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Ihab Habib, John Coles, Mark Fallows, Stan Goodchild

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Human campylobacteriosis related to cross-contamination during handling of raw chicken meat: application of quantitative risk assessment to guide intervention scenarios analysis in the Australian context

Ihab Habib ^{a, b, c*}, John Coles ^d, Mark Fallows ^d, Stan Goodchild ^d

^a *Veterinary Medicine Department, College of Food and Agriculture, United Arab Emirates University (UAEU), Al Ain P.O. Box 1555, UAE*

^b *School of Veterinary Medicine, Murdoch University, 90 South Street, Murdoch Western Australia 6150, Australia*

^c *High Institute of Public Health, Alexandria University, 165 ElHoreya Road, Alexandria, Egypt*

^d *Department of Health Western Australia, 189 Royal Street, East Perth Western Australia 6004, Australia*

*** Corresponding author at:**

College of Food and Agriculture, UAEU, Al Ain, United Arab Emirates

E-mail address: i.habib@uaeu.ac.ae (I. Habib)

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ABSTRACT

Quantitative Microbiological Risk Assessment (QMRA) is a methodology used to organize and analyze scientific information to both estimate the probability and severity of an adverse event as well as prioritize efforts to reduce the risk of foodborne pathogens. No QMRA efforts have been applied to *Campylobacter* in the Australian chicken meat sector. Hence, we present a QMRA model of human campylobacteriosis related to the occurrence of cross-contamination while handling raw chicken meat in Western Australia (WA). This work fills a gap in *Campylobacter* risk characterization in Australia and enables benchmarking against risk assessments undertaken in other countries. The model predicted the average probability of the occurrence of illness per

serving of salad that became cross-contaminated from being handled following the handling of fresh chicken meat as 7.0×10^{-4} (90% Confidence Interval [CI] $\pm 4.7 \times 10^{-5}$). The risk assessment model was utilized to estimate the likely impact of intervention scenarios on the predicted probability of illness (campylobacteriosis) per serving. Predicted relative risk reductions following changes in the retail prevalence of *Campylobacter* were proportional to the percentage desired in the reduction scenario; a target that is aiming to reduce the current baseline prevalence of *Campylobacter* in retail chicken by 30% is predicted to yield approximately 30% relative risk reduction. A simulated one-log reduction in the mean concentration of *Campylobacter* is anticipated to generate approximately 20% relative risk reductions. Relative risk reduction induced by a one-log decrease in the mean was equally achieved when the tail of the input distribution was affected—that is, by a change (one-log reduction) in the standard deviation of the baseline *Campylobacter* concentration. A scenario assuming a 5% point decrease in baseline probability of cross-contamination at the consumer phase would yield relative risk reductions of 14%, which is as effective as the impact of a strategic target of 10% reduction in the retail prevalence of *Campylobacter*. In conclusion, the present model simulates the probability of illness predicted for an average individual who consumes salad that has been cross-contaminated with *Campylobacter* from retail chicken meat in WA. Despite some uncertainties, this is the first attempt to utilize the QMRA approach as a scientific basis to guide risk managers toward implementing strategies to reduce the risk of human campylobacteriosis in an Australian context.

Keywords: *Campylobacter*; Public Health; Risk mitigation; Western Australia

1. INTRODUCTION

Campylobacter spp. are a common cause of bacterial gastroenteritis in humans. A few hundred of these bacteria can induce clinical gastrointestinal symptoms. Infection is generally manifested as self-limiting diarrhea that lasts three to five days, although, in some cases, infection may progress to bloody diarrhea and pose life-threatening consequences (Hansson et al., 2018). Extensive research in some countries has identified that cross-contamination occurring

while handling fresh (rather than frozen) broiler chickens is a significant source of *Campylobacter* infection (Boysen et al., 2014; Havelaar et al., 2007; Mughini Gras et al., 2012). Compared to cross-contamination, several studies have indicated that undercooking plays a limited role in the foodborne exposure to *Campylobacter* (Al-Sakkaf, 2015; Lubber, 2009). In the context of *Campylobacter* and chicken, cross-contamination in the kitchen environment may occur directly from fresh meat either on cooked meal components or indirectly via hands, cutting boards, and/or knives (Lubber et al., 2006).

A study in Australia reported that 75% (95% confidence interval [CI] 67%–83%) of cases of *Campylobacter* infection might be due to foodborne transmission (Hall et al., 2005). Stafford et al. (2008) estimated that the foodborne risk factor with the highest attributable risk to *Campylobacter* infection in persons ≥ 5 years of age in Australia was cooked chicken, with an estimated median of 21.2%; followed by undercooked chicken, with an estimated median of 8.1%. Despite its significance as a critical food safety challenge, there have been few published reports on *Campylobacter* in the Australian poultry meat in the past ten years. *Campylobacter* was found in 87.8% and 93.2% of surveyed chicken portions and carcasses sampled at the retail level in New South Wales and South Australia, respectively (2005 to 2006) (Pointon et al., 2008). A national survey coordinated by Food Standards Australia New Zealand (FSANZ) indicated that, in Western Australia and New South Wales, the *Campylobacter* concentration of positive whole chicken carcasses sampled at the end of abattoir processing (rather than retail) was, on average, $0.70 \log_{10} \text{ CFU/cm}^2$. In Queensland, where only counts $>100 \text{ CFU/ml}$ were quantified, the mean concentration was $1.45 \log_{10} \text{ CFU/cm}^2$ (FSANZ, 2010). Nationally representative quantitative data based on the enumeration of *Campylobacter* per gram of chicken are insufficient in Australia. Such data are required to fill a gap in the assessment of the

microbial safety of Australian poultry and could be used as an input for further development of Quantitative Microbial Risk Assessment (QMRA) of *Campylobacter* in Australia.

QMRA is a valuable tool for the characterization of complex exposure pathways that contribute to adverse human health outcomes (Nauta et al., 2009). Hurdles concerning harmonization of data collection and the uncertainty about contamination levels in poultry across the different states are among several factors challenging the development of a “national” risk assessment approach for *Campylobacter* in chicken in Australia (Pointon et al., 2008). The Australian chicken meat industry is predominantly vertically integrated; with the two largest integrated chicken companies supply more than 70% of Australia’s meat chickens (ACMF, 2017). The objective of this study is to predict, using a Monte-Carlo simulation model, the risk of illness per serving due to the transfer of *Campylobacter* as a result of cross-contamination from contaminated chicken to other foods (e.g., salad) in an Australian home kitchen context. This result is used as baseline to assess and compare the relative effects of interventions on the public health risk. Scenarios are presented to examine the impact of specific intervention strategies on the probability of illness per serving. The model was developed using baseline microbiological data generated explicitly in Western Australia (WA), a state occupying the entire western third of Australia. In 2016, WA produced 110-115,000 tons of poultry meat, 19% of which were interstate imports (Western Australian Agriculture Authority, 2017). This QMRA model provides information to risk managers in the Australian chicken meat industry and controlling authorities to help with future refining and benchmarking of effective strategies to reduce the risk of human campylobacteriosis.

2. MATERIALS AND METHODS

2.1. The scope of the QMRA model – pathogen/food pathway and setting

The considered pathogens refer to thermophilic species of *Campylobacter*. In Australia, the dominating species isolated from chicken meat are *C. jejuni* and *C. coli* (Pointon et al., 2008). The food source in this model was fresh (rather than frozen) chicken meat (whole or in parts) presented for retail consumption in WA. The frozen chicken meat was not included because freezing poultry for commercial and retail distribution has been associated with low *Campylobacter* exposure risk (Hansson et al., 2018). The food pathway was simulated using a modular process model approach (Figure 1), which focuses on quantifying *Campylobacter* exposure from retail through to human consumption as a function of consumer handling behavior during meal preparation (Nauta and Christensen, 2011). We did not include the effects of transportation to and storage within the kitchen prior to food preparation on the contamination levels of chicken meat because *Campylobacter* spp. do not multiply at room temperature and lower (Hansson et al., 2018). Given that chicken meat is typically cooked prior to consumption, direct exposure to undercooked chicken meat is assumed to be less critical than is exposure by cross-contamination (Havelaar et al., 2007; Luber et al., 2006); hence, we did not include exposure due to undercooking in the present model. Cross-contamination is defined as the transmission of pathogens from naturally contaminated sources to the finished product.

