Instrumental Variables with Multiple Time Periods

Pedro Picchetti*

July 29, 2024

Abstract

The instrumental variables (IV) method has been widely studied in cross-sectional settings. However, many practical applications involve panel data, in settings where a unit's treatment status may turn on or off over time. In the presence of dynamics, that is, if past treatments affect current potential outcomes, standard methods are no longer valid. This paper proposes the nonparametric identification of dynamic causal effects in a potential outcomes framework in which potential outcomes depend on the treatment path taken by a unit through time and each IV instruments its contemporary treatment. I provide a nonparametric estimator that is unbiased over the randomization distribution and derive its finite population limiting distribution as the sample size increase. Monte Carlo Simulations assert the desirable finite-sample properties of the estimators. An application of the estimator shows that there is substantial time-varying heterogeneity on the effects of law enforcement on illegal deforestation, but the effects are not persistent through time.

Keywords: Panel data, Dynamic causal effects, Instrumental Variables, Finite-population.

JEL Codes: C01, C13, C21.

*I thank Cristine Pinto, Jonathan Roth, Peter Hull, Sergio Firpo, Sukjin Han, Toru Kitagawa, Vitor Possebom, as well as participants in the Bristol Econometric Study Group 2024, the New York Camp Econometrics XVIII, the Brown Econometrics Seminar and the Insper Students Seminar. I gratefully acknowledges the financial support from FAPESP 2021/13708-8 and FAPESP 2022/13229-5. All errors are my own. **Pedro Picchetti:** Department of Economics, Insper Institute of Education and Research, Rua Quata, 300, São Paulo, SP, Brazil 04546-042. E-mail: pedrop3@al.insper.edu.br

1 Introduction

In a seminal paper, Imbens and Angrist (1994) showed that the instrumental variables (IV) estimand in cross-sectional settings can be interpreted as the Local Average Treatment Effect (LATE), defined as the average treatment effect for the subpopulation that has its treatment status shifted by an excluded instrument, the so-called compliers.

However, applications of the IV method using panel data are also pervasive in the literature. In such cases, it is common to find the following static 2SLS specification stacked across time periods:

$$Y_{i,t} = \alpha + \beta D_{i,t} + \varepsilon_{i,t}$$

$$D_{i,t} = \kappa + \theta Z_{i,t} + \eta_{i,t}$$

where α and κ are constants, $D_{i,t}$ is a binary non-absorbing treatment[1] of interest in period t, $Z_{i,t}$ is the instrument in period t and $\varepsilon_{i,t}$ is the error term for a linear outcome model that imposes treatment effects that are constant over time, while $\eta_{i,t}$ is the error term from a linear model for the first stage.

As a leading case, consider this stylized example, inspired by the setting of Assunção et al. (2023) (hereafter referred to as AGR), in which the authors investigate the effects of a satellite law enforcement program, the DETER program, on deforestation in the Brazilian Amazon.

In this example, $Y_{i,t}$ is the deforested area of municipality i in the year t, $D_{i,t}$ is an indicator for "intense" law enforcement in municipality i in year t measured as a function of the number of fines issued by the DETER program, and $Z_{i,t}$ is an indicator for "intense" cloud coverage in municipality i and year t[2].

[1] Absorbing treatments are treatments that are not "forgotten" by units and remains constant in post-treatment periods (e.g a policy change, a change in the minimum wage, etc...)

[2] In the original AGR setting, the treatment is defined by the number of fines and the instrument is defined by the average cloud coverage of the municipal area. I propose this binarization for the sake of the exposition

Beyond the AGR setting, such an specification is common in applied research. To document this practice, I searched the Web of Science database for articles published between 2000 and 2023 containing the words "instrumental variable" and "panel data" in the abstract, title, or topic words. The restricted search for the following journals: *Econometrica*, *Journal of Political Economy*, *Quarterly Journal of Economics*, *American Economic Review*, *Review of Economic Studies* and *Review of Economics and Statistics*, found 33 articles matching the criteria.

Some prominent examples are in the development literature (Acemoglu et al., 2019), inequality (Aghion et al., 2018), education Jackson et al. (2015), trade (Blanchard and Matschke, 2015) and behavioral economics (Stango and Zinman, 2022).

The well-know results from Imbens and Angrist (1994) imply that the static 2SLS specification yields a LATE in the absence of dynamics, that is, if $Z_{i,t}$ only affects $D_{i,t}$ and $Y_{i,t}$, but not $D_{i,t'}$ and $Y_{i,t'}$ for $t \neq t'$. However, in practice we might often expect dynamic effects of treatment. I show that the static specification generally does not identify well defined causal effects if the instrument $Z_{i,t}$ is correlated to past values of the instrument and past treatments affect the outcome $Y_{i,t}$ as in a Robins (1986) model. Similar results have been derived in de Chaisemartin and Lei (2023) and Shen et al. (2024) in related, but different dynamic IV settings [3].

In this paper, I propose a novel identification approach for dynamic causal effects using instrumental variables and panel data when potential outcomes that are defined in terms of the sequence of treatments taken by a unit. As in Robins (1986, 1987), potential outcomes for unit i in period t depend on the path of treatments taken until period t, $Y_{i,t}(D_{i,1}, ..., D_{i,t})$. I then consider the effect of changing the path of treatments. In particular, I focus on what Bojinov et al. (2021) called the lag-p dynamic causal effect, defined as the difference

[3] An intuitive alternative would be a multivariate 2SLS specification with the sequence of endogenous treatments until period t being instrumented by the sequence of instruments until period t. I show that this approach identifies a weighted average of different dynamic causal effects in which weights can be negative. Much like in the multiple treatments IV literature (Kirkeboen et al., 2016; Mountjoy, 2022), multivariate 2SLS estimates only hold a clear causal interpretation in the absence of treatment effect heterogeneity.

between the outcomes from following different treatment paths from period $t-p$ to t , fixing the assignments for unit i to follow the observed path up to time $t-p-1$.

The strategy consists in identifying the potential outcomes associated to different treatment paths separately, by exploiting all possible variations in the assignment path from period $t-p$ to t under a "multiple differences" Wald-like estimand. That is, potential outcomes associated to contemporary treatments ($p=0$) are identified by exploiting the variation in assignment in period t holding the path until t-1 fixed, which takes the form of a simple difference. Potential outcomes associated to a path of treatments from $t-1$ to t ($p=1$) are identified exploiting variations in the path of assignment in a Wald "difference-in-differences" format.

The approach takes a purely design-based approach to uncertainty and is fully agnostic about functional forms. I provide nonparametric estimators that are unbiased over the randomization distribution induced by the random design and asymptotically normal as the finite population grows to infinity. The limiting distribution can be used to perform conservative tests on weak null hypotheses of no average dynamic causal effects. I propose the construction of a conservative dynamic version of the Bloom (1984) Confidence Interval for hypothesis testing. Monte Carlo Simulation studies illustrate the desirable finite-sample properties of the proposed estimators.

Finally, I use the proposed estimators to revisit AGR and analyze the dynamic effects of law enforcement on illegal deforestation in the Brazilian Amazon. The results suggest there are dynamic causal effect of law enforcement, which persist through time until a certain point when they fade out. The estimates show that law enforcement curbs illegal deforestation until two years after the actual detection of illegal practice.

Related Literature: This paper relates to several strands of the causal inference literature. There is an extensive literature on dynamic treatment settings, specifically from the biostatistics literature (Robins (1986), Murphy et al. (2001),Hernan and Robins (2023)). There is also a more recent interest from econometricians in such settings (Bojinov et al.

(2021); Arkhangelsky and Imbens (2022)). The aforementioned works focus on identification under the assumption that treatment assignment is sequentially randomized and that compliance is perfect. By contrast, I consider settings where there is an instrument and imperfect compliance.

There is also a large literature on the identification of dynamic treatment effects using instrumental variables, dating back to Arellano and Bond (1991). Abbring and Heckman (2007), Heckman and Navarro (2007) and Heckman et al. (2016), focus on the identification of time-to-treatment effects in settings with absorbing treatments. This paper differs from the ones mentioned above by focusing on non-absorbing treatments and taking a fully nonparametric approach to identification without relying on identification-at-infinity arguments or parametric factor models.

When it comes to sequential treatment settings, Pham and Chen (2017) propose a nonparametric identification approach that relies on the knowledge of the compliance type from each individual in the sample. Han (2021) and Han (2023) focus on how the support of the instrument and covariates allows the identification of average causal effects in sequential regimes. Sotra and Syrgkanis (2024) focuses on identification of dynamic causal effects in dynamic IV settings with one-sided noncompliance, and Shen et al. (2024) studies Panel IV regressions when the instrument follows a first-order autoregressive (AR-1) model. This paper also focus on sequential treatment regimes, but relies on different assumptions for identification. Namely, by restricting how the path of assignments affects potential treatments, I provide a new identification approach that avoids two-way exclusion restrictions and restrictions on noncompliance. Moreover, estimation and inference is fundamentally different from the aforementioned ones due to the fact that it does not rely on superpopulation arguments.

In a certain way, the sequential treatment regime is a setting with multiple treatments, multiple potential outcomes and multiple instruments. In that sense, this paper is related to the recent advances in the literature regarding multiple treatments in IV settings. (Kirkeboen

et al. (2016); Mountjoy (2022)).

Finally, the paper relates to the literature on design-based inference in IV settings (Imbens and Rubin (2015); Kang et al. (2018); Rambachan and Roth (2024), Hull and Borusyak (2024)). While these papers focus solely on estimators for the case where cross-sectional data is available, I am the first to consider design-based inference for IV settings with panel data.

Outline of the paper: Section 2 provides the intepretation of static and multivariate 2SLS specifications, as well as the proposed estimand in a framework with only two-periods. Section 3 defines the dynamic potential outcomes framework, states the identification assumptions that are used throughout the paper and define the target causal parameters. Section 4 provides the general estimand for the identification of dynamic causal effects. In Section 5 I provide a nonparametric estimator for the dynamic causal effects and derive its finite-population asymptotic distribution. Section 6 shows how the randomization distribution can be modified to incorporate common features from applied work using panel data. Section 7 presents the Monte Carlo simulations and Section 8 the empirical application. Section 9 concludes.

Notation: For an integer $t \geq 1$ and a random variable R_t, we write $R_{1:t} = (R_1, ..., R_t)$. Sets of observation units and time periods are respectively defined in a compact form as $[N] := \{1, ..., N\}$ and $[T] := \{1, ..., T\}$.

2 Identification with $T = 2$

As a leading case, consider a two-period model in which we observe at each period a binary treatment status ($D_{i,t}$), a binary instrument ($Z_{i,t}$) and a scalar outcome ($Y_{i,t}$). In the first, period, there are two possible treatments and thus two potential outcomes ($Y_{i,1}(1)$ and $Y_{i,1}(0)$) and one causal effect of interest. There are two possible assignments and thus two potential treatments ($D_{i,1}(1)$ and $D_{i,1}(0)$). Under the standard Imbens and Angrist (1994)

monotonicity assumption hold, individuals can be divided into three groups with respect to $Z_{i,1}$, always-takers (AT_1), never-takers (NT_1) and compliers (C_1).

In the second period, there are four potential outcomes associated to the four possible treatment paths. The realized outcome $Y_{i,2}$ has the following relation to the four potential outcomes:

$$Y_{i,2} = D_{i,1}D_{i,2}Y_{i,2}(1,1)+(1-D_{i,1})D_{i,2}Y_{i,2}(0,1)+D_{i,1}(1-D_{i,2})Y_{i,2}(1,0)+(1-D_{i,1})(1-D_{i,2})Y_{i,2}(0,0)$$

The vector of assignments $Z_{i,1:2}$ also takes four values. Suppose that the potential treatments at each period are functions of their contemporary treatments. Furthermore, suppose that the Imbens and Angrist (1994) assumptions hold for each period. Individuals can be divided into nine groups with respect to their compliance towards the assignment path. The individuals that comply in both periods ($C_{1:2}$) are called path-compliers.

(Linear Toy Model): For the sake of concreteness, suppose the outcome is generated by a linear causal model where potential outcomes are assumed to be homogeneous. Potential outcome for unit i in period 2 is

$$Y_{i,2}(D_{i,1:2}) = \beta_0 + \beta_1 D_{i,1} + \beta_2 D_{i,2}$$

Here, β_0 is the fully untreated potential outcome of unit i $\beta_0+\beta_1$ is the potential outcome associated to taking treatment in the first period only, $\beta_0 + \beta_2$ is the potential outcome associated to taking treatment in period 2, and $\beta_0+\beta_1+\beta_2$ is the potential outcome associated to the path of full exposure.

Suppose that in the first period the share of compliers is α_1, in the second period the share is α_2, and that the first stage of each period is independent, and thus the joint first stage is the product of first-stages so that the share of path-compliers is simply $\alpha_1\alpha_2$:

$$\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{G_{i,1}=C_1\right\}=\alpha_1,\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{G_{i,2}=C_2\right\}=\alpha_2,\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{G_{i,1:2}=C_{1:2}\right\}=\alpha_1\alpha_2$$

2.1 Interpretation of the Static Wald Estimator

In most applications of IV methods, researches usually rely on a static specification of the Wald estimator, that is, there is an outcome equation in which the outcome in period t is specified as a function of an endogenous treatment taken in period t, instrumented by an excluded variable also realized in period t. I will refer to such specification as the Time-t static Wald estimator. When $T=2$, let $\sum_{i=1}^{N}Z_{i,2}=N_1$ denote the number of units exposed to the instrument in period 2, $\sum_{i=1}^{N}(1-Z_{i,2})=N_0$ denote the number of units unexposed to treatment in period 2 and $\lambda_{i,2}=\mathbb{P}\left(Z_{i,2}=1\right)$ denote the marginal probability of being exposed to treatment in period 2 for individual i. I analyze the two-stage difference-in-means static Wald estimator, $\widehat{\beta}_2^{Wald}=\frac{\widehat{\beta}_2^{RF}}{\widehat{\beta}_2^{FS}}$, where:

$$\widehat{\beta}_2^{RF}=\frac{1}{N_1}\sum_{i=1}^{N}Z_{i,2}Y_{i,2}-\frac{1}{N_0}\sum_{i=1}^{N}(1-Z_{i,2})Y_{i,2}$$
$$\widehat{\beta}_2^{FS}=\frac{1}{N_1}\sum_{i=1}^{N}Z_{i,2}D_{i,2}-\frac{1}{N_0}\sum_{i=1}^{N}(1-Z_{i,2})D_{i,2}$$

Next, I characterize the causal decompostion of the Time-t static Wald estimand under Assumptions 1-4. Proposition 1 shows that the estimator suffers from omitted variable bias (OVB) under the maintained assumptions:

Proposition 1 *Suppose that the dynamic treatment potential outcomes model hold, and that for each period that assumptions from Imbens and Angrist (1994) for the first-stage hold. Then,*

$$\frac{\mathbb{E}\left[\widehat{\beta}_2^{RF}\right]}{\mathbb{E}\left[\widehat{\beta}_2^{FS}\right]} = \underbrace{\overline{\tau}_{C_2,2}(1,0;0)}_{lag-0\ effect} + \underbrace{\Lambda}_{OVB}$$

where

$$\Lambda = \frac{1}{|C_2|}\left(\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(D_{i,1}(Z_{i,1}), D_{i,2}(1))\right] + \frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(D_{i,1}(Z_{i,1}), D_{i,2}(0))\right]\right)$$

To put it simply, Proposition 1 shows that in the presence of serial correlation of the instrument, the Time-t static Wald estimator can suffer from omitted variable bias, see Section 1 of Appendix A for the proof. If there is serial correlation and the potential outcome $Y_{i,2}$ depends on the past treatment $D_{i,1}$, then $\lambda_{i,2}$ might be related to $Y_{i,2}(D_{i,1}(Z_{i,1}, D_{i,2}(1))$ and $Y_{i,2}(D_{i,1}(Z_{i,1}, D_{i,2}(0))$ through the dependence of $\lambda_{i,2}$ on $Z_{i,1}$.

Consider the bias terms that form Λ, they could be equal to zero under three conditions: (i) there's no dynamic effect of treatment, $D_{i,1}$ does not affect $Y_{i,2}$ so the correlation between $Z_{i,2}$ and $Z_{i,1}$ is inconsequential; (ii) there are no compliers in period 1, so that $D_{i,1}(1) = D_{i,1}(0)$ and once again the serial correlation is inconsequential or (iii) the instrument is fully randomized at each period, so that there is no serial correlation of the instrument. If one of the three conditions above is satisfied, then the estimand can be interpreted as the time-2 local lag-0 dynamic causal effect, the causal effect in period 2 from taking treatment in period 2 for compliers in period 2.

