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The public health impact of different microbiological criteria approaches for *Salmonella* in chicken parts

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ABSTRACT

Non-typhoidal *Salmonella* is a significant foodborne pathogen causing over a million illnesses each year in the United States. Poultry is one of the food commodities most frequently associated with *Salmonella* infections. While government, research, and industry efforts have reduced *Salmonella* contamination in poultry to some extent, the incidence of salmonellosis has not changed significantly and is still above the public health goals of Healthy People 2020, and novel and more comprehensive approaches are needed. In this paper, the public health impact of implementing different microbiological criteria (MC) for *Salmonella* in chicken parts was evaluated using a quantitative risk assessment approach. Four hypothetical scenarios, including a no-action baseline and three alternative scenarios, were considered. Scenario 1 modeled a prevalence-based microbiological criterion based on the proportion of positive samples in an establishment, Scenario 2 modeled a microbiological criterion based on the concentration of *Salmonella* in samples, and Scenario 3 modeled a combination of the two. With exception of the baseline, all three scenarios assumed that different interventions would be adopted for non-compliant establishments (Scenario 1) or lots (Scenario 2), with Scenario 3 combining establishment-level and lot-level interventions. The product was assumed to be sold to consumers as raw, and contamination via undercooked product as well as cross contamination in consumer kitchens were considered as potential exposure routes. Risk was characterized by the probability of illness and the preventable fraction of risk, which was calculated for each scenario in comparison with the baseline. Simulation results show that, depending on the parameters of specific sampling strategies, both prevalence-based and concentration-based MC coupled with interventions could significantly lower risk (range of 60–88% in mean preventable fraction of risk). Overall, while the model is preliminary and subject to the stated limitations, it is likely that a combination approach including establishment-level and lot-level interventions would be highly effective in reducing risk and, therefore, benefit public health. The effectiveness of all MC was impacted by several assumptions and model parameters. In particular, the prevalence MC threshold and the concentration reduction associated with the establishment-level intervention impacted the preventable fraction of risk for Scenario 1, and the concentration MC threshold and the variability across lots impacted the risk outcomes for Scenario 2. Overall, high variance in risk outputs was observed, mainly associated with a high variance in concentration inputs. This model provides a risk-based approach to test different MC approaches for chicken parts at both lot and establishment levels, and over a wide range of scenarios of input contamination distributions, interventions, and consumer behaviors. Model estimates, as well as the ability to distinguish between variability and uncertainty, could be improved by additional data on the distribution of *Salmonella* concentrations across and within establishments.

1. Introduction

Nontyphoidal *Salmonella* spp. cause an estimated 1.2 million illnesses, 23,000 hospitalizations, and 450 deaths annually in the United States (Scallan et al., 2011). Food is the main source of *Salmonella* infections, being responsible for about 94% of cases (Scallan et al., 2011). Among the different types of foods most frequently attributed as the source of *Salmonella* outbreaks in the United States, chicken is estimated to be third and accounted for about 10.4% of all *Salmonella* infections in 2013, preceded only by seeded vegetables (16.6%) and

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eggs (11.5%) (IFSAC, 2017). Outbreaks linked to contamination of *Salmonella* in chicken parts and other poultry products have also captured national headlines (CDC, 2011a, 2014, 2018b; Huffstutter, 2014; Grinnell et al., 2013) and propelled the United States Department of Agriculture Food Safety and Inspection Service (USDA-FSIS) to develop and implement, for the first time, pathogen reduction performance standards for *Salmonella* and *Campylobacter* in chicken parts (USDA, 2015a). The chicken industry and large retailers are also taking measures to reduce the contamination of *Salmonella* in chicken parts (Kowalcyk et al., 2018); however, the incidence of salmonellosis (16.0 cases per 100,000) is still well above CDC's Healthy People 2020 objective of 11.4 cases per 100,000 population (US DHHS, 2017), and has not seen substantial reductions in the last two decades (CDC, 2017, 2006, 2005).

The control of *Salmonella* contamination in poultry can be quite complex, e.g. due to the number of serovars and their different ecologies, requiring a multi-hurdle approach that spans from farm to fork (CDC, 2011b). The implementation of microbiological criteria (MC) for a specific food product is one of the many tools used by industry and regulators to verify process control (NRC, 1985). Codex Alimentarius defines MC as the acceptability of a product or a food lot based on the presence or absence of a pathogen, the number of microorganisms including parasites, and/or the quantity of their toxins/metabolites (concentration-based) per unit(s) of mass, volume, area, or lot (CAC, 1997). The Codex further states that MC should be scientifically valid and, whenever possible, based on risk analysis (CAC, 1997). MC play an important role in food safety but, due to limitations associated with microbiological sampling and testing of foods, they cannot by themselves ensure the safety of a product (CAC, 1997). MC need to be a component of a comprehensive preventative food safety system that controls contamination at the source and is based on Pathogen Reduction; Hazard Analysis and Critical Control Point Systems (PR/HACCP) and good manufacturing practices (GMP) (CAC, 1997). Several countries have implemented MC to reduce foodborne illness. For instance, New Zealand recently established MC to help control *Campylobacter* contamination in poultry products, and observed reductions in human illnesses (New Zealand Ministry for Primary Industries, 2017). The European Union has also adopted MC for several pathogens and food products, including poultry (EC, 2007). In the U.S., while not explicitly called MC, *Salmonella* performance standards for selected meat and poultry products based on product testing were established in 1996 as part of the PR/HACCP Final Rule (USDA, 1996). The incidence of *Salmonella* infections reported to public health officials decreased in the years immediately following the implementation of PR/HACCP, but has plateaued in recent years (CDC, 2017, 2006, 2005).

While it is recognized that level of contamination affects risk, MC based on presence/absence of a target organism in a specified percentage of samples (i.e. prevalence-based) are more common than MC that account for the actual number of organisms in a sample (i.e. concentration-based MC) (Swart et al., 2013). Risk assessment studies have been conducted to examine the public health impact of utilizing a MC based on a specified threshold concentration of *Campylobacter* in broiler chicken meat in Europe (Seliwiorstow et al., 2016; Nauta et al., 2015; Swart et al., 2013, 2012), and recently for *Salmonella* in ground turkey in the United States (Sampedro et al., 2018), but to date no comparable risk assessment has been conducted for *Salmonella* in chicken parts under conditions specific to the US.

This study explored the public health impact of specific prevalence- and concentration-based MC for *Salmonella* in raw chicken parts using a probabilistic risk assessment model. Four hypothetical scenarios, including a no-action baseline, were considered. Scenario 1 modeled a prevalence-based MC based on the proportion of positive lots in an establishment; Scenario 2 modeled MC based on the concentration of *Salmonella* in samples, and Scenario 3 modeled a combination of the two. Steps from the end of the production/processing chain to consumer consumption were modeled to quantify the mean probability of illness and preventable fraction for each scenario.

2. Materials and methods

2.1. Modeling framework

The fate of *Salmonella* in raw chicken was modeled from the point of sampling (i.e. before packaging) through consumption, using a probabilistic Quantitative Microbial Risk Assessment (QMRA) approach, to assess the public health impact of implementing prevalence-based and concentration-based MC as a strategy to trigger risk-reduction interventions and help control *Salmonella* in processing. For all alternative scenarios, the baseline scenario (no MC, no intervention) is considered the starting point for assessing compliance. Failure to meet the prevalence-based MC resulted in the implementation of an establishment-level intervention such as an in-depth food safety audit aimed at identifying areas for improvement, increasing compliance and lowering *Salmonella* contamination in subsequent years. Failure to meet the concentration-based MC resulted in the implementation of a lot-level intervention that reduces the risk of *Salmonella* contamination associated with the product lot to zero. Four hypothetical scenarios based on the same initial contamination inputs were considered (Table 1):

- *Baseline Scenario:* No MC is implemented, there is no pre-market intervention to increase compliance or reduce public health risk, and product enters the market without any specific interventions tied to the *Salmonella* contamination status.
- *Alternative Scenario 1:* One sample per lot is analyzed using a specified laboratory protocol to test for *Salmonella* presence or absence. If the proportion of detected samples for the establishment exceeds the prevalence-based MC, the establishment is scored as non-compliant and undergoes an establishment-level intervention (e.g. a food safety audit) that is assumed to lower contamination by a set amount across all product lots in the establishment.
- *Alternative Scenario 2:* One sample per lot is tested to determine *Salmonella* concentration in the sample. If the sample concentration exceeds a set concentration-based MC threshold, the product lot is scored as non-compliant and undergoes a lot-level intervention that completely mitigates the public health risk associated with the non-compliant product lot.
- *Alternative Scenario 3:* One sample per lot is tested to determine *Salmonella* concentration in the sample. If any sample in an establishment exceeds a set concentration threshold, the establishment is scored as non-compliant and undergoes an establishment-level intervention (e.g. a food safety audit) as in Scenario 1, which is assumed to lower contamination by a set amount across all product lots in the establishment. After the establishment-level intervention (e.g. in year 2), one sample per lot is tested according to the same concentration-based MC, and any lots exceeding the concentration threshold are scored as non-compliant and undergo a lot-level intervention as in Scenario 2.

Table 1
Summary of scenarios considered in the study.

Features	Alternative scenarios			
	Baseline	Scenario 1	Scenario 2	Scenario 3
MC approach				
None	X			
Prevalence-based		X		X
Concentration-based			X	X
Intervention				
None	X			
Establishment-level intervention		X		X
Lot-level intervention			X	X