A cross-contamination pathway was considered in our QMRA, based on a quantitative model described by Kusumaningrum et al. (2004) for simulating transfer from contaminated chicken carcasses via unwashed surfaces to salad vegetables in the domestic kitchen. This model provided the full mathematical details for conversion of the level of contamination on cucumber slices (expressed in CFU per square centimeter) based on the consumption size (expressed in a weight unit (grams)) (Kusumaningrum et al., 2004). Cucumber slices were used as a

model/surrogate for salad vegetables (Figure 1). This route was chosen because cucumber is a typical salad component that is frequently cut to small pieces or slices. The likelihood of cucumber slices become cross-contaminated with *Campylobacter* from fresh chicken exists if both were prepared using unwashed common surfaces (kitchen bench or cutting board) or knives. It should be noted that the simulated scenario adopted in the present study is specific to the chicken cross-contaminated cucumber that is included in the salad. Other routes of cross-contamination (e.g., fingers contamination) might vary; hence, the results based on this cucumber-specific model might not be generalized for all pathways of cross-contamination occurring while handling raw fresh chicken.

2.2. Exposure assessment

2.2.1. Retail

The model was developed using inputs data from a baseline microbiological survey on *Campylobacter* in WA retail. Our group purposively designed that survey with the aim of generating data to feed the future development of QMRA; the full details of this survey are published elsewhere (Habib et al., 2019). Raw poultry products (n = 315) were purchased for a year (2016–2017) from retail supermarkets in metropolitan Perth, WA. *Campylobacter* concentration was determined by a direct plating method in all samples, whereas in 59.0% (186/315) of the samples, testing was done using enrichment culture in conjunction with direct plating, to reduce uncertainty arising from testing methods misclassification (Habib et al., 2019).

The prevalence at retail (P_{ret}) was 53.7% (100 *Campylobacter*-positive samples out of 186 total samples tested) and was modeled using a *Beta* distribution (Table 1). Using a standard direct plating method, *Campylobacter* were recovered from almost 23.8% [75/315] of the retail chicken

meat samples; the results were used to feed into the QMRA model. The quantification (N_{ret}) input variable (Table 1) was the 10-based log of the concentration at retail ($\log N_{ret}$) which is modeled using a *Normal* distribution with mean=1.82 log CFU/g and standard deviation (SD)=2.26 (Habib et al., 2019). The number of *Campylobacter* (CFU) on one serving of consumed meat, $N_{serving}$, is defined by a Poisson distribution ($N_{serving} \sim Poisson(N_{ret} \times W_{serving})$) (Table 1); where \sim represents “is distributed from,” and $W_{serving}$ represents the serving sizes (Nauta and Christensen, 2011). $W_{serving}$ refers to the typical chicken portion size consumed by Australian adults based on the 2011–12 National Nutrition and Physical Activity Survey (NNPAS), where a portion was defined as the amount of foods consumed per eating occasion (Zheng et al., 2016). Based on NNPAS data, chicken serving sizes in the present model are sampled from a *Log-Normal* distribution with mean=142 g, SD=127, and the distribution was truncated assuming a maximum chicken portion size of 1 kg [such high limit has a very low probability] (Neves et al., 2018). The retail module was used to estimate the number of organisms that “arrive in the kitchen” and hence are potentially transferred via cross-contamination.

2.2.2. Consumer handling and meal preparation

For the consumer phase module, we utilized the quantitative *Campylobacter* cross-contamination model (from retail chicken to salad vegetables, e.g., cucumber slices) of Kusumaningrum et al. (2004). The prevalence of salad vegetable contamination (P_v) and an estimation of the level of that contamination (C_v) on salad serving in relation to CFU per square centimeter (cm^2) of the chicken (surface concentration) are determined as follows:

$$P_v = P_{ret} \times P_{cross}$$

$$C_v = N_{serving} \times T1/100 \times T2/100$$

Where P_{ret} = a probability distribution describing the prevalence of contaminated chicken meat at retail; P_{cross} = a probability distribution describing the frequency of cross-contamination in domestic kitchens; $N_{serving}$ = the level of microorganisms on contaminated chicken carcasses (CFU/cm² carcass); $T1$ = the transfer rate (in %) from chicken risk serving to surfaces; and $T2$ = the transfer rate (in %) from surfaces to salad vegetables. The inputs describing the transfer rates for *Campylobacter*, ($T1$) and ($T2$), were included in the model using log-normal and logistic probability distributions (Table 1), respectively, based on distribution parameters described in the QMRA model of Signorini et al. (2013); these parameters are based on data of experimental cross-contamination using “naturally contaminated” chicken meat rather than spiked samples (Luber et al., 2006). Realizing that only the cells that are present on the outer layer of the meat can be transferred (initiate the cross-contamination process), the variable $Wx_{serving}$ was included in the model to mind the number of cells on outer contact side (=15%) of chicken serving that can give rise to transmission of the pathogen (Table 1). The fixed value of the variable $Wx_{serving}$ was driven from a risk assessment study by Uyttendaele et al. (2006), based on their calculations of the outer contact side of chicken meat preparation that can give rise to transmission of the *Campylobacter*.

A distribution describing the frequency of cross-contamination in domestic kitchens, P_{cross} , was estimated based on a “national” Australian food safety telephone survey that involved a sample of 1,203 randomly selected Australian households (Jay et al., 1999). The study indicated that the percentage range of incorrect responses to hypothetical food preparation situations was 18.0 to 81.4%. For the survey question “when you cut raw vegetables or any raw produce and need to use that surface again for something you are not going to cook (or something that has already been cooked)”, an unsafe food handling practice (reuse the surface as is, or wipe the

surface with a damp cloth) that would lead to cross-contamination of the ready-to-eat food with microorganisms from the raw food would be performed by 38.1% of respondents. Hence, P_{cross} was modeled using a *Pert* distribution with a minimum of 18%, a mode of 38% and a maximum of 81%. Studies on the Australian consumers' handling and cooking raw poultry are scarce. We do acknowledge that the study of Jay et al. (1999) was undertaken in Australia almost twenty years ago; however, the *Pert* distribution constructed based on this study is still comparable (around the mode value (38%)) with results from other, more recent, studies in neighboring New Zealand, as well as from different industrialized countries. For instance, a New Zealand study (2007) has indicated that 28-41% of consumers allow cross-contamination to occur in their kitchen (Gilbert et al., 2007). A study in the U.S. reported that nearly 70% of adult grocery shoppers washing or rinsing raw poultry before cooking it, a potentially unsafe practice because "splashing" of contaminated water may lead to the transfer of pathogens (e.g., *Campylobacter*) to other kitchen surfaces (Kosa et al., 2015).

The number of *Campylobacter* per serving of salad (C_e) was calculated by multiplying the level of contamination on cucumber slices (C_v) (CFU per cm^2) by the cucumber serving size (cm^2) (Kusumaningrum et al., 2004). The variability in serving weight of cucumber was modeled using a *Log-Normal* distribution (with mean=26 g, SD=12g), based on the typical portion size consumed by Australian adults as indicated in the 2011–12 NNPAS (Zheng et al., 2016). Because the consumption size was expressed in a weight unit (grams) while the level of contamination on cucumber slices was expressed in CFU per square centimeter, the weight of the serving size was transformed to square centimeters using a transformation formula described by Kusumaningrum et al. (2004):

$$T_{w-s} (\text{cm}^2 \text{ serving}) = (w_{\text{consumption}}/w_{\text{slice}}) \times \pi d^2/4$$

Where $w_{consumption}$ = the weight of vegetable consumption (grams); w_{slice} = the weight of a cucumber slice with an approximate thickness of 0.3 cm (grams); and d = the diameter of the cucumber slice (cm). The w_{slice} and d values of the cucumber slices were 6.4 ± 0.8 g, and 4.0 ± 0.2 cm, respectively as measured experimentally (Kusumaningrum et al., 2004).

