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NEIGHBORHOOD SANITATION AND INFANT MORTALITY

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ABSTRACT

In this paper, we shed new light on a long-standing puzzle: In India, Muslim children are substantially more likely than Hindu children to survive to their first birthday, even though Indian Muslims have lower wealth, consumption, educational attainment, and access to state services. Contrary to the prior literature, we show that the observed mortality advantage accrues not to Muslim households themselves but rather to their neighbors, who are also likely to be Muslim. Investigating mechanisms, we provide a collage of evidence suggesting externalities due to poor sanitation are a channel linking the religious composition of neighborhoods to infant mortality.

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In this paper, we address 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 such as piped water and health infrastructure, compared to the majority Hindus.[1] By age one, mortality among Muslims is 17% lower than among Hindus, with an additional 1.1 infants per 100 surviving. Bhalotra, Valente and van Soest (2010) named this robust and persistent pattern a "puzzle," showing that individual and household characteristics could not explain it.[2]

After replicating the fact that Muslim children have a large survival advantage in India, we show that the mortality difference can be accounted for by two facts. First, compared to the typical Hindu infant, the typical Muslim infant lives in a neighborhood where a larger share of her neighbors are Muslim. Second, both Hindu and Muslim infants are more likely to survive in neighborhoods with high shares of Muslim neighbors. The natural question, then, is: what makes neighborhoods disproportionately inhabited by Muslims better places for the health of (Muslim and Hindu) children? We show, consistent with the well-known relative disadvantage of Muslims in India (Sachar et al., 2006 and Deolalikar, 2008), that neighborhoods with high shares of Muslim households are associated with *worse* characteristics that predict infant health along many observable dimensions—with the important exception of sanitation.

Despite relative economic advantage, India's majority Hindu population is 25 percentage points more likely to defecate in the open—that is, in open places such as in fields, behind bushes, or near roads—than the minority Muslim population. This Hindu-Muslim behavioral difference implies that the fraction of a household's neighbors who are Muslim is strongly correlated with the local sanitation environment to which the household is exposed. For example, in nationally representative data, Hindus residing in neighborhoods that are 10% Muslim are exposed to a local open defecation prevalence of 63%, while Hindus residing in neighborhoods that are 90% Muslim are exposed to a local open defecation prevalence of 46%. To better understand this variation, we draw on a survey by Coffey et al. (2014) in rural northern India that was designed to elicit richer detail on stated and

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

[2] 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.

revealed preferences toward latrine ownership and use. While both Hindus and Muslims in India are significantly more likely to defecate in the open than similarly poor people in other developing countries, there is a significant gap in attitudes, beliefs, and behaviors between these two groups in India. Hindus are more 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, Hindus are less likely to *use* it. The idea that latrine demand could be significantly influenced by social factors beyond price and wealth accords with recent qualitative work in India (Coffey et al., 2017) and experimental interventions in Bangladesh (Guiteras et al., 2014).[3]

After establishing that the mortality advantage accrues, in fact, to both Hindu and Muslim neighbors of Muslims and after observing that local sanitation is strongly associated with the share of Muslim neighbors, we provide a collage of evidence consistent with poor sanitation as the channel linking religious composition of neighborhoods to infant mortality. We show (i) that the partial correlation between Muslim neighbors and mortality diminishes once one controls for the sanitation practices of neighbors; (ii) that in the minority of neighborhoods where there is no open defecation, there is no mortality advantage associated with a higher proportion of Muslim neighbors; (iii) that where there is non-negligible open defecation, regional variation in the strength of the Hindu-Muslim difference in defecation behaviors predicts the regional strength of the Muslim mortality advantage; (iv) that other child outcomes with a specific biological link to the bacteria and worms spread by fecal matter (in particular, anemia and low current weight) are correlated with Muslim neighbors in the same pattern as infant mortality; and (v) that child health inputs (diet, hand washing, access to piped water) and child health outcomes (vaccinations, institutional births) that should not be affected by open defecation do not positively covary with the Muslim share in the neighborhood.

As a final exercise, we attempt to understand the potential size of the apparent mortality externalities of open defecation by instrumenting for neighborhood sanitation with the religious composition of neighbors, holding constant own religious identity and own latrine/toilet use. These regressions have the advantage that the coefficient of interest is identified off of a component of the variation in neighbors' behavior that is carefully documented in the paper. However, even though we show that the observed correlates of Muslim neighborhoods would tend to bias against the finding of a

[3] Coffey et al. (2017) document significant resistance to the adoption of effective, low-cost improvements to sanitation in India.

negative externality under this strategy, the neighborhoods where Muslims are concentrated may be different in unobservable ways, so these results are presented with caution. With that caveat, 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 6 infants per 1,000, or about 8% of the population mean infant mortality rate (IMR).[4] By replicating the IV results within various subsets of the data—for example, boys and girls separately, first-borns and later-borns separately, and rural and urban neighborhoods separately—we show that these results are not confounded by phenomena like differential son preference by Hindus and Muslims, birth order explanations, or differential sorting of Muslims to urban areas.

The implied importance of open defecation for infant mortality in the modern Indian context fits with evidence by Bleakley (2007), Cutler and Miller (2005), and Watson (2006) regarding the role of sanitation and water in determining health and human capital outcomes in the historical US.[5] In particular, Cutler and Miller (2005) show that clean water and sanitation interventions in US citites explain large shares of the decline in infant mortality in the US at the turn of the twentieth century.[6] Our estimates are also consistent with the most credible data on cause of death during early-life in India, which show that a large share (22%) of non-neonatal child deaths in India are due to diarrhea, which is just one symptom of the diseases transmitted via open defecation.[7]

It is difficult to overstate the potential importance of this issue. Today, more than a billion people continue to defecate in the open. Ending this practice has become a target of governments, NGOs, and private foundations. For example: ending open defecation was adopted in September 2015 among the UN's new Sustainable Development Goals; in India in 2014, the central government launched a 100-day construction plan during which one toilet or latrine was to be constructed every second; from 2007 to 2014, the World Bank Group committed an average of over $3 billion per year

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

[5] 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.

[6] Cutler and Miller (2005) show that the introduction of clean water technologies in US cities beginning around 1900 reduced the infant mortality rate by 87 deaths per thousand, or 46 percent of the base of 189 per thousand. Naively applying our IV estimates would predict that cutting open defecation in half from 66 percent to 33 percent would reduce infant mortality by 20 deaths per thousand ($= -0.33 \cdot 61.4$). This would be a 28 percent reduction in the baseline Indian infant mortality rate of 72 deaths per thousand during our sample period.

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

to water and sanitation; and since 2005, the Gates Foundation has invested hundreds of millions of dollars to improve sanitation in developing countries, including efforts to "reinvent the toilet." In parallel, researchers have investigated the economic (Guiteras, Levinsohn and Mobarak, 2015) and social (Guiteras et al., 2014) determinants of persistent open defecation. The rationale underlying much of this recent attention is that private demand for toilets and latrines may be below the social optimum, possibly because of inaccurate beliefs about the health benefits of improved sanitation, or because latrine use has an important public goods component. The public goods aspect of sanitation is indeed plausible. Epidemiological evidence and recent work by Duflo et al. (2015) suggest clear pathways by which exposure to fecal pathogens introduced by neighbors could lead to acute net malnutrition and ultimately death.[8] Nonetheless, the research significantly lags the policy action and investment.[9]

Our paper develops important new facts on sanitation in the modern developing world—in part by simply documenting striking patterns in stated and revealed preferences over latrines in India. But our study also contributes new evidence to a long-running debate: 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. The disease environment, including water and sanitation, has been found to be an important determinant of health and human capital outcomes around the world (Spears and Lamba, 2014; Gertler et al., 2015; Headey et al., 2015). Our study is unique in the economics literature in providing *even suggestive* evidence of the external impacts of open defecation on mortality. Establishing whether the harm from open defecation is primarily external is an important

[8]We discuss the epidemiological evidence in detail in Section 5. Duflo et al. (2015) show that an integrated water and sanitation improvement program in rural India reduced diarrhea episodes by 30-50%.

[9]There are several reasons why, despite significant interest in and spending on this issue, the externalities of open defecation are not yet well understood. For one, OLS estimates are likely to be biased because neighborhoods with worse sanitation practices are likely to be worse in other unobservable dimensions as well. Further, efforts to estimate the impacts of sanitation via field experiments have been complicated by difficulties in generating a first-stage effect on latrine use. In many places there exist deeply rooted preferences against using latrines and toilets, even those freely provided. 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, 2016; Clasen et al., 2015; Gertler et al., 2015). Indeed, the difficulty of changing latrine use behavior, combined with the large number of neighborhood clusters required to measure mortality externalities, may explain the lack of any experimental evidence on sanitation and mortality to date.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.

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 replicating the Muslim mortality puzzle and showing that the mortality advantage in fact accrues to both Hindu and Muslim households in Muslim-concentrated neighborhoods. Section 3 then documents how neighborhood characteristics vary with religious composition, suggesting the possibility of a sanitation channel. Section 4 draws on several household survey datasets to understand these neighborhood differences in light of striking differences between Hindu and Muslim respondents in stated beliefs and revealed preferences regarding the use of latrines. Section 5 provides a variety of corroborating evidence supporting a sanitation channel, and Section 6 puts the magnitudes of the apparent sanitation externalities in context.

2 The Puzzle of Muslim Mortality Advantage

2.1 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 15 to 49. Respondents report birth histories, including deaths and stillbirths, from which we calculate infant and neonatal mortality rates. The NFHS 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 1 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.[10] 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.[11] Throughout the paper,

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

[11] We focus on these mortality outcomes as they are measured closest in time to the open defecation measurement that

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.

Infant mortality is high across India, and consistent with previous studies, there is a large and significant Muslim survival advantage. Table 1 shows that across both groups during our sample period, more than 6 children in 100 died 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% higher among Hindus. Neonatal mortality shows a similar pattern, with a 19% survival deficit. This is despite Muslims having lower educational attainment and wealth, measured in this survey by assets. Though the NFHS does not measure consumption, the India Human Development Survey, which is used to supplement the main analysis and is described below, shows that mean and median consumption are higher among Hindus than Muslims as well.[12]

Below, we show that neighborhood characteristics are important in understanding the lower mortality rates of Muslim infants. We exploit the two-stage sampling structure of the survey design to identify neighborhoods as primary sampling units (PSUs).[13] The median survey PSU contains observations on 27 households, which are sampled from PSU-level frames of about 100-200 households. In rural areas, these PSU frames may be the entire village. In urban areas, they are census enumeration blocks.[14] Therefore, PSU identifiers allow us to construct neighborhood-level variables that reflect the characteristics of small localities. For each PSU j in the NFHS, we calculate the fraction of sample respondents that are Muslim, $\overline{M_j}$, referred to below as Muslim concentration or Muslim share.

2.2 The Puzzle

Figure 1 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-

comprises our variable of interest.

[12]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.

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

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

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.[15] 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 1 shows that infant mortality is steeply decreasing in both measures. The Muslim advantage documented by Shariff (1995), Bhat and Zavier (2005), Bhalotra and van 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 1 shows that about an 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 11.1 (per thousand) in Table 1. The confidence intervals in the figure are not adjusted for clustering, though as we show in regressions clustered at the neighborhood level in Table 2, the mortality gap is strongly statistically significant and persists after conditioning on a wider set of person, household, and neighborhood characteristics.

2.3 The Solution: Neighborhoods

In Table 2, we analyze the extent to which neighborhood composition can statistically account for the Hindu-Muslim mortality gap, net of the religious identity of the respondent household. In the table, observations are children (live births). We estimate regressions of the form

$$Y_{ijt} = \alpha_1 M_{ijt} + \alpha_2 \overline{M_{jt}} + \phi_{jt} + f(X_{ijt}) + \epsilon_{ijt}, \tag{1}$$

[15] 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 1; (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.

where i indexes live births, j indexes survey PSUs, and t indexes the three NFHS survey rounds.[16] The dependent variable, Y_{ijt}, is an individual-level mortality indicator for infant or neonatal death, as indicated in the column headers of Table 2. It is 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.[17] The regressor of interest is an indicator for whether the household in which the child lived is Muslim, M_{ijt}. In some specifications, we include controls for the fraction of the PSU that is Muslim $\overline{M_j}$, or PSU fixed effects, ϕ_{jt}. Standard errors are clustered by PSU.

The top panel includes no covariates X_{ijt} beyond indicators for the survey round. The bottom panel adds controls for a set of demographic and socio-economic characteristics that the literature on the determinants of early-life health in India has highlighted.[18] The first column in the table offers another illustration of the widely-documented mortality puzzle. In Panel A, the Muslim coefficient replicates the unconditional mean infant mortality difference discussed above. In the corresponding column of Panel B, the addition of individual, household, and PSU-level variables has essentially no net effect on the Muslim coefficient.

To capture the possibility of neighborhood effects, we include PSU fixed effects in columns 2 and 6 of Table 2. These controls attenuate the own Muslim coefficient and render it statistically insignificant. Contrary to the focus in the prior literature on the characteristics of Muslim respondents themselves, these columns show that it is the places where Muslims are more likely to reside that is most strongly associated with low infant mortality, and that this neighborhood effect is similar for Hindu and Muslim households. The respondent household's religious identity no longer predicts its child's survival. In the next two columns within each set of specifications (columns 3 and 7 in Panels A and B), we control for just one PSU-level variable: the fraction of the PSU that is Muslim ($\overline{M_j}$). Doing so has nearly the same effect on the own-Muslim coefficient as including a neighborhood fixed effect. Additionally controlling for indicators for all-Muslim and all-Hindu neighborhoods in columns 4 and 8 replicates the same statistically insignificant point estimate for the coefficient on own household Muslim as the specifications that are linear in $\overline{M_j}$. In all cases—infant and neona-

[16] All specifications include survey round fixed effects.

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

[18] These controls, labeled "extended controls" in Table 2, include a full set of birth order indicators interacted with sex (Pande and Jayachandran, 2013); indicators for household ownership of each of the seven assets asked about throughout survey rounds, the standard strategy for controlling for SES using these data (Filmer and Pritchett, 2001); an indicator for whether the mother lives with her husband's parents (Coffey, Khera and Spears, 2013); indicators for child's birth month (Doblhammer and Vaupel, 2001) and birth year; an urban indicator; household size; and several other individual and PSU-level variables. See the Table 2 notes for the full control list.

tal mortality with and without extended controls—controlling for the religious composition of the neighborhood removes the association between a household's own religion and the child's mortality. These results imply that what has been widely documented as a Muslim survival advantage is actually an advantage accruing to the neighbors of Muslims.

Figure 2 displays corresponding semi-parametric plots of infant mortality versus Muslim concentration. Consistent with a negative coefficient on Muslim concentration in Table 2, the negative slope in $\overline{M_j}$ in the figure indicates that infant mortality is lower for both Hindus and Muslims in neighborhoods with a greater proportion of Muslim neighbors. Also consistent with Table 2, the Muslim mortality advantage, reflected in the vertical distance between the regression lines, is smaller (about 4 deaths per thousand) and statistically insignificant once one holds constant the religious composition of a household's neighbors (Panel A). After additionally conditioning on own household latrine use—a behavior that figures prominently in the analysis below—the point estimates converge even more closely (Panel B).

3 Characteristics of Muslim Neighborhoods

The results in Table 2 raise an obvious question: What is different about neighborhoods where Muslims are likely to reside that confers a mortality advantage to the (Hindu *and* Muslim) residents of those places? In this section, we investigate how neighborhoods in this context vary with religious composition. We show a striking pattern in which Muslim-dominated neighborhoods appear to be *worse* in terms of characteristics that predict infant health along many observable dimensions—with the singular exception of sanitation.

3.1 Better or Worse Places?

Hindus and Muslims are largely segregated by neighborhood in India, but a substantial minority of neighborhoods are mixed. Figure 3 plots the histogram of Muslim share, $\overline{M_j}$, across PSUs, with the point mass at zero (= 65%) excluded to maintain a readable scale. The histogram shows that between the extremes of perfect segregation of the majority Hindu and minority Muslim populations, there is substantial variation in $\overline{M_j}$. In particular, 31% of survey primary sampling units (PSUs) contain some fraction of Muslim residents strictly between zero and one. These mixed neighborhoods are what

identify neighborhood religious composition effects separately from own religion in the regressions of Table 2.

