Dynamics and Control of Infections Transmitted From Person to Person Through the Environment

Sheng Li, Joseph N. S. Eisenberg, Ian H. Spicknall, and James S. Koopman

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The environment provides points for control of pathogens spread by food, water, hands, air, or fomites. These environmental transmission pathways require contact network formulations more realistically detailed than those based on social encounters or physical proximity. As a step toward improved assessment of environmental interventions, description of contact networks, and better use of environmental specimens to analyze transmission, an environmental infection transmission system model that describes the dynamics of human interaction with pathogens in the environment is presented. Its environmental parameters include the pathogen elimination rate, \( \mu \), and the rate humans pick up pathogens, \( \rho \), and deposit them, \( q \). The ratio, \( \rho N / \mu \) (\( N \) equals population size), indicates whether transmission is density dependent (low ratio), frequency dependent (high ratio), or in between. Transmission through frequently touched fomites, such as doorknobs, generates frequency-dependent patterns, while transmission through thoroughly mixed air or infrequently touched fomites generates density-dependent patterns. The environmental contamination ratio, \( \alpha / \gamma \), reflects total agent deposition per infection and outbreak probability, where \( \gamma \) is defined as the recovery rate. These insights provide theoretical contexts to examine the role of the environment in pathogen transmission and a framework to interpret environmental data to inform environmental interventions.

Abbreviations: EITS, environmental infection transmission system; SIR, susceptible-infectious-recovered.

Human infections that pass from one person to another commonly do so through environmental media, such as air, fomites, food, hands, and water. Infection transmission models for non-vector-borne infections, however, rarely specify the mode of transmission or the vehicle that carries infection from one person to another. Instead, most models assume that the dynamic details of environmental transmission can be approximated by a point contact process (1–5). With the exception of sexually transmitted diseases, however, the environment often plays a major role in transmission, especially for enteric and respiratory diseases. Moreover, these environmental processes provide important points of intervention. To promote a transmission system framework that explicitly accounts for environmental process dynamics, we present a transmission model with environmental components that mediate transmission. We call this an environmental infection transmission system (EITS) model.

A number of approaches have been presented for incorporating environmental processes of non-vector-borne infections in transmission models. One approach is to collapse across environmental dynamics, resulting in a static description of the environment (6). Another approach is to explicitly formulate environmental pathogen dynamics. This has been done for water-mediated transmission in deterministic (7–11) and stochastic (12) formulations, as well as for air- and fomite-mediated transmission of influenza (13). We both generalize and abstract these approaches in our EITS framework.

Much of the previous work on defining transmission rates in this area has focused on the household to define contact and to estimate transmission probabilities (14–17). Other
attempts to define contact have relied on conversational encounters (3, 18, 19), direct touching (3), or simultaneous presence in a room (4, 5). Conversational contact is likely relevant to airborne transmission. Direct touching could transmit environmentally acquired pathogens. The simultaneous presence of both transmission modes could generate vastly different transmission probabilities in different venues depending upon environmental conditions, human behaviors in those venues, the survival characteristics of the agent as it transits in air or on fomites, and the dose required to initiate infection.

By explicitly modeling environmental processes that mediate transmission, our EITS models differentiate air, water, and fomite pathways of transmission (and even different classes of fomite transmission). We will demonstrate how this framework provides paths to the following: 1) developing transmission parameters that can be independently measured in environmental field studies, including survival rates of pathogens in the environment, the transfer coefficient from fomites to hands, and many more; 2) formulating transmission processes specific for air, water, food, and fomites in a manner that facilitates assessment of potential environmental control effects and the interpretation of environmental pathogen measurements; and 3) developing mechanistic theory on environmentally based transmission rates, analogous to how vector-borne transmission rates are largely defined by entomologic factors that are easily measured in the field.

**MATERIALS AND METHODS**

**Model assumptions**

We present a basic EITS model with the following assumptions:

1. All individuals are identical except that one is either $S$ (completely susceptible), $I$ (infected and infectious), or $R$ (completely immune).
2. The total population size is constant.
3. The environment is a single, fixed-size, homogeneous compartment.
4. Humans are the only source of pathogens, and individuals are uniformly exposed to pathogens in the environment.
5. In the environment, pathogens instantaneously and thoroughly mix, and they do not replicate.
6. Once picked up from the environment, pathogens can instantaneously infect $S$ at a rate that is independent of prior pathogen pick up.
7. Pathogen levels in the environment diminish via first-order dynamics through pick up of pathogens by humans, die off, and environmental decontamination.

