



A mathematical model for pathogen cross-contamination dynamics during produce wash



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ABSTRACT

One of the main challenges for the fresh-food produce industry is to ensure that the produce is free from harmful pathogens. A potential area of risk is due to cross-contamination in a sanitizing chlorine wash-cycle, where the same water is used to wash contaminated as well as non-contaminated produce. However, this is also an area where effective intervention strategies are possible, provided we have a good understanding of the mechanism of cross-contamination. Based on recent experimental work by Luo, Y. et al. A pilot plant scale evaluation of a new process aid for enhancing chlorine efficacy against pathogen survival and cross-contamination during produce wash, *International Journal of Food Microbiology*, 158 (2012), 133–139, we have built mathematical models that allow us to quantify the amount of cross-contamination of *Escherichia coli* O157:H7 from spinach to lettuce, and assessed the efficacy of the associated wash-cycle protocols.

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1. Introduction

Produce washing is an important step in the fresh produce supply chain that is designed to improve cosmetic appearance, remove unwanted materials such as dirt and plant exudates and reduce the incoming microbial load (Gil and et al., 2009). However, wash water can act as a secondary source of contamination, enabling pathogens on incoming produce to disperse to multiple lots if not adequately sanitized. While many studies have explored sanitization options ranging from ultrasound and ultraviolet radiation to the synergistic effect of ozone and organic acids, in practice, chlorine remains the most widely used (Davidson and et al., 2013; Gil and et al., 2009; Luo and et al., 2012).

Despite its pervasive use, the underlying mechanisms that govern the concentration dynamics of hypochlorous acid and its role in preventing pathogen cross-contamination during the wash process are not completely understood. Part of the problem is that many experiments are conducted at the lab scale under particular

conditions and therefore results from these studies are difficult to synthesize. In their review of fresh cut produce sanitation, Gil et al. (Gil and et al., 2009) suggest that, “A standardized experimental approach to study the efficacy of different sanitizing treatments is needed considering as much as possible the commercial processing conditions.”

This is where mathematical modeling can play a fundamental role as it, along with relevant data, can be used to test mechanistic hypotheses as well as provide quantifiable links between specific processing parameters and resulting contamination levels with economy and scientific rigor. Furthermore, modeling can provide a well-defined reference point from which to compare various sanitization strategies even among differing wash conditions and particular produce/pathogen combinations.

From this perspective we approach the study in (Luo and et al., 2012), using the resulting time series data and experimental procedure to build and test a mechanistic model of the wash process. In particular, we construct a simple mathematical model, that captures the essential mechanism for chlorine decay in the wash tank as well as the cross-contamination dynamics of pathogen transfer from the wash water to shredded lettuce.

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2. Materials and methods

2.1. Pilot plant experiment

A brief description of the procedure in (Luo and et al., 2012) provides basic information for our model: baby spinach leaves, inoculated on average with $10^{4.9}$ CFU/g of *Escherichia coli* O157:H7, and shredded lettuce were placed (adjacent to each other without mixing) on a conveyor belt and discharged simultaneously into an immersion wash tank (volume given by $V = 3.2 \times 10^6$ ml). The entry rate of the shredded lettuce was approximately 45 kg/min and the spinach to lettuce ratio was 0.2%. The produce remained in the wash tank for an average of 26 s. In order to control pathogen build up in the wash water, sodium hypochlorite was added every $\tau = 12$ minutes with increasing dose volumes over a continuous wash period of approximately 36 min. Water quality, free chlorine concentration, pathogen survival and cross-contamination were monitored. The experiment was repeated three times. The data values used in this paper are the average of these measurements.

2.2. Chlorine dynamics in the wash tank

As the results in (Luo and et al., 2012) indicate, maintaining a stable level of free chlorine (FC) concentration in the process water is difficult. While this is due to a variety of factors, we considered the effects of the organic load on the chlorine concentration. Fresh-cut produce, entering the wash tank, introduces a significant amount of organic material, increasing the chemical oxygen demand (COD) in the water. Based on the data in (Luo and et al., 2012), the chemical oxygen demand increased linearly with the amount of lettuce entering the tank (on a time scale of about 36 min). Therefore, we modeled the rate of increase of COD by

$$O' = k_0 \quad (1)$$

where the $'$ denotes the derivative with respect to time, O (mg/L) is the COD in the wash water and k_0 is a constant with units (mg/(L min)).

To model the FC dynamics in the process water, we built the following equation

$$C' = -\lambda_c C - \beta_c OC + \sum_{k=1}^N r_k \chi_{[k\tau, k\tau + \tau_0]} \quad (2)$$

where C' indicates the change in FC in the wash water with respect to time and C is the concentration (mg/L) of FC available. As chlorine reacts with organic matter, there is a rapid depletion of FC in the system. For the majority of “chlorination reactions, the elementary reaction can be formulated as $\text{HOCl} + \text{B} \rightarrow \text{products}$, where B is an organic or inorganic compound” (Deborde and von Gunten, 2008). Using the COD in the wash water as a measure of the concentration of the organic material present and because the reactivity of HOCl with organics is usually second order (Deborde and von Gunten, 2008), we modeled the loss of FC as the second term in (2) where β_c is the second order rate constant.