To investigate how neighborhood-level characteristics covary with Muslim concentration, Figure 4 plots local polynomial regressions in which a set of ten variables capturing characteristics of children, parents, and their neighborhoods that are likely to be relevant health inputs, outputs, and correlates are regressed on $\overline{M_j}$. For ease of interpretation, we define each of the variables such that its range is $[0, 1]$ and higher values predict better child health outcomes.[19] The dependent variables include asset wealth (as the fraction of the seven assets reported in all survey rounds) and indicators for household electrification, household piped water, institutional delivery of the child, any child vaccinations, family possession of a national health card, mother having a say in child's healthcare, mothers' literacy, and latrine use. In Panel A, we include all PSUs in the data. In Panel B, we restrict the sample to the set of mixed-religion PSUs ($0 < \overline{M_j} < 1$).

Other than latrine use, the characteristics in Figure 4 are generally flat or declining in $\overline{M_j}$, indicating a mortality *dis*advantage of Muslim neighborhoods. The pattern is particularly clear and approximately linear in Panel B of Figure 4, which excludes religiously homogenous PSUs. The bottom panels of Figure 4 summarize this intuition by predicting infant mortality on the basis of the neighborhood-level means of the variables examined in Panels A and B, other than latrine use. We regress IMR on these variables, then apply the coefficient estimates to each PSU's characteristics to generate predictions.[20] The predicted IMR (dashed line) is monotonically increasing in Muslim concentration, for both the full sample (Panel C) and for the subset of mixed-religion PSUs (Panel D). The actual IMR (solid line) exhibits exactly the opposite pattern, decreasing in the proportion of Muslim neighbors.

For completeness, Appendix Table A1 reports the coefficients from the linear regressions corresponding to the local polynomials plotted in Panel B of Figure 4. 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 4 and Tables A1 and A2 show that among variables likely to cause or to be correlated with child health outcomes, almost all are either uncorrelated with $\overline{M_j}$ or are changing in a way that would predict *worse* child health as

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

[20] We also normalize the constant in the regression so that the mean predicted IMR exactly replicates the mean observed IMR.

Muslim concentration increases. In Section 5.3, we turn to an additional dataset, the India Human Development Survey, to show that Muslim concentration is likewise uncorrelated with diet and hygiene behaviors that are not observable in our main dataset. The finding that Muslim-concentrated neighborhoods are no better and possibly worse for infant survival along observables also accords with observations about the relative disadvantage of India's Muslim minority (Sachar et al., 2006; Deolalikar, 2008).

A clear exception to this pattern in which higher Muslim concentration predicts neutral or worse PSU characteristics is latrine use, defined as one minus the indicator for open defecation.[21] 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%. In aggregate, given the patterns of residential sorting, exposure to local open defecation is higher for the typical Hindu child by a margin of 21 percentage points (Table 1, row 4). The relationship between $\overline{M_j}$ and local open defecation (hereafter abbreviated OD) is primarily driven by variation in religious composition interacting with stable differences across Hindu and Muslim households in their probability of practicing open defecation. In Appendix Figure A1, we show that, within the Hindu and Muslim subsamples, there is also a small gradient in which both groups are more likely to safely dispose of feces as $\overline{M_j}$ increases.[22]

3.2 Neighborhood Sanitation and IMR

In light of Figure 4, we examine the correlation between the sanitation differences across Hindu and Muslim neighborhoods and the observed Hindu-Muslim mortality differences. To precisely control for neighbors' behavior separately from that of the respondent household, we calculate a neighbor OD rate, $\overline{OD_{ij}^{-i}}$, where the superscript $-i$ indicates a leave-out mean, taken over all households in PSU j other than the respondent household.

In Figure 5, we plot local polynomial regressions of IMR on $\overline{OD_{ij}^{-i}}$, separately for Hindus and Muslims. These plots parallel those from Figure 2 in which Muslim share was the regressor of interest, and ask whether the mortality association with sanitation follows the same pattern as the mortality association with religious composition. Panel A displays the raw correlation and Panel B

[21] Urban/rural status and household size show some association, but not after controlling for own household characteristics (own latrine use and own religion).

[22] This fact highlights the importance of controlling for the own household's use of latrines or toilets in any analysis examining the external effects of neighborhood-level sanitation.

controls for the household's own latrine use.[23] In both panels, the slope is similar across the two religious groups. At higher levels of $\overline{OD_{ij}^{-i}}$ in Panel A, point estimates suggest a possible Hindu-Muslim difference conditional on $\overline{OD_{ij}^{-i}}$, though that pattern is not robust and reverses in Panel B with the addition of a control for own household OD.[24]

In a series of supplemental tests in Table A3, we regress mortality outcomes on a Muslim indicator, local Muslim share, and own and neighbors' OD. The exercise is not intended to recover well identified causal effects of open defecation.[25] These "horserace" regressions address a more basic question: Does the partial correlation between Muslim neighbors and own child mortality diminish once one controls for the sanitation practices of neighbors? We find that it does. Consistent with Figure 5, adding controls for own household and neighbors' OD attenuates the coefficient on $\overline{M_j}$ to a magnitude that is economically small and statistically insignificant.

The result that neighborhood open defecation is strongly correlated with IMR is a very robust pattern.[26] A natural question in this context is whether differences in son preference and intra-household resource allocation between Hindus and Muslims could confound the results. To address this possibility, in Panel A of Appendix Table A4 we examine the correlation between local OD and IMR, splitting 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 once the measures of open defecation are included. We also investigate whether the apparent relationship is confounded by the pattern documented in Pande and Jayachandran (2013) in which sex $\times$ birth order interactions predict child health in India. The behavior has the potential for importance here, because Hindu and Muslim household sizes in India differ, and therefore so do the mean birth orders across groups. Nonetheless, this phenomenon does not explain the patterns here: All regressions in Panel B of Table 2 include the full set of sex indicators interacted with birth order indicators as controls, and Panel B of Table A4 shows that the

[23] Throughout, *latrine use* is used as a shorthand for latrine and toilet use, either of which can safely dispose of excreta.

[24] As in all of the local polynomial regression plots, the 95% confidence intervals drawn in the figure are not corrected for clustering and will overstate the statistical significance of Hindu-Muslim differences. We rely on Table A3 to assess the statistical significance of the OD-IMR cross-sectional relationship and Hindu-Muslim differences in it.

[25] Further below, we estimate IV specifications to get a sense of magnitudes of the effect of $\overline{OD_{ij}^{-i}}$ on IMR under strong identification assumptions that we make explicit.

[26] In Appendix Section A.2, we describe an alternative approach to statistically accounting for the mortality gaps. There, we estimate counterfactual Hindu mortality rates after nonparametrically reweighting the sample of Hindu children to match the joint distribution of characteristics in the Muslim child sample. Compared to the linear regressions 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. Appendix Table A5 reports the results of the exercise, which are closely consistent with Tables 2 and A3.

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 1 to differ significantly across religious groups, in Panel C of Table A4 we replicate the regression 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 overall patterns.

3.3 Heterogeneity in the Muslim Advantage

The facts above suggest that a channel operating behind the mortality advantage of Muslims may consist of two pieces. First, compared to the typical Hindu infant, the typical Muslim infant lives in a neighborhood where a larger share of her neighbors are Muslim. Second, Muslim neighbors are much less likely to defecate in the open. Below in Section 5.1 we discuss the plausibility of a biological mechanism linking neighbors' OD to infant mortality via fecal pathogens. Here, we first examine some basic statistical predictions of this interpretation that do not require a deep understanding of the biological mechanism.

First, we note that if the channel linking mortality and religious composition of neighborhoods is indeed better sanitation, then in the subset of neighborhoods in which OD is rare, and the channel is therefore closed off, Muslim neighbors should not predict lower mortality. Table 3 tests this idea, dividing the sample of survey PSUs according to whether there is non-zero open defecation, and re-running the mortality regressions from Table 2 within these partitions.[27]

There is a clear pattern in the point estimates across this division: In places with zero reported open defecation, Muslim identity has an insignificant and often *positive* association with morality. In places where open defecation is present, Muslim identity has a significantly negative association with morality. This general pattern holds whether one examines the correlation between own religion and IMR (Panel A), neighbors' religion and IMR (Panel B), or the partial correlations of own and neighbors' religion in the same regression (Panel C). It is striking that in places where the OD channel cannot operate by construction of the subsample, Muslim own identity and Muslim neighbor identity

[27] This partitioning—and more generally relying on measures of open defecation based on household-level responses—overstates the precision with which we can observe open defecation at the *person* level. This is because the type of household-level reports recorded in the NFHS do not account for the possibility of discordant latrine use behaviors among members of the same household. As we discuss in detail below in Section 4, there can be substantial within-household differences in latrine use. Thus, even setting aside measurement error due to the PSU mean OD being calculated from a small sample, there is error in this measurement. Person-level open defecation is rare in the PSUs in column 1 of Table 3, but likely not a precise zero.

are actually correlated with *higher* IMR, in clear contrast to the national, aggregate relationship that motivates this paper. The sign reversal is consistent with Figure 4, which shows that other than latrine use, neighborhoods with many Muslims tend to be worse in terms of features that would predict child health and human capital.

An alternative test in a similar spirit asks whether the Muslim neighborhood mortality effects are weaker in places where the "first stage" relationship between religious identity and sanitation practice is weaker. India is a large, geographically diverse country of 1.2 billion people. We exploit spatial heterogeneity across the states of India in the degree to which religious identity predicts revealed preference for open defecation. To do so, we divide the main survey data into 79 state-by-survey round cells.[28] For each cell, we calculate the means of IMR and OD separately for Hindus and Muslims. This allows us to calculate a first stage relationship $(\mathbf{E}[OD|\text{Hindu}] - \mathbf{E}[OD|\text{Muslim}])$ and a reduced form relationship $(\mathbf{E}[IMR|\text{Hindu}] - \mathbf{E}[IMR|\text{Muslim}])$ separately within each cell. In Panel A of Figure 6, we plot these statistics against each other. Each scatter point is a state-by-survey round, and marker sizes are proportional to sample size. Most of the points lie in the right-hand portion of the graph because in most places, the prevalence of OD among Hindus exceeds that of Muslims, consistent with the national averages. But behind those national averages lies some heterogeneity, and Panel A shows that only in the places where there is a strong behavioral difference in latrine use between Hindus and Muslims is there a strong association between religious identity and infant mortality. A regression line fit to the data points (in red) passes through the origin, indicating that in expectation, there is no relationship between religious identity and infant mortality in places where Hindus and Muslims have similar sanitation practices.

In Panel B of Figure 6, we retain the same horizontal axis, but on the vertical axis plot the strength of the religion-IMR relationship, as revealed in a regression of infant mortality on a Muslim indicator. This is the same regression that is reported in the first column of Table 3, but is here estimated separately for each state-by-round cell. The vertical axis measures the t-statistics for the coefficients, rather than the coefficients themselves, in order to reduce the visual noise associated with any large but statistically insignificant estimates generated within the 79 cells of the partitioned sample. (Appendix Figure A2 plots the analogous graph using coefficients themselves.) Here again, the strength and direction of the statistical relationship between religious identity and mortality is clearly related

[28] The number of states increased from 25 to 29 across the years represented in the survey rounds.

to the strength and direction of the first stage relationship: In the many places where Hindus are more likely to OD (right of the origin), Muslims, who tend to collocate near other Muslims, have an aggregate mortality advantage. In the few places where Hindus are less likely to OD (left of the origin), Muslims, who tend to collocate near other Muslims, have an aggregate mortality disadvantage.

Taken together, Table 3 and Figures 6 and A2 support the idea that sanitation may be the channel linking religious composition of neighborhoods to infant mortality. Where there is no OD, there is no Muslim mortality advantage. And where there is non-zero OD, the strength of the Hindu-Muslim difference in OD predicts the strength of the mortality advantage. This clearly suggests a mechanism in which neighborhood religious composition is predictive of IMR via its association with open defecation.

4 Hindu-Muslim Differences in Demand for Latrine Use

Given the observed association between the religious composition of neighborhoods and local sanitation, in this section we draw on several data sources to better understand Hindu-Muslim differences in attitudes and behaviors related to latrine/toilet use. Perhaps surprisingly, we show that open defecation in this context is not merely a matter of latrine affordability or access, but rather involves clear consumer preference heterogeneity.

More than half of the Indian population, over 600 million people, defecates in the open. Muslim Indians, though they are more likely to defecate in the open than the poor rural populations of much of the world, are much less likely to do so than Hindu Indians. In our main dataset, only 32% of Hindu children live in households that use latrines or toilets, compared to 57% of Muslim children. This gross difference cannot be explained by affordability because Hindus are the richer group: Appendix Figure A3 shows that conditional on any level of asset wealth, Hindus are more likely to be exposed to open defecation and to defecate in the open themselves, compared to Muslims.[29] Importantly, although exposure to open defecation declines in wealth, even Hindus and Muslims in relatively wealthy households still practice OD at non-negligible rates and are still exposed to

[29]The figure also shows that this disparity holds conditioning instead on mother's height—thus paralleling Figure 1, which showed that Hindu IMR was higher at any level of asset wealth or mother's height. The associations of sanitation with asset wealth and mother's height strikingly resemble the associations of infant mortality with asset wealth and mother's height. Linear regressions confirm that the gaps are statistically significant ($p < .01$) in both panels. In panels C and D of Appendix Figure A3, we plot own, rather than PSU, OD on the vertical axes. The pattern is nearly identical, highlighting that even relatively wealthy Hindus (and to a lesser extent relatively wealthy Muslims) are still likely to practice OD in this context. The richest Hindus by the asset wealth measure have OD rates in excess of 30%.

significantly high levels of open defecation by their neighbors.

To investigate these patterns in more detail, we turn briefly in Table 4 to data from the Sanitation Quality, Use, Access, & Trends (SQUAT) survey, which was collected 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, as distinct from latrine ownership. The survey is also unique in having a structure that permits reporting of different latrine use patterns by different members of the same household. The survey is described in more detail in Coffey et al. (2014).[30]

Table 4 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. 26% of Hindus whose households own functional latrines choose not to use them, compared to 10% of Muslims. These statistics mask some within-household heterogeneity: Over 40% of households that had a working latrine had at least one member who nevertheless defecated in the open.

These statistics are especially useful in disentangling access to latrines from preferences over latrine *use*. People from households that owned a latrine used by another household member unambiguously had the option not to defecate in the open. 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. We investigate further in Appendix Figure A4, where we compare latrine use behaviors at the individual (rather than household) level and separately by ownership of a working latrine. The figure shows that whether one examines all respondents, or just those residing in latrine-owning households, Hindus are more likely to defecate in the open.

To provide additional context to the fact that high socioeconomic status does not predict zero open defecation in this setting, we draw on another nationally representative survey, the India Hu-

[30]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.

man Development Survey, which has the advantage of being more recent than our main dataset and of allowing additional measures of household socioeconomic status. (See Section 5.3 for a fuller description of these data.) Among rural households in the 2012 IHDS in which the most highly-educated adult member holds a bachelor's degree or higher, 32% of households report practicing open defecation. For rural households in which the most highly-educated adult completed secondary school, the statistic is 51%. This contrasts with neighboring Bangladesh (in its 2011 DHS survey), where only 1% of rural households in which the most highly-educated adult completed *any* schooling beyond secondary defecated in the open, and where only 4% of households with a member who completed secondary school defecated in the open.

