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ABSTRACT

In the developing world, there has been significant policy interest in recent years in ending open defecation—that is, defecation in fields, behind homes, and near roads. This attention is in part motivated by a belief that the private demand for latrines and toilets is below the social optimum. We investigate the infant mortality externalities of poor sanitation by exploiting differences in the demand for latrines between Muslim and Hindu households in India: Indian Muslims, despite being poorer, are 25 percentage points more likely than Indian Hindus to use latrines or toilets. Instrumenting for local sanitation with the religious composition of neighborhoods, we show large infant mortality externalities of neighbors defecating in the open. Estimates of these neighbor effects are similar regardless of the household's own latrine use and own religion. Our findings are informative of the external harm generated by the roughly 1 billion people today who defecate in the open.

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1 Introduction

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.[1] 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, since 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, India's central government launched a 100-day construction plan in 2014 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." The economic justification for such investments relies on an implicit assumption that the private demand for toilets and latrines is below the social optimum, possibly because of inaccurate beliefs about the health benefits of latrine use, or because latrine use has an important public goods component. The public goods aspect of sanitation is indeed plausible. The epidemiological evidence suggests a clear pathway by which exposure to fecal pathogens introduced by neighbors could lead to acute net malnutrition and ultimately death.[2]

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. Efforts to estimate effects of latrine use on health outcomes via field experiments have been complicated by the fact that in many places there exist deeply rooted preferences against using latrines and toilets, even those freely provided.[3] Indeed, the difficulty in generating a first stage effect on latrine use, combined with the large number of clusters required

[1] See Cutler and Miller (2005) for an overview.

[2] We discuss the epidemiological evidence in detail in Section 3.

[3] 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., 2015; Gertler et al., 2015). See also Guiteras, Levinsohn and Mobarak (2015) on limits to traditional latrine promotion programs.

to measure mortality externalities, may explain the lack of any experimental evidence on mortality effects to date.[4]

In this paper, we provide the first econometric evidence on the infant mortality externalities of poor sanitation by exploiting a systematic difference in the demand for 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 revealed preference for open defecation among Hindus has been noted for at least a century and is widely recognized among current residents of rural India. This pattern of behavior 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 primary sampling units that are 10% Muslim are exposed to a local open defecation rate of 63%, while Hindus residing in primary sampling units that are 90% Muslim are exposed to a local open defecation rate of 46%.

This variation in local sanitation is, as we show, not positively correlated with measures of health inputs or economic well-being across the localities differentially settled by the groups. Instead, with the exception of sanitation, neighborhoods in which the minority Muslim populations are concentrated have observable characteristics that would imply worse infant health outcomes. This is consistent with the well-known disadvantage of the Muslims in India.[5]

We exploit the pattern of latrine demand differences between Hindu and Muslims, instrumenting for local sanitation with the religious composition of a neighborhood. 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 compared to the majority Hindus. 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,

[4]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.

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

Valente and van Soest (2010), is hard to reconcile with the well-developed literature on the importance of parental income and education in predicting child health and mortality.[6] Nonetheless, 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 of Muslim mortality advantage.[7] We show that this large difference can be entirely accounted for by latrine use: Own religious identity is correlated with own child survival because it is correlated with the religious identity of neighbors, and the religious identity of neighbors is correlated with child survival only because it predicts the local sanitation environment. In short, the Muslim mortality advantage is actually an advantage accruing to the neighbors of Muslims.

In OLS regressions, we find that a 10 percentage point reduction in the fraction of neighbors defecating in the open is associated with a decline in infant mortality of 2.7 to 2.9 infants out of 1000. IV regressions that isolate only the variation in local open defecation arising from the religious composition of one's neighbors—controlling for own religion and own sanitary practice—generate even larger point estimates of 6.5 infants per 1,000, or about 9% of the population mean IMR. These results are robust to a variety of alternative hypotheses, including differential son preference by Hindus and Muslims, sex $\times$ birth order explanations, and differential sorting of Muslims to urban areas. We also demonstrate that our findings are not driven by other behaviors of Muslim neighbors with the potential for externalities, such as hand washing.

These effects are large. 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 so is associated with about the same infant mortality reduction as the difference between the bottom and top quintiles of (own) wealth. Our estimates are also large relative to the prior literature identifying the impacts on infant and child mortality of intensive neonatal medical interventions (Almond, Chay and Lee, 2005), hospital integration (Almond, Chay and Greenstone, 2006), privatizing water services (Galiani, Gertler and Schargrodsky, 2005), and airborne pollutants (Currie and Neidell, 2005).[8]

[6] For overviews of the literature and applications in the US, see Cutler and Lleras-Muney (2010) and Geruso (2012).

[7] 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.

[8] Estimates in the literature include: a reduction of 6 to 13 deaths per thousand among blacks over months 1-12 of the integration of a hospital in Mississippi (Almond, Chay and Greenstone, 2006); a reduction of 10.7 deaths per thousand over months 0-12 of crossing the very low birth weight threshold in US (1.5 kg), thus triggering extra medical care (Almond, Chay and Lee, 2005); a reduction of 5.4 deaths per thousand over months 0-4 due to privatizing water in Argentina (Galiani, Gertler and Schargrodsky, 2005); and an effect of 2.4 deaths per thousand over months 0-12 of 1 ppm of CO pollution in California (Currie and Neidell, 2005).

Nonetheless, the idea that open defecation could explain such an important fraction of infant mortality in the modern Indian context is consistent with the evidence 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; Watson, 2006). The estimates are also consistent with the most credible data on cause of death for early-life mortality in India, which shows that 22% of non-neonatal child deaths (deaths from 1-59 months) in India are due to diarrhea, which is one symptom of the intestinal diseases transmitted via open defecation.[9] Indeed, we confirm in a mechanism check that low weight among infants, which reflects recent bouts of diarrhea, responds to our identifying variation in the same pattern as mortality.

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 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, decreasing in the fraction of one's neighbors who are Muslim).

Because it may be at odds with many readers' intuition that a relatively richer group with higher educational attainment would systematically choose open defecation, we supplement the aggregate national statistics on latrine use with an original survey collected by one of this study's authors in rural northern India in 2013 and 2014 to elicit richer data on stated and revealed preferences toward latrine ownership and use. The survey corroborates the observed demand patterns in national data. We shows 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, we show that Hindus are less likely to *use* it. The notion that demand for latrine ownership and use is not merely a matter of affordability also accords with recent qualitative work in India (Coffey et al., 2014*a*) and experimental interventions in Bangladesh (Guiteras et al., 2014) that have documented significant resistance to the adoption of effective, low-cost improvements to water quality and sanitation.

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

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 and human capital outcomes in the history of development in the US (Cutler and Miller, 2005, Watson, 2006, Bleakley, 2007, and Alsan and Goldin, 2015) and around the world (e.g. Spears, 2012 and Alsan, 2015).[10] Our study is unique in examining the external effects of open defecation on mortality. Establishing whether open defecation harm 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 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

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 most recent wave of the National Family Health Survey (NFHS) of India show that as of 2005, 68% of Hindu households defecate in

[10] 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. Alsan and Goldin (2015), in a study subsequent to ours, document that sewer and water improvements in Massachusetts beginning in the late 19th century accounted for a large share of the overall decline in infant mortality in the following decades. Alsan (2015) finds an important indirect role of the disease environment in Africa on precolonial institutions and subsequent growth. Spears (2012) evaluated a large sanitation project that constructed pit latrines in rural areas of India, finding improvements in child height and mortality, though the study could not distinguish between the impacts of own behavior and that of nearby neighbors.

the open—e.g., in fields, near streets, or behind bushes. In comparison, only 43% of the relatively poorer Muslim households do so.[11]

To investigate these patterns in more detail, we turn briefly to the Sanitation Quality, Use, Access, & 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*).[12]

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

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

[12] 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.