Linear Toy Model (continued). Consider the linear toy model introduced above. The expected value of the Time-t static Wald estimator over the randomization distribution amounts to

$$\frac{\mathbb{E}\left[\widehat{\beta}_2^{RF}\right]}{\mathbb{E}\left[\widehat{\beta}_2^{FS}\right]} = \beta_2 + \frac{\beta_1\alpha_1\left(\mathbb{E}\left[Z_{i,1}=1|Z_{i,2}=1\right] - \mathbb{E}\left[Z_{i,1}=1|Z_{i,2}=0\right]\right)}{\alpha_2}$$

It is clear that the ratio of means equals β_2 only if there is no dynamic effect of treatment

($\beta_1 = 0$), if there are no compliers in previous periods ($\alpha_1 = 0$), or the instrument is fully randomized at each period ($\mathbb{E}[Z_{i,1} = 1|Z_{i,2} = 1] = \mathbb{E}[Z_{i,1} = 1|Z_{i,2} = 0]$).

Translating to the AGR setting, the estimates for the effect of present law enforcement on illegal deforestation from the static 2SLS specification are biased if there are dynamic effects of law enforcement if the probability of a municipality experiencing "intense" cloud coverage depends on its history of past cloud coverage.

2.2 Multivariate 2SLS Decomposition

An intuitive alternative to the static approach would be a multivariate 2SLS specification, in which the path of treatments is instrumented by the path of assignments, that is, the IV estimate of

$$Y_{i,2} = \beta_0 + \beta_1 D_{i,1} + \beta_2 D_{i,2} + u_{i,2}$$

would seemingly be able to deliver dynamic and present causal effects. In order to analyze the multivariate 2SLS estimator, define the following sample moment:

$$\widehat{\Omega} = \frac{1}{N} \sum_{i=1}^{N} \begin{pmatrix} Y_{i,2} \\ D_{i,1} \\ D_{i,2} \\ Z_{i,1} \\ Z_{i,2} \end{pmatrix} \begin{pmatrix} Y_{i,2} \\ D_{i,1} \\ D_{i,2} \\ Z_{i,1} \\ Z_{i,2} \end{pmatrix}'$$

The partitioned version of the matrix above can be expressed as

$$\widehat{\Omega} = \begin{pmatrix} \Omega_{YY} & \Omega_{YD_1'} & \Omega_{YD_2'} & \Omega_{YZ_1'} & \Omega_{YZ_2'} \\ \Omega_{D_1Y} & \Omega_{D_1D_1'} & \Omega_{D_1D_2'} & \Omega_{D_1Z_1'} & \Omega_{D_1Z_2'} \\ \Omega_{D_2Y} & \Omega_{D_2D_1'} & \Omega_{D_2D_2'} & \Omega_{D_2Z_1'} & \Omega_{D_2Z_2'} \\ \Omega_{Z_1Y} & \Omega_{Z_1D_1'} & \Omega_{Z_1D_2'} & \Omega_{Z_1Z_1'} & \Omega_{Z_1Z_2'} \\ \Omega_{Z_2Y} & \Omega_{Z_2D_1'} & \Omega_{Z_2D_2'} & \Omega_{Z_2Z_1'} & \Omega_{Z_2Z_2'} \end{pmatrix}$$

The well known closed form solution for the multivariate 2SLS is

$$\begin{pmatrix} \widehat{\beta}_1^{2SLS} \\ \widehat{\beta}_2^{2SLS} \end{pmatrix} = \begin{pmatrix} \Omega_{Z_1 D_1'} & \Omega_{Z_1 D_2'} \\ \Omega_{Z_2 D_1'} & \Omega_{Z_2 D_2'} \end{pmatrix}^{-1} \begin{pmatrix} \Omega_{Z_1 Y} \\ \Omega_{Z_2 Y} \end{pmatrix}$$

Define the expected value of the multivariate 2SLS as $\tilde{\beta}_1$ and $\tilde{\beta}_2$, respectively. Proposition 2 discusses their interpretation:

Proposition 2 *Suppose that the dynamic treatment potential outcomes model hold, and that for each period that assumptions from Imbens and Angrist (1994) for the first-stage hold. Then,* $\tilde{\beta}_1 = \mathbb{E}\left[\widehat{\beta}_1^{2SLS}\right]$ *and* $\tilde{\beta}_2 = \mathbb{E}\left[\widehat{\beta}_2^{2SLS}\right]$ *are linear combinations of the following causal effects:*

1. $Y_{i,2}(1,1) - Y_{i,2}(0,0)$
2. $Y_{i,2}(0,1) - Y_{i,2}(0,0)$
3. $Y_{i,2}(1,0) - Y_{i,2}(0,0)$

Involving five different groups of compliers in different periods: path-compliers $(C_{1:2})$*, compliers for period 1 but not for period 2* $((C_1, AT_2), (C_1, NT_2))$*, and compliers for period 2 but not period 1* $((AT_1, C_2), (NT_1, C_2))$*.*

Proposition 2 shows that without further restrictions, IV estimation of a multivariate dynamic specification does not identify any dynamic causal effect. The decomposition is shown in Section 2 of Appendix A.

This result is similar to the no-identification result of multivariate 2SLS in IV settings with multiple treatments and multiple instruments (Kirkeboen et al., 2016; Mountjoy, 2022). In such settings, the estimates from multivariate 2SLS only identify well-defined causal parameters if treatment effects are homogeneous across individuals.

In the dynamic setting, the coefficients are linear combinations of potential outcomes that are compliers in one the periods, but might be noncompliers in the other. Hence, treatment

effects must be homogeneous across individuals and time periods. Therefore, multivariate 2SLS has the undesirable consequence of mixing comparisons and is only valid in the absence of individual and time-varying heterogeneity in causal effects.

2.3 Separate Identification Approach

To overcome the limitations of the standard static Wald estimand and the multivariate 2SLS, I develop an alternative dynamic IV approach that separately identifies causal effects of different treatment paths for individuals that are compliers for the path of interest.

Instead of attempting to use the path of instruments to recover multiple causal effects such as in the multivariate specification, which has the undesirable consequence of mixing comparisons across different treatment paths and groups of compliers, I propose a separate identification approach that isolates mean potential outcomes for path compliers. Taking the difference between two of these separate components generates a dynamic causal effect for path-compliers.

I illustrate the procedure by showing how the proposed causal estimand identifies the mean causal effect of full exposure for path-compliers, that is, $\frac{1}{N}\sum_{i\in C_{1:2}}(Y_{i,2}(1,1)-Y_{i,2}(0,0))\frac{1}{|C_{1:2}|}$. A causal estimand is defined by Abadie et al. (2020) as an estimand written in terms of potential outcomes and potential treatments. Even though it cannot be inferred from realized outcomes and treatments, it is useful to build the descriptive estimand that is built using realized outcomes and treatments, and subsequently, the estimator.

In the case of $T = 2$, there are four potential outcomes and four potential treatments that can be arranged in order to identify the mean potential outcomes. Below, I show that a "difference-in-differences" Wald-like causal estimand exploiting the variations in the path of assignment recovers the mean potential outcomes for path-compliers.

I begin with the identification of the potential outcome associated to full exposure, $Y_{i,2}(1,1)$. Consider the "difference-in-differences" causal estimand for the first stage:

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,1}(1)D_{i,2}(1) - D_{i,1}(1)D_{i,2}(0) - (D_{i,1}(0)D_{i,2}(1) - D_{i,1}(0)D_{i,2}(0)) \tag{1}$$

First, note that under Assumptions 1-4,

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,1}(z_1)D_{i,2}(z_2) = \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,1}(z_1) = 1, D_{i,2}(z_2) = 1\right\}$$

Table 1 shows how the fully treated path appears for different groups of compliers under different assignment paths. Always-takers for both periods (AT_1, AT_2) will take the fully treated sequence under the four possible assignments, compliers for the two periods only take the fully treated path under assignment to treatment for the full path. The third column shows what is identified under the first-difference, referred to as Δ^1, of the fully treated path. That is, the quantity that is identified by exploiting the variation in assignment in period 2, keeping assignment in period 1 fixed. The fourth column shows the "difference-in-differences", that is, the difference in Δ^1 across different assignments in period 1, referred to as Δ^2.

Table 1: Separate Identification Approach - Modified First-stage $(D_{i,1:2} = (1,1))$

Potential Path	Groups	Δ^1	Δ^2
$\mathbf{1}\{D_{i,1}(1) = 1, D_{i,2}(1) = 1\}$	$(AT_1, AT_2), (C_1, AT_2), (AT_1, C_2), (C_1, C_2)$		
$\mathbf{1}\{D_{i,1}(1) = 1, D_{i,2}(0) = 1\}$	$(AT_1, AT_2), (C_1, AT_2)$	$(AT_1, C_2), (C_1, C_2)$	
			(C_1, C_2)
$\mathbf{1}\{D_{i,1}(0) = 1, D_{i,2}(1) = 1\}$	$(AT_1, AT_2), (AT_1, C_2)$		
$\mathbf{1}\{D_{i,1}(0) = 1, D_{i,2}(0) = 1\}$	(AT_1, AT_2)	(AT_1, C_2)	

Therefore, it follows that the causal estimand for the modified first-stage expressed in equation (1) identifies $\sum_{i=1}^{N} \mathbf{1}\{G_{i,1:2} = C_1, C_2\} = |C_{1:2}|$, which is the size of the group of individuals individuals that are compliers in both periods.

Now, let's look at the modified reduced form causal estimand associated to the fully treated path. Similar to the first stage, we have under Assumptions 1-4 that

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,1}(z_1)D_{i,2}(z_2)Y_{i,2}(1,1) = \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,1}(z_1)=1, D_{i,2}(z_2)=1\right\} Y_{i,2}(1,1)$$
$$= \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,1}(z_1)=1, D_{i,2}(z_2)=1\right\} Y_{i,2}(1,1)$$

Therefore, using the same reasoning as in the first stage one finds that the "difference-in-differences" causal estimand for the reduced form identifies

$$\frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{G_{i,1:2}=(C_1,C_2)\right\} Y_{i,2}(1,1) = \frac{1}{N}\sum_{i\in C_{1:2}} Y_{i,2}(1,1)$$

and the ratio of these causal estimands identifies

$$\frac{1}{N}\sum_{i\in C_{1:2}} Y_{i,2}(1,1)\frac{1}{|C_{1:2}|}$$

which is a design-based expression for the local average response function associated to full exposure for compliers in periods 1 and 2.

Now, consider the potential outcome associated to full control. Table 2 shows how the fully untreated path appears for different groups of compliers under different assignment paths. Never-takers for both periods (NT_1, NT_2) will take the fully untreated sequence under the four possible assignments, compliers for the two periods only take the fully untreated path under assignment to control for the full path.

Table 2: Separate Identification Approach - Modified First-stage ($D_{i,1:2} = (0,0)$)

Potential Path	Groups	Δ^1	Δ^2
$\mathbf{1}\{D_{i,1}(1)=0, D_{i,2}(1)=0\}$	(NT_1, NT_2)		
$\mathbf{1}\{D_{i,1}(1)=0, D_{i,2}(0)=0\}$	$(NT_1, NT_2), (NT_1, C_2)$	(NT_1, C_2)	
			$-(C_1, C_2)$
$\mathbf{1}\{D_{i,1}(0)=0, D_{i,2}(1)=0\}$	$(NT_1, NT_2), (C_1, NT_2)$		
$\mathbf{1}\{D_{i,1}(0)=0, D_{i,2}(0)=0\}$	$(NT_1, NT_2), (C_1, NT_2), (NT_1, C_2), (C_1, C_2)$	$(NT_1, C_2), (C_1, C_2)$	

It is easy to see that the first stage estimand identifies $-\frac{1}{|C_{1:2}|}$, and the reduced form

identifies $-\sum_{i\in C_{1:2}} Y_{i,2}(0,0)$. And thus, the separate identification approach allows us to identify $\sum_{i\in C_{1:2}} Y_{i,2}(0,0)\frac{1}{|C_{1:2}|}$.

Linear Toy Model (Continued). In the linear toy model introduced above, the dynamic causal effects associated to full exposure is $\beta_1 + \beta_2$. The first stage associated to the fully exposed potential outcome identifies

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,1}(1)D_{i,2}(1) - D_{i,1}(1)D_{i,2}(0) - (D_{i,1}(0)D_{i,2}(1) - D_{i,1}(0)D_{i,2}(0)) = \alpha_1^1\alpha_2^2$$

The reduced form identifies

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,1}(1)D_{i,2}(1)Y_{i,2} - D_{i,1}(1)D_{i,2}(0)Y_{i,2}$$
$$- (D_{i,1}(0)D_{i,2}(1)Y_{i,2} - D_{i,1}(0)D_{i,2}(0)Y_{i,2}) = (\beta_0 + \beta_1 + \beta_2)\alpha_1^1\alpha_2^2$$

And thus, the "difference-in-differences" Wald-like estimand identifies $\beta_0 + \beta_1 + \beta_2$.

The first stage associated to the fully unexposed potential outcome identifies

$$\frac{1}{N}\sum_{i=1}^{N}(1 - D_{i,1}(1))(1 - D_{i,2}(1)) - (1 - D_{i,1}(1))(1 - D_{i,2}(0))$$
$$- ((1 - D_{i,1}(0))(1 - D_{i,2}(1)) - (1 - D_{i,1}(0))(1 - D_{i,2}(0))) = -\alpha_1^1\alpha_2^2$$

The reduced form identifies

$$\frac{1}{N}\sum_{i=1}^{N}(1 - D_{i,1}(1))(1 - D_{i,2}(1))Y_{i,2} - (1 - D_{i,1}(1))(1 - D_{i,2}(0))Y_{i,2}$$
$$- ((1 - D_{i,1}(0))(1 - D_{i,2}(1))Y_{i,2} - (1 - D_{i,1}(0))(1 - D_{i,2}(0))Y_{i,2}) = -\beta_0\alpha_1^1\alpha_2^2$$

And thus β_0 is identified. Taking the difference between these two quantities identifies $\beta_1 + \beta_2$.

Why does the "difference-in-differences" approach work? The first difference identifies compliers in period 2, but this individuals are from different subgroups in period 1. For instance, in the case of full exposure, the first difference associated to the $Y_{i,2}(1,1)$ potential identifies compliers in period 2, but this compliers can be either compliers or always-takers in period 1.

When it comes to the $Y_{i,2}(0,0)$ outcome, however, the first difference identifies compliers in period 2 that can be either compliers in period 1 or never-takers in period 1. Hence, the difference between the first difference quantities yields forbidden comparisons between heterogeneous groups in period 1.

The second-difference cancels out noncompliers in period 1 for each potential outcome, identifying the different potential outcomes for individuals that are compliers for both periods, and therefore, comparable.

3 Framework

3.1 Assumptions

Consider a balanced panel in which N units are observed over T periods of time. For each unit $i \in [N]$ and time period $t \in [T]$, we observe a binary instrumental variable $Z_{i,t} \in \{0,1\}$, a binary treatment status $D_{i,t} \in \{0,1\}$ and a real-valued scalar outcome $Y_{i,t}$.

Without further restrictions, potential outcomes of unit i in period t are a function of the full panel of treatments and assignments, $Y_{i,t}(d_{1:N,1:T}, z_{1:N,1:T})$, and potential treatments are a function of the full panel of assignments, $D_{i,t}(z_{1:N,1:T})$. The next assumptions are invoked for identification.

Assumption 1 (No Spillovers and No-Anticipation) For all $i \in [N]$, $t \in [N]$,

$$Y_{i,t} = \sum_{(d_{1:t},z_{1:t})\in\{0,1\}^{t\times t}} \mathbf{1}\left\{D_{i,1:t} = d_{1:t}, Z_{i,1:t} = z_{1:t}\right\} Y_{i,t}(d_{1:t}, z_{i:t})$$

$$D_{i,t} = \sum_{z_{1:t}\in\{0,1\}^{t}} \mathbf{1}\left\{Z_{i,1:t} = z_{1:t}\right\} D_{i,t}(z_{1:t})$$

Assumption 1 imposes that the potential outcome of unit i in period t depends only on the treatment and assignment paths of unit i until period t, ruling out the possibility of spillover of both treatments and assignments across units, as well as future treatments affecting past potential outcomes. It also imposes that the potential treatment from unit i in period t depends only on the assignment paths of unit i until period t. To put it shortly, Assumption 1 imposes both SUTVA and no-anticipation.

In the AGR setting, Assumption 1 implies that deforestation in a municipality in a certain year depends only on the path of law enforcement for the that municipality until that year, thus ruling out spillovers in deterrence effects and the possibility that expectations of future law enforcement affect current deforestation decisions. It also implies that future cloud coverage does not affect the present capacity of detection from the satellite monitoring deforestation.

One of the fundamental assumptions of IV settings is the exclusion restriction. Its dynamic version is stated below, alongside an additional exclusion restriction for the first stage.

Assumption 2 (Exclusion Restrictions): $Y_{i,t}(d_{i,1:t}, z_{i,1:t}) = Y_{i,t}(d_{i,1:t})$ and $D_{i,t}(z_{1:t}) = D_{i,t}(z_t)$, for all $i \in [N]$ and $t \in [T]$.

The first part of Assumption 2 is the standard exclusion restriction for dynamic IV settings. It states that the path of assignments does not affect potential outcomes directly. Assignments only affect potential outcomes to the extent that they affect treatment choices. Mapping it to the application, it implies that the history of cloud coverage over a municipality affects illegal deforestation in that municipality only through its effect on law enforcement capabilities.

