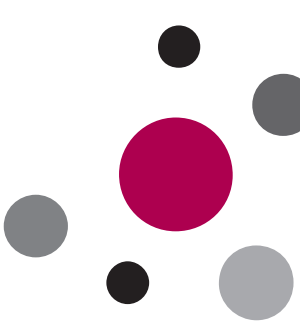

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Adam Glynn
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Generalized Nonlinear Difference-in-Difference-in-Differences*

Adam N. Glynn† Nahomi Ichino‡

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Abstract

Difference-in-difference-in-differences (DiDiD) allow for the correction of unmeasured confounding and function as a robustness check for difference-in-differences (DiD) techniques. However, this technique is not scale invariant and requires that the outcome variable be measured on units for which the treatment could have had no effect in either the pre-treatment or post-treatment periods. Athey and Imbens (2006) provides a scale invariant, nonlinear DiD approach known as Changes-in-Changes (CiC). Sofer et al. (2016) extends CiC by showing that pre-treatment outcome measures are a special case of placebo (negative) outcomes and proposes a generalization of CiC called Negative Outcome Control (NOC). We develop a generalized nonlinear DiDiD approach we call NOCNOC that can be used either in the traditional DiDiD setting or when a placebo outcome is available in the pre and post-treatment data. We show that NOCNOC can correct for bias in DiDiD, CiC, and NOC. We apply this method to a study of whether exposure to candidate debates affected Nepalese citizens' sense of political efficacy.

1 Introduction

Difference-in-difference-in-differences (DiDiD) allow for the correction of unmeasured confounding. However, this technique is not scale invariant and requires the outcome variable to be measured on "treated" observations for which the treatment could have had no effect in either the pre-treatment or post-treatment periods. Often, these observations are individuals who would have been ineligible for the treatment, for example, due to a minimum age requirement or maximum age limit.

In this paper, we discuss a generalized nonlinear DiDiD approach. This builds on Sofer, Richardson, Colicino, Schwartz & Tchetgen (2016), which showed how the changes-in-changes model (CiC) of non-linear DiD (Athey & Imbens 2006) could be generalized to allow the use of placebo outcomes in lieu of pre-treatment measurements of the outcome. The Sofer et al. (2016) approach, which they call negative outcome control (NOC), relies on placebo outcomes – outcomes that the treatment should not affect and for which the confounding should equal the confounding of primary outcomes on the quantile scale. For example, Sofer et al. (2016) uses body mass index (BMI) as a placebo outcome to help estimate the effect of black carbon exposure due to air pollution on fibrinogen (blood inflammation). There is likely unmeasured confounding of the relationship between black carbon and fibrinogen because those living in areas exposed to high levels of black carbon are also likely to have other risk factors for fibrinogen. BMI may serve as a useful placebo outcome because it is unlikely to be directly affected by black carbon, but it is likely to share many of the same confounding factors as fibrinogen.

We extend the Athey & Imbens (2006) and Sofer et al. (2016) approaches to the DiDiD context and demonstrate this in detail using placebo (negative) outcomes. We develop a method to relax the Sofer et al. (2016) assumption of quantile-quantile primary-placebo equi-confounding by using pre- and post-treatment data. This approach, which we call the NOCNOC, is non-linear, scale invariant, and can be used in traditional DiDiD settings or with placebo outcomes. We show using simulations that NOCNOC can correct for bias in traditional linear DiDiD, CiC, and NOC. We also apply this method to analyze whether exposure to candidate debates affected the political efficacy of Nepalese citizens. We use political knowledge as a placebo outcome, despite the fact that first, exposure to the debates may have had some small effect on knowledge, and second, it is difficult to determine *ex ante* whether cross-sectional confounding would be equal for political efficacy and knowledge. Using pre-treatment measures of both political efficacy and knowledge with our NOCNOC estimator, we demonstrate that exposure to candidate debates likely had no effect on political efficacy, even though DiD analysis with placebo tests in conventional practice suggested a positive effect.

2 Review of Changes-in-Changes (CiC) and Negative Outcome Control (NOC)

Define the outcome variable Y_{at} for action/treatment group $a = \{0, 1\}$ (control, treatment) at time $t = \{0, 1\}$ (pre-treatment, post-treatment). Then the linear DiD estimator can be written as the following:

$$\begin{aligned}&(\widehat{E}[Y_{11}] - \widehat{E}[Y_{01}]) - (\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]) \\ &= \widehat{E}[Y_{11}] - (\widehat{E}[Y_{01}] + (\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]))\end{aligned} \tag{1}$$

This is an estimator for the average treatment effect for the treated (ATT) in the post-treatment period, which can be defined in terms of the missing potential outcomes $Y_{11}(0)$, the outcome that would have occured for the treated units in the post-treatment period if they had been assigned control. With this notation, the ATT is:

$$E[Y_{11}] - E[Y_{11}(0)] \tag{2}$$

Note that $\widehat{E}[Y_{11}]$ in (1) is simply the mean outcome among the treated units in the post-treatment period and is a plug-in estimator for $E[Y_{11}]$ in (2). The difficult task is the estimation of the second term in (2), the mean of the missing potential outcomes $E[Y_{11}(0)]$. The linear DiD approach estimates this quantity with $\widehat{E}[Y_{01}]+(\widehat{E}[Y_{10}]-\widehat{E}[Y_{00}])$ from (1), where $\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]$ is the correction for the confounding. Note also that although we do not include notation for covariates in this paper, nonparametric use of covariates could be easily incorporated by conditioning the expectations on values of the covariates.

2.1 Changes-in-Changes (CiC)

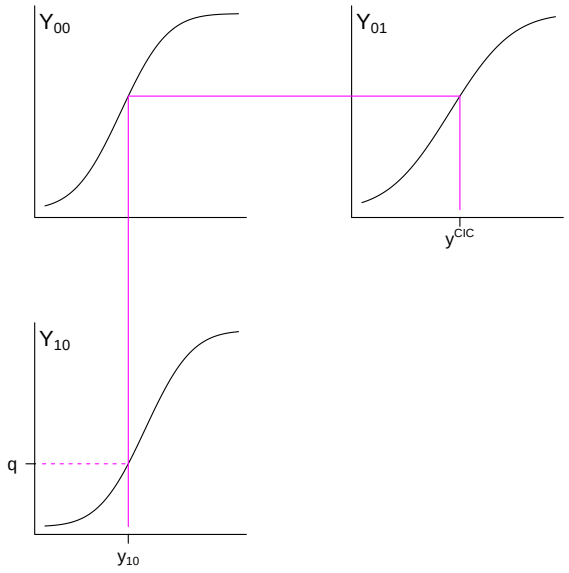
Athey & Imbens (2006)'s CiC procedure is a nonlinear DiD approach to the correction for unmeasured confounding. It generalizes the linear DiD in the following way:

$$\widehat{E}[Y_{11}] - \frac{1}{k_{10}} \sum_{i=1}^{k_{10}} \hat{F}^{-1}_{Y_{01}}(\hat{F}_{Y_{00}}(Y_{10,i})) \tag{3}$$

where k_{10} is the number of treated units in the pre-treatment period, $\hat{F}_{Y_{00}}$ represents a consistent estimator of the CDF from the control units in the pre-treatment period, and $\hat{F}^{-1}_{Y_{01}}$ represents a consistent esimator of the inverse CDF from the control units in the post-treatment period. Note that the second term from (3) is analogous to the second

term in the linear DiD estimator in (1) and estimates the second term of the ATT in (2). Note also that although we do not include notation for covariates, nonparametric use of covariates could be easily incorporated by conditioning the CDFs and inverse CDFs on values of the covariates.

