

The Causal Relationship between Sanitation and
Child Height
Effects at Household and Community Levels

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1 Introduction

Height is an important measure of population health and human capital, with links to labor market outcomes (Case and Paxson, 2006; Vogl, 2012), mortality (Jousilahti et al., 2000), and cognitive achievement (Spears, 2012). While genetics play a major role in determining maximum height, early life nutrition and disease environment strongly influence the extent to which this height potential is achieved (JH, 1999). One major determinant of the disease environment, particularly in less-developed countries, is exposure to poor sanitation in the form of open defecation. India has one of the highest rates of open defecation, with over 60% of its rural population and 44% of the overall population lacking toilet or latrine facilities in 2015 (WHO/UNICEF JMP, 2015).

India also has high rates of stunting, with 38% of children in 2015 under the age of five being too short for their age (National Family Health Survey-4, 2016). Indeed, children in India are shorter, on average, than children in Africa, who are poorer, on average, and one proposed explanation for this “Asian enigma” is the high rates of open defecation in India (Spears, 2013). Recent evidence in the form of a randomized field experiment (Hammer and Spears, 2016) and an ecological analysis at the district level (Spears et al., 2013) suggests that there is a causal relationship between the sanitation environment at the neighborhood level and height-for-age among children in India. This study uses data from three household surveys to study the causal effects of open defecation at both the household and neighborhood levels in India using a sample of over 70,000 children from three rounds of a national household survey.

1.1 Causal questions

In this study, we examine the effects of open defecation (hereafter OD) at the household and neighborhood levels on height for age of Indian children aged three and younger. Specifically, we ask and answer the following questions: (1) What is the effect of living in a household with a latrine in neighborhoods where the OD rate is low? (2) What is the effect of living in a household with a latrine in neighborhoods where the OD rate is high? (3) What is the effect of a high versus low neighborhood-level OD rate on children who are likely to live in a household with a latrine?

Note that we use the terms OD and latrine use interchangeably; a household practicing OD is assumed to not use a latrine or toilet, and vice versa. The first three of the above questions closely parallel those asked by Hong and Raudenbush (2006) in a study of the effect of kindergarten retention on reading outcomes. We adopt their analytical framework for studying multilevel observational data as described in Section 2 below.

1.2 Data

The data we use come from three rounds of the National Family Health Survey (NFHS). The NFHS is a cross-sectional survey conducted in 1992-93, 1998-99, and 2005-06 intended to yield estimates of indicators of maternal and child health, nutrition, and reproductive health valid for the rural and urban parts of Indian states. The NFHS includes anthropometric measurements of height for children born to interviewed mothers in the three years prior to the survey, as well as numerous mother- and household-level covariates. The outcome variable is the height-for-age z -score: a child’s height converted into a z -score relative to the 2006 WHO healthy reference population. While children are

naturally nested within mothers and households, we ignore this level of nesting in our analysis. Because heights are measured only for children born in the three years prior to the survey, 90% of mothers and 89% of households in our sample have data for only one child. Conversely, 81% of the children in our sample are the only measured child of their mother and 89% are the only measured child from their household.

For the purposes of this study, we define “neighborhood” to be the primary sampling unit (PSU) to which each household belongs. In rural areas, the PSUs are generally villages of up to 500 households (larger villages are split in two), and in urban areas the PSUs are census enumeration blocks (CEBs) consisting of 150-200 households (technically, the urban PSUs are census wards in which the CEBs are nested, but since one CEB is selected per ward, we can think of the CEB as the PSU (Roy and Acharya, 2016)). Within selected PSUs, a sample of roughly 15-60 households is taken. To ensure adequate PSU-level sample sizes, we discard PSUs with fewer than 15 sampled households. We define OD at the household level as households reporting that their toilet facility is “no facility/bush/field”. See Appendix A for more details on our sample.

2 Causal inference framework

2.1 Relaxing SUTVA

We use the potential outcomes framework of Rubin (1978) to define causal effects and the estimands. In a population of N children, $i = 1, \dots, N$, we let z_i denote the treatment status of the household in which child i lives: $z_i = 1$ if the household practices OD and $z_i = 0$ if it does not. We let $\mathbf{z} = (z_1, \dots, z_N)$ denote the vector of treatment assignments for all N children. Since the treatment is binary, $z_i \in \{0, 1\}$ and $\mathbf{z} \in \{0, 1\}^{2N}$. The potential outcome of child i , were their household’s treatment status z_i and the other households’ treatments $\mathbf{z}_{-i} = (z_1, \dots, z_{i-1}, z_{i+1}, \dots, z_N)$, is $Y_i(\mathbf{z})$.

A fundamental assumption that makes causal identification possible in the potential outcomes framework is the stable unit treatment value assumption (SUTVA). SUTVA states that there is a single version of the treatment and that the potential outcome of one unit is independent of the treatment assignment of the other units (Rubin, 1986). In other words, SUTVA allows us to simplify the definition of potential outcome and write $Y_i(\mathbf{z}) \equiv Y_i(z_i, \mathbf{z}_{-i}) = Y_i(z_i)$. In our context, SUTVA implies that the potential outcome for a child depends only on the sanitation status of their own household and not on that of their neighbors.

A key methodological challenge in this study is that SUTVA is an unreasonable assumption. Because OD by definition occurs outdoors, the practice of OD by one household necessarily exposes others in the neighborhood to possible contact with human waste. It is therefore entirely plausible that the sanitation behavior of the neighbors of a given household can affect the potential outcome of a child in that household, regardless of the sanitation status of that household.