The second part of Assumption 2 imposes that potential treatments in period t depend only on the instruments in period t. Although it might seem strong in a dynamic setting, it is justifiable in many experimental and observational settings. In AGR, the capacity of the satellite to detect illegal deforestation at a given period depends on the cloud coverage below the satellite at that period. Previous coverage does not affect the current capacity to detect deforestation.

When considering the validity of Assumption 2 in other contexts, one can think, for example, of a multi-period experiment with imperfect compliance in which past assignments do not provide any "encouragement" to present treatment due to institutional aspects of the experiment. A canonical case would be the Fast Track Prevention Program (Conduct Problems Prevention Research Group, 1992), a randomized trial to prevent conduct disorders and drug use in children at risk studied by Murphy et al. (2001). Interventions take place at the end of each semester starting from first grade and noncompliance occurs for almost 53% of the population. The interventions take place at the school and the student's household, and previous assignments to the intervention do not provide access to the intervention in subsequent periods. Now, consider an example for observational settings. In a criminal justice setting with repeated offenders, sentences in a period depend only on the leniency of the judge assigned to trial in that period, and not on previous judges characteristics[4].

The fundamental behavioral assumption in IV settings is the monotonicity assumption, which is provided below.

Assumption 3 (Monotonicity): For all $i \in [N]$ and $t \in [T]$, $D_{i,t}(1) \geq D_{i,t}(0)$.

Monotonicity states that at each period units assigned to treatment ($Z_{i,t} = 1$) are almost-sure to take treatment as units assigned to control ($Z_{i,t} = 0$). Under Assumption 3, units can be divide into three groups at each period of time defined by how units treatment choice in period t relates to treatment assignment in period t: Always-takers (AT_t), Never-takes (NT_t) and Compliers (C_t). Note that an individual can be part of a group in a given period, it does

[4]One can think that the lenience of past judges do not affect current sentences, but past sentences might. This is not a violation of Assumption 2, and cases such as this are discussed in Section 6.2

not need to be in the same group through the whole path of assignments. In the application, Assumption 3 implies that municipalities with intense cloud coverage at a certain period are are less likely to have intense law enforcement in that period than the ones with little cloud coverage [5].

Throughout the paper, I follow a design-based approach for inference. Potential treatments and potential outcomes from a finite-population are taken as given, and uncertainty in the setting arises from randomness in the assignment of the instrument.

Let $\mathcal{F}_{1:N,1:T}$ denote the collection of potential outcomes and potential treatments for all units and all periods of time. Assumption 4 states that the assignment of the instrument at each period are individualistic and depend only on observed past assignments. Let $R_{-i,t} = (R_{1,t}, ..., R_{i-1,t}, R_{i+1,t}, ..., R_{N,t})$

Assumption 4 (Sequential Randomization): For all $i \in [N]$, $t \in [T]$,

$$\mathbb{P}\left(Z_{i,t} = z_t | Z_{-i,1:t}, \mathcal{F}_{1:N,1:T}\right) = \mathbb{P}\left(Z_{i,t} = z_t | Z_{i,1:t-1} = z_{1:t-1}\right)$$

Assumption 4 is the finite-population framework analog of the mean-independence of the instrument assumption that is usually invoked using super-population arguments in dynamic IV settings(Han, 2021, 2023). It states that treatment assignment in period t is "as-good-as-random" conditional on the path of assignments from period 1 to period $t-1$[6]. In our applied setting, it implies that cloud coverage in a certain period is independent from the panel of potential law enforcement and potential deforestation conditional on the path of cloud coverage until that period.

3.2 Target Parameters

We are interested in the identification of dynamic causal effects, which are parameters that compare potential outcomes for unit i at period t along different treatment paths. Define

[5]For the application, it is more adequate to state Assumption 3 with the inverted inequality, $D_{i,t}(1) \leq D_{i,t}(0)$, which does not affect the validity of the results exposed in this paper.

[6]Modified versions of Assumption 4 are discussed in Section 6

the dynamic causal effect of a treatment path versus an alternative treatment path in period t as $\tau_{i,t}(d_{i,1:t}, \tilde{d}_{i,1:t}) = Y_{i,t}(d_{i,1:t}) - Y_{i,t}(\tilde{d}_{i,1:t})$.

The number of potential outcomes grows exponentially with the periods of time. For the sake of tractability, it is common to focus on lag-p dynamic causal effect as defined in Bojinov et al. (2021). For $0 \leq p \leq t$, and $\mathbf{d}, \tilde{\mathbf{d}} \in \{0,1\}^{p+1}$, the lag-p dynamic causal effect is defined as

$$\tau_{i,t}(\mathbf{d}, \tilde{\mathbf{d}}; p) := Y_{i,t}(d^{obs}_{i,1:t-p-1}, \mathbf{d}) - Y_{i,t}(d^{obs}_{i,1:t-p-1}, \tilde{\mathbf{d}})$$

Which can be interpreted as the causal effect of taking a treatment path from period $t-p$ to p versus an alternative path, keeping the path until period $t-p-1$ fixed.

The lag-p dynamic causal effect can be used to construct generalized impulse response functions, which are weighted averages of the lag-p dynamic causal effects, defined as

$$\tau^{\dagger}_{i,t}(\mathbf{d}, \tilde{\mathbf{d}}; p) = \sum_{\mathbf{v} \in \{0,1\}^p} a_{\mathbf{v}} \left\{ Y_{i,t}(d^{obs}_{1:t-p-1}, (\mathbf{d}, \mathbf{v})) - Y_{i,t}(d^{obs}_{1:t-p-1}, (\tilde{\mathbf{d}}, \mathbf{v})) \right\}$$

where $a_{\mathbf{v}}$ are non-stochastic weights chosen by the researcher. By varying the values of the choice for the lag p, the generalized impulse response function can be used to construct event-study plots. In that sense, the generalized impulse response function parameter holds a similar interpretation to the event study parameters estimated in Difference-in-Differences settings (Goodman-Bacon, 2021; Callaway and Sant'Anna, 2021) as the effect of the length of exposure to treatment.

There are different ways to aggregate such parameters in order to highlight different aspects of heterogeneity in dynamic causal effects. As in cross-sectional IV settings, the average causal effects are only identifiable for the group of compliers. I define two target parameters for the path of compliers. The time-t lag-p local dynamic causal effect and the total lag-p local dynamic causal effect are defined respectively as

$$\overline{\tau}_{C_{t-p:t},t}(\mathbf{d},\tilde{\mathbf{d}};p) = \sum_{i\in C_{t-p:t}} \tau_{i,t}(\mathbf{d},\tilde{\mathbf{d}};p)\frac{1}{|C_{t-p:t}|}$$

$$\overline{\tau}_{C_{t-p:t}}(\mathbf{d},\tilde{\mathbf{d}};p) = \frac{1}{T-p}\sum_{t=p+1}^{T}\sum_{i\in C_{t-p:t}} \tau_{i,t}(\mathbf{d},\tilde{\mathbf{d}};p)\frac{1}{|C_{t-p:t}|}$$

These estimands extend to the generalized impulse response function by analogously defining $\overline{\tau}^{\dagger}_{C_{t-p:t},t}$ and $\overline{\tau}^{\dagger}_{C_{t-p:t}}$.

Next, I show how the dynamic IV can be used to identify dynamic causal effects for units that are contemporary compliers in the periods of interest. The identification of the generalized impulse response function follows directly.

4 Identification with General T

4.1 The Role of the Time Structure in Identification

Dynamic IV settings and Multiple Treatment IV settings share some common features. Namely, there are multiple potential outcomes and hence multiple treatment effects, from which it follows that standard IV methods (e.g Wald estimands, Multivariate 2SLS) fail to recover relevant causal effects in the presence of heterogeneity. However, the identification approach is fundamentally different in these settings. One fundamental difference, is that the multiple differences Wald-like estimand identifies dynamic causal effects without any additional assumptions, which is not the case in multiple treatments settings, which rely in additional restrictions, such as assuming irrelevant alternatives (Kirkeboen et al., 2016), or comparable compliers (Mountjoy, 2022).

What makes identification in the dynamic setting fundamentally different from the multiple treatment setting? The answer lies in the sequential nature of choice and compliance.

In the multiple treatments settings, there are mutually exclusive and exhaustive treat-

ments, and shifts in the instruments for a treatment affect compliers coming from different treatments. In Mountjoy (2022), for example, shifts in the distance of a community college make compliers that would not enroll in any superior education and compliers that would enroll in a 4-year college move to the community college option. A shift in an instrument affects multiple margins.

In the dynamic setting, treatment paths are mutually exclusive and exhaustive, but period-specific treatment choices are not, and the instrument is assigned at the period-specific treatment level, that is, there is a path of instruments, not instruments for the path. Thus, the time structure in the setting allows the identification of compliers for a sequence of periods under the multiple differences estimands.

Under no-anticipation, shifts in $Z_{i,t}$ do not affect treatment choices in periods 1 up to t-1. Keeping the path of instruments $z_{1:t-1}$ fixed, variations in $Z_{i,t}$ identify the compliers in period t. Compliers in period t mimic their assignment in period t. Hence, variation in instruments from previous periods do not affect their decision in period t. Thus, keeping $z_{1:t-2}$ fixed, the joint variation of $Z_{i,t-1}$ and $Z_{i,t}$ in the difference-in-differeces format identifies that share of individuals that comply in both t-1 and t. Taking a triple difference would identify compliers from t-2 to t, so on and so forth.

4.2 A General Expression for the Multiple Differences Estimand

Here, I focus on the identification of the local lag-p response functions, which are mean potential outcomes for compliers from period $t-p$ to period t, along the observed treatment path until period $t-p-1$. I define them as

$$m_{t,(\mathbf{d})} = \frac{1}{|C_{t-p:t}|} \sum_{i \in C_{t-p:t}} Y_{i,t}(d^{obs}_{1:t-p-1}, \mathbf{d})$$

where $\mathbf{d}$ is the treatment path of interest from $t-p$ to t.

Before introducing the main identification result, I define here an expression for the

general multiple difference in means across assignment paths, which I will refer to hereafter as the $p+1$ difference in means (Δ^{p+1}).

Definition (Δ^{p+1}): Define the $p+1$-th difference across assignment paths from period t-p onwards of conditional means of a random variable R as

$$\Delta^{p+1}\left(R_{i,t}(z_{t-p:t})\right) = \sum_{z_{t-p:t}\in\{0,1\}^{p+1}} \sum_{i\in\mathcal{Z}_{1:t-p-1}} (-1)^{\prod_{k\in\{t-p:t\}}(z_k+1)} R_{i,t}(z_k)$$

It is easy to see that when $p=0$, the expression takes form as a difference in means for units with $Z_{i,t}=1$ versus units with $Z_{i,t}=0$, holding the assignment from period 1 to period $t-1$ fixed. For the case $p=1$, the expression takes the form of a difference-in-differences across assignment paths from period $t-1$ to period t, keeping the path until period $t-2$ fixed. For $p=2$, it takes the form a triple difference. Thus, the $p+1$ difference can be used to exploit all possible variations in assignment paths until period t.

Before defining the causal estimand, one more assumption is required for the proposed object to be well-defined:

Assumption 5 (Relevance): For all $i\in[N]$, $t\in[T]$ and $\mathbf{z}\in\{0,1\}^{p+1}$,

$$\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})=\mathbf{d}\right\} \neq 0$$

Assumption 5 is a dynamic version of the standard relevance assumption from cross-sectional IV settings. In the case of $p=0$, it amounts $\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(1)-D_{i,t}(0)\neq 0$, which is the standard relevance assumption (Kang et al., 2018).

Theorem 1 shows that a Wald-like causal estimand built using the $p+1$ difference of the modified outcome in period t and a first stage for a treatment path from period $t-p$ to p equals the local lag-p response function.

Theorem 1 *Suppose Assumptions 1-5 hold. Then for any $p\leq t$,*

$$\frac{\Delta^{p+1}\left(\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,t-p:t}(\boldsymbol{z})=\boldsymbol{d}\right\}Y_{i,t}(d^{obs}_{i,1:t-p-1},\boldsymbol{d})\right)}{\Delta^{p+1}\left(\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,t-p:t}(\boldsymbol{z})=\boldsymbol{d}\right\}\right)}=m_{t,(\boldsymbol{d})}$$

The theorem shows that the $p+1$ difference estimand identifies the local lag-p response function conditional on the assignment path until period t-p-1. Integrating over the assignment paths identifies the local lag-p response function. Two local lag-p response functions generate a local lag-p dynamic causal effect. See Section 3 of Appendix A for a proof by induction.

Building on the identification result for the causal estimand, I propose a descriptive estimand which takes the form of a Horvitz-Thompson estimand, which is built using a propensity score for the path of the instrument, given by $\pi_{i,t-p}(\mathbf{z})=\mathbb{P}\left(Z_{i,t-p:t}=\mathbf{z}|Z_{i,1:t-p-1}=z_{1:t-p-1}\right)$.

Next, I assume that the assignment mechanism is probabilistic.

Assumption 6 (Probabilistic Assignment): For all $i\in[N]$, $t\in[T]$, there exists $C^L<C^U\in(0,1)$ such that $C^L<\pi_{i,t-p}(\mathbf{z})<C^U$ for all $\mathbf{z}\in\{0,1\}^{p+1}$.

Assumption 6 is also known as the common support assumption, and it states that individuals have a positive probability of following all possible assignment paths.

Theorem 2 shows that a Wald-like Horvitz-Thompson estimand identifies the local lag-p response function.

Theorem 2 *Define*

$$\tilde{m}_{t,(\boldsymbol{d})}=\frac{\tilde{m}^{RF}_{t,(\boldsymbol{d})}}{\tilde{m}^{FS}_{t,(\boldsymbol{d})}}=\frac{\Delta^{p+1}\left(\sum_{i=1}^{N}\frac{Y_{i,t}\mathbf{1}\{D_{i,t-p:t}=\boldsymbol{d}\}}{\pi_{i,t-p:t}(\boldsymbol{z})}\right)}{\Delta^{p+1}\left(\sum_{i=1}^{N}\frac{\mathbf{1}\{D_{i,t-p:t}=\boldsymbol{d}\}}{\pi_{i,t-p:t}(\boldsymbol{z})}\right)}$$

Under Assumptions 1-6,

$$\frac{\mathbb{E}\left[\tilde{m}^{RF}_{t,(\boldsymbol{d})}\right]}{\mathbb{E}\left[\tilde{m}^{FS}_{t,(\boldsymbol{d})}\right]}=m_{t,(\boldsymbol{d})}$$

Theorem 2 provides the main identification result, see Section 4 of Appendix A. The quantity $\tilde{m}_{t,(\mathbf{d})}$ is a function of the observable data that can be used to recover the local

lag-p response function. Time-t local lag-p dynamic causal effects are identified by taking the difference between two local lag-p response functions identified separately.

4.3 Identification under Sequential Monotonicity

The results presented above rely on a monotonicity assumption combined with an exclusion restriction for the first stage. At each period individuals are assigned to treatment control and choose their treatment status based on the contemporary assignment, that is, the observed treatment in period t is a function of two potential treatments: $D_{i,t} = Z_{i,t}D_{i,t}(1) + (1 - Z_{i,t})D_{i,t}(0)$. Although this sort of monotonicity assumption appears in the dynamic IV literature (Pham and Chen, 2017), it is also common to define treatment choice in period t as a function of the path of instruments until period t and assume sequential monotonicity:

Assumption 4' (Sequential Monotonicity): For all $i \in [N]$, $t \in [T]$, $D_{i,t}(z_{1:t-1}, 1) \geq D_{i,t}(z_{1:t-1}, 0)$.

Identification under sequential monotonicity is challenging as individuals that are compliers to the contemporary instrument coming from a path $z_{1:t-1}$ might not be compliers coming from an alternative path $z'_{1:t-1}$ (Sotra and Syrgkanis, 2024). Hence, dynamic causal effects are not identified without further assumptions. A simple solution would be to assume that although the path of instruments might affect treatment choice, it does not affect compliance towards the contemporary instrument.

Assumption 7 (Path-independent compliance): For all $i \in [N]$, $t \in [T]$, $D_{i,t}(z_{1:t-1}, 1) > D_{i,t}(z_{1:t-1}, 0)$ implies $D_{i,t}(z'_{1:t-1}, 1) > D_{i,t}(z'_{1:t-1}, 0)$.

Although hard to motivate from a design-based perspective, Assumption 5 is readily satisfied in specifications of the first stage in period t where the path of instruments $Z_{i,1:t}$ is separable, or at least that the instrument $Z_{i,t}$ is separable from $Z_{i,1:t-1}$, i.e, $D_{i,t} = f(Z_{i,1:t}) = f_1(Z_{i,t}) + f_2(Z_{i,1:t-1})$, which are common specifications in the applied literature[7].

In a way, Assumptions 4' and 5 combined are equivalent to Assumption 4, in the sense

[7]Linear specifications for the first stage are pervasive in the literature and readily satisfy the separability assumption

that they allow compliance to be described solely in terms of the behavior with respect to the contemporary assignment. Assuming that there are no path-specific compliers, however, can be quite strong. The reasonableness of Assumption 4' can be assessed by calculating the compliance rates towards the contemporary instrument coming form different assignment paths, as in Marcus et al. (2014).