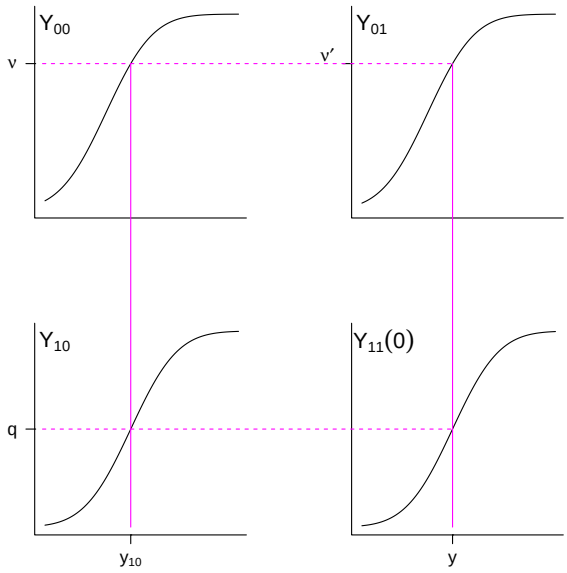
Figure 1: CiC procedure for imputing $Y(0)$ for quantile q ($F^{-1}_{Y_{11}(0)}(q)$)



Consider what the second term of (3) is doing for a particular quantile q. For example, suppose we want to impute the missing potential outcome for $q = .4$. The associated CiC procedure is presented in Figure 1. We take the y value associated with the .4 quantile among the treated units in the pre-treatment period (y_{10} in the figure). We calculate what quantile that value would take among the control units in the pre-treatment period (roughly .5 in the figure). We then assume the imputed value should be at the same quantile among the controls in the post-treatment period as in the pre-treatment period (indicated by the horizontal line between the Y_{00} and Y_{01} in the figure). Finally, we calculate the y value associated with the .5 quantile among the controls in the post-treatment period (the y^{CiC} in the figure).

Intuitively, the CiC procedure uses the pre-treatment period to measure confounding on the quantile scale by showing how far the quantiles move from the treatment group to the control group (.4 to .5 in the figure). This is then used to impute the missing potential outcome in the post-treatment period. Athey & Imbens (2006) provides a set of assumptions under which (3) is consistent for ATT, and Sofer et al. (2016) weakens these assumptions. Here we provide an illustrative reduced form of the Sofer et al.

Figure 2: CiC quantile-quantile pre-post equi-confounding assumption for quantile q



(2016) assumptions extracted from their proof.[1] The underlying assumption is that the confounding in the pre-treatment period equals the confounding in the post-treatment period on the quantile scale (i.e., time-invariant confounding). For a particular quantile q, this assumption is depicted in Figure 2, where the move from quantile q among the treated units in the pre-treatment period to quantile ν among the control units in the pre-treatment period is assumed to mirror the move from quantile q in the missing potential outcomes to quantile ν' among the control units in the post-treatment period. However, for ATT, this assumption only needs to hold on average across the quantiles.

2.2 Negative Outcome Control (NOC)

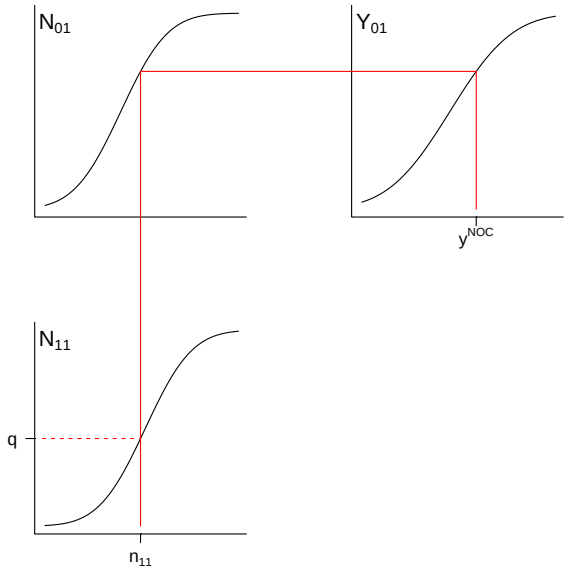
Without covariates, Sofer et al. (2016)'s NOC procedure is very similar to the CiC procedure. The pre-treatment outcomes, Y_{10} and Y_{00}, are replaced with post-treatment placebo (negative) outcomes, N_{11} and N_{01}. Sofer et al. (2016)'s application, for example, is cross-sectional in the post-treatment period: fibrinogen (the primary outcome Y) and BMI (the placebo/negative outcome N) are both measured after exposure to black carbon. Hence, the NOC procedure is the following:

[1]The more expansive assumptions of Athey & Imbens (2006) and Sofer et al. (2016) provide more detail in terms of data generating processes that would satisfy these reduced form assumptions.

$$\widehat{E}[Y_{11}] - \frac{1}{k_{11}} \sum_{i=1}^{k_{11}} \hat{F}_{Y_{01}}^{-1}(\hat{F}_{N_{01}}(N_{11,i})) \tag{4}$$

where k_{11} is the number of treated units in the post-treatment period, $\hat{F}_{N_{01}}$ represents a consistent estimator of the CDF for the placebo outcome from the control units in the post-treatment period, and $\hat{F}_{Y_{01}}^{-1}$ represents a consistent estimator of the inverse CDF for the primary outcome for the control units in the post-treatment period. Note that the second term in (4) is analogous to the second term in (3).