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 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*).[13] 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.[14] 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

[13]This is documented in qualitative study in rural north India by Coffey et al. (2014*a*). The study describes how a Hindu man in Haryana reported to interviewers: "[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." In contrast, a Muslim woman from Uttar Pradesh reported: "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 [to use a latrine]. If we have a latrine in the house, we will use it."

[14]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.

documented since at least the 19th century.[15] By definition, open defecation is not limited to a confined 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

[15] See Freedman, 1991 for examples.

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.[16] 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.

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

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 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 by assets. While 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 is higher among Hindus than Muslims.[17]

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 the variation in local average open defecation arising from the religious composition of a household's neighbors in the local primary sampling unit (PSU).[18] Despite significant residential sorting along religious lines, 31% of sample PSUs contain some fraction of Muslim residents strictly between zero and one. The median survey PSU contains observations on 27 households, which are sampled from PSU-level frames of about 100-200 households.[19] 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

[17] 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.

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

[19] Our data do not contain the sampling frame, but according to the DHS (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).

($\overline{OD}_j$) and the mean open defecation rate of neighbors in the PSU ($\overline{OD_{ij}^{-i}}$), where the superscript $-i$ indicates that the mean is 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 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 figure 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 plotting a local polynomial regression of $\overline{OD}_j$ on $\overline{M}_j$ at the individual level. The figure shows that 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.[20] 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 ($0 < \overline{M} < 1$).

[20] We confirm the sign of the relationships between these variables and mortality in Appendix Table A2 in a regression that controls simultaneously for each of the outcome measures plotted in the figure.

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 religiously 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, most are either uncorrelated with $\overline{M_j}$ or are changing in a way that would predict *worse* child health as Muslim concentration increases.

The only exceptions to this pattern are $\overline{OD_j}$ (our variable of interest), urban/rural status, and household size. Muslims are more likely to locate in urban areas, and for this reason we control for urban/rural status in the analysis below. As a robustness check, we also replicate results separately in urban and rural areas. In our sample, Muslims tend to have larger families, creating a mechanical relationship with $\overline{M_j}$. We control for household size below, 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, addressing 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, is confounding the results.

The finding that Muslim-dominated neighborhoods are worse for infant survival along observables also accords with popular beliefs and observations about the relative disadvantage of the Muslim minority India.[21] In sum, the evidence suggests that instrumenting for $\overline{OD_j}$ with $\overline{M_j}$ would, if anything, understate the effects of interest.[22] As we show, the IV estimates are large, implying that the lower bound is informative in this case.

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

[22] 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 $\overline{M_j}$ 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.

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 ($\overline{OD_{ijt}^{-i}}$):

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

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

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

Here i indexes live births, j indexes survey PSUs, and t indexes the three NFHS survey rounds. 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.[23] M_{ijt} is an indicator for the child residing in a Muslim household. 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 and always include a fully saturated set of interacted sex $\times$ birth order indicators. We cluster standard errors by PSU.

In Section 5, we begin by exploiting the variation linking in $\overline{OD_{ijt}^{-i}}$ with $\overline{M_{jt}}$ to solve the long-noted puzzle that Muslim children in India have dramatically 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 isolate only the variation in neighborhood sanitation arising from the religious identity of neighbors by instrumenting for $\overline{OD_{ijt}^{-i}}$ with $\overline{M_{jt}}$. The latrine use polynomial plotted in Panel B of Figure 2 visually depicts our instrument. In Appendix Table A3, we report the linear regression coefficients that precisely describe the first stage coefficients corresponding to our IV analysis. 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

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

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.

The IV specifications below always control for own latrine use and own religion. Including own religion addresses any potential confounding variables that are correlated with religion and have primarily private benefits, such as differing Hindu and Muslim diets. To address the possibility that some other externality associated with the hygiene behaviors of Muslim neighbors is generating our findings, we examine the extent to which Hindus and Muslims differ along other hygiene behaviors that could plausibly produce externalities.

Finally, we present two tests of the externality channel we describe. First, acute (net) malnutrition is an intermediate health outcome that should precede deaths caused by exposure to poor sanitation and therefore should follow the same pattern as our mortality results. We examine effects on acute malnutrition, measured by weight. Second, because water and food are two key pathways through which poor sanitation causes infections in children, our hypothesis generates the sharp prediction that breastfeeding—which filters fecal pathogens whereas water and other foods do not—should be more effective in terms of infant survival where $\overline{OD}$ is higher (and therefore, where one's neighbors are Hindus, rather than Muslims).

5 The Mortality Puzzle and Solution

This section presents evidence that patterns of latrine use can completely account for the large Muslim mortality advantage that has been documented in India, despite the relative 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 sur-

veys, 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.[24] 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. For each outcome, the first specification in the series (columns 1 and 4) includes no controls for open defecation or the religious composition of the neighborhood. Then controls are sequentially added for the religious composition of the PSU (columns 2 and 5) and then own and neighbors' open defecation (columns 3 and 6).

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

[24] 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. 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.

determinants of early-life health in India has highlighted: 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 DHS 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 indicator for the child being a multiple birth; an urban indicator; and household size.

Table 3 shows that open defecation can fully account for the respective 11.0 and 7.6 (per thousand) Hindu-Muslim gaps in infant and neonatal mortality, which are estimated as the coefficients on the Muslim indicator in columns 1 and 4. First, controlling for the fraction of the PSU that is Muslim removes the association between own religion and mortality in columns 2 and 5. Then incrementally adding controls for own household open defecation (OD_{ij}) and the fraction of neighbors that openly defecate ($\overline{OD_{ij}^{-i}}$) attenuates both the Muslim identity coefficient and 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. These results imply that the Muslim survival advantage that has been widely documented as a puzzle is actually an advantage accruing to the neighbors of Muslims, and further that this Muslim neighbor advantage operates via a latrine use channel.[25]

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 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, since Hindu and Muslim household sizes in

[25]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.

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 Appendix Table A5 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 Appendix Table A6 we replicate the main 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 A.2, we describe an alternative approach to statistically explaining the mortality gaps. There, we estimate counterfactual Hindu mortality rates after non-parametrically 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 A7 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, the association between the religious composition of neighborhoods and the local sanitation environment can fully and robustly account for a mortality gap that has been deemed a puzzle in the literature. 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 Externalities

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 pre-

dicts 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).

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. In contrast, in Panel A, the nonparametric plots jump at the endpoints ($\overline{M} = 0$ and $\overline{M} = 1$) for many variables, suggesting that all-Muslim and all-Hindu neighborhoods may be systematically different from mixed-religion neighborhoods.

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. Unlike Table 3, these OLS specifications do not control for the instrument, $\overline{M_j}$. Columns 2 and 6 report instrumented coefficients with no controls. Columns 3 and 7 add controls for each household's own latrine use and its own religion, and columns 4 and 8 add the set of extended controls from Panel B of Table 3.

