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ORIGINAL ARTICLE

Process risk model for *Salmonella* and ground chicken

T.P. Oscar

United States Department of Agriculture, Agricultural Research Service, Poultry Food Safety Research Worksite, Center for Food Science and Technology, University of Maryland Eastern Shore, Princess Anne, MD, USA

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Correspondence
Thomas P. Oscar, United States Department of Agriculture, Agricultural Research Service, Poultry Food Safety Research Worksite, Room 2111, Center for Food Science and Technology, University of Maryland Eastern Shore, Princess Anne, MD 21853, USA.
E-mail: thomas.oscar@usda.gov

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Abstract

Aims: To develop a process risk model (PRM) for evaluating the safety of individual lots of ground chicken (GC) contaminated with *Salmonella* (*Salm*).
Methods and Results: Data for prevalence, number and serotype of *Salm* were collected with 25 g samples of GC using a combination of methods (whole sample enrichment, quantitative polymerase chain reaction, cultural isolation and serotyping). These data were used to develop a predictive model for *Salm* contamination of GC as a function of serving size from 25 to 300 g. This model was combined with a model for thermal inactivation of *Salm* in GC and a dose–response model for *Salm* to develop a PRM in Excel that was simulated with NeuralTools and @Risk. Of 100, 25 g samples of GC examined, 19 tested positive for *Salm*. Three serotypes were isolated: Infantis (n = 13), Enteritidis (n = 5) and Typhimurium (n = 1). The number of *Salm* ranged from 0 to 2·56 log with a median of 0·93 log per 25 g of GC. The PRM predicted that *Salm* prevalence would increase ($P < 0{\cdot}05$) from 19 to 57% to 82 to 93% as serving size increased from 25 to 100 g to 200 to 300 g. However, the total number of *Salm* in a 100-kg lot of GC and total severity of illness (TSI) were not affected ($P > 0{\cdot}05$) by serving size. The PRM was also used to evaluate effects of serving size distribution, cooking, food consumption behaviour, consumer demographics and *Salmonella* virulence on TSI.
Conclusions: How a lot of GC is partitioned and consumed does not affect TSI. Scenario analysis demonstrated that the PRM can integrate prevalence, number and serotype data for *Salm* with consumer handling, consumption and demographics data to identify safe and unsafe lots of GC for improved food safety and public health.
Significance and Impact of the Study: Process-risk models like the one developed in this study represent a new, holistic approach to food safety that holds great promise for improving public health and reducing food recalls.

Introduction

Salmonella are a leading cause of foodborne illness in the United States and throughout the world. The Centers for Disease Control and Prevention (CDC) estimates that each year *Salmonella* causes 1 027 561 cases of salmonellosis that result in 19 366 hospitalizations, and 378 deaths in the United States (Scallan *et al.* 2011). Worldwide, *Salmonella* causes an estimated 93 757 000 cases of salmonellosis and 155 000 deaths per year with 80 318 000 cases from food (Majowicz *et al.* 2010). The annual cost of foodborne salmonellosis in the United States due to increased healthcare costs and lost worker productivity is estimated at 4·43 billion dollars (Scharff 2012).

Symptoms of salmonellosis include headache, fever, abdominal pain, vomiting, diarrhoea and in severe cases dehydration, bacteraemia and death (Baird-Parker 1990). Although most cases of salmonellosis are mild and will resolve without medical treatment in otherwise healthy adults, the disease is more often fatal in the young, the old, the immunocompromised and those with pre-

existing health conditions like diabetes, cancer and liver disease. Thus, it is important to consider variations in consumer resistance when evaluating risk of salmonellosis from food.

Salmonella have been isolated from a variety of food products including eggs (Humphrey *et al.* 1989), chicken (Huang *et al.* 2016), turkey (Peng *et al.* 2016), beef (Zhao *et al.* 2001), pork (Sinell *et al.* 1990), shrimp (Heinitz *et al.* 2000), milk (Fleet *et al.* 1991), tomatoes (Gupta *et al.* 2007), lettuce (Horby *et al.* 2003), cucumbers (Angelo *et al.* 2015) and almonds (Danyluk *et al.* 2007) as well as many others. However, risk of salmonellosis depends not only on presence of *Salmonella* but also on the number and serotype (Jones *et al.* 2008). In addition, dose–response models of human feeding trial data (Oscar 2004a), outbreak investigation data (Bollaerts *et al.* 2008) and human feeding trial and outbreak investigation data (Oscar 2017b) indicate that ability of *Salmonella* to cause disease in humans differs among serotypes and strains. Thus, it is important to consider variation of *Salmonella* virulence when assessing risk of salmonellosis from food.

