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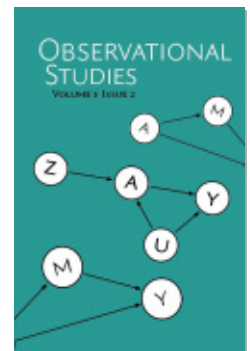
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The Regression Discontinuity Design Under Interference: A Local Randomization-based Approach

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1. Introduction

In recent years, the regression discontinuity (RD) design originally pioneered in educational psychology (Thistlethwaite and Campbell, 1960) has been rediscovered and has been the focus of much methodological development (see, e.g., Lee, 2008; Imbens and Lemieux, 2008). One particularly promising avenue of research, typified by Cattaneo, Frandsen, and Titiunik (2015), considers a local randomization-based model for the RD design. In the local randomization-based approach to the RD design, it is hypothesized that, within some finite *window* of an administrative threshold (e.g., a test score or age cutoff) that determines treatment assignment, subjects are “as-if” randomly assigned to treatment and control.

Despite recent methodological advances for the RD design, comparatively little attention has been given to the cases where there may be interference (Cox, 1958) between subjects (i.e., some subjects’ treatment status may causally affect other subjects’ outcomes). One notable exception is Cattaneo, Titiunik, and Vazquez-Bare (2016), which provides a software implementation of Rosenbaum (2007)’s interference-robust confidence intervals for Fisher (1935)-type causal inference with the RD design under a local randomization assumption. In this short note, we investigate the properties of the RD design for Neyman (1923)-type causal inference under a simple local randomization-based model when we allow for interference of arbitrary and unknown structure. We show that under a local randomization assumption, the difference-in-means estimator as applied to subjects in the window near the cutoff is unbiased for a regime-specific causal effect. This causal effect is equivalent to Hudgens and Halloran (2008)’s average direct effect for the subpopulation of subjects within the window near the threshold. For topics of study where interference is likely widespread

(e.g., evaluating effects of vaccines), our result may help to provide a formal basis for the use of the RD design.

2. Results

We first define our causal model and setting. Suppose we have a finite population U of N subjects indexed by $i = 1, \dots, N$. Define a treatment assignment vector, $\mathbf{z} = (z_1, \dots, z_N)'$, where $z_i \in \{0, 1\}$ specifies which treatment value (0 = control, 1 = treatment) that subject i receives. Suppose that associated with each subject i are 2^N fixed potential outcomes, $y_i(\mathbf{z})$, defined over all 2^N vectors \mathbf{z} such that $z_j \in \{0, 1\}, \forall j \in \{1, 2, \dots, N\}$. For example, $y_2((0, 1, 0, \dots, 0))$ would represent subject 2's potential outcome if she were treated, but no other subjects were treated. Note that this makes no assumptions about interference (or its absence): for each subject i , her outcome y_i is allowed to depend on her own or any combination of the other subjects' treatment statuses.

To proceed, we will make assumptions about the manner in which \mathbf{z} is assigned and how some potential outcomes are revealed. In particular, we will assume that \mathbf{Z} is a binary random vector of length N . The observed data then consist of a single realization from $(\mathbf{Y}, \mathbf{Z}, \mathbf{X})$, where it is assumed that the outcome vector $\mathbf{Y} = (y_1(\mathbf{Z}), y_2(\mathbf{Z}), \dots, y_N(\mathbf{Z}))$. (\mathbf{X} is discussed in the following paragraph; note that potential outcomes $y_i(\mathbf{z})$ are assumed not to depend on \mathbf{X} .) The key idea behind the local randomization-based approach is to assume that, for a range of subjects with values of a “running variable” (e.g., age in days) that are close to the administrative threshold (e.g., the minimum age for which treatment is administered), treatment is administered in an as-if random manner. We formalize this notion as follows.

Assume that associated with each subject i is a random variable X_i denoting the difference between subject i 's running variable and the threshold. We assume that whether or not a subject is treated depends solely on whether or not she is above the threshold: let $Z_i = 1$ (the subject is treated) if $X_i \geq 0$ (the subject is above the threshold), else let $Z_i = 0$ (the subject is in the control condition). In the event that we have noncompliance, let Z_i instead denote treatment eligibility and all effects should be interpreted as intention-to-treat effects. The joint distribution of $\mathbf{X} = (X_1, X_2, \dots, X_N)$ fully determines the joint distribution of \mathbf{Z} , hence knowledge of \mathbf{X} implies knowledge of \mathbf{Z} . Here we introduce our key modeling assumption, so as to be consistent with the local randomization approach. Let b be the bandwidth, or the maximum distance (e.g., number of days) from the threshold at which we would still consider a subject to be close to the threshold. Denote the window $W_0 = [-b, b]$, and define the conditional distribution function $F_{\mathbf{x}, W_0}$ as the empirical distribution function of X_i for all subjects such that $X_i \in W_0$. To simplify our definition of local randomization, without loss of generality, assume that $\Pr(X_i = X_j) = 0, \forall i, j : X_i, X_j \in W_0$, so that no two observed age values within the window are exactly identical. Our local randomization assumption requires that $F_{\mathbf{x}, W_0}(t) = F_0(t), \forall \mathbf{x} \in \text{Supp}(\mathbf{X})$ and $\forall t; \Pr(X_i \in W_0) \in \{0, 1\}, \forall i \in \{1, \dots, N\}$, $\Pr(\mathbf{X} = \mathbf{x}) = \frac{1}{N_0!}, \forall \mathbf{x} \in \text{Supp}(\mathbf{X})$, where $N_0 = \sum_{i=1}^N \mathbf{I}(X_i \in W_0)$: implying that all permutations of the running variable values (and therefore treatment values) within the window W_0 are equiprobable. Further assume that $0 < \sum_{i: X_i \in W_0} Z_i < N_0$, so that at least one subject within the window is treated, and at least one subject within the window is in the control group.