5 Estimation and Inference

5.1 Correcting the Wald Estimator

Proposition 1 shows that the static "difference-in-means" Wald estimator fails to recover relevant causal effects in the presence of dynamics. Thus, any estimator for such specification will be biased. However, the Wald estimator can be easily modified to account for the path of instruments and hence recover local lag-0 dynamic causal effects.

I propose a simple two-stage Horvitz-Thompson estimator, which is built using a propensity score for the path of the instrument[8]

The nonparametric estimator for $\tau_{i,t}(1,0;0)$ is

$$\widehat{\tau}_{i,t}(1,0;0) = \frac{\widehat{\tau}^{RF}_{i,t}(1,0;0)}{\widehat{\tau}^{FS}_{i,t}(1,0;0)}$$

where

$$\widehat{\tau}^{RF}_{i,t}(1,0;0) = \frac{Z_{i,t}Y_{i,t}}{\pi_{i,t}(1)} - \frac{(1-Z_{i,t})Y_{i,t}}{\pi_{i,t}(0)}$$
$$\widehat{\tau}^{FS}_{i,t}(1,0;0) = \frac{Z_{i,t}D_{i,t}}{\pi_{i,t}(1)} - \frac{(1-Z_{i,t})D_{i,t}}{\pi_{i,t}(0)}$$

Both the time-t lag-0 dynamic causal effect and the total lag-0 dynamic causal effect can

[8]For now, the propensity score is known, in Section 5.3 I discuss inference when the propensity score for the path needs to be estimated.

be estimated by plugging in $\widehat{\tau}_{i,t}(1,0)(0)$:

$$\widehat{\overline{\tau}}_{C_t,t}(1,0;0) = \frac{\frac{1}{N}\sum_{i=1}^{N}\widehat{\tau}_{i,t}^{RF}(1,0;0)}{\frac{1}{N}\sum_{i=1}^{N}\widehat{\tau}_{i,t}^{FS}(1,0;0)}$$
$$\widehat{\overline{\tau}}_{C_t}(1,0;0) = \frac{\frac{1}{NT}\sum_{t=1}^{T}\sum_{i=1}^{N}\widehat{\tau}_{i,t}^{RF}(1,0;0)}{\frac{1}{NT}\sum_{t=1}^{T}\sum_{i=1}^{N}\widehat{\tau}_{i,t}^{FS}(1,0;0)}$$

Theorem 5 shows that the estimators are unbiased over the randomization distribution, and asymptotically normal as the population size grows larger.

Theorem 3 *Suppose that potential outcomes are bounded. Under Assumptions 1-6,*

$$\frac{\sqrt{N}\left\{\widehat{\overline{\tau}}_{C_t,t}(1,0;0) - \overline{\tau}_{C_t,t}(1,0;0)\right\}}{\sigma_t(1,0;0)} \xrightarrow{d} \mathcal{N}(0,1),\ as\ N \to \infty$$

and

$$\frac{\sqrt{NT}\left\{\widehat{\overline{\tau}}_{C_t}(1,0;0) - \overline{\tau}_{C_t}(1,0;0)\right\}}{\sigma(1,0;0)} \xrightarrow{d} \mathcal{N}(0,1),\ as\ NT \to \infty$$

where $\sigma_t(1,0;0)$ and $\sigma(1,0;0)$ are defined in Section 4 of Appendix A.

The variances of $\widehat{\overline{\tau}}_{C_t,t}(1,0;0)$ and $\overline{\tau}_{C_t}(1,0;0)$ are the appropriate averages of the variance of $\widehat{\overline{\tau}}_{C_t,t}(1,0;0)$, which are generally not estimable as they depends on individual potential outcomes and potential treatments under both treatment and counterfactual. However, Lemma 8 in Appendix B shows that the variance of the reduced form and the first stage are bounded from above by a term that is estimable.

For hypothesis testing, I propose the estimation of a conservative Bloom (1984) confidence interval, built with estimates of the upper bound of the variance of the reduced form, and the square of the estimate of the first stage.

The upper bound for the variance of the estimator of the reduced form $\widehat{\tau}_{i,t}^{RF}(1,0;0)$ is

$$\left(\gamma_{i,t}^{RF}(1,0;0)\right)^2 = \frac{Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))^2}{\pi_{i,t}(1)} + \frac{Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))^2}{\pi_{i,t}(0)}$$

The resulting $1-\alpha$ confidence intervals for the time-t lag-0 dynamic causal effect and the total lag-0 dynamic causal effect are, respectively,

$$\widehat{\overline{\tau}}_{C_t,t}(1,0;0) \pm z_{1-\alpha/2}\sqrt{\left\{\frac{\frac{1}{N}\sum_{i=1}^{N}\left(\gamma_{i,t}^{RF}(1,0;0)\right)^2}{\left(\frac{1}{N}\sum_{i=1}^{N}\widehat{\tau}^{FS}(1,0;0)\right)^2}\right\}}$$

$$\widehat{\overline{\tau}}_{C_t}(1,0;0) \pm z_{1-\alpha/2}\sqrt{\left\{\frac{\frac{1}{NT}\sum_{t=1}^{T}\sum_{i=1}^{N}\left(\gamma_{i,t}^{RF}(1,0;0)\right)^2}{\left(\frac{1}{NT}\sum_{t=1}^{T}\sum_{i=1}^{N}\widehat{\tau}^{FS}(1,0;0)\right)^2}\right\}}$$

Bloom (1984) intervals exhibit good performance in terms of coverage rates for compliance rates greater than 10%. See Kang et al. (2018) for a thorough discussion about inference using IV in cross-sectional settings.

5.2 Nonparametric Estimator for Dynamic Causal Effects

For the general lag-p dynamic causal effect, I propose the separate estimation of lag-p dynamic response functions through a multiple-differences Horvitz-Thompson type of estimator. That is, $\widehat{\tau}_{i,t}(\mathbf{d},\tilde{\mathbf{d}};p) = \widehat{m}_{i,t}(\mathbf{d}) - \widehat{m}_{i,t}(\tilde{\mathbf{d}})$, where $\widehat{m}_{i,t}(\mathbf{d}) = \frac{\widehat{m}_{i,t}^{RF}(\mathbf{d})}{\widehat{m}_{i,t}^{FS}(\mathbf{d})}$ with

$$\widehat{m}_{i,t}^{RF}(\mathbf{d}) = \Delta^{p+1}\left(\frac{\mathbf{1}\left\{Z_{i,t-p:t} = \mathbf{z}\right\}\mathbf{1}\left\{D_{i,t-p:t} = \mathbf{d}\right\}Y_{i,t}}{\pi_{i,t-p:t}(\mathbf{z})}\right)$$
$$\widehat{m}_{i,t}^{FS}(\mathbf{d}) = \Delta^{p+1}\left(\frac{\mathbf{1}\left\{Z_{i,t-p:t} = \mathbf{z}\right\}\mathbf{1}\left\{D_{i,t-p:t} = \mathbf{d}\right\}}{\pi_{i,t-p:t}(\mathbf{z})}\right)$$

Plugging the unit i, period t estimates for the lag-p local response function as shown in Section 5.1 leads to estimates of the time-t lag-p local response function and the total lag-p

response function. The local lag-p response functions are estimated, respectively, by

$$\widehat{m}_{t,(\mathbf{d})} = \frac{\frac{1}{N}\sum_{i=1}^{N}\widehat{m}_{i,t}^{RF}(\mathbf{d})}{\frac{1}{N}\sum_{i=1}^{N}\widehat{m}_{i,t}^{FS}(\mathbf{d})}$$
$$\widehat{m}_{(\mathbf{d})} = \frac{\frac{1}{N(T-p)}\sum_{t=p+1}^{T}\sum_{i=1}^{N}\widehat{m}_{i,t}^{RF}(\mathbf{d})}{\frac{1}{N(T-p)}\sum_{t=p+1}^{T}\sum_{i=1}^{N}\widehat{m}_{i,t}^{FS}(\mathbf{d})}$$

The appropriate lag-p dynamic causal effects are generated by the difference of the estimates for the response functions. Theorem 5 shows that the estimators are unbiased over the randomization distribution, and asymptotically normal as the population size grows larger.

Theorem 4 *Suppose that potential outcomes are bounded. Under Assumptions 1-6,*

$$\frac{\sqrt{N}\left\{\widehat{\overline{\tau}}_{C_{t-p:t},t}(\boldsymbol{d},\tilde{\boldsymbol{d}};p) - \overline{\tau}_{C_{t-p:},t}(\boldsymbol{d},\tilde{\boldsymbol{d}};p)\right\}}{\sigma_t(\boldsymbol{d},\tilde{\boldsymbol{d}};p)} \xrightarrow{d} \mathcal{N}(0,1),\ as\ N\to\infty$$

and

$$\frac{\sqrt{N(T-P)}\left\{\widehat{\overline{\tau}}_{C_{t-p:t}}(\boldsymbol{d},\tilde{\boldsymbol{d}};p) - \overline{\tau}_{C_{t-p:t}}(\boldsymbol{d},\tilde{\boldsymbol{d}};p)\right\}}{\sigma(\boldsymbol{d},\tilde{\boldsymbol{d}};p)} \xrightarrow{d} \mathcal{N}(0,1),\ as\ NT\to\infty$$

where $\sigma_t(\boldsymbol{d},\tilde{\boldsymbol{d}};p)$ and $\sigma(\boldsymbol{d},\tilde{\boldsymbol{d}};p)$ are defined in Section 5 of Appendix A.

For hypothesis testing, conservative Bloom (1984) confidence intervals are constructed using the upper bound for the variance of the reduced forms for the lag-p response functions and estimates of the first stage for the lag-p response functions.

The upper bound for the variance of the estimator of the reduced form is

$$= \sum_{\mathbf{z}\in\{0,1\}^{p+1}} \frac{\left(Y_{i,t}(d_{1:t-p-1}^{obs},\mathbf{d})\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})=\mathbf{d}\right\}\right)^2}{\pi_{i,t-p:t}(\mathbf{z})} + \sum_{\mathbf{z}\in\{0,1\}^{p+1}} \frac{\left(Y_{i,t}(d_{1:t-p-1}^{obs},\tilde{\mathbf{d}})\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})=\tilde{\mathbf{d}}\right\}\right)^2}{\pi_{i,t-p:t}(\mathbf{z})}$$
$$\left(\gamma_{i,t}^{RF}(\mathbf{d})\right)^2 + \left(\gamma_{i,t}^{RF}(\tilde{\mathbf{d}})\right)^2$$

The resulting $1-\alpha$ confidence intervals for the time-t lag-p dynamic causal effect and the total lag-p dynamic causal effect are, respectively,

$$\widehat{\overline{\tau}}_{C_{t-p:t},t}(\mathbf{d},\tilde{\mathbf{d}};p) \pm z_{1-\alpha/2}\sqrt{\left\{\frac{\frac{1}{N}\sum_{i=1}^{N}\left(\gamma_{i,t}^{RF}(\mathbf{d})\right)^2+\left(\gamma_{i,t}^{RF}(\tilde{\mathbf{d}})\right)^2}{\frac{1}{N}\sum_{i=1}^{N}\left(\widehat{m}_{i,t}^{FS}(\mathbf{d})\right)^2+\left(\widehat{m}_{i,t}^{FS}(\tilde{\mathbf{d}})\right)^2}\right\}}$$

$$\widehat{\overline{\tau}}_{C_{t-p:t}}(\mathbf{d},\tilde{\mathbf{d}};p) \pm z_{1-\alpha/2}\sqrt{\left\{\frac{\frac{1}{N(T-p)}\sum_{t=p+1}^{T}\sum_{i=1}^{N}\left(\gamma_{i,t}^{RF}(\mathbf{d})\right)^2+\left(\gamma_{i,t}^{RF}(\tilde{\mathbf{d}})\right)^2}{\frac{1}{N(T-p)}\sum_{t=p+1}^{T}\sum_{i=1}^{N}\left(\widehat{m}_{i,t}^{FS}(\mathbf{d})\right)^2+\left(\widehat{m}_{i,t}^{FS}(\tilde{\mathbf{d}})\right)^2}\right\}}$$

5.3 Inference When the Propensity Score is Unknown

So far, in the construction of the estimators the propensity score for the path of instruments is assumed to be known. Although such an assumption is reasonable in experimental settings, it is inadequate for most observational studies in which the propensity score needs to be estimated. In such cases, the feasible nonparametric estimator is built using estimates for the propensity score. That is,

$$\widehat{m}_{i,t}^{RF}(\mathbf{d}) = \Delta^{p+1}\left(\frac{\mathbf{1}\left\{Z_{i,t-p:t}=\mathbf{z}\right\}\mathbf{1}\left\{D_{i,t-p:t}=\mathbf{d}\right\}Y_{i,t}}{\widehat{\pi}_{i,t-p:t}(\mathbf{z})}\right)$$
$$\widehat{m}_{i,t}^{FS}(\mathbf{d}) = \Delta^{p+1}\left(\frac{\mathbf{1}\left\{Z_{i,t-p:t}=\mathbf{z}\right\}\mathbf{1}\left\{D_{i,t-p:t}=\mathbf{d}\right\}}{\widehat{\pi}_{i,t-p:t}(\mathbf{z})}\right)$$

Next, I assume there is an unbiased estimator for the propensity score that converges at the $\sqrt{N}$-rate.

Assumption 7 (Estimator for the Propensity Score): For all $i \in [N]$, $t \in [T]$, $\mathbf{z} \in \{0,1\}^{p+1}$ there is an estimator $\widehat{\pi}_{i,t-p}(\mathbf{z})$ such that

$$\mathbb{E}\left[\widehat{\pi}_{i,t-p}(\mathbf{z})\right] = \pi_{i,t-p}(\mathbf{z}), \frac{1}{N}\sum_{i=1}^{N}\widehat{\pi}_{i,t-p}(\mathbf{z}) - \frac{1}{N}\sum_{i=1}^{N}\pi_{i,t-p}(\mathbf{z}) = O_p(N^{-1/2})$$

Although the estimator relies on an estimate for the propensity score, as long as it is unbiased and converges at the $\sqrt{N}$ rate, it will have the asymptotic distribution as the one with the known propensity score as the population size grows to infinity. See Appendix C for the result.

6 Extensions

In this section, I briefly discuss some extensions and how to modify the estimand and estimators in order to correctly account for the modifications in the identifying assumptions.

6.1 Covariates

Most applications of IV methods with panel rely on conditional versions of the identifying assumptions, whether they are baseline or time-varying. For example, it is common in multi-period experiments to assume that assignment to treatment is randomized within strata of baseline covariates. In that case, the sequential randomization assumption is modified to take the following form. Let X denote a vector of baseline covariates (e.g. gender, race). Then, sequential randomization holds conditional on covariates if $\mathbb{P}\left(Z_{i,t} = z_t | Z_{1:N,1:T}, X_{1:N}, \mathcal{F}_{1:N,1:T}\right) = \mathbb{P}\left(Z_{i,t} = z_t | Z_{i,1:t-1} = z_{1:t-1}, X_i = x\right)$, for all $i \in [N]$, $t \in [T]$.

When covariates are time-varying, they can be incorporated into the sequential randomization assumption in two ways. The first is to assume that sequential randomization holds conditional on contemporary covariates. In such cases, the randomization distribution can be expressed as $\mathbb{P}\left(Z_{i,t} = z_t | Z_{1:N,1:T}, X_{1:N,1:T}, \mathcal{F}_{1:N,1:T}\right) = \mathbb{P}\left(Z_{i,t} = z_t | Z_{i,1:t-1} = z_{1:t-1}, X_{i,t} = x_t\right)$, for all $i \in [N]$, $t \in [T]$.

If sequential randomization holds conditional on the path of covariates until period t, then the distribution can be expressed as

$$\mathbb{P}\left(Z_{i,t} = z_t | Z_{1:N,1:T}, X_{1:N,1:T}, \mathcal{F}_{1:N,1:T}\right) = \mathbb{P}\left(Z_{i,t} = z_t | Z_{i,1:t-1} = z_{1:t-1}, X_{i,1:t} = x_{1:t}\right)$$

for all $i \in [N]$, $t \in [T]$. In all of three cases, the estimand can be easily modified by adequately incorporating the covariates in the adapted propensity score $\pi_{t-p}(\mathbf{z})$. Such procedure can be interpreted as a nonparametric analogue of the so-called 'g-formula' (see Hernan and Robins (2023) for a review).

6.2 Intermediary Realizations

Another common specification in dynamic treatment settings is to assume that sequential randomization holds conditional on the path of all realizations, that is, $Z_{i,t}$ is independent from the panel of potential outcomes and treatments conditional on the path of instruments, treatments, and outcomes until period $t-1$:

$$\mathbb{P}\left(Z_{i,t} | Z_{1:N,1:T}, \mathcal{F}_{1:,N,1:T}\right) = \mathbb{P}\left(Z_{i,t} | Z_{i,1:t-1} = z_{1:t-1}, D_{i,1:t-1} = d_{1:t-1}, Y_{i,1:t-1} = y_{1:t-1}\right)$$

In such cases, the identification of the first stage of treatment paths becomes more challenging, as the identification of the joint distribution of treatments as proposed in Sections 3 and 4 is no longer valid if there are treatment effects from previous periods. However, the procedure can be modified, and the path of compliers can be identified using the general product rule.

