An Individual-Carcass Model for Quantifying Bacterial Cross-Contamination in an Industrial Three-Stage Poultry Scalding Tank

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An individual-carcass model for quantifying bacterial cross-contamination in an industrial three-stage poultry scalding tank

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1. Introduction

Foodborne illness is a major public health concern and economic burden to the world. Of an estimated 582 million cases of foodborne illness, 96 million cases are caused by Campylobacter spp. (Kirk et al., 2015) including 27,738 deaths. While most cases attributed to Campylobacter spp. are diarrheal, more severe events such as Guillain-Barré syndrome are also associated with Campylobacter spp. infections. An estimated 20-150 cases are reported per 100,000 people in industrialized nations each year (Christine et al., 2008). High prevalence and counts of Campylobacter on retail poultry products continue to pose a threat to consumers (Müller-Julie et al., 2010; Suzuki and Yamamoto, 2009). In addition, it is estimated that 24%-46% of processors will not pass 2015 FSIS-USDA Campylobacter performance standards for chicken products (Food Safety US Department of Agriculture and Inspection Service, 2015).

Poultry raised on the farm are transported to the slaughterhouse and then passed on to retail and consumer markets. Foodborne pathogens such as Campylobacter and Salmonella can persist on birds throughout this “farm-to-fork” process, resulting in contaminated poultry products. The poultry slaughterhouse is a site of concern with regard to the possible spread, growth, and persistence of these pathogens. In particular, the World Health Organization has declared the poultry scalding, chilling, defathering, and evisceration processes as sites of major cross-contamination and hence are of key importance (FAO/WHO, 2009). To assist with global efforts in reducing the burden of foodborne disease, we proceed using techniques of mathematical modeling to study pathogens levels in the processing plant.

Here we investigate the high-speed industrial scalding stage where carcasses are hung on a processing line and pulled through heated “scald water”. The scald water thermally inactivates pathogens; however, it is also a medium for which bacteria may transfer between
carcasses. The current state-of-the-art mathematical models describing poultry scalding are designed to predict pathogen levels in a single-pass scald tank; however, modern scalding systems are comprised of three scalding tanks which broilers pass through sequentially (Osiriphun et al., 2012; McCarthy et al., 2018). These existing mathematical modeling works quantify average bacteria levels over all carcasses in the scalding tank for mathematical and computational convenience.

In this work, we adopt a mathematical model quantifying pathogen levels on each carcass in the scalding tank. There are several advantages of this individual-carcass modeling approach over previously used averaging approaches. National government performance standards for Campylobacter and Salmonella are in terms of individual carcass pathogen levels, hence our modeling framework may be of use to industrial processors and policy-makers (Food Safety US Department of Agriculture and Inspection Service, 2015). Pathogen levels on each carcass varies considerably thus this is a natural step in moving toward model realism. Second, with such a model many different scenarios may be weighed in terms of the risk they pose in terms of individual carcass contamination and carcass-to-carcass transmission. One key advantage is that we may quantify the effects of cross-contamination directly, subject to various scenarios. This was an open problem and strong limitation of existing models posed previously (McCarthy et al., 2018).

The objectives of this work are to develop, parametrize, and validate a mathematical model describing the transfer and inactivation dynamics of pathogens on individual carcasses in a three-stage scalding tank. In particular, we aim for this modeling framework to be amenable to integration in Quantitative Microbial Risk Assessment (QMRA) models and have the capacity to weigh various Hazard Analysis and Critical Control Point (HACCP) measures. Long-term goals in this series include the integration of causal, mechanistic models of this form into a complete farm-to-fork QMRA framework. In this work, we provide details of model development; parametrize the model according to specific pathogen C. jejuni; and provide forward validation against industrial processing data. We also illustrate how the model may be applied using configurations for a specific industrial poultry processor in Canada.

2. Model development: an individual-carcass model

We develop an individual-carcass model (ICM) for describing the bacteria transfer and inactivation in a modern three-stage scalding tank. The formulation we present is similar in scope to an existing model describing the contamination dynamics in a poultry chilling tank (McCarthy et al., 2017). We also note that this model may be classified more generally as an individual-based model, where each carcass represents an individual. We then apply the scald model to a specific strain of Campylobacter (C. jejuni); however, the formulation here may be adapted to model for other mesophilic pathogens such as Escherichia coli and S. Typhimurium. The model developed herein quantifies bacteria levels on each carcass passing through a three-stage scalding tank and also bacteria levels in the water of each successive scald tank. During the residence time of carcasses in each scald tank, there is opportunity for transfer (carcass-to-water and vice versa) and thermal inactivation of bacteria. However, we assume that there is no physical contact between carcasses in the tank, hence there is no direct route for bacterial cross-contamination. A key feature of this model is that cross-contamination at the individual level can be investigated.

2.1. Mechanisms for transfer and inactivation

Contaminated carcasses enter the scald tank with pathogens attached to their skin and feathers. While the carcasses move through scalding tanks, pathogens shed into the scald water as a result of turbulence, shear forces, and diffusion. Heated scald water thermally inactivates bacteria attached to the carcasses and in the scald water. These basic mechanisms were studied in models previously (McCarthy et al., 2018; Osiriphun et al., 2012). One objective of this work is to quantify the effects of cross-contamination or carcass-to-carcass transmission directly, hence an additional feature of the model we develop is considering bacterial attachment from scald water to carcasses. In other words, we shift directions from a qualitative view to a more quantitative view. This approach is closer to the actual dynamics seen in the scald tank.

2.2. Modeling approach

We denote the bacteria level on the $i^{th}$ carcass as $C_i$ colony forming units (CFU), which is subject to change while the carcass resides in each of the three scald tanks. We denote the time for which the $i^{th}$ carcass enters scald tank 1 as $t_i^1$ and carcasses have a dwell time of $t_i^1 + t_i^2 + t_i^3$ in each successive scald tank. Therefore the $i^{th}$ carcass leaves the first scald tank at time $t_i^1$. We also account for the transition time between scald tanks. That is, the $i^{th}$ bird enter scald tank 2 a short time $\varepsilon$ after exiting scald tank 1 and so we have $t_i^2 = t_i^1 + \varepsilon$. Finally, the $i^{th}$ bird passes through all three scald tanks at time $t_i^3 = t_i^1 + t_i^2 + t_i^3$. In other words, we assume for $t_i^k \leq t_i \leq t_i^k+1$ the carcasses are subject to bacteria transfer and inactivation.

Carcass surface inactivation rate $S_i$ Denote the rate at which bacteria are thermally inactivated on the surfaces of carcasses by $S_i$. Here the indices $i,j$ refer to the inactivation rate on the $i^{th}$ carcass residing in the $j^{th}$ scald tank, respectively.

$$S_i^j = \begin{cases} S_i & t_i^j \leq t_i \leq t_i^j+1 \\ 0 & \text{otherwise} \end{cases} \quad \text{(1)}$$

The pathogen killing potential on carcass surfaces has been shown experimentally to depend on chlorine levels (Yang et al., 2001). Hence we consider the inactivation on carcass surfaces due to the addition of chlorine, and denote this by rate constant $h_s$. In other words, we model the total rate of chlorine inactivation on carcass $C_i$ by the term $h_sC_i$.

