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ABSTRACT

Ending open defecation in the developing world has gained significant policy attention recently, motivated by the idea that private demand for latrines lies below the social optimum. We investigate the mortality externalities of poor sanitation by exploiting differences in latrine demand between Muslim and Hindu households in India: Muslims, despite being poorer, are 25 percentage points more likely than Hindus to use latrines or toilets. Instrumenting for local sanitation with the religious composition of neighborhoods, we estimate large infant mortality externalities. Our findings are informative of the external harm generated by the one billion people today who practice open defecation.

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The past two centuries have produced the fastest declines in human mortality ever experienced. Much of this gain has come from declines in infant deaths. Economic historians examining the epidemiological transition to low infant mortality in developed countries have debated whether it reflects changes in nutrition, medical care, or the disease environment. A leading candidate explanation is improvement in sanitation: the safe confinement of human feces. Understanding the role of sanitation in driving health and mortality is relevant even today, because more than a billion people continue to defecate in the open—for example, in fields, behind bushes, or near roads.

In this paper we examine the infant mortality externalities of open defecation. Ending open defecation has become a target of governments, NGOs, and private foundations. For example: ending open defecation was adopted in September 2015 among the UN's new Sustainable Development Goals; in India in 2014, the central government launched a 100-day construction plan during which one toilet or latrine was to be constructed every second; from 2007 to 2014, the World Bank Group committed an average of over $3 billion per year to water and sanitation; and since 2005, the Gates Foundation has invested hundred of millions of dollars to improve sanitation in developing countries, including efforts to "reinvent the toilet." In parallel, researchers have investigated the economic (Guiteras, Levinsohn and Mobarak, 2015) and social (Guiteras et al., 2014) determinants of persistent open defecation. The rationale underlying much of this recent attention is that private demand for toilets and latrines may be below the social optimum, possibly because of inaccurate beliefs about the health benefits of improved sanitation, or because latrine use has an important public goods component. The public goods aspect of sanitation is indeed plausible. Epidemiological evidence and recent work by Duflo et al. (2015) suggest clear pathways by which exposure to fecal pathogens introduced by neighbors could lead to acute net malnutrition and ultimately death.[1]

There are several reasons why, despite significant interest in and spending on this issue, the externalities of open defecation are not yet well understood. For one, OLS estimates are likely to be biased because neighborhoods with worse sanitation practices are likely to be worse in other unobservable dimensions as well. Further, efforts to estimate the impacts of sanitation via field experiments have been complicated by difficulties in generating a first-stage effect on latrine use: In many places there exist deeply rooted preferences against using latrines and toilets, even those freely provided.[2]

[1] We discuss the epidemiological evidence in detail in Section 3. Duflo et al. (2015) show that an integrated water and sanitation improvement program in rural India reduced diarrhea episodes by 30-50%.

[2] The difficulty in generating a large first stage effect has been demonstrated by three recent field experiments in rural India, each intending to estimate the effect of open defecation on child height (Hammer and Spears, 2013; Clasen et al.,

Indeed, the difficulty of changing latrine use behavior, combined with the large number of neighborhood clusters required to measure mortality externalities, may explain the lack of any experimental evidence on sanitation and mortality to date.[3]

In this paper, we provide the first econometric evidence on the infant mortality externalities of poor sanitation by exploiting a systematic difference in latrine use between Hindus and Muslims in India. More than half of India's population of 1.2 billion do not use toilets or latrines, either of which can serve to safely dispose of waste. Despite relative economic advantage, India's majority Hindu population is 25 percentage points more likely to defecate in the open than the minority Muslim population. This Hindu-Muslim behavioral difference implies that the fraction of a household's neighbors who are Muslim is strongly correlated with the local sanitation environment to which the household is exposed. For example, in nationally representative data, Hindus residing in neighborhoods that are 10% Muslim are exposed to a local open defecation rate of 63%, while Hindus residing in neighborhoods that are 90% Muslim are exposed to a local open defecation rate of 46%. In contrast to this difference in sanitation practices, neighborhoods where Muslim populations are concentrated are no different or are worse in terms of other observable characteristics that predict infant health outcomes, consistent with the well-known relative disadvantage of Muslims in India.[4]

To better understand the latrine use preferences that we exploit for identification, we fielded a survey in rural northern India that was designed to elicit richer detail on stated and revealed preferences toward latrine ownership and use. We show that Hindus, but not Muslims, are likely to report that using a toilet or latrine near their home is "impure" and that defecating away from the home in the open is part of a healthy lifestyle. Even conditional on ownership of a working latrine, our survey shows Hindus are less likely to *use* it. The idea that latrine demand could be significantly influenced by factors beyond price and wealth accords with recent qualitative work in India (Coffey et al., 2014*a*) and experimental interventions in Bangladesh (Guiteras et al., 2014). Such studies have documented significant resistance to the adoption of effective, low-cost improvements to water quality and sanitation in similar settings.

Our main analysis instruments for local sanitation with the religious composition of a neighbor-

2015; Gertler et al., 2015).

[3]The number of neighborhood clusters required to detect even economically large mortality effects via a field experiment is large both because of the plausible effect size relative to the variance in mortality and because measuring local externalities necessarily implies randomization at the level of the locality, not the individual. We discuss the issue in Appendix A.1.

[4]See, for example, Sachar et al. (2006) and Deolalikar (2008).

hood. We motivate the plausibility of our research design by first showing that accounting for this latrine demand difference solves a long-standing puzzle in the development and health literature: In India, Muslim children are substantially more likely than Hindu children to survive to their first birthday, even though Muslims have lower wealth, consumption, and educational attainment, and face worse access to state services liked piped water and health infrastructure, compared to the majority Hindus.[5] By age one, mortality among Muslims is 17% lower than among Hindus, with an additional 1.1 infants per 100 surviving. Bhalotra, Valente and van Soest (2010) named this robust and persistent pattern a "puzzle," showing that individual and household characteristics could not explain it.[6] In a series of reduced-form OLS regressions, we show that this large difference can be entirely accounted for by two facts: (i) Compared to the typical Hindu infant, the typical Muslim infant lives in a neighborhood where a larger share of her neighbors are Muslim; and (ii) Muslim neighbors are much less likely to defecate in the open.

In IV regressions, we find that a 10 percentage point reduction in the fraction of neighbors defecating in the open is is associated with a decline in infant mortality of 6.5 infants per 1,000, or about 9% of the population mean infant mortality rate (IMR).[7] These regressions control for own religion and own sanitary practice and are identified solely off of the variation in local open defecation arising from the religious composition of one's neighbors. By replicating our main IV results within various subsets of the data—for example, boys and girls separately, first-borns and later-borns separately, and rural and urban neighborhoods separately—we show that our findings are robust and are not confounded by phenomena like differential son preference by Hindus and Muslims, birth order explanations, or differential sorting of Muslims to urban areas.

More generally, we show that the variation in local sanitation that arises from these preference differences is not positively correlated with health inputs or economic well-being across the localities differentially settled by the groups. Instead, to the extent observables other than sanitation covary

[5]This phenomenon, which has existed since at least the 1960s and which has been documented by Shariff (1995), Bhat and Zavier (2005), Bhalotra and Soest (2008), and Bhalotra, Valente and van Soest (2010), is hard to reconcile with the well-developed literature on the importance of parental income in predicting child health and mortality. For overviews of the literature and applications in the US, see Cutler and Lleras-Muney (2010) and Geruso (2012).

[6]Bhalotra, Valente and van Soest (2010) carefully demonstrated that individual and household characteristics, development expenditure, and village-level health services and health infrastructure could not account for the Muslim mortality advantage.

[7]To give a sense of how large a 10 percentage point decline in open defecation would be, we note that despite India's rapid economic growth over the last two decades and a large government investment specific to open defecation (Spears, 2013), open defecation rates have fallen in India by no more than 1 percentage point per year between 2001 and 2011 on average (Government of India, 2011).

at all with Muslim concentration, they would imply worse child health outcomes, suggesting that our estimates may be lower bounds on the true effects. Because diet and washing are two important health behaviors that may be expected to covary with religion and affect infant health, we turn to an additional dataset to demonstrate that our instrument is uncorrelated with variation in washing and diet at the neighborhood level. For example, although Hindus are more likely to be vegetarian than Muslims, Hindus living around many Muslims are not differentially likely to eat meat than Hindus living around few Muslims, which is the variation we exploit.

The effects we estimate are large, consistent with prior evidence on the importance of water and sanitation in driving infant health outcomes. In the context of our data, the difference between a locality where all residents defecate in the open and a locality where no one does is associated with about the same infant mortality reduction as the difference between the bottom and top quintiles of (own) wealth. The implied importance of open defecation for infant mortality in the modern Indian context fits with evidence by Cutler and Miller (2005) on the role of clean water and sanitation in explaining large shares of declining infant mortality in the US at the turn of the twentieth century.[8] Our estimates are also consistent with the most credible data on cause of death during early-life in India, which show that a large share (22%) of non-neonatal child deaths in India are due to diarrhea, which is just one symptom of the intestinal diseases transmitted via open defecation.[9] Indeed, we confirm in a mechanism check that low current weight among infants, which reflects recent bouts of diarrhea, responds to our identifying variation in the same pattern as mortality does. A mechanism check on the interactive effects of breastfeeding and local sanitation likewise supports the causal channel we describe.[10]

Besides informing current policy, our results contribute to the broader literature on water, sanitation, and disease environment, which have been found to be important determinants of health

[8] Cutler and Miller (2005) show that the introduction of clean water technologies in US cities beginning around 1900 reduced the infant mortality rate by 46% and accounted for 74% of the total decline in US infant mortality at the turn of the century. To compare to India, IMR in India declined by 25 deaths per thousand over a decade (2001 to 2011) while open defecation simultaneously declined by about 8 percentage points. Naively applying our IV estimates would predict a change in IMR of -5.2 $(= 65 \times -0.08)$, which would account for about 20% of the total decline in IMR over that period.

[9] See Million Death Study Collaborators (2010).

[10] In this type of disease environment, exclusive breastfeeding has the potential to significantly impact health and human capital, with greater relative gains where sanitation is worse. This is because breastfeeding creates a natural barrier against germs, even if the nursing mother ingests those germs. We investigate the interaction of breastfeeding and local sanitation, under the hypothesis that infants who consume water and non-breastmilk food are more likely to ingest the fecal pathogens introduced by neighbors than those who exclusively breastfeed. Our analysis confirms that the efficacy of breastfeeding is increasing in the fraction of one's neighbors who defecate in the open—and for that reason only, increasing in the fraction of one's neighbors who are Hindu.

and human capital outcomes around the world (Spears and Lamba, 2014; Gertler et al., 2015; Headey et al., 2015), as well is in the historical of development of the US (Cutler and Miller, 2005; Watson, 2006; Bleakley, 2007).[11] Our study is unique in the literature in examining the external impacts of open defecation on mortality. Establishing whether the harm from open defecation is primarily external is an important starting point for understanding behavior that is socially suboptimal and for justifying any policy intervention on the grounds of efficiency.

The remainder of the paper is organized as follows: We begin in Section 2 by describing the context of open defecation in India and by documenting the large difference in demand for latrine use between Hindus and Muslims. In Section 3 we provide more detail on the plausible channel linking open defecation to infant deaths. Section 4 describes the main dataset and research design. In Section 5, we resolve the Muslim mortality puzzle and motivate our IV analysis. Section 6 reports the IV results and presents several tests of the channel we describe. Section 7 concludes.

2 Hindu-Muslim Differences in Demand for Latrine Use

> *Far from his dwelling let him remove urine and excreta*
>
> –The Laws of Manu (a Hindu sacred text), Chapter 4 verse 151[12]

We begin by presenting summary statistics documenting the pattern of behavior central to our research design. More than half of the Indian population, over 600 million people, defecate in the open, without the use of a latrine or toilet. The prevalence of open defecation (hereafter OD) is particularly high among India's Hindu majority. Data from the National Family Health Survey (NFHS) of India show that 68% of Hindu households defecate in the open—e.g., in fields, near streets, or behind bushes. In comparison, only 43% of the relatively poorer Muslim households do so.[13]

To investigate these patterns in more detail, we turn briefly to the Sanitation Quality, Use, Access,

[11] Cutler and Miller (2005) examined the introduction of clean water technologies into US cities, estimating large effects on infant and child mortality. Watson (2006) studied federal interventions to improve water and sanitation on US Indian reservations, finding effects on local infant mortality and spillovers to neighboring localities. Bleakley (2007) studied the eradication of hookworm in school-aged children in the US South at the turn of the twentieth century, documenting impacts on school enrollment and attendance and on later-life income.

[12] For a parallel example from a Muslim sacred text with opposing advice, the Mishkat-al-Masabih includes the passage: *Muadh reported God's messenger as saying,"Guard against the three things which produce cursing: relieving one self in watering-places, in the middle of the road and in the shade."*

[13] We describe our main analysis dataset, the NFHS, in more detail below.

& Trends survey, which was collected by one of this study's authors in rural northern India in 2013 and 2014. Unlike our main analysis dataset, the NFHS, this survey was specifically designed to elicit preferences over latrine use. We use it here to provide a clearer context and understanding of the stark demand differences we exploit below. The survey is described in more detail in Coffey et al. (2014*b*).[14]

Table 1 summarizes responses at the individual and household levels, with Hindu means in column 1 and Muslim means in column 2. Consistent with other data sources, the first row of the table shows that Hindus are significantly more likely than Muslims to practice OD—that is, they are less likely to use a latrine or toilet. This difference in revealed preferences is deeply embedded: The second row shows that a substantial minority of Hindus who reside in a household with a working latrine nonetheless choose to defecate in the open. 25% of Hindus who own functional latrines choose not to use them, compared to 10% of Muslims. These findings are consistent with accounts from Indian commentators (e.g. Ramaswami, 2005) and evaluations by researchers (e.g. Barnard et al., 2013) that toilets constructed or paid for by the government often remain unused or repurposed by Hindus.

The roots of these behavioral differences are difficult to trace and are beyond the scope of this paper. Sanitation practices may have evolved differently across Muslim and Hindu communities for purely secular reasons, and could have been privately or socially optimal given the context under which they arose (Mobarak, Levinsohn and Guiteras, 2014). Even specific religious instruction with respect to sanitation and hygiene that we observe today may have been established long ago—codifying then-existing norms, rather than establishing those norms. Regardless of the historical path, we show here that religion is a highly predictive marker for group differences.

While we take no position on whether religion causes OD *per se*, it is common for Indians to discuss waste disposal choices with reference to religious purity. Row 4 of Table 1 shows that a substantial fraction of both Hindu and Muslim respondents self-report that a religious leader has told them explicitly where to defecate. The last two rows of Table 1 show that Hindus are more likely than Muslims to respond that OD away from the home is pure, while using a latrine near the home

[14]See the Sanitation Quality, Use, Access, & Trends (SQUAT) Survey. The SQUAT survey was designed to be representative of rural open defecation practices in five states of north India: Bihar, Haryana, Madhya Pradesh, Rajasthan, and Uttar Pradesh. These states are home to 40% of the population of India, and to 45% of households in India without a toilet or latrine. Surveyors interviewed 3,235 adults about their defecation practices and views on latrines and latrine use, and collected individual level latrine use data for 22,787 household members.

is not pure. All Hindu-Muslim differences in the table are statistically significant at the 5% level.