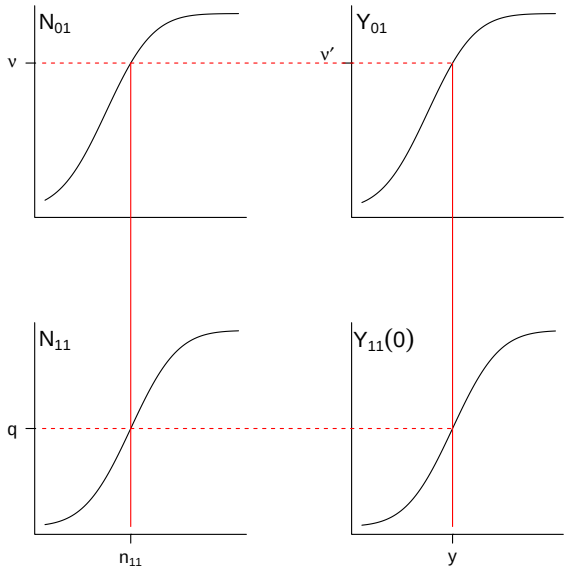
Figure 3: NOC procedure for imputing $Y(0)$ for quantile q ($F_{Y_{11}(0)}^{-1}(q)$)



Again, we can build intuition about the NOC by considering what the second term of (4) is doing for a particular quantile q. For example, suppose we want to impute the missing potential outcome for $q = .4$. The associated NOC procedure is presented in Figure 3. We take the y value associated with the .4 quantile for the placebo outcome among the treated units in the post-treatment period (n_{11} in the figure). We calculate what quantile that placebo outcome value would take among the control units in the post-treatment period (roughly .6 in the figure). We then assume the imputed value should be at the same quantile among the controls for the primary outcome in the post-treatment period as among the placebo outcome in the same post-treatment period (horizontal line between the N_{01} and Y_{01} in the figure). Finally, we calculate the y value associated with the .6 quantile among the controls in the post-treatment period (the y_{NOC} in the figure).

The formal statement of this assumption is stated below (and as before, the support of the control units must contain the support of the treated units):

Figure 4: NOC quantile-quantile placebo-primary equi-confounding assumption for quantile q



Intuitively, the placebo outcome plots measure the confounding on the quantile scale by showing how far the quantiles move from the treatment group to the control group (.4 to .6 in the figure). This makes sense as a measure of confounding because the treatment should not affect a placebo outcome. This .6 is then used to impute the missing potential outcome in the post-treatment period. Again, the underlying assumption for the NOC procedure is a quantile-quantile equi-confounding assumption, except that now the equality of confounding is assumed between the placebo outcome and the primary outcome. In the CiC procedure, the equal confounding is assumed across time. This NOC assumption is depicted for a particular quantile q in Figure 4. Finally, as before we only need this assumption to hold on average when estimating ATT.

3 Generalizing the DiDiD Approach

If we allow N to denote either a placebo outcome as before, or alternatively measurements of Y on units for which the treatment should have no effect (e.g., those that miss

an eligibility cutoff), the standard linear DiDiD estimator can be written as the following:

$$
\begin{aligned}
&(\widehat{E}[Y_{11}] - \widehat{E}[Y_{01}]) - (\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]) - \{(\widehat{E}[N_{11}] - \widehat{E}[N_{01}]) - (\widehat{E}[N_{10}] - \widehat{E}[N_{00}])\} \\
&= (\widehat{E}[Y_{11}] - \widehat{E}[Y_{01}]) - (\widehat{E}[N_{11}] - \widehat{E}[N_{01}]) - \{(\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]) - (\widehat{E}[N_{10}] - \widehat{E}[N_{00}])\} \qquad (5) \\
&= \widehat{E}[Y_{11}] - \Big[\widehat{E}[Y_{01}] + (\widehat{E}[N_{11}] - \widehat{E}[N_{01}]) - \{(\widehat{E}[N_{10}] - \widehat{E}[N_{00}]) - (\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}])\}\Big] \qquad (6)
\end{aligned}
$$

Again, this is an estimator for ATT in the post-treatment period ($E[Y_{11}] - E[Y_{11}(0)]$). If we write the estimator as in (5), then the first half of the estimator ($(\widehat{E}[Y_{11}] - \widehat{E}[Y_{01}]) - (\widehat{E}[N_{11}] - \widehat{E}[N_{01}])$) can be seen as a linear NOC approach in the post-treatment period, while the second half ($\{(\widehat{E}[Y_{10}] - \widehat{E}[Y_{00}]) - (\widehat{E}[N_{10}] - \widehat{E}[N_{00}])\}$) can be seen as a linear NOC approach in the pre-treatment period. Alternatively, in (6) $\widehat{E}[Y_{11}]$ is a plug-in estimator for the first term of ATT and the linear DiDiD approach estimates the second term of ATT with everything in the large square brackets from (6). One thing this notation makes clear is that (6) has the potential to be quite biased when N is measured on a different scale than Y, which is likely to be the case when N is a placebo outcome.

Given the apparent problems with the linear DiDiD estimator, we introduce a non-linear DiDiD estimator for ATT that we call the NOCNOC estimator:

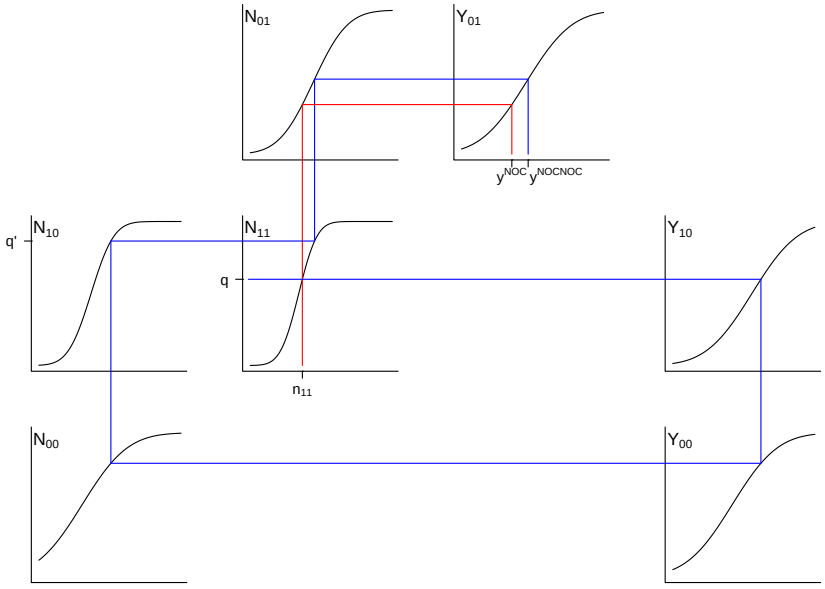
$$
\frac{1}{k_{11}} \sum_{i=1}^{k_{11}} Y_{11,i} - \frac{1}{k_{10}} \sum_{i=1}^{k_{10}} \hat{F}_{Y_{01}}^{-1}(\hat{F}_{N_{01}}(\hat{F}_{N_{11}}^{-1}(\hat{F}_{N_{10}}(\hat{F}_{N_{00}}^{-1}(\hat{F}_{Y_{00}}(Y_{10,i})))))) \qquad (7)
$$

Note that this estimator has a similar form to (6), and hence the fundamental idea behind the NOCNOC estimator is to correct inconsistency in the NOC estimator in the post-treatment period, with estimates from the NOC estimator in the pre-treatment period. In this sense, NOCNOC will work well when the differential in confounding between the primary and placebo outcome is time-invariant.