**EITS deterministic compartmental model**

As shown in Figure 1, our EITS deterministic compartmental model includes 2 types of state entities: 1) humans, which are divided into $S$, $I$, and $R$ states, and 2) live pathogens in the environment, $E$. The model is based on the following ordinary differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= -Sp\pi E \\
\frac{dI}{dt} &= Sp\pi E - \gamma I \\
\frac{dR}{dt} &= \gamma I \\
\frac{dE}{dt} &= \alpha I - E((S + I + R)\rho + \mu).
\end{align*}
\]

$\rho$ is the fraction of $E$ picked up by each person per unit of time; $\pi$ is the probability that a susceptible individual becomes infectious per pathogen $E$ picked up; $\gamma$ is the rate per individual and per unit of time of recovery from and acquisition of immunity to infection; $\alpha$ is the number of pathogens per unit of time deposited into the environment by an infectious individual; and $\mu$ is the rate at which pathogens are eliminated from the environment by any means (naturally dying, being killed by decontamination processes, or being cleaned or otherwise removed from the environment).

We used Berkeley Madonna software (20) to numerically solve the ordinary differential equations in equation 1.

**Stochastic compartmental model**

Our EITS stochastic model is Markovian. All state entities and transmission rates are defined similarly to those in the EITS deterministic compartmental model. One difference is that the state variables ($S$, $I$, $R$, $E$) are discrete integers in the stochastic model rather than continuous as in the deterministic compartmental model. Our stochastic models allow for only a single event to occur at any given time; the specific event is randomly determined by the transition rates (Table 1). In deterministic compartmental models, on the other hand, events happen continuously and simultaneously. The outputs of these 2 model structures converge as the initial numbers of infected individuals and environmental pathogens are large. As these initial values decrease, the chance of stochastic die out increases. This phenomenon does not occur in a deterministic model.

The Gillespie algorithm (21) is applied to simulate the stochastic transmission process and to randomly execute a single event at various time steps on a continuous time scale. This model was coded and run in JAVA software (Sun Microsystems, Inc., Santa Clara, California).
Choice of parameter values

To illustrate the behavior of the EITS, we chose to parameterize our model for influenza (Table 2) such that the environment corresponds to 1) frequently touched fomites, such as a door handle, that are touched by many different individuals; 2) infrequently touched fomites, such as floors or ledges; or 3) air inside a building. Any real situation might incorporate all 3 of these pathways simultaneously. We isolated these 3 conditions for the sake of clarifying the dynamics related to each condition separately. The population size, \( N (N = S + I + R) \), represents the number of people in a public indoor venue. We used a point estimate for the recovery rate derived from previous influenza models (22, 23). Except for the recovery rate, \( \gamma \), all parameters vary by route of transmission. Infectivity, \( \pi \), differs between air and fomite because the different routes of infection, inhalation for air and touched membrane for fomites, have different dose-response characteristics as illustrated from empirical studies (24–26). Estimates on environmental elimination rates, \( \mu \), are based on experimental studies in air (27) and on nonporous surfaces (28). Considering the particle size distribution of excretions, only the smaller particles stay suspended in air and are respirable, whereas larger particles rapidly settle onto surfaces where they may be picked up; thus, there is a route-specific deposit rate, \( \alpha \), for each pathway. We assume that frequently touched fomites have smaller surface areas than infrequently touched fomites and, therefore, receive proportionally less contamination. The deposit rate is governed by physical and behavioral factors, such as sneezing or cough rates and deposition and aerosolization fractions, among others that are derived from a variety of empirical studies (29–35). Analogously, the pick-up rate, \( \rho \), is governed by physical and behavioral factors, such as breathing, touching rates, and transfer efficiencies among surfaces, hands, and membranes (36–41). In order to compare across the 3 scenarios, we parameterized the scenarios to have the same \( R_0 \). For more parameterization details, refer to the Web-only supplement accompanying this paper. (This information is posted on the Journal’s website (http://aje.oxfordjournals.org/).)