The possibility that open defecation would ever be chosen if a working toilet or latrine were available may be surprising to many readers, but these patterns, and indeed the Hindu-Muslim behavior difference itself, are well known to many residents of rural India. Coffey et al. (2014*a*) documents this fact in a qualitative study in rural northern India, recording, for example, how a Muslim woman from Uttar Pradesh described the differences: "Even if Hindus have made a latrine, still they go out to defecate in the open. Now for our people [Muslims], it's not a problem. If we have a latrine in the house, we will use it."[15] Moreover, this behavior has long been recognized and documented publicly: Cultural scholars attribute the modern persistence of OD among Hindus in India to the persistence of the Hindu caste system, with its ritual avoidance of excreta (Ramaswami, 2005; Bathran, 2011). Recently, Hindu politicians across the political spectrum have publicly recognized this pattern.[16] And nearly a century ago, Gandhi campaigned to change Indian behavior with respect to excreta disposal, famously declaring, "Sanitation is more important than independence."

In short, the prominence of OD among Hindus is not merely a matter of the affordability of latrines and toilets. Instead, Hindus report and reveal clear preferences against using latrines. Indeed, we show below in nationally representative data that the Hindu-Muslim demand difference holds at all levels of wealth. This demand difference is key in our identification of the mortality externalities of latrine use.

3 Sanitation and Health

Here we briefly outline the mechanism linking infant mortality to externalities associated with open defecation (OD), drawing on the economic and epidemiological literature. Bacteria and parasites, such as worms, live in feces. Fecal matter in the local environment gets onto feet and hands and into mouths directly. It can also contaminate food and water. These pathogenic processes have been documented since at least the 19th century.[17] By definition, open defecation is not limited to a con-

[15] Coffey et al. (2014*a*) also reports how a Hindu man in Haryana described his beliefs about the health benefits of open defecation: "[By defecating in the open] one can stretch the body, one can go out for a walk. You can also prevent yourself from getting diseases. If a latrine is in the house, bad smells will come, germs will grow. Latrines in the house are like...hell. The environment becomes completely polluted."

[16] Hindu politicians from both major political parties in India have echoed this sentiment with the slogan: "Toilets are more important than [Hindu] Temples." Union Rural Development Minister Jairam Ramesh of the Congress party made the statement in October 2012. From the BJP, Gujarat Chief Minister and then-candidate for Prime Minister Narendra Modi made an identical statement in October 2013.

[17] See Freedman, 1991 for examples.

fined or designated area. Feces are disposed of in crop fields, near homes, and in and along roads, implying widespread scope for disease transmission and potential harm, regardless of whether an individual chooses to use a latrine himself.

For children, infections caused by fecal pathogens can reduce net nutrition through caloric loss to diarrhea and parasites, as well as by expending calories to combat infections. Acute malnutrition due to infectious disease is well established in the epidemiological literature (Kielmann and McCord, 1978; Mosley and Chen, 1984) and recognized among economists (e.g. Cutler, Deaton and Lleras-Muney, 2006). Epidemiological evidence also suggests that exposure to fecal pathogens could lead to enteropathy—a chronic intestinal problem that prevents the proper absorption of calories and micronutrients (Humphrey, 2009; Petri et al., 2008; Mondal et al., 2011; Lin et al., 2013). The resulting acute malnutrition manifests as wasting (low weight) and can lead to death (Black, Morris and Bryce, 2003). We check this mechanism directly in Section 6 by examining the intermediate outcomes of weight-for-height and weight-for-age.

For neonates (0-1 month old), the process is somewhat different but with the potential for similar mortality effects. Maternal exposure to fecal pathogens could cause neonatal mortality by reducing the quality of maternal net nutrition during gestation, in turn reducing uterine growth and birth weight. This possibility is highlighted by the recent finding in Prendergast et al. (2014) of a correlation between in-utero growth, growth hormones at birth, and mothers' exposure to open defecation in Zimbabwe.

The public goods features of sanitation have been highlighted in the economics literature. Mobarak, Levinsohn and Guiteras (2014) investigated the determinants of latrine use in Bangladesh via a field experiment, finding an important role for local complementarities. Other studies have examined health outcomes impacted by the public goods problems of sanitation, though these have focused almost exclusively on worms, which comprise just one channel by which OD could affect health. In the Kenyan context, Miguel and Kremer (2004) studied the public goods problems associated with intestinal worms transmitted by contact with fecal matter. In the historical US context, Bleakley (2007) examined the impact of efforts by the Rockefeller Foundation at the turn of the century to eradicate widespread hookworm infections in the US South, which were spread by contact with human feces and caused anemia and stunting in children.

Taken together, the economic literature supports the notion of important externalities of OD,

while the epidemiological evidence suggests that health impacts could be significant and include mortality effects.

4 Data and Research Design

In this section, we describe our data and estimation strategy, in which the identifying variation is generated by Hindu-Muslim differences in latrine preferences combined with heterogeneity across localities in the composition of residents. Our variation is uniquely suited to estimating local mortality externalities because it generates substantial variance in open defecation at the neighborhood-level.

4.1 Data

For our main analysis, we use data from three rounds of the National Family Health Survey (NFHS) of India: 1992/1993, 1998/1999, and 2005/2006. The NFHS (India's version of the Demographic and Health Survey) is a large, nationally representative survey that collects data from women aged 13 to 49. Respondents report birth histories, including deaths and stillbirths, from which we calculate infant and neonatal mortality rates. The NFHS is also includes information on household assets, household physical infrastructure, and health behaviors. With respect to the disposal of excreta, the respondents are asked about the type of toilet facility, if any, the household usually uses. We code a household as practicing open defecation (OD) if they report using no facility, or using a bush or a field.

Table 2 tabulates the summary statistics for our main analysis sample, which consists of Hindus and Muslims in all waves of the NFHS. Corresponding to the analysis below, children (live births) are the unit of observation.[18] Our primary outcomes of interest are the infant mortality rate (IMR) and the neonatal mortality rate (NMR), defined respectively as the number of deaths among children less than one year old and less than 1 month old, scaled per 1,000 live births. We focus on these mortality outcomes as they are measured closest in time to the open defecation measurement that comprises our variable of interest.

Infant mortality is high across India, and consistent with previous studies, there is a large and significant Muslim survival advantage. Table 2 shows that across both groups, more than 6 children in 100 will die before their first birthday. Comparison of the Hindu and Muslim means shows that

[18]Therefore, these averages are representative of young children and their households, not of all of India

for every hundred live births, 1.1 fewer Hindu children will survive to age one, implying infant mortality is 17 percent higher among Hindus. Neonatal mortality shows a similar pattern, with a 19 percent survival deficit. This is despite Muslims having lower educational attainment and wealth, measured in this survey by assets. Though the NFHS does not measure consumption, the India Human Development Survey, which is used in a series of robustness checks below, shows that mean and median consumption are higher among Hindus than Muslims as well.[19]

With respect to open defecation, 32% of Hindu children in the sample live in households that use latrines or toilets, compared to 57% of Muslim children. The summary statistics on infrastructure and assets in Table 2 show that Hindu—but not Muslim—households are much more likely to have electricity than to use a private or public latrine. In addition to being more likely to use latrines themselves, Muslims are more likely to have neighbors who do so: Because residents tend to collocate along religious lines, local open defecation is higher for Hindu children by a margin of 21 percentage points.

4.2 Identifying Variation

We exploit variation in the local sanitation environment that arises from the religious composition of a household's neighbors. Despite significant residential sorting along religious lines, 31% of survey primary sampling units (PSUs) contain some fraction of Muslim residents strictly between zero and one.[20] The median survey PSU contains observations on 27 households, which are sampled from PSU-level frames of about 100-200 households.[21] Therefore, PSU means reflect the characteristics of small localities.

For each PSU j in the NFHS, we calculate the fraction of sample residents that are Muslim and call this Muslim concentration ($\overline{M_j}$). We also calculate the mean open defecation rate in the PSU ($\overline{OD}_j$) and the mean open defecation rate of neighbors in the PSU ($\overline{OD_{ij}^{-i}}$), where the superscript $-i$ indicates a leave-out mean, taken over all households in PSU j other than the respondent household.

Figure 1 plots the histogram of Muslim concentration, $\overline{M_j}$, across PSUs, with the point mass at

[19] The unconditional mean and median consumption among Hindus in the IHDS is 985 and 710 rupees per month per capita, respectively. The unconditional mean and median consumption among Muslims is 831 and 623 rupees per month per capita, respectively.

[20] The NFHS is a two-stage random sample, first sampling PSUs and then households.

[21] Our data do not contain the sampling frame, but according to the NFHS-3 report, rural PSUs are villages of "usually about 100 to 200 households." Large villages above 500 households were split into three possible PSUs. Urban PSUs are census enumeration blocks (approximately 150-200 households).

zero (= 65%) excluded to maintain a readable scale. Although Muslims make up a small fraction of our births sample, there is support in the distribution at very high levels of Muslim concentration, including all-Muslim localities. The histogram shows that between the extremes of perfect segregation, there is substantial variation in $\overline{M_j}$. Figure 1 also illustrates the identifying variation in sanitation by overlaying a local polynomial regression of $\overline{OD_j}$ on $\overline{M_j}$ at the individual level. In neighborhoods with close to zero Muslim residents, open defecation averages nearly 70%. This decreases monotonically in Muslim concentration, with residents in all-Muslim neighborhoods facing local open defecation rates below 40%.

The pattern in Figure 1 suggests instrumenting for $\overline{OD_j}$ with $\overline{M_j}$. This strategy would yield unbiased estimates of the external effect of local open defecation on mortality if the religious composition of the PSU predicts own-infant mortality only through its association with nearby neighbors' open defecation. However, because the Muslim minority population is widely documented as being disadvantaged relative to Hindus (e.g., Sachar et al., 2006; Deolalikar, 2008), a reasonable prior is that Muslims would reside in neighborhoods that are systematically worse for child health along dimensions other than sanitation.

To investigate how neighborhood-level characteristics covary with Muslim concentration, we examine the extent to which variation in Muslim concentration across PSUs predicts observables. Figure 2 plots local polynomial regressions in which a set of ten individual-level variables capturing characteristics of children, parents, and their neighborhoods are regressed on $\overline{M_j}$. For ease of interpretation, we define each of the variables such that their range is $[0, 1]$ and higher values predict better child health outcomes.[22] The dependent variables include asset wealth (as the fraction of the seven assets reported in all survey rounds) and indicators for household electrification, household piped water, institutional delivery of the child, any child vaccinations, family possession of a national health card, mother having a say in child's healthcare, mothers' literacy, and latrine use. In Panel A, we include all PSUs in the data. In Panel B, we restrict the sample to the set of mixed-religion PSUs ($0 < \overline{M_j} < 1$).

Figure 2 shows that latrine use (defined as one minus the indicator for open defecation) is highly positively correlated with $\overline{M_j}$, while other characteristics are relatively flat or declining in $\overline{M_j}$. The pattern is particularly clear and approximately linear in Panel B of Figure 2, which excludes reli-

[22] We confirm the sign of the relationships between these variables and mortality in Appendix Table A2.

giously homogenous PSUs. Appendix Table A1 reports the coefficients from the linear regressions corresponding to the local polynomials plotted in Panel B of Figure 2. The table also performs this test for additional household, parental, and child characteristics beyond those plotted in the figure. In Appendix Table A2, we confirm the sign of the relationships between each of these characteristics and infant mortality by regressing IMR on each of the dependent variables in Table A1. Together, Figure 2 and Tables A1 and A2 show that among variables likely to cause or to be correlated with child health outcomes, all are either uncorrelated with $\overline{M_j}$ or are changing in a way that would predict *worse* child health as Muslim concentration increases. In Section 6.2, we turn to an additional dataset, the India Human Development Survey, to show that our instrument is likewise uncorrelated diet and hygiene behaviors that are not contained in our main dataset.

The *only* exception to this pattern in which higher Muslim concentration predicts neutral or worse PSU characteristics is $\overline{OD_j}$, our variable of interest. Urban/rural status and household size show some association, but not after controlling for own household characteristics (own latrine use and own religion). Our IV estimates below are identified off of the composition of neighbors only, always controlling for own household OD and own household religion. Nonetheless, because Muslims are more likely to locate in urban areas, we control for urban/rural status in the analysis below. As a robustness check, we also replicate all results separately in urban and rural areas. Likewise, Muslims in our sample tend to have larger families, and because mean household size is in turn mechanically linked to mean birth order, we also show that results are robust to stratifying the analysis by birth order, estimating results separately by first births and second or later births. This removes the possibility that the differential composition of mean birth orders across the Hindu and Muslim sub-samples, combined with differential investment in children by birth order, could be confounding the results.

The finding that Muslim-dominated neighborhoods are no better and possibly worse for infant survival along observables also accords with popular beliefs and observations about the relative disadvantage of the Muslim minority India.[23] In sum, the evidence suggests that instrumenting for $\overline{OD_j}$ with $\overline{M_j}$ would, if anything, understate the effects of interest. Formally, we do not rely on the exclusion restriction holding with equality, but rather as an inequality.[24] As we show below, the IV

[23] For example, Sachar et al. (2006) and Deolalikar (2008).

[24] Proof, for the analytically tractable case of no other controls: Assume that the true data generating process for infant mortality is $IMR = \beta \cdot \overline{OD} + \delta \cdot X_{bad}$, where β is positive and X_{bad} is an omitted variable that is positively correlated with

estimates are sufficiently sized that the lower bound is informative in this case.

4.3 Econometric Framework

We organize our analysis at the level of the child, constructing mortality rates from birth history information on around 280,000 Hindu and Muslim children in India over the three survey rounds. We regress mortality outcomes on variables capturing a household's own open defecation (OD_{ijt}) and neighbors' open defecation within the PSU ($\overline{OD_{ijt}^{-i}}$):

$$y_{ijt} = \beta_1 \widehat{\overline{OD_{ijt}^{-i}}} + \beta_2 OD_{ijt} + \alpha_1 M_{ijt} + f(X_{ijpt}) + \epsilon_{ijt}. \tag{1}$$

We instrument for the mean open defecation rate among neighbors with the fraction of PSU households that are Muslim:

$$\overline{OD_{ijt}^{-i}} = \gamma_1 \overline{M_{jt}} + \gamma_2 OD_{ijt} + \gamma_3 M_{ijt} + f(X_{ijpt}) + \mu_{ijt}. \tag{2}$$

Here i indexes live births, j indexes survey PSUs, and t indexes the three NFHS survey rounds.[25] The main dependent variable y_{ijt} is an individual-level mortality indicator scaled so that coefficients reflect impacts on deaths per thousand: y_{ijt} is either 0 if a child survived to the specified age or 1,000 if she did not.[26]

Identification in Eq. (1) arises from the religious composition of neighbors only. The latrine use polynomial plotted in Panel B of Figure 2 visually depicts Eq. (2), for the case of no controls and non-parametric estimation. M_{ijt} is an indicator for the child residing in a Muslim household, which absorbs any unobserved health behaviors or child investments that could be correlated with a household's own religion, such as differing Hindu and Muslim diets. We additionally control for a variety of person, household, and PSU-level characteristics in $f(X_{ijt})$ to demonstrate robustness. These are described in more detail below. We cluster standard errors by PSU.

In Appendix Table A3, we report the linear regression coefficients from Eq. (2) that describe the first stage of our IV analysis. Consistent with the visual evidence in Figure 2, the instrument is strong,

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$\overline{M_j}$ and with IMR. Then estimating β via an IV that omits δ yields: $\widehat{\beta_{IV}} = \frac{\text{cov}(IMR, \overline{M})}{\text{cov}(\overline{OD}, \overline{M})} = \beta + \frac{\delta \cdot \text{cov}(X_{bad}, \overline{M})}{\text{cov}(\overline{OD}, \overline{M})} < \beta$, where the last inequality follows from sign of the second term: The numerator is positive and the denominator is negative.