NOCNOC is depicted in Figure 5. Suppose we want to estimate $Y_{11}(0)$ for a particular quantile q. This is accomplished by starting with the quantile q pre-treatment primary outcome in the treated group (Y_{10}) and asking what quantile q' among the pre-treatment values of the placebo in the treated group (N_{10}) would have produced that Y_{10}. This process can be seen by following the blue path in the lower part of Figure 5. Then the estimated q' is used instead of q to start the NOC process in the post-treatment period. This process can be seen by the blue path in the upper part of Figure 5. For comparative purposes, NOC process is represented by the red path in the upper part of of Figure 5. The fundamental difference between the procedures is that NOC starts with quantile q in

the post-treatment placebo distribution (N_{11}) and NOCNOC uses quantile q' in the post-treatment placebo distribution (N_{11}), having estimated this quantile in the pre-treatment period.

Figure 5: NOCNOC estimator procedure for imputing $Y(0)$ for quantile q ($F^{-1}_{Y_{11}(0)}(q)$)



The reduced form assumptions of the NOCNOC estimator are presented in Assumptions 1a and 1b below:

Assumption 1a.

$$F_{N_{11}}(F^{-1}_{N_{01}}(F_{Y_{01}}(F^{-1}_{Y_{11}(0)}(q)))) = F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(F^{-1}_{Y_{10}}(q)))), q \in [0,1]$$

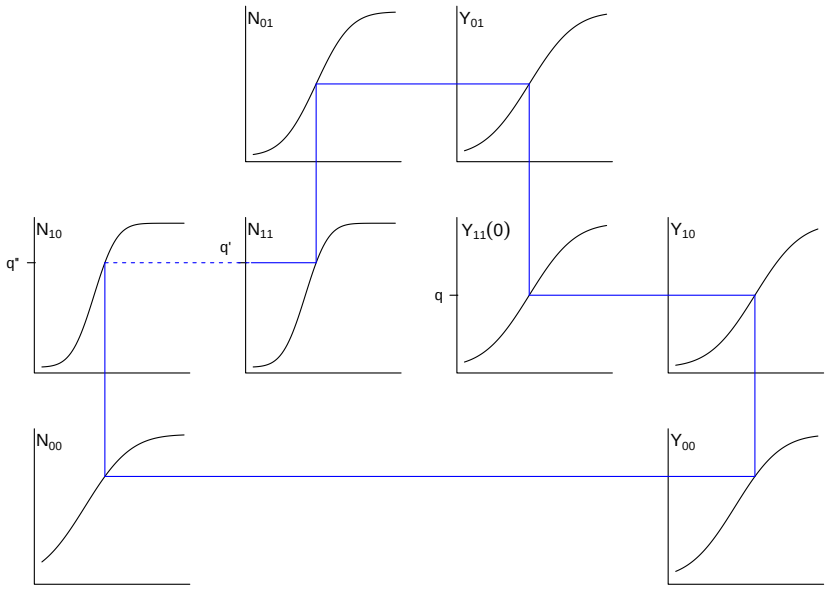
Assumption 1b.

$$\begin{aligned} \textit{if } 0 < f_{Y_{10}}(y_{10}),\ \textit{then } & 0 < F_{Y_{00}}(y_{10}) < 1, \\ & 0 < F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(y_{10}))) < 1,\ \textit{and} \\ & 0 < F_{N_{01}}(F^{-1}_{N_{11}}(F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(y_{10}))))) < 1 \end{aligned}$$

Assumption 1a is visualized in Figure 6 for a particular value of q. The assumption holds in this case, with the output from the left hand side of Assumption 1a depicted on the figure as q', the output from the right hand side of Assumption 1a depicted on the figure as q'', and $q' = q''$. The assumption means that the confounding generated

on the quantile scale is equal for the NOC process in the post-treatment period and the pre-treatment period.

Figure 6: Placebo outcome NOCNOC assumption for imputing $Y(0)$ for quantile q $(F^{-1}_{Y_{11}(0)}(q))$



Assumption 1b states the support conditions for the NOCNOC estimator, and these ensure that the CDFs never output negative or positive infinity. For the NOCNOC, the support for the treated units in the pre-treatment period must be contained in the support of the controls in the pre-treatment period. In addition, quantiles implied by the treated units in the pre-treatment period must also be in the support of the placebo outcomes in the manner presented.

Alternatively, Assumption 1a can be microfounded in terms of unmeasured confounders as in the Sofer et al. (2016) proof:

Assumption 2a.

$$N_{at}(a) = N_{at} \; for \; a = 0, 1$$
$$Y_{at}(a) = Y_{at} \; if \; A = a$$

Assumption 2b.

$$A_t \perp\!\!\!\perp Y_t(0)|U_t \text{ for } t = 0, 1$$
$$A_t \perp\!\!\!\perp N_t|W_t \text{ for } t = 0, 1$$

Assumption 2c.

$$Y_{at}(0) = h_{yt}(U_{at}) \text{ where } h_{yt}(U_{at}) \text{ is monotone increasing}$$
$$N_{at} = h_{nt}(W_{at}) \text{ where } h_{nt}(W_{at}) \text{ is monotone increasing}$$

Assumption 2d.

$$F_{W_{11}}(F^{-1}_{W_{01}}(F_{U_{01}}(F^{-1}_{U_{11}(0)}(q)))) = F_{W_{10}}(F^{-1}_{W_{00}}(F_{U_{00}}(F^{-1}_{U_{10}}(q)))), q \in [0, 1]$$

If Assumptions 2a, 2b, 2c, and 2d hold, then the proof in Sofer et al. (2016) implies that Assumption 1a holds. These assumptions also illustrate our approach to simulation in the next section. The following theorem establishes the consistency of the NOCNOC estimator. The proof is in Appendix A.

