

Emerging Issues in Sanitation: Herd Protection, Sharing Between Households, and Joint  
Effects

by

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Dedicated to my daughter June Deann, for teaching me the value of a single child's life while  
I study the lives of millions

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## **Abstract**

Sanitation (toilets and latrines) is considered one of the most important health advancements of modern times, yet over one third of the world still lacks basic access to sanitation services. In the coming year, the international community will likely adopt a global goal of reaching universal access by the year 2030. While much is known about the health benefits of improved sanitation, this dissertation seeks to address three emerging issues related to sanitation coverage: herd protection, sharing between households, and the joint effects of drinking water and sanitation. In chapter two, we explore the concept of herd protection, which occurs when an infectious disease intervention indirectly benefits those that do not receive it. We review the literature and highlight herd protective effects from interventions such as vaccines, insecticide treated bednets, and deworming drugs. We then use a mathematical model to highlight the mechanisms through which improving sanitation in some households can provide herd protection to the entire community. In chapter three, we build off of the conceptual work of chapter two by assessing herd protection from sanitation in 24 rural villages in northern coastal Ecuador. We find that children from neighborhoods with higher sanitation coverage were taller than children from areas with lower levels of coverage. In chapter four, we address the topic of sanitation facilities that are shared by multiple households, which is an increasingly common practice in urban slums and rural communities. Using data from, 51 Demographic and Health



Surveys, we show that using such a facility is associated with an modest increase in diarrhea prevalence, but the effect varied across countries. In chapter five, we investigate the independent and joint effects of drinking water and sanitation. Using data from 217 Demographic and Health Surveys, we find that these services are largely independent, suggesting that they should be combined to maximize the benefit. We observe that the effect of water and sanitation varies across countries, and the effect of sanitation has diminished over time. This dissertation uses a variety of methods to highlight the importance of access to adequate sanitation at the household and at the community level.

## **Chapter I**

### Introduction

#### **Sanitation and Health**

In 2007, the British Medical Journal commissioned a series of papers, each arguing the merits of given medical advancements. These papers covered topics such as the discovery of antibiotics, the development of X-ray imaging, and vaccines, and readers were asked to vote on which they considered to be most important medical milestone since 1840. The winner of that poll was the ‘sanitary revolution’ (1). Improvements in sanitation and drinking water have long been considered a primary cause for the declines in mortality seen in the industrialized world during the 19<sup>th</sup> and 20<sup>th</sup> centuries (2). In England, for example, mortality from diarrhea declined rapidly after 1911, coinciding with the introduction of widespread water chlorination (3, 4).

There are many ways through which sanitation may have an impact on human health, most importantly of which is the interruption of transmission of fecal-oral pathogens. The well-known ‘F’ diagram (5) highlights several of the fecal-oral pathways which different pathogens may exploit in different contexts (Figure 1.1). Food, fingers, fields, fluids (drinking water), and flies may all serve as environmental intermediaries between contaminated feces and a susceptible host. Improved sanitation captures and contains human excrement, preventing fecal contamination from reaching the

environment. It is expected, therefore, that all environmental intermediaries will have less contamination in the presence of good sanitation. Treating drinking water, on the other hand, has a more specific effect. It prevents contaminated water from reaching a host, either directly or via food.

The most studied outcome of ingesting enteric pathogens is diarrhea, defined by the World Health Organization as 3 or more loose or watery stools in a 24-hour period (6). Historically, diarrhea was the leading cause of death among children under 5 years of age worldwide, causing an estimated 4.6 million deaths per year between 1950 and 1980 (7). Due to improved case management and the development of oral rehydration therapy, diarrhea mortality has dropped substantially over the past 40 years (8). A 1992 review estimated that diarrhea mortality among children during the 1980s was 3.3 million deaths per year (9). Through the 1990s and the early 21<sup>st</sup> century, mortality has continued to decline, and in 2011, there were approximately 700,000 diarrheal disease deaths (10). Despite the success of oral rehydration therapy, diarrhea remains one of the leading causes of childhood death worldwide (11), and rates of illness have not declined (9, 10, 12). A reduction in diarrhea incidence will require scaling up of high quality interventions, including drinking water, sanitation, hygiene, and immunization. Inadequate sanitation is responsible for an estimated 20% of the diarrheal disease burden worldwide (13).

Diarrhea occurs when the absorptive or secretory functions of the small intestine are interrupted. These interruptions may be an increase in secretion of fluids into the gut, a reduction in fluid absorption, or inflammation of the mucosal lining. There are a variety of causes, such as toxins and chronic diseases, but the most common is infection, though some bacteria produce diarrheagenic toxins. The main health risk of acute diarrhea is

dehydration, which can usually be remedied with oral rehydration therapy (14). Persistent diarrhea, which lasts for 14 days or longer, is more difficult to treat, and though it accounts for only a fraction of all cases of diarrhea, it is responsible for one half of diarrheal deaths (15). Persistent diarrhea also leads to long-term disruption of intestinal absorption resulting in short-term or long-term malnutrition (16).

Another important outcome of ingesting fecal pathogens is environmental enteropathy, sometimes called tropical enteropathy. It is characterized by a blunting of intestinal villi and increased permeability of the small intestine (17). While persistent diarrhea plays an important role in intestinal absorptive capacity, environmental enteropathy is attributed to chronic exposure to enteric pathogens, and persons with environmental enteropathy are often asymptomatic. A more severe and symptomatic condition, tropical sprue, has been documented before and throughout the 20<sup>th</sup> century. It was not until the 1960s that medical advances allowed for a more thorough investigation of the small intestine, leading to the discovery of a milder and asymptomatic form (18). Shortly thereafter the condition was documented in American soldiers and Peace Corp volunteers who had close contact with local populations in tropical countries (19, 20), as well as persons in South Asia (18), and to a lesser extent Africa (21).

Though environmental enteropathy is asymptomatic, it is not without consequence. The limited efficacy of oral vaccines (oral poliovirus vaccine, rotavirus vaccine, live attenuated cholera vaccine, and a *Shigella* vaccine) in developing countries is largely attributed to decreased gut function found in those with environmental enteropathy (22). Recently, this condition has also gained attention as a potential source for growth faltering in children (17, 23-25). This occurs as a result of both the decreased absorptive capacity of

the gut as well as the increased energy expenditure of the gut's chronic immune stimulation. Childhood stunting (low height-for-age) affected 26% of children under 5 worldwide in 2011 and played a contributing role in over 1 million deaths (26). Stunting is also a risk factor for poor outcomes later in life, including behavioral problems, underachievement in school, and chronic diseases such as diabetes (27-30). While child growth is obviously influenced by fetal exposure (30), food security (31), and diet (32), inadequate access to water, sanitation, and hygiene is also recognized to play an important role (33, 34). In the next section, I explore several important enteric pathogens, and the relationship between their biology and epidemiology.

## **Pathogens**

A variety of pathogens can infect the gut and cause diarrhea. While this dissertation does not seek to identify specific pathogens responsible for infection, it is nevertheless important to recognize how different enteric pathogens are transmitted, and how these transmission characteristics may determine the efficacy of a sanitation intervention. The GEMS study, a case-control study, was recently carried out in 7 countries (The Gambia, Mali, Mozambique, Kenya, India, Bangladesh, and Pakistan) to identify the etiology of diarrhea in children (35). The most common causes of diarrhea in the first year of life were rotavirus (attributable fraction [AF]: 16.3-27.8%), *Cryptosporidium* (AF: 5.3-14.7%), enterotoxigenic *E. coli* (AF: 1.4-7.0%), and *Shigella* (AF: 0.0-13.2%), but there was still variability across study sites (35). This pathogen profile was similar for older preschool children, though *Cryptosporidium* was rare in children after the 2<sup>nd</sup> year of life. Other studies have also highlighted the global importance of rotavirus (36), *Shigella* (36, 37),

ETEC (36, 38, 39), and *Vibrio cholerae* (40). In this section, I review several of these key pathogens and assess the potential role of sanitation in blocking their transmission.

### *Rotavirus*

Before the introduction of a vaccine, rotavirus infections were ubiquitous in children throughout the world, causing millions of hospitalizations and between 427,000 and 611,000 deaths each year (41-44). Historically, the incidence of rotavirus infection has been nearly the same in high-income and low-income countries (45, 46), though low-income countries have much higher rotavirus mortality rates (44).

The success of rotavirus as a pathogen can be largely explained by virology. Rotavirus is a non-enveloped double stranded RNA virus. It is characterized by high secretion rates ( $10^{11}$  particles per g or ml of feces), excellent survival in the environment, and a low infectious dose (10-100 virus particles) (47). While envelope viruses are especially susceptible to desiccation (drying out), heat, and detergents, non-enveloped virus like rotavirus can survive for weeks or even months on solid, non-porous surfaces and is resistant to many disinfectants (48, 49). The lipid bilayer of viral envelopes assist the virus in entering a host cell, but it also makes it difficult for the virus to survive outside of the host. The low infectious dose is also due to the virus' structure. Along with being non-enveloped, the 3-layer protein capsid allows it to survive the extreme pH in the human gut.

Transmission of rotavirus occurs via the fecal-oral route. There is little evidence, however, to suggest that improvements in drinking water or sanitation can impact transmission, as rates of infection are similar in high- and low-income countries (45, 46). Nevertheless, few studies have directly investigated the impact of sanitation on rotavirus

infection. Direct person-to-person transmission along with fomites probably plays the primary role (49), suggesting that proper hand hygiene can interrupt rotavirus transmission. Indeed, several studies have shown that alcohol-based hand sanitizers can reduce rotavirus transmission in hospital settings (50, 51) and the rate of gastrointestinal illness in community settings (52, 53). Despite evidence of its efficacy, adequate hand hygiene is difficult to maintain. Immunization, therefore, is considered to be most effective public health intervention against rotavirus. The two licensed rotavirus vaccines, Rotarix and RotaTeq, have shown a very high level of efficacy in high-income countries and Latin America (54). In low-income African countries, the vaccine has shown a lower efficacy, which has contributed to the slower rollout of the vaccine(55). Even with a lower efficacy, the vaccine is predicted to have a large public health impact in these higher burden countries.

### *Giardia and Cryptosporidium*

*Giardia* is among the most common human parasites, infecting up to 5-30% of children with diarrhea (56, 57). While infection is common, a recent study in 5 sites found that it was not associated with moderate or severe diarrhea (35). A systematic review also found that *Giardia* infection was not associated with acute diarrhea, but it was, however, associated with persistent diarrhea (58). Asymptomatic infection is fairly common, suggesting that host immunity plays an important role in the development of disease.

*Giardia* is a genus of protozoa, with two life stages: the infectious cyst and the mature trophozoite (59). During an infection, the trophozoite moves from the small bowel towards the colon, where encystation occurs. Infectious cysts are excreted in the stool in

large quantities (60), and a person may shed cysts for several weeks. The cysts can survive for days to months in the environment (61), because the cyst wall is impermeable to most molecules, and metabolism can slow or even stop (62). The infectious dose is very low, with studies suggesting as few as 1-10 cysts (63, 64).

In the United States and other high-income countries, *Giardia* transmission occurs predominantly through small water systems (65). Chlorination is much less effective for deactivating giardia cysts than for other enteric pathogens, such as *E. Coli*. Effective protection requires high levels of chlorine and up to one hour for deactivation (66). Filtration can be an effective means to removing giardia cysts from drinking water, but they are small and require filtration pores of 1  $\mu\text{m}$  (57). In developing country contexts, transmission occurs via contaminated food, water, or fomites. Studies in industrialized countries have shown that *Giardia* is readily transmitted in childcare settings (67), suggesting that poor hygiene can play a role in transmission.

*Cryptosporidium* was first identified in humans in 1976 (68, 69), and it was thereafter considered an important cause of diarrhea among those with HIV/AIDS and other immunocompromised persons (70, 71). Among those with HIV, it was an important cause of death before the advent of effective antiretroviral therapy (72, 73). Among immune competent persons, it was thought to cause asymptomatic infection or mild diarrhea. A recent study, however, identified *Cryptosporidium* as a major cause of moderate and severe diarrhea among children in low-income countries (35).

Similar to *Giardia*, *Cryptosporidium* is a protozoan parasite. Its infectious oocysts are excreted in feces for several weeks in large quantities (74). These oocysts survive for several weeks in water and soil, but survival is diminished by warmer temperatures and



the presence of microbes (61). These oocysts are substantially smaller than giardia cysts, requiring filters of less than 1  $\mu\text{m}$ , though even these are not completely effective (75). The infectious dose is also small; one study estimated an ID<sub>50</sub> (number sufficient to cause infection in 50% of people) of 132 oocysts (76). *Cryptosporidium* oocysts are even more resistant to chlorination than giardia cysts (57, 77), making chlorination virtually ineffective.

*Cryptosporidium* has been responsible for many outbreaks in the United States associated with drinking water supplies and swimming pools (78-80). One outbreak was documented in Nevada in spite of a water treatment system that exceeded government regulations (81), highlighting the difficulty of preventing transmission. Similar to *Giardia*, secondary transmission of *Cryptosporidium* can also occur within households and within childcare settings, suggesting that direct person-to-person contact can play a role (82, 83).

### *Vibrio Cholerae*

Cholera disease is characterized by profuse watery diarrhea, often called 'rice stool,' and vomiting. Historically, the disease was concentrated in the Ganges River Delta, and global pandemics occurred beginning in the 19<sup>th</sup> century. In fact, the seminal work of John Snow, which led to the removal of the handle of the Broad Street Pump in London, occurred during the third documented global pandemic of cholera. Globally, cholera tends to be geographically isolated, and most cases occur in endemic areas of South-Asia and Sub-Saharan Africa (40). Before the recent outbreak in Haiti, it was estimated that cholera accounted for 2.9 million (2.8 million in endemic countries and 87,000 in epidemic

countries) cases of disease and 92,000 (91,000 in endemic countries and 2,500 in epidemic countries) deaths worldwide (40).

Cholera is caused by the bacteria *Vibrio cholera*, which colonizes the upper small intestine. Disease is caused by the production of a toxin, cholera toxin, which increases chloride secretion and decreases sodium chloride absorption (84). This drastically increases the amount of fluid secreted into the intestine, and can cause dehydration rapidly. With a stool output of up to 1L per hour, the disease can cause death from dehydration in a matter of hours. The case fatality rate is very high, about 50%, but is drastically reduced in the presence of oral and/or intravenous rehydration therapies (85).

*Vibrio cholerae* is transmitted on the fecal-oral route predominantly via contaminated water, though direct person-to-person and food-borne transmission has been documented. The infectious dose is quite high, at least  $10^8$  organisms (86, 87). The organism not only survives very well in water, but it can thrive, reproduce, and evolve in aquatic and marine ecosystems (88-90). Due its water-borne nature, treating drinking water is obviously effective at preventing cholera. Improving sanitation, which prevents the initial contamination of water sources, has been shown to be just as effective (91).

#### *Pathogenic E. Coli and Shigella*

*Escherichia coli* is a very diverse species of bacteria found in the gut of most animals. Most strains of *E. coli* are commensals, and actually contribute to digestion in the lower intestine. Some strains, however, can cause disease in humans, including diarrhea. The most common pathogenic strain is enterotoxigenic *E. coli* (ETEC), which is the most common bacterial cause of diarrhea worldwide (92), causing an estimated 280 million

cases of diarrhea among children under five each year (39). The bacteria binds to the cells in the small intestine and produces at least one of two toxins, a heat-stable toxin or a heat-labile toxin which is very similar in structure and function to cholera toxin (38, 93). The toxin results in a profuse watery diarrhea often clinically similar to cholera, though it may also be milder or even asymptomatic.

The infectious dose of ETEC is relatively high, at least  $10^6$  organisms (94). In high-income countries, cases of ETEC associated diarrhea are usually attributable to contaminated food (95). In lower-income countries, contaminated food and water are thought to be the primary routes of transmission (96). ETEC is found in surface water in areas with poor sanitation (97), and has been shown to survive for months in water, though it is often not detected or underreported (98). Sanitation facilities can prevent the contamination of drinking water and food and have been shown to be protective against ETEC diarrhea(99).

Unlike ETEC, enterotoxigenic *E. coli* (EPEC) does not produce a toxin, but it adheres to the cells of the small intestine and causes lesions. EPEC has long been considered an important cause of diarrhea in low-income countries. The recent GEMS study found that EPEC infection was common in both cases and controls and was associated with diarrhea in only one of five study sites (35). The relatively high prevalence in controls can be explained by partial immunity conferred by previous infection. The high infectious dose, at least  $10^8$  organisms (100), suggests that contaminated water and food play a major role in transmission. Improved sanitation, along with piped drinking water and breastfeeding, has been shown to be protective against EPEC diarrhea (101). Less is known about the epidemiology of enteroinvasive *E. coli* (EIEC). It has a relatively high infectious dose of  $10^6$

organisms (102). Contaminated drinking water and food have long been considered the primary modes of transmission, but direct person-to-person contact has also been implicated in some situations (96, 103).

*Shigella* is a genus of bacteria very similar to EIEC. It is clinically unique in that it causes both diarrhea and dysentery. *Shigella* accounts for over 160 million cases (1.1 million deaths) of diarrhea or dysentery per year (37). It is very genetically similar to EIEC, but has a much lower infectious dose, as few as 10 organisms (104). This low infectious dose and the organism's ability to survive and multiply on food make it readily transmitted via contaminated food (105, 106), though flies and fomites may also play a role (107, 108).

### **Monitoring Access to Drinking Water and Sanitation**

Access to drinking water and sanitation services has increased substantially over the past decades. In 2000, world leaders met at the Millennium Summit and in 2001 adopted the UN Millennium Declaration, a commitment to end extreme poverty by 2015. Along with the declaration, several targets were created known as the Millennium Development Goals (MDGs), with deadlines in 2015. The target for drinking water and sanitation, Target 7C, was officially adopted in 2006 and states, "to halve<sup>1</sup>, by 2015, the proportion of people without sustainable access to safe drinking water and basic sanitation" (109, 110). The task of tracking global access to drinking water and sanitation was assigned to the World Health Organization and UNICEF Joint Monitoring Programme (JMP). Access to safe drinking water has increased worldwide from 76% in 1990 to 89% in 2012, surpassing the 2015 MDG target of 88%. Global access to improved sanitation has

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<sup>1</sup> The halving refers to the initial levels in 1990

also increased (from 45% in 1990 to 64% in 2012), though it falls short of the MDG target of 75% (111). While these worldwide gains in access are encouraging, there remains substantial heterogeneity and inequality between and within countries (111-113). Many countries have made substantial progress during the MDG period, while others have stagnated.

During the MDG period, the JMP was faced with several methodological challenges. During 2014, I worked as a consultant for the JMP and prepared a background paper summarizing and addressing some of these methodological challenges. A description of much of this work can be found in a report of a task force meeting (114). First, the official JMP method of linear regression (109, 111) failed to capture non-linear patterns of growth exhibited by some countries. Specifically, by using semi-parametric methods I identified countries that exhibited saturation (approaching 100% coverage), stagnation (showing little evidence of any increase in coverage), and acceleration (an initial period of no progress followed by steady progress). Figure 1.2 shows examples of some of the non-linear trajectories observed in the JMP dataset. Across the different indicators, between 15 and 38% of countries with more than 10 data points showed evidence of non-linearity. This new modeling approach provided additional information about potential increase in disparity among countries. I explored several alternatives to linear regression, such as the piecewise linear model and the generalized additive model. These methods are fairly straightforward, and can accurately capture the variety of trajectories in the JMP dataset.

Second, due to the formulation of the MDG Target 7C, countries have faced a moving 2015 target. As countries' data are updated, the official JMP projection changes, leading to a change in the estimate of the 1990 baseline, and, therefore, a change in the 2015 target.

This problem is exacerbated by non-linearity. By using a model that accounts for curvature, such as a generalized additive model, the effect of the moving baseline is minimized.

Third, while many countries had a good number of data points, others had very few, making even the most simple longitudinal assessment difficult. Multilevel modeling has been proposed as a way to borrow information between countries and use a regional trend when country-level data are sparse (115). I thoroughly evaluated this method and came to the conclusion that neighboring countries do not have similar trajectories; therefore, borrowing information between countries would not yield accurate estimates.

With the end of the MDG period in 2015, the emphasis is being shifted to the Sustainable Development Goals (SDGs) with new targets for the year 2030 (116-118). Table 1.2 displays the proposed WASH target for the SDGs. This new target introduces several new themes: 1) universal access and elimination as targets, 2) monitoring of open defecation, 3) the inclusion of hygiene (hand washing), 4) the inclusion of schools and health facilities, and 5) monitoring inequality. Many of the lessons learned during the MDG period will be translatable to the SDGs, but some challenges will persist. While the amount of data available to the JMP during the MDG period has increased rapidly, the challenge of data scarcity will be renewed. For example, data on open defecation is less common than on the improved/unimproved dichotomy.

Our approach to capture nonlinearities will provide a nice platform to address the fifth theme of the SDG mention above. Inequalities can be monitored through a method similar to the semi-parametric approach described above. Highlighting the divide between countries that are reaching 100%, making progress, and stagnating provides information for the JMP to identify inequalities.

## **Dissertation Objectives**

Hundreds of epidemiologic studies have investigated the association between sanitation and human health. The majority of these studies focus on diarrhea morbidity, mortality or nutritional status, mostly among children. An early systematic review by Esrey et al (119) found that improvements in excreta disposal led to a median reduction in diarrhea morbidity of 22%. The review also found a median reduction in all cause mortality of 21%, though most of these studies did not disentangle the effects of drinking water and sanitation. Another review of the early literature found that sanitation was a more important determinant of diarrhea morbidity, mortality, and nutritional outcomes than drinking water (120). In 2005, Fewtrell et al (121) conducted a more complete systematic review and meta-analysis of drinking water, sanitation, and hygiene intervention trials. Only two studies assessed a sanitation intervention alone, yielding a pooled reduction in diarrhea of 32%. A series of cross-sectional surveys using data from the Demographic and Health Surveys found a more modest impact of sanitation on health (33). The study reported pooled odds ratios of 0.87 for diarrhea, 0.85 for neonatal mortality, and 0.73 for stunting. While this body of evidence suggests a strong link between sanitation and human health, there are still important aspects of sanitation interventions that are understudied.

In this dissertation, I approach several distinct subjects to improve our understanding of the sanitation process. Chapters 2 and 3 are focused on the concept of herd protection, while Chapters 4 and 5 are focused on the risks associated with sharing sanitation between households, and the joint effects of levels of sanitation and water quality.

Herd protection arises when an infectious disease intervention provides some level of protection to non-recipients of that intervention. This may occur if the intervention prevents infection in a susceptible individual or if it reduces the contagiousness of an infectious individual. Vaccines and other interventions have shown evidence of herd protection against a variety of diseases. Sanitation should also provide herd protection, since it safely disposes of human excrement preventing environmental contamination. Neighboring households, therefore, should receive some indirect benefit when a household improves their sanitation practices. The vast majority of epidemiologic studies (33, 119-121) do not account for the indirect benefits of sanitation, and as a result, their results are likely underestimates of the true protective effect of sanitation. A few studies, mostly cross-sectional, have attempted to measure the herd protective effect of sanitation. In Chapter II, I create a mathematical framework for relating the herd protective impact of sanitation to that of vaccines and other interventions, such as insecticide treated bednets. In Chapter III, I use a longitudinal study in rural Ecuador to assess the effect of sanitation coverage in the community on child growth.

A shared sanitation facility is a latrine or toilet that is shared by more than one household. It may be a communal toilet or owned by a single household but used by neighbors or relatives. In 2012, approximately 11% of the world's population used a shared facility as their primary toilet or latrine (111). One reason that worldwide access to sanitation appears to lag behind water is a key difference in how access is defined. While public sources of drinking water (e.g., public taps) may be considered improved, any type of shared sanitation facility is considered unimproved, even if it is using an improved form of technology (Table 1.1). Cumming et al (122) show that after accounting for this



difference in definition, access for both services has increased by nearly the same amount. The classification of shared sanitation as unimproved stems from several concerns. First, communal latrines may be less hygienic, as they have more users and a managing institution may be lacking. Second, shared facilities may be less accessible, and long lines or inadequate safety may lead potential users to engage in a more convenient but less hygienic practice, such as open defecation. As shown by a recent review (123), the epidemiologic literature linking shared sanitation to disease is sparse. In Chapter IV, I use cross-sectional data from 51 Demographic and Health Surveys to assess the association between shared sanitation and the prevalence of diarrhea among children.

Sanitation and drinking water interventions interrupt the transmission of enteric pathogens by blocking different pathways (Figure 1). It is unclear, however, whether these two interventions are redundant services preventing the same cases of illness, or if they act independently or even synergistically. A few studies have investigated this potential interaction, with mixed results (124-127). In Chapter V, I use cross-sectional data from 217 Demographic and Health Surveys to look at the independent and joint effects of sanitation and drinking water on the prevalence of diarrhea. I also look at how these effects vary between countries and over time. Finally in Chapter VI, I conclude by summarizing these studies, their strengths and limitations, and future directions for research on sanitation and health.