While there are multiple types of organic (and inorganic) material in the wash water: bacteria, plant juices, soil, etc. and β_c most likely depends on the chlorine reaction with each of these, we assume β_c represents an average type rate (Deborde and von Gunten, 2008). Also, β_c is a function of pH, but we assumed the pH is constant, maintained by citric acid (this is a typical procedure in the fresh processing industry) (Deborde and von Gunten, 2008; Luo and et al., 2012). Furthermore, referring to the first term in (2), λ_c is the natural decay rate of chlorine in tap water.

Usually, wash systems have some kind of dosing scheme to replenish the loss of FC. Following the study in (Luo and et al., 2012), we considered a dosing strategy with a fixed period $\tau = 12$ min. Combining these ideas, we used the third term in (2) to account for the addition of FC to the process water. Here χ is the indicator function, taking the value 1 on time interval $[k\tau, k\tau + \tau_0]$ for some small time increment τ_0 and value zero elsewhere, N is the number of doses added, and $r_k > 0$ reflects the rate increase of FC from each dose.

2.3. Cross-contamination dynamics in the wash tank

In order to quantify the concentration of pathogen in the process water, X_W (MPN/ml), we constructed the following equation

$$X'_W = \beta_{WS} - \beta_{LW} X_W \frac{L}{V} - \alpha X_W C \quad (3)$$

The data (see Fig. 4 in (Luo and et al., 2012)), suggested that the level of *E. coli* remaining on the baby spinach during washing equilibrates quickly during the process, indicating that shed rate of *E. coli* from the baby spinach into the wash water is approximately constant. In terms of our model, we treated the spinach merely as a pathogen delivery vehicle, implying that there is a constant rate of *E. coli* being added to the wash water. Representing this rate by β_{WS} (MPN/(ml min)), the rate of increase of pathogen in the wash water is described by the first term in equation (3).

On the other hand, we considered the binding rate and the inactivation rate via FC as the two mechanisms that describe how pathogens are removed from the wash water. For the binding rate, see the second term of (3), we assumed that the successful contact and attachment of the pathogen to the produce occurs at a rate that is proportional to product of X_W and L/V where L is the amount of lettuce (kg) in the wash tank, V is the tank volume and β_{LW} (ml/(g min)) is the proportionality constant (in other words, the produce and pathogen are thoroughly mixed in the process water). Again, working from a well-mixing perspective, we modeled the inactivation of suspended pathogen via free chlorine indicated in the third term of (3) where C is the concentration of FC and α has units (l/(mg min)).

Finally, the contamination dynamics for the lettuce depend on the binding rate (i.e. the rate at which pathogen in the water binds to the lettuce), the FC inactivation of pathogen attached to the lettuce as well as the average time the lettuce spends in the wash tank. We modeled this as

$$X'_L = \beta_{LW} X_W - \alpha X_L C - c_1 X_L \quad (4)$$

where X_L (MPN/g) quantifies the amount of pathogen on the lettuce in the tank. The first term in (4) indicates the rate increase of pathogen transferring from the water to the lettuce, the second term reflects the inactivation of pathogen on the lettuce due to FC. For the third term, we assumed that the exit time of the lettuce from the wash tank is exponentially distributed with mean $1/c_1$. That is, $1/c_1$ (min) reflects the average dwell time for the lettuce in the wash tank. Note that we did not include produce to produce type transmission of the pathogen.

2.4. Complete model

Combining the dynamics of the water chemistry and pathogen transmission, our model is defined by the following system of equations:

$$\begin{aligned}
O' &= k_0 \\
C' &= -\lambda_C C - \beta_C OC + \sum_{k=1}^N r_k X_{[k\tau, k\tau+\tau_0]} \\
X'_W &= \beta_{WS} - \beta_{LW} X_W \frac{L}{V} - \alpha X_W C \\
X'_L &= \beta_{LW} X_W - \alpha X_L C - c_1 X_L
\end{aligned} \quad (5)$$

on the phase space where C , O , X_W , and X_L are all nonnegative. It is clear by inspection that the model is positively invariant on this space, indicating that the solutions make sense in an industrial context. See Table 1 for a complete list of the model parameters and their respective units.

2.5. Parameter fitting

All parameter values used in our model are reported in Table 1, and all simulations and optimization methods for fitting were implemented in MATLAB R2010a (The Mathworks, Inc.). We obtained some of these values from the literature. However, other parameters like L , k_0 and β_{WS} were specific to our model, and the ones such as β_C , α and β_{LW} , were not readily available from the literature for the experimental conditions used. These parameter values were determined as follows:

The produce is discharged into the wash tank at a constant rate N_1 g/min. Moreover, the average wash time is $1/c_1$ min, and the spinach to lettuce ratio is given by θ , we deduced that the amount of lettuce (g) in the tank is a constant, given by

$$L = (1 - \theta)N_1/c_1 \quad (6)$$

Next, the rate of change of COD from equation (5) is linear in time, and a fitting of the data from (Luo and et al., 2012) yielded a value for the slope k_0 of this line as 32.3 mg/(L min).