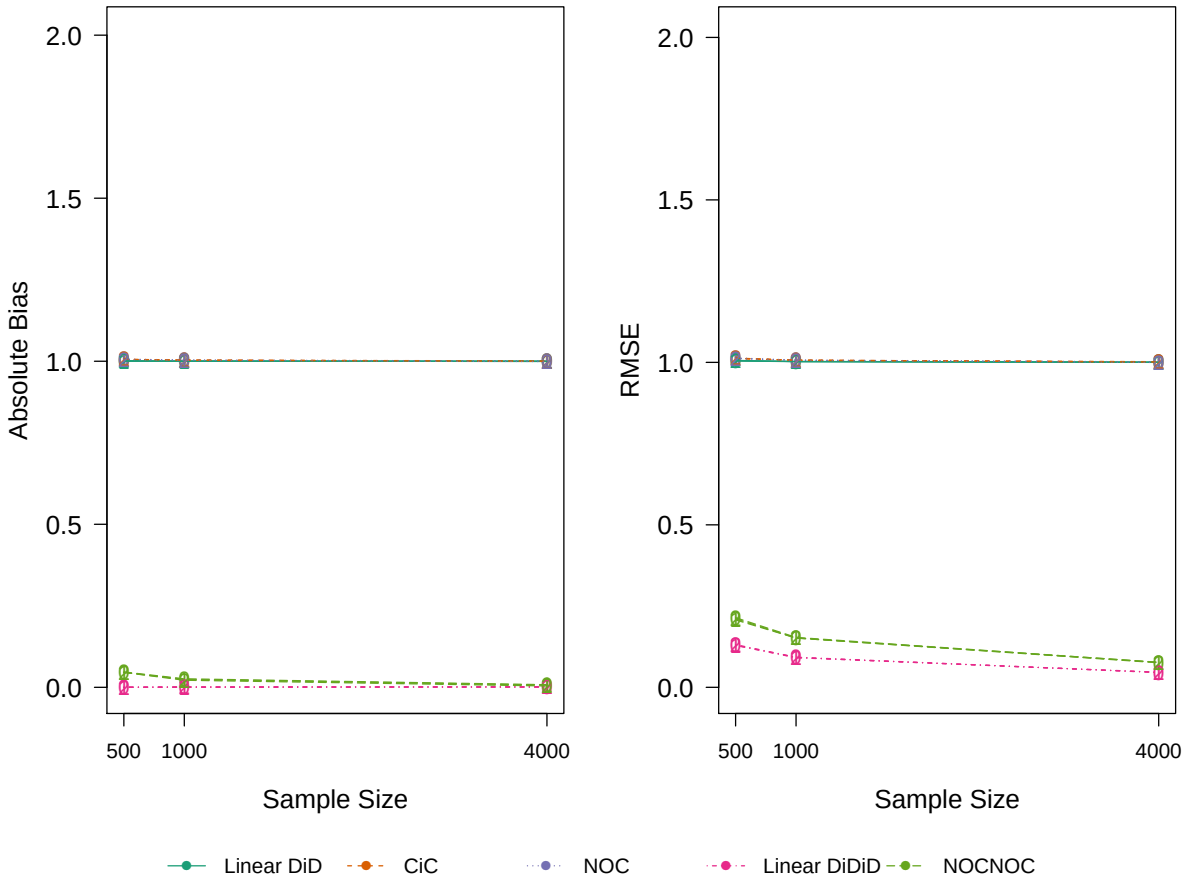
Theorem 1. *Under Assumptions 1a and 1b, the NOCNOC estimator is consistent for ATT.*

4 Simulation Study

In order to assess the performance of the NOCNOC estimator, we ran a number of simulations where pre- and post-treatment data were available for both a primary outcome and a placebo outcome. In the first set of simulations, we generated data consistent with the linear DiDiD model. These results are presented in Figure 7, and we see that as expected the NOCNOC does well in comparison to the linear DiDiD, although the linear approach is more efficient. In the second set of simulations, we generated data consistent with the CiC model. These results are presented in Figure 8, and we see that CiC does well, while NOC, and the linear DiDiD perform quite poorly for some specifications. NOCNOC does well but the CiC approach is more efficient. In the third set of simulations, we generated data consistent with the NOC model. These results are presented in Figure 9, and we see that NOC does well, while CiC and the linear DiDiD perform poorly. Again, NOCNOC does well but the NOC approach is slightly

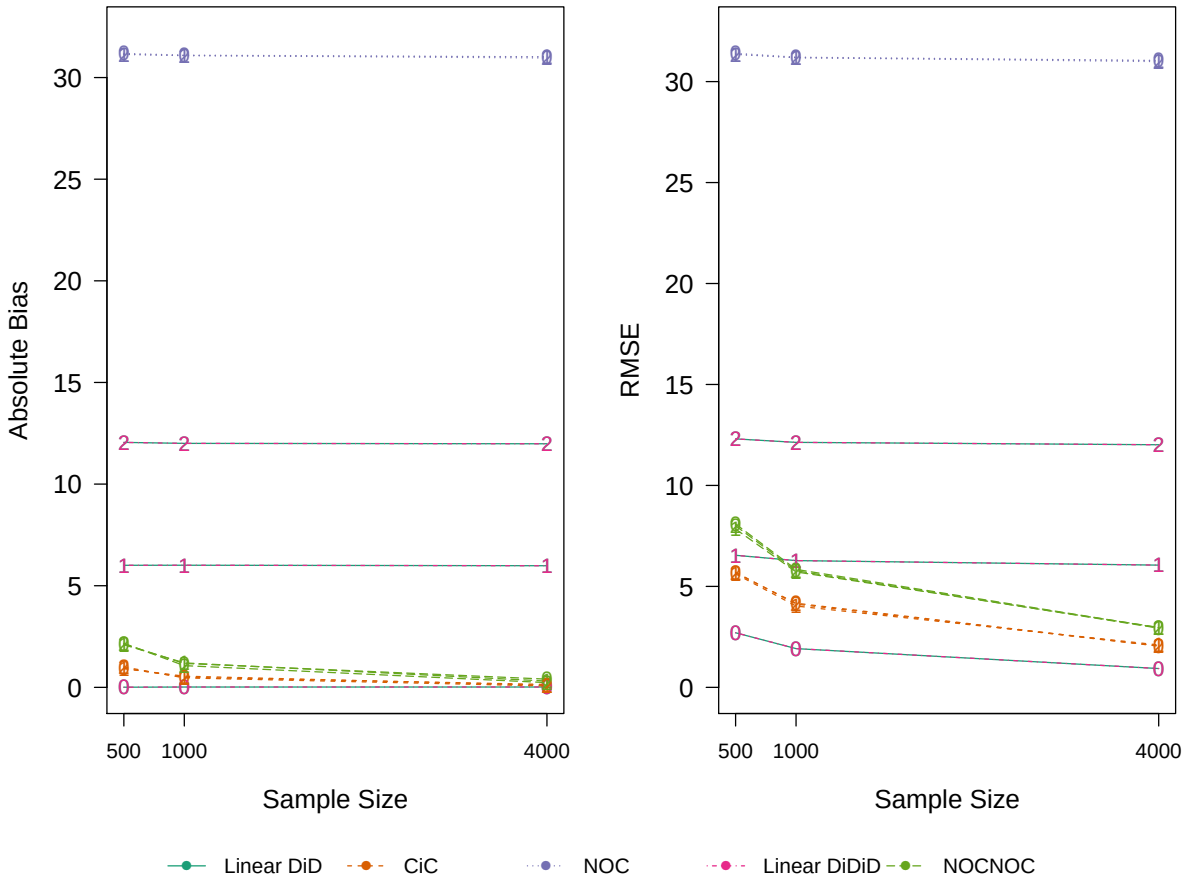
more efficient. Finally, we present simulations with generated data consistent with only the NOCNOC model. These results are presented in Figure 10, and only the NOCNOC estimator performs well.

Figure 7: Simulation results for data consistent with a linear DiDiD model.



The results of these simulation studies provide some guidance as to the potential choice of estimator. The main finding is that for bias, the NOCNOC estimator nearly weakly dominates all other estimators over all simulations at large sample sizes. In particular, all the other estimators have catastrophic failures for at least some of the simulations, while NOCNOC never does. Furthermore, the NOCNOC has reasonable root mean squared error, so not a great amount of efficiency is lost in using the most robust model.