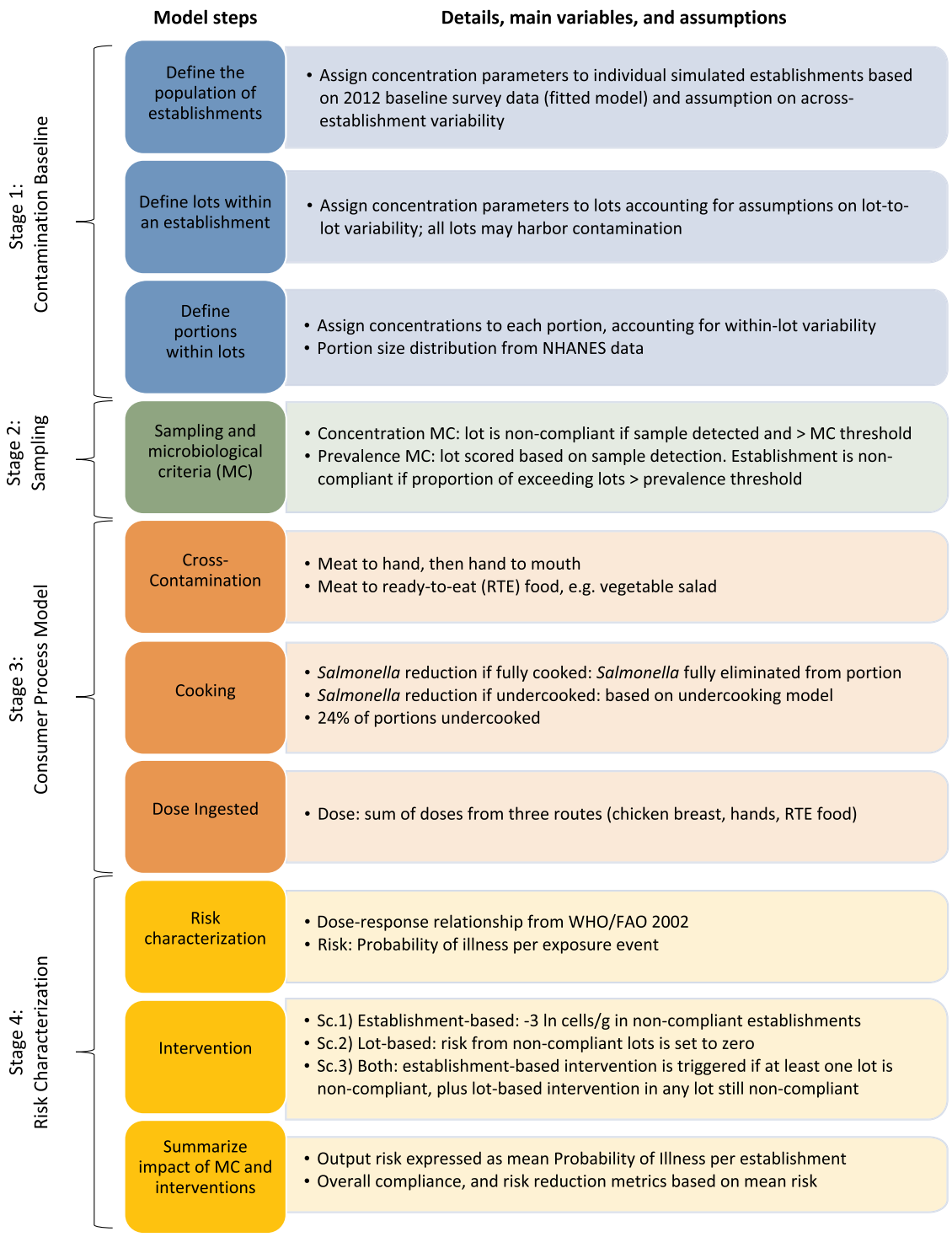

Fig. 1. Sequence of main steps considered in the model.

The no-action baseline scenario was chosen as a clear and neutral starting point, to keep the comparison between baseline and each scenario general and not tied to a specific regulation or practice. Alternative Scenario 1 was chosen as an example of prevalence-based MC at establishment level, which is related to the approach currently adopted in U.S. performance standards for *Salmonella* in multiple chicken and turkey products. Alternative Scenario 2 was chosen as an example of concentration-based MC at lot level, an approach that is implemented in other countries and for other food products. Alternative Scenario 3 was chosen as a two-tiered combination of Scenarios 1 and 2 that, while not currently prescribed by U.S. regulations, provides a stricter multi-barrier approach.

The main steps considered in the simulation are shown in Fig. 1. The product is assumed to be sold to consumers as raw, not-ready-to-eat, and the model focused on the following exposure routes: (1) consumption of cooked or partially cooked chicken meat; (2) cross-contamination of hands and subsequent hand-mouth contact during meal preparation; and (3) cross-contamination with ready-to-eat (RTE) food, e.g. a vegetable salad, during meal preparation (IFSAC, 2017). The inputs and parameters are described in Table 2.

Table 2
Variables and parameters included in the risk model.

Variable	Description	Distribution	Parameter	Data source
Lot weight	Weight of an average lot of chicken parts	Constant	2000 lbs (907.2 Kg)	Industry expert, personal communication
Lots per establishment	Number of 2000-lb product lots produced by an establishment in a simulated year	Constant	500	Approximate estimate based on USDA FSIS data (obtained via FOIA)
Portions per lot	Number of portions in a 2000-lb lot	Constant	Lot weight/mean portion size = 6820 (rounded) where mean portion size ~ 133 g/day	Estimated
Salmonella concentration in chicken breast portions>	Number of cells per unit of product, at the point of sampling before packaging	Lognormal distribution, fitted to 2012 baseline survey data	$\mu_{fitted} = -7.728$ ln cells/g (see Supplementary Material S.3 for details)	USDA (2012) obtained via FOIA
Salmonella concentration parameters at overall establishment-level	Concentration distribution parameters for an establishment	Parameters were set to be compatible with overall concentration and prevalence fitted distributions	$\mu_{establishment}$ = Normal (μ_{fitted}, $\sigma_{across\_establishment}$) where $\sigma_{across\_establishment} = 1$ $\sigma_{within\_establishment}$ = lognormal(1.224,0.30)	Estimated and assumption (see text and Supplementary Material S.3)
Salmonella concentration variability across lots	Proportion of overall concentration variance that is attributed to lot-to-lot variability	Constant, lot-to-lot variance assumed 70% of overall variance in establishment	$\sigma_{across\text{-}lots}^2 = 0.50 \times \sigma_{within\_establishment}^2$	Approach based on Swart et al. (2013)
Salmonella concentration variability within lots	Proportion of overall concentration variance that is attributed to variability within each lot	Constant, within-lot variance assumed 30% of overall variance in establishment	$\sigma_{portions}^2 = 0.50 \times \sigma_{within\_establishment}^2$	Assumption Swart et al. (2013) and EFSA (2011)
Simulated average *Salmonella* concentration in a contaminated lot (step 1)	Concentration parameters of lots, accounting for lot-to-lot variability		$\mu_{lot} \sim$ Normal($\mu_{establishment}$, $\sigma_{across\text{-}lots}$)	Estimated
Simulated *Salmonella* concentration in portions within a lot (step 2)	How each portion within a lot is assigned an average concentration parameter accounting for within-lot variability	Concentration in portions ~ lognormal (μ_{lot}, $\sigma_{portions}$)	Concentration in portions = exp(Normal(μ_{lot}, $\sigma_{portions}$) truncated at 10^{-3} cells/g	Estimated
Number of samples per lot	Number of distinct samples collected per lot	Constant	1	Assumption
Concentration threshold	Lot is "exceeding" if sample is detected and concentration > concentration threshold	Constant	0.1 cells/g (subject to what-if scenario analysis)	Assumption
Establishment prevalence threshold (PMC)	Proportion of "exceeding" lots over one simulated year (500 lots)	Constant	15% (subject to what-if scenario analysis)	Assumption
Portion size (breast)	Amount consumed per exposure event (day) by individuals that consumed chicken breast	Lognormal (μ, σ)	μ: 4.76 ln g σ: 0.55 ln g Truncated at 1st and 99th percentiles (32.53 and 419.17 g)	CDC (2016)
Salmonella contamination in chicken breast portion	Average number of cells present in apportion of chicken breast, when reaching the consumer's home	Numeric value (average number of cells)	Contamination on portion = Concentration in portion (cells/g) × Portion Size (g)	Estimated
Cross-contamination from meat to hands	Probability of transfer meat to hand	Triangular (Min, Mode, Max)	Probability of transfer meat to hand = Triangular (1.13/100, 6.54/100, 26.06/100,) Contamination on hand = Probability of transfer meat to hand × Contamination on portion	Hoelzer et al. (2012)
Contamination on fingers	Proportion of contamination on fingertips	Numeric value	Proportion of hand contamination that is on fingers = 0.06 Contamination on fingers = 0.06 × Contamination on hand	AuYeung et al. (2008) and Rusin et al. (2002)
Cross-contamination from hand to mouth	Proportion transferred from hand (fingers) to mouth	Uniform (Min, Max)	Range: Min 0.34, Max 0.41	AuYeung et al. (2008) and Rusin et al. (2002)
Dose from hand-to-mouth route	Average number of *Salmonella* cells ingested by touching mouth with hand	Numeric value	Dose hand to mouth = Proportion transferred hand to mouth × Contamination on fingers	Estimated
Cross-contamination from meat to board	Probability of transfer meat to board	Triangular	Triangular (min: 3.02/100, Max: 30.96/100, mode: 7.50/100)	Smadi and Sargeant (2013)
Cross-contamination from board to RTE food	Probability of transfer from board to RTE food	Normal fitted to Log10 of transfer proportions	μ: −1.42; σ: 0.52	Hoelzer et al. (2012)

(*continued on next page*)

Table 2 (*continued*)

Variable	Description	Distribution	Parameter	Data source
Dose from RTE food	Dose ingested with cross-contaminated RTE food (assumes RTE food ingested by one person)	For each transfer: probability of transfer (i.e. transfer rate) is multiplied by the contamination in the original surface.	Contamination on board = Contamination on meat × Probability of transfer meat to board Contamination on RTE food = Contamination on board × Probability of transfer board to RTE	Estimated
Cooking Log reduction	Reduction in Log cells of *Salmonella* due to fully cooking chicken breast according to recommendations	Constant	Full cooking is assumed to completely eliminate *Salmonella* contamination from a portion	NACMCF (2007)
Proportion of portions undercooked	% of portions that are not fully cooked to achieve the stated cooking reduction	Constant	Prob(undercooked) = 0.24 Portion is fully cooked (1/0) ~ Binomial (1,1-Prob(undercooked))	Maughan et al., (2016)
Undercooking USDA (2014b)	Reduction in Log cells of *Salmonella* associated with undercooking in home ovens	Estimated	Temperature = uniform (55,70) °Celsius Time = Uniform (20,30) minutes z_value = 5.34188 Dref = 5.72 min Tref = 60 °C Log_{10} reduction = Time × (10^((Temperature -Tref)/z_value))/Dref	Smadi and Sargeant (2013), Chardon and Evers (2017), and USDA (2014), Van Asselt and Zwietering (2006), and Murphy et al. (2004)
Dose from chicken breast	Average *Salmonella* cells remaining on a portion after cooking, and ingested by the consumer	Numeric value	For undercooked portions: Dose from chicken meat = 10^ (Log_{10} Contamination in portion - Log_{10} reduction) For fully cooked portions: Dose = 0	Estimated
Overall Dose	Average number of cells ingested by the consumer (assumes all three doses are ingested by the same person)	Numeric value	Total dose: [dose from hands] + [*dose from* RTE *food*] + [dose from chicken breast]	Estimated
Dose-response	Probability of infection associated with a portion, based on the number of *Salmonella* cells (dose) ingested	Beta-Poisson	P(illness) = 1-(1 + dose × SF/ β)^(- α) where α: 0.1324 β: 51.45 SF: 5×10^{-5} (scaling factor, estimated)	WHO/FAO (2002)
Preventable Fraction of Risk (PF)	Ratio of mean Probability(illness) per exposure event, between a scenario and the baseline	Numeric value (ratio)	PF = Mean $Prob(illness)_{scenario}$ / Mean $Prob(illness)_{baseline}$	Estimated

2.2. Product selection and definition

Chicken parts are typically defined to include chicken breasts (bone-in or boneless, skin or skinless), legs (thigh and drumstick), thighs, drumsticks and wings. Since the way consumers prepare chicken parts varies significantly, this study chose to focus on a single part, chicken breast with and without skin. Breasts are the most frequently consumed chicken part in the United States, according to the U.S. National Health and Nutrition Examination Survey (NHANES) database dietary intake data. The USDA (2012) Nationwide Microbiological Baseline Survey on Raw Chicken Parts (USDA, 2012) did not find differences in the prevalence of *Salmonella* in chicken parts with and without skin. See Supplemental Materials S.1 for the code used to extract data from NHANES and estimate consumption rates.