Our analytical strategy for relaxing SUTVA is inspired by the approach taken by Hong and Raudenbush (2006), hereafter HR, in a study of the causal effect of kindergarten retention (i.e. repeating kindergarten) on reading outcomes, a context in which SUTVA is also an implausible assumption. Hong and Raudenbush (2006) handle this complication by assuming that the collective effects of \mathbf{z}_{-i} on $Y_i(\mathbf{z})$ can be summarized by z_i and a

scalar function $v(\mathbf{z})$, so that

$$Y_i(\mathbf{z}) \equiv Y_i(z_i, \mathbf{z}_{-i}) = Y_i(z_i, v(\mathbf{z})). \quad (1)$$

Following HR, we use $v(\mathbf{Z}) = V$ to denote the binary random variable representing the neighborhood-level OD rate that takes on values $v(\mathbf{z}) = v = 0$ in low-OD neighborhoods and $v(\mathbf{z}) = v = 1$ in high-OD neighborhoods. Empirically, we use the average OD rate by survey round and rural-urban status to classify neighborhoods as high- or low-OD. See Appendix A for details.

Given two sets of possible treatment assignments for a single unit and all units, (z, \mathbf{z}) and (z', \mathbf{z}') , a generic causal estimand is of the form $\mathbb{E}[Y(z, v(\mathbf{z})) - Y(z', v(\mathbf{z}'))]$. This formulation relies on three main assumptions as described by HR: a) causal inferences are generalized conditional on current neighborhood residence; b) there is no interference between neighborhoods, and c) treatment assignment at both the household and neighborhood levels are strongly ignorable conditional on observed covariates. See Appendix B for a more detailed description of these assumptions.

2.2 Subpopulations and estimable causal quantities

An important feature of HR's analysis of kindergarten retention rates is their division of children into subpopulations depending on their risks of retention under high and low school-level retention rates. They make this division because some potential outcomes are not defined for some children: some children may only be retained under a high school-level retention rate but not under a low one, while other may never be retained regardless of the school-level rate.

The identification of subpopulations is relevant in our context as well. In rural north India, notions of purity, pollution, caste, and religion play an integral role in deep-seated sanitation preferences that make ending OD particularly challenging (Coffey et al., 2014, 2015). OD rates in India as a whole are starkly different by both rural vs. urban residency and by religion. Data from NFHS-3 in 2005-06 show that 17% of urban households practiced OD, compared to 74% of rural households. The same data also show that nearly 60% of Hindu households practiced OD, while only 40% of the relatively poorer Muslim households did.

Let q_1 denote the probability of a household practicing OD under a high neighborhood OD rate and q_0 denote the probability under a low rate. Assume that $q_1 \geq q_0$ for all households. In parallel with HR, we identify three subpopulations of households (equivalently, children, as we ignore the clustering of children in households):

- (A) Households at risk of OD under a low neighborhood OD rate ($q_1 \geq q_0 > 0$)
- (B) Households at risk of OD under a high neighborhood OD rate but not a low rate ($q_1 \geq q_0 = 0$)
- (C) Households at no risk even under a high neighborhood OD rate ($q_1 = q_0 = 0$)

See Table 3 of Appendix C for more details on the potential outcomes and causal effects defined for these subpopulations.

Because of the observational nature of our data, only certain causal quantities are estimable. Specifically, our estimands parallel those in HR and are as follows (see Appendix

C for details). First, we estimate the conditional effect of household OD for children in subpopulation (A) who live in low-OD neighborhoods,

$$\delta_{Z0} = \mathbb{E}[Y_A(1, 0) - Y_A(0, 0) \mid V = 0, \mathbf{X}, \mathbf{W}], \quad (2)$$

where \mathbf{X} and \mathbf{W} are child- and neighborhood-level covariates, respectively. Second, we consider the union of subpopulations (A) and (B), which we denote (AB). Children in subpopulation (AB) are those who are ever at risk for living in households that practice OD ($q_1 > 0$). For this ever-at-risk subpopulation, we estimate the conditional effect of household OD in high-OD neighborhoods for children actually living in high-OD neighborhoods:

$$\delta_{Z1} = \mathbb{E}[Y_{AB}(1, 1) - Y_{AB}(0, 1) \mid V = 1, \mathbf{X}, \mathbf{W}]. \quad (3)$$

The third estimand is the average causal effect of high vs. low neighborhood OD level for children in subpopulation (C), that is, the effect of neighborhood OD for children at no risk of living in households that practice OD:

$$\delta_{V0} = \mathbb{E}[Y_C(1, 1) - Y_C(0, 1) \mid V = 0, \mathbf{W}]. \quad (4)$$

3 Estimation and inference

3.1 Propensity scores

We follow HR in using propensity score stratification to estimate the causal effects δ_{Z0} , δ_{Z1} , and δ_{V0} . We estimate propensity scores at two levels. First, we use logistic regression to estimate the neighborhood-level propensity for a high OD rate conditional on observed neighborhood covariates:

$$Q = \mathbb{P}(V = 1 \mid \mathbf{W}).$$

We then estimate q_1 and q_0 , the propensities for household-level OD for households in high- and low-OD neighborhoods, respectively, and use them to identify subpopulations (A), (B), and (C); see Appendix D for details. We use R and the **Rstan** package for all analyses.