Consider the leading case of $T = 2$. Note that there is no history affecting the first stage in period 1. Hence, potential treatments are readily identified by $\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(z_1)$ and can readily be estimated using the Horvitz-Thompson estimator $\frac{1}{N}\sum_{i=1}^{N} \frac{D_{i,1}\mathbf{1}\{Z_{i,1}=z_1\}}{\pi_{i,1}(z_1)}$.

$$\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1}\mathbf{1}\left\{D_{i,2}(z_2)=1\right\}=\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1}D_{i,2}(z_2)=\int_{y_1}\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1,Y_{i,1}=y_1}D_{i,2}(z_2)dF_{Y_{i,1}}(y_1)$$

Hence, the potential treatment path $D_{i,1}(z_1)=1, D_{i,2}(z_2)=1$ is identified using a design-based analogue of the general product rule:

$$\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,1}(z_1)=1, D_{i,2}(z_2)=1\right\}=\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1}\mathbf{1}\left\{D_{i,2}(z_2)=1\right\}\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,1}(z_1)=1\right\}$$

Once, these quantity is identified, identification of compliers for the path follows the same "difference-in-differences" format as presented in Section 3.

When it comes to the reduced form, note that

$$\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1,Y_{i,1}=y_1}D_{i,2}(z_2)Y_{i,2}=\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1,Y_{i,1}=y_1}Y_{i,2}(1,1)\mathbf{1}\left\{D_{i,2}(z_2)=1\right\}$$

And, therefore, the reduced form of the potential outcome associated to full exposure for individuals with assignment path (z_1, z_2) is identified by

$$\frac{1}{N}\int_{y_1}\sum_{i:D_{i,1}(z_1)=1,Y_{i,1}=y_1}D_{i,2}(z_2)Y_{i,2}dF_{Y_{i,1}}(y_1)\mathbf{1}\left\{D_{i,1}(z_1)=1\right\}=\frac{1}{N}\sum_{i:D_{i,1}(z_1)=1}D_{i,2}(z_2)Y_{i,2}\mathbf{1}\left\{D_{i,1}(z_1)=1\right\}$$

And one can proceed with Wald-like modified "difference-in-differences" to identify the local lag-p response function.

Hence, the Horvitz-Thompson estimator can be easily modified to account for the alternative sequential randomization assumption. Consider, once again, the estimation of the local lag-p response function associated to full exposure, $Y_{i,2}(1,1)$. This time, we

work with two different propensity scores instead of the generalized propensity score. Let $\pi_{i,1}(\mathbf{z}_1) = \mathbb{P}(Z_{i,1} = \mathbf{z}_1)$ and $\pi_{i,2}(\mathbf{z}_2) = \mathbb{P}(Z_{i,2} = \mathbf{z}_2 | Y_{i,1} = y_1, D_{i,1}(\mathbf{z}_1) = 1)$. Then, the first stage is consistently estimated by

$$\widehat{m}^{FS}_{t,(1,1)} = \frac{1}{N}\sum_{i=1}^{N} \Delta^2 \left(\frac{D_{i,2}\mathbf{1}\{Z_{i,2} = \mathbf{z}_2\}}{\pi_{i,2}(\mathbf{z}_2)} \frac{D_{i,1}\mathbf{1}\{Z_{i,1} = \mathbf{z}_1\}}{\pi_{i,1}(\mathbf{z}_1)} \right)$$

and the reduced form is estimated by

$$\widehat{m}^{RF}_{t,(1,1)} = \frac{1}{N}\sum_{i=1}^{N} \Delta^2 \left(\frac{D_{i,1}D_{i,2}Y_{i,2}\mathbf{1}\{Z_{i,1:2} = (\mathbf{z}_1, \mathbf{z}_2)\}}{\pi_{i,2}(\mathbf{z}_2)\pi_{i,1}(\mathbf{z}_1)} \right)$$

Therefore, estimation and inference can be easily adapted to different versions of the sequential randomization assumption.

7 Monte Carlo Simulations

In this section, I show the desirable finite-sample properties of the proposed nonparametric estimator for the local dynamic causal effects.

For the sake of simplicity, I consider a balanced panel setting with $N = 1000$ and $T = 2$. The simulation focuses on the lag-p dynamic causal effects with $p = t$, that is, the dynamic causal effects associated to the whole treatment path in the setting. I simulate a continuous covariate that has a Gaussian distribution and a binary treatment instrument that is sequentially randomized following a Bernoulli distribution:

$$\mathbb{P}(Z_{i,t} = z_t | Z_{i,1:t-1} = z_{1:t-1}, Y_{i,1:t-1} = y_{1:t-1}, X_i = x) \propto \prod_{i \in [N]} p_{i,t}^{z_{i,t}} (1 - p_{i,t})^{1 - z_{i,t}}$$

I set $p_{i,t} = p_i = 0.6$. For the choice model. Outcomes in period t are specified to have the following linear working model:

$$Y_{i,t} = \delta_t' Y_{i,1:t-1} + \beta_{1:t}' D_{i,1:t} + \theta X_i + U_t(D_{i,1:t})$$

I set $\beta_t = 1$ and $\beta_{1:t-1} = 0.5$.

In Table 1, I compare the performance of the proposed Horvitz-Thompson estimator (HT) with the performance of the static 2SLS specification (2SLS), taking the local lag-0 dynamic causal effect as the target parameter.

Table 3: Simulation results for the Lag-0 dynamic causal effect

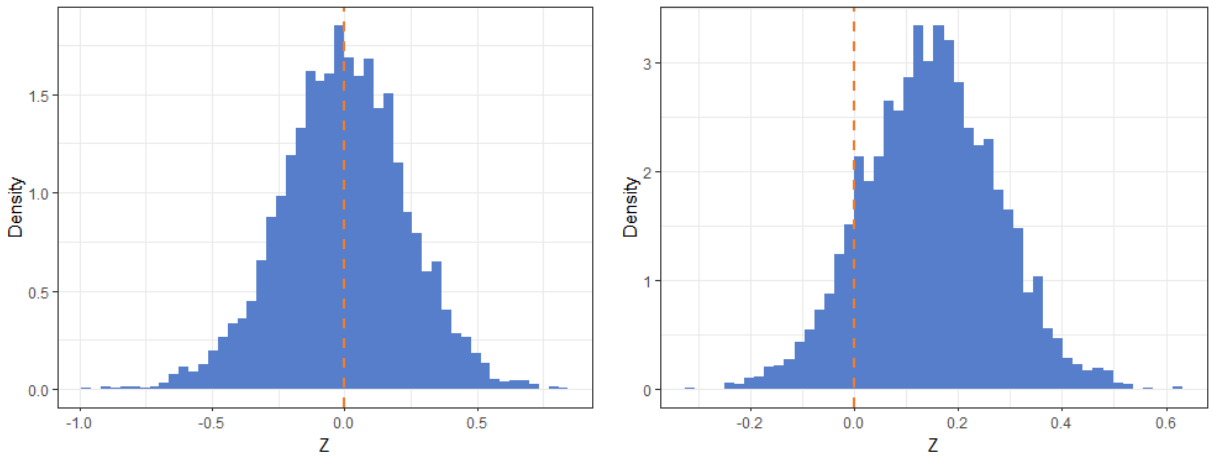
Time-t effect	t=1		t=2		Total	
Estimator	HT	2SLS	HT	2SLS	HT	2SLS
Av. Bias	-0.003	0.003	0.027	0.368	0.015	0.156
Med. Bias	-0.002	0.003	0.034	0.392	0.019	0.163
RMSE	0.097	0.081	0.119	0.484	0.237	0.169
Cover	0.944	0.948	0.952	0.612	0.955	0.784
CIL	0.356	0.223	0.508	0.285	0.441	0.258

Note: Simulations based on 10.000 Monte Carlo experiments with sample size $N = 1.000$ and $T = 2$. CIs for the 2SLS estimator were built using the standard estimated variance using the Delta Method.

The first two columns show the results for the first period. When $t = 1$, dynamic plays no role in the model. Hence, both the nonparametric estimator and the static 2SLS show little to none Monte Carlo bias. Moreover, the coverage is close to the desired 95%, with the 2SLS estimator showing a tighter Confidence Interval on average. When it comes to the second period, the Horvitz-Thompson estimator remains unbiased in the Monte Carlo exercise, as shown by the third column. The static 2SLS estimator is severely biased, with coverage far from the desired 95%. The last two columns stack the lag-0 estimates across the two time periods. Thus, the results can be interpreted as a weighted average of the time-t results, which explains why the performance of the 2SLS estimator is better than in the second period alone. As the number of periods grows larger, however, one should expect the 2SLS estimator to perform increasingly worse with the number of time periods.

In Table 2, I present the simulation results for different time-t local lag-1 dynamic causal effects. The lag-1 effects can only be estimated for period 2. I present results for the "difference-in-differences" modified Horvitz-Thompson estimator (HT) and a multivariate

Figure 1: Simulated Randomization Distributions



Note: The figure on the left shows the simulated randomization distribution of the Horvitz-Thompson estimator for the total local lag-0 dynamic causal effect. The figure on the right shows the simulated randomization distribution of the static 2SLS estimator for the total local lag-0 dynamic causal effect. Simulations based on 10.000 Monte Carlo experiments with sample size $N = 1.000$ and $T = 2$. CIs for the 2SLS estimator were built using the standard estimated variance using the Delta Method.

2SLS estimator (MV2SLS) such as the one specified in Section 3. I consider the performance of the estimators with respect to effects of full exposure in the first two columns, exposure in the second period in the third and fourth columns, and exposure in the first period in the last two columns.

Unsurprisingly, the multivariate 2SLS specification yields substantially biased estimates for the three different target parameters. Coverage is closer to the desired 95% than in the simulations for the static 2SLS. However, it is never greater than 81.5%.

Figure 1 plots the randomization distribution for total lag-0 dynamic effect estimated by the HT estimator (left) and the 2SLS estimator (right). Both estimators converge to a normal distribution under the Monte Carlo exercise. However, the distribution of the 2SLS estimator is clearly not centered around the true parameter.

The proposed nonparametric estimator exhibits great Monte Carlo performance. The average bias, median bias and root mean-squared error for the causal effects are small, and the coverage of the conservative confidence interval is close to the desired 95% coverage. Confidence interval lengths are fairly stable across the considered treatment paths.

Overall, the Monte Carlo Simulations assert the desirable finite-sample performance of

Table 4: Simulation results for the Lag-1 dynamic causal effects

Lag-1 effect	((1,1),(0,0))		((0,1),(0,0))		((1,0),(0,0))	
Estimator	HT	MV2SLS	HT	MV2SLS	HT	MV2SLS
Av. Bias	-0.0008	0.1968	-0.0039	0.1306	-0.0051	0.1641
Med. Bias	-0.0012	0.1970	0.0002	0.1312	-0.0046	0.1646
RMSE	0.0772	0.1975	0.0762	0.1404	0.0845	0.1937
Cover	0.938	0.813	0.956	0.798	0.928	0.815
CIL	0.8721	0.3019	0.8023	0.3145	0.8674	0.3222

Note: Simulations based on 10.000 Monte Carlo experiments with sample size $N = 1.000$ and $T = 2$. CIs for the 2SLS estimator were built using the standard estimated variance using the Delta Method.

the proposed estimators for dynamic causal effects over the randomization, while bringing evidence of the inadequacy of the standard 2SLS methods in the presence of time-varying heterogeneity.

8 Application: Law Enforcement and Deforestation

8.1 AGR (2023)

In this section, I revisit the work of Assunção et al. (2023) (hereafter referred to as AGR, 2023). The authors investigate the effects of a satellite law enforcement program, the DETER program, on deforestation in the Brazilian Amazon.

In 2004, the Brazilian government launched the Action Plan for the Prevention and Control of Deforestation in the Legal Amazon (PPCDAm). In the early 2000s, Brazil was the country that cleared the most tropical forest area in both absolute and relative terms (Hansen et al., 2008). The program was a response to the growing concerns regarding the dangers of deforestation of tropical forests.

One of the main innovations of the PPCDAm was the development of a satellite-based system that regularly collected and processed georeferenced imagery on Amazon land cover to detect forest loss, the Real-Time System for Detection of Deforestation (DETER). Deforestation alerts that came from DETER imagery became the cornerstone for law enforcement. Officers visited alert sites and applied sanctions if evidence of illegal deforestation was found.

Law enforcement officers would typically issue a fine for every environmental infraction they detected, but other forms of penalty, such as he seizure and destruction of products and equipment, are also contemplated by law.

AGR (2023) estimate the effects of law enforcement, as a function of the number of fines issued by the DETER satellite, on deforestation using panel data from 2006 to 2016. To address the issue of reverse causality between deforestation and law enforcement, the authors use an IV approach, leveraging variation in cloud coverage as exogenous variation on law enforcement. The intuition behind the strategy is that cloud coverage blocks visibility in satellite imagery and thereby limits DETER's capacity to detect changes in land cover patterns and therefore law enforcement.

8.2 Serial Correlation of the Instrument

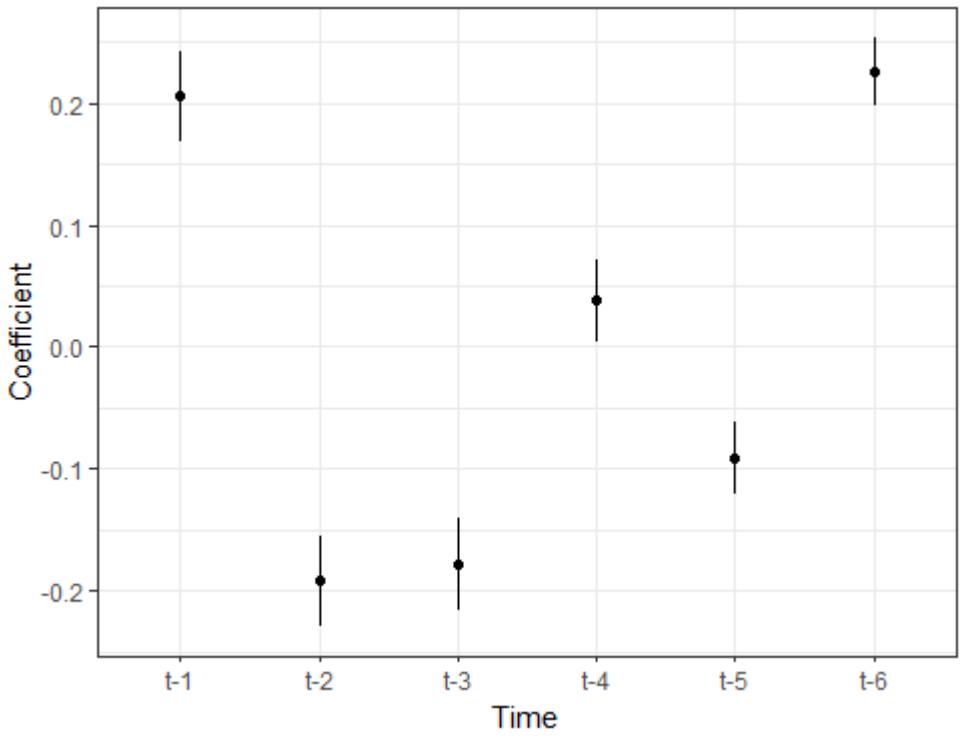
The 2SLS regressions from AGR (2023) can be mapped to the static Wald estimator as presented in Section 3.1, with the introduction of covariates. From proposition 1, it follows that the estimates are unbiased as long as cloud coverage in a given period is independent from cloud coverage at previous periods.

Figure 1 plots the estimates of the coefficients and the confidence intervals associated to serial correlation of the instrument in a regression where cloud coverage in period t is the dependent variable, and lags of cloud coverage and municipal fixed effects are the regressors. The coefficients ate statistically significant up to the 6-th lag. Therefore, the estimates for AGR are biased if either there are no dynamic effects of law enforcement or cloud coverage previous to period t do not affect law enforcement before period t.

8.3 Dynamic Effects of Law Enforcement

To address the issue of serial correlation of the instrument, I use the multiple-differences Horvitz-Thompson estimator presented in Section 5.2 to estimate the impulse response functions in order to analyze the dynamics effects of law enforcement. Since the procedure is

Figure 2: Serial Correlation of Cloud Coverage



Note: Figure 2 plots the results of the regression of cloud coverage in period t vs cloud coverage in previous periods and municipal fixed effects.

suited for binary instruments and binary treatments, I binarize the treatment creating an indicator of "intense" law enforcement for municipalities in which the number of fines in a given year is greater than that year's average, and a binary instrument for "intense" cloud coverage under the same reasoning. The binarized results can be interpreted under the light of Schuler et al. (2024)[9].