2.3. Hazard characterization

The dose-response is the relationship between the ingested levels of *Campylobacter* (C_e) per salad serving and the probabilities of consequent human campylobacteriosis (probability of infection (P_{inf}), and the probability of illness given (conditional probability) infected (P_{ill})). First, the probability of illness per *Campylobacter* dose (P_{dr}) in a salad serving was estimated using the Beta-Poisson Model developed by the Joint FAO/WHO activities on Risk Assessment of Microbiological hazards in food (FAO/WHO, 2009). A Beta-Poisson model was adjusted to estimate the probability of infection per one bacterium ($P_{inf}(I)$) for an individual consuming a meal with a specific dose (C_e) (Lindqvist and Lindblad, 2008; Signorini et al., 2013).

In the presented model, exposure is a function of the proportion of meals where fresh chicken meats are prepared along with salads. Thus, with the probability of illness per serving of salad ($P_{ill-riskserv}$) was estimated by multiplying the predicted P_{dr} value by the probability that salad vegetables are contaminated (P_v) (Kusumaningrum et al., 2004; Signorini et al., 2013).

2.4. Simulation setting and software

The model was created in Microsoft Excel 2010 with the add-on package @Risk (version 7.5, Palisade Corporation, New York, USA). A Monte-Carlo simulation with Latin-Hypercube sampling was carried out to simulate the distribution of contamination probabilities and levels of *Campylobacter* spp. in salad vegetables as a result of cross-contamination. The predicted probability of illness per salad serving ($P_{ill-riskserv}$) was estimated through 50,000 iterations. Each

iteration predicts a probability of illness for a random scenario of cross-contamination during consumer handling and meal preparation. The number of iterations provided adequate convergence (<1% change in the simulation statistics) (Cassin et al., 1998). A sensitivity analysis was conducted, using the Spearman rank correlation coefficient (r), to determine the impact of each input variable and the uncertain variables on the predicted model output ($P_{ill-riskserv}$). The closer the value of r is to 1, the higher the correlation, and thus the more important the factor is for the variability in the process (Busschaert et al., 2011).

2.5. What-if scenarios

The risk assessment model was utilized to estimate the likely impact of intervention strategies scenarios on the probability of illness (campylobacteriosis) per serving of salad cross-contaminated after handling of fresh chicken meat. Guided by analyzing the model sensitivity analysis, eight scenarios for reducing the predicted probability of illness were examined, as presented in Table 2. These scenarios are aiming at: (i) reducing the prevalence of *Campylobacter*-positive fresh retail chicken meat, (ii) reducing the concentration of *Campylobacter* spp. on retail chickens, and (iii) consumer campaign aiming toward improving the range of incorrect hypothetical food preparation situations leading to cross-contamination. Note that the intervention strategies scenarios (i) and (ii) are assumed to be implemented somewhere along the production chain, at primary production or during industrial processing. Hence, they do not affect the consumer phase model itself. The ratio of the predicted value in an intervention scenario to that in the baseline scenario is used to measure the expected effectiveness of the intervention. Thus, relative risk reductions were calculated for the what-if scenarios, for both models before and after an intervention, using 50,000 iterations in @Risk.

Relative risk reduction was presented as one minus the quotient of *Campylobacter* probabilities of illness *after* implementation of the control measure and *before* (Nauta et al., 2007).

3. RESULTS

An intermediate prediction of the model, the concentration of *Campylobacter* in a cross-contaminated salad serving after handling of fresh chicken meat, is given in Fig. 2 [A]. The predicted average log concentration of total *Campylobacter* per serving of salad (C_e) was 2.76 \log_{10} CFU/serving (SD= 1.50 \log_{10}). The average prevalence of such contaminated salad servings (P_v) was predicted by the model to be 22.4% (Fig. 2[B]).

Fig. 3 shows the simulated cumulative density function of the probability of illness per serving of cross-contaminated salad after handling of fresh chicken meat. Each iteration predicted a probability of illness for a single serving. The presented Monte-Carlo simulation summarizes the results from 50,000 iterations, including both the variability between servings and the uncertainty about the estimate. The range of this probability extended from 10^{-14} to 10^{-3} , with an average of 7.0×10^{-4} (the 90% Confidence Interval (CI) from @ risk is reported as $\pm 4.7 \times 10^{-5}$, although the value of skewness= 5.0 indicates that CI is not symmetrical around the average estimate). The distribution in Fig. 3 indicates the central tendency (50%) of the distribution at risk 10^{-4} . Log probability of illness was chosen as a convenient representation of the probability of risk, which is so concentrated near zero that it was not useful to display on a linear scale. All of the following results related to sensitivity analysis and relative risk reduction estimation were done utilizing the simulated value of the probability of illness (on a linear scale and not log probability of illness).

The sensitivity analysis indicated that the predicted probability of illness per salad serving cross-contaminated with *Campylobacter* after handling of fresh chicken meat was most sensitive ($r= 0.82$) to the input distribution (in the dose-response model) describing the probability of infection from an ingested *Campylobacter* (Fig. 4). Other factors included; probability of cross-contamination in domestic kitchens, number of *Campylobacter* in retail fresh chicken meat, probability of illness given infection, variability in serving weight of cucumber, transfer rates from surfaces to salad vegetables, prevalence of *Campylobacter* in retail fresh chicken meat, and transfer rates from chicken serving to surfaces (Fig. 4).

The model was used to evaluate the effect that a change in an assumption will have on the predicted risk to human health. Figure 5 presents a comparison of the efficacy of the eight (hypothetical) scenarios (Table 2). The per-serving probability of illness under the original model was considered the baseline and the effectiveness of the various strategies was expressed as the predicted relative risk reductions. As shown in Fig. 5, predicted relative risk reductions after a change in prevalence were proportional to the percentage of the desired percentage of reduction; for instance, a strategic target aiming to reduce the current baseline prevalence of *Campylobacter* at retail chicken by 30% is predicted to yield around 30% relative risk reduction. Considering quantitatively set targets; a one-log decrease in the mean concentrations of *Campylobacter*, which is equivalent to a 10-fold reduction of the concentrations, is predicted to yield around 20% relative risk reductions (Fig. 5). Relative risk reduction induced by a one-log decline in the mean was equally achieved when the tail of the input distribution is affected—that is, by a change (one-log reduction) in the standard deviation of the baseline *Campylobacter* concentration (Fig. 5). The previously described intervention strategies were assumed to be

implemented somewhere along the production chain, at primary production or during industrial processing.

A consumer concerned intervention approach was also evaluated. A scenario assuming a 5% point reduction (from 38% to 33%, which is relatively a 13% decrease) in the probability of cross-contamination, compared to the baseline model, would yield relative risk reductions of 14% (Fig. 5); this was almost as effective as the impact of a strategic target of 10% reduction in *Campylobacter* prevalence at the retail chicken (going from 100/186 to 90/186; which is equal to only a 5.4% point reduction).

4. DISCUSSION

The model described in this research predicted the distribution of probability of illness attributable to *Campylobacter* cross-contamination during the handling of fresh chicken meat in an Australian context (WA). The model predicted risk by integrating current microbial food safety data, evidence from relevant literature, and techniques of QMRA. The model utilized probability distributions to more effectively describe variability and uncertainty in the estimates of model parameters (Montville and Schaffner, 2005). The presented baseline risk model is a representation of “the most likely situation” based on dedicated microbiological survey data on *Campylobacter* contamination in retail chicken meat in WA between 2016 and 2017 (Habib et al., 2019).

