

Salmonella Prevalence Alone Is Not a Good Indicator of Poultry Food Safety

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Salmonella is a leading cause of foodborne illness (i.e., salmonellosis) outbreaks, which on occasion are attributed to ground turkey. The poultry industry uses *Salmonella* prevalence as an indicator of food safety. However, *Salmonella* prevalence is only one of several factors that determine risk of salmonellosis. Consequently, a model for predicting risk of salmonellosis from individual lots of ground turkey as a function of *Salmonella* prevalence and other risk factors was developed. Data for *Salmonella* contamination (prevalence, number, and serotype) of ground turkey were collected at meal preparation. Scenario analysis was used to evaluate effects of model variables on risk of salmonellosis. Epidemiological data were used to simulate *Salmonella* serotype virulence in a dose-response model that was based on human outbreak and feeding trial data. *Salmonella* prevalence was 26% ($n = 100$) per 25 g of ground turkey, whereas *Salmonella* number ranged from 0 to 1.603 with a median of 0.185 log per 25 g. Risk of salmonellosis (total arbitrary units (AU) per lot) was affected ($p \leq 0.05$) by *Salmonella* prevalence, number, and virulence, by incidence and extent of undercooking, and by food consumption behavior and host resistance but was not ($p > 0.05$) affected by serving size, serving size distribution, or total bacterial load of ground turkey when all other risk factors were held constant. When other risk factors were not held constant, *Salmonella* prevalence was not correlated ($r = -0.39$; $p = 0.21$) with risk of salmonellosis. Thus, *Salmonella* prevalence alone was not a good indicator of poultry food safety because other factors were found to alter risk of salmonellosis. In conclusion, a more holistic approach to poultry food safety, such as the process risk model developed in the present study, is needed to better protect public health from foodborne pathogens like *Salmonella*.

KEY WORDS: Food safety; ground turkey; poultry; risk assessment; *Salmonella* prevalence

1. INTRODUCTION

Salmonella are a leading cause of foodborne illness outbreaks (Kirk, Ford, Glass, & Hall, 2014; Majowicz et al., 2010) that on occasion are attributed to poultry meat (Antunes, Mourao, Campos, & Peixe, 2016; Routh et al., 2015; Uyttendaele et al., 2009). The current approach to food safety in the poultry

industry consists of two approaches. First, pathogen reduction and process control (Hazard Analysis and Critical Control Points) are used to reduce consumer exposure to foodborne pathogens (e.g., *Salmonella* and *Campylobacter*). Success is based on compliance with performance standards based on pathogen prevalence (Ebel & Williams, 2019; Simonsen et al., 1987; Williams & Ebel, 2012). Second, active surveillance and next-generation sequencing methods are used to identify and source foodborne illness outbreaks with subsequent recall of implicated food to reduce its harm to public health (Anonymous, 2014; Chai, Cole, Nisler, & Mahon, 2017). The limitation of the first approach is that pathogen prevalence is only

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one of several risk factors that determine food safety (Lambertini, Ruzante, Chew, Apodaca, & Kowalczyk, 2019; Membre & Boue, 2018). Consequently, this approach is not good at identification of unsafe food before it is distributed to consumers. This is confirmed by outbreaks of illness from food that has passed inspection (Anonymous, 2013; Chai et al., 2017; Jackson, Griffin, Cole, Walsh, & Chai, 2013). The limitation of the second approach is that harm to public health occurs before corrective action is taken.

One way to improve food safety is to use a more comprehensive or holistic approach. One that better identifies unsafe lots of food before they are shipped to consumers. Development of farm-to-table models that predict food safety as a function of multiple risk factors is one approach that can be used to accomplish this (Akil & Ahmad, 2019; Al-Sakkaf, 2019; Casulli, Calhoun, & Schaffner, 2019; Farakos et al., 2019). Recently, a farm-to-table model that predicts safety of individual lots of ground chicken contaminated with *Salmonella* was developed and used to demonstrate the importance of considering multiple risk factors when evaluating food safety (Oscar, 2019). In the current study, a similar process risk model for *Salmonella* and ground turkey was developed with two important modifications. First, epidemiological data were used to more precisely simulate differences in *Salmonella* serotype virulence. Second, a new thermal line of death model was used to better simulate *Salmonella* death and survival during cooking. The process risk model was used to evaluate *Salmonella* prevalence and other factors (i.e., *Salmonella* number, *Salmonella* virulence, serving size, serving size distribution, total bacterial load, incidence of undercooking (Luber, 2009; Porto-Fett et al., 2019; Swart, van Leusden, & Nauta, 2016), extent of undercooking, food consumption behavior, and host resistance) for their effects on risk of salmonellosis and to compare risk of salmonellosis from individual lots of ground turkey and ground chicken shipped to the same distribution channel and consumer population.

2. MATERIALS AND METHODS

2.1. Data Collection

One-pound packages of ground turkey were purchased from a local retail store (Salisbury, MD) between December 26, 2017 and April 23, 2018. Data for prevalence, number, and serotype of *Salmonella*

in 25 g samples were obtained using a combination of whole sample enrichment (WSE), quantitative polymerase chain reaction (qPCR), cultural isolation, and serotyping as described in a previous study (Oscar, 2019). In brief, a standard curve (Fig. 1) for log number of *Salmonella* in 25 g of ground turkey as a function of cycle threshold (C_T) value from qPCR (iQ-Check for *Salmonella enterica*, Bio-Rad Laboratories, Hercules, CA) was developed using the Weibull model in Prism (version 8.3, GraphPad Software Inc., San Diego, CA) as follows:

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

where $C_{T_{\max}}$ was the maximum observed C_T (36.15), x was the log number of *Salmonella* in 25 g, a (0.01187 ± 0.00325 ; best-fit $\pm$ standard error) was a regression coefficient, and b (0.5251 ± 0.0255) was a shape parameter, and goodness-of-fit, as assessed by the adjusted coefficient of determination (R^2) was 0.979 with 19 degrees of freedom. The interpolation function of Prism was used to determine log number of unknown 25 g samples (Table I). The standard curve was developed with a single strain of *Salmonella* Infantis, which was isolated from ground turkey in the present study. Samples (1 mL) for qPCR were collected at 6 hours of WSE in 400 mL of buffered peptone water (BPW; Microbiology International, Frederick, MD) incubated at 40 °C and 80 rpm.

Salmonella were isolated from WSE (1 mL) by nonselective enrichment for an additional 24 hours at 40 °C followed by selective enrichment of WSE (10 μ L) in 1 mL of Rappaport Vassiliadis broth (Becton, Dickinson and Co., Sparks, MD) for 24 hours at 42 °C followed by selective plating (1 μ L) on xylose lysine tergitol 4 agar (XLT4; Becton, Dickinson) for 24 hours at 40 °C. One presumptive colony per XLT4 plate or sample was regrown in BPW and confirmed as positive for *Salmonella* using an antibody-based test strip (Reveal 2.0 for *Salmonella*, Neogen Corp., Lansing, MI) before being sent to a *Salmonella* reference laboratory (U. S. Department of Agriculture, National Veterinary Services Laboratory, Ames, IA) for serotyping.

2.2. Process Risk Model

The process risk model was developed in Excel (version 2016, MicroSoft Corp., Redmond, WA) and was simulated using @Risk (version 7.6.1, Palisade Corp., Ithaca, NY), a spreadsheet add-in program that allowed input variables to be described as

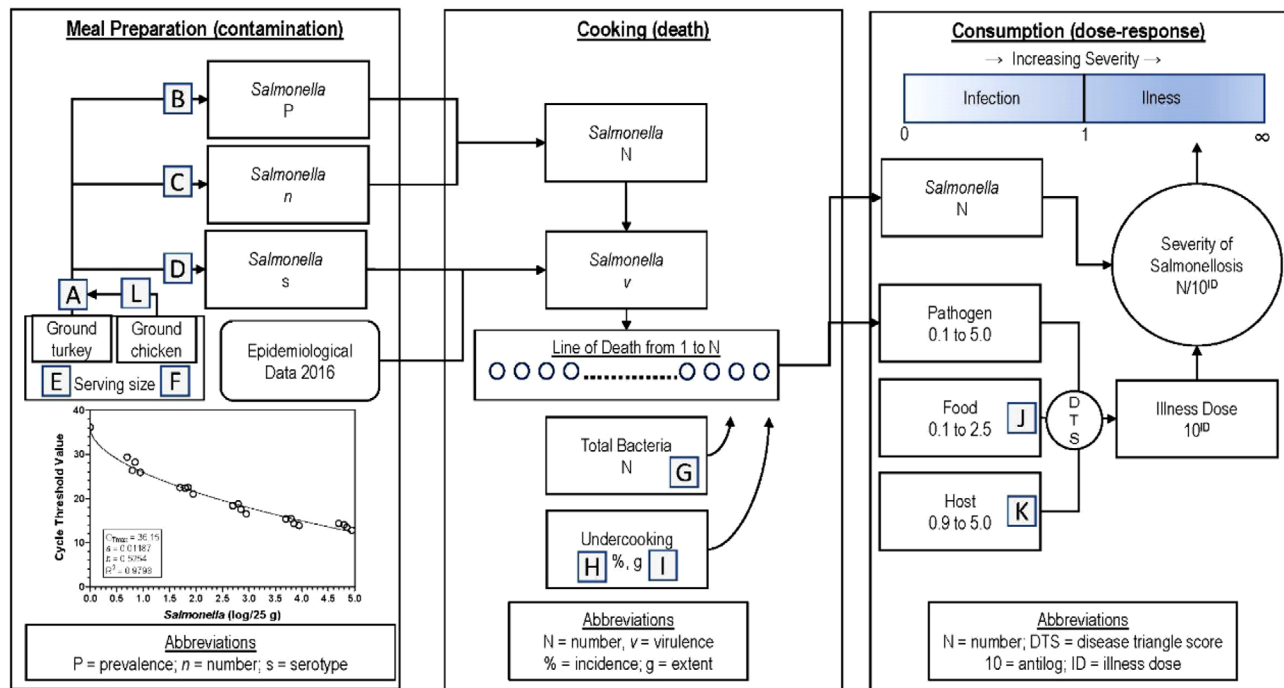


Fig 1. Schematic diagram of the process risk model used to simulate risk of salmonellosis from ground turkey or ground chicken. The locations in the model where input distributions were changed to simulate test scenarios B to L are indicated in text boxes. The baseline scenario was scenario A.

probability distributions rather than by single values. The process risk model consisted of four spreadsheets: (1) data entry (Fig. 2), (2) meal preparation (contamination) submodel (not shown), (3) cooking (death) submodel (not shown), and (iv) consumption (dose-response) submodel (not shown). A schematic diagram of the process risk model is shown in Fig. 1.

2.2.1. Meal Preparation (Contamination) Submodel

Data for prevalence, number, and serotype and virulence were used to develop a submodel for *Salmonella* contamination of ground turkey at meal preparation. The submodel predicted *Salmonella* prevalence, number, and serotype and virulence as a function of serving size from 25 to 325 g in 25 g increments using a previously published method (Oscar, 2004c, 2019).

Serving size was simulated using a discrete distribution. In baseline scenario A, which simulated a serving size of 25 g, the discrete distribution was: {25,50,75,100,125,150,175,200,225,250,275,300,325}, {100,0,0,0,0,0,0,0,0,0,0,0}

where the numbers in the left set of brackets were the serving sizes in grams (discrete distribution

outputs) and the numbers in the right set of brackets were the frequency of occurrence of each serving size. In test scenario E (Table II) a serving size of 125 g was simulated by changing this distribution to: {25,50,75,100,125,150,175,200,225,250,275,300,325}, {0,0,0,0,100,0,0,0,0,0,0,0} and in test scenario F (Table II) a serving size distribution was simulated by changing this distribution to: {25,50,75,100,125,150,175,200,225,250,275,300,325}, {0,25,0,0,50,0,0,25,0,0,0,0}.

Sampedro, Wells, Bender, and Hedberg (2018) reported that ground turkey consumption ranges from 85 to 170 g with a mean of 113 g. Thus, test scenarios E and F simulated the mean and range of ground turkey servings that occur in practice. A serving size of 113 g could not be simulated exactly because the model only simulated serving sizes in 25 g increments from 25 g to 325 g. However, it is possible to simulate a mean serving size of 112.5 g by using various combinations of serving sizes, such as 100 (50%) and 125 (50%) g.