Shed rate from carcasses to scald water $k_i^j$ Each broiler carcass in the scald tanks is subject to turbulence in the form of aeration and shear forces. As a result, bacteria on the surface of carcasses are shed into the water. Here we consider some fixed rate of bacteria shedding, $k_i^j$ (1/min). Here, the indices $i,j$ refer to shedding of the $i^{th}$ carcass in the $j^{th}$ scald tank, ensuring that shedding only occurs when the carcass is in the $j^{th}$ scalding tank. We consider a uniform bacterial shed rate $k$ across all three scald tanks.

$$k_i^j = \begin{cases} k & t_i^j \leq t_i \leq t_i^j+1 \\ 0 & \text{otherwise} \end{cases} \quad \text{(1)}$$

We also consider the attachment of pathogens in scald water to carcasses at some constant rate $\beta$ (1/min). We assume that the amount of attachment to carcass skins is proportional to the current bacteria levels in the scald water as justified in (McCarthy et al., 2018). As a result of turbulence from countercurrent, aeration, and carcass mass moving through the scald tank we assume that sheeding and attachment are uniform in all scald tanks. Hence the term $10^5 \theta_i^j W_i^j$ represents the attachment of bacteria on carcass $i$ from scald tank $j$. The factor $10^5$ represents a unit conversion from milliliters to liters.

$$\beta_i^j = \begin{cases} \beta & t_i^j \leq t_i \leq t_i^j+1 \\ 0 & \text{otherwise} \end{cases} \quad \text{(1)}$$

From these mechanisms described above for bacterial surface thermal inactivation, sheeding, and attachment from scald water, we have the following equation governing pathogen levels of each carcass $C_i$.

$$C_i(t) = C_i(t_{i-1}) (k_i^j W_i^j + \beta_i^j W_i^j - S_i^j W_i^j - h_s C_i) + 10^5 \theta_i^j W_i^j t_i^j \leq t_i \leq t_i^j+1 \quad \text{for} \quad i = 1, \ldots, N. \quad \text{(1)}$$

$$0$$
Scald water contamination and inactivation dynamics. Here we aim to capture the contamination dynamics of bacteria in scald water. Let \( W_j \) (CFU/ml) denote the bacteria concentration in scald water in scald tank \( j \). Bacteria finds its way into the scald water in each tank by means of shedding from resident carcass surfaces (skin and feathers). As a result of the thermal sensitivity of foodborne pathogens in temperature ranges relevant to scalding, we consider a rate of thermal inactivation \( I_{in} \) in the scald water (McCarthy et al., 2018).

Also, organic material (excreta, blood, fat, proteins, etc.) in each successive scald tank decreases (Cason et al., 1999). This reduced protection from organic results in higher kill rates in the latter stage tanks. In other words, bacteria is less protected from organic material. The parameter \( \alpha \) is the percent increase of kill rate in scald water from these reduced organic material levels. We therefore consider a baseline kill rate in the first tank \( (\alpha_1 = 0) \), so that \((1 + \alpha_1)I_{in} = I_{in} \). Reduced organic load in the latter tanks enhances the effective thermal kill rate \((\alpha_2, \alpha_3 > 0) \). To illustrate note that \((1 + \alpha_2)I_{in} > I_{in} \) and also \((1 + \alpha_3)I_{in} > I_{in} \). Similarly, we consider a fractional increase of effective bacterial kill rate by the addition of chlorine in scald water by \( h_{ch} \) in tanks \( j = 1.2.3 \). Lastly, in each tank there is clean water inflow and scald water outflow at rate \( \dot{q} \) L/min and a tank volume of \( V_j \) in each tank \( j \), hence bacteria leaves each scald tank at rate \( W_jg/T_j \) (McCarthy et al., 2018). Similarly, the \( \text{pH} \) and temperature in each tank are denoted \( \text{pH}^j \) and \( T^j \), respectively. Hence in scald tanks \( j = 1.2.3 \) we have the following model equation describing the contamination dynamics in the scald water.

\[
W_j = \frac{1}{10^{7.7}} \left( \sum_{i=1}^{N} \frac{k_{ji}}{C_i} \right) - \left( W_j \sum_{i=1}^{N} \frac{1}{C_i} \right) - (1 + \alpha)(1 + h_{ch})I_{in}W_j - \frac{g_j}{T_j}W_j
\]

\[
(2)
\]

2.3. Complete model

Combining equations (1) and (2) for \( j = 1.2.3 \) and initial conditions reflecting the beginning of a scalding shift, the complete IBM becomes:

\[
C^j = \left\{
\begin{array}{ll}
-(k_i^j + S_i^j + h_{ch}^j)C_i - (k_i^j + S_i^j + h_{ch}^j)C_i & \text{for } t < t^j_i \\
C_i - (k_i^j + S_i^j + h_{ch}^j)C_i & \text{for } t = 1,...,N \\
0 & \text{otherwise}
\end{array}
\right.
\]

\[
W^j_i = \frac{1}{10^{7.7}} \left( \sum_{i=1}^{N} \frac{k_{ji}}{C_i} \right) - \left( W_j \sum_{i=1}^{N} \frac{1}{C_i} \right) - (1 + \alpha)(1 + h_{ch})I_{in}W_i - \frac{g_j}{T_j}W_i
\]

\[
W^j_0 = \frac{1}{10^{7.7}} \left( \sum_{i=1}^{N} \frac{k_{ji}}{C_i} \right) - \left( W_j \sum_{i=1}^{N} \frac{1}{C_i} \right) - (1 + \alpha)(1 + h_{ch})I_{in}W_0 - \frac{g_j}{T_j}W_0
\]

\[
(3)
\]

Initial conditions: Each carcass may have different initial contamination levels, which is one of the main advantages of the IBM. Let the pre-scald pathogen levels of carcass \( i \) by \( a_i \) CFU. So we have the resulting initial condition \( C_i(0) = a_i \) CFU. For the water contamination levels, we start the scalding shift at \( t = 0 \) and assume the scald tanks are clean and free of pathogens. In other words, we have \( W_i(0) = W_j(0) = W_0(0) = 0 \) CFU/ml.

2.4. Well-posedness

To show that the model is well-posed, we first consider the linear system

\[
\frac{dx}{dt} = A(t)x(t) + v(t)
\]

where \( x(t) \) and \( v(t) \) are \( n \)-dimensional vector-valued functions and \( A(t) \) is an \( n \times n \) matrix. We make use of the following theorem from (Filipov, 1988).

Let all the elements of the matrix \( A(t) \) and the vector-valued function \( v(t) \) be summable (i.e. Lebesque integrable) on each segment contained in the interval \((a, b) \). Then for all \( t_0 \in (a, b) \) the solution of the system with an arbitrary initial condition \( x(t_0) = x_0 \) exists on the whole interval \((a, b) \) and is unique.