RESULTS

Mathematical analysis of EITS model structure and behavior

In the EITS model, pathogens are picked up by humans at a rate, \( \rho N \), and eliminated from the environment at a rate, \( \mu \). The fraction of live environmental pathogens picked up by humans, therefore, is

\[
f_E = \frac{\rho N}{\rho N + \mu}.
\]

This fraction ranges from near zero when elimination is much larger than the pick up to near 1 if the pick up is much larger than elimination. Another important metric is the average time pathogens persist in the environment, \( t_E \).

<table>
<thead>
<tr>
<th>Event</th>
<th>Result</th>
<th>Transition Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>((S, I, R, E) \rightarrow (S - 1, I + 1, R, E))</td>
<td>(S \times \rho \times E \times \pi)</td>
</tr>
<tr>
<td>Removal</td>
<td>((S, I, R, E) \rightarrow (S - 1, I - 1, R + 1, E))</td>
<td>(I \times \gamma)</td>
</tr>
<tr>
<td>Depositing</td>
<td>((S, I, R, E) \rightarrow (S, I, R, E + 1))</td>
<td>(I \times \alpha)</td>
</tr>
<tr>
<td>Pathogen decrease</td>
<td>((S, I, R, E) \rightarrow (S, I, R, E - 1))</td>
<td>(E \times (\rho \times (S + I + R) + \mu))</td>
</tr>
</tbody>
</table>

Abbreviation: EITS, environmental infection transmission system.

Table 1. Event and Transition Rates for the EITS Stochastic Model

<table>
<thead>
<tr>
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<td>Infection</td>
<td>((S, I, R, E) \rightarrow (S - 1, I + 1, R, E))</td>
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<tr>
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</tr>
</tbody>
</table>

Abbreviation: EITS, environmental infection transmission system.

Notes:
- Based on empirical probability distribution.
- Based on exponential dose-response model.
- Based on breathing rates and touching rates.
- Based on size distribution from sneezing, where pre-evaporative particles with a diameter of <20 \( \mu \)m are assumed to remain in air.

Abbreviation: EITS, environmental infection transmission system.

Table 2. Parameter Values for an Influenza EITS Model Based on Data From the Literature

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Parameter Estimates</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recovery rate, 1/day ((\gamma))</td>
<td>Air</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>Frequently Touched Fomite</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>Infrequently Touched Fomite</td>
<td>0.2</td>
</tr>
<tr>
<td>Infectivity ((\pi))</td>
<td>Air</td>
<td>0.0517</td>
</tr>
<tr>
<td></td>
<td>Frequently Touched Fomite</td>
<td>0.0000693</td>
</tr>
<tr>
<td></td>
<td>Infrequently Touched Fomite</td>
<td>0.0000693</td>
</tr>
<tr>
<td>Elimination rate, 1/day ((\mu))</td>
<td>Air</td>
<td>8.64</td>
</tr>
<tr>
<td></td>
<td>Frequently Touched Fomite</td>
<td>2.88</td>
</tr>
<tr>
<td></td>
<td>Infrequently Touched Fomite</td>
<td>2.88</td>
</tr>
<tr>
<td>Deposit rate, pathogens/infected/day ((\alpha))</td>
<td>Air</td>
<td>693</td>
</tr>
<tr>
<td></td>
<td>Frequently Touched Fomite</td>
<td>5,244</td>
</tr>
<tr>
<td></td>
<td>Infrequently Touched Fomite</td>
<td>1,040,177</td>
</tr>
<tr>
<td>Pick up, 1/person/day ((\rho))</td>
<td>Air</td>
<td>0.0000877</td>
</tr>
<tr>
<td></td>
<td>Frequently Touched Fomite</td>
<td>0.297</td>
</tr>
<tr>
<td></td>
<td>Infrequently Touched Fomite</td>
<td>0.0000145</td>
</tr>
</tbody>
</table>

Abbreviation: EITS, environmental infection transmission system.

References for model parameterization are in the Web-only supplement (http://aje.oxfordjournals.org/).
For our 3 environmental transmission pathways (air, frequently touched fomites, infrequently touched fomites), the parameter values defined in Table 2 generate fractions of live pathogens picked up by humans of 0.01%, 99.04%, and 0.50%, respectively. They generate average persistence times of 0.115, 0.003, and 0.345 days (Table 3). These values reflect the fact that frequently touched fomites are picked up at a high rate and, therefore, have a low environmental persistence time compared with infrequently touched fomites. Air is more similar to infrequently touched fomites with respect to persistence and fractional pick up. The specific relation between these 2 transmission routes depends on pathogen-environment-specific factors.