2.3. Salmonella contamination in product

The concentration of *Salmonella* in chicken breast at the point of sampling (i.e. immediately before packaging) was estimated using data obtained from the USDA (2012) Nationwide Microbiological Baseline Survey on Raw Chicken Parts (Table 3, Fig. 2, raw data provided in Supplementary Materials S.2). Samples collected for the USDA survey first underwent a presence/absence screening test consisting of rinsing approximately 4 lbs (1818 g, variability unknown) of chicken parts in 400 ml of liquid medium (rinsate); 30 ml of this rinsate were enriched and scored as detected/non-detected. All positive chicken breast samples (211 out of 783) were enumerated via 3 × 3 Most Probable Number (MPN) assay using three serial dilutions and three replicates per dilution, although only a portion of positive samples were further enumerated for most parts (FSIS Appendix 2.05). A summary of the entire dataset, including all parts for context, is provided in Table 3.

Two types of information were leveraged from the 2012 microbiological baseline survey for chicken parts: (1) the result of the presence/absence screening test for all samples of chicken breast, and (2) tube scores from the MPN assay for samples that tested positive (USDA, 2014a). A Bayesian latent variable hierarchical model analogous to the approach of Williams and Ebel (2012) was used to estimate the distribution of *Salmonella* concentration in chicken breasts. The distribution was assumed to be lognormal, and model convergence was tested using the Heidelberger and Welch diagnostic test. The variance of the fitted distribution accounted for the combined variability from all sources represented in the dataset, including the variability across establishments, the lot-to-lot variability within each establishment, and the variability across individual samples or portions within each lot. The model was coded in JAGS (JAGS, 2016) and R (the code, including priors and settings, is available in the Supplementary Material section S.3) (R Core Team, 2016).

2.4. Portion size

The probability of consumption and distribution of portion sizes (g consumed per day) were estimated using the "What We Eat in America" dietary data from the 2013–2014 cycle of NHANES (CDC, 2016). Participants consuming chicken breast with and without skin (DRXFDCD codes available in the Supplemental Materials S.1) were identified using Day 1 of the two-day dietary recall data. Since NHANES oversamples certain subpopulations, each individual is assigned a sample weight that indicates the number of people in the general U.S. population that the individual represents (CDC, 2016). Therefore, the probability of consumption was calculated by dividing the sum of portion weights for those consuming the identified products by the sum of the weights for all participants. The distribution of portion sizes was estimated using methods recommended for estimating usual daily intake from NHANES data (Ahluwali et al., 2016; Dwyer et al., 2003; Tooze et al., 2006). Due to the lower proportion of individuals consuming chicken breasts on both days, only weighted data from the first

Table 3
Summary of prevalence and concentration data from the FSIS 2012 baseline survey of *Salmonella* in chicken parts, overall and for breasts only (USDA 2012).

Variable	All chicken parts	Breast only
Collection period	Jan–Aug 2012	Jan–Aug 2012
Number of establishments	445	288
Total no. samples screened	2496	783[b]
No. samples positive at screening	657 (26.2%)	211 (26.9%)
No. MPN-enumerated samples quantifiable via MPN	444 of 657	138 of 211
No. MPN-enumerated samples below the limit of quantification of MPN assay (< 0.03 MPN/ml)[a]	201	68
No. MPN-enumerated samples above the limit of quantification of MPN assay (>11 MPN/ml)[a]	12	5
Mean of quantified MPN samples (MPN/g)	0.15	0.2
Standard deviation (MPN/g)	0.35	0.46
Median (MPN/g)	0.020	0.020
Minimum (MPN/g)	<0.0066	<0.0066
Maximum (MPN/g)	>2.42	>2.42

[a] If all tubes in the MPN assay were negative, the sample was estimated to be below the MPN quantification limit of 0.03 MPN/ml (0.0066 MPN/g). If all tubes in the MPN assay were positive, the sample was estimated to be >11 MPN/ml (2.42 MPN/g). FSIS reports MPN results per ml of the 400-ml rinsate. In this table, concentrations were converted from MPN/ml of rinsate to MPN/g of chicken sample by multiplying by 0.22 (400/1818 = 0.22 ml/g). MPN tube scores were used to fit the concentration distribution used in the simulation.

[b] All 783 samples were used to estimate the concentration distribution. One screen-negative sample was excluded from the calculations on prevalence by establishment, as its establishment ID was missing (see Supplementary materials).

day of dietary recall for consumers of chicken breast with and without skin were used to estimate the weighted mean and median usual daily intake as well as the standard error of the weighted mean and selected percentiles using PROC SURVEYMEANS in SAS software version 9.4 (SAS Institute, Cary NC). The SAS code is available in the Supplementary Material S.1. The population-weighted data were fitted with a lognormal distribution using the "rrisk" package in R. In the simulation, the distribution was truncated at the 1% and 99% quantiles (32.6 g and 419.6 g) to eliminate unrealistic extreme values.

2.5. Simulating establishments, lots, and portions

The main steps in the simulations are outlined in Fig. 1. A population of 5000 establishments was simulated for each of the four scenarios. For simplicity, all establishments were assumed to produce 500 product lots per year, which is approximately the mean yearly production volume in U.S. chicken part establishments (FSIS FOIA data). Product lots were simulated for each establishment (2.5×10^6 lots overall) and followed from packaging to consumption for each scenario, as described in the following sections. Each lot was assumed to be 2000 lbs (907.2 kg), which is consistent with industry practices, and estimated to include 6820 portions (based on the mean portion size of approximately 133 g/day). Contamination in individual portions was modeled and the mean probability of illness per serving was calculated.

Establishments were simulated by randomly assigning each establishment a mean *Salmonella* concentration ($\mu_{establishment}$) from a normal distribution of parameters derived from fitting the 2012 Baseline Survey data for chicken breasts ($\mu_{fitted} = -7.774$, $\sigma_{across\_establishment}$ assumed to be 1, see Supplementary Material S.3). The overall variability in the fitted concentration distribution was assumed to be due to the combination of across-establishment and within-establishment components. The within-establishment variability ($\sigma_{within\_establishment}$) was randomly drawn from a lognormal distribution of parameters (1.224, 0.30) empirically derived to create a simulated scenario that reasonably matched both the distributions of concentrations and prevalence (see Table 2 and Supplementary Material S.3). The within-establishment variability was further partitioned between across-lot and within-lot components. Since only a limited number of samples per establishment was available from the 2012 baseline survey (range of 1–13, mean 2.7 samples screened), it was not possible to characterize the across-lot variability for individual establishments from the data. In addition, only one sample was collected per lot in the 2012 baseline survey and, consequently, there is no information on the variability within lots. Therefore, in keeping with other risk assessments (Swart et al., 2013; EFSA, 2011), it was assumed that 50% of the variability was due to variability across lots, and 50% was due to within-lot variability.

To simulate lots, it was first assumed that all lots could be contaminated, i.e. no lot is assumed *a priori* to be free of *Salmonella*. Since concentrations for each portion are drawn from lognormal distributions, they are all positive numbers; however, very low concentrations, which make up a large proportion of all samples, are not detected using standard testing protocols and, therefore, are *de facto* indistinguishable from zero (for instance, 73% of the raw concentration data were non-detects). Each lot was randomly assigned a concentration distribution, reflecting the assumption that the overall establishment variance in concentration was equally partitioned between the lot-to-lot variance and the portion-to-portion variance within the lot. The implications of this assumption were tested in what-if scenarios. The mean lot concentration (μ_{lot}) was randomly drawn from the overall concentration distribution for the considered establishment ($\mu_{establishment}$ and $\sigma_{across\text{-}lots}$). Lot-to-lot variability ($\sigma_{across\text{-}lots}^2$) was assumed to be the same for all establishments and set as 50% of the overall concentration variance in the establishment, following the framework in Swart et al. (2013):

$$\sigma_{across-lots}^2 = 0.50 \times \sigma_{within\_establishment}^2$$

To simulate portions, concentrations for each portion were then drawn from the concentration distribution for the considered lot (parameters μ_{lot} and $\sigma_{portions}$). Portion-to-portion variability ($\sigma_{portions}^2$) was calculated as 50% of the overall concentration variance in the establishment, i.e. equal to $\sigma_{across\text{-}lots}^2$:

$$\sigma_{portions}^2 = 0.50 \times \sigma_{within\_establishment}^2$$

When assigning concentrations to individual portions, the concentration distribution was truncated at 10^3 cells/g, a conservative assumption based on a maximum observed MPN being a right-censored value of 11 MPN/ml of rinsate, i.e. approximately 2.42 MPN/g of product. Each portion was also assigned a portion size (g) randomly drawn from the distribution derived from the NHANES data. To estimate the number of cells in each portion, the concentration of *Salmonella* in each portion (cells/g) was exponentiated to convert it from the natural logarithm scale to the absolute (non-ln) scale and then multiplied by the portion size. Since the dose-response relationship used in the study utilizes mean doses, concentrations were not converted to integers. It is recognized that very low theoretical mean concentrations would result in a negligible probability of illness.

It was assumed every lot is sampled immediately before packaging. Sampling frequency was assumed to be one 1818-g (± 10%) sample

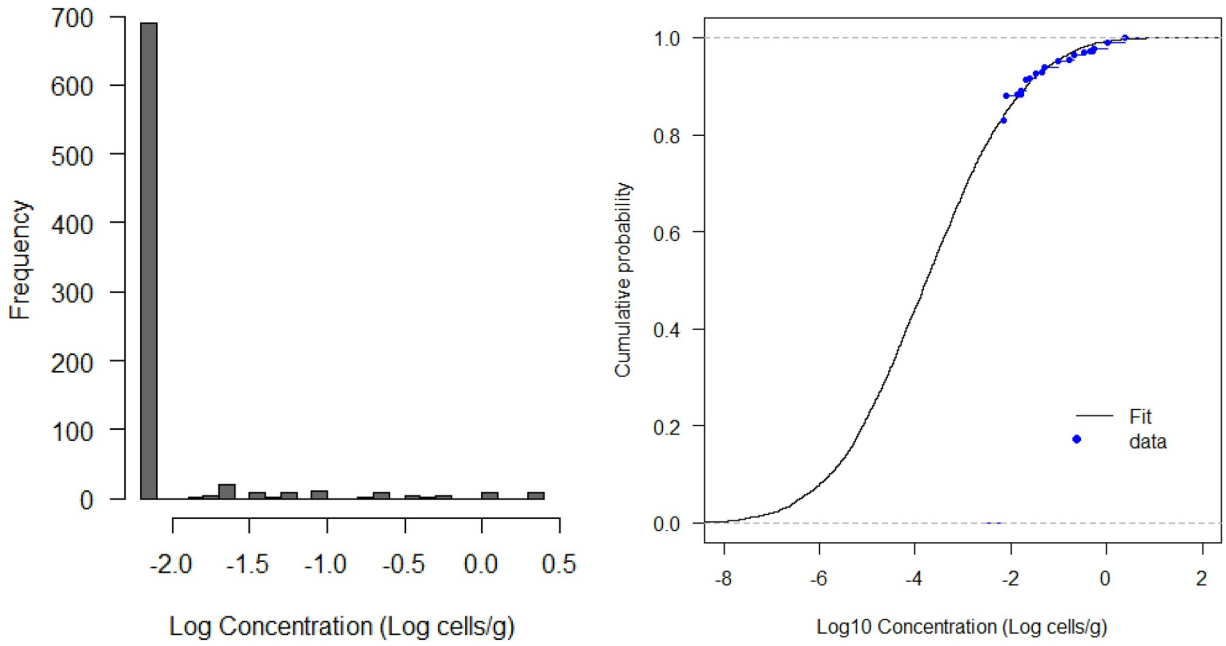

Fig. 2. Distribution of Salmonella concentration in chicken breast. *Left panel:* MPN data from the 2012 baseline survey dataset; censored values are set at the lower and upper limit of quantification for the MPN assay. *Right panel:* cumulative distribution of the fitted lognormal distribution of Salmonella concentration, in Log cells/g, and MPN/g data.

