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Enhanced Surveillance on Food-Borne Disease Outbreaks: Dynamics of Cross-Contamination in Biocidal Wash Procedure

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1. Introduction

Food borne diseases associated with the consumption of fresh produce continue to cause serious difficulties for public health. Recently, there have been a number of significant outbreaks both in North America and Europe. For instance, the 2006 *Escherichia coli* contamination of bagged spinach resulted in many hospita lizations in the US (and Canada) and three deaths (Sander, 2006). In 2008, an uncommon serotype of *Salmonella enterica*, known as Saintpaul, caused over 1000 cases of food poisoning across the US, finally being linked to jalapeño and serrano peppers from Mexico (Taylor et al., 2010). The year 2011 was particularly tough as the US suffered from at least six outbreaks associated to fresh produce, one of which involved cantaloupes contaminated with listeriosis, resulting in the second most deadly outbreak ever to occur in the US (CDC, 2011). Furthermore, in the summer of 2011, Europe was hit with the deadliest outbreak in recent history linked to sprouts grown from imported fenugreek seeds con taminated with *E. coli* O104:H4. Germany, being the epicenter, reported 45 deaths as of July 27 of that year (ECDC).

Clearly outbreaks in these countries have had tremendous socio economic impact. In order to mitigate these effects, disease surveillance must be able to quickly detect both geographic and temporal occurrence of such contamination. As many studies highlight disinfection as a crucial juncture in the supply chain, we look to examine the contamination dynamics of fresh produce that can occur in commercial wash procedures (Gil et al., 2009). While washing is designed to ensure the safety of a product, wash water may provide a secondary source of contamination or promote cross contamination (Tomás Callejas et al., 2012). In line with this, we build and analyze a novel three stage model of a typical wash procedure in a fresh produce processing facility.

In terms of mathematics, we show that contamination levels converge rapidly to an equilibrium, which we can describe via closed form expressions involving only model parameters, in each of the three stages. For biological implications, we identify

parameters to which this equilibrium is sensitive. In particular, we show how the mean wash time and sanitizer concentration crucially affect the degree of produce contamination and further we provide guidelines on how these parameters can be practically controlled to avoid misidentification of the source of contamina tion during a potential disease outbreak.

The paper is organized as follows: in Section 2 we describe the basic assumptions, parameters and the three stage model. Next, in Section 3, we show that the model converges to a unique, component wise positive equilibrium. Using these dynamics, in Section 4 we apply the model to a wash procedure involving fresh cut romaine lettuce (or similar leafy greens) contaminated with *E. coli* O157:H7. Also in Section 4, we justify ranges for model parameters and perform sensitivity analysis. Determining the mean wash time and free chlorine concentration to be key parameters with regards to produce contamination, we explore how these can be constrained to prevent misidentifying the initial food vehicle associated to an outbreak. Finally, we discuss how our model can be augmented to be more realistic as well as its link ability to global supply chain models. Note that in light of recent outbreaks in North America, a model framework similar to the one we adopt in this paper (for studying the contamination of fresh produce during processing) can be applied to contamination dynamics of meat (especially ready to eat products) at the processing juncture. For more details concerning meat hygiene and safety risks see Sofos and Geornaras (2010).

2. Three stage wash model

Suppose two farms produce either the same type of fresh produce or two different types (denoted by P1 and P2, respec tively), which are transported to a processing center to be washed, packaged and then shipped along various routes in the supply chain. Suppose also that farm 1 has a source of contam ination (possibly via compost or irrigation water) causing a portion of P1 to be contaminated before coming into the center (here we assume that contamination levels are sufficient to lead to a possible outbreak) (Beuchat, 2006). We want to study the dynamics of pathogen spread among P1 and the possible cross contamination of P2 during the wash processing procedure. To do so, we propose the following three stage model: Before, Washing and After. The B stage includes the pre washing of the produce, the W stage concerns the principle wash (with sanitizer) and the A stage reflects the dewatering step.

2.1. B stage

We suppose the pre wash involves a non immersion process, such as a municipal water spray. Because the produce is not submerged in water, we assume that the spread of contamination occurs via produce produce contact. To model such transmission, we rely simply on the principle of mass action which, when applied in this context, states that the amount of contaminated produce grows at a rate proportional to the product of the amount of clean produce with the amount of contaminated produce. The underlying assumption here is that the contaminated pro duce totally mixes with the clean produce (while incoming contamination usually is "patchy", due to a lack of commercial processing data and because we do not want to overcomplicate the model, we assume uniform mixing). If we let S_{1B} and I_{1B} denote the susceptible and contaminated densities of P1, then $I'_{1B} = \beta_{1B} S_{1B} I_{1B}$, where β_{1B} is the contamination rate. Adjusting this equation to account for the inflow and outflow of produce into the pre wash stage and including the dynamics for S_{1B}, we have

$$S'_{1B} = \beta_{1B} S_{1B} I_{1B} + \rho N_1 \quad b_1 S_{1B},$$
$$I'_{1B} = \beta_{1B} S_{1B} I_{1B} + (1 \quad \rho) N_1 \quad b_1 I_{1B},$$

where $N_1 > 0$ is the incoming rate of P1, $0 < \rho < 1$ and $1/b_1 > 0$ is the average time the produce spends in pre wash. Our assump tion on ρ indicates that a portion of P1 comes into the B stage already contaminated. Finally, for simplicity we assume that P2 comes into the B stage clean and thus ignore any other contam ination sources that may be involved (note that before pre washing, cross contamination could occur through cutting procedures, handling, etc., however, in our paper we do not consider these possibilities). See Fig. 1 for a schematic of the contamination dynamics in the B stage.