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.[31] Regardless of the historical path, today 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 4 shows that a substantial fraction of both Hindu and Muslim respondents self-report that a religious leader has told them explicitly where to defecate. The table shows 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 SQUAT data also allow us to observe plainly-stated beliefs about whether sanitation (latrine use) confers a health benefit to children. Respondents were asked "Imagine two villages, one in which everyone defecates in the open and one in which everyone uses a latrine. In which village do you think children would be healthier?" The final row of Table 4 shows that 40% of Hindus reported that open defecation was healthier, compared to 27% of Muslims. Hindus and Muslims gave statistically identical answers to the other questions in this module, which asked whether children would

[31] *The Laws of Manu*, a Hindu sacred text, advises defecating away from the home (Chapter 4 verse 151): "Far from his dwelling let him remove urine and excreta." In contrast, the Mishkat-al-Masabiha, a Muslim sacred text, offers opposing advice: "Muadh reported God's messenger as saying, 'Guard against the three things which produce cursing: relieving one self in watering-places, in the middle of the road and in the shade.' "

be healthier in villages where people ate rice or wheat and whether children would be healthier in villages where households used kerosene or dung as fuel.[32]

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. (2017) documents this fact in a qualitative study in rural northern India, recording, for example, how a Muslim woman from Uttar Pradesh described the differences: "Even if Hindus have made a latrine, still they go out to defecate in the open. Now for our people [Muslims], it's not a problem. If we have a latrine in the house, we will use it."[33,34]

In short, the pattern of especially poor sanitation in Hindu-concentrated neighborhoods is not merely a matter of affordability of or access to latrines and toilets. Instead, many Indians (Muslim and Hindu) report and reveal clear preferences against using latrines, and this clear preference for open defecation is particularly strong among the Hindu majority.

5 Corroborating Evidence for the Open Defecation Channel

We next present a collage of evidence evaluating sanitation externalities as the possible channel linking the religious composition of neighborhoods to infant mortality. We begin by briefly outlining the biological mechanism, drawing on the prior literature. That discussion then guides several empirical tests with respect to intermediate health outcomes where effects of poor sanitation should be detectable, and which would not easily be explained by other phenomenon. We conclude with a series of falsification tests.

[32] In this module of three questions, the order was: rice/wheat, followed by dung/kerosene, followed by OD/latrine. Respondents could answer that one of the two options within each set was healthier for children in the village, or that the two options had the same impact. Only the sanitation question showed a differential response pattern by religious identity. The F-stats, are, in the same order, 0.5, 2.7, and 9.7. We also note that this question about the health perceptions surrounding sanitation was asked before it was revealed that the survey was specifically focused on sanitation.

[33] Coffey et al. (2017) also reports how a Hindu man in Haryana described his beliefs about the health benefits of open defecation: "[By defecating in the open] one can stretch the body, one can go out for a walk. You can also prevent yourself from getting diseases. If a latrine is in the house, bad smells will come, germs will grow. Latrines in the house are like...hell. The environment becomes completely polluted."

[34] 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 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. And nearly a century ago, Gandhi campaigned to change Indian behavior with respect to excreta disposal, famously declaring, "Sanitation is more important than independence."

5.1 Biological Mechanism

Bacteria and parasites, such as intestinal worms, live in feces. Fecal matter in the local environment gets onto feet and hands and into mouths directly. It can also contaminate food and water. These pathogenic processes have been documented since at least the 19th century.[35] 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). It may also manifest as anemia as the body fails to absorb the nutrients necessary for red blood cell production (Rosenberg and Bowman, 1982). We check this mechanism directly below by examining the intermediate outcomes of blood hemoglobin, 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

[35] See Freedman, 1991 for examples.

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. Taken together, the economic literature supports the potential of important externalities of OD, while the epidemiological evidence suggests that health impacts could be significant and include mortality effects.

5.2 Tests of the Fecal Pathogens Channel

Anemia/Hemoglobin. In Table 5 we examine a health outcome with a direct connection to the type of intestinal disease spread by open defecation. Anemia, defined as low levels of blood hemoglobin, can be caused by diets lacking iron, vitamin B12 and folic acid, all of which are necessary for the production of red blood cells. Intestinal parasites also importantly contribute to low blood hemoglobin in developing country settings, by causing blood loss in the stool, lack of appetite, increased motility (including via diarrhea), and competition for nutrients. Intestinal parasites also cause damage to the intestinal wall lining, which can inhibit the absorption of nutrients critical in red blood cell production (Rosenberg and Bowman, 1982). The impacts of intestinal parasites on hemoglobin can be large even relative to the impacts of diet or micronutrient supplementation. For example, in a randomized control trial among Kenyan children, Friis et al. (2003) found that a single dose of deworming medicine was as effective in raising hemoglobin levels as the daily administration of micronutrient supplements, including iron, for a period of eight months. In the economics literature, Bleakley (2007) shows that a hookworm eradication campaign in the US South at the turn of the twentieth century raised school attendance and later-life incomes, with reduced anemia as the assumed channel.[36]

The dependent variable in the first four columns of Table 5 is the child's blood hemoglobin, in g/dL, measured from a surveyor-collected blood sample analyzed by a portable device in the field. Mean hemoglobin in the sample is 10.3 g/dL, very near to the WHO threshold for moderate anemia ($<$ 10 g/dL). The analysis sample is significantly smaller than in the main analysis because hemoglobin was measured only in the most recent NFHS survey round and because these measurements were taken only for children alive at the time of the interview and between one and five years

[36] In Bleakley (2007), anemia was the assumed channel, though it was not possible in that study to directly observe blood hemoglobin in the historical data. In a cross-country comparison of recent micro data in which hemoglobin was measured in blood samples, Coffey, Geruso and Spears (2017) show that, at the country level, children's hemoglobin and sanitation are correlated, even after controlling for household-level diet data and national malaria incidence.

old. Table 5 shows that Muslim children are observed to have higher hemoglobin on average (column 1), but this relationship disappears after controlling for neighborhood fixed effects (column 2) or the fraction of Muslims in the neighborhood (column 3). In column 4 the association between neighborhood religious composition and blood hemoglobin becomes smaller and insignificant after partialing-out the contribution of poor sanitation. This pattern precisely tracks the pattern of Tables 2 and A3.

It is important to understand that the list of other potential causes of low hemoglobin here is very limited. As discussed in Coffey, Geruso and Spears (2017), the literature points to just diet, malaria, and the type of intestinal infections spread by poor sanitation as being plausible causes of anemia in this context. And we are able to investigate diet directly, which we do below in Section 5.3, where we show that conditional on own religion, own diet is uncorrelated with the religious composition of neighborhoods.

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

We follow the standard practice (e.g. Schmidt et al., 2010) of using surveyor-measured weight to capture acute malnutrition and recent diarrhea. Weight, rather than self-reported diarrheal disease, is recommended due to widely recognized problems in survey-reported diarrhea. We discuss the problems with survey-reported diarrhea in this context in more detail in Appendix A.3, and provide statistics in our own dataset highlighting these issues. Weight-for-age is the particular measure of recent diarrhea recommended by Schmidt et al. (2010). To evaluate robustness, we also examine weight-for-height.[37] We operationalize both measures as z-scores scaled to the World Health Organization's child growth standards.

Columns 5 through 12 in Table 5 repeat the exercise performed for hemoglobin, but now with measures of child weight as the dependent variables. The sample includes all children up to 24

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

months for whom a weight measurement was taken. Due to the DHS/NFHS surveying scheme, these detailed anthropometry measures exist for only a small subset of our main analysis sample. In the first three columns, the dependent variable is weight-for-age. We report OLS regressions.[38] The extended controls are identical to those in the bottom panel of Table 2 with an additional control for height.[39]

The results for weight follow the now-familiar pattern: In columns 5 and 9, an indicator for Muslim households predicts significantly higher z-scores (i.e., heavier children). Then, additionally controlling for Muslim concentration in the locality ($\overline{M_j}$) or controlling for a PSU fixed effect attenuates the coefficient on the Muslim indicator to insignificance. And finally, incrementally controlling for the local sanitation environment attenuates the coefficient on $\overline{M_j}$ as well. This pattern of results supports the mechanism outlined above, as it shows that the fraction of Muslim neighbors is correlated with child malnutrition, but not after partialing out the association between religious composition of the neighborhood or neighborhood open defecation.

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

Table 6 examines various combinations of interactions between religion, breastfeeding, and sanitation in OLS regressions in which the dependent variable is IMR. The regressor of interest is an indicator for exclusive breastfeeding during the infant's first six months of life if she survived, or until death if she died. Controls are as in Table 2. Column 1 reports results for a specification interacting breastfeeding with the Muslim indicator. Unsurprisingly, the main effect of exclusive breastfeeding is large and negative—i.e., breastfeeding is associated with reduced mortality. But the significant positive coefficient estimate for the interaction term *breastfed* $\times$ *Muslim* indicates that breastfeeding is

[38] 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.

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

less beneficial to Muslim children than Hindu children, on average.

This non-intuitive pattern again appears to be due to the correlation between own religious identity and that of neighbors and due to the association of both with sanitation. After additionally controlling for $\overline{M_j}$ or $\overline{OD_{ij}^{-i}}$ and the interaction of each with breastfeeding, the positive coefficient on *breastfed* $\times$ *Muslim* becomes insignificant (column 3) or flips sign (column 6). Overall, comparing columns 1 through 3, in which there are no controls for sanitation, with columns 5 through 7, in which these controls are included, suggests that the associations of the *breastfed* $\times$ *Muslim* and *breastfed* $\times$ *fraction Muslim* with mortality are operating via open defecation: Living near more Hindus predicts a greater protective effect of breastfeeding only until the correlation between religious composition and sanitation is controlled for. These regressions indicate that Muslim children, on average, benefit less from breastfeeding only because they tend to live in better local sanitation environments, while Hindu children, on average, face environments where the protection conferred by breastfeeding matters more.[40]

Appendix Table A6 shows that although the main effect of breastfeeding varies significantly as a function of baseline IMR, the coefficient on the interaction of breastfeeding with PSU fraction Muslim is remarkably similar across PSUs split by high versus low infant morality rates. Thus, these results are not merely driven by a stronger effect of breastfeeding in locales where IMR is higher.

5.3 Falsification Tests

Panels A and B of Figure 4 showed that two child health outcomes that should not be driven by local sanitation are indeed flat (institutional deliveries) or declining (vaccinations) in fraction Muslim, a clear contrast with the overall IMR patterns revealed in Panels C and D of that figure, in which survival increases in fraction Muslim. In this section, we investigate whether the religious composition of neighbors is correlated with other important observable health behaviors, inputs, and outcomes that would not be expected to be importantly affected by open defecation. Although there is no reason in principle that sanitation-related and and non-sanitation-related child inputs and outcomes should not be positively correlated, the special institutional feature of our setting is that higher concentration of Muslims in a locality predicts better sanitation but otherwise predicts worse or neutral

[40] Like the main results in Table 2, estimates in Table 6 are identified off of the religion of neighbors, not the religion of the child's own household. Therefore, any unobserved differences in inputs like weaning foods between Hindu and Muslim households would be captured by controls for *Muslim* and *Muslim* $\times$ *breastfed*.

neighborhood characteristics, allowing us to potentially separate which channel is operating.

Diet. In Table 7 we ask whether Hindus living around many Muslims are likely to have different diets than Hindus living around few Muslims. Because our main dataset, the NFHS, was not designed to measure consumption and contains relatively little information on hygiene behaviors, we turn again to the India Human Development Survey (IHDS) of 2012.[41] Summary statistics for this supplemental dataset are included in Appendix Table A7. For the set of variables common to both the IHDS and NFHS, such as urban residence, open defecation, and access to piped water, Table A7 shows that the IHDS replicates the same Hindu-Muslim differences found in the NFHS, which are displayed in Table 1.

The dependent variables in Table 7 are listed in the column headers and include measures of meat, eggs, and dairy consumption, measured per household per month. For each of these diet variables, we first regress diet on *own* religion to demonstrate that the consumption data are rich enough to capture differences across Muslim and Hindu households. For example, column 1 shows that Muslims are a precisely estimated 15 percentage points more likely (on a base of 25 percentage points) to consume meat compared to Hindus. Such differences are expected, and not problematic.

We add ($\overline{M}_j$) as a regressor in the second and third columns for each dependent variable. Panel A shows that there is no correlation between $\overline{M}_j$ and the extensive margin of eating any meat, or between $\overline{M}_j$ and the intensive margin of quantity of meat consumed. In Panel B, we examine two other calorie- and protein-rich foods: eggs and milk. For all diet variables, the coefficients are small, not significantly different from zero, and robust to the inclusion of controls.[42] This is true even though large, precisely estimated diet differences are apparent between Hindu and Muslim households for each consumption outcome in the table.

Table 7 is direct evidence against the notion that Hindus living around Muslims have systematically different diets than Hindus living around other Hindus, and similarly against the notion that Muslims living around Hindus have systematically different diets than Muslims living around other Muslims. Thus, there is no empirical evidence that our results are likely to be confounded by issues related to diet, including our results regarding hemoglobin in Table 5.

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

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

Other Hygiene and Water. Diet is likely to have primarily private benefits, but hygiene practices like hand washing could generate externalities in principle. If these practices covary with Muslim concentration across localities, then our estimates could be reflecting a different hygiene-related externality associated with the religious composition of a neighborhood. In fact, Hindu and Muslim households do not appear to differ with respect to these health behaviors. For example, the summary statistics from the IHDS data in Table A7 reveal that unlike OD (practiced by 51% of Hindus and 31% of Muslims in the IHDS data), Hindu and Muslim households do not differ in hand washing (72% vs. 70%) or purifying water (11% vs. 12%).

In Table 8, we report on regressions in which the dependent variables are various measures of hygiene and water quality from the IHDS. The regressor of interest is Muslim share, $\overline{M}_j$. In the first eight columns the dependent variables are always washing hands, sometimes washing hands, always purifying water, and sometimes purifying water. In columns 9 and 10, the dependent variable is piped public water to the home, for which a similar measure is available in the NFHS. For completeness, in columns 11 and 12, we replicate the Figure 4 regression of OD on $\overline{M}_j$ using the IHDS data. The last four columns are shaded, since these can be compared directly to results in the NFHS dataset—although in a different time period.

Columns 1 through 8 show that there is no correlation between measures of washing or water purifying and $\overline{M}_j$. The only significant difference we find in the table—besides OD—is that Muslim concentration is associated with *less* access to public piped water. This is consistent with our analysis in the NFHS data presented above (see Figure 4 and Table A1). Both the literature (Jayachandran and Kuziemko, 2010) and our own analysis (Table A2) indicate that lower access to piped water in this region implies a Muslim disadvantage with respect to health. The difference in piped water likely reflects the inferior access to state services faced by Muslims. It appears that the Hindu-Muslim difference in latrine use, therefore, is not merely a marker for a wider array of other important hygiene behaviors with public goods qualities.

Cough and Fever. Although all types of infectious disease may respond to poor sanitation exposure due to a weakened immune system,[43] respiratory infections and fevers may be be less responsive

[43] As Cutler and Miller (2005) explain: "It is difficult to determine which infectious diseases should have responded to clean water technologies, however. Hiram Mills, a member of the Massachusetts State Board of Health, and J. J. Reincke, a health officer in Hamburg, Germany, independently observed a marked reduction in overall mortality, not just mortality from waterborne diseases, on the introduction of water filtration more than a century ago. ... The biological mechanism underpinning the so-called Mills-Reincke phenomenon would presumably be that contaminated water weakens the immune system, making one more susceptible to other contagions."

than symptoms specifically related to the gut. As a falsification test, we examine here whether fever and cough respond to neighborhood religious composition differently than diarrhea, a symptom with a more direct (and presumably quantitatively stronger) link to sanitation.

As discussed above, our primary measure of recent diarrhea in children is based not on the survey reports of mothers, but on current weight. This follows the recommendation from the medical literature (Schmidt et al., 2010) of preferring surveyor-measured weight to capture recent diarrhea even when self-reports of the symptom are available. Our own analysis in Table A8 suggests that a mother's education may significantly affect what she decides "counts" as diarrhea in her child. To overcome differences in the perceptions of illness across mothers of different socioeconomic status, we compare the relative reporting of three different types of symptoms by the same mother.

