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, mortality, and cognitive achievement (cite here). 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 (CITE). One major determinant of the disease environment, particularly in less-developed countries, is exposure to poor sanitation in the form of open defecation. Globally, over a billion people practice open defecation, nearly 60% of whom are in India (WHO/UNICEF JMP, 2015). 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, 2015-2016, 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). Evidence in the form of a randomized field experiment (Hammer and Spears, 2016) and an ecological analysis at the district level (Spears et al., 2013a) 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 four household surveys to study the causal effects of open defecation at both the household and community levels in India using a sample of over XXX children.

1.1 Sanitation and child health in India

Previous studies have established strong links between sanitation and child health in India, particularly at the neighborhood level. Geruso and Spears (2015) estimate that a 10 percentage-point reduction in neighborhood-level open defecation "is associated with a decline in infant mortality of 6.5 infants per 1,000". Children in India tend to be exceptionally short for their height, particularly compared to children in sub-Saharan African countries, where children are poorer, on average; this puzzle is known as the "Asian enigma", and some studies suggest that open defecation, which is substantially more prevalent in India than in sub-Saharan Africa, can account for much of this discrepancy (Spears, 2013). Within India, an ecological analysis at

the district level (Spears et al., 2013b) estimates that a 10 percentage-point increase in open defecation is associated with a 0.7 percentage point increase in stunting and severe stunting. A recent randomized field experiment in rural Maharashtra, India, that subsidized construction of household pit latrines and promotes sanitation at the village level yielded an increase of about 0.3 height-for-age standard deviations among children children under five after 18 months (Hammer and Spears, 2016). This estimate is close to the 0.2 height-for-age standard deviation increase associated with the Total Sanitation Campaign, an Indian government program dedication to building latrines and promoting sanitation in India (Spears, 2012).

1.2 Causal questions

In this study, we examine the effects of open defecation 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 open defecation rate is low?
2. What is the effect of living in a household with a latrine in neighborhoods where the open defecation rate is high?
3. What is the effect of a high versus low neighborhood-level open defecation rate on children who are likely to live in a household with a latrine?
4. What is the dose-response relationship between neighborhood-level open defecation and height for age?
5. How does the dose-response relationship vary with household-level sanitation?

Note that we use the terms open defecation and latrine use interchangeably; a household practicing open defecation 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 follow and expand upon their analytical framework as described in Section 2 below.

1.3 Data

The data we use come from three rounds of the National Family Health Survey (NFHS) and the fourth round of the District-Level Household Survey (DLHS). 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 DLHS is also cross-sectional but was designed to be representative at the district level. We use the fourth round of the DLHS (DLHS-4), conducted in 2012-14. The DLHS-4 is the only round to include anthropometric measurements of height. It also omits nine of the less developed Indian states, which were instead covered by the Annual Health Survey (AHS). The AHS has much larger sample sizes than the DLHS and was designed to produce more accurate district-level estimates in these states (Dandona et al., 2016).

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)).

2 Methods

We use the potential outcomes framework of Rubin (1978) to define causal effects and the estimands. In a population of N children, $i = 1, \ldots, N$, we let z_i denote the treatment status of the household in which child i lives: $z_i = 1$ if the household practices open defecation and $z_i = 0$ if it does not. (Here we implicitly assume that each household has exactly one child. This is not exactly true, but X% of the children in our data belong to distinct households.) We let $\mathbf{z} = (z_1, \ldots, z_N)$ 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 $\mathbf{z}_{-i} = (z_1, \ldots, z_{i-1}, z_{i+1}, \ldots, z_N)$, is $Y_i(\mathbf{z})$. In this study, our outcome is the height-for-age z-score: a child's height converted into a z-score relative to the 2006 WHO healthy reference population.