A discrete distribution was used to simulate *Salmonella* serotype prevalence (virulence). In baseline scenario A (Fig. 2 and Table II), which was based on data collected using 25 g samples

Table I. Isolation Date, Cycle Threshold (C_T) Value, Serotype, 2016 Rank, 2016 Cases, Virulence Score, and Log Number of *Salmonella* in 25-g Samples of Ground Turkey

Date	C_T	Serotype	Rank	Cases	Virulence	Log Number
January 22, 2018	29.08	Infantis	6	1,281	4.5	0.492
January 29, 2018	32.71	Typhimurium	3	4,581	4.8	0.125
January 29, 2018	32.18	Typhimurium	3	4,581	4.8	0.164
January 29, 2018	30.59	Typhimurium	3	4,581	4.8	0.312
January 29, 2018	34.84	Typhimurium	3	4,581	4.8	0.020
January 29, 2018	32.36	Typhimurium	3	4,581	4.8	0.150
January 29, 2018	31.67	Reading	>20	221	2.7	0.206
January 29, 2018	30.20	Typhimurium	3	4,581	4.8	0.355
January 29, 2018	30.81	Typhimurium	3	4,581	4.8	0.289
January 29, 2018	33.12	Typhimurium	3	4,581	4.8	0.098
February 12, 2018	33.36	Hadar	>20	205	2.5	0.084
February 20, 2018	32.50	Typhimurium	3	4,581	4.8	0.140
February 20, 2018	33.57	Typhimurium	3	4,581	4.8	0.072
February 20, 2018	35.60	Typhimurium	3	4,581	4.8	0.004
February 20, 2018	29.64	Typhimurium	3	4,581	4.8	0.421
February 26, 2018	34.05	Typhimurium	3	4,581	4.8	0.049
March 19, 2018	30.57	Hadar	>20	205	2.5	0.890
March 19, 2018	26.50	Infantis	6	1,281	4.5	1.603
March 19, 2018	23.01	Kentucky	>20	63	0.8	1.147
March 19, 2018	25.13	Kentucky	>20	63	0.8	0.982
March 19, 2018	25.99	Infantis	6	1,281	4.5	0.825
March 19, 2018	26.88	Infantis	6	1,281	4.5	0.314
April 9, 2018	35.79	Rough_O:eh:-	>20	220	2.7	0.002
April 16, 2018	26.96	4, 5, 12:i:-	5	2,179	4.6	0.811
April 23, 2018	36.15	Reading	>20	221	2.7	0.000
April 23, 2018	32.48	Reading	>20	221	2.7	0.141

of ground turkey, the discrete distribution was: $\{0,1,2,3,4,5,6,7\},\{74,4,13,3,2,2,1,1\}$ where the distribution outputs (frequency of occurrence) were 0 = none (74%), 1 = Infantis (4%), 2 = Typhimurium (13%), 3 = Reading (3%), 4 = Hadar (2%), 5 = Kentucky (2%), 6 = 4,5,12:i:- (1%), and 7 = Rough_O:eh:- (1%). In test scenario B (Table II), input values for this discrete distribution were altered to simulate a higher prevalence of *Salmonella* at meal preparation but with the same ratio of serotypes among contaminated servings. In test scenario D (Table II), the discrete distribution for *Salmonella* prevalence at meal preparation was altered to simulate the same overall prevalence as in baseline scenario A but with a single serotype (i.e., Kentucky) of *Salmonella* rather than seven different serotypes of *Salmonella*. In test scenario L (Table II), a different discrete distribution $\{0,1,2,3\},\{81,13,1,5\}$ was used to simulate *Salmonella* serotype prevalence in ground chicken that was based on results from a previous study (Oscar, 2019) with 25 g samples and where 0 = none (81%); 1 = Infantis (13%); 2 = Typhimurium (1%); and 3 = Enteritidis (5%).

To translate *Salmonella* serotype prevalence into *Salmonella* virulence (v) for use in the consumption (dose-response) submodel, epidemiological data for *Salmonella* from the U. S. Centers for Disease Control and Prevention in 2016 (www.cdc.gov/nationalsurveillance/pdfs/2016-report-508.pdf) were used. For serotypes ranked in the top 20 of human clinical isolates, virulence in arbitrary units (AU) per serving from 3.1 to 5 in 0.1 increments was calculated as follows:

$$v = 5.1 - 0.1(r),$$

where r was the ranking from 1 to 20. For serotypes ranked outside the top 20, virulence (0.1–3 in 0.1 increments of AU per serving) was calculated as follows:

$$v = 3.1 \times (c/257),$$

where c was the number of cases for the serotype and 257 was the number of cases for the 20th ranked serotype, which was *Salmonella* Anatum.

The VLOOKUP function of Excel was used to assign the appropriate virulence value, which was 4.5

Inputs					Incidence		Extent			
Unit Operation	Pathogen Event	Other	Serotype	Virulence	No	Yes	Minimum	Most Likely	Maximum	Unit
Processing Plant		Lot Size						907		kg
Meal Preparation	Contamination	Salmonella	None	0		74	0	0.185	1.603	log
			Infantis	4.5		4				
			Typhimurium	4.8		13				
			Reading	2.7		3				
			Hadar	2.5		2				
			Kentucky	0.8		2				
			4,[5],12:i:-	4.6		1				
			Rough_O:eh:-	2.7		1				
		Total Bacteria					4.4	6.4	8.4	log
Cooking	Death	Undercooking			80	20	0.0	1.0	5.0	g
Consumption	Dose-Response	Food Factors					0.1	1.5	2.5	au
		Host Resistance					0.9	3.5	5.0	au
		Serving Size						25		g
		Servings per Lot						36,280		

Outputs								
Unit Operation	Pathogen Event	Human Event	Prevalence	Number	Virulence	DTS	ID	Severity
Meal Preparation	Contamination		1	2	4.8			
Cooking	Death		1	2	4.8			
Consumption	Dose-Response					9.7	2.95	0.002259666

Fig 2. Data entry page for the process risk model used to simulate risk of salmonellosis from ground turkey or ground chicken. The model was developed in an Excel notebook and was simulated with @Risk, a spreadsheet add-in program. The input settings are for baseline scenario A for ground turkey. The output results are for a single serving (25 g) of ground turkey. Red, input by user; blue, calculated by model; black, fixed label or value.

for Infantis, 4.8 for Typhimurium, 2.7 for Reading, 2.5 for Hadar, 0.8 for Kentucky, 4.6 for 4,5,12:i:-, 2.7 for Rough_O:eh:-, and 5.0 for Enteritidis (Table II). Virulence values were rounded to one decimal place so that they could be used in the disease triangle, dose-response model for *Salmonella* (Oscar, 2019). In addition, if a serving was contaminated with more than one serotype, a composite score was calculated. For example, if a serving was contaminated with 2 cells of Hadar ($v = 2.5$) and 3 cells of Typhimurium ($v = 4.8$), the composite score was $3.9 = ((2/5) \times 2.5) + ((3/5) \times 4.8)$.

A pert distribution (minimum, most likely, maximum) was used to simulate *Salmonella* number for ground turkey, which ranged from 0 to 1.603 log with a most likely (median) value of 0.185 log per 25 g. Similarly, a pert distribution (0, 0.93, 2.562) was used to simulate *Salmonella* number (log/25 g) for ground chicken in test scenario L (Table II). In test scenario D (Table II), a modified pert distribution (0, 0.685,

2.103) was used to simulate a higher *Salmonella* number (log/25 g) for ground turkey.

Serving size from 25 to 325 g in 25 g increments was simulated by using 13 pairs of the same discrete distribution for *Salmonella* serotype prevalence and the same pert distribution for *Salmonella* number. During simulation of the model, each discrete distribution and pert distribution pair was randomly sampled. However, the output of the pert distribution was only used to calculate *Salmonella* number for the serving being simulated when the output of the corresponding discrete distribution was > 0 indicating that the 25 g portion was contaminated with *Salmonella*. In addition, the serving size simulated determined which outputs of the discrete and pert distributions pairs were used to calculate *Salmonella* contamination (prevalence, number, and serotype and virulence) for the serving being simulated. For example, if the serving size was 125 g, the outputs of the 8 discrete and corresponding 8 pert distributions for 25 g

Table II. Input Settings for Baseline Scenario A and Test Scenarios B to L

Scenario	Meat	Model Variable	Baseline Scenario A	Test Scenario B to L
B	Turkey	Prevalence (%)	74	37
C	Turkey	Number (log/25 g)	0, 0.185, 1.603	0, 0.685, 2.103
D	Turkey	Serotype (%)	4, 13, 3, 2, 2, 1, 1	0, 0, 0, 0, 26, 0, 0
E	Turkey	Serving size (g (%))	25 (100)	125 (100)
F	Turkey	Serving size distribution (g (%))	25 (100)	50 (25), 125(50), 200 (25)
G	Turkey	Total bacteria (log/25 g)	4.4, 6.4, 8.4	3.4, 5.4, 7.4
H	Turkey	Incidence of undercooking (%)	20	40
I	Turkey	Extent of undercooking (g)	0, 1, 5	0, 2, 10
J	Turkey	Food consumption behavior (AU/25 g)	0.1, 1.5, 2.5	0.6, 2.0, 2.5
K	Turkey	Host resistance (AU/25 g)	0.9, 3.5, 5.0	1.9, 4.5, 5.0
L	Chicken	Prevalence (%)		81
		Number (log/25 g)		0, 0.93, 2.562
		Serotype (%)		13, 1, 5, 0, 0, 0, 0

portions from 150 to 325 g were ignored in the calculation of the *Salmonella* contamination results.

Salmonella prevalence (P), number (n), and serotype and virulence (v) per 25 g portion in a serving were simulated by linking discrete and pert distributions (Oscar, 1998, 2004b):

$$P = 0, n = 0, \text{ and } v = 0 \quad \text{if Discrete} = 0$$

$$P = 1, n = 10^{\text{Pert}}, \text{ and } v = 0.1 \text{ to } 5 \quad \text{if Discrete} > 0,$$

where 10^{Pert} was the antilog of the pert distribution output for log number. *Salmonella* number was rounded to the nearest whole number because it is not possible to have a fraction of a cell and summed across 25 g portions for the serving size simulated to obtain *Salmonella* number (N) per serving at meal preparation. In addition, outputs of *Salmonella* number and serotype virulence at meal preparation served as inputs to the cooking submodel (Fig. 1).

2.2.2. Cooking (Death) Submodel

Death and survival of *Salmonella* during cooking of ground turkey was simulated as a function of their rare, random, variable, and uncertain location within the food matrix and total bacterial population. Differences in thermal resistance among serotypes of *Salmonella* (Bermudez-Aguirre & Corradini, 2012; Murphy, Duncan, Johnson, Davis, & Smith, 2002) were ignored in deference to their random location in the food matrix and total bacterial population because a cell with higher thermal resistance can die before a cell with lower thermal resistance if by random chance it is located closer to the heat source. Con-

sequently, death and survival of bacteria including *Salmonella* during cooking of ground turkey was simulated as occurring in a thermal death timeline without assignment of a specific mechanism of death or survival with respect to time, temperature, and thermal resistance.

To further illustrate this line of thought and to justify scenario G, three scenarios were considered. In scenario 1, a pure culture (1 mL) of 100 cells of *Salmonella* was heated to kill one cell per second. The thermal inactivation curve was linear, and death of *Salmonella* was nonrandom. In scenario 2, a mixed culture (1 mL) of one cell of *Salmonella* and 99 cells of other bacteria was heated to kill one cell per second. The thermal inactivation curve was linear but time of death for *Salmonella* was random; it occurred within any one second interval from 0 to 100 seconds. In scenario 3, a mixed culture (1 mL) of one cell of *Salmonella* and 999 cells of other bacteria was heated to kill one cell per second. The thermal inactivation curve was linear but time of death for *Salmonella* was random; it occurred within any one second interval from 0 to 1,000 seconds. Therefore, the higher number of other bacteria could by random chance extend the life of the *Salmonella* cell up to 1,000 seconds. Thus, total bacterial load could be an important risk factor for salmonellosis (justification for scenario G). Together these scenarios show that when *Salmonella* was a minority member of the native microflora, its time of thermal death was random. Thus, it was appropriate to simulate thermal death of *Salmonella* as a random event.

