Herd Protection from Drinking Water, Sanitation, and Hygiene Interventions

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Abstract. Herd immunity arises when a communicable disease is less able to propagate because a substantial portion of the population is immune. Nonimmunizing interventions, such as insecticide-treated bednets and deworming drugs, have shown similar herd-protective effects. Less is known about the herd protection from drinking water, sanitation, and hand hygiene (WASH) interventions. We first constructed a transmission model to illustrate mechanisms through which different WASH interventions may provide herd protection. We then conducted an extensive review of the literature to assess the validity of the model results and identify current gaps in research. The model suggests that herd protection accounts for a substantial portion of the total protection provided by WASH interventions. However, both the literature and the model suggest that sanitation interventions in particular are the most likely to provide herd protection, since they reduce environmental contamination. Many studies fail to account for these indirect effects and thus underestimate the total impact an intervention may have. Although cluster-randomized trials of WASH interventions have reported the total or overall efficacy of WASH interventions, they have not quantified the role of herd protection. Just as it does in immunization policy, understanding the role of herd protection from WASH interventions can help inform coverage targets and strategies that indirectly protect those that are unable to be reached by WASH campaigns. Toward this end, studies are needed to confirm the differential role that herd protection plays across the WASH interventions suggested by our transmission model.

INTRODUCTION

Enteric pathogens are a major source of disease burden worldwide leading to diarrhea, subclinical environmental enteropathy, malnutrition, and death.1 These pathogens are largely transmitted via the fecal-oral route with a variety of environmental intermediaries, such as drinking water, soil, food, fomites, and hands. Drinking water, sanitation, and hand hygiene (WASH) interventions have been shown to be protective against enteric infections,1 but little attention is given to the herd-protective effects that they may provide. Herd protection occurs when an infectious disease intervention provides indirect protection to nonrecipients. Studies that fail to account for herd protection will lead to an underestimate of the total protective effectiveness of the intervention.

Halloran and Struchiner2 provided a useful framework for conducting studies to measure these indirect effects (Figure 1). These study designs require different populations with varying levels of intervention coverage. The direct effect is the protective efficacy of the intervention, and is measured by comparing intervention groups within population A, as is done in a simple randomized controlled trial where individuals are the unit of randomization. The direct effect can be estimated at each level of intervention coverage by the equation $1 - D_1/D_0$, where $D_1$ represents the disease risk in the intervention group and $D_0$ represents the disease risk in the nonintervention or control group in the same population. The indirect effect represents the herd protection provided by the intervention and is measured through cross-population studies. It is estimated at each level of coverage by the equation $1 - D_0/D_p$, where $D_0$ represents the disease risk in a population where the intervention is entirely absent (coverage = 0%). The total effect is the combination of the direct and indirect effects. The overall effect is the risk in population A (the weighted average of $D_1$ and $D_0$) compared with the risk in population B ($D_p$). The majority of cluster-randomized trials measure either the overall effect or the total effect but without disaggregating the direct and indirect effects.3

The framework of Halloran and Struchiner2 has been used to show evidence of herd protection for a variety of infectious disease interventions, such as vaccines4–5 and insecticide-treated bednets to prevent malaria,3–8 mass deworming drugs to treat helminth infection,9,10 highly active retroviral therapy to treat human immunodeficiency virus (HIV)/acquired immunodeficiency syndrome (AIDS)11,12 and antibiotics to treat trachoma.13,14 A few studies15–18 have shown that WASH technologies may have community-wide benefits, the practices of one household having a beneficial effect on neighbors. Use of this framework, however, assumes comparability (no confounding) between populations with different levels of intervention, which can be difficult to achieve or prove. Although a few of these studies were rigorously done, many others have serious methodological limitations, such as failure to adjust for this group-level confounding or failure to account for the individual level of the exposure in question.