Let \((a, b) = (-1, 1^2 + 1) \) and now we make use of the above theorem. As a result, we have existence and uniqueness of solutions of the scalding model (3) for \( t = 0 \) min to \( t = 1^2 + 1 \) min. That is, solutions exist from the beginning of the scalding process until the last carcass exits the last scald tank. Also, it may be easily shown that for nonnegative parameter values and initial conditions, solutions to the scald model (3) remain nonnegative.

2.5. Parameter estimation

Here we estimate transfer and inactivation parameters for the IBM for the specific pathogen \( C. jejuni \). Scald model (3) is composed from three main types of mechanisms: (I) those that involve typical processing procedures for industrial scalding, (II) bacteria transfer between compartments and inactivation in each compartment, and (III) parameters associated with input contamination levels and within-flock-prevalence. Parameters, along with relevant values/ranges, corresponding to Type I, Type II, or Type III mechanisms are categorized below.

2.5.1. Type I parameters

We assume a fixed mass \( m_a \) (kg) for all incoming broiler carcasses. Here we have \( m_a = 2 \) kg as in (McCarty et al., 2017, 2018). From a high-speed plant in Thailand, a clean water flow rate of \( g = 172 \) L/min and tank size \( V_j = 5900 \) liters are reported (Osrinphun et al., 2012). We then assume \( \dot{q}_j = 5900 \) liters and \( \dot{g}_j = 172 \) L/min for all three stage tanks \( j = 1.2.3 \).

2.5.2. Type II parameters

Bacteria shedding rate \( k \) Estimates of bacterial shed rate were obtained in (Osrinphun et al., 2012) and a similar range also obtained in (McCarty et al., 2018). We therefore fix \( k_i^j = 1.05 \) min\(^{-1} \) as found in (Osrinphun et al., 2012) for each bird \( i \) in scald tank \( j \). In other words, we assume that the rate of bacterial shedding into the scald water is the same in each tank.

Bacteria inactivation on carcass surfaces \( S^j \)

We estimate the thermal surface kill rate \( S^j \) of \( C. jejuni \) by adopting the Bigelow model (Bigelow, 1921). The Bigelow model is typically expressed as \( D(T) = D_{ref}10^{-T/T_{ref}/t} \). To proceed, we fit the Bigelow model to six previously estimated inactivation rates from poultry scalding-specific experiments (Osrinphun et al., 2012). The resulting coefficients from curve fitting are \( D_{ref} = 1.85 \) min, mean \( z^-\text{value} = 16.06 \) and 95% CI for the \( z^-\text{value} \) is \((8.48,23.64) \) using the reference temperature 54.15°C. For details of this curve fitting process refer to Section 6.2 in the Appendix. We also note that \( S^j = ln(10)/D(T) \) when the \( D^-\text{values} \) are converted to inactivation rate for use in scald model (1).

Organic material levels and \( \text{pH} \) indeed impact this kill rate (Berrang et al., 2011; Yang et al., 2001); however, the dominant factor determining the kill rate on carcass surfaces is scald water temperature. Note the kill rates yielded from fitting are similar to those synthesized in (Berrang et al., 2011; McCarthy et al., 2018; Yang et al., 2001). We estimate the chlorine’s additive effect of inactivating pathogens on the carcass surface \( h_{ch} = [0.006, 0.02] \) using a control pathogen inactivation experiment conducted in tap water and also an experiment conducted in poultry process water (with an organic load) (Yang et al., 2001). In particular, D-values were reported for chlorine inactivation on chicken skin in tap water (control) and in poultry process water. The organic load buffers pathogen inactivation, hence we consider \( h_{ch} \) within a range derived from both experimental rates. One explanation for this reduced killing potential is due to inactivation of chlorine near the carcass surfaces by organic material (Yang et al., 2001). In the former
In the scalding water, we see $D = 344 \text{min}$ corresponding to $h_s = 0.0067$ also using the same relationship between $D$-value and inactivation rate $I$. Hence, we consider $h_s$ in the range $[0.0067, 0.023] / \text{min}$.  

3. Simulation

Here we provide a simulation scheme that we use for validation and application of the scald model (3) informed with parameters and initial conditions from Table 1 as outlined below.

3.1. Simulation setups

Note that all model parameter values in Table 1 are fixed values except for the bacteria kill rate in scald water $I_s$ and the attachment rate from scald water to carcasses $\beta$. Surface thermal kill rate $I_s$ and chlorine inactivation rate $h_s$. To explore these parameter ranges and their impact on post-scald contamination levels, we numerically solve model (3) twice according to setup I (low-risk) or II (high-risk) below:

3.1.1. Setup I (I)

When $I_s$ is on the high end, $\beta$ is on the low end, $h_s$ is high, and $I_s$ is high, the carcass contamination levels are lowest (Fig. A5). Here the kill rate of the viable bacteria in the water is high combined with the relatively slow rate of attachment resulting in lowest net effects of cross-contamination. The results for this simulation are shown in the Appendix (Fig. A5).

3.1.2. Setup II (II)

When $I_s$ is on the low end, $\beta$ is on the high end, $h_s$ is low, and $I_s$ is low, the post-scald carcass contamination levels are highest. Here the kill rate of bacteria in the scald water is low while $\beta$ is on high end; hence, attachment occurs more rapidly. This range of the parameter space yields the highest levels of cross-contamination. As a result, we numerically solve scald model (3) twice; one for each scenario described above.

3.1.3. Monte Carlo simulations

In addition to the low-risk and high-risk simulations, we perform uncertainty analyses in the form of Monte Carlo simulations. That is, we observe the changes in model output with respect to sampling different regions of the parameter space. In particular, we use Latin Hypercube Sampling to sample $I_s$, $\beta$, $I_s$, and $h_s$ from their respective ranges and numerically solve model (3) each time. Monte Carlo simulations are performed in Section 4.1, 4.2, and 4.3 and are shown in Figs. 1B, 2B and 2.

In the case of setup (I), (setup II) and the Monte Carlo simulations described above, $I_s$, $\beta$, $I_s$, and $h_s$ are now known, remaining model parameters for scald model (3) are known and come from Table 1. The initial pathogen levels on each carcass are sampled from a distribution as described in Section 2.5.3. Scald model (3) is now informed with a complete set of fixed model parameters and initial conditions for $W_j$ for $j = 1, 2, 3$ and $C_i$ for $i = 1, 2, \ldots, N$. We numerically compute solutions for
Table 1
Parameter values and ranges found in Section 2.5 informing the scald model (\textsuperscript{3}). Simulation for the complete IBM determined by these parameters is carried out in Section 3.