A QMRA approach for *Campylobacter* in chicken meat could be much needed now more than ever in the Australian context. Australia has one of the highest rates of *Campylobacter* infection in the developed world; being around ten times that of the United States and double that seen in the European Union (Moffatt et al., 2017). In April 2017, the Australia and New Zealand

Ministerial Forum on Food Regulation identified the development of a strategy to reduce foodborne illness, mainly related to *Campylobacter* in Australia, noting New Zealand has an existing *Campylobacter* strategy, as a priority area for 2017–2021 (Anonymous, 2017). To meet such evolving food safety priority in Australia, there is a need to develop a science-based, objective-oriented strategy in collaboration between controlling authorities and industry, with input from research and community. To this end, QMRA has emerged as a useful tool in enabling food safety risk manager to consider and compare management and control options (Lammerding and Paoli., 1997). Together with other means, for example, risk ranking, epidemiology (e.g., source-attribution) and economic analysis, QMRA can provide a sound scientific foundation for “risk-based” management systems and control measures (Havelaar et al., 2007). Around twenty QMRAs have been published for *Campylobacter* in broiler chickens over the past two decades by some independent researchers and national food safety agencies worldwide (Chapman et al., 2016; Nauta et al., 2009). However, there are no published models for *Campylobacter* risk assessment in chicken meat in Australia. Thus, the present work fills a gap in the quantitative understanding of *Campylobacter* food safety risk in the Australian context and enables benchmarking against risk assessments undertaken in other countries. The exact numeric results of the presented model are interesting to compare with different model-based approaches; however, it should not be considered as the ultimate output, giving that several simplifications and assumptions were necessary for the risk assessment. We believe that the results from the present model are best suited if considered as baseline incidences that can be used to assess and compare the relative effects of interventions on the public health risk.

In the present risk assessment, the predicted probability of human campylobacteriosis per serving (average of 7.0×10^{-4}) was in agreement with the probability reported in the model of

Uyttendaele et al. (2006) for the risk assessment of *Campylobacter* cross-contamination (and undercooking) during handling chicken meat preparations in Belgium, which was estimated at 7.84×10^{-4} . Also, our result is in line with the average probability (3.32×10^{-4}) predicted by Signorini et al. (2013) in a model of human campylobacteriosis through consumption of salad cross-contaminated with *Campylobacter* from broiler meat in Argentina. The later model predicted the prevalence of salad contaminated with *Campylobacter* to be 32.9%, compared to 22.4% as predicted by our model in Western Australia (Fig. 2[B]). The predicted probability of illness should be interpreted carefully. A particular cross-contaminated serving may pose no risk or a very high risk to an individual considering the process by which it arrived on the plate of the consumer. Some individuals may always experience a more upper or lower risk, due to their age, particular immune-competence, hygienic practices during meal preparation, and eating habits (Cassin et al., 1998). With this regard, it should be noted that Australians consumption pattern of chicken meat is way more than a lot of other nations; the per-capita annual consumption of chicken meat was 44.5 kilograms per person in 2017, setting Australians as the fourth-highest ranked chicken consumers in the world, behind Israel, United States, and Saudi Arabia (OECD, 2017). Considering such high consumption pattern, along with the well-established role of broiler chickens as one of the primary sources of campylobacteriosis (Kaakoush et al., 2015), it is not a total surprise that Australia reports a high number of human campylobacteriosis. In 2016, the reported (all sources) Australian national notification rate of campylobacteriosis was 99.9 cases per 100,000 population, compared to 133.2 reported cases per 100,000 population in WA (Zheng et al., 2016). Because the estimation of risk was not the ultimate purpose of our QMRA, we did not include analyses necessary for the evaluation of predicted annual risk or number of human cases. Thus, the probability of illness predicted by the present model surrogates the risk

experienced by an average person, consuming salad cross-contaminated with *Campylobacter* from retail chicken meat in WA.

The presented QMRA was developed to provide a mean to analyze the relationship between *Campylobacter* probability of illness and possible intervention scenarios. A risk manager is likely to be more interested in the sensitivity analysis (Fig. 4) and comparison of intervention strategies (Fig. 5), than the risk distribution itself (Fig. 3). Sensitivity analysis of the model input parameters identified several factors which contribute significant uncertainty to the ‘total uncertainty’ of the risk of illness prediction. As indicated in Figure 4, a group of these factors is related to the dose-response relationship (probability of illness per a consumed *Campylobacter*, and the probability of illness given infection). Similar to our finding, inputs related to the dose-response relationship were noted as the most important predictive factors of the estimated probability of human campylobacteriosis in the QMRA models of Lindqvist and Lindblad (2008) in Sweden, Signorini et al. (2013) in Argentina, and Uyttendaele et al. (2006) in Belgium, using the same dose-response model that we applied in the present study. The uncertainty arising from the single-hit assumption in the Beta-Poisson dose-response relationship is an inherent caveat in *Campylobacter* risk assessment (Nauta et al., 2007). Data on the infective dose of *Campylobacter* have been widely assumed based on a single human feeding study, which unfortunately provides incomplete and biased information on the dose-response relationship (Black et al., 1998). Such methodological caveat is a well-recognized knowledge gap, as noted by several (inter)national *Campylobacter* risk assessment studies (Chapman et al., 2016).

In addition to the inherent methodological issue associated with *Campylobacter* dose-response relationship, the sensitivity analysis identified several factors related to the consumer phase. The probability of cross-contamination in domestic kitchens was on top of such

consumer-related predictive factors (Fig. 4). Some studies emphasized the importance of cross-contamination, compared to undercooking, as the main kitchen route by which humans are exposed to the pathogen (Al-Sakkaf, 2015; Fischer et al., 2007). Since *Campylobacter* is heat-sensitive, it poorly survives the heat treatment of typical cooking conditions of chicken meat (Al-Sakkaf and Jones, 2012). Data on chicken meat cooking preferences are scarce in Australia. However, a survey of domestic food handling practices in Australia's neighbor New Zealand indicated that none of the respondents (n= 128) preferred raw and rare cooking conditions of chicken roast (Gilbert et al., 2007); supporting our model assumption not to include undercooking given its neglected role compared to cross-contamination. In domestic kitchen, cross-contamination could support the transfer of *Campylobacter* from fresh chicken to ready-to-eat foods and other surfaces, especially in a setting where there is a relatively high chance of preparing chicken before salad, combined with lower chance of washing hands and cutting board (Fischer et al., 2007; Luber et al., 2006). A study by Jay et al. (1999) indicated that almost 25% of 1,203 randomly selected Australian households respondents failed to identify that washing hands before handling food and during food preparation as an important factor in reducing the risk of cross-contamination and possible foodborne illness. The work of Jay et al. (1999) concluded that "*although Australians appear to understand that the home kitchen could be a source of foodborne disease, they generally lack the knowledge to ensure that food preparation in the home is performed so that the risk of illness is minimized*". Hence, available Australian data on consumer food safety practices combined with our sensitivity analysis of the key predictive factors in our model, demonstrate the need for considering consumer education regarding safe food handling practices and its possible impact on reducing the probability of cross-contamination in domestic kitchens.

To guide the selection of a risk mitigation strategy, possible interventions may be deduced from variables that possess predictive contributions to risk (Cassin et al., 1998). The probability of cross-contamination in domestic kitchens may theoretically be a controllable variable through investment in consumer awareness and kitchen hygiene campaigns (Milton and Mullan, 2012). Sensitivity analysis also points to other consumer-related factors (Fig. 4), as transfer rates from chicken to surfaces and from surfaces to salad will consequently be affected by the controlling of cross-contamination—given that the transfer of *Campylobacter* in a kitchen is conditional to the initial occurrence of a cross-contamination incidence. Mylius et al. (2007) identified that the risk of *Campylobacter* infection is proportional to the probability of preparing chicken prior to preparing salad and is negatively related to the probability of washing hands and using a cutting board. The transfer of *Campylobacter* throughout the kitchen environment may be managed by enhancing food safety culture among food preparers—for instance, washing hands before, during, and after meal preparation, separating between fresh chicken meat and ready-to-eat foods, and washing surfaces and cutting boards (Al-Sakkaf, 2015; Havelaar et al., 2007; Jay et al., 1999; Milton and Mullan, 2012).