To encourage more empirical research on the indirect effects of WASH interventions, we present a stochastic mathematical model of enteric pathogen transmission in a community. This model serves to illustrate the different mechanisms through which WASH interventions can provide community-wide protective effects, that is, herd protection. We then compare the findings of our model to the existing literature to highlight current gaps in empirical research.

METHODS

Model structure. We simulated a community of 500 individuals, nested within 100 households. Individuals are modeled as discrete entities using a stochastic framework. The quantity of pathogens in the environment is modeled as continuous using ordinary differential equations.19 Individuals are categorized as susceptible, infectious, or immune, and immunity is assumed to be permanent (SIR model). Many enteric pathogens do not provide permanent immunity, but during short-term outbreaks, such as those simulated in this model, the
difference between permanent and temporary immunity is insignificant. All transmission of pathogens occurs via the environment. Infectious individuals can transmit pathogens to susceptible individuals by either of two pathways (Figure 2). First, the infectious individual sheds pathogens into their household environment at rate $\delta$. Susceptible individuals pick up pathogens from their household environment at rate $\rho$. This household environment represents household surfaces, stored drinking water, or any other pathogen-harboring area located within the household. Second, infectious individuals shed pathogens in the community environment at rate $\phi$. All susceptible individuals in the community pick up pathogens from the community environment at rate $\alpha$. The community environment can represent an unprotected source of drinking water such as a pond, common or shared areas such as schools, or any other pathogen-harboring area accessed by people from multiple households. Pathogen survival in both the household and community environment is determined by the parameter $\mu$. The model was coded in R version 3.0.2. For details, see the Supplemental Appendix A.

**Simulation analysis.** Each simulation begins with a population that is entirely susceptible, except for one infectious individual; this is representative of a new pathogen strain being introduced into a community with no prior protection.
We then simulate an epidemic. The primary outcome of interest is the cumulative incidence, defined as the proportion infected or immune at the end of the epidemic.

To estimate the effect of intervention coverage, we simulate these epidemics at varying levels of intervention coverage in the community. In the first scenario, no households in the community are using the intervention, that is, everyone is in the control group. In subsequent scenarios, we increase the percentage of households using the intervention by increments of 10%, until coverage reaches 100%. At each level of coverage, the model is simulated 100 times, for a total of 1,100 simulations. The median cumulative incidence at each level of coverage is used to calculate the protective efficacy of the intervention.

To estimate the amount of herd protection, the direct, indirect, total, and overall effects of the intervention are measured using the framework presented in Figure 1.\(^1\) The direct effect is the protective efficacy of the intervention that a simple randomized controlled trial would measure. It is estimated at each level of intervention coverage by the equation \(1 - D_1/D_0\), where \(D_1\) represents the disease risk in the intervention group at a given level of coverage and \(D_0\) represents the disease risk in the control group (those not receiving the intervention) at that same level of coverage. The indirect effect represents the herd protection provided by the intervention. It is estimated at each level of coverage by the equation \(1 - D_0/D_0^*\), where \(D_0^*\) represents the disease risk in a population where the intervention is entirely absent (coverage = 0%). The total effect is the combination of the direct and indirect effects, and is estimated at each level of coverage by the equation \(1 - (w_0D_0 + w_1D_1)/D_0^*\), where \(w_0\) and \(w_1\) are weights equal to the proportion of individuals in population A that are in the control and intervention groups, respectively.

**WASH interventions.** We separately model a drinking water intervention, a sanitation intervention, and a hand hygiene intervention. Each of these interventions is applied at the household level with no within-household heterogeneity and compliance is assumed to be 100%. We conceptualize these interventions in our model as resulting in a reduction of the rate of pathogen shedding into the environment and/or a reduction of the rate of pathogen pickup from the environment. Specifically, the drinking water intervention reduces the value of \(\alpha\), the rate of pathogen pickup from the community environment. The sanitation intervention reduces the value of \(\delta\), the rate of shedding pathogens into community environment. Finally, the hand hygiene intervention reduces the value of \(\phi\) and \(\rho\), the rate of shedding into the household environment and the rate of pathogen pickup from the household environment, respectively. These interventions are shown in Figure 2 as grey boxes, suggesting that they partially block these pathways.