<table>
<thead>
<tr>
<th>Model variable</th>
<th>Units</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$C_i$</td>
<td></td>
<td>Pathogen level on surface of carcass $i$</td>
</tr>
<tr>
<td>$W_j$</td>
<td></td>
<td>Pathogen concentration of scald water in tank $j$</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Name</th>
<th>Description</th>
<th>Parameter Range, reference</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_s$</td>
<td>Scald tank volume, \cite{Osiriphun2012}</td>
<td>5900</td>
<td>L</td>
</tr>
<tr>
<td>$g$</td>
<td>Water overflow, \cite{Osiriphun2012}</td>
<td>172</td>
<td>L/min</td>
</tr>
<tr>
<td>$m_i$</td>
<td>Typical carcass mass, \cite{Allen2003}</td>
<td>2</td>
<td>kg</td>
</tr>
<tr>
<td>$t_{d1}$</td>
<td>dwell time in tank 1, \cite{Berrang2000}</td>
<td>0.833</td>
<td>min</td>
</tr>
<tr>
<td>$t_{d2}$</td>
<td>dwell time in tank 2, \cite{Berrang2000}</td>
<td>0.833</td>
<td>min</td>
</tr>
<tr>
<td>$t_{d3}$</td>
<td>dwell time in tank 3, \cite{Berrang2000}</td>
<td>0.833</td>
<td>min</td>
</tr>
<tr>
<td>$w$</td>
<td>delay between carcass exit and entry of successive tanks (estimated)</td>
<td>2/60</td>
<td>min</td>
</tr>
<tr>
<td>$pH$</td>
<td>pH in scald tanks 1,2,3 (estimated)</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>$k$</td>
<td>Carcass to water bacterial shed rate \cite{Osiriphun2012}</td>
<td>1.65</td>
<td>1/min</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Attachment from scald water to carcass surfaces \cite{Lillard1986}</td>
<td>[0.001, 0.018]</td>
<td>1/min</td>
</tr>
<tr>
<td>$\eta_0(T_s, T_i)$</td>
<td>Baseline thermal kill rate in scald water \cite{McCarthy2018}</td>
<td>(0.74, 8.89)</td>
<td>1/min</td>
</tr>
<tr>
<td>$\eta_1$</td>
<td>Fractional increase of scald water kill rate with 59 ppm chlorine \cite{Berrang2011}</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>$\delta_1$</td>
<td>Thermal surface kill rate in tanks 1,2,3, estimated from \cite{Osiriphun2012}</td>
<td>1.49(1.41, 1.75)</td>
<td>1/min</td>
</tr>
<tr>
<td>$\eta_2$</td>
<td>Surface kill rate via chlorine addition \cite{Yang2001}</td>
<td>0.00067, 0.023</td>
<td></td>
</tr>
<tr>
<td>$\eta_3$</td>
<td>Fractional increase in inactivation from organic (tank 1) \cite{Yang2001}</td>
<td>0.31</td>
<td></td>
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<tr>
<td>$\eta_4$</td>
<td>Fractional increase in inactivation from organic (tank 2) \cite{Yang2001}</td>
<td>0.56</td>
<td></td>
</tr>
<tr>
<td>$J_1$</td>
<td>Organic material in tank 1, \cite{Cason1999}</td>
<td>13</td>
<td>mg/ml</td>
</tr>
<tr>
<td>$J_2$</td>
<td>Organic material in tank 2, \cite{Cason1999}</td>
<td>6.5</td>
<td>mg/ml</td>
</tr>
<tr>
<td>$J_3$</td>
<td>Organic material in tank 3, \cite{Cason1999}</td>
<td>3.02</td>
<td>mg/ml</td>
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<table>
<thead>
<tr>
<th>Type II Parameters</th>
<th>Description</th>
<th>Parameter Range, reference</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>$p$</td>
<td>Within flock prevalence, \cite{Berghaus2013}</td>
<td>0.68</td>
<td></td>
</tr>
<tr>
<td>$\eta_i$</td>
<td>$C. jejuni$ positive carcass input distribution, \cite{Berrang2000}</td>
<td>Normal(1.02 × 10^5, σ^2)</td>
<td>CFU</td>
</tr>
<tr>
<td>$\eta_i$</td>
<td>$C. jejuni$ negative carcass input distribution \cite{Berrang2000}</td>
<td>Uniform(0,999)</td>
<td>CFU</td>
</tr>
</tbody>
</table>

this system of N + 3 ordinary differential equations using the Matlab function ode45, which is an implementation of the Runge-Kutta method. Similar results may be obtained using a variety of software and numerical differential equation solution methods.

3.2. Model forward validation

Here we describe the forward validation process of scald model (\textsuperscript{3}) against industrial processing data from Berrang and Dickens \cite{Berrang2000}. We inform the model parameters in the same setting to carcass pre-scald contamination levels (Type III) as well as processing configurations (Type I) to replicate those in \cite{Berrang2000}. The pre-scald pathogen levels $o$, (incoming levels on each bird $i$) reflect the pre-scalp sample mean and variance found in \cite{Berrang2000}. Just 30 birds were sampled in \cite{Berrang2000}, hence we use prevalence from a more comprehensive Campylobacter survey including on-farm levels on 658 birds \cite{Berghaus2013}. This figure may more accurately depict the prevalence on a typical farm in the United States. We then numerically solve the scald model (\textsuperscript{3}) twice, in accordance with setup (I) and setup (II) described above. Using two numerical solutions of the scald model, we obtain a range for the mean output bacteria level $\mu$ \in [1.15 × 10^3, 2.6 × 10^4] CFU/carcass which captures the experimental mean of $\mu = 1.89 × 10^4$ CFU/carcass \cite{Berrang2000}. The simulation results are shown in Fig.
Fig. 2. Pathogen levels on individual broiler carcasses. A) Time dynamics of individual carcass pathogen levels during scalding in a high-risk scenario corresponding to setup II (T1, low, T2, low, β, high, and k1, low). Each curve and color is associated with pathogen levels on a single carcass. For example, carcass #1500 enters (black curve) enters the scald at about 8 min and leaves the scald at 3.33 min later. B) Monte Carlo simulations depicting the distributions of post-scald pathogen levels for individual carcasses. A and B illustrate how lowering pre-scald mean levels and prevalence may dramatically reduce individual post-scald pathogen levels below the limit of detection. The pathogen levels on individual carcasses #1, #1000, #2000, and #3000 are shown and may be compared with the levels in Fig. 1, observing the lower mean pathogen levels in this scalding scenario.

As in the Appendix. Note we carry out the estimation of parameters related to bacteria transfer and inactivation (Type II parameters) without using industrial slaughterhouse data and we validate the model against (Berrang and Dickens, 2000). A key feature of the scald model (3) is that it simplifies many complicated phenomena to a mathematically tractable form using few transfer and inactivation parameters which we can reliably estimate.

4. Scal model application

Here we show how our scalding framework may be applied by using poultry plant scalding process specifications, namely scalding temperature and pH. Effectively the scald model (3) gives us a metric for systematically comparing various input prevalence and pathogen levels for weighing control strategies and guiding management for pathogen levels on the farm. To demonstrate the use of this quantitative link we provide model simulations corresponding to various pre-scald contamination levels. We also explore the implications of this link in terms of risk assessment and scenario analysis in Section 4.3.4.