[25] All specifications include survey round fixed effects.

[26] This construction merely scales mortality rates and coefficients to match the standard of expressing rates per 1,000.

with first stage F-statistics always exceeding weak instrument thresholds. We also estimate the first stage separately within subsamples defined by child's birth order, child's sex, child's *own* religion, and the location of the household in a rural or urban setting in order to demonstrate that the first stage relationship is not driven by an association with any of these variables. Table A3 shows that the first stage effect is strong, precisely estimated, and consistent across subsamples.

In Section 5, we begin by exploiting the association between $\overline{OD_{ijt}^{-i}}$ and $\overline{M_{jt}}$ to solve the long-noted puzzle that Muslim children in India have significantly higher survival rates than Hindus, despite Muslim parents having lower income, education, and status, as well as worse access to state services. In large part, this analysis motivates the plausibility of the IV strategy. In Section 6, we report our main results that instrument for $\overline{OD_{ijt}^{-i}}$ with $\overline{M_{jt}}$.

5 Motivating Puzzle

This section presents reduced form evidence on the relationship between the religious composition of neighbors and own child mortality. We show that patterns of latrine use, driven by the composition of neighbors, can completely account for the large Muslim mortality advantage that has been documented in India, despite the relative economic disadvantage of Muslims there. This section also informs and motivates the IV analysis in Section 6, which exploits the same variation that solves this puzzle.

5.1 The Puzzle

Figure 3 illustrates the mortality puzzle that was most clearly documented by Bhalotra, Valente and van Soest (2010): At all levels of socioeconomic status, mortality is lower among Muslim children than among Hindu children. The figure plots infant mortality, separately by religious group, against two alternative summary measures of household economic well-being. The NFHS, like all DHS surveys, does not measure income or consumption. Therefore, in Panel A we follow the literature (see, for example, Filmer and Pritchett, 2001) in using asset ownership as a proxy for wealth, and collapsing seven categories of asset ownership into a single-dimensional wealth rank within the sample.[27]

[27] We cannot use the pre-constructed asset index included in the NFHS dataset because it is constructed including measures of sanitation. Therefore, following Filmer and Pritchett (2001), we construct a household's asset rank by (1) partitioning the sample into $128 = 2^7$ bins of indicators for ownership of seven assets listed in Table 2; (2) ranking the bins by the average infant mortality rate in each bin; and (3) assigning each household the median rank within the sample of its bin.

This gives the horizontal axis a clear rank interpretation. As an alternative measure of parental endowment, we use mother's height along the horizontal axis in Panel B. Maternal adult height predicts maternal adult health and reflects maternal economic well-being earlier in the mother's life (Case and Paxson, 2008; Steckel, 2009).

Consistent with asset ownership and mother's height capturing meaningful variation in endowments that is correlated with child survival, Figure 3 shows that infant mortality is steeply decreasing in both measures. The Muslim advantage documented by Shariff (1995), Bhat and Zavier (2005), Bhalotra and Soest (2008), and Bhalotra, Valente and van Soest (2010) is apparent in the large and statistically significant mortality differences at any fixed level of either measure of well-being. To put the size of the mortality difference in context, holding either wealth rank or maternal height constant at their medians, Figure 3 shows that about additional 10 out of 1000 infants in Hindu households will die before age one compared to Muslim infants. The figure is consistent with the unconditional difference in means of 11.0 (per thousand) in Table 2.

5.2 The Solution

In Table 3, we analyze the extent to which open defecation (OD) can explain the puzzle. The dependent variables are indicated in the column headers. Observations are children (live births). The dependent variable, an indicator for death, is scaled as described in Section 4 so that coefficient estimates indicate mortality effects per thousand. The top panel includes no covariates beyond indicators for the survey round. The bottom panel adds controls for a set of demographic and socio-economic characteristics that the literature on the determinants of early-life health in India has highlighted.[28]

Table 3 shows that neighbors' open defecation can fully account for the respective 9.8 and 7.1 (per thousand) Hindu-Muslim gaps in infant and neonatal mortality. These mean differences are estimated as the coefficients on the Muslim indicator in columns 1 and 4. Controlling for the fraction of the PSU that is Muslim in columns 2 and 5 removes any association between a household's own religion and the child's mortality. Then incrementally adding controls for own household open defe-

Thus, the household of child 200,000 has more and better assets than 200,000 of the approximately 300,000 children in our sample. Unlike a principal component index, this measure has units with a clear interpretation.

[28]These controls, labeled "extended controls" in Table 3, include a full set of birth order indicators interacted with sex (Pande and Jayachandran, 2013); indicators for household ownership of each of the seven assets asked about throughout survey rounds, the standard strategy for controlling for SES using these data (Filmer and Pritchett, 2001); an indicator for whether the mother lives with her husband's parents (Coffey, Khera and Spears, 2013); indicators for child's birth month (Doblhammer and Vaupel, 2001) and birth year; an urban indicator; household size; and several other individual and PSU-level variables. See the Table 3 notes for the full control list.

cation (OD_{ij}) and the fraction of neighbors that openly defecate ($\overline{OD_{ij}^{-i}}$) attenuates the coefficient on the concentration of Muslim neighbors ($\overline{M_j}$) to a magnitude both economically small and statistically insignificant. The same pattern holds irrespective of the addition of controls in the bottom panel.[29]

These results imply that what has been widely documented as a Muslim survival advantage is actually an advantage accruing to the neighbors of Muslims. The solution to the mortality puzzle consists of two facts. First, compared to the typical Hindu infant, the typical Muslim infant lives in a neighborhood where a larger share of her neighbors are Muslim. Second, Muslim neighbors are much less likely to defecate in the open.

The result that open defecation accounts for the mortality gap is robust to a variety of alternative hypotheses. A natural question in this context is whether differences in son preference and intra-household resource allocation between Hindus and Muslims could confound the results. To address this possibility, in Panel A of Appendix Table A4 we replicate the main IMR results of Table 3, but split the sample by child's sex. The table shows that the Hindu-Muslim gaps in infant mortality are similar across boys and girls. More importantly, in both the boy and girl subsamples, these gaps attenuate to insignificance in exactly the same pattern as in the main table once the measures of open defecation are included. We also investigate whether our results are confounded by the pattern documented in Pande and Jayachandran (2013) in which sex $\times$ birth order interactions predict child health in India. The behavior has the potential for importance here, because Hindu and Muslim household sizes in India differ, and therefore so do the mean birth orders across groups. Nonetheless, this phenomenon does not confound the relationship between IMR and sanitation: All regressions in Panel B of Table 3 include the full set of sex indicators interacted with birth order indicators as controls, and Panel B of Table A4 shows that the pattern of our results holds whether restricting the sample to only first births or to only second or later births. Because residence in an urban area was shown in Table 2 to differ significantly across religious groups, in Panel C of Table A4 we replicate the results separately for urban and rural areas, allowing more flexibility in how urban status is held constant. The results within the urban and rural subsamples are closely consistent with the main findings.

In Appendix Section A.2, we describe an alternative approach to statistically explaining the mortality gaps. There, we estimate counterfactual Hindu mortality rates after nonparametrically

[29]The neonatal mortality result is likely owing to mother's exposure to open defecation while the child was in utero due to the impacts on mother's net nutrition during gestation. Prendergast et al. (2014) provides suggestive evidence of this mechanism, finding a correlation between in-utero growth and mothers' exposure to open defecation in Zimbabwe.

reweighting the sample of Hindu children to match the joint distribution of characteristics in the Muslim child sample. Compared to the linear regression above, this exercise more flexibly controls for the entire distribution of open defecation exposure, matching the probability mass in 20 bins defining the sanitation environment: 10 bands of local (PSU) open defecation interacted with household's own latrine use indicator. The reweight also has the advantage of more flexibly allowing for correlation between OD and the other controls. Appendix Table A5 reports the results of the exercise, which are closely consistent with Table 3. As in the main table, a large mortality gap persists after reweigthing on the joint distribution of characteristics that do not include open defecation. But reweighting according to a finer partition that interacts groupings of these variables with the distribution of sanitation exposure in 20 bins can completely account for the Hindu-Muslim mortality gap.

In summary, Tables 3, A4, and A5 show that the association between the religious composition of neighborhoods and the local sanitation environment can fully and robustly account (in a statistical sense) for the mortality gap that has been deemed a puzzle by Bhalotra, Valente and van Soest (2010) and others. Next, we exploit this variation in local religious composition to estimate the external impacts of open defecation on child mortality in an IV framework.

6 Main Results

In this section we begin by presenting our main estimates and discussing their economic importance. We then perform a series of falsification tests to show that our instrument is not positively correlated with better diet, water quality, or hygiene behaviors like hand washing. Finally, we present two tests of the externality channel we claim by (i) examining effects of open defecation on acute malnutrition, and (ii) investigating the interactions between breastfeeding and the child's local sanitation environment.

6.1 IV Estimates

The nature of the demand difference we exploit offers a unique advantage: Our setting is one in which a higher concentration of Muslims in a locality predicts better sanitation but otherwise predicts worse neighborhood characteristics. This allows us to exploit variation in sanitation that is

plausibly not positively correlated with confounding factors promoting child survival, and suggests that regressions that instrument for $\overline{OD_{ij}^{-i}}$ with Muslim concentration ($\overline{M_j}$) will tend to understate the child mortality externalities of open defecation (OD).

For the IV analysis, we restrict attention to the mixed-religion PSU sample, over which the relationship between between $\overline{M}_j$ and other PSU characteristics, including $\overline{OD}_j$, is monotonic and approximately linear, as shown in Panel B of Figure 2. Besides tracking the linearity of the first stage, restricting to mixed-religion PSUs confers the identification advantage that only respondents who have settled or remained in mixed neighborhoods contribute to estimating our coefficients. This could be important if the types of Hindus and Muslims who are willing to collocate with the other group are systematically different from those who are not. Panel A of Figure 2 suggests that all-Muslim and all-Hindu neighborhoods may indeed differ from mixed-religion neighborhoods, because the nonparametric plots jump at the endpoints ($\overline{M} = 0$ and $\overline{M} = 1$) for many variables.

Table 4 presents the results of the IV analysis. Column headers in the table describe the dependent variable (IMR or NMR), the sample restriction, and the regression model. Columns 1 and 5 report OLS estimates over the sample of mixed religion PSUs for comparison. Columns 2 and 6 report instrumented coefficients with no controls other than survey round. Columns 3 and 7 add controls for each household's own latrine use and its own religion, and columns 4 and 8 add the same set of extended controls used in Panel B of Table 3.[30] First stage estimates are presented in Appendix Table A3, with first stage F-statistics always exceeding weak instrument thresholds.

Table 4 shows that instrumenting for $\overline{OD_{ij}^{-i}}$ with $\overline{M_j}$ yields point estimates larger than the OLS regressions. To put the size in context, the IV coefficient in column 4 implies that a 10 percentage point reduction in the fraction of neighbors defecating in the open is associated with a decline in infant mortality of 6.5 children out of 1000. For neonatal mortality in column 8, the figure is 4.7 deaths per 1000. This magnitude is similar across specifications that vary the control set, suggesting that the variation in open defecation that arises from the composition of neighbors is not strongly correlated

[30] All regressions include indicators for survey rounds. The extended controls are identical to those in Table 3 and include a full set of birth order indicators interacted with sex, indicators for household ownership of each of the seven assets asked about throughout NFHS survey rounds, an indicator for piped water, an indicator for whether the mother lives with her husband's parents, indicators for child's birth month and birth year, an indicator for the child being a multiple birth, an urban indicator, household size, mother's education in years, and an indicator for mother's literacy, as well as PSU-level means of household assets, household electricity, household piped water, whether births occurred in an institution, whether mothers had birth assistance, whether children were ever vaccinated, household possession of a health card, and father's education in years. Several of the variables included as PSU-level means could not be controlled for at the individual level because the DHS/NFHS surveying scheme did not ask these of all respondents.

with any individual-level behaviors or characteristics that affect mortality. The IV estimates may be larger than the OLS due to measurement error in the PSU-level variable $\overline{OD_{ij}^{-i}}$, which is calculated over only the survey-sampled households (less than 50% of households are sampled in the typical PSU).

Because Figure 2 and Tables A1 and A2 showed that, if anything, bias would push our IV results towards zero, these estimates may imply a lower bound on true effects.[31] Nonetheless, the effects are economically large, so that the lower bound is informative here. In the context of our data, the difference between a locality in which all of a household's neighbors defecate in the open and a locality where none of them do is associated with a larger reduction in child mortality than the difference between households at the bottom and top quintiles of asset wealth (see Figure 3).

The implied importance of open defecation for infant mortality in the modern Indian context is consistent with evidence by Cutler and Miller (2005) and Watson (2006) on the role of clean water and sanitation in explaining large shares of declining infant mortality in the history of the US. Cutler and Miller (2005) show that the introduction of clean water technologies in US cities beginning around 1900 reduced the infant mortality rate by 46% and accounted for 74% of the total decline in US infant mortality at the turn of the century.[32,33] To compare to India, IMR in India declined by 25 deaths per thousand over a decade (2001 to 2011) while open defecation simultaneously declined by about 8 percentage points. Naively applying our IV estimates would predict a change in IMR of -5.2 ($= 65 \times -0.08$), which would account for about 20% of the total decline in IMR over that period.[34]

Our estimates are also consistent with the most credible data on cause of death during early-life in India: Using a census of all Indian deaths occurring from 2001 to 2003, Million Death Study Collaborators (2010) show that 22% of non-neonatal child deaths (deaths from 1-59 months) in India

[31] See footnote 24 for a proof.

[32] Watson (2006), studying sanitation programs in the United States in the 1960, where infant mortality was lower and the therefore marginal effects would be expected to be smaller compared to India, finds that "sanitation interventions explain almost forty percent of the convergence in Native American and White infant mortality rates in reservation counties since 1970."

[33] Galiani, Gertler and Schargrodsky (2005) studying water privatization in Argentina, found a decline in all-cause child mortality by 8%, with effects reaching as high as 26% in the poorest places, which are likely to be better comparisons to India with respect to poverty. Given that interventions like water privatization could have such large effects on mortality, it is unsurprising to find similarly large effects of open defecation. Among other plausible channels, open defecation is likely to contaminate drinking water.

[34] Alternatively, consider the implied effect of cutting the open defecation rate in half in the typical neighborhood in our sample, from roughly 60% of households to 30%. Setting aside all complications of predictions out-of-sample, and ignoring the potential for reinforcing or countervailing behavioral responses to such a dramatic shift in the local environment, this would imply a reduction in IMR of 27% $\left(= \frac{65 \times 30\%}{72}\right)$. (The parameter 65 is taken from column 4 of Table 4. The denominator 72 is the weighted average IMR of Hindus and Muslims from row 1 of Table 2.)

are due to diarrhea, which is just one of the potential pathways by which intestinal worms and bacterial infections that are transmitted via open defecation could affect infant mortality.[35] Indeed, we confirm in a mechanism check below that low current weight among infants, which reflects recent bouts of diarrhea, responds to our identifying variation in the same pattern as mortality.

Although ours is the first paper able to identify mortality effects of local sanitation, recent work exploring other human capital impacts of open defecation have found effects with similarly large magnitudes. Gertler et al. (2015) perform a meta-analysis of open defecation-height experiments, and estimate a reduction of 0.046 height-for-age standard deviations from a 10 percentage point reduction in open defecation. To compare the size of these height effects to our mortality estimates, one can scale the estimates using the correlation between IMR and height across Indian villages. In the DHS, Indian villages with one more IMR point (.0010) have children that are 0.0034 normalized height-for-age standard deviations shorter. Using this conversion factor to roughly translate between outcomes, our estimate of a .0065 mortality improvement for a 10 percentage point decline open defecation (Table 4, col 4) would be consistent with a 0.022 height-for-age effect. This effect size translated from our mortality estimates is roughly half of the Gertler et al. (2015) experimental effect sizes, implying our estimates, though economically large, are smaller by this metric.

