ORIGINAL ARTICLE

The impact of shared sanitation facilities on diarrheal diseases with and without an environmental reservoir: a modeling study

Matthew R. Just^{a†}, Stephen W. Carden^a , Sheng Li^b, Kelly K. Baker^c , Manoj Gambhir^d and Isaac Chun-Hai Fung^e

^aDepartment of Mathematical Sciences, Georgia Southern University, Statesboro, GA, USA; ^bCUNY School of Public Health, City University of New York, New York City, NY, USA; ^cDepartment of Occupational and Environmental Health, College of Public Health, The University of Iowa, Iowa City, IA, USA; ^dEpidemiological Modelling Unit, Faculty of Medicine, Nursing and Health Sciences, Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia; ^eDepartment of Epidemiology and Environmental Health Sciences, Jiann-Ping Hsu College of Public Health, Georgia Southern University, Statesboro, GA, USA

ABSTRACT

Epidemiological studies have identified an increased risk of diarrheal diseases associated with using shared sanitation facilities. We hypothesized that this might be related to differences in transmission routes of pathogens. We proposed a mathematical model of two fictitious pathogens, one transmitted with an environmental reservoir and one without. We assumed that individuals susceptible to one pathogen are not susceptible to the other, and therefore, decoupled the two models. We initialized the model with 99% individuals being susceptible. We sampled the parameter space using Latin Hypercube Sampling. We simulated 10,000 parameter sets. We varied the effective shared sanitation coverage (the product of latrine coverage and users' compliance). Our results show that, in our hypothetical scenario, across all levels of effective coverage of shared sanitation, the median final cumulative incidence of diarrheal disease was higher than that of zero coverage. Our simulation findings suggest that increasing effective coverage of shared sanitation may have limited benefits against diarrhea-causing pathogens with an environmental reservoir and may lack benefit against diarrhea-causing pathogens without an environmental reservoir given increased human contacts if latrines are poorly maintained.

KEYWORDS

Diarrhea; Mathematical model; Sanitation

Introduction

Sanitation facilities enable proper disposal of human excreta, reduce the contamination of the environment by open defecation [1], and thereby prevent the indirect transmission of certain diarrhea-causing pathogens via an environment reservoir. Striving to “ensure access to water and sanitation for all” (Goal 6, Sustainable Development Goals), the United Nations aims to “achieve access to adequate and equitable sanitation and hygiene for all and end open defecation, paying special attention to the needs of women and girls and those in vulnerable situations” [2]. Intervention programs that aim to increase latrine coverage in poor villages and urban slums in less industrialized countries may help reduce diarrhea disease burden [1]. A recent “WASH Benefits” cluster-randomized trial conducted in Bangladesh found that children in households receiving sanitation interventions experienced 39% reduction in 7-day diarrhea prevalence compared with controls [3]. However, in a sister study in Kenya researchers found that the sanitation intervention did not lower diarrhea prevalence

compared with the active controls [4]. According to the WHO/UNICEF Joint Monitoring Program, shared latrines of otherwise improved design are not considered as safe as private latrines, because shared latrines are thought to be more poorly maintained than private facilities, and thus may increase exposure of users to feces of other users [5]. There is epidemiological evidence that shared sanitation access may increase the burden of diarrheal diseases. A meta-analysis of twelve studies reporting on diarrhea included in a systematic review identified an increased risk of 44% for those who shared sanitation facilities compared with those who used individual household latrines [6]. Two matched case-control studies in two separate cholera outbreaks in a refugee camp in Kakuma, Kenya, found increased risk of watery diarrhea for individuals (both children and adults) who shared latrines with other households [7,8]. Prevalence data from Demographic and Health Surveys from 51 countries showed that children from households sharing sanitation facilities have a higher diarrheal disease burden than those using non-shared facilities [9]. A multinational

CONTACT Isaac Chun-Hai Fung cfung@georgiasouthern.edu

[†]Current affiliation: Department of Mathematics, University of Georgia, Athens, GA 30602

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case-control study also found that young children from households who shared sanitation facilities with other households had a higher risk of moderate-to-severe diarrhea [10]. While both Fuller et al. [9], and Baker et al. [10], were measuring the risk of diarrheal diseases under the age of five who are unlikely users of shared latrines, such observed associations suggest the possibility of an increased risk of diarrhea for latrine users (of age five and above) who may transmit the pathogens and increased the risk of diarrhea for the young children in the same households. More recently, in a cross-sectional study in Bangladesh, researchers used sentinel toy balls as an indicator to measure fecal contamination of the household environment. They found that households whose improved sanitation was shared by two to five households had a higher fecal contamination compared with private improved sanitation, but the adjusted association was not significant [11].

