreadsheet and was simulated using @Risk. The retail-to-table pathway was modeled as a series of unit operations and associated pathogen events that included initial contamination at retail, growth during consumer transport, thermal inactivation during cooking, cross-contamination during serving, and dose response after consumption. Published data as well as predictive models for growth and thermal inactivation of *Salmonella* were used to establish input settings. Noncontaminated chickens were simulated so that the QRAM could predict changes in the incidence of *Salmonella* contamination. The incidence of *Salmonella* contamination changed from 30% at retail to 0.16% after cooking to 4% at consumption. *Salmonella* growth on chickens during consumer transport was the only pathogen event that did not impact the risk of salmonellosis. For the scenario simulated, the QRAM predicted 0.44 cases of salmonellosis per 100,000 consumers, which was consistent with recent epidemiological data that indicate a rate of 0.66–0.88 cases of salmonellosis per 100,000 consumers of chicken. Although the QRAM was in agreement with the epidemiological data, surrogate data and models were used, assumptions were made, and potentially important unit operations and pathogen events were not included because of data gaps and thus, further refinement of the QRAM is needed.
Published by Elsevier B.V.

Keywords: Chicken; Predictive microbiology; Quantitative microbial risk assessment; *Salmonella*; Monte Carlo simulation

1. Introduction

The rate of salmonellosis in the United States is between 15 and 20 cases per 100,000 people (Bryan and Doyle, 1995). Approximately 10% of salmonellosis cases are caused by poultry meat with 6.6% from turkey and 4.4% from chicken (Bryan and Doyle, 1995) for a rate of 0.66–0.88 cases of salmonellosis per 100,000 consumers of chicken.

Preventing outbreaks and sporadic cases of salmonellosis from chicken requires a holistic approach to chicken safety. One such holistic approach is risk assessment, which involves hazard identification, exposure assessment, dose–response assessment, and risk characterization. The typical approach taken is

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0168-1605/$ - see front matter. Published by Elsevier B.V.
doi:10.1016/j.ijfoodmicro.2003.12.002

to construct a quantitative risk assessment model (QRAM) in a computer spreadsheet using probability distributions to model the variability and uncertainty of important risk factors, such as time, temperature and pathogen density. The QRAM is then simulated using a spreadsheet add-in program that randomly samples the probability distributions and uses the random numbers generated to perform calculations and generate output distributions (Vose, 1998). Examples of QRAM for food pathogens using this approach and which are published in the scientific literature are listed in Table 1.

Two of the QRAMs in Table 1 are for *Salmonella* and chicken. Whiting (1997) developed a QRAM for *Salmonella* and cooked poultry patties that was designed to demonstrate how predictive models for pathogen growth and survival and Monte Carlo simulation could be used to assess the risk of salmonellosis. The QRAM considered the effects of initial contamination, growth during storage, death during cooking, amount of chicken consumed, and dose response on the probability of salmonellosis (Whiting, 1997). Predictive models for growth of *Salmonella* in laboratory medium (Gibson et al., 1988) and death of *Salmonella* in eggs (Annellis et al., 1954) were used as surrogate models for the growth and thermal inactivation steps in the QRAM (Whiting, 1997).

Table 1
Quantitative risk assessment models for food pathogens that use Monte Carlo simulation and are published in the scientific literature

Pathogen	Food commodity	Reference
Salmonella enteritidis	Pasteurized liquid eggs	Whiting and Buchanan, 1997
Escherichia coli O157:H7	Ground beef hamburgers	Cassin et al., 1998
Generic	Generic	McNab, 1998
Salmonella spp.	Cooked poultry patty	Whiting, 1997
Salmonella spp.	Whole chicken	Oscar, 1998
Bacillus cereus	Chinese-style rice	McElroy et al., 1999
Listeria monocytogenes	Smoked salmon and trout	Lindqvist and Westoo, 2000
Salmonella enteritidis	Shell eggs	Whiting et al., 2000
Escherichia coli O157:H7	Raw fermented sausages	Hoornstra and Notermans, 2001
Salmonella spp.	Turkey cordon bleu	Bemrah et al., 2002
Staphylococcus aureus	Unripened cheese	Lindqvist et al., 2002
Escherichia coli O157:H7	Apples	Duffy and Schaffner, 2002

Oscar (1998) constructed a QRAM for *Salmonella* and whole chickens that used Monte Carlo simulation methods to model the effects of initial contamination after processing, growth during distribution, thermal inactivation during cooking, cross-contamination during serving and host resistance on the probability of salmonellosis. The objective of Oscar (1998) was to provide a QRAM for assessing the risk of salmonellosis from an individual batch of whole chickens, where input settings would be based on locally collected data. However, in the QRAM of Oscar (1998), the input settings were empirically derived, and thus, how existing data and predictive models can be used to define the input settings was not demonstrated. Consequently, in the current study, the QRAM of Oscar (1998) was modified because of data gaps and then used to demonstrate how existing data and predictive models can be used to define the input settings and to assess the risk of salmonellosis from whole chickens.

2. Materials and methods

2.1. Model design

A QRAM for *Salmonella* and ready-to-cook whole chickens (Fig. 1), hereafter referred to as whole chickens, was constructed in an Excel (Microsoft, Redmond, WA) spreadsheet and was simulated using @Risk (version 4.0, Palisade, Newfield, NY), a spreadsheet add-in program. The retail-to-table pathway was modeled as a series of unit operations and associated pathogen events that included initial contamination at retail (node 1), growth during consumer transport (node 2), thermal inactivation during cooking (node 3), cross-contamination during serving (node 4), and dose response after consumption (node 5). The cell addresses, formulas and input settings used in the QRAM are shown in Table 2.

Pathogen events were modeled using PERT distributions (nodes 3 and 5) or by linking discrete and PERT distributions to model rare pathogen events

	A	B	C	D	E	F	G
1	Node	Unit Operation	Pathogen Event	Output	Units	Input (incidence)	Input (extent)
2	1	Retail	Initial Contamination	0	MPN/chicken	0	1.12
3	2	Transport	Growth	0	MPN/chicken	0	0.05
4	3	Cooking	Thermal Inactivation	0	MPN/chicken		-21.54
5	4	Serving	Cross-contamination	0	MPN/chicken	0	0.08
6	5	Consumption	Dose-Response	0			3.33

Fig. 1. A quantitative risk assessment model (QRAM) for *Salmonella* and whole chickens was constructed in an Excel spreadsheet and was simulated using @Risk. The formulas used in the QRAM are shown in Table 2 and results for an iteration of the QRAM are shown here and in Fig. 3.

(nodes 1, 2 and 4; Oscar, 1998). PERT distributions, defined by minimum, median or mean, and maximum values, were used to model extent of pathogen events.