To understand the economic importance of our results, it is useful to consider external harm created by a single household defecating in the open. Because about one in every ten Indian households has an infant born each year, the coefficient on $\overline{OD_{ij}^{-i}}$ is approximately equal to the sum of harm (across all neighbors) imposed by a single household that chooses open defecation, aggregated over 10 years.[36] Also, although we do not claim that we can econometrically identify the *private* benefits of latrine use (the coefficient on OD_i) with our identification strategy, we note that OLS estimates of the private harm of open defecation in Panel B of Table 3 are about one tenth the size of the IV estimates of the external harm (the coefficient on $\overline{OD_{ij}^{-i}}$) in Table 4.[37]

[35] In addition, Million Death Study Collaborators (2010) show that an even larger fraction, 33%, of neonatal deaths (deaths from 0-1 months) are attributable to low birthweight. As we discuss in Section 3, there is clear epidemiological evidence that exposure to fecal pathogens reduces the body's net absorption of nutrients. In mothers, poorer net nutrition would have direct impacts on child's birthweight. Prendergast et al. (2014) find suggestive evidence on birthweight specifically, showing a correlation between in-utero growth and mothers' exposure to open defecation in Zimbabwe.

[36] Consider the simple case in which all households contain one infant. Because the contribution of any household to the regressor $\overline{OD^{-i}}$ is weighted by its share in the PSU, the harm caused by one household defecating in the open on any other single household is $\frac{1}{N-1} \cdot \beta$, where N is the number of households in the PSU and β is the coefficient on $\overline{OD^{-i}}$. The total external harm summed across the other $N-1$ households is then $N-1 \cdot \frac{1}{N-1} \cdot \beta = \beta$. In practice, about one in every 10 Indian households has an infant born each year, so that the total harm equals β over a 10-year window.

[37] Both the private and external harm would have the same 1 in 10 scaling described in Footnote 36, due to the roughly

To examine the robustness of our IV results to more flexibly controlling for potential confounders, we re-estimate the IV regressions over different partitions of our analysis sample. Appendix Table A6 estimates the IV regression separately for Muslim and Hindu children (in each case continuing to instrument with the religious composition of *all* neighbors). The additional flexibility and smaller sample reduces statistical precision, but these results by subgroup are consistent with the overall effect size in Table 4 and with each other. Similarly, Table A6 shows that within subsamples of only girls or boys, only first-borns or later-borns, and only urban or rural PSUs, the IV results are never statistically different and are often quantitatively similar.

Finally, it would be interesting to evaluate whether there was any interaction between own household latrine use and that of neighbors. *A priori*, it is unclear whether an interaction should exist: The disposal of feces in this context is distributed along roads, in fields, and near homes—that is, it is not relegated to areas trafficked only by those who do not use latrines. This implies that the effect of neighbors' open defecation may be invariant to own household latrine use. Our estimation strategy, which focuses on neighbor effects, does not allow us identify the interaction term, as doing so would require a second instrument (for own open defecation, OD_i). With that caveat, in Appendix Table A7, we split the sample according to whether the respondent household uses a latrine or defecates in the open and then re-estimate the IV coefficients in the two subgroups. The table shows that the IV estimates among households practicing open defecation and households using latrines are statistically indistinguishable and numerically similar (79.0 and 63.1, respectively), offering no strong evidence of an interaction effect.

6.2 Falsification Tests: Diet, Water, and Washing

In this section, we investigate whether our instrument—the religious composition of neighbors—is correlated with important, observable health behaviors like diet and hygiene.[38] The potential for bias would arise only if *conditional on own religion*, our neighborhoods instrument predicts such behaviors. In contrast, differences across Muslim and Hindu households pose no problem for our identification strategy: Any systematic behavioral differences between Hindus and Muslims, such as vegetarianism or different washing practices, are absorbed by the controls for own household religion that are

10% probability of any household containing an infant.

[38]Our interpretation of the IV results as a lower bound relies on an assumption that Muslim neighbors do not positively impact child health through some channel other than latrine use.

included in all regressions. Our IV estimates are identified solely off of neighbors' religion.

We ask, for example, whether Hindus living around many Muslims are more likely to eat meat than Hindus living around few Muslims. Because our main dataset, the NFHS, was not designed to measure consumption and contains relatively little information on hygiene behaviors, we turn briefly to the India Human Development Survey (IHDS) of 2012.[39] Summary statistics for this supplemental dataset are included in Appendix Table A8. For the set of variables common to both the IHDS and NFHS, such as urban residence, open defecation, and access to piped water, Table A8 shows that the IHDS replicates the same Hindu-Muslim differences found in the NFHS, which are displayed in Table 2.

6.2.1 Diet

In Table 5, we investigate the relationship between diet and the composition of the neighborhood ($\overline{M}_j$) in OLS regressions. The dependent variables are listed in the column headers and include measures of meat, eggs, and dairy consumption, measured per household per month. For each of these diet variables, we begin by regressing diet on *own* religion to demonstrate that the consumption data capture large differences across Muslim and Hindu households. For example, column 1 shows that Muslims are a precisely estimated 15 percentage points more likely (on a base of 25 percentage points) to consume meat compared to Hindus. Such differences are expected, and not problematic.

To test for a violation of our identifying assumption, we add our instrument ($\overline{M}_j$) as a regressor in the second and third columns for each dependent variable. Panel A shows that there is no correlation between $\overline{M}_j$ and the extensive margin of eating any meat, or between $\overline{M}_j$ and the intensive margin of quantity of meat consumed. In Panel B, we examine two other calorie- and protein-rich foods: eggs and milk. For all diet variables, the coefficients on our instrument are small, not significantly different from zero, and robust to the inclusion of controls.[40] This is true even though large, precisely estimated diet differences are apparent between Hindu and Muslim households for each consumption outcome in the table.

Table 5 is direct evidence against the notion that Hindus living around Muslims have systemat-

[39] The IHDS contains richer hygiene information and a complete consumption module, but it cannot be used to construct mortality rates that are similarly reliable to those from the NFHS. Specifically, we are limited by the fact that complete birth histories were not recorded for all women of childbearing age.

[40] We control for log per capita per consumption and the urban classification of the household, which could be important in principle because richer and urban households in India are more likely to eat meat.

ically different diets than Hindus living around other Hindus, and similarly against the notion that Muslims living around Hindus have systematically different diets than Muslims living around other Muslims. Thus, there is no empirical evidence that our results are likely to be confounded by issues related to diet.

6.2.2 Other Hygiene and Water

Diet is likely to have primarily private benefits, but hygiene practices like hand washing could generate externalities in principle. If these practices covary with Muslim concentration across localities, then our estimates could be reflecting some other hygiene-related externality associated with the religious composition of a neighborhood, rather than the open defecation externality we claim.

We begin by noting that Hindu and Muslim households do not appear to differ with respect to these health behaviors. For example, the summary statistics from the IHDS data in Table A8 reveal that unlike OD (practiced by 51% of Hindus and 31% of Muslims in the IHDS data), Hindu and Muslim households do not differ in hand washing (72% vs. 70%) or purifying water (11% vs. 12%).

To search for violations of our identifying assumption, in Table 6, we report on regressions in which the dependent variables are various measures of hygiene and water quality from the IHDS. The regressor of interest is our instrument, $\overline{M}_j$. In the first eight columns the dependent variables are always washing hands, sometimes washing hands, always purifying water, and sometimes purifying water, none of which are available in our main dataset, the NFHS. In columns 9 and 10, the dependent variable is piped public water to the home, for which a similar measure is available in the NFHS. For completeness, in columns 11 and 12, we show that our first stage holds in the IHDS data by regressing OD on $\overline{M}_j$. The last four columns are shaded, since these can be compared directly to results in the NFHS dataset.

Columns 1 through 8 show that there is no correlation between measures of washing or water purifying and our instrument, $\overline{M}_j$. The only significant difference we find in the table—besides columns 11 and 12, which confirm the robustness of our identifying variation in another dataset—is that Muslim concentration is associated with *less* access to public piped water. This is consistent with our analysis in the NFHS data presented above (see Figure 2 and Table A1). Both the literature (Jayachandran and Kuziemko, 2010) and our own analysis (Table A2) indicate that lower access to piped water in this region implies a Muslim disadvantage with respect to health, again contributing

to the notion that our IV estimates are, if anything, biased downward.[41] The difference in piped water likely reflects the inferior access to state services faced by Muslims. In sum, there is no evidence that other hygiene behaviors are confounding our estimates. The Hindu-Muslim difference in latrine use, therefore, is not merely a marker for a wider array of other important hygiene behaviors with public goods qualities.

6.3 Unobservable Features of Mixed Neighborhoods

Individuals who are willing to live in more diverse neighborhoods could be systematically different from those who are not. We investigate the possibility here as it relates to our identification. We begin by noting that any cosmopolitan advantage in health could not even *in principle* spuriously generate our results. This is because our instrument does not measure the religious diversity of a PSU, it measures the share that is Muslim, which has an asymmetric relationship to religious diversity for Hindus and Muslims. For example, because most Hindus collocate with other Hindus and most Muslims collocate with other Muslims, for the typical Muslim, an increase in $\overline{M}_j$ implies a less religiously diverse neighborhood. In contrast, for the typical Hindu, an increase in $\overline{M}_j$ implies a more religiously diverse neighborhood. This predicted asymmetry stands in contrast to the symmetry of the effects we estimate in Appendix Table A6, where we split the sample by own religion and find positive effects for both groups.[42]

To show that unobservables associated with willingness to live in a diverse or cosmopolitan neighborhood are not driving our results, in Appendix Table A9 we replicate our main IV analysis from Table 4, but include an additional control for diversity. In particular, we control for the fraction of neighbors who are religiously dissimilar from the respondent household. This religious dissimilarity variable moves in opposite directions for Hindu and Muslim households as $\overline{M}_j$ increases. The regressions show that coefficients on religious dissimilarity in IMR and NNM regressions are small and indistinguishable from zero, and that the effects of interest are almost numerically unchanged.

[41] Appendix Table A2 shows that piped water is strongly negatively correlated with infant mortality in our main analysis dataset, the NFHS.

[42] Appendix Table A6, which presents coefficients estimated separately for the Hindu and Muslim subsamples, also provides evidence against the alternative diversity explanation. Though IV estimates in the smaller samples generated by the subgroups less imprecise, Panel C of Table A6 shows that infants within both groups do better when the share of neighbors who are Muslims is larger. This is contrary to the diversity hypothesis, which would predict effects working in opposite directions for the two subsamples. Such a hypothesis would imply that, counter to our findings, Muslims living in a neighborhood with a 50 percent Muslim share would experience lower infant mortality than Muslims living in a neighborhood with a 100 percent Muslim share.

The effect on IMR of $\overline{OD_{ij}^{-i}}$ changes from 64.9 (t-stat = 2.8) to 65.3 (t-stat = 2.9) after the inclusion of the additional religious dissimilarity control.

6.4 Mechanisms: Tests of the Fecal Pathogens Channel

The external effects of open defecation are hypothesized to operate via infection by fecal pathogens introduced by neighbors. As a test of this channel, in this section we examine (i) whether acute malnutrition, which is an intermediate outcome resulting from infectious disease and which precedes death caused by infection, follows the same pattern as the mortality results presented above, and (ii) whether breastfeeding effects interact with local open defecation in a way consistent with the fecal pathogens hypothesis.

6.4.1 Acute Malnutrition

A main channel by which exposure to fecal pathogens may cause death is by affecting net nutrition—that is, calories consumed net of calories lost to diarrheal disease and parasites and expended in combating infections. Acute malnutrition is a well-known mechanism linking infectious disease to infant death (Kielmann and McCord, 1978; Mosley and Chen, 1984), our outcome of interest. If open defecation is causing deaths via infection, acute malnutrition should also be observable in weight. This intermediate outcome may also be of independent interest, because acute malnutrition could impact the human capital accumulation of surviving children.

We follow the standard practice (e.g. Schmidt et al., 2010) of using surveyor-measured weight to capture acute malnutrition and recent diarrhea. Weight, rather than self-reported diarrheal disease, is recommended due to widely recognized problems in survey-reported diarrhea. We discuss the problems with survey-reported diarrhea in this context in more detail in Appendix A.3, and provide statistics in our own dataset highlighting these issues. Weight-for-age is the particular measure of recent diarrhea recommended by Schmidt et al. (2010). To evaluate robustness, we also examine weight-for-height.[43] We operationalize both measures as z-scores scaled to the World Health Organization's child growth standards.

Table 7 displays regression estimates analogous to the mortality regressions in Table 3, but with measures of child weight as the dependent variables. The sample includes all children up to 24

[43]Height is an appropriate denominator for normalizing weight when analyzing acute malnutrition because height reflects long-term, but not recent, net nutritional and disease experience.

months for whom a weight measurement was taken. Due to the DHS/NFHS surveying scheme, these detailed anthropometry measures exist for only a small subset of our main analysis sample. In the first three columns, the dependent variable is weight-for-age. We report OLS regressions.[44] The extended controls are identical to those in the bottom panel of Table 3 with an additional control for height.[45] These OLS regressions offer an opportunity for a falsification test of our identifying assumption: In OLS regressions, if Muslim concentration generates lower mortality only through its association with neighborhood OD, then we should observe that Muslim concentration is not positively correlated with other health outcomes, conditional on controls for OD.

The results in Table 7 follow the same pattern as in the mortality regressions: In columns 1 and 4, an indicator for Muslim households predicts significantly higher z-scores (i.e., heavier children). Then, additionally controlling for Muslim concentration in the locality ($\overline{M_j}$) attenuates the coefficient on the Muslim indicator to insignificance. And finally, incrementally controlling for the local sanitation environment via $\overline{OD_{ij}^{-i}}$ and OD_{ij} attenuates the coefficient on ($\overline{M_j}$) as well. This pattern of results supports the identifying assumption in the IV analysis above, as it shows that the fraction of Muslim neighbors is correlated with child malnutrition, but only through the open defecation channel. The size of the point estimates in column 3 indicates that increasing PSU-level open defecation by 10 percentage points (approximately 0.25 standard deviations) is associated with a decline in weight-for-age of 0.026 standard deviations and a decline in weight-for-height of 0.032 standard deviations.[46] These coefficients are of plausible magnitude. They imply that entirely eliminating open defecation from India would increase child weight-for-age by approximately 0.16 standard deviations, eliminating 8% of India's total shortfall relative to the healthy WHO reference population.

To examine the robustness of this result to alternative parameterizations, in Appendix Figure A2 we plot local polynomial regressions of weight-for-age on local open defecation separately for Hindu and Muslim children. The exercise is described in detail in Appendix Section A.4. These plots show

[44] Corresponding IV estimates lacked precision, with confidence intervals including both zero and very large effects, possibly due to the significantly smaller sample. The sample here is smaller because weight in the NFHS is only consistently measured for children below age 3 at the time of the survey, whereas in our main analysis, we calculate IMR over a larger sample by using the mother's responses to retrospective questions about the timing of births and deaths over a longer look back period.

[45] We control for height-for-age to ensure that results are not spuriously driven by *chronic* malnutrition, which would be reflected in height. In practice, removing the height control does not affect the pattern of results in the table.