Table 4 shows that instrumenting for $\overline{OD_{ij}^{-i}}$ with $\overline{M_j}$ yields point estimates larger than the OLS regressions, though less precisely estimated. To put the size in context, the IV coefficient in column 4 implies that a 10 percentage point reduction in the fraction of neighbors openly defecating is associated with a decline in infant mortality of 6.5 children out of 1000. For neonatal mortality in column 8, the figure is 4.9 deaths per 1000. This magnitude is similar across specifications that vary the control set. 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), though it is important to note that the IV coefficients are not statistically different from the OLS.

Because Figure 2 and Tables A1 and A2 showed that, if anything, bias push our IV results towards zero, these estimates likely imply a lower bound. Nonetheless, the effects are economically large. 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). These effects are also large relative to the wider literature on the determinants

of mortality at young ages in the US and abroad.[26]

Another way to view the size of effects is in terms of the external harm created by a single household defecating in the open. 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.[27] While 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 Table 3 are about one third and one sixth the size, respectively, of the OLS and IV estimates of the external harm (the coefficient on $\overline{OD_{ij}^{-i}}$) in Table 4.[28]

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 A8 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 A8 shows that splitting the sample by girls and boys, first-borns and later-borns, or urban and rural PSUs generates quantitatively similar IV results.

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, not relegated to areas trafficked only by those who don't 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 OD_i). In unreported OLS regressions, we expand the specifications

[26] Estimates in the literature include: a reduction of 5.4 deaths per thousand over months 0-4 due to privatizing water in Argentina (Galiani, Gertler and Schargrodsky, 2005); a reduction of 6 to 13 deaths per thousand among blacks over months 1-12 from the integration of a hospital in Mississippi (Almond, Chay and Greenstone, 2006); a reduction of 10.7 deaths per thousand over months 0-12 of crossing the very low birth weight threshold in US (1.5 kg), thus triggering extra medical care (Almond, Chay and Lee, 2005); and an effect of 2.4 deaths per thousand over months 0-12 of 1 ppm of CO pollution in California (Currie and Neidell, 2005).

[27] 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 openly defecating 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.

[28] Both the private and external harm would have the same 1 in 10 scaling described in Footnote 27, due to the roughly 10% probability of any household containing an infant.

in Table 3 to estimate this interaction, though we find no statistically significant effects.

6.2 Robustness: External Effects of Other Hygiene

Our interpretation of the IV results relies on an exclusion restriction that Muslim neighbors do not positively impact child health through some channel other than latrine use. The health and mortality benefits generated by latrine use could be similar in principle to benefits conferred by other proper hygiene practices. Indeed, experimental evidence by Luby et al. (2005) in South Asia has shown that hand washing can impact diarrhea and pneumonia. To address the possibility that the external effects we estimate are driven by some other hygiene-related public good associated with the religious composition of a neighborhood, in this section we show that Hindus and Muslims do not in fact differ significantly along hygiene behaviors other than latrine use.

Our main dataset contains very limited information regarding hygiene practices other than latrine use. For that reason, we turn briefly to the India Human Development Survey (IHDS) of 2004-2005, which contains better measures of hand washing and the treatment of water, but for which we cannot construct similarly reliable mortality rates.[29] Our goal with the IHDS is therefore to examine whether Hindu-Muslim differences exist in other hygiene behaviors.

In Table 5, we regress indicators for several hygiene and water quality variables from the IHDS on an indicator for being a Muslim household. Results are displayed in two panels, with the top panel reporting differences in the unconditional means in a regression that includes no controls, and the bottom panel controlling for log household consumption and an indicator for urban status. In the first column, we replicate the pattern from our main analysis that Muslims are less likely to OD. Column 2 shows that there is no association between religious identity and hand washing after defecating. Column 3 shows there is no association between religious identity and hand washing with soap. Column 4 shows Muslims are no more likely than Hindus to purify their water. Column 5 shows the only economically large or statistically significant difference in the table besides latrine use: Muslims are significantly less likely than Hindus to have water piped to their homes. Both the literature (Jayachandran and Kuziemko, 2010) and our own analysis (Table A2) suggest that lower access to piped water in this region implies a Muslim disadvantage with respect to health, operating

[29] Specifically, we are limited by the fact that complete birth histories were not recorded for all women of childbearing age.

against our findings.[30] The difference in piped water likely reflects the inferior access to state services faced by Muslims. In sum, the table shows that differences in human waste disposal between Hindus and Muslim appear not to carry over to advantages in even a single other category of hygiene or water. The difference in latrine use, therefore, is not merely a marker for other important hygiene behaviors with public goods qualities.

Also, although it is not possible to completely rule out the possibility that PSU-level OD is correlated with some unobservable local characteristic that simultaneously predicts Muslim concentration and mortality, Table 3 in the previous section also provides evidence that PSU-level OD is not strongly correlated with other observables that predict mortality. The coefficient estimates for $\overline{OD_{ij}^{-i}}$ are remarkably stable between specifications with no controls and those with controls. Comparing Panels A and B in Table 3 reveals that the estimates without and with controls are 29.4 versus 27.1 in the IMR regression and 17.8 versus 17.9 in the NMR regression, respectively. In contrast, coefficients on the household's own use of a latrine (OD_{ij}) attenuate significantly with the inclusion of controls in Panel B, suggesting that own—but not neighbors'—use is correlated with other parent or household determinants of child health.

6.3 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.

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 combatting 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

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

in weight. This intermediate outcome may also be of independent interest, since acute malnutrition could impact the human capital accumulation of surviving children.

We follow the standard practice (e.g. Schmidt et al., 2010) of using on 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). As an alternative measure of acute malnutrition, we also examine weight-for-height. 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. We operationalize both measures as z-scores scaled to the World Health Organization's child growth standards.

Table 6 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 12 months for whom a weight measurement was taken. 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.[31] The extended controls are identical to those in the bottom panel of Table 3 with an additional control for height.[32] 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 6 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 san-

[31] 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.

[32] Weight-for-height regressions additionally control for height-for-age to ensure that the result is not spuriously driven by any heterogeneity in height or *chronic* malnutrition. Weight-for-age regressions do not include the additional control.

itation 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 0.052 standard deviations and a decline in weight-for-height of 0.046 standard deviations.[33]

To examine the robustness of this result to alternative parameterizations, in Figure 4 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.[34] 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 6. 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 4. 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. Together, the results in Table 6 and Figure 4 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

[33]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.

[34]The control set used to generate residuals is the same as the extended controls in Tables 3 and 6, with the addition of own household open defecation.

association with $\overline{OD^{-i}}$.

Breastfeeding. 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 7 examines interactions between religion, breastfeeding, and OD 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, exclusive breastfeeding has a large negative effect on mortality incidence. But the significant positive coefficient estimate for the interaction term breastfed $\times$ Muslim indicates that breastfeeding is less effective among Muslim children, on average. Put differently, breastfeeding appears to be differentially beneficial to the health of Hindu children compared to Muslim children.

However, additionally controlling for $\overline{OD_{ij}^{-i}}$ and its interaction with breastfeeding in column 2, shows that, again, this is merely due to the correlation between own religious identity and that of neighbors. Muslim children, on average, benefit less from breastfeeding because they tend to live in better local sanitation environments, while Hindu children, on average, face environments where the protection conferred by breastfeeding matters more. The positive coefficients on the indicator for Muslim and its interaction with breastfeeding become insignificant and change signs with the addition of open defecation controls. Although not statistically significant, the sign reversal 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.