2.2. W stage

Following the pre wash stage, both P1 and P2 move separately into the main wash stage, where the produce types are cleaned by an immersion produce washer (see Pao et al., 2012 for more details). While P1 and P2 follow distinct processing lines, we suppose that the wash water is re circulated between these lines, which is a standard practice in the fresh produce industry (Bhagwat, 2006). Because of this, the wash water may become contaminated, leading to a cross contamination event. The main point here is that contaminated produce can shed pathogens into the wash water, which can then spread to uncontaminated produce (thus our system allows for more than one "transmission pathway", see Tien and Earn, 2010, for a related model). Note that in the following development, we ignore produce to produce contact in the wash stage as well as the possibility that pathogens may be able to grow in the wash water.

Let W represent the pathogen concentration in the wash water which is shed from contaminated P1 at a rate $\alpha > 0$. Let $1/\mu$ be the mean pathogen lifetime in the water. This is regulated by the addition of a sanitizing agent to the water as we assume that $\mu > 0$ (a constant) depends on the concentration of sanitizer used to treat the wash water. If we define I_{1W} and S_{1W} as the contaminated and uncontaminated amounts of P1 in the produce washer, then the change of pathogen concentration in the wash water is given by the following equation:

$$W' = \alpha I_{1W} \quad \mu W.$$

Furthermore, as contaminated water can potentially spread pathogens to clean P1, we model the growth rate of contaminated P1 at this stage by $I'_{1W} = \beta_{1W} S_{1W} W$, where $\beta_{1W} > 0$ is the trans mission rate from water to produce. Again, our incidence rate is based on the notion of mass action (which will be discussed in more detail when we derive a range for β_{1W} in Section 4). Including the flow of produce through the washer (i.e. let $1/c_1$ be the mean duration of the wash phase for P1, which will be

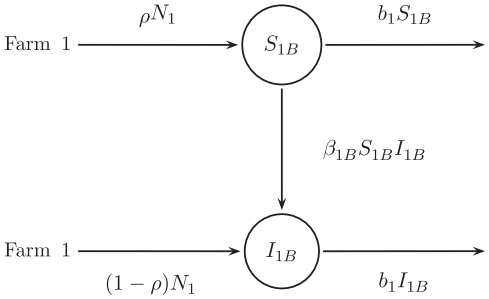

Fig. 1. I_{1B} is the contaminated amount of P1 in stage B. It increases via direct inflow from Farm 1 and via direct contact at rate β_{1B}. The average pre-wash time is $1/b_1$.

referred to as the "mean wash time" for the sake of brevity) and the dynamics of S_{1W}, we obtain

$$S'_{1W} = \beta_{1W}S_{1W}W + b_1S_{1B} - c_1S_{1W},$$
$$I'_{1W} = \beta_{1W}S_{1W}W + b_1I_{1B} - c_1I_{1W}.$$

Not only can clean P1 be contaminated by pathogens in the wash water, but if the sanitizer is limited in its efficacy, pathogens not killed by the treatment can contaminate P2 via re circulation. Therefore, following the same incidence rate assumptions as above, the cross contamination dynamics are given as follows:

$$S'_{2W} = \beta_{2W}S_{2W}W + N_2 - c_2S_{2W},$$
$$I'_{2W} = \beta_{2W}S_{2W}W - c_2I_{2W},$$

where S_{2W} and I_{2W} are the uncontaminated and contaminated amounts of P2, respectively, in the wash stage, $\beta_{2W} > 0$ is the cross contamination rate, $N_2 > 0$ in flow rate of P2, and $1/c_2$ is the mean wash time for the produce. It is important to mention here that we assume that contaminated P2 does not shed pathogens back into the wash water, and therefore, the equation for W' above does not depend on I_{2W}. Fig. 2 illustrates the wash stage for both P1 and P2.

2.3. A stage

Immediately after washing, P1 and P2 are dewatered along their respective lines. While continued cross contamination between P1 and P2 can potentially occur through equipment contact, food handlers working both lines, packaging and even pests, we assume that contamination spread in the A stage can only continue within each of the respective produce types. In particular, if we assume the produce is dewatered via centrifuga tion, because of the thorough mixing of produce with respect to contact with the centrifuge, we assume that the incidence rate again follows a mass action principle. Setting, S_{1A}, S_{2A}, I_{1A}, and I_{2A} to be the susceptible and contaminated amounts of P1 and P2, respectively, and including the in and out flow of produce, the contamination dynamics are given by

$$S'_{1A} = \beta_{1A}S_{1A}I_{1A} + c_1S_{1W} - d_1S_{1A},$$
$$I'_{1A} = \beta_{1A}S_{1A}I_{1A} + c_1I_{1W} - d_1I_{1A},$$
$$S'_{2A} = \beta_{2A}S_{2A}I_{2A} + c_2S_{2W} - d_2S_{2A},$$
$$I'_{2A} = \beta_{2A}S_{2A}I_{2A} + c_2I_{2W} - d_2I_{2A},$$

where β_{1A}, $\beta_{2A} > 0$ are the transmission rates, and $1/d_1$ and $1/d_2$ are the mean dewatering times for P1 and P2, respectively. Fig. 3 provides a schematic of the contamination route in the A stage.

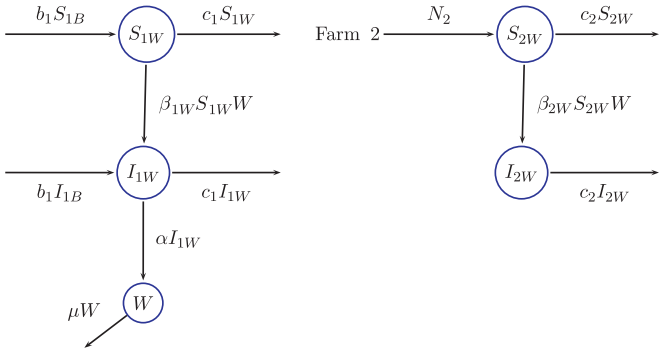

Fig. 2. Subscripts 1 and 2 indicate contamination routes for P1 and P2, respectively, during the wash stage. I_{1W} (the contaminated amount of P1 in the wash stage) contaminates the wash water at rate α. The W compartment indicates contaminated wash water with which both S_{1W} and S_{2W} have contact due to recirculation. I_{2W} gives the level of cross-contamination of S_{2W} which occurs at rate β_{2W}. Compartments with subscript "W" are outlined in blue indicating contact with contaminated water. (For interpretation of the references to color in this figure caption, the reader is referred to the web version of this article.)