The cooking submodel assumptions were: (1) bacteria were evenly distributed among portions (25 g) of a serving (25–325 g in 25 g increments)

before cooking (Dhananjayan, Han, Acton, & Dawson, 2006), (2) portions of a serving were cooked independently, (3) proper cooking resulted in death of all bacteria, (4) undercooking resulted in survival of bacteria, and (5) location of *Salmonella* in the thermal death timeline was random.

The following methods were used to simulate these assumptions. For the first assumption, Dhananjayan et al. (2006) reported that total bacteria (log/g) in ground turkey was 3 (4.4 log/g (log/25 g) at day 0, 5 (6.4 log/g (log/25 g) at day 3 of cold (4 °C) storage, and was spoiled at 7 (8.4 log/g (log/25 g) at day 9 of cold (4 °C) storage. Therefore, a single pert distribution (4.4, 6.4, 8.4) was used in the baseline scenario A (Table II) to simulate the assumption that total bacteria (log) were evenly distributed among portions (25 g) of a serving (25–325 g in 25 g increments). In test scenario G (Table II), a single pert distribution (3.4, 5.4, 7.4) was used to simulate a lower number (log) of total bacteria per portion (25 g). This scenario simulated “What if” consumers stored the ground turkey for less time in their refrigerators before they cooked it. The total bacterial numbers simulated in scenario G were within those reported in the scientific literature (Dhananjayan et al., 2006) on which the baseline scenario A was based.

During proper refrigerated storage (4 °C) of poultry meat, *Salmonella* number will not change because 4 °C is below the minimum temperature for growth (Oscar, 2014, 2015). However, during improper refrigerated storage (15 °C) of poultry meat, *Salmonella* number will increase because 15 °C is above the minimum temperature for growth (Oscar, 2017b). On the other hand, during proper (4 °C) and improper (15 °C) refrigerated storage of poultry meat, the number of psychotropic spoilage microflora will increase at lower and higher growth rates, respectively (Baranyi, Robinson, Kaloti, & Mackey, 1995; del Rio, Gonzalez de Caso, Prieto, Alonso-Calleja, & Capita, 2008; McClure, Baranyi, Boogard, Kelly, & Roberts, 1993). In the present study, *Salmonella* number of ground turkey was determined at meal preparation or after refrigerated storage. Thus, samples with higher numbers of *Salmonella* (Table I) could represent ground turkey that was not properly refrigerated. Also, in the current study, the number of native microflora at meal preparation or after refrigerated storage was simulated based on published data for ground turkey (Dhananjayan et al., 2006). The pert distribution used for total bacterial load simulated a full range of scenarios for refrigerated storage from freshly

ground and stored meat (4.4 log/g) to nearly spoiled meat (8.8 log/g) from extended storage. Thus, behavior of *Salmonella* and native microflora before meal preparation (refrigerated storage) was simulated at meal preparation as an intrinsic property of the distributions used to simulate this unit operation (meal preparation) and pathogen event (contamination).

For the second assumption, Sampedro et al. (2018) in a risk assessment for *Salmonella* and ground turkey reported that 20% of consumers eat ground beef cooked to a doneness of medium rare (6%) or medium (14%), which would likely result in a portion of the serving being raw after cooking. Therefore, 13 discrete distributions (one for each 25 g portion in a serving from 25 to 325 g) for incidence of undercooking ($\{0, 1\} \{80, 20\}$) were used in the baseline scenario A (Table II) to simulate the assumption that each portion of a serving was cooked independently and where 0 = proper cooking, 1 = undercooking, 80 = incidence of proper cooking, and 20 = incidence of undercooking. In test scenario H (Table II), a different discrete distribution ($\{0, 1\} \{60, 40\}$) was used to simulate a higher incidence of undercooking.

For the third assumption, a formula (if the output of the discrete distribution = 0 then *Salmonella* number = 0) that returned a value of zero was used to simulate the assumption that proper cooking resulted in death of all bacteria including *Salmonella*.

For the fourth assumption, a pert distribution (0, 1, 5) was used in the baseline scenario A (Table II) to simulate extent of undercooking as the amount (g) of a portion (25 g) that was raw after cooking or received insufficient heat treatment to kill any bacteria. In test scenario I (Table II), a modified pert distribution (0, 2, 10) was used to simulate a higher extent of undercooking.

A model that predicts log reduction was not used because such model does not exist for the native microflora of ground turkey. The model that is needed to fill this data gap in the present process risk model is one that predicts the number of bacterial survivors per 25 g as a function of initial number per 25 g, and time and temperature of cooking. The predictions from this model could be used outside the process risk model to predict the probability distributions for incidence (discrete) and extent (pert) of undercooking (Oscar, 2004b). More specifically, the thermal inactivation model for native microflora would predict the equivalent amount of ground turkey (g/25g) that did not receive enough heat to kill any bacteria. For example, if the model predicted the number

of bacterial survivors was 1,000 per 25 g and the initial number of bacteria was 25,000 per 25 g then the equivalent amount of ground turkey that did not receive enough heat to kill any bacteria would be 1 g. The reason that the model was not available for the present study was that quantitative microbial risk assessment (QMRA) is an iterative process. Sometimes it is necessary to build the process risk model first and then use it as a guide to obtain the needed data for development of submodels.

For the fifth assumption, the RANDBETWEEN function of Excel was used to simulate the assumption that the location of *Salmonella* in the thermal line of death was random where the range was from 1 to N , the total number of bacteria in the 25 g portion.

A model for thermal death of *Salmonella* in ground chicken with native microflora as a dynamic function of time (0–10 minutes), temperature (50–100 °C), and initial number (2–5 log/g) (Oscar, 2017a) that was used in a process risk model (PRM) for *Salmonella* and ground chicken (Oscar, 2019) was not used in the present study because it did not adequately simulate thermal death of *Salmonella* as a function of the natural microbial ecology of the ground poultry meat.

2.2.3. Consumption (Dose-Response) Submodel

The disease triangle, dose-response model for *Salmonella* (Oscar, 2019), which is based on human outbreak (Teunis et al., 2010) and feeding trial (Oscar, 2004a) data (Fig. 3), was used to simulate the severity or risk of salmonellosis per serving and overall. In brief, when a person consumes a foodborne pathogen like *Salmonella*, their response falls on a continuum of increasing severity from no response to infection to illness to hospitalization to death (Fig. 1). To simulate this continuum, a disease triangle score (DTS) was calculated:

$$DTS = F + H + P$$

where F was the output (0.1–2.5 AU) of a pert distribution for food consumption behavior, H was the output (0.9–5.0 AU) of a pert distribution for host resistance, and P was the output (0.1–5.0 AU) from the cooking submodel for *Salmonella* serotype and virulence (v). In the baseline scenario A (Table II), the pert distribution for F was 0.1, 1.5, 2.5 and the pert distribution for H was 0.9, 3.5, 5.0. In test scenario H (Table II), the modified pert distribution for F was 0.6, 2.0, 2.5, which simulated more high risk

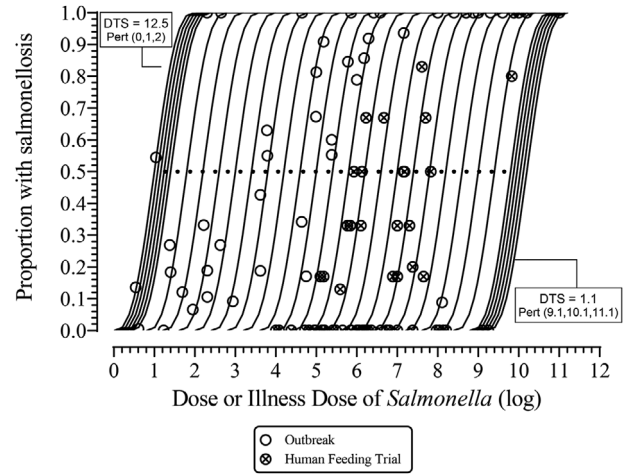


Fig 3. Human outbreak and feeding trial data used to model dose-response as a function of disease triangle score (DTS) from 12.5 to 1.1 in 0.1 increments. Each DTS corresponds to a different pert distribution or dose-response curve.

food consumption behaviors like consuming antacids (Oscar, 2017b; Smith, 2003). In test scenario I (Table II), the modified pert distribution for H was 1.9, 4.5, 5.0, which simulated more high risk consumers like immunocompromised individuals.

The DTS was used to calculate a pert distribution for log illness dose (ID):

$$\text{minimum ID} = 0 + ((12.5 - DTS) \times 0.8)$$

$$\text{most likely ID} = 1 + ((12.5 - DTS) \times 0.8)$$

$$\text{maximum ID} = 2 + ((12.5 - DTS) \times 0.8)$$

which resulted (Fig. 3) in one of 115 possible pert distributions for illness dose (log) that ranged from (0,1,2) for a DTS of 12.5 (highest risk) to (9.1,10.1,11.1) for a DTS of 1.1 (lowest risk). The severity of salmonellosis (SS) in AU per serving was calculated as follows (Oscar, 1998):

$$SS = 0 \quad \text{if } N = 0$$

$$SS = N/10^{\text{ID}} \quad \text{if } N > 0$$

where N was the number of *Salmonella* on the serving after cooking or at consumption and 10^{ID} was the antilog of the log illness dose. The total severity of salmonellosis in AU per lot (907 kg) was calculated as the sum of SS for all servings simulated.

It is possible to use the current process risk model to predict the number of infections (<1), illnesses (≥ 1 to <10), severe illnesses or hospitalizations (≥ 10 to <100), and deaths (≥ 100) where the numbers in parentheses are the ratio of dose consumed (number of *Salmonella*) to illness dose (number of *Salmonella* that cause an illness). In addition, the model could be used to predict the probability of an outbreak (two or more cases of illness per lot) by determining the proportion of replicate simulations of a scenario that predicted two or more cases of salmonellosis. However, these approaches were not included in this study because the poultry industry might not want to use a model that predicts health outcomes.

When a person consumes a pathogen dose (≥ 0 log) with their food, their peak response falls on a continuum from no infection to infection to illness to death (Fig. 1). For modeling purposes, in a human feeding trial (McCullough & Eisele, 1951a, 1951b, 1951c, 1951d), the individual dose-response is first classified as no illness or illness based on a set of criteria. Then the proportion of subjects with illness in each dose group is calculated and graphed as a function of dose. These data are then fitted to a dose-response model (Latimer, Jaykus, Morales, Cowen, & Crawford-Brown, 2001; Oscar, 2004a) that predicts the proportion of ill subjects as a function of dose. Thus, the individual dose-response is a binary event (illness or no illness), whereas the population dose-response curve is the cumulative distribution of these binary events. Therefore, when a subject is ill from consuming a dose of pathogen, the dose consumed was equal to or greater than the number of pathogens needed to cause an illness for that pathogen $\times$ food $\times$ host interaction and when a portion of the population is ill from exposure to a pathogen, it means that the number of pathogens consumed equaled or exceeded the dose of pathogen needed for those pathogen $\times$ food $\times$ host interactions to cause an illness. Thus, it is valid to simulate the pathogen $\times$ food $\times$ host interaction as a binary event by randomly assigning an illness dose (threshold dose) to each serving simulated.

2.3. Scenario Analysis

A scenario is a unique set of inputs or input distributions in a process risk model. Changing one input or input distribution in the baseline model creates a new scenario. For example, increasing the most likely value of the pert distribution for *Salmonella* number of ground turkey (25 g) from

0.185 log to 0.685 log would create a new scenario. Twelve scenarios were simulated (Table II) in the present study to evaluate effects of model variables on risk of salmonellosis; they were: (A) baseline, (B) *Salmonella* prevalence, (C) *Salmonella* number, (D) *Salmonella* virulence, (E) serving size, (F) serving size distribution, (G) total bacterial load, (H) incidence of undercooking, (I) extent of undercooking, (J) food consumption behavior, (K) host resistance, and (L) ground chicken. In each scenario, a single input distribution (scenarios B to K) or multiple input distributions (scenario L) in the baseline scenario was(were) altered to create the test scenarios that were used to determine whether the model variable being evaluated was an important risk factor for salmonellosis. In addition, scenario L was simulated, as explained in more detail later, to test the null hypothesis that *Salmonella* prevalence at meal preparation is a good indicator of poultry food safety. The location of the modified input distribution(s) within the process risk model is(are) shown in Fig. 1 for each scenario.