Process risk modelling is a holistic approach to food safety that holds great promise for improving public health. The cornerstone of a process risk model (PRM) is data that describe the distribution of the pathogen among servings of the food at some point in the production chain. Important considerations are that pathogen prevalence, number and serotype in food depend on sample size and pathogens are not evenly distributed among or within food servings (Oscar 2004b). Thus, to accurately predict consumer exposure and response to the pathogen, prevalence, number and serotype data are needed for different size samples of food and the lower limit of detection of the enumeration method must be one cell per serving.

Obtaining data for prevalence, number and serotype of *Salmonella* for different sample sizes with a lower limit of detection of one cell per serving can be accomplished in a time and cost-effective manner using a combination of methods. First, whole sample enrichment (WSE), quantitative polymerase chain reaction (qPCR), cultural isolation and serotyping can be used to obtain prevalence, number and serotype data for one sample size (Oscar 2014). Second, the data for one sample size can be used to develop a model that predicts prevalence, number and serotype as a function of serving size (Oscar 2004b). This approach was used in the current study to develop a predictive model for *Salmonella* contamination of ground chicken (GC) as a function of serving size before cooking (i.e. at meal preparation). This model was then combined with a predictive model for thermal inactivation of *Salmonella* in GC during cooking (Oscar 2017a) and a disease triangle, dose–response model for *Salmonella* after consumption (Oscar 2017b) to create a PRM for *Salmonella* and GC. The PRM was then used to evaluate effects of serving size, serving size distribution, cooking, food consumption behaviour, consumer demographics (i.e. immunity) and *Salmonella* virulence on consumer exposure to *Salmonella* and total severity of illness (TSI) from individual lots of GC.

Materials and methods

Materials

All isolates of *Salmonella* used to develop the standard curve for enumeration were obtained from the US Department of Agriculture, Agricultural Research Service culture collection at the Poultry Food Safety Research Worksite at the University of Maryland Eastern Shore (curator: T. P. Oscar). Real-time, polymerase chain reaction test kits for *Salmonella* (iQ-Check) were from Bio-Rad Laboratories (Hercules, CA). Buffered peptone water (BPW) was from Microbiology International (Frederick, MD). Rappaport Vassiliadis (RV) broth, xylose lysine (XL) agar and tergitol four (T4) were from Becton Dickinson (Sparks, MD).

One-pound packages of a single brand of GC with 1% vinegar and rosemary extract added and 8% fat were purchased from a local retail store in Salisbury, Maryland, USA. The pH of GC was obtained with a pH spear (Oakton Instruments, Vernon Hills, IL), and was 5·94 ± 0·22 (mean ± SD) with a range from 5·66 to 6·29.

Software applications used to graph, model and analyse data were Excel 2016 (MicroSoft Corporation, Redmond, WA), NeuralTools 7.6 and @Risk 7.6 (Palisade Corporation, Ithaca, NY) and Prism 8 (GraphPad Software, Inc., San Diego, CA).

Data collection

Data for prevalence, number and serotype of *Salmonella* in GC (25 g) were obtained using a published method (Oscar 2014) that involves WSE, qPCR, cultural isolation and serotyping. In brief, a standard curve for predicting log number of *Salmonella* in 25 g samples of GC as a function of cycle threshold (C_T) value from qPCR was developed using the Weibull model in Prism (Fig. 1):

$$C_T = C_{T_o} - \left(\frac{x}{a}\right)^b$$

where C_{T_o} at 0 log per 25 g was 39·6 (fixed value), x was the log number of *Salmonella* per 25 g, a was 0·01234 ± 0·00558 (best fit value ± standard error), b was 0·5 ± 0·04, the coefficient of determination (R^2) was

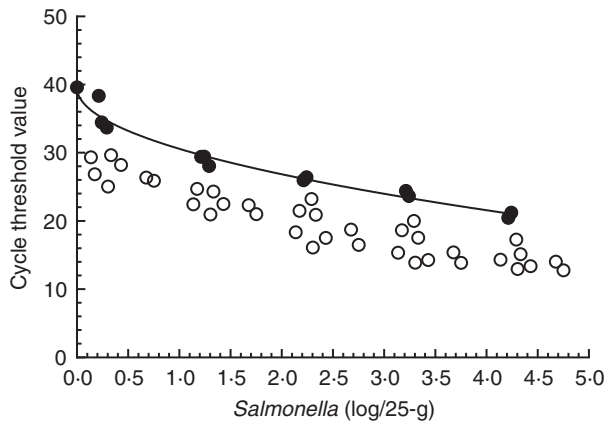

Figure 1 Standard curve for enumeration of *Salmonella* in ground chicken by whole sample enrichment, real-time, polymerase chain reaction. The line is the standard curve, whereas the data represented by the closed symbols (●) were used to develop the standard curve, whereas the data represented by the open symbols (○) were not used to develop the standard curve.