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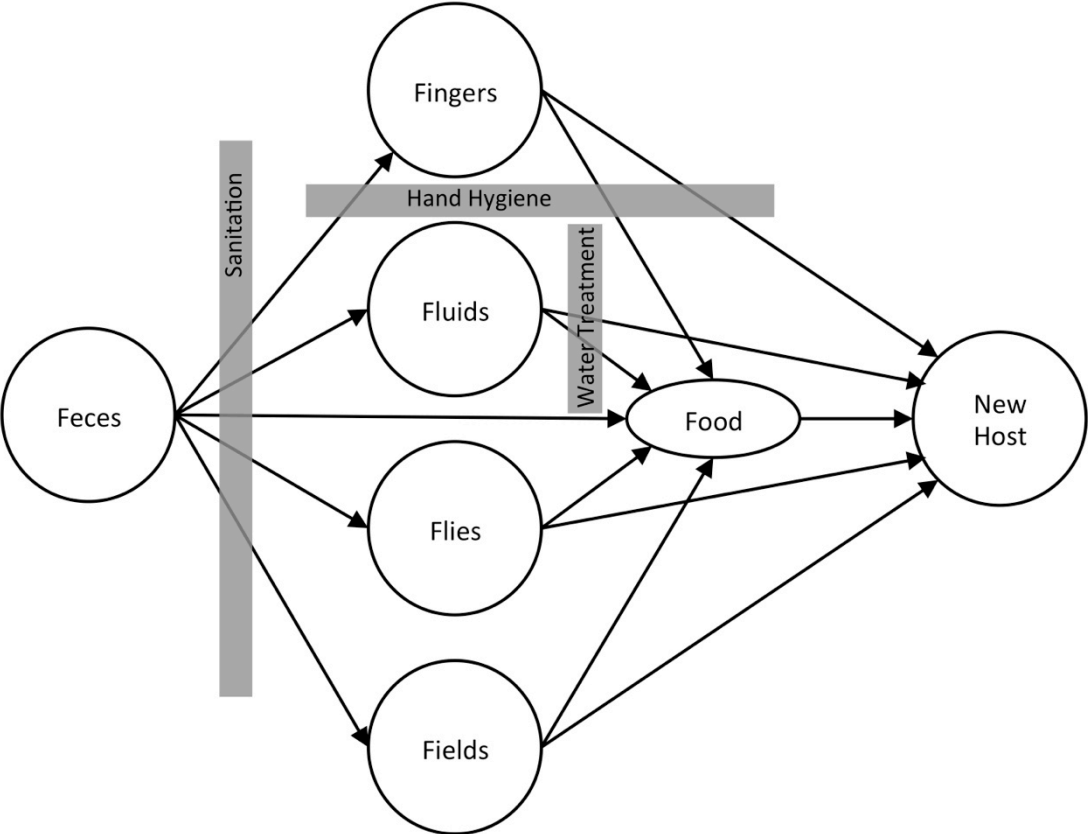


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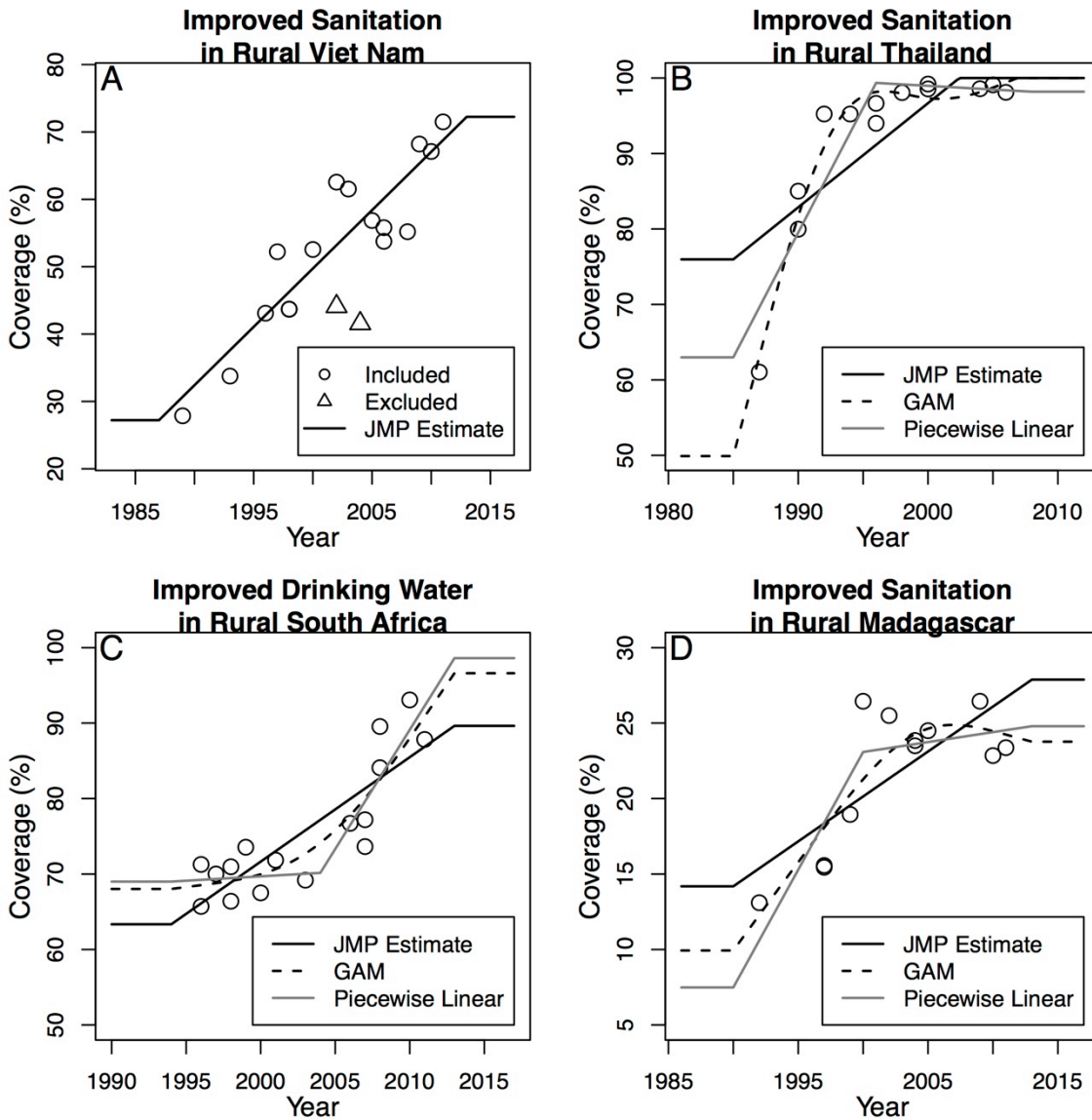
**Figure 1.1** The 'F' Diagram (5), and the potential for sanitation, water treatment, and hand hygiene to interrupt fecal-oral transmission.



**Table 1.1** World Health Organization/UNICEF Joint Monitoring Programme definitions of improved and unimproved drinking water and sanitation(111).

<b>Drinking Water</b>	<b>Sanitation</b>
<i>Improved</i>	<i>Improved</i>
<ul style="list-style-type: none"> <li>• Piped water into dwelling, yard, or plot</li> <li>• Public tap or standpipe</li> <li>• Tubewell or borehole</li> <li>• Protected dug well</li> <li>• Protected spring</li> <li>• Rainwater collection</li> </ul>	<ul style="list-style-type: none"> <li>• Flush or pour-flush to piped sewer system, septic tank, or pit latrine</li> <li>• Ventilated improved pit latrine</li> <li>• Pit latrine with slab</li> <li>• Composting toilet</li> </ul>
<i>Unimproved</i>	<i>Unimproved</i>
<ul style="list-style-type: none"> <li>• Unprotected dug well</li> <li>• Unprotected spring</li> <li>• Cart with small tank or drum</li> <li>• Tanker truck</li> <li>• Surface water</li> <li>• Bottled water</li> </ul>	<ul style="list-style-type: none"> <li>• Flush or pour-flush to elsewhere</li> <li>• Pit latrine without slab/open pit</li> <li>• Bucket</li> <li>• Hanging toilet or hanging latrine</li> <li>• Shared facilities of any time</li> <li>• No facilities, bush or field</li> </ul>

**Figure 1.2** Different trajectories of access to drinking water and sanitation. A - Example of the WHO/UNICEF Joint Monitoring Programme (JMP) method on a linear trajectory. B - Saturation, C - Acceleration, D - Deceleration, comparing the official JMP estimate with a generalized additive model (GAM) and a piecewise linear regression.



## Chapter II

### The mathematical theory of herd protection: A case study of sanitation interventions

#### Abstract

Herd immunity arises when a communicable disease is unable, or less able to propagate because a substantial portion of the population is immune either naturally or through vaccination. More generally, treatment interventions such as deworming drugs and environmental interventions like insecticide treated bednets show similar protective effects among non-recipients. Herd immunity through vaccine intervention is part of a broader concept that we call herd protection. To illustrate the broader mechanisms of herd protection we first summarize existing empiric evidence for herd protection presented in the literature and second construct and analyze a mathematical model in which enteric pathogens are transmitted through the household environment and a shared community environment. Herd protection is evident because as coverage of the intervention increases, the risk of infection declines among non-intervention households. The magnitude of herd protection depends on the extent to which improved sanitation prevents the spread of infection. Herd protection has many applications in infectious disease control, and likely exists for any intervention that either prevents infection in the unprotected or reduces an infectious individual's contagiousness. Studies that do not account for herd protection will underestimate the total protective effect of an intervention.

## **Introduction**

### *Herd Immunity*

Herd immunity is a foundational concept in public health and the basis of many vaccination strategies. The underlying principle is that a communicable disease is unable, or less able, to propagate through a population because a substantial portion of the population is immune (1-3). Early in the 20th century, epidemiologists recognized that the periodicity observed in measles epidemics was due to the accumulation of susceptible individuals during the inter-epidemic period (4, 5). An implication of this theory is that the risk in susceptible children is lower when a higher proportion of the population is immune.

For many infections immunity can be acquired via vaccination. For example, consider an unvaccinated population with a single infectious individual (Figure 2.1 - Panel A). In such a population, infection can spread uninhibited. However, when a portion of the population receives a vaccine with sterilizing immunity (Figure 2.1 - Panel B), the contacts of the infectious individual are more likely to be immune, reducing the number of subsequent infections. If coverage with the vaccine is high enough, transmission can be interrupted and the infection dies out. Many vaccines are not sufficiently efficacious to reach such a level of population immunity due to either waning immunity (e.g., pertussis) or low take rate (e.g., cholera or typhoid fever). Regardless, transmission can be attenuated and the risk of disease in the unvaccinated will decrease, as demonstrated for oral cholera vaccines in Bangladesh (Table 2.1).



### *Herd Protection*

Many infectious disease interventions provide protection to non-recipients without inducing immunity. This occurs if the intervention prevents infection in susceptible individuals or reduces an infectious individual's contagiousness. In the literature, this phenomenon has been called herd protection (6), herd immunity (7), herd effects (8, 9), indirect effects (10), mass effects (11), community effects (12), externalities (13, 14), dependent happenings (15), among other things. The term *herd protection* may be more accurate, as it implies a population mechanism similar to that of a vaccine with sterilizing immunity, but not necessarily requiring that the intervention induce immunity. Herd protection occurs when an intervention provides some level of protection to non-recipients of that intervention. Analogous to herd immunity, if coverage of an intervention is high enough, transmission can be interrupted and the pathogen eliminated. Several interventions have shown evidence of herd protection (Table 2.1).

School-based deworming programs led to decreases in helminthic infection among nearby children that did not receive deworming drugs (13, 16). Here, herd protection occurs because treated children stop shedding infectious eggs into the soil. Susceptible children, whether treated or not, are then less likely to become infected because there are fewer eggs in the soil (Figure 2.1 - Panels C and D).

Mass antibiotic distribution among children reduces the prevalence of trachoma infection among older individuals that did not receive treatment (6). This occurs because infectious children are cleared of their infections and are no longer contagious. Susceptible individuals in the population will then have fewer infectious contacts.

Insecticide treated bednets (ITNs) can also have protective effects among non-users (11, 12, 17). There are several mechanisms through which ITNs can provide benefits to non-users (Figure 2.1 - Panels E and F). First, ITNs kill mosquitos, and thus reduce the density of the local mosquito population. Second, ITNs act as a barrier between susceptible individuals and infectious mosquitoes, acting in the same way as a sterilizing vaccine by effectively reducing the susceptible fraction in the population. Third, ITNs act as a barrier between infected individuals and susceptible mosquitoes, lowering parasite burden among mosquitoes and leading to fewer infections among people not using ITNs.

#### *Sanitation and Herd Protection*

A sanitation intervention (i.e. improved latrines and toilets) has the potential to provide herd protection against diarrheal diseases. Enteric pathogens are predominantly transmitted via the fecal-oral route and often have environmental intermediaries, such as drinking water or fomites. Without improved sanitation, an infectious individual may contribute to high levels of environmental contamination in their household and in the surrounding community (Figure 2.1 - Panel G). Susceptible individuals are then exposed to enteric pathogens in their household or their community (18). However, when infectious individuals use improved sanitation, their excrement is better contained and less able to contaminate the household and/or surrounding community (Figure 2.1 - Panel H). Susceptible persons in other households, regardless of their own sanitation practices, will face lower levels of contamination in the community environment.

Herd protection is manifested when one or more pathways of transmission are interrupted through an intervention. Pathogens that cause diarrhea can exploit many

different pathways, including food, water, hands, and fomites. In this paper, we examine different mechanisms of transmission to explore the role that each plays in the manifestation of herd protection. Due to the lack of empirical research on herd protection from sanitation, we use a mathematical model, which serves as an explanatory tool to guide further research.

The dynamics exhibited in these examples are similar and can be summarized in the following equation for  $R_o$ , based on an environmental infection transmission system model (EITS) described previously (19):

$$R_o = \pi \cdot \frac{\delta}{\gamma} \cdot \frac{\rho N}{\rho N + \mu}$$

where  $\pi$  = infectivity,  $\gamma$  = rate of recovery from infection,  $\delta$  = shedding rate into the environment,  $\rho$  = pick up or ingestion rate,  $\mu$  = pathogen die-off rate in the environment, and  $N$  = total human population. In this formulation environmental processes attenuate  $R_o$  analogous to vaccine coverage, reducing the risk of infection among all individuals in the population. Herd protection can also lead to elimination when that attenuation forces  $R_o < 1$ .

## **Methods**

### *Model Structure*

We simulate a community of 500 individuals, nested within 100 households. Individuals are categorized as susceptible, infectious, or immune, and immunity is assumed to be permanent (SIR model). All transmission of pathogens occurs via the environment (19). Infectious individuals can transmit pathogens to susceptible individuals by either of

two pathways (Figure 2.2). 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  $\mu$ . Individuals are modeled as discrete entities using a stochastic framework, and pathogens in the environment are modeled as continuous using ordinary differential equations (20). The model was coded in R version 3.0.2 (21).

### *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. In the first scenario, no households in the community are using the intervention. 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 1100 runs. The median cumulative incidence is used to calculate the protective efficacy of the intervention.

To estimate the herd protection from sanitation, the direct, indirect, and total effects of the intervention are measured using the framework presented by Halloran and Struchiner (Figure 2.3) (10). 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  $1 - \text{Risk}_1 / \text{Risk}_0$ , where  $\text{Risk}_1$  represents the risk in the intervention group and  $\text{Risk}_0$  represents the risk in the non-intervention group. The indirect effect represents the herd protection provided by the intervention. It is estimated at each level of coverage by the equation  $1 - \text{Risk}_0 / \text{Risk}_{0^*}$ , where  $\text{Risk}_{0^*}$  represents the 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  $1 - \text{Risk}_1 / \text{Risk}_{0^*}$ .

### *Sanitation Interventions*

Sanitation is a household-level intervention. In our model, infectious individuals in households with better sanitation have lower rates of shedding into their own household environment and/or into the community environment compared to those in households with worse sanitation. This is represented in Figure 2.2 by the darker shaded lines among households practicing open defecation, indicating  $\phi_1 < \phi_0$  and  $\delta_1 < \delta_0$ .

Sanitation practices can vary dramatically. Here we consider three potential practices: open defecation, use of low quality latrines, and use of high quality flush toilets. The relative size of the shedding rate parameters depends on which two sanitation

scenarios are being compared. For example, open defecation will result in the highest levels of community contamination. A low quality latrine concentrates excrement near the household, but may not properly contain it. This results in less community contamination but no impact on household contamination, relative to open defecation. Relative to open defecation, high quality flush toilets will concentrate excrement, but also properly contain it. This will result in the lowest rates of shedding into both the household and the community environments. Shedding parameter values are shown in Table 2.2. 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 based on the following criteria: 1) the baseline risk without the intervention is approximately 70%, 2) the intervention yields a direct efficacy of between 10-20% (22-24), and 3) the indirect effect will in some circumstances dominate the direct effect.

## **Results**

As described in the methods, we examine three intervention scenarios: 1) improving sanitation from practicing open defecation to using a low quality latrine (resulting in diminished community-level contamination but ongoing household-level contamination); 2) improving sanitation from using a low quality latrine to using a high quality latrine (resulting in both diminished household- and community-level contamination); and 3) improving sanitation from practicing open defecation to using a high quality latrine. Specific incidence levels presented in the results are based on the assumptions detailed in the methods section.

### *Low Quality Latrines versus Open Defecation*

When everyone is practicing open defecation, the median cumulative incidence is 71.6 per 100 persons (Figure 2.4 - Panel A). As coverage of low quality latrines increases, the cumulative incidence in both users and non-users declines. When coverage reaches 100% (everyone is using a low quality latrine), the median risk is 50.8 per 100, providing a total protective efficacy of 29.1% ( $0.291 = 1 - 0.508/0.716$ ). This effect is entirely attributable to the herd protection of the intervention, as the direct effect is negligible (Figure 2.4 - Panel D).

A typical epidemiologic study measuring only the direct effect would fail to detect any benefit from this intervention. Though the risk declines in both groups, there is no difference between the groups. This occurs because users and non-users have similar rates of shedding pathogens into their household environments (Table 2.2). Levels of contamination in the two types of households will be similar, so individuals face a similar risk of infection.

Although the intervention provides no direct benefit to the users, the risk in both users and non-users declines as coverage increases (Figure 2.4 - Panel C) because the intervention reduces the shedding of pathogens into the community environment (Table 2.2). As a result, the community environment has fewer pathogens, and every individual in the village benefits equally.

### *High Quality Toilets versus Low Quality Latrines*

When everyone uses a low quality latrine, the median cumulative incidence is 50.8 per 100 persons (Figure 2.5 - Panel A). As more households use high quality toilets, the

cumulative incidence declines until it reaches 29.1 per 100, a total reduction of 42.7%. This reduction is attributable to both a direct benefit to the user and herd protection (Figure 2.5 - Panel D). Also, as more households use high quality toilets, the epidemic is more likely to die out before reaching a substantial portion of the population. This is evident by the greater thickness of the violin plots at lower values of cumulative incidence (Figure 2.5 - Panels A and B).

At every level of coverage, the risk is always higher among users of low quality latrines than among users of high quality toilets (Figure 2.5 - Panel C). This occurs because of the reduced rate of shedding into the household environment among users of high quality toilets. The direct protective efficacy ranges between 9.3% and 20.0%.

As coverage of the high quality toilet intervention increases, the cumulative incidence of infection declines among both groups of households. This decline, a manifestation of herd protection, does not occur because of a reduced rate of shedding into the community environment. Instead, it is attributable to less transmission within households using high quality toilets, and, therefore, fewer overall infectious individuals. Fewer infectious individuals results in lower amounts of contamination in the community environment, which benefits all equally.

### *High Quality Toilets versus Open Defecation*

When everyone is practicing open defecation, the cumulative incidence is 71.6 per 100 persons (Figure 2.6 - Panel A). As coverage of high quality toilets increases, the risk in the overall population declines to 29.1 per 100 (Figure 2.6 - Panel B). This is a total reduction in risk of 59.4%; herd protection (the indirect effect) accounts for more than



75% of this reduction. Similarly to what was seen above, stochastic die out of the epidemic is much more likely when coverage of high quality toilets is greater than 70% (Figure 2.6 - Panels A and B).

The direct benefit in this scenario occurs for the same reasons it occurs when comparing high with low quality toilets, namely, there is a reduced rate of shedding into the household environment. The indirect benefit, however, can be explained by two mechanisms found in the above examples. First, users of high quality toilets have lower rates of shedding into the community environment. This results in a lower risk among everyone in the village. Second, users of high quality toilets have lower rates of shedding into the household environment. Similar to what occurs when comparing high quality toilets to low quality latrines, this reduced household shedding results in fewer overall cases, which reduces the cumulative amount of shedding into the community environment.

## **Discussion**

While evidence of herd protection has been seen for vaccines, de-worming drugs, ITNs, and to some extent sanitation, it should exist for any intervention that either prevents infection in a susceptible individual or reduces an infectious individual's contagiousness. The causal mechanism will vary by intervention and by disease, but the overarching concept of herd protection is unvarying: the intervention benefits both users and non-users. Failure to account for this indirect effect will result in an underestimate of the actual protective effect of the intervention (7, 12). In some scenarios, the intervention provides no direct benefit to the user. Without accounting for herd protection, such an intervention would appear entirely ineffective. One such intervention is the potential transmission

blocking malaria vaccine, which provides no direct benefit to the recipient, but may provide indirect benefits to the surrounding community (25, 26).

Sanitation interventions have the potential to provide herd protection via two mechanisms. First, if the intervention reduces shedding rates into the household, there will be fewer cases due to within-household transmission. Fewer secondary cases will result in less cumulative environmental contamination in the community. Second, if improved sanitation can reduce the rate of shedding into the community environment, all surrounding households will benefit regardless of their own sanitation practices. The opposite is also true: low quality sanitation in one household will have an adverse effect on the surrounding households.

Few studies have sought to estimate the herd protective effect of sanitation. Barreto et al (27) conducted two cohort studies in Salvador, Brazil, one before a city-wide sanitation campaign and one after. The study attributed the 21% reduction in diarrhea prevalence to the increase in sewer-connected toilet coverage (from 26% to 80%). Household toileting did not explain the reduction. Another smaller study in a rural village in Zimbabwe showed that the rate of diarrhea was lower among children that practiced open defecation when their nearest neighbor had an improved pit latrine (28). In a cluster-based household survey in rural India, higher levels of sanitation coverage at the cluster (village) level was associated with a lower prevalence of diarrhea after accounting for the sanitation practices of the household (14). They found that the prevalence was 47% lower among children in a household with improved sanitation in a village with 100% coverage compared to a child in a household without improved sanitation in a village with 0% coverage. 75% of that overall benefit was attributable to the indirect effect. They made no

attempt, however, to account for differences in village characteristics (village-level confounders). Buttenheim et al. used a cluster randomized trial in an urban slum to show that the percent of households using an improved latrine has a beneficial impact on the short-term nutritional status of children (29). Corsi et al. used data from the 2004 Demographic and Health Survey in Bangladesh and observed a strong association between the percentage of households with a modern toilet and nutritional indicators for children, but that association disappeared when they controlled for household and community characteristics (30). A longitudinal study of 1,233 children in urban Brazil found that the duration of diarrhea episodes were shorter when a greater percentage of households were connected to the sewer system (31). These studies suggest that sanitation interventions may provide some level of herd protection. Future studies should seek to better quantify the direct and indirect effects and adjust for community-level confounders.

As in all modeling exercises, our findings could be sensitive to a relaxation of our simplifying assumptions. We assumed different pathogens are able to exploit different pathways to varying degrees. Cholera has a high infectious dose and thrives in surface water enhancing its ability to survive in our so-called 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.

We also simplified the details of sanitation practices, which vary substantially across countries and even within individuals. We chose 3 generic practices for the purposes of isolating the benefits of sanitation. Our analysis does not seek to estimate the actual amount of herd protection from a given sanitation intervention. The actual amount of both

direct and indirect effects 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. Also, the degree of environmental connectivity between households will vary by setting and pathogen. For example, the transmission of cholera is enhanced when households are connected via surface water (32).

This conceptual framework has important policy implications. If sanitation has a herd protective effect, then it is much more cost effective than previously thought. Also, sanitation campaigns often aim to achieve 100% coverage in communities, but 100% compliance is elusive (33). Immunization policy is often based on reaching a threshold of vaccination coverage at which transmission will be interrupted and the disease eliminated. It is unclear, however, if such a threshold exists for sanitation.

Interventions for the control of infectious diseases can provide indirect protection to non-users. Although the mechanism behind herd protection varies by pathogen and transmission cycle, the goal of providing sufficient coverage to interrupt transmission will be the same. Sanitation interventions, for example, are largely focused on preventing environmental contamination that has the potential create risks at the community level. Sanitation, however, does not operate in isolation. As enteric pathogens exploit multiple pathways that are interdependent, further studies should focus on herd protection in multiple interventions including water, sanitation, and hygiene (34).

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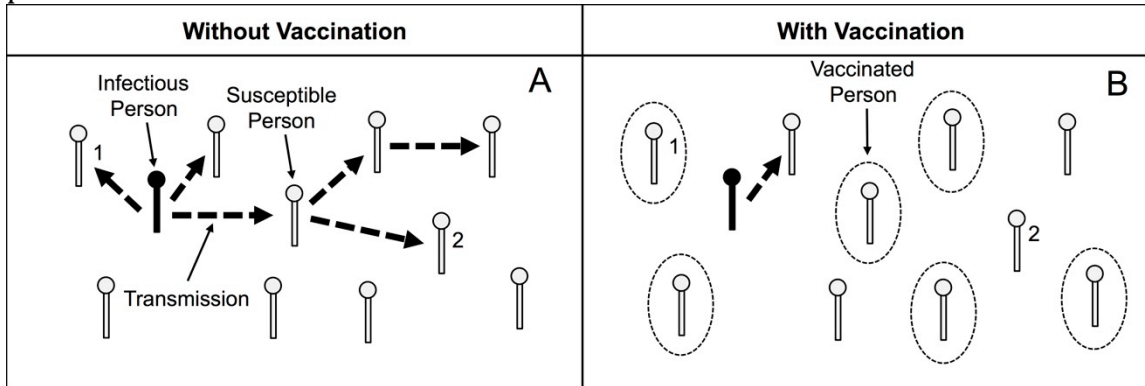
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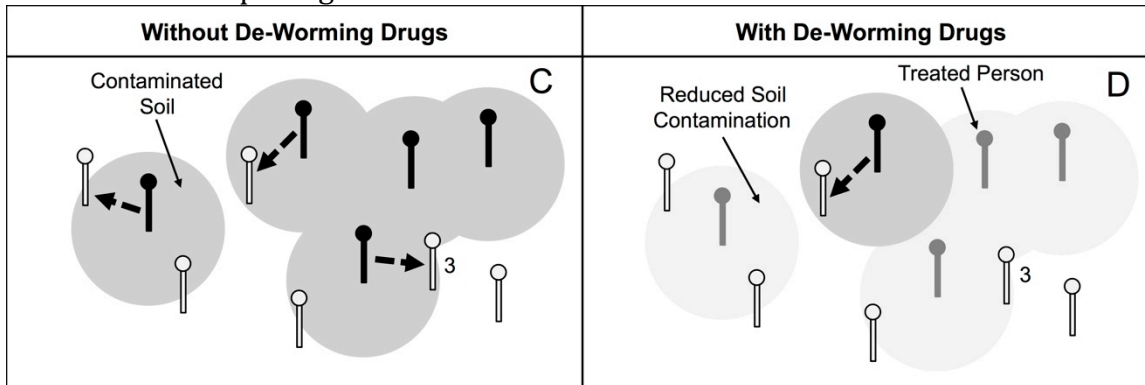
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**Figure 2.1** Four examples of herd protection.