Table 2
Cell addresses and formulas used in the quantitative risk assessment model for *Salmonella* and whole chickens (Fig. 1)

Unit operation	Distribution	Cell	Formula
Retail	Output	D2	=IF(F2=0,0, ROUNDDOWN (POWER(10,G2),0))
Transport	Output	D3	=IF(F3=0,D2, ROUNDDOWN (POWER(10,G3)*D2,0))
Cooking	Output	D4	=ROUNDDOWN (POWER (10,G4)*D3,0))
Serving	Output	D5	=IF(F5=0,D4, ROUNDDOWN ((G5*D3)+D4,0))
Consumption	Output	D6	=D5/(ROUNDDOWN (POWER(10,G6),0))
Retail	Input (incidence)	F2	=RiskDiscrete({0,1}, {70,30})
Transport	Input (incidence)	F3	=RiskDiscrete({0,1}, {99.98,0.02})
Serving	Input (incidence)	F5	=RiskDiscrete({0,1}, {72,28})
Retail	Input (extent)	G2	=RiskPert(0, 1, 2.7)
Transport	Input (extent)	G3	=RiskPert(0.0005, 0.04, 0.15)
Cooking	Input (extent)	G4	=RiskPert(−96, −8.1, −0.83)
Serving	Input (extent)	G5	=RiskPert(0.021, 0.057, 0.24)
Consumption	Input (extent)	G6	=RiskPert(1, 3, 7)

The PERT distribution was selected as the continuous distribution for extent of pathogen events because it is easy to define and because its shape is flexible as it can vary in shape from a normal distribution to a lognormal distribution that is skewed to the right or to the left.

During simulation of the QRAM, @Risk randomly sampled the discrete and PERT distributions for nodes 1, 2 and 4. However, only when the pathogen event occurred or when the discrete distribution returned a 1 was the random number from the PERT distributions for nodes 1, 2 and 4 used to calculate the model outputs. A consequence of this model design was that the sensitivity analysis provided by @Risk was compromised because not all of the randomly selected values from the PERT distributions for nodes 1, 2 and 4 were used to calculate the model outputs although these values were used to calculate correlations among the inputs and outputs in the sensitivity analysis.

Predictive models for growth (Oscar, 2002) and thermal inactivation (Murphy et al., 2002) of *Salmonella* were used outside the QRAM to convert time and temperature data into input settings. In addition, noncontaminated chickens were included in the simulations to allow the QRAM to predict changes in the incidence of *Salmonella* contamination.

2.2. Input settings

2.2.1. Node 1—retail

The first node in the QRAM simulated the initial contamination of whole chickens with *Salmonella* at retail. Recent and extensive reviews (Bryan and Doyle, 1995; Waldroup, 1996) of the scientific literature indicate that the incidence of *Salmonella* con-

tamination of whole chickens is variable among studies because of differences in chicken production and processing practices (Waldroup et al., 1992), as well as differences in sampling and detection methods (Jetton et al., 1992; Waldroup et al., 1992; Jorgensen et al., 2002). In fact, in their extensive review, Bryan and Doyle (1995) reported that the incidence of *Salmonella* contamination of whole chickens ranged from 0% to 100% with a median value of 30%. In the current study, the median value of 30% reported by Bryan and Doyle (1995) was used to define the incidence of *Salmonella* contamination of whole chickens at retail.

Six studies were found in the scientific literature that enumerated *Salmonella* on whole chickens and all of these studies used the most probable number (MPN) method (Table 3). The MPN of *Salmonella* on contaminated whole chickens is low, often below 10 MPN, whereas the maximum MPN per chicken ranges from >300 to >1100. A minimum value of 1 MPN (by definition, the minimum level of contamination that is possible), a median value of 10 MPN (a value close to the central tendency of the most likely data in Table 3), and a maximum value of 450 MPN (the median value for the maximum data in Table 3) per chicken were used to define the input settings for the PERT distribution for the extent of *Salmonella* contamination of ready-to-cook whole chickens at retail (Table 2).

Table 3
Summary of studies that have determined the most probable number (MPN) of *Salmonella* on whole chickens

Sample source	Extent			Units	Reference
	Minimum	Most likely	Maximum		
Processing plant		7		MPN/chicken	Whittemore, 1993
Processing plant		<6		MPN/chicken	Waldroup et al., 1992
Processing plant	1	<30	>300	MPN/chicken	Surkiewicz et al., 1969
Retail outlet		<10	>1100	MPN/chicken	Dufrenne et al., 2001
Retail outlet		<35		MPN/chicken	Izat et al., 1991
Retail outlet	10	69	450	MPN/chicken	Hawa et al., 1984

2.2.2. Node 2—consumer transport

The second node simulated growth of *Salmonella* on whole chickens during consumer transport from the retail store to the home refrigerator. It was assumed that the chickens were individually packaged and thus, cross-contamination did not occur during consumer transport or during storage in the home refrigerator. To establish input settings, surrogate time and temperature data for consumer transport of fresh meat from retail outlets to home refrigerators (Anonymous, 2002) were used in a recently published growth model for *Salmonella typhimurium* and sterile-cooked (autoclaved) chicken (Oscar, 2002). The growth model uses PERT distributions for time and temperature, and Monte Carlo simulation to predict the incidence and the minimum, median and maximum log cycle increase of potential growth events for *Salmonella* on sterile-cooked (autoclaved) chicken (Oscar, 2002). For growth to occur during consumer transport, the chicken must be contaminated with *Salmonella* and experience time and temperature conditions that allow growth to occur.

Time and temperature data from 943 consumer transport events of fresh meat in the consumer survey (Anonymous, 2002) were analyzed and it was found that the time of consumer transport ranged from 0.2 to 6.3 h with a median time of 1 h. The temperature of fresh meat when it arrived in the consumer's home ranged from −3.9 to 21.1 °C with a median temperature of 7.8 °C. These values were used to define the input settings for the PERT distributions for time and temperature in the growth model (Oscar, 2002), which was then simulated. A predicted incidence of potential growth events during consumer transport of 0.02% was obtained and was used to define the incidence of growth events for this node (Table 2). The growth model also predicted that the extent of potential growth events would range from 0.0005 to 0.15 log cycles with a median value of 0.04 log cycles; these values were used to define the input settings for the PERT distribution for the extent of potential *Salmonella* growth during consumer transport (Table 2).

Although surrogate time and temperature data from a consumer survey (Anonymous, 2002) and a surrogate growth model for sterile-cooked (autoclaved) chicken (Oscar, 2002) were used to establish input settings for this node, no assumptions were made as to the mechanism whereby a particular log cycle increase

of *Salmonella* occurred on whole chickens during consumer transport. Rather, it was recognized that many combinations of time, temperature (e.g., fluctuating temperature), chicken (e.g., fat %), *Salmonella* (e.g., physiological state) and human (e.g., food handling practices) risk factors could interact and account for a particular log cycle increase.

2.2.3. Node 3—cooking

The third node simulated the thermal inactivation of *Salmonella* during cooking of whole chickens in a home oven. Thermal inactivation of *Salmonella* depends on a number of risk factors, such as time, temperature (Mazzotta, 2000; Murphy et al., 2002), product shape and size (Murphy et al., 1999), strain of *Salmonella* (Bayne et al., 1965; Murphy et al., 1999), method of cooking (Brown et al., 1998) and physiological state of *Salmonella* (Doyle and Mazzotta, 2000). Temperature data for cooked poultry (Anonymous, 2002) and a thermal inactivation model for *Salmonella* and chicken patties (Murphy et al., 2002) were used to establish input settings for this node.