[46] The surviving children for whom we can observe a weight measurement constitute a selected sample, because these children were strong enough to survive until the time of observation. This "culling" would tend to bias our estimates of the coefficient on $\overline{OD_{ij}^{-i}}$ in the weight regressions toward zero, following the logic in Almond and Currie (2011) that estimates of the impacts of health shocks are generally conservative when those shocks also increase mortality.

that the effects of $\overline{OD^{-i}}$ on child weight (i.e., the non-parametric slopes) are large and essentially identical across Hindus and Muslims. Together, the results in Table 7 and Figure A2 are consistent with a disease externality mechanism in which fecal pathogens cause diarrhea and acute malnutrition prior to death. And because $\overline{M_j}$ has no residual predictive power for child weight after conditioning on $\overline{OD^{-i}}$, these results support the identifying assumption that $\overline{M_j}$ impacts survival only through its association with neighbors' latrine use.

6.4.2 Breastfeeding Interactions

Water and prepared food are two key pathways through which poor sanitation causes infections in children. Breastfeeding, which interrupts this pathway, is known to be protective against the transmission of such infections, and previous studies have shown important interactions in this context between the efficacy of breastfeeding and the quality of a household's water supply (see Jayachandran and Kuziemko, 2010). Here, we examine whether the efficacy of breastfeeding is increasing in the fraction of neighbors who defecate in the open.

Table 8 examines interactions between religion, breastfeeding, and sanitation in OLS regressions in which the dependent variable is IMR. The regressor of interest is an indicator for exclusive breastfeeding during the infant's first six months of life if she survived, or until death if she died. Controls are as in Table 3. Column 1 reports results for a specification interacting breastfeeding with the Muslim indicator. Unsurprisingly, the main effect of exclusive breastfeeding is large and negative—i.e., breastfeeding is associated with reduced mortality. But the significant positive coefficient estimate for the interaction term *breastfed* $\times$ *Muslim* indicates that breastfeeding is differentially less beneficial to Muslim children than Hindu children, on average.

This non-intuitive pattern is again merely due to the correlation between own religious identity and that of neighbors. After additionally controlling for $\overline{OD_{ij}^{-i}}$ and its interaction with breastfeeding in column 2, the positive coefficients on the indicator for Muslim and its interaction with breastfeeding become insignificant and change signs.[47] These regressions indicate that Muslim children, on average, benefit less from breastfeeding only because they tend to live in better local sanitation environments, while Hindu children, on average, face environments where the protection conferred by

[47] Although not statistically significant, the sign reversal on is consistent with the notion that conditional on open defecation, Muslim households might face somewhat worse environments. For example, as shown above, Muslim households are significantly less likely to have piped water, which Jayachandran and Kuziemko (2010) have shown as having potentially important implications for the relative benefits of breastfeeding.

breastfeeding matters more.[48]

The main coefficient of interest in Table 8 is on $\overline{OD_{ij}^{-i}} \times$ *breastfed* in column 2. Increasing PSU-level open defecation by 10 percentage points increases the efficacy of breastfeeding (decreases IMR) by about 25 percent of the main effect of breastfeeding $\left(= \frac{250 \times 10\%}{105}\right)$. This interaction effect is consistent with the mechanism we describe: breastfeeding filters the fecal pathogens that would otherwise be ingested by infants.[49]

7 Conclusion

As of 2014, more than a billion people worldwide continue to defecate in the open, without the use of even basic latrines. While governments and others have invested heavily in reducing the practice in recent years, there has been disproportionately little evidence of the causal impact of open defecation on mortality. We view our study as informing ongoing policy efforts to reduce open defecation around the world, as well as contributing to the economic literature concerned with the impacts of infectious disease on health and human capital.

The pattern we document solves an existing puzzle in the literature—that in India Muslim children suffer lower rates of mortality than Hindu children, despite being poorer on average. We show that this operates entirely via the tendency of Muslims to reside near other Muslims, combined with the typically higher rates of latrine use among this group of neighbors. Tests of the mechanisms linking open defecation to infant death, including contamination of food and water and acute malnutrition due to intestinal disease, support our interpretations.

More broadly, our study provides insights into the public goods aspect of sanitation. This study is the first to provide econometric evidence on the mortality externalities of open defecation, exploiting variation arising from the religious composition of neighbors. Understanding this public goods component is important for policy interventions motivated by the efficiency concern that private demand is below the social optimum. Practically, our results indicate that many infants die each year due to poor sanitation in their localities. In the context of India, a back-of-the-envelope calculation

[48] Like the reduced form results in Table 3, estimates in Table 8 are identified off of the religion of neighbors, not the religion of the child's own household. Therefore, any unobserved differences in inputs like weaning foods between Hindu and Muslim households would be captured by controls for *Muslim* and *Muslim* $\times$ *breastfed* in column 2.

[49] The size of the point estimate on $\overline{OD_{ij}^{-i}} \times$ *breastfed*, which is equal and opposite to the coefficient on $\overline{OD_{ij}^{-i}}$, implies that ingesting contaminated food or water is an important channel by which infants are harmed by neighbors' open defecation.

within the range of variation supported by the data suggests that reducing mean open defecation by 10 percentage points (one quarter of a standard deviation across localities) would reduce IMR by 3 deaths per thousand, or about 4% of the mean mortality rate. With an estimated 26 million children born in India each year, this equates to 78,000 deaths annually. The sheer size of these effects highlights the need for further investigation into the externalities of sanitation in the developing world.

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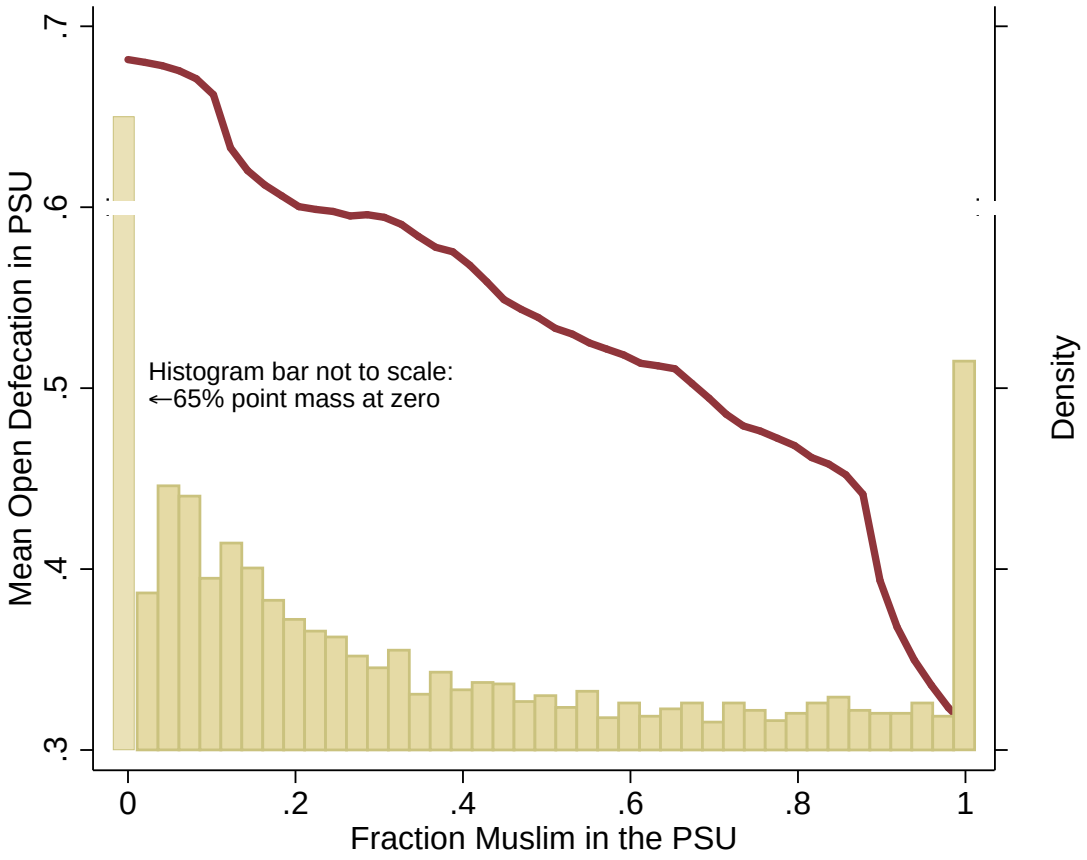
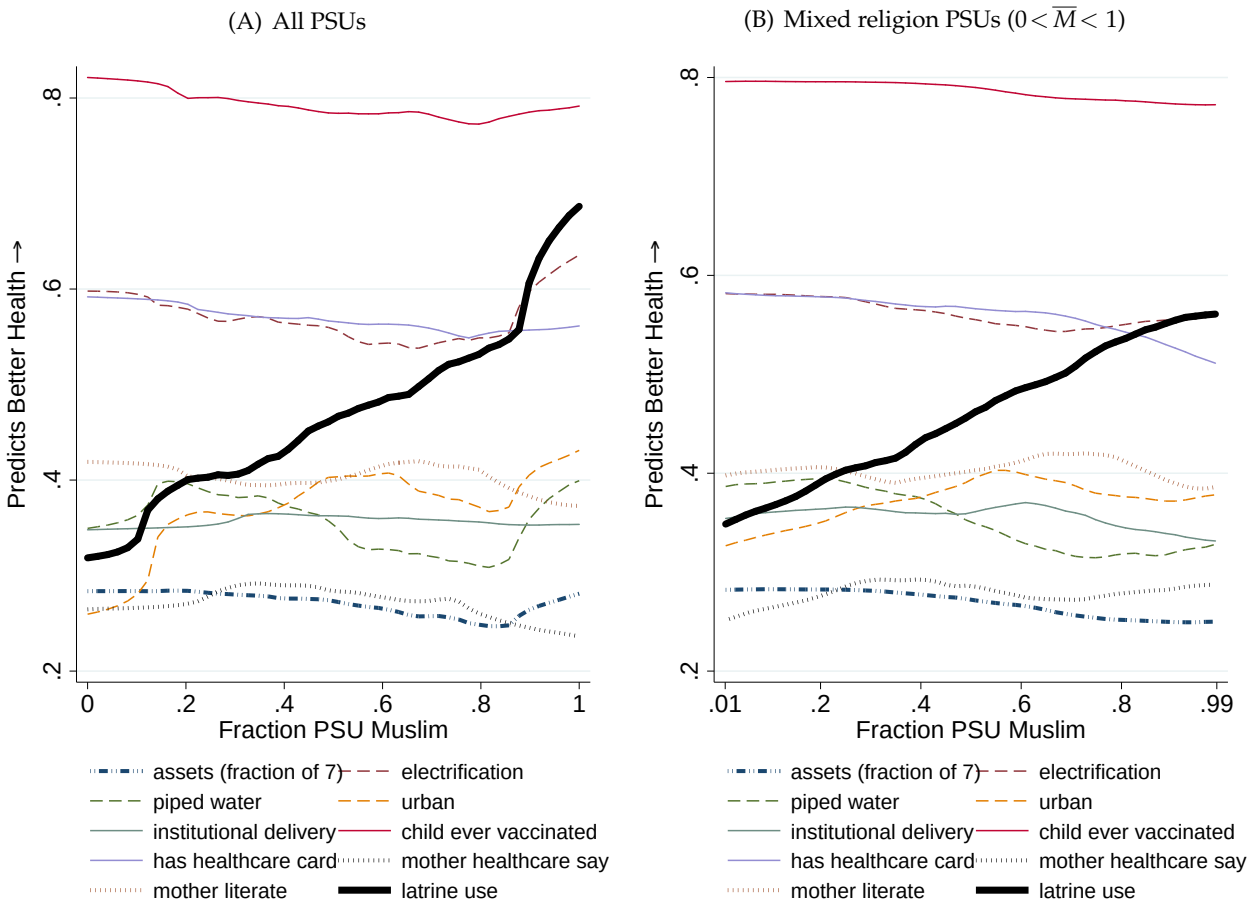
Figure 1: Identifying Variation: Muslim Concentration ($\overline{M}$) and Open Defecation ($\overline{OD}$) Across PSUs



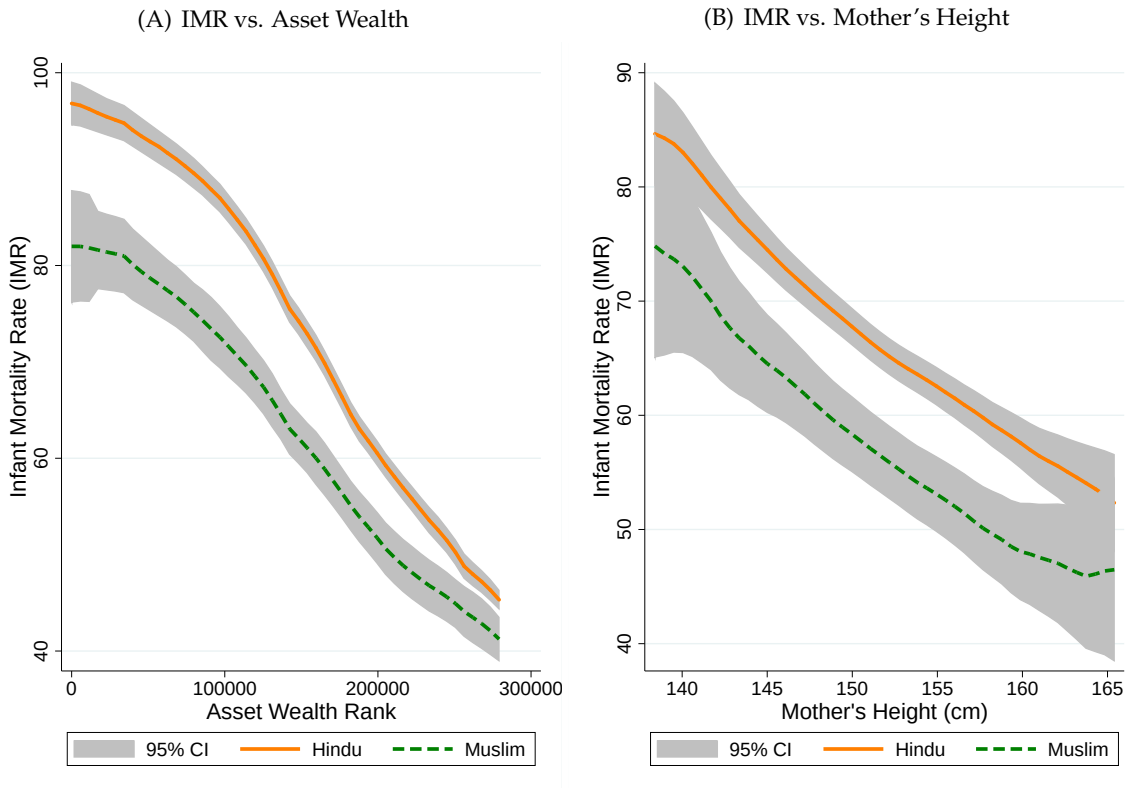
Note: Measured along the left vertical axis, the figure plots a local polynomial regression of PSU-level open defecation ($\overline{OD_j}$) on Muslim concentration ($\overline{M_j}$) in the PSU. Measured along the right vertical axis, the figure shows the histogram of Muslim concentration across primary sampling units (PSUs) in the NFHS survey. Observations are PSUs. Most PSUs are perfectly segregated along religious lines, with either 0% or 100% Muslim concentration. The point mass representing 100% Hindu, which comprises about two thirds of all PSUs, is not drawn to scale.