0·9704 and degrees of freedom was 11. The interpolation function of Prism was used to determine x for unknown samples.

Samples for qPCR were collected at 6 h of WSE in 400 ml of BPW incubated at 40°C and 80 rev min^{-1}. The standard curve had an enumeration range from 0 to 4·24 log or 1 to 17 348 per 25 g. The standard curve was developed with *Salmonella* serotypes Newport, Kentucky, and 8,20:-:z_6, which were isolated from chicken parts in previous studies (Oscar 2013, 2014, 2017b), and with serotype Infantis, which was isolated from GC in the present study. The highest C_T values at each dose of *Salmonella* were used to develop the standard curve and provided a conservative (fail-safe) estimate of log number as explained and justified in previous studies (Oscar 2014, 2017b).

Salmonella were isolated from WSE by nonselective enrichment in BPW (24 h at 40°C) followed by selective enrichment in RV broth (24 h at 42°C) followed by selective plating on XLT4 agar (24 h at 40°C). One colony per plate corresponding to one isolate per positive sample was collected and serotyped by a *Salmonella* Reference Center (US Department of Agriculture, Animal and Plant Health Inspection Service, National Veterinary Services Laboratory, Ames, IA).

Contamination model

Data for prevalence, number and serotype, and a published modelling method (Oscar 2004b) were used to develop a predictive model (Fig. 2) for *Salmonella* contamination of GC as a function of serving size from 25 to 300 g. In brief, the model was developed in Excel and was simulated with @Risk. A discrete distribution ({0,1,2,3},{81,13,5,1}), where 0 was none, 1 was Infantis, 2 was Enteritidis and 3 was Typhimurium, was used to model prevalence and a uniform distribution (0,2·562) was used to model the extent (i.e. log number) of *Salmonella* contamination per 25 g.

Serotype virulence was modelled using a published method (Oscar 2017b) for dose–response in which serotypes are classified as low-low (animal adapted; e.g. Pullorum), low (>top 20 human clinical isolate), normal (top 11–20 human clinical isolate), high (top 6–10 human clinical isolate) or high-high (top 1–5 human clinical isolate) risk based on their ranking as a human clinical isolate by the CDC. The contamination model assigns a numerical value for serotype virulence from 1 (low-low) to 5 (high-high) in increments of one for each serving simulated. If the serving is contaminated with more than one serotype of different virulence, then a composite score is calculated based on the number of each serotype present in the serving. For example, if a 50-g serving is contaminated with 10 cells of Enteritidis (high-high = 5) and 10 cells of Infantis (normal = 3), the composite score = ((10/20)*5) + ((10/20)*3) = 4·0. Finally, the contamination model had an input cell for lot size that was used to calculate the number of servings to be simulated for a given scenario of serving size or serving size distribution.

Process risk model

The PRM was developed by combining the contamination model (Fig. 2) with a published (Oscar 2017a) thermal inactivation model for *Salmonella* and a published (Oscar 2017b) disease triangle, dose–response model for *Salmonella*. The thermal inactivation model (Fig. 3) is a neural network model that predicts death of *Salmonella* to elimination in GC as a function of time (0–10 min), temperature (52–100°C) and initial number (2–5·2 log). The model also predicts the meat temperature and death rate profiles. The model was developed in Excel using NeuralTools. Pert distributions were used to simulate time and temperature of cooking, whereas the output of the contamination model (Fig. 2) was used to simulate initial number in the thermal inactivation model (Fig. 3).

The dose–response model (Fig. 4) had to be modified to accommodate the serotype prediction from the newly developed contamination model (Fig. 2). In the original version, disease triangle scores (meal+*Salmonella*+consumer) ranged from 2·5 to 12·5 in 0·5 increments and there were 21 corresponding pert distributions for illness dose. In the modified version, disease triangle scores

Prevalence	Log number	Frequency	Size	Subsample	Cumulative	Serotype	Risk Score	Outputs	Settings
2	1·825	1	25	67	67	Infantis	3·000	Serving, g	Mean serving, g
0	1·174	0	50	0	67			25	25·0
0	0·303	0	75	0	67			Number	Lot size, kg
2	1·938	0	100	87	154	Infantis		67	100
0	0·089	0	125	0	154			Serotype	Servings
0	1·348	0	150	0	154			3·0	4,000
0	0·393	0	175	0	154				
0	0·008	0	200	0	154				
0	0·880	0	225	0	154				
0	0·344	0	250	0	154				
0	1·613	0	275	0	154				
2	0·131	0	300	1	155	Infantis			

Figure 2 Screenshot of the Excel model for predicting *Salmonella* contamination of ground chicken as a function of serving size at meal preparation. The red font is used for input cells, whereas the blue font is used for output cells that contain formula.