Aside from methodological and consumer-related factors discussed above, sensitivity analysis (Fig. 4) also pointed to the impact of both concentration and prevalence of *Campylobacter* in retail fresh chicken meat on driving the estimate of the probability of illness. Reduction in *Campylobacter* prevalence in retail chicken requires interventions implementation along the production chain, at primary production or during industrial processing (Hansson et al., 2018; Havelaar et al., 2007; Wagenaar et al., 2013). In our model, the number of *Campylobacter* in retail fresh chicken was ranked third out of the eight predictive factors highlighted in the sensitivity analysis (Fig. 4). The importance of the number of *Campylobacter* in retail was more

evident compared to the predictive impact of *Campylobacter* prevalence (Fig. 4). This finding, stresses the importance of evaluating the applicability and implications of mitigation strategies aiming toward reducing the numbers (mean and/or standard deviation) of *Campylobacter* in retail chicken meat. Chicken meat carrying low numbers of *Campylobacter* pose a lower risk of human infection than those carrying a higher concentration (Duarte and Nauta, 2015). A Danish risk assessment estimated that a reduction of $2 \log_{10}$ CFU/g would reduce the human incidence of infection with *Campylobacter* 30 folds (Rosenquist et al., 2003). Thus, investing in refining a quantitative *Campylobacter* monitoring and process hygiene target could help the Australian chicken meat industry in prioritizing risk-based corrective actions, as well as tracing sources of unacceptable contamination.

Guided by sensitivity analysis, we simulated some possible risk mitigation scenarios, demonstrating the possible application of QMRA for decision-making (Table 2). Using this approach for comparison of possible options de-emphasizes the importance of the actual risk estimate, and emphasizes the relative risk estimates under possible intervention options (Cassin et al., 1998; Signorini et al., 2013). Thus, an advantage of such approach is that the uncertainty of the relative risk is expected to be substantially smaller than the estimate of the risk itself because the uncertainty in the numerator and the denominator may cancel out (Nauta et al., 2007).

Our results show that the differences between the predicted relative risk reductions depend on the parameter targeted by the mitigation scenario (Fig.5). The reader should keep in mind that the reported risk reduction values (Fig. 5) should not be generalized to all chicken-attributed cases (contracted through other ways and settings). Results of the mitigation scenarios in the present work are specific to the risk of infection from chicken cross-contaminated

cucumber that is included in the salad. Relative risk reductions after a change in prevalence (with the distribution of concentration unaltered) was found to be proportional to the percentage of the desired reduction in *Campylobacter* prevalence in retail chicken meat (Fig. 5); similar to what was noted by Nauta and Christensen (2011) comparing seven different published consumer phase models. If the mitigation strategy changes the distribution of concentrations, the models show variable risk reduction rates (Fig. 5). Our result comes to concordance with conclusions from various researchers confirming the importance of the tail of the distribution of *Campylobacter* concentrations, which is driven by the proportion of highly contaminated chicken meats (Busschaert et al., 2010; Busschaert et al., 2011; Nauta et al., 2012). Our recent baseline microbiological survey on *Campylobacter* counts in retail chicken (n=315) in WA revealed that 76.2% of the chicken portions and carcasses were contaminated with <10 CFU/g, and 18.7% of the samples were contaminated with ≥ 100 CFU/g (Habib et al., 2019). In Australia, the use of chlorine in the chiller water during poultry processing is a very well established control point; poultry processing establishments are commonly multistage counterflow with the use of chlorine at a level up to 5 ppm of free available chlorine (Duffy et al., 2014). It has been proposed by Nauta and Christensen (2011) that most practical control measures targeting *Campylobacter* concentrations during industrial poultry processing, like the use of chlorine in the chiller water, are believed to affect the mean concentrations and not the tail of the distribution. However, it could be argued that the use of chlorine, and similar chemical interventions, might “shift” the distribution but not the upper percentiles (i.e., stretching of the distribution and hence large SD).

The probability of cross-contamination in domestic kitchens was on top of the consumer-related predictive factors indicated by sensitivity analysis of the baseline model (Fig. 4). A scenario that assumed a 5% point reduction (theoretical value) in the probability of cross-

contamination (from 38 to 33%, which is relatively a 13% decrease) in domestic kitchens yielded a relative risk reduction similar to that induced by a scenario assumed 10% reduction (going from 100/186 (53.7%) to 90/186 (48.3%); which is equal to only a 5.4% point reduction) in the prevalence of *Campylobacter* in retail chicken meat (Fig. 5). Modification of the probability of cross-contamination is only possible by changing the behavior of those who prepare the food (Havelaar et al., 2007). Several studies have recommended that education is a crucial step in preventing foodborne illness in the domestic environment (Al-Sakkaf, 2015; Jay et al., 1997). However, few educational or psychosocial interventions have been designed and implemented to improve food-safety knowledge, attitudes, and behaviors (Redmond and Griffith, 2006). It is important to note that self-report behavior changes should be verified through observational studies, which have been known to be a very challenging issue in ascertaining the impact of food-safety educational campaigns (Milton and Mullan, 2012).

In recent years, several countries started to develop their own target-oriented, evidence-based *Campylobacter* risk management strategies. The development of such approaches was partly based on the utility of QMRA to formulate voluntary targets, process hygiene criteria (PHC), performance objectives (POs) or even to set microbiological criteria (MC) for *Campylobacter* in chicken meat. Results from a QMRA study by the European Food Safety Authority indicate that adoption of critical limits of 500 or 1000 CFU/g of neck and breast skin, of batches of fresh broiler meat, would lead to 90% and 50% reduction of campylobacteriosis public health risk, respectively (EFSA, 2011). In the Netherlands, using risk assessment model and economic analysis, a PHC has been proposed, with a critical limit set at 1000 CFU/g breast skin, with none of 5 samples per batch exceeding this limit ($c=0$), to reduce the number of human cases by 2/3 (RIVM, 2013). In the UK, the Food Standard Agency jointly with the industry set

together an agreed target to reduce the prevalence of the most contaminated chickens (those with >1000 CFU per g chicken skin) to below 10% at the end of the slaughter process (PHE/FSA, 2017). In the US, for chicken parts and comminuted chicken, the Federal Food Safety and Inspection Service (FSIS) set up a pathogen reduction performance standard for *Campylobacter* designed to reduce human illness. The US Government program “Healthy People 2020” aims to reduce human illnesses from *Salmonella* by 25% and *Campylobacter* by 33% by the year 2020 (FSIS, 2015). The above examples demonstrate the utility of risk assessment as a tool that allows consideration and allocation of resources to potential risk reduction strategies that may be effective. Risk assessment at the same time help with identifying priorities for focused and longer-term research to understand the risk better, and intervene for its management. In Australia, a PHC was established for *Campylobacter* in 2016, with a limit <10,000 CFU for whole chicken carcass at the end of processing (after final chill and just before to dispatch). A “moving window limit” failure occurs when the log count for seven or more out of 45 samples in the moving window are higher than the established limit of <10,000 CFU/carcass (FSANZ, 2018). This PCH might assist the food operators in verifying that the whole process is under control; however, it is not designed to be linked with a targeted reduction in human illness.

A limitation of the present modeling approach is that it has not yet considered the cost of the proposed interventions. Intervention costs must be considered when making policy decisions (Havelaar et al., 2007), which we regard as a priority for the next step of model improvement. This may be achieved through future consultation with stakeholders along the pre-harvest level, production chain, industrial processing, and public health authorities. Another limitation of the model is in the uncertainty regarding data on the Australian consumer handling of chicken as well as in other practices during food preparation at home. In the present model, the probability

estimate of cross-contamination in domestic kitchens was based on a study (Jay et al., 1999) that was undertaken nearly twenty years ago. To reduce the overall uncertainty in the present model, there exists an urgent need to gather more recent data on chicken meat handling and other hygienic practices in modern Australian home kitchens. In addition, there exists a need to generate more updated knowledge on food safety behavior, behavioral intention, attitudes, knowledge, and microbial transfer data applied to the situation in Australia.