The NFHS contains mothers' subjective reports of three infectious disease symptoms: fever, cough, and diarrhea. We estimate differences in the relative reporting of these symptoms within a mother across localities that vary in Muslim concentration. In a stacked regression in which an observation is a child $\times$ symptom, we regress an indicator for a positive report of the symptom on mother $\times$ child fixed effects. Appendix Figure A5 plots residuals from that regression—separately by symptom—against the religious composition of the PSU in which the child resides. The figure shows that for the fever and cough symptoms, residuals are clearly increasing in fraction Muslim. Diarrhea residuals, in contrast, are clearly decreasing in fraction Muslim. This means that mothers in neighborhoods with higher Muslim shares are differentially less likely to report their child as having recent diarrhea, relative to their reporting of recent fever and cough and relative to mothers in neighborhoods with fewer Muslims. Appendix Section A.4 describes the exercise in more detail and also reports on a parametric regression approach that yields the same pattern of results.[44]

6 Sizing the Externalities

In this section we attempt to understand the potential size of the sanitation externalities suggested by the evidence above. To do so, we instrument for the local sanitation environment with the religious composition variable, $\overline{M_j}$. This strategy would yield unbiased estimates of the externalities of interest if PSU religious composition predicted own-household infant mortality only through its association

[44]The parametric version of this mother fixed effect regression in Table A12 yields a coefficient on diarrhea $\times$ fraction Muslim that is significant at $p < .09$.

with nearby neighbors' open defecation. In contrast, it is clear from Figure 4 that the neighborhoods where Muslims are concentrated are somewhat different along several other observable dimensions. Even though the evidence above points toward Muslim neighborhoods being, if anything, worse in terms of observable correlates of child health (other than sanitation), we cannot rule out the possibility of important unobservables. We therefore view the IV analysis as informative primarily because these regressions are identified off of a subset of the variation in open defecation that is carefully documented above.

We regress mortality outcomes on variables capturing a household's own open defecation (OD_{ijt}) and neighbors' open defecation within the PSU ($\overline{OD_{ijt}^{-i}}$):

$$Y_{ijt} = \beta_1 \widehat{\overline{OD_{ijt}^{-i}}} + \beta_2 OD_{ijt} + \beta_3 M_{ijt} + f(X_{ijt}) + \epsilon_{ijt}. \tag{2}$$

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

$$\overline{OD_{ijt}^{-i}} = \gamma_1 \overline{M_{jt}} + \gamma_2 OD_{ijt} + \gamma_3 M_{ijt} + f(X_{ijt}) + \mu_{ijt}. \tag{3}$$

As above, i indexes live births, j indexes survey PSUs, and t indexes the three NFHS survey rounds. The dependent variables are infant and neonatal mortality. Note that the indicator for the child residing in a Muslim household, M_{ijt}, absorbs any unobserved health behaviors or child investments that could be correlated with a household's own religion, such as differing Hindu and Muslim diets. We 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 4. That plot visually depicts a non-parametric version of the first stage regression for the case of no controls.[45] Parametric first stage estimates are presented in Appendix Table A9, with first stage F-statistics always exceeding weak instrument thresholds. We also estimate the first stage separately within subsamples defined by child's birth order, child's sex, child's *own* religion,

[45] Besides tracking the linearity of the first stage, restricting to mixed-religion PSUs confers the identification advantage that only respondents who have settled or remained in mixed neighborhoods contribute to estimating our coefficients. This could be important if the types of Hindus and Muslims who are willing to collocate with the other group are systematically different from those who are not. Panel A of Figure 4 suggests that all-Muslim and all-Hindu neighborhoods may indeed differ from mixed-religion neighborhoods, because the nonparametric plots jump at the endpoints ($\overline{M} = 0$ and $\overline{M} = 1$) for many variables.

and the location of the household in a rural or urban setting in order to demonstrate that the first stage relationship is not driven by an association with any of these variables. Table A9 shows that the first stage effect is strong, precisely estimated, and consistent across subsamples.

Table 9 presents the results of the IV analysis. Results for IMR are displayed in Panel A. Results for NMR are in Panel B. Columns 1 and 2 in each panel report OLS estimates for comparison. Columns 3 through 7 report IV estimates with various control sets.[46,47] Table 9 shows that instrumenting for $\overline{OD_{ij}^{-i}}$ with $\overline{M_j}$ yields point estimates of about 50-60 infant deaths per thousand. To put the size in context, the IV coefficient in column 7 implies that a 10 percentage point reduction in the fraction of neighbors defecating in the open is associated with a decline in infant mortality of 6.1 children out of 1000. For neonatal mortality (Panel B), the figure is 4.4 deaths per thousand. The magnitude of the estimated externality is similar across specifications that vary the control set, suggesting that the variation in open defecation that arises from the composition of neighbors is not strongly correlated with any individual-level behaviors or characteristics that affect mortality.[48]

These 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 1). To understand the economic importance of our results, it is useful to consider external harm created by a single household defecating in the open. Because about one in every ten Indian households has an infant born each year, the coefficient on $\overline{OD_{ij}^{-i}}$ is approximately equal to the sum of harm (across all neighbors) imposed by a single household that chooses open defecation, aggregated over 10 years.[49]

The implied importance of open defecation for infant mortality in the modern Indian context is

[46] All regressions include indicators for survey rounds. The extended controls are identical to those in Table 2.

[47] The coefficient on own household OD becomes insignificant and unstable in the IV. This accords with the fact that own latrine use and neighbors' latrine use are highly correlated, as Appendix Figure A1 shows.

[48] 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, and because, as reported in Table 4 from the supplementary SQUAT data, even within a household there is variation in use of latrines that our main data cannot capture. Computations in the spirit of regression calibration indicate that measurement error in local area open defecation could plausibly account for the IV coefficients being approximately twice as large as our OLS coefficients, in large part because own household Muslim, own open defecation, and survey round are correlated with the "signal" in true PSU open defecation.

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

consistent with evidence by Cutler and Miller (2005) and Watson (2006) on the role of clean water and sanitation in explaining large shares of declining infant mortality in the history of the US.[50] Cutler and Miller (2005) show that the introduction of clean water technologies in US cities beginning around 1900 reduced the infant mortality rate by 87 deaths per thousand, or 46 percent of the base of 189 per thousand. To put our findings in perspective, naively applying our IV estimates (Table 9, column 7), would predict that cutting open defecation in half from 66% to 33% would reduce infant mortality by 20 deaths per thousand in India $(= -0.33 \times 61.4)$. This would be a 28 percent reduction in the baseline Indian infant mortality rate of 72 deaths per thousand during our sample period.[51] Our estimates are also consistent with the most credible data on cause of death during early-life in India: Using a census of all Indian deaths occurring from 2001 to 2003, Million Death Study Collaborators (2010) show that 22% of non-neonatal child deaths (deaths from 1-59 months) in India are due to diarrhea, which is just one of the potential pathways by which intestinal worms and bacterial infections that are transmitted via open defecation could affect infant mortality.[52]

Although ours is the first paper to provide even suggestive evidence of mortality externalities associated with open defecation, recent work exploring other human capital impacts of open defecation has found effects with similarly large magnitudes. Gertler et al. (2015) perform a meta-analysis of open defecation-height experiments, and estimate a reduction of 0.046 height-for-age standard deviations from a 10 percentage point reduction in open defecation. To compare the size of these height effects to our mortality estimates, one can scale the estimates using the correlation between IMR and height across Indian villages. In the NFHS, Indian villages with one more IMR point (.0010) have children that are 0.0034 normalized height-for-age standard deviations shorter. Using this conver-

[50] Watson (2006), studying sanitation programs in the United States in the 1960s, where infant mortality was lower and the therefore marginal effects would be expected to be smaller compared to India, finds that "sanitation interventions explain almost forty percent of the convergence in Native American and White infant mortality rates in reservation counties since 1970." Galiani, Gertler and Schargrodsky (2005) studying water privatization in Argentina, found a decline in all-cause child mortality by 8%, with effects reaching as high as 26% in the poorest places, which are likely to be better comparisons to India with respect to poverty. Given that interventions like water privatization could have such large effects on mortality, it is unsurprising to find similarly large effects of open defecation. Among other plausible channels, open defecation is likely to contaminate drinking water.

[51] This calculation sets aside all complications of predictions out-of-sample, such as the potential for reinforcing or countervailing behavioral responses to such a dramatic shift in the local environment. The denominator 72 is the weighted average IMR of Hindus and Muslims from row 1 of Table 1.)

[52] As we discuss in Footnote 43, there is a plausible pathway from poor sanitation to *all* infectious disease, not merely diarrhea. In addition, Million Death Study Collaborators (2010) show that an even larger fraction (33%) of neonatal deaths (deaths from 0-1 months) are attributable to low birthweight. As we discuss in Section 5.1, there is clear epidemiological evidence that exposure to fecal pathogens reduces the body's net absorption of nutrients. In mothers, poorer net nutrition would have direct impacts on child's birthweight. Prendergast et al. (2014) find suggestive evidence on birthweight specifically, showing a correlation between in-utero growth and mothers' exposure to open defecation in Zimbabwe.

sion factor to roughly translate between outcomes, our estimate of a .0061 mortality improvement for a 10 percentage point decline open defecation (Table 9, col 4) would be consistent with a 0.021 height-for-age effect. This effect size translated from our mortality estimates is roughly half of the Gertler et al. (2015) experimental effect sizes, implying our estimates, though economically large, are smaller by this metric.

To 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. Dividing the sample into various partitions pushes the limit of what the data is powered to detect, but in Appendix Table A10, we estimate 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. The point estimate in the Muslim subsample is less than half the size of corresponding point estimate in the Hindu subsample, though not statistically different from the Hindu effect or from zero. Similarly, Table A10 shows that within subsamples of only girls or boys, only first-borns or later-borns, and only urban or rural PSUs, the IV results are never statistically different and are often quantitatively similar.[53]

Finally, we note that individuals who are willing to live in more diverse neighborhoods could be systematically different from those who are not. However, any "cosmopolitan" advantage in health could not even *in principle* spuriously generate our results. This is because the instrument here does not measure the religious diversity of a PSU, it measures the share that is Muslim, which has an asymmetric relationship to religious diversity for Hindus and Muslims. For example, because most Hindus collocate with other Hindus and most Muslims collocate with other Muslims, for the typical Muslim, an increase in $\overline{M}_j$ implies a less religiously diverse neighborhood. In contrast, for the typical Hindu, an increase in $\overline{M}_j$ implies a more religiously diverse neighborhood. This predicted asymmetry stands in contrast to the symmetry of the effects we estimate in Appendix Table A10, where we split the sample by own religion and find positive effects for both groups.[54]

[53] It would be interesting to evaluate whether there was any interaction between own household latrine use and that of neighbors. *A priori*, it is unclear whether an interaction should exist: The disposal of feces in this context is distributed along roads, in fields, and near homes—that is, it is not relegated to areas trafficked only by those who do not use latrines. This implies that the effect of neighbors' open defecation may be invariant to own household latrine use. Our estimation strategy, which focuses on neighbor effects, does not allow us identify the interaction term, as doing so would require a second instrument (for own open defecation, OD_i).

[54] This is contrary to the diversity hypothesis, which would predict effects working in opposite directions for the two subsamples. Such a hypothesis would imply that, counter to our findings, Muslims living in a neighborhood with a 50 percent Muslim share would experience lower infant mortality than Muslims living in a neighborhood with a 100 percent Muslim share.

To show that unobservables associated with willingness to live in a diverse or cosmopolitan neighborhood are not driving our results, in Appendix Table A11 we replicate our main IV analysis from Table 9, but include an additional control for diversity, defined as the fraction of neighbors who are religiously dissimilar from the respondent household. This religious dissimilarity variable moves in opposite directions for Hindu and Muslim households as $\overline{M_j}$ increases. The regressions show that coefficients on religious dissimilarity in IMR and NNM regressions are small and indistinguishable from zero, and that the effects of interest are almost numerically identical to the specifications in Table 9.

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 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 via the tendency of Muslims to reside near other Muslims, but that both Hindus and Muslims appear to gain advantage from Muslim neighbors. Investigating channels, we observe that Indian Muslims, despite being more likely to defecate in the open than Muslims in neighboring Pakistan or Bangladesh, practice open defecation at significantly lower rates than Indian Hindus, implying that the neighbors of Muslims are on average exposed to better sanitation environments in this context. Tests of the mechanisms linking open defecation to infant death, including contamination of food and water and acute malnutrition due to intestinal disease, lend support to this interpretation.

More broadly, our study provides insights into the public goods aspect of sanitation. Understanding this public goods component is important for policy interventions motivated by the efficiency concern that private demand is below the social optimum. The clear stated and revealed preferences towards open defecation reported in Section 4 suggest that the gap between individual perceptions regarding latrine use and the objective external harms of open defecation are signifi-

cant. Practically, our results suggest 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 6 deaths per thousand, or about 8% of the mean mortality rate. With an estimated 26 million children born in India each year, this equates to 156,000 deaths annually. The sheer size of these potential effects highlights the need for further investigation into the externalities of sanitation in the modern developing world.

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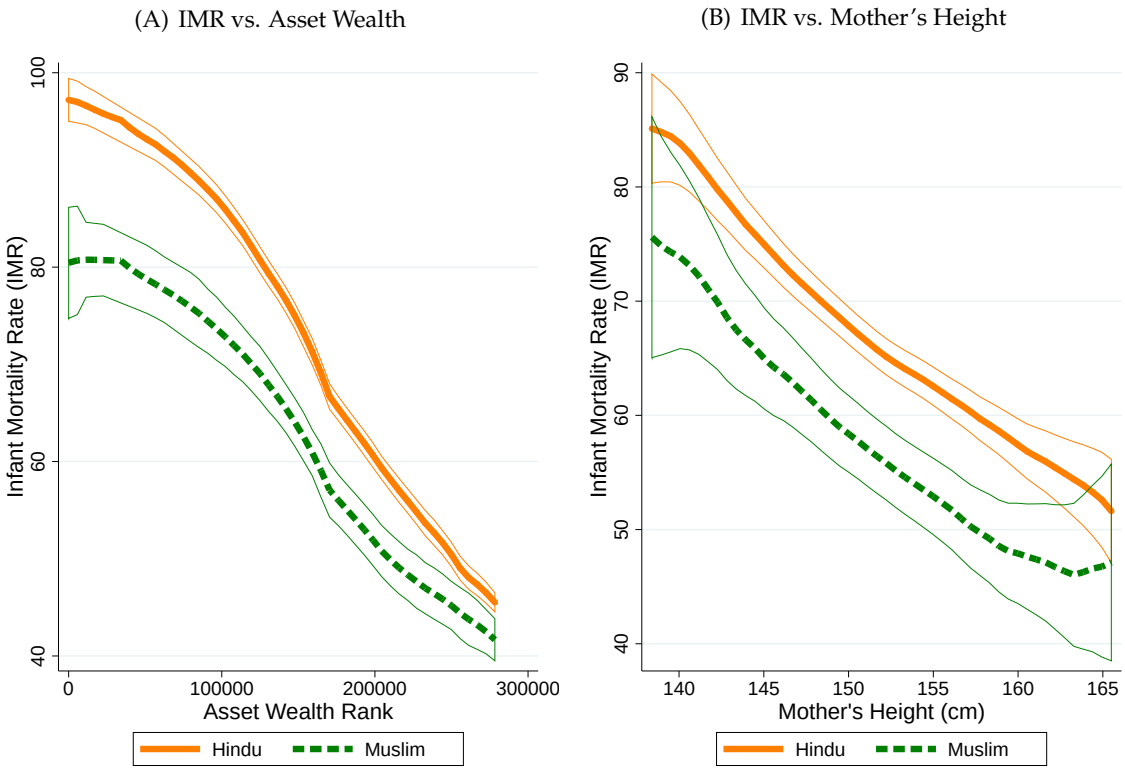
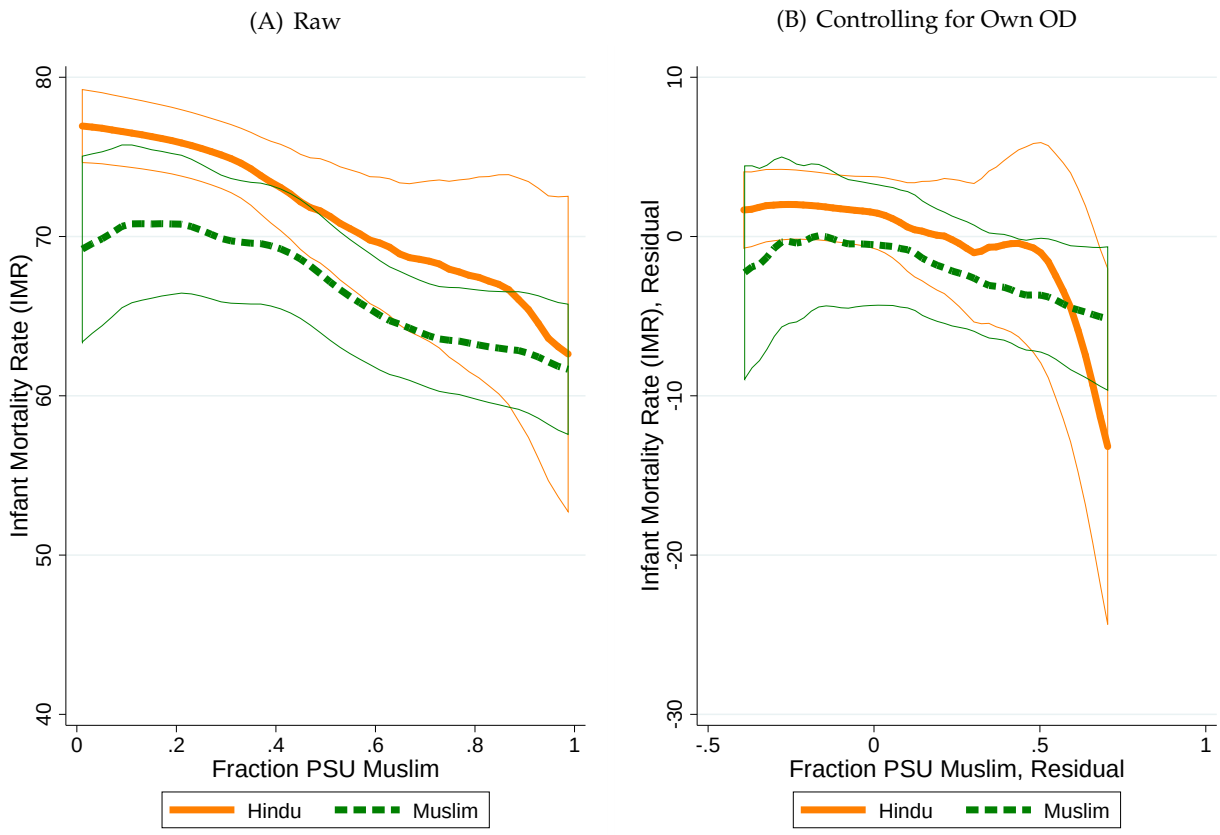
Figure 1: Puzzle: At All Levels of Parental Wealth and Health, Hindu IMR is Higher