A cooking model was created in an Excel spreadsheet and was simulated using @Risk (Fig. 2). A PERT distribution was used to model the variability and uncertainty of final cooked temperature, which was reported to range from 26 to 93 °C with a mean of 62 °C (Anonymous, 2002). However, PERT settings of 55, 62 and 70 °C (Fig. 2) were used because the thermal inactivation model (Murphy et al., 2002) only had a temperature range of 55–70 °C. Time of cooking and method of cooking were not reported in the consumer survey (Anonymous, 2002). Consequently, it was assumed that chickens were cooked in a home oven and that *Salmonella* were exposed to the final cooked temperature for a minimum of 15 min, a median of 30 min, and a maximum of 45 min before the cooking model was simulated.

Results of the cooking model simulation indicated that the log cycle reduction of *Salmonella* ranged from −96 to −0.83 with a median value of −8.1; these values were used to define the PERT distribution for cooking (Table 2). Again, although surrogate data and models were used to establish input settings, no assumptions were made as to the mechanism whereby a particular log cycle reduction of *Salmonella* occurred during cooking. Rather, it was recognized that many combinations of time, temperature, cooking environment (e.g., relative humidity), chicken (e.g., fat %), *Salmonella* (e.g., strain) and human (e.g., doneness preference) risk factors could account for a particular log cycle reduction.

Throughout the model, *Salmonella* were treated as discrete entities by rounding down fractions of *Salmonella* to 0 (Table 2). Thus, if the number of *Salmonella* that survived cooking was a fraction less than 1, then it was assumed that all of the *Salmonella* had been killed and that the chicken was *Salmonella*-free.

2.2.4. Node 4—serving

The fourth node simulated cross-contamination of cooked chickens with *Salmonella* during serving. Here, it was assumed that cooked chickens were served and consumed before *Salmonella* that survived cooking could grow. In this node, incidence refers to the percentage of chickens that were mishandled by consumers. For example, cutting of cooked chicken with utensils used to prepare raw chicken.

	A	B	C
1		Outputs	Formula
2	Final Temperature, °C	62.2	=RiskPert(55,62,70)
3	D-value, min	3.57	=POWER(10,8.7344-(0.1316*B2))
4	Cooking Time, min	30.0	=RiskPert(15,30,45)
5	Log Cycle Reduction	8.39	=RiskOutput() + B4/B3

Fig. 2. A simulation model for thermal inactivation of *Salmonella* on whole chickens during cooking in a home oven (node 3 in Fig. 1) was constructed in an Excel spreadsheet and was simulated using @Risk. Results for an iteration of the model are shown. Input settings for the PERT distribution for extent of *Salmonella* death on whole chicken during cooking (Table 2) were derived from the output distribution of this model, which is in cell B5.

A summary of consumer surveys of cooked food handling practices that could lead to cross-contamination and a summary of laboratory studies that quantified transfer rates of *Salmonella* or surrogate bacteria from raw chicken to hands or surfaces were summarized (Table 4) and used to establish input settings for this node (Table 2). The incidence of food handling mistakes that could lead to cross-contamination averaged 28% among three consumer surveys (Table 4) and was the value used to define the incidence of cross-contamination events for this node (Table 2). Likewise, average values among studies of 0.021, 0.057 and 0.24 for the minimum, median and maximum transfer rates, respectively, of *Salmonella* were used to define the PERT distribution (Table 2) for cross-contamination during serving.

In this node, only self-contamination events were modeled. In other words, for a chicken that was mishandled during serving to experience an increase in *Salmonella* load, the chicken had to be contaminated with *Salmonella* before cooking. This model design was used to provide a link between the level of *Salmonella* contamination entering the consumer's home and consumer exposure. Again, no assumptions were made as to the mechanism whereby a specific transfer of *Salmonella* occurred. Rather, it was recognized that many combinations of chicken handling (e.g., contact time with a contaminated surface), *Salmonella* (e.g., strain variation) and human (e.g., type of food handling error) risk factors could account for a particular transfer rate of *Salmonella* from raw chicken to cooked chicken.

2.2.5. Node 5—consumption

The fifth node simulated response of consumers to consumption of *Salmonella*. A PERT distribution was used to simulate illness dose of the consumption event. During simulation, @Risk randomly assigned an illness dose to each chicken simulated. No assumptions were made as to the mechanism for a particular illness dose. Rather, it was recognized that many combinations of food matrix (e.g., fat %), *Salmonella* (e.g., strain virulence) and host (e.g., immunity) risk factors could account for a particular illness dose. However, to simplify interpretation of model outputs, it was assumed that four people consumed each chicken and that one person consumed all the *Salmonella* on a contaminated chicken.

Dose–response data for nontyphoid *Salmonella* in humans are limited to a large feeding trial with healthy men (McCullough and Eisele, 1951a,b,c). Data from the study have been extensively modeled (Rose et al., 1995; Coleman and Marks, 1998; Teunis et al., 1999; Latimer et al., 2001) and indicate differences in virulence among strains of *Salmonella*. The lowest dose causing illness in healthy men ranges from 10^5 to 10^{10} for the 13 strains tested (Blaser and Newman, 1982). However, estimated doses of *Salmonella* ingested in outbreaks that may have involved less resistant consumers, more virulent strains of *Salmonella* and/or more permissive meals ranges from

Table 4
Summary of consumer food-handling surveys and cross-contamination studies for *Salmonella* and surrogate bacteria from the scientific literature

Category	Transfer from	Transfer to	Incidence (%)	Minimum (%)	Median (%)	Maximum (%)	Reference
Mishandling			17				Worsfold and Griffith, 1997
Mishandling			33.5				Altekruse et al., 1995
Mishandling			33.5				Jay et al., 1999
Salmonella	Chicken skin	Stainless steel		3.0	5.0	10.0	Carson et al., 1987
Surrogate	Chicken	Hand		1.8	8.7	41.7	Chen et al., 2001
Surrogate	Chicken	Cutting board		3.0		32.4	Chen et al., 2001
Surrogate	Chicken	Hand		0.6	3.5	10.4	Montville et al., 2001

10^1 to 10^{11} with a dose of $<10^3$, usually causing illness (Blaser and Newman, 1982; Vought and Tatini, 1998). Based on these data, the input settings for the PERT distribution for illness dose (Table 2) were a minimum of 1 log MPN, a median of 3 log MPN and a maximum of 7 log MPN. Dose response was modeled as a discrete event. For an illness to occur from consumption of a cooked chicken, the dose of *Salmonella* consumed had to exceed the illness dose that was randomly assigned to that chicken or iteration by @Risk. Thus, the outcome of the dose response was discrete: no illness or illness.

2.3. Model simulation

The scenario of the QRAM defined in Table 2 was simulated with @Risk settings of Latin Hypercube sampling (similar results were obtained when Monte Carlo sampling was used), 10,000 iterations and a random number generator seed of 1. The random number generator seed is a number that initiates the selection of random numbers by @Risk. Each random number generator seed generates a unique outcome of the model. By simulating the QRAM with different random generator seeds, one can assess the uncertainty of model outputs, which is important for QRAM, like the present one that contain rare pathogen events.