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. It is 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. Indeed, as discussed previously, several studies have found evidence of causal effects of neighborhood-level open defecation rates. Because open defecation by definition occurs outdoors, the practice of open defecation by one household necessarily exposes others in the neighborhood to possible contact with human waste. Furthermore, the effect of household-level open defecation may vary with the neighborhood-level rate. If it is analogous to vaccination, then the protective effect of latrine use at the household level may be weak in a neighborhood where open defecation rates are low and stronger in neighborhoods where it is higher. Alternatively, the opposite could be true: the benefits of household-level latrine use in a neighborhood with high open defecation rates may be insignificant compared to the negative effects of high exposure to waste but striking in a neighborhood with low open defecation rates. Either way, as the neighborhood-level open defecation varies from low to high – that is, as more and more neighbors are "assigned" to the treatment status of open defecation – it is not plausible that the potential outcome of a given child in a given household would remain unchanged.

2.1 Causal inference framework

Our analytical framework 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, another context in which SUTVA is 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

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

HR restrict $v(\mathbf{z})$ to be binary, $v(\mathbf{z}) = 0$ for neighborhoods with low rates of open defecation and $v(\mathbf{z}) = 1$ for neighborhoods with high rates. In this study, we generalize their approach and allow $v(\mathbf{z})$ to take on values in $[0, 1]$. 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 $\mathbb{E}[Y(z, \mathbf{z}) - Y(z', \mathbf{z}')]$. 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. Following HR, we briefly describe these three assumptions in the context of our study.

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 $\mathbf{s} = (s_1, \ldots, s_N)$ and $s_i \in \{1, \ldots, J\}$ denotes which of the J neighborhoods unit i is assigned to. The estimand would then be

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

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 $\mathbf{z}_j = (z_{1j}, \ldots, z_{n_j j})$ for the vector of treatment assignments for the n_j children in neighborhood j and $\mathbf{s}_j = (j, \ldots, j)$ 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 $\mathbf{W}$.

2.2 Estimable causal quantities

We now consider which causal quantities can be estimated from our data under the assumption of strong ignorability. Again following the presentation of HR, we can think of our observational study as an approximation of a two-stage randomized experiment. Both stages use a randomized blcok design, first at the cluster level and then at the unit level within clusters. Thus, neighborhoods are first randomized to a continuous OD level $V = v \in [0, 1]$ within blocks defined by $\mathbf{W}$ with probability $Q = Q(\mathbf{W})$:

$$Q = \mathbb{P}(V = v \mid \mathbf{W}). \tag{2}$$

Given the neighborhood OD rate, 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

$$q_v = \mathbb{P}(Z = 1 \mid V = v, \mathbf{X}, \mathbf{W}), \tag{3}$$

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

$$\mathbb{P}(Z = z, V = v \mid \mathbf{X}, \mathbf{W}) = \mathbb{P}(Z = z \mid V = v, \mathbf{X}, \mathbf{W})\mathbb{P}(V = v \mid \mathbf{W}). \tag{4}$$

Under the two-stage randomized experiment,

2.3 Generalized propensity score

2.3.1 Framework

Methods for generalizing propensity scores for nonbinary treatments have been developed by Imai and van Dyk (2004) and Hirano and Imbens (2005). In contrast to the propensity score developed by Rosenbaum and Rubin (1983), which assumes a binary treatment, these generalizations allow for the treatment to be categorical, ordinal, continuous, or even multivariate (Imai and van Dyk, 2004). We follow the approach of Hirano and Imbens (2005) that defines the generalized propensity score and describing its properties for a continuous treatment.

We briefly describe the generalized propensity score (GPS) as developed by Hirano and Imbens (2005), closely following their presentation. We have N units, $i = 1, \ldots, N$, each assigned a treatment t that takes values in $\mathcal{T}$. For a binary treatment, $\mathcal{T} = \{0, 1\}$, but for our purposes we take $\mathcal{T} = [0, 1]$. Our goal is to estimate the average dose-response function (DRF)

$$\mu(t) = \mathbb{E}[Y(t)]. \tag{5}$$

In parallel with the binary treatment case, the utility of the GPS depends on several assumptions. First, we assume that treatment is weakly unconfounded conditional on observed covariates X, so that