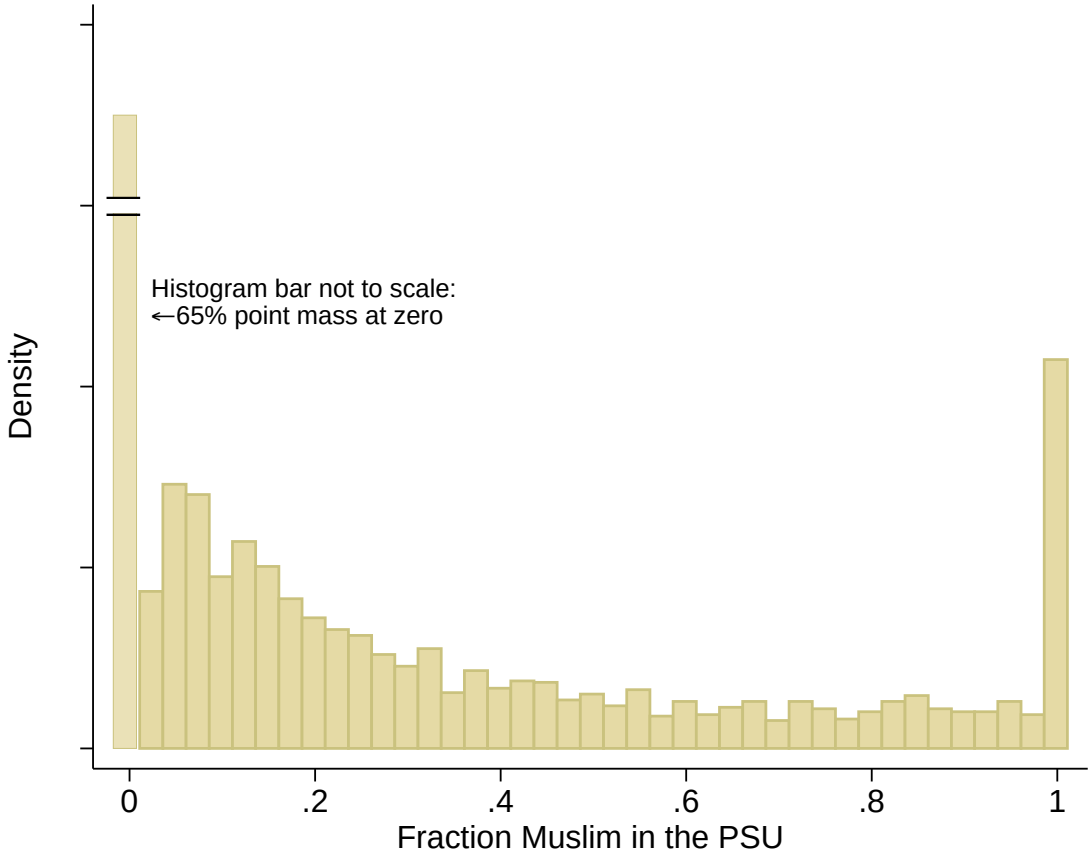
Note: Figure plots local regressions of infant mortality on measures of economic well-being. The dependent variable is an indicator for death in the first year of life $\times$ 1000. The left panel plots mortality against asset wealth rank, constructed as described in the text. The right panel plots mortality against mother's height. Observations are live births. Thinner lines in the figure correspond to 95% confidence intervals that are not adjusted for clustering. Table 2 reports standard errors that account for clustering.

Figure 2: Infant Mortality Decreases with Muslim Neighbors for Both Hindus and Muslims



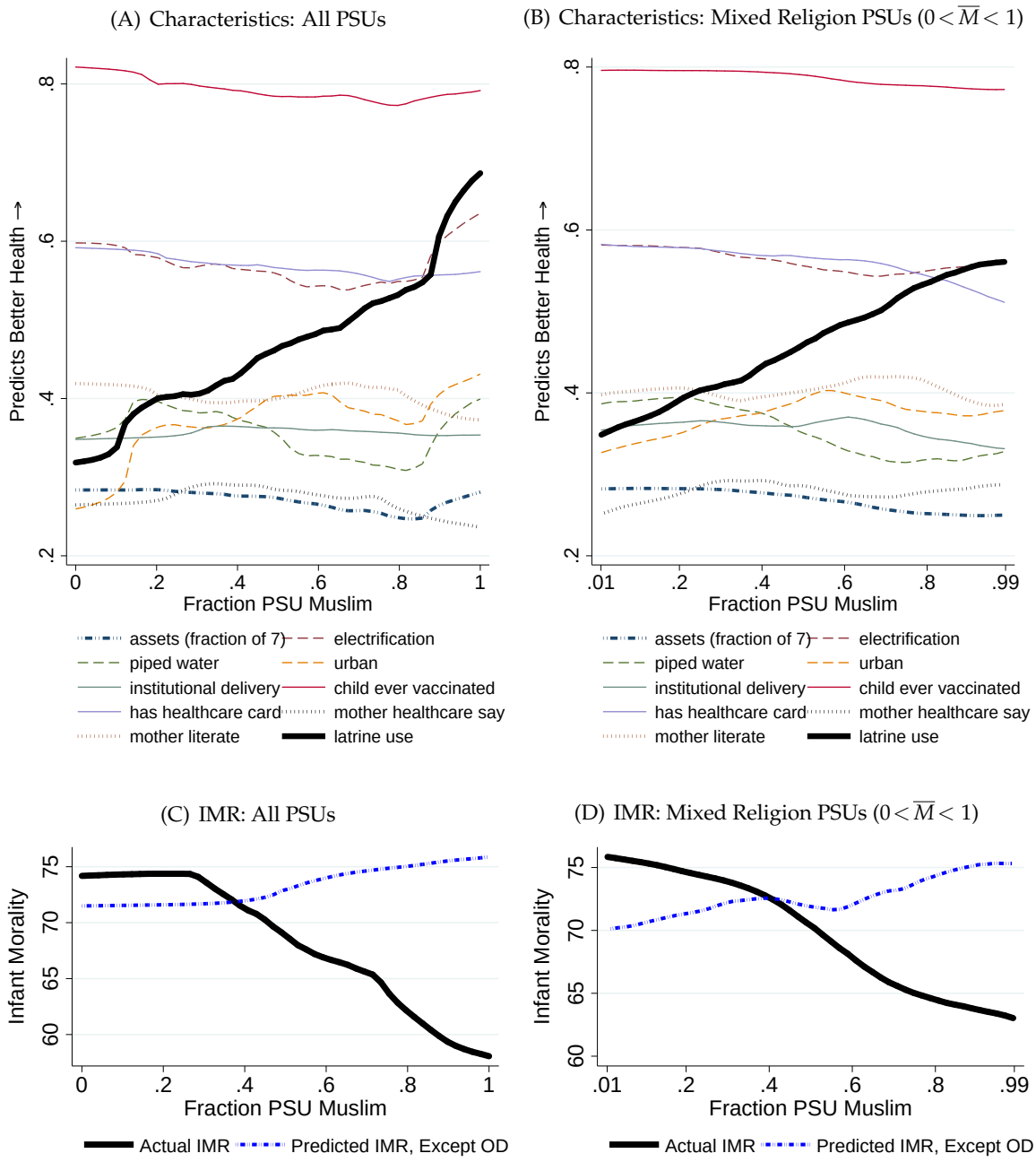
Note: Figure plots local regressions of infant mortality on the fraction of the neighborhood that is Muslim. The dependent variable is an indicator for death in the first year of life $\times$ 1000. Neighborhoods are defined as survey PSUs. Panel A plots the raw relationship, separately for Hindus and Muslims. Panel B plots residuals after conditioning on own open defecation. Observations are live births in mixed-religion PSUs ($0 < \overline{M} < 1$). Thinner lines in the figure correspond to 95% confidence intervals that are not adjusted for clustering. Table 2 reports standard errors that account for clustering.

Figure 3: Muslim Share ($\overline{M}$) in Survey Primary Sampling Units



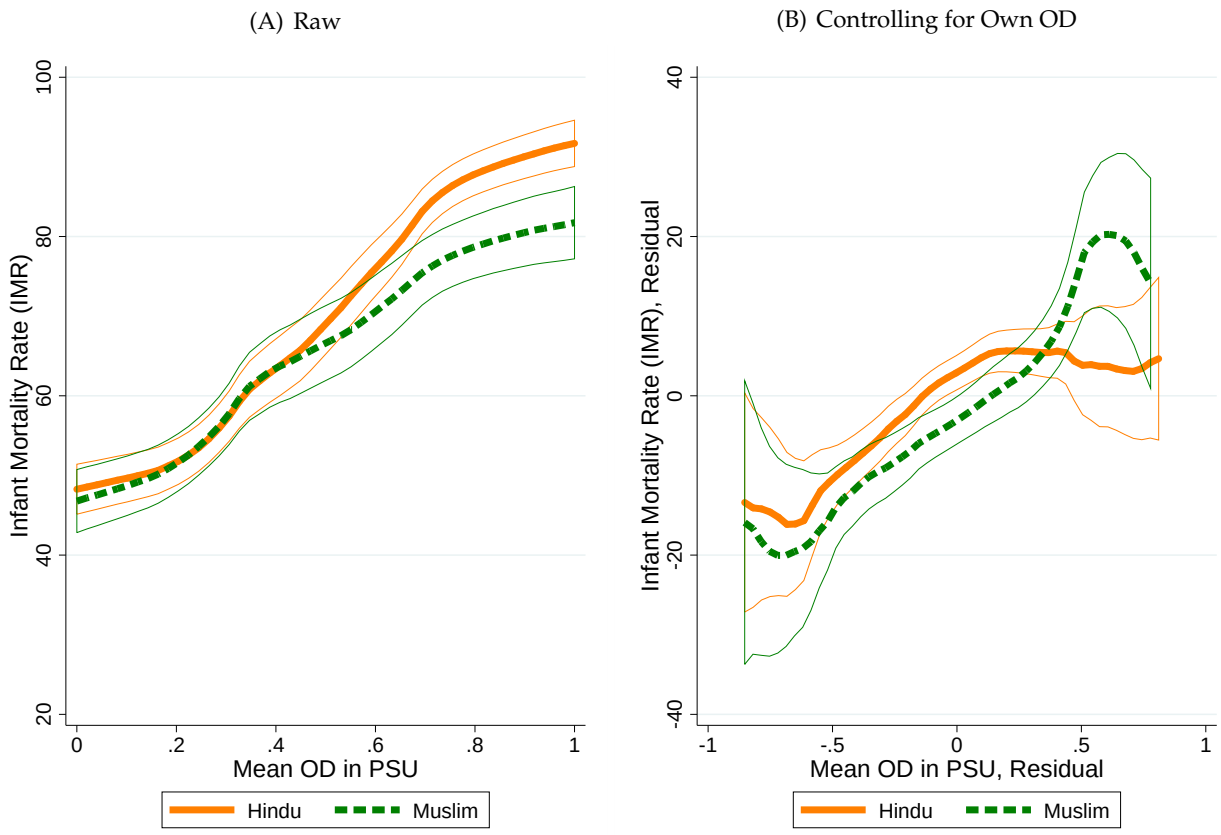
Note: Figure shows the histogram of Muslim concentration across primary sampling units (PSUs) in the NFHS survey. Observations are PSUs. Most PSUs are perfectly segregated along religious lines, with either 0% or 100% Muslim shares. The point mass representing 100% Hindu, which comprises about two thirds of all PSUs, is not drawn to scale.