In QMRA, a baseline scenario is established using published data, unpublished data, surrogate data, and expert opinion (Cassin, Lammerding, Todd, Ross, & McColl, 1998; Casulli et al., 2019; Oscar, 2004b). Risk factors in the baseline scenario are represented by probability distributions because they are variable and uncertain. “What if” scenarios supported or not supported by data are simulated to determine effects and potential effects, respectively, of interventions on risk. Scientific surveys of hazards in food (Bailey et al., 2001; Fearnley, Raupach, Lagala, & Cameron, 2011; Habib, Coles, Fallows, & Goodchild, 2019; Madden, Moran, Scates, McBride, & Kelly, 2011; Mazengia et al., 2014; Surkiewicz, Johnston, Moran, & Krumm, 1969; Waldroup, Rathgeber, & Forsythe, 1992; Yang et al., 2017) and consumer surveys (Evans, 1992; James, Evans, & James, 2008; Jay, Comar, & Govenlock, 1999; Kennedy et al., 2005; Kosa, Cates, Bradley, Chambers, & Godwin, 2015) of food handling practices indicate that probability distributions for risk factors (temperature abuse, undercooking, cross-contamination, host resistance) differ among distribution channels and as a function time (e.g., season) due to cultural, climatic, and human diversity. Thus, it is appropriate to simulate differences in risk factors among distribution channels when evaluating effects of performance standards on food safety. To the best of my knowledge there are no studies supporting the notion that risk factors are the same among all distribution channels.

2.4. Model Simulation

The model was simulated with @Risk settings of Latin Hypercube sampling, Mersenne Twister, and 50 simulations (i.e., replicate simulations) per scenario. A different seed was used to initiate each simulation and produced a unique outcome of the model scenario being simulated. In addition, a different seed was used for each replicate simulation to characterize variability and uncertainty of model outputs. Iterations or servings simulated depended on lot size and mean serving size. Lot size was fixed at 2,000 pounds or 907 kg per the ground turkey industry standard (Sampedro et al., 2018). Mean serving size was used to determine the number of servings to simulate when a distribution of serving sizes was simulated. Ten scenarios (A to D, and G to L) involved 25 g servings or 36,280 servings per 907 kg lot and two scenarios (E and F) involved 125 g servings or 7,256 servings per 907 kg lot.

2.5. Data Analysis

The outputs of the model were probability distributions. A total of 9,000 probability distributions for model outputs were generated (i.e., 50 simulations $\times$ 15 model outputs $\times$ 12 scenarios). This resulted in 282,984,000 model outputs. To summarize this vast amount of information, summary statistics from the probability distributions for model outputs and scenarios were used to calculate the 15 dependent variables for statistical analysis. Dependent variables evaluated were *Salmonella* prevalence (%), *Salmonella* virulence (mean AU), and *Salmonella* number (mean, maximum, and total) at meal preparation and after cooking, and severity of salmonellosis (mean, maximum, total AU), illness dose (mean), and exposures (total) at consumption.

2.6. Statistical Analysis

Output distributions for model outputs (dependent variables) for scenarios A to L were evaluated for normality using the D'Agostino-Pearson omnibus K2 test at $p \leq 0.05$. Output distributions that passed ($p \leq 0.05$) the test for normality were analyzed by one-way analysis of variance (ANOVA). When ANOVA was significant ($p \leq 0.05$), means of test scenarios B to L were compared to mean of the baseline scenario A using Dunnett's multiple comparison test corrected for multiple comparisons at $p \leq 0.05$. Output distributions that failed ($p > 0.05$) the

D'Agostino-Pearson omnibus K2 test for normality were analyzed by a nonparametric method (Kruskal-Wallis test). When the Kruskal-Wallis test was significant ($p \leq 0.05$), medians of test scenarios B to L were compared to the median of the baseline scenario A using Dunn's multiple comparison test corrected for multiple comparisons at $p \leq 0.05$. All statistical analyses were performed in Prism.

3. RESULTS

3.1. Baseline Scenario A for Ground Turkey

Baseline scenario A simulated risk of salmonellosis from 25 g servings of ground turkey. The input settings (Fig. 2 and Table II) were based on published studies and new data collected by WSE-qPCR, cultural isolation, and serotyping (Table I). *Salmonella* prevalence at meal preparation was 26% per 25 g, whereas median *Salmonella* number (log/25 g) was 0.18 (range: 0–1.6). *Salmonella* virulence ranged from 0.8 AU for serotype Kentucky (low) to 4.8 AU for serotype Typhimurium (high). These data were used to simulate *Salmonella* contamination (prevalence, number, serotype, virulence) of ground turkey as a function of serving size (25–325 g). Simulation results were compared to test scenarios B to L to evaluate effects of model variables on risk of salmonellosis.

3.2. Test Scenario B for *Salmonella* Prevalence

An increase of *Salmonella* prevalence at meal preparation from 26.0% to 41.3% per 25 g (Fig. 4(a)) had the following significant effects on model outputs: (1) an increase of *Salmonella* prevalence after cooking from 0.31% to 0.50% per 25 g (Fig. 4(b)), (2) an increase of *Salmonella* exposures at consumption from 112 to 181 per lot (Fig. 4(c)), (3) an increase of *Salmonella* number at meal preparation from 28,255 to 44,906 per lot (Fig. 5(a)), (4) an increase of *Salmonella* number after cooking from 336 to 539 per lot (Fig. 5(b)), and (5) an increase of total severity of salmonellosis from 0.38 to 0.56 AU per lot (Fig. 5(c)). Thus, *Salmonella* prevalence at meal preparation was an important risk factor for salmonellosis.

3.3. Test Scenario C for *Salmonella* Number

An increase of *Salmonella* number at meal preparation from 28,255 to 91,659 per lot (Fig. 5(a))

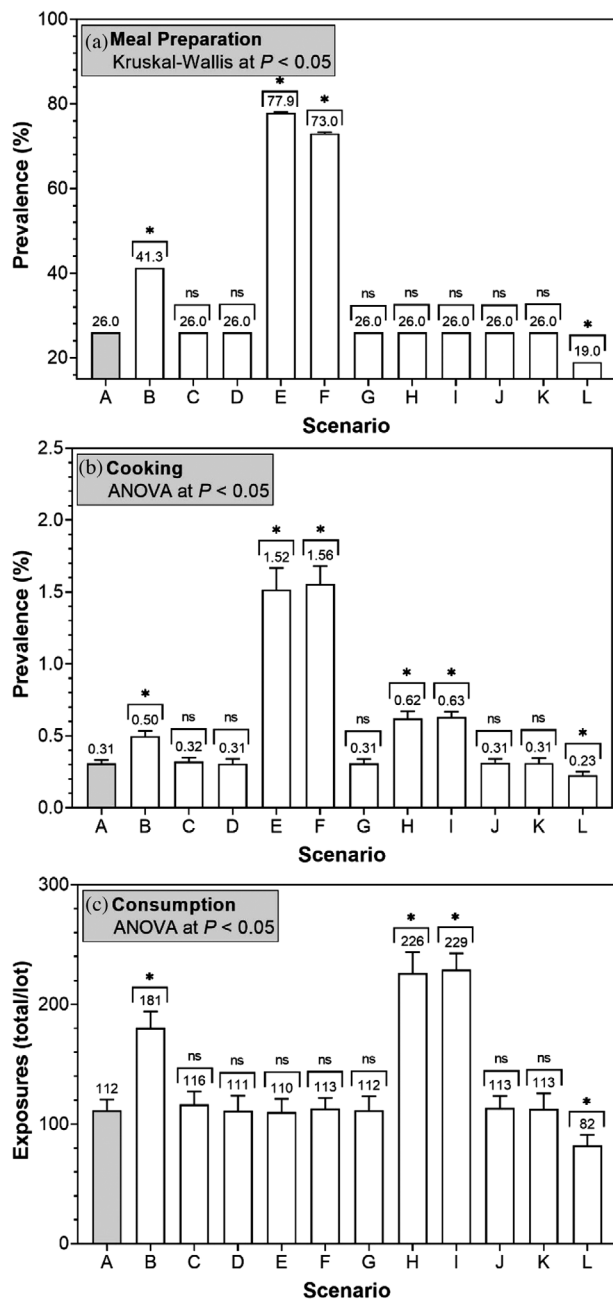


Fig 4. Simulation results for the baseline scenario A and test scenarios B to L: (a) *Salmonella* prevalence at meal preparation, (b) *Salmonella* prevalence after cooking, and (c) *Salmonella* exposures at consumption. Bars (error bars) are median (95% confidence intervals) values for Kruskal-Wallis test or mean (standard deviation) values for one-way analysis of variance (ANOVA). Scenarios B to L were compared to scenario A using Dunn's multiple comparison test for Kruskal-Wallis and Dunnett's multiple comparison test for ANOVA where a superscript above a bar of ns, nonsignificant ($p > 0.05$); *, significant ($p \leq 0.05$).

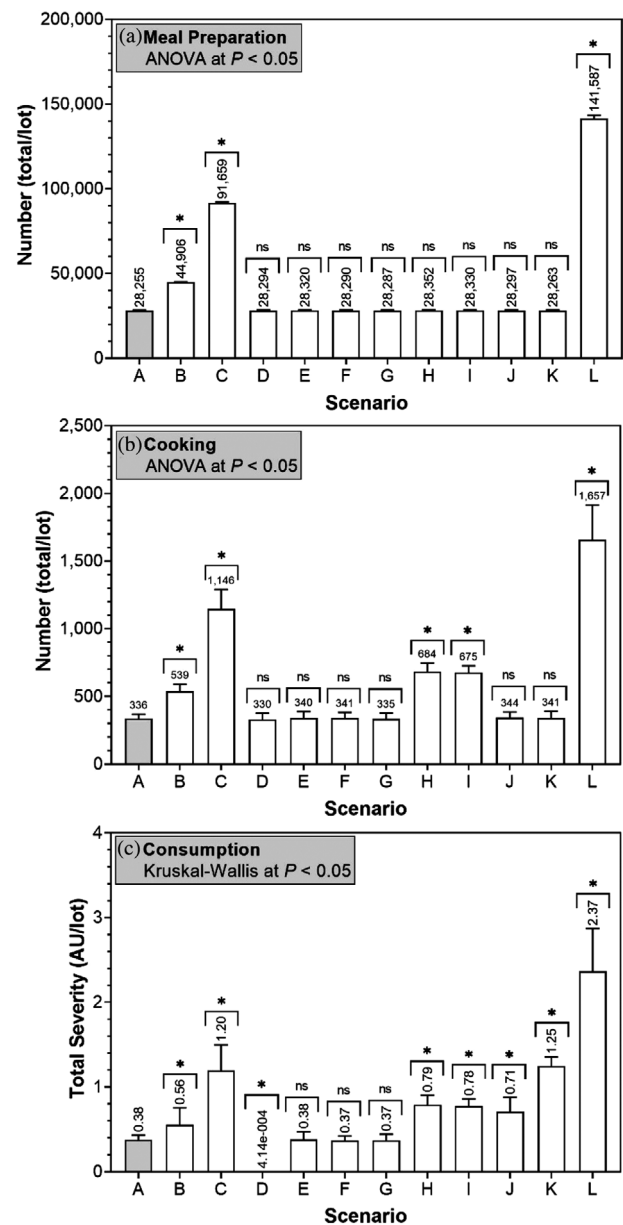


Fig 5. Simulation results for the baseline scenario A and test scenarios B to L: (a) *Salmonella* number at meal preparation, (b) *Salmonella* number after cooking, and (c) total severity of salmonellosis at consumption. Bars (error bars) are median (95% confidence intervals) for Kruskal-Wallis test or mean (standard deviation) for one-way analysis of variance (ANOVA). Scenarios B to L were compared to scenario A using Dunn's multiple comparison test for median values (Kruskal-Wallis) or Dunnett's multiple comparison test for mean values (ANOVA) where a bar superscript of ns, nonsignificant ($p > 0.05$); *, significant ($p \leq 0.05$).

had the following significant effects on model outputs: (1) an increase of *Salmonella* number after cooking from 336 to 1,146 per lot (Fig. 5(b)), and (2) an increase of total severity of salmonellosis from 0.38 to 1.20 AU per lot (Fig. 5(c)). Thus, *Salmonella* number at meal preparation was an important risk factor for salmonellosis.