Figure 2: Greater Muslim Concentration ($\overline{M}$) Predicts Neutral or Worse PSU Characteristics, Except Latrine Use



Note: Figure plots local linear regressions. The dependent variables are household-level characteristics, and the regressor is Muslim concentration ($\overline{M_j}$), which is measured at the PSU-level. Each dependent variable is defined such that higher values predict better infant health outcomes. The signs of the relationships between these variables and infant mortality are confirmed in Appendix Table A2. The dependent variables include asset wealth (as the fraction of the seven assets reported in all survey rounds) and indicators for household electrification, household piped water, institutional delivery of the child, any child vaccinations, family possession of a national health card, mother having a say in child's healthcare, mothers' literacy, and latrine use. Panel A includes all PSUs in the sample. Panel B includes only mixed-religion PSUs ($0 < \overline{M} < 1$). Observations are live births.

Figure 3: Motivating Puzzle: At All Levels of Parental Wealth and Health, Hindu IMR is Higher



Note: Figure plots local linear regressions of infant mortality on measures of economic well-being. The dependent variable is an indicator for death in the first year of life $\times$ 1000. The left panel plots mortality against asset wealth rank, constructed as described in the text. The right panel plots mortality against mother's height. 95% confidence intervals are shaded in gray. Observations are live births.

Table 1: Stated and Revealed Preferences over Latrine Ownership and Use

	Unit of Observation	Hindu Mean	Muslim Mean
		(1)	(2)
Open defecation, unconditionally	all persons in household	0.73 (0.00)	0.46 (0.01)
Open defecation, conditional on owning latrine	all persons in household	0.25 (0.01)	0.10 (0.01)
Owns latrine	household	0.34 (0.01)	0.52 (0.04)
Says religious leader ever told them where to defecate	respondent	0.16 (0.01)	0.33 (0.04)
Says open defecation far from home is pure	respondent	0.53 (0.01)	0.40 (0.04)
Says latrine use near home is pure	respondent	0.46 (0.01)	0.54 (0.04)

Note: Table reports means and standard errors of survey responses from the Sanitation Quality, Use, Access, & Trends (SQUAT) Survey, 2013-2014. Responses are stratified by religious group. The table contains information on 22,787 individuals in 3,235 sampled rural households in Bihar, Madhya Pradesh, Rajasthan, and Uttar Pradesh. The unit of observation differs across rows and includes either all persons in the household, whether interviewed or told about; the household itself; or the survey respondent (one per household). Standard errors of the means, clustered by village, are shown in parentheses. All across-group comparisons are statistically significant at the 5% level.

Table 2: Summary Statistics: Hindus and Muslims in the NFHS

	Hindu Subsample		Muslim Subsample	
	Mean	SD	Mean	SD
	(1)	(2)	(3)	(4)
infant mortality rate (IMR), year 1	74.1	261.9	63.0	243.0
neonatal mortality rate (NMR), month 1	47.5	212.6	39.8	195.6
household open defecation	0.68	0.47	0.43	0.49
local (PSU) open defecation	0.66	0.38	0.45	0.38
local (PSU) fraction Muslim	0.06	0.14	0.69	0.31
household has electricity	0.59	0.49	0.59	0.49
household has piped water	0.35	0.48	0.37	0.48
household is urban	0.28	0.45	0.40	0.49
household has radio	0.36	0.48	0.36	0.48
household has TV	0.33	0.47	0.31	0.46
household has refrigerator	0.10	0.30	0.10	0.30
household has bicycle	0.46	0.50	0.41	0.49
household has motorcycle	0.12	0.33	0.10	0.29
household has car	0.02	0.14	0.02	0.12
mother's height (cm)	151.5	5.8	152.0	5.8
mother no education	0.58	0.49	0.64	0.48
mother completed primary	0.27	0.45	0.21	0.40
child breastfed for at least six months	0.91	0.28	0.91	0.28
child's birth order	2.46	1.17	2.74	1.20
child is female	0.48	0.50	0.49	0.50
observations (live births)	232,123		46,300	

Note: Table displays summary statistics for our main analysis sample, rounds 1, 2, and 3 of the NFHS. Neonatal and infant mortality are defined, respectively, as the number of deaths among children less than one month old and less than one year old, scaled per 1,000 live births. Observations are children (live births).

Table 3: OLS Estimates: Neighborhood Composition, Local Sanitation, and Mortality

	Panel A: Survey Round FEs only					
dependent variable:	Infant Mortality (IMR)			Neonatal Mortality (NMR)		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-3.8	-2.5	-7.1**	-2.5	-1.7
	(1.5)	(2.3)	(2.3)	(1.2)	(2.0)	(2.0)
PSU fraction Muslim		-9.6**	4.3		-7.4**	1.3
		(3.0)	(3.0)		(2.5)	(2.5)
own household OD			19.7**			12.1**
			(1.7)			(1.4)
PSU mean OD (except own)			26.5**			17.1**
			(2.3)			(1.8)
mean of dep. var.	72.2	72.2	72.2	46.2	46.2	46.2
observations (live births)	278,423	278,423	278,423	278,423	278,423	278,423
	Panel B: Extended Controls					
dependent variable:	Infant Mortality (IMR)			Neonatal Mortality (NMR)		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.5**	-4.2+	-3.6	-5.8**	-2.1	-1.6
	(1.5)	(2.3)	(2.3)	(1.2)	(1.9)	(1.9)
PSU fraction Muslim		-8.6**	-4.6		-6.1*	-2.8
		(2.9)	(3.0)		(2.5)	(2.5)
own household OD			5.6**			4.8**
			(1.8)			(1.4)
PSU mean OD (except own)			10.2**			8.2**
			(3.0)			(2.4)
extended controls	X	X	X	X	X	X
mean of dep. var.	72.2	72.2	72.2	46.2	46.2	46.2
observations (live births)	278,423	278,423	278,423	278,423	278,423	278,423

Note: Table reports results from a series of OLS regressions. The dependent variable in columns 1 through 3 is infant mortality (year 1). The dependent variable in columns 4 through 6 is neonatal mortality (month 1). Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). PSU mean OD is calculated over all households in the PSU other than the respondent household. All regressions include survey round fixed effects. Extended controls in the bottom panel include a full set of birth order indicators interacted with sex, indicators for household ownership of each of the seven assets asked about throughout NFHS survey rounds, an indicator for piped water, an indicator for whether the mother lives with her husband's parents, indicators for child's birth month and birth year, an indicator for the child being a multiple birth, an urban indicator, household size, mother's education in years, and an indicator for mother's literacy, as well as PSU-level means of household assets, household electricity, household piped water, whether births occurred in an institution, whether mothers had birth assistance, whether children were ever vaccinated, household possession of a health card, and father's education in years. See the text for additional details. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table 4: Main Results: IV Estimates Exploiting PSU-level Variation in the Composition of Neighbors

dependent variable:	Infant Mortality (IMR)				Neonatal Mortality (NMR)			
sample restriction:		Mixed Religion PSUs				Mixed Religion PSUs		
specification:	OLS	IV	IV	IV	OLS	IV	IV	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PSU mean OD (except own)	28.4**	74.9**	56.6+	64.9**	17.2**	53.0**	45.6+	46.6*
	(3.6)	(14.3)	(32.6)	(22.8)	(2.8)	(11.0)	(26.1)	(18.4)
own household OD	19.5**		3.3	-14.8+	13.1**		-3.2	-8.3
	(2.6)		(18.7)	(8.1)	(2.1)		(15.1)	(6.6)
own household Muslim	X		X	X	X		X	X
extended controls				X				X
mean of dep. var.	72.2	72.2	72.2	72.2	46.2	46.2	46.2	46.2
first stage F-stat		81.1	90.6	204.9		81.1	90.6	204.9
observations (live births)	104,090	104,090	104,090	104,090	104,090	104,090	104,090	104,090

Note: Table reports results from a series of OLS and IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). IV estimates instrument $\overline{OD_{ij}^{-i}}$ with $\overline{M_j}$. Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). Columns 1 and 5 report results from regressions similar to the OLS regressions in Table 3 for comparison, but exclude the instrument $\overline{M}$ and are restricted to the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). Columns 2 and 6 report instrumented coefficients in regressions with no controls. Columns 3 and 7 add controls for own religious identity and own OD. Columns 4 and 8 add extended controls as described in the Table 3 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table 5: Falsification Tests: Instrument ($\overline{M}_j$) Does Not Predict Diet Differences Across PSUs

	Panel A: Meat Eating					
dependent variable:	Non-Vegetarian			Meat (kg), conditional on > 0		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	0.15**	0.15**	0.14**	0.89**	0.90**	0.84**
	(0.02)	(0.02)	(0.02)	(0.15)	(0.21)	(0.20)
PSU fraction Muslim		0.01	0.01		-0.02	-0.01
		(0.04)	(0.04)		(0.29)	(0.30)
IHDS Controls			X			X
mean of dep. var.	0.25	0.25	0.25	3.35	3.35	3.35
observations (households)	37,195	37,195	37,195	9,235	9,235	9,235
	Panel B: Other Diet Variables					
dependent variable:	Eggs (doz)			Milk (liters)		
	(7)	(8)	(9)	(10)	(11)	(12)
Muslim	5.16**	4.27**	4.32**	-2.89**	-2.27**	-1.95**
	(0.56)	(0.47)	(0.47)	(0.90)	(0.61)	(0.58)
PSU fraction Muslim		1.53	1.51		-1.08	0.53
		(0.96)	(0.98)		(1.66)	(1.56)
IHDS Controls			X			X
mean of dep. var.	4.86	4.86	4.86	18.24	18.24	18.24
observations (households)	37,195	37,195	37,195	37,195	37,195	37,195

Note: Table reports results from OLS regressions, using data from the India Human Development Survey of 2012. Dependent variables are listed in the column headers, and include an indicator for consuming meat and continuous measures of meat consumed (kg), eggs consumed (dozens), and milk consumed (liters), per household per month. All columns control for own religion. IHDS controls include an urban indicator and log of consumption, measured as rupees per month per capita. Observations are households. Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table 6: Falsification Tests: Instrument ($\overline{M}_j$) Does Not Predict Other Hygiene Behaviors Across PSUs

dependent variable:	Always Wash Hands after Defecating		Usually/Always Wash Hands after Defecating		Always Purify Water		Usually/Always Purify Water		Piped Public Water to Home		Open Defecation	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
PSU fraction Muslim	0.05	0.03	-0.01	-0.01	0.02	0.01	0.01	-0.01	-0.07*	-0.11**	-0.19**	-0.14**
	(0.04)	(0.04)	(0.01)	(0.01)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.02)	(0.04)	(0.03)
IHDS Controls		X		X		X		X		X		X
mean of dep. var.	0.72	0.72	0.96	0.96	0.11	0.11	0.20	0.20	0.29	0.29	0.49	0.49
observations (households)	36,608	36,608	36,608	36,608	36,608	36,608	36,608	36,608	36,608	36,608	36,608	36,608

Note: Table reports results from OLS regressions, using data from the India Human Development Survey of 2012. Dependent variables are listed in the column headers, and include indicators for washing hands after defecating, for purifying water, for own household open defecation, and for having public piped drinking water into the home. All columns control for own religion. IHDS controls include an urban indicator and log of consumption, measured as rupees per month per capita. Observations are households. Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table 7: Test of Mechanism: ($\overline{OD}$) and Acute Net Malnutrition

	dependent variable:					
	Weight-for-Age Z-score			Weight-for-Height Z-score		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	0.128**	0.057	0.046	0.135**	0.033	0.021
	(0.028)	(0.042)	(0.042)	(0.032)	(0.046)	(0.045)
PSU fraction Muslim		0.118*	0.013		0.170**	0.044
		(0.055)	(0.056)		(0.062)	(0.063)
own household OD			-0.145**			-0.166**
			(0.033)			(0.036)
PSU mean OD (except own)			-0.264**			-0.319**
			(0.056)			(0.063)
extended controls	X	X	X	X	X	X
mean of dep. var.	-1.89	-1.89	-1.89	-0.94	-0.94	-0.94
observations (live births)	21,636	21,636	21,636	21,164	21,164	21,164

Note: Table reports results from a series of OLS regressions. The dependent variable in columns 1 through 3 is weight-for-age. The dependent variable in columns 4 through 6 is weight-for-height. Current weight is an outcome reflecting acute malnutrition, which is an important mechanism by which poor sanitation causes infant mortality. Both outcomes are scaled as z-scores relative to 2006 WHO child growth standards. PSU mean OD is calculated over all households in the PSU other than the respondent household. Controls are as described in the Table 3 notes, with the addition of height-for-age. All regressions include survey round fixed effects. The sample includes all children under 2 years. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table 8: Test of Mechanism: Interaction Between Breastfeeding Efficacy and $\overline{OD}$

dependent variable:	Infant Mortality (IMR)	
	(1)	(2)
Muslim	-38.4**	5.8
	(14.0)	(13.7)
breastfed X Muslim	31.5*	-16.5
	(14.1)	(13.8)
PSU OD (except own)		233.1**
		(14.0)
PSU OD (except own) X breastfed		-250.2**
		(13.7)
breastfed	-245.5**	-104.6**
	(6.4)	(8.5)
extended controls	X	X
observations (live births)	83,702	83,702

Note: Table reports results from OLS regressions. The dependent variable is IMR, scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). The breastfed indicator is equal to one if the infant was exclusively breastfed during the first six months of life if she survived, or until death if she died. PSU mean OD is calculated over all households in the PSU other than the respondent household. Controls are as described in the Table 3 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

APPENDIX

A.1 Sample Sizes Needed to Experimentally Identify Infant Mortality Externalities

In footnote 1 in the introduction, we noted that the number of neighborhood clusters required to detect even economically large infant mortality effects of open defecation (OD) via a field experiment is large, both because of the plausible effect size relative to the variance in mortality and because measuring local externalities necessarily implies randomization at the level of the locality, not the individual. We also noted that it can be difficult to generate a first stage effect on latrine use via experimental interventions. In this section, we illustrate these issues.

We begin with a standard power calculation to determine the number of localities (clusters) required to detect an external effect of OD on IMR. Assume we wish to detect a minimum effect size of 3.5 infant deaths per thousand, which is 5% of mean IMR and a little larger than our OLS estimate of 2.7 to 2.9 deaths averted per 10 percentage point reduction in local open defecation. The calculation results in 8,622 clusters, based on a simple two-sided test.

With the NFHS data, we can alternatively perform a more detailed calculation for the required sample size and cluster count via Monte Carlo simulation. Unlike the standard power calculation, this method naturally incorporates any heterogeneity in infant mortality that is present across clusters. For the simulation, we again assume that the true effect of a 10 percentage point reduction in local OD is equal to 5 percent of mean infant mortality, or 3.5 deaths per thousand. To implement the Monte Carlo simulation, we iterate over the following procedure, varying the number of sample clusters (N_c) included. We use PSUs from the NFHS data described in Section 4 as our clusters.

1. Randomly select, with replacement, N_c clusters to include in the simulation.
2. Randomly assign half the included clusters to treatment and half to control.
3. Randomly identify 5 percent of infants in treated clusters and replace their infant mortality with zero, thus leaving observations for live children unchanged. This changes the mean IMR in each treatment cluster by 5 percent of the mean on average.
4. Regress infant mortality on a treatment indicator, clustering standard errors.

In practice, we vary N_c from 2,000 to 10,500 clusters in increments of 100, with 50 iterations at each value of N_c. Appendix Figure A1 plots the relationship between sample size and power delivered by the simulation. The horizontal axis shows the cluster count, and the vertical axis measures the fraction of simulations resulting in a significant treatment effect at the 5% level. The graph reveals that between 9,000 and 10,000 clusters are needed to achieve power $= .80$. This closely aligns with the analytical derivation of the required sample size of 8,622 clusters.