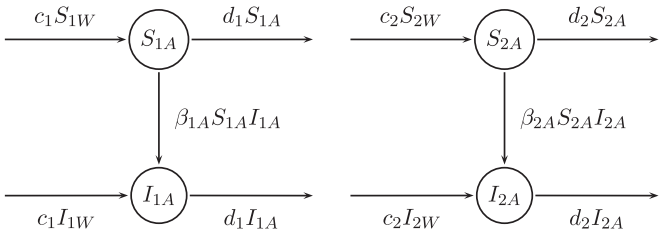

Fig. 3. Subscripts 1 and 2 indicate contamination routes for P1 and P2 in the "After" stage, respectively. The produce moves from the wash stage to be dewatered. The respective contamination rates are β_{1A} and β_{2A}.

2.4. Complete model

Combining the dynamics in the B, W and A stages, our contamination model is defined by the following system of equations:

$$\begin{aligned}
S'_{1B} &= \beta_{1B}S_{1B}I_{1B} + \rho N_1 - b_1S_{1B},\\
I'_{1B} &= \beta_{1B}S_{1B}I_{1B} + (1-\rho)N_1 - b_1I_{1B},\\
S'_{1W} &= \beta_{1W}S_{1W}W + b_1S_{1B} - c_1S_{1W},\\
I'_{1W} &= \beta_{1W}S_{1W}W + b_1I_{1B} - c_1I_{1W},\\
W' &= \alpha I_{1W} - \mu W,\\
S'_{2W} &= \beta_{2W}S_{2W}W + N_2 - c_2S_{2W},\\
I'_{2W} &= \beta_{2W}S_{2W}W - c_2I_{2W},\\
S'_{1A} &= \beta_{1A}S_{1A}I_{1A} + c_1S_{1W} - d_1S_{1A},\\
I'_{1A} &= \beta_{1A}S_{1A}I_{1A} + c_1I_{1W} - d_1I_{1A},\\
S'_{2A} &= \beta_{2A}S_{2A}I_{2A} + c_2S_{2W} - d_2S_{2A},\\
I'_{2A} &= \beta_{2A}S_{2A}I_{2A} + c_2I_{2W} - d_2I_{2A},
\end{aligned} \tag{2.1}$$

on the phase space $\Omega' = \{S_{1B}, S_{1W}, S_{1A}, I_{1B}, I_{1W}, I_{1A}, S_{2W}, I_{2W}, S_{2A}, I_{2A}, W \geq 0 : S_{1B} + I_{1B} = N_1/b_1, S_{1W} + I_{1W} = N_1/c_1, S_{1A} + I_{1A} = N_1/d_1, S_{2W} + I_{2W} = N_2/c_2, S_{2A} + I_{2A} = N_2/d_2\}$. Refer to Tables 1 and 2 for a com plete list of model variables and parameters with their respective units.

3. Analysis of model

Proposition 1. *System (2.1) is positively invariant on Ω'.*

Proof. To see this, let $n_1 = S_{1B} + I_{1B}$, $n_2 = S_{1W} + I_{1W}$, $n_3 = S_{1A} + I_{1A}$, $n_4 = S_{2W} + I_{2W}$, and $n_5 = S_{2A} + I_{2A}$. Then we have

$$\begin{aligned}
n'_1 &= N_1 - b_1n_1,\\
n'_2 &= b_1n_1 - c_1n_2,\\
n'_3 &= c_1n_2 - c_2n_3,\\
n'_4 &= N_2 - c_2n_4,\\
n'_5 &= c_2n_4 - d_2n_5,
\end{aligned} \tag{3.1}$$

where $(N_1/b_1, N_1/c_1, N_1/d_1, N_2/c_2, N_2/d_2)$ is a fixed point for system (3.1). So for $(n_1(0), n_2(0), n_3(0), n_4(0), n_5(0)) = (N_1/b_1, N_1/c_1, N_1/d_1, N_2/c_2, N_2/d_2)$, we see that

$$\begin{aligned}
&(S_{1B} + I_{1B}, S_{1W} + I_{1W}, S_{1A} + I_{1A}, S_{2W} + I_{2W}, S_{2A} + I_{2A})\\
&= (N_1/b_1, N_1/c_1, N_1/d_1, N_2/c_2, N_2/d_2)
\end{aligned}$$

for all $t \geq 0$.

Now let y_i be one of the phase space variables in Ω'. If $y_i = 0$, then by direct computation $y'_i \geq 0$. Thus we conclude that solution trajectories starting in Ω' remain in Ω'. Also, because $I_{1W} < N_1/c_1$, we see that from equation of W in Section 2.4, $W(t)$ is decreasing for any $W > \alpha N_1/\mu c_1$. This means that $W(t)$ is bounded and we conclude that any solution to the complete system is bounded.

Thus, solutions with initial conditions in Ω' exist for all $t>0$ (Hirsch and Smale, 1974). □

To simplify our analysis, we restrict our system to involve only the contaminated variables and consider the following:

$$
\begin{aligned}
I'_{1B} &= \beta_{1B}(N_1/b_1 \quad I_{1B})I_{1B}+(1 \quad \rho)N_1 \quad b_1 I_{1B},\\
I'_{1W} &= \beta_{1W}(N_1/c_1 \quad I_{1W})W+b_1 I_{1B} \quad c_1 I_{1W},\\
I'_{1A} &= \beta_{1A}(N_1/d_1 \quad I_{1A})I_{1A}+c_1 I_{1W} \quad d_1 I_{1A},\\
I'_{2W} &= \beta_{2W}(N_2/c_2 \quad I_{2W})W \quad c_2 I_{2W},\\
I'_{2A} &= \beta_{2A}(N_2/d_2 \quad I_{2A})I_{2A}+c_2 I_{2W} \quad d_2 I_{2A},\\
W' &= \alpha I_{1W} \quad \mu W,
\end{aligned}
\tag{3.2}
$$

where the phase space for (3.2) is $\Omega=\{I_{1B},I_{1W},I_{1A},I_{2W},I_{2A}, W\geq 0\}\subset\Omega'$.