3. Results

3.1. Individual iteration

3.1.1. Exposure assessment

Fig. 3 shows the results for iteration or chicken #6632 in the simulation of the QRAM. Results of this iteration are presented in detail to demonstrate how the simulation results were generated and to show how the random and rare nature of pathogen events in the QRAM affected consumer exposure and response to *Salmonella* on whole chickens.

For nodes 1, 2 and 4, when the discrete distribution for these rare pathogen events returned a 0, the pathogen event was considered to not have occurred and consequently, the QRAM ignored the random number from the PERT distribution in its calculation of the output results. For example, in iteration #6632,

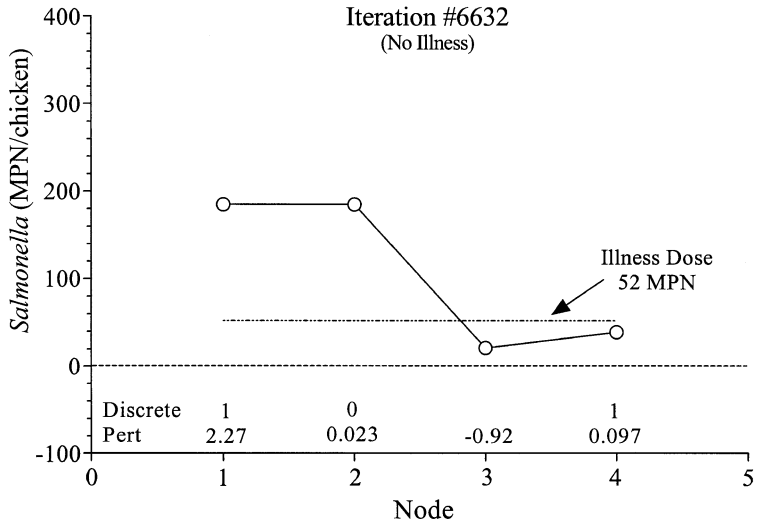

Fig. 3. Results of iteration #6632 from the simulation of the quantitative risk assessment model (QRAM) for *Salmonella* and whole chickens (Fig. 1). For rare pathogen events (nodes 1, 2 and 4), the QRAM ignored the output of the PERT distribution when calculating output results when the discrete distribution returned a zero indicating that the pathogen event did not occur. For this iteration, the randomly assigned illness dose of 52 MPN was greater than the dose of 39 MPN on the chicken at consumption and thus, the QRAM predicted that no illness would occur from consumption of chicken #6632.

the output of the discrete distribution for node 1 was 1, and the output of the PERT distribution was 2.27 log MPN or 185 MPN (Fig. 3). Because the random number from the discrete distribution for initial contamination was 1, the QRAM did not ignore the number from the PERT distribution and thus, chicken #6632 was assigned an initial *Salmonella* load of 185 MPN.

	A	B	C	D	E	F
2	Name	Node 1	Node 2	Node 3	Node 4	Node 5
3	Description	Output	Output	Output	Output	Output
4	Cell	D2	D3	D4	D5	D6
5	Minimum	1	1	1	1	-1.#QNAN
6	Maximum	363	363	21	39	-1.#QNAN
7	Mean	26.5	26.5	4.69	3.62	-1.#QNAN
8	Std Deviation	39.1	39.1	5.67	4.10	-1.#QNAN
9	Variance	1,527	1,527	32.1	16.8	-1.#QNAN
10	Skewness	3.28	3.28	1.63	3.05	-1.#QNAN
11	Kurtosis	17.0	17.0	4.7	18.5	-1.#QNAN
12	Errors Calculated	0	0	0	0	0
13	Mode	3	3	1	1	1
14	5% Perc	2	2	1	1	-1.#QNAN
15	10% Perc	2	2	1	1	-1.#QNAN
16	15% Perc	3	3	1	1	-1.#QNAN
17	20% Perc	4	4	1	1	-1.#QNAN
18	25% Perc	5	5	1	1	-1.#QNAN
19	30% Perc	6	6	1	1	-1.#QNAN
20	35% Perc	7	7	2	1	-1.#QNAN
21	40% Perc	9	9	2	2	-1.#QNAN
22	45% Perc	10	10	2	2	-1.#QNAN
23	50% Perc	12	12	2	2	-1.#QNAN
24	55% Perc	14	14	2	2	-1.#QNAN
25	60% Perc	17	17	2	2	-1.#QNAN
26	65% Perc	20	20	3	3	-1.#QNAN
27	70% Perc	25	25	5	4	-1.#QNAN
28	75% Perc	31	31	5	5	-1.#QNAN
29	80% Perc	38	38	8	6	-1.#QNAN
30	85% Perc	49	49	10	7	-1.#QNAN
31	90% Perc	66	66	13	8	-1.#QNAN
32	95% Perc	104	104	21	12	-1.#QNAN
33	Filter Minimum	1	1	1	1	1
34	Filter Maximum					
35	Type (1 or 2)	1	1	1	1	1
36	# Values Filtered	7,000	7,000	9,984	9,601	10,000
37	Unfiltered	3,000	3,000	16	399	0
38	Incidence	30%	30%	0.16%	3.99%	0%
39	Total	79,509	79,509	75	1,446	

Fig. 4. Filtered output results from 10,000 iterations of the quantitative risk assessment model for *Salmonella* and whole chickens (Fig. 1). Filtering, which removed the zero iterations, made it possible to calculate the incidence of *Salmonella* contamination (cells B38, C38, D38 and E38), the total MPN of *Salmonella* after each unit operation and pathogen event (cells B39, C39, D39 and E39) and the cases of salmonellosis per 10,000 chickens (cell F37).

In contrast, in node 2, the output for the discrete distribution was 0, which indicated that the pathogen event, growth during consumer transport, did not occur, and thus, the QRAM ignored the output of the PERT distribution, which called for a 0.023 log cycle increase in MPN of *Salmonella*, in its calculation of the model output (Fig. 3). Thus, the *Salmonella* load of chicken #6632 after consumer transport was the same as the initial contamination or 185 MPN.

In node 3, the QRAM randomly assigned a log cycle reduction value of −0.92 (Fig. 3) from the PERT distribution for cooking (Table 2). This small log cycle reduction value indicated that chicken #6632 was not thoroughly cooked and thus, *Salmonella* may have survived the cooking process. For iteration #6632, the QRAM predicted that 21 of the 185 *Salmonella* on the chicken, before cooking, had survived the thermal inactivation step.