Following the experiment in (Luo and et al., 2012), we let σ (MPN/g) be the average amount of *Escherichia coli* on the incoming inoculated spinach. Also, we defined X_S (MPN/g) to be the average level of pathogen remaining on the spinach during washing. Since the rate of spinach coming into the tank is θN_1 (g/min) we calculated the rate of pathogen addition to the wash water, β_{WS} (MPN/(ml min)), as

$$\beta_{WS} = \frac{(\sigma - X_S)\theta N_1}{V} \quad (7)$$

In equation (2) for the FC levels in the tank, C only depends on itself and COD levels, as we have assumed that it does not depend on the pathogen levels in the tank. From (Hua and et al., 1999), we obtained the natural decay rate λ_C of FC as 1.7×10^{-3} /min at 5° C. For the FC depletion rate, β_C , due to the organic load, and the chlorine dosing parameters, r_1 , r_2 and r_3 , we used the subroutine “fminsearch” in MATLAB, to fit equation (2) to the full 36 min of data from (Luo and et al., 2012). Parameter values for β_C and r_1 , r_2 and r_3 are listed in Table 1.

Following these parameter fits, we used the resulting FC levels in the rate equations in model (5) to determine the pathogen levels in the water and on the lettuce in order to optimize for the parameters α and β_{LW} . Again, we used the full 36 min data set from (Luo and et al., 2012) and the subroutine “fminsearch” in MATLAB.

3. Results and discussion

3.1. Model fitting

Fig. 1(a) shows the amount of free chlorine levels, and Fig. 1(b)

and (c) show the pathogen levels in the water and on the lettuce, respectively, using both the data in (Luo and et al., 2012) as well as our model described in equation (5) with parameter values coming from Table 1.

We observe from Fig. 1(a) that our model fits the FC levels very well, with a root mean square error (RSME) of about 0.48. Also, it captures most of the dynamics of the pathogen levels, with a scaled (in order to equally weight the residuals) RSME of about 1.8, for the model fitting in Fig. 1(b) and (c). However, the last two data points are not explained well by our model. This further has the effect of lowering the solution peaks obtained using our model in the respective figures for the pathogen levels, indicating why these peaks do not quite match the data there. If we remove the last two time points from the data for the pathogen levels in the water on the lettuce, and run an optimization to fit for the parameters α and β_{LW} , we obtain the results shown in Fig. 2(a) and (b). In this case, $\alpha=0.52$ and $\beta_{LW}=0.47$ (with a scaled RSME reduced to around 1.5), which are not very different from the values obtained from using the full data set. From an experimental view, it is not entirely clear what conditions affected these final data points.

3.2. Comparing experimental results from varying scales

Given that our model describes most of the underlying mechanisms involved in the produce wash, it is useful as a reference point to compare parameters obtained from experiments at different scales. For instance, using lab-scale data from (Nou and et al., 2011) as well as from (Shen and et al., 2013), we calculated β_C , the depletion rate of FC in process water due to the organic load. On the lab-scale, these data indicated that $\beta_C \approx 2 \times 10^{-3}$ L/(mg min), whereas our model informed by data in (Luo and et al., 2012), reported that $\beta_C = 5.38 \times 10^{-4}$ L/(mg min). This suggested that lab scale experiments represent this mechanism relatively well.

However, when considering the inactivation rate of *E. coli* via FC, α L/(mg min), there was a larger discrepancy between the two experimental scales. Unpublished lab-scale data for pathogen inactivation suggested that for suspended *E. coli* levels at 8 Log CFU, α was on the order of 300–500 L/(mg min), in comparison with our model prediction that $\alpha=0.75$ L/(mg min). Part of this discrepancy may be linked with the fact that the incoming pathogen levels shed into the wash water are relatively low (≤ 5 Log CFU, as in (Luo and et al., 2012)). This difference in magnitude suggests the importance of future experiments, examining pathogen inactivation of FC, to use low pathogen concentrations in the wash water.

In terms of cross-contamination, this discrepancy was also present when comparing lab and semi-commercial experiments. For instance, following the experiment in (Luo and et al., 2011), 30 g of lettuce inoculated with 10^4 CFU/g of *E. coli* O157:H7 was added to 3000 ml of water and 120 g of uninoculated lettuce was added immediately after. The mixture was then manually agitated for 30 s and then measurements for *E. coli* transfer were made. Using this data, we calculated the average transfer rate, β_{LW} ml/(g min), to be approximately 30.6. The value obtained from our model fit from data in (Luo and et al., 2012) was $\beta_{LW} = 0.38$ ml/(g min), indicating that cross-contamination occurs at a much lower rate on the commercial scale most likely due to multiple factors that cannot be readily controlled.