Figure 4: Greater Muslim Share ($\overline{M}$) Predicts Neutral or Worse PSU Characteristics, Except Latrine Use



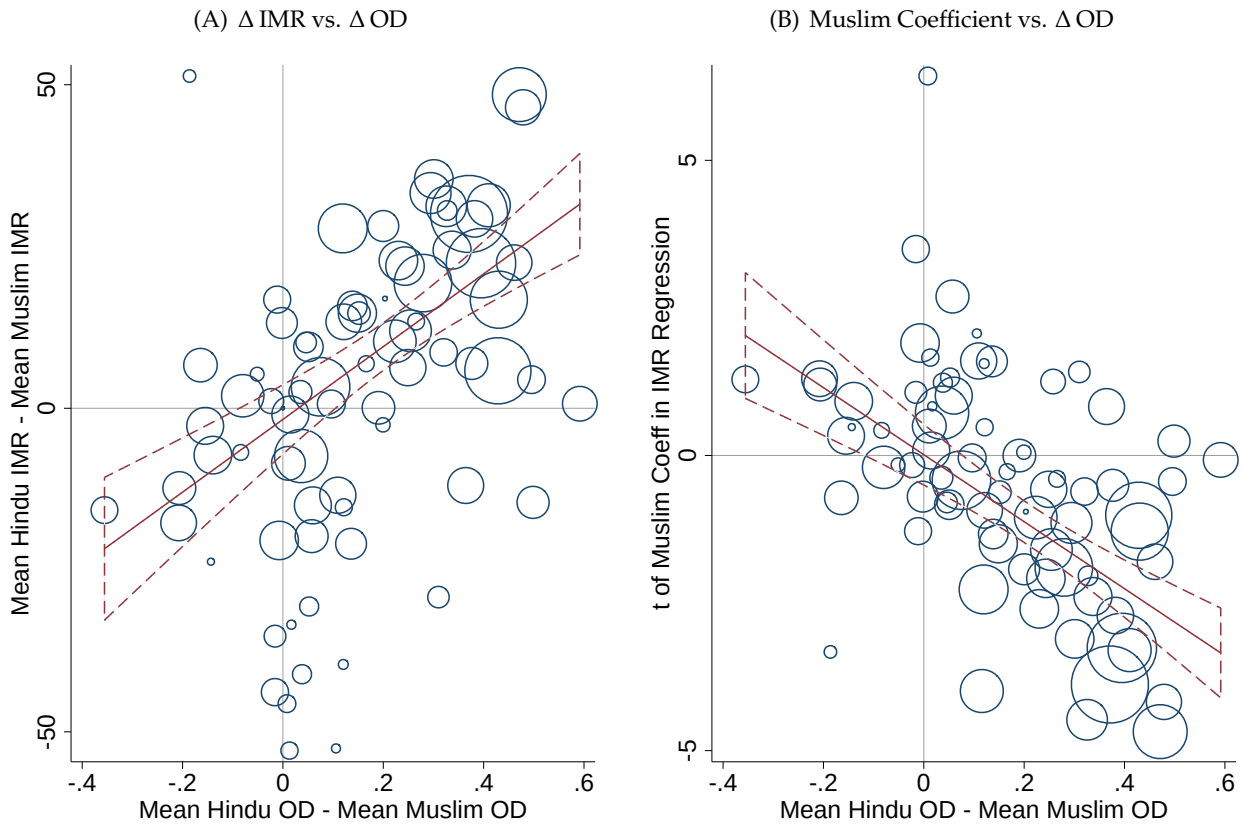
Note: Figure plots local regressions. The dependent variables in Panels A and B are household-level characteristics, and the regressor is Muslim concentration in the PSU ($\overline{M_j}$). Each dependent variable is defined such that higher values predict better infant health outcomes. The signs of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

Figure 5: Neighborhood Sanitation and Infant Mortality



Note: Figure plots local regressions of infant mortality on the fraction of the neighborhood that defecates in the open. The dependent variable is an indicator for death in the first year of life $\times$ 1000. Neighborhoods are defined as survey PSUs. Panel A plots the raw relationship, separately for Hindus and Muslims. Panel B plots residuals after conditioning on own open defecation. Observations are live births in mixed-religion PSUs ($0 < \overline{M} < 1$). Thinner lines in the figure correspond to 95% confidence intervals that are not adjusted for clustering. Table A3 reports standard errors that account for clustering.

Figure 6: Hindu-Muslim IMR Gap Tracks Heterogeneity in First Stage Relationship Across States



Note: Figure shows how heterogeneity in the "first stage" relationship between religious identity and open defecation is reflected in the reduced-form relationship between religious identity and IMR. Scatter points are Indian states × survey rounds, with marker sizes proportional to sample sizes. Panel A plots the Hindu-Muslim mean IMR difference versus the Hindu-Muslim mean difference in OD for each state × round. Panel B plots the t-statistic on an indicator for Muslim in an individual-level regression in which the dependent variable is IMR. That regression follows the specification in Panel A, column 1 of Table 2 but is estimated separately for each state × round cell. OLS regressions fit to the scatterpoints are displayed in red in each panel, along with 95% confidence intervals.

Table 1: Summary Statistics: Hindus and Muslims in the NFHS

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

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

Table 2: Main Result: Neighborhood Composition Predicts Mortality

	Panel A: Survey Round FEs only							
dependent variable:	Infant Mortality (IMR)				Neonatal Mortality (NMR)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Muslim	-9.8**	-4.1	-3.8	-3.8	-7.1**	-2.8	-2.5	-2.5
	(1.5)	(2.4)	(2.3)	(2.3)	(1.2)	(2.0)	(2.0)	(2.0)
PSU fraction Muslim			-9.6**	-11.8**			-7.4**	-9.4**
			(3.0)	(4.2)			(2.5)	(3.3)
PSU FEs		X				X		
indicators for All Muslim & No Muslim				X				X
mean of dep. var.	72.2	72.2	72.2	72.2	46.2	46.2	46.2	46.2
observations (live births)	278,423	278,423	278,423	278,423	278,423	278,423	278,423	278,423
	Panel B: Extended Controls							
dependent variable:	Infant Mortality (IMR)				Neonatal Mortality (NMR)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Muslim	-9.5**	-3.4	-4.2	-4.2	-5.8**	-1.7	-2.1	-2.1
	(1.5)	(2.4)	(2.3)	(2.3)	(1.2)	(2.0)	(1.9)	(1.9)
PSU fraction Muslim			-8.6**	-13.3**			-6.1*	-9.9**
			(2.9)	(3.9)			(2.5)	(3.2)
extended controls	X	X	X	X	X	X	X	X
PSU FEs		X				X		
indicators for All Muslim & No Muslim				X				X
mean of dep. var.	72.2	72.2	72.2	72.2	46.2	46.2	46.2	46.2
observations (live births)	278,423	278,423	278,423	278,423	278,423	278,423	278,423	278,423

Note: Table reports results from OLS regressions. The dependent variable in columns 1 through 4 is infant mortality (year 1). The dependent variable in columns 5 through 8 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). All regressions include survey round fixed effects. Columns 4 and 8 control both for the Muslim share of the PSU and for indicators for all-Muslim and all-Hindu neighborhoods. Extended controls in the bottom panel include a full set of birth order indicators interacted with sex, indicators for household ownership of each of the seven assets asked about throughout NFHS survey rounds, an indicator for piped water, an indicator for whether the mother lives with her husband's parents, indicators for child's birth month and birth year, an indicator for the child being a multiple birth, an urban indicator, household size, mother's education in years, and an indicator for mother's literacy, as well as PSU-level means of household assets, household electricity, household piped water, whether births occurred in an institution, whether mothers had birth assistance, whether children were ever vaccinated, household possession of a health card, and father's education in years. See the text for additional details. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table 3: Heterogeneity in the Muslim Advantage, by Local OD Prevalence

	Panel A: Own Religion			
dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
sample restriction:	PSU OD = 0	PSU OD > 0	PSU OD = 0	PSU OD > 0
	(1)	(2)	(3)	(4)
own household Muslim	1.6	-8.7**	1.5	-6.5**
	(3.9)	(2.2)	(3.3)	(1.8)
χ^2 test for equality of coeffs.	p=0.02		p=0.03	
	Panel B: Neighbors' Religion			
dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
sample restriction:	PSU OD = 0	PSU OD > 0	PSU OD = 0	PSU OD > 0
	(1)	(2)	(3)	(4)
PSU fraction Muslim	1.6	-16.0**	-1.1	-12.0**
	(6.6)	(4.0)	(5.2)	(3.0)
χ^2 test for equality of coeffs.	p=0.02		p=0.07	
	Panel C: Own and Neighbors' Religion			
dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
sample restriction:	PSU OD = 0	PSU OD > 0	PSU OD = 0	PSU OD > 0
	(1)	(2)	(3)	(4)
own household Muslim	1.6	-4.6	3.2	-3.3
	(4.8)	(2.6)	(4.2)	(2.2)
PSU fraction Muslim	0.0	-11.4*	-4.3	-8.7*
	(8.3)	(4.7)	(6.7)	(3.7)
χ^2 test: own household Muslim	p=0.26		p=0.16	
χ^2 test: PSU fraction Muslim	p=0.23		p=0.57	
OD mean	0.00	0.60	0.00	0.60
PSUs	631	2,445	631	2,445
observations (live births)	13,546	90,544	13,546	90,544

Note: Table reports results from OLS regressions run over subsamples of the NFHS data. Columns divide the sample into partitions according to the mean of household-level open defecation in the PSU, either zero or greater than zero. The sample consists of mixed-religion PSUs ($0 < \overline{M} < 1$). Household-level observations of open defecation reported in the NFHS do not account for the discordant latrine use behaviors among household members. The dependent variable in columns 1 and 2 is infant mortality (year 1), and in columns 3 and 4 is neonatal mortality (month 1). Survey round FEs are included in all specifications. See Table 2 for additional notes. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table 4: Stated and Revealed Preferences over Latrine Ownership and Use

	Unit of Observation	Hindu Mean	Muslim Mean
		(1)	(2)
Open defecation, unconditionally	all persons in household	0.73	0.45
		(0.00)	(0.01)
Open defecation, conditional on owning latrine	all persons in household	0.26	0.10
		(0.01)	(0.01)
Owns latrine	household	0.34	0.52
		(0.01)	(0.04)
Says religious leader ever told them where to defecate	respondent	0.16	0.33
		(0.00)	(0.01)
Says open defecation far from home is pure	respondent	0.53	0.40
		(0.01)	(0.04)
Says latrine use near home is pure	respondent	0.45	0.54
		(0.01)	(0.04)
Says open defecation is healthier for village children	respondent	0.40	0.27
		(0.01)	(0.03)

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

Table 5: Tests of Mechanism: Anemia and Current Weight

dependent variable:	Blood Hemoglobin (g/dL)				Weight-for-Age Z-score				Weight-for-Height Z-score			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Muslim	0.151**	-0.015	0.020	0.005	0.128**	0.009	0.056	0.046	0.135**	0.016	0.032	0.021
	(0.034)	(0.049)	(0.047)	(0.046)	(0.028)	(0.059)	(0.042)	(0.042)	(0.032)	(0.062)	(0.046)	(0.045)
PSU fraction Muslim			0.199**	0.090			0.093	-0.009			0.123	0.001
			(0.062)	(0.063)			(0.074)	(0.075)			(0.084)	(0.085)
own household OD				-0.256**				-0.139**				-0.159**
				(0.059)				(0.033)				(0.036)
PSU mean OD (except own)				-0.155**				-0.274**				-0.326**
				(0.034)				(0.055)				(0.063)
extended controls	X	X	X	X	X	X	X	X	X	X	X	X
PSU FEs		X				X				X		
indicators for All Muslim & No Muslim			X	X			X	X			X	X
mean of dep. var.	10.3	10.3	10.3	10.3	-1.89	-1.89	-1.89	-1.89	-0.94	-0.94	-0.94	-0.94
observations (children)	26,330	26,330	26,330	26,330	21,636	21,636	21,636	21,636	21,164	21,164	21,164	21,164

Note: Table reports results from OLS regressions. The dependent variables are indicated at the column header and are either blood hemoglobin in g/dL, or weight-for-age or weight-for-height scaled as WHO z-scores. Hemoglobin levels are affected by intestinal parasites. Current weight is an outcome reflecting acute malnutrition, and is recommended in the medical literature as the preferred measure for recent bouts of diarrhea. PSU mean OD is calculated over all households in the PSU other than the respondent household. Controls are as described in the Table 2 notes. Observations are children (live births). Due to the DHS/NFHS surveying scheme, the hemoglobin and anthropometry measurements exist for only a small subset of our main analysis sample. The sample in columns 1 through 4 includes all observations for which hemoglobin was collected in the NFHS data. The sample in columns 5 through 12 includes all children up to 24 months for whom a weight measurement was taken. Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table 6: Test of Mechanism: Interaction Between Breastfeeding Efficacy and $\overline{OD}$

dependent variable:	Infant Mortality (IMR)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Muslim	-38.4**		-23.6		9.9		-28.3
	(14.0)		(22.6)		(13.7)		(22.0)
breastfed X Muslim	31.5*		24.3		-20.8		29.0
	(14.1)		(22.7)		(13.8)		(22.1)
PSU fraction Muslim		-48.0**	-24.4			32.2	60.6*
		(18.3)	(29.1)			(18.1)	(28.6)
PSU fraction Muslim X breastfed		35.5	11.2			-51.2**	-80.3**
		(18.1)	(29.1)			(17.8)	(28.5)
PSU OD (except own)				233.8**	233.6**	236.6**	236.8**
				(13.7)	(14.0)	(14.1)	(14.1)
PSU OD (except own) X breastfed				-246.6**	-250.5**	-256.3**	-256.5**
				(13.5)	(13.7)	(13.7)	(13.7)
breastfed	-245.5**	-246.2**	-246.2**	-110.7**	-105.1**	-96.8**	-96.7**
	(6.4)	(6.7)	(6.7)	(7.7)	(8.5)	(8.8)	(8.8)
extended controls	X	X	X	X	X	X	X
indicators for All Muslim & No Muslim		X	X			X	X
observations (live births)	83,702	83,702	83,702	83,702	83,702	83,702	83,702

Note: Table reports results from OLS regressions. The dependent variable is IMR, scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). The breastfed indicator is equal to one if the infant was exclusively breastfed during the first six months of life if she survived, or until death if she died. PSU mean OD is calculated over all households in the PSU other than the respondent household. Controls are as described in the Table 2 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table 7: Falsification Tests: $\overline{M}_j$ Does Not Predict Diet Differences Across PSUs

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

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

Table 8: Falsification Tests: $\overline{M}_j$ Does Not Predict Other Hygiene Behaviors Across PSUs

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

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

Table 9: IV Estimates Exploiting PSU-level Variation in the Composition of Neighbors

dependent variable:	Panel A: Infant Mortality (IMR)						
sample restriction:	Mixed Religion PSUs						
specification:	OLS	OLS	IV	IV	IV	IV	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
PSU mean OD (except own)	44.0**	23.9**	68.6**	52.1**	45.8	51.0**	61.4**
	(2.6)	(3.6)	(15.1)	(17.6)	(31.6)	(16.1)	(21.5)
own household Muslim		-3.5		-3.9	-3.3	-3.6	-4.8*
		(2.0)		(2.3)	(2.0)	(2.4)	(2.1)
own household OD		20.2**			7.9		-14.1
		(2.6)			(17.9)		(7.9)
extended controls						X	X
mean of dep. var.	72.0	72.0	72.0	72.0	72.0	72.0	72.0
first stage F-stat			74.4	74.8	97.0	200.3	230.7
observations (live births)	104,090	104,090	104,090	104,090	104,090	104,090	104,090

dependent variable:	Panel B: Neonatal Mortality (NMR)						
sample restriction:	Mixed Religion PSUs						
specification:	OLS	OLS	IV	IV	IV	IV	IV
	(8)	(9)	(10)	(11)	(12)	(13)	(14)
PSU mean OD (except own)	29.4**	16.1**	52.1**	41.3**	42.5	38.4**	44.1*
	(2.0)	(2.9)	(11.7)	(14.1)	(25.5)	(13.0)	(17.4)
own household Muslim		-2.9		-2.5	-2.6	-1.4	-2.1
		(1.6)		(1.9)	(1.6)	(2.0)	(1.8)
own household OD		13.5**			-1.4		-7.8
		(2.2)			(14.4)		(6.4)
extended controls						X	X
mean of dep. var.	46.6	46.6	46.6	46.6	46.6	46.6	46.6
first stage F-stat			74.4	74.8	97.0	200.3	230.7
observations (live births)	104,090	104,090	104,090	104,090	104,090	104,090	104,090

Note: Table reports results from OLS and IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$). IV estimates instrument $\overline{OD_{ij}^{-i}}$ with $\overline{M_j}$. Mortality variables are scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). The sample consists of mixed-religion PSUs ($0 < \overline{M} < 1$). Extended controls are as described in the Table 2 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

APPENDIX

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

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

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

With the NFHS data, we can alternatively perform a more detailed calculation for the required sample size and cluster count via Monte Carlo simulation. Unlike the standard power calculation, this method naturally incorporates any heterogeneity in infant mortality that is present across clusters. For the simulation, we again assume that the true effect of a 10 percentage point reduction in local OD is equal to 5 percent of mean infant mortality, or 3.5 deaths per thousand. To implement the Monte Carlo simulation, we iterate over the following procedure, varying the number of sample clusters (N_c) included. We use PSUs from the NFHS data described in Section 2 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 A6 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.[55] 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 2, 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.[56] For each reweight on some combination these of characteristics, we follow three steps:

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

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

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

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

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

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

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

A.3 Problems with Survey-Reported Diarrhea

The NFHS contains information on mothers' reports of diarrhea in their children. This type of survey measure is likely to contain significant biases that may be correlated with our regressors of interest. For example, because the reporting of diarrhea depends on whether the reporting mother recognizes a loose stool as diarrhea, differences in reporting across children is correlated with the education level of their mothers. Appendix Table A8 illustrates this fact, regressing reported diarrhea on mother's education, where the omitted category is no education. The table also includes regressions where weight-for-age is the dependent variable. The table shows that reported diarrhea is only weakly correlated with education, even though children of higher educated mothers tend to show fewer measurable symptoms of the problem: Point estimates indicate that mothers with some education are weakly *more* likely to report diarrhea than those with no education (columns 1 and 2). This is despite the fact that weight moves in the predicted pattern, increasing with education. Columns 3 and 4 show that the weight of children is strongly correlated with mother's education.