The main coefficient of interest is on $\overline{OD_{ij}^{-i}} \times$ breastfed in column 2, which indicates that 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 $\left(= \frac{250 \times 10\%}{104}\right)$. This in-

teraction effect is consistent with the notion that breastfeeding filters the fecal pathogens that would otherwise be ingested by infants. 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 a main channel by which infants are harmed by neighbors' open defecation.

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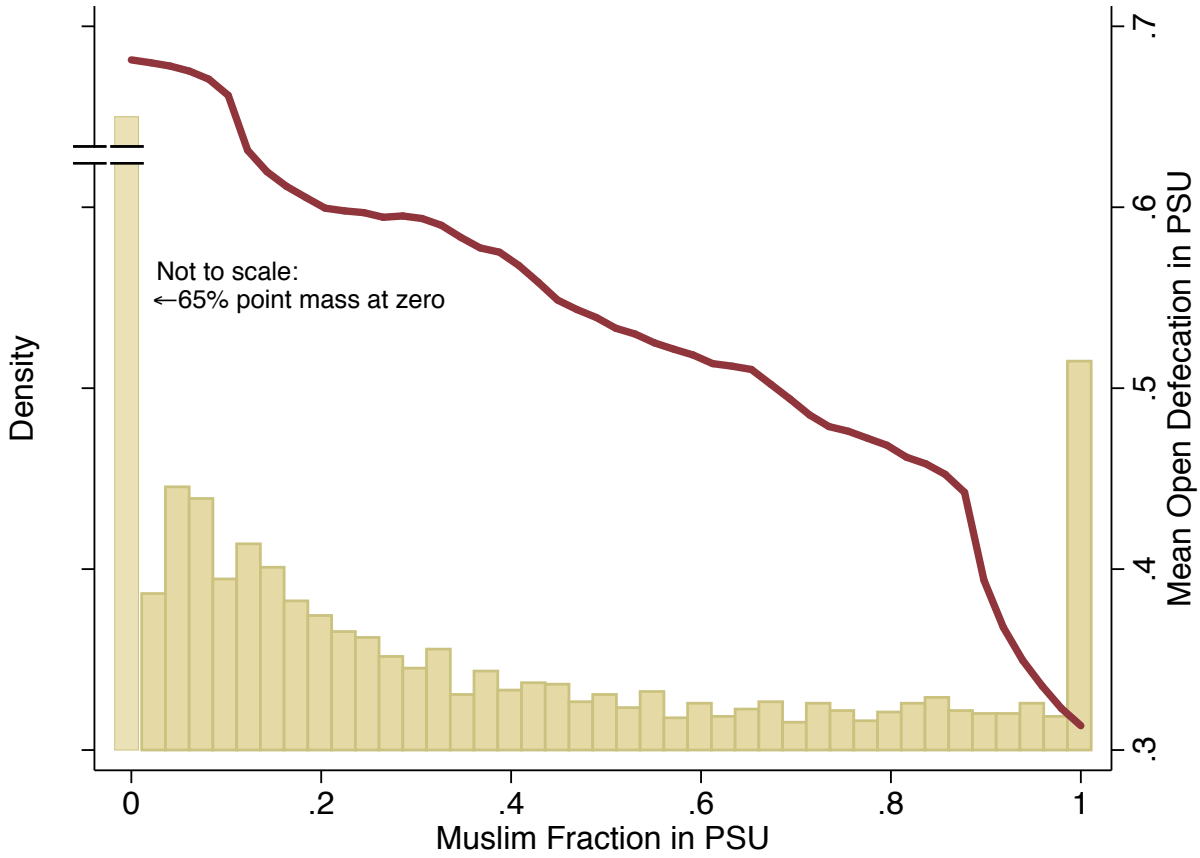
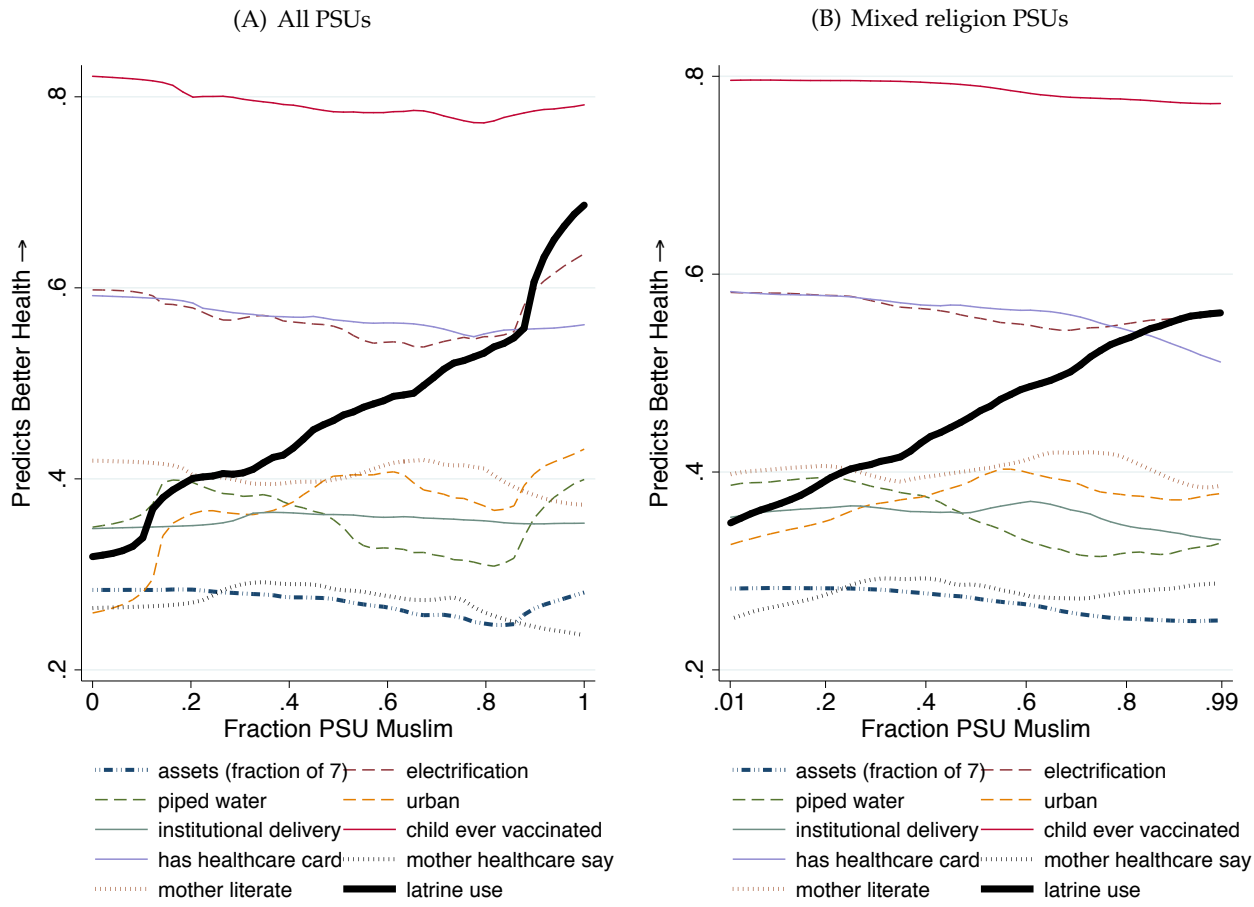
Figure 1: Identifying Variation: Muslim Concentration ($\overline{M}$) and Open Defecation ($\overline{OD}$) Across PSUs