Parameter values for each of these interventions along with the baseline practices of the control group are shown in Table 1. The goal of this model is to provide a conceptual framework for herd protection and not to estimate the magnitude of direct or indirect effects. Therefore, parameter values were chosen to generate a baseline risk of approximately 70% and a total effect \((1 - D_1/D_0^*\) at 100% coverage) of approximately 35% based on estimates from systematic reviews.\(^22\)–\(^32\)

**Literature review.** To identify studies that investigated herd protection from WASH interventions, we searched PubMed and Google Scholar for articles using the following search criteria: “herd protection,” “herd effect,” “indirect effect,” “mass effect,” “community effect,” “externalities,” or “neighborhood,” as well as key words for each intervention: “sanitation,” “toilet,” or “latrine,” “water” and “hygiene,” or “handwashing.” After selecting relevant articles from these search results, we also considered articles in their citation network (those articles cited by or citing the original article). In

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### Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n)</td>
<td>The number of households in the community</td>
<td>100</td>
</tr>
<tr>
<td>(\mu)</td>
<td>The number of individuals per household</td>
<td>5*</td>
</tr>
<tr>
<td>(\gamma)</td>
<td>The rate at which pathogens die in the environment</td>
<td>1/10</td>
</tr>
<tr>
<td><strong>Control group parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\alpha)</td>
<td>The rate at which susceptible individuals in the control group pick up pathogens from the community environment</td>
<td>(1 \times 10^{-6})</td>
</tr>
<tr>
<td>(\phi)</td>
<td>The rate at which infectious individuals in the control group shed pathogens into the community environment</td>
<td>78</td>
</tr>
<tr>
<td>(\delta)</td>
<td>The rate at which infectious individuals in the control group shed pathogens into their own household environment</td>
<td>395</td>
</tr>
<tr>
<td>(\rho)</td>
<td>The rate at which susceptible individuals in the control group pick up pathogens from their household environment</td>
<td>(1 \times 10^{-5})</td>
</tr>
<tr>
<td><strong>Intervention group parameters</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\alpha_1)</td>
<td>The rate at which susceptible individuals using a drinking water intervention pick up pathogens from the community environment</td>
<td>(0.73 \times 10^{-6})</td>
</tr>
<tr>
<td>(\phi_1)</td>
<td>The rate at which infectious individuals using a sanitation intervention shed pathogens into the community environment</td>
<td>56.94</td>
</tr>
<tr>
<td>(\delta_1)</td>
<td>The rate at which infectious individuals using a hygiene intervention shed pathogens into their own household environment</td>
<td>173.8</td>
</tr>
<tr>
<td>(\rho_1)</td>
<td>The rate at which susceptible individuals using a hygiene intervention pick up pathogens from their household environment</td>
<td>(0.44 \times 10^{-5})</td>
</tr>
</tbody>
</table>

*All rates are per day.

* Normally distributed, with a mean of 5 and a standard deviation of 2.
RESULTS

Drinking water. When the entire community is among the control group, the median risk of infection in the community across 100 simulations is 69.6%. As coverage of the drinking water intervention increases, the risk in both the intervention group and the control group decreases (Figure 3, Panel A). When coverage reaches 100%, the median risk in the community is 44.5%, providing a total protective efficacy of 36.1% \((0.361 = 1 - 0.445/0.696)\). At all levels of coverage, the risk in the intervention group is lower than that of the control group (Figure 3, Panel A). The direct protective efficacy of the drinking water intervention is about 15%, and is relatively constant across all levels of coverage (Figure 3, Panel B). As coverage increases, however, the indirect effect becomes more pronounced and approaches 20% at high levels of coverage.