There is a limited availability of quantitative baseline data on *Campylobacter* levels in chicken meat in WA. In the past ten years, there is only one quantitative baseline survey of *Campylobacter* in WA; this study reported that 18.7% of the tested chicken meat samples were contaminated with $\geq 2 \log_{10}$ CFU/g, and very few samples were contaminated with $\geq 3 \log_{10}$ CFU/g (Habib et al., 2019). Gathering more quantitative data on *Campylobacter*, especially on chicken carcasses, is very crucial to describe better the variability in contamination levels in Australian context. In future research, given that more baseline data will be generated, it will be important to consider the intervention scenario targeting reducing the upper tail of *Campylobacter* counts distribution; the effect of such intervention is expected be more robust than the scenarios investigating the effect of modifying only the standard deviation of *Campylobacter* counts. The QMRA procedure is not static because the used data, assumptions, and models may be changed when new information (e.g., updated knowledge on consumer handling practices) becomes available (Kusumaningrum et al., 2004). A general limitation in the QMRA methodology is that this approach may be restrictive primarily when applied to foods manufactured in diverse ways and wherein multiple approaches are utilized to manage risks. In such cases, the establishment of intermediate targets may be more desirable and more practical (FAO/WHO, 2009).

5. CONCLUSION

Despite some uncertainties, this model can be used as a scientific basis by risk managers to decide upon strategies to reduce the risk of human campylobacteriosis in WA. Risk management does not end with the selection of an appropriate control measure; rather, it must be followed up with monitoring activities to determine the level of compliance. Too high a level of stringency may reduce the level of compliance, whereas a very high degree of compliance may be achieved with an approach that is slightly less stringent. In addition to monitoring the level of compliance, risk measures must be monitored for efficacy, and after some time, they must be reviewed and revised as needed. In the present work, we have introduced a simple QMRA framework that allows the consideration and comparison of such scenarios to facilitate the selection of the most appropriate risk management option.

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Fig.1. The conceptual framework of the QMRA model of *Campylobacter* spp. related to cross-contamination during handling of fresh chicken meat

Fig.2. Predicted [A] concentration (\log_{10} CFU) of *Campylobacter* in a serving of cross-contaminated salad after handling of fresh chicken meat (C_e); and, [B] prevalence of contaminated salad servings (P_v)

Fig.3. Cumulative ascending distribution for the log probability of illness predicted for *Campylobacter* consumed in a serving of cross-contaminated salad after handling of fresh chicken meat

Fig.4. Sensitivity analysis of the output variable probability of illness predicted for *Campylobacter* consumed in a serving of cross-contaminated salad after handling of fresh chicken meat

Fig.5. Relative risk reduction in the probability of illness (campylobacteriosis) per a serving of cross-contaminated salad after handling of fresh chicken meat. The eight different scenarios reflect the effect of intervention-based hypothetical targets on $P_{ill-riskserv}$ after implementation of the control measure and before (baseline model).

Fig. 1. The conceptual framework of the QMRA model of *Campylobacter* spp. related to cross-contamination during handling of fresh chicken meat

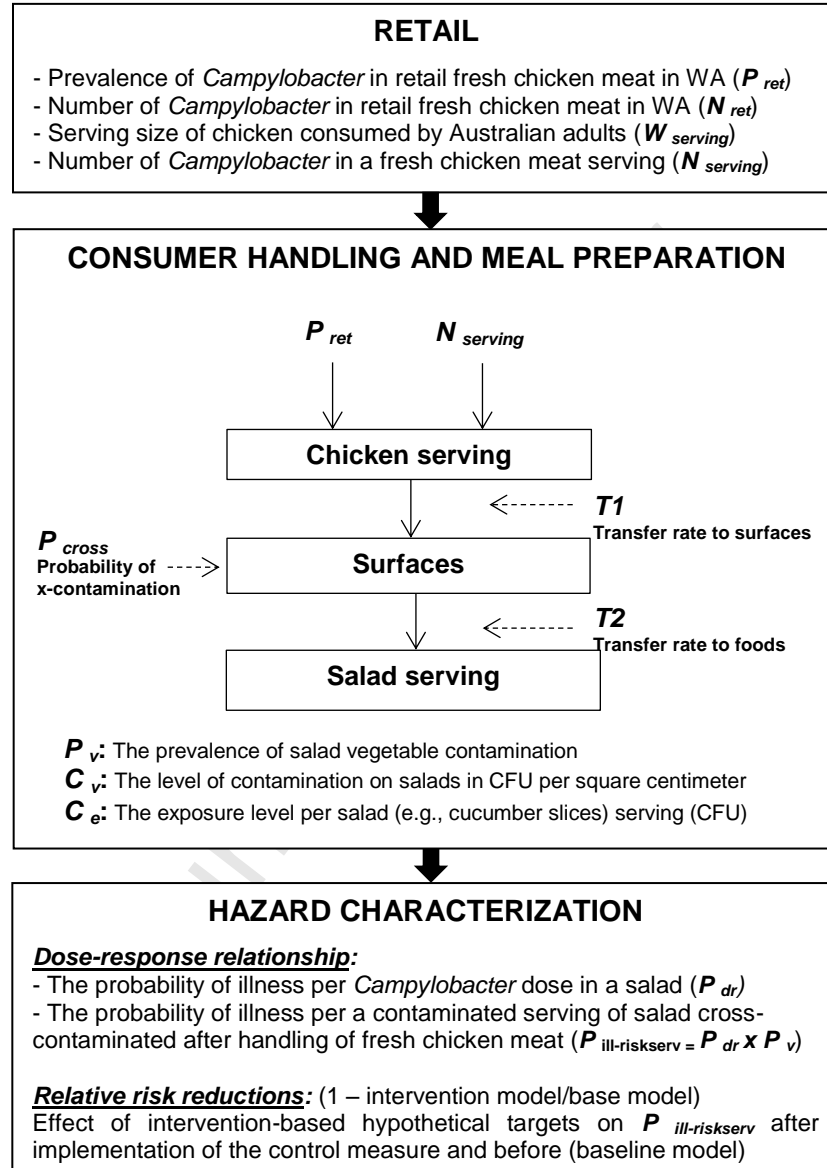


Table 1

Parameters and default values applied in the baseline Western Australia QMRA model

Variable	Description	Units	Distribution/Model
P_{ret}	Prevalence of <i>Campylobacter</i> in retail fresh chicken meat in WA	%	Beta (α_1 ; α_1); where α_1 = positive samples + 1; α_2 = total tested samples - positive samples + 1 RiskBeta (101; 87) (Data source: Habib et al., 2019)
$N_{ret-log}$	Number (log-normal, base 10) of <i>Campylobacter</i> in retail fresh chicken meat in WA	Log ₁₀ CFU/g	Normal (Mean; SD) RiskNormal (1.82; 2.26) (Data source: Habib et al., 2019)
N_{ret}	Number (exact) of <i>Campylobacter</i> in retail fresh chicken meat in WA	CFU/g	$=10^N N_{ret-log}$
$W_{serving}$	The typical serving size of chicken consumed by Australian adults	g	LogNormal (Mean; SD) RiskLogNormal (142g; 127g) Maximum 1000g (Data source: Zheng et al., 2016)
$Wx_{serving}$	Number of cells on the outer contact side (=15%) of chicken serving that can give rise to transmission of the pathogen	g	15% (Fixed) \times $W_{serving}$ (Data source: Uyttendaele et al., 2006)
$N_{serving-ex}$	Number (exact) of <i>Campylobacter</i> in a fresh chicken meat serving	CFU/serving	$N_{serving} \sim$ Poisson ($N_{ret} \times Wx_{serving}$)
$N_{serving-log}$	Number (log-normal, base 10) of <i>Campylobacter</i> in a fresh chicken meat serving	Log ₁₀ CFU/serving	Log ₁₀ ($N_{serving-ex}$)
P_{cross}	The probability of cross-contamination in domestic kitchens		Pert (Minimum; Most likely; Maximum) RiskPert (18%; 38%; 81%) (Data source: Jay et al., 1999)
P_v	The prevalence of salad vegetable contamination	%	$= P_{ret} \times P_{cross}$
$T1$	The transfer rates from chicken risk serving to surfaces	%	RisklogNormal (12.7; 7.0087; shift(0.54332)) Mean of the distribution = 13% (Data source: Signorini et al., 2013)
$T2$	The transfer rates from surfaces to salad vegetables	%	$=$ RiskLogistic (-0.42502; 11.002; shift (3.8223))