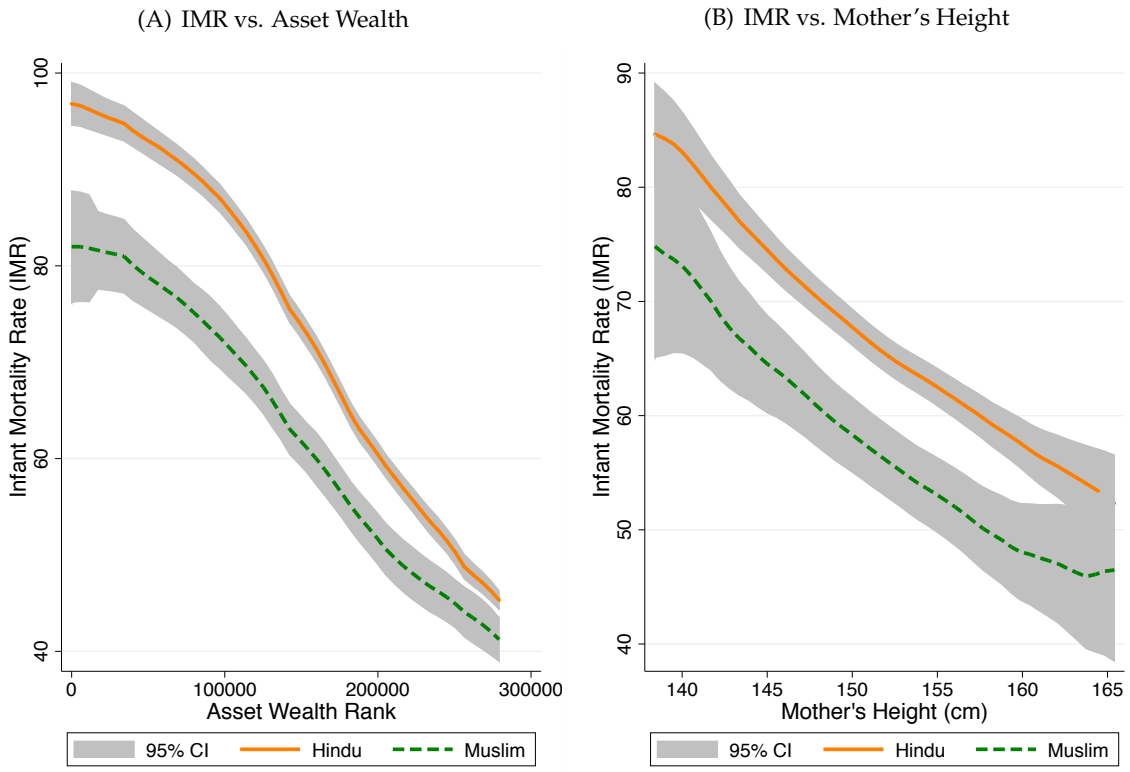
Note: Measured along the left vertical axis, the figure shows the histogram of Muslim concentration across primary sampling units (PSUs) in the NFHS survey. In the histogram observations are PSUs. Most PSUs are perfectly segregated along religious lines, with either 0% or 100% Muslim concentration. The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

Figure 2: Household Characteristics Varying with Muslim Concentration in the PSU ($\overline{M}$)



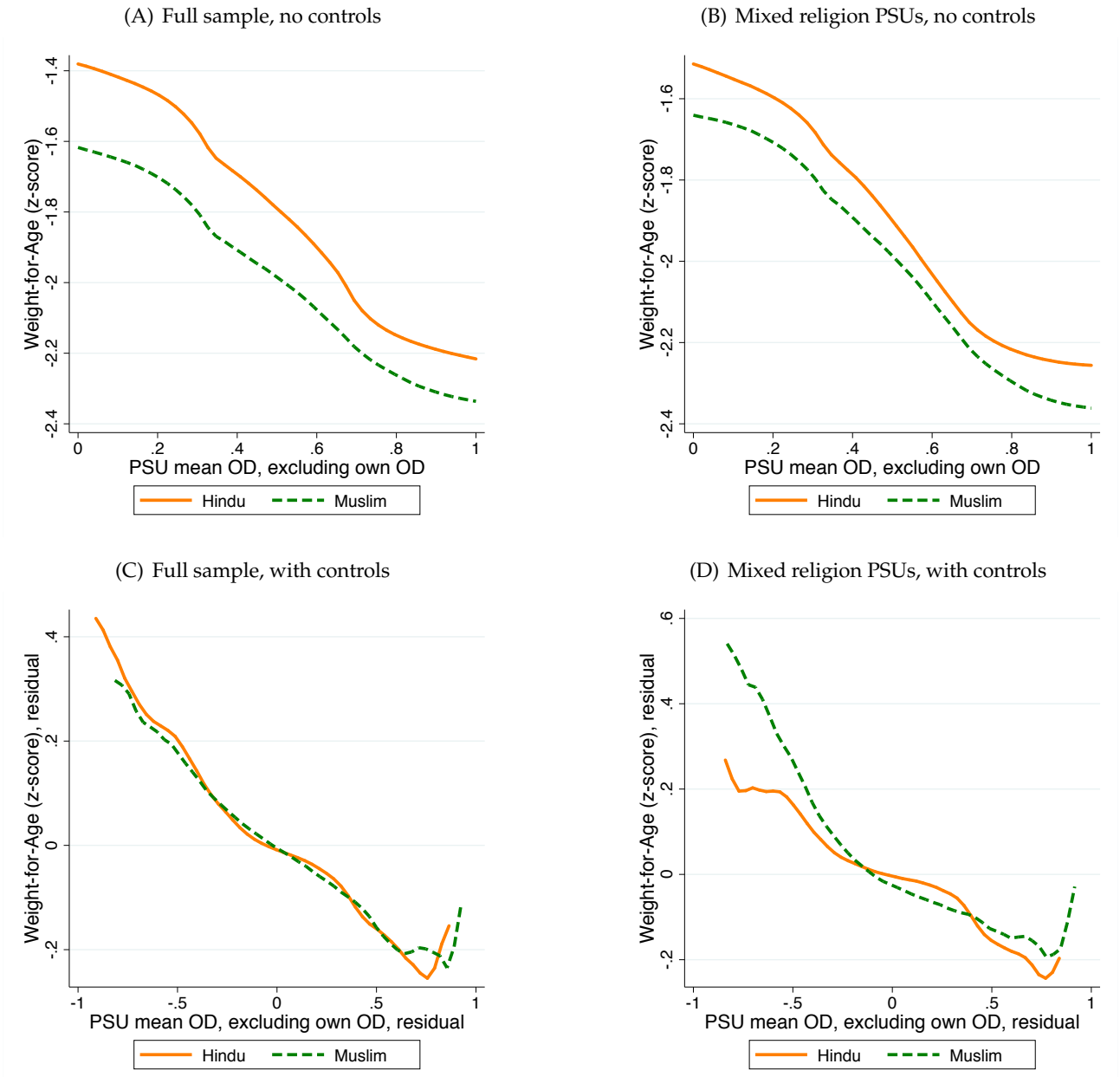
Note: Figure plots local linear regressions. The dependent variables are household-level characteristics, and the regressor is Muslim concentration ($\overline{M_j}$), which is measured at the PSU-level. Each dependent variable is defined such that higher values predict better child health outcomes. The signs of the relationships between these variables and 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: The Hindu-Muslim Mortality Puzzle, Conditioning on Parental Wealth and Health



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.