Several studies have attempted to show evidence of herd protection from drinking water (Table 2 and Suppl. Table 1). All of these studies used cross-sectional national household surveys such as the Demographic and Health Surveys (dhsprogram.org) and typically used the percentage of households in a survey cluster with an improved source of drinking water as an independent variable. Six studies from three publications17,33,34 assessed the effect of community-level drinking water on child or infant mortality by re-constructing cohorts of children based on 5- or 10-year birth histories, two studies35,36 assessed child malnutrition (height for age), and one study assessed low birth weight.37 Most showed a protective effect, though the results were not always consistent.

These studies had important methodological limitations. First, many did not adjust for community-level confounding variables. Just as socioeconomic status is an important confounder at the household level,38 communities with differences in WASH coverage are likely different in many other important ways. Failure to account for potential confounding variables at the community level makes causal inference dubious. Second, many did not account for WASH access at the household level. Failure to account for household-level drinking water makes the assessment of herd protection difficult. Household-level access may be confounding the protective effect of community-level access, so the protective effect at the community level is actually a mixture of the direct and indirect effects, with no ability to disentangle to two. A strength of our simulation approach is that it eliminates any potential confounding by providing perfect experimental conditions. Finally, some studies were often not focused on estimating the unbiased protective effect of drinking water, but simply attempted to show that community-level variables in general have explanatory power, with little discussion or understanding of the pathogen transmission process. Only one study37 did not include any of these limitations, and it showed no protective effect of community-level coverage of improved drinking water.

Sanitation. As coverage of the sanitation intervention increases, the risk of infection in both the intervention group and the control group decreases (Figure 4, Panel A). When coverage of the sanitation intervention reaches 100%, the median risk is 44.7 per 100 persons, providing a total protective efficacy of 35.8% \((0.358 = 1 - 0.447/0.696)\). This total effect is entirely attributable to the indirect effect, as the direct effect is negligible (Figure 4, Panel B). In other words, sanitation provides no direct benefit to the user, but protects the entire community equally.

Several studies have suggested that sanitation provides herd protection to neighboring households (Table 3 and Suppl. Table 2). The majority of these were also from cross-sectional national household surveys. Outcomes in these studies varied from diarrhea,15,39-41 prevalence of parasitic infection,42 infant or child mortality,17,34,43 child stunting,16,35,44 and child wasting.45 The majority of these studies had the same limitations mentioned for the drinking water studies, namely insufficient adjustment for community-level confounders, no adjustment for household-level sanitation, and no discussion of interrupting pathogen transmission. There were, however, a few exceptions.15-17,42 Barreto and others15 conducted two cohort studies in Salvador, Brazil, one before a city-wide sanitation campaign and one after. The study attributed the entirety of the 21% reduction in diarrhea prevalence to the increase in sewer-connected toilet coverage in the neighborhood (26–80%). Increases in household-level

![Figure 3](image-url)  
**Figure 3.** Model simulations showing the effect of a drinking water intervention across different levels of intervention coverage in the community. At each level of coverage of the intervention, the stochastic model was simulated 100 times. Panels A and B show the median values for the cumulative incidence (proportion infected) and protective efficacy, respectively, where \(D_1\) represents the disease risk in the intervention group, \(D_0\) represents the disease risk in the control group, and \(D_{1*}\) represents the disease risk in a separate population where there is no intervention (see Figure 1 and text for more details).
toileting did not explain the reduction. Our model results confirm these findings. Within the same study setting, Barreto and others also assessed the prevalence of various intestinal parasites before and after the city-wide sanitation campaign. For the three parasites studied, the prevalence of each dropped substantially, and 25–40% of the reduced prevalence was attributable to the increase in sewer-connected toilet coverage in the neighborhood. Fuller and others used a cohort study in rural Ecuador to show that the total effect of sanitation was a 72% lower prevalence of stunting, and 94% of this total effect was attributable to the indirect effect of sanitation coverage. Finally, Van de Poel and others used data from six Demographic and Health Surveys in sub-Saharan Africa, and found that sanitation coverage at the community level was surprisingly a risk factor for infant mortality in urban areas; sanitation coverage in rural areas was not associated with infant mortality.