3.4. Test Scenario D for *Salmonella* Virulence

A decrease of *Salmonella* virulence at meal preparation from 3.94 to 0.80 AU per serving (Fig. 6(a)) had the following significant effects on model outputs: (1) a decrease of *Salmonella* virulence after cooking from 3.94 to 0.80 AU per serving (Fig. 6(b)), (2) an increase of illness dose at consumption from 4.05 to 6.55 log per serving (Fig. 6(c)), and (3) a decrease of total severity of salmonellosis from 0.38 to 0.000414 AU per lot (Fig. 5(c)). Thus, *Salmonella* virulence at meal preparation was an important risk factor for salmonellosis.

3.5. Test Scenario E for Serving Size

An increase of serving size from 25 to 125 g had the following significant effects on model outputs: (1) an increase of *Salmonella* prevalence at meal preparation from 26.0% per 25 g to 77.9% per 125 g (Fig. 4(a)), and (2) an increase of *Salmonella* prevalence after cooking from 0.31% per 25 g to 1.52% per 125 g (Fig. 4(b)). However, total severity of salmonellosis did not change (Fig. 5(c)). Thus, serving size was not a significant risk factor for salmonellosis.

3.6. Test Scenario F for Serving Size Distribution

An increase of serving size distribution from 25 to 125 g (mean) had the following significant effects on model outputs: (1) an increase of *Salmonella* prevalence at meal preparation from 26.0% per 25 g to 73.0% per 125 g (Fig. 4(a)), and (2) an increase of *Salmonella* prevalence after cooking from 0.31% per 25 g to 1.56% per 125 g (Fig. 4(b)). However, total severity of salmonellosis did not change (Fig. 5(c)). Thus, serving size distribution was not a significant risk factor for salmonellosis.

3.7. Test Scenario G for Total Bacterial Load

A decrease of total bacterial load by one log per 25 g had no significant effects on any model outputs

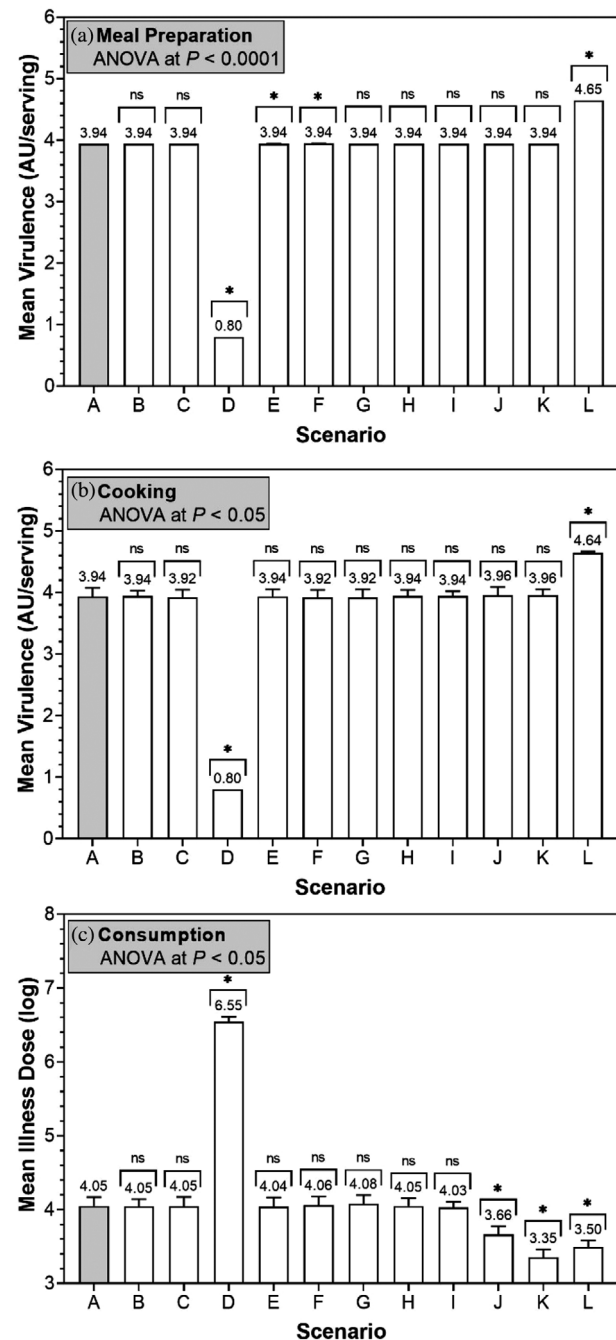


Fig 6. Simulation results for the baseline scenario A and test scenarios B to L: (a) *Salmonella* virulence at meal preparation, (b) *Salmonella* virulence after cooking, and (c) illness dose at consumption. Bars (error bars) are mean (standard deviation). Results were analyzed by one-way analysis of variance (ANOVA). Scenarios B to L were compared to scenario A using Dunnett's multiple comparison test where a bar superscript of ns, nonsignificant ($p > 0.05$); *, significant ($p \leq 0.05$).

(Figs. 4–6). Thus, total bacterial load was not a significant risk factor for salmonellosis.

3.8. Test Scenario H for Incidence of Undercooking

An increase of undercooking from 20% to 40% per 25 g had the following significant effects on model outputs: (1) an increase of *Salmonella* prevalence after cooking from 0.31% to 0.62% per 25 g (Fig. 4(b)), (2) an increase of *Salmonella* exposures from 112 to 226 per lot (Fig. 4(c)), (3) an increase of *Salmonella* number after cooking from 336 to 684 per lot (Fig. 5(b)), and (4) an increase of total severity of salmonellosis from 0.38 to 0.79 AU per lot (Fig. 5(c)). Thus, incidence of undercooking was a significant risk factor for salmonellosis.

3.9. Test Scenario I for Extent of Undercooking

An increase of extent of undercooking had the following significant effects on model outputs: (1) an increase of *Salmonella* prevalence after cooking from 0.31% to 0.63% per 25 g (Fig. 4(b)), (2) an increase of *Salmonella* exposures at consumption from 112 to 229 per lot (Fig. 4(c)), (3) an increase of *Salmonella* number after cooking from 336 to 675 per lot (Fig. 5(b)), and (4) an increase of total severity of salmonellosis from 0.38 to 0.78 AU per lot (Fig. 5(c)). Thus, extent of undercooking was a significant risk factor for salmonellosis.

3.10. Test Scenario J for Food Consumption Behavior

An increase in the proportion of high risk food consumption behaviors had the following significant effects on model outputs: (1) a decrease of illness dose from 4.05 to 3.66 log per serving (Fig. 6(c)), and (2) an increase of total severity of salmonellosis from 0.38 to 0.71 AU per lot (Fig. 5(c)). Thus, food consumption behavior was a significant risk factor for salmonellosis.

3.11. Test Scenario K for Host Resistance

An increase in the proportion of high risk consumers had the following significant effects on model outputs: (1) a decrease of illness dose from 4.05 to 3.35 log per serving (Fig. 6(c)), and (2) an increase in total severity of salmonellosis from 0.38 to 1.25 AU

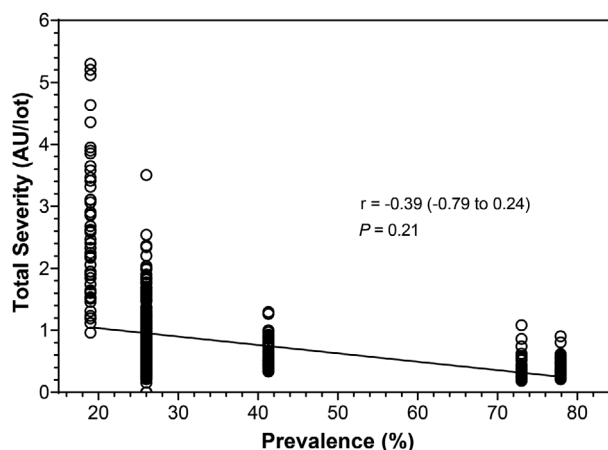


Fig 7. Correlation (r) between *Salmonella* prevalence at meal preparation and total severity of salmonellosis per lot for scenarios A to L. Values in parentheses are the 95% confidence interval for r .

per lot (Fig. 5(c)). Thus, host resistance was a significant risk factor for salmonellosis.

3.12. Test Scenario L for Ground Chicken

Compared to ground turkey at meal preparation, ground chicken significantly differed as follows: (1) lower *Salmonella* prevalence at 19% per 25 g (Fig. 4(a)), (2) higher *Salmonella* number at 141,587 per lot (Fig. 5(a)), and (3) higher *Salmonella* virulence at 4.65 AU per serving (Fig. 6(a)). These differences resulted in the following significant effects on model outputs: (1) lower *Salmonella* prevalence after cooking at 0.23% per 25 g (Fig. 4(b)), (2) lower *Salmonella* exposures at consumption at 82 per lot (Fig. 4(c)), (3) higher *Salmonella* virulence after cooking at 4.65 AU per serving (Fig. 6(b)), (4) higher *Salmonella* number after cooking at 1,657 per lot (Fig. 5(b)), (5) lower illness dose at consumption at 3.50 log per serving, and (6) higher total severity of salmonellosis at 2.37 AU per lot. Thus, ground chicken had a higher risk of salmonellosis than ground turkey even though it had a lower *Salmonella* prevalence at meal preparation.

3.13. Test Scenarios A to L

Total severity of salmonellosis per lot of ground turkey was graphed as a function of *Salmonella* prevalence at meal preparation to evaluate the correlation between these model outputs (Fig. 7). The results indicated that risk of salmonellosis was not

correlated ($r = -0.39$; $p = 0.21$) with *Salmonella* prevalence at meal preparation.

4. DISCUSSION

Salmonella contamination of ground turkey is an important public health issue because it has resulted in recent outbreaks of salmonellosis and product recalls. For example, an outbreak of salmonellosis from ground turkey occurred between December 19, 2018 and March 16, 2019 (www.cdc.gov/salmonella/schwarzengrund-03-19/). The outbreak involved three states, seven cases, one hospitalization, and no deaths. The victims (86% female) ranged in age from 1 to 71 years with a median age of 49 years. The outbreak strain (*Salmonella* Schwarzengrund) was identified by whole-genome sequencing of clinical and food isolates and was resistant to multiple antibiotics. The company issued a recall of 78,164 pounds of product on March 13, 2019. The ground turkey sampled in the present study, which was not from the implicated company, was found to harbor seven different serotypes of *Salmonella* but not Schwarzengrund.

Another outbreak of salmonellosis from ground turkey occurred between February 27, 2011 and September 13, 2011 (www.cdc.gov/salmonella/ground-turkey-11-10-2011.html). This outbreak involved 36 states, 136 cases, 37 hospitalizations, and 1 death. The victims (55% male) ranged in age from 1 to 90 years with a median age of 23 years. The outbreak strain (*Salmonella* Heidelberg) was resistant to multiple antibiotics and was subtyped by pulsed field gel electrophoresis. The implicated company issued product recalls on August 3, 2011 (36 million pounds) and on September 11, 2011 (185,000 pounds). The serotype (Heidelberg) responsible for this outbreak was not isolated in the present study, which tested ground turkey that was not from the company implicated in this outbreak.

A third outbreak involving *Salmonella* Hadar and ground turkey occurred between December 27, 2010 and March 24, 2011 (www.cdc.gov/salmonella/2011/turkey-burger-4-4-2011.html). This outbreak involved 10 states, 12 cases, 3 hospitalizations, and 0 deaths. The outbreak strain was resistant to multiple antibiotics. The age of victims (63% female) ranged from 1 to 86 years with a median age of 26 years. The company responsible for the outbreak issued a product recall on April 1, 2011 of 54,960 pounds. Serotype Hadar was isolated from ground turkey in the present study. However, it was not isolated from the company implicated in this outbreak.