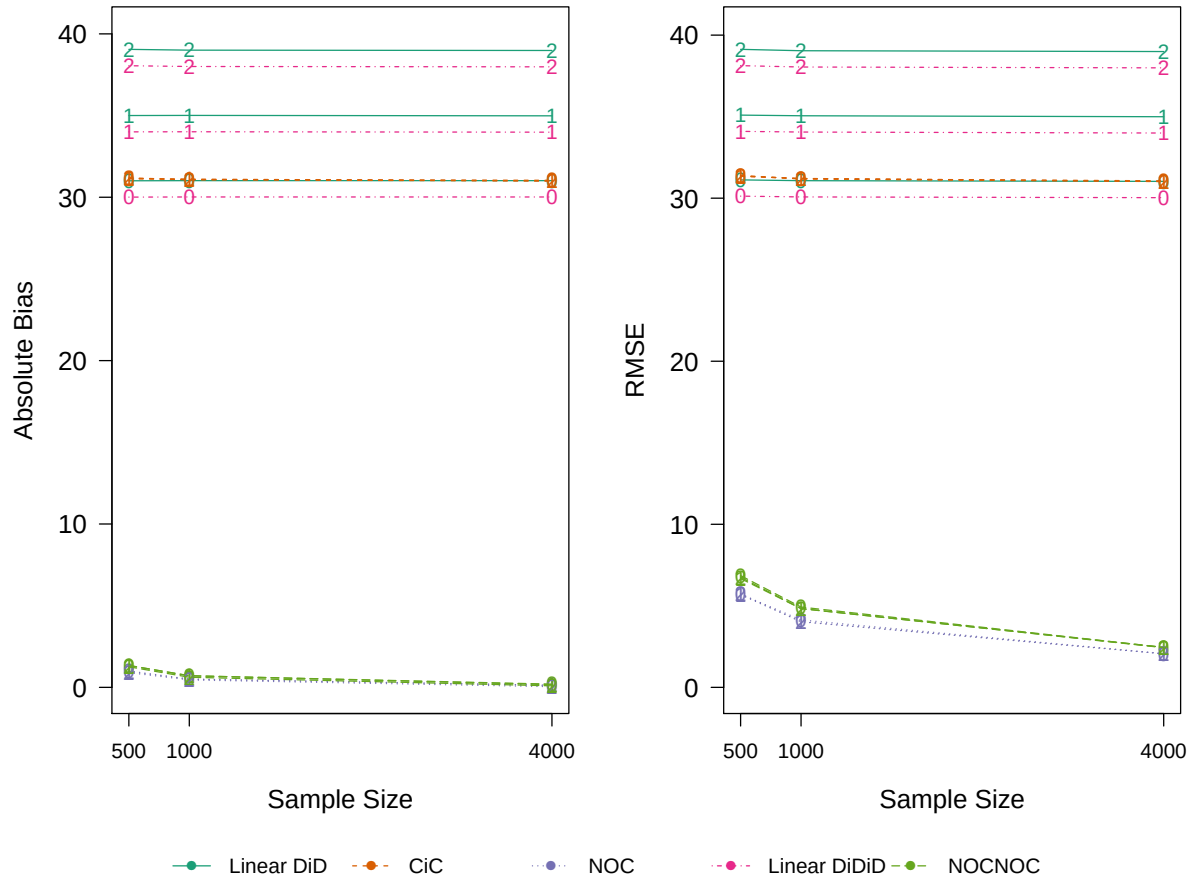
Figure 8: Simulation results for data consistent with a CiC model.



5 Debate Exposure and Political Efficacy in Nepal

Nepal is a country of approximately 29 million that experienced civil war from 1996 to 2006 and was a monarchy until 2008. In December 2017, Nepal held local and national elections for the first time under a new constitution that established it as a federal, multi-ethnic republic. As part of an effort to strengthen the connections between citizens and their representatives and to encourage issue-oriented rather than personality-centered politics, the Samriddhi Foundation, a Nepalese civil society organization, hosted televised debates among candidates for the House of Representatives (the directly-elected lower house of parliament) for three single-member constituencies within Kanchanpur, Jhapa, and Sunsari districts in November 2017. A community radio station in each of these rural districts invited 1000 randomly-selected citizens to a public venue to view a screening of the recorded candidate debate for their area and/or participate in small-group discussions

Figure 9: Simulation results for data consistent with an NOC model.