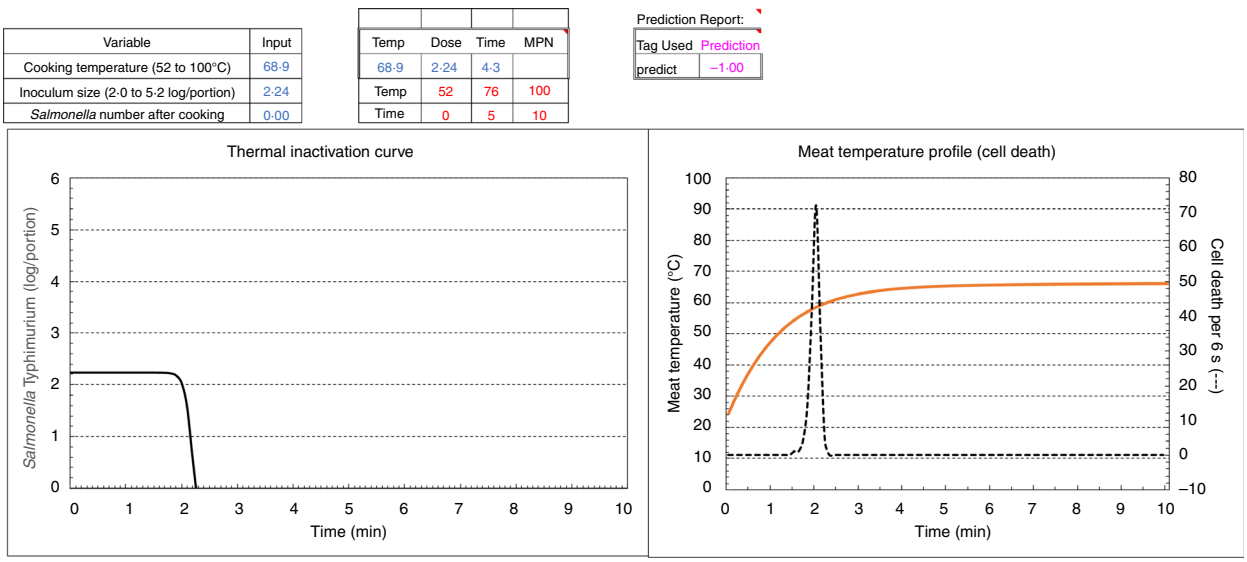

Figure 3 Screenshot of the Excel model for predicting death of *Salmonella* in ground chicken as a function of time, temperature and initial number during cooking. The red font is used for input cells, whereas the blue font is used for output cells that contain formula.

ranged from 2·5 to 12·5 in 0·1 increments with 101 corresponding pert distributions for illness dose.

Process risk model simulation

The PRM was developed in Excel and was simulated with NeuralTools and @Risk. Inputs for Scenario A (control) for a serving size of 25 g are shown in Fig 2. A series of test scenarios (B–D) was simulated to evaluate effects of serving size on model outputs. Scenarios B, C and D simulated serving sizes of 100, 200 and 300 g, respectively.

A second set of scenarios (E–I) was simulated to demonstrate that the PRM could simulate serving size distribution, food handling (i.e. cooking), food consumption behaviour, consumer demographics and serotype virulence. Scenario E (control) was a single modification of Scenario A that used a discrete distribution ({100,200,300}),({80,15,5}) to simulate a distribution of serving sizes of 100, 200 and 300 g.

Four test scenarios were simulated using a single modification of Scenario E. Scenario F simulated more thorough cooking of GC using a modified pert distribution for temperature (52, 80, 100°C) in the thermal inactivation model (Fig. 3). Scenario G simulated risker food consumption behaviour (e.g. increased use of antacids) using a modified discrete distribution ({0·5,1,1·5,2,2·5}), ({5,15,40,30,10}) for meal in the dose–response model (Fig. 4). Scenario H simulated a higher risk consumer population using a modified discrete distribution ({1,2,3,4,5}),({0,10,10,60,20}) for consumer in the dose–response model (Fig. 4).

Scenario I simulated contamination with a low virulence serotype (i.e. Kentucky) using a modified discrete distribution

Disease triangle, dose-response model for *Salmonella*			
Dose			1
Illness dose		4·76	57 290
Meal	Low		
Salmonella	Normal		
Consumer	Normal		
Health outcome		Severity	0·000

Illness dose (log_{10}) sub-model						
Discrete distribution (porportion, outcome value)						
	Proportion per risk category					
Factor	low-low	low	normal	high	high-high	
Meal	5·00	15·00	60·00	15·00	5·00	
Consumer	5·00	15·00	60·00	15·00	5·00	
	Outcome valuse per risk category					Discrete
Factor	low-low	low	normal	high	high-high	output
Meal	0·5	1	1·5	2	2·5	1·0
Salmonella	1	2	3	4	5	3·0
Consumer	1	2	3	4	5	3·0
		lookup value →				7·0
	lookup value		Pert distribution (log_{10})			
		Pert output	minimum	median	maximum	
	12·5	1·03	0·00	1·00	2·00	
	12·4	1·26	0·08	1·08	2·08	
	12·3	1·20	0·16	1·16	2·16	