The risks versus benefits of shared latrines remain unclear. On the one hand, reduced practice of open defecation will reduce widespread contamination of the environment and reduce risk of diarrhea at the community level. On the other hand, sharing a latrine with other users may increase exposure to feces of other users and increase exposure to a less hygienic latrine environment that increases the risk of infection via fomites. Additionally, there are many species of pathogens that cause diarrheal diseases. These pathogens may vary in how they survive and decay in the environment and how they were transported in the environment. Some spread primarily through environmental routes and with environmental reservoirs (such as contaminated water sources). Others are primarily transmitted between humans via close contacts and fomites without any environment reservoir. Pathogen-specific transmission ecologies may also contribute to observations of whether shared latrines are safe or unsafe.

Mathematical models can help explain complicated transmission scenarios and shed light on the key determinants and processes of the transmission dynamics of infectious diseases. Infectious disease transmission dynamics is non-linear and the risk of infections depends on factors that are changing dynamically during an outbreak (e.g. the number of infectious individuals in the outbreak). Despite their assumptions and limitations, when applied appropriately, mathematical models can provide key insights into epidemiological questions and can simulate different scenarios for policy-makers to consider in the decision-making process [12].

In this paper, we hypothesize that the observed association between diarrheal disease burden and the effective coverage of shared sanitation facilities might in part be explained by an increase of transmission of some types of diarrhea-causing pathogens that are primarily transmitted between humans without any environmental reservoir, which counteracts some of the benefits of

feces containment. When considering the cost-effectiveness of rolling out shared sanitation facilities, the risk of increasing human-to-human transmission of diarrhea-causing pathogens while decreasing environmental transmission must be factored. To achieve the Goal 6 of the Sustainable Development Goals [2] and to attain the goal of reducing cholera deaths by 90% by 2030 as pledged in the 2017 declaration to ending cholera [13], the interacting effects of sanitation and pathogen-specific transmission patterns is particularly important for predicting how well global development will address emerging disease issues. We propose a mathematical model to illustrate our hypothesis.

Methods

Rationale for the conceptualization of the model

We built a mathematical model using ordinary differential equations (ODEs). We assumed a theoretical population that is stratified by effective coverage with a shared sanitation intervention (either covered or not) and by risk of one of the two fictitious pathogens. One is a fictitious pathogen that is indirectly transmitted via a water body where it persists for a long time. The other is a fictitious pathogen that is directly transmitted between human beings. A system of ODEs models the spread of both the environmentally transmitted pathogen as well as the directly transmitted pathogen. We make an explicit simplifying assumption that individuals who would be infected with the pathogen with environmental reservoir would not be infected with the pathogen that is without environmental reservoir, and vice versa. We also assumed that there is no cross-immunity between the two pathogens. Therefore, the two sub-models are de-coupled from each other.

In order to parameterize our models of two fictitious pathogens, we chose parameter values from published mathematical models of norovirus and *Vibrio cholerae* for our directly and indirectly transmitted pathogens respectively. While we fully acknowledge that both pathogens can transmit via both direct and indirect routes, the facts that *V. cholerae* can replicate in water bodies and that norovirus does not replicate outside the human bodies provide a basic justification of our simplifying assumptions. Furthermore, given the high infectivity/pathogenicity of norovirus (a dose as low as about 10 viable virions as opposed to at least 100–1000 times that amount for *V. cholerae*), published models of norovirus often incorporate the simplifying assumption of direct transmission (i.e. risk of transmission being proportional to risk of contact with infected individuals instead of being proportional to the environmental concentration of a pathogen) [14]. This practice substantiated our choice of using norovirus model parameter values to parameterize our fictitious pathogen that is directly transmitted between human beings.

Figure 1. Schematics of our mathematical models of (a) a fictitious pathogen with an environmental reservoir and (b) a fictitious pathogen without an environmental reservoir.