Table 5 presents the results. The dependent variable is the ratio between a municipality's deforested area in a year and its total area. The mean of the ratio for the whole panel is about 0.007. The first column of Table 1 present the results from AGR (2023). The estimate should be interpreted as a "naive" estimate for the lag-0 dynamic causal effect. The second column shows the estimated lag-0 dynamic causal effect using the HT estimator. The estimated causal effect is over two times greater than the one from AGR (2023). The difference in the results, however, cannot be interpreted solely as the correction for OVB since the authors work with a continuous version of the treatment and a linear specification for first stage and reduced form. Nevertheless, the results in column 2 corroborate the initial finding that law enforcement successfully curbs deforestation, even when accounting for serial correlation in the instrument.

Table 5: Dynamic Effects of Law Enforcement: Impulse Response Functions

		lag-p			
	AGR(2023)	0	1	2	3
Point estimate	-0.0244	-0.0513	-0.0211	0.0334	0.0126
95% CI	(-0.049, -0.001)	(-0.102, -0.001)	(-0.042, -0.001)	(-0.077, 0.120)	(-0.029, 0.034)
Baseline	0.0071	0.0071	0.0071	0.0071	0.0071
Observations	5210	5210	4689	4168	3647
Municipalities	521	521	521	521	521

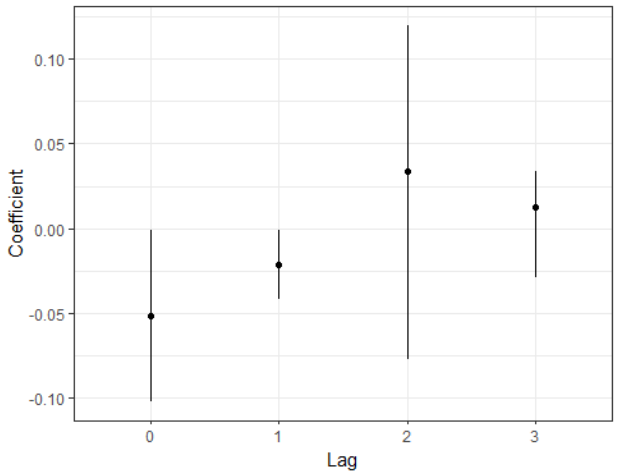
Note: The dependent variable is the ratio between deforested area in a year and the municipality area. The set of control variables contains precipitation and temperature (weather), PRODES cloud coverage and other nonobservable areas (satellite visibility), and agricultural commodity prices.

Column 3 presents the estimate for the lag-1 impulse response function. The estimated coefficient shows that lagged law enforcement also successfully curbs deforestation. Thus,

[9]As a robustness check, I use the estimator under different binarization procedures. See Appendix D for the results.

it can be interpreted as evidence of dynamic effects of law enforcement. Columns 4 and 5 present the results of the impulse response functions for the lag-2 and lag-3 dynamic causal effects. The estimates, however, are not statistically significant.

Figure 3: Event-Study: Dynamic Effects of Law Enforcement



Note: Figure 3 plots the results displayed in the columns 2-5 from Table 5.

The interpretation of these results is not straightforward, however. One could interpret the evidence of dynamic effects of law enforcement as evidence of persistence of deterrence effects through time. Law enforcement curbs deforestation by causing potential offenders to update their beliefs about the probability of getting caught and thus their expected costs from engaging in the illegal activity. However, the effect fades out, which could indicate that law enforcement does not provide a permanent shock on the perception of potential offenders.

On the other hand, law enforcement could have lead to to the loss of capital goods and machinery used in deforestation, which may have reduced potential offenders' ability to commit future offenses. If that's the case, the observed pattern of the dynamic effects of law enforcement could be suggesting that law enforcement affects the means to engage in illegal deforestation, but with time potential offenders reequip themselves with the necessary machinery and go back to their illegal deforestation activities. The empirical strategy does not allow us to disentangle the underlying mechanisms driving the results and that should

is an effort for future research.

Still, the results presented contribute to the literature on the enforcement of environmental regulation in developing countries. While Greenstone and Hanna (2014) and AGR (2023) study such phenomenon, to the extent of my knowledge this is the first paper analyzing the dynamics effects of environmental regulation. Dynamics are particularly important in the context of environmental regulation, as understanding how the effects of regulation vary with time is fundamental for improving the efforts against illegal deforestation. Yet, further inquiries are necessary in order to understand how to improve the efforts against deforestation in the Brazilian Amazon, since the mechanisms driving the effects are not clear.

9 Conclusion

This paper develops a finite-population framework for the identification of dynamic causal effects using instrumental variables with panel data.

First, I show that the usual approach to IV settings with panel data, which I called the static Wald estimator, does not generally estimates well define causal parameters unless the instrument is fully randomized at each period of time. I show that Multivariate 2SLS is also not generally valid.

Second, I present a modification of the Wald estimator that identifies contemporary treatment effects under sequential randomization, and provide a general framework for the identification of dynamic causal effects in which mean potential outcomes are identified separately. I introduce a nonparametric estimator, and derive its finite population asymptotic distribution.

I then show how covariates and intermediary outcomes and treatments can be introduced in the framework by accounting for them in the randomization distribution.

Monte Carlo simulation studies assert the desirable finite-sample property of the estimators for both the local and the marginal dynamic causal effects.

Finally, I use the estimators to analyze the dynamic effects of law enforcement on illegal deforestation in the Brazilian Amazon. Results suggest that there is substantial time-varying heterogeneity in the effects of law-enforcement and that while the effects of law enforcement on illegal deforestation persist for some time, they are not permanent.

References

Abadie, A., Athey, S., Imbens, G. W., and Wooldridge, J. M. (2020). Sampling-based versus design-based uncertainty in regression analysis. *Econometrica*, 88(1):265–296.

Abbring, J. H. and Heckman, J. J. (2007). Chapter 72 econometric evaluation of social programs, part iii: Distributional treatment effects, dynamic treatment effects, dynamic discrete choice, and general equilibrium policy evaluation. volume 6 of *Handbook of Econometrics*, pages 5145–5303. Elsevier.

Acemoglu, D., Naidu, S., Restrepo, P., and Robinson, J. A. (2019). Democracy does cause growth. *Journal of Political Economy*, 127(1):47–100.

Aghion, P., Akcigit, U., Bergeaud, A., Blundell, R., and Hemous, D. (2018). Innovation and Top Income Inequality. *The Review of Economic Studies*, 86(1):1–45.

Arellano, M. and Bond, S. (1991). Some Tests of Specification for Panel Data: Monte Carlo Evidence and an Application to Employment Equations. *The Review of Economic Studies*, 58(2):277–297.

Arkhangelsky, D. and Imbens, G. W. (2022). Doubly robust identification for causal panel data models. *The Econometrics Journal*, 25(3):649–674.

Assunção, J., Gandour, C., and Rocha, R. (2023). Deter-ing deforestation in the amazon: Environmental monitoring and law enforcement. *American Economic Journal: Applied Economics*, 15(2):125–56.

Blanchard, E. and Matschke, X. (2015). U.S. Multinationals and Preferential Market Access. *The Review of Economics and Statistics*, 97(4):839–854.

Bloom, H. S. (1984). Accounting for no-shows in experimental evaluation designs. *Evaluation Review*, 8(2):225–246.

Bojinov, I., Rambachan, A., and Shephard, N. (2021). Panel experiments and dynamic causal effects: A finite population perspective. *Quantitative Economics*, 12(4):1171–1196.

Callaway, B. and Sant'Anna, P. (2021). Difference-in-Differences with multiple time periods. *Journal of Econometrics*, 225(2):200–230.

Conduct Problems Prevention Research Group, . (1992). A developmental and clinical model for the prevention of conduct disorder: The fast track program. *Development and Psychopathology*.

de Chaisemartin, C. and Lei, Z. (2023). More robust estimators for instrumental-variable panel designs, with an application to the effect of imports from china on us employment. *Working Paper*.

Goodman-Bacon, A. (2021). Difference-in-differences with variation in treatment timing. *Journal of Econometrics*, 225(2):254–277.

Greenstone, M. and Hanna, R. (2014). Environmental regulations, air and water pollution, and infant mortality in india. *American Economic Review*, 104(10):3038–72.

Hahn, J. (1998). On the role of the propensity score in efficient semiparametric estimation of average treatment effects. *Econometrica*, 66(2):315–331.

Han, S. (2021). Identification in nonparametric models for dynamic treatment effects. *Journal of Econometrics*, 225(2):132–147. Themed Issue: Treatment Effect 1.

Han, S. (2023). Optimal dynamic treatment regimes and partial welfare ordering. *Journal of the American Statistical Association*, 0(0):1–11.

Hansen, M. C., Stehman, S. V., Potapov, P. V., Loveland, T. R., Townshend, J. R. G., DeFries, R. S., Pittman, K. W., Arunarwati, B., Stolle, F., Steininger, M. K., Carroll, M., and DiMiceli, C. (2008). Humid tropical forest clearing from 2000 to 2005 quantified by using multitemporal and multiresolution remotely sensed data. *Proceedings of the National Academy of Sciences*, 105(27):9439–9444.

Heckman, J. J., Humphries, J. E., and Veramendi, G. (2016). Dynamic treatment effects. *Journal of Econometrics*, 191(2):276–292. Innovations in Measurement in Economics and Econometrics.

Heckman, J. J. and Navarro, S. (2007). Dynamic discrete choice and dynamic treatment effects. *Journal of Econometrics*, 136(2):341–396. The interface between econometrics and economic theory.

Hernan, M. A. and Robins, J. M. (2023). Causal inference: What if. volume 1, pages 1–312. CRC Press.

Hirano, K., Imbens, G. W., and Ridder, G. (2003). Efficient estimation of average treatment effects using the estimated propensity score. *Econometrica*, 71(4):1161–1189.

Hull, P. and Borusyak, K. (2024). Negative weights are no concern in design-based specifications. *Working Paper*.

Imbens, G. W. and Angrist, J. D. (1994). Identification and estimation of local average treatment effects. *Econometrica*, 62(2):467–475.

Imbens, G. W. and Rubin, D. B. (2015). *REGULAR ASSIGNMENT MECHANISMS WITH NONCOMPLIANCE: ANALYSIS*, page 511–586. Cambridge University Press.

Jackson, C. K., Johnson, R. C., and Persico, C. (2015). The Effects of School Spending on Educational and Economic Outcomes: Evidence from School Finance Reforms *. *The Quarterly Journal of Economics*, 131(1):157–218.

Kang, H., Peck, L., and Keele, L. (2018). Inference for Instrumental Variables: A Randomization Inference Approach. *Journal of the Royal Statistical Society Series A: Statistics in Society*, 181(4):1231–1254.

Kirkeboen, L. J., Leuven, E., and Mogstad, M. (2016). Field of Study, Earnings, and Self-Selection*. *The Quarterly Journal of Economics*, 131(3):1057–1111.

Marcus, P. M., Ogden, S. L., Gren, L. H., Childs, J. C., Pretzel, S. M., Lamerato, L. E., Walsh, K., Rozjabek, H. M., Mabie, J., Thomas, B., and Riley, T. (2014). Non-compliance with the initial screening exam visit in the prostate, lung, colorectal and ovarian (plco) cancer screening trial. *Preventive Medicine*, 67:82–88.

Mountjoy, J. (2022). Community colleges and upward mobility. *American Economic Review*, 112(8):2580–2630.

Murphy, S. A., van der Laan, M. J., and Robins, J. M. (2001). Marginal mean models for dynamic regimes. *Journal of the American Statistical Association*, 96(456):1410–1423.

Pham, T. T. and Chen, W. (2017). The instrumental variable method for estimating local average treatment regime effects. *Working Paper*.

Rambachan, A. and Roth, J. (2024). Design-based uncertainty for quasi-experiments. *Working Paper*.

Robins, J. (1986). A new approach to causal inference in mortality studies with a sustained exposure period—application to control of the healthy worker survivor effect. *Mathematical Modelling*, 7(9):1393–1512.

Robins, J. (1987). A graphical approach to the identification and estimation of causal parameters in mortality studies with sustained exposure periods. *Journal of Chronic Diseases*, 40:139S–161S.

Schuler, A., Lee, K. J., and Hubbard, A. (2024). Bridging binarization: Causal inference with dichotomized continuous treatments. *Working Paper*.

Shen, S., Choi, J., and Seong, D. (2024). Panel instrumental variable regression models with varying-intensity repeated treatments: Theory and the china syndrome application. *Working Paper*.

Sotra, R. B. and Syrgkanis, V. (2024). Dynamic local average treatment effects. *Working Paper*.

Stango, V. and Zinman, J. (2022). We Are All Behavioural, More, or Less: A Taxonomy of Consumer Decision-Making. *The Review of Economic Studies*, 90(3):1470–1498.

Appendix A

Proof of Proposition 1

I begin with the decomposition of the fist stage β_2^{FS}. We have that, under Assumptions 1-5,

$$\begin{aligned}
\mathbb{E}\left[\beta_2^{FS}\right] &= \mathbb{E}\left[\frac{1}{N_1}\sum_{i=1}^{N} Z_{i,2}D_{i,2} - \frac{1}{N_0}\sum_{i=1}^{N}(1-Z_{i,2})D_{i,2}\right] \\
&= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}D_{i,2}(1) - \frac{1}{N_0}\sum_{i=1}^{N}(1-\lambda_{i,2})D_{i,2}(0) \\
&= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right) + \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}D_{i,2}(0) - \frac{1}{N_0}\sum_{i=1}^{N}(1-\lambda_{i,2})D_{i,2}(0) \\
&= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right) + \frac{NN}{N_0N_1}\left(\frac{1}{N}\sum_{i=1}^{N}\left(\lambda_{i,2}-\frac{N_1}{N}\right)D_{i,2}(0)\right) \\
&= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right) + \frac{NN}{N_0N_1}\mathbb{C}ov\left[\lambda_{i,2}, D_{i,2}(0)\right] \qquad (2)
\end{aligned}$$

Note that

$$\frac{1}{N}\sum_{i=1}^{N}\left(D_{i,2}(1)-D_{i,2}(0)\right)$$

$$=\frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right)-\frac{1}{N_1}\sum_{i=1}^{N}\left(\lambda_{i,2}-\frac{N_1}{N}\right)\left(D_{i,2}(1)-D_{i,2}(0)\right)$$

$$\frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right)-\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(1)-D_{i,2}(0)\right]$$

$$\frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right)-\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(1)\right]+\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(0)\right]$$

And thus,

$$\frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(D_{i,2}(1)-D_{i,2}(0)\right)=\frac{1}{N}\sum_{i=1}^{N}\left(D_{i,2}(1)-D_{i,2}(0)\right)+\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(1)\right]-\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(0)\right]$$

Substituting the expression above into (1) and using the second part of Assumption 2 and Assumption 3 yields

$$\beta_2^{RF}=\frac{1}{N}\sum_{i=1}^{N}\left(D_{i,2}(1)-D_{i,2}(0)\right)+\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(1)\right]+\frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2},D_{i,2}(0)\right]$$

$$=\frac{1}{N}\sum_{i=1}^{N}\left(D_{i,2}(1)-D_{i,2}(0)\right)=\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{G_{i,2}=C_2\right\}=\frac{1}{N}\left|C_2\right|$$

Now, let's consider the reduced form. We have

$$\mathbb{E}\left[\beta_2^{RF}\right] = \mathbb{E}\left[\frac{1}{N_1}\sum_{i=1}^{N} Z_{i,2}Y_{i,2} - \frac{1}{N_0}\sum_{i=1}^{N}(1-Z_{i,2})Y_{i,2}\right]$$

$$= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}Y_{i,2}(d_1^{obs}, D_{i,2}(1)) - \frac{1}{N_0}\sum_{i=1}^{N}(1-\lambda_{i,2})Y_{i,2}(d_1^{obs}, D_{i,2}(0))$$

$$= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(Y_{i,2}(d_1^{obs}, D_{i,2}(1)) - Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right)$$

$$+\frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}Y_{i,2}(d_1^{obs}, D_{i,2}(0)) - \frac{1}{N_0}\sum_{i=1}^{N}(1-\lambda_{i,2})Y_{i,2}(d_1^{obs}, D_{i,2}(0))$$

$$= \frac{1}{N_1}\sum_{i=1}^{N}\lambda_{i,2}\left(Y_{i,2}(d_1^{obs}, D_{i,2}(1)) - Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right) + \frac{NN}{N_0N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right]$$

Using the same algebraic manipulation as in the first stage, the expression simplifies to

$$\beta_2^{RF} = \frac{1}{N}\sum_{i=1}^{N}\left(Y_{i,2}(d_1^{obs}, D_{i,2}(1)) - Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right)$$

$$+\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(1))\right] + \frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right]$$

$$= \frac{1}{N}\sum_{i\in\mathcal{C}_2}\left(Y_{i,2}(d_1^{obs}, 1) - Y_{i,2}(d_1^{obs}, 0)\right)$$

$$+\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(1))\right] + \frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right]$$