Figure 4 Screenshot of the Excel model for predicting dose–response of consumers to *Salmonella* ingestion in ground chicken after cooking. The red font is used for input cells, whereas the blue font is used for output cells that contain formula.

for serotype prevalence ({0,1},{81,19}) in the contamination model (Fig. 2). Serotype Kentucky was simulated because it is a common serotype found in chicken (Parveen *et al.* 2007) but rarely causes human clinical cases of salmonellosis. Thus, it was classified as low risk (value = 2) in the simulations and served as a contrasting comparison with Scenario E that simulated only normal (i.e. Infantis) and high-high (i.e. Enteritidis and Typhimurium) risk serotypes.

The PRM was simulated with @Risk settings of Latin Hypercube sampling, Mersenne Twister and six simulations per scenario using random number generator seeds of 1, 7, 17, 29, 37 and 45. The number of servings simulated depended on serving size(s) simulated and was calculated by the contamination model (Fig. 2) based on lot size and mean serving size.

Statistical analysis

Simulation results were analysed in Prism by repeated measures, one-way, analysis of variance. Sphericity (equal variance of differences) was assumed. When there was a significant ($P < 0·05$) effect of scenario, means were compared to the control scenario (i.e. A or E) using Fisher's least significance difference test at $P < 0·05$. Dependent variables analysed were prevalence at meal preparation, total number per lot at meal preparation, prevalence at consumption, total number per lot at consumption, median illness dose, mean serotype score and TSI (TSI = Σ(dose consumed/illness dose)).

Results

Contamination data

Nineteen of 100 (19%) 25-g samples of GC examined tested positive for *Salmonella* by qPCR and cultural isolation following WSE (Table 1). Three serotypes were isolated: Infantis ($n = 13$), Enteritidis ($n = 5$) and Typhimurium ($n = 1$). Infantis is ranked 12th by CDC as a human clinical isolate and therefore, was classified as normal risk (value = 3) per the dose–response model (Fig. 4). Enteritidis is ranked 1st and Typhimurium is ranked 2nd by CDC as human clinical isolates and thus, were classified as high-high risk (value = 5) per the dose–response model.

The log number of *Salmonella* ranged from 0 to 2·56 with a median of 0·93 per 25 g (Table 1). The best fitting distribution to these data was the uniform distribution as determined using the best fit option of @Risk and Akaike's information criterion (results not shown). Thus, a uniform distribution (0,2·56) was used to simulate log number of *Salmonella* per 25 g of GC in the contamination model (Fig. 2). Any growth of *Salmonella* that occurred between grinding or production of GC and meal preparation would be accounted for in these enumeration data.

Scenarios A to D

Simulation results of the PRM for Scenarios A–D indicated that prevalence of *Salmonella* in GC at meal preparation (Fig. 5a) increased ($P < 0·05$) as a function of

Table 1 Number and serotype of *Salmonella* in 25-g samples of ground chicken

Date	Cycle threshold value	Serotype	Log number
9/11/2017	29·90	Infantis	1·161
9/11/2017	31·52	Infantis	0·806
9/18/2017	34·03	Infantis	0·383
9/18/2017	35·27	Infantis	0·231
9/18/2017	29·65	Infantis	1·222
9/18/2017	30·92	Infantis	0·930
9/18/2017	34·77	Infantis	0·288
10/23/2017	30·25	Infantis	1·079
10/23/2017	39·60	Infantis	0·000
10/23/2017	32·28	Infantis	0·661
10/23/2017	29·18	Infantis	1·340
10/30/2017	35·41	Infantis	0·217
11/13/2017	36·85	Infantis	0·093
11/20/2017	26·64	Enteritidis	2·072
11/20/2017	25·19	Enteritidis	2·562
11/20/2017	25·70	Enteritidis	2·384
11/20/2017	25·61	Enteritidis	2·415
11/20/2017	26·61	Enteritidis	2·082
12/4/2017	32·98	Typhimurium	0·541

serving size from 19·0 ± 0% (mean ± standard deviation) for 25 g (Scenario A) to 56·8 ± 0·9% for 100 g (Scenario B) to 81·9 ± 0·5% for 200 g (Scenario C) to 92·6 ± 0·9% for 300 g (Scenario D).