After cooking, the consumer mishandled chicken #6632, as indicated by a discrete distribution value of 1 (Fig. 3). Because the uncooked chicken was contaminated with 185 MPN of *Salmonella*, this food-handling mistake had the potential of increasing the *Salmonella* load of the cooked chicken as the result of a cross-contamination event. The transfer rate for this potential cross-contamination event was 0.097 (Fig. 3). Because the *Salmonella* load of the uncooked chicken was greater than 11 MPN, which was the minimum *Salmonella* load required for 1 MPN of *Salmonella* to be transferred from the uncooked chicken to the cooked chicken for a transfer rate of 0.097 (i.e., 11 MPN × 0.097 = 1.07 or 1 MPN after rounding off), the *Salmonella* load of the cooked chicken #6632 increased by 18 MPN (i.e., 0.097 × 185 MPN before cooking) during serving. Thus, the total *Salmonella* load of the cooked chicken at consumption was 39 MPN (i.e., 21 MPN after cooking + 18 MPN during serving).

3.1.2. Dose–response assessment

The final node in the QRAM simulated the response of consumers to *Salmonella* ingestion. To simplify the interpretation of the model outputs, it was assumed that four people ate each chicken but that one person ingested all of the *Salmonella* on the chicken. Dose response for the consumption event was modeled as a discrete event where illness did not

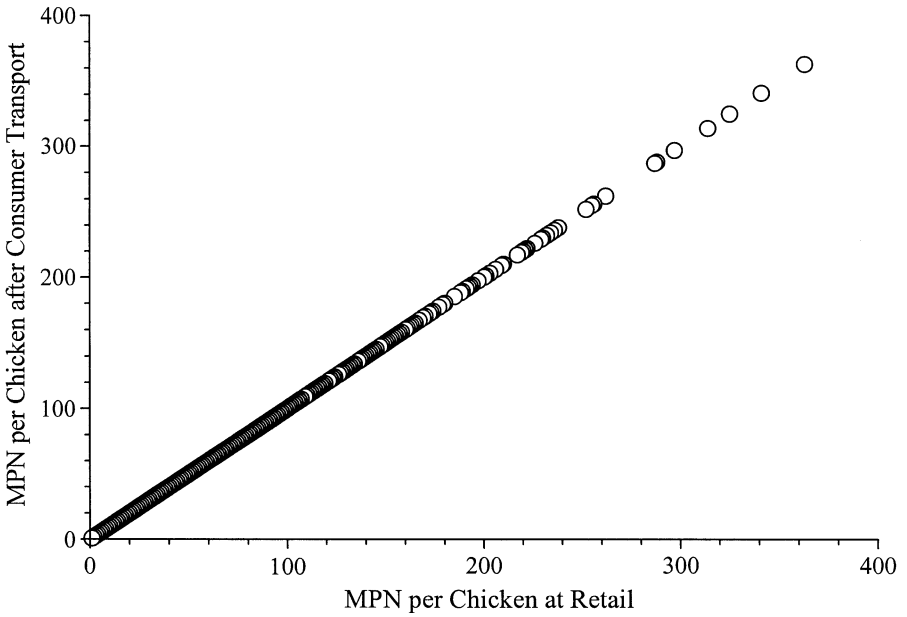

Fig. 5. Scatter plot of the most probable number (MPN) of *Salmonella* per contaminated chicken after consumer transport versus the MPN of *Salmonella* per contaminated chicken at retail. All of the points fall on the line of unity, indicating that *Salmonella* growth did not occur on any chickens during consumer transport.

occur if the dose consumed was less than the illness dose, whereas illness occurred if the dose consumed was equal to or greater than the illness dose. By random chance, an illness dose of 52 MPN was assigned to chicken #6632 (Fig. 3). There are many possible explanations for this low illness dose, such as a highly permissive meal (e.g., postmeal antacid pill), a highly virulent strain of *Salmonella* (e.g., acid-tolerant) and/or a highly susceptible consumer (e.g., elderly person). Regardless of the mechanism, the illness dose was greater than the dose of *Salmonella* consumed and thus, the QRAM predicted that no illness would occur from consumption of chicken #6632.

3.2. All iterations

3.2.1. Exposure assessment

To further illustrate how the rare and random nature of pathogen events affected the outputs of the QRAM, the output results from all 10,000 iterations were examined after filtering to remove the 0 values (Fig. 4). As expected, 7000 of the 10,000 output values for node 1, initial contamination, were removed by filtering because only 30% or 3000 of the 10,000 chickens or iterations were initially contaminated with *Salmonella* (Fig. 4). Results of the simulation indicated that the MPN of *Salmonella* per contaminated chicken ranged from 1 (cell B5 in Fig. 4) to 363 (cell B6 in Fig. 4) with a mean of 26.5 (cell B7 in Fig. 4) and a median of 12 (cell B23 in Fig. 4). The total MPN of *Salmonella* was 79,509 (cell B39 in Fig. 4; mean × 3000) per 10,000 chickens.

When the MPN of *Salmonella* on the 3000 contaminated chickens at retail was graphed against the MPN of *Salmonella* on the same 3000 chickens after consumer transport, a straight line with no points above the line of unity was obtained (Fig. 5). This result indicated that no growth of *Salmonella* had occurred on any of the chickens during consumer transport. In fact, the total MPN of *Salmonella* after consumer transport was 79,509 per 10,000 chickens (cell C39 in Fig. 4), which was the same as the *Salmonella* load at retail (cell B39 in Fig. 4).

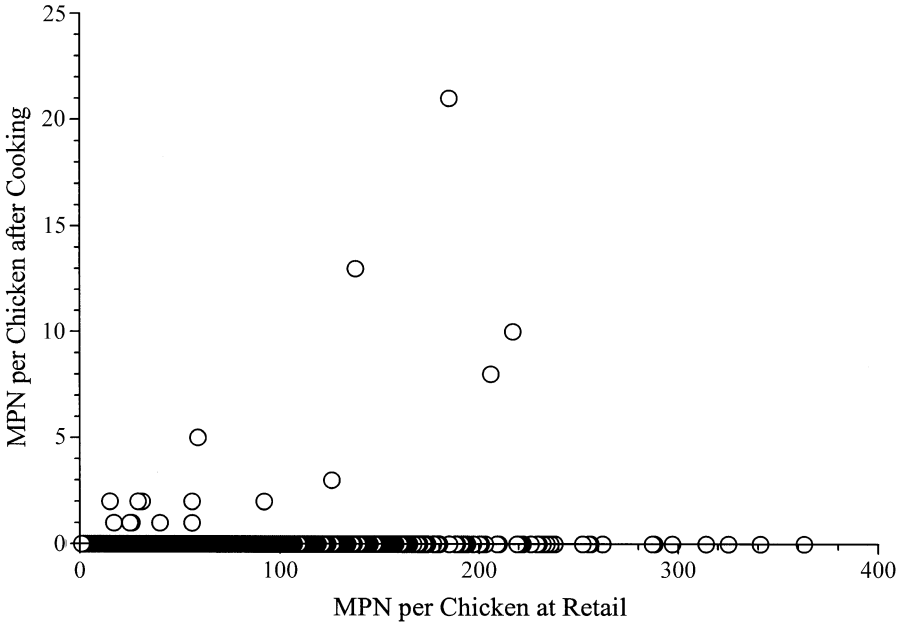

Fig. 6. Scatter plot of the most probable number (MPN) of *Salmonella* per contaminated chicken after cooking versus the MPN of *Salmonella* per contaminated chicken at retail. Points on the *x*-axis represent contaminated chickens that were properly cooked and contained no *Salmonella* after cooking. For the remaining chickens, the results demonstrated that survival of *Salmonella* during cooking was a random event and that after cooking, chickens with low initial MPN (< 100) of *Salmonella* may have higher MPN of *Salmonella* than some of the chickens with high initial MPN (>100) of *Salmonella*.