Taking the ratio between β_2^{RF} and β_2^{FS} yields

$$\frac{\mathbb{E}\left[\widehat{\beta}_2^{RF}\right]}{\mathbb{E}\left[\widehat{\beta}_2^{FS}\right]} = \overline{\tau}_{C_2,2}(1,0;0) + \frac{1}{|C_2|}\left(\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(1))\right] + \frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(d_1^{obs}, D_{i,2}(0))\right]\right)$$

$$= \overline{\tau}_{C_2,2}(1,0;0) + \frac{1}{|C_2|}\left(\frac{N}{N_1}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(D_{i,1}(Z_{i,1}), D_{i,2}(1))\right] + \frac{N}{N_0}\mathbb{C}ov\left[\lambda_{i,2}, Y_{i,2}(D_{i,1}(Z_{i,1}), D_{i,2}(0))\right]\right)$$

$$= \overline{\tau}_{C_2,2}(1,0;0) + \Lambda$$

which concludes the proof

Proof of Proposition 2

The parameters $\tilde{\beta}_1$ and $\tilde{\beta}_2$ solve the following set of equations:

$$\mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1\right]\tilde{\beta}_1 + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\tilde{\beta}_2 = \mathbb{E}\left[\mathbf{Z}_1'\mathbf{Y}\right] \tag{3}$$

$$\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_1\right]\tilde{\beta}_1 + \mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]\tilde{\beta}_2 = \mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}\right] \tag{4}$$

Multiply (3) by $\mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}$ to obtain

$$\mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\mathbb{E}\left[\mathbf{Z}_2\mathbf{D}_1\right]\tilde{\beta}_1 + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\tilde{\beta}_2 = \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}\right] \tag{5}$$

Substitute (4) into (1) to obtain

$$\left[\mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1\right] - \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\left[\mathbf{Z}_2'\mathbf{D}_1\right]\right]\tilde{\beta}_1 = \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}\right] \tag{6}$$

Note that

$$\mathbf{Y} = \mathbf{Y}(0,0) + \mathbf{D}_1(1-\mathbf{D}_2)\left(\mathbf{Y}(1,0) - \mathbf{Y}(0,0)\right) + (1-\mathbf{D}_1)\mathbf{D}_2\left(\mathbf{Y}(0,1) - \mathbf{Y}(0,0)\right) + \mathbf{D}_1\mathbf{D}_2\left(\mathbf{Y}(1,1) - \mathbf{Y}(0,0)\right)$$

And thus,

$$\mathbb{E}\left[\mathbf{Z}_1'\mathbf{Y}\right] = \mathbb{E}\left[\mathbf{Z}_1'\mathbf{Y}(0,0)\right] + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1(1-\mathbf{D}_2)(\mathbf{Y}(1,0)-\mathbf{Y}(0,0))\right]$$
$$+\mathbb{E}\left[\mathbf{Z}_1'(1-\mathbf{D}_1)\mathbf{D}_2(\mathbf{Y}(0,1)-\mathbf{Y}(0,0))\right] + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1\mathbf{D}_2(\mathbf{Y}(1,1)-\mathbf{Y}(0,0))\right]$$

and

$$\mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}\right] = \mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}(0,0)\right] + \mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_1(1-\mathbf{D}_2)(\mathbf{Y}(1,0)-\mathbf{Y}(0,0))\right]$$
$$+\mathbb{E}\left[\mathbf{Z}_2'(1-\mathbf{D}_1)\mathbf{D}_2(\mathbf{Y}(0,1)-\mathbf{Y}(0,0))\right] + \mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_1\mathbf{D}_2(\mathbf{Y}(1,1)-\mathbf{Y}(0,0))\right]$$

And so, under Assumption 4,

$$\begin{aligned}
&\mathbb{E}\left[\mathbf{Z}_1'\mathbf{Y}\right] - \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\mathbb{E}\left[\mathbf{Z}_2'\mathbf{Y}\right] \\
&= \mathbb{E}\left[\mathbf{Z}_1'\mathbf{Y}(0,0)\right] + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1(1-\mathbf{D}_2)(\mathbf{Y}(1,0)-\mathbf{Y}(0,0))\right] \\
&+\mathbb{E}\left[\mathbf{Z}_1'(1-\mathbf{D}_1)\mathbf{D}_2(\mathbf{Y}(0,1)-\mathbf{Y}(0,0))\right] + \mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_1\mathbf{D}_2(\mathbf{Y}(1,1)-\mathbf{Y}(0,0))\right] \\
&-\mathbb{E}\left[\mathbf{Z}_1'\mathbf{D}_2\right]\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_2\right]^{-1}\Big\{\mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_1(1-\mathbf{D}_2)(\mathbf{Y}(1,0)-\mathbf{Y}(0,0))\right] \\
&+\mathbb{E}\left[\mathbf{Z}_2'(1-\mathbf{D}_1)\mathbf{D}_2(\mathbf{Y}(0,1)-\mathbf{Y}(0,0))\right] + \mathbb{E}\left[\mathbf{Z}_2'\mathbf{D}_1\mathbf{D}_2(\mathbf{Y}(1,1)-\mathbf{Y}(0,0))\right] \qquad \textit{dotted}
\end{aligned}$$

Substituting the expression back into (5) yields the solution for $\tilde{\beta}_1$. Taking $\tilde{\beta}_1$ and substituting into (3) yields the solution for $\tilde{\beta}_2$. Thus, the parameters are weighted averages of three different causal effects, for different complier groups.

Proof of Theorem 1

Given $\mathcal{F}_{1:N,1:T}$, define the following causal parameters

$$m_{t,(\mathbf{d})} = \frac{m^{RF}_{t,(\mathbf{d})}}{m^{RF}_{t,(\mathbf{d})}} = \frac{\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z}) = \mathbf{d}\right\} Y_{i,t}(d^{obs}_{i,1:t-p-1}, \mathbf{d})}{\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z}) = \mathbf{d}\right\}}$$

The proof is conducted by induction. I show that the result holds for the baseline case $p = 0$ ad then show that if it holds for a general p, then it must also hold for $p + 1$.

Consider the case where $p = 0$ and $D_{i,t} = 1$. Results for the identification in the cross-sectional setting can be modified to show that under Assumptions 1-4, the first stage identifies

$$\begin{aligned} &\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(1) - D_{i,t}(0) \\ &= \frac{1}{N}\sum_{i=1}^{N} \left(\mathbf{1}\left\{G_{i,t} = AT_t\right\} + \mathbf{1}\left\{G_{i,t} = C_t\right\}\right) - \mathbf{1}\left\{G_{i,t} = AT_t\right\} \\ &\qquad\qquad \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{G_{i,t} = C_t\right\} \end{aligned}$$

from which it follows that

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(1) - D_{i,t}(0) = \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{G_{i,t} = C_t\right\} = \frac{1}{N}\frac{1}{|C_t|}$$

For the reduced form estimand of the modified outcome, note that under Assumptions 1-4,

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(1)Y_{i,t}(d^{obs}_{1:t-p-1},1) - D_{i,t}(0)Y_{i,t}(d^{obs}_{1:t-p-1},1)$$

$$= \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{D_{i,t}(1)=1\right\} Y_{i,t}(d^{obs}_{1:t-p-1},1) - \mathbf{1}\left\{D_{i,t}(0)=1\right\} Y_{i,t}(d^{obs}_{1:t-p-1},1)$$

$$= \frac{1}{N}\sum_{i=1}^{N} Y_{i,t}(d^{obs}_{1:t-p-1},1)\left(\mathbf{1}\left\{G_{i,t}=AT_t\right\} + \mathbf{1}\left\{G_{i,t}=C_t\right\}\right) - Y_{i,t}(d^{obs}_{1:t-p-1},1)\mathbf{1}\left\{G_{i,t}=AT_t\right\}$$

$$= \frac{1}{N}\sum_{i=1}^{N} Y_{i,t}(d^{obs}_{1:t-p-1},1)\mathbf{1}\left\{G_{i,t}=C_t\right\}$$

from which it follows that

$$\frac{1}{N}\sum_{i=1}^{N} D_{i,t}(1)Y_{i,t}(d^{obs}_{1:t-p-1},1) - D_{i,t}(0)Y_{i,t}(d^{obs}_{1:t-p-1},1) = \frac{1}{N}\sum_{i\in C_t} Y_{i,t}(d^{obs}_{1:t-p-1},1)$$

and the ratio identifies

$$\frac{\sum_{i\in C_t} Y_{i,t}(d^{obs}_{1:t-p-1},1)}{|C_t|} = m_{t,(1)}$$

The result for the case where $D_{i,t}=0$ can be demonstrated analogously, once we note that the first stage identifies

$$\frac{1}{N}\sum_{i=1}^{N}(1-D_{i,t}(1)) - (1-D_{i,t}(0))$$

$$= \frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{G_{i,t}=NT_t\right\} - \left(\mathbf{1}\left\{G_{i,t}=NT_t\right\} + \mathbf{1}\left\{G_{i,t}=C_t\right\}\right)$$

$$= -\frac{1}{N}\sum_{i=1}^{N} \mathbf{1}\left\{G_{i,t}=C_t\right\} = -\frac{1}{N}\frac{1}{|C_t|}$$

and the modified reduced form identifies

$$-\frac{1}{N}\sum_{i=1}^{N} Y_{i,t}(d^{obs}_{1:t-p-1},0)\mathbf{1}\left\{G_{i,t}=C_t\right\} = -\frac{1}{N}\sum_{i\in C_t} Y_{i,t}(d^{obs}_{1:t-p-1},0)$$

For the general case, I prove the result only for $D_{i,t-p-1}=1$. The case for $D_{i,t-p-1}=0$ and can be derived using the intuition from the displayed result above. I begin with the first stage. Note that under Assumption 1,

$$\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(z_{t-p-1:t})=(1,\mathbf{d})\right\}\right)$$
$$\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,z_{t-p:t})=(1,\mathbf{d})\right\}\right)-\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,z_{t-p:t})=(1,\mathbf{d})\right\}\right)$$

Assume the result holds for p. Under Assumptions 1-4, the modified first stage can be written as

$$\begin{aligned}\frac{1}{N}\sum_{i=1}^{N}&\mathbf{1}\left\{D_{i,t-p-1}(1)=1,G_{i,t-p:t}=C_{t-p:t}\right\}-\mathbf{1}\left\{D_{i,t-p-1}(0)=1,G_{i,t-p:t}=C_{t-p:t}\right\}\\&=\frac{1}{N}\sum_{i=1}^{N}\left(\mathbf{1}\left\{G_{i,t-p-1:t}=AT_{t-p-1},C_{t-p:t}\right\}+\mathbf{1}\left\{G_{i,t-p-1:t}=C_{t-p-1:t}\right\}\right)\\&\qquad-\mathbf{1}\left\{G_{i,t-p-1:t}=AT_{t-p-1},C_{t-p:t}\right\}\\&=\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{G_{i,t-p-1:t}=C_{t-p-1:t}\right\}=\frac{1}{N}\left|C_{t-p-1:t}\right|\end{aligned}$$

and thus,

$$\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(z_{t-p-1:t})=(1,\mathbf{d})\right\}\right)=\frac{1}{N}\left|C_{t-p-1:t}\right|$$

For the reduced form, note that under Assumption 1,

$$
\begin{aligned}
&\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(z_{t-p-1:t})=(1,\mathbf{d})\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\right)\\
=&\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,z_{t-p:t})=(1,\mathbf{d})\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\right)\\
&-\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,z_{t-p:t})=(1,\mathbf{d})\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\right)
\end{aligned}
$$

Assuming the result holds for p, if Assumptions 2-4 further hold, we can write the result displayed above as

$$
\begin{aligned}
&\frac{1}{N}\sum_{i=1}^{N}\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,z_{t-p:t})=(1,\mathbf{d})\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\right)\\
&-\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,z_{t-p:t})=(1,\mathbf{d})\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\right)\\
=&\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,t-p-1}(1)=1,G_{i,t-p:t}=C_{t-p:t}\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\\
&-\mathbf{1}\left\{D_{i,t-p-1}(0)=1,G_{i,t-p:t}=C_{t-p:t}\right\}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\\
=&\frac{1}{N}\sum_{i=1}^{N}Y_{i,t}(d^{obs}_{1:t-p-2},1,\mathbf{d})\mathbf{1}\left\{G_{i,t-p-1:t}=C_{t-p-1:t}\right\}
\end{aligned}
$$

Hence, it follows that

$$
\frac{\sum_{i=1}^{N}\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(z_{t-p-1:t})=(1,\mathbf{d})Y_{i,t}\right\}\right)}{\sum_{i=1}^{N}\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(z_{t-p-1:t})=(1,\mathbf{d})\right\}\right)}=m_{t,(\mathbf{d})}
$$

Which concludes the proof.

Proof of Theorem 2

The proof follows trivially from Theorem 1 once is noted that for a given assignment path $z_{t-p:t}$, under Assumptions 1-6,

$$\mathbb{E}\left[\frac{1}{N}\sum_{i=1}^{N}\frac{\mathbf{1}\left\{D_{i,t-p:t}=\mathbf{d}\right\}}{\pi_{i,t-p:t}(z_{t-p:t})}\right]=\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,t-p:t}(z_{t-p:t})=\mathbf{d}\right\}$$

and

$$\mathbb{E}\left[\frac{1}{N}\sum_{i=1}^{N}\frac{\mathbf{1}\left\{D_{i,t-p:t}=\mathbf{d}\right\}Y_{i,t}}{\pi_{i,t-p:t}(z_{t-p:t})}\right]=\frac{1}{N}\sum_{i=1}^{N}\mathbf{1}\left\{D_{i,t-p:t}(z_{t-p:t})=\mathbf{d}\right\}Y_{i,t}(d^{obs}_{i,1:t-p-1},\mathbf{d})$$

The remainder of the proof follows using the reasoning from Theorem 1.

Proof of Theorem 3

If potential outcomes are bounded, then the standard Lindeberg condition holds. The first result follows from the triangular array central limit theorem, as in Bojinov et al. (2021).

The variance $\left(\sigma_t(1,0;0)\right)^2$ is simply

$$\left(\sigma_t(1,0;0)\right)^2=\frac{1}{N}\sum_{i=1}^{N}\left(\sigma_{i,t}(1,0;0)\right)^2$$

where $\left(\sigma_{i,t}(1,0;0)\right)^2$ is defined in Lemma 1 from Appendix B.

The second result follows from Theorem 3.2 in Bojinov et al. (2021). The variance is given by

$$\left(\sigma(1,0;0)\right)^2=\frac{1}{NT}\sum_{t=1}^{T}\sum_{i=1}^{N}\left(\sigma_{i,t}(1,0;0)\right)^2$$

Proof of Theorem 4

The result follows the same reasoning as the one in Theorem 5. This time, however, we have

$$\Big(\sigma_t(\mathbf{d},\tilde{\mathbf{d}};p)\Big)^2=\frac{1}{N}\sum_{i=1}^{N}\Big(\sigma_{i,t}(\mathbf{d},\tilde{\mathbf{d}};p)\Big)^2$$

and

$$\left(\sigma(\mathbf{d}, \tilde{\mathbf{d}}; p)\right)^2 = \frac{1}{N(T-p)} \sum_{t=p+1}^{T} \sum_{i=1}^{N} \left(\sigma_{i,t}(\mathbf{d}, \tilde{\mathbf{d}}; p)\right)^2$$

See Lemma 2 from Appendix B for the necessary results.