For a lot size of 100 kg of GC, the servings simulated were 4000 for Scenario A (25 g), 1000 for Scenario B (100 g), 500 for Scenario C (200 g) and 333 for Scenario D (300 g). The total number of *Salmonella* per 100-kg lot was not affected ($P > 0·05$) by serving size (Fig. 5b). This was an expected result because no matter how a lot of GC is partitioned, the total number of *Salmonella* should remain the same. The total number of *Salmonella* per 100 kg of GC for Scenarios A–D ($n = 24$) was 46 730 ± 1958 (mean ± SD).

Prevalence of *Salmonella* in GC after cooking or at consumption (Fig. 5c) was lower than before cooking and increased ($P < 0·05$) as a function of serving size from 1·13 ± 0·09% for 25 g (Scenario A) to 3·43 ± 0·4% for 100 g (Scenario B) to 5·23 ± 0·56% for 200 g (Scenario C) to 6·31 ± 0·73% for 300 g (Scenario D). The total number of *Salmonella* consumed per 100-kg lot of GC was not affected ($P > 0·05$) by serving size (Fig. 5d). The total number of *Salmonella* consumed per l00-kg lot of GC for Scenarios A–D was 2346 ± 613 (mean ± SD; $n = 24$).

An illness dose was only assigned to servings that were contaminated with *Salmonella* at consumption. The input settings for *Salmonella*, meal and consumer in the PRM were the same for Scenarios A–D and thus, median illness dose (Fig. 5e) was not affected ($P > 0·05$) by scenario. The median illness dose for Scenarios A–D was 40 615 ± 20 487 (mean ± SD; $n = 24$).

Likewise, the serotype value used in the dose–response model was not affected ($P > 0·05$) by serving size because the input values for this variable were the same for Scenarios A–D. The mean serotype value for Scenarios A–D was 3·6 ± 0·02 (mean ± SD; $n = 24$).

Finally, TSI was not affected ($P > 0·05$) by serving size (Fig. 5f) and for Scenarios A–D was 2·47 ± 2·65 (mean ± SD; range: 0·17–11·1; $n = 24$). Thus, how the 100-kg lot of GC was partitioned and consumed did not affect its public health impact.

Scenarios E to I

Scenarios E–I simulated the same serving size distribution among consumers, which was 80% consumed 100 g (like a cooked ¼-lb burger), 15% consumed 200 g and 5% consumed 300 g of GC. Prevalence (Fig. 6a) and total number (Fig. 6b) of *Salmonella* that contaminated GC at meal preparation were the same for all scenarios because the same input settings for contamination at meal preparation were used for all five scenarios. Prevalence of *Salmonella* at meal preparation was 62·2 ± 0·7% (mean ± SD; $n = 24$), whereas the total number of *Salmonella* per 100 kg of GC was 46 629 ± 2545 (mean ± SD; $n = 24$).

More thorough cooking of GC (Scenario F) reduced ($P < 0·05$) the prevalence of *Salmonella* at consumption (Fig. 6c) from 3·88 ± 0·49% for Scenario E to 2·67 ± 0·38% for Scenario F. More thorough cooking of GC (Scenario F) also reduced ($P < 0·05$) the total number of *Salmonella* consumed (Fig. 6d) from 2458 ± 880 in Scenario E to 1806 ± 825 in Scenario F. Although more through cooking (Scenario F) reduced consumer exposure to *Salmonella* from GC, median illness dose (Fig. 6e) and TSI (Fig. 6f) were not affected ($P > 0·05$) by more thorough cooking. Median illness dose was 35 627 ± 24 211 for Scenario E and 43 592 ± 46 717 for Scenario F, whereas TSI was 5·25 ± 4·54 for Scenario E and 4·66 ± 4·89 for Scenario F.

The food, beverages and supplements (e.g. antacid) consumed with GC during or after the meal can affect illness dose and severity of illness. This possibility was simulated in Scenario G by increasing the percentage of high-risk food consumption events. Although consumers engaged in higher risk food consumption behaviour in Scenario G, median illness dose (Fig. 6e) and TSI (Fig. 6f) were not different ($P > 0·05$) from the control scenario (Scenario E). Median illness dose was 35 627 ± 24 211 for Scenario E and was 37 860 ± 29 193 for Scenario G, whereas TSI was 5·25 ± 4·54 for Scenario E and was 6·70 ± 6·88 for Scenario G.