3.3. Quantifying residual FC

In order to keep the process water free of pathogens and hence minimize cross-contamination during produce washing, there must be sufficient residual FC in the water. As pointed out in (Gómez-López and et al., 2014; Shen and et al., 2013), in experiments with increasing COD levels, this residual FC concentration is the essential factor for

Table 1

List of parameters and their values. All the values were obtained using data from (Luo and et al., 2012) except λ_c , which was obtained from (Hua and et al., 1999).

| Type | Parameter | Description | Values & units |
|-----------------------------|--------------|---|-----------------------------------|
| From (Luo and et al., 2012) | c_1 | Reciprocal of average wash time | 2.3/min |
| | σ | Pathogen level on spinach | $10^{4.9}$ MPN/g |
| | V | Volume of wash tank | 3.2×10^6 ml |
| | N_1 | Incoming rate of produce | 45,000 g/min |
| | τ | Chlorine dosing period | 12 min |
| | τ_0 | Duration of dose | 2 min |
| | θ | Ratio of spinach to lettuce | 0.2% |
| Calculated | L | Amount of lettuce in wash tank | 19,526 g |
| | λ_c | Natural decay rate of FC | 1.7×10^{-3} /min |
| | β_{WS} | Effective pathogen rate entering water | 1.95 MPN/(ml min) |
| | k_0 | COD increase rate | 32.3 mg/(L min) |
| Model fit | β_c | Depletion rate of FC in wash water | 5.38×10^{-4} L/(mg min) |
| | r_1 | Add. rate of FC at dose 1 | 12.75 mg/(ml (min) ²) |
| | r_2 | Add. rate of FC at dose 2 | 7.47 mg/(ml (min) ²) |
| | r_3 | Add. rate of FC at dose 3 | 5.56 mg/(ml (min) ²) |
| | A | Inactivation rate of pathogen via FC | 0.75 L/(mg min) |
| | β_{LW} | Pathogen binding rate: water to lettuce | 0.38 ml/(g min) |

controlling pathogen inactivation, as opposed relying on ORP, for instance. Furthermore, “understanding the dynamic interactions between organic load and FC concentration is critical to developing practical sanitization strategies for maintaining safety of fresh-cut produce” (Shen and et al., 2013). Referring to Fig. 1(a), it is clear that the rise in the COD levels was the main cause for the FC levels to fall rapidly, and this subsequently caused the pathogen levels both in the water and the lettuce to rise. Because our model is informed by the direct quantification of these interactions, as opposed to merely a correlative description, it has predictive power and could be used, for instance, to deduce that any technique used to lower the reaction rate between the free chlorine and the COD, would have a considerable impact on controlling the pathogen levels. Furthermore, given such a technique, our model could directly predict the scope of this control, especially for extend wash times. That is, our model coupled together with streamlined experiments (as in Luo and et al., 2012) could be used to test optimal chlorine sanitization strategies for lengthy wash times that would otherwise be costly and difficult to monitor. Fig. 3 uses the model to predict the dynamics of the chlorine and pathogen levels after two additional chlorine dosing cycles (i.e. up to 60 min), assuming a linear rise in COD levels and a similar chlorine dosing scheme as in (Luo and et al., 2012).

3.4. Model validation and predictability

In order to validate our model described in equation (5), we used the first 12 min of data from (Luo and et al., 2012) to determine our model parameters and then compared the model predictions with the remaining 24 min of FC and pathogen concentration data from (Luo and et al., 2012). To determine the parameters β_c , r_1 , α , and β_{LW} , we used data from the first 12 min of the experiment in (Luo and et al., 2012). In particular, we used the subroutine ‘fminsearch’ in MATLAB to minimize the least square error for the parameter fits. This procedure yielded the follow values: first using equations (1) and (2), we calculated $\beta_c = 5.26 \times 10^{-4}$ and $r_1 = 13.08$, RSME of 2.6 and then using the resulting FC levels, we ran the optimization with equations (3) and (4) to obtain $\beta_{LW} = 0.74$ and $\alpha = 0.50$, with weighted root mean square error (RMSE) of 0.63.

To use our model against the remaining 24 min of data coming from (Luo and et al., 2012), we needed values for r_2 and r_3 , the effective addition rates of FC following doses 2 and 3 respectively. Since these values are dependent on the physical addition of chlorine to the process water, we used equation (2) and only the FC data at 12 and 14 min as well as 24 and 26 min coming from (Luo and et al., 2012) (i.e. data from the dosing periods). We found

that $r_2 = 7.18$ and $r_3 = 5.01$ (calculations not shown).