			<p>Mean of the distribution = 4%</p> <p>(Data source: Signorini et al., 2013)</p>
$w_{consumption}$	The variability in serving weight of cucumber	g	<p>RiskLogNormal (26; 12)</p> <p>(Data source: Kusumaningrum et al., 2004)</p>
$Trans_{w-s}$	The cucumber risk serving size; the weight (grams) of the serving size was transformed to cm^2	cm^2 serving	Formula = $(w_{consumption}/w_{slice}) \times \pi d^2/4$
C_v	Estimated levels on contaminated salad	CFU/ cm^2	$= N_{serving} \times T1/100 \times T2/100$
C_e	Dose of <i>Campylobacter</i> per risk serving of salad	CFU/serving	$= C_v \times Trans_{w-s}$
$P_{inf(1)}$	The probability of infection from one <i>Campylobacter</i>	—	<p>Beta (0.21; 59.95)</p> <p>(Data source: FAO/WHO, 2002)</p>
P_{inf}	The probability of infection/serving of salad	—	<p>Beta-Poisson Model</p> <p>$1-(1-P_{inf(1)})^{C_e}$</p> <p>(Data source: FAO/WHO, 2002)</p>
$P_{ill/inf}$	The probability of illness given infection (conditional)	—	<p>RiskBeta (30, 61)</p> <p>(Data source: Black et al., 1988)</p>
P_{dr}	The probability of illness per serving of contaminated salad	—	$= P_{inf} \times P_{ill/inf}$
$P_{ill-riskserv}$	The probability of illness per serving of salad cross-contaminated after handling of fresh chicken meat	—	$= P_{dr} \times P_v$

Fig.2. Predicted [A] concentration (\log_{10} CFU) of *Campylobacter* in a serving of cross-contaminated salad (C) and [B] prevalence of contaminated salad vegetables (D).

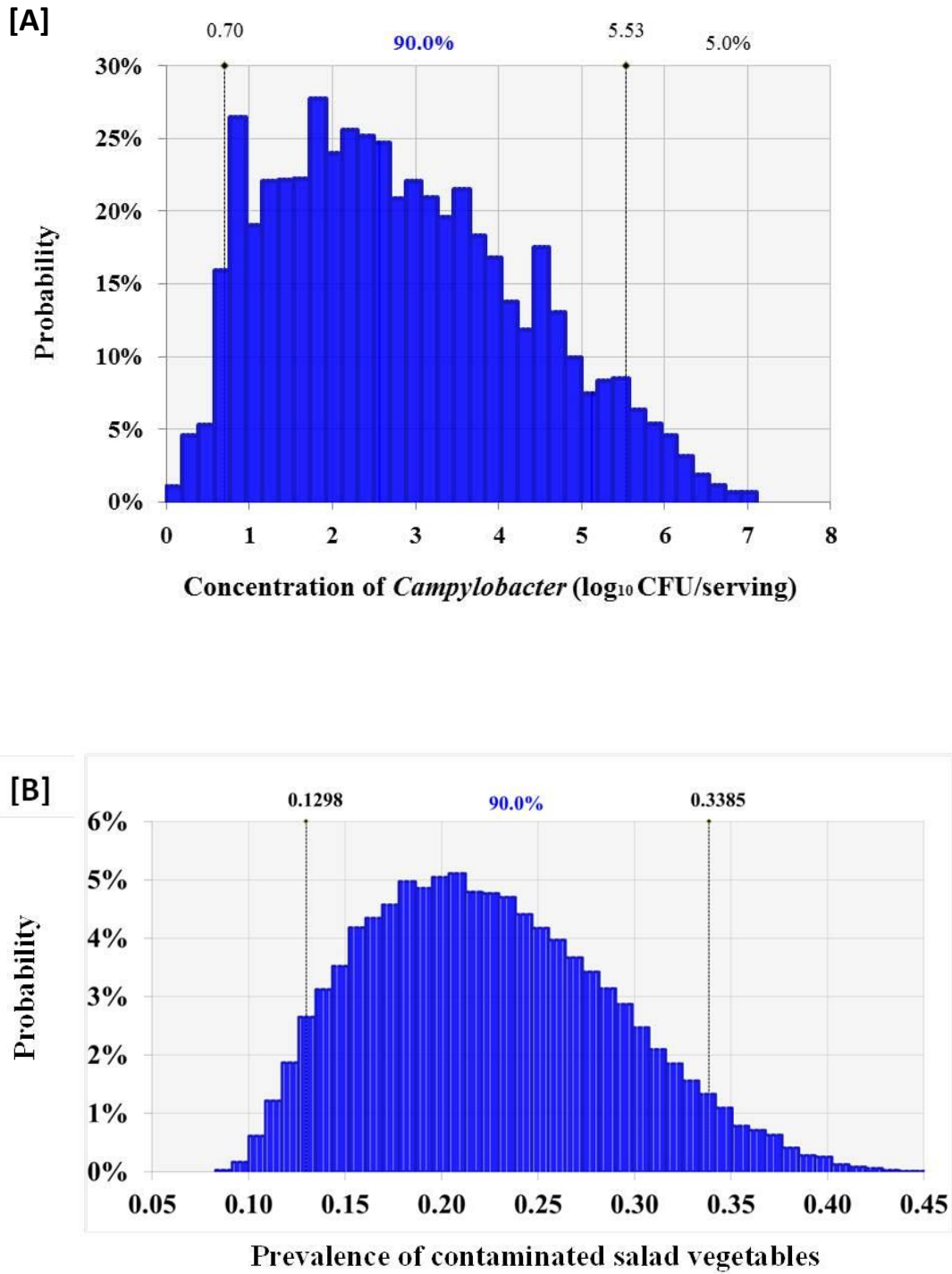


Fig. 3. Cumulative ascending distribution for the log probability of illness predicted for *Campylobacter* consumed in a serving of cross-contaminated salad after handling of fresh chicken meat

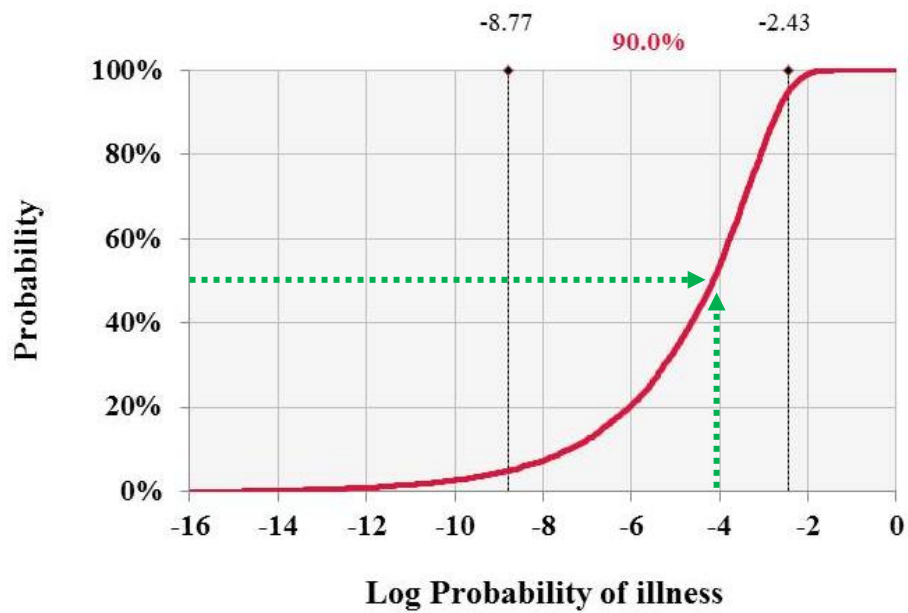


Table 2

Evaluated *Campylobacter* risk mitigation strategies and hypothetical targets compared to the baseline model and the associated new input distribution assumptions