Test Scenario H simulated consumption of the GC by a higher risk consumer population. Here, reduced consumer resistance was reflected in a lower ($P < 0·05$) median

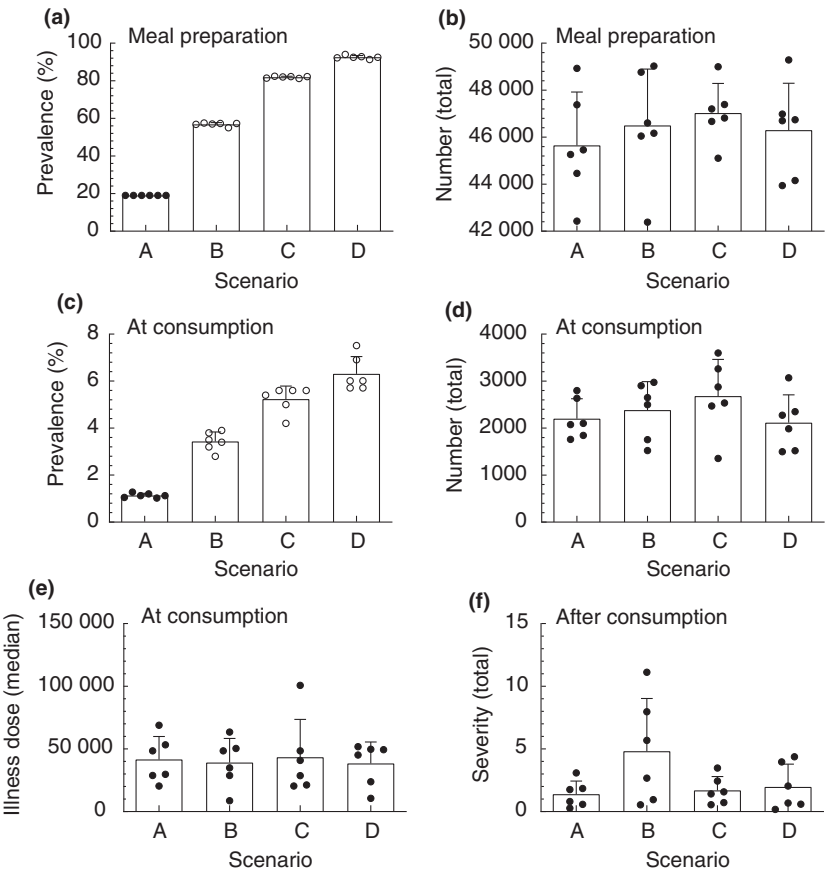

Figure 5 Simulation results for Scenarios A–D. Bars with open (○) symbols differ from Scenario A at $P < 0{\cdot}05$.

illness dose (Fig. 6e) and increased ($P < 0{\cdot}05$) TSI (Fig. 6f). Median illness dose was 35 627 ± 24 211 for Scenario E and 4656 ± 2380 for Scenario H, whereas TSI was 5·25 ± 4·54 for Scenario E and 10·2 ± 4·6 for Scenario H.

The final test scenario (Scenario I) simulated consumption of GC contaminated exclusively with a low-risk serotype of *Salmonella* (i.e. serotype Kentucky). Here, reduced virulence of *Salmonella* resulted in a lower ($P < 0{\cdot}05$) serotype risk score (i.e. 3·62 ± 0·02 for Scenario E and 2·00 ± 0·00 for Scenario I), a higher ($P < 0{\cdot}05$) median illness dose (Fig. 6e) and a decreased ($P < 0{\cdot}05$) TSI (Fig. 6f). Median illness dose was 35 627 ± 24 211 for Scenario E and 603 674 ± 264 825 for Scenario I, whereas TSI was 5·25 ± 4·54 for Scenario E and was 0·046 ± 0·033 for Scenario I.

Discussion

A common approach for simulating serving size in a PRM is to express pathogen number per gram throughout the model and then multiple this number by serving size at consumption to obtain the dose consumed (Bemrah *et al.* 2003; Smadi and Sargeant 2013). This approach assumes that the pathogen is evenly distributed in the food. However, in the present study, *Salmonella* were not evenly distributed among 25 g samples of GC. Therefore, the common approach to simulating serving size was not used in the current study. Rather, a model was developed that predicted the number of *Salmonella* as a function of serving size and then the number of *Salmonella* that survived cooking of the serving size simulated was used as the dose consumed.

Another common approach in process risk modelling is to use a sigmoid-shaped dose–response curve to simulate consumer response to *Salmonella* exposure (Smadi and Sargeant 2013; Zhu *et al.* 2017). However, when a food is contaminated with multiple serotypes or strains of *Salmonella* of different virulence, the dose–response curve is not sigmoid in shape (Oscar 2004a). Therefore, to provide a better simulation of dose–response, a model (i.e. disease triangle, dose–response model) that is based on outbreak and human feeding trial data and that produces nonsigmoid-shaped dose–response curves and that considers differences in virulence among serotypes of *Salmonella*, differences in food factors and difference in resistance among consumers was modified and used in the present study (Oscar 2017b).