It is not hard to show that (3.2) has a unique positive equilibrium

$$E^*=(I^*_{1B},I^*_{1W},I^*_{1A},I^*_{2W},I^*_{2A},W^*)\in\Omega$$

where

$$I^*_{1B}=\frac{N_1\beta_{1B} \quad b_1^2}{2b_1\beta_{1B}}+\frac{\sqrt{(N_1\beta_{1B} \quad b_1^2)^2+4(1 \quad \rho)N_1 b_1^2\beta_{1B}}}{2b_1\beta_{1B}}, \tag{3.3}$$

$$I^*_{1W}=\frac{N_1\beta_{1W}\alpha \quad c_1^2\mu}{2c_1\beta_{1W}\alpha}+\frac{\sqrt{(N_1\beta_{1W}\alpha \quad c_1^2\mu)^2+4b_1\mu c_1^2\beta_{1W}\alpha I^*_{1B}}}{2c_1\beta_{1W}\alpha}, \tag{3.4}$$

$$I^*_{1A}=\frac{N_1\beta_{1A} \quad d_1^2}{2d_1\beta_{1A}}+\frac{\sqrt{(N_1\beta_{1A} \quad d_1^2)^2+4c_1 I^*_{1W} d_1^2\beta_{1A}}}{2d_1\beta_{1A}}, \tag{3.5}$$

$$I^*_{2W}=\frac{\beta_{2W}\frac{\alpha}{\mu}I^*_{1W}N_2/c_2}{c_2+\beta_{2W}\frac{\alpha}{\mu}I^*_{1W}}, \tag{3.6}$$

$$I^*_{2A}=\frac{N_2\beta_{2A} \quad d_2^2}{2d_2\beta_{2A}}+\frac{\sqrt{(N_2\beta_{2A} \quad d_2^2)^2+4c_2 I^*_{2W} d_2^2\beta_{2A}}}{2d_2\beta_{2A}}, \tag{3.7}$$

$$W^*=\frac{\alpha}{\mu}I^*_{1W}. \tag{3.8}$$

Because I_{1B} decouples from the other equations in system (3.2), and because I_{1B} converges to I^*_{1B} as $t\to\infty$, we consider the following system:

$$
\begin{aligned}
I'_{1W} &= \beta_{1W}(N_1/c_1 \quad I_{1W})W+b_1 I_{1B} \quad c_1 I_{1W},\\
I'_{1A} &= \beta_{1A}(N_1/d_1 \quad I_{1A})I_{1A}+c_1 I_{1W} \quad d_1 I_{1A},\\
I'_{2W} &= \beta_{2W}(N_2/c_2 \quad I_{2W})W \quad c_2 I_{2W},\\
I'_{2A} &= \beta_{2A}(N_2/d_2 \quad I_{2A})I_{2A}+c_2 I_{2W} \quad d_2 I_{2A},\\
W' &= \alpha I_{1W} \quad \mu W.
\end{aligned}
\tag{3.9}
$$

In light of the fact that our original system is positively invariant on Ω', it is easy to see that the decoupled system (3.9) is positively invariant on Ω. It follows by inspection that the off diagonal terms of the Jacobian matrix of system (3.9) are non negative on Ω, indicating that system (3.9) is monotone on Ω (Smith, 1995).

We are interested in the relative long term dynamics of system (2.1) in order to understand how contamination occurs over an extended period of continual washing. Because system (3.9) is monotone, we can use Theorem C in Jiang (1994) to show that any solution, with non negative not identically zero initial data, of (2.1) converges to E^*. That is, E^* is the unique globally asymptotic positive steady state of (2.1).

4. Application of model to *E. coli*0157:H7 contamination of fresh-cut romaine lettuce

In this section, we apply our model to predict the spread of *E. coli* O157:H7 among fresh cut romaine lettuce during a com mercial biocidal wash procedure (let P1 represent romaine lettuce and P2 represent romaine lettuce from a different source or another type of leafy green). In this instance, we suppose that chlorine is being used as the sanitizing agent in the wash water. Using experimental data and estimation techniques, we can establish a feasible range for each of the parameters in our model. However, because the actual parameter values are not precisely known, we make use of uncertainty and sensitivity analysis. Focusing on the wash procedure, we want to know which parameters have the most affect on the amount of contaminated produce. Within the range of these parameters, numerics indicate that solutions of our model (in Section 2.4) converge quickly to the steady state E^* (less than ≈ 0.15 days), allowing us to ignore the transient dynamics and look at the effects of parameter variance on I^*_{1W} and I^*_{2W} (we justify this in Section 4.2). Our results show that $1/c_1$, the mean wash time for P1, and $1/\mu$, the mean pathogen lifetime (which we link to the free chlorine concentration below) are key parameters in this respect.