The incidence of potential growth events during consumer transport was only 0.02% or 2 chances per 10,000 iterations. Considering that 7000 of the chickens initially contained no *Salmonella*, the probability of a potential growth event occurring on a contaminated chicken was only 0.006% or 6 chances in 100,000. Thus, it was not surprising that the *Salmonella* load of the 10,000 chickens did not change in node 2.

In contrast, cooking had a dramatic effect on the incidence and extent of *Salmonella* contamination of whole chickens. Cooking reduced the number of contaminated chickens from 3000 (cell B37 in Fig. 4; 30%) to 16 (cell D37 in Fig. 4; 0.16%) and the MPN of *Salmonella* per contaminated chicken ranged from 1 (cell D5 in Fig. 4) to 21 (cell D6 in Fig. 4) with a mean of 4.7 (cell D7 in Fig. 4) and a median of 2 (cell D23 in Fig. 4) after cooking. The total MPN of *Salmonella* was 75 (cell D39 in Fig. 4) per 10,000 cooked chickens.

It was by random chance which chickens were cooked in a manner that eliminated *Salmonella*. In fact, after cooking, some of the chickens that had less than 100 MPN of *Salmonella* at retail had higher MPN of *Salmonella* than those that had greater than 100 MPN of *Salmonella* at retail (Fig. 6).

The final node in the exposure assessment simulated cross-contamination of cooked chicken with *Salmonella* from uncooked chicken during serving. The QRAM was designed such that this was a self-contamination event and thus, in order for a chicken to gain *Salmonella* during serving, the chicken had to be contaminated with *Salmonella* before cooking. This was done to establish a link between the MPN of *Salmonella* entering the consumer's home and the MPN of *Salmonella* transferred to the cooked chicken. In order for *Salmonella* to be transferred from uncooked to cooked chickens, the MPN before cooking had to be greater than 5 because the maximum rate of transfer was 0.24 and fractions of

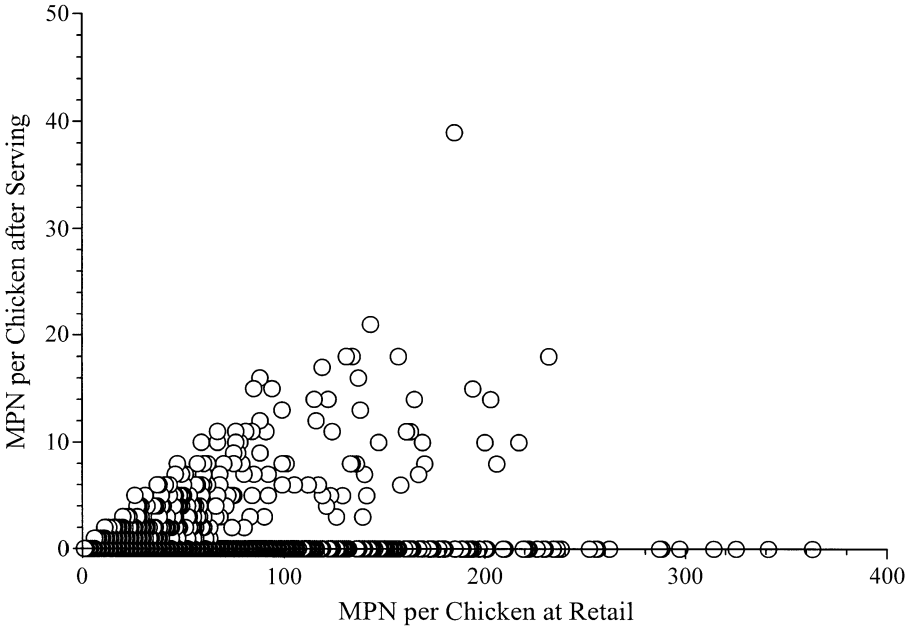

Fig. 7. Scatter plot of the most probable number (MPN) of *Salmonella* per contaminated chicken after serving versus the MPN of *Salmonella* per contaminated chicken at retail. A link between MPN of *Salmonella* after consumer transport (node 2), which had no effect on the MPN of *Salmonella* at product testing (Fig. 5), and MPN of *Salmonella* transferred during mishandling of chicken during serving was built into the model. Thus, as expected, a positive relationship was observed between the MPN of *Salmonella* after serving and the MPN of *Salmonella* at retail. Again, results of this step in the QRAM demonstrated the random nature of pathogen events that resulted in many chickens with low initial MPN (< 100) of *Salmonella* having higher MPN of *Salmonella* after serving or at consumption than some of the chickens with high initial MPN (>100) of *Salmonella*.

Salmonella that were less than 1 were rounded down to 0. Thus, if 4 MPN were present before cooking and the transfer rate was 0.24, the MPN transferred would be 0.96, which would be rounded down to 0 in the QRAM because *Salmonella* were treated as discrete entities.

Although noncontaminated chickens at retail could not become contaminated at any point in the retail-to-table pathway, contaminated chickens at retail that became *Salmonella*-free during cooking could become *Salmonella*-positive again after cooking, as a result of cross-contamination during serving. In fact, the number of contaminated chickens increased from 16 (cell D37 in Fig. 4; 0.16%) after cooking to 399 (cell E37 in Fig. 4; 3.99%) after serving. The MPN of *Salmonella* per contaminated chicken after serving ranged from 1 (cell E5 in Fig. 4) to 39 (cell E6 in Fig. 4) with a mean of 3.6 (cell E7 in Fig. 4) and a median of 2 (cell E23 in Fig. 4). The total MPN of *Salmonella* among the 10,000 chickens was 1446 (cell E39 in Fig. 4) after serving or at consumption.

When the MPN of *Salmonella* per chicken after serving was graphed against the MPN of *Salmonella* at retail, a positive relationship between initial and final contamination was observed (Fig. 7). This was not unexpected because most of the *Salmonella* on the cooked chickens at consumption came from cross-contamination during serving, which was linked to *Salmonella* contamination before cooking. Clearly, cross-contamination during serving was an important risk factor.