per 2000-lb (907.2 kg) lot (USDA, 2016). Each sample was assumed to be rinsed in 400 ml of liquid medium (rinsate), and that 30 ml of the rinsate was enriched and used to screen for presence/absence. The number of cells in each sample was calculated by multiplying the concentration in the sample by the sample weight. The mean cell concentration in the rinsate was calculated by dividing the number of cells in the sample by the amount of the rinsate (400 ml).

2.6. MC compliance metrics

For all alternative Scenarios 1–3, the same baseline scenario (no MC, no intervention) was used as the starting point for assessing compliance. First, the baseline scenario was simulated and the probability of illness was estimated for this "baseline year". Then the alternative scenarios were simulated, where interventions were applied based on the observed rate of non-compliance in the baseline year. For each simulated lot, the number of cells in the rinsate are assumed to follow a Poisson distribution and the probability of detecting *Salmonella* in a sample was calculated as:

$$P_{detection} = 1 - \exp(-\text{Conc}_{rinsate} \times \text{Vol}_{rinsate})$$

where $\text{Conc}_{\text{rinsate}}$ is the mean concentration assigned to the rinsate (cells/ml), and $\text{Vol}_{\text{rinsate}}$ is the volume of rinsate enriched to assess presence/absence (30 ml). The detection of *Salmonella* in the sample (1/0) was simulated by drawing from a binomial distribution with sample size of 1 (i.e. a Bernoulli trial) and probability of success equal to $P_{\text{detection}}$. If *Salmonella* was detected in the sample, the corresponding lot was scored as "detected".

2.6.1. Prevalence-based MC (Scenario 1)

The proportion of "detected" lots in the baseline scenario was calculated for each establishment by dividing the number of "detected lots" by the number of lots produced in the year (i.e. 500). If the proportion of "detected" lots exceeded the prevalence-based MC, the establishment was scored as non-compliant and the establishment-level intervention was applied in the subsequent year.

2.6.2. Concentration-based MC (Scenario 2)

If *Salmonella* was detected in a sample, the concentration of the sample was compared with the threshold concentration. If the sample concentration exceeded the concentration threshold, the associated production lot was scored as non-compliant. If *Salmonella* was not detected, or detected but with concentration below the concentration threshold, the lot was scored as compliant.

2.6.3. Combined MC (Scenario 3)

Samples were tested and lots scored according to the concentration-based MC. If one or more lots were found to be "exceeding" in the baseline scenario (i.e. more than 0.2% of the 500 lots in the establishment), the establishment was scored as non-compliant. In the subsequent year, after the establishment-level intervention was applied to non-compliant establishments, individual lots exceeding the concentration-based MC were scored as non-compliant and underwent the lot-level intervention as in Scenario 2.

2.7. Intervention scenarios

In this study, two main intervention approaches were considered, and assumed to be triggered by non-compliance with either the prevalence-based MC or the concentration-based MC (Table 1).

2.7.1. Establishment-level intervention

Non-compliant establishments were assumed to undergo a comprehensive establishment-level intervention that resulted in a concentration reduction for all product lots in the establishment in the subsequent year. In the main risk scenarios, we assumed a uniform reduction of 3 ln cells/g (approximately 1.3 decimal Logs) in the mean of the concentration distribution for each lot in the establishment (μ_{lot}); portion-to-portion variability was assumed to remain the same. This intervention mimics a simplified "feedback loop" where information on establishment non-compliance resulting from the prevalence-based MC in the first data collection period (e.g. year 1) informs control efforts that reduce contamination levels in the second, post-intervention period (e.g. year 2). The impact of the assumption of a 3 ln cells/g reduction was tested in the what-if scenario analysis.

2.7.2. Lot-level intervention

Non-compliant lots were assumed to be subjected to a theoretical risk reduction measure that eliminated any contamination from all portions in the lot (best-case intervention scenario), resulting in zero risk for those lots. This was modeled by substituting the estimated risk for all portions in the non-compliant lots to zero.

2.8. Retail handling and transportation

The model assumed no change (i.e. no growth or die-off) in *Salmonella* prevalence or concentration during transportation to retailers, at retail, or during transportation from retail to homes. Portions were assumed to be independent, with no cross-contamination occurring between chicken breast portions placed in the same package.

2.9. Consumer handling

Upon entering the consumer's home, the raw unfrozen product was assumed to undergo the following steps: (1) either immediate meal preparation, or refrigeration for a defined time duration (no change in *Salmonella* levels was assumed); no freezing or thawing scenarios were considered in this model; (2) temporary storage of the raw product at room temperature before cooking (no change in *Salmonella* levels was assumed); (3) touching raw chicken breast with hands and subsequently touching the mouth (hand-to-mouth route); (4) cross contamination between raw chicken breast and a RTE product, such as a vegetable salad, resulting from raw meat touching a cutting board, and subsequent contact between the contaminated board and RTE vegetables; (5) cooking a chicken breast, resulting in a reduction in *Salmonella* levels (kill step); (6) eating a cooked chicken breast; (7) eating the RTE food (“salad” route).

Three exposure routes were considered in the model. The first route of exposure was direct consumption of chicken breast with or without skin. Two additional exposure routes associated with cross-contamination in consumers’ kitchens were also considered (hand-to-mouth and salad). The dose per exposure route was calculated by estimating the average number of cells transferred via each route; transfer coefficients between surfaces are shown in Table 2. The proportion of cells transferred at each step was assigned by randomly drawing a value from the distribution of the transfer coefficient. The number of cells transferred in that step was then calculated by multiplying the number of cells available for transfer by the assigned transfer coefficient. The number of cells transferred was then subtracted from the number of cells available in the previous step. For example, if the contamination level for a given portion was 100 cells and the randomly drawn transfer coefficient was 10% for the transfer from breast to hands, 10 cells would be transferred from the portion to the hands, reducing the contamination level for the portion to 90 cells and making only 10 cells available for transfer to the mouth and/or the board. It was assumed that only one portion was handled during one food preparation event and that hand-to-mouth transfer occurred before hand-to-board-to-salad transfer. For cross contamination with RTE food, it was assumed that the entire salad portion affected by bacterial transfer was consumed by one person; hence, the salad portion is not specified.

2.10. Consumer risk reduction

It is assumed that the chicken breast (with or without skin) was cooked in home ovens. Fully cooked portions (i.e. an internal temperature of 165°F or 71 °C) were assumed to have zero risk. It was further assumed that 24% of chicken breast portions were improperly cooked (Maughan et al., 2016). An individual portion was scored as undercooked using a Bernoulli trial with probability equal to a 24%. The reduction in *Salmonella* levels associated with undercooking was modeled following the approach and assumptions of Chardon and Evers (2017) and Smadi and Sargeant (2013). For undercooked portions, the cooking temperature was assumed to be uniformly distributed between 55 and 70 °C (Smadi and Sargeant, 2013). Cooking time was assumed to follow a uniform distribution between 20 and 30 min, as recommended by FSIS guidance for roasting boneless chicken breast (USDA, 2014b). The Log cells/g reduction associated with undercooking was calculated using the approach by Chardon and Evers (2017) as shown in Table 2.

2.11. Risk characterization

The total dose per portion was calculated by summing the dose across the three exposure routes. The probability of illness per portion was estimated using the WHO/FAO (2002) beta-Poisson dose-response equation for *Salmonella* (Table 2). Variability in dose-response was not considered. A scaling factor was used to multiply the dose, in order for the magnitude of risk outcomes in the baseline to be compatible and lower than epidemiological estimates (approximate average of 3.5 cases per 1 M lbs for all chicken parts, based on calculations and on data reported by Scallan et al., 2011; IFSAC, 2017; NCC, 2018). The impact on public health associated with each scenario was expressed as the probability of illness per portion, averaged and tracked at establishment-level (representing 1 M lbs of product). The significance of differences in the probability of illness between baseline and each post-intervention scenario, as well as between scenarios, was assessed using the Anderson-Darling test. The mean residual risk after intervention was calculated as the ratio between the mean probability of illness across establishments with and without intervention:

$$Residual\ Risk = \frac{mean(P(ill)_{establishment\_post-intervention})}{mean(P(ill)_{establishment\_baseline})}$$

The mean preventable fraction of risk was calculated as (1-Residual Risk).

2.12. What-if scenario analysis and sensitivity analysis

Uncertainty analyses were conducted to assess the impact of different thresholds for the prevalence- and concentration-based MC and *Salmonella* concentration assumptions, such as the parameter μ and variance partitioning, on model outcomes. Additional ad-hoc scenario analyses were carried out to test different assumptions on intervention compliance rate, cooking, undercooking, and cross-contamination. A sensitivity analysis was carried out over variables aggregated at establishment-level, using Spearman correlation.

Each of the four scenarios considered were modeled using 5000 Monte Carlo iterations (i.e. 5000 simulated establishments). The number of Monte Carlo iterations was tested for convergence: using results from 10,000 iterations as reference, 5000 iterations were sufficient to bring the coefficient of variation of the estimate of risk from run to run to below 4% for the mean, and below 5% for the 95th percentile. The model was built in the R language (R Core Team, 2016), and run in the R version 3.5.0; the code is provided in Supplemental Materials S.4.

3. Results

3.1. Salmonella contamination in product

The concentration of *Salmonella* in raw chicken breasts was modeled using a lognormal distribution. The distribution was derived from 783 chicken breast samples collected from 288 establishments for the FSIS 2012 baseline survey; 211 samples were positive at screening and also enumerated (Table 3). The fitted concentration distribution (Fig. 2) formed the basis for the contamination input variables used in the risk model. The mean fitted parameters were $\mu = -7.728$ ln cells/g and $\sigma = 3.166$ ln cells/g, corresponding to a mean of 0.066 cells/g, and a median of 4.4×10^{-4} cells/g.