Fig. 4 shows the model predictions against the data for the remaining 24 min (note that the model fit and data from the first 12 min are included as the model was run for the full 36 min). The scaled RSME (in order to equally weight the residuals) for predicted vs observed C, X_W and X_L was approximately 3.3. Fig. 4(a) shows that the model nicely captures the mechanisms for FC dynamics. However, two points are worth noting. First, the depletion of FC from 2 to 10 min of data indicates a variation from exponential decay, as assumed by the model. This may have to do with the fact that the FC was not yet thoroughly mixed throughout the process water. Our model fit overmatched the data from about 7 to 12 min and this translated into the under-matching of the model fit in Fig. 4(b). That is, the *E. coli* level in the water at 12 min was predicted to be slightly lower than observed.

The second aspect concerns the FC level during the 34–36 min time span. Fig. 4(a) shows that the predicted FC level was lower than the corresponding data. This is curious as the data indicated that the FC level increased even though there was no external dosing. Although our model described the rest of the dynamics of the pathogen levels quite well (Fig. 4(b) and (c)), the model prediction under-matched the observed FC level during the 34–36 min interval, which was a major contributor to the RSME. From an experimental view, it is not entirely clear what conditions affected these final two data points.

Table 2 offers a comparison between parameters fit from the first 12 min of data from (Luo and et al., 2012) and parameters fit from the full data set. Notice that the two sets of values are very similar, indicating that model has predictive value and describes the main mechanisms quite well. The largest discrepancies concern α and β_{LW} . Table 2 shows that α is lower when fit to the first 12 min of data. As above, this is most likely due to the fact that thorough mixing of the FC had not yet occurred in the wash water. In terms of β_{LW} , data for X_L (the pathogen level on the lettuce) at time 24 min as well as 34–36 min are lower than might be expected (Fig. 4(c)). These points have the effect of lowering the value of β_{LW} when using the full data set for fitting. As the values of the parameters did not significantly differ when we used data points up to the first 12 min, we did not try to fit the data using more time values, say up to the first 24 min, and then try to make predictions with our model.

3.5. Quantitative microbial risk assessment (QMRA)

In terms of controlling cross-contamination during processing of fresh produce, intervention strategies ideally need to be informed by both pathogen prevalence and concentration at

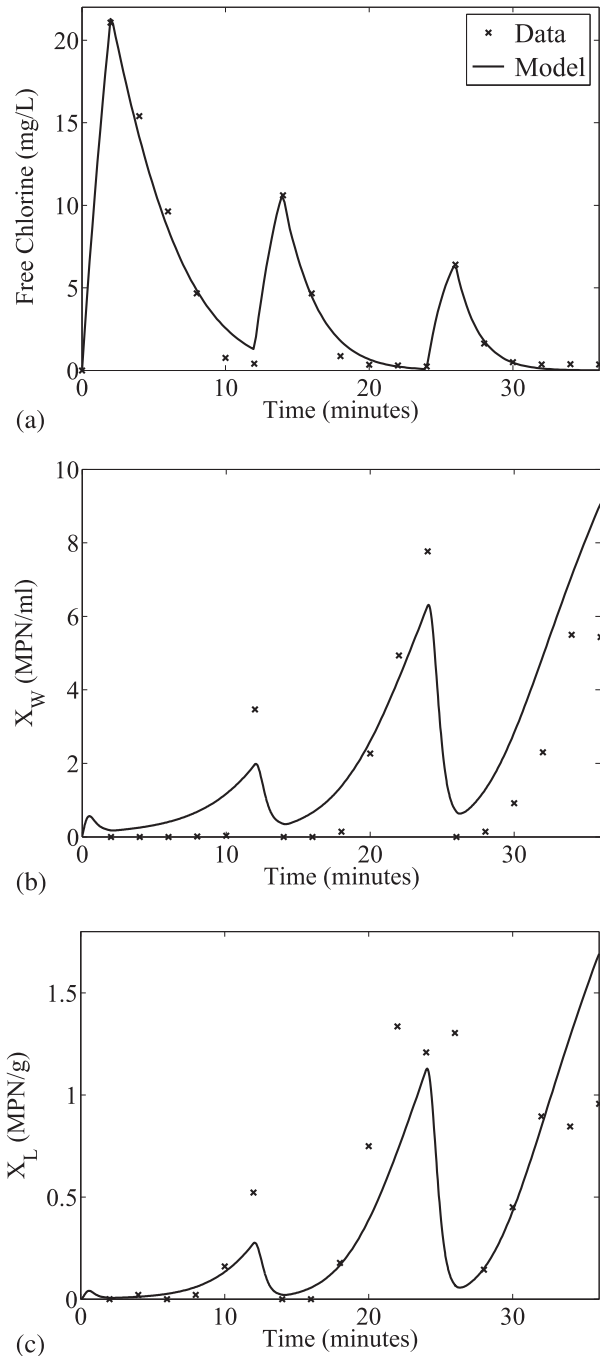


Fig. 1. Time plots of (a) Free chlorine levels, (b) *E. coli* levels in the water, and (c) *E. coli* levels on the lettuce. The solid line is from the model described in equation (5), and the \times values are the data points from (Luo and et al., 2012).

various stages. Typically, stochastic/agent-based models have been employed to address these concerns, quantifying risk over a variety of factors. For instance, the FDA has developed models such as FDA-iRisk and QPRAM (Quantitative Produce Risk Assessment Model) (<https://irisk.foodrisk.or> and Febr 19, 2015). iRisk is a freely available, web-based, risk modeling tool that can address local risk questions at the farm level as well as larger scale issues at the supply chain level, tracing risk from farm to fork. QPRAM is an agent-based model that focuses on the risk levels at a particular farm or processing facility.