**Hand hygiene.** As coverage of the hand hygiene intervention increases, the risk in both the intervention group and the control group decreases (Figure 5, Panel A). When coverage reaches 100%, the median risk in the community is 43.1%, providing a total protective efficacy of 38.1% (0.381 = 1 – 

**Table 2**

<table>
<thead>
<tr>
<th>Setting</th>
<th>Specific outcome</th>
<th>Conceptualization of drinking water coverage</th>
<th>Effect on outcome</th>
<th>Limitations*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Sub-Saharan African countries: urban</td>
<td>Infant mortality</td>
<td>Proportion of households in survey cluster with water from a tap, protected well, bottle, or vendor</td>
<td>Probit regression coefficient = 0.064 (P &gt; 0.05)</td>
<td>–</td>
</tr>
<tr>
<td>6 Sub-Saharan African countries: rural</td>
<td>Infant mortality</td>
<td>Proportion of households in survey cluster with water from a tap, protected well, bottle, or vendor</td>
<td>Probit regression coefficient = 0.007 (P &gt; 0.05)</td>
<td>–</td>
</tr>
<tr>
<td>Nigeria: national</td>
<td>Infant mortality</td>
<td>% of households in survey cluster with piped water (low, medium, high)</td>
<td>Medium vs. low, HR = 0.67 (P &lt; 0.05); high vs. low, HR = 0.87 (P &lt; 0.05)</td>
<td>b, c</td>
</tr>
<tr>
<td>Nigeria: national</td>
<td>Child mortality</td>
<td>% of households in survey cluster with piped water (low, medium, high)</td>
<td>Medium vs. low, HR = 1.01 (P &gt; 0.05); high vs. low, HR = 1.42 (P &lt; 0.05)</td>
<td>b, c</td>
</tr>
<tr>
<td>Brazil: northeast</td>
<td>Child mortality</td>
<td>% of households in municipality with regular network water or % with well water</td>
<td>Network water: HR = 0.30 (P &gt; 0.05); well water: HR = 0.34 (P &lt; 0.05)</td>
<td>b</td>
</tr>
<tr>
<td>Brazil: south and southeast</td>
<td>Child mortality</td>
<td>% of households in municipality with regular network water</td>
<td>Network water: HR = 30.9 (P &gt; 0.05); well water: HR = 4.54 (P &lt; 0.05)</td>
<td>b</td>
</tr>
<tr>
<td><strong>Nutrition</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Nigeria: national</td>
<td>Stunting</td>
<td>Safe water, community level (no vs. yes, undefined)</td>
<td>OR = 1.08 (95% CL = 0.74–1.15)</td>
<td>b, c</td>
</tr>
<tr>
<td>Malawi: national</td>
<td>Stunting</td>
<td>% of households in survey cluster with protected water source (&gt; 73% vs. ≤ 73%)</td>
<td>OR = 0.83 (95% CL = 0.55–1.01)</td>
<td>a, c</td>
</tr>
<tr>
<td>Ghana: national</td>
<td>Low birth weight</td>
<td>% of households in survey cluster with access to safe water (high vs. low, undefined)</td>
<td>OR = 0.74 (95% CL = 0.57–0.96)</td>
<td>b, c</td>
</tr>
</tbody>
</table>

HR = hazard ratio; OR = odds ratio; WASH = drinking water, sanitation, and hand hygiene. No studies could be related to the Halloran framework. Studies are grouped by outcome. Additional study details can be found in the Supplemental Appendix B.