3.2.2. Dose–response assessment

During simulation of the model, @Risk randomly assigned an illness dose to each chicken simulated. Fig. 8 shows the illness dose assigned to each of the 3000 contaminated chickens. In node 5, the MPN of *Salmonella* on the chicken at consumption (Fig. 7) was divided by the randomly assigned illness dose (Fig. 8), and if the value was less than 1, the QRAM predicted that no illness would occur, whereas if the value was equal to or greater than 1, then the QRAM predicted that an illness would occur. In the current simulation, none of the 10,000 chickens had a *Salmonella* load at consumption that exceeded the randomly assigned illness dose and thus, the QRAM

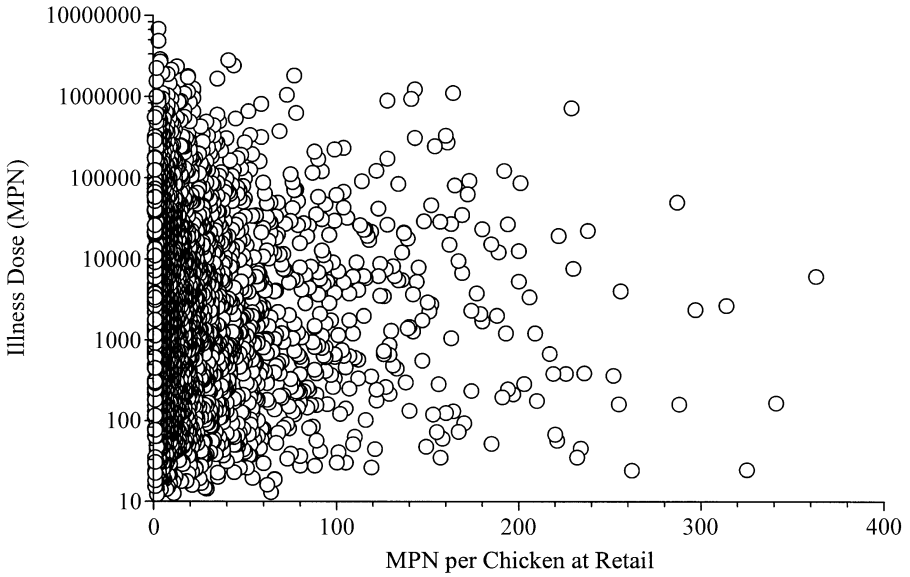

Fig. 8. Scatter plot of the illness dose for the consumption event versus the MPN of *Salmonella* on contaminated chickens at retail. Results of this part of the QRAM demonstrated the random nature of illness dose, which is the outcome of the interaction among the food matrix, *Salmonella* virulence and host resistance. Which chicken is consumed with a permissible meal, which chicken contains a highly virulent strain(s) of *Salmonella*, or which chicken is consumed by someone with low immunity are random events that are well modeled by Monte Carlo simulation techniques using the concept of illness dose, which is the dose of *Salmonella* ingested that causes illness in the host.

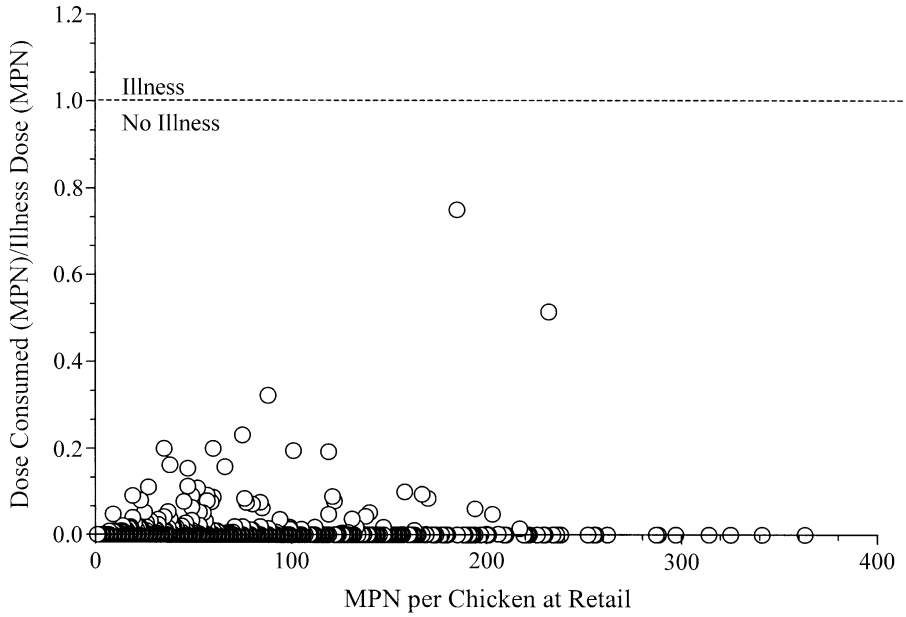

Fig. 9. Scatter plot of the ratio of dose consumed to illness dose versus the most probable number of *Salmonella* per contaminated chicken at retail. None of the chickens contained a dose of *Salmonella* that exceeded the randomly assigned illness dose (i.e., ratio < 1.0). Thus, the QRAM predicted that no illnesses would result from consumption of the 10,000 chickens.

predicted that no case of salmonellosis would occur (Fig. 9). However, 5% to < 10% of the chickens had a *Salmonella* load that was greater than 10 MPN (Fig. 7), which was the minimum illness dose (Fig. 8). Thus, it was possible for some of the chickens to cause salmonellosis; it just did not happen by random chance. In other words, the results of the QRAM indicated that the risk of salmonellosis was less than 1 per 10,000 chickens but not 0.

3.2.3. Risk characterization

To more precisely determine the risk of salmonellosis for the batch of whole chickens simulated, the QRAM was simulated with the same input and @Risk settings except that 1,000,000 iterations were performed and the QRAM was simulated with four different random number generator seeds (i.e., 1, 7, 29 and 83) to assess the uncertainty (i.e., standard error of the mean [S.E.M.]) of the QRAM prediction. Results of the simulations indicated that the risk of salmonellosis was 17, 21, 19 and 14 cases per 1,000,000 chickens for random number generator seeds of 1, 7, 29 and 83, respectively, for a mean (S.E.M.) of 17.8 (1.5) cases per 1,000,000 chickens. To calculate the rate of salmonellosis per 100,000 consumers, the results were divided by 4, because it was assumed that 4 people ate each chicken, and by 10 (i.e., 1,000,000/100,000). Thus, the predicted rate of salmonellosis for the scenario simulated was 0.44 (0.004) per 100,000 consumers of chicken. If other batches of chicken (i.e., other scenarios) had been evaluated using the QRAM, it would have been possible to determine whether the current batch of chickens was high or low risk relative to the other batches of chickens evaluated. However, including other scenarios (i.e., scenario analysis) was beyond the scope of this study but will be the focus of a future study.

4. Discussion

Quantitative risk assessment models have great potential as decision analysis tools for the chicken industry. However, predictions of QRAM are only as good as the data used to develop and define them. The current QRAM for *Salmonella* and whole chickens was not complete because it did not contain poten-

tially important unit operations and pathogen events in the retail-to-table pathway, such as nonthermal inactivation during cold storage and growth during cooling of cooked chicken. Moreover, it was difficult to model the pathogen events included in the QRAM because of data gaps and incomplete predictive models. In many cases, surrogate data (e.g., time and temperature data for transport of fresh meat) and surrogate models [e.g., growth model for sterile-cooked (autoclaved) chicken] had to be used and assumptions had to be made where data did not exist (e.g., time of exposure at the final cooked temperature) in order to model the pathogen events. Minimizing these types of data gaps and assumptions are important steps towards producing QRAM that provides better predictions. Nonetheless, the predicted rate of 0.44 cases of salmonellosis per 100,000 consumers of chicken is in agreement with recent epidemiological data indicating that the overall rate of salmonellosis is 15–20 cases per 100,000 people, of which, 4.4% or 0.66–0.88 cases per 100,000 are from chicken (Bryan and Doyle, 1995). Although the predicted rate of salmonellosis is in agreement with the recent epidemiological data, one should not automatically conclude that the QRAM provides reliable predictions because it is easy to adjust assumptions and input settings in the QRAM so that its overall prediction agrees with target values. Thus, it is important to validate not only the final output but also the inputs and outputs of each unit operation and pathogen event in the QRAM.