Model structure

To simulate our fictitious pathogen with an environmental reservoir, we used a published cholera mathematical model as our template [15–17]. Susceptible individuals (S) may be infected by the environment (W) through the oral-fecal route at a daily contact rate per day per unit of pathogen concentration in the environment with κ being the concentration at which there is a 50% infection probability, $\beta_c \frac{W}{W+\kappa}$. Infected individuals (I) shed pathogens at a per capita rate of ϕ per day into the environment, and recover at a rate of γ and become recovered individuals (R). The pathogens in the environment are represented in the model as bacterial concentration in the water body (W, number of cells per mL of water) and they decay at a rate of μ (Figure 1).

To simulate our fictitious pathogen without an environmental reservoir, we used a published norovirus mathematical model as our template [14]. Susceptible individuals (S) became infected with the pathogen through direct contact with symptomatic infectious individuals (I) at a per capita rate of $\beta_n(1 + \alpha^2)$ per day and with exposed (E) and asymptotically infectious (A) individuals at a reduced per capita rate of 5% of $\beta_n(1 + \alpha^2)$ per day, where α indicates “effective coverage” of the intervention (defined below). Exposed individuals (E) are individuals who are in the latent period (infected but yet to be infectious) of an average length of $1/\sigma$ days. Symptomatic infectious individuals stay infectious for an average period of $1/\kappa$ days before they become asymptotically infected (A). Individuals stay asymptotically infected for an average period of $1/\rho$ days before they recovered (Figure 1). We do not include waning immunity, natural birth or death, or population

migration into this model, as our simulation is a single outbreak scenario.

Parameter values selection

Approximate ranges for the essential parameters were compiled from the literature and were summarized in Table 1. To select the appropriate values in the parameter space for our simulations, we used Latin Hypercube sampling to generate a realm of possible scenarios using values drawn from the feasible parameter space [18]. Because of the uncertainty involving many of the parameters, we sample values in the parameter space. Likewise, we included the initial concentration of the environmentally transmitted pathogen in the water environment in our Latin Hypercube sampling (within the range of 10^3 and 10^7 cells per mL) [16]. In total, we generated 10,000 sets of parameter values for this study. It is important to note that the population in each compartment was modeled as a fraction of the total population, which is equal to 1. Thus, there is no need to specify the total number of population.

Outbreak Scenario

The outbreak scenario was implemented for each set of parameter values. This was a single outbreak scenario in a totally susceptible population. We did not assume any prior immunity against these two pathogens in this population. The scenario represented an outbreak that would happen immediately after the implementation of a shared sanitation infrastructure intervention. The simulations lasted for 365 days.

Table 1. List of model parameters with explanations.

Parameter	Biological meaning	Range	Units	Reference
<i>Norovirus</i>				
β_n	Transmission rate per susceptible (norovirus)	0.66–1.26	Days ⁻¹ person ⁻¹	Simmons, et al.[14]
$1/\sigma$	Duration of incubation (norovirus)	0.5–1.5	Days	Atmar, et al.[32]
$1/\delta$	Duration of symptoms (norovirus)	1.5–2.5	Days	Atmar, et al.[32]
$1/\rho$	Duration of asymptomatic infection (norovirus)	9.5–10.5	Days	Rockx, et al.[33]
<i>Cholera</i>				
β_c	Rate of contact with pathogens in the environment (cholera)	1	Days ⁻¹	Grad, et al.[16]
$1/\gamma$	Duration of infection (cholera)	2.9–14	Days	Grad, et al.[16]
ϕ	Pathogen shedding rate (cholera)	0.01–10	Cells*mL ⁻¹ *person ⁻¹ *days ⁻¹	Grad, et al.[16]
$1/\mu$	Length of time of survival of pathogen in the environment (inverse of decay rate) (cholera)	3–41	Days	Grad, et al.[16]
κ	Concentration of pathogen that yields 50% chance of infection (cholera)	10 ⁵ –10 ⁶	Cells*mL ⁻¹	Grad, et al.[16]
<i>Both</i>				
α	Effective coverage of shared sanitation	0–1	This is a proportion and is dimensionless	

Initialization of the outbreak

The outbreak was initialized through a small fraction of infectious individuals in the populations. The initial states of the human populations were prescribed *a priori* in our models. In the model of environmentally transmitted pathogen, 1% of the population was initially infected with 99% being susceptible. In the model of person-to-person transmitted pathogen, 0.5% of the population was initially in the exposed category and 0.5% in the infected category and 99% susceptible. These values were arbitrarily chosen for illustration purposes.