Note that these power calculations will somewhat understate the required sample size because they do not account for the fact that within a cluster, externalities can only be measured on the subset of households that were not assigned the latrine treatment. Here, we have used the size of the whole cluster to simulate the externality, whereas the correct experiment would measure the externality within only the "leave-out" households in treatment clusters.

Calculating costs requires making additional assumptions about the efficacy of a hypothetical latrine intervention, on which very little data exists. For illustration, we note that Barnard et al. (2013) provides evidence on this question by examining a small number of Indian villages where latrines were built under the central government's Total Sanitation Campaign in the late 2000s. Barnard et al. (2013) shows that among individuals owning a latrine following the implementation of the program in their village, less than half were using the latrines.

To calculate a lower-bound estimate of the cost of an experiment that detected the mortality externalities of a latrine intervention, consider an intervention that converts non-latrine users to latrine users at a success rate of 50% by constructing a latrine and providing some information about its benefits and use at a cost of $500 USD per household.[50] With approximately 9,000 clusters and average cluster sizes of 200 households, this implies 40 interventions in each of the 4,500 treatment localities in order to generate the 10 percentage point first stage effect on latrine use. The cost of implementing the treatment alone (leaving out surveying and other costs) would equal $90 million.

A.2 Details of Non-Parametric Decomposition

As an alternative approach to statistically explaining the mortality gaps, we estimate counterfactual Hindu mortality rates after non-parametrically reweighting the sample of Hindu children to match the characteristics of Muslim children. Compared to the linear regressions in Section 5, this non-parametric approach has the advantage of more flexibly allowing correlation between open defecation and other controls.

Following DiNardo, Fortin and Lemieux (1996), we first reweight the Hindu sample according to a partition based on variables other than open defecation and report counterfactual outcomes. We then reweight according to a finer partition that interacts groupings of these variables with our sanitation variable. Here, sanitation (exposure to open defecation) is defined flexibly as an interaction between own and neighbors' latrine use. In particular, we divide both samples into 20 bins b of exposure to open defecation: 10 bands of local (PSU) open defecation interacted with household open defecation. Other variables are binned as follows: 3 survey rounds, 2 urban statuses, 8 bins of asset ownership, 3 terciles of household size, and 4 quartiles of birth order.[51] For each reweight on some combination these of characteristics, we follow three steps:

1. Within each sample $s \in \{Hindu, Muslim\}$ and each bin b, compute ω_b^s, the fraction of sample s in bin b, using survey design weights.

2. For each observation in the Hindu sample, create new counterfactual weights by multiplying the observation's survey sampling weight by the ratio $\frac{\omega_b^{Muslim}}{\omega_b^{Hindu}}$ for the bin b of which it is a member.

3. Compute a counterfactual mean Hindu mortality rate under the Muslim distribution of characteristics using these new weights.

Table A5 reports counterfactual Hindu infant mortality rates with the new weights. The first row displays the unweighted difference in means and the reweight on the marginal distribution of open defecation alone. The rest of the table explores the explanatory power of local open defecation when added sequentially after reweighting with respect to other factors. Row 1 shows that matching on open defecation alone completely accounts for, and even reverses, the direction of the gap. Sanitation non-parametrically accounting for 108 percent of the IMR gap is consistent with the fact that Hindu children come from richer families, on average, and would therefore be expected to have lower mortality. In the remaining rows, reweighting on various sets of covariates that do not include OD continues to generate a large mortality gap. Then, adding sanitation to the set of reweighting variables has a large incremental effect and explains the entire gap in most cases. The single case in

[50] The $500 figure follows Duflo et al. (2015) who report an approximate construction cost of $440 per latrine plus annual maintenance.

[51] The requirement in any reweighting exercise to create joint distributions that include full support in both subsamples limits the number of dimensions over which we can jointly reweight in a fixed sample size. See Geruso (2012) for a fuller discussion of this limitation.

which it fails to do so is the specification that includes a count of joint household assets, but does not control for the fact the Muslims live in larger households.

A.3 Problems with Survey-Reported Diarrhea

The NFHS contains information on mothers' reports of diarrhea in their children. This type of survey measure is likely to contain significant biases that may be correlated with our regressors of interest. For example, because the reporting of diarrhea depends on whether the reporting mother recognizes a loose stool as diarrhea, differences in reporting across children is correlated with the education level of their mothers. Appendix Table A10 illustrates this fact, regressing reported diarrhea on mother's education, where the omitted category is no education. The table also includes regressions where weight-for-age is the dependent variable. The table shows that reported diarrhea is only weakly correlated with education, even though children of higher educated mothers tend to show fewer measurable symptoms of the problem: Point estimates indicate that mothers with some education are weakly *more* likely to report diarrhea than those with no education (columns 1 and 2). This is despite the fact that weight moves in the predicted pattern, increasing with education. Columns 3 and 4 show that the weight of children is strongly correlated with mother's education.

We also note that in the NFHS data, the reported incidence of diarrhea fluctuates significantly across survey rounds: In our sample it is 11% in the 1992/1993 round, up to 19% in the 1998/1999 round, and then back down to 11% in the 2005/2006 round. This non-monotonicity over time stands in stark contrast to the wide evidence from elsewhere, including the Census of India, that infant mortality—which is largely accounted for by diarrheal disease (Million Death Study Collaborators, 2010)—was steadily declining in India over this time period. For these reasons, we focus our analysis on surveyor-measured weight-for-age, following the standard practice (Schmidt et al., 2011). For more detail on the problems with survey-reported diarrhea, see Schmidt et al. (2011).

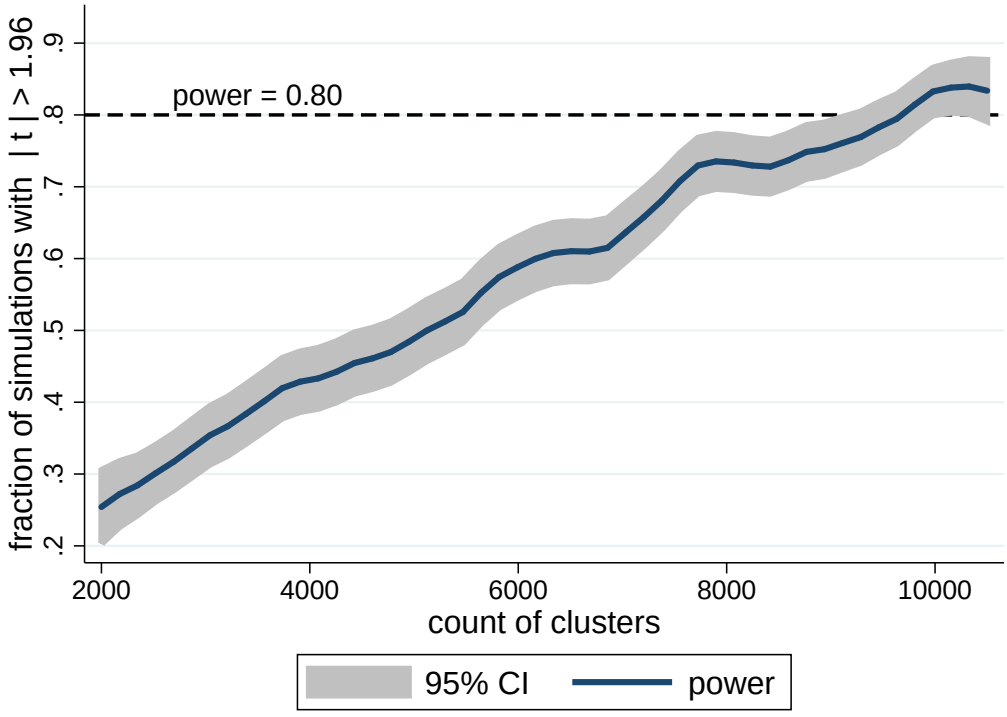
A.4 Acute Malnutrition Channel: Non-Parametric Estimates

To examine the robustness of the Table 7 results to alternative parameterizations, in Figure A2 we plot local polynomial regressions of weight-for-age on local open defecation separately for Hindu and Muslim children. The left panels include all observations. The right panels include only the mixed-religion PSUs that are used in the IV analysis. The top panels include no controls, and the bottom panels add controls by first separately regressing weight-for-age and $\overline{OD}_{ij}^{-i}$ on the controls, and then performing the local polynomial regression on the residuals from those regressions.[52] In the figure, higher values of the dependent variables indicate higher child weight, so these plots are strongly decreasing in $\overline{OD}^{-i}$.

In Panels A and B, Hindu infants appear to have a nutrition advantage after conditioning on local sanitation, but not other controls. In other words, controlling for $\overline{OD}^{-i}$ reverses the Muslim advantage seen in columns 1 and 4 of Table 7. This may reflect that within a PSU, Hindu families are richer on average and can better compensate for acute malnutrition—at least among infants who will ultimately survive and generate a weight measurement in our data. This advantage disappears once the standard set of controls are added in the bottom two panels of Figure A2. These plots show that the effects of $\overline{OD}^{-i}$ on child weight (i.e., the non-parametric slopes) are large and essentially identical across Hindus and Muslims.

[52] The control set used to generate residuals is the same as the extended controls in Tables 3 and 7, including own household open defecation.

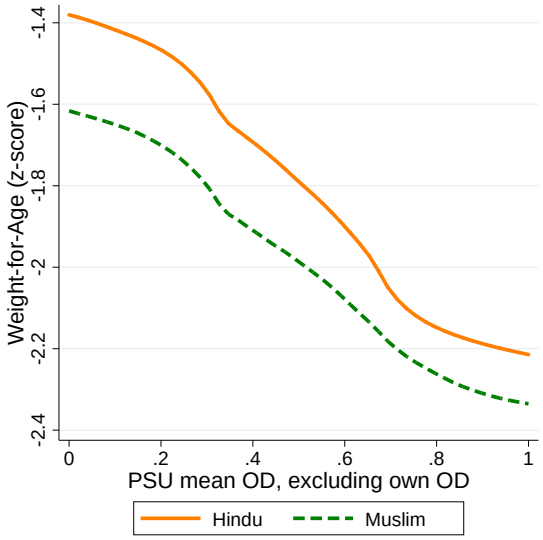
Figure A1: Experimental Sample Size Needed to Identify the Mortality Externalities of OD



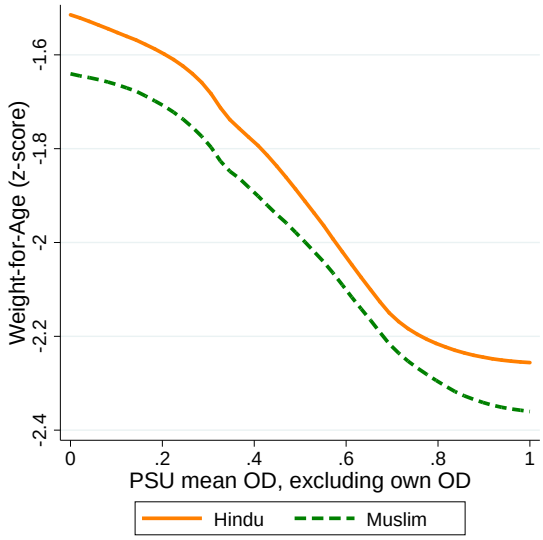
Note: Figure plots statistical power against the number of clusters for a hypothetical experiment that generates infant mortality reductions equal to 5 percent of the mean infant mortality rate via a cluster-level externality. Observations are generated by sampling NFHS survey data, following a Monte Carlo procedure described in Appendix A.1. The line in the figure is a local polynomial regression of the simulation result on the cluster count.

Figure A2: Test of Mechanism: $\overline{OD}$ and Acute Net Malnutrition

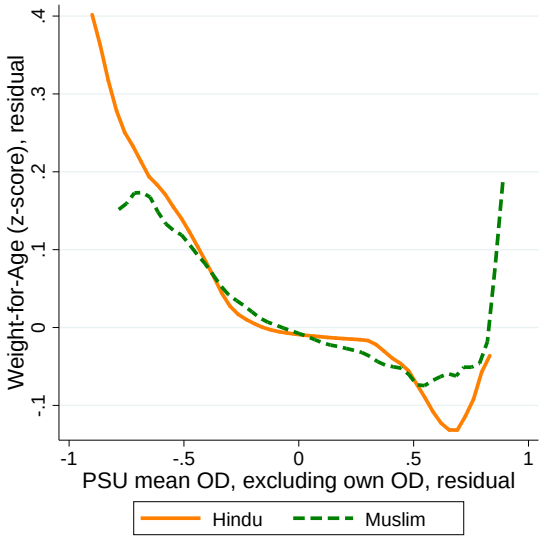
(A) Full sample, no controls



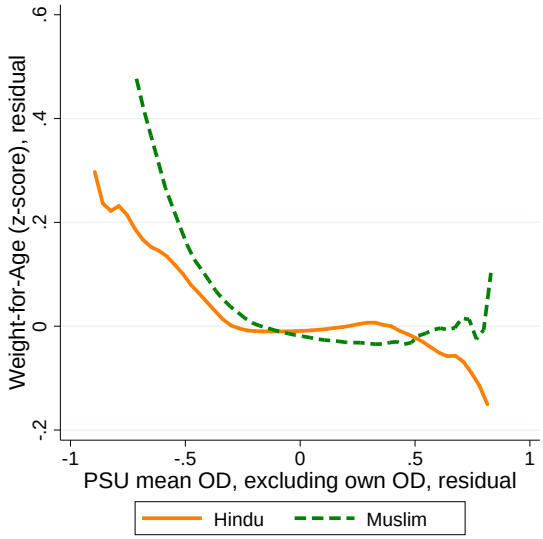
(B) Mixed religion PSUs, no controls



(C) Full sample, with controls



(D) Mixed religion PSUs, with controls



Note: Figure plots local linear regressions of weight on local area open defecation. Weight is an outcome reflecting acute malnutrition, which is an important mechanism by which poor sanitation causes infant mortality. The dependent variables are the weight-for-age z-score, scaled to the World Health Organization's child growth standards, or its residual. The top panels (A and B) display plots with no controls. The bottom panels (C and D) display semi-parametric regressions using residuals generated by first regressing the independent and dependent variables on the extended controls defined in the Table 3 notes. The left panels (A and C) include the full sample, and the right panels (B and D) include only mixed-religion PSUs ($0 < \overline{M} < 1$). Observations are live births.