3.2 Causal effects

In (2), we defined δ_{Z0} , the causal effect of being in an OD household in a low-OD neighborhood. Given the assumptions outlined above and in the appendices, we can condition on q_0 in place of \mathbf{X} and \mathbf{W} , and (2) is thus equivalent to

$$\delta_{Z0} = \mathbb{E}[Y_A(1, 0) - Y_A(0, 0) \mid V = 0, q_0].$$

To estimate δ_{Z0} , we use a hierarchical linear regression similar to the approach taken by HR:

$$Y_i \sim N \left(\beta_0 + u_{j[i]} + (\delta_{Z0} + \Delta_{Z0j[i]})z_i + \sum_{g=1}^G \beta_g q_{0gi} + \beta_9 \text{logit}(\widehat{q_{0i}}), \sigma^2 \right)$$

$$\begin{pmatrix} u_j \\ \Delta_{Z0j} \end{pmatrix} \sim N \left[\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} \tau_u & \tau_{u \cdot Z_0} \\ \tau_{u \cdot Z_0} & \tau_{Z0} \end{pmatrix} \right].$$

Here i indexes households/children, j indexes neighborhoods and $j[i]$ denotes the neighborhood to which household i belongs. The outcome Y_i is the height-for-age z -score, z_i is the household-level OD indicator, and q_{0gi} , $g = 1, \dots, G$ are indicators for the $G = 10$ strata of q_0 . Following HR, we also include the estimate \hat{q}_{0i} on the logit scale for additional bias reduction. Here u_j is a neighborhood-level random intercept that captures baseline differences in height-for-age across neighborhoods, while Δ_{Z0j} is the neighborhood-level variation in the treatment effect. Neighborhood variation in the household-level treatment effect is captured by τ_{Z0} , and we can also estimate the covariance between Δ_{Z0j} and u_j with $\tau_{u \cdot Z0}$.

Among children in low OD neighborhoods, the adjusted within-stratum difference in means of height-for-age z -scores between children in OD and non-OD households is $\hat{\delta}_{Z0} = -0.17$ (SE 0.03). This household-level OD effect varied across neighborhoods, with $\hat{\tau}_{Z0} = 0.73$. If Δ_{Z0j} really were normally distributed, then we would expect 95% of the household-level OD effects to range from -1.60 to 1.27 ($\hat{\delta}_{Z0} \pm 1.96\hat{\tau}_{Z0}$) in low-OD neighborhoods.

Similarly, our definition of δ_{Z1} given in (3) is equivalent to

$$\delta_{Z1} = \mathbb{E}[Y_{AB}(1, 1) - Y_{AB}(0, 1) \mid V = 1, q_1].$$

We use a model analogous to the one above, restricting ourselves to children in subpopulation (AB) – that is, those for whom we estimate $q_1 > 0$. For children in subpopulation (AB) who are at risk of living in OD households in high-OD neighborhoods, the estimated treatment effect is $\hat{\delta}_{Z1} = -0.18$ (SE 0.02). Here again we observe significant variation across neighborhoods, with $\hat{\tau}_{Z1} = 0.71$, which would lead to a 95% range of -1.56 to 1.21 for household-level OD effects in high-OD neighborhoods. This is quite similar to the case for low-OD neighborhoods, suggesting that the effect of household-level OD does not vary much by (binary) neighborhood OD level.

For estimating δ_{V0} , we use children in subpopulation (C) for whom we estimate $q_1 = 0 = q_0$. Our hierarchical model in this case is

$$\begin{aligned} Y_i &\sim N \left(\beta_0 + u_{j[i]} + \delta_{V0}v_j + \sum_{h=1}^H \beta_h Q_{hj} + \beta_9 \text{logit}(\widehat{Q}_j), \sigma^2 \right) \\ u_j &\sim N(0, \tau), \end{aligned}$$

where v_j denotes the neighborhood OD level and Q_{hj} are indicators for the $H = 10$ strata of \widehat{Q}_j . We estimate that the neighborhood-level effect of high vs low OD levels among children not at risk of living in OD households is $\delta_{V0} = 0.98$ (SE 0.67). This estimate is not statistically significant, meaning we cannot detect an effect of high vs low OD neighborhoods on height-for-age among children at low risk of living in OD households.

4 Discussion

Given the stubbornly high rates of OD in India and the growing evidence of the causal relationship between sanitation and health, further research into the specific causal mechanisms of how sanitation affects height is urgently needed. Specifically, research on potential effect modifiers and variability in the treatment effect of OD at both household and neighborhood levels will enable policymakers to better target resources into expanding

latrine use and mitigating the negative effects of OD.

While observed neighborhood OD rates are naturally continuous over $[0, 1]$, we have dichotomized them into low and high, perhaps unnecessarily throwing away some information. A better approach might be to extend the framework of HR to allow the neighborhood rate to vary continuously from 0 to 1. Such an extension would require the use of the generalized propensity score (Hirano and Imbens, 2005), which extends the propensity score and its balancing properties to nonbinary treatments to estimate the neighborhood OD propensity Q .

See Appendix E for a discussion of the limitations of this study.

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Appendix A NFHS data

Figure A shows the distribution of height-for-age z -scores by survey round and household OD status. Height is measured for children born in the three years prior to the survey to ever-married women aged 15–49. The z -scores are calculated relative to the 2006 WHO reference population and are adjusted for age and sex. Extreme values of the z -scores ($|z| > 6$), likely resulting from measurement and/or transcription error, have been dropped. Table 1 displays the sample size by survey round.