Effective coverage of shared sanitation

We defined *effective coverage* as the average proportion of the population compliant with a shared sanitation intervention. This parameter is the product of both the infrastructure (latrine coverage) in a given population and accompanying socio-behavioral factors of compliance (the percentage of individuals who consistently use the latrines). We “implemented” the intervention (the expansion of effective coverage of shared sanitation facilities) in varying levels for each of the given sets of parameters (assuming that they represent different socio-environmental settings). The proportion of compliant individuals affects the transmission of each disease differently. As compliant individuals no longer contribute pathogens into the environment, the $\phi(1-\alpha)$ term in Figure 1(a) shows that the rate of pathogen contribution decreases linearly with α . Also, compliant individuals increase the transmission rate of the directly transmitted pathogen, but this increase is not linear. More individuals using shared sanitation increases the number of human-to-human interactions super linearly, represented mathematically by the quadratic term $\beta(1+\alpha^2)$ in Figure 1(b). Our approach is similar to that adopted by Hu et al. [19], in which contact scales with the square of density.

Assumption of shared latrines as fomites

Our model assumed that the risk of transmission of the (norovirus-like) pathogen without an environmental

reservoir being proportional to the risk of contact with infected individuals. To put it in terms of field epidemiology, we are assuming that the shared latrines are poorly maintained and unhygienic, and the surfaces of the latrine infrastructure serve as fomites transmitting the pathogen. As aforementioned, mathematically, this assumption was translated into the simplifying assumption of human-to-human transmission that was incorporated into our mathematical model.

Measurements used in the simulation

We used the final cumulative incidence of diarrheal disease (the fraction of the hypothetical population that ever experienced diarrheal disease within 365 days) at the end of a single simulated outbreak to measure the effects of the effective coverage of the shared sanitation. We calculate the median and the interquartile range of the prevalence of diarrheal disease across the 10,000 parameter configurations across the range of effective coverage of shared sanitation (from zero to one).

Sensitivity analyses

We set each parameter to its mid-range value of the uncertainty range. Then, each parameter is set to its minimum and maximum values respectively, while the other parameters are held constant. An outbreak is simulated and the final cumulative incidence and peak prevalence, as well as the percent change from the baseline, are obtained for each case. The results of the sensitivity analyses are presented in the Online Supplementary Materials.

We implemented the model using MATLAB (MathWorks, Inc., Natick, Massachusetts, USA). For technical details, please refer to the Online Supplementary materials.

Results

Figure 2 presents the simulation results of our models of two fictitious pathogens, with and without environmental reservoirs. The figure presents the median (with

Figure 2. The prevalence of diarrhea across different levels of effective coverage of shared sanitation of 10,000 scenarios of parameter combinations in a model of two fictitious pathogens with and without an environmental reservoir respectively.

Notes: The black line represents the total prevalence of diarrhea. The blue broken line represents diarrhea caused by the pathogen that is transmitted without an environmental reservoir. The red dotted line represents diarrhea caused by the pathogen that is transmitted via an environmental reservoir. Lines represent medians; shaded areas represent inter-quartile ranges (IQR).

inter-quartile range) of the final cumulative incidence of diarrhea across all the 10,000 parameter configurations through a range of effective coverage of shared sanitation from 0 (open defecation by everyone) to 1 (full effective coverage when everyone has access to and consistently uses shared latrines), assuming that the outbreak occurs immediately after the implementation of the intervention. In this hypothetical scenario, there is no optimal effective coverage of shared sanitation at which the median of the final cumulative incidence of diarrhea would be lower than that of 0% effective coverage of shared sanitation. While the median of diarrheal disease final cumulative incidence caused by the fictitious pathogen that was transmitted via an environmental reservoir decreases slightly as the effective coverage of shared sanitation increases, the median of diarrheal disease final cumulative incidence caused by the fictitious pathogen that is transmitted between humans without an environmental reservoir increases to a larger extent that causes the overall increase in the total final cumulative incidence of diarrheal diseases. In other words, if we assume that an increase in effective coverage of shared sanitation will increase personal contact with feces of other individuals at latrines and lead to an increase in diarrhea caused by pathogens that are transmitted person-to-person, diarrheal disease burden will probably *increase* as more and more individuals use shared sanitation facilities.

For each of the feasible parameter configurations in our simulation, we also studied the difference in the total cumulative incidence of diarrhea between full compliance and no compliance. In our simulation results, should a community adopt shared sanitation fully as compared to no sanitation at all, they would experience an increase in the final diarrhea cumulative incidence in all 10,000 sets of parameter values (as a fraction of the total normalized population: Minimum: 0.0141, first quartile: 0.0557, median: 0.0845, third quartile: 0.1273, and maximum: 0.4355).