The basic reproductive number, \( R_0 \), represents the expected number of secondary cases caused by introducing a single primary case into a totally susceptible population. As shown in the Web supplement, \( R_0 \) can be written as follows:

\[
R_0 = \frac{\alpha \times \frac{pN}{\gamma} \times \pi}{(S + I + R)\rho + \mu}.
\]

\( R_0 \) can be considered as the product of 1) total pathogens deposited by an infectious individual during his/her contagious period, \( \frac{\alpha}{\gamma} \times \frac{pN}{\rhoN + \mu} \times \pi \); 2) the proportion of pathogens picked up while still alive, \( \frac{\alpha}{\gamma} \times \frac{pN}{\rhoN + \mu} \); and 3) the infectivity of pathogens, \( \pi \). Based on the parameter values presented in Table 2, \( R_0 \) = 1.8 for the 3 model scenarios.

When the total pickup rate is much larger than the elimination rate (\( pN >>> \mu \)), \( R_0 \) approaches \( \frac{\alpha}{\gamma} \times \frac{pN}{\rhoN + \mu} \) and is independent of population size, \( N \). This results in a frequency-dependent mass action formulation and corresponds to a frequently touched fomite, such as a door handle or a frequently used workspace. When the total pick-up rate is much smaller than the elimination rate (\( pN << \mu \)), \( R_0 \) approaches \( \frac{\alpha}{\gamma} \times \frac{pN}{\rhoN + \mu} \times \pi \) and is proportional to the population size, \( N \). This corresponds to a density-dependent mass action formulation, such as either 1) airborne transmission with rapid, thorough mixing of air or 2) surface contamination where individuals infrequently touch the surface and the majority of agents die or are disinfected before the next person touches it. This density-dependent formulation is similar to the case addressed by Noakes et al. (6) in an airborne transmission model. Therefore, according to the ratio, \( \rhoN/\mu \), the EITS model can characterize airborne- or fomite-mediated transmission and, within fomite-mediated transmission, either frequently or nonfrequently touched surfaces. Multiple pathways can also be modeled, each with unique parameterizations.

By rearranging equation 4, we get

\[
R_0 = \pi \times \left( \frac{\alpha}{\gamma} \times \frac{pN}{\rhoN + \mu} \right). \tag{5}
\]

This reformulation of \( R_0 \) brings out 2 important ratios: \( (\rhoN)/\mu \), the environmental persistence ratio, an indicator of the importance of pick up compared with environmental elimination of pathogens from the environment; and \( \alpha/\gamma \), the contamination ratio, a measure of the pathogen deposition magnitude from an infectious individual. The condition, \( \rhoN/\mu > 1 \), corresponds to frequently touched fomites. Under this condition, environmental contamination, \( \alpha/\gamma \), is more likely to be picked up than to die off. The condition, \( \rhoN/\mu < 1 \), corresponds to infrequently touched fomites. Under this condition, environmental processes attenuate pathogen levels before humans are exposed.

These frequency- and density-dependent relations can also be appreciated by transforming our EITS model into an instantaneous contact model corresponding to the classic Kermack-McKendrick susceptible-infectious-recovered (SIR) model (42). To this end, we assume that the dynamics of \( E \) can be ignored, such that \( dE/dt = 0 \). Under these conditions:

\[
E = \frac{\alpha I}{(S + I + R)\rho + \mu}.
\]

Substituting \( E \) into equation 1, we have

\[
\frac{dS}{dt} = -\frac{\alpha pN}{\rhoN + \mu} SI,
\]

\[
\frac{dI}{dt} = \frac{\alpha pN}{\rhoN + \mu} SI - \gamma I,
\]

\[
\frac{dR}{dt} = \gamma I,
\]

where the term \( \frac{\alpha pN}{\rhoN + \mu} \) is equivalent to the single transmission rate parameter in the classic SIR model. The \( R_0 \) of this instantaneous contact SIR model is

\[
R_0 = N \left( \frac{\alpha pN}{\rhoN + \mu} \right) \left( \frac{1}{\gamma} \right). \tag{7}
\]

This is the same as that derived previously from our EITS model with environmental dynamics. The Kermack-McKendrick SIR model is formulated as density-dependent contact, and its \( R_0 \) is proportional to the population size. As in the EITS model, the formulation of this instantaneous contact SIR model can be considered as either density- or
frequency-dependent contact on the basis of the environmental persistence ratio, \( \rho \mathcal{N} / \mu \).