Appendix B

Proof of Lemma 1

Define $W_{i,t-p:t}(\mathbf{z}) = \pi_{i,t-p:t}(\mathbf{z})^{-1}\mathbf{1}\left\{Z_{i,t-p:t} = \mathbf{z}\right\}$. From Lemma A.1 in Bojinov et al. (2021), we have

$$\begin{aligned}
\mathbb{E}\left[W_{i,t-p:t}(\mathbf{z})\right] &= 1 \\
\mathbb{V}\left[W_{i,t-p:t}(\mathbf{z})\right] &= \pi_{i,t-p:t}(\mathbf{z})^{-1}(1 - \pi_{i,t-p:t}(\mathbf{z})) \\
\mathbb{C}ov\left[W_{i,t-p:t}(\mathbf{z}), W_{i,t-p:t}(\tilde{\mathbf{z}})\right] &= -1
\end{aligned}$$

I analyze the properties of the estimator for each stage separately before analyzing the properties of the two-stage estimator. I begin with the first stage. Define

$$u_{i,t}^{FS} = \widehat{\tau}_{i,t}^{FS} - \tau_{i,t}^{FS} = D_{i,t}(1)(W_{i,t}(1) - 1) - D_{i,t}(0)(W_{i,t}(0) - 1)$$

Lemma A.1 from Bojinov et al. (2021) implies that $\mathbb{E}\left[u_{i,t}^{FS}\right] = 0$. Hence, the error terms from the estimator are a martingale difference sequence and so, uncorrelated through time. Now, let's look at the variance:

$$
\begin{aligned}
\mathbb{V}\left[u_{i,t}^{FS}\right] &= D_{i,t}(1)^2\mathbb{V}\left[W_{i,t}(1)\right] + D_{i,t}(0)^2\mathbb{V}\left[W_{i,t}(0)\right] \\
&\quad -2D_{i,t}(1)D_{i,t}(0)\mathbb{C}ov\left[W_{i,t}(1), W_{i,t}(0)\right] \\
&= D_{i,t}(1)^2\pi_{i,t}(1)^{-1}(1-\pi_{i,t}(1)) + D_{i,t}(0)^2\pi_{i,t}(0)^{-1}(1-\pi_{i,t}(0)) \\
&\quad +2D_{i,t}(1)D_{i,t}(0) \\
&= \frac{D_{i,t}(1)^2}{\pi_{i,t}(1)} + \frac{D_{i,t}(0)^2}{\pi_{i,t}(0)} - (D_{i,t}(1) - D_{i,t}(0))^2 \\
&= \left(\gamma_{i,t}^{FS}(1,0)(0)\right)^2 - (D_{i,t}(1) - D_{i,t}(0))^2 = \left(\sigma_{i,t}^{FS}(1,0)(0)\right)^2
\end{aligned}
$$

Now, consider the reduce form. Define

$$
u_{i,t}^{RF} = \widehat{\tau}_{i,t}^{RF} - \tau_{i,t}^{RF} = Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))(W_{i,t}(1)-1) - Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))(W_{i,t}(0)-1)
$$

We have $\mathbb{E}\left[u_{i,t}^{RF}\right] = 0$. Hence, the error terms from the estimator are a martingale difference sequence and so uncorrelated through time. Now, let's look at the variance:

$$
\begin{aligned}
\mathbb{V}\left[u_{i,t}^{RF}\right] &= Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))^2\mathbb{V}\left[W_{i,t}(1)\right] + Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))^2\mathbb{V}\left[W_{i,t}(0)\right] \\
&\quad -2Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))\mathbb{C}ov\left[W_{i,t}(1), W_{i,t}(0)\right] \\
&= Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))^2\pi_{i,t}(1)^{-1}(1-\pi_{i,t}(1)) + Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))^2\pi_{i,t}(0)^{-1}(1-\pi_{i,t}(0)) \\
&\quad +2Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0)) \\
&= \frac{Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1))^2}{\pi_{i,t}(1)} + \frac{Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0))^2}{\pi_{i,t}(0)} - (Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1)) - Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0)))^2 \\
&= \left(\gamma_{i,t}^{RF}(1,0;0)\right)^2 - (Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(1)) - Y_{i,t}(d_{1:t-1}^{obs}, D_{i,t}(0)))^2 = \left(\sigma_{i,t}^{RF}(1,0;0)\right)^2
\end{aligned}
$$

From the results above, it follows that

$$\frac{\mathbb{E}\left[u_{i,t}^{RF}\right]}{\mathbb{E}\left[u_{i,t}^{FS}\right]} = 0$$

We apply the Uniform Delta Method to obtain

$$\mathbb{V}\left[u_{i,t}\right] = g^{'} \begin{pmatrix} \left(\sigma_{i,t}^{RF}(1,0;0)\right)^2 \\ \left(\sigma_{i,t}^{FS}(1,0;0)\right)^2 \end{pmatrix} g := \left(\sigma_{i,t}(1,0;0)\right)^2$$

Where g is the gradient of $h(x,y) = x/y$ evaluated at $(\tau_{i,t}^{RF}, \tau_{i,t}^{FS})$, which concludes the proof.

Proof of Lemma 2

Define

$$u_{i,t}^{FS}(\mathbf{d}) = \widehat{m}_{i,t}^{FS}(\mathbf{d}) - m_{i,t}^{FS}(\mathbf{d}) = \Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})\right\}\left(W_{i,t-p:t}(\mathbf{z}) - 1\right)\right)$$
$$u_{i,t}^{RF}(\mathbf{d}) = \widehat{m}_{i,t}^{RF}(\mathbf{d}) - m_{i,t}^{RF}(\mathbf{d}) = \Delta^{p+1}\left(Y_{i,t}(d_{1:t-p-1}^{obs}, \mathbf{d})\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})\right\}\left(W_{i,t-p:t}(\mathbf{z}) - 1\right)\right)$$

We have $\mathbb{E}\left[u_{i,t}^{FS}(\mathbf{d})\right] = \mathbb{E}\left[u_{i,t}^{RF}(\mathbf{d})\right] = 0$ for all $\mathbf{d} \in \{0,1\}^{p+1}$. Furthermore, define $u_{i,t}^{RF}(\mathbf{d}, \tilde{\mathbf{d}}; p) = u_{i,t}^{RF}(\mathbf{d}) + u_{i,t}^{RF}(\tilde{\mathbf{d}})$ and $u_{i,t}^{FS}(\mathbf{d}, \tilde{\mathbf{d}}; p)$ analogously. It follows that $\mathbb{E}\left[u_{i,t}^{RF}(\mathbf{d}, \tilde{\mathbf{d}}; p)\right] = \mathbb{E}\left[u_{i,t}^{FS}(\mathbf{d}, \tilde{\mathbf{d}}; p)\right] = 0$.

Now let's consider the variance. I prove the result by induction for the reduced form and first stage of each potential outcome separately, and build on it to derive the properties of the estimator.

First, let's consider the case when $p = 0$ and $D_{i,t} = 1$ (the case for $D_{i,t} = 0$ is analogous). For the first stage, it's been proved earlier that

$$\begin{aligned}\mathbb{V}\left[u_{i,t}^{FS}(1)\right] &= \frac{D_{i,t}^2(1)}{\pi_{i,t}(1)} + \frac{D_{i,t}^2(0)}{\pi_{i,t}(0)} - (D_{i,t}(1) - D_{i,t}(0))^2 \\ &= \left(\gamma_{i,t}^{FS}(1)\right)^2 - (D_{i,t}(1) - D_{i,t}(0))^2 = \left(\sigma_{i,t}^{FS}(1)\right)^2\end{aligned}$$

Now let's consider the reduced form. Using a reasoning similar from the one in the lag-0 case, we obtain

$$\begin{aligned}\mathbb{V}\left[u_{i,t}^{RF}(1)\right] &= \left(Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(1)\right)^2 \mathbb{V}\left[W_{i,t}(1)\right] + \left(Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(0)\right)^2 \mathbb{V}\left[W_{i,t}(0)\right] \\ &\quad -2Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(1)Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(0)\mathbb{C}ov\left[W_{i,t}(1), W_{i,t}(0)\right] \\ &= \frac{\left(Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(1)\right)^2}{\pi_{i,t}(1)} + \frac{\left(Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(0)\right)^2}{\pi_{i,t}(0)} \\ &\quad -(Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(1) - Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(0))^2 \\ &= \left(\gamma_{i,t}^{RF}(1)\right)^2 - (Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(1) - Y_{i,t}(d_{1:t-1}^{obs}, 1)D_{i,t}(0))^2 = \left(\sigma_{i,t}^{RF}(1)\right)^2\end{aligned}$$

Now, I proceed by deriving the expression for a general $p+1$, assuming that the result holds for p. For the first stage,

$$
\begin{aligned}
\mathbb{V}\left[u_{i,t}^{FS}(\mathbf{d})\right] &= \mathbb{V}\left[\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(\mathbf{z})=\mathbf{d}\right\}\left(W_{i,t-p-1}(\mathbf{z})-1\right)\right)\right] \\
&= \mathbb{V}\left[\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,\mathbf{z}_-)=\mathbf{d}\right\}\left(W_{i,t-p-1}(1,\mathbf{z}_-)-1\right)\right)\right. \\
&\quad \left.-\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,\mathbf{z}_-)=\mathbf{d}\right\}\left(W_{i,t-p-1}(0,\mathbf{z}_-)-1\right)\right)\right. \quad do \\
&= \mathbb{V}\left[\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,\mathbf{z}_-)=\mathbf{d}\right\}\left(W_{i,t-p-1}(1,\mathbf{z}_-)-1\right)\right)\right] \\
&\quad +\mathbb{V}\left[\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,\mathbf{z}_-)=\mathbf{d}\right\}\left(W_{i,t-p-1}(0,\mathbf{z}_-)-1\right)\right)\right] \\
&\quad -2\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(1,\mathbf{z}_-)=\mathbf{d}\right\}\right)\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p-1:t}(0,\mathbf{z}_-)=\mathbf{d}\right\}\right)\mathbb{C}ov\left[W_{i,t-p-1}(1,\mathbf{z}_-),W_{i,t-p-1}(0,\mathbf{z}_-)\right] \\
&= \sum_{\mathbf{z}_-\in\{0,1\}^{p+1}} \frac{\mathbf{1}\left\{D_{i,t-p-1}(1,\mathbf{z}_-)\right\}=\mathbf{d}}{\pi_{i,t-p-1}(1,\mathbf{z}_-)} - \Delta^{p+1}\left(D_{i,t-p-1}(1,\mathbf{z}_-)\right)^2 \\
&\quad + \sum_{\mathbf{z}_-\in\{0,1\}^{p+1}} \frac{\mathbf{1}\left\{D_{i,t-p-1}(0,\mathbf{z}_-)\right\}=\mathbf{d}}{\pi_{i,t-p-1}(0,\mathbf{z}_-)} - \Delta^{p+1}\left(D_{i,t-p-1}(0,\mathbf{z}_-)\right)^2 \\
&\quad +2\Delta^{p+1}\left(D_{i,t-p-1}(1,\mathbf{z}_-)\right)\Delta^{p+1}\left(D_{i,t-p-1}(0,\mathbf{z}_-)\right) \\
&= \sum_{\mathbf{z}\in\{0,1\}^{p+2}} \frac{\mathbf{1}\left\{D_{i,t-p-1}(\mathbf{z})\right\}=\mathbf{d}}{\pi_{i,t-p-1}(\mathbf{z})} \\
&\quad -\left(\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1}(1,\mathbf{z}_-)\right\}=\mathbf{d}\right)-\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1}(0,\mathbf{z}_-)\right\}=\mathbf{d}\right)\right)^2 \\
&= \left(\gamma_{i,t}^{FS}(\mathbf{d})\right)^2 - \left(\Delta^{p+2}\left(\mathbf{1}\left\{D_{i,t-p-1}(\mathbf{z})\right\}=\mathbf{d}\right)\right)^2 \\
&= \left(\sigma_{i,t}^{FS}(\mathbf{d})\right)^2
\end{aligned}
$$

Analogously, for the reduced form it can be shown that

$$
\begin{aligned}
\mathbb{V}\left[u_{i,t}^{RF}(\mathbf{d})\right] &= \sum_{\mathbf{z}\in\{0,1\}^{p+2}} \frac{\left(Y_{i,t}(d_{1:t-p-2}^{obs},\mathbf{d})\mathbf{1}\left\{D_{i,t-p-1:t}(\mathbf{z})=\mathbf{d}\right\}\right)^2}{\pi_{i,t-p-1}(\mathbf{z})} \\
&\quad -\left(\Delta^{p+2}\left(Y_{i,t}(d_{1:t-p-2}^{obs},\mathbf{d})\mathbf{1}\left\{D_{i,t-p-1:t}(\mathbf{z})=\mathbf{d}\right\}\right)\right)^2 \\
&= \left(\gamma_{i,t}^{RF}(\mathbf{d})\right)^2 - \left(\Delta^{p+2}\left(Y_{i,t}(d_{1:t-p-2}^{obs},\mathbf{d})\mathbf{1}\left\{D_{i,t-p-1:t}(\mathbf{z})=\mathbf{d}\right\}\right)\right)^2 = \left(\sigma_{i,t}^{RF}(\mathbf{d})\right)^2
\end{aligned}
$$

From the results above, we have

$$\mathbb{V}\left[u_{i,t}(\mathbf{d})\right] = g^{'}\begin{pmatrix}\left(\sigma_{i,t}^{RF}(\mathbf{d})\right)^2\\ \left(\sigma_{i,t}^{FS}(\mathbf{d})\right)^2\end{pmatrix} g = \left(\sigma_{i,t}(\mathbf{d})\right)^2$$

Where g is the gradient of $h(x,y) = x/y$ evaluated at $(m_{i,t}^{RF}(\mathbf{d}), m_{i,t}^{FS}(\mathbf{d}))$

The variance for $\widehat{\tau}_{i,t}(\mathbf{d}, \tilde{\mathbf{d}}; p)$ is simply $\left(\sigma_{i,t}(\mathbf{d})\right)^2 + \left(\sigma_{i,t}(\tilde{\mathbf{d}})\right)^2$, which concludes the proof.

Appendix C

In order to derive the asymptotic properties of the estimator with an estimated propensity score, first I define the estimation error in the first stage as

$$u_{i,t}^{FS}(\mathbf{d}) = \widehat{m}_{i,t}^{FS}(\mathbf{d}) - m_{i,t}^{FS}(\mathbf{d}) = \Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})\right\}\left(\widehat{W}_{i,t-p:t}(\mathbf{z}) - 1\right)\right)$$

which can be written as

$$\begin{aligned} u_{i,t}^{FS}(\mathbf{d}) &= \Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})\right\}\left(W_{i,t-p:t}(\mathbf{z}) - 1\right)\right) \\ &+\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t}(\mathbf{z})\right\}\left(\widehat{W}_{i,t-p:t}(\mathbf{z}) - W_{i,t-p:t}(\mathbf{z})\right)\right)\end{aligned}$$

If we assume that $\mathbb{E}\left[\widehat{\pi}_{i,t-p:t}(\mathbf{z})\right] = \pi_{i,t-p:t}(\mathbf{z})$, then it follows that $\mathbb{E}\left[\widehat{W}_{i,t-p:t}(\mathbf{z})\right] = W_{i,t-p:t}(\mathbf{z})$.

If we further assume that $\widehat{\pi}_{i,t-p:t}(\mathbf{z}) - \pi_{i,t-p:t}(\mathbf{z}) = O_p(N^{-1/2})$, then using Hahn (1998), Hirano et al. (2003) we obtain the following approximation:

$$\sqrt{N}\left(\widehat{m}_t^{FS}(\mathbf{d}) - m_t^{FS}(\mathbf{d})\right) = \frac{1}{\sqrt{N}}\sum_{i=1}^{N}\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t} = \mathbf{d}\right\}\left(W_{i,t-p:t}(\mathbf{z}) - 1\right)\right) + o_p(1)$$

A similar result can be shown for $\widehat{m}_t^{RF}(\mathbf{d})$. Therefore, it follows that

$$\frac{\sqrt{N}\left(\widehat{m}_t(\mathbf{d}) - m_t(\mathbf{d})\right)}{\sigma_t(\mathbf{d})} \xrightarrow{d} \mathcal{N}(0,1)$$

where is obtained using the uniform Delta method and

$$\frac{\sqrt{N}\left(\widehat{\overline{\tau}}_t(\mathbf{d},\tilde{\mathbf{d}};p) - \overline{\tau}_t(\mathbf{d},\tilde{\mathbf{d}};p)\right)}{\sigma_t(\mathbf{d},\tilde{\mathbf{d}};p)} \xrightarrow{d} \mathcal{N}(0,1)$$

where $\sigma_t(\mathbf{d},\tilde{\mathbf{d}};p)$ is the same from the case with known propensity score.

The same can be shown for total lag-p dynamic causal effects, once we note that the following approximation holds:

$$\sqrt{N(T-p)}\left(\widehat{m}^{FS}(\mathbf{d}) - m^{FS}(\mathbf{d})\right) = \frac{1}{\sqrt{N(T-p)}}\sum_{t=p+1}^{T}\sum_{i=1}^{N}\Delta^{p+1}\left(\mathbf{1}\left\{D_{i,t-p:t} = \mathbf{d}\right\}\left(W_{i,t-p:t}(\mathbf{z}) - 1\right)\right) + o_p(1)$$

Appendix D

As a robustness check, I estimate the results presented in Table 5 using different methods for the binarization of the instrument and the treatment. In Table 6 I present the results when treatment and instrument are binarized using the median of fines and cloud coverage instead of the mean.

Table 6: Impulse Response Functions - Median

		lag-p			
	AGR(2023)	0	1	2	3
Point estimate	-0.0244	-0.0504	-0.0151	0.0134	0.0196
95% CI	(-0.049, -0.001)	(-0.097, -0.002)	(-0.037, -0.001)	(-0.017, 0.041)	(-0.033, 0.058)
Baseline	0.0071	0.0071	0.0071	0.0071	0.0071
Observations	5210	5210	4689	4168	3647
Municipalities	521	521	521	521	521

Note: The dependent variable is the ratio between deforested area in a year and the municipality area. The set of control variables contains precipitation and temperature (weather), PRODES cloud coverage and other nonobservable areas (satellite visibility), and agricultural commodity prices.

As a final robustness check, I binarize the treatment using an indicator for municipalities

that received a number of fines greater than zero. The results are presented in Table 7 below.

Table 7: Impulse Response Functions - Greater than zero

		lag-p			
	AGR(2023)	0	1	2	3
Point estimate	-0.0244	-0.0641	-0.0350	0.0134	0.0026
95% CI	(-0.049, -0.001)	(-0.123, -0.004)	(-0.070, -0.005)	(-0.085, 0.018)	(-0.049, 0.008)
Baseline	0.0071	0.0071	0.0071	0.0071	0.0071
Observations	5210	5210	4689	4168	3647
Municipalities	521	521	521	521	521

Note: The dependent variable is the ratio between deforested area in a year and the municipality area. The set of control variables contains precipitation and temperature (weather), PRODES cloud coverage and other nonobservable areas (satellite visibility), and agricultural commodity prices.

Overall, the estimates are fairly stable across specifications, with the exception of the impulse response function for the lag-1 dynamic causal effect, which is not statistically significant under the binarization procedure for Table 7.