*Potential limitations include: a—did not adjust or insufficiently adjusted for community-level socioeconomic status, b—did not include the corresponding WASH exposure at the household or individual level, c—discussion of social context but not of disease transmission process.
<table>
<thead>
<tr>
<th>Setting</th>
<th>Specific outcome</th>
<th>Conceptualization of sanitation coverage</th>
<th>Effect on outcome</th>
<th>Overall effect (protective efficacy)</th>
<th>Total effect (protective efficacy)</th>
<th>Indirect effect (protective efficacy)</th>
<th>% of overall/total effect due to the indirect effect</th>
<th>Limitations*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diarrhea</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Brazil: urban15</td>
<td>Diarrhea prevalence</td>
<td>Primary exposure is the intervention (after vs. before). Potential mediator is proportion of households in area with sewer connection</td>
<td>PR (after vs. before intervention) = 0.78; 100% mediated by area sewer coverage</td>
<td>22%</td>
<td>–</td>
<td>22%</td>
<td>100%</td>
<td>–</td>
</tr>
<tr>
<td>India: rural39</td>
<td>Diarrhea prevalence</td>
<td>Proportion of households in a village with access to improved sanitation with no sanitation, whether the nearest neighbor had improved latrine</td>
<td>Estimated with quadratic</td>
<td>–</td>
<td>47%</td>
<td>35%</td>
<td>75%</td>
<td>a</td>
</tr>
<tr>
<td>Zimbabwe: rural40</td>
<td>No. of episodes of diarrhea in 45 weeks</td>
<td>Among children with no sanitation, whether the nearest neighbor had improved latrine</td>
<td>Mean difference: 1.13 fewer episodes when nearest neighbor has improved latrine</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>a</td>
</tr>
<tr>
<td>Brazil: urban41</td>
<td>Duration (days) of diarrheal episodes</td>
<td>% of households with sewer connection: very low (&lt; 34.1%), low (34.147.1%), normal (47.2–55.0%), good (&gt; 55.0%)</td>
<td>Mean difference (days): normal vs. good 0.07, low vs. good 0.23, very low vs. good 0.47</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>a, b</td>
</tr>
<tr>
<td><strong>Parasitic infection</strong></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Brazil: urban42</td>
<td>Prevalence of <em>Ascaris lumbricoides</em></td>
<td>Primary exposure is the intervention (after vs. before). Potential mediator is proportion of households in area with sewer connection</td>
<td>PR (after vs. before) = 0.57; 40% mediated by area sewer coverage</td>
<td>43%</td>
<td>–</td>
<td>17%</td>
<td>40%</td>
<td>–</td>
</tr>
<tr>
<td>Brazil: urban42</td>
<td>Prevalence of <em>Trichuris trichiura</em></td>
<td>Primary exposure is the intervention (after vs. before). Potential mediator is proportion of households in area with sewer connection</td>
<td>PR (after vs. before) = 0.38; 30% mediated by area sewer coverage</td>
<td>62%</td>
<td>–</td>
<td>19%</td>
<td>30%</td>
<td>–</td>
</tr>
<tr>
<td>Brazil: urban42</td>
<td>Prevalence of <em>Giardia duodenalis</em></td>
<td>Primary exposure is the intervention (after vs. before). Potential mediator is proportion of households in area with sewer connection</td>
<td>PR (after vs. before) = 0.41; 25% mediated by area sewer coverage</td>
<td>59%</td>
<td>–</td>
<td>15%</td>
<td>25%</td>
<td>–</td>
</tr>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Sub-Saharan African countries: urban12</td>
<td>Infant mortality</td>
<td>Proportion of households in survey cluster with any toilet facility</td>
<td>Probit regression coefficient: 0.314 ($P &lt; 0.01$)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>6 Sub-Saharan African countries: rural17</td>
<td>Infant mortality</td>
<td>Proportion of households in survey cluster with any toilet facility</td>
<td>Probit regression coefficient: 0.008 ($P &gt; 0.05$)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>India: national43</td>
<td>Infant mortality</td>
<td>Proportion of households in survey cluster NOT practicing open defecation</td>
<td>27.1 fewer deaths per 1,000 children</td>
<td>–</td>
<td>35†§</td>
<td>27†§</td>
<td>77%</td>
<td>a</td>
</tr>
<tr>
<td>Brazil: northeast44</td>
<td>Child mortality</td>
<td>% of households in municipality with sewage connection or % with any sanitation</td>
<td>Sewage connection: HR = 0.08 ($P &lt; 0.05$); any sanitation: HR = 0.29 ($P &lt; 0.05$)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>b</td>
</tr>
<tr>
<td>Brazil: south and southeast44</td>
<td>Child mortality</td>
<td>% of households in municipality with sewage connection or % with any sanitation</td>
<td>Sewage connection: HR = 0.67 ($P &gt; 0.05$); any sanitation: HR = 0.33 ($P &gt; 0.05$)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>b</td>
</tr>
</tbody>
</table>
At all levels of coverage, the risk in the intervention group is lower than that of the control group (Figure 5, Panel A). The direct protective efficacy of hand hygiene is about 15%, and is relatively constant across all levels of coverage (Figure 5, Panel B). As coverage increases, however, the indirect effect becomes more pronounced, becoming roughly equal to the direct effect at 70% coverage.