USDA-FSIS performance standards for Campylobacter on broiler carcasses are in terms of prevalence after chilling (Food Safety US Department of Agriculture and Inspection Service, 2015). More specifically, carcasses are sampled from a poultry processor weekly and microbiological analysis is performed. If greater than 8 of 51 of these post-chill carcass samples are Campylobacter positive, then the processor does not meet the performance standard for this period (Food Safety US Department of Agriculture and Inspection Service, 2015). Here we demonstrate how scald model (3) may be used to help processors meet these standards. In particular, we illustrate how the pre-scald distribution of pathogens on carcasses impacts the output carcass prevalence for varying pre-scald pathogen levels. The power here is that the model may inform maximum allowable pre-scald prevalence and pathogen levels in accordance with these standards through numerical experimentation. Here we work with the scald model applied to the 2015 USDA-FSIS Campylobacter performance standards. In Section 4.3.4 we discuss how to apply this model to other microbial performance standards.

4.1. Model applications to a specific industrial poultry processor in Canada

We apply the scald model to a specific processing plant in Canada whose scalding configurations were provided to us by the Public Health Agency of Canada and Canadian Poultry and Egg Processors Council. We refer to this specific plant as Plant A (as in McCarthy et al., 2018). These scalding configurations come from a recent survey and hence may represent a typical high-speed industrial processing scenario. The scalding process configuration of plant A is as follows: the pH in all three scald tanks is 9, the temperature in scald tanks 1, 2, and 3 are T1 = 51.66°C, T2 = 53.33°C, T3 = 58.89°C respectively, and carcasses have dwell times t1 = 1 min, t2 = 1 min, t3 = 1.33 min in tanks 1, 2, and 3.

We simulate scalding shifts for Plant A with the above process parameters and all other parameters remain the same as in Section 3.2. In other words, we “numerically scald” the same carcasses in a series of numerical experiments. Numerical results of the low- and high-risk simulations are included in Table 2. We also illustrate the time dynamics of pathogen levels on individual carcasses using the high-risk setup II in Fig. 1A. Lastly, the results of Monte Carlo simulation are shown in Fig. 1B. In the Monte Carlo simulations we provide 100 realizations of the scald model (3) to simulate the post-scald distributions of C. jejuni on individual carcasses. In Fig. 1A and B, we see that post-scald pathogen levels are similar regardless of pre-scald levels. Even more, most individual carcass pathogen levels approach the mean post-scald over all carcasses. The post-scald prevalence is above 8/51 ≈ 0.16, hence Plant A may be at risk of failing USDA-FSIS Campylobacter performance standards (Food Safety US Department of Agriculture and Inspection Service, 2015). Further reduction in pathogen levels may be required in later processing stages with this processing configuration.

4.2. Altering pre-scald C. Jejuni prevalence and pathogen levels

Bacterial contamination on the broiler surfaces is carried from the farm to transport and subsequently into the slaughterhouse. We now illustrate how the scald model may be applied to weigh these various scenarios. Specifically, we look at how post-scald pathogen levels are affected by adjusting the incoming prevalence p and mean input level µ CFU/carcass. We modify the pre-scald pathogen distribution on carcasses as follows: The C. jejuni positive carcasses are contaminated with an average of µ = 10^3 CFU/carcass and also the within-flock prevalence is reduced from 68% to 30%. This reduced prevalence replicates 30% pre-scald Campylobacter prevalence found in Denmark according to the European Food Safety Authority’s analysis of the 2008 EU baseline survey for Campylobacter in broilers (European Food Safety Authority, 2010).

We present the same set of simulation configurations as in Section 4.1 with this augmented pre-scald distribution. Results of the low-risk and high-risk simulations are included in Table 2. Time dynamics of pathogen levels on individual carcasses using high-risk setup I are shown in Fig. 2A. Also, Monte Carlo simulation results are shown in Fig. 2B.

We see altering the pre-scald prevalence and mean pathogen level
reduces the post-scald pathogen levels on nearly all carcasses below the detection limit of 1000 CFU/carcass (≤ 0% post-scald prevalence). In particular, the altered pre-scald distribution brings Plant A into passing territory for the 2015 USDA-FSIS Performance standards (Food Safety US Department of Agriculture and Inspection Service, 2015). Hence it may be possible that small changes in policy and management on the farm may correspond to very different levels post-scald and post-processing. Hence decision-making regarding processing compliance with performance standards may be guided with the scald model (3). In Section 4.3 we look more closely at post-scald prevalence and how it depends on pre-scald carcass contamination levels, revealing that the effects of the mean of prescal pathogen levels dominates influence of the pre-scald prevalence.

4.3. A closer look at post-scald prevalence levels

Recall that the estimates for the C. jejuni kill rate in scald water \( I_w \) and attachment rate to carcasses \( \beta \) are in terms of particular ranges. In the previous simulations (Figs. 1A and 2A) we choose these parameters according to the lowest and highest opportunity for cross-contamination (high-risk setup I and low-risk setup II). To gain insights into the typical output contamination levels, we perform Monte Carlo simulations as outlined in Section 3. We repeat this process for pre-scald prevalence over the full spectrum between 0%—100% and for mean values on positive carcass \( \mu = 10^6, \mu = 10^7 \) CFU/carcass. In particular, we see a threshold pre-scald prevalence level that separates the pre-scald levels into two distinct regions: one which post-scald prevalence levels are typically below the 2015 USDA-FSIS Campylobacter performance standard and another where post-scald prevalence are typically above the performance standard (Food Safety US Department of Agriculture and Inspection Service, 2015). Above a higher input prevalence all carcasses will be C. jejuni positive. The power here is that the model may inform maximum allowable prevalence and input mean levels for processors to meet performance standards. Here when \( \mu = 10^6 \) CFU/carcass we see that the post-scald prevalence is most frequently 0% (Fig. 3A); however, few parameter sets yield high post-scald prevalence (Fig. 3A). In particular, when \( p < 0.50 \) then typically Plant A will pass the USDA-FSIS Campylobacter performance standard assuming no additional contamination with Campylobacter occurs at later stages of processing (Food Safety US Department of Agriculture and Inspection Service, 2015). On the other hand, notice that when \( \mu = 10^7 \) CFU/carcass, typically the post-scald prevalence is above the performance standard limit when input prevalence is above 10% (Fig. 3B). However there are exceptions at all post-scald prevalences which correspond to 0% post-scald prevalence due to high inactivation rates and limited opportunity for cross-contamination. Depicted is 100 simulations for each input prevalence, exploring the parameter space with Latin Hypercube Sampling. For details regarding Latin Hypercube Sampling see (Marino et al., 2008) (see Fig. 3).

We now turn to the high-risk scenario (setup II) and look at how post-scald prevalence depends on pre-scald contamination. We conduct model simulations over a wide range of pre-scald configurations: \( 10^3 \leq \mu \leq 10^6 \) CFU/carcass and 0-100% prevalence \( p \) (Fig. 4). We see interesting behavior when we vary pre-scald configurations in these ranges. In particular, the pre-scald input mean C. jejuni levels \( \mu \) dominates prevalence \( p \) in terms of affecting post-scald prevalence. We also see the emergence of a sharp transition from 0% to 100% post-scald prevalence as the pre-scald mean increases above 10^5 CFU. The key here is that with only process operating procedures, this threshold and risk map may be generated with the scald model (3). This threshold mean contamination level may be of use for guiding maximum allowable pathogen levels on the farm, which we discuss further in Section 4.3.4.