While these models are promising, parameters at some key

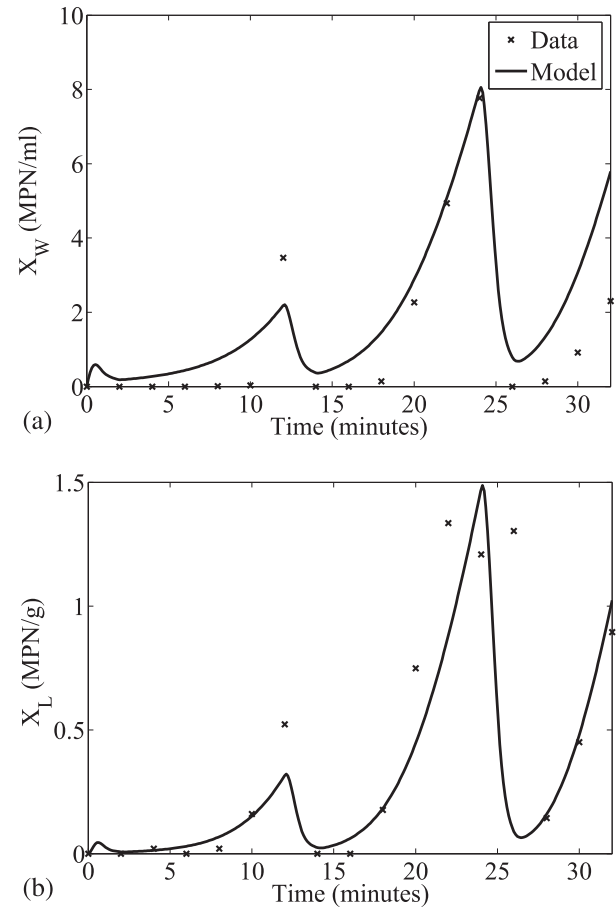


Fig. 2. Time plots of (a) *E. coli* levels in the water, and (b) *E. coli* levels in the lettuce, after removing the last two data points in Fig. 1(b) and (c). The solid line is from the model described in equation (5), and the \times values are the data points from (Luo and et al., 2012). There is no change in the free chlorine when we remove the last two data points.

steps are either unknown or loosely estimated. Therefore, the risk outputs from these models may lack sufficient confidence. This is where mechanistic modeling can provide significant information. By elucidating the mechanisms of cross-contamination dynamics at focused spatial/temporal hubs in the supply chain, these models can narrow specific parameters of the larger scale risk models. For instance, in (Rodríguez and et al., 2011), a stochastic model for cross-contamination of *Escherichia coli* O157:H7 during lettuce processing was developed to understand the prevalence and concentration of *E. coli* in bags of post-processed fresh-cut lettuce. Transfer coefficients describing the pathogen transfer for various scenarios involving produce, equipment and process water were estimated by fitting probability distributions to relevant data, providing the backbone of the model.

At the decontamination step, however, the chlorine concentration was assumed constant during a full day of production (Rodríguez and et al., 2011). In light of the aforementioned discussion concerning the depletion of FC via the organic load, it seems important to use pathogen transfer coefficients during the produce wash that reflect these dynamics. This is where our model could play a vital role. By using data from (Buchholz and et al., 2012a; Buchholz and et al., 2012b), for instance, β_{WS} could be adjusted to reflect various levels of pathogen entering the wash tank. Then, tuning the parameters of our model to fit the details of the particular wash procedure (such as wash time, produce wash rate, volume of the wash tank, etc.) our model outputs could be