These outbreaks, which occurred from ground turkey that passed inspection, show that the current approach to identifying unsafe lots of food at the processing plant using *Salmonella* prevalence as an indicator of food safety is not completely successful. They also show that types of *Salmonella* and consumer demographics differ among outbreaks. In the current study, it was shown that in addition to *Salmonella* prevalence, *Salmonella* virulence and number, incidence and extent of undercooking, food consumption behavior, and host resistance were important risk factors for salmonellosis from ground turkey. Thus, it was concluded that *Salmonella* prevalence alone was not a good indicator of poultry food safety because there were other risk factors that needed to be considered when evaluating safety of individual lots of ground turkey before they were shipped to consumers. This was further demonstrated in the present study by comparing risk of salmonellosis from ground turkey and ground chicken. Here, even though ground chicken had a lower *Salmonella* prevalence (19%/25g) than ground turkey (26%/25g) at meal preparation, it posed a higher risk of salmonellosis because it was contaminated with higher numbers of more virulent serotypes of *Salmonella* than ground turkey.

The current performance standard for ground turkey (Anonymous, 2016) is no more than 13.5% positive samples over a 52-week moving window period. The ground turkey examined in the present study was obtained at retail from December 26, 2017 to April 23, 2018. During this 17-week period, *Salmonella* prevalence at meal preparation following proper storage (4 °C for 6 hours) was 26% (26/100) per 25 g. It can reasonably be assumed that the ground turkey had passed inspection since it was obtained at a retail outlet. Thus, it should have had a *Salmonella* prevalence <13.5%. However, Simmons, Fletcher, Berrang, and Cason (2003) found that when the number of *Salmonella* on a chicken carcass is low, the whole rinse and aliquot method used by the chicken industry underestimates *Salmonella* prevalence when compared to whole carcass enrichment (WCE) because its lower limit of detection (LLD) is higher than that of WCE, which has an LLD of one cell of *Salmonella* per carcass. In the present study, a WSE method was used that has an LLD of one cell of *Salmonella* per sample (Oscar, 2016). Thus, the use of a more sensitive detection method (i.e., WSE) may explain the higher than expected *Salmonella* prevalence observed in the present study.

Cui, Guran, Harrison, Hofacre, and Alali (2015) examined ground turkey for *Salmonella* prevalence and number. They reported a *Salmonella* prevalence of 14.5% and a mean number (95% confidence interval) of 1.9 (1.1–2.6) log per 25 g. In the present study, *Salmonella* prevalence of ground turkey was 26% per 25 g, whereas *Salmonella* number ranged from 0 to 1.6 log per 25 g with a median of 0.18 log per 25 g. The lower *Salmonella* prevalence and higher mean *Salmonella* number reported by Cui et al. (2015) could be due to a different source of ground turkey and/or differences in data collection methods. Nonetheless, both studies indicate that ground turkey is contaminated with low-to-moderate levels of *Salmonella*.

Sampedro et al. (2018) conducted a risk assessment for *Salmonella* and ground turkey. They summarized results from testing performed by the U. S. Department of Agriculture, Food Safety and Inspection Service for the years 2010–2016. They reported an overall *Salmonella* prevalence of 11.9% and a mean *Salmonella* number of 0.16 log per g, which is equivalent to 1.56 log per 25 g, if one assumes a uniform distribution of the pathogen. This result agrees with that of Cui et al. (2015) but is higher than observed in the present study. Methods with LLD > 1 cell per sample will underestimate *Salmonella* prevalence and overestimate *Salmonella* number by excluding samples below the LLD from calculation of the mean. This may explain the difference in results from the present study and those of Sampedro et al. (2018) and Cui et al. (2015).

In the risk assessment model of Sampedro et al. (2018) sensitivity analysis was used to identify model inputs that had the most influence on risk of salmonellosis (i.e., model output). *Salmonella* number was identified as one of the most influential inputs. Thus, Sampedro et al. (2018) proposed that the performance standard for ground turkey could be improved by basing it on both *Salmonella* prevalence and *Salmonella* number. In the present study, the scenario analysis showed that *Salmonella* prevalence and *Salmonella* number at meal preparation were important risk factors for salmonellosis from ground turkey. Thus, results of the current study support the proposal of Sampedro et al. (2018) that the performance standard for ground turkey could be improved by basing it on both *Salmonella* prevalence and *Salmonella* number.

In the risk assessment model of Sampedro et al. (2018) for *Salmonella* and ground turkey, two dose-response models were used; one for low-virulent

strains and one for high-virulent strains. Similarly, Oscar (2017b) considered low-low, low, normal, high, and high-high virulent serotypes of *Salmonella* in a dose-response model for *Salmonella* and chicken parts. Subsequently, Oscar (2019) expanded this dose-response model to accommodate composite virulence from servings of ground chicken contaminated with more than one serotype of *Salmonella*. In the present study, a further refinement of the Oscar (2019) model was made. Namely, epidemiological data were used to more precisely define differences in virulence among serotypes of *Salmonella* in the dose-response model of Oscar (2019). Because the scenario analysis in the present study indicated that *Salmonella* virulence was an important risk factor for salmonellosis from ground turkey and based on previous risk assessment studies (Oscar, 2004a, 2017b, 2019; Sampedro et al., 2018) that consider *Salmonella* virulence in dose-response models, it is proposed here that the performance standard for ground turkey could be further improved by basing it not only on *Salmonella* prevalence and number, but also on *Salmonella* virulence.

Risk assessments for poultry (Ebel & Williams, 2015, 2019) show that a reduction in *Salmonella* prevalence at the processing plant results in a similar reduction in salmonellosis when poultry meat is shipped to the same distribution channel and consumer population or when all other risk factors are held constant. Similarly, in the present study, results from scenarios A and B showed that a change in *Salmonella* prevalence at meal preparation resulted in a similar change in risk of salmonellosis when ground turkey was shipped to the same distribution channel and consumer population or when all other risk factors were held constant. However, when ground turkey was shipped to different distribution channels and consumer populations or when other risk factors were not held constant, risk of salmonellosis was not correlated with *Salmonella* prevalence at meal preparation. Thus, *Salmonella* prevalence alone was not a good indicator of poultry food safety when multiple risk factors were considered. This conclusion is in agreement with other studies (Oscar, 1998, 2004b, 2011b, 2012a, 2012b, 2016, 2017b, 2018b, 2019; Sampedro et al., 2018).

Salmonella are a minority member of the native microflora of ground turkey. In the present study, the number of *Salmonella* in ground turkey ranged from 1 to 40 cells per 25 g, whereas native microflora ranged from 25,000 to 250,000,000 per 25 g. Traditional thermal inactivation studies (Gurtler, Juneja,

Jones, & Purohit, 2019; Juneja et al., 2016) create an artificial ecology by introducing a large and nonecological number of *Salmonella*. The result is the appearance of nonrandom thermal inactivation of the pathogen. In nature, where *Salmonella* is a minority member of the native microflora of food, thermal inactivation is a random event because it depends on the random location of *Salmonella* in the food matrix relative to the heat source. Thus, the new method (thermal line of death model) used in the present study more accurately simulated death and survival of *Salmonella* during cooking of ground turkey because it considered the natural ecology of *Salmonella* in ground turkey and the random location of *Salmonella* in ground turkey relative to the heat source.

Another important consideration for risk assessment and food safety is serving size. In the present study (results not shown) and in a previous study for *Salmonella* and ground chicken (Oscar, 2019), *Salmonella* prevalence increased in a nonlinear manner as a function of serving size, whereas total severity of salmonellosis per lot was not affected by serving size demonstrating that *Salmonella* prevalence by itself is not a good indicator of food safety. Moreover, these findings indicate that when reporting and discussing results for *Salmonella* prevalence it is important to express prevalence as a function of sample size. In the present study, *Salmonella* prevalence for ground turkey was 26% when the sample size was 25 g and was predicted to be 77.9% when the sample or serving size was 125 g. Moreover, the nonlinear relationship between *Salmonella* prevalence and serving size means that linear extrapolation of *Salmonella* prevalence from one sample size to another is not warranted or valid. In fact, when sample or serving size is large enough, *Salmonella* prevalence will be 100%. Hopefully, in the future, researchers will report results for prevalence of foodborne pathogens as a function of sample size, which will make comparisons among studies more meaningful.

Sampedro et al. (2018) simulated serving size in their risk assessment model for *Salmonella* and ground turkey with a pert distribution (85,113,170 g) based on a personal communication with the turkey industry. The model developed in the present study for ground turkey predicted prevalence, number, and serotype and virulence of *Salmonella* as a function of serving size from 25 to 325 g in 25 g increments. Thus, it covers the range of serving sizes of ground turkey that are typically eaten by consumers. Also, as shown in test scenario F, the current model can simulate a

distribution of serving sizes, which is more typical of what occurs in practice when a batch (lot) of ground turkey is shipped to and consumed by consumers.

Sampedro et al. (2018) simulated a lot size of 907 kg in their risk assessment model for *Salmonella* and ground turkey. This was based on a personal communication with the ground turkey industry, which uses 2,000-pound (907 kg) lots in their production scheme. Thus, this is a good lot size to simulate and, therefore, was used in the present study. The current study and a previous study (Oscar, 2019) with ground chicken and the study of Sampedro et al. (2018) are some of the first modeling studies to simulate risk of salmonellosis as a function of lot size. This is important because it represents a future approach that could be used in the food industry to assess the public health impact of different lots of food before they are distributed to consumers. Thus, it has practical application and significance.

Risk managers are often interested in seeing the distribution of risk when making decisions on how best to address and mitigate a risk to public health. In the present study, there were two classes of distributions of risk for each scenario. First, there was a distribution of risk for severity of salmonellosis per serving. There were 50 of these output distributions per scenario or 600 of these distributions in the current study. It was not possible to show all these distributions. Consequently, a second class of output distribution for risk of salmonellosis (i.e., total severity of salmonellosis per lot) was generated using the summary statistics of the first class of output distributions. There were 12 of these output distributions, which were summarized in a simple bar graph (Fig. 5(c)). These output distributions characterized the variability and uncertainty of the risk of salmonellosis within a scenario and allowed an objective evaluation of the effect of model variables on risk of salmonellosis (i.e., food safety) because the results could be subjected to statistical analysis. Thus, by simplifying a large and complex set of data and simulation results into a simple and objective bar graph, risk managers can be provided with an easily understood risk assessment that can help them make important food safety decisions that impact public health.

Total severity of salmonellosis per lot was used as the primary risk metric in the present study because it integrated all model variables into a single measure of food safety for use by risk managers. Moreover, it was the summation of the area under the probability distribution for severity of salmonellosis per serving where most of the values were zero as *Salmonella*

prevalence after cooking ranged from 0.23% per 25 g in test scenario L to 1.56% per 25 g in test scenario F (Fig. 4(b)). According to the D'Agostino-Pearson omnibus K2 test, the distribution for total severity of salmonellosis per lot among simulations ($n = 50$ per scenario) of the model did not follow a normal distribution. Thus, results among scenarios were objectively interpreted and compared for statistical significance ($p \leq 0.05$) using a nonparametric test (Kruskal-Wallis) and multiple comparison test of medians (Dunnett's). This approach to risk assessment provides risk managers with a valid and objective means of confidently and clearly applying simulation results from the model to their food safety decisions aimed at protecting public health. It should also be helpful in communicating the risk management strategy and rationale to stakeholders to generate public support for changes in public health policy.