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

A.4 Cough, Fever, and Diarrhea

In Section 5.3 we describe an exercise that examines whether fever and cough respond to neighborhood religious composition differently than diarrhea. We attempt to address differences across mothers of different socioeconomic status in the subjective reporting of symptoms by comparing relative reporting of the three types of symptoms conditional on a mother $\times$ child fixed effect. We reshape the data to "stack" three observations per child, one for each symptom: fever, cough, and diarrhea. The dependent variable is an indicator for whether the mother reported that the child was recently ill with that symptom. We regress an indicator for a positive report of the symptom on mother $\times$ child fixed effects.

$$\begin{aligned}\text{Ill recently}_{ijt} = \alpha + \theta_{ijt} + \gamma_1 \text{cough}_{ijt} + \gamma_2 \text{diarrhea}_{ijt} \\ + \psi_1 \text{cough}_{ijt} \times \overline{M_{jt}} + \psi_2 \text{diarrhea}_{ijt} \times \overline{M_{jt}} + \epsilon_{ijt}. \end{aligned} \tag{4}$$

where α_{ijt} are mother $\times$ child FEs. Note that the main effect of $\overline{M_{jt}}$ is absorbed by α_{ijt}. The variables fever_{ijt} and $\text{fever}_{ijt} \times \overline{M_{jt}}$ are the excluded categories. In this regression, cough_{ijt} and diarrhea_{ijt} take on values of one if the observation corresponds to that symptom type, regardless of whether the mother reported the symptom as present ($\text{Ill recently}_{ijt} = 1$) or absent ($\text{Ill recently}_{ijt} = 0$). Table A12 reports the coefficients, which show that relative to fever, diarrhea is more strongly negatively associated with fraction Muslim. Cough, in contrast, is not.

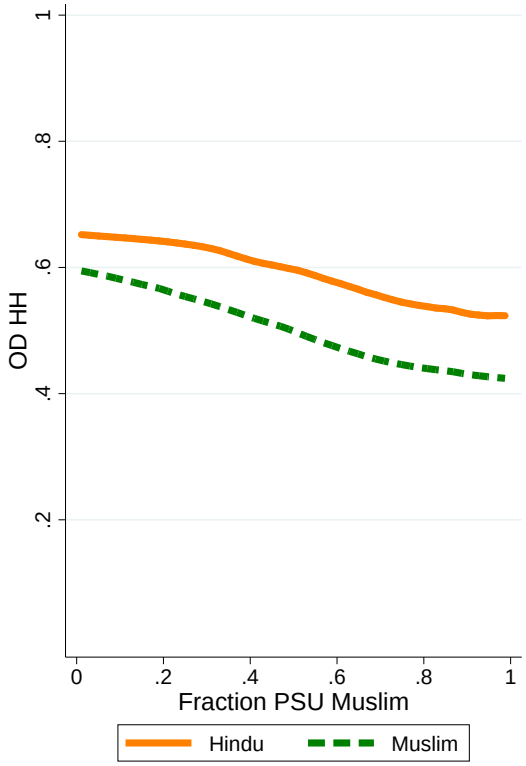
Appendix Figure A5 reports on a non-parametric version of this exercise, plotting residuals from a regression of symptom indicators on mother $\times$ child fixed effects, separately by symptom, against

the religious composition of the PSU. The equation generating the residuals is:

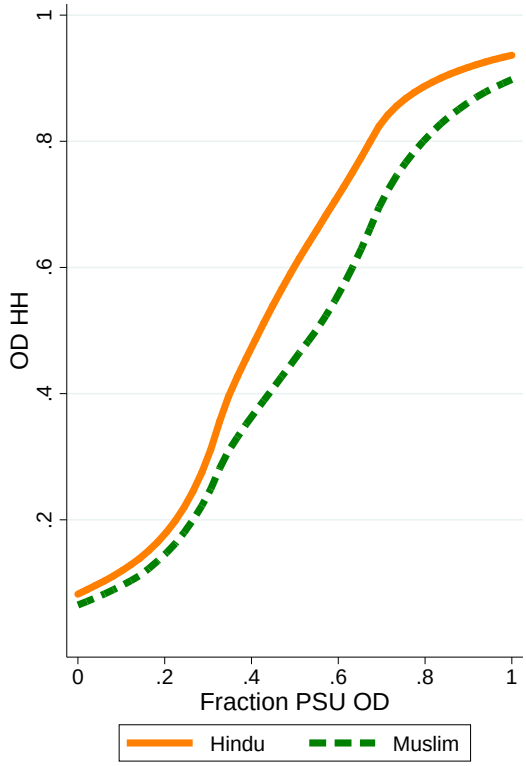
$$\text{Ill recently}_{ijt} = \alpha_{ijt} + \gamma_1 \text{fever}_{ijt} + \gamma_2 \text{cough}_{ijt} + \gamma_3 \text{diarrhea}_{ijt} + \epsilon_{ijt}. \quad (5)$$

Figure A1: Correlation between Own and Neighbors' Open Defecation

(A) Household OD vs $\overline{M_j}$

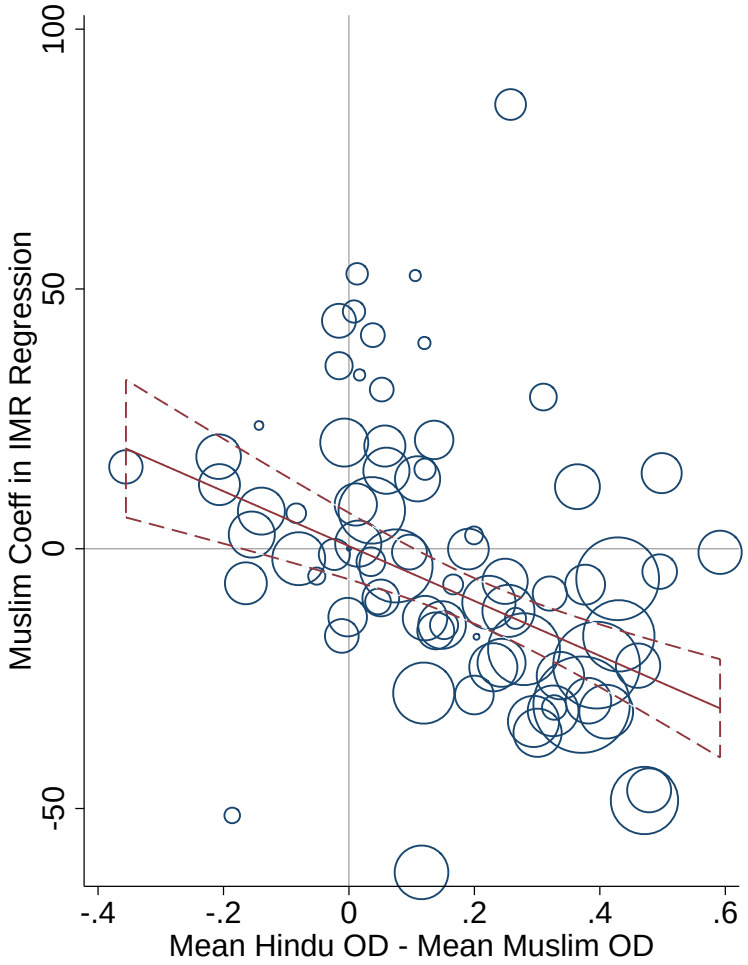


(B) Household OD vs Neighbors' OD ($\overline{OD_{ij}^{-i}}$)



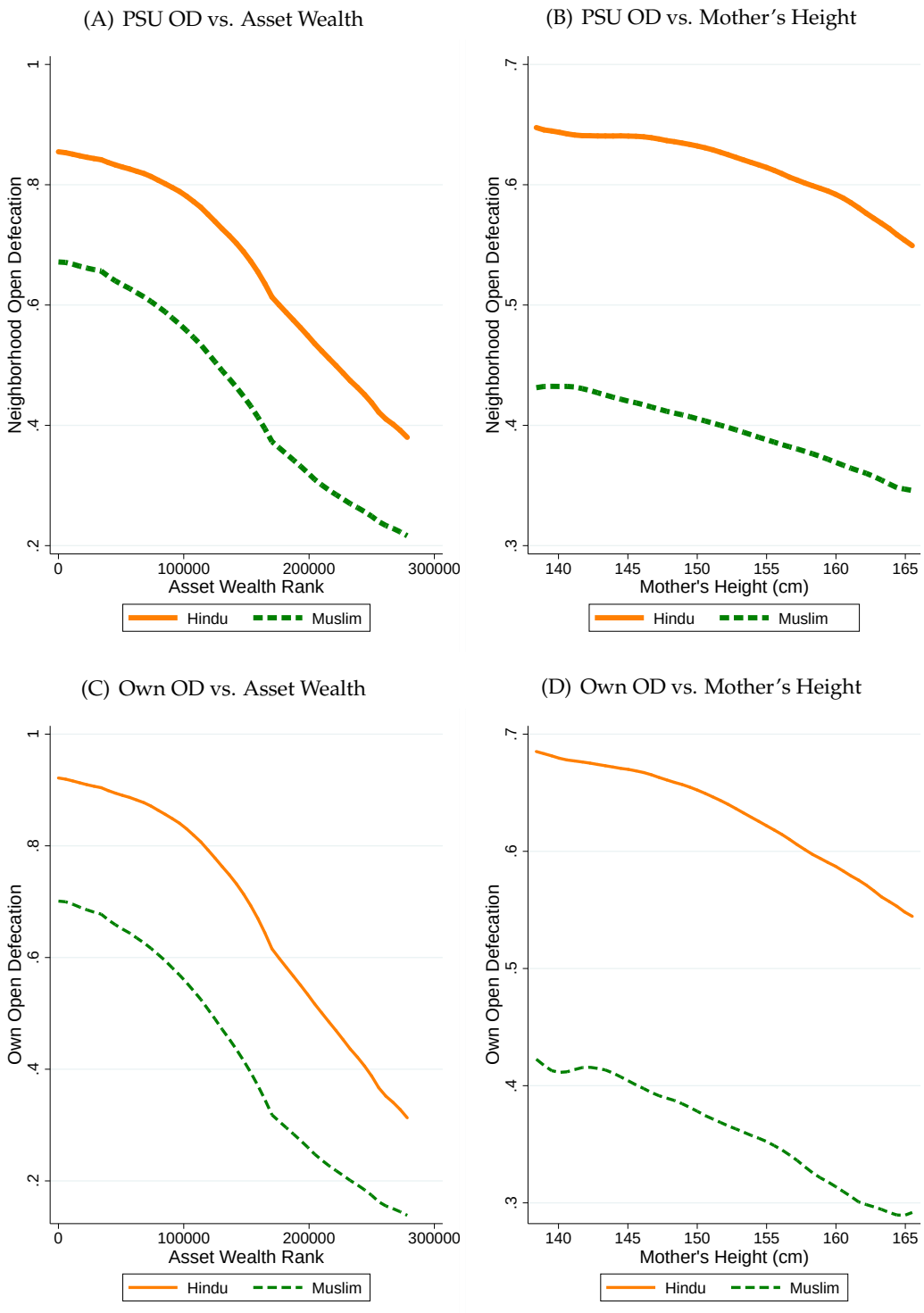
Note: Figure plots local regressions of household OD on the Muslim share of the neighborhood (Panel A) and the fraction of the PSU that defecates in the open (Panel B). Neighborhoods are defined as survey PSUs. Observations are live births.

Figure A2: Hindu-Muslim IMR Gap Tracks Heterogeneity in First Stage Relationship Across States



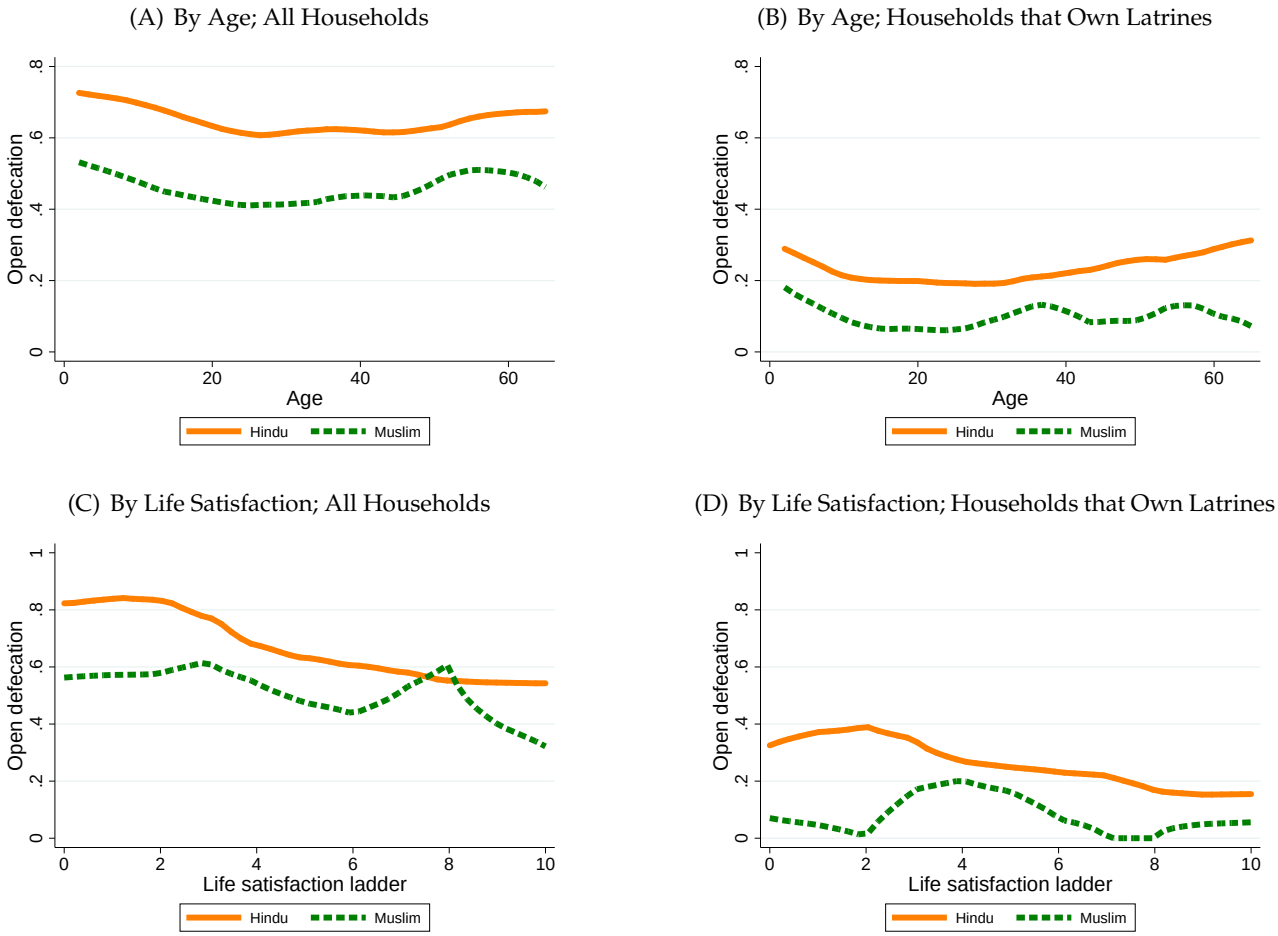
Note: Figure shows how heterogeneity in the first stage relationship between religious identity and open defecation is reflected in the reduced-form second stage relationship between religious identity and IMR. Scatter points are Indian states × survey rounds, with marker size proportional to sample size. The vertical axis measures the Muslim coefficient in an individual-level regression in which the dependent variable is IMR, estimated separately in each state × round. A linear regression is displayed in red, along with 95% confidence intervals. See Figure 6.