We found no studies showing that hand hygiene in one household provides a benefit to neighboring households. One study showed that when the food preparer practices handwashing, then children in the household are less likely to have diarrhea. This highlights that the indirect effects from WASH interventions may occur not only between households as shown by our model and literature review but also within households. It also raises an important point of within-household heterogeneity of WASH practices. Although these interventions are often applied at the household level, household members may comply to differing degrees, particularly young children that often use diapers instead of defeating directly into a pit latrine.

**DISCUSSION**

WASH interventions can provide both direct protection to the user and indirect (herd) protection to the entire community. Household-level drinking water interventions aim to reduce the number of pathogens that the user ingests, providing a clear direct benefit to that user. Individuals from neighboring households, however, may receive an indirect benefit because there are fewer total infections in the community, which will result in less cumulative environmental contamination, though the rates of shedding are unchanged. This type of indirect effect should arise in any communicable disease system whenever an intervention reduces the prevalence of infection, as susceptible persons in the community will be less likely to have contact with an infectious individual, whether that contact is direct or environmentally mediated.

Our model simulations and literature review suggest that the indirect effects from a sanitation intervention are a larger component of the total effect compared with those from a drinking water or hand hygiene intervention. If sanitation can reduce the rate of shedding pathogens into the community environment, all surrounding households will benefit regardless of their own sanitation practices. In our model, sanitation provides no direct benefit to the household. This is in some ways similar to the transmission-blocking malaria vaccines currently in development, wherein the recipient of the vaccine would not be immune to infection or symptoms, but the parasite would be unable to be transmitted to a susceptible mosquito. The only benefit to the vaccinated...
person is the indirect effect shared by everyone in the community. Sanitation also has the potential to provide a direct benefit to the household if the intervention can reduce contamination of the household environment.

Hand hygiene is unique in that it can both reduce the contagiousness of an infected individual and protect a susceptible individual. The direct effect occurs because the person washing their hands is reducing the number of pathogens that they will ingest. The indirect effect occurs for two reasons. The first is similar to the mechanism of the household drinking water intervention in that by preventing new infections, it reduces the overall number of pathogens excreted into the environment. The second is like that of sanitation, namely a reduced shedding rate into the environment.