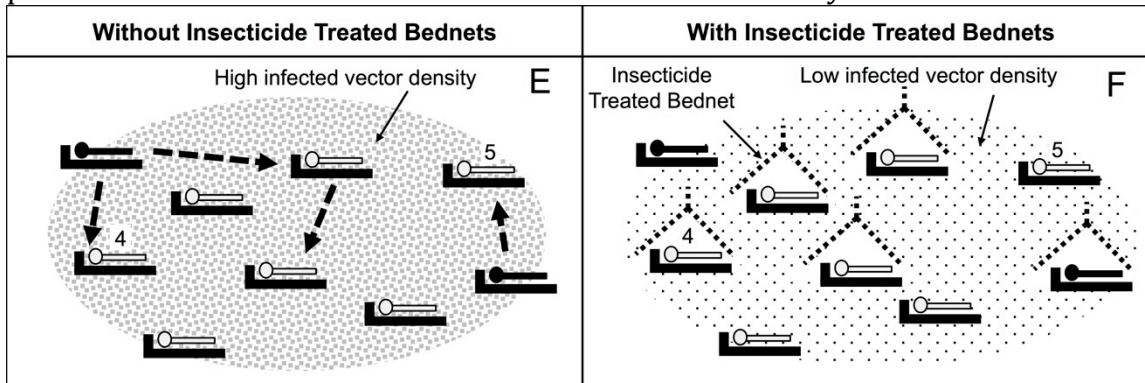
Panels A & B - A pathogen that is transmitted person-to-person. Person 1 receives direct protection from the vaccine. Person 2 is not vaccinated but receives indirect (herd) protection because others are vaccinated.



Panels C & D - Helminth infection transmitted through the soil. Treated individuals shed fewer eggs into the soil, so Person 3 receives indirect (herd) protection because they encounter fewer pathogens in the soil.

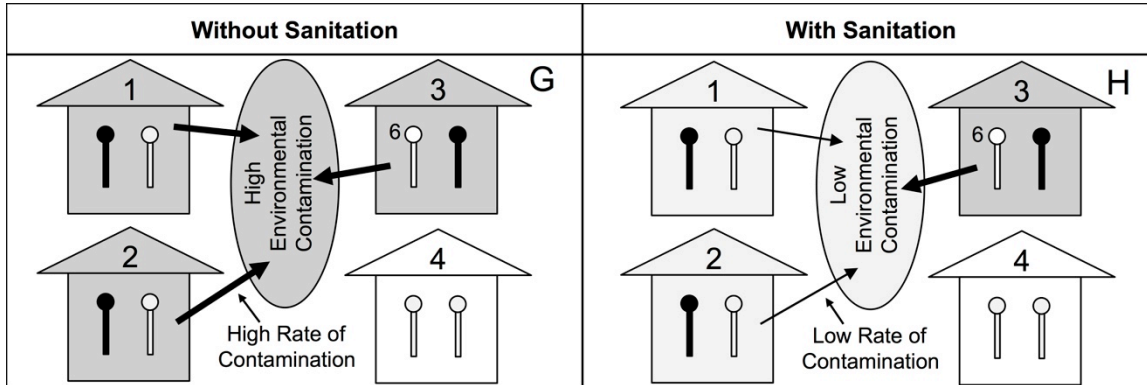


Panels E & F - Malaria infection transmitted through mosquitoes. Person 4 receives direct protection by using a bednet. Person 5 does not use a bednet, but receives indirect (herd) protection because others use bednets and the vector density is reduced.





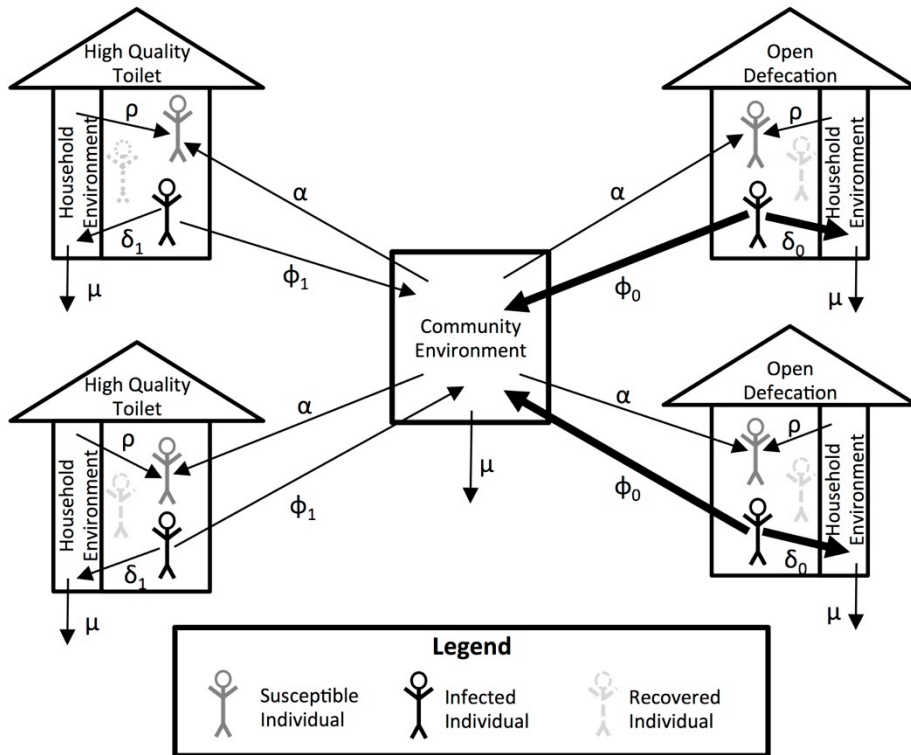
Panels G & H - Enteric pathogens transmitted through the environment. All households in Panel G have poor sanitation and infected individuals contaminate both their own household and the community environment. In Panel H, households 1 and 2 have improved sanitation, which results in less environmental contamination. Person 6 still uses poor sanitation, but receives indirect (herd) protection because they encounter fewer pathogens in the environment.



**Table 2.1** Summary of key studies assessing herd protection

<b>Study</b>	<b>Intervention</b>	<b>Outcome</b>	<b>Evidence of Herd Protection</b>
Ali et al 2005 (7)	Individual-randomized oral cholera vaccine	Cholera	The risk of cholera was lower among both placebo and vaccine recipients when vaccine coverage within 500m of their household was higher. For each 1% increase in vaccine coverage, the odds of cholera declined by 2-4%.
Hawley et al 2003 (12)	Village-randomized Insecticide Treated Bednet Distribution	Clinical Malaria, parasitemia, anemia, hemoglobin levels, child mortality	Persons in intervention villages had significantly better outcomes than those in control villages, and persons in control villages within 300m of intervention villages had significantly better outcomes than those from villages >300m from intervention villages. Among control households, health outcomes were better when the proportion of households within 300m using ITNs was higher.
Miguel and Kremer 2000 (13)	School-randomized albendazole treatment in children	Moderate or heavy helminth infection	Within schools randomized to receive treatment, the prevalence was 14 percentage points lower among those that actually received treatment compared to those that did not (direct effect). The prevalence was 12 percentage points lower among those from treatment schools that did not receive treatment compared to those from control schools (indirect effect). Among those from control schools, the prevalence was lower when nearby children (within <3km) were from treatment schools.
House et al 2009 (6)	Mass azithromycin treatment in children ≤10 years of age	Trachoma infection in persons >11 years of age (persons not receiving treatment)	Prevalence of infection among the untreated population declined from 15.5% (10.7-20.3) to 8.2% (5.1-11.4), a relative reduction of 47% (33-57, p=0.002).

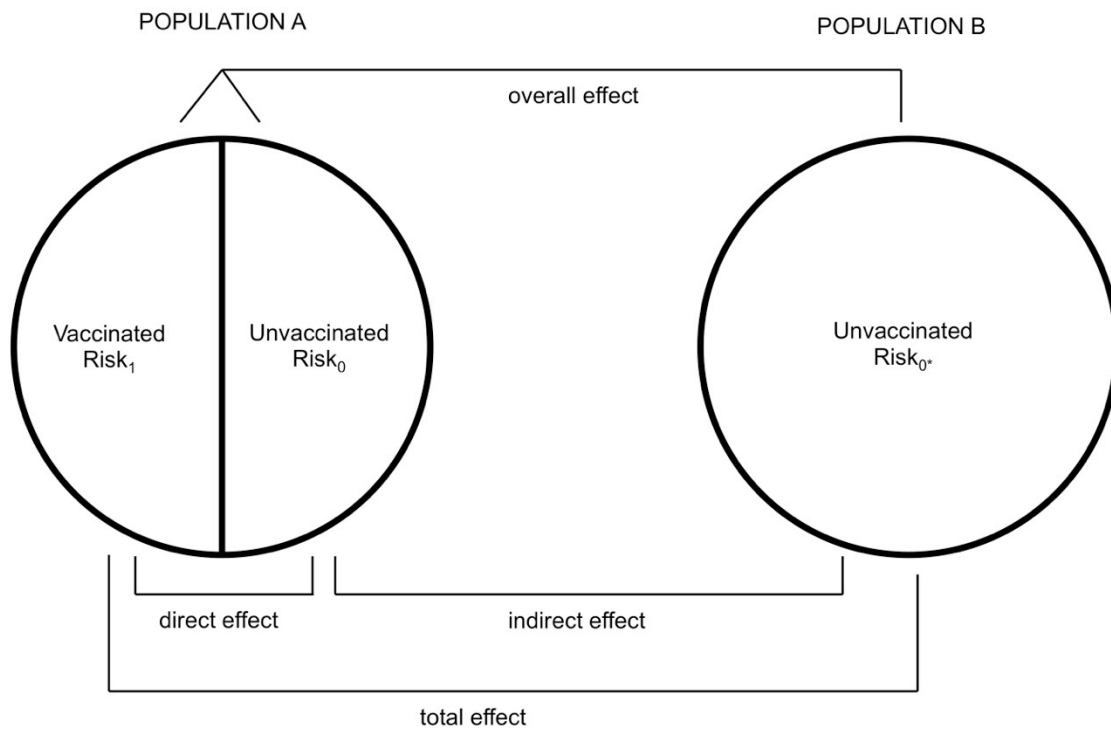
**Figure 2.2** Framework of household transmission model.



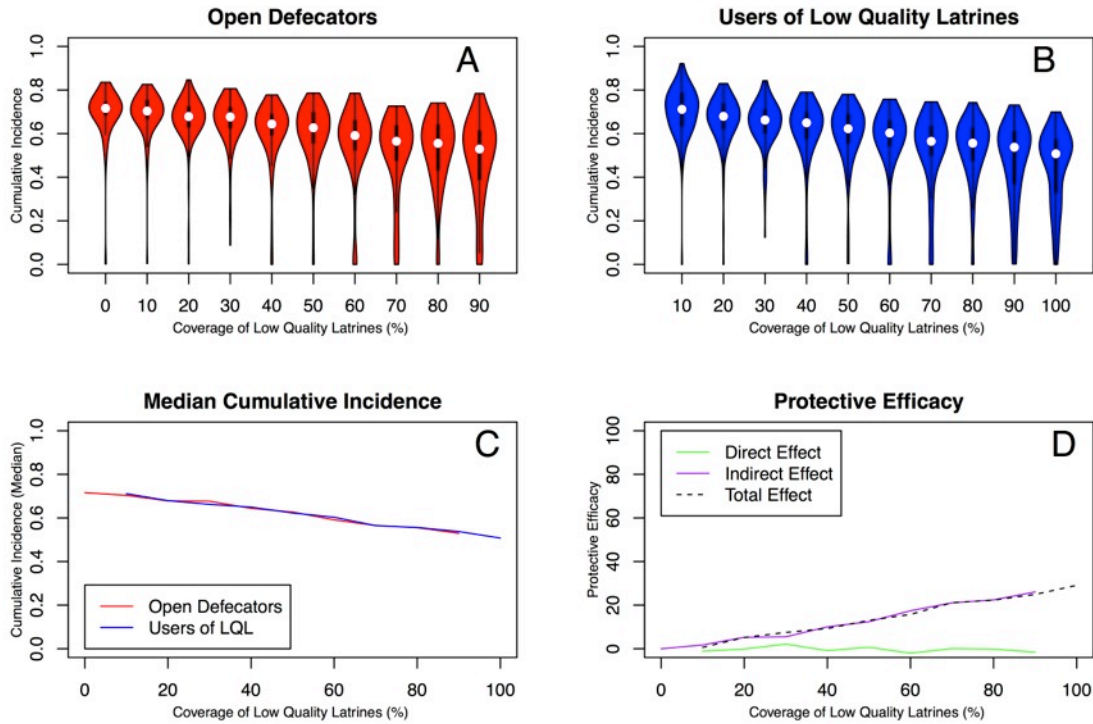
**Table 2.2** Parameter values and their description used in the analysis. All rates are per day.

Parameter	Description	Value
-	The number of households in the community	100
-	The number of individuals per household	$\sim N(5, sd=2)$
$\alpha$	The rate at which susceptible individuals pick up pathogens from the community environment	$1/10^6$
$\rho$	The rate at which susceptible individuals pick up pathogens from their household environment	$1/10^5$
$\phi_{OD}$	The rate at which infectious individuals practicing open defecation shed pathogens into the community environment	80
$\phi_{LQL}$	The rate at which infectious individuals using low quality latrines shed pathogens into the community environment	60
$\phi_{HQL}$	The rate at which infectious individuals using high quality toilets shed pathogens into the community environment	60
$\delta_{OD}$	The rate at which infectious individuals practicing open defecation shed pathogens into their own household environment	400
$\delta_{LQL}$	The rate at which infectious individuals using low quality latrines shed pathogens into their own household environment	400
$\delta_{HQT}$	The rate at which infectious individuals using high quality toilets shed pathogens into their own household environment	200
$\mu$	The rate at which pathogens die in the environment	$1/10$
$\gamma$	The rate at which infectious individuals recover from infection	$1/3$

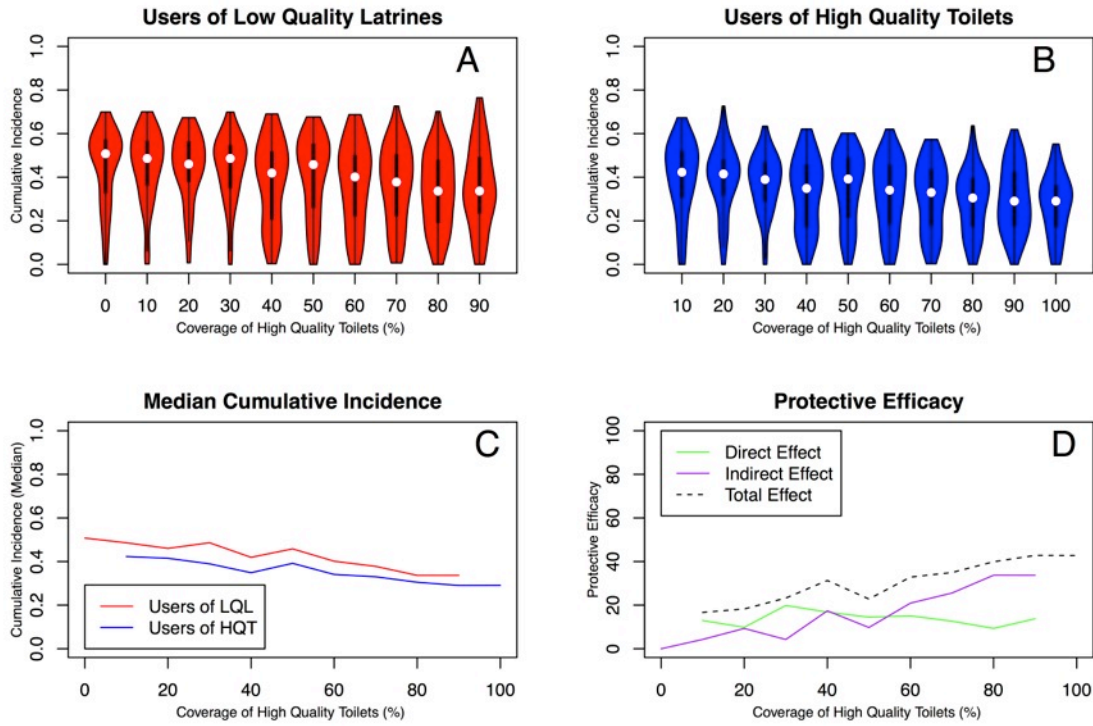
**Figure 2.3** The framework proposed by Halloran and Struchiner (10) for measuring the herd protection from an intervention.



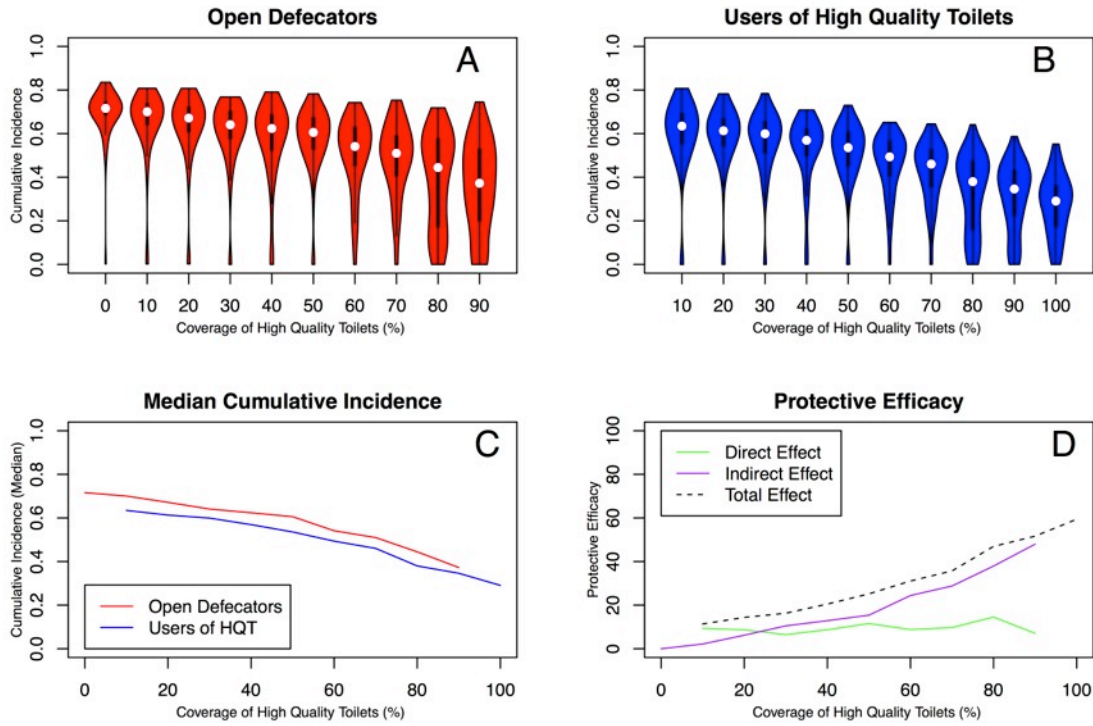
**Figure 2.4** The effect of low quality latrines compared to open defecation in a rural village. At each level of coverage of low quality latrines, the model was simulated 100 times. Panels A and B are violin plots showing the distribution of the cumulative incidence at each level of coverage among those practicing open defecation and those using low quality latrines, respectively. The width of the violin plot reflects the density of the distribution and a box-and-whisker plot is nested within the violin plot. Plots C and D show the median values for the cumulative incidence and protective efficacy, respectively.



**Figure 2.5** The effect of high quality toilets compared to low quality latrines in a rural village. At each level of coverage of high quality toilets, the model was simulated 100 times. Panels A and B are violin plots showing the distribution of the cumulative incidence at each level of coverage among those using low quality latrines and those using high quality toilets, respectively. The width of the violin plot reflects the density of the distribution and a box-and-whisker plot is nested within the violin plot. Plots C and D show the median values for the cumulative incidence and protective efficacy, respectively.



**Figure 2.6** The effect of high quality toilets compared to open defecation in a rural village. At each level of coverage of high quality toilets, the model was simulated 100 times. Panels A and B are violin plots showing the distribution of the cumulative incidence at each level of coverage among those practicing open defecation and those using high quality toilets, respectively. The width of the violin plot reflects the density of the distribution and a box-and-whisker plot is nested within the violin plot. Plots C and D show the median values for the cumulative incidence and protective efficacy, respectively.





### Chapter III

I get height with a little help from my friends: The herd protective effect of neighborhood sanitation on child growth in rural Ecuador.

#### Abstract

Sanitation can prevent childhood stunting by preventing diarrhea, helminth infections, and environmental enteropathy. Most studies of sanitation and nutrition focus on the sanitation environment of the household, ignoring any potential neighborhood effect. From 2008 to 2013, we took repeated anthropometric measurements on 1,314 children under five years of age in 24 rural Ecuadorian villages. Using mixed effects linear regression, we estimate the household and neighborhood effects of sanitation on child growth (height-for-age). Children from households with improved sanitation were on average 0.34 centimeters taller (95% CL -0.09-0.77) than those from households with unimproved sanitation, after adjusting for age, sex, and household socioeconomic variables. Sanitation at the neighborhood level had a much bigger impact, as those with 100% coverage in their neighborhood were 1.68 cm taller (95% CL 0.30-3.06) than those with 0% coverage. The protective effect of neighborhood sanitation is manifested during the second year of life, the time at which growth faltering is most likely to occur. Our study highlights that a household's sanitation practices can provide herd protection to overall

community. Studies which fail to account for the positive externalities that sanitation provides will underestimate the overall protective effect.

## **Introduction**

Childhood stunting (low height-for-age) affected 26% of children under 5 worldwide in 2011, contributing to over 1 million deaths (1). Other than mortality, childhood stunting is also an important risk factor for outcomes later in life, including behavioral problems, underachievement in school, and chronic diseases such as diabetes (2-5). Child growth is influenced by many factors, including fetal exposures (5), food security (6), and micronutrient deficiencies (7), and inadequate access to water, sanitation, and hygiene (8, 9).

Increasing evidence suggests that a poor sanitation environment leads to not only diarrhea (10-12) and helminth infection (13) but also persistent exposure to pathogens responsible for environmental enteropathy (14-16), a chronic subclinical infection of the gut characterized by atrophy of the intestinal villi and decreased absorptive capacity (17). All three of these conditions reduce nutrient absorption and promote an immune response that increases energy expenditure, resulting in slower growth.

Most studies of sanitation and nutrition, however, focus on the sanitation environment of the household (8, 9, 18, 19), ignoring any effect of neighboring households. As shown in Chapter 2, sanitation can provide positive externalities, i.e. herd protection, whereby improved sanitation in one household prevents infection in nearby households by reducing contamination of the shared environment. We undertook this longitudinal study

to estimate the effect of sanitation at the household and neighborhood level on child growth.

## **Methods**

### *Study Population*

The study took place in 24 rural villages in the Esmeraldas province of northwestern Ecuador. These villages lie along several river systems near the town of Borbón, and many are still not accessible by road. The population is predominantly Afro-Ecuadorian, though some villages have a high concentration of Chachis, an indigenous group. Between December 2008 and July 2013, each village was visited four times.

### *Anthropometry*

Anthropometric data were collected for all children under 5 years of age at each of the study visits. At each observed time point, height or length was measured in centimeters. Height-for-age z scores (HAZ) were calculated using WHO Anthro software (20). The z scores are standardized by age (in months) and gender. Observations were excluded if a z score was  $>6$  or  $<-6$ . Binary indicators for moderate and severe stunting were created based on Z scores of less than -2 and less than -3, respectively. Chachi children were excluded from the analysis because their anthropometry was substantially different from that of other children.

### *Sanitation Variables*

Sanitation information was collected for each household during each of the 4 study visits. We classified each household's sanitation access as unimproved (no facility, pit latrine without a slab, pit latrines without a seat) or improved (pit latrines with a slab and seat, pour-flush and flush toilets). During each visit, the GPS location of the household was recorded or verified. For each household at each study visit, sanitation coverage was calculated as the proportion of households within a 500-meter radius that have improved sanitation. Other distances were considered (e.g., 250, 750, and 1000 meters), but we selected 500 meters based on the housing density and size of the villages.

### *Covariates*

During each study visit, information is gathered on educational attainment, asset ownership, and housing construction. For each household, the maximum number of years of completed education of all persons was used. Principal components analysis was used to create a wealth index for each household for each visit based on the following variables: house tenancy, house construction, roof material, floor material, source of lighting, source of drinking water, and ownership of assets (television, stove, refrigerator, blender, stereo, DVD player, computer, washing machine, solar panel, generator, bicycle, motorcycle, car, canoe, cell phone, chainsaw, business, farm, cattle). From this index, we then created wealth quintiles. For each household, we also calculated the mean wealth index of other households within 500 meters. Based on the assumption that wealth and sanitation practices are stable over time, missing data on sanitation and wealth were imputed using values from previous or later study visits. Anthropometric data was not imputed.

## *Statistical Analysis*

Mixed effects linear regression was used to model the growth curves (height in cm) and HAZ of children. Age was included in these models using a restricted cubic spline with knots at 0.75, 1, 1.25, 1.5, and 3.5 years. These models account for repeated observations by including a random intercept and a random slope for the linear age term for each child. We ran 4 separate models, including different covariates in each. In the first model, we include only the household's sanitation (improved versus unimproved). In the second, we also adjust for the child's age and sex, and the household's wealth. In the third model, we include sanitation coverage within 500 meters and the mean wealth index of households within 500 meters. In the fourth model, we include an interaction term for household sanitation and sanitation coverage.