**Analyses of dynamics of the EITS model**

Figure 2 compares the influenza dynamics for the 3 EITS model scenarios (air, frequently touched fomites, and infrequently touched fomites) with the SIR model configuration shown in equation 6. The final cumulative incidence is similar for all 3 EITS transmission pathways and the SIR configuration, reflecting that they were all parameterized to have the same \( R_0 \) (Figure 2A). The SIR configuration dynamics are the fastest, although frequently touched fomite transmission exhibits similar dynamics. Air transmission dynamics are slower, and the infrequently touched fomite transmission is even slower.

These dynamic differences are reflected in the different environmental persistence times, \( \frac{1}{\rho \mathcal{N} / \mu} \) for the 3 EITS model scenarios. Environmental persistence time is greatest for the infrequently touched fomites and, therefore, their time to peak infection prevalence is longer than for the other scenarios (Figure 2B). On the other hand, the pathogen environmental persistence time is short in the case of the frequently touched fomites, so their time to peak infection prevalence is the shortest.

The specific timing and dynamics will vary by the specific parameterization. For example, decreasing the pick-up rate and the elimination rate by the same fraction (\( \rho \mathcal{N} / \mu \) remains constant) results in slower dynamics, lower peak prevalence, and unchanged final cumulative incidence. However, the general features of the EITS model are that 1) environmental pathogen dynamics will slow the epidemic curve, 2) environmental transmission will attenuate peak incidence and prevalence, and 3) different routes of environmental transmission will exhibit different dynamics.

**Intervention effects in the EITS deterministic compartment model**

To gain insights into environmental infection control dynamics, we assess 2 types of interventions. The first affects the elimination rate parameter, \( \mu \), which corresponds to environmental decontamination. The second affects the pathogen pick-up rate parameter, \( \rho \). The pick-up rate, \( \rho \), has no simple intervention analog but could be thought of as a behavioral change that decreases environmental contact, an altered transfer efficiency, or a dilution of the environmental surface area to be touched. To illustrate intervention effectiveness in different scenarios and how interventions impact dynamics and risk, we examine 2 simple scenarios: 1) increasing the elimination rate, \( \mu \), by 25%; and 2) reducing the pathogen pick-up rate, \( \rho \), by 25%. As shown in Figure 3, both interventions have little effect on the dynamics of epidemics for the frequently touched fomite scenario. Frequently touched objects, such as doorknob handles, have a high pick-up rate, so that a small increase in the elimination rate or a decrease in the pick-up rate will have very little impact on transmission. This can also be explained by noting that \( R_0 \) for frequently touched fomites is approximately independent of both the elimination and pick-up rates. For the air and the infrequently touched fomite scenarios, however, these 2 interventions can lead to lower cumulative incidence, slower dynamics, and a smaller peak of live pathogens in the environment. Although reducing pathogen pick up is more effective than environmental decontamination, the efforts required by these 2 interventions will affect the choice of intervention. The reason why these 2 interventions are effective for air and infrequently touched fomites is that the elimination rate and pick-up rate are approximately proportional to \( R_0 \) in these 2 scenarios.

**Stochastic model analyses**

The EITS stochastic model was first analyzed to explore how an environmental contamination event affects the probability of an outbreak. To this end, we varied the initial level of environmental contamination to assess its impact on the probability of an outbreak’s occurring, defined as the proportion of simulations that resulted in attack rates of >0.05
The relation between the outbreak probability and \( E(t=0) \) is sigmoidal; that is, above a threshold contamination level, there is a region in which the probability of an epidemic increases exponentially with \( E(t=0) \). At higher initial contamination levels, the probability of an outbreak levels off. Using the probability of outbreak as an additional risk measure in microbial risk assessments may be an important complement to the currently used measures that generally rely on mean values.