4.1. Parameter ranges

We first establish baseline values for parameters involved with P1. From Luo et al. (2011), we suppose in the W stage that the wash water is held at an average of 22 °C and the produce to water ratio averages at 1:20. Assuming the wash tank is 4800 L (see Barrera et al., 2012 for comparable tank sizes), we must have 240 kg of P1 present in the tank at any given time. This implies that $N_1=240$ kg. Furthermore, assume that the plant can process up to 24,000 kg of P1 per day (again, see Barrera et al., 2012 for comparable processing rates). This means that the average wash time is 0.01 days, which implies that $1/c_1=0.01$ days (or 30 s, see Luo et al., 2011). For simplicity, we also set $1/b_1=0.01$ days. Setting $\rho=0.95$, we assume 5% of P1 coming into the pre wash stage is contaminated. Because we want to focus on the dynamics of contamination in the W stage, we set $\beta_{1B}=0.1$ mL $(\text{CFU})^{-1}$ $(\text{day})^{-1}$, which allows the contamination in the pre wash stage to stay less than or equal to $\approx 6\%$ of 240 kg (see Table 1 in Barrera et al., 2012) and we ignore the dewatering stage. For ranges about these baseline values, see Table 3. Justi fication for the ranges of β_{1W}, β_{2W}, α and μ in Table 3 are provided.

4.1.1. Mean pathogen lifetime in wash water $1/\mu$

Using data from Zhao and et al. (2001) we can model the kill rate of *E. coli* O157:H7 in solution relative to the concentration of free chlorine via exponential decay. While Zhao and et al. (2001) considered only concentrations of free chlorine up to 2 mg/L, using MATLAB's curve fit tool (cftool), we fit a function for μ in terms of the free chlorine concentration c, given by

$$\mu(c)=957.9c^{0.172}.$$

Table 1
Variables with units for the wash model.

S_{1B},S_{1W},S_{1A}	Susceptible density of P1 in each stage	kg
I_{1B},I_{1W},I_{1A}	Contaminated density of P1 in each stage	kg
S_{2W},S_{2A}	Susceptible density of P2 in each stage	kg
I_{2W},I_{2A}	Contaminated density of P2 in each stage	kg
W	Pathogen concentration in water	CFU mL ^{1}

Using this function and allowing the free chlorine concentration to vary on [0.02, 126] mg/L, we build an allowable range for μ as $500 \le \mu \le 2200$. Thus $1/\mu$ stays in the range [0.00045,0.002] day.

4.1.2. Pathogen shed rate α

Luo et al. (2011) found that the reduction of *E. coli* O157:H7 populations on romaine lettuce was significantly linked to the free chlorine concentration used. Based on their data and the aforementioned curve fit tools, we describe the connection between α and c as follows:

$$\alpha(c) = \ 12.77c^{-0.6129} + 207.8.$$

Again, allowing the free chlorine concentration to vary on [0.02, 126] mg/L, the corresponding range for α is [67, 207] (CFU) kg ^{1} day ^{1} mL ^{1}.

4.1.3. Water produce transmission rate β_{1W}

The usual definition of transmission in epidemic models, derived originally from collision theory for chemical reactions, states that the transmission rate = the average number of contacts per unit time between susceptible and infected individuals $\times$ the probability of "successful contact" (Brauer, 2008). In terms of our model, we want to determine both the average number of contacts a cut piece of lettuce makes with *E. coli* in solution and the probability of attachment.

The number of contacts relies critically on the produce to water ratio and we fix this to be 1:20 as above. Assuming that this concentration is maintained throughout the wash tank, we suppose that (1/20,000) kg of produce can be present in 1 mL of water. Also, the lettuce are cut into pieces with dimensions: 25.4 mm $\times$ 25.4 mm $\times$ 0.03 mm (Luo et al., 2011; Thomas, 2010), which translates into 0.08 pieces/mL. To calculate the average number of contacts, we want to calculate the number of "mL" of solution "0.08 pieces" of lettuce hits along its path through the wash tank. Currently we have no data for the average path length of a piece, but we estimate the distance traveled to range anywhere from 2 to 10 m during one wash period. Simplifying this further, we assume that our "mL" pieces of water are cubes and once a piece of lettuce hits a "face" of the mL cube, it travels normal to that face through the mL. This means then the number of mL hit is the distance traveled by the lettuce divided by the cross sectional length of the mL. Applying this reasoning, and the fact that 0.08 of a piece of lettuce has volume 0.0015 mL, we see that 0.08 of a lettuce piece hits on average 1.97 9.84 mL per wash period.

To estimate the probability of attachment, we extrapolate from an experiment in Luo et al. (2011). Using their data, we calculate that the number amount of bacteria found on 120 g of initially clean lettuce (when washed with 30 g of inoculated lettuce, see Luo et al., 2011 for details) divided by the average number of pathogen shed during one wash period is 0.0095. That is, 0.95% of *E. coli* in solution successfully attached to the clean lettuce pieces.

As we assumed above, the plant processes 24,000 kg/day of lettuce, meaning that there are 100 wash periods per day. In light of this, our range for the number of contacts is 197 984 mL/day. Combining this with the probability of attachment, we see that $\beta_{1W} \in [2,10]$ mL (CFU)$^{-1}$ (day)$^{-1}$.

4.2. Sensitivity analysis

To find which parameters most affect I^*_{1W} and I^*_{2W}, we use Latin hypercube sampling (LHS) to build a sample matrix of parameter input values. Here we suppose that each parameter is sampled randomly from a uniform distribution across its respective range. Using a sample size of $n=500$, and then rank transforming the sample matrix and corresponding outputs for I^*_{1W} and I^*_{2W}, we calculate the partial rank correlation coefficients (PRCCs) asso ciated to each parameter (see Marino et al., 2008). In general, the PRCCs, valued between 1 and +1, provide a measure of monotonicity between each parameter and a selected output.

Before discussing the PRCCs that link each parameter to I^*_{1W} and I^*_{2W}, we substantiate why we can ignore transient dynamics and focus solely on the steady state amounts of contamination in the wash stage. Using LHS as described above, with $n=500$, we perform Monte Carlo simulations to calculate the corresponding outputs for $I_{1W}(t)$ and $I_{2W}(t)$ for $t \in [0,0.3]$ days. For visual clarity, after fitting a normal distribution to the respective outputs for $I_{1W}(t)$ and $I_{2W}(t)$, we calculate the 95% confidence intervals. In order to avoid redundancy, we illustrate the confidence interval vs time for P1 only, see Fig. 4. Referring to Fig. 4, notice that the transient dynamics are relatively fast as the solution $I_{1W}(t)$ is very close to steady state within ≈ 0.1 days (the behavior is similar for $I_{2W}(t)$ and is on the order of 0.15 days).