Figure 4: Test of Mechanism: Effects of $\overline{OD}$ on Acute Net Malnutrition



Note: Figure plots local linear regressions of weight, an outcome reflecting acute malnutrition, on local area open defecation. 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 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	0.725	0.456
		(0.003)	(0.013)
open defecation, conditional on owning latrine	all persons in household	0.252	0.099
		(0.006)	(0.010)
owns latrine	household	0.335	0.516
		(0.010)	(0.036)
says religious leader ever told them where to defecate	respondent	0.159	0.326
		0.008	0.036
says open defecation far from home is pure	respondent	0.526	0.397
		(0.010)	(0.036)
says latrine use near home is pure	respondent	0.455	0.538
		0.010	-0.037

Note: Table reports means and standard errors of survey responses from the Sanitation Quality, Use, Access, & Trends (SQUAT) Survey, 2013. 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, NFHS 1, 2, & 3

	Hindu Subsample		Muslim Subsample	
	Mean	SD	Mean	SD
	(1)	(2)	(3)	(4)
infant mortality rate (IMR), year 1	74.0	261.8	63.0	243.0
neonatal mortality rate (NMR), month 1	47.4	212.6	39.9	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	0.36	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	0.63	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)	232,863		46,462	

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: No Controls					
dependent variable:	Infant Mortality (IMR)			Neonatal Mortality (NMR)		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-11.0**	-3.8	-2.5	-7.6**	-2.5	-1.7
	(1.5)	(2.3)	(2.3)	(1.2)	(1.9)	(1.9)
PSU fraction Muslim		-11.5**	4.2		-8.2**	1.4
		(3.0)	(3.0)		(2.5)	(2.5)
own household OD			19.9**			12.2**
			(1.7)			(1.4)
PSU mean OD (except own)			29.4**			17.8**
			(2.3)			(1.8)
mean of dep. var.	72.1	72.1	72.1	45.6	45.6	45.6
observations (live births)	279,325	279,325	279,325	279,325	279,325	279,325
	Panel B: Extended Controls					
dependent variable:	Infant Mortality (IMR)			Neonatal Mortality (NMR)		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-6.1**	-2.5	-1.5	-3.8**	-1.0	-0.3
	(1.5)	(2.3)	(2.3)	(1.2)	(1.9)	(1.9)
PSU fraction Muslim		-5.9*	2.3		-4.5+	1.1
		(2.9)	(3.0)		(2.4)	(2.5)
own household OD			8.2**			6.2**
			(1.7)			(1.4)
PSU mean OD (except own)			27.1**			17.9**
			(2.7)			(2.2)
extended controls	X	X	X	X	X	X
mean of dep. var.	72.1	72.1	72.1	45.6	45.6	45.6
observations (live births)	279,325	279,325	279,325	279,325	279,325	279,325

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. 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 DHS survey rounds; 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; and household size. Observations are children (live births). Standard errors are clustered at the PSU level.

Table 4: IV Estimates: Variation in Open Defecation Arising from the Religious Composition of Neighbors

dependent variable:	Infant Mortality (IMR)				Neonatal Mortality (NMR)			
sample restriction:		Mixed Religion PSU				Mixed Religion PSU		
specification:	OLS	IV	IV	IV	OLS	IV	IV	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PSU mean OD (except own)	25.0**	75.0**	56.5+	65.3*	17.1**	53.0**	45.5+	48.6*
	(4.4)	(14.3)	(32.5)	(29.8)	(2.8)	(11.0)	(26.1)	(24.2)
own household OD	7.9**		3.3	-7.8	13.2**		-3.2	-5.2
	(2.6)		(18.7)	(11.8)	(2.1)		(15.1)	(9.6)
Muslim	-3.2		-3.6+	-2.1	-2.9+		-2.7+	-0.5
	(2.0)		(2.0)	(2.1)	(1.6)		(1.6)	(1.7)
extended controls	X			X	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		80.9	90.6	98.1		80.9	90.6	98.1
observations (live births)	104,309	104,309	104,309	104,309	104,309	104,309	104,309	104,309

Note: Table reports results from a series of OLS and IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). 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. Observations are children (live births). Standard errors are clustered at the PSU level.

Table 5: Hindus and Muslim Differences Along Other Hygiene Behaviors

dependent variable:	OD	Wash Hands	Wash Hands with Soap	Purify Water	Has Piped Water
	(1)	(2)	(3)	(4)	(5)
PSU fraction Muslim	-0.212**	-0.008	0.023	0.046	-0.184**
	(0.032)	(0.011)	(0.028)	(0.034)	(0.031)
ln(consumption)	-0.219**	0.004**	0.189**	0.107**	0.107**
	(0.008)	(0.001)	(0.008)	(0.008)	(0.007)
urban	-0.322**	0.005*	0.293**	0.166**	0.413**
	(0.015)	(0.002)	(0.016)	(0.016)	(0.018)
observations (households)	41,192	41,339	41,360	41,100	41,491

Note: Table reports results from OLS regressions, using data from the India Human Development Survey of 2004-2005. Dependent variables are listed in the column headers, and include an indicators for own household open defecation, for washing hands after defecating, for washing hands with soap, for purifying water, and for having piped water into the home. Controls include only those displayed in the table. Consumption is measured as rupees per month per capita. Observations are children (live births). Standard errors are clustered at the PSU level.

Table 6: Test of Mechanism: Effect on Acute Net Malnutrition

	dependent variable:					
	Weight-for-Age Z-score			Weight-for-Height Z-score		
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	0.058*	0.020	0.005	0.091**	0.011	-0.003
	(0.027)	(0.040)	(0.039)	(0.032)	(0.046)	(0.045)
PSU fraction Muslim		0.063	-0.098+		0.133*	-0.023
		(0.053)	(0.053)		(0.062)	(0.062)
own household OD			-0.182**			-0.185**
			(0.031)			(0.035)
PSU mean OD (except own)			-0.519**			-0.459**
			(0.048)			(0.056)
extended controls	X	X	X	X	X	X
mean of dep. var.	-1.90	-1.90	-1.90	-0.94	-0.94	-0.94
observations (live births)	23,965	23,965	23,965	21,204	21,204	21,204

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. 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. Columns 4 through 6 additionally control for height-for-age. The sample includes all children under 2 years. Observations are children (live births). Standard errors are clustered at the PSU level.

Table 7: Test of Mechanism: Interaction Between Breastfeeding Efficacy and $\overline{OD}$

dependent variable:	Infant Mortality (IMR)	
	(1)	(2)
breastfed	-245.1**	-104.3**
	(6.4)	(8.5)
Muslim	-32.2*	14.6
	(14.1)	(13.8)
breastfed X Muslim	29.7*	-17.7
	(14.2)	(13.8)
PSU OD (except own)		255.5**
		(13.9)
PSU OD (except own) X breastfed		-250.2**
		(13.7)
extended controls	X	X
observations (live births)	83,895	83,895

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 breastfeeding 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. Observations are children (live births). Standard errors are clustered at the PSU level.