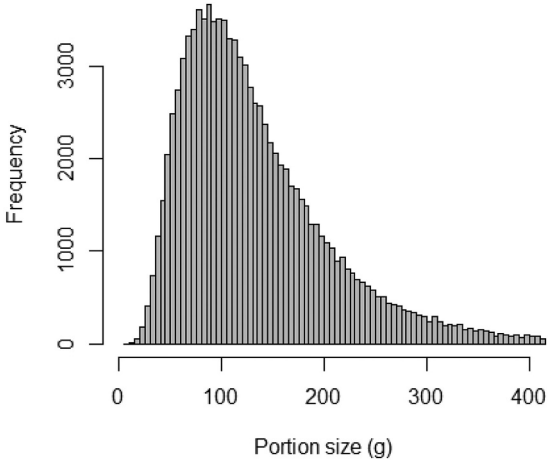

Fig. 3. Distribution of total grams of chicken breast consumption on day 1 of the dietary recall assessment in the 2013–2014 cycle of NHANES (before truncation at the 1% and 99% percentiles).

3.2. Portion size

Five hundred ninety-nine (599) of 8661 participants reported consuming roasted, broiled, baked or fried chicken breasts on Day 1 of the dietary recall assessment in the 2013–2014 cycle of NHANES. The weighted consumption rate was 7.5%. Only 35 participants reported consuming more than one portion on Day 1, so the total grams consumed was assumed to be representative of a single consumption event. The weighted mean and median portion size were 139.82 and 117.12 g/day, respectively, with the distribution being approximately lognormal (Fig. 3). The cumulative percentiles from the empirical distribution of weighted consumption amounts were fitted with a lognormal distribution with parameters $\mu = 4.76$ ln g/day and $\sigma = 0.55$ ln g/day, where μ and σ are the mean and standard deviation of the underlying normal distribution. Portion sizes were drawn from this lognormal distribution.

3.3. Risk scenario results

Results for scenarios 1, 2, and 3, as well as the baseline, are presented below and summarized in Table 4 and Fig. 4. In general, the estimated mean probability of illness decreased, and the preventable fraction increased as interventions were added to the scenarios. As expected, Scenario 3, which included both establishment- and lot-level interventions, had the lowest probability of illness and highest preventable fraction compared to the baseline. Risk outcomes for all scenarios were right-skewed and presented a large variance. The distributions of post-intervention risk estimates (mean probability of illness per establishment) for the main scenarios were significantly different from each other and from the baseline.

3.3.1. Baseline scenario

The baseline scenario, with no establishment- or lot-level interventions, resulted in a mean probability of illness of 2.46×10^{-7} (90% CI: 6.42×10^{-10}, 1.24×10^{-6}) per exposure event.

3.3.2. Scenario 1. Prevalence-based MC with establishment-level intervention

When setting a 15% prevalence-based MC based on a presence/absence test using a 4-lb sample (which corresponds to an approximate theoretical and non-probabilistic limit of detection of 0.007 cells/g), 87.1% of establishments were scored as non-compliant and underwent a food safety assessment that resulted in a 3 ln (~ 1.3 Log cells/g) reduction across the entire establishment in year 2. The mean probability of illness after intervention was estimated to be 5.73×10^{-8} (90% CI: 1.07×10^{-10}, 2.87×10^{-7}) per portion. Compared to baseline, the mean residual risk fraction was 0.233 and the preventable fraction was 0.767. When a concentration threshold was added to the testing assay used in Scenario 1, with a threshold of 0.1 cells/g (to be consistent with Scenarios 2 and 3), the mean residual risk was 1.38×10^{-7} and the preventable fraction was 0.461. When a 1 Log cells/g reduction was applied to all establishments, instead of only the non-compliant ones, the mean residual risk was 7.80×10^{-8}, and the preventable fraction was 67.6%.

3.3.3. Scenario 2. Concentration-based MC with lot-level intervention

Assuming a 0.1 cells/g concentration threshold, 7.1% of all lots were scored as non-compliant and underwent a lot-level intervention that eliminated *Salmonella* contamination from all portions in the lot. The post-intervention mean probability of illness was estimated to be approximately 9.11×10^{-8} (90% CI: 7.09×10^{-10}, 4.80×10^{-7}) per exposure event. Compared to baseline, the residual risk was 0.212 and the mean preventable fraction was 0.602. However, the large variance in the risk outcome (as can be seen from the 90% CI) obscured the difference between baseline and Scenario 2, as well as among other scenarios.

3.3.4. Scenario 3. Concentration-based MC with establishment and lot-level interventions

As in Scenario 2, 7.1% of lots were scored as non-compliant. The mean probability of illness after both establishment-level and lot-level interventions was estimated to be approximately 6.07×10^{-8} (90% CI: 5.62×10^{-11}, 1.67×10^{-7}) per exposure event. Compared to baseline, the residual risk was 0.125 and the preventable fraction was 0.875.

A sensitivity analysis, conducted by correlating the mean probability of illness per 1 M lbs with model variables at establishment-level showed a high correlation with the variability within establishments ($\sigma_{within\_establishment}$) (coefficient of correlation CC: 0.92 for Scenario 1, CC:0.93 for Scenario 2), and a moderate correlation with the establishment concentration parameter ($\mu_{establishment}$) (CC: 0.17 for Scenario 1, CC: 0.30 for Scenario 2). As expected, the proportion of detected lots in an establishment shows a higher degree of correlation with risk outputs in Scenario 1 (CC: 0.81), where the intervention is triggered by prevalence, than in Scenario 2 (CC: 0.30) where the intervention is triggered by concentration levels.

3.4. What-if scenario analysis

The impact of assumptions (initial level of lot contamination, concentration threshold, thresholds for the prevalence- and concentration-based MC, concentration reduction associated with the establishment-level intervention, cooking, cross-contamination, variance partitioning)

Table 4
Comparison of risk outcomes for the baseline (without intervention) and the three main alternative scenarios (post intervention).

	Mean $P_{illness}$	90% Confidence Interval for $P_{illness}$	Preventable fraction
Baseline (no intervention)	2.46×10^{-07}	6.42×10^{-10}; 1.24×10^{-06}	–
Scenario 1	5.73×10^{-08}	1.07×10^{-10}; 2.87×10^{-07}	0.767
Scenario 2	9.11×10^{-08}	7.09×10^{-10}; 4.80×10^{-07}	0.602
Scenario 3	6.07×10^{-08}	5.62×10^{-11}; 1.67×10^{-07}	0.875

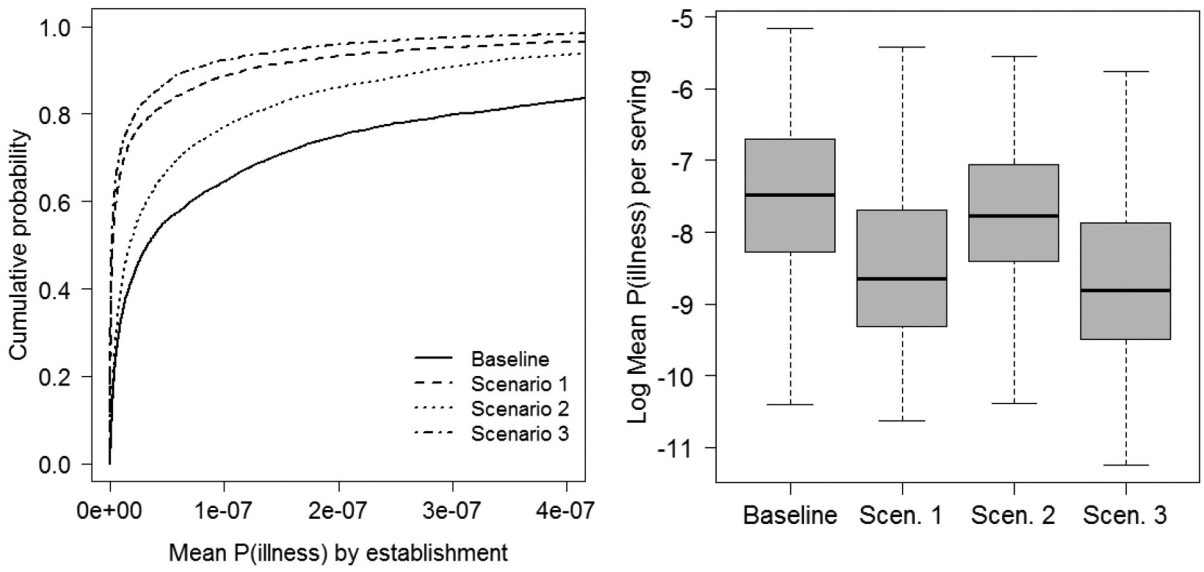

Fig. 4. Comparison of risk outcomes for the three main alternative scenarios, and the baseline without intervention. In the right panel, boxes represent the 25th and 75th percentile, the horizontal band in the box the median, and whiskers are set at ± 1.5 times the interquartile range from the median.

on the probability of illness and preventable fraction were explored using what-if scenarios. Results for the what-if scenarios are summarized below and in Table 5. As in the main risk scenarios, Scenario 3 had the lowest probability of illness and highest preventable fraction (compared to baseline) in all what-if scenarios.

3.4.1. Impact of initial contamination level

In the main scenarios, the initial level of contamination across all establishments is assumed to be a lognormal distribution with a mean (μ) of −7.728 ln cells/g and a median of 4.4×10^{-4} cells/g. Alternative values were tested to assess the impact of different initial contamination levels. The trend in the risk outcome and preventable fraction as a function of the mean are shown in Fig. 5. As expected, the mean probability of illness increases as the baseline contamination increases across all three scenarios for concentrations below −5 ln cells/g. Once the mean level of initial contamination exceeds approximately −5 ln cells/g, the trends differ across scenarios. In Scenario 1, at these concentration levels, all establishments have a prevalence above 15% and, as the mean increases, and the fixed 3-ln reduction remains the same, the preventable fraction starts decreasing. For Scenarios 2 and 3, the trend is driven by the lot-level intervention in that, as the concentration increases, more lots become non-compliant and their risk is reduced to zero; hence the preventable fraction keeps increasing. In the (unrealistic) case where the majority of lots are non-compliant, the mean probability of illness trend flattens out at the residual risk level of the remaining compliant lots, and the preventable fraction approaches 1.