Now making use of the steady state outputs I^*_{1W} and I^*_{2W}, Fig. 5 illustrates the PRCCs and the corresponding parameters which have the most influence on the contamination amounts of P1 and P2 in the wash stage. c_1 and c_2 have the highest magnitude among all PRCCs which make sense as $1/c_1$ and $1/c_2$ reflect the mean wash times for P1 and P2, respectively. However, it is noteworthy that I^*_{2W} is also highly sensitive to c_1. This means that the average

Table 2
Parameters with units for the wash model.

β_{1B}	Direct cont. rate for P1 in stage B	kg ^{1} day ^{1}
N_1,N_2	Arrival rate of P1 and P2, resp.	kg day ^{1}
$1/b_1$	Mean period of P1 in stage B	Day
β_{1W},β_{2W}	Water–produce transmission rate	mL (CFU) ^{1} day ^{1}
$1/c_1$	Mean wash time for P1	Day
$1/c_2$	Mean wash time for P2	Day
α	Pathogen shed rate	CFU kg ^{1} day ^{1} mL ^{1}
$1/\mu$	Pathogen lifetime in wash water	Day
β_{1A},β_{2A}	Direct cont. rate for P1 and P2 in stage A	kg ^{1} day ^{1}
$1/d_1$	Dewatering period for P1	Day ^{1}
$1/d_2$	Dewatering period for P2	Day ^{1}

Table 3
Parameter ranges and units with references.

Parameter	Description	Range	Reference
β_{1B}	Cont. rate for P1 in B	[0.05, 0.3] kg ^{1} day ^{1}	Barrera et al. (2012)
$1-\rho$	% Incoming contaminated P1	[0.01, 0.15]	
N_1,N_2	Arrival rate of P1 and P2	[18,000, 30,000] kg day ^{1}	Barrera et al. (2012)
$1/b_1$	Pre-wash period of P1	[0.007, 0.02] day	Barrera et al. (2012), Luo et al. (2011), and Tomás-Callejas et al. (2012)
β_{1W},β_{2W}	Water–produce transmission rate	[2, 10] mL (CFU) ^{1} day ^{1}	
$1/c_1$	Mean wash time for P1	[0.007, 0.02] day	Barrera et al. (2012), Luo et al. (2011), and Tomás-Callejas et al. (2012)
$1/c_2$	Mean wash time for P2	[0.007, 0.02] day	Barrera et al. (2012), Luo et al. (2011), and Tomás-Callejas et al. (2012)
α	Pathogen shed rate	[67, 207] CFU kg ^{1} day ^{1} mL ^{1}	
$1/\mu$	Pathogen lifetime in wash water	[0.00045, 0.002] day	

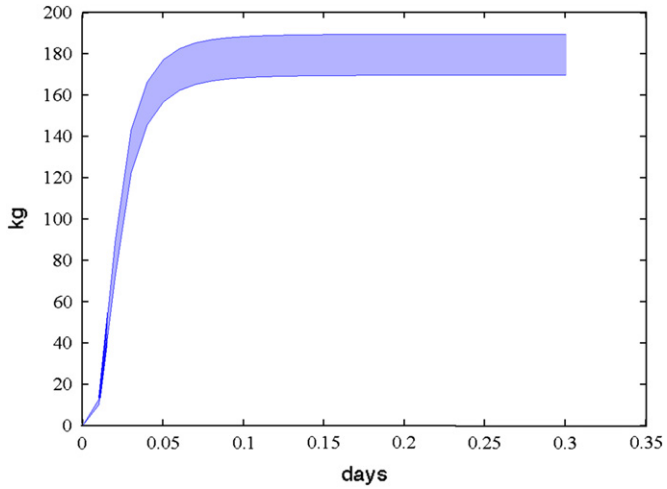

Fig. 4. Dynamics of amount of contaminated P1 (kg) vs time (days) in the Wash stage. The blue region indicates 95% confidence interval for $I_{1W}(t)$. Notice the transient dynamics are relatively short, on the order of ≈ 0.1 days. (For interpretation of the references to color in this figure caption, the reader is referred to the web version of this article.)

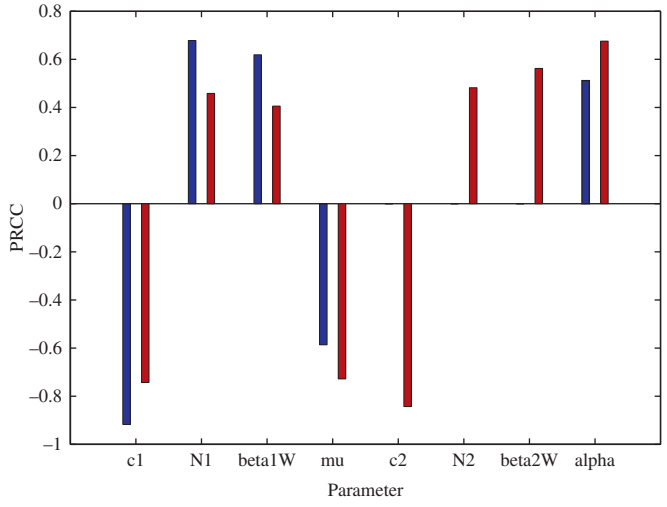

Fig. 5. The blue bars give the PRCC between the corresponding parameter and I^*_{1W}. The red bars give the PRCC between the corresponding parameter and I^*_{2W}. Note that each of the coefficients listed are significant ($p < 0.01$). (For interpretation of the references to color in this figure caption, the reader is referred to the web version of this article.)

wash time for P1 has a significant affect on the contamination amount of P2. Furthermore, both outputs are quite sensitive to μ which again is reasonable as μ is directly connected to the free chlorine concentration used. While it is clear in Fig. 5 that other parameters play a significant role, we want to focus on c_1 and μ as these parameters are the most influential on I^*_{1W} and I^*_{2W} simultaneously and because these parameters are subject to processing control.