4.3.1. Scald model dynamics

The scald model (3) has a convenient mathematical form allowing for simple analysis. In fact, pathogen levels on each negative and positive carcasses may be approximated with explicit expressions. The idea is that we can predict pathogen levels on carcasses in each individual tank and repeat for all three tanks. We leverage these approximate solutions to develop rules of thumb for mitigating cross-contamination and discuss further implications in Section 4.3.4. Similarly, note that when \( W_i(t) = \infty \), \( j = 1, 2, 3 \), and constants \( \eta_j, \zeta_j \) may be solved explicitly in each tank. Hence, if we have time series data for concentrations for \( W_i(t) \) then carcass contamination may be inferred if their pre-scald levels are known. Similarly, ranges for \( W_i(t) \) may be used to explore output pathogen levels. Also, we may weigh the efficacy of altered water recycling strategies on post-scald pathogen levels. We discuss potential applications for the model for use with emerging sensor technologies in Section 4.3.4.

4.3.2. Maximum MLR reduction

Lastly, we introduce a metric to quantify the maximum possible reduction of pathogen levels on carcasses during scalding. In the absence of cross-contamination, that is either bacterial attachment from scald water, \( \beta = 0 \) or \( W_i(t) = W_i(t) = W_i(t) = 0 \) CFU, pathogen levels on carcasses will be at its lowest. We then denote the maximum log_{10} reduction of a carcass by

\[
\text{MLR} = t^*(k_i^1 + h_i + S_i^1) + t^*(k_i^2 + h_i + S_i^2) + t^*(k_i^3 + h_i + S_i^3).
\]

(5)

Note carcasses entering the scald tank with pathogen levels above MLR cannot become decontaminated. Here MLR depends only on dwell time in each scalding tank and scald water temperature. With our parameter estimates in Section 2.5, the MLR may be easily calculated. See Section 4.3.4 for the details.
Fig. 3. Post-scald prevalence and its relationship to input prevalence for input mean values A) $\mu = 10^9$ and B) $\mu = 10^7$ CFU/carcass. A) The vast majority of pre-scald scenarios result in 0% post-scald. B) The majority of pre-scald prevalences result in 100% post-scald prevalence. In the red line represents the 2015 USDA-FSIS Campylobacter performance standard (maximum 8 of 51 positive). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

4.3.3. Control guidance

In the scald model (3) the change in bacteria level on a given carcass is determined by two rates: the decay rate on the carcass $k + h_i + S_i$ and the rate bacteria attaches from the scald water $10^2T_i\beta$. In other words $(k + h_i + S_i)^{-1}$ is the average time in minutes that bacteria remains on the carcass and $(10^2T_i\beta)^{-1}$ is the average time it takes for a single bacteria in scald water to attach to a given carcass. Essentially the following control quantity $C_j^d$ captures these two characteristic time scales. $C_j^d$ may be thought of as an attracting state which changes in time.

$$C_j^d = C_j(W_j(t)) = \frac{10^2T_i\beta W_j(t)}{k + h_i + S_i}$$  \hspace{1cm} (6)

The following two mathematical results relate the control quantity $C_j^d$ to prevalence, namely whether or not positive carcasses stay positive and negative carcasses stay negative.

i) A C. jejuni positive carcass $i$ entering a scald tank $j$ will remain C. jejuni positive in tank $j$ if $C_j^d > detection$ for $t \in [t_i^j, t_f^j]$.

ii) A C. jejuni positive negative $i$ entering a scald tank $j$ will remain C. jejuni negative in tank $j$ if $C_j^d < detection$ for $t \in [t_i^j, t_f^j]$.

Proof: Please refer to Section 6.5 for proofs of results i) and ii).

These results i) and ii) give relationships between detection level and $W_j(t)$, since we have estimates of the remaining parameters. In particular, i) and ii) give conditions on scald water concentration which ensure that positive carcasses remain positive and negative carcasses remain negative in a given scald tank. Hence we may use $C_j^d$ to find a maximum concentration of pathogens in the scald water to prevent negative carcasses from being contaminated. One result of this condition is that pathogen levels in the third and final scald tank $W_j$ must consistently remain low to obtain post-scald prevalence below 100%.

4.3.4. Risk assessment and risk management

Typical laboratory testing takes several days to obtain bacteria counts from samples. However, online biosensors and potentially automated methods are emerging, reducing the time required to retrieve sample bacteria counts (Massad-IvanirGiorGi et al., 2016). These online sensors may be implemented to sample scald process water. Currently the fastest sensors take roughly 30 min to estimate pathogen levels from a water sample. However as this detection time decreases, it may allow for the development of automated alert systems in which groups of highly contaminated birds may be located before they leave the processing plant. As we discussed in Section 4.3.1, the scald model (3) may be used to infer pathogen levels on carcasses if the levels of bacteria in

Fig. 4. A, B) High-risk simulation (Setup II: low $I_i$, high $\beta$, low $I_{ii}$, low $h_{ii}$) illustrating the effects of pre-scald mean and prevalence on post-scald prevalence. The pre-scald mean has a greater impact than pre-scald prevalence in terms of influence on post-scald prevalence. Also, we see threshold mean values between roughly $\mu = 5.5$ and $6.5 \log 10$ CFU/carcass where the post-scald prevalence sharply increases.
the scal}d water and pre-scal}d pathogen levels are known. Similarly, equation (2) with discrete-time measurements of the bacterial concentration in the scal}d water along with pH levels, scal}ding temperature, and water flow rates can be used to estimate the average pathogen counts over all carcasses in each scal}d tank. Hence our parameter estimates, modeling framework, and tools we develop may be used to more rapidly (and automatically) infer average pathogen levels on carcasses. Overall, there is an emerging space for potential application synthesizing mathematical modeling, automated on-line sampling methods, and (bio)sensor networks. Furthermore, this application space becomes more broad with improving technology; namely, the decreased bacterial enumeration time of sensors.

The scal}d model and framework developed herein may assist in the policymaking and risk assessment processes. For instance, given a target post-scal}d C. jejuni count and/or prevalence the scal}d model may guide maximum allowable levels on the farm by only informing the model with scal}d water temperatures, pH, and carcass dwell times. For instance, if a carcass enters the scal}der with pathogen levels above the maximum log_{10} reduction then they will remain contaminated above detection levels. Scal}ing prepares carcasses for defeathering and later evisceration and chilling which are all major sites of cross-contamination (FAO/WHO, 2009). Hence a potential strategy for mitigating foodborne illness risk is bringing all carcasses below the detection limit in the scal}ding stage, as scal}ing occurs early in processing. The scal}d model may help guide processors and policymakers with such decision-making. Moreover, the model was designed specifically for this purpose. In terms of risk analysis, the scal}d model (3) fills in an important component of a poultry consumer process model. The present authors have developed a similar individual carcass model has been developed for the poultry chilling operation (McCarthy et al., 2017). The defeathering and evisceration may also be approached with similar individual-carcass modeling techniques to aid in understanding and mitigating cross-contamination. At present time models for defeathering and evisceration are less tractable than the scal}ing and chilling processes.