To assess spatial correlation, we created empirical semivariograms of the residuals from the 4th model (21). A fifth model was then fit with a random intercept and age slope for each child and spatial correlation of residuals using an exponential covariance function. Finally, to assess the impact of sanitation on the prevalence of stunting, we fit a mixed effects logistic regression for both moderate ( $HAZ < -2$ ) and severe stunting ( $HAZ < -3$ ). All analysis was conducted using the lme4 (22) and nlme (23) packages in R version 3.0.2.

## **Results**

### *Summary Statistics*

A total of 1,314 children were included in the analysis for a total of 2,225 observations. Table 3.1 shows summary statistics across each of the 24 villages. Overall, the prevalence of moderate stunting was 14.0%, but this ranged from 0% in village 31 to

58.3% in village 30. 73.9% of all households had access to improved sanitation, and this ranged from 30.2% in village 26 to 95.3% in village 9.

### *Height in Centimeters*

During the first year of life, children in this cohort were on average equal to the WHO standard population (Figure 3.1). During the second year of life, however, growth stalled, leading to 3.4 and 2.7 cm deficit by age 24 months among boys and girls, respectively.

Children from households with improved sanitation were 0.41 (95%CL -0.01-0.84) cm taller than children from household with unimproved sanitation (Table 3.2, Model 1). Differences in socioeconomic status explained some of this difference (Table 3.2, Model 2). Additional years of education in the household did not translate into taller children, but ownership of assets did. Children from households in higher wealth quintiles were consistently taller than those in lower wealth quintiles (Table 3.2, Models 2-5).

The sanitation status of households within 500 meters was a strong predictor of child height. Children from areas with 100% coverage were 1.68 (95%CL 0.30-3.06) cm taller than children from areas with 0% coverage, after controlling for all other covariates (Table 3.2, Model 3). The results of Model 4 allow us to test whether the effect of sanitation coverage depends on the sanitation access of the household. The effect of sanitation coverage (100% compared to 0% coverage) when the household has unimproved sanitation is a difference of 1.05 (95%CL -0.91-3.02) cm and 2.06 (95%CL 0.40-3.71) cm when the household has improved sanitation (Table 3.2, Model 4). The difference of these

two effects was not statistically significant. Accounting for the spatial correlation of observations did not have a meaningful effect on the point estimates (Table 3.2, Model 5).

Figure 3.2 shows predicted growth curves among boys by level of sanitation coverage adjusted for all covariates. These predictions are based off a model that includes an age-sanitation interaction, thus allowing the effect of sanitation to vary across age. There seems to be little effect of sanitation coverage during the first year of life, and children in our study grow along the WHO standard trajectory. During the second year, however, children in our study experience a lull in growth, which is more pronounced among children low sanitation coverage. By age 24 months, children with 100% sanitation coverage are 3.07 (95%CL 1.23-4.91) cm taller than those with 0% coverage. This translates into a HAZ difference of 1.02 (95%CL 0.43-1.61) z scores. This deficit decreases but remains in the fourth and fifth years of life.

#### *Height-for-Age Z Scores (HAZ)*

The lull in growth during the second year of life translates into a rapid decline in HAZ (Figure 1). Similar to the results in Table 3.2, the sanitation facilities of the household had a small effect on the HAZ of children (Table 3.3, Model 1). Children from households with improved sanitation had, on average, a height-for-age Z score 0.13 greater (95%CL 0.00-0.25) than children from households without improved sanitation. Adjusting for the child's age and sex, and the household's wealth and education had little effect on the coefficient for household sanitation (Table 3.3, Model 2). While the results of Table 3.2 showed that boys were approximately 0.75 cm taller than girls, the results of Table 3.3 show that boys have approximately 0.15 lower HAZ than girls.

After accounting for sanitation coverage and wealth at the neighborhood level, the effect of improved sanitation at the household is substantially attenuated (Table 3.3, Model 8). After controlling for the sanitation facility of the household and other household and child characteristics, children had a Z score that was 0.49 higher (95%CL 0.06-0.88) when 100% of their neighbors had improved sanitation compared to when 0% of the neighbors had improved sanitation. Similar to the results of Model 4, the interaction term in Model 9 suggests that the effect of sanitation coverage is not different between households with and without improved sanitation. Accounting for spatial correlation had little effect on the model estimates.

#### *Moderate and Severe Stunting*

Table 3.4 shows the results of mixed effects logistic regression for moderate and severe stunting. These results reinforce the findings from Tables 3.2 and 3.3. Children from households with improved sanitation had a slightly lower odds of being moderately stunted (OR 0.70, 95%CL 0.43-1.15). Sanitation coverage in the neighborhood reduced the odds of moderate stunting 5 fold (OR 0.21, 95%CL 0.05 - 0.84, Table 3.4, Model 11). The results for severe stunting are similar (OR 0.29, 95%CL 0.05-1.72), but are no longer statistically significant (Table 3.4, Model 12).

## **Discussion**

We have shown that sanitation provides substantial protection against childhood stunting. Improved sanitation at the household provided a small benefit, but it was eclipsed



by the much stronger effect of neighborhood sanitation coverage. Had we only accounted for household sanitation as many studies do, we would have drastically underestimated the overall benefit of sanitation. Also, this herd protective effect manifested during the second year of life, when a child's growth is most likely to falter (24), suggesting that sanitation can play an important role in prevention. While sanitation showed a strong protective effect, children with the optimal sanitation scenario were still stunted, suggesting the importance of other pathways such as breastfeeding and micronutrients.

Other studies have shown some evidence of herd protection from sanitation. Bутtenheim (25) followed 153 children in Bangladesh slums for 1 year for changes in weight-for-height, a short-term indicator of nutritional status. Improved sanitation at the household level did not have a statistically significant impact on weight-for-height, but there was a 0.1 z score increase for each 10 percentage point increase in neighborhood sanitation coverage. Using data from a cross-sectional household survey in Peru, Alderman et al (26) compared the HAZ for 2,084 children. They also saw no significant effect of a household's sanitation, but children from sample clusters with 100% sanitation coverage had 0.47 greater HAZ ( $p < 0.05$ ) than children from clusters with 0% coverage. Corsi et al (27) used data from the Demographic and Health Survey in Bangladesh, and compared both HAZ and weight-for-age among 5,731 children. They did not, however, disentangle the effects of water and sanitation, and the protective community effect of water and sanitation disappeared after adjusting for other community-level covariates. Using a much larger survey in rural India, Andres et al (28) observed an effect of both household sanitation and community sanitation on the prevalence of diarrhea.

Our study makes several key contributions to the literature. First, we employ a longitudinal study on a large sample of children allowing a more robust construction of growth curves. With the exception of Bütünheim (25), all other studies on this topic have been cross-sectional (26-28) or ecological in nature (29, 30). Second, we sampled all households in the villages along with the GPS location of each household. National household surveys use a multiple stage sampling design, where neighborhood sanitation coverage is calculated by the non-self mean of sanitation in the survey cluster. Because all households in a survey cluster are not sampled, the estimate of sanitation coverage is susceptible to random sampling error, which will bias the results the null. Also, surveys clusters may vary in size geographically, a problem that we addressed by defining neighborhoods with a 500 meter radius.

Because this is an observational study, it is susceptible to confounding. Just as households with improved sanitation are typically different in many ways than households with unimproved sanitation, communities with high sanitation coverage are different than those with low coverage. Many of these differences may also be risk factors for stunting. We have attempted to capture these differences by controlling for education, household wealth, and community wealth. Information on breastfeeding, handwashing, and food security was unavailable limiting our ability to draw inferences from our study. Other studies have adjusted for socioeconomic status, education, breastfeeding, food security, and handwashing, but none have adjusted for all simultaneously. Randomized trials, which have been useful in assessing the impact of household sanitation, may be impractical for assessing the effect of sanitation coverage.

In conclusion, we have shown that sanitation coverage at the neighborhood level has a stronger impact on child height than sanitation at the household level. As with other diseases and interventions, these externalities suggest that community context should not be ignored, for failure to do so will lead to an underestimate of the overall protective effect of sanitation. Future studies should investigate the causal link between sanitation coverage and child growth by incorporating symptomatic diarrhea, helminth infection, and environmental enteropathy.

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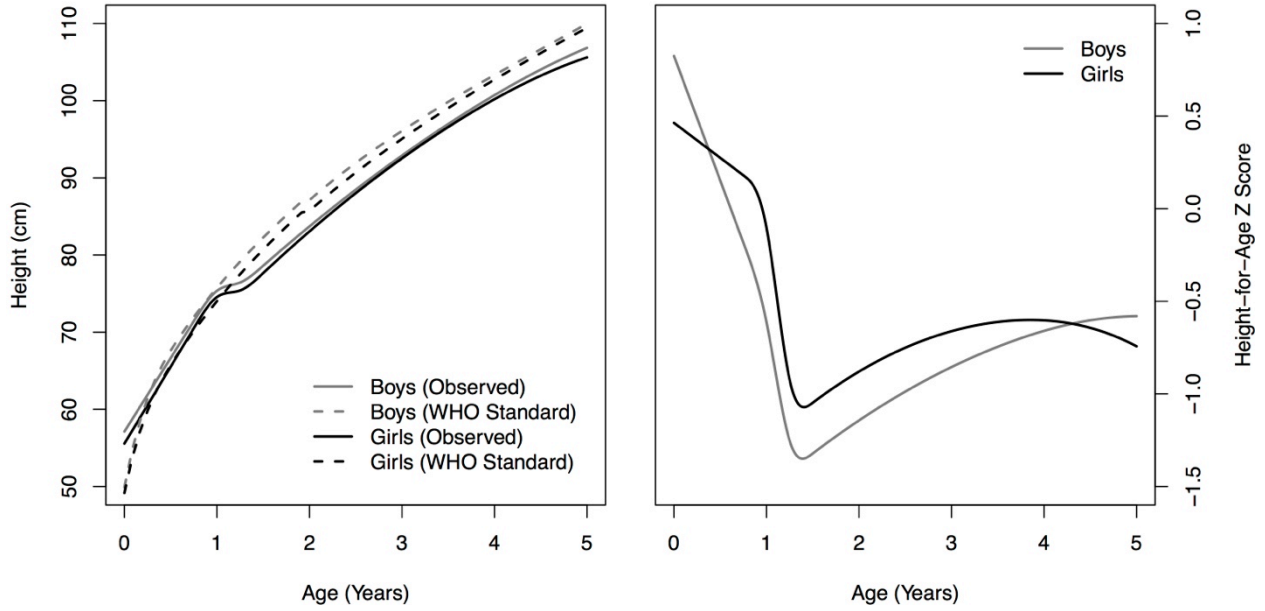
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**Table 3.1** Summary statistics of children < 5 years of age in 24 villages in coastal northern Ecuador, 2008-2011.

Village ID	Number of Children	Number of Observations	Height-for-Age Z Score <sup>1</sup>	Moderate Stunting <sup>1,2</sup>	Severe Stunting <sup>1,3</sup>	Improved Sanitation <sup>1,4</sup>	Wealth Index <sup>1</sup>	Years of Education <sup>1,5</sup>
3	85	136	-1.11	23.5%	9.6%	63.6%	0.65	6.5
4	130	198	-0.49	14.1%	3.5%	86.8%	1.51	9.1
5	177	291	-0.71	13.7%	4.5%	80.0%	1.36	8.4
7	13	30	-0.28	6.7%	3.3%	92.1%	1.90	6.7
8	30	52	-0.39	13.5%	1.9%	83.8%	0.76	9.5
9	66	127	-0.51	15.0%	10.2%	95.3%	1.95	7.9
10	32	48	-0.17	6.3%	2.1%	66.5%	0.56	7.0
11	39	68	-0.20	5.9%	4.4%	93.4%	1.78	6.5
13	23	44	-0.19	11.4%	4.5%	36.2%	0.12	7.2
15	23	44	-0.15	9.1%	2.3%	60.0%	0.28	6.6
16	17	26	-0.27	7.7%	0.0%	72.9%	-0.80	6.7
17	81	135	-0.48	8.9%	0.7%	84.5%	0.58	8.6
19	121	213	-0.43	6.6%	1.4%	64.8%	-0.63	8.6
20	32	60	-0.81	16.7%	1.7%	82.1%	-0.51	7.0
21	42	67	-1.05	16.4%	7.5%	94.5%	-1.36	9.2
24	105	192	-0.52	9.9%	4.2%	86.7%	-0.07	6.9
25	26	43	-0.29	2.3%	0.0%	75.0%	0.84	6.0
26	55	90	-1.43	33.3%	12.2%	30.2%	-0.31	6.6
27	62	96	-0.89	20.8%	7.3%	45.9%	0.32	7.3
28	66	93	-0.60	9.7%	1.1%	77.0%	1.36	7.6
29	78	140	-0.92	22.1%	5.7%	63.8%	1.01	8.1
30	7	12	-1.87	58.3%	8.3%	72.4%	-0.86	9.0
31	1	1	-0.94	0.0%	0.0%	40.2%	-4.26	12.0
32	9	19	-0.25	10.5%	5.3%	47.1%	-1.18	8.6
Total	1,314	2,225	-0.63	14.0%	4.6%	73.9%	0.61	7.8

<sup>1</sup>Mean or proportion of all observations<sup>2</sup>Height-for-age Z Score < -2<sup>3</sup>Height-for-age Z Score < -3<sup>4</sup>All households, not just those with children<sup>5</sup>Maximum of all persons in the household

**Figure 3.1** Mean height-for-age z-score growth trajectories, coastal northern Ecuador, 2008-2011. Predicted values were estimated from a mixed effect linear model with a restricted cubic spline for age, a random intercept for each child, and a random age slope for each child.





**Table 3.2** Predictors of height (in centimeters) among children < 5 years of age in rural northern Ecuador, 2008-2011. Coefficients (and standard errors) represent height differences in cm and were estimating using a mixed effects linear model. All models include restricted cubic spline terms for age, a random intercept for each child, and a random age slope for each child. Model 5 includes also accounts for exponential spatial covariance between observations.

	Model 1	Model 2	Model 3	Model 4	Model 5
Household Sanitation (Improved <sup>1</sup> vs Unimproved)	0.41 (0.22)	0.34 (0.22)	0.18 (0.23)	-0.51 (0.82)	-0.43 (0.84)
Sanitation Coverage <sup>2</sup>			1.68* (0.70)	1.05 (1.00)	0.96 (1.02)
Household Sanitation <sup>1</sup> X Sanitation Coverage <sup>2</sup>				1.01 (1.15)	1.03 (1.18)
Neighborhood Wealth Index <sup>3</sup>			-0.16 (0.13)	-0.16 (0.13)	-0.17 (0.14)
Male Child vs. Female Child		0.75*** (0.23)	0.75*** (0.23)	0.75*** (0.23)	0.70** (0.23)
Years of Education (Maximum of the Household)		0.04 (0.03)	0.03 (0.03)	0.03 (0.03)	0.06 (0.03)
Household Wealth Quintile <sup>4</sup>					
2 vs 1		0.42 (0.34)	0.44 (0.35)	0.45 (0.35)	0.46 (0.36)
3 vs 1		0.52 (0.36)	0.56 (0.37)	0.57 (0.37)	0.60 (0.38)
4 vs 1		1.06** (0.37)	1.09** (0.39)	1.09** (0.39)	1.14** (0.40)
5 vs 1		1.11** (0.39)	1.16** (0.42)	1.17** (0.42)	1.09* (0.43)
Intercept	55.94	54.69	53.60	53.99	53.65
Range Parameter (meters)	-	-	-	-	0.02
Observations	2,225	2,225	2,225	2,225	2,225
Number of Children	1,314	1,314	1,314	1,314	1,314
AIC	12,613	12,606	12,606	12,605	12,685

Standard errors in parentheses. \*\* p<0.01, \* p<0.05

<sup>1</sup> Defined as JMP Improved, but ignoring sharing.

<sup>2</sup> Defined as the proportion of households within a 500-meter radius that have improved sanitation.

<sup>3</sup> Defined as the mean wealth index of households within a 500-meter radius.

<sup>4</sup> 5 is the wealthiest quintile, 1 is the poorest.

**Table 3.3** Predictors of height-for-age z scores among children < 5 years of age in rural northern Ecuador, 2008-2011. Coefficients (and standard errors) were estimating using a mixed effects linear model. All models include restricted cubic spline terms for age, a random intercept for each child, and a random age slope for each child. Model 5 includes also accounts for exponential spatial covariance between observations.

	<b>Model 6</b>	<b>Model 7</b>	<b>Model 8</b>	<b>Model 9</b>	<b>Model 10</b>
Household Sanitation (Improved <sup>1</sup> vs Unimproved)	0.13 (0.06)	0.10 (0.07)	0.05 (0.07)	-0.28 (0.24)	-0.20 (0.25)
Sanitation Coverage <sup>2</sup>			0.47* (0.21)	0.17 (0.30)	0.31 (0.30)
Household Sanitation <sup>1</sup> X Sanitation Coverage <sup>2</sup>				0.49 (0.34)	0.36 (0.35)
Neighborhood Wealth Index <sup>3</sup>			-0.05 (0.04)	-0.05 (0.04)	-0.06 (0.04)
Male Child vs. Female Child		-0.15* (0.07)	-0.15* (0.07)	-0.15* (0.07)	-0.17* (0.07)
Years of Education (Maximum of the Household)		0.02 (0.01)	0.01 (0.01)	0.01 (0.01)	0.01 (0.01)
Household Wealth Quintile <sup>4</sup>					
2 vs 1		0.13 (0.10)	0.13 (0.10)	0.14 (0.10)	0.12 (0.11)
3 vs 1		0.14 (0.11)	0.16 (0.11)	0.17 (0.11)	0.16 (0.11)
4 vs 1		0.29** (0.11)	0.30* (0.12)	0.30** (0.12)	0.29* (0.12)
5 vs 1		0.31** (0.12)	0.33** (0.13)	0.34** (0.13)	0.33* (0.13)
Intercept	0.59	0.38	0.07	0.26	0.19
Range Parameter (meters)	-	-	-	-	0.002
Observations	2,225	2,225	2,225	2,225	2,225
Number of Children	1,314	1,314	1,314	1,314	1,314
AIC	7,210	7,225	7,230	7,230	7,308

Standard errors in parentheses. \*\* p<0.01, \* p<0.05

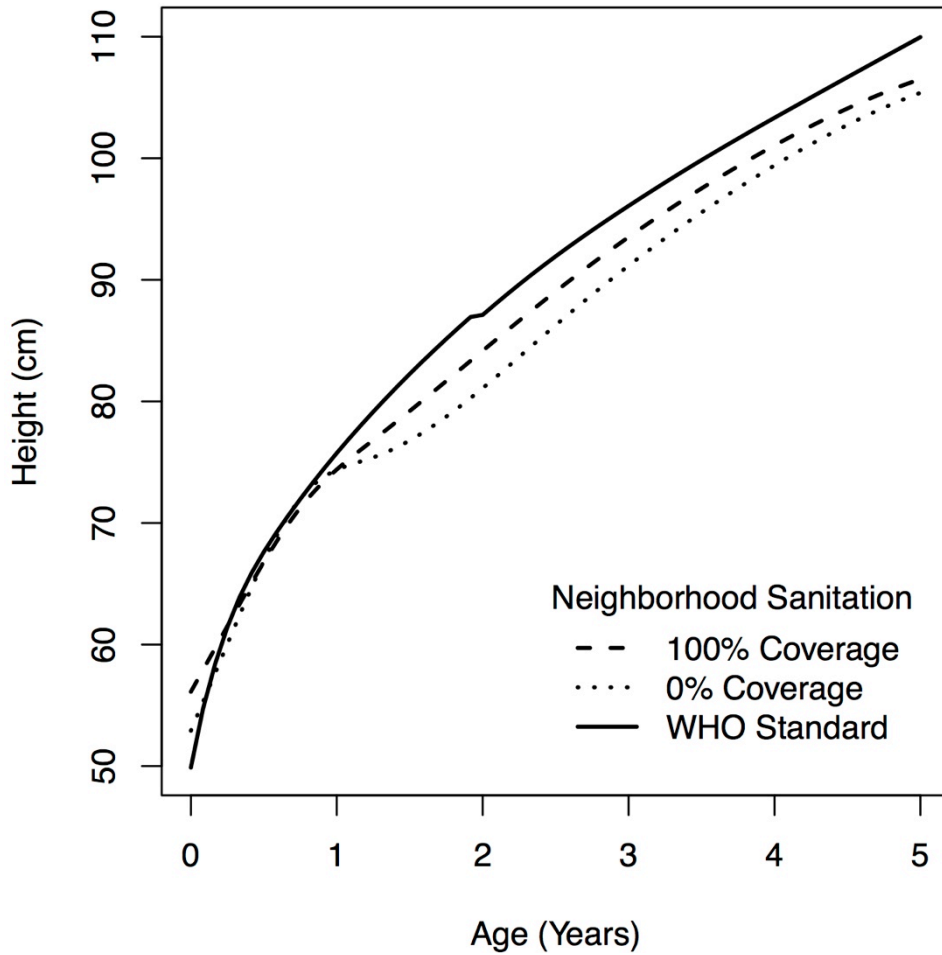
<sup>1</sup> Defined as JMP Improved, but ignoring sharing.

<sup>2</sup> Defined as the proportion of households within a 500-meter radius that have improved sanitation.

<sup>3</sup> Defined as the mean wealth index of households within a 500-meter radius.

<sup>4</sup> 5 is the wealthiest quintile, 1 is the poorest.

**Figure 3.2** Predicted height in CM among boys by level of sanitation in the surrounding households, northern Ecuador, 2008-2011. Adjusted for household sanitation, household education, household wealth, neighborhood wealth. Age was modeled using a restricted cubic spline with knots at 0.5, 0.75, 1.25, 2.5, and 4 years.



**Table 3.4** Odds ratios and 95% Confidence Limits for moderate and severe stunting among children < 5 years of age in rural northern Ecuador, 2008-2011. All models include a random intercept for each child.

	<b>Model 11</b>	<b>Model 12</b>
	<b>Moderate Stunting</b>	<b>Severe Stunting</b>
Household Sanitation (Improved <sup>1</sup> vs Unimproved)	0.70 (0.43-1.15)	0.65 (0.33-1.27)
Sanitation Coverage <sup>2</sup>	0.21 (0.05-0.84)	0.29 (0.05-1.72)
Neighborhood Wealth Index <sup>3</sup>	1.16 (0.89-1.53)	1.46 (1.01-2.11)
Years of Education (Maximum of the Household)	1.01 (0.94-1.08)	1.03 (0.94-1.12)
Household Wealth Quintile <sup>4</sup>		
2 vs. 1	1.45 (0.65-3.27)	1.16 (0.39-3.47)
3 vs. 1	0.93 (0.40-2.15)	0.55 (0.17-1.73)
4 vs. 1	0.69 (0.29-1.68)	0.56 (0.17-1.88)
5 vs. 1	0.80 (0.31-2.06)	0.68 (0.19-2.35)
Male Child vs. Female Child	2.05 (1.29-3.26)	1.67 (0.93-3.00)
Age of Child in Years		
1 vs. 0	8.03 (4.07-15.86)	2.93 (1.23-6.96)
2 vs. 0	4.86 (2.49-9.50)	2.23 (0.92-5.40)
3 vs. 0	1.98 (1.01-3.86)	1.42 (0.57-3.53)
4 vs. 0	1.68 (0.81-3.48)	1.43 (0.53-3.86)

<sup>1</sup> Defined as JMP Improved, but ignoring sharing.

<sup>2</sup> Defined as the proportion of households within a 500-meter radius that have improved sanitation.

<sup>3</sup> Defined as the mean wealth index of households within a 500-meter radius.

<sup>4</sup> 5 is the wealthiest quintile, 1 is the poorest.

## Chapter IV

The association between shared sanitation and diarrheal disease among children in 51 countries

### Abstract

Shared sanitation is defined as “unimproved” due to concerns that it creates unsanitary conditions; this policy is being reconsidered by the World Health Organization and UNICEF. We assessed whether sharing a toilet facility was associated with an increased prevalence of diarrhea among children < 5 years of age. We use data from Demographic and Health Surveys conducted in 51 countries. Crude and adjusted prevalence ratios (PR) for diarrhea, comparing children from households that used a shared facility to children from households that used a non-shared facility, were estimated for each country and pooled across countries. The unadjusted PR varied across countries, ranging from a 2.15 to 0.65. The pooled PR was 1.09 (95% confidence limit 1.06-1.12); differences in socioeconomic status explained approximately one half of this elevated prevalence (adjusted PR=1.05, 95% confidence limit 1.02-1.08). Shared sanitation appears to be a risk factor for diarrhea though differences in socioeconomic status are important. The heterogeneity across countries, however, suggests that the social and economic context matters.

## **Introduction**

Diarrheal disease is a major cause of morbidity and mortality, particularly in low- and middle-income countries (1). Inadequate sanitation, water, and hygiene are the most significant risk factors for diarrheal disease, responsible for an estimated 1.9 million deaths worldwide (2). Since the adoption of the Millennium Development Goals (MDGs), access to improved sanitation has increased around the globe. However, approximately 37% of the world's population (2.5 billion people) still lacks access to improved sanitation (3). This includes an estimated 761 million people who rely on public or other "shared" sanitation facilities.

In order to track changes in water and sanitation, including progress towards international targets such as the MDGs, WHO and UNICEF created the Joint Monitoring Programme (JMP) for Water Supply and Sanitation. Apart from monitoring, the JMP was tasked with creating a uniform definition of 'improved' and 'unimproved' sanitation to be used across countries. JMP's definition of 'improved' sanitation currently includes flush or pour-flush toilets, pit latrines with a slab, ventilated improved pit latrines, and composting toilets, while 'unimproved' sanitation includes open defecation, pit latrines without a slab, buckets, hanging toilets or latrines, or a flush/pour flush toilet that flushes to an unsanitary destination (3). Due to concerns about cleanliness and accessibility, facilities that are shared by 2 or more households are classified as 'unimproved,' regardless of the level of technology used (3).