We now define our causal estimands. Denote the individual average potential outcome under treatment z for all subjects $i : X_i \in W_0$,

$$\bar{Y}_i(z) = \frac{\sum_{\mathbf{z} \in \text{Supp}(\mathbf{Z})} y_i(\mathbf{z}) \mathbf{I}(\mathbf{z}_i = z)}{\sum_{\mathbf{z} \in \text{Supp}(\mathbf{Z})} \mathbf{I}(\mathbf{z}_i = z)}.$$

Note that this estimand is regime specific: it generally depends on the joint distribution of \mathbf{Z} . Depending on the treatment allocation scheme, then the individual average potential outcome would change. (E.g., when 90% of subjects in the population are treated, then both $\bar{Y}_i(0)$ and $\bar{Y}_i(1)$ might be different from when 10% of subjects in the population are treated.) While the treatment allocation scheme for subjects outside of W_0 is left completely unspecified, the allocation scheme for these subjects nevertheless contributes to the definition of each $\bar{Y}_i(z)$. Individual average potential outcomes marginalize over the treatment assignments for all units, not simply those within the window near the threshold.

Then our primary target is the average direct effect (Hudgens and Halloran, 2008) of treatment for subjects who are close to the threshold:

$$\tau = N_0^{-1} \sum_{i: X_i \in W_0} \bar{Y}_i(1) - \bar{Y}_i(0).$$

Or, put simply, our target is the difference between the average outcome we would expect to see in treated subjects (across all subjects within the window and across all randomizations) and the average outcome we would expect to see in control subjects (across all subjects within the window and across all randomizations). When there is interference between subjects, this estimand is conditional not only on the subjects being close to the threshold in the running variable, but also on the exact way in which the treatment is assigned to all subjects in the finite population U .

We now show that τ is estimable and that the difference-in-means estimator as applied to all subjects $i : X_i \in W_0$ is unbiased for the average direct effect τ among these subjects. Formally, the difference-in-means estimator for subjects local to the threshold,

$$\hat{\tau} = \frac{\sum_{i: X_i \in W_0} Y_i Z_i}{\sum_{i: X_i \in W_0} Z_i} - \frac{\sum_{i: X_i \in W_0} Y_i (1 - Z_i)}{\sum_{i: X_i \in W_0} (1 - Z_i)}.$$

Then, under the assumption of local randomization, the difference-in-means estimator as applied to subjects local to the threshold is unbiased for the average direct effect for subjects near the threshold:

$$\mathbb{E}[\hat{\tau}] = \frac{1}{N_0} \left[\sum_{i: X_i \in W_0} \bar{Y}_i(1) - \sum_{i: X_i \in W_0} \bar{Y}_i(0) \right],$$

where the result trivially follows from linearity of expectations.

Inference on the average direct effect for subjects near the threshold requires considerably more structure. As a sufficient condition for root- n consistency, asymptotic normality, and the existence of conservative variance estimators, asymptotics based on a growing number of strata of bounded size suffice, given (i) partial interference (i.e., subjects do not

interfere with subjects outside of their stratum, see Sobel 2006) and (ii) suitable regularity conditions on the values of potential outcomes, $y_i(\mathbf{z})$, and the within-stratum running variable distributions, $F_0(t)$. An alternative sufficient condition, without requiring a large number of independent strata, is given by stratified interference and suitable regularity conditions. Liu and Hudgens (2014) and Aronow and Samii (2016) provide details. One further alternative, as suggested by Cattaneo, Frandsen, and Titiunik (2015) and Cattaneo, Titiunik, and Vazquez-Bare (2016), is to impose more structure on causal effects and use a variant of Fisher (1935)’s exact test, which may be preferable particularly when N_0 is small.

3. Discussion

Our results have illustrated that the RD design can yield credible causal inferences in the context of studies with interference, though — as is usually the case under interference — the estimated causal effect may have a nuanced interpretation. We note here three possible avenues for future methodological work in the context of interference, including (i) exploring results analogous to ours in the setting of the standard econometric limit-based RD design (Imbens and Lemieux, 2008); (ii) derivation of the properties of “fuzzy” instrumental variables-type estimators that account for imperfect compliance (Angrist and Lavy, 1999; Angrist and Pischke, 2009); (iii) exploiting natural variation in local treatment saturation to estimate indirect, total and overall effects as in Hudgens and Halloran (2008).

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