Another common approach in process risk modelling is to express dose–response as a probability of illness

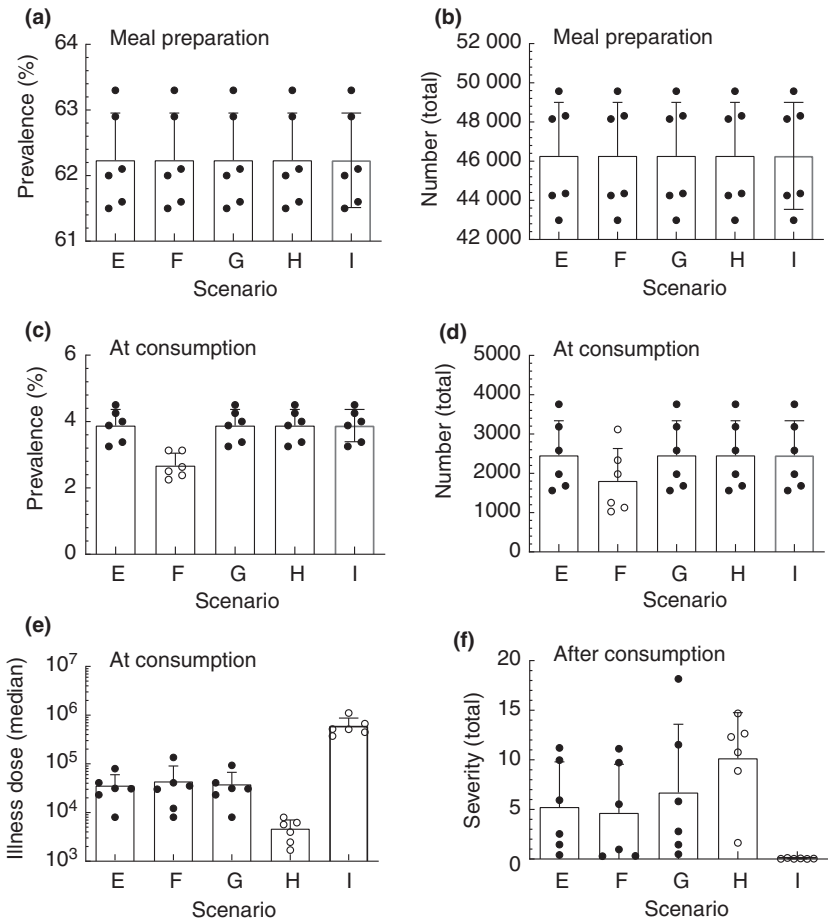

Figure 6 Simulation results for Scenarios E–I. Bars with open (○) symbols differ from Scenario E at $P < 0{\cdot}05$.

(Straver *et al.* 2007; Smadi and Sargeant 2013). However, when a consumer ingests *Salmonella*, they do not have a probability of becoming ill. Rather, their response falls on a continuum from no response to death. In the present study, the dose–response continuum or severity of illness was simulated using the ratio of dose consumed to illness dose where a ratio of one was equivalent to an illness (i.e. a diagnosed case of salmonellosis). The sum of severity of illness for all servings in the lot or TSI was used as the primary risk endpoint in the current PRM.

Total severity of illness per lot of GC was highly variable among replicate simulations of the same scenario in the present study. This occurred because of the rare, random, variable and uncertain nature of events in the risk pathway. This variability was characterized by running multiple simulations of a scenario using different random number generator seeds. This allowed a statistical comparison of scenarios using a repeated measure, one-way, analysis of variance approach. Importantly, it was observed that significant changes in prevalence and/or number of *Salmonella* were not always consistent with significant changes in TSI indicating that consumer exposure (i.e. prevalence and/or number) is not a good indicator of food safety. Rather, the best indicator of food safety is a risk endpoint that considers consumer exposure and response to *Salmonella* exposure (i.e. TSI).

Another common approach in process risk modelling is to simulate the national production of chicken (Maijala *et al.* 2005; Uyttendaele *et al.* 2009; Smadi and Sargeant 2013). In contrast, in the present study, process risk modelling was used to simulate the local production of a single lot of GC. This approach was taken because the current approach to chicken safety is focused on determining whether a processing plant is producing safe or unsafe chicken. Thus, using a PRM at the processing plant would be a good approach to identify that rare lot of chicken that might cause an outbreak. If PRM was used in this manner throughout the chicken industry the combined results would be a simulation of the national production of the food commodity.

The current approach to food safety uses performance standards for *Salmonella* that are based on prevalence. This approach can be improved by also considering number, serotype and postprocessing risk factors, such as

consumer food handling practices, serotype virulence and consumer resistance and food consumption practices. A PRM like the one developed in the present study can be used to integrate prevalence, number, serotype and post-processing risk factor data into a more complete evaluation of chicken safety. By taking this more holistic approach to food safety, the chicken industry will be better able to identify safe and unsafe lots of chicken at the processing plant and in the process better protect public health while reducing the chance of food recalls.

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Disclaimer

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Conflict of Interest

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