Recently, the JMP's Task Force on Sanitation proposed a change in this policy that would allow sanitation facilities to be considered as "improved"-and therefore scored toward the MDG and other international sanitation targets-provided they meet the other

criteria and are shared by no more than 5 households or 30 persons, whichever is fewer (4). In 2010, an estimated 11% of the world's population used a shared facility that would otherwise be considered improved, and that percentage is rising (3). There is relatively little evidence, however, on whether and at what circumstances sharing sanitation facilities actually poses a health risk to those that use them. Also, public latrines are considered by some to be the only viable option in many urban slums (3, 5-7).

Due to this trend towards shared sanitation, more empirical data are needed to determine whether such facilities increase the risk of disease, and if so, to quantify that risk, identify the causal pathway and explore ways of mitigating it. A recent systematic review reported that shared sanitation may be a risk factor for diarrhea and other adverse health outcomes when compared to individual household latrines (8). The review identified 8 studies (2 cross-sectional and 6 case control); shared sanitation was the focus in only two of these studies (9, 10), while the others simply reported statistical associations with little to no mention of potential mechanisms. While these studies report an association between shared sanitation and diarrhea, the review noted substantial deficiencies in the methodological quality of most studies, including the failure to account for some potential sources of confounding, unclear comparisons, and failure to distinguish between different types of sanitation technology and ownership.

The objective of our study is to determine whether the prevalence of diarrhea is higher among those that share a toilet facility compared to those that use a facility that is not shared. We use data from 51 low- and middle-income countries that represents much of the developing world. We also define 'shared sanitation' 3 different ways. Finally, we

rigorously assess the extent to which confounding plays a role in the association between sharing and diarrhea.

## **Methods**

We use data from the Demographic and Health Surveys ([measuredhs.com](http://measuredhs.com)) completed between 2001 and 2011. Surveys completed before 2001 were excluded. As a result, our findings will better reflect current circumstances and be more able to inform an ongoing policy debate. In order to achieve a representative sample at the subnational level, these cross-sectional surveys use a 2-stage stratified random sample of households. Countries are divided into enumeration areas (clusters), and then households are randomly selected within each cluster with different probability of selection within different clusters. The surveys ask a variety of questions about demographics, reproductive health, and child health. For countries that had multiple surveys in this time period, we use only the most recent one to prevent the overrepresentation of single countries. We selected the 51 recent surveys from low- and middle-income countries that included data on disease outcome, exposure, and potential confounders (Table 4.1).

For any children < 5 years of age in the household, the caretaker reported whether said child had diarrhea in the past 2 weeks. The surveys do not use a specific case definition. Each caretaker also reported the type of toilet facility that the household uses. For each survey, we classified each potential response as being improved or unimproved based on the definitions provided by the JMP but ignoring sharing (3). Responses considered to be improved were then further classified based on whether or not the facility utilized flush technology, yielding 3 categories: unimproved facility, improved latrine, and



improved flush or pour-flush toilet. Caretakers then reported whether or not their facility was shared by other households. We used this information to create 3 different measures of sharing. First, a binary definition of sharing was used, where a toilet facility was classified as shared if more than 1 household used it. Those with no facility were excluded. We then accounted for the number of households that share the facility, creating three exposure categories: 1) facilities that are not shared, 2) facilities shared by 1 to 5 households, and 3) facilities shared by 6 or more households. Again, those with no facility were excluded. The data describing the number of households sharing, however, was only available in 40 of the 51 surveys (Table 4.1). Finally, we use the JMP's sanitation ladder, made up of 4 categories: 1) no facility, 2) unimproved facility, 3) shared but otherwise improved facility, and 4) improved facility that is not shared. Log-binomial regression, accounting for the complex sampling strategy, was used to generate the unadjusted (crude) and adjusted prevalence ratios (PR) and 95% confidence intervals (CI) for diarrhea. The prevalence ratios represent the relative difference in diarrhea prevalence among children from households with a shared facility compared to children from households with a facility that is not shared.

Households that use a shared sanitation facility are likely different in many respects than households that have their own facility. To account for these differences, we made a list of potential confounding variables to include in the analysis. Characteristics of the household assessed were type of sanitation facility (unimproved, improved latrine, improved flush or pour-flush toilet; improved being defined by JMP, but ignoring sharing), improved water source (as defined by JMP), household ownership of assets (electricity, radio, television, refrigerator, bicycle, motorcycle/scooter, car/truck, improved cooking

fuel, and improved floor surface), urban/rural residence, the mother's age, the mother's educational attainment, the highest level of education in the household, the number of children < 5 years of age in the household. Characteristics of the child assessed were age, gender, vaccination status, and whether the child had a health card. The DHS includes many more variables, but we selected this group because each captures a different aspect of socioeconomic status. We chose this list of confounders a priori and analyzed each of them individually and in groups to assess their impact on the prevalence ratio(s) for shared sanitation and diarrhea. For the sake of parsimony, we only included variables that made a substantial impact on the PR in our final model, namely the type of sanitation facility (unimproved facility, improved latrine, or improved flush toilet), mother's age and education, the highest level of education in the household, and household ownership of assets.

We conducted both country-specific and pooled analyses. In the pooled analyses, surveys were combined by the WHO-defined regions of the world (Africa, Latin America and the Caribbean, South-East Asia, Western Pacific, Eastern Mediterranean and Europe), and dummy variables for each survey were included. Because of geographic proximity and the small number of countries in the Western Pacific region, South-East Asia and Western Pacific were combined as a single region. Because they contained relatively fewer countries, the Eastern Mediterranean and Europe regions were also combined. This resulted in 4 distinct regions. We analyzed the data stratified by region to detect any regional patterns or differences. We also conducted an overall pooled analysis by using data from all 51 surveys along with survey fixed effects.

All data management and analysis was conducted using STATA 11.2 (StataCorp, College Station, TX).

## **Results**

There were 435,205 children under the age of 5 included in the analysis (Table 4.1). Of these, 30.9% were from households with no sanitation facility. Of children from households with a facility, 45.1% were from households with a facility that was improved (ignoring sharing), and 29.9% were from households that used a shared facility. The amount of sharing varied substantially across countries. The lowest level of sharing was in Armenia (1.4% of those with a facility) and the highest was in Ghana (87.3%). When all 51 surveys were combined, the overall prevalence of diarrhea was 14.3%. Diarrhea prevalence varied substantially across countries, from 4.5% in Maldives to 26.2% in Bolivia.

In the majority of countries, the prevalence of diarrhea was higher among households that used a shared toilet facility (Figure 1). This effect of sharing, however, varied across countries. The point estimates of the unadjusted PRs ranged from 0.65 (Nigeria) to 2.15 (Moldova), though only 16 of the 51 unadjusted PRs were statistically significantly different from 1. After adjusting for confounders, many of the point estimates moved towards the null, but some did not. The adjusted PRs ranged from 0.80 (Armenia) to 2.04 (Moldova). There was an apparent clustering of countries in West Africa that showed protective effects, particularly Nigeria, Cameroon, Mali, Senegal, and Liberia. To highlight this geographic pattern, in Table 4.2 we present the Africa and global estimates with and without West Africa.

We observed 9% higher prevalence among households that used a shared toilet facility (Crude PR=1.09, 95%CI: 1.06-1.12) when pooling the data across all 51 countries (Table 4.2). In absolute terms, this represents a prevalence difference of 1.2 (95%CL: 0.8-1.6) percentage points. Adjusting for confounding attenuated the effect (Adjusted PR=1.05, 95%CI: 1.02-1.08). This relationship is consistent across 3 of the 4 regions. Only the Latin America and Caribbean region differed, where adjusting for confounding eliminated the effect. In the Eastern Mediterranean and Europe region we observed the largest harmful effect (Adjusted PR=1.20, 95%CI: 1.06-1.36). The level of attenuation after adjustment for confounding differed slightly by region. The estimates did not appear to differ when stratified by urban and rural areas (data not shown).

As mentioned above, there exists substantial heterogeneity among countries within each region (Figure 1). This heterogeneity is best illustrated in the African region (Figure 1 and Table 4.3). The pooled prevalence ratio for a number of countries within Africa are either protective (Nigeria, Cameroon, Mali, Senegal, and Liberia: Adjusted PR=0.86, 95%CI: 0.80-0.93) or exhibit no effect (Sao Tome and Principe, Namibia, Congo, Burkina Faso, and Burundi). In the remaining subsets of African countries, those that use a shared toilet have a 10-32% higher prevalence of diarrhea than those that do not share (Table 4.3). The African countries that exhibited a protective effect are all located in West Africa. The patterns within other regions of the world appear similar. In Europe, there was a large degree of heterogeneity both between and within countries, possibly attributable to small sample size.

The second way in which we examine the impact of sharing on prevalence is by stratifying exposure by those that share with 5 or fewer households and those that share

with 6 or more households. These data were available for only 40 of the 51 surveys (Table 4.1). Except for Africa, the regional estimates were not statistically significant after adjustment for confounders (Table 4.4). Each sharing category had an elevated prevalence compared to the not shared reference group, but the prevalence of diarrhea was not statistically different when comparing a facility that is shared with 1 to 5 households to a facility that is shared with 6 or more. Only in the South-East Asia, Western Pacific, Eastern Mediterranean and Europe did there appear to be a dose-response relationship. In other regions, the prevalence of diarrhea did not differ based on the number of households sharing. Therefore, the stratified data provide little evidence for a dose response relationship and no support for a threshold of households for which sharing does not present an increased risk of diarrhea.

The JMP's sanitation ladder is another useful way to examine the impact of sharing on prevalence. By using this classification, households that share sanitation facilities that are otherwise improved can be compared to those that use improved facilities that are not shared. When all 51 surveys are pooled, sharing appeared to be harmful even when the facility is improved (Table 4.5). The prevalence of diarrhea was 10% lower among households that used a non-shared improved facility compared to facilities that were shared but otherwise improved (Crude PR=0.90, 95%CI: 0.87-0.93). Adjusting for confounding modestly attenuated that effect (Adjusted PR=0.95, 95%CI: 0.91-0.99). The strongest effect observed was in Eastern Mediterranean and Europe (Adjusted PR=0.83, 95%CI: 0.72-0.94) and Africa when West Africa is excluded, (Adjusted PR=0.81, 95%CI: 0.75-0.87). In Latin America and the Caribbean and South-East Asia and Western Pacific the adjusted effect was not significant.

The results are less consistent when comparing sharing (otherwise improved) with either no facility or unimproved facility (either shared or not shared) (Table 4.5). Whereas Eastern Mediterranean and Europe exhibited a protective effect for both no facility and unimproved facility (Adjusted PR=0.81, 95%CI: 0.69-0.94 and Adjusted PR=0.75, 95%CI: 0.63-0.89 respectively), the other regions either did not have significant results (South-East Asia and Western Pacific), was protective in one category (Africa), or was harmful in one category (Latin America and the Caribbean).

## **Discussion**

Our global pooled analysis shows that there was an increased prevalence of diarrhea associated with shared sanitation. This is generally in agreement with the few studies that have been done (9-16), though the effect we observe is more modest and is attenuated after adjusting for confounding. However, we also report a high level of between-country heterogeneity, which limits the ability to make inferences from our pooled estimates or from the pooled estimates from previous studies.

One strength of our study is the ability to look at differences across a wide array of countries. In the majority of countries, sharing appears to be harmful. However, In Nigeria and Cameroon, sharing actually appears to be quite protective, and in many other countries there was no difference in diarrhea prevalence attributable to sharing. These findings are consistent with the recent systematic review that found sharing latrines to be associated with increased risk (though not always significant) of diarrhea in 10 countries but protective in 1 (Bangladesh) (8). Other research has shown substantial differences among countries in the effectiveness of water, sanitation and hygiene interventions to prevent

disease (17). Such variability between countries, and possibly within countries, makes a single, uniform, global policy particularly difficult. Future research is needed to elucidate the circumstances under which sharing is more harmful.

Confounding appears to play an important role in the relationship between shared sanitation and diarrheal disease. Country-specific and pooled prevalence ratios were substantially attenuated when socioeconomic indicators were included in the models. Because households that share are generally of a lower socioeconomic status than those that do not share, they are at increased risk of diarrhea due to poverty in general, not necessarily because of sharing (18). These lower-income households are more likely to have inadequate hygiene practices and consume contaminated food. The type of toilet facility (unimproved latrine, improved latrine, or flush toilet) also explained some of the observed association between shared sanitation and diarrhea but was less important than the socioeconomic variables. In this dataset, shared facilities were less likely to be improved than non-shared facilities, and less likely to use flush technology if improved. The results of Table 4.5, which directly account for type of facility, show similar levels of elevated prevalence associated with sharing. Although confounding explains some of the observed difference it does not explain all of the differences. Furthermore, the importance of confounding varied across regions, greater in South-East Asia and Western Pacific as well as in the Americas than in Africa, the Eastern Mediterranean, and Europe.

In many countries, the adverse effect of sharing was strong even after adjusting for confounding. For example, in Madagascar the prevalence of diarrhea was 44% higher (95%CL: 12%-86%) among those with shared facilities compared to those with facilities that are not shared, after controlling for socioeconomic variables. In such settings, shared

toilets may contribute to the transmission of diarrheal disease. Further research is necessary to substantiate these findings, evaluating whether and to what extent shared sanitation actually increases the risk of disease. Stronger study designs using incidence of diarrhea will allow for more robust causal inference in this regard. It is also important to identify the mechanism of transmission and how this can be mitigated. Transmission could be occurring because shared facilities, particularly those that are communally owned, may be more difficult to clean and maintain. Often, some type of institution is required to keep the public facility in good operating condition (5-7, 19-21). When such institutions are insufficient or lacking, the quality of the facility suffers. Also, shared facilities of all types may be overused and increase the amount of epidemiologic contact between users. Other than cleanliness, people may periodically choose to practice open defecation, or some other less hygienic means of excrement disposal, when shared facilities are deemed unsafe or inconvenient to due to distance or long lines. Shared latrines may also fill up more rapidly and require more frequent emptying, which raises additional concerns about unsafe sludge management, creating another source of exposure.

In some countries, sharing appears to be protective, a seemingly counterintuitive result. The protective effect was particularly strong in Nigeria, where the prevalence ratio was substantially protective even after adjusting for confounding. Cameroon also initially showed a protective effect, but it was substantially attenuated after adjusting for confounders. Other countries, namely Mali, Senegal, and Liberia, showed a modest protective effect. Interestingly, these countries are clustered in West Africa, while countries in Sub-Saharan Africa generally show benign to harmful effects. Further research is necessary to confirm the validity of this protective effect and, if so, the reasons therefor.



The nature of shared sanitation is often quite different between rural and urban areas.<sup>3</sup> Sharing in rural areas is often characterized by sharing with a few neighbors or relatives. In urban areas, particularly in urban slums, many of the shared facilities may be public and used by a large number of households. Unfortunately, the DHS data does not allow for enough geographic resolution to differentiate between urban slums and other urban areas, which may explain why we did not detect a difference in the effect of shared sanitation between urban and rural areas.

Our study design has other limitations. It is well documented that using a 2-week recall period understates disease status, resulting in bias. Some studies (22-24) have suggested that a 2- or 3-day recall period will minimize this bias, but Arnold et al. (25) argue that 1 week is optimal when accounting for both bias and variance. While the 2-week recall period used in the DHS is not ideal, any bias in our results should be towards the null, as long as disease misclassification is unrelated to exposure. Second, like any cross-sectional study, causal inference is limited. Reverse causation seems unlikely in this situation, but we cannot rule out residual confounding. However, the DHS collect many potential confounding variables that we were able to use in these analyses. In particular, we were able to examine how much of the potential increase in harmful effect measured in the analysis was due to confounding by socioeconomic status and how much was likely due to an actual increase risk when sharing sanitation. Additional information on handwashing, hygiene practices and food contamination would enhance these analyses. Also, diarrheal diseases are often seasonal. Cross-sectional studies are unable to detect seasonal trends. Even so, in order for season to be a confounder, it would need to be associated with the exposure (sharing) not just the outcome. Additionally, the DHS relies on self-reporting of

shared sanitation. A compound may be made up of several households of the same family sharing the same facility. In such situations, sharing (and the number of households sharing) may be underreported.

These results provide additional evidence that shared sanitation is generally a risk factor for diarrhea among children. As a result, our results provide support for the existing policy of the JMP to treat shared sanitation as “unimproved”. However, our results also provide no evidence of a minimum threshold of households that can share a latrine without increasing the risk. Thus, our findings provide no support for the proposed change in the JMP policy that would encourage sharing of latrines by treating latrines shared among 5 or fewer households as “improved”.

At the same time, there are settings where the relationship is neutral, and in a few it appears to be protective. This heterogeneity among countries suggests that the specific social and economic context matters. As the number of shared latrines is large and likely to increase, particularly in urban settings, it is important to ascertain under what circumstances sharing can be undertaken safely. Also, because the overall increase in prevalence is modest, shared sanitation could potentially be a low cost intervention. While shared facilities are clearly not optimal, for the same cost, higher coverage rates could be achieved with shared sanitation compared to private facilities. The higher coverage rates achieved could offset any losses to effectiveness.

One clear conclusion from this analysis is that confounding likely plays an important role in the association between sharing and diarrhea. Adjusting for socioeconomic status attenuates the estimated harmful effect of sharing, suggesting that alternative transmission pathways accounts for some of the differences. However, adjusting for socioeconomic

status does not account for all of the differences observed, suggesting that shared sanitation may contribute to the transmission of diarrheal diseases due to issues of cleanliness and maintenance, overuse, or due to users occasionally opting for less hygienic means of excreta disposal. Future research should attempt to identify the circumstances that make sharing harmful or protective, better understand confounding and its role, and seek to elucidate the mechanism through which sharing could increase the risk of diarrhea. This information will be crucial to help inform policy decisions.

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**Table 4.1.** Summary statistics of children < 5 years of age, 51 Demographic and Health Surveys, 2001-2011.

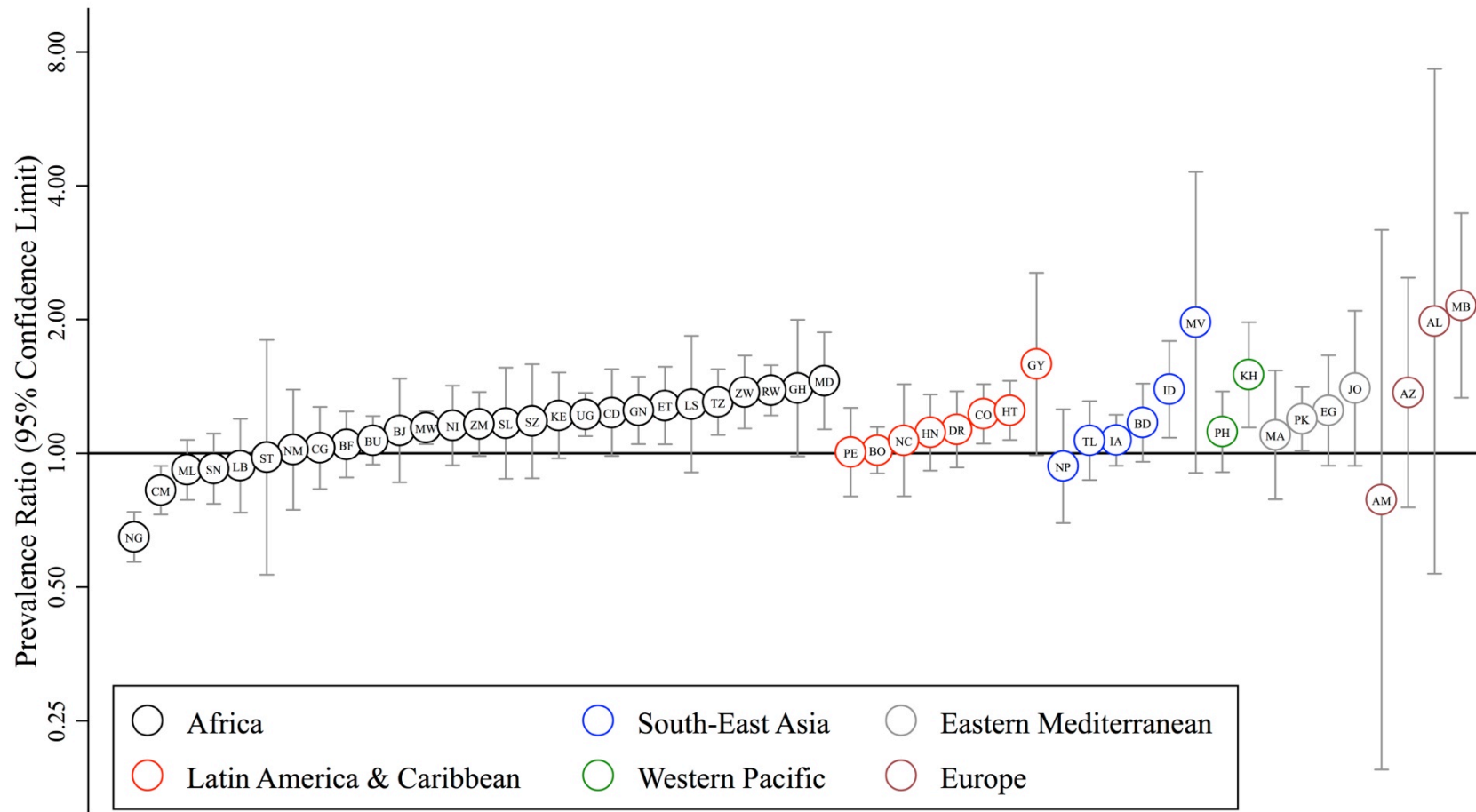
Country (year)	Sample Size (n)	Prevalence of Diarrhea (%)	No Toilet Facility (%)	Improved Toilet Facility (%) <sup>a</sup>	Shared Toilet Facility (%) <sup>b</sup>	Shared with >5 Households (%) <sup>c</sup>
<b>All Countries</b>	435,205	14.3	30.9	45.1	29.9	22.2
<b>Africa</b>	220,000	15.4	32.1	31.5	41.8	24.2
Benin (2006)	14,270	9.2	68.6	18.1	69.5	56.1
Burkina Faso (2010)	13,487	14.9	68.1	25.1	51.0	14.0
Burundi (2011)	7,147	25.2	3.0	40.0	15.9	9.0
Cameroon (2011)	9,932	21.8	8.4	53.9	29.4	18.9
Congo (Brazzaville) (2005)	4,047	14.1	11.9	17.1	60.6	-
Congo Democratic Republic (2007)	7,678	16.5	11.6	37.2	55.3	-
Ethiopia (2003)	10,441	13.6	43.1	12.6	27.7	19.0
Ghana (2008)	2,733	20.1	27.6	60.5	87.3	80.8
Guinea (2005)	5,316	16.4	30.1	25.4	60.8	-
Kenya (2009)	5,533	16.8	18.1	39.9	49.4	29.0
Lesotho (2010)	3,322	11.4	41.8	31.7	36.2	34.2
Liberia (2007)	4,930	20.8	59.8	23.5	76.0	64.3
Madagascar (2009)	11,444	8.4	49.9	3.9	63.8	15.2
Malawi (2010)	17,966	17.6	11.1	11.8	42.8	7.0
Mali (2006)	12,070	13.6	19.8	20.4	45.0	3.2
Namibia (2007)	4,238	13.4	58.1	37.8	25.2	36.5
Niger (2006)	7,922	21.3	80.5	8.8	39.3	41.9
Nigeria (2008)	24,733	10.4	30.7	51.8	40.2	41.8
Rwanda (2011)	8,330	13.1	1.3	72.9	19.7	5.4
Sao Tome and Principe (2009)	1,807	15.9	62.1	37.7	20.3	34.4
Senegal (2011)	11,060	21.1	19.1	55.8	24.3	8.6
Sierra Leone (2008)	4,783	13.6	23.9	39.7	77.6	35.1
Swaziland (2007)	2,325	14.3	22.0	28.7	33.6	30.5
Tanzania (2010)	6,995	14.9	18.9	14.8	30.6	13.5
Uganda (2011)	7,015	24.1	11.1	29.8	39.7	23.3
Zambia (2007)	5,582	15.8	27.4	30.8	40.1	9.9
Zimbabwe (2011)	4,894	13.6	32.2	57.1	47.5	17.8
<b>Latin America and the Caribbean</b>	75,910	16.1	18.0	66.1	17.9	8.5
Bolivia (2008)	8,135	26.2	32.5	37.9	33.7	10.3
Colombia (2010)	17,220	12.7	8.4	85.1	13.3	-
Dominican Republic (2007)	10,285	14.8	5.8	90.2	18.0	-
Guyana (2009)	2,027	10.1	1.4	87.8	13.6	3.6
Haiti (2006)	5,358	24.4	41.1	24.7	49.7	12.8
Honduras (2006)	10,198	16.0	21.9	58.5	15.2	2.0
Nicaragua (2001)	6,536	13.0	22.2	27.9	8.6	-
Peru (2008)	16,151	13.8	17.9	78.9	13.7	7.7
<b>South-East Asia</b>	85,276	10.7	46.5	43.2	25.1	22.0
Bangladesh (2007)	5,201	10.1	8.6	37.6	45.1	11.2
India (2006)	45,144	8.9	62.3	34.3	32.5	25.2
Indonesia (2007)	17,292	13.8	25.1	56.0	14.1	45.0
Maldives (2009)	3,678	4.5	0.7	96.9	2.1	22.6
Nepal (2011)	4,754	13.9	48.8	43.6	30.9	6.9
Timor-Leste (2010)	9,207	15.6	37.5	50.5	16.9	4.5
<b>Western Pacific</b>	13,837	12.4	40.3	56.2	24.5	7.5
Cambodia (2011)	7,670	15.0	61.6	36.1	19.7	7.5
Philippines (2008)	6,167	9.1	12.9	82.0	27.3	-
<b>Eastern Mediterranean</b>	33,605	14.4	11.7	83.6	7.0	1.6
Egypt (2008)	9,992	8.4	0.4	99.5	5.7	1.6
Jordan (2007)	9,791	15.9	0.0	99.5	3.1	-
Morocco (2004)	5,746	11.9	20.8	78.6	7.9	-
Pakistan (2007)	8,076	21.6	32.6	49.3	15.5	-
<b>Europe</b>	6,577	8.2	0.0	84.2	6.3	20.4
Albania (2009)	1,562	5.4	0.0	93.8	2.2	0.0
Armenia (2010)	1,433	8.7	0.0	77.9	1.4	9.2
Azerbaijan (2006)	2,116	10.7	0.1	80.7	10.0	24.6
Moldova (2005)	1,466	7.1	0.0	85.3	9.6	-

<sup>a</sup>Based on the JMP categorization, but ignoring sharing.