3.4.2. Impact of the prevalence-based MC assumptions

Scenario 1 assumes that establishments with more than 15% of samples detected undergo an establishment-level intervention. To assess the impact of this parameter, we varied the prevalence-based MC threshold from 2% to 50%. Decreasing the prevalence-based MC from 40% to 30% would increase the preventable fraction by approximately 40%, whereas decreasing the prevalence-based MC from 30% to 10% would increase the preventable fraction by 12%. For prevalence-based MCs below 25% (Fig. 7), the preventable fraction trend flattened out, suggesting that there is marginal improvement for decreasing the prevalence-based MC to below this level, in the specific context of the assumed concentration distribution. When the prevalence-based MC was increased to 50% or above, no illnesses were prevented by employing the establishment-level intervention, as all establishments were compliant and therefore the establishment-level intervention driving *Salmonella* reduction in this scenario was not triggered. These absolute values of risk and preventable fraction are driven by the high prevalence intrinsic in the fitted input concentrations (which leads to 87.1% of establishments being non-compliant with a 15% prevalence threshold); however, trends in Fig. 7 would be similar and shifted to the left with a lower input prevalence. While a compliance rate of 100% was assumed in the main scenario, when this rate was reduced to 40% (i.e. a non-compliant establishment had a 40% probability of implementing the intervention), which was an assumption used by FSIS in establishing performance standards (USDA 2015b), the preventable fraction was reduced to 29.1%.

3.4.3. Impact of the concentration-based MC assumptions

In Scenarios 2 and 3, the default concentration-based MC is assumed to be 0.1 cells/g. To assess the impact of this assumption, a range of concentration-based MC thresholds between 0.001 cells/g and 100 cells/g were considered (Fig. 6). As expected, the mean probability of illness decreased as the concentration threshold decreased. For Scenario 2, the preventable fraction ranged from 0.818 for a concentration-based MC of 0.001 cells/g to 0.089 for a concentration-based MC of 100 cells/g, highlighting the impact of the MC concentration threshold. Since concentrations above 100 cells/g are unlikely to occur, a MC based on such threshold would result in the treatment of very few lots, and hence would not appreciably reduce risk compared to the baseline. For Scenario 3, the preventable fraction ranged from 0.928 for a concentration-based MC of 0.001 cells/g to 0.663 for a concentration-based MC of 100 cells/g, suggesting a high public health impact regardless of the MC threshold level within the considered range. For both scenarios, the preventable fractions flattened out and exceeded a preventable fraction of 0.75 for concentration MC thresholds at and below 0.01 cells/g, suggesting marginal improvement for reducing the concentration-based MC to below 0.01 cells/g, in the context of the assumed input concentration distribution. Given that most laboratory assays have a limit of detection at or below 0.01 cells/g, this suggests that the results are robust regardless of the assay employed. For Scenario 3, the preventable fraction remained above 0.75 even if the concentration-based MC was increased to 10 cells/g, suggesting that the results seen for Scenario 3 are fairly robust against changes in the concentration-based MC.

Table 5
Comparison of risk outcomes for what-if scenarios for the three main alternative scenarios (post intervention).

What-if scenario	Baseline[a] Mean $P_{illness}$	Scenario 1 Mean $P_{illness}$	Preventable fraction	Scenario 2 Mean $P_{illness}$	Preventable fraction	Scenario 3 Mean $P_{illness}$	Preventable fraction
Input concentration (μ)							
−10	8.50E−08	2.58E−08	0.696	4.46E−08	0.506	1.30E−08	0.851
−7.7 (Main Scenario)	2.46E−07	5.73E−08	0.767	9.11E−08	0.602	6.07E−08	0.875
−5	9.27E−07	2.19E−07	0.764	2.56E−07	0.738	8.55E−08	0.906
−3	2.36E−06	5.22E−07	0.767	4.08E−0	0.833	1.85E−07	0.924
−1	6.07E−06	1.51E−06	0.752	5.61E−07	0.909	3.21E−07	0.947
1	1.42E−05	3.86E−06	0.729	6.14E−07	0.957	4.79E−07	0.966
Establishment intervention – prevalence threshold							
2%	2.46E−07	6.26E−08	0.762	N/A	N/A	N/A	N/A
5%	2.46E−07	5.89E−08	0.763	N/A	N/A	N/A	N/A
10%	2.46E−07	6.32E−08	0.753	N/A	N/A	N/A	N/A
15% (Main Scenario)	2.46E−07	5.73E−08	0.767	N/A	N/A	N/A	N/A
20%	2.46E−07	5.76E−08	0.763	N/A	N/A	N/A	N/A
30%	2.46E−07	9.06E−08	0.636	N/A	N/A	N/A	N/A
40%	2.46E−07	1.96E−07	0.256	N/A	N/A	N/A	N/A
50%	2.46E−07	2.33E−07	0.038	N/A	N/A	N/A	N/A
Lot Intervention – concentration threshold (cells/g)							
0.001	2.46E−07	5.73E−08	0.767	4.79E−08	0.818	1.82E−08	0.928
0.01	2.46E−07	6.27E−08	0.758	5.57E−08	0.773	1.58E−07	0.911
0.1 (Main Scenario)	2.46E−07	5.73E−08	0.767	9.11E−08	0.602	6.07E−08	0.875
1	2.46E−07	6.09E−08	0.761	1.40E−07	0.400	6.18E−08	0.830
10	2.46E−07	6.42E−08	0.754	1.96E−07	0.224	6.35E−08	0.781
100	2.46E−07	6.30E−08	0.756	2.40E−07	0.089	9.32E−08	0.663
Establishment intervention – Log reduction in concentration (μ of establishment)							
0.5	2.58E−07	1.50E−07	0.420	N/A	N/A	7.04E−08	0.735
1	2.46E−07	8.09E−08	0.671	N/A	N/A	4.59E−08	0.833
1.3 (Main Scenario) = 3 ln	2.46E−07	5.73E−08	0.767	N/A	N/A	6.07E−08	0.875
1.5	2.60E−07	5.04E−08	0.806	N/A	N/A	2.53E−08	0.895
2	2.49E−07	2.83E−08	0.887	N/A	N/A	1.47E−08	0.938
Undercooking							
Undercooked = 1 Log reduction	2.41E−07	1.47E−07	0.389	1.01E−07	0.600	6.80E−08	0.726
Undercooked = 2 Log reduction	2.34E−07	8.79E−08	0.625	1.04E−07	0.604	4.77E−08	0.812
24% undercooked (Main Scenario)	2.46E−07	5.73E−08	0.767	9.11E−08	0.602	6.07E−08	0.875
10% undercooked	2.15E−07	5.19E−08	0.759	8.35E−08	0.602	2.70E−08	0.874
Cross-contamination –							
1 portion handled (Main Scenario)	2.46E−07	5.73E−08	0.767	9.11E−08	0.602	6.07E−08	0.875
No cross-contamination	9.98E−08	2.33E−08	0.766	3.95E−08	0.609	1.25E−08	0.873
Variance Partitioning Assumptions							
Fraction of Variance within lot: 0.01	4.46E−07	1.10E−07	0.754	1.52E−09	0.997	4.95E−10	0.999
Fraction of Variance within lot: 0.30	2.69E−07	6.42E−08	0.762	5.03E−08	0.832	1.56E−08	0.942
Fraction of Variance within lot: 0.50 (Main Scenario)	2.46E−07	5.73E−08	0.767	9.11E−08	0.602	6.07E−08	0.875
Fraction of Variance within lot: 0.70	2.54E−07	6.32E−08	0.751	1.54E−07	0.604	4.11E−08	0.830
Fraction of Variance within lot: 0.99	2.31E−07	5.51E−08	0.761	2.06E−07	0.178	5.65E−08	0.777

[a] Risk outcomes for both the baseline and the considered scenario were generated for each model run. For clarity, the same value of the baseline risk is presented for scenarios where the considered "what-if" variable does not impact the baseline.

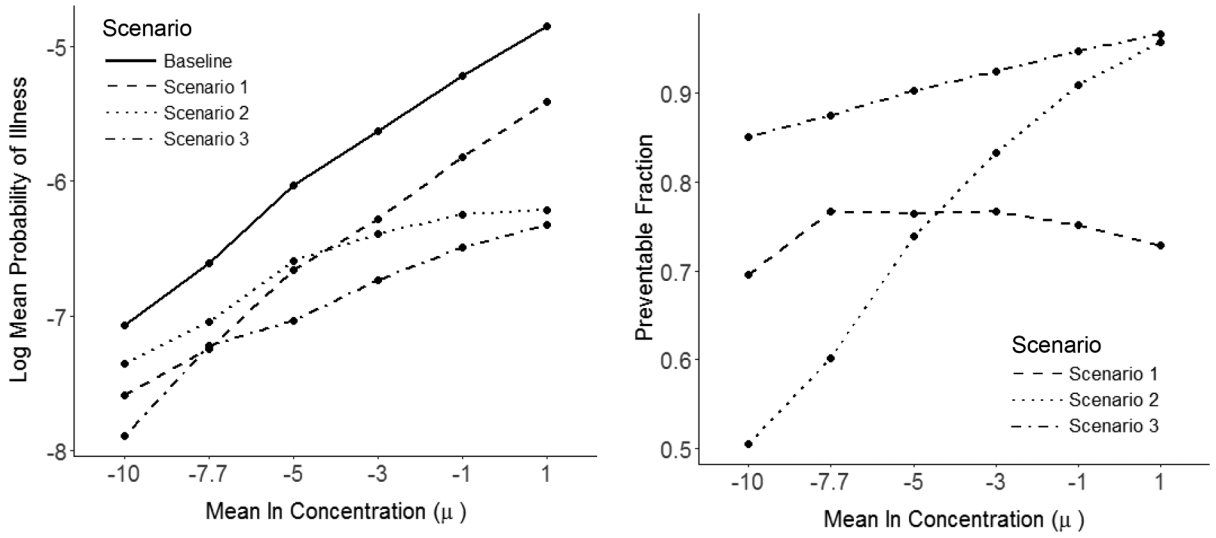

Fig. 5. Impact of different MC concentration thresholds on the mean risk (mean probability of illness per serving) and overall preventable fraction of risk.

3.4.4. Impact of reduction associated with the establishment-level intervention

Scenarios 1 and 3 include an establishment-level intervention that assumes a 3 ln cells/g (i.e. 1.3 Log cells/g) reduction in concentration in the post-intervention period (year 2). Increasing the reduction associated with the establishment-level intervention from 0.5 to 2 Log cells/g resulted in approximately linear decreases in the probability of illness, and in corresponding increases in the preventable fraction from approximately 42–89%.

3.4.5. Impact of cooking assumptions

In all main scenarios, it is assumed that 24% of portions are undercooked and that undercooked portions result in a wide range of Log reductions. To examine the impact of these assumptions, we varied the proportion of undercooked portions from 24% to 10% and the Log reduction for undercooked portions from 0 to 3 (**Table 5**). Results were fairly insensitive to changes in the fraction of undercooked portions. For instance, decreasing the proportion of undercooked chicken breasts from 24% to 10% only marginally decreased the mean probability of illness. This is likely due to the fact that a large fraction of undercooked portions still undergoes a high level of reduction (approximately 60% underwent at least a 7 Log reduction) and the fact that the RTE cross-contamination route accounts for a large portion of the overall risk. Limiting the Log reduction for undercooking to 1 or 2 Logs resulted in an increase in risk and a decrease in the preventable fraction for Scenarios 1 and 3. In Scenario 2, cooking assumptions affected the majority of lots (all but the 7.1% non-compliant) in the same way in both baseline and post-intervention scenario, hence having marginal impact on the preventable fraction.