Total severity of salmonellosis per lot was predicted using a simulation model composed of three unit operations (pathogen events) and submodels: (1) one for *Salmonella* contamination (prevalence, number, serotype, virulence) of ground turkey or ground chicken at meal preparation, (2) one for death and survival of *Salmonella* during cooking of ground turkey or ground chicken, and (3) one for dose-response of consumers to *Salmonella* exposure from cooked ground turkey or ground chicken after consumption. The model was used to make a valid comparison of the safety of two lots of ground poultry meat; one for ground turkey (i.e., the baseline scenario A) and one for ground chicken (i.e., test scenario L). The comparison was valid because data for *Salmonella* contamination (prevalence, number, and serotype) of ground turkey and ground chicken were collected at the same point in the farm-to-table continuum (i.e., meal preparation) using the same methods (i.e., WSE, qPCR, cultural isolation, and serotyping). Because *Salmonella* prevalence was different for ground turkey (26%/25g) and ground chicken (19%/25g), it was possible to test the null hypothesis that *Salmonella* prevalence alone is a good indicator of poultry food safety. However, because total severity of salmonellosis was higher ($p \leq 0.05$) for ground chicken than ground turkey even though ground chicken had a lower prevalence of *Salmonella* than ground turkey at meal preparation, the null hypothesis was rejected and the alternative hypothesis that *Salmonella* prevalence alone is not a good indicator of poultry food safety was accepted. This conclusion was supported by other evidence. Namely, simulation of other scenarios indicated that total severity

of salmonellosis per lot was affected ($p \leq 0.05$) by other model variables or risk factors: (1) *Salmonella* number, (2) *Salmonella* virulence, (3) incidence of undercooking, (4) extent of undercooking, (5) food consumption behavior, and (6) host resistance. In addition, there was no correlation between *Salmonella* prevalence at meal preparation and risk of salmonellosis when ground turkey was shipped to different distribution channels and consumers' populations or when multiple risk factors were considered. Thus, although *Salmonella* prevalence at meal preparation was an important risk factor for salmonellosis, it was not a good indicator of poultry food safety because it was only one of several factors that determine risk of salmonellosis. The same conclusion was reached in previous studies (Oscar, 1998, 2004b, 2011b, 2016, 2017b, 2018b, 2019; Sampredo et al., 2018).

The *Salmonella* contamination data for ground turkey in the present study were collected at meal preparation. Thus, they are a summation of all unit operations and pathogen events that occurred between egg formation in the hen and meal preparation. Growth of *Salmonella* in ground turkey between grinding at the processing plant and meal preparation is accounted for in these data and may explain the samples with the highest numbers of *Salmonella*. However, if the model were applied at the processing plant, which is the intended future application, it would need to be expanded to include unit operations and pathogen events from the processing plant to meal preparation, such as retail transport (growth), retail storage (growth), consumer transport (growth), and consumer storage (growth). Models that predict growth of *Salmonella* in ground turkey or ground chicken at times and temperatures encountered in these unit operations are available (Oscar, 2006, 2009, 2011a) or could be developed. Outputs from these models could be used to predict how *Salmonella* number may change between grinding at the processing plant and meal preparation as was done in a previous study (Oscar, 2004b).

The current model can be improved by including unit operations (pathogen events) for cross-contamination of ready-to-eat (RTE) food (e.g., cooked ground poultry meat or salad fruits and vegetables) with *Salmonella* from raw ground turkey or ground chicken followed by *Salmonella* growth on RTE food during meal preparation. Data for natural cross-contamination of RTE food with *Salmonella* from ground turkey and ground chicken were not collected and are not available in the scientific literature but could be collected in the future using

WSE, qPCR, cultural isolation, and serotyping as was done in a previous study (Oscar, 2017b) with whole chicken parts (i.e., wings, breasts, thighs, and drumsticks) harvested from whole chickens sold in flow-pack wrappers. In addition, existing models for growth of chicken isolates of *Salmonella* on cooked chicken (Oscar, 1999a, 1999b, 2002) or on Roma tomatoes (Oscar, 2018a) or on Romaine lettuce (Oscar, 2020) as a function of times and temperatures encountered during meal preparation can be used to forecast how *Salmonella* number on RTE food may change between cross-contamination and consumption.

Total severity of salmonellosis per lot of ground turkey or ground chicken was not affected ($p > 0.05$) by serving size or serving size distribution in the present study. This result agrees with a previous study (Oscar, 2019) with ground chicken and a slightly different but similar model. Although mean and maximum *Salmonella* number at meal preparation were higher ($p \leq 0.05$) in test scenario E for serving size (125 g) and in test scenario F for serving size distribution (mean of 125 g) than in the baseline scenario A for a serving size of 25 g (results not shown), mean and maximum *Salmonella* number after cooking were the same ($p > 0.05$) for test scenarios E and F and baseline scenario A (results not shown). Thus, cooking, which decreased *Salmonella* prevalence and number and made exposure to *Salmonella* at consumption a rare event, might explain why serving size was not an important risk factor for salmonellosis. Furthermore, total number of *Salmonella* per lot (907 kg) was not affected ($p > 0.05$) by serving size (scenario E) or serving size distribution (scenario F), which makes sense because the total number of *Salmonella* per lot should be the same regardless of how the lot is partitioned. This result agrees with a previous study (Oscar, 2019) with ground chicken that used a similar modeling approach.

In a previous study (Oscar, 1998), whole chickens with lower numbers of *Salmonella* at the processing plant exit posed higher risk of salmonellosis than whole chickens with higher numbers of *Salmonella* at the processing plant exit when they were temperature abused during distribution, undercooked, and consumed by someone from the high risk population. These findings have broader implications for the present approach to poultry food safety. They indicate that the processing plant with the highest prevalence of *Salmonella* on its poultry meat is not necessarily the processing plant that poses the highest risk of salmonellosis. Rather, poultry meat from a pro-

cessing plant with a lower prevalence of *Salmonella* could pose a higher risk of salmonellosis if it has a higher number of *Salmonella* and/or more virulent serotypes of *Salmonella* and/or if the poultry meat is shipped to a distribution channel with a higher incidence and/or extent of temperature abuse and/or a higher incidence and/or extent of undercooking and/or a higher incidence and/or extent of cross-contamination of RTE food and/or a higher proportion of higher risk food consumption behaviors, and/or a higher proportion of high risk consumers. Thus, it is important to consider multiple risk factors when assessing the risk of salmonellosis from individual lots of poultry meat at the processing plant before they are shipped to consumers. These results and conclusions agree with those of the present study indicating that poultry food safety is a function of multiple risk factors and not just one. Thus, rather than use a single risk factor like *Salmonella* prevalence to determine poultry food safety, it would be better to use multiple risk factors to determine poultry food safety. One way to do this is to apply a process risk model, like the one developed in the present study, at the processing plant exit to integrate pathogen (*Salmonella*) contamination (prevalence, number, and serotype and virulence) data and postprocessing risk factors (temperature abuse, undercooking, cross-contamination, food consumption behavior, and host resistance) into an objective decision about the safety of individual lots of food before they are shipped to consumers. Such an approach would improve public health by maximizing food safety and food security by ensuring that unsafe food is not consumed, and by ensuring that safe food is not destroyed.

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REFERENCES

- Akil, L., & Ahmad, H. A. (2019). Quantitative risk assessment model of human salmonellosis resulting from consumption of broiler chicken. *Diseases*, 7(1), 19. <https://doi.org/10.3390/diseases7010019>.
- Al-Sakkaf, A. (2020). Comparison of three modelling approaches to predict the risk of campylobacteriosis in New Zealand. *Microbial Risk Analysis*, 62. <https://doi.org/10.1016/j.mran.2019.06.001>.
- Anonymous. (2013). Outbreak of *Salmonella* Heidelberg infections linked to a single poultry producer - 13 States, 2012–2013. *MMWR Morbidity and Mortality Weekly Report*, 62(27), 553–556.
- Anonymous. (2014). Foodborne diseases active surveillance network (FoodNet): FoodNet surveillance report for 2012 (final report). Atlanta, Georgia: U.S. Department of Health and Human Services, CDC.
- Anonymous. (2016). New performance standards for *Salmonella* and *Campylobacter* in not-ready-to-eat comminuted chicken and turkey products and raw chicken parts and changes to related agency verification procedures: Response to comments and announcement of implementation schedule. Federal Register: U.S. Government.
- Antunes, P., Mourao, J., Campos, J., & Peixe, L. (2016). Salmonellosis: The role of poultry meat. *Clinical Microbiology and Infection*, 22, 110–121.
- Bailey, J. S., Stern, N. J., Fedorka-Cray, P., Craven, S. E., Cox, N. A., Cosby, D. E., ... Musgrove, M. T. (2001). Sources and movement of *Salmonella* through integrated poultry operations: A multistate epidemiological investigation. *Journal of Food Protection*, 64(11), 1690–1697.
- Baranyi, J., Robinson, A., Kaloti, A., & Mackey, B. M. (1995). Predicting growth of *Brochothrix thermosphacta* at changing temperature. *International Journal of Food Microbiology*, 27, 61–75.
- Bermudez-Aguirre, D., & Corradini, M. G. (2012). Inactivation kinetics of *Salmonella* spp. under thermal and emerging treatments: A review. *Food Research International*, 45, 700–712.
- Cassin, M. H., Lammerding, A. M., Todd, E. C. D., Ross, W., & McColl, R. S. (1998). Quantitative risk assessment for *Escherichia coli* O157:H7 in ground beef hamburgers. *International Journal of Food Microbiology*, 41, 21–44.
- Casulli, K. E., Calhoun, S., & Schaffner, D. W. (2019). Modeling the risk of salmonellosis from consumption of peanuts in the United States. *Journal of Food Protection*, 82(4), 579–588.
- Chai, S. J., Cole, D., Nisler, A., & Mahon, B. E. (2017). Poultry: The most common food in outbreaks with known pathogens, United States, 1998–2012. *Epidemiology and Infection*, 145(2), 316–325.
- Cui, Y., Guran, H. S., Harrison, M. A., Hofacre, C. L., & Alali, W. Q. (2015). *Salmonella* levels in turkey neck skins, drumstick bones, and spleens in relation to ground turkey. *Journal of Food Protection*, 78(11), 1945–1953.
- delRio, E., Gonzalez de Caso, B., Prieto, M., Alonso-Calleja, C., & Capita, R. (2008). Effect of poultry decontaminants concentration on growth kinetics for pathogenic and spoilage bacteria. *Food Microbiology*, 25(7), 888–894.
- Dhananjayan, R., Han, I. Y., Acton, J. C., & Dawson, P. L. (2006). Growth depth effects of bacteria in ground turkey meat patties subjected to high carbon dioxide or high oxygen atmospheres. *Poultry Science*, 85(10), 1821–1828.
- Ebel, E. D., & Williams, M. S. (2015). When are qualitative testing results sufficient to predict a reduction in illnesses in a microbiological food safety risk assessment? *Journal of Food Protection*, 78(8), 1451–1460.
- Ebel, E. D., & Williams, M. S. (2020). Assessing the effectiveness of revised performance standards for *Salmonella* contamination of comminuted poultry. *Microbial Risk Analysis*, 14. <https://doi.org/10.1016/j.mran.2019.05.02>.
- Evans, J. (1992). Consumer handling of chilled foods: Perceptions and practice. *International Journal of Refrigeration*, 15(5), 290–298.
- Farakos, S. M. S., Pouillot, R., Davidson, G. R., Johnson, R., Son, I., Anderson, N., & Van Doren, J. M. (2019). A quantitative risk assessment of human salmonellosis from consumption of walnuts in the United States. *Journal of Food Protection*, 82(1), 45–57.
- Fearnley, E., Raupach, J., Lagala, F., & Cameron, S. (2011). *Salmonella* in chicken meat, eggs and humans: Adelaide, South Australia, 2008. *International Journal of Food Microbiology*, 146(3), 219–227.
- Gurtler, J. B., Juneja, V. K., Jones, D. R., & Purohit, A. (2019). Thermal inactivation kinetics of three heat-resistant *Salmonella* strains in whole liquid egg. *Journal of Food Protection*, 82(9), 1465–1471.
- Habib, I., Coles, J., Fallows, M., & Goodchild, S. (2019). A baseline quantitative survey of *Campylobacter* spp. on retail chicken portions and carcasses in Metropolitan Perth, Western Australia. *Foodborne Pathogens and Disease*, 16(3), 180–186.
- Jackson, B. R., Griffin, P. M., Cole, D., Walsh, K. A., & Chai, S. J. (2013). Outbreak-associated *Salmonella enterica* serotypes and food commodities, United States, 1998–2008. *Emerging and Infectious Disease*, 19(8), 1239–1244.
- James, S. J., Evans, J., & James, C. (2008). A review of the performance of domestic refrigerators. *Journal of Food Engineering*, 87, 2–10.
- Jay, L. S., Comar, D., & Govenlock, L. D. (1999). A national Australian food safety telephone survey. *Journal of Food Protection*, 62, 921–928.
- Juneja, V. K., Valenzuela-Melendres, M., Heperkan, D., Bautista, D., Anderson, D., Hwang, C. A., ... Torrentera-Olivera, N. (2016). Development of a predictive model for *Salmonella* spp. reduction in meat jerky product with temperature, potassium sorbate, pH, and water activity as controlling factors. *International Journal of Food Microbiology*, 236, 1–8.
- Kennedy, J., Jackson, V., Blair, I. S., McDowell, D. A., Cowan, C., & Bolton, D. J. (2005). Food safety knowledge of consumers and the microbiological and temperature status of their refrigerators. *Journal of Food Protection*, 68(7), 1421–1430.
- Kirk, M., Ford, L., Glass, K., & Hall, G. (2014). Foodborne illness, Australia, circa 2000 and circa 2010. *Emerging and Infectious Disease*, 20(11), 1857–1864.
- Kosa, K. M., Cates, S. C., Bradley, S., Chambers, I. V., & E., & Godwin, S. (2015). Consumer-reported handling of raw poultry products at home: Results from a national survey. *Journal of Food Protection*, 78(1), 180–186.
- Lambertini, E., Ruzante, J., Chew, R., Apodaca, V. L., & Kowalczyk, B. B. (2019). The public health impact of different microbiological criteria approaches for *Salmonella* in chicken parts. *Microbial Risk Analysis*, 12(8), 44–59.
- Latimer, H. K., Jaykus, L. A., Morales, R. A., Cowen, P., & Crawford-Brown, D. (2001). A weighted composite dose-response model for human salmonellosis. *Risk Analysis*, 21(2), 295–305.
- Luber, P. (2009). Cross-contamination versus undercooking of poultry meat or eggs—which risks need to be managed first? *International Journal of Food Microbiology*, 134(1–2), 21–28.
- Madden, R. H., Moran, L., Scates, P., McBride, J., & Kelly, C. (2011). Prevalence of *Campylobacter* and *Salmonella* in raw chicken on retail sale in the republic of Ireland. *Journal of Food Protection*, 74(11), 1912–1916.
- Majowicz, S. E., Musto, J., Scallan, E., Angulo, F. J., Kirk, M., O'Brien, S. J., ... Hoekstra, R. M. (2010). The global burden of nontyphoidal *Salmonella* gastroenteritis. *Clinical Infectious Diseases*, 50(6), 882–889.
- Mazengia, E., Samadpour, M., Hill, H. W., Greeson, K., Tenney, K., Liao, G., ... Meschke, J. S. (2014). Prevalence, concentrations, and antibiotic sensitivities of *Salmonella* serovars in