In this work, we focus on standards set by the FSIS-USDA which is in terms of Campylobacter prevalence. We note that other similar national performance standards exists and the scal}d model and techniques illustrated herein may be applied similarly to assist with decision-making. For instance, New Zealand has experienced high incidence of Campylobacteriosis and has more elaborate performance standards for poultry consisting of three components: alerts associated with highly contaminated birds, moving window targets, and quarterly mean values (Massad-Ivani/Giorgi et al., Segal). With the scal}ding framework we develop, the procedure we present may easily be augmented to make recommendations for these standards as well as providing worldwide utility. More particularly, given process specifications and pre-scal}d counts, numerical experiments which illuminate precisely the expected number of birds above critical pathogen levels, test a moving target window over all carcasses. Throughout the paper we refer to risk of USDA-FSIS failure in terms of prevalence post-scal}d. It is possible that pathogens are introduced onto carcasses via cross-contamination from equipment or viscera rupture during evisceration, raising post-chill prevalence. Prevalence may also be reduced in subsequent stages changing the plant’s pass or fail status.

5. Conclusions

The scal}d model (3) provides a quantifiable link between pre-scal}d pathogen levels, scal}ding processing parameters, and post-scal}d pathogen levels. The model we present may be a useful tool for risk assessment where experiments are costly, time consuming, or impractical. The general modeling framework and technique presented may be used to inform quantitative microbial risk assessment (QMRA) models with mechanistic detail. In particular, given pre-scal}d pathogen levels on carcasses and scal}ding configurations (e.g., scal}ding temperature, pH levels, etc.) the post-scal}d levels can be predicted using our scal}d model and fed into subsequent components of the QMRA model. In particular, post-scal}d levels may be used as inputs in the defeathering stage of a QMRA model, which may then be fed into the evisceration stage, and so on. Hence, this general framework may be used to determine best practices and policy in accordance with compliance standards in the United States (Food Safety US Department of Agriculture and Inspection Service, 2015) and European Union Reg. No. 2073/2005 (Althaus et al., 2017). Furthermore, many scenarios (e.g., logistic slaughter) may be evaluated in terms of the output contamination and risk they pose. For instance, influences on the farm (e.g., flock prevalence, incoming contamination levels) as well as processing configuration may also be weighed.

Future directions The model may be adapted to account for changing organic material levels, which would give insights for the time of the scal}ding shift for which pathogen levels may be highest or cross-contamination is most likely. We predict that the organic material levels may impact the inactivation of bacteria toward the end of a scal}ding shift as there is a constant influx of organic material and relatively little outflow due to water recycling. The current model is set up for the end of day organic levels which pose the highest threat.

Overall, in this work we use data from various sources and experiments to inform model parameters based on availability. We synthesized the bacterial attachment rate to carcasses ∫ using laboratory scale data collected from excised poultry skin (Lillard, 1986). This attachment rate is not well understood and may be influenced by many factors including scal}d water pH and temperature (Lillard, 1986; NGUYEN et al., 2010). Laboratory scale experiments which give more information as attachment rates change with temperature, pH, and other factors would help clarify understanding this rate of attachment. Also, industrial experiments which give pathogen levels for an individual carcass before and after scal}ding would give further insight on this attachment rate. We note that Markov Chain Monte Carlo (MCMC) techniques may be used to estimate Type II parameters describing transfer and inactivation. This methodology may give a more realistic picture of the transfer and inactivation time scales in the model. These statistical parametrization methods may be used to, for example, determine if the scal}ding process enhances or inhibits bacterial attachment.

The high variation of thermal kill rate in scal}d water with respect to the C. jejuni strain, especially at high temperatures, yields corresponding high variation in post-scal}d prevalence due to vast differences of cross-contamination effects felt during processing. Obtaining a narrower range for these rates with further inactivation experimentation would clarify model prediction. Then, having strain specific thermal inactivation information as well as specific strain presence in each processor may be useful for control purposes in each plant. Also, complete processing configurations such as line speeds, clean water flow rates, scal}d tank volumes, contamination levels, organic material levels are convenient for informing Type II transfer and inactivation parameters in such mechanistic models. Wider availability of these items may also assist in model parametrization and increase predictive power.

In this work, the scal}d model is formulated with several assumptions. First, we assume that cross-contamination does not occur via physical contact between carcasses in the scal}ding tanks. Also we incorporate several well-mixing assumptions in the model, i) bacteria attaches to all carcasses at the same rate and ii) bacteria is well-mixed in the scal}d water, i.e. the bacterial concentration in the scal}d water is uniform over the entire scal}d tank. The results relating the control parameter C∗ and C. jejuni levels on individual carcasses in Section 4.3.3 are mathematical consequences of these assumptions. We note that when the spatial uniformity in bacteria in scal}d water assumption is relaxed then these results may require adjustment. Realistically, highly contaminated carcasses may contribute more to the pathogen levels of their neighbors in the scal}d tank; however, this is not considered in the
present model. Measurements of bacterial concentrations at different locations (e.g., the scalding entrance and exit) may indicate that space should be accounted for. It is also possible that the counterflow introduces a bacterial gradient as well as organic material gradient in the scald water in each tank. However, at the present time this data is not available.

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A. Appendix

A.1. Effects of organic material levels on carcass inactivation

*C. jejuni* inactivation rates were recovered by comparing bacterial populations after heating in tap water (control) and scalding water after 10 h of continuous processing (Yang et al., 2001). Inactivation rates in scald water were significantly reduced in the experiments conducted in presence of the scald water (Yang et al., 2001). We suggest that the observed inactivation buffering effect of scald water is also dampened by the changes in water chemistry, namely the pH of scald water. The levels of *C. jejuni* recovered from chicken skins were higher in the presence of organic material as opposed to those in tap water (Yang et al., 2001). However this may be due to altered pH of scald water (McCarthy et al., 2018). Since tap water is neutral pH and scald water is acidic in the absence of additives due to organic material presence. We expect a similar buffering effect of bacteria would be found on broiler carcass skins, where organic material levels play a dominant role. However, the effect of pH dominates the effects of organic material levels on inactivation on carcass skins. Similar inactivation rates of pathogen inactivation are estimated using all known data sources (McCarthy et al., 2018; Osiriphan et al., 2012; Yang et al., 2001). Overall, we see that organic material may have a stronger influence than pH of kill rate on carcass skins. These experiments suggest that the effects of organic material on inactivation are in fact small compared to those from scald water pH (Yang et al., 2001). With all these factors in mind, as expected, the temperature is the dominant player in determining inactivation rates in scald water and on carcasses.