3.4.6. Impact of cross-contamination assumptions

In all main scenarios, it is assumed that the consumer handles one chicken breast, and that the same person is also exposed to the two other routes. When scenarios without cross-contamination were considered, the absolute risk value decreased by approximately half order of magnitude, while the preventable fraction did not change substantially, as expected since both scenarios and baseline were similarly affected.

3.4.7. Impact of variance partitioning

In all main scenarios, it was assumed that within-establishment variability is equally distributed across and within lots (i.e. 50% of variability is attributed to each). An uncertainty analysis was conducted to assess the impact of this assumption on the effectiveness of different MCs. Changing these assumptions did not impact the results for Scenario 1, which was expected since results for this scenario are primarily driven by the overall distribution for an establishment. In contrast, changing the assumptions did impact results for Scenarios 2 and 3, with the effectiveness of all MCs decreasing as the component of within-lot variability increased (Table 5). These results highlight the potentially significant impact of variability within establishments.

4. Discussion

In this study, a probabilistic quantitative risk assessment model was developed to evaluate the public health impact of implementing three different MC for *Salmonella* in chicken parts, and more specifically chicken breasts: 1) a prevalence-based MC (Scenario 1); 2) a concentration-based MC (Scenario 2); and 3) a combination of a prevalence-based and concentration-based MC (Scenario 3). Overall, implementation of a MC reduced the mean probability of illness in our model. The implementation of a prevalence-based MC and an establishment-level intervention (Scenario 1) reduced the mean probability of illness by 76.7% in the main scenario when compared to no MC and no interventions (Baseline Scenario). Implementation of a concentration-based MC (with a 0.1 cells/g concentration threshold) and a lot-level intervention (Scenario 2) reduced the mean probability of illness associated with a single exposure event by 60.2%, compared to no MC (Baseline Scenario). Combining the prevalence-based and concentration-based MC and implementing both establishment-level and lot-level interventions (Scenario 3) reduced the mean probability of illness by 87.5%. The results were fairly consistent across the considered what-if scenarios, suggesting that the overall results are robust against deviations from the model assumptions, although it is recognized that the reduction in risk is a function of the specific set of assumptions considered. It is important to note that the relative magnitude of the reduction was dependent on scenario-specific parameters, and hence the three main scenarios are presented as examples of different approaches, and are not meant to be ranked or construed as the optimal set of MC parameters. Overall risk outcomes from all three scenarios were significantly different from the baseline and from each other. However, the high variability in risk outcomes, associated with high variance in the input concentration, suggests caution in deriving conclusions based on mean estimates, and highlights the importance of better characterizing inputs to distinguish variability from uncertainty.

The model developed in this study allows the application of different MC and interventions to a common baseline, explicitly considering lot-level and establishment-level approaches. Results show that

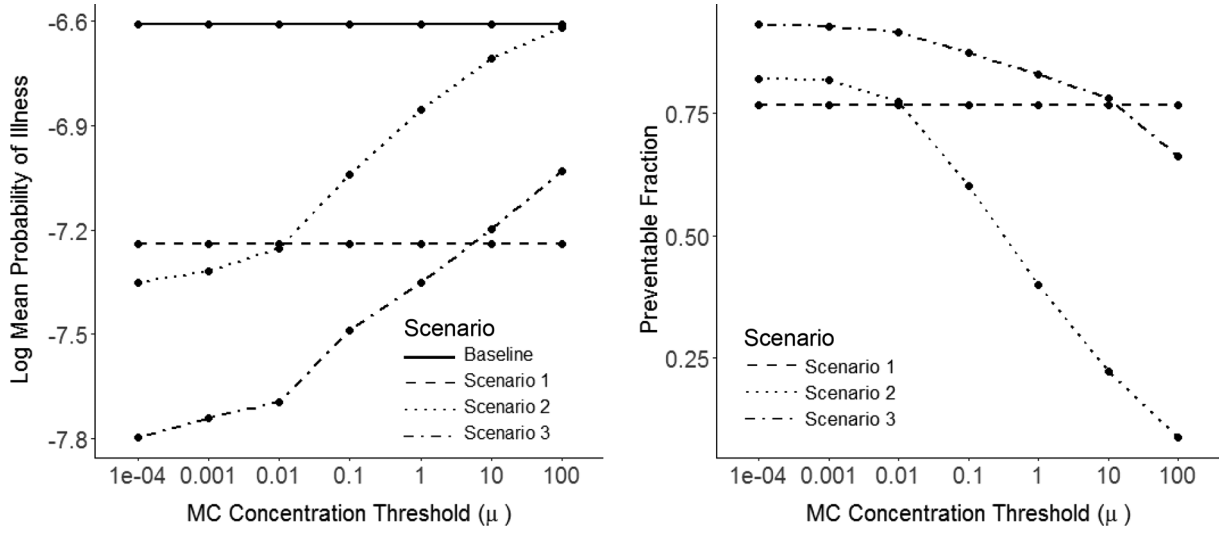

Fig. 6. Impact of the distribution of establishment concentration on the mean risk (mean probability of illness per serving) and overall preventable fraction of risk.

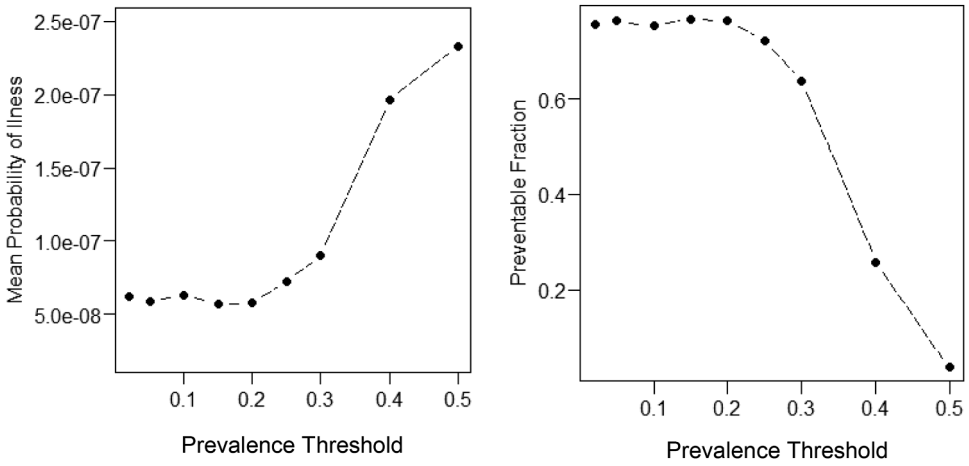

Fig. 7. Impact of the prevalence-based MC for Scenario 1 on the mean risk (mean probability of illness per exposure event) and overall preventable fraction of risk. The default prevalence threshold in the main Scenario 1 is 0.15.

both approaches have the potential to reduce risk. Other studies that have sought to assess different approaches for controlling foodborne pathogens in poultry, specifically *Salmonella* and *Campylobacter*, using risk assessment models have also found net public health benefits from the implementation of MC (Swart et al., 2013; USDA, 2015b; Sampedro et al., 2018). In particular, FSIS assessed the public health impact of a performance standard based on prevalence and concluded that implementing a performance standard of a maximum of 8 positives out of 52 samples (15.4%) would lead to a 25% reduction in salmonellosis incidence associated with chicken parts, compared to the 2006–08 baseline (USDA, 2015b). There are some key differences between the FSIS approach and the one used in this study, which limits the comparability of results. The FSIS model was based on the distribution of prevalence instead of concentrations, did not make explicit assumptions on the partition of variability across establishments, lots, and portions, and also considered different sampling frequencies than this study (USDA, 2015b). Our model was based on the distribution of concentrations, with the assumption that prevalence and concentration of *Salmonella* are dependent; that is, higher concentration levels result in higher likelihood of detection and hence higher prevalence rates. The compliance factor also differed between the two studies, with FSIS assuming a compliance fraction of 40% (product from establishments that would go from failing to passing the performance standard) whereas we assumed a best-case scenario of 100% compliance. The different assumptions about compliance may partially explain the higher relative risk reduction in Scenario 1 (76.7%) when compared with the FSIS risk assessment (25%), in addition to assumptions on concentration inputs. When we reduced the compliance fraction to 40%, the preventable fraction was 29.1%, which is only slightly higher than the preventable fraction in the FSIS risk assessment.

Our findings are also consistent with those from Swart et al. (2013) which examined the public health impact of using a concentration-based MC and a lot-level intervention to control *Campylobacter* in poultry in the Netherlands. In their study, using a MC of 1000 cells/g and applying an intervention that reduced risk to zero in non-compliant lots yielded an overall risk reduction of 67% and 72% for 2009 and 2010 respectively, with a range of 30–90% across individual slaughterhouses. While considering a different pathogen, their approach was similar to the one adopted in the current study. For instance, their model was based on the distribution of concentrations, used a forward risk model including consumer handling and a dose-response relationship, and made explicit assumptions on how variability in concentrations was partitioned between across-lots and within-lots. In contrast to our study, Swart et al. (2013) had sufficient data to characterize each establishment individually, and hence did not need to make assumptions about variability across establishments.

Our findings are consistent with those in Sampedro et al. (2018), which estimated a significant reduction of *Salmonella* risk associated with ground turkey when a lot-based intervention was applied based on a concentration-based MC. This study considered exposure at both homes and restaurants, with home cooking being estimated as resulting in higher risk. It also made the explicit distinction between high-virulence and low-virulence serovars. Differently from our study, Sampedro et al. (2018) assumed that concentration in a sample represents the concentration of a lot, i.e. that all portions from the lot have a uniform concentration equal to that of the sample, with no within-lot variability. They also assumed that non-detected samples were completely free of *Salmonella*. In addition, this study considered undercooking but not cross-contamination. While exploring a different range of scenarios and assumptions, this study highlights the potential effectiveness of concentration-based MC, suggesting they should be considered as a possible tool within a comprehensive intervention approach.

The sensitivity of the sampling protocol and testing assay can significantly impact the effectiveness of any MC. In reality, it is unlikely that more than one sample per lot is collected and, if this is the case, prevalence can only be calculated over a set of lots, as assumed in the considered scenarios. In addition, the presence/absence and concentration testing assays may have different sensitivity. The presence/absence assay, modeled after the FSIS assay, was assumed to have a theoretical non-probabilistic limit of detection of 0.007 cells/g, i.e. 1 cell × 400 ml / (1818 g × 30 ml). In contrast, while the concentration assay applied to samples positive to the presence/absence test was assumed to correctly estimate concentrations at all levels without loosing signal or precision, the discriminant between compliance and non-compliance in the concentration-based MC was a sample threshold of 0.1 cells/g, much higher than 0.007 cells/g. To compare scenarios using the same assay, a what-if was considered that included a concentration threshold step for Scenario 1 in addition to the detection step, and prevalence was defined not as the proportion of samples detected with a presence/absence test, but as the proportion of samples exceeding a set concentration threshold (as also done in Scenario 3). In practice, this modification corresponds to raising the theoretical detection limit of the presence/absence test from 0.007 cells/g to 0.1 cells/g. In this case, the prevalence-based MC coupled with an establishment-level intervention had a lower preventable fraction than a lot-level intervention (preventable fraction of 46.1% in the modified Scenario 1 vs. 60.2% in Scenario 2). This is in contrast to the main Scenarios, where the preventable fraction was higher for Scenario 1 than Scenario 2, highlighting the impact of the laboratory assay sensitivity.