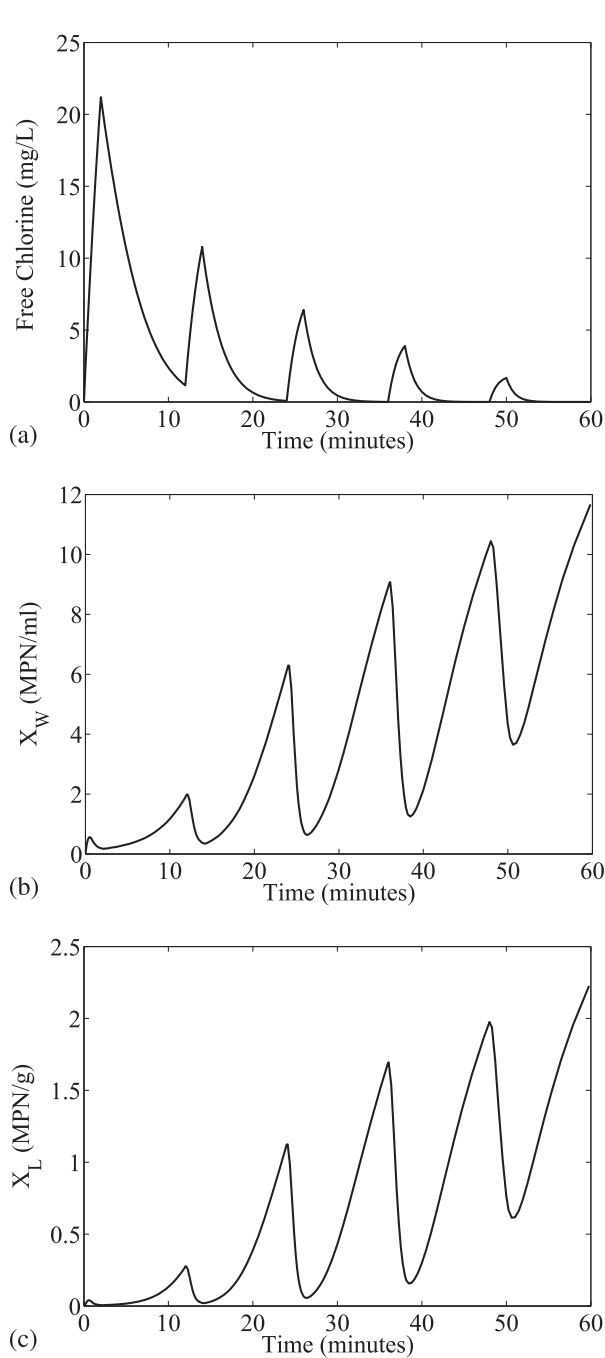


Fig. 3. Simulations over time of (a) free chlorine levels, (b) *E. coli* levels in the water, and (c) *E. coli* levels in the lettuce by numerically solving for the variables described in equation (5), after two additional chlorine dosing cycles.

used to calculate pathogen transfer. As an example of this, Fig. 5(a) and (b) compares our model predictions for *E. coli* levels in the water and on the lettuce exiting the wash tank, linked to two different shed rates of say, for example, 0.25 MPN/(ml min) of pathogens into the water and 2.5 MPN/(ml min) of pathogens into the water. We have chosen values for β_{WS} which differ by an order of magnitude in order to illustrate the sensitivity of the model to this shed rate. Note that all other parameters are fixed with values listed in Table 2. The advantage here is two fold: first, the transfer coefficients associated to the wash step would have a mechanistic basis and second, our model could allow for easy and economic

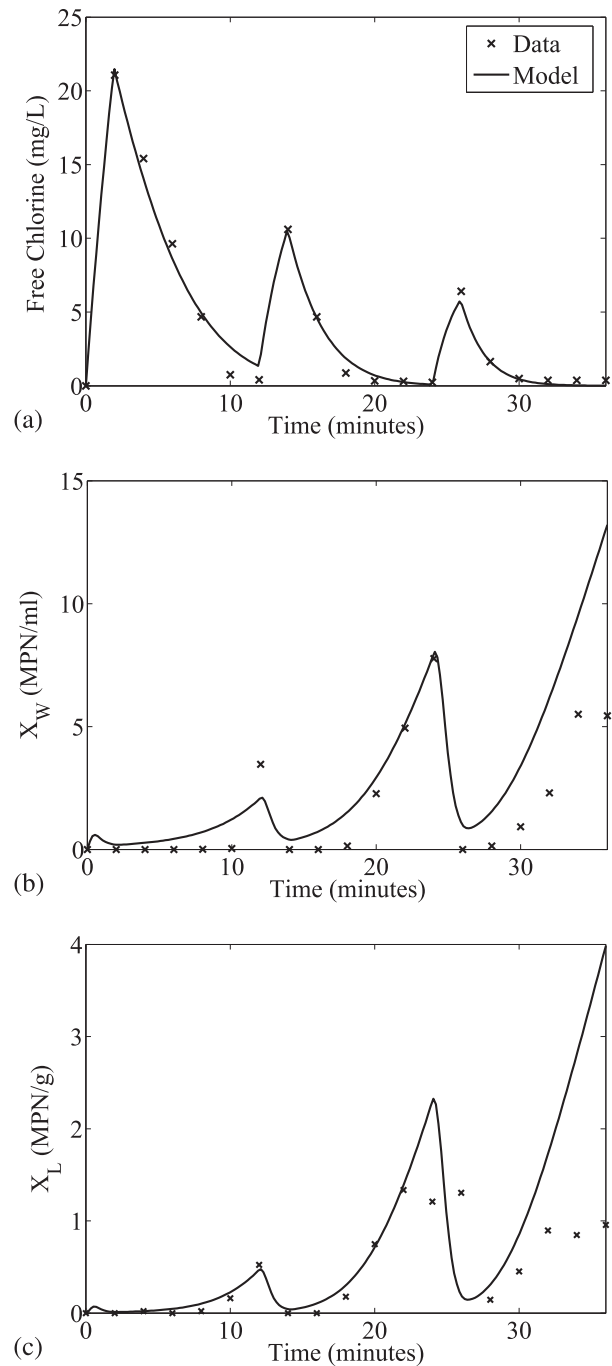


Fig. 4. Time plots of (a) Free chlorine levels, (b) *E. coli* levels in the water, and (c) *E. coli* levels on the lettuce. The solid line is the model, described in equation (5), fit (for the first 12 min) and then the model prediction (for the last 24 min) and the x values are the data points from (Luo and et al., 2012).