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. 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.[35] 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 A7 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 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.

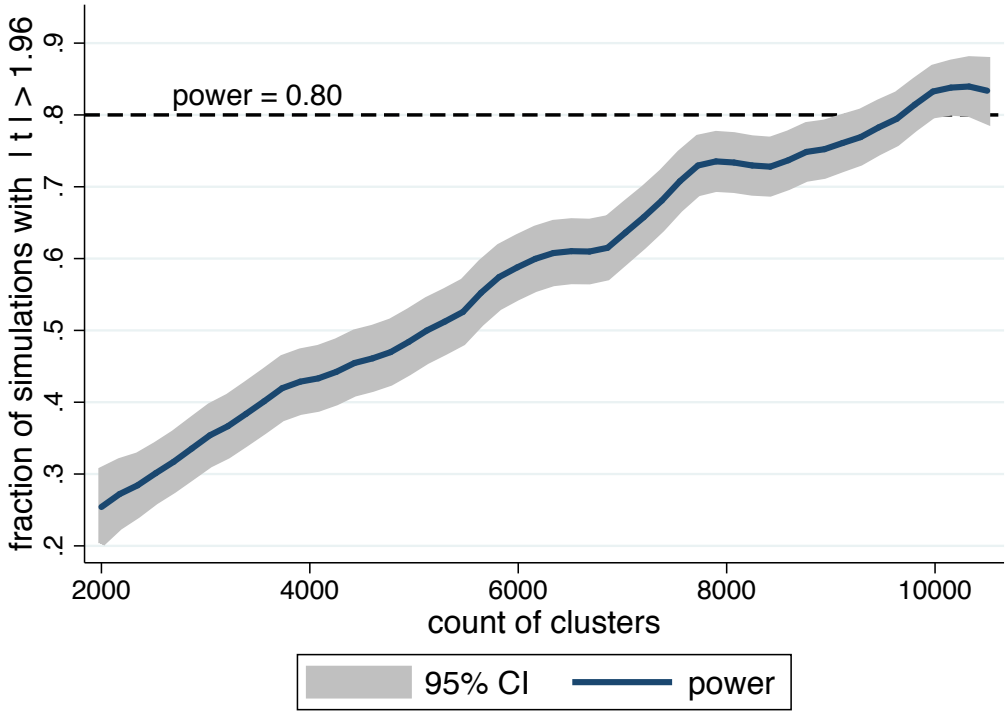
[35] The requirement in any reweighting exercise to create joint distributions that include full support in both subsamples limits the number of dimensions over which we can jointly reweight in a fixed sample size. See Geruso (2012) for a fuller discussion of this limitation.

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 A9 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 morality—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).

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 induces infant mortality effects equal to 10 percent of mean infant mortality 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.

Table A1: Household-Level Correlates of Muslim Concentration in the PSU

dependent Varible:	assets (fraction of 7)	electricity	piped water	urban	institutional delivery
	(1)	(2)	(3)	(4)	(5)
Fraction PSU Muslim	-0.039***	-0.041	-0.091**	0.077*	-0.012
	(0.010)	(0.029)	(0.029)	(0.035)	(0.025)
mean of dep. var.	0.281	0.592	0.358	0.300	0.351
observations (live births)	104,000	104,000	104,000	104,000	31,286

dependent Varible:	child ever vaccinated	family has healthcard	mother has healthcare say	mother literate	house has dirt floor
	(6)	(7)	(8)	(9)	(10)
Fraction PSU Muslim	-0.028	-0.062**	0.032	0.005	0.067
	(0.021)	(0.023)	(0.024)	(0.022)	(0.049)
mean of dep. var.	0.813	0.584	0.265	0.413	0.486
observations (live births)	30,111	30,215	26,487	104,000	27,131

dependent Varible:	birth assistance	mother education in years	father education in years	birth order	household size
	(11)	(12)	(13)	(14)	(15)
Fraction PSU Muslim	-0.047	-0.394*	-0.542***	0.581***	0.758***
	(0.025)	(0.181)	(0.079)	(0.054)	(0.141)
mean of dep. var.	0.432	3.182	3.340	2.921	7.320
observations (live births)	31,305	104,000	77,308	104,000	104,000

dependent Varible:	postnatal checkup	child immediately breastfed	mother weighed while pregnant	mother owns money	clean cooking fuel
	(16)	(17)	(18)	(19)	(20)
Fraction PSU Muslim	0.000	0.051*	-0.032	0.004	-0.117**
	(0.025)	(0.022)	(0.040)	(0.040)	(0.043)
mean of dep. var.	0.091	0.233	0.691	0.434	0.298
observations (live births)	3,168	13,975	5,817	27,136	27,124

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.

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	institutional delivery
	(1)	(2)	(3)	(4)	(5)
coefficient in IMR regression	-76.8***	-35.5***	-22.9***	-27.0***	-26.4***
	(3.9)	(2.1)	(2.1)	(2.1)	(2.7)
observations (live births)	104,309	104,309	104,020	104,309	31,286
Regressor:	child ever vaccinated	family has healthcard	mother has healthcare say	mother literate	house has dirt floor
	(6)	(7)	(8)	(9)	(10)
coefficient in IMR regression	-116.7***	-57.8***	0.900	-38.0***	28.8***
	(4.8)	(2.8)	(3.6)	(1.9)	(3.5)
observations (live births)	30,111	30,215	26,487	104,309	27,131
Regressor:	birth assistance	mother education in years	father education in years	birth order	household size
	(11)	(12)	(13)	(14)	(15)
coefficient in IMR regression	-29.2***	-4.8***	-2.4***	4.3***	-2.9***
	(2.7)	(0.2)	(0.5)	(0.5)	(0.2)
observations (live births)	31,305	104,177	77,308	104,309	104,309
Regressor:	postnatal checkup	child immediately breastfed	mother weighed while pregnant	mother has own money	clean cooking fuel
	(16)	(17)	(18)	(19)	(20)
coefficient in IMR regression	-24.7**	-7.0*	-15.4***	2.600	-29.4***
	(8.3)	(3.2)	(4.6)	(3.3)	(3.5)
observations (live births)	3,168	13,975	5,817	27,136	27,124

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 some survey questions were asked to only subsets of respondents. Standard errors are clustered at the PSU level.

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 own religion					
	Full Sample		Muslims		Hindus	
PSU fraction Muslim	-0.121**	-0.130**	-0.112**	-0.137**	-0.135**	-0.122**
	(0.0129)	(0.0133)	(0.0181)	(0.0178)	(0.0155)	(0.0153)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,309	104,309	34,138	34,138	70,171	70,171
	Panel B: split by child sex					
	Full Sample		Boys		Girls	
PSU fraction Muslim	-0.121**	-0.130***	-0.119**	-0.131**	-0.124**	-0.129**
	(0.0129)	(0.0133)	(0.0131)	(0.0134)	(0.0136)	(0.0138)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,309	104,309	53,875	53,875	50,434	50,434
	Panel C: split by PSU location					
	Full Sample		Urban		Rural	
PSU fraction Muslim	-0.121**	-0.130**	-0.0542**	-0.0579**	-0.176**	-0.168**
	(0.0129)	(0.0133)	(0.0186)	(0.0185)	(0.0177)	(0.0180)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,309	104,309	37,314	37,314	66,995	66,995
	Panel D: split by child birth order					
	Full Sample		First Birth		Second or Higher Birth	
PSU fraction Muslim	-0.121**	-0.130**	-0.107**	-0.118**	-0.127**	-0.134**
	(0.0129)	(0.0133)	(0.0130)	(0.0132)	(0.0136)	(0.0139)
own religion and OD	X	X	X	X	X	X
extended controls		X		X		X
observations (live births)	104,309	104,309	27,077	27,077	77,232	77,232

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 ($0 < \overline{M} < 1$). Observations are children (live births). Standard errors are clustered at the PSU level.