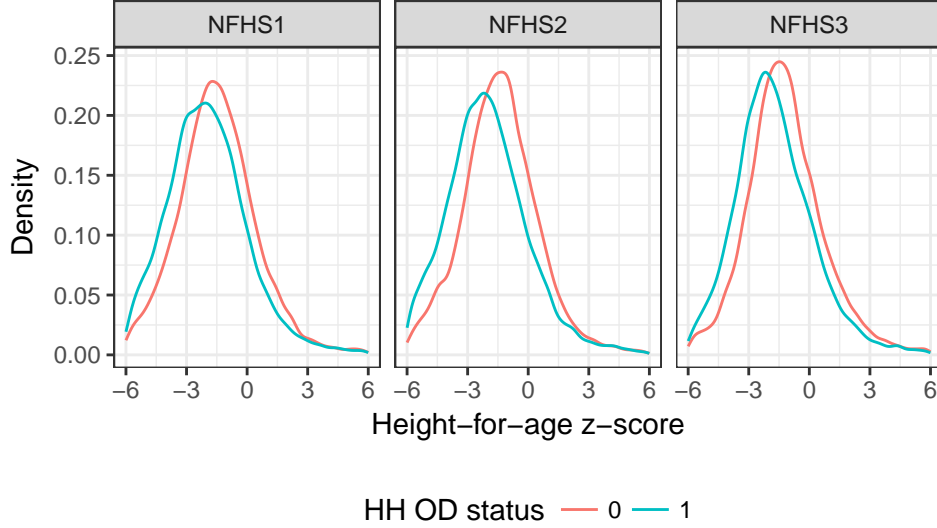


Figure 1: Distribution of height-for-age z -scores by survey round and household OD status.

Round	N
NFHS1	20993
NFHS2	25727
NFHS3	24896

Table 1: Sample size by survey round.

Table 2 displays the OD rates by survey round and rural-urban residence, with rates for all India shown for reference. NFHS-1 was conducted in 1992-93, NFHS-2 in 1998-99, and NFHS-3 in 2005-06.

	NFHS-1	NFHS-2	NFHS-3
Rural	87.1	80.8	74.0
Urban	24.1	19.2	16.8
All India	69.7	63.7	55.4

Table 2: OD rates (%) by survey round and rural-urban residency.

Appendix B Causal assumptions

In this section, we provide details on the main assumptions underlying our causal framework as established by Hong and Raudenbush (2006).

Intact neighborhoods. The generic estimand above implicitly ignores the assignment of individuals to neighborhoods. If we were to take this assignment into account, we would include the vector of neighborhood assignments \mathbf{s} in the potential outcome, where $\mathbf{s} = (s_1, \dots, s_N)$ and $s_i \in \{1, \dots, J\}$ denotes which of the J neighborhoods unit i is assigned to. The estimand would then be

$$\mathbb{E}[Y(z, \mathbf{z}, \mathbf{s}) - Y(z', \mathbf{z}', \mathbf{s}')],$$

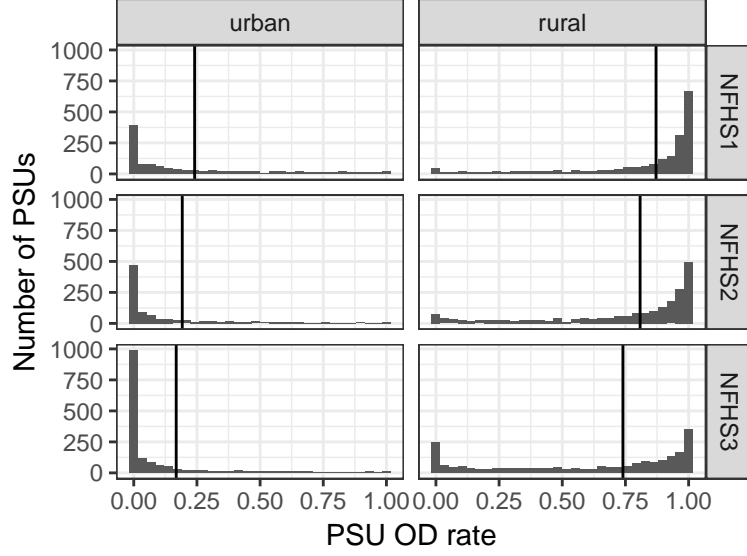


Figure 2: PSU (neighborhood) OD rates by survey round and rural-urban residency. Vertical lines denote mean OD rate. Only PSUs with at least 15 sampled households are included.

where \mathbf{s} and \mathbf{s}' are two vectors of possible neighborhood assignments. However, we know that in the real world, people are not assigned to neighborhoods at random, and we wish to estimate causal effects conditional on actual neighborhood membership. We therefore condition our estimands on the observed neighborhood assignment \mathbf{s}^* :

$$\mathbb{E}[Y(z, \mathbf{z}, \mathbf{s}^*) - Y(z', \mathbf{z}', \mathbf{s}^*) \mid \mathbf{S} = \mathbf{s}^*].$$

For clarity of notation, we consider all estimands to be conditional on $\mathbf{S} = \mathbf{s}^*$ from this point forward and do not explicitly write out this conditioning.

No interference between neighborhoods. We assume that the potential outcome for a child can be affected by the treatment assignments of other children in the same neighborhood, but not by the treatment assignments of children from other neighborhoods. We therefore write $\mathbf{z}_j = (z_{1j}, \dots, z_{n_jj})$ for the vector of treatment assignments for the n_j children in neighborhood j and $\mathbf{s}_j = (j, \dots, j)$ for the corresponding neighborhood assignments. The potential outcome for a child is then

$$Y_i(z_i, v(\mathbf{z}), \mathbf{s}^*) = Y_i(z_i, v(\mathbf{v}_{j[i]}), \mathbf{s}_{j[i]}^*) \equiv Y_i(z_i, v),$$

where the notation $j[i]$ refers to the neighborhood j that unit i belongs to. We let $V = v(\mathbf{Z})$ denote the random variable taking on values $v(\mathbf{z}) = v \in [0, 1]$.