There are many studies reporting the results of cluster-randomized trials of drinking water,39,50 sanitation,51,52,55 and hand hygiene interventions,46,53,54 which we have not included in our review of the literature. There are several motivations for the use of this study design, one of which is the potential to account for indirect effects or herd protection.3 Published articles, however, have reported the results in terms of the total effect or the overall effect, without disaggregating the direct and indirect effects. Although these studies do not underestimate the total impact of an intervention, they do not provide a complete picture of the type of impact WASH may have. Specifically, understanding the indirect effects can be essential for disease control strategies, especially if the indirect effect is nonlinear with increases in coverage, as is the case with a herd immunity threshold. Also, identifying herd protection and not just a total effect suggests that these interventions may protect households unreached by intervention campaigns. It is possible that these cluster-randomized trials could be reanalyzed to assess herd protection from various WASH interventions,4 though the benefits of randomization would likely be lost.

As in all modeling exercises, our findings could be sensitive to a relaxation of our simplifying assumptions. First, WASH-related practices vary substantially across communities, between individuals and even within individuals. Our model assumes 100% compliance with the intervention, though recent studies have shown that efficacious WASH interventions yield no benefit if high levels of coverage are not accompanied by high levels of compliance.51,52,55 Second, many enteric pathogens are transmitted person to person, but our model does not explicitly capture this pathway. Although we did not model transmission as a traditional mass action, environmentally mediated transmission partially captures this phenomenon, as some person-to-person transmission may actually occur via fomites and other objects. Third, due to very few quality studies on the subject, our analysis did not seek to estimate the actual amount of herd protection from a given intervention. Also, this will vary substantially across different settings. For example, open defecation in a rural setting may occur in the bush, far from human dwellings. Such a practice may create little to no risk for other individuals in the village.56 The degree of environmental connectivity57 between households will vary by setting and pathogen. The magnitude of the indirect effect largely depends on the relative proportion of transmission that occurs between households compared with transmission within households. Drinking water interventions, for example, will provide a much smaller indirect effect if contamination from one household is less able to reach a shared environment. The degree of herd protection will also depend on the type of pathogens circulating in the community, as different pathogens are able to exploit different pathways to varying degrees. Cholera, for example, has a high infectious dose and thrives in surface water enhancing its ability to survive in water-based community environment. Shigella is less able to survive in the environment and has a much lower infectious dose, allowing it to be readily transmitted via food and hands and possibly increasing within-household transmission. Also, we have oversimplified what are often complex interventions. For example, hand hygiene (handwashing) interventions are only a subset of what could be considered hygiene. The same could be said for drinking water and sanitation interventions, which often focus on infrastructure, but could also include behavior change. This simplification, however, serves to highlight the important mechanisms through which each class of intervention may provide protection to nonrecipients. Finally, we have simulated short-term epidemics of enteric pathogens. In reality, these pathogens are often endemic or become endemic after an outbreak.

Our model was constructed with the intent of illustrating the mechanisms through which WASH interventions may
provide indirect protection. To date, data illustrating herd protection for water sanitation and hygiene are still relatively limited. As more high-quality empirical studies are produced, it will be possible to obtain more robust estimates of model’s parameters and outcomes, and thereby address more detailed questions pertaining to the mechanisms driving herd protection. Data may also provide insight into how generalizable herd protection is across different environmental and social conditions as well as to different pathogens.

Our study has important implications for both future research and program delivery. Assessment of WASH interventions has largely ignored community interdependence.\textsuperscript{58} Studies assessing the health benefits of WASH interventions without accounting for herd protection will likely underestimate the total efficacy, particularly for sanitation interventions whose benefits may be largely due to their indirect effects. Future studies should seek to quantify these additional benefits, which would alter the cost-effectiveness calculations of decision makers. Also, WASH programs often aim to achieve 100% coverage in communities, but 100% coverage, not to mention 100% compliance, is elusive.\textsuperscript{58} Immunization policy is often based on reaching a threshold of vaccination coverage, often less than 100%, at which transmission will be interrupted and the disease eliminated. It is unclear, however, whether such a threshold exists for WASH interventions. Interventions for the control of infectious diseases can provide indirect protection to nonusers. Although the mechanism behind herd protection varies by pathogen and transmission cycle, the goal of providing sufficient coverage to interrupt transmission transcends these differences.

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