Table A4: Robustness of Table 3: Split by Child Sex

	Panel A: No Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		Boys		Girls	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-11.0**	0.0	-10.2**	0.0	-11.7**	0.2
	(1.5)	(1.5)	(2.0)	(2.0)	(2.0)	(2.0)
own household OD		19.9**		20.6**		19.3**
		(1.7)		(2.3)		(2.4)
PSU mean OD (except own)		29.0**		25.1**		33.4**
		(2.3)		(2.9)		(3.0)
mean of dep. var.	72.2	72.2	73.7	73.7	70.5	70.5
observations (live births)	279,325	279,325	144,722	144,722	134,603	134,603
	Panel B: Extended Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		Boys		Girls	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-6.1**	-0.2	-4.9*	0.6	-7.4**	-1.1
	(1.5)	(1.5)	(2.0)	(2.0)	(1.9)	(2.0)
own household OD		8.1**		8.6**		7.7**
		(1.7)		(2.3)		(2.4)
PSU mean OD (except own)		26.9**		23.8**		30.2**
		(2.7)		(3.5)		(3.5)
extended controls	X	X	X	X	X	X
mean of dep. var.	72.2	72.2	73.7	73.7	70.5	70.5
observations (live births)	279,325	279,325	144,722	144,722	134,603	134,603

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. Panel A includes no controls. Panel B includes the controls described in the Table 3 notes. Observations are children (live births). Standard errors are clustered at the PSU level.

Table A5: Robustness of Table 3: Split by Birth Order

	Panel A: No Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		First Birth		Second or Higher Birth	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-11.0**	0.0	-7.6**	4.1	-11.8**	-1.0
	(1.5)	(1.5)	(2.7)	(2.7)	(1.7)	(1.7)
own household OD		19.9**		22.4**		19.4**
		(1.7)		(2.9)		(2.0)
PSU mean OD (except own)		29.0**		41.2**		24.5**
		(2.3)		(3.7)		(2.6)
mean of dep. var.	72.2	72.2	74.4	74.4	71.3	71.3
observations (live births)	279,325	279,325	76,498	76,498	202,827	202,827
	Panel B: Extended Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		First Birth		Second or Higher Birth	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-6.1**	-0.2	-6.0*	0.8	-6.4**	-1.0
	(1.5)	(1.5)	(2.7)	(2.7)	(1.6)	(1.7)
own household OD		8.1**		7.5*		8.2**
		(1.7)		(3.1)		(2.0)
PSU mean OD (except own)		26.9**		38.0**		22.2**
		(2.7)		(4.4)		(3.1)
extended controls	X	X	X	X	X	X
mean of dep. var.	72.2	72.2	74.4	74.4	71.3	71.3
observations (live births)	279,325	279,325	76,498	76,498	202,827	202,827

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. Panel A includes no controls. Panel B includes the controls described in the Table 3 notes. Observations are children (live births). Standard errors are clustered at the PSU level.

Table A6: Robustness of Table 3: Split by Urban/Rural Status

	Panel A: No Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		Urban		Rural	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-11.0**	0.0	-4.3*	-1.6	-9.7**	1.4
	(1.5)	(1.5)	(2.1)	(2.1)	(2.0)	(2.1)
own household OD		19.9**		21.7**		19.2**
		(1.7)		(3.1)		(2.0)
PSU mean OD (except own)		29.0**		23.7**		32.0**
		(2.3)		(4.6)		(3.3)
mean of dep. var.	72.2	72.2	52.0	52.0	80.8	80.8
observations (live births)	279,325	279,325	83,696	83,696	195,629	195,629
	Panel B: Extended Controls					
dependent variable:	IMR					
sample restriction:	Full Sample		Urban		Rural	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-6.1**	-0.2	-6.1**	-3.6+	-7.2**	1.2
	(1.5)	(1.5)	(2.1)	(2.1)	(2.0)	(2.1)
own household OD		8.1**		8.4**		8.7**
		(1.7)		(3.1)		(2.1)
PSU mean OD (except own)		26.9**		17.6**		30.3**
		(2.7)		(4.6)		(3.3)
extended controls	X	X	X	X	X	X
mean of dep. var.	72.2	72.2	52.0	52.0	80.8	80.8
observations (live births)	279,325	279,325	83,696	83,696	195,629	195,629

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. Panel A includes no controls. Panel B includes the controls described in the Table 3 notes. Observations are children (live births). Standard errors are clustered at the PSU level.

Table A7: Nonparametric Reweight: Counterfactual Hindu Mortality Under Muslim Exposure to Open Defecation

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

Reweighting Variables					Reweight Results				
round	urban	joint household assets	household 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$1000), using the empirical Hindu distribution of exposure to OD and the Muslim distribution of other characteristics. Column 3 presents counterfactual mortality rates for Hindu children after matching the Muslim joint distribution of exposure to OD and the indicated characteristics. The distribution of open defecation is defined over 20 bins of exposure: 10 bands of local (PSU) open defecation interacted with household open defecation. Other characteristics are binned as follows: 3 survey rounds, 2 urban statuses, 8 bins of asset ownership, 3 terciles of household size, and 4 quartiles of birth order. The final row matches the distribution of characteristics across 11,520 (=20$\times$3$\times$2$\times$8$\times$3$\times$4) cells.

Table A8: Robustness of IV Results: Splits by Subsamples

dependent variable:	Infant Mortality (IMR)		
	(1)	(2)	(3)
	Panel A: split by own religion		
	Full Sample	Muslim	Hindu
PSU mean OD (except own)	65.26	43.80	95.21
	(29.84)	(36.79)	(47.20)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,309	34,138	70,171
	Panel B: split by child sex		
	Full Sample	Boys	Girls
PSU mean OD (except own)	65.26	57.32	73.97
	(29.84)	(38.64)	(40.29)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,309	53,875	50,434
	Panel C: split by PSU location		
	Full Sample	Urban	Rural
PSU mean OD (except own)	65.26	76.42	66.98
	(29.84)	(97.74)	(30.89)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,309	37,314	66,995
	Panel D: split by child birth order		
	Full Sample	First Birth	Second or Higher Birth
PSU mean OD (except own)	65.26	69.57	63.70
	(29.84)	(56.55)	(33.19)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,309	27,077	77,232

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 own religion and own OD and the extended controls as described in the Table 3 notes. Observations are children (live births). Standard errors are clustered at the PSU level.

Table A9: Self-Reported Diarrhea Versus Objective Measures of Acute Malnutrition

	dependent variable:			
	respondent reported diarrhea		weight-for-height z-score	
	(1)	(2)	(3)	(4)
Mother some education	0.008	0.012+	0.342**	0.147**
	(0.007)	(0.007)	(0.027)	(0.026)
Mother high education	-0.021**	-0.007	0.919**	0.393**
	(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)	25,709	25,709	25,709	25,709

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. Observations are children (live births). Standard errors are clustered at the PSU level.