A.2. Details of estimating the pathogen kill rate on carcass surfaces

From the Bigelow model we have $D(T) = D_{0} 10^{-1/(T-T_{0})}$ (Gaillard et al., 1998). To inform this inactivation model $D(T)$, we use industrial-scale scalding experiments (Osiriphan et al., 2012). Scalding shifts were conducted at several temperatures and carcasses were sampled for subsequent microbiological analysis (Osiriphan et al., 2012). With the estimated *C. jejuni* counts on carcasses, estimates for carcass surface inactivation rates were provided for each temperature (Osiriphan et al., 2012). Here we use standard least square curve fitting to fit $D(T)$ to this data with reference temperature $T_{0} = 54.15^\circ C$. We use Matlab's curve fitting package and find the following coefficients $D_{0}$ and z-values mean (95% CI) to be $z = 16.66$ (8.48, 23.64). For use in the scald model (1), we convert $D(T)$ in units (min) to inactivation rate (1/min) using the relationship $I(T) = ln(10/D(T))$. In other words, we find $S' = I(T)$. In Monte Carlo simulations (Figs. 1B, 2B and 3A, and 3B) we consider the variation in $I_{r}$ as a result of this uncertainty in the z-value.

A.3. Details of estimating influence of organic material on kill rate in scald water

Here we elaborate on the parameter estimation process for $\alpha$, the percent increase of kill rate in scald water from reduced organic material levels. The scald water inactivation parameter $I_{r}pH, T)$ is fit to agree with data taken in scald water containing 13 g/l solids (McCarthy et al., 2018; Humphrey and Lanning, 1987). As a result of these buffering effects, this inactivation rate of free bacteria in scald water is higher with less organic material than the reference level of $J = 13 g/l$. We model these effects by fitting a surface $\phi(T, J)$ to the *C. jejuni* inactivation data reported for 50°C and 55°C. In each temperature cross-section (that is, for a fixed $J$), $\phi(T, J)$ is exponential in temperature. We then join these temperature cross-sections with a line from zero organic load to high levels of organic material.

We find the resulting surface

$$\phi(T, J) = \frac{a_{1}e^{-b_{1}T} - a_{2}e^{-b_{2}T}}{J_{d}} + \frac{a_{3}e^{-b_{3}T}}{J_{d}}$$

(A7)

To estimate the buffering effects in each tank $\alpha$, we first set $\Delta J_{d} = 13 g/l$ and $J_{d} = 0$ to replicate experimental settings (Yang et al., 2001). Now, we use $\phi(T, J)$ to obtain an estimate on $\alpha$ for any corresponding scalding temperature $T$ and organic load level $J$. For organic load levels in each tank, we set $J_{d} = 13 g/l$ as experimentally measured in (Humphrey and Lanning, 1987). Since organic material levels are typically halved in sequential scald tanks we estimate $J_{d} = 6.5 g/l$ and $J_{d} = 3.25 g/l$ (Cason et al., 1999). The resulting percentage increases in bacterial kill rate to be $\alpha = 0$, $\alpha = 0.31$, $\alpha = 0.56$ in scald tanks 1, 2, and 3, respectively.

A.4. Attachment to carcass surfaces $\beta$

To estimate the range of this attachment rate, we use laboratory-scale experimental data (Lillard, 1986). In this experiment, excised chicken carcass skins were immersed in solution and exposed to known *S. Typhimurium* concentrations (Lillard, 1986). We then use the reported pathogen levels recovered from carcass skins after immersion for 30 s and 30 min and the equation $C' = 10^{5}\beta l_{r}W$ from scald model (3). From this equation and assuming $W$ is constant over the course of the experiment, we have $C(t_{f}) - C(t_{0}) = 10^{5}\beta l_{r}W(t_{f} - t_{0})$ which relates $\beta$ and experimental settings. Now, using the experimental data we find $\beta$ corresponding to the slow and fast kinetics above. To convert the units of $\beta$ from attachment rate per 12 cm$^2$ to a whole broiler carcass, we use the following mass-to-surface area relationship
where \( w \) is weight of a carcass in g (United States Department of Agriculture, 1996). The pathogen levels recovered after 30 s give an estimate of the upper bound on \( \beta \) (fast kinetic) and 30 min pathogen levels immersion reports to estimate the lower bound on \( \beta \) (slow kinetic). We then estimate attachment rate \( \beta = [0.001, 0.021] \/ \text{min.} \)

Fig. A5. C. jejuni levels on individual broiler carcasses. Solutions correspond to numerical solutions of scaled model (3) with parameters from Table 1. A) Depict Setup (I: \( I_0 \) high end, \( \beta \) low end, \( k_i \) high, \( t_i \) high) results. Here the mean post-scaled C. jejuni level on broiler carcasses is \( \mu = 1.15 \times 10^7 \) CFU and nearly all carcasses end up contaminated above the detection level of 1000 CFU/carcass (99% prevalence).

A.5 Proofs of mathematical results in Section 4.3.3

i) A C. jejuni positive carcass \( i \) entering a scald tank \( j \) will remain C. jejuni positive in tank \( j \) if

\[
C_i^j > \text{detection for } t \in [t_i^j, t_{i+}^j]
\]

Proof: Let \( L = \inf \{C_i^j : t \in [t_i^j, t_{i+}^j]\} \). Hence we have \( C_i^j \geq L \) and therefore \( W(t) \geq (k_i + S_i)L/(10^6 \beta_i^j) \) for \( t \in [t_i^j, t_{i+}^j] \). Now, the contamination level on carcass \( i \) changes in tank \( j \) is given by Equation (1). Combining the preceding bound on \( W(t) \) as well as Equation (1) we have \( C_i^j \geq 0 \) when \( C = L \).

\[
C_i^j = -(k_i + S_i)C_i + 10^6 \beta_i^j W_j(t) \geq -(k_i + S_i)C_i + 10^6 \beta_i^j \frac{(k_i + S_i)L}{10^6 \beta_i^j} = 0.
\]

Since \( C_i^j > \text{detection} \) we have \( C_j(t) > \text{detection} \) for \( t \in [t_i^j, t_{i+}^j] \) and therefore carcass \( i \) remains C. jejuni positive.

ii) A C. jejuni positive negative \( i \) entering a scald tank \( j \) will remain C. jejuni negative in tank \( j \) if

\[
C_i^j < \text{detection}, \text{ for } t \in [t_i^j, t_{i+}^j]
\]

Proof: Let \( M = \sup L \{C_i^j : t \in [t_i^j, t_{i+}^j]\} \). Hence we have \( C_i^j \leq M \) and therefore \( W(t) \leq (k_i + S_i)M/(10^6 \beta_i^j) \) for \( t \in [t_i^j, t_{i+}^j] \). Now, the contamination level on carcass \( i \) changes in tank \( j \) is given by Equation (1). Combining the preceding bound on \( W(t) \) as well as Equation (1) we have \( C_i^j \leq 0 \) when \( C = M \).

\[
C_i^j = -(k_i + S_i)C_i + 10^6 \beta_i^j W_j(t) \leq -(k_i + S_i)C_i + 10^6 \beta_i^j \frac{(k_i + S_i)M}{10^6 \beta_i^j} = 0.
\]

Since \( C_i^j < \text{detection} \), we have \( C_j(t) \leq \text{detection} \) for \( t \in [t_i^j, t_{i+}^j] \) and therefore carcass \( i \) remains C. jejuni negative in tank \( j \).

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