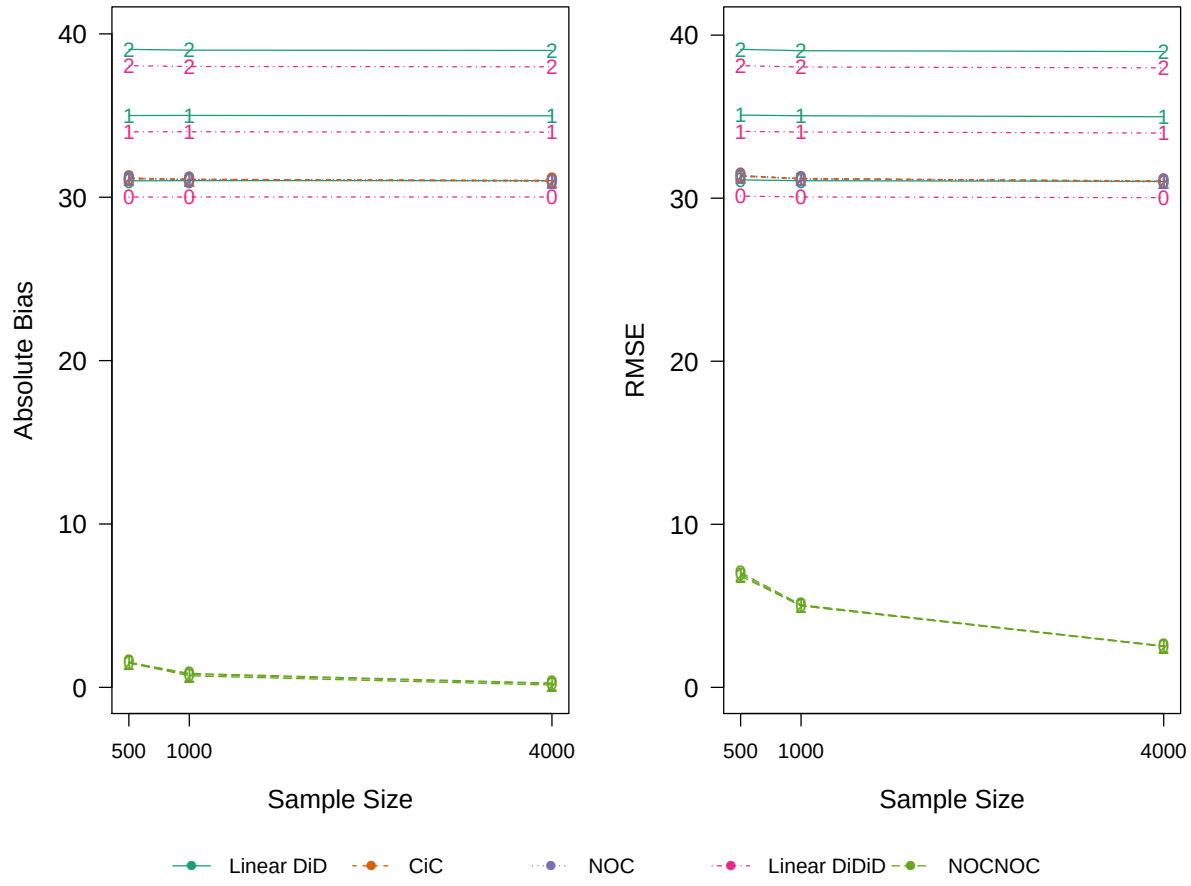


about the candidates and issues.[2] Recent randomized studies in Ghana, Sierra Leone, and Uganda, where candidate debates are also relatively novel, have found that exposure to candidate debates (sometimes followed by community discussion) increased voters' knowledge about the candidates and their policies and affected how they voted in some contexts (Bidwell, Casey & Glennerster 2016, Brierley, Kramon & Ofosu 2018, Platas & Raffler 2017).

To illustrate our method for DiDiD with placebo outcomes, we assess whether exposure to the candidate debates ahead of these historic elections affected a related but different outcome – citizens' sense of political efficacy. Political efficacy – the sense that one can influence politics and government (external) and that one can understand politics and government (internal) – is associated with political participation (Campbell, Gurin

[2]The debates were edited for length and to even out the screen time across candidates.

Figure 10: Simulation results for data consistent with a NOCNOC model.



& Miller 1954, Almond & Verba 1963, Rosenstone & Hansen 1993, Verba, Schlozman & Brady 1995). Low efficacy could create the danger that citizens will fail participate in politics and hold their politicians accountable, leading politicians to learn that they can fail to serve voters and instead serve private interests with little electoral consequence. But involvement in political activities itself could enhance citizens' sense of efficacy (Finkel 1985, Valentino, Gregorowicz & Groenendyk 2009), and we consider the impact of exposure to these debates.

This analysis uses data for 223 respondents who attended one of the events and were randomized into the debate screening condition, along with 510 respondents who signed up for but did not attend the events, for whom we have measures of all items used to construct primary and placebo outcomes both pre- and post-treatment. These respondents were initially interviewed at their homes in November 2017; signed up for an

event to be held between November 21 and 27, 2017; and were re-interviewed mostly at their homes in January/February 2018. Excluded are those attendees who were assigned to other treatment arms that included small-group discussions as part of the larger study and those participants who had signed up for dates for which we had to cancel the events. We use pre- and post-treatment data collected on the same units, but this is not necessary for our methods. It is only necessary that the primary and placebo outcomes are measured on the same units within each cross-section.

A first tack at this question would compare the efficacy of attendees and non-attendees of these events after the treatment, and we find that the difference-in-means is 0.0733 (0.0247).[3] Although all respondents signed up for an event date that was convenient for them, attendance was not randomized and we expect those who attended these events to differ from those who do not in important respects. Those who have less interest in politics or have lower efficacy and think that the event will have smaller benefits for them are less likely to take the time to travel and participate in these events. Indeed, differences between attendees and non-attendees in efficacy at baseline provide some evidence for this confounding problem. At baseline, the mean efficacy index were 0.5669 for attendees and 0.5345 for non-attendees, respectively (Table 1). This gives us a difference-in-difference estimate of 0.0408 (0.0231), an apparent effect significant at the 90% level. Furthermore, even if we use the more robust Athey & Imbens (2006)'s CiC model we get an ATT estimate of 0.0448 (0.0245, standard error from 1000 bootstrap samples).[4] It appears from these estimates that debates have an effect on efficacy.

However, even the CiC estimate is only valid if the quantile-quantile pre-post equiconfounding assumption holds on average. Fortunately, we have a placebo outcome that can be used to assess this assumption. This is an index of knowledge of aspects of politics and government that were *not* discussed in the debate, which is likely to suffer from confounding similar to political efficacy. Respondents were asked how many levels of government Nepal has under the new constitution, how many legislative bodies Nepal has at the national level under the new constitution, and asked to list as many as they

[3]Our political efficacy index is a measure of both external and internal efficacy. External efficacy, the sense that one can influence politics and government, is measured on a 5-point scale from strongly agree to strongly disagree with the statement "I feel I can influence political decisions that affect my life." Internal political efficacy, which is the sense that one can understand political affairs, is measured in two ways. The first is on a 5-point scale from strongly agree to strongly disagree with the statement "I feel I am as well-informed about politics and government as most people." The second is whether the respondent agrees more with the statement "Politics is complicated and I usually do not understand what politicians are doing," or "Most of the time I understand what politicians are doing." These items are rescaled so that each has a minimum of 0 and maximum of 1 and averaged to generate an index of political efficacy.

[4]To apply this model, we add a small amount of random noise (0.04σ) to break ties and create a more continuous measure of our outcomes. Then for each attendee, we determine at what quantile this pre-treatment efficacy level would fall in the distribution of pre-treatment efficacy for non-attendees.

Table 1: Pre- and Post-Treatment Data from Nepal Debate Study

Number of attendees (treated)	223
Number of non-attendees (control)	510
Post-treatment:	
Political efficacy index (treated)	mean 0.5643, min 0, max 1
Political efficacy index (control)	mean 0.4910, min 0, max 1
Knowledge index (treated)	mean 3.4215, min 0, max 11
Knowledge index (control)	mean 2.9980, min 0, max 12
Pre-treatment:	
Political efficacy index (treated)	mean 0.5669, min 0.0833, max 1
Political efficacy index (control)	mean 0.5345, min 0, max 1
Knowledge index (treated)	mean 3.1300, min 1, max 14
Knowledge index (control)	mean 2.7922, min 1, max 13

could of the responsibilities and power of local governments under the new constitution.[5] The debates were held after the local government elections and featured only candidates for the federal-level House of Representatives. They did not mention the elections for the provincial-level State Assemblies being held concurrently or the federal-level National Assembly which were to be held later. Therefore, we expect the debates or discussion to not affect knowledge on these particular items, unlike for knowledge on candidate platforms and other information that were presented during the debates. Political efficacy and knowledge are closely related, since those with less interest in politics or lower efficacy are less likely to seek out information, pay attention to information, or participate in activities that would expose them to information that they don't expect to understand well or find useful.

A DiD analysis on knowledge constitutes a classic placebo test. It appears that we "pass" this placebo test with a statistically insignificant estimate of 0.0856 (0.1855). Although these tests are widely used, it is unclear how well they protect us against unmeasured confounding. In a recent advance, Hartman & Hidalgo (2018) have proposed moving away from null hypotheses of no difference in variables that should be unaffected by the treatment, and instead testing a null hypothesis of difference in those variables against an alternative hypothesis of equivalence between treated and control groups.