In our sensitivity analyses, we found that the final cumulative incidence is most sensitive to the initial concentration in the environment of the pathogen with an environment reservoir, and the transmission rate of the pathogen that is transmitted directly between individuals (see Online Supplementary Materials for details).

Discussion

Our results showcase the importance of maintaining the hygiene of latrines, especially if they are shared by different households. If our assumption that unhygienic latrines serve as fomites for norovirus-like pathogens holds, increasing the effective coverage of shared latrines will increase the “contacts” between humans (via fomites) and therefore will increase the risk of diarrhea caused by such pathogens. Our results explain in part the association between the use of shared sanitation and the increase in diarrheal disease burden as observed in epidemiological studies such as Baker et al. [10], that shared sanitation increases the transmission of diarrhea-causing pathogens that are transmitted between humans without any environmental reservoirs and such increases are not compensated by the modest reduction of transmission of diarrhea-causing pathogens with environmental reservoirs.

Our hypothesis is only one among several that might explain the association between the use of shared sanitation facilities and the increase in diarrheal disease burden. Among other factors, geography and demographics are found to be associated with the use of shared sanitation facilities. An analysis of survey data from 84 low and middle income countries found that shared sanitation is more common in Africa and South-East Asia, and that it is more common among the poor, the urban residents, those with more young children and those whose head of household received no formal education [20].

The challenge of maintaining of a hygienic shared sanitation facility is not to be overlooked either. A survey of 570 households in 30 slums in Orissa, India, found that, as compared to individual household latrines, shared facilities were less clean, less likely to be functional and more likely to have feces and flies [21]. To the contrary, a study of sanitation facilities of 662 randomly selected residential properties from 35 low-income districts in

Dar es Salaam, Tanzania, found that shared sanitation facilities were more likely to be “safe and sustainable” and functional than non-shared sanitation facilities [22]. This observation might in part be explained by the possibility of pooling resources across low-income households to maintain a functional latrine. Likewise, in a recent study in Tanzania, researchers found that non-shared latrines were less likely to be clean and to be built with floors built with permanent materials, washable floors and lockable doors [23]. Furthermore, seasonality and its associated cycles of human behaviors are known to affect the hygienic conditions of shared latrines too. A study in Kampala, Uganda, found that residents were able to maintain their shared latrines in a more hygienic condition during the dry seasons as compared to the wet seasons [24]. All these issues would factor into the intertwined web of causality behind the observed increased risk of diarrheal disease associated with the use of shared sanitation facilities as found in recent studies [6,9,10]. In our study, we assumed that the latrines serve as fomites. Future studies can incorporate varying hygienic condition (or maintenance level) of latrines as variables in the mathematical models to test the effectiveness of cleaning shared latrines as an intervention. However, the major challenge is the dearth of quantitative data that informs the mathematical relationship between the frequency of cleaning latrines (the intervention), the cleanliness of the latrines (e.g. as measured by the amount of pathogens found on the surfaces of the latrines), and the risks of disease transmission specific to such pathogens.

We also acknowledge that the notion of “shared sanitation” itself encompasses a spectrum of sanitation facilities and usage patterns [25]. For example, Rheinländer and colleagues argued that shared sanitation facilities can be categorized into a number of categories, such as (a) those shared between households that know each other, (b) public toilets and (c) institutional toilets (e.g. those in workplaces) [26]. Different types of shared facilities may have different attributes in terms of user demographics and hygienic conditions and their role in disease transmission or its control may be different. For example, a survey of 295 households in 30 urban slums in Orissa, India, found that compared to neighbor-shared latrines, households using communal latrines had a lower income; with more people; received less education; were less likely to have access to piped water and were more likely to have a household member who openly defecated [27].