Several assumptions were made in our model for simplification reasons and/or lack of current research, which may limit the generalizability of the results presented here. These assumptions and limitations are discussed below.

First, to simplify the consumer handling portion of the model, we did not consider all chicken parts. Consumption rates, portion sizes, and consumer handling/cooking behaviors can differ significantly depending on chicken part product. Including all these combinations was beyond the scope and resources available for this project, so the analysis was limited to chicken breasts since they are the most commonly consumed chicken part in the United States. The risk reductions associated with the prevalence-based and concentration-based MC may differ by chicken part and, thus, the risk reductions seen for chicken breasts may not be generalizable to all chicken parts.

Second, the model was based on publicly available data for *Salmonella* prevalence and concentration that was several years old and was limited in scope. Presence/absence and concentration (MPN) data were derived using data collected from a relatively small number of establishments over a specified period of time in 2012. A large proportion (73%) of the data points were non-detects, resulting in a large variance in the fitted concentration distribution (Fig. 2). Also, when we modeled limited prevalence data with a beta distribution, approximately 85% of the fitted curve fell above a prevalence of 15%, which may not be realistic and is likely due to the low number of samples available per establishment. Including only establishments with more than 1 or more than 2 samples did not reduce the issue. Most notably, this bias in the fitted prevalence led to a high proportion of non-compliant establishments in Scenario 1. As a result, the concentration inputs in this model should be considered a theoretical example to illustrate the approach and should not be considered representative of current or recent contamination patterns. Further, since a limited number of samples was collected for each establishment, it was not possible to estimate concentration variability across production lots from the same establishment, or within individual portions from the same production lot. As a result, assumptions about the distribution of variability across and within establishments and lots were made, which would need to be substantiated by data. In addition, data on establishment production volume was not available, which prevented the development of weighted prevalence estimates. Finally, the data were collected 6 years ago and prior to the implementation of performance standards for chicken parts. Since *Salmonella* contamination is not static and it is likely that the newly implemented performance standards resulted in improvements within the poultry industry, the overall prevalence and contamination rates derived from this data may no longer represent current rates for *Salmonella* in chicken parts. The prevalence observed in the data could be viewed as an “upper limit” for the range of prevalence that can be expected. To address these limitations, what-if analyses were conducted to explore the impact of the initial concentration of *Salmonella* in chicken breasts (Fig. 5, Table 5).

Third, we made a simplifying assumption that only one sample is collected per lot and that all lots are sampled, yielding 500 samples per establishment. It is well recognized that foodborne pathogens are heterogeneously distributed in food and, particularly, in meat and poultry products. As a result, it is unlikely that a single sample would be representative of an entire production lot. In addition, the probability of detection, or the probability of a sample to exceed a concentration threshold, are highly dependent on variability within a lot. As seen from the what-if analyses on variance partitioning, the risk reduction for Scenario 2 (lot-level) was much higher when the within-lot variability was lower, i.e. more uniform, and, hence, even one sample could more reliably represent the contamination level in the whole lot. While a more detailed assessment of these sampling parameters was not conducted in this study, the model could be further applied to explore a wider range of sampling protocols, e.g. involving multiple samples per lot, sampling only a subset of lots, or adopting a sampling frequency based on previous testing outcomes.

Fourth, to keep the focus of the study on the considered risk management question, a simplifying assumption was made that no growth or die-off occurred between the time when the sample was collected and the time when the consumer prepared the chicken breast (i.e. it was assumed the product was maintained at or below 40°F throughout the process). Evidence suggests that there is variation in the temperature of retail refrigerators as well as home refrigerators, and therefore fresh meat and poultry could be exposed to temperatures above 41°F (Bruhn et al., 2014; EcoSure, 2008), allowing for growth of *Salmonella* to potentially occur. The consumer handling portion of the model also did not consider freezing and thawing practices or the associated potential die-off, growth, and cross contamination. Theoretically, a growth event could increase the preventable fraction. However, any growth would occur in both the baseline year and in the post-intervention year, thus limiting the effect of this assumption on the relative risk estimates.

Fifth, NHANES data was used to estimate the distribution of usual intake of chicken breasts for the U.S. population with the assumption that the 24-h dietary recall data collected from NHANES participants was representative of the entire U.S. population, which it may not be. Misreporting and underreporting of food intake using 24-h dietary recalls, including those used by NHANES, is not uncommon and can lead to biased estimates of usual intake (Ahluwalia et al., 2016; Dwyer, 2003). To reduce this potential bias, NHANES collects two 24-h dietary recalls. Given the frequency of consumption (7.5% of NHANES participants), chicken breast is considered a ubiquitously consumed food, which is defined to be foods that are consumed every day by more than about 5% of the population (CDC, 2011c). Advanced methods have been developed to estimate usual intake of ubiquitously consumed foods, but these methods require that a sufficient number of participants reported consuming the food on at least two days (Herrick et al., 2018). Of the 599 participants who reported consuming chicken breasts on Day 1, 283 (47.25%) also reported consuming chicken breasts on Day 2. Since this is less than the 85% deemed sufficient for using more sophisticated methods for estimating usual intake, we conducted a basic analysis that utilized only one day of 24-h recall data to estimate usual intake of chicken breast for this study, which may have under- or over-estimated portion sizes and, consequently, affected the potential dose of *Salmonella* ingested. Further, since only 35 participants reported consuming chicken breasts multiple times on Day 1, we used the total grams consumed during the day to estimate portion size, which likely over-estimated the usual intake for a portion. Exploring the impact of these assumptions on the results was outside the scope of this project but would be an opportunity for future research.

Sixth, assumptions were made in estimating exposure and probability of illness. For instance, only three potential pathways for exposure (ingesting undercooked chicken breast meat, touching the meat and then touching the mouth, and ingesting a cross-contaminated ready-to-eat dish such as a vegetable salad) were modeled, and a dose-response relationship based on outbreak data not related to U.S. poultry was used. There are several other possible exposure routes (e.g. handling at retail) that were not included because they would have shifted the focus to complex consumer stage models that were beyond the scope of this study. For the exposure pathways included, there was limited information on cooking practices, cross-contamination behaviors, and transfer rates specific to *Salmonella* in chicken breasts. As a result, data from studies on *Listeria monocytogenes* were used as a surrogate, which may result in biased estimates of the ingested dose. The existing dose-response model for *Salmonella* used here is known to overestimate risk, and does not consider factors such as acquired immunity or increased susceptibility of vulnerable populations. For example, a nonlinear relationship between the frequency of exposure to *Campylobacter jejuni* and the risk of illness has been previously observed, suggesting that acquired immunity may mitigate risk (Havelaar and Swart, 2014). A scaling factor was used to approximately match the baseline risk to epidemiological estimates and mitigate overestimation; however, since it was applied to both baseline and

intervention scenarios, the preventable fraction estimate was not substantially affected.

Seventh, we assumed that lots exceeding the concentration-based MC would be further processed using control methods that were able to ensure the safety of the product. Therefore, for simplification the risk of *Salmonella* infection in those further processed products was assumed to be reduced to zero, which may not be representative of current feasible pre-market control measures. Even so, this approach provides a way to clearly partition risk between compliant and non-compliant lots, and provides an upper best-case estimate of risk reduction. It bears repeating that the effect of a concentration-based MC is highly dependent not only on the concentration threshold, but also on the concentration distribution across and within product lots.

Finally, the establishment-level intervention (a review of the establishment's food safety plan) was assumed to result in a fixed 3 ln cells/g reduction (1.3 decimal Logs) of *Salmonella* in the post-intervention period; no assumptions were made as to how establishments achieved this reduction. As expected, the mean probability of illness decreased as the Log reduction associated with the establishment-level intervention increased. Increasing the decimal Log reduction for the establishment-level intervention from 0.5 to 2 increased the preventable fraction from 0.420 to 0.887, suggesting that changes are incremental and could be substantial. It is recognized that the observed risk reduction is driven by the high proportion of simulated establishments (87%) that exceeded the prevalence MC threshold (15%), and that different testing assays and prevalence thresholds could lead to different risk reduction levels. In addition, actual interventions may change not only average concentration levels, but also variability or the shape of the concentration distribution. We considered a range of establishment-wide reduction in what-if analyses, and the model could be further applied to test other interventions.

Overall, this work presents a risk-based approach to assessing the effectiveness of different MC and provides a model that could be used to further explore relevant factors and inputs. There are several data gaps that, if addressed, would significantly improve the current model. Improving our understanding of the distribution of concentration of *Salmonella* in chicken parts within and across establishments and lots would be beneficial but would require more data than regulatory agencies typically collect. Similarly, the model would be improved with additional information on industry practices, particularly in terms of sampling and within-establishment actions taken in response to sampling results. As part of their normal production activities, poultry establishments presumably collect a significant amount of information that, in conjunction with novel data collection efforts, could be leveraged to fill these data gaps.

In decision-making around risk management options, it is important to remember that there are two dimensions of risk: the likelihood and the severity. To date, most food sampling strategies have considered how often a product is contaminated (i.e. likelihood of contamination) but have not considered how much contamination is present (i.e. severity of risk). We found that utilizing a concentration-based MC to mitigate the risk associated with products with higher levels of contamination reduced risk of illness. While specific risk management applications for product with high levels of contamination were not explored, this study can inform decision-making around the implementation of a prevalence-based versus concentration-based MC.

5. Conclusion

In summary, study findings suggest the following: (1) Both prevalence-based and concentration-based MC may substantially lower risk from *Salmonella* in chicken parts; (2) A combination approach including establishment-level and lot-level interventions could be highly effective in reducing risk; (3) While scenario outcomes cannot be compared directly to each other due to differences in assumptions, trends emerge when considering the impact of individual variables, e.g. the MC threshold; (4) The high variance in the risk outcomes suggests caution in interpreting results based on mean estimates; (5) The model is limited by several assumptions and data gaps, for instance in the distribution of concentrations, the association between prevalence and concentration, and differences among establishments and among lots; increased data collection would help characterize these variables and strengthen estimates by reducing uncertainty; (6) Overall, while the model is preliminary and subject to the stated limitations, it is likely that public health would be improved if any of the considered MC approaches were adopted, with the greatest benefit arising from a combination approach.

Declaration of Competing Interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome. We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We confirm that we have given due consideration to the protection of intellectual property associated with this work and that there are no impediments to publication, including the timing of publication, with respect to intellectual property. In so doing we confirm that we have followed the regulations of our institutions concerning intellectual property. We understand that the Corresponding Author is the sole contact for the Editorial process (including Editorial Manager and direct communications with the office). He/she is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs. We confirm that we have provided a current, correct email address which is accessible by the Corresponding Author and which has been configured to accept email from kowalcyk.1@osu.edu.

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Supplementary materials

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