Strongly ignorable treatment assignment. We assume that the assignments of intact neighborhoods to open defecation level $V = v$ and children to household latrine use status $Z = z$ are ignorable conditional on observed neighborhood-level covariates \mathbf{W} and child- and household-level covariates \mathbf{X} . Under this assumption, we can write

$$\mathbb{E}[Y(z, v) \mid Z = z, V = v, \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}] = \mathbb{E}[Y(z, v) \mid \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}],$$

allowing us to estimate the conditional average causal effect

$$\mathbb{E}[Y(z, v) \mid \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}] - \mathbb{E}[Y(z', v') \mid \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}]$$

with the observed quantity

$$\mathbb{E}[Y(z, v) \mid Z = z, V = v, \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}] - \mathbb{E}[Y(z', v') \mid Z = z', V = v', \mathbf{X} = \mathbf{x}, \mathbf{W} = \mathbf{w}]$$

Together, the assumptions of intact neighborhoods, no interference between neighborhoods, and strong ignorability allow us to conceive of our observational data as approximating a two-stage experiment. In the first stage, neighborhoods are randomly assigned an open defecation rate within blocks defined by \mathbf{W} . In the second stage, households within neighborhoods are randomly assigned to binary latrine use within blocks defined by \mathbf{X} and \mathbf{W} .

Appendix C Causal estimands and strong ignorability

Table 3 displays the potential outcomes and causal effects of interest for subpopulations of households defined by their probabilities of OD under high and low neighborhood-level OD rates. This table parallels Table 2 of HR.

Subpopulation	Probabilities of OD	Potential outcomes	Causal effects of interest
(A)	$q_1 \geq q_0 > 0$	$Y(1, 1), Y(0, 1), Y(1, 0), Y(0, 0)$	$\mathbb{E}[Y(1, 1) - Y(0, 1)]$ $\mathbb{E}[Y(1, 0) - Y(0, 0)]$
(B)	$q_1 \geq q_0 = 0$	$Y(1, 1), Y(0, 1)$	$\mathbb{E}[Y(1, 1) - Y(0, 1)]$
(C)	$q_1 = q_0 = 0$	$Y(0, 1), Y(0, 0)$	$\mathbb{E}[Y(0, 1) - Y(0, 0)]$

Table 3: Potential outcomes and causal effects for subpopulations of households

To understand which causal effects are estimable under strong ignorability, we imagine that our observational data are in fact from two-stage randomized experiment. Neighborhoods are first randomized to a binary OD level $V = v \in \{0, 1\}$ within blocks defined by neighborhood covariates \mathbf{W} with probability $Q = Q(\mathbf{W})$:

$$Q = \mathbb{P}(V = v \mid \mathbf{W}). \quad (5)$$

Given the neighborhood OD level, households with neighborhoods are randomly assigned an OD status, $Z = 1$ for practicing OD and $Z = 0$ for latrine use, within blocks defined by \mathbf{X} and \mathbf{W} . The household-level probability of $Z = 1$ is then $q_v = q_v(\mathbf{X}, \mathbf{W})$, where

$$q_v = \mathbb{P}(Z = 1 \mid V = v, \mathbf{X}, \mathbf{W}), \quad (6)$$

and a child's probability of living in a household with OD status $Z = z$ and neighborhood with OD level $V = v$ is

$$\mathbb{P}(Z = z, V = v \mid \mathbf{X}, \mathbf{W}) = \mathbb{P}(Z = z \mid V = v, \mathbf{X}, \mathbf{W})\mathbb{P}(V = v \mid \mathbf{W}). \quad (7)$$

Of course, we could also have factored the above as

$$\mathbb{P}(Z = z, V = v \mid \mathbf{X}, \mathbf{W}) = \mathbb{P}(V = v \mid Z = z, \mathbf{X}, \mathbf{W})\mathbb{P}(Z = z \mid \mathbf{x}, \mathbf{W}). \quad (8)$$

But if our data really did come from a two-stage randomized experiment, then we can write the first term on the right-hand side of (8) as $\mathbb{P}(V = v \mid Z = z, \mathbf{X}, \mathbf{W}) = \mathbb{P}(V = v \mid \mathbf{W})$. Since (7) and (8) are equal, this implies that

$$\mathbb{P}(Z = z \mid V = v, \mathbf{X}, \mathbf{W}) = \mathbb{P}(Z = z \mid \mathbf{X}, \mathbf{W}),$$

meaning we can estimate the unobserved quantity on the right hand side with the observed quantity on the left hand side.

Unfortunately, our data are not actually from a two-stage randomized experiment: clearly, neighborhoods are not assigned at random to high or low OD rates. We therefore cannot use a model for q_1 fit to data from children in high-OD neighborhoods ($V = 1$) to estimate q_1 for children in low-OD neighborhoods. The same holds for q_0 . As a result, we must modify our causal estimands δ_{Z0} and δ_{Z1} to condition on the observed neighborhood OD level. In other words, we estimate the conditional effect of household-level OD for households in low (high) OD neighborhoods for children who actually live in low (high) OD neighborhoods.

Appendix D Propensity score models

To estimate Q , the propensity for a neighborhood having a high OD rate, we use a hierarchical logistic regression model that accounts for the nesting of neighborhoods within states:

$$\begin{aligned} Q_j &= \mathbb{P}(V_j = 1 \mid \mathbf{W}) \\ \text{logit}(Q_j) &= \alpha_0 + \alpha_1 \mathbf{1}(NFHS2) + \alpha_2 \mathbf{1}(NFHS3) + \\ &\quad \beta_1 \mathbf{1}(\text{rural}_j) + \beta_2 \text{assets}_j + \beta_3 \text{water}_j + \beta_4 \text{muslim}_j + \beta_5 \text{hindu}_j + u_{s[j]} \\ u_s &\sim N(0, \sigma_u^2), \end{aligned}$$

where j indexes neighborhoods, s indexes states, $s[j]$ denotes the state for neighborhood j , $\mathbf{1}(NFHS2)$ and $\mathbf{1}(NFHS3)$ are indicators for NFHS survey rounds, $\mathbf{1}(\text{rural}_j)$ is an indicator for a rural neighborhood, assets_j is the average proportion of seven specific assets measured in each survey round owned by households in the neighborhood, water_j is the proportion of households with an improved water source, muslim_j is the proportion of Muslim households, hindu_j is the proportion of Hindu households, and u_s is a state varying intercept.