Level	Strategy	Baseline distribution	Scenario/Hypothetical target
Retail	Reduction of the prevalence of <i>Campylobacter</i> in retail fresh chicken meat	RiskBeta (101; 87)	RP10% ; 10% prevalence reduction =RiskBeta((90+1),(186-90+1)) RP20% ; 20% prevalence reduction =RiskBeta((80+1),(186-80+1)) RP30% ; 30% prevalence reduction =RiskBeta((70+1),(186-70+1))
	Reduction of the mean log concentration of <i>Campylobacter</i> in retail fresh chicken meat	RiskNormal (1.82; 2.26)	RM0.5 ; Mean log, 0.5 log unit lower RM1.0 ; Mean log, 1 log unit lower RS0.5 ; SD, 0.5 log unit lower RS1.0 ; SD, 1 log unit lower
Consumer	Consumer campaign aiming at improving the probability of cross-contamination in domestic kitchens	RiskPert (18%; 38%; 81%)	Hygiene 5%* RiskPert (13%, 33%, 75%); Assuming ~ 5% point decrease in the probability of cross-contamination in domestic kitchens.

* This scenario implies a 5% point decrease (the value of the mean goes from 38% to 33%, which is relatively a 13% decrease).

Fig. 4. Sensitivity analysis of the output variable probability of illness predicted for *Campylobacter* consumed in a serving of cross-contaminated salad after handling of fresh chicken meat

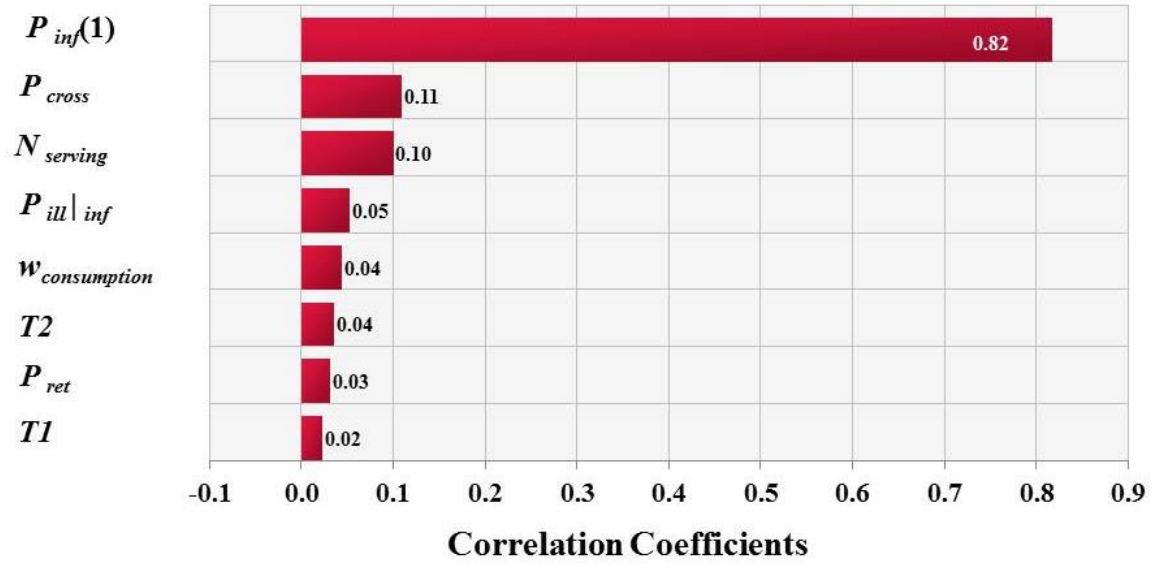
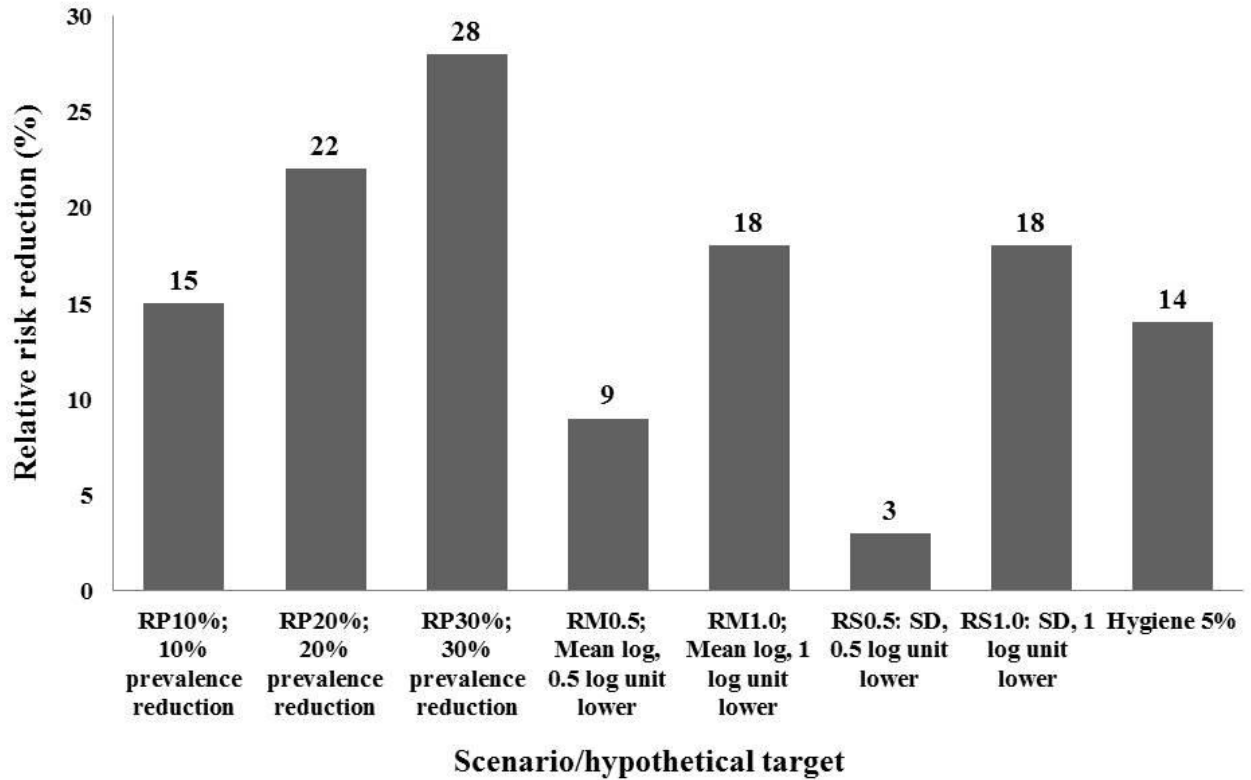


Fig. 5. Relative risk reduction in the probability of illness (campylobacteriosis) per a serving of cross-contaminated salad after handling of fresh chicken meat. The different scenarios reflect the effect of intervention-based hypothetical targets on $P_{ill-riskserv}$ after implementation of the control measure and before (baseline model).



HIGHLIGHTS

- The first published QMRA model for *Campylobacter* in Australian chicken meat
- The model predicts the probability of illness per serving of salad cross-contaminated after handling chicken
- The model was utilized to estimate the likely impact of some intervention scenarios
- The present QMRA is presented to help risk managers in deciding strategies to reduce campylobacteriosis

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