- poultry from retail establishments in Seattle, Washington. *Journal of Food Protection*, 77(6), 885–893.
- McClure, P. J., Baranyi, J., Boogard, E., Kelly, T. M., & Roberts, T. A. (1993). A predictive model for the combined effect of pH, sodium chloride and storage temperature on the growth of *Brochothrix thermosphacta*. *International Journal of Food Microbiology*, 19, 161–178.
- McCullough, N. B., & Eisele, C. W. (1951a). Experimental human salmonellosis. I. Pathogenicity of strains of *Salmonella meleagridis* and *Salmonella anatum* obtained from spray-dried whole egg. *Journal of Infectious Disease*, 88, 278–289.
- McCullough, N. B., & Eisele, C. W. (1951b). Experimental human salmonellosis. III. Pathogenicity of strains of *Salmonella newport*, *Salmonella derby*, and *Salmonella bareilly* obtained from spray-dried whole egg. *Journal of Infectious Disease*, 89, 209–213.
- McCullough, N. B., & Eisele, C. W. (1951c). Experimental human salmonellosis. IV. Pathogenicity of strains of *Salmonella pullorum* obtained from spray-dried whole egg. *Journal of Infectious Disease*, 89, 259–265.
- McCullough, N. B., & Eisele, C. W. (1951d). Experimental human salmonellosis. II. Immunity studies following experimental illness with *Salmonella meleagridis* and *Salmonella anatum*. *Journal of Immunology*, 66, 595–608.
- Membre, J. M., & Boue, G. (2018). Quantitative microbiological risk assessment in food industry: Theory and practical application. *Food Research International*, 106, 1132–1139.
- Murphy, R. Y., Duncan, L. K., Johnson, E. R., Davis, M. D., & Smith, J. N. (2002). Thermal inactivation D- and z-values of *Salmonella* serotypes and *Listeria innocua* in chicken patties, chicken tenders, franks, beef patties, and blended beef and turkey patties. *Journal of Food Protection*, 65(1), 53–60.
- Oscar, T. P. (1998). The development of a risk assessment model for use in the poultry industry. *Journal of Food Safety*, 18, 371–381.
- Oscar, T. P. (1999a). Response surface models for effects of temperature and previous growth sodium chloride on growth kinetics of *Salmonella* Typhimurium on cooked chicken breast. *Journal of Food Protection*, 62, 1470–1474.
- Oscar, T. P. (1999b). Response surface models for effects of temperature and previous temperature on lag time and specific growth rate of *Salmonella* Typhimurium on cooked ground chicken breast. *Journal of Food Protection*, 62, 1111–1114.
- Oscar, T. P. (2002). Development and validation of a tertiary simulation model for predicting the potential growth of *Salmonella typhimurium* on cooked chicken. *International Journal of Food Microbiology*, 76, 177–190.
- Oscar, T. P. (2004a). Dose-response model for 13 strains of *Salmonella*. *Risk Analysis*, 24, 41–49.
- Oscar, T. P. (2004b). A quantitative risk assessment model for *Salmonella* and whole chickens. *International Journal of Food Microbiology*, 93, 231–247.
- Oscar, T. P. (2004c). Simulation model for enumeration of *Salmonella* on chicken as a function of PCR detection time score and sample size: Implications for risk assessment. *Journal of Food Protection*, 67, 1201–1208.
- Oscar, T. P. (2006). Validation of a tertiary model for predicting variation of *Salmonella* Typhimurium DT104 (ATCC 700408) growth from a low initial density on ground chicken breast meat with a competitive microflora. *Journal of Food Protection*, 69, 2048–2057.
- Oscar, T. P. (2009). Predictive model for survival and growth of *Salmonella* Typhimurium DT104 on chicken skin during temperature abuse. *Journal of Food Protection*, 72, 304–314.
- Oscar, T. P. (2011a). Development and validation of a predictive microbiology model for survival and growth of *Salmonella* on chicken stored at 4 to 12°C. *Journal of Food Protection*, 74(2), 279–284.
- Oscar, T. P. (2011b). Plenary lecture: Innovative modeling approaches applicable to risk assessments. *Food Microbiology*, 28(4), 777–781.
- Oscar, T. P. (2012a). Food risk analysis. In O. A. Oyarzabal & S. Backert (Eds.), *Microbial food safety* (pp. 175–188). New York: Springer.
- Oscar, T. P. (2012b). Innovative modeling approaches for risk assessments in food safety. In X. Yan, V. K. Juneja, P. M. Fratamico, & J. L. Smith (Eds.), *Omics, microbial modeling and technologies for foodborne pathogens* (pp. 389–422). Lancaster: DEStech Publications, Inc.
- Oscar, T. P. (2014). General regression neural network model for behavior of *Salmonella* on chicken meat during cold storage. *Journal of Food Science*, 79, M978–M987.
- Oscar, T. P. (2015). Neural network model for survival and growth of *Salmonella enterica* serotype 8,20::z₆ in ground chicken thigh meat during cold storage: Extrapolation to other serotypes. *Journal of Food Protection*, 78, 1819–1827.
- Oscar, T. P. (2016). Acquisition of data by whole sample enrichment, real-time polymerase chain reaction for development of a process risk model for *Salmonella* and chicken parts. *Journal of Nutrition and Food Sciences*, 6, 538.
- Oscar, T. P. (2017a). Modeling the effect of inoculum size on the thermal inactivation of *Salmonella* Typhimurium to elimination in ground chicken thigh meat. *American Journal of Food Science and Technology*, 5(4), 135–142.
- Oscar, T. P. (2017b). Risk of salmonellosis from chicken parts prepared from whole chickens sold in flow pack wrappers and subjected to temperature abuse. *Journal of Food Protection*, 80(9), 1496–1505.
- Oscar, T. P. (2018a). Development and validation of a neural network model for predicting growth of *Salmonella* Newport on diced Roma tomatoes during simulated salad preparation and serving: Extrapolation to other serotypes. *International Journal of Food Science and Technology*, 53, 1789–1801.
- Oscar, T. P. (2018b). Short-term and long-term effects of pathogen reduction interventions on salmonellosis from whole chickens. *Food Science and Nutrition*, 6, 2515–2522.
- Oscar, T. P. (2019). Process risk model for *Salmonella* and ground chicken. *Journal of Applied Microbiology*, 127(4), 1236–1245.
- Oscar, T. P. (2020). Predictive model for growth of *Salmonella* Newport on Romaine lettuce. *Journal of Food Safety*, 40, e12786.
- Porto-Fett, A. C. S., Shoyer, B. A., Shane, L. E., Osoria, M., Henry, E., Jung, Y., & Luchansky, J. B. (2019). Thermal inactivation of *Salmonella* in pate made from chicken liver. *Journal of Food Protection*, 82(6), 980–987.
- Routh, J. A., Pringle, J., Mohr, M., Bido, S., Arends, K., Adams-Cameron, M., ... Gieraltowski, L. (2015). Nationwide outbreak of multidrug-resistant *Salmonella* Heidelberg infections associated with ground turkey: United States, 2011. *Epidemiology and Infection*, 143(15), 3227–3234.
- Sampedro, F., Wells, S. J., Bender, J. B., & Hedberg, C. W. (2018). Developing a risk management framework to improve public health outcomes by enumerating *Salmonella* in ground turkey. *Epidemiology and Infection*, 147(e69), 1–8.
- Simmons, M., Fletcher, D. L., Berrang, M. E., & Cason, J. A. (2003). Comparison of sampling methods for the detection of *Salmonella* on whole broiler carcasses purchased from retail outlets. *Journal of Food Protection*, 66, 1768–1770.
- Simonsen, B., Bryan, F. L., Christian, J. H. B., Roberts, T. A., Tompkin, R. B., & Siliker, J. H. (1987). Prevention and control of salmonellas through application of HACCP. *International Journal of Food Microbiology*, 4, 227–247.
- Smith, J. L. (2003). The role of gastric acid in preventing foodborne disease and how bacteria overcome acid conditions. *Journal of Food Protection*, 66(7), 1292–1303.

- Surkiewicz, B. F., Johnston, R. W., Moran, A. B., & Krumm, G. W. (1969). A bacteriological survey of chicken eviscerating plants. *Food Technology*, 23(August), 80–85.
- Swart, A. N., van Leusden, F., & Nauta, M. J. (2016). A QMRA model for *Salmonella* in pork products during preparation and consumption. *Risk Analysis*, 36(3), 516–530.
- Teunis, P. F., Kasuga, F., Fazil, A., Ogden, I. D., Rotariu, O., & Strachan, N. J. (2010). Dose-response modeling of *Salmonella* using outbreak data. *International Journal of Food Microbiology*, 144(2), 243–249.
- Uyttendaele, M., Baert, K., Grijspeerdt, K., De Zutter, L., Horion, B., Devlieghere, F., ... Debevere, J. (2009). Comparing the effect of various contamination levels for *Salmonella* in chicken meat preparations on the probability of illness in Belgium. *Journal of Food Protection*, 72(10), 2093–2105.
- Waldroup, A. L., Rathgeber, B. M., & Forsythe, R. H. (1992). Effects of six modifications on the incidence and levels of spoilage and pathogenic organisms on commercially processed postchill broilers. *Journal of Applied Poultry Research*, 1, 226–234.
- Williams, M. S., & Ebel, E. D. (2012). Estimating changes in public health following implementation of hazard analysis and critical control point in the United States broiler slaughter industry. *Foodborne Pathogens and Disease*, 9(1), 59–67.
- Yang, X., Jin, K., Yang, F., Yuan, G., Liu, W., Xiang, L., ... Cao, G. (2017). Nontyphoidal *Salmonella* gastroenteritis in Baoshan, Shanghai, China, 2010 to 2014: An etiological surveillance and case-control study. *Journal of Food Protection*, 80(3), 482–487.