We then stratify the estimates $\text{logit}(\hat{Q}_j)$ into ten groups of roughly equal size. Table 4 shows the number of neighborhoods by stratum and OD status (low/high), and Figure 3 plots the density of $\text{logit}(\hat{Q}_j)$ by stratum and OD status. While the distribution of $\text{logit}(\hat{Q}_j)$ is quite variable even within strata, the balance of covariates between low- and high-OD neighborhoods is adequate. Figure 4 compares the within-stratum distribution of survey round and rural residence between high- and low-OD neighborhoods, while Figures 5 to 8 show the distributions of household asset ownership, proportion of households with improved water, proportion Muslim, and proportion Hindu. These figures show that we have achieved reasonable balance of the covariates within strata of $\text{logit}(\hat{Q}_j)$.

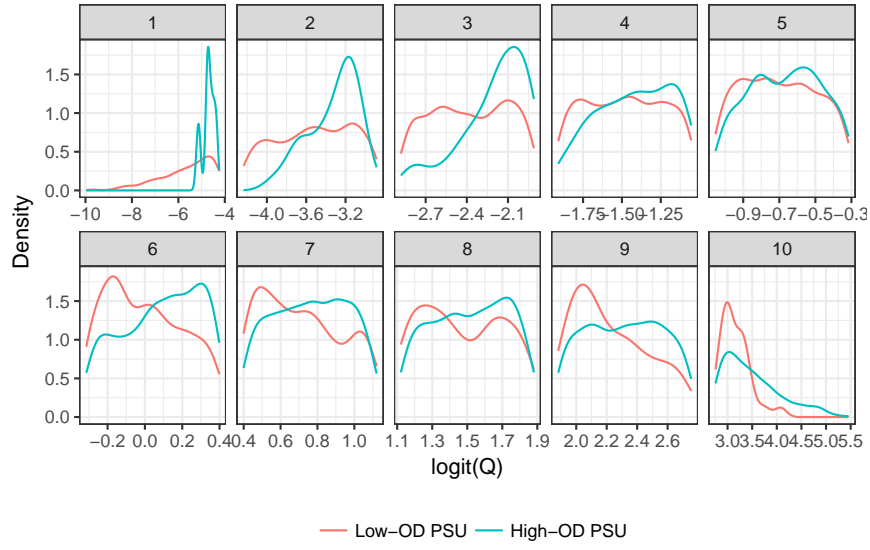


Figure 3: Density of $\text{logit}(\hat{Q}_j)$ by stratum and OD status.

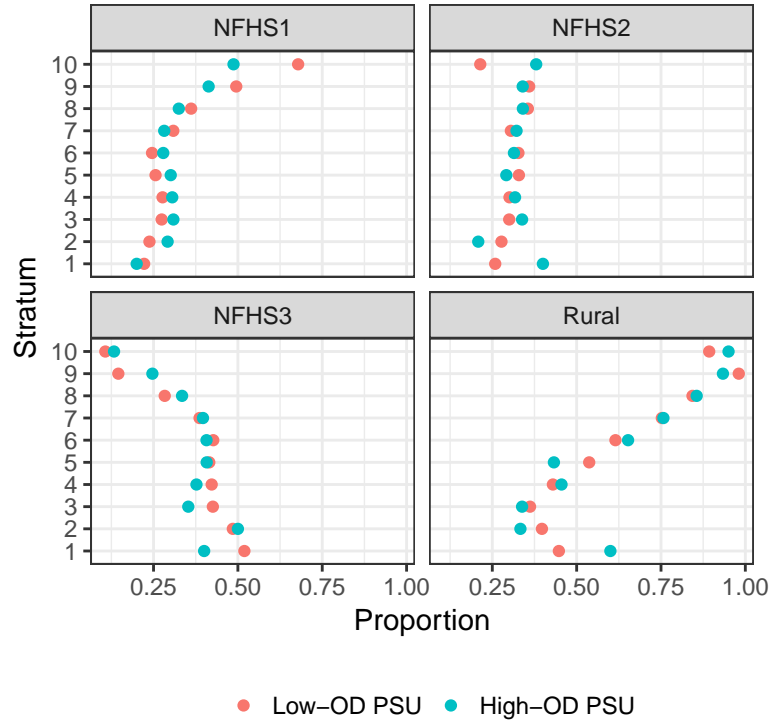


Figure 4: Balance of survey rounds and rural residence within strata of $\text{logit}(\hat{Q}_j)$.

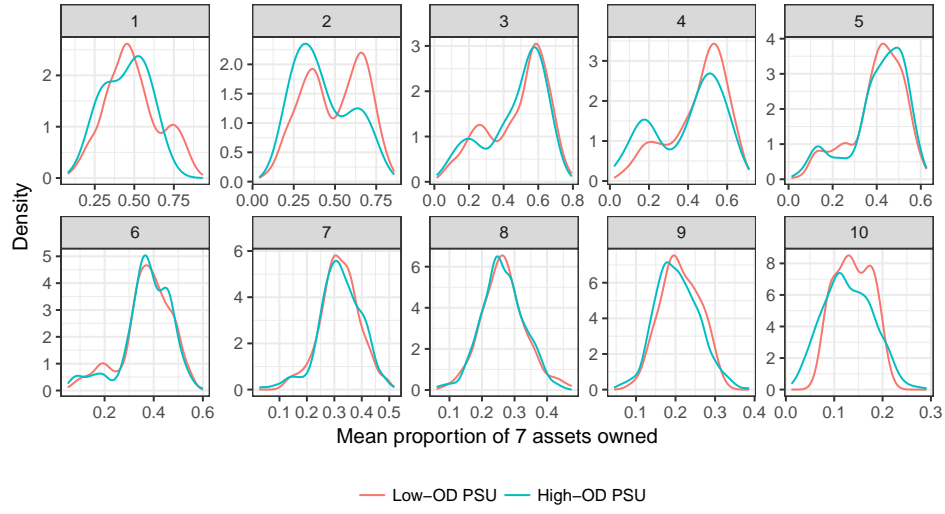


Figure 5: Density of asset ownership by stratum and OD status.