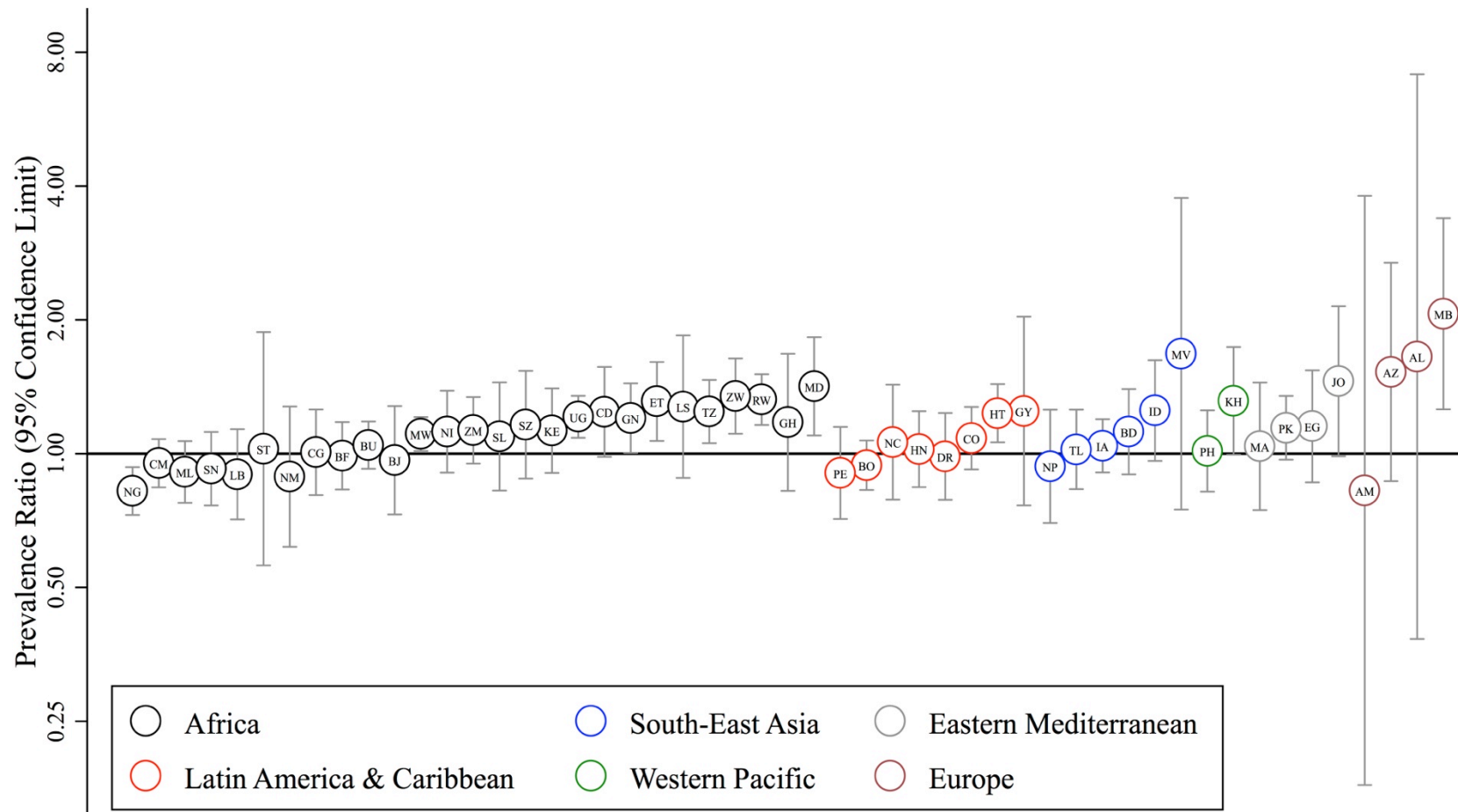
<sup>b</sup>Among households that have a sanitation facility.

<sup>c</sup>Among households with a shared sanitation facility. '-' indicates data not collected.

**Figure 4.1.** Crude prevalence ratios (and 95% confidence intervals) for diarrhea, comparing those with shared toilet facilities to those with non-shared facilities. Presented for each Demographic and Health Survey (n=51) conducted between 2001 and 2011.



**Figure 4.2.** Adjusted<sup>a</sup> Prevalence Ratios (and 95% confidence intervals) for diarrhea, comparing those with shared toilet facilities to those with non-shared facilities. Presented for each Demographic and Health Survey (n=51) conducted between 2001 and 2011.



<sup>a</sup>Adjusted for type of facility (flush toilet, 'improved' latrine, 'unimproved' latrine) mother's age and education, highest level of education in the household, and ownership of assets.



**Table 4.2.** The effect of shared sanitation pooled across countries. Prevalence ratios and 95% confidence intervals for diarrhea, comparing households with shared toilet facilities to households with facilities that are not shared. Data from 51 Demographic and Health Surveys, 2001-2011.

<b>Region and Subset of Countries</b>	<b>Crude PR (95% CI)</b>	<b>Adjusted<sup>a</sup> PR (95% CI)</b>
Africa	1.07 (1.03-1.10)	1.05 (1.01-1.09)
West Africa <sup>b</sup>	0.89 (0.84-0.94)	0.91 (0.86-0.97)
Excluding West Africa <sup>b</sup>	1.19 (1.14-1.25)	1.15 (1.11-1.21)
Latin America and the Caribbean	1.11 (1.04-1.19)	1.03 (0.97-1.10)
South-East Asia and Western Pacific	1.16 (1.06-1.26)	1.09 (1.01-1.19)
Eastern Mediterranean and Europe	1.26 (1.11-1.42)	1.20 (1.06-1.36)
All Regions Combined	1.09 (1.06-1.12)	1.05 (1.02-1.08)
Excluding West Africa <sup>b</sup>	1.17 (1.14-1.21)	1.11 (1.08-1.15)

PR, Prevalence Ratio; 95%CI, 95% confidence interval.

<sup>a</sup>Adjusted for type of facility (flush toilet, 'improved' latrine, 'unimproved' latrine) mother's age, mother's educational attainment, highest level of education in the households, and asset ownership.

<sup>b</sup>West Africa defined as: Benin, Burkina Faso, Cameroon, Ghana, Guinea, Mali, Nigeria, Senegal, and Sierra Leone.

**Table 4.3.** Heterogeneity of the effect of sharing within Africa. Countries are grouped based on quintiles of the crude prevalence ratio. Prevalence ratios and 95% confidence intervals for diarrhea, comparing households with shared toilet facilities to households with facilities that are not shared.

<b>Countries</b>	<b>Crude PR (95% CI)</b>	<b>Adjusted<sup>a</sup> PR (95%CI)</b>
Nigeria, Cameroon, Mali, Senegal, Liberia	0.82 (0.76-0.88)	0.86 (0.80-0.93)
Sao Tome and Principe, Namibia, Congo, Burkina Faso, Burundi	1.05 (0.96-1.15)	1.00 (0.92-1.10)
Benin, Malawi, Niger, Zambia, Sierra Leone	1.15 (1.07-1.23)	1.10 (1.03-1.18)
Swaziland, Kenya, Uganda, DR Congo, Guinea	1.23 (1.12-1.34)	1.19 (1.09-1.30)
Ethiopia, Lesotho, Tanzania, Zimbabwe, Rwanda, Ghana, Madagascar	1.35 (1.25-1.45)	1.32 (1.22-1.42)

PR, Prevalence Ratio; 95%CI, 95% confidence interval.

<sup>a</sup>Adjusted for type of facility (flush toilet, 'improved' latrine, 'unimproved' latrine) mother's age, mother's educational attainment, highest level of education in the household, and asset ownership.

**Table 4.4.** The number of households sharing a toilet facility and the prevalence ratios for diarrhea among children < 5 years of age. Data from 40 Demographic and Health Surveys, 2001-2011.

<b>Region</b>	<b>Sharing Category<sup>a</sup></b>	<b>Crude PR (95% CI)</b>	<b>Adjusted<sup>b</sup> PR (95% CI)</b>
Africa	With ≤ 5 households	1.06 (1.02-1.10)	1.04 (1.00-1.08)
	With > 5 households	1.01 (0.95-1.08)	1.02 (0.95-1.09)
West Africa <sup>c</sup>	With ≤ 5 households	0.88 (0.82-0.94)	0.89 (0.83-0.95)
	With > 5 households	0.81 (0.73-0.90)	0.87 (0.79-0.96)
Excluding West Africa <sup>c</sup>	With ≤ 5 households	1.20 (1.15-1.25)	1.15 (1.10-1.20)
	With > 5 households	1.20 (1.10-1.31)	1.17 (1.08-1.28)
Latin America and the Caribbean	With ≤ 5 households	1.09 (1.00-1.18)	1.04 (0.95-1.13)
	With > 5 households	1.10 (0.88-1.38)	1.01 (0.81-1.26)
South-East Asia and Western Pacific	With ≤ 5 households	1.13 (1.03-1.25)	1.08 (0.98-1.18)
	With > 5 households	1.27 (1.05-1.55)	1.21 (0.99-1.46)
Eastern Mediterranean and Europe	With ≤ 5 households	1.25 (0.93-1.67)	1.15 (0.85-1.54)
	With > 5 households	1.48 (0.67-3.29)	1.36 (0.63-2.94)
All Regions Combined	With ≤ 5 households	1.08 (1.04-1.11)	1.04 (1.00-1.07)
	With > 5 households	1.05 (0.99-1.12)	1.03 (0.97-1.09)
Excluding West Africa <sup>c</sup>	With ≤ 5 households	1.16 (1.12-1.21)	1.10 (1.07-1.15)
	With > 5 households	1.20 (1.12-1.30)	1.14 (1.06-1.23)

PR, Prevalence Ratio; 95%CI, 95% confidence interval.

<sup>a</sup>The reference category is those that use a 'Not Shared' facility.

<sup>b</sup>Adjusted for type of facility (flush toilet, 'improved' latrine, 'unimproved' latrine) mother's age, mother's education, highest level of education in the household, and ownership of assets.

<sup>c</sup>West Africa defined as: Benin, Burkina Faso, Cameroon, Ghana, Guinea, Mali, Nigeria, Senegal, and Sierra Leone.

**Table 4.5.** The sanitation ‘ladder’ and the prevalence of diarrhea. Prevalence ratios and 95% confidence intervals for diarrhea, by level of the JMP Sanitation Ladder. Data from 51 Demographic and Health Surveys, 2001-2011.

<b>Region</b>	<b>Sanitation Ladder Category</b>	<b>Crude PR (95% CI)</b>	<b>Adjusted<sup>a</sup> PR (95% CI)</b>
Africa	No Facility	1.06 (1.01-1.11)	0.95 (0.90-1.00)
	Unimproved Facility (Shared or Not Shared)	1.03 (0.98-1.08)	0.96 (0.92-1.01)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.93 (0.88-0.98)	0.95 (0.90-1.00)
West Africa <sup>b</sup>	No Facility	1.14 (1.07-1.23)	0.98 (0.91-1.05)
	Unimproved Facility (Shared or Not Shared)	1.18 (1.10-1.27)	1.05 (0.98-1.13)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	1.11 (1.03-1.20)	1.10 (1.02-1.19)
Excluding West Africa <sup>b</sup>	No Facility	0.97 (0.91-1.04)	0.91 (0.84-0.98)
	Unimproved Facility (Shared or Not Shared)	0.91 (0.85-0.97)	0.88 (0.82-0.93)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.78 (0.72-0.83)	0.81 (0.75-0.87)
Latin America and the Caribbean	No Facility	1.24 (1.14-1.35)	1.12 (1.03-1.22)
	Unimproved Facility (Shared or Not Shared)	1.13 (1.03-1.24)	1.09 (0.99-1.19)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.88 (0.81-0.95)	0.96 (0.89-1.04)
South-East Asia and Western Pacific	No Facility	1.07 (0.98-1.17)	1.04 (0.94-1.14)
	Unimproved Facility (Shared or Not Shared)	1.02 (0.90-1.14)	0.99 (0.88-1.12)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.90 (0.82-0.98)	0.95 (0.87-1.04)
Eastern Mediterranean and Europe	No Facility	0.85 (0.73-0.98)	0.81 (0.69-0.94)
	Unimproved Facility (Shared or Not Shared)	0.75 (0.63-0.89)	0.75 (0.63-0.89)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.78 (0.69-0.90)	0.83 (0.72-0.94)
All Regions Combined	No Facility	1.08 (1.04-1.12)	0.99 (0.95-1.02)
	Unimproved Facility (Shared or Not Shared)	1.03 (0.99-1.07)	0.98 (0.94-1.02)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.90 (0.87-0.93)	0.95 (0.91-0.99)
Excluding West Africa <sup>b</sup>	No Facility	1.04 (1.00-1.09)	0.98 (0.94-1.03)
	Unimproved Facility (Shared or Not Shared)	0.97 (0.92-1.01)	0.94 (0.90-0.98)
	Shared Facility (Otherwise Improved)	1.00 (Ref.)	1.00 (Ref.)
	Improved Facility (Not Shared)	0.83 (0.80-0.87)	0.89 (0.85-0.93)

PR, Prevalence Ratio; 95%CI, 95% confidence interval.

<sup>a</sup>Adjusted for mother’s age, mother’s educational attainment, highest level of education in the households, and asset ownership.

<sup>b</sup>West Africa defined as: Benin, Burkina Faso, Cameroon, Ghana, Guinea, Mali, Nigeria, Senegal, and Sierra Leone.

## Chapter V

### The joint effects of water and sanitation on diarrheal disease: A multi-country analysis of the Demographic and Health Surveys

#### Abstract

Drinking water and sanitation are effective interventions for preventing diarrheal disease. Because they may block similar or distinct transmission pathways, they may be redundant services preventing the same cases of diarrhea, or act independently, or even synergistically. These effects may also vary across countries and over time. We used data from 217 Demographic and Health Surveys conducted in 90 countries between 1986 and 2013 and modified Poisson regression to assess the impact of water and sanitation infrastructure on the prevalence of diarrhea among children under five. The impact of water and sanitation varied across surveys, and adjusting for socioeconomic status drove these estimates towards the null. Sanitation had a greater effect than water infrastructure when all 217 surveys were pooled; however, the impact of sanitation diminished over time. Based on survey data from the past ten years, we saw no evidence for benefits in improving drinking water or sanitation alone, but we estimated a 6% reduction of both combined (prevalence ratio = 0.94, 95% confidence limit 0.91-0.98). Water and sanitation interventions should be combined to maximize the number of cases of diarrheal disease prevented in children under five. Further research should identify the sources of variability

seen between countries and across time. These national surveys likely include substantial measurement error in the categorization of water and sanitation, making it difficult to interpret the roles of other pathways.

## **Introduction**

Diarrheal diseases are a leading cause of death in children under five in developing countries worldwide (1), accounting for over 700,000 child deaths in 2011 (2). The frequency of diarrheal diseases in developing countries is largely attributed to a lack of clean water and adequate sanitation (3). While the network of water quality, human waste disposal, health status, and disease transmission has been meticulously documented and is widely understood (4, 5), the joint effects of multiple interventions in preventing disease are not well understood.

Four key studies have investigated the interaction between water and sanitation services. Esrey (6) used cross-sectional data from 8 Demographic and Health Surveys (DHS) to show that 1) improved water supply had no meaningful effect on health if improved sanitation was not concurrent and 2) larger impacts were seen with both interventions than the improvements to water or sanitation alone. In a cohort study among Filipino infants, VanDerslice and Briscoe (7) reported that improved water was most protective when a community had better sanitation. Similarly, a meta-analysis by Gundry et al. showed that the protective effect of improved water interventions was stronger when a greater proportion of households had access to improved sanitation (8). Finally, mathematical modeling suggests that water quality improvements may have little to no impact when sanitation conditions are poor (9). The different conclusions in these four

studies may be the result of differences in underlying contextual factors, such as social and environmental conditions. Studies have also shown that improved sanitation infrastructure may have a greater impact on diarrheal disease than improved water infrastructure (6, 10). However, the extent of the impact from these facilities has varied, possibly because of context.

Extending upon this work, as well as a prior DHS analysis that examined water and sanitation benefits separately (10), we use data from 217 DHS to examine both the independent and joint effects of improved water and sanitation. In addition, we examine whether these effects vary geographically (such as between countries), over time, or between rural and urban areas. These standardized surveys provide the opportunity to address these questions on a large scale.

## **Methods**

We used data from DHS surveys (measuredhs.com) completed between 1986 and 2013. These are country-specific surveys on population demographics and health that have been conducted in over 90 developing countries using standardized household questionnaires. In some instances, we include only surveys that were completed in the past 10 years (2003-2013), and when a country had multiple surveys in the past 10 years, we used only the most recent. This selection was based on the desire to achieve a balance between using a dataset that is most relevant to current conditions, having a sufficient sample size to conduct our analysis, and preventing some countries from being overrepresented. These surveys typically employ a two-stage sampling strategy wherein a country is divided into enumeration areas (clusters), and then households are randomly

selected within each cluster. Other household surveys, such as the Multiple Indicator Cluster Surveys, were considered. However, we opted to limit our analysis to DHS surveys in order to limit differences in survey methodology.

Household characteristics, demographics, and health information were obtained from eligible women ages 15-49 in each household surveyed, although in some countries, only ever-married women (age 15-49) were interviewed. Childhood diarrhea was ascertained by asking mothers whether each child under five years of age in the household had experienced diarrhea in the two weeks preceding the interview. No specific case definition of diarrhea was used.

Water and sanitation sources for each household were measured by asking the respondent about the “main source of drinking water” and the “kind of toilet facilities” that were used by household members. We then classified sanitation facilities and sources of drinking water as being either improved or unimproved using the classification system of the Joint Monitoring Programme (JMP) for Water and Sanitation (11): 1) improved water sources were defined as a protected spring, protected well, tubewell or borehole, public tap, piped water to yard, piped water into dwelling or rainwater; 2) unimproved water sources were defined as an unprotected spring or well, tanker truck or bottled water, or surface water; 3) an improved sanitation facility was defined as a sewer system, flush toilet (or pour-flush toilet to pit latrine, septic tank, or to an unknown location), septic tank, composting toilet, ventilated improved pit latrine, or pit latrine with a slab and 4) an unimproved sanitation facility was defined as a flush or pour flush to elsewhere (i.e., open gutter), hanging toilet or hanging latrine, pit latrine without a slab, bucket, bush, field or no facilities. To assess the independent and joint effects of improved water and improved



sanitation, we classified households in the following way: 1) uses unimproved water and unimproved sanitation (neither improved); 2) uses improved water and unimproved sanitation (improved water only); 3) uses unimproved water and improved sanitation (improved sanitation only), and 4) uses improved water and improved sanitation (both improved).

Several potential confounders were included in the analysis. For each survey, a socioeconomic status (SES) index was constructed using principal components analysis (12) of the mother's age and education, household asset ownership (cooking fuel, floor material, electricity, radio, television, refrigerator, bicycle, motorcycle or scooter, and a car or truck), the highest education in the household, and whether or not the child had a health card. Within each survey, SES quintiles were derived based on the index. Many DHS datasets include a wealth index/quintile variable; however, we chose to create our own since those provided by the DHS typically included drinking water source and sanitation facilities in the index. In addition to the SES index, we adjusted for the child's age in years, the child's sex (female versus male), and whether the household was in an urban or rural area.

Modified poisson regression, accounting for complex sampling, was used to estimate unadjusted and adjusted prevalence ratios (PR) and 95% confidence limits (95%CL) for the prevalence of diarrhea in children under five years of age. We first estimate these PRs for each specific survey. For the pooled analyses, data from multiple surveys were combined, and a single model was used which included a fixed effect for each survey (survey dummies). To test for longitudinal trends, we first ran a pooled model for each year, which included all surveys conducted during that year, the year previous, or the following year.

To highlight within country time trends, we used a multilevel linear regression with the log PR of each survey as the dependent variable and year as a continuous predictor. These models also included a random intercept and slope for each country. All statistical analysis was conducted using STATA 11.2 (StataCorp LP, College Station, TX).

For assessing interaction, we determined whether the observed joint effect of both water and sanitation together was greater than, equal to, or less than the expected joint effect (13, 14). For multiplicative interaction, the expected joint effect is the product of the two independent effects ( $PR_{11} = PR_{10} * PR_{01}$ ). For additive interaction, the expected joint effect is the sum of the two independent effects minus one ( $PR_{11} = PR_{10} + PR_{01} - 1$ ). The 95% CL for the expected joint effect was calculated using the delta method (15). A synergistic interaction would be evident if the observed joint effect of both exposures exceeds the expected effect. This implies that the effect of one exposure is greater in the presence of the other. If, however, the observed effect is equal to the expected effect, then the two exposures are likely independent; the effect of each does not depend on the presence of the other. Another possible outcome is some form of antagonism, where the observed joint effect is less than the expected effect, suggesting that the effect of each exposure is diminished in the presence of the other. This implies that the two exposures are acting on the same pathway and preventing the same cases.

## **Results**

### *Survey Specific Results*

217 surveys from 74 countries had data on diarrhea, source of drinking water, sanitation facility, and necessary covariates (data by country and survey year can be seen

in Table 5.1). The prevalence of diarrhea among children <5 years of age ranged from 4.4% (Maldives, 2009) to 39.6% (Senegal, 1986), and the median across surveys was 16.1%. Coverage of improved water and sanitation services also varied across countries. For example, in 37 surveys, many from African countries, showed that >50% of children lived in households lacking access to both improved water and sanitation, whereas in 50 surveys >50% of children had access to both services.

The effect of improved drinking water varied substantially across surveys, even after adjusting for potential confounders (Figure 5.2). The strongest protective effect was observed in Vietnam (2002) (PR 0.51, 95%CL 0.28-0.91), and the strongest harmful effect, though not statistically significant, was observed in Armenia (2000) (PR 1.64, 95%CL 0.76-3.56). A total of 23 surveys showed a significant protective effect of improved drinking water, while 190 had effects that overlapped the null, and 4 had a harmful effect. Adjustment for confounders tended to attenuate the effect of improved water (unadjusted PRs for each survey can be seen in Figure 5.1).

The adjusted effect of improved sanitation also varied across surveys, with PRs ranging from 0.40 (95%CL 0.20-0.82) in Kazakhstan (1999) to 1.93 (95%CL 1.03-3.63) in Armenia (2005) (Figure 5.2). 41 surveys showed a significant protective effect, 168 had effects overlapping the null, and 7 showed a statistically significant harmful effect. Jordan (2012) had too few children in the unimproved category to estimate the effect of sanitation. Similar to what was seen for improved drinking water, adjustment for confounders had a mostly attenuating effect on the impact of improved sanitation (unadjusted PRs can be seen in Figure S1).

### *Pooled Results*

When pooling data across all 217 surveys, the unadjusted prevalence of diarrhea was 8.9% lower (PR 0.92, 95%CL 0.91-0.93) among those with improved drinking water compared to those without (Table 5.2, Model 1). When accounting for differences in household SES and access to sanitation services, the effect was attenuated (PR 0.97, 95%CL 0.96-0.99; Table 5.2, Model 3). Adjusting for the child's age, sex, and urban/rural residence had no effect on the impact of improved water, suggesting that these covariates are not confounders (Table 5.2, Model 4). The unadjusted effect of improved sanitation (PR 0.85, 95%CL 0.84-0.86) was stronger than that of drinking water (Table 5.2, Model 2). This effect was also attenuated after accounting for household SES (PR 0.93, 95%CL 0.92-0.95; Table 5.2, Model 3) but remained stronger than the adjusted effect of improved drinking water. There was little evidence of a difference in the effect of water or sanitation between urban and rural areas.

### *Longitudinal Trends*

When excluding surveys conducted prior to 2003, the adjusted effect of improved sanitation was smaller (Table 5.3, Model 8). A gradual attenuation of the effect of sanitation over time can be observed in Figure 5.3. For example, when pooling surveys conducted between 1989 and 1991, the adjusted effect of improved sanitation was 0.89 (95%CL 0.83-0.95), and for surveys conducted between 2006 and 2008 the effect was null (PR 0.99, 95%CL 0.95-1.02). Because this trend may be due to the inclusion of different countries from varying time points, we used a multilevel model to investigate within-country trends.

The predicted prevalence ratio for improved sanitation can be seen to increase over time ( $p=0.09$ ) and approach the null in recent years, suggesting that this attenuation occurred within countries. The effect of improved drinking water appeared to be relatively constant over time.

### *Independent and Joint Effects of Water and Sanitation*

When all 217 surveys were combined, the prevalence of diarrhea was lower when a household had either improved water, improved sanitation, or both compared to when they had neither service (Table 5.4, Model 9). The unadjusted independent effect of improved sanitation, however, was stronger than that of improved water. Adjusting for confounders resulted in a marked attenuation of both independent effects and the joint effect (Table 5.4, Model 10). The adjusted independent effect of improved water was statistically significant but small (PR 0.98, 95%CL 0.97-0.99). The adjusted independent effect of sanitation was somewhat larger (PR 0.95, 95%CL 0.92-0.97). Having both improved water and sanitation resulted in 9.9% lower prevalence of diarrhea (PR 0.90, 95%CL 0.89-0.92). When considering only surveys from the past 10 years, having both improved water and sanitation resulted in a 5.9% lower prevalence of diarrhea.