testing (as opposed to extensive experiments) to determine how significantly the organic load affects the contamination results within the larger stochastic model.

4. Conclusions

This study is an initial step towards understanding and quantifying the underlying mechanisms involved in commercial scale washing of fresh cut produce. We constructed a mathematical model that is able to continuously describe the dynamics of water

Table 2

Comparison of the parameters of the model from fits using the first 12 min of data (column 2), and the full data set (column 3) from (Luo and et al., 2012). Units for the various parameters are the same as in Table 1.

| Parameter | Description | Fit to first 12 min data set | Fit to 36 min data set |
|--------------|---|------------------------------|------------------------|
| β_c | Depletion rate of FC in wash water | 5.26×10^{-4} | 5.38×10^{-4} |
| r_1 | Add. rate of FC at dose 1 | 13.08 | 12.75 |
| r_2 | Add. rate of FC at dose 2 | 7.18 | 7.47 |
| r_3 | Add. rate of FC at dose 3 | 5.01 | 5.56 |
| α | Inactivation rate of pathogen via FC | 0.50 | 0.75 |
| β_{LW} | Pathogen binding rate: water to lettuce | 0.74 | 0.38 |

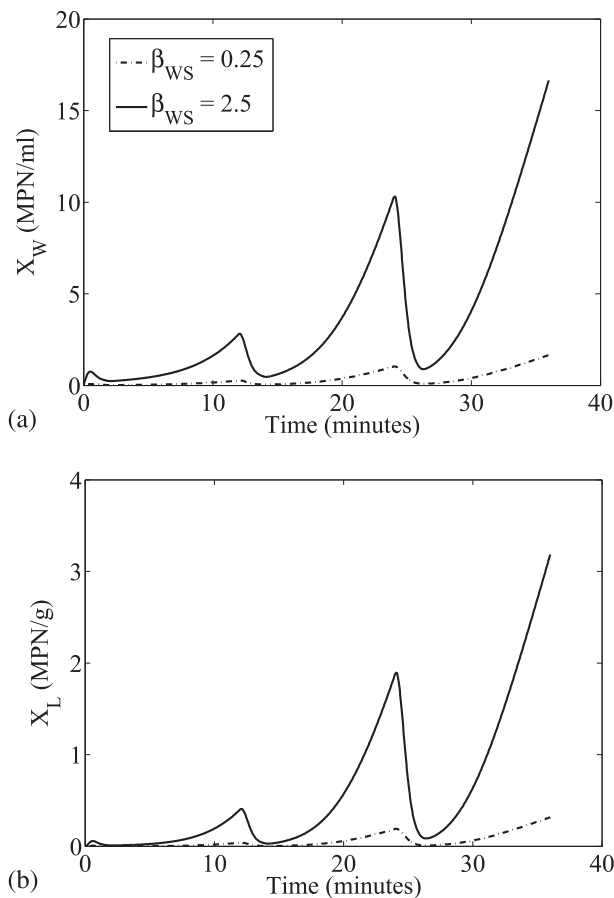


Fig. 5. Simulations of (a) *E. coli* levels in the water, and (b) *E. coli* levels in the lettuce after numerically solving the rates described in equation (5) using $\beta_{WS} = 0.25$ MPN/(mL min) (dashed lines) and $\beta_{WS} = 2.5$ MPN/(mL min) (solid lines). All other parameter values are as in Table 2. The free chlorine levels are the same as in Fig. 1.

chemistry and pathogen cross-contamination during the wash procedure outlined in (Luo and et al., 2012). The highlights of our model are its simplicity, its ability to capture most of the mechanisms that account for FC fluctuation and pathogen transfer during fresh produce washing, and as discussed in Section 3.4, its ability to predict the dynamics of the FC and pathogen levels. We also have shown that our model can serve as a benchmark to help compare decontamination experiments at different scales as well as identify

particular assumptions that can inform streamlined future experiments. In addition, coupled with stochastic QMRA models, our mechanistic modeling regime can provide a foothold toward a more standardized approach for food safety and the evaluation of intervention strategies. Finally, we expect that our model framework, that is, our mechanistic description of FC depletion and pathogen transfer, can be used to understand cross-contamination during wash procedures that involve other produce/pathogen pairs.

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