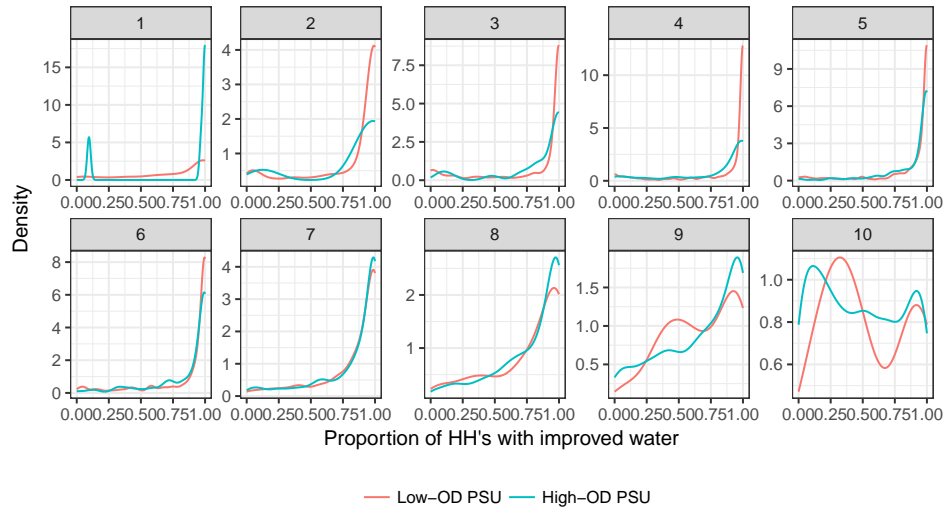


Figure 6: Density of proportion of households with improved water source by stratum and OD status.

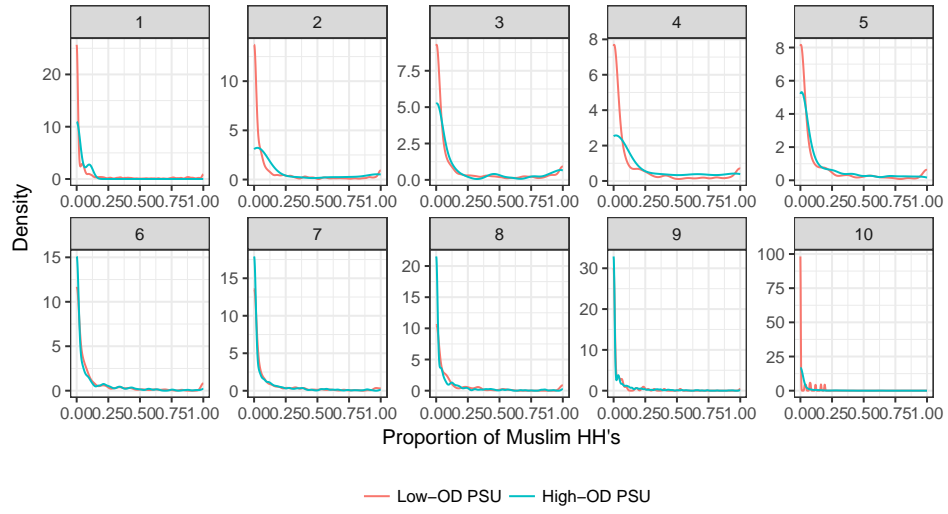


Figure 7: Density of proportion of Muslim households by stratum and OD status.

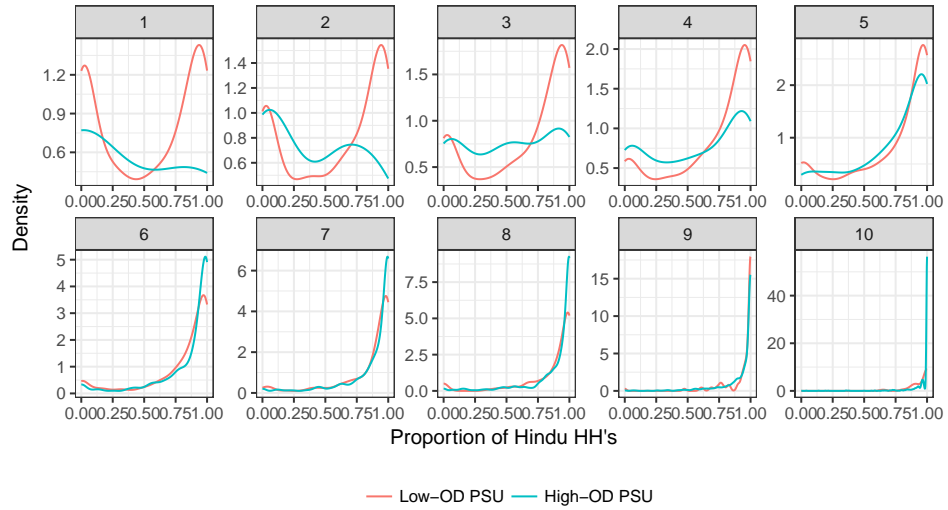


Figure 8: Density of proportion of Hindu households by stratum and OD status.