The results of Model 10 suggest very little interaction on the multiplicative scale. The expected joint effect, under the assumption of no multiplicative interaction, was 0.93 ( $0.98 \times 0.95$ ), only slightly larger than the observed joint effect of 0.90 ( $p$ -value for difference=0.039). Similarly, Model 10 shows little interaction on the additive scale, where the expected joint effect of 0.92 ( $0.98 + 0.95 - 1$ ), only slightly different from the observed ( $p=0.058$ ). This is evidence that water and sanitation are likely operating primarily on

different pathways. When considering only surveys from the past 10 years, however, Model 12 shows synergistic interaction for water and sanitation. Neither alone had any effect on the prevalence of diarrhea; however, both together had a protective effect. On the multiplicative and additive scales, the observed effect was larger than the expected effect ( $p=0.042$  and  $p=0.041$ , respectively).

## **Discussion**

Our findings confirm the results of previous studies that water and sanitation infrastructure reduce the risk of diarrheal disease in children and that water and sanitation likely operate independently. We found, however, that the individual effect of improved sanitation and improved water in our overall sample was smaller than that found in two previous meta-analyses. Fewtrell et al. (5) and Esrey et al. (16) reported reduced risks of diarrheal disease of 32% and 22% for sanitation interventions and 22% and 17% for water interventions, respectively, in contrast to the 7% and 3% reductions reported by our study (Table 5.2, Model 4). One significant difference is that these previous meta-analyses were summaries of intervention trials, many of which were of short duration with unblinded participants. Such intervention trials often attempt to measure the efficacy of an intervention under idealized conditions as opposed to its “real-world” effectiveness, which is often smaller.

Other cross-sectional studies (6, 10) using the DHS reported smaller effect sizes than those of the meta-analyses. For example, Fink et al. (10) used data from 171 surveys and found that intermediate and high quality water compared to low quality water reduced the odds of diarrhea by 8% and 9%, respectively. High and intermediate quality sanitation

had slightly stronger reductions of 8% and 13%, respectively. Our results, however, showed even smaller effect sizes than these previous studies, especially for improved drinking water. There are three primary reasons for this: First, our analysis includes more up-to-date data from the DHS. Early surveys tended to show a much stronger effect of improved sanitation, while more recent surveys were more likely to show a null effect. Second, we estimated prevalence ratios, instead of prevalence odds ratios as previous studies had done. The odds ratio will be exaggerated relative to the prevalence ratio, especially when the outcome is not rare, as is the case in our data. Third, we use a different classification scheme for improved/unimproved water and sanitation. Our results suggest that the JMP scheme may not capture disease risk as well as technology classification schemes used in other studies. For example, when using the infrastructure categorization scheme of Fink et al. (10), we see a stronger protective effect of flush and pour-flush technology, but little effect of latrine technologies (see Appendix 5.1). These differences are largely due to the fact that Fink et al. (10) use a three-level categorization which allows for a more extreme contrast. For drinking water, there is the additional difference that some technologies, such as bottled water, are classified by the JMP as unimproved but as the highest category by Fink et al. (10).

Confounding presents a substantial challenge for observational studies of water and sanitation. Households with unimproved services are much more likely to be of a lower SES, and therefore have higher risk of disease due to pathways other than water or sanitation, such as hygiene or contaminated food. Because SES is highly correlated with water and sanitation services, it is difficult to differentiate the effects. Our results show the presence of substantial confounding – the effect of improved drinking water is largely

explained by differences in SES, yet SES was still highly protective even after adjusting for water and sanitation. If our measurement of water and sanitation technologies does not accurately capture the risks they pose, then SES may be capturing some of the true risk associated with water and sanitation in addition to capturing the effect of other pathways.

Our results also highlight the heterogeneity of the effect of improved water and sanitation across surveys. In many surveys, improved infrastructure is protective, in others it has no effect, and in a few it appears to be harmful. In the presence of high heterogeneity, a single effect measure is less useful and can even be misleading. This heterogeneity has several potential sources. First, classifying water and sanitation technologies across a variety of settings is a challenging task. Some of the observed heterogeneity may be due to a differing degree of measurement error between surveys. Second, in the absence of any type of bias or measurement error, sampling error will still result in some variability in the effect size across studies, even though the true underlying parameter of interest is the same. Lastly, it is likely that the effect of these improved services varies across time and place.

We have shown that the effect of improved sanitation has attenuated over the past 25 years, even within the same country. This finding is unique to our study, and may explain some of the variability seen across surveys. One possible explanation for this observed attenuation is that environmental contamination has decreased over time. This would reduce the fraction of cases attributable to poor sanitation, thus reducing its effectiveness at preventing disease. This and other explanations, such as the adoption of sub-optimal technology, are beyond the scope of this analysis but should be a focus of future research.



Our results underscore the importance of both water and sanitation for preventing diarrheal disease in children under five. Water and sanitation also provide other important health and non-health benefits to users, such as privacy and safety, warranting more investigation. When examining the independent and joint effects among all 217 surveys, either water or sanitation alone has a modest protective effect, and the joint effect of both together is roughly what is expected based on the independent effects. However, when using surveys from the past 10 years, water and sanitation infrastructure appear to be synergistic. In contrast to meta-analyses that reported combining interventions provide no additional benefit beyond what is seen with a single intervention (5, 16), these results support findings by Balthazar et al.(17), Esrey (6), and VanDerslice (7) that combined interventions are more protective in reducing diarrheal episodes than single interventions. Although these data provide no evidence of protection in the presence of either improved water or sanitation infrastructure alone; when in combination the services are protective. These two findings have a single underlying message – both interventions combined are better than a single intervention.

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**Table 5.1** The prevalence of diarrhea and access to improved drinking water and sanitation services among children < 5 years of age in 217 Demographic and Health Surveys, 1986-2013.

Country (Year)	Prevalence of Diarrhea (%)	Access to Improved Services (%)			
		Neither Improved	Improved Drinking Water Only	Improved Sanitation Only	Both Improved
<b>Africa</b>					
Benin (1996)	26.2	47.0	38.0	8.3	6.7
Benin (2001)	13.8	31.0	42.0	4.8	22.2
Benin (2006)	9.2	29.3	52.5	3.5	14.6
Benin (2012)	6.4	20.3	50.4	3.4	25.9
Burkina Faso (1992)	20.5	85.3	13.6	0.5	0.5
Burkina Faso (1999)	20.3	52.9	46.6	0.2	0.4
Burkina Faso (2003)	21.1	42.8	41.7	2.5	13.0
Burkina Faso (2010)	14.9	23.3	51.6	2.1	23.1
Burundi (1987)	17.5	30.5	61.5	2.5	5.5
Burundi (2011)	25.1	16.6	43.5	8.9	31.0
CAR (1994)	23.1	54.0	31.6	9.2	5.2
Cameroon (1991)	18.4	55.8	37.6	0.2	6.4
Cameroon (1998)	19.0	18.3	55.7	3.5	22.5
Cameroon (2004)	17.0	45.1	24.6	16.9	13.4
Cameroon (2011)	21.7	24.0	22.2	10.7	43.0
Chad (1996)	22.0	68.1	24.6	3.8	3.5
Chad (2004)	26.9	61.1	34.7	1.1	3.1
Comoros (1996)	23.4	6.9	71.6	1.2	20.3
Congo (Brazzaville) (2005)	14.3	39.8	43.1	2.1	14.9
Congo (Brazzaville) (2011)	19.3	23.9	38.7	3.1	34.4
Congo, Democratic Republic (2007)	16.8	40.2	22.6	14.5	22.8
Cote d'Ivoire (1994)	22.0	47.5	18.1	11.5	22.8
Cote d'Ivoire (1999)	22.6	48.8	29.6	4.3	17.2
Cote d'Ivoire (2012)	18.5	20.8	37.3	4.1	37.8
Ethiopia (1992)	24.1	78.6	20.9	0.0	0.4
Ethiopia (1997)	18.1	39.8	51.1	2.8	6.2
Ethiopia (2003)	13.5	49.2	38.0	4.2	8.6
Gabon (2000)	17.2	20.1	36.3	1.1	42.5
Gabon (2012)	16.8	6.6	32.0	1.7	59.8
Ghana (1988)	26.9	62.0	31.8	1.1	5.1
Ghana (1993)	20.1	46.4	36.5	3.8	13.2
Ghana (1999)	18.4	41.4	34.1	5.9	18.5
Ghana (2003)	15.6	34.0	38.6	4.9	22.5
Ghana (2008)	20.1	11.7	27.9	11.6	48.8

Guinea (1999)	21.8	71.1	10.5	7.3	11.1
Guinea (2005)	16.3	62.5	12.3	10.5	14.8
Guinea (2012)	16.6	20.8	38.4	5.0	35.8
Kenya (1989)	13.0	65.4	28.2	0.3	6.2
Kenya (1993)	14.1	59.1	28.3	2.7	9.8
Kenya (1998)	17.3	68.0	17.5	3.6	10.9
Kenya (2003)	16.4	58.3	27.5	3.3	10.9
Kenya (2009)	16.8	33.0	26.8	9.3	30.9
Lesotho (2004)	14.5	27.2	53.3	3.7	15.8
Lesotho (2009)	11.6	21.9	45.9	3.5	28.6
Liberia (2007)	20.8	33.5	43.3	2.9	20.3
Madagascar (1992)	12.6	63.1	5.7	22.0	9.2
Madagascar (1997)	27.4	82.9	12.7	2.4	1.9
Madagascar (2004)	10.1	46.0	7.6	27.1	19.3
Madagascar (2008)	8.4	63.9	32.2	1.0	2.9
Malawi (1992)	22.1	51.8	45.2	0.3	2.8
Malawi (2000)	17.9	36.2	61.2	0.0	2.6
Malawi (2004)	22.6	38.9	58.2	0.2	2.7
Malawi (2010)	17.7	20.5	67.8	1.3	10.5
Mali (1987)	35.6	29.0	2.1	59.2	9.7
Mali (1996)	25.6	64.9	26.3	4.7	4.1
Mali (2001)	19.0	52.7	31.8	5.6	10.0
Mali (2006)	13.5	40.5	38.9	4.6	16.0
Mozambique (1997)	20.9	76.1	21.4	0.5	2.0
Mozambique (2003)	14.5	39.9	11.0	18.4	30.8
Mozambique (2011)	11.2	46.2	32.5	4.4	16.8
Namibia (1992)	22.6	48.4	25.5	0.4	25.8
Namibia (2000)	13.4	23.0	44.9	0.3	31.8
Namibia (2007)	13.4	13.6	47.8	1.1	37.4
Niger (1992)	28.6	79.2	7.8	4.6	8.5
Niger (1998)	38.6	62.3	25.9	3.9	8.0
Niger (2006)	21.3	59.4	31.9	1.6	7.2
Niger (2012)	14.4	32.7	49.5	1.5	16.3
Nigeria (1990)	18.0	68.4	24.7	1.2	5.7
Nigeria (1999)	15.7	59.0	23.0	6.5	11.5
Nigeria (2003)	19.2	60.3	25.7	3.2	10.8
Nigeria (2008)	10.3	28.7	19.4	19.7	32.2
Rwanda (1992)	22.0	4.6	1.2	72.5	21.7
Rwanda (2000)	17.2	57.0	33.6	2.3	7.1
Rwanda (2005)	14.4	2.2	1.4	62.9	33.5
Rwanda (2008)	13.9	24.6	18.5	34.6	22.4
Rwanda (2011)	13.3	8.6	18.6	19.5	53.4
Sao Tome and Principe (2008)	16.1	4.8	57.7	1.7	35.9

Senegal (1986)	39.6	67.6	4.8	15.6	12.0
Senegal (1993)	21.3	36.0	32.6	15.7	15.7
Senegal (1997)	15.5	32.6	35.8	19.3	12.3
Senegal (2005)	23.3	26.8	31.0	6.0	36.2
Senegal (2011)	21.1	18.5	25.7	5.6	50.2
Senegal (2012)	14.8	20.2	23.4	8.0	48.4
Sierra Leone (2008)	13.6	39.9	20.1	13.3	26.7
Sudan (1989)	30.2	46.3	48.9	0.3	4.5
Swaziland (2006)	14.1	29.3	41.2	6.0	23.5
Tanzania (1992)	13.5	69.0	28.5	0.6	2.0
Tanzania (1996)	14.2	66.0	31.5	0.5	2.1
Tanzania (1999)	13.0	38.3	59.3	0.1	2.3
Tanzania (2004)	13.1	56.4	39.2	0.8	3.7
Tanzania (2010)	14.9	51.7	33.4	4.4	10.5
Togo (1988)	30.1	20.5	45.7	7.7	26.1
Togo (1998)	31.3	50.1	41.3	1.7	6.9

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#### Latin America and the Caribbean

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Bolivia (1989)	30.3	40.6	16.1	11.6	31.7
Bolivia (1994)	29.7	40.0	35.8	1.3	22.9
Bolivia (1998)	19.3	20.8	17.8	10.1	51.2
Bolivia (2004)	22.6	24.5	44.8	0.8	29.9
Bolivia (2008)	26.2	15.9	45.7	0.7	37.7
Brazil (1986)	17.2	18.8	6.1	14.1	60.9
Brazil (1991)	15.4	37.6	19.1	7.8	35.5
Brazil (1996)	13.3	20.8	19.5	10.2	49.5
Colombia (1986)	19.2	15.7	12.2	7.2	64.9
Colombia (1990)	12.6	7.7	12.2	2.6	77.5
Colombia (1995)	16.9	14.0	16.1	3.2	66.8
Colombia (2000)	14.1	10.1	13.0	5.8	71.1
Colombia (2005)	14.4	7.0	9.8	8.0	75.2
Colombia (2010)	12.7	6.2	8.8	10.6	74.4
Dominican Republic (1986)	26.2	7.9	13.7	10.6	67.8
Dominican Republic (1991)	17.0	6.2	7.5	17.8	68.5
Dominican Republic (1996)	16.2	6.1	11.9	30.4	51.7
Dominican Republic (1999)	16.6	18.2	32.8	32.9	16.2
Dominican Republic (2002)	14.2	4.6	7.2	55.2	33.0
Dominican Republic (2007)	14.9	4.6	5.1	65.0	25.3
Dominican Republic (2007)	18.0	20.1	25.5	26.0	28.3
Guatemala (1987)	16.7	29.1	10.4	26.0	34.4
Guatemala (1995)	21.0	5.6	18.4	15.1	60.9
Guatemala (1998)	13.4	10.9	55.3	9.2	24.6
Guyana (2009)	10.0	4.5	7.8	33.1	54.6

Haiti (1994)	28.2	44.8	15.0	21.3	18.9
Haiti (2000)	26.8	30.7	30.1	11.7	27.5
Haiti (2006)	24.3	37.8	37.3	7.6	17.3
Haiti (2012)	21.3	30.5	20.0	27.3	22.3
Honduras (2006)	16.1	17.1	24.4	21.9	36.7
Honduras (2012)	18.0	10.7	17.1	34.0	38.2
Mexico (1987)	22.8	31.7	1.4	30.4	36.6
Nicaragua (1998)	14.3	40.0	40.1	1.6	18.3
Nicaragua (2001)	13.3	62.2	9.9	13.3	14.6
Paraguay (1990)	8.2	64.3	14.7	2.5	18.5
Peru (1986)	32.5	44.9	25.9	2.8	26.4
Peru (1991)	18.4	27.0	15.9	11.0	46.1
Peru (1996)	18.1	23.6	12.3	16.2	47.8
Peru (2000)	15.5	17.2	12.1	17.2	53.5
Peru (2005)	14.0	11.9	9.3	15.9	62.9
Peru (2009)	14.1	8.4	9.5	13.4	68.7
Peru (2010)	14.9	7.6	9.3	13.8	69.3
Peru (2011)	14.0	6.5	7.5	15.6	70.4
Peru (2012)	12.3	5.5	7.6	13.0	73.9
Trinidad and Tobago (1987)	6.2	8.8	45.1	1.0	45.1

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#### South-East Asia

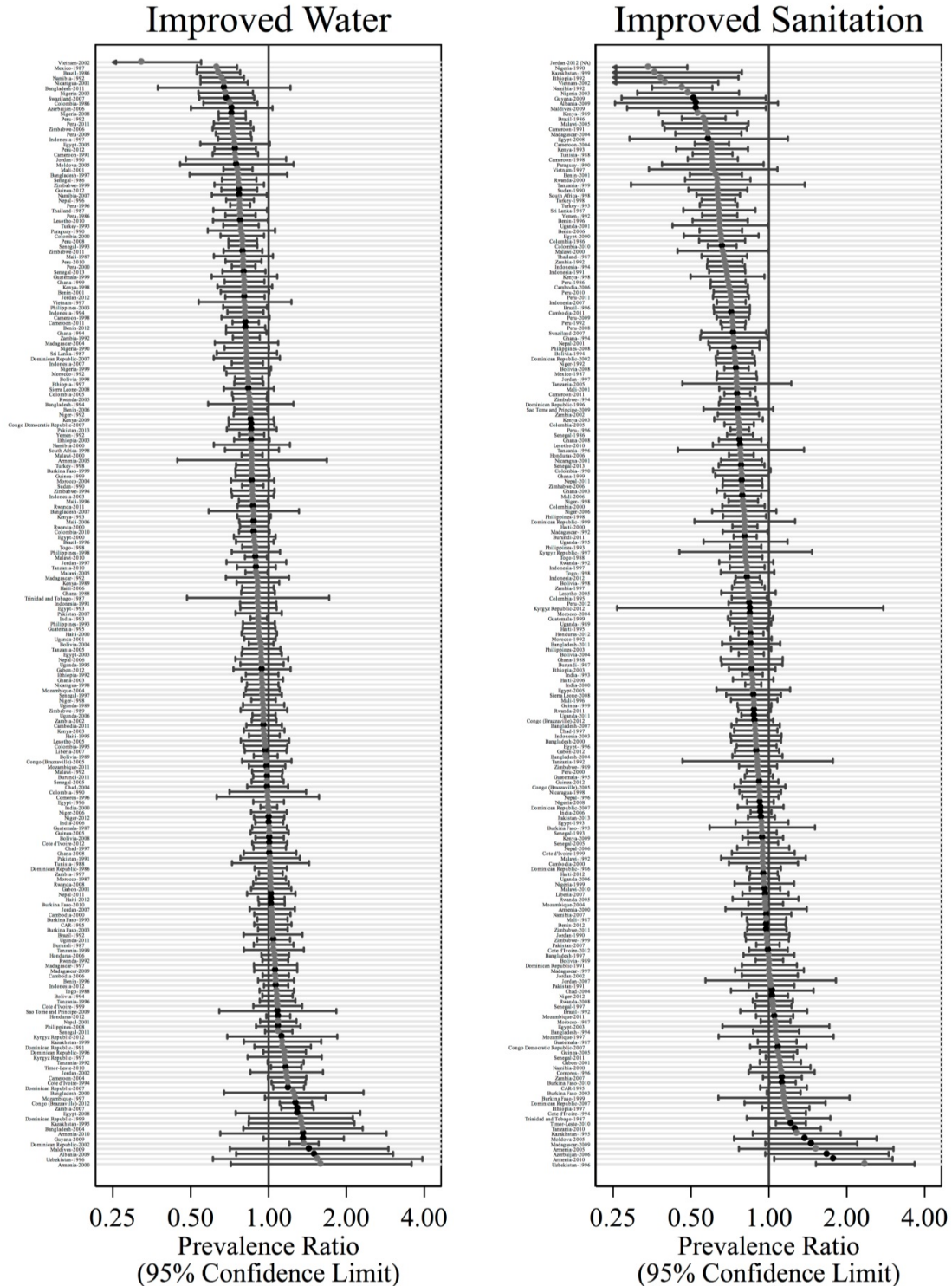
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Bangladesh (1994)	12.4	4.4	34.2	3.3	58.1
Bangladesh (1996)	7.7	4.3	67.3	0.6	27.8
Bangladesh (1999)	6.2	2.9	64.0	0.9	32.2
Bangladesh (2004)	7.5	2.8	75.1	0.4	21.6
Bangladesh (2007)	9.8	2.2	60.2	1.0	36.6
Bangladesh (2011)	4.6	0.7	47.7	0.8	50.8
India (1992)	10.0	29.7	50.8	2.9	16.6
India (1998)	19.0	17.8	53.5	4.0	24.7
India (2006)	9.0	11.1	54.6	2.4	31.8
Indonesia (1991)	11.2	44.7	11.2	27.6	16.5
Indonesia (1994)	12.2	19.8	33.0	8.4	38.9
Indonesia (1997)	10.5	16.3	28.7	10.6	44.4
Indonesia (2002)	11.1	24.2	22.5	15.6	37.7
Indonesia (2007)	13.8	23.6	20.5	20.1	35.8
Indonesia (2012)	14.4	17.0	15.0	35.8	32.3
Maldives (2009)	4.4	0.2	2.8	14.2	82.8
Nepal (1996)	27.6	30.2	55.8	4.4	9.6
Nepal (2001)	20.6	24.8	66.5	1.0	7.7
Nepal (2006)	12.0	15.0	55.0	2.5	27.5
Nepal (2011)	13.9	8.0	48.5	3.0	40.6
Sri Lanka (1987)	6.1	31.2	31.6	7.8	29.3

Thailand (1987)	16.0	36.0	7.8	26.4	29.8
Timor-Leste (2009)	15.7	24.3	25.2	12.0	38.5
<b>Western Pacific</b>					
Cambodia (2000)	19.0	69.2	25.7	1.7	3.4
Cambodia (2005)	19.7	40.1	38.2	7.3	14.4
Cambodia (2010)	15.0	32.7	31.0	12.3	24.0
Philippines (1993)	10.1	18.4	24.9	13.6	43.1
Philippines (1998)	7.5	9.3	15.4	6.9	68.3
Philippines (2003)	10.8	6.9	13.5	10.5	69.1
Philippines (2008)	9.1	6.8	11.3	23.9	58.0
<b>Eastern Mediterranean</b>					
Egypt (1992)	13.4	5.3	6.2	19.6	68.9
Egypt (1995)	16.0	3.6	4.3	18.8	73.4
Egypt (2000)	7.1	1.3	1.9	14.6	82.3
Egypt (2003)	18.9	0.6	0.8	12.5	86.2
Egypt (2005)	18.4	0.1	2.3	2.5	95.1
Egypt (2008)	8.5	0.0	0.5	2.6	96.9
Jordan (1990)	8.5	3.8	73.2	0.3	22.8
Jordan (1997)	18.1	1.5	6.6	4.1	87.8
Jordan (2002)	14.8	0.5	11.3	8.2	80.0
Jordan (2007)	16.0	0.1	0.3	22.6	77.0
Jordan (2012)	15.6	0.0	0.1	44.9	55.0
Morocco (1987)	29.0	42.2	8.2	15.3	34.3
Morocco (1992)	12.7	42.8	11.4	9.6	36.2
Morocco (2004)	12.0	14.1	7.3	14.4	64.2
Pakistan (1990)	14.6	17.5	53.6	0.9	28.0
Pakistan (2007)	21.9	5.0	45.7	2.8	46.5
Pakistan (2012)	22.6	4.5	28.1	5.2	62.2
Tunisia (1988)	20.6	29.1	1.8	65.6	3.4
<b>Europe</b>					
Albania (2008)	5.4	1.2	5.0	16.0	77.8
Armenia (2000)	7.8	8.4	36.0	1.1	54.4
Armenia (2005)	16.8	1.3	5.7	3.1	89.8
Armenia (2010)	8.8	5.2	16.6	1.3	76.8
Azerbaijan (2006)	10.6	6.6	12.9	16.8	63.7
Kazakhstan (1995)	16.0	16.9	55.0	0.1	27.9
Kazakhstan (1999)	13.6	0.6	0.1	46.8	52.5
Kyrgyz Republic (1997)	17.8	30.5	58.3	0.0	11.3
Kyrgyz Republic (2012)	5.2	0.3	1.3	13.9	84.6
Moldova (2005)	7.5	1.8	12.9	8.8	76.4
Turkey (1993)	24.8	9.3	6.7	19.6	64.4
Turkey (1998)	30.1	12.5	3.8	27.1	56.6

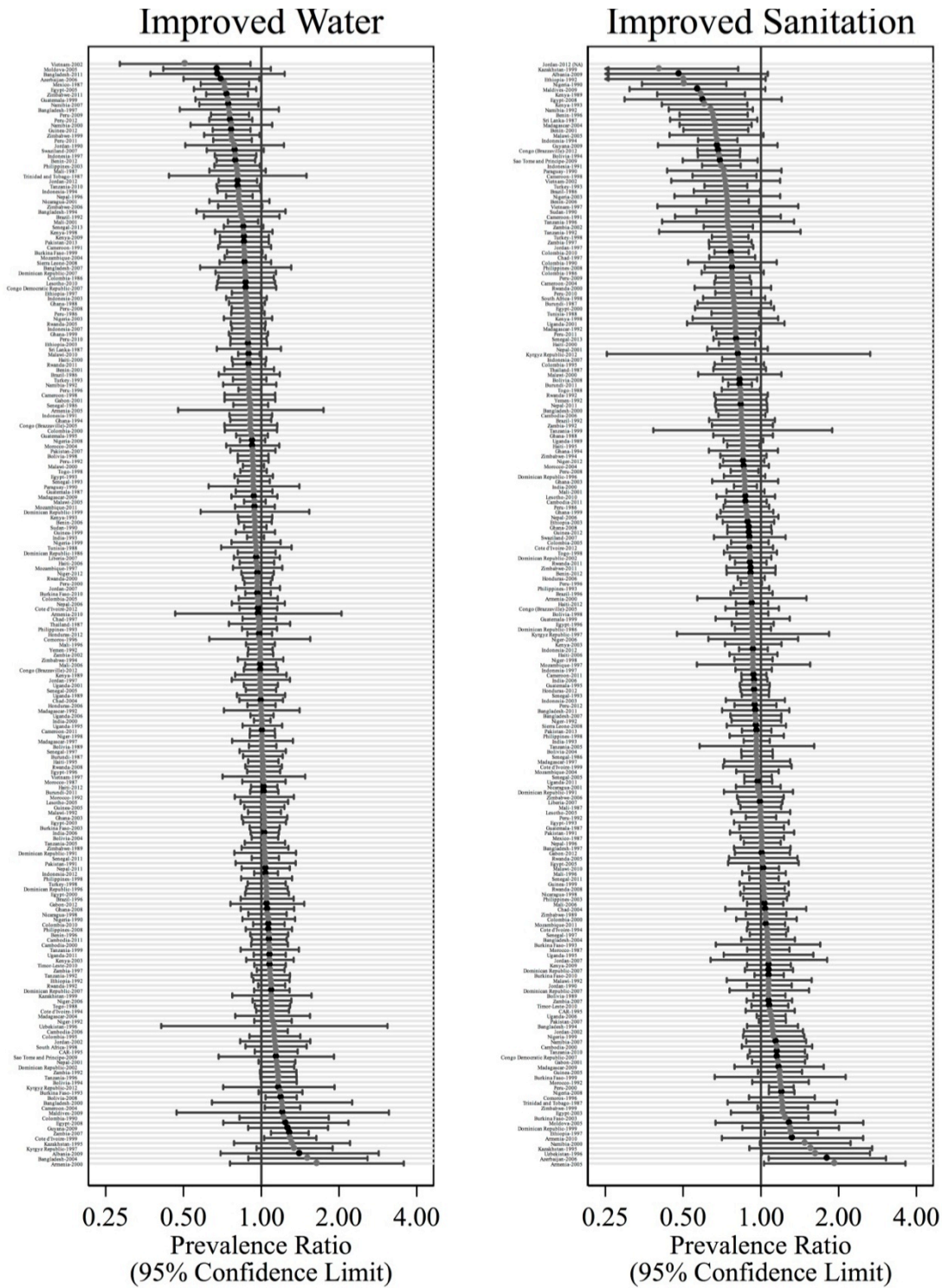


**Figure 5.1** The unadjusted effect of improved drinking water and improved sanitation in each of the 217 Demographic and Health Surveys, 1986-2013. Prevalence ratios are for diarrhea comparing those with the improved service to those without. Black markers indicate the most recent surveys for countries with surveys completed since 2003.