4.3. Implications for disease surveillance due to wash time and sanitizer concentration

In Section 4.2, we showed that $1/c_1$ (mean wash time) and $1/\mu$ (mean pathogen lifetime in wash water) have significant influ ence on the amount of contaminated produce, both P1 and P2, in the wash tanks. Fortunately, both these parameters can be practically controlled during processing. By fixing either c_1 or μ at various points along their respective ranges and letting all other parameters vary, we gain predictive information towards the amount of contamination of P1 and P2 as well as insight into potential source misidentification. In particular, we build sample matrices (as in Section 4.2), as c_1 and μ vary along their respective ranges, generating a range of outputs (I^*_{1W} and I^*_{2W}) for each fixed c_1 and μ. We then fit a normal distribution to the range of values of I^*_{1W} and I^*_{2W} (for each fixed c_1 and μ) and compute the 95% confidence intervals for both outputs. See Figs. 6 and 7 for the display of these results.

Examining Fig. 6, we draw two conclusions. First we see that even with short mean wash times (less than 24 s), the chlorine sanitizer is not able to eliminate the *E. coli* as contaminated P1 in the wash stage stabilizes at around 75 kg and cross contamination of P2 stabilizes at around 100 kg. Furthermore, in terms of the proportion of contaminated P1, if we let $N_1 = 24{,}000$ kg/day (the average value of N_1 on its range), using the data in Fig. 6, we see that this proportion on average ranges from about 30 to 80% as the mean wash time varies from about 20 to 60 s. So we see that decreasing the average wash time for P1 decreases the proportion of contam ination among P1, but because the wash process is continuous, the build up of *E. coli* in the wash water seems to only be marginally controlled by the sanitizer, despite the wash time.

The second notion concerns potential for misidentification of the food vehicle which is originally contaminated. For instance, if an *E. coli* outbreak is suspected, the identification of the food vehicle initially connected to the contamination is crucial for implementing effective disease control. However, cross contamination may pose difficulties for such diagnoses. To illus trate this, consider that P1 comes into a processing plant with significant contamination. If the sanitizer is not able to effectively eliminate the pathogens in the process water, our model shows that P2 will also become contaminated. Now depending on various parameters in our model, the amount of contaminated P2 in the wash stage can eventually surpass that of P1. This means that the plant could at some point be shipping out a higher volume of contaminated P2 than P1. While the complexity of the supply chain and human behavior may lead to a variety of outcomes, because higher amounts of contaminated P2 than P1 are moving into the supply chain, we consider the potential situation in which P2 will be associated more strongly to the outbreak when statistical studies are conducted. The problem here is that P1 may go unnoticed as the initial food vehicle for sometime. The lack of control associated to the contamination source would then cause more illness and economic problems.

In terms of Fig. 6, we see that the 95% confidence interval (CI) for values of I^*_{1W} (indicated by the blue region) intersects the 95% CI for I^*_{2W} (red region) for $95 < c_1 < 125$, i.e. for mean wash times between 30 and 24 s. This overlap (indicated in purple) implies the likelihood of confusion about which produce is the primary food vehicle. Furthermore, we see that for $c_1 > 125$ (mean wash time less than 24 s) the contamination of both P1 and the cross contamination of P2 is minimized, but following the above reasoning, P2 will most likely be misidentified as the initial food vehicles as the CI for I^*_{2W} lies completely above the CI for I^*_{1W}. For $c_1 < 95$ (mean wash time > 30 s), we see the reverse situation as the CI for I^*_{1W} lies completely above the CI for I^*_{2W}. Therefore, as contaminated P1 moves down the supply chain in greater numbers than P2, the original food vehicle will most likely be identified correctly. In reality, the initially contaminated food vehicle would be unknown. In light of this fact, we can use the results in Fig. 6 to avoid misidentification, by setting the mean wash times for both P1 and P2 to be greater than 30 s.

The other parameter of significance is μ as it connects directly to the free concentration of chlorine sanitizer used to treat the process water. Our results align clearly with the fact that if the concentration of sanitizer is increased (i.e. increase in μ), then the

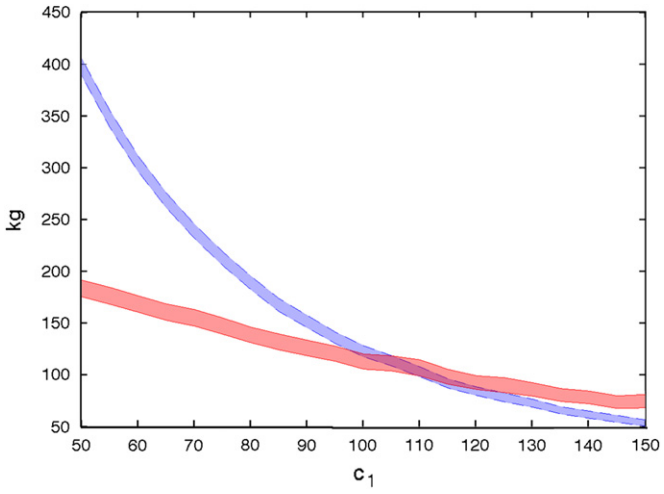

Fig. 6. Contamination amounts of P1 and P2 (kg) vs c_1 (1/day). The blue region indicates 95% confidence interval for I^*_{1W} and the red region illustrates 95% confidence interval for I^*_{2W}. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