$$Y(t) \perp T \mid X \quad \text{for all } t \in \mathcal{T}.$$

Here the term "weak" refers to the fact that we do not require joint independence of all potential outcome $\{Y(t)\}_{t \in \mathcal{T}}$. Let $r(t, x) = f_{T|X}(t \mid x)$ denote the conditional density of the treatment given the covariates. The GPS is then this conditional density evaluated at the observed values of T and X:

$$R = r(T, X). \tag{6}$$

In practice, the GPS can be estimated with any parametric or nonparametric method. A simple example is a linear regression of T on X: $T_i \mid X_i \sim N(\beta_0 + \beta_1^T X_i, \sigma^2)$. Recent studies have explored machine learning approaches like Super Learners (Kreif et al., 2015) and boosting (Zhu et al., 2015). However, as Kluve et al. (2012) point out, the primary concern with modeling the GPS is that it achieves adequate covariate balance; just as with the propensity score for a binary treatment, the specific modeling approach is less important than obtaining scores that yield balance on the observed covariates.

As Hirano and Imbens (2005) describe, a mechanical property of the GPS endow it with a balancing property, namely that within strata defined by $r(t, X)$, the probability that $T = t$ is independent of X: $X \perp \mathbf{T} = \mathbf{t} \mid r(t, X)$. This mechanical property does not require unconfoundedness. However, in conjunction with weak unconfoundedness, this property implies that treatment assignment is unconfounded conditional on the GPS:

$$Y(t) \perp T \mid r(t, X). \tag{7}$$

In other words, we need no longer condition on the (likely high-dimensional) covariate X to achieve unconfoundedness of the treatment; conditioning on the GPS is sufficient.

We can now use the GPS to obtain an unbiased estimate of the DRF $\mu(t)$. The first step is to estimate the conditional expectation of the outcome given the treatment and the GPS,

$$\beta(t, r) = \mathbb{E}[Y \mid T = t, R = r]. \tag{8}$$

This step requires us to specify a model for Y as a function of T and R. Hirano and Imbens (2005) proposed a linear regression of Y on T and R with quadratic terms:

$$Y_i \mid T_i, R_i \sim N(\alpha_0 + \alpha_1 T_i + \alpha_2 T_i^2 + \alpha_3 R_i + \alpha_4 R_i^2 + \alpha_5 T_i R_i, \sigma^2),$$

Of course, more flexible nonparametric models can also be used; recent studies have employed splines (Zhu et al., 2015) and Super Learners (Kreif et al., 2015). Whatever the model we choose, the estimation is done using the observed T_i and estimated $\widehat{R}_i = \widehat{r}(T_i, X_i)$ for each individual. As Hirano and Imbens (2005) emphasize, $\beta(t, r)$ does not have a causal interpretation.

The second step is to estimate the DRF itself, $\mu(t) = \mathbb{E}[\beta(t, r)]$, where the expectation is taken over the GPS evaluated at the treatment level of interest t and observed covariates X, $r(t, X)$. With an estimate of $\mu(t)$ in hand, we can calculate the causal effect of dose t relative to a baseline or control dose t', $\mathbb{E}[Y(t) - Y(t')]$, or the marginal treatment effect function $d\mu/dt$. In practice, we estimate $\mu(t)$ as

$$\widehat{\mu}(t) = \frac{1}{N} \sum_{i=1}^{N} \beta_i(t, \widehat{r}(t, X_i))$$

2.3.2 Multilevel treatments

The main methodological contribution of this study is combining use of the GPS for continuous treatments (Hirano and Imbens, 2005) with the framework that allows us to view an observational study as an approximation of a two-stage experiment (Hong and Raudenbush, 2006).

2.3.3 Implementation

3 Results

4 Discussion

4.1 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 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, X% of the children in our data belong to unique households, Y%

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 the size of XXX) 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 sample size in each community is roughly XX% (interquartile range XX% to XX%). 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.

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