Table A1: Correlates of Muslim Concentration Across PSUs

dependent varible:	assets (fraction of 7)		electricity		piped water		urban	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fraction PSU Muslim	-0.039***	-0.076***	-0.041	-0.155***	-0.091**	-0.169***	0.077*	-0.032
	(0.010)	(0.008)	(0.029)	(0.023)	(0.029)	(0.026)	(0.035)	(0.029)
HH Muslim & HH OD		X		X		X		X
mean of dep. var.	0.275	0.275	0.569	0.569	0.368	0.368	0.357	0.357
observations (live births)	104,090	104,090	104,090	104,090	104,090	104,090	104,090	104,090

dependent Varible:	mother education in years		mother literate		household size		birth order	
	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
Fraction PSU Muslim	-0.392*	-0.258	0.005	-0.003	0.756***	0.173	0.580***	0.255***
	(0.181)	(0.141)	(0.022)	(0.018)	(0.141)	(0.145)	(0.054)	(0.054)
HH Muslim & HH OD		X		X		X		X
mean of dep. var.	3.014	3.014	0.400	0.400	7.553	7.553	3.054	3.054
observations (live births)	104,090	104,090	104,090	104,090	104,090	104,090	104,090	104,090

dependent Varible:	father education in years		child ever vaccinated		family has healthcard		institutional delivery	
	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)
Fraction PSU Muslim	-0.542***	-0.386***	-0.027	-0.006	-0.061**	-0.038	-0.011	-0.047*
	(0.079)	(0.080)	(0.021)	(0.021)	(0.023)	(0.022)	(0.025)	(0.021)
HH Muslim & HH OD		X		X		X		X
mean of dep. var.	3.313	3.313	0.790	0.790	0.568	0.568	0.357	0.357
observations (live births)	77,122	77,122	30,078	30,078	30,182	30,182	31,252	31,252

dependent Varible:	birth assistance		mother has own money		mother has healthcare say		clean cooking fuel	
	(25)	(26)	(27)	(28)	(29)	(30)	(31)	(32)
Fraction PSU Muslim	-0.047	-0.089***	0.004	0.035	0.032	0.016	-0.116**	-0.147***
	(0.025)	(0.021)	(0.040)	(0.042)	(0.024)	(0.026)	(0.043)	(0.032)
HH Muslim & HH OD		X		X		X		X
mean of dep. var.	0.433	0.433	0.441	0.441	0.275	0.275	0.328	0.328
observations (live births)	31,271	31,271	27,123	27,123	26,474	26,474	27,111	27,111

Note: Table reports results from a series of OLS regressions in which the dependent variable is a characteristic of a household, parent, or child, and the single regressor is the fraction of the PSU in which the child resides that is Muslim ($\overline{M}$). The sample is limited to mixed religion PSUs ($0 < \overline{M} < 1$) over which the IV analysis is defined. Observations are children (live births), and sample size varies across regressions because some survey questions were asked to only subsets of respondents. Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A2: Correlates of IMR: Signing the Potential Biases (see Table A1)

dependent variable:	Infant Mortality (IMR)			
Regressor:	assets (fraction of 7)	electricity	piped water	urban
	(1)	(2)	(3)	(4)
coefficient in IMR regression	-76.9***	-35.6***	-22.7***	-27.0***
	(3.9)	(2.1)	(2.1)	(2.1)
observations (live births)	104,090	104,090	104,090	104,090
Regressor:	mother education in years	mother literate	household size	birth order
	(5)	(6)	(7)	(8)
coefficient in IMR regression	-4.8***	-37.9***	-2.9***	4.3***
	(0.2)	(1.9)	(0.2)	(0.5)
observations (live births)	104,090	104,090	104,090	104,090
Regressor:	father education in years	child ever vaccinated	family has healthcard	institutional delivery
	(9)	(10)	(11)	(12)
coefficient in IMR regression	-2.4***	-116.7***	-57.8***	-26.5***
	(0.5)	(4.8)	(2.8)	(2.7)
observations (live births)	77,122	30,078	30,182	31,252
Regressor:	birth assistance	mother has own money	mother has healthcare say	clean cooking fuel
	(13)	(14)	(15)	(16)
coefficient in IMR regression	-29.2***	2.600	0.900	-29.4***
	(2.7)	(3.3)	(3.6)	(3.5)
observations (live births)	31,271	27,123	26,474	27,111

Note: Table reports results from a series of OLS regressions in which the dependent variable is IMR. Each column reports a separate regression of IMR on a single regressor, which is listed in the column header. Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). The sample is limited to mixed religion PSUs ($0 < \overline{M} < 1$) to correspond to Table A1. Observations are children (live births), and sample size varies across regressions because of the design of the DHS questionnaire, which asked some questions to only subsets of respondents. Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A3: Robustness of First Stage Result: Splits by Subsamples

dependent variable:	PSU mean OD (except own)					
	(1)	(2)	(3)	(4)	(5)	(6)
	Panel A: split by child sex					
	Full Sample		Boys		Girls	
PSU fraction Muslim	-0.121**	-0.174**	-0.119**	-0.174**	-0.124**	-0.174**
	(0.013)	(0.012)	(0.013)	(0.012)	(0.014)	(0.013)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,090	104,090	53,779	53,779	50,311	50,311
	Panel B: split by child birth order					
	Full Sample		First Birth		Second or Higher Birth	
PSU fraction Muslim	-0.121**	-0.174**	-0.107**	-0.161**	-0.127**	-0.178**
	(0.013)	(0.012)	(0.013)	(0.012)	(0.014)	(0.013)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,090	104,090	27,020	27,020	77,070	77,070
	Panel C: split by own religion					
	Full Sample		Muslims		Hindus	
PSU fraction Muslim	-0.121**	-0.174**	-0.113**	-0.179**	-0.135**	-0.166**
	(0.013)	(0.012)	(0.018)	(0.016)	(0.015)	(0.014)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,090	104,090	34,052	34,052	70,038	70,038
	Panel D: split by PSU location					
	Full Sample		Urban		Rural	
PSU fraction Muslim	-0.121**	-0.174**	-0.054**	-0.103**	-0.177**	-0.202**
	(0.013)	(0.012)	(0.019)	(0.017)	(0.018)	(0.016)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,090	104,090	37,209	37,209	66,881	66,881

Note: Table reports results from a series of OLS regressions in which the dependent variable is the mean of neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). The regressor of interest is the fraction of the PSU that is Muslim ($\overline{M}$). Column 1 reports results from the full sample. Columns 2 and 3 and Columns 4 and 5 estimate the identical OLS regression for each of the subsamples defined in the panel headers. All regressions control for own religion and own OD. Regressions in columns 4 and 6 include the extended controls as described in the Table 3 notes. All regressions include only the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A4: Robustness of Table 3: Splits by Child Sex, Birth Order, and Urban/Rural

Panel A: split by child sex						
dependent variable:	IMR					
sample restriction:	Full Sample		Boys		Girls	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-9.0**	-1.3	-10.6**	-3.9
	(1.5)	(2.3)	(2.0)	(3.2)	(2.0)	(3.1)
own household OD		19.7**		20.2**		19.2**
		(1.7)		(2.3)		(2.4)
PSU mean OD (except own)		26.5**		22.3**		31.1**
		(2.3)		(3.0)		(3.1)
mean of dep. var.	72.2	72.2	73.7	73.7	70.6	70.6
observations (live births)	278,423	278,423	144,269	144,269	134,154	134,154

Panel B: split by child birth order						
dependent variable:	IMR					
sample restriction:	Full Sample		First Birth		Second or Higher Birth	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-6.7*	-1.4	-10.5**	-2.6
	(1.5)	(2.3)	(2.7)	(4.2)	(1.7)	(2.7)
own household OD		19.7**		22.1**		19.3**
		(1.7)		(2.9)		(2.0)
PSU mean OD (except own)		26.5**		39.2**		21.8**
		(2.3)		(3.8)		(2.6)
mean of dep. var.	72.2	72.2	74.4	74.4	71.4	71.4
observations (live births)	278,423	278,423	76,253	76,253	202,170	202,170

Panel C: split by PSU location						
dependent variable:	IMR					
sample restriction:	Full Sample		Urban		Rural	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-3.7+	-4.6	-8.8**	-1.4
	(1.5)	(2.3)	(2.1)	(3.4)	(2.0)	(3.1)
own household OD		19.7**		21.6**		18.9**
		(1.7)		(3.1)		(2.0)
PSU mean OD (except own)		26.5**		21.8**		27.4**
		(2.3)		(4.7)		(3.4)
mean of dep. var.	72.2	72.2	52.0	52.0	80.9	80.9
observations (live births)	278,423	278,423	83,344	83,344	195,079	195,079

Note: Table reports results from a series of OLS regressions. The dependent variable is IMR, scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). Columns 1 and 2 repeat the main results from Table 3 for reference. Columns 3 through 6 replicate the regressions in the subsamples defined in the column headers. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A5: Nonparametric Reweight: Counterfactual Hindu Mortality Under Muslim Exposure to Open Defecation

Hindu Raw Mean:	Muslim Raw Mean:	Raw Gap to Explain:
73.93	63.17	10.76

Reweighting variables					Reweight Results				
Round	Urban	House-hold Assets	House-hold Size	Birth Order	Reweight without OD	Residual Gap to Explain	Reweight with OD	Incremental Effect of OD Reweight	Percent Explained
					(1)	(2)	(3)	(4)	(5)
					73.93	10.76	62.31	11.62	108%
X					72.63	9.46	62.29	10.34	109%
X	X				70.52	7.35	61.95	8.57	117%
X	X	X			72.72	9.55	65.58	7.14	75%
X	X	X	X		69.58	6.41	61.76	7.82	122%
X	X	X	X	X	70.32	7.15	62.68	7.64	107%

Note: Table presents a nonparametric decomposition of the extent to which sanitation differences can account for infant mortality differences between Hindus and Muslims. Xs in the left of the table indicate the characteristics over which the reweight of the joint distribution is performed. Column 1 presents counterfactual mortality rates for Hindu children ($\times$1000), using the empirical Hindu distribution of exposure to OD and the Muslim distribution of other characteristics. Column 3 presents counterfactual mortality rates for Hindu children after matching the Muslim joint distribution of exposure to OD and the indicated characteristics. The distribution of open defecation is defined over 20 bins of exposure: 10 bands of local (PSU) open defecation interacted with household open defecation. Other characteristics are binned as follows: 3 survey rounds, 2 urban statuses, 8 bins of asset ownership, 3 terciles of household size, and 4 quartiles of birth order. The final row matches the distribution of characteristics across 11,520 (=$20\times3\times2\times8\times3\times4$) cells.

Table A6: Robustness of IV Results in Table 4: Splits by Subsamples

dependent variable:	Infant Mortality (IMR)		
	(1)	(2)	(3)
	Panel A: split by child sex		
	Full Sample	Boys	Girls
PSU mean OD (except own)	64.9**	58.5*	72.0*
	(22.8)	(29.7)	(30.5)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	53,779	50,311
	Panel B: split by child birth order		
	Full Sample	First Birth	Second or Higher Birth
PSU mean OD (except own)	64.9**	69.2	62.7*
	(22.8)	(42.4)	(25.4)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	27,020	77,070
	Panel C: split by own religion		
	Full Sample	Muslim	Hindu
PSU mean OD (except own)	64.9**	35.1	91.9**
	(22.8)	(28.3)	(35.3)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	34,052	70,038
	Panel D: split by PSU location		
	Full Sample	Urban	Rural
PSU mean OD (except own)	64.9**	74.9	68.8**
	(22.8)	(55.6)	(26.4)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	37,209	66,881

Note: Table reports results from a series of IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). Column 1 reports results from the full sample IV regression in column 4 of Table 4 for comparison. Columns 2 and 3 estimate the identical IV regression over each of the subsamples defined in the panel headers. All regressions include only the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). All regressions control for survey round fixed effects, own religion, own OD and the extended controls as described in the Table 3 notes, except for the single variable on which the sample is split in each panel (sex, birth order, etc.). Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A7: No Evidence of Interaction Between Neighbors' OD and Own Household OD

dependent variable:	Infant Mortality (IMR)		
	Full Sample	Household Defecates in the Open	Household Uses Latrine
	(1)	(2)	(3)
PSU mean OD (except own)	64.9**	79.0*	63.1+
	(22.8)	(31.5)	(32.8)
own religion	X	X	X
extended controls	X	X	X
observations (live births)	104,090	60,959	43,131

Note: Table reports results from a series of IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). Column 1 reports results from the full sample IV regression in column 4 of Table 4 for comparison. Columns 2 and 3 estimate the identical IV regression over each of the subsamples defined in the panel headers. All regressions include only the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). All regressions control for survey round fixed effects, own religion, and the extended controls as described in the Table 3 notes. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A8: Summary Statistics from Supplementary Dataset: The India Human Development Survey

	Hindu Subsample		Muslim Subsample	
	Mean	SD	Mean	SD
	(1)	(2)	(3)	(4)
household open defecation	0.51	0.50	0.31	0.46
local (PSU) open defecation	0.50	0.35	0.33	0.34
local (PSU) fraction Muslim	0.05	0.12	0.66	0.33
household has piped water	0.30	0.46	0.28	0.45
household is urban	0.30	0.46	0.44	0.50
ln(per capita consumption)	9.90	0.68	9.83	0.61
non-vegetarian household	0.23	0.42	0.40	0.49
meat, household kg per month conditional on any	3.15	3.65	4.14	3.92
eggs, household dozen per month	4.17	10.98	9.73	18.64
milk, household liters per month	18.64	29.27	15.41	24.16
always wash hands after defecating[a]	0.72	0.45	0.70	0.46
usually or always wash hands after defecating[a]	0.96	0.18	0.96	0.20
always purify water[a]	0.11	0.32	0.12	0.33
usually or always purify water[a]	0.20	0.40	0.21	0.41
observations	32,572		4,623	

Note: Table displays summary statistics for the supplemental dataset used in Section 6.2, the 2012 round of the India Human Development Survey (IHDS). Observations are households.

[a] The sample sizes for the wash and water variables are slightly smaller than for the rest of the table because these were observed in the female questionnaire, rather than the main household questionnaire. These sample sizes are 32,254 and 4,550 for Hindus and Muslims, respectively.

Table A9: Robustness of IV Results: Mortality is Uncorrelated with Residing in a PSU that is Religiously Dissimilar from the Respondent Household

dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
sample restriction:	Mixed Religion PSU		Mixed Religion PSU	
specification:	IV	IV	IV	IV
	T4 - Col 4		T4 - Col 8	
	(1)	(2)	(3)	(4)
PSU mean OD (except own)	64.9**	65.3**	46.6*	46.7*
	(22.8)	(22.8)	(18.4)	(18.5)
own household OD	-14.8+	-14.8+	-8.3	-8.3
	(8.1)	(8.1)	(6.6)	(6.6)
fraction of PSU religiously dissimilar		-4.5		-1.0
		(3.9)		(3.2)
extended controls	X	X	X	X
mean of dep. var.	72.2	72.2	46.2	46.2
first stage F-stat	204.9	205.4	204.9	205.4
observations (live births)	104,090	104,090	104,090	104,090

Note: Table reports results from a series of IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$), instrumented with $\overline{M}_j$. The sample is restricted to the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). Columns 1 and 3 repeat results from Table 4 for comparison. Columns 2 and 4 add a control for the fraction of the responden'ts neighborhood that is religiously dissimilar, which equals $\overline{M}_j$ for Hindu households and $1 - \overline{M}_j$ for Muslim households. Extended controls are as described in the Table 3 notes. All regressions control for survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.

Table A10: Reliability of Self-Reported Diarrhea vs. Objective Measures of Acute Malnutrition

	dependent variable:			
	respondent reported diarrhea		weight-for-height z-score	
	(1)	(2)	(3)	(4)
Mother some education	0.008	0.013+	0.341**	0.099**
	(0.007)	(0.007)	(0.027)	(0.026)
Mother high education	-0.021**	-0.002	0.918**	0.326**
	(0.005)	(0.007)	(0.021)	(0.025)
extended controls		X		X
mean of dep. var.	0.17	0.17	-1.90	-1.90
observations (live births)	25,684	25,684	25,684	25,684

Note: Table reports results from a series of OLS regressions. In Columns 1 and 2, the dependent variable is the respondent's report of diarrhea in the child. In Columns 3 and 4, the dependent variable is the surveyor-measured weight and height, converted to a weight-for-height z-score according to the World Health Organization child growth standard. Some education corresponds to some primary education. High education corresponds to greater than primary education. The omitted category is no education. Extended controls are as described in the Table 3 notes. All regressions control for survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. + $p < 0.1$, * $p < 0.05$, ** $p < 0.01$.