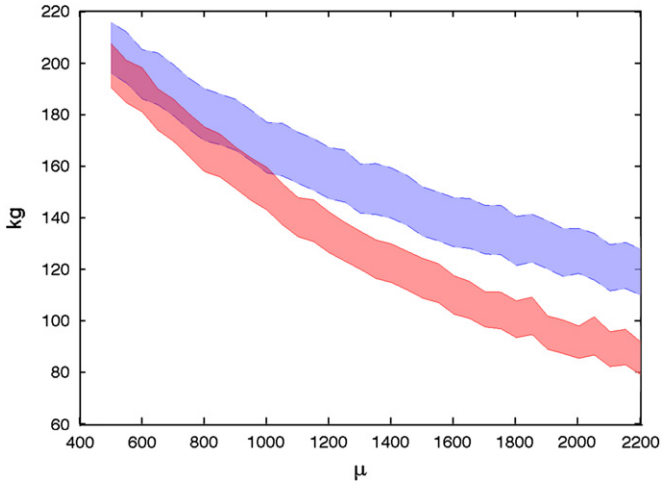

Fig. 7. Contamination amounts of P1 and P2 (kg) vs μ (1/day). The blue region indicates 95% confidence interval for I^*_{1W} and the red region illustrates 95% confidence interval for I^*_{2W}. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

contamination amounts of both P1 and P2 will decrease. However, we want to use our model to again inform control against misidentification of the contamination source. In this instance, suppose P1 is cut romaine lettuce and P2 is another type of cut produce which has a higher degree of surface roughness than P1 (e.g. shredded cabbage). It has been shown that an increase in surface roughness leads to an increased rate of *E. coli* attachment (Wang et al., 2009). Therefore, we assume the range of β_{2W} (the water produce transmission rate) is slightly increased, varying in the range [3, 12] mL $(\text{CFU})^{-1}$ $(\text{day})^{-1}$. Applying our model to this situation, we perform similar analysis as with c_1 except now we fix μ and vary all other parameters. The results are illustrated in Fig. 7. Here we see that for $\mu < 1100$ (free chlorine concentration ≤ 2.2 mg/L), the 95% CI for I^*_{1W} and the 95% CI for I^*_{2W} intersect or are quite close (see purple region in Fig. 7). On the other hand, for $\mu > 1100$ (free chlorine concentration > 2.2 mg/L, the CI for I^*_{1W} (blue) lies completely above that of I^*_{2W} (red). These results suggest that using free chlorine concentration less than 2.2 mg/L will likely result in, at worst, the misidentification of the contamination source, and at best, confusion about the source.

5. Discussion

Many studies suggest that the disinfection stage of processing is one of the most important points in the production chain, affecting the "quality, safety, and shelf life of the end product" (Gil et al., 2009). One reason for this is that wash water may become contaminated during the course of processing and serve as a secondary source of contamination. In order to understand this phenomenon, researchers typically conduct studies at the lab scale, discussing the effects that parameters such as free chlorine concentration, pH, temperature, etc., have on contamination during a single wash period (Gil et al., 2009). However, on a commercial scale, the wash process is continual, and therefore, the effects of this dynamic are important to explore.

Our model is setup to describe a continuous wash process of fresh produce, allowing us to predict contamination and cross contamination amounts over an extended period of time. Notice, however, that our assumptions in the wash stage, namely, that we ignore produce to produce contact, pathogen growth in the wash water, and shedding from contaminated P2, suggest that our results are an underestimation. For example, keeping the significance of this underestimation in mind, when applied to the case of fresh cut romaine lettuce, our model shows that even when a small proportion (1 5%) of incoming lettuce is contaminated with *E. coli* O157:H7, acceptable levels of free chlorine (we consider free chlorine concentrations up to ≈ 125 mg/L) are not able to eliminate the pathogen in solution. In fact after only about 20 wash cycles, the contamination level in solution stabilizes above a positive threshold, causing substantial amounts of contaminated lettuce to move into the dewatering stage and eventually into the supply chain. The underlying reason here is that the pathogen population is able to sufficiently compound in the wash water as contaminated produce continuously enters the wash stage. Furthermore, if another processing line is connected via water recirculation, our results predict that produce along this secondary line will suffer from cross contamination. While using separate water sources for each line would be ideal for preventing cross contamination, recirculation is a standard practice in the food industry as managing water usage and waste is expensive. Taking both our model and this practice into account, it would be interesting to find a strategy that simultaneously minimizes water consumption and contamination levels.

As a result of quantifying the dynamics of produce contamination, our system can also give insight towards potential source misidentification. In particular, we apply the model to washing fresh cut romaine lettuce together with the same type of lettuce from a different farm or another type of leafy green with greater surface roughness. Considering *E.coli* 0157:H7 as the contaminant, our results indicate that keeping the free chlorine concentration above 2.2 mg/L and washing all produce for at least 30 s will with strong likelihood prevent misidentification of the initial food vehicle. With sufficient data, we can use our model to make similar conclusions about the free chlorine level and length of wash time associated to other types of produce.

For a more realistic approach, certain model parameters should include time dependence. For instance, sanitizer concentrations usually change due to periodic dosing and the presence of organic matter in the wash water (Gil et al., 2009). By allowing μ to be an appropriate periodic function of time, we could more closely approximate this phenomenon. Also, as the sanitizer is not instantly effective when added, incorporating a delay in μ would be significant.

Another important step involves adding processing parameters to the equation for W in (Section 2.4) such as temperature, turbidity, organic load, pH, agitation/mixing, etc. Not only are these details important for optimizing specific wash conditions,

but the results from our augmented model would be able to provide a benchmark against which various wash and sanitization procedures can be compared. This is currently an unresolved issue in the food industry as "the lack of a standardized methodology and validation procedure makes it difficult to select the most adequate sanitizing strategies for the disinfection of fresh cut produce" (Gil et al., 2009). In order to fit our model to address this dilemma, relevant processing data are needed.

Finally, in terms of the big picture, our three stage model can connect to a multi scale model that includes supply chain net work and shipping dynamics. This larger scale model can then be used to simulate outbreak events on a global level, providing spatial and temporal maps of contamination "hubs" or "hot spots". Such information would be vital for effective food borne disease control and prevention.

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