Instead of using placebo outcomes to test for the validity of the research design, Sofer et al. (2016)'s negative outcome control (NOC) approach uses a placebo outcome that is assumed to have the same confounding as the primary outcome on the quantile scale to correct for the confounding. The Sofer et al. (2016) procedure is represented in Figure 3. We would take the knowledge index level for an attendee n_{11} and find its quantile amongst

[5]Each correct answer is given one point, with a possible maximum of 16.

the distribution of knowledge for the non-attendees. Then the imputed counterfactual outcome y^{NOC} for the attendee with n_{11} would be the value at that same quantile in the distribution of political efficacy for the non-attendees. Attendees with knowledge index values greater (smaller) than that observed amongst non-attendees will be assigned the largest (smallest) efficacy value observed amongst non-attendees as their counterfactual efficacy values. The average of the differences between the observed outcomes and these counterfactual outcomes for the attendees is the NOC approach's estimated ATT, 0.0040 (0.0287).

Table 2: Summary of Results

	Estimate	S.E.
Mean difference in post-treatment political efficacy (Y)	0.0733	(0.0247)
Linear DiD on political efficacy (Y)	0.0408	(0.0231)
CiC on political efficacy (Y)	0.0448	(0.0245)
Linear DiD on knowledge (N)	0.0856	(0.1855)
Post-treatment NOC	0.0040	(0.0287)
NOCNOC	0.0258	(0.0387)

We can use our NOCNOC estimator to relax the primary-placebo equi-confounding assumption to an assumption of time-invariant differential primary-placebo confounding. The NOCNOC procedure is visualized with the blue line segments in Figure 5 in earlier Section 3. For each attendee's pre-treatment efficacy value, we determine at what quantile in the distribution of the pre-treatment efficacy for the non-attendees it would fall. Then we find the knowledge level associated with that quantile, and then where this knowledge level would be in the distribution of pre-treatment knowledge for attendees. This second quantile then becomes the starting point for the second portion of the procedure, which is the NOC procedure. There is no treatment effect in the pre-treatment period, so we are effectively backing out the confounding on the quantile scale through the first portion of the procedure to carry through in the second portion. By subtracting the mean NOCNOC estimates for the counterfactual outcomes from the mean post-treatment efficacy for the attendees, we get an ATT estimate of 0.0258 (0.0387, standard error from 1000 bootstrap samples). This again indicates that exposure to debate screenings had no effect on political efficacy.

6 Conclusion

In this paper, we extended the Athey & Imbens (2006) and Sofer et al. (2016) approaches to DiD to a DiDiD context. This generalized nonlinear DiDiD approach we

call NOCNOC can be used either in the traditional DiDiD setting or when a placebo outcome is available in the pre and post-treatment data. We show that NOCNOC can correct for bias in DiDiD, CiC, and NOC. In the application, we used these methods to analyze whether exposure to candidate debates discussions affected the political efficacy of Nepalese citizens, using political knowledge as a placebo outcome. Using pre-treatment measures of both political efficacy and knowledge, we found that exposure to candidate debates likely had no effect on political efficacy, even though traditional DiD analysis with placebo tests in conventional practice suggested a positive effect.

A Proof of Theorem 1

We can rewrite Assumption 1a in the following way:

$$
\begin{aligned}
F_{N_{11}}(F^{-1}_{N_{01}}(F_{Y_{01}}(F^{-1}_{Y_{11}(0)}(q)))) &= F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(F^{-1}_{Y_{10}}(q)))), q \in [0,1] \\
F^{-1}_{N_{01}}(F_{Y_{01}}(F^{-1}_{Y_{11}(0)}(q))) &= F^{-1}_{N_{11}}(F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(F^{-1}_{Y_{10}}(q))))), q \in [0,1] \\
F_{Y_{01}}(F^{-1}_{Y_{11}(0)}(q)) &= F_{N_{01}}(F^{-1}_{N_{11}}(F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(F^{-1}_{Y_{10}}(q)))))), q \in [0,1] \\
F^{-1}_{Y_{11}(0)}(q) &= F^{-1}_{Y_{01}}(F_{N_{01}}(F^{-1}_{N_{11}}(F_{N_{10}}(F^{-1}_{N_{00}}(F_{Y_{00}}(F^{-1}_{Y_{10}}(q))))))), q \in [0,1]
\end{aligned}
$$

where the right hand side of the last equation corresponds to the second term in the NOCNOC estimator. If we further have the support conditions in Assumption 1b, then all observed values of Y_{10} will produce non-infinite values of this expression. Finally, if we have consistent estimators of the component CDFs and inverse CDFs via standard statistical theory, then NOCNOC provides a consistent estimator of ATT.

B Simulation Details

For Figure 7, we generated data from the following model:

$$
\begin{aligned}
Y_{at} &= U_{at} + a\beta, \text{ where } U|A \sim N(\eta_0 - \delta(1-t) + \theta_1 at + \theta_2(1-t)a, \frac{3-a}{2}) \\
N_{at} &= W_{at}, \text{ where } W|A \sim N(\eta_0 - \delta(1-t) + \theta_3 at + \theta_4(1-t)a, \frac{3-a}{2})
\end{aligned}
$$

for $a = \{0,1\}$, $t = \{0,1\}$. $\boldsymbol{\theta}$ is set to $(3,2,2,1)$.

For Figures 8 – 10, we generated data from the following model:

$$
\begin{aligned}
Y_{at} &= (U_{at}+1)^2 + a\beta, \text{ where } U|A \sim N(\eta_0 - \delta(1-t) + \theta_1 at + \theta_2(1-t)a, \frac{3-a}{2}) \\
N_{at} &= W_{at}, \text{ where } W|A \sim N(\eta_0 - \delta(1-t) + \theta_3 at + \theta_4(1-t)a, \frac{3-a}{2})
\end{aligned}
$$

for $a = \{0,1\}$, $t = \{0,1\}$. $\boldsymbol{\theta}$ is set to $(3,3,2,2)$ for Table 8, to (3,2,3,2) for Table 9, and to (3,2,2,1) for Table 10.

In all models, the treatment effect is β and additive. U and W are the unobserved confounders for the treatment with the primary and placebo outcomes, respectively. Y and N are each functions of time (pre/post-treatment) and an unobserved confounder, and both strictly monotonically increasing in the confounder. The confounding bias across treatment groups, over time, and across outcomes is given by the differences in the

means of U and W. To ensure support, we set the treatment effect at $\beta = 1$, $\eta_0 = 12$, $\delta = \{0, 1, 2\}$, and standard deviations of the unmeasured confounders are larger for the controls than for the treated group. We generate data for $n_1 = 500$, 1000, and 4000 treated observations and $n_0 = 2n_1$ control observations. We compare bias and RMSE of the linear differences-in-differences estimator, the CiC estimator, the NOC estimator, the linear DiDiD estimator, and the NOCNOC estimator, with 5000 simulations.

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