Note: For display purposes, confidence limits are truncated if the lower limit is < 0.25. This is indicated by an arrowhead.

**Figure 5.2** The adjusted effect of improved drinking water and improved sanitation in each of the 217 Demographic and Health Surveys, 1986-2013. Prevalence ratios are for diarrhea comparing those with the improved service to those without, adjusted for child's age and sex, household wealth quintile, and urban/rural residence. Black markers indicate the most recent surveys for countries with surveys completed since 2003.



Note: For display purposes, confidence limits are truncated if the lower limit is < 0.25. This is indicated by an arrowhead.

**Table 5.2** Prevalence ratios (and 95% Confidence Limits) for diarrhea among children < 5 years of age in 217 Demographic and Health Surveys, 1986-2013. All models include survey fixed effects and account for complex sampling design. Models 1-4 include all 217 surveys.

	<b>All Surveys</b>			
	<b>Model 1</b>	<b>Model 2</b>	<b>Model 3</b>	<b>Model 4</b>
<i>Improved Water vs. Unimproved</i>	0.919 (0.908 - 0.929)		0.973 (0.961 - 0.985)	0.971 (0.960 - 0.983)
<i>Improved Sanitation vs. Unimproved</i>		0.852 (0.841 - 0.863)	0.932 (0.918 - 0.945)	0.929 (0.915 - 0.942)
<i>Female Child vs. Male Child</i>				0.927 (0.919 - 0.935)
<i>Age of child in years</i>				
1 vs. 0				1.27 (1.256 - 1.285)
2 vs. 0				0.833 (0.822 - 0.844)
3 vs. 0				0.534 (0.525 - 0.543)
4 vs. 0				0.386 (0.379 - 0.393)
<i>SES Quintile<sup>1</sup></i>				
2 vs. 1			0.992 (0.978 - 1.006)	0.981 (0.967 - 0.995)
3 vs. 1			0.975 (0.960 - 0.990)	0.958 (0.943 - 0.972)
4 vs. 1			0.933 (0.919 - 0.948)	0.907 (0.892 - 0.921)
5 vs. 1			0.795 (0.781 - 0.810)	0.767 (0.752 - 0.782)
<i>Rural vs. Urban</i>				0.96 (0.946 - 0.975)
<i>N</i>	1,584,397	1,581,441	1,577,881	1,577,881

<sup>1</sup>Quintile 1 represents the lowest level of SES, and quintile 5 represents the highest. SES = socioeconomic status

**Table 5.3** Prevalence ratios (and 95% Confidence Limits) for diarrhea among children < 5 years of age in Demographic and Health Surveys, 2003-2013. All models include survey fixed effects and account for complex sampling design. Models 5-8 include only the most recent surveys for countries with a survey completed since 2003.

	<b>Last 10 Years</b>			
	<b>Model 5</b>	<b>Model 6</b>	<b>Model 7</b>	<b>Model 8</b>
<i>Improved Water vs. Unimproved</i>	0.944 (0.922 - 0.966)		0.977 (0.954 - 1.000)	0.975 (0.952 - 0.999)
<i>Improved Sanitation vs. Unimproved</i>		0.902 (0.880 - 0.924)	0.967 (0.942 - 0.994)	0.961 (0.935 - 0.988)
<i>Female Child vs. Male Child</i>				0.929 (0.913 - 0.945)
<i>Age of child in years</i>				
1 vs. 0				1.326 (1.296 - 1.357)
2 vs. 0				0.866 (0.843 - 0.889)
3 vs. 0				0.559 (0.541 - 0.577)
4 vs. 0				0.396 (0.382 - 0.410)
<i>SES Quintile<sup>1</sup></i>				
2 vs. 1			0.995 (0.967 - 1.024)	0.978 (0.951 - 1.007)
3 vs. 1			0.969 (0.940 - 0.999)	0.947 (0.919 - 0.977)
4 vs. 1			0.938 (0.908 - 0.968)	0.905 (0.876 - 0.935)
5 vs. 1			0.828 (0.799 - 0.859)	0.786 (0.757 - 0.817)
<i>Rural vs. Urban</i>				0.941 (0.914 - 0.969)
<i>N</i>	491,539	491,689	491,201	491,201

<sup>1</sup>Quintile 1 represents the lowest level of SES, and quintile 5 represents the highest. SES = socioeconomic status

**Table 5.4** The independent and joint effects of water and sanitation. Prevalence ratios (and 95% Confidence Limits) for diarrhea among children < 5 years of age in 217 Demographic and Health Surveys. All models are modified poisson regressions, include survey fixed effects, and account for complex sampling design. Models 9-10 include all 217 surveys. Models 11-12 include only those surveys completed since 2003.

	All Surveys		Last 10 Years	
	Model 9	Model 10	Model 11	Model 12
Neither Improved	1.000 (Ref.)	1.000 (Ref.)	1.000 (Ref.)	1.000 (Ref.)
Improved Water Only	0.954 (0.941 - 0.967)	0.979 (0.965 - 0.993)	0.978 (0.949 - 1.007)	0.993 (0.964 - 1.023)
Improved Sanitation Only	0.881 (0.863 - 0.901)	0.945 (0.924 - 0.966)	0.942 (0.903 - 0.983)	0.996 (0.954 - 1.041)
Both Improved Water and Sanitation	0.816 (0.803 - 0.829)	0.901 (0.885 - 0.918)	0.875 (0.848 - 0.904)	0.941 (0.908 - 0.975)
Female Child vs. Male Child		0.927 (0.919 - 0.935)		0.929 (0.913 - 0.945)
Age of child in years				
1 vs. 0		1.270 (1.256 - 1.285)		1.326 (1.296 - 1.357)
2 vs. 0		0.833 (0.822 - 0.844)		0.866 (0.843 - 0.889)
3 vs. 0		0.534 (0.525 - 0.543)		0.559 (0.541 - 0.577)
4 vs. 0		0.386 (0.379 - 0.393)		0.396 (0.382 - 0.410)
SES Quintile <sup>1</sup>				
2 vs. 1		0.980 (0.966 - 0.994)		0.978 (0.951 - 1.006)
3 vs. 1		0.957 (0.943 - 0.972)		0.947 (0.919 - 0.976)
4 vs. 1		0.906 (0.892 - 0.921)		0.904 (0.875 - 0.934)
5 vs. 1		0.767 (0.753 - 0.782)		0.786 (0.757 - 0.816)
Rural vs. Urban		0.960 (0.946 - 0.974)		0.941 (0.914 - 0.969)
N	1,577,881	1,577,881	491,201	491,201
Multiplicative Interaction				
Expected Joint Effect <sup>2</sup>	0.841 (0.816-0.865)	0.925 (0.897-0.952)	0.921 (0.865-0.977)	0.990 (0.928-1.052)
p-value for interaction <sup>3</sup>	0.019	0.039	0.043	0.042
Additive Interaction				
Expected Joint Effect <sup>4</sup>	0.835 (0.809-0.862)	0.924 (0.895-0.952)	0.920 (0.862-0.977)	0.990 (0.927-1.052)
p-value for interaction <sup>3</sup>	0.090	0.058	0.059	0.041

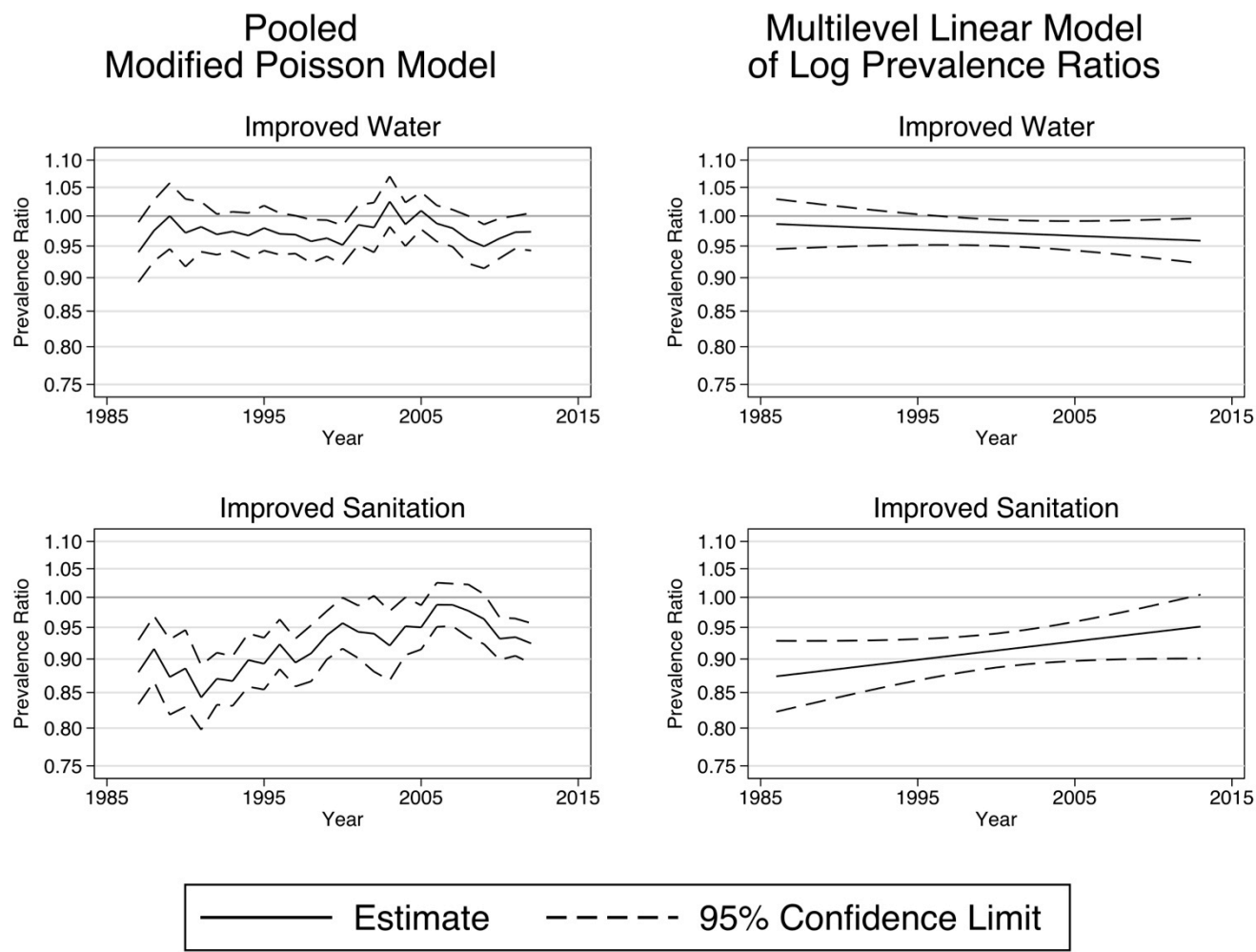
<sup>1</sup>SES=socioeconomic status. <sup>1</sup>Quintile 1 represents the lowest level of SES, and 5 represents the highest.

<sup>2</sup>Product of the two independent effects (PR<sub>Water</sub> X PR<sub>Sanitation</sub>). 95% Confidence limits calculated using the delta method.

<sup>3</sup>p-values calculated using the delta method.

<sup>4</sup>Sum of the two independent effects - 1 (PR<sub>Water</sub> + PR<sub>Sanitation</sub> - 1). 95% Confidence limits calculated using the delta method.

**Figure 5.3** The effect of improved drinking water and improved sanitation over time in 217 Demographic and Health Surveys, 1986-2013. Prevalence ratios are for diarrhea comparing those with the improved service to those without, adjusted for child's age and sex, household wealth quintile, and urban/rural residence. In the pooled modified poisson results, pooled models were run for each year, excluding all surveys except those conducted during that year, the year before, or the subsequent year. In the multilevel model results, a linear model of the log prevalence ratio was estimated only for countries with >1 survey.



**Appendix 5.1** Comparison with the water and sanitation categorization scheme of Fink et al. (10)

*Categorization Scheme*

<b>Level</b>	<b>Drinking Water</b>	<b>Sanitation Facilities</b>
<i>Low</i>	Rain water or surface water (ponds, streams, rivers, lakes, irrigation canals, etc)	No facility (open defecation)
<i>Intermediate</i>	Ground water (wells, boreholes, and springs), both protected and unprotected sources	Latrines (both improved and unimproved technologies)
<i>High</i>	Piped water or purchased water	Flush or pour-flush toilets (regardless of the flush destination)

When using the water and sanitation categorization scheme of Fink et al. (10), a stronger relationship is revealed even after adjustment for confounders. Compared to those with low quality water sources, children from households with intermediate and high quality water sources had a lower prevalence of diarrhea (Adjusted PR 0.951, 95%CL 0.935-0.968 and Adjusted PR 0.941, 95%CL 0.923-0.960, respectively). Intermediate quality sanitation facilities provided little benefit relative to those of low quality (Adjusted PR 0.970, 95%CL 0.955-0.987), but high quality facilities showed a substantial protective effect (Adjusted PR 0.871, 95%CL 0.855-0.889).

## **Chapter VI**

### Discussion

#### **Summary**

Sanitation has been among the most important public health intervention of modern times. Many studies have confirmed the health benefits of improved sanitation, but billions of people still lack access. We have used a variety of study designs to show the value of sanitation for improving human health, highlighting many aspects of sanitation that have thus far been overlooked. Specifically, we have investigated the herd protective effects of sanitation, the risks associated with shared sanitation, and the potential effect modification between sanitation and drinking water.

#### **Herd Protection**

We have highlighted, both theoretically and empirically, that improved sanitation in one household can provide herd protection to surrounding households. Because past studies have overlooked these indirect benefits, the true effectiveness of sanitation is likely much greater than our current estimates. By accounting for herd protection, the overall protective effect of sanitation is much larger, making it a much more cost-effective intervention. Other infectious disease interventions have received much more international support after the realization of their herd protective effects. Perhaps the best example is



the oral cholera vaccine, which when first evaluated (direct effect only), did not gain overwhelming support for its use (1-3). After the recognition of additional benefits from herd immunity (4), however, the WHO altered to recommendations to encourage more widespread use of the vaccine (5).

The mathematical modeling in Chapter II serves as an exercise to highlight the mechanisms through which sanitation may provide herd protection. The mechanism of reduced shedding rates into community environments may be the most important and the most straightforward. When sanitation can adequately capture excrement and prevent contamination of neighborhoods, the entire neighborhood will benefit. This can also be interpreted in a negative sense: a single person practicing open defecation may be putting the entire community at risk. The other mechanism whereby sanitation provides herd protection is more convoluted. By reducing shedding rates into household environments, sanitation can prevent within household transmission. This results in fewer infected individuals, which leads to lower cumulative pathogen load in the community environment. The relative importance of these two pathways will depend on pathogen being transmitted and the contextual setting of the community. Pathogens that are readily transmitted via water (e.g., *Vibrio cholerae*, *Giardia*, and *Cryptosporidium*), may be more likely to show stronger herd protective effects from sanitation. Other pathogens that are less likely to be transmitted via water (e.g., *Shigella* and rotavirus) may exploit within household transmission pathways, limiting the herd protective effects of sanitation. Future empirical research on sanitation and diarrhea should seek to identify which pathogens are blocked.

Our empirical work in Chapter III shows the importance of study design for capturing the total effect of sanitation. A simple analysis that ignored herd protection

would have drastically underestimated the effect of sanitation on child growth. Special study designs that can compare individuals from different types of neighborhoods with different levels of coverage are required. Our study holds two advantages over previous studies on this topic. First, we use a longitudinal study design and monitor growth of a cohort of children over time. Most previous studies were cross-sectional in nature. Second, we censused entire communities to gain a more accurate measure of sanitation at the household and community level. Most previous studies were cluster-based surveys, and only subsets of households were sampled in a given cluster. Also, many of these surveys were large house-household surveys, such as the Demographic and Health Surveys, which have an underlying level of measurement error in sanitation exposures. This study, however, is limited by the fact that we did not measure breastfeeding practices nor food security and diet, both of which may be confounding this relationship. A study is currently underway to provide more details of the causal relationship between sanitation, other exposures, environmental enteropathy, and child growth (6).

The evidence that improved sanitation (both at the household and community level) leads to better child growth outcomes is a timely contribution to the literature. There is increasing interest in the role of sanitation in the development of environmental enteropathy, which can lead to worse outcomes such as stunting or death. Environmental enteropathy has also been implicated in the poor performance of oral vaccines for rotavirus, polio, typhoid, and cholera. All of these vaccines have shown a greater efficacy in high-income countries than in low-income countries where they are most needed. While there are likely many causes to their reduced efficacy, improving the sanitation environment in these countries may have a two-fold impact on enteric infections: by

reducing exposure to enteric pathogens and by improving gut health and increasing efficacy of these vaccines. Current polio eradication efforts could be greatly enhanced by increased investments in toilets and latrines.

### **The Demographic and Health Surveys**

We have presented two separate analyses using data from the Demographic and Health Surveys (DHS). These datasets have proven useful in the presentation of descriptive statistics by international monitoring organizations, such as the JMP's reporting of access to drinking water and sanitation. The surveys have also been used in hundreds of journal articles in PubMed. The vast majority of these articles use a single survey in a given country. One study used the 2004 Bangladesh survey to show that sanitation coverage at the cluster level is associated with a child growth (7). A few studies, however, use multiple surveys. Fink et al (8) used every available survey (n=187) to look at the association between drinking water, sanitation, and various child health outcomes. This paper presented very valuable global estimates pooled across dozens of countries and several decades and has been highly cited. In both of our pieces using the DHS data, we also used every available survey (n=217) to present pooled global estimates.

We also, however, recognized a relatively high level of heterogeneity across countries, suggesting that these pooled estimates were an oversimplification of global situation. We found that in several countries, shared sanitation was actually protective even though the global estimate showed a moderate level of risk. This underlying heterogeneity has several potential sources. First, it may be representative of true heterogeneity. Contextual factors that vary across countries or over time may be modifying

the effect of these exposures. Second, there is an inherent level of sampling error, and a certain level of heterogeneity is expected. This may explain much of the pattern that we see but not all. For example, it does not explain the longitudinal trend we observed in the effect of improved sanitation. Third, while effort is made to make these surveys uniform, there is likely measurement error both of the exposures and of the outcome (diarrhea). These surveys rely on self-reporting, which may lead to misclassification of sanitation infrastructure. Even if misclassification of the exposures is non-differential with respect to the disease (and vice versa), the degree of error may vary across surveys, creating observed heterogeneity where there may be none. Misclassification of diarrhea likely occurs because there is no specific case definition of diarrhea, and most surveys employ a 2-week recall period (9).

Accounting for confounding proved to be a substantial challenge with the DHS datasets. All observational studies of sanitation and drinking water are subject to substantial confounding, since use of these services is highly correlated with socioeconomic status (SES), which is a risk factor for diarrhea via pathways such as contaminated food, hand hygiene, and breastfeeding. In both of our studies, adjusting for socioeconomic status resulted in substantial attenuation of the measures of association. The somewhat puzzling result was that socioeconomic status had a stronger effect on diarrhea than the sanitation and water exposures. This may be because alternative transmission pathways, such as food and hygiene are more important than drinking water or sanitation. It may also occur if our measure of drinking water and sanitation do not accurately capture the risk from these exposures. In this case, our measure of SES may be a better indicator of risk from these exposures.

## **Shared Sanitation**

Our work on shared sanitation and the risk of diarrhea fills an important gap in the literature on this topic. As discussed in Chapter IV, a systematic review found that very few studies addressed this issue, and many of those that did had serious methodological flaws (10). We attempted to address several specific policy questions. First, does using a shared facility increase the risk of diarrhea? We found that the answer to this question varies across the globe, but on average, the prevalence of diarrhea was 5-10% higher among users of a shared facility compared to those using an equivalent facility that is not shared. Second, does risk increase as the number of users increases? We found no association between the prevalence of diarrhea and the number of households using the facility. Third, how does the risk associated with shared sanitation compare to that of open defecation and other sanitation practices? For the global average, we found a gradient of risk along the sanitation ladder in the expected direction.

An important finding was that the observed relationship between shared sanitation and diarrhea was substantially confounded by socioeconomic status. It should come as no surprise that those using a shared facility tend to be poorer than those that have their own facility (11). Most previous studies failed to properly account for confounding (10). After adjustment for socioeconomic status, we saw substantial attenuation in the measure of association between sharing and diarrhea. The association, however, was not nullified, suggesting that there is some increased risk from sharing. Though the effect size was small, it is important to keep in mind the amount of measurement error inherent in the DHS data.

The effect of sharing varied across countries. This makes it difficult to decide on a single global policy. It also highlights the need for field studies that can capture a greater level of detail and nuance in sharing practices. Specifically, studies should focus on ownership, management, and maintenance to identify situations where sharing may be safe, though perhaps not ideal.

We also found that using shared sanitation tended to be safer than practicing open defecation. Future research could focus on the cost effectiveness of shared sanitation interventions. Shared sanitation can reach more individuals at a lower cost, but there are some increased risks. These analyses could focus on whether shared sanitation, particularly in urban slums and other areas with very low coverage, is more cost effective than private sanitation.

Our work does not shed light on the mechanism through which using a shared facility increases risk of disease. Transmission may occur within the facility if it is dirty. In this case, fomite transmission would be important. Also, contamination may reach the wider community if the shared facility is not emptied and cleaned in a safe way or if potential users opt for less hygienic practices such as open defecation. Future research could employ laboratory data to determine whether sharing is associated with infection with certain pathogens. An association with water-borne pathogens such as *V. cholera* or *Cryptosporidium* might suggest that transmission is occurring outside of the facility. An association with a fomite-transmitted pathogen, such as rotavirus, might suggest that transmission is occurring within the facility.

## **Joint Effects**

In Chapter V, we provided globally pooled estimates of the effects of water and sanitation on diarrhea. Fink et al (8) published a similar analysis in 2011. We extended their work by using additional recent surveys, showing differences among survey-specific estimates, and identifying a decreasing longitudinal trend in the effect of sanitation. Our pooled estimates were somewhat smaller than theirs, and we provided several reasons for this, the most interesting of which was the longitudinal trend in the effect of sanitation. In previous decades, the DHS datasets suggest that the effect of improved sanitation was strong, but that effect has decreased substantially in recent years. While this observation could be an anomaly, it also warrants further research, as potential explanations for this decline have important implications. Future studies should seek to identify whether the quality of improved sanitation is gradually declining or whether environmental conditions are improving.

We also extended the work done by and Esrey (12) in 1996 investigating the statistical interaction of drinking water and sanitation. As shown on the F Diagram (Figure 1.1), these two services may act on the same or different pathways of disease transmission. If low quality sanitation results in contaminated drinking water, then water treatment and improved sanitation may be preventing the same cases of diarrhea. Empirically, the effect of a single intervention alone would be just as strong as both interventions together. If this pattern existed in the data, it would have important implications for these infrastructure programs. For example, more cases of diarrhea would be averted if sanitation programs targeted areas without improved drinking water. The data, however, show that there was little effect modification between sanitation and water. This suggests that both interventions are needed to prevent the most number of cases of disease. We did not

inspect the heterogeneity of this interaction across countries, but we did see that in more recent years a synergistic pattern appears where neither intervention alone had much of an effect, but both together were protective. This suggests that both are needed to prevent disease. Because the transmission of enteric pathogens can vary dramatically by context, further research should seek to identify whether drinking water and sanitation show different types of interaction for some pathogens or under different circumstances.

## **Conclusion**

Access to sanitation and drinking water has increased substantially over the past few decades (13). The proposed Sustainable Development Goals aim for universal access by 2030 (14). Experience suggests that global progress will likely slow, as more and more resources are required to reach the most disadvantaged populations. The work of this dissertation is especially relevant. Through our work on herd protection, we highlight the importance of sanitation coverage, but also the possibility that, similar to vaccines, 100% coverage may not be necessary to eliminate transmission. Shared sanitation is an increasingly population sanitation option, especially in urban slums. We show that there is increased risk with this practice, but that it is an incremental improvement from open defecation. Finally, sanitation and drinking water are complementary services and their effects do not cancel each other. Efforts should be made to increase coverage of these services in the same areas.



## References

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