Stratum	Low OD	High OD
1	986	5
2	967	24
3	923	68
4	824	167
5	672	319
6	452	538
7	295	696
8	166	825
9	103	888
10	28	963

Table 4: Number of neighborhoods in strata of $\text{logit}(\hat{Q}_j)$.

We now turn to modeling q_1 and q_0 . We again use a hierarchical logistic regression model with household-level predictors and a neighborhood-level varying intercept. For q_1 , we estimate the model

$$\begin{aligned} \text{logit}(q_{1i}) = & \beta_0 + \beta_1 \mathbf{1}(NFHS2) + \beta_2 \mathbf{1}(NFHS3) + \\ & \beta_3 \mathbf{1}(rural_i) + \beta_4 assets_i + \beta_5 water_i + \beta_6 muslim_i + \beta_7 hindu_i + u_{j[i]} \\ u_j \sim & N(0, \sigma_u^2), \end{aligned}$$

where i now indexes households and j indexes neighborhoods. The predictor $assets_i$ is the proportion of the seven assets owned by household i , $water_i$ is an indicator for having an improved water source, and $muslim_i$ and $hindu_i$ are indicators for Muslim and Hindu households, respectively. We estimate $\text{logit}(\hat{q}_1)$ using only the children in high-OD neighborhoods.

Following HR, we can identify the subpopulation that each child belongs to given these quantities. To identify subpopulation (AB), children ever-at-risk for living in households that practice OD, we stratify the estimated values $\text{logit}(\hat{q}_1)$ into 10 strata. We then take the minimum observed value of $\text{logit}(\hat{q}_1)$ among children in OD households and classify children in non-OD households whose $\text{logit}(\hat{q}_1)$ is less than this minimum as belonging to subpopulation (C). All other children whose $\text{logit}(\hat{q}_1)$ is above the minimum are considered part of subpopulation (AB).

We estimate $\text{logit}(\hat{q}_0)$ using analogous modeling and stratification strategies, this time using only children in low-OD neighborhoods. We calculate the minimum value of $\text{logit}(\hat{q}_0)$ among children in non-OD households and classify all children in low-OD neighborhoods with $\text{logit}(\hat{q}_0)$ above this minimum as belonging to subpopulation (C). Figures 9 and 10 show the estimated values of $\text{logit}(\hat{q}_1)$ and $\text{logit}(\hat{q}_0)$ by household OD status.

Appendix E Limitations

One limitation of the present study is that we have ignored the clustering of children within households, thus implicitly treating children in the same community as independent. Children within the same household are likely to be more similar than children in different households, so if the data for a community has many children clustered in only

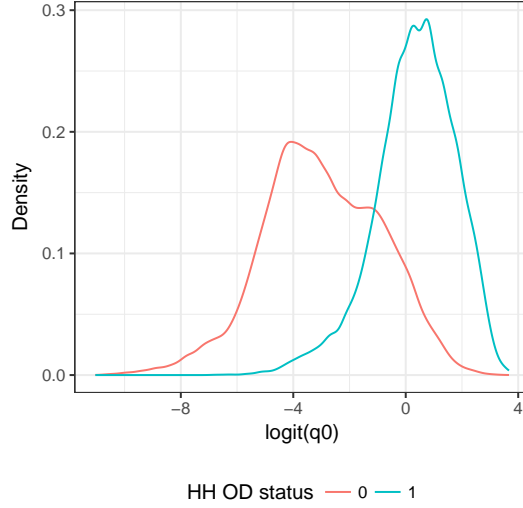


Figure 9: Density of $\text{logit}(\hat{q}_0)$ for OD and non-OD households in low-OD neighborhoods.

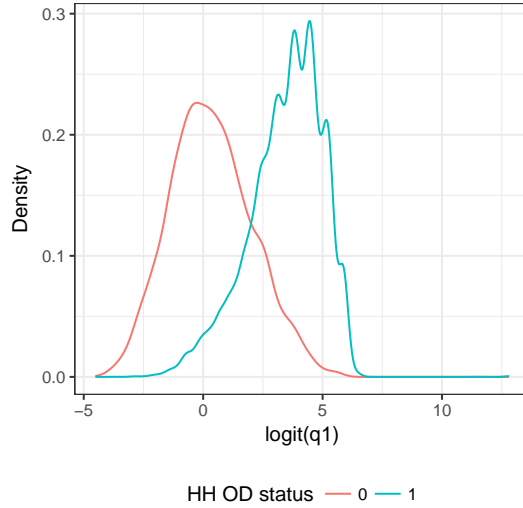


Figure 10: Density of $\text{logit}(\hat{q}_1)$ for OD and non-OD households in high-OD neighborhoods.

a few households, they are less informative than they would be for a community with the same number of children with one child per household. However, 89% of the children in our data belong to unique households and 81% to unique mothers.

Our models do not take the state of residence into account due to computational constraints. However, the large differences in OD rates between states make this an important source of variation to account for. Careful consideration of how to best capture the relationship between states, neighborhoods, and survey rounds will yield more precise estimates.

We also do not know the geographic size of the PSUs. While the DHS manual tells us that urban PSUs are generally census enumeration blocks (roughly 150-200 households) and rural PSUs are villages of 500 or fewer households, there is likely to be variation in the geographic size and population density of the PSUs. In that case, our results are likely to be lower bounds, as the median sample size per PSU is 29 households (interquartile range 22 to 33). A recent study using both cross-national data from 172 countries and

subnational data from Bangladesh suggests that the negative effects of neighborhood-level open defecation are magnified by high population density (Hathi et al., 2017). However, an epidemiological study in Guatemala did not find that population density was an important determinant of intestinal infections or a strong effect modifier for household-level sanitation (Jarquin et al., 2016), so evidence remains mixed.