Strengths and limitations of our mathematical model

We illustrate our hypothesis and highlight our key message with a theoretical mathematical model of two fictitious pathogens, one with and one without an environmental reservoir. For the purpose of this study, we

applied the simplifying assumption to our model, in which we model our fictitious pathogen with no environmental reservoir after the environmental transmission of cholera, and our fictitious pathogen without environmental reservoir after the person-to-person transmission of norovirus, because of their well-understood epidemiology and the availability of published mathematical models of these pathogens [14–17]. Both *V. cholerae* and norovirus affect individuals beyond the age of 5, unlike certain pathogens such as rotavirus that is primarily a disease of toddlers. Coinfection of these *V. cholerae* and norovirus is not very common, primarily because *V. cholerae* is not very common. We understand that there are dozens of species of pathogens that caused diarrhea. We acknowledge that both *V. cholerae* and norovirus could be transmitted by more than one route. Our goal is not to represent reality in all its details, but to create a simple model of a specific example that illustrates our hypothesis and highlights our key message. Given the uncertainty associated with each of our model parameters, we sampled our parameter space through Latin Hypercube sampling [18].

Another limitation of our choices of parameter values for our fictitious pathogens is the difference in basic reproduction number (the number of secondary infections caused by one infected individual introduced to an entirely susceptible population) between cholera (e.g. <2.7 in one study using data from Zimbabwe) [28] and norovirus (ranges from around 1.6 to 4.9 based on data from developed countries) [29].

In a deterministic model, the model framework and parameter values determine the results. A limitation of our study is that some of our choices are motivated by intuition and simplicity of the model. In a sense, a particular model represents a small part of the myriad possibilities of the complex reality of the world that we live in. An avenue of possible future research is collecting data that can inform model choices through observation rather than intuition. Nevertheless, our results do present a scenario that is in line with the hypothesis, that shared sanitation facilities could facilitate the transmission of certain pathogens that do not have environmental reservoirs and could over-compensate the reduction of other diarrhea-causing pathogens that have environmental reservoirs. Our hypothesis is at least a *plausible* explanation for the observed phenomenon of the implementation of shared sanitation facilities leading to an increase in overall incidence of diarrhea diseases. We do not claim that this is the underlying explanation for all circumstances. It is certainly plausible under *certain* circumstances, and we illustrate it through our mathematical model.

In our model, we did not attempt to include details of the diversity of shared sanitation facilities and their usage. Essentially, the parameter of *effective coverage* in our model is a product of both infrastructure coverage

and behavioral compliance. This simplification highlights the essence of our hypothesis without including overbearing mathematical details in our model. We also did not attempt to incorporate variation in maintenance (or hygienic level) of latrines given the dearth of data pertinent to the quantitative relationship between latrine cleanliness and the human-to-human transmission rate of a norovirus-like pathogen. Likewise, we acknowledge that shared sanitation facilities exist in many forms, from private latrines shared by several households to public latrines in markets or slums [25,26]. While such heterogeneity is not included in our simple model, its effect on disease outcomes can be explored in future studies.

We limited the time frame of our simulations to one year. We acknowledged the limitations of our model as it did not incorporate seasonal changes in the force of infection and it did not include waning immunity. We chose to explore the outbreak scenario starting with a population that was 99% susceptible and we did not explore the endemic scenario with prior immunity in the population. Yet the simplicity of our model helps illustrate our hypothesis.

Mathematical models offer theoretical insights to our understanding of epidemiology of diarrheal diseases [17,30], while other methods, such as quantitative microbial risk assessments, would allow us to quantify the risk of exposure to diarrhea-causing pathogens that are transmitted via different routes [31]. Our mathematical model highlights the theoretical possibility of our hypothesis and future field studies will be able to provide empirical evidence to substantiate or refute our hypothesis.

Conclusions

If shared latrines are poorly maintained and serve as fomites for diarrhea-causing pathogens, an increase in their coverage and use may increase the cumulative incidence of diarrhea. Our mathematical model serves to illustrate this key policy implication.

Authors' contributions

ICHF proposed the hypothesis and conceived the study; MRJ conducted the mathematical modeling analysis under ICHF's supervision; SWC edited MRJ's Matlab codes and re-did the mathematical modeling analysis after MRJ's graduation; SWC, SL, KKB, and MG provided intellectual inputs to the study design; MRJ wrote the first draft of the manuscript; MRJ created Figure 1 and SWC and ICHF edited it; SWC created Figure 2; SWC, SL, KKB, MG, and ICHF critically revised the manuscript for intellectual content. ICHF serves as the senior author.

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No potential conflict of interest was reported by the authors.

ORCID

Stephen W. Carden <http://orcid.org/0000-0002-4793-7883>
 Kelly K. Baker <http://orcid.org/0000-0002-3774-2628>
 Isaac Chun-Hai Fung <http://orcid.org/0000-0001-5496-2529>

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