Figure A3: At All Levels of Parental Wealth and Health, Hindu OD Exposure is Higher



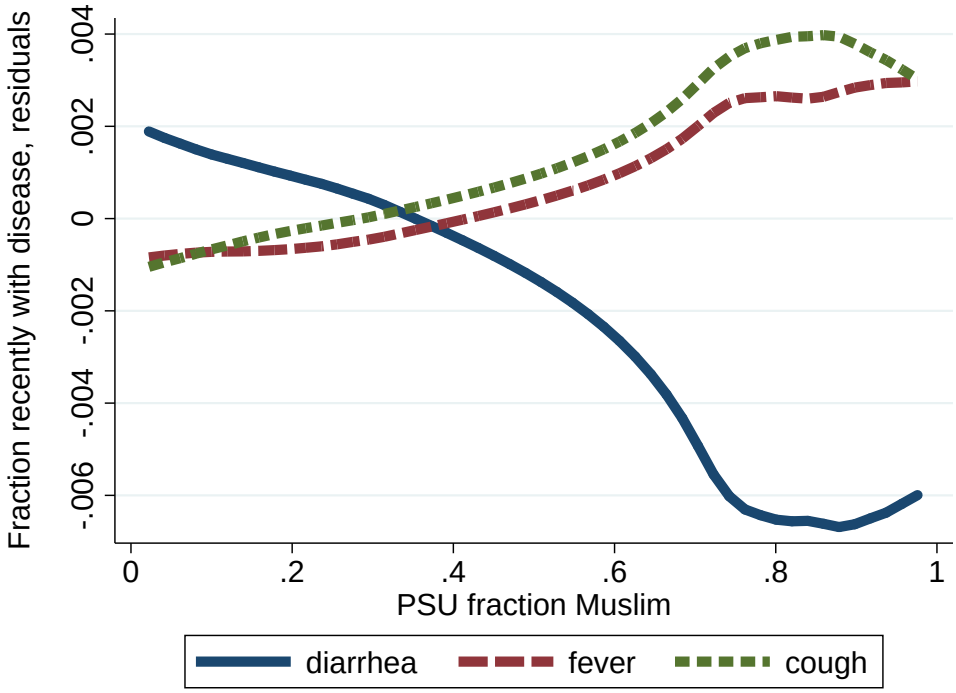
Note: Figure plots local regressions of neighborhood OD (Panels A and B) and household OD (Panels C and D) on the economic wellbeing of the household, proxied by asset wealth and mother's height. Neighborhoods are defined as survey PSUs. Observations are live births.

Figure A4: Hindu-Muslim Differences in OD: Unconditional and Conditional on Owning a Latrine



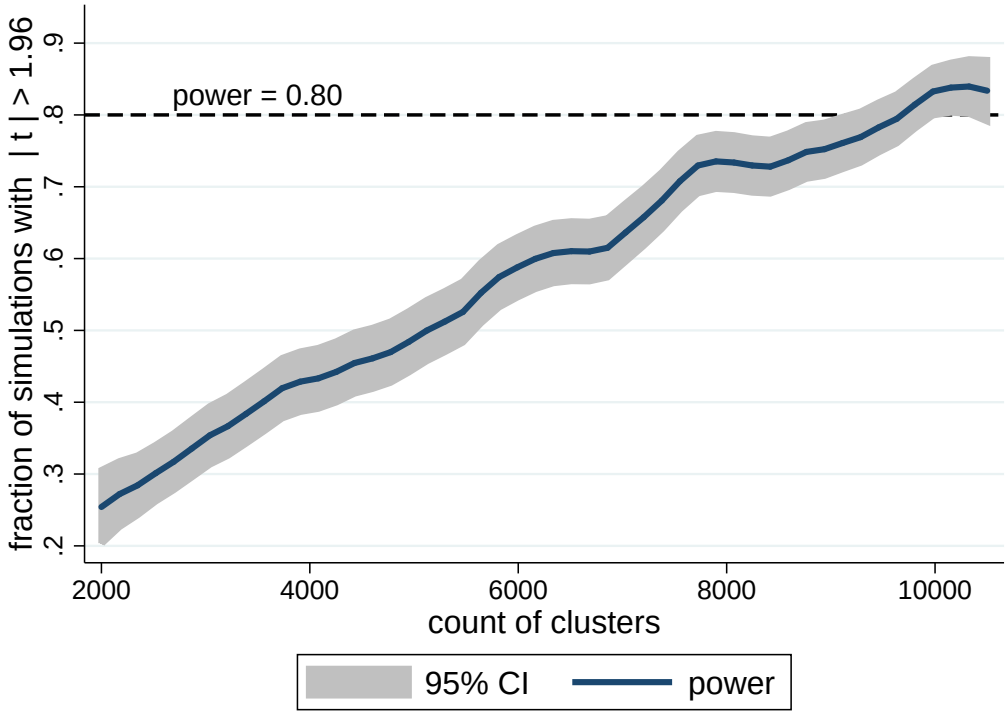
Note: Figure plots local linear regressions in which the dependent variable is person-specific open defecation. The sample in the left panels includes all household members for whom a response was recorded. The sample in the right panels includes only respondents in households that own a latrine, illustrating the preference for open defecation even when latrines are clearly available. The rows, respectively, condition on age and a life satisfaction "ladder" question (0 to 10, 10 being the most satisfied). The dataset used to construct these plots is the SQUAT survey. Observations are individuals. See Table 4 for additional data notes.

Figure A5: Symptoms by Fraction Muslim: Residuals from Mother × Child FE Regressions



Note: Figure plots local regressions of symptom residuals on $\overline{M_j}$. Residuals are generated from a stacked regression in which an observation is a child × symptom, for each of three symptoms: fever, cough, and diarrhea. An indicator for a positive report of the symptom is regressed on mother × child fixed effects and on indicators for each of the three symptom types. Residuals are plotted separately by symptom. See Appendix A.4 for additional details.

Figure A6: Experimental Sample Size Needed to Identify the Mortality Externalities of OD



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

Table A1: Correlates of Muslim Share Across PSUs

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

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

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

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

Note: Table reports results from 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 ($0 < \overline{M} < 1$). Observations are children (live births), and sample size varies across regressions because some survey questions were asked to only subsets of respondents. Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

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

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

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

Table A3: Horserace Regressions: $\overline{OD_j}$, $\overline{M_j}$, and Mortality

dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
	(1)	(2)	(3)	(4)
Muslim	-2.5	-3.6	-1.7	-1.6
	(2.1)	(2.3)	(1.7)	(1.9)
PSU fraction Muslim	4.6	-4.7	1.5	-2.8
	(2.7)	(3.0)	(2.2)	(2.5)
PSU mean OD (except own)	25.6**	9.4**	16.8**	8.0**
	(2.1)	(3.0)	(1.7)	(2.4)
own household OD	20.2**	5.9**	12.3**	4.9**
	(1.6)	(1.8)	(1.3)	(1.4)
extended controls		X		X
mean of dep. var.	72.2	72.2	46.2	46.2
observations (live births)	278,423	278,423	278,423	278,423

Note: Table reports results from OLS regressions. PSU mean OD is calculated over all households in the PSU other than the respondent household. Controls are as described in the Table 2 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

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

	Panel A: split by child sex					
dependent variable:	IMR					
sample restriction:	Full Sample		Boys		Girls	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-9.0**	-1.3	-10.6**	-3.8
	(1.5)	(2.3)	(2.0)	(3.2)	(2.0)	(3.1)
own household OD		20.2**		20.4**		20.1**
		(1.7)		(2.3)		(2.4)
PSU mean OD (except own)		25.8**		22.0**		29.9**
		(2.3)		(3.0)		(3.1)
indicators for All Muslim & No Muslim		X		X		X
mean of dep. var.	72.2	72.2	73.7	73.7	70.6	70.6
observations (live births)	278,423	278,423	144,269	144,269	134,154	134,154

	Panel B: split by child birth order					
dependent variable:	IMR					
sample restriction:	Full Sample		First Birth		Second or Higher Birth	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-6.7*	-1.4	-10.5**	-2.6
	(1.5)	(2.3)	(2.7)	(4.2)	(1.7)	(2.7)
own household OD		20.2**		22.7**		19.8**
		(1.7)		(3.0)		(2.0)
PSU mean OD (except own)		25.8**		38.4**		21.1**
		(2.3)		(3.8)		(2.7)
indicators for All Muslim & No Muslim		X		X		X
mean of dep. var.	72.2	72.2	74.4	74.4	71.4	71.4
observations (live births)	278,423	278,423	76,253	76,253	202,170	202,170

	Panel C: split by PSU location					
dependent variable:	IMR					
sample restriction:	Full Sample		Urban		Rural	
	(1)	(2)	(3)	(4)	(5)	(6)
Muslim	-9.8**	-2.5	-3.7	-4.6	-8.8**	-1.3
	(1.5)	(2.3)	(2.1)	(3.4)	(2.0)	(3.1)
own household OD		20.2**		22.4**		19.3**
		(1.7)		(3.1)		(2.1)
PSU mean OD (except own)		25.8**		19.3**		26.4**
		(2.3)		(4.8)		(3.4)
indicators for All Muslim & No Muslim		X		X		X
mean of dep. var.	72.2	72.2	52.0	52.0	80.9	80.9
observations (live births)	278,423	278,423	83,344	83,344	195,079	195,079

Note: Table reports results from 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 2 for reference. Columns 3 through 6 replicate the regressions in the subsamples defined in the column headers. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

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

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

Reweighting variables					Reweight Results				
Round	Urban	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 (=$20\times3\times2\times8\times3\times4$) cells.

Table A6: Breastfeeding $\times$ $\overline{M_j}$ Interactions in High and Low IMR Neighborhoods

dependent variable:	Infant Mortality (IMR)	
sample restriction:	PSU IMR < Median	PSU IMR ≥ Median
	(1)	(2)
Muslim	23.4	-43.4
	(21.1)	(36.0)
breastfed X Muslim	-25.9	47.3
	(21.1)	(36.4)
PSU fraction Muslim	44.1	32.2
	(27.1)	(48.4)
PSU fraction Muslim X breastfed	-45.4	-59.1
	(27.0)	(48.5)
PSU OD (except own)	142.2**	137.4**
	(14.7)	(23.5)
PSU OD (except own) X breastfed	-148.8**	-172.6**
	(14.7)	(23.5)
breastfed	-28.5**	-254.0**
	(6.8)	(18.8)
extended controls	X	X
indicators for All Muslim & No Muslim	X	X
PSUs	7,288	7,288
observations (live births)	42,775	40,927

Note: Table reports results from OLS regressions. The dependent variable is IMR, scaled as described in the text to generate coefficients that indicate impacts on rates $\times$ 1000 (deaths per 1000 children). The breastfed indicator is equal to one if the infant was exclusively breastfed during the first six months of life if she survived, or until death if she died. PSU mean OD is calculated over all households in the PSU other than the respondent household. The sample is split across the columns according to PSU-level mean IMR above or below the median. Controls are as described in the Table 2 notes. All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table A7: Summary Statistics from Supplementary Dataset: The India Human Development Survey

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

Note: Table displays summary statistics for the supplemental dataset used in Section 5.3, the 2012 round of the India Human Development Survey (IHDS). Observations are households.

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

Table A8: Reliability of Self-Reported Diarrhea vs. Objective Measures of Acute Malnutrition

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

Note: Table reports results from 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 2 notes. All regressions control for survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table A9: Robustness of First Stage Result: Splits by Subsamples

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

Note: Table reports results from 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 2 notes. All regressions include only the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). All regressions include survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table A10: Robustness of IV Results in Table 9: Splits by Subsamples

dependent variable:	Infant Mortality (IMR)		
	(1)	(2)	(3)
	Panel A: split by child sex		
	Full Sample	Boys	Girls
PSU mean OD (except own)	61.4**	55.2*	68.4*
	(21.5)	(28.0)	(29.0)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	53,779	50,311
	Panel B: split by child birth order		
	Full Sample	First Birth	Second or Higher Birth
PSU mean OD (except own)	61.4**	66.4	59.1*
	(21.5)	(40.7)	(24.0)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	27,020	77,070
	Panel C: split by own religion		
	Full Sample	Muslim	Hindu
PSU mean OD (except own)	61.4**	33.5	86.9**
	(21.5)	(26.9)	(33.4)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	34,052	70,038
	Panel D: split by PSU location		
	Full Sample	Urban	Rural
PSU mean OD (except own)	61.4**	66.4	66.1**
	(21.5)	(49.0)	(25.4)
own religion and own OD	X	X	X
extended controls	X	X	X
observations (live births)	104,090	37,209	66,881

Note: Table reports results from 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 7 of Table 9 for comparison. Columns 2 and 3 estimate the identical IV regression over each of the subsamples defined in the panel headers. All regressions include only the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). All regressions control for survey round fixed effects, own religion, own OD and the extended controls as described in the Table 2 notes, except for the single variable on which the sample is split in each panel (sex, birth order, etc.). Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table A11: Robustness of IV Results: Mortality is Uncorrelated with Residing in a PSU that is Religiously Dissimilar from the Respondent Household

dependent variable:	Infant Mortality (IMR)		Neonatal Mortality (NMR)	
sample restriction:	Mixed Religion PSU		Mixed Religion PSU	
specification:	IV	IV	IV	IV
	T9 - Col 7		T9 - Col 14	
	(1)	(2)	(3)	(4)
PSU mean OD (except own)	61.4**	61.8**	44.1*	44.2*
	(21.5)	(21.6)	(17.4)	(17.5)
own household OD	-14.1	-14.1	-7.8	-7.8
	(7.9)	(7.9)	(6.4)	(6.4)
fraction of PSU religiously dissimilar		-4.7		-1.1
		(3.9)		(3.2)
extended controls	X	X	X	X
mean of dep. var.	72.2	72.2	46.2	46.2
first stage F-stat	230.7	231.1	230.7	231.1
observations (live births)	104,090	104,090	104,090	104,090

Note: Table reports results from IV regressions of mortality on neighbors' open defecation in the PSU ($\overline{OD_{ij}^{-i}}$), instrumented with $\overline{M}_j$. The sample is restricted to the mixed-religion PSU sample over which the IV is defined ($0 < \overline{M} < 1$). Columns 1 and 3 repeat results from Table 9 for comparison. Columns 2 and 4 add a control for the fraction of the respondent's neighborhood that is religiously dissimilar, which equals $\overline{M}_j$ for Hindu households and $1 - \overline{M}_j$ for Muslim households. Extended controls are as described in the Table 2 notes. All regressions control for survey round fixed effects. Observations are children (live births). Standard errors are clustered at the PSU level. * $p < 0.05$, ** $p < 0.01$.

Table A12: Diarrhea, Fever, and Cough

dependent variable:	Infant Mortality (IMR)
	(1)
cough	-0.0397**
	(0.0035)
diarrhea	-0.1275**
	(0.0063)
cough X PSU fraction Muslim	0.0001
	(0.0092)
diarrhea X PSU fraction Muslim	-0.0293
	(0.0172)
mother X child FEs	X
observations (children X symptoms)	189,735

Note: Table reports results from an OLS regression. Observations are children $\times$ symptoms, so that the sample stacks three observations per child, one for each symptom: fever, cough, and diarrhea. Mother $\times$ child fixed effects are included. The coefficients are relative to the excluded categories—fever and fever $\times$ $\overline{M_j}$. The sample consists of mixed-religion PSUs ($0 < \overline{M} < 1$). * $p < 0.05$, ** $p < 0.01$.