We also explored the influences of human contamination, through shedding from infectious individuals, on the dynamics of epidemics (simulation details defined in Figure 5). An infectious individual sheds, on average, \( \frac{a}{c} \) pathogens into the environment during his/her contagious period. As shown in Figure 5, when fixing \( R_0 \) and the initial conditions, increasing the contamination ratio, \( \frac{a}{c} \), results in an increase in the probability of an outbreak. The relation in Figure 5 holds for any proportional change in the elimination rate, pick-up rate, and infectivity parameter values where \( R_0 \) remains constant, suggesting that, as long as \( R_0 \) is constant, these 3 parameters do not have significant influence on the probability of an outbreak. The reason that the contamination ratio can affect the probability of an outbreak, even when \( R_0 \) is constant, is that, when infectious individuals excrete fewer pathogens into the environment, reflected by a small contamination ratio, \( \frac{a}{c} \), this smaller number of environmental pathogens has a higher chance of extinction, preventing the initiation of an outbreak.

The results shown in Figures 4 and 5 also hold for both the infrequently touched fomite and air scenarios (data not shown).

**DISCUSSION**

Commonly, the mechanisms of transmission through the environment are not explicitly formulated in non-vector-borne infectious disease population dynamic models, except for some models that focus on enclosed hospital settings (31) or water-borne outbreaks (7–12). In this paper, we
present a basic conceptual framework of environmentally mediated population infection transmission for non-vector-borne infectious diseases by incorporating environmental mechanisms into epidemic models.

By use of the EITS framework presented here, the transmission rate and \( R_0 \) are formulated by well-defined and measurable environmental factors, similar to how vector-borne transmission is formulated by using entomologic factors. Although we focus on air and fomite transmission, a potential generalization of this framework would be incorporating pathogen dynamics within different environmental settings, such as other vectors, food, and water.

The adoption of an EITS framework provides a theoretical basis for understanding and modeling intervention efficacy in realistically detailed situations involving diverse venues where transmission takes place. The parameters of the EITS model reflect physical events on which data can be readily gathered by using newly developed methodologies and for which a considerable body of data and theory already exists. In contrast, contact rates and transmission probabilities in specific venues are abstract and not feasibly measurable in most situations, except in uniform places with repeated and prolonged contact, such as households.

The EITS framework also helps to identify and relax unrealistic mass action assumptions, such as no time passing between pathogens leaving one person and reaching another. It also provides a way to conceptualize the extent to which transmission is a density- or frequency-dependent contact process (1, 2). In reality, most transmissions occur between these 2 extremes, and the EITS formulation reflects this. As the environmental persistence ratio, \( pN/\mu \), increases, transmission becomes more density dependent and, as this ratio decreases, it becomes more frequency dependent.

Another theoretically valuable focus is found in the contamination ratio, \( u/\gamma \), a measure of the total amount of pathogens shed by infectious individuals. This ratio and the magnitude of a contamination event are both indicators of the probability that an outbreak will occur, suggesting that stochastic “die out” is more likely when environmental contamination is low.

EITS models will eventually help us to define the role of different transmission modes in sustaining or amplifying transmission. Multiple transmission modes have been recognized for many other infections than those in our example, such as cholera, hepatitis A, and cryptosporidiosis (43–45). The EITS model framework provides leverage for using detailed environmental data and well-established parameters reflecting pathogen characteristics to analyze different transmission modes and the role they play in endemic and epidemic situations.

Although including more realistic details than classic SIR models, the EITS models presented here are abstract in order to serve heuristic purposes. Future EITS models will relax current model assumptions. The specific choices of which assumptions to relax will depend on the research question. For example, with only a single environmental compartment, our models may not accurately capture contact patterns or pathogen dynamics in the environment. In fact, our preliminary analysis pointed out that a key way to relax the homogeneous environment assumption is to distinguish frequently touched fomites from infrequently touched fomites. Additionally, a more refined understanding of transmission patterns might require model structure that accounts for detailed contact patterns between people and the environment.

Our simple EITS models identify key elements and important data gaps of the environmental infection transmission system. They provide an initial step in motivating improved environmental measurements that would complement human case data and might be more informative and more cost-effective to gather than such data. EITS models incorporating more realistic details than those presented here can be used to help design future environmental data collection efforts. They also can provide a basis for analyzing focused environmentally based interventions, such as decontamination of specific surfaces, water, or air, as well as hygiene and sanitation efforts.

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