Although it is common for microbial risk assessors to create QRAM in computer spreadsheets and simulate them using a spreadsheet add-in program, such as @Risk, differences in design and simulation approach exist among QRAMs for food pathogens (Schlundt, 2000). In most QRAMs, incidence and extent of pathogen events are simulated separately throughout the QRAM and then combined at the end (Whiting and Buchanan, 1997; Lammerding and Fazil, 2000), whereas in other QRAMs, they are simulated together throughout the model (Oscar, 1998). In other words, in some QRAMs, only effects of pathogen events on contaminated servings are simulated (Whiting and Buchanan, 1997; Lammerding and Fazil, 2000), whereas in other QRAMs, effects of pathogen events on noncontaminated servings are also simulated (Oscar, 1998). Inclusion of noncontaminated servings in simulations of QRAM is necessary when cross-contamination events are important risk factors and when pathogen incidence is a desired output of the QRAM, such as in the present study. A disadvantage of including noncontaminated servings in the simulations of QRAM, especially those with rare pathogen events, is that extensive iteration is needed to generate enough nonzero outputs for meaningful decision analysis; also especially when a pathogen reduction step, such as cooking, is included that drives the incidence of nonzero iterations to very low numbers. In fact, 1,000,000 iterations of the present QRAM were needed to determine the risk of salmonellosis for whole chickens.

Another difference in the design of QRAM for food pathogens among studies is the use of predictive models for growth and death of pathogens inside (Whiting and Buchanan, 1997; Cassin et al., 1998; McNab, 1998) or outside (this study) of the QRAM. In the current study, predictive models for growth and thermal inactivation of *Salmonella* were used outside the QRAM in an effort to keep the QRAM simple. In addition, only discrete and PERT distributions were used to keep the QRAM design uniform and simple so that it could be easily defined and modified. In fact, it is very easy to expand or contract the current QRAM by adding and deleting unit operations and pathogen events. However, with each unit operation and pathogen event that is added, the cost of acquiring data increases. Thus, the proper balance must be established between how detailed the QRAM is and how much it will cost to collect data to properly define its input settings. A disadvantage of using predictive models outside the QRAM is that important risk factors, such as time, temperature, and food pH, are excluded from the sensitivity analysis of the QRAM provided by @Risk. In the current study, this was not an issue because the sensitivity analysis provided by @Risk was compromised, as discussed earlier, by using a combination of discrete and PERT distributions to model rare pathogen events.

Simulation results of the current study suggest that growth of *Salmonella* on whole chickens during consumer transport was the only unit operation and pathogen event in the QRAM that did not have a significant impact on the risk of salmonellosis. Thus, the QRAM could be simplified by eliminating this unit operation and pathogen event. On the other hand,

cross-contamination during serving was identified as an important risk factor.

Similar to results of a previous QRAM for *Salmonella* and whole chickens (Oscar, 1998), chickens with high initial levels of *Salmonella* did not result in the greatest consumer exposure to *Salmonella*. Rather, chickens with lower initial levels of *Salmonella* resulted in higher consumer exposure when they were undercooked and mishandled during serving.

A third difference in the design of QRAM for food pathogens is whether a probability (Whiting and Buchanan, 1997; Oscar, 1998; Cassin et al., 1998; McNab, 1998; McElroy et al., 1999) or discrete (current study) approach is used to simulate consumer exposure and response. Which approach is best depends on the objective of the QRAM. In the current QRAM, the discrete approach was used because cross-contamination during serving was an important risk factor and a QRAM was desired that could predict the incidence of *Salmonella* contamination after each unit operation and pathogen event. For instance, if the probability of consumer exposure to *Salmonella* on whole chickens approach had been used, the incidence of *Salmonella* contamination would have remain unchanged at 30% throughout the retail-to-table pathway because fractions of *Salmonella* of less than 1 after cooking would have been carried forward as probabilities or fractions rather than being rounded down to 0 as in the discrete approach. Thus, when the discrete approach was used, *Salmonella* incidence changed from 30% at retail and consumer transport to 0.16% after cooking to 3.99% after serving or at consumption. A potential advantage of the discrete approach may be in the validation of the QRAM as it is easier to determine *Salmonella* incidence than *Salmonella* MPN after each unit operation and pathogen event.

Routine application of QRAM to assess the impact of local differences in risk factors on the microbiological safety of whole chickens faces many challenges. Perhaps the biggest issue is that contamination of whole chickens with *Salmonella* is a rare event. Consequently, a large number of samples must be examined before enough positive chickens are encountered to produce reliable distributions of *Salmonella* contamination for QRAM. In the current QRAM, initial contamination of whole chickens was modeled as a rare event by linking a discrete distribution for *Salmonella* incidence with a PERT distribution for *Salmonella* MPN. With the advent of rapid methods for detection of *Salmonella* (Bennet et al., 1998; Bailey, 1998; Mandrell and Wachtel, 1999), it is becoming increasingly possible to routinely determine the incidence of *Salmonella* contamination of whole chickens. In contrast, determining the MPN of *Salmonella* on a routine basis is still very difficult. Nonetheless, it should be possible to establish a national baseline distribution for the MPN of *Salmonella* contamination of whole chickens and make this distribution available on the Internet to those wanting to use QRAM to assess chicken safety. Thus, local data on the incidence of *Salmonella* contamination could be collected and combined with the baseline distribution for MPN of *Salmonella* to simulate the initial contamination of whole chickens with *Salmonella*. Likewise, predictive models for growth, nonthermal inactivation, thermal inactivation, cross-contamination and dose response of *Salmonella* on whole chickens could be developed and made available to risk assessors in programs, such as the USDA, ARS Pathogen Modeling Program (www.arserrc.gov/mfs/) and the UK Food MicroModel (McClure et al., 1994), for use with locally collected time and temperature and consumer survey data to define the other input settings in the QRAM. In this way, it may be possible and practical to use QRAM on a routine basis to assess local differences in the microbiological safety of chicken contaminated with *Salmonella*.

5. Conclusions

Quantitative risk assessment modeling is a holistic approach that has a great potential as a decision analysis tool for the chicken industry. The advantage of QRAM over other approaches, such as in-plant HACCP, is that postprocessing risk factors, such as chicken-handling practices and consumer demographics, are considered in the evaluation of the microbiological safety of chicken. Ignoring important postprocessing risk factors may result in the mislabeling of whole chickens, resulting in the destruction of safe chickens and the shipment of unsafe chickens; neither situation would benefit public health.

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