Revisiting Event Study Designs*

Kirill Borusyak, Harvard University
Xavier Jaravel, Stanford University

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Abstract

A broad empirical literature uses "event study" research designs for treatment effect estimation, a setting in which all units in the panel receive treatment but at random times. We make four novel points about identification and estimation of causal effects in this setting and show their practical relevance. First, we show that in the presence of unit and time fixed effects, it is impossible to identify the linear component of the path of pre-trends and dynamic treatment effects. Second, we propose graphical and statistical tests for pre-trends. Third, we consider commonly-used "static" regressions, with a treatment dummy instead of a full set of leads and lags around the treatment event, and we show that OLS does not recover a weighted average of the treatment effects: long-term effects are weighted negatively, and we introduce a different estimator that is robust to this issue. Fourth, we show that equivalent problems of under-identification and negative weighting arise in difference-in-differences settings when the control group is allowed to be on a different time trend or in the presence of unit-specific time trends. Finally, we show the practical relevance of these issues in a series of examples from the existing literature, with a focus on the estimation of the marginal propensity to consume out of tax rebates.

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1 Introduction

A broad empirical literature in labor economics, public finance, finance and empirical macroeconomics uses event study research designs for treatment effect estimation, a setting in which all units in the panel receive treatment but at random times. We make a series of novel points about identification and estimation of causal effects in such a setting, which are closely related to the well-known age-cohort-time problem. We then establish the practical relevance of these points in light of the existing literature and in a specific application, estimating the impulse response function of consumption expenditures to tax rebate receipt.

We first show that *in the presence of unit and time fixed effects, it is impossible to identify the linear component of the path of pre-trends and dynamic treatment effect*s. Identification of the dynamic causal effects only up to a linear trend is particularly problematic because researchers usually want to test for the absence of pre-trends prior to the event and to document changes in the outcome variable after treatment in a non-parametric way. Intuitively, the path of pre-trends and dynamic treatment effects is identified only up to a linear trend because, "within a treatment unit", one cannot disentangle the passing of absolute time from the passing of time relative to the treatment event. We show formally that the collinearity problem that arises in this setting is effectively the same as the age-cohort-time problem that others have studied.

We then propose two approaches to address this underidentification issue. Our first strategy is to restrict the pre-trends in the fully dynamic specification, while keeping unit fixed effects. Our second strategy consists in replacing unit fixed effects with unit random effects. We develop statistical tests for the validity of both approaches, as well as a graphical test for the first approach.

Next, we turn to estimation of the average treatment effect, with a particular focus on specifications that are meant to average all dynamic treatment effects post treatment. We show that the specification that is commonly used in the literature estimates an average of treatment effects that severely overweighs short-term effects and weighs long-term effects negatively. This issue can be serious, such that *the estimated average treatment effect can be outside of the convex hull of the true dynamic treatment effects*. We introduce a simple estimator that is robust to this concern.

Both of the aforementioned problems, underidentification and negative weighting, stem from a collinearity problem in event study designs without a control group: the ability of unit and time fixed effects to recover "exposure time" (time since the first treatment). We demonstrate that equivalent problems may arise in other empirical designs, for instance in settings with a control group where the control group is allowed to have its own linear time trend. We consider other generalizations of our points, in particular negative weighting arising from treatment effect heterogeneity across treated units.

Finally, we establish the empirical relevance of these various points by describing a series of recent and influential papers in the literature that run precisely the specifications whose undesirable properties we point out in this paper. Moreover, we use extended data following Parker et al. (2013) to estimate the impulse response function of consumption expenditures to tax rebate receipt, using the random timing of tax rebate receipt across households. In this application, we find that the underidentification and the negative weighting issues have strong empirical revelance.[1]

The remainder of this paper is organized as follows. In Section 2, we describe our setting, the key specifications we study, and how they are consistent with a causal model. Section 3 presents the underidentification problem and our solutions, while Section 4 describe the negative weighting issue and how to address it. Section 5 considers a variety of extensions. Finally, Section 6 relates our points to the existing literature and presents the estimation of the marginal propensity to consume out of tax rebates as an application.

[1] The Empirical Relevance section is being revised and is available from the authors upon request.

2 Setup

2.1 Data-generating Process

Consider a panel of $i = 1, \ldots, N$ units in which the outcome Y_{it} is observed for $t = 1, \ldots, T$ periods ("calendar time"), or possibly for a subset thereof. In our main setting, every unit receives treatment in some period E_i within the sample and stays treated forever.[2] Units with the same treatment period are referred to as a cohort. Let $K_{it} = t - E_i$ denote the "relative time"—the number of periods relative to the event. The indicator variable for being treated can therefore be written as $D_{it} = \mathbf{1}\{t \geq E_i\} = \mathbf{1}\{K_{it} \geq 0\}$.

Empirical papers using this event study setup often pursue some of the following three goals. They first estimate whether the treatment has an effect on average. Second, they test for pre-trends to lend credibility to the research design. Finally, they may study in more depth the dynamics of the causal effect. With these goals in mind, we choose a class of data-generating processes which is very flexible on the dynamics but abstracts away from a variety of other specification issues:[3]

$$Y_{it} = \tilde{\alpha}_i + \tilde{\beta}_t + \sum_{k=-\infty}^{\infty} \tilde{\gamma}_k \mathbf{1}\{K_{it} = k\} + \tilde{\varepsilon}_{it}. \tag{1}$$

Here $\{\tilde{\gamma}_k\}$ for $k < 0$ correspond to pre-trends, and for $k \geq 0$—to dynamic effects k periods relative to the event.[4] The average effect is $\sum_{k=0}^{\infty} \omega_k \tilde{\gamma}_k$ for some weighting scheme $\{\omega_k\}$, but researchers rarely specify it based on their economic question and instead rely on the regression to do something reasonable. Tildes indicate the parameters of the "true model", reflecting the data generating process, and later on we express the estimands of commonly-used regression specification in terms of these parameters.[5] $\tilde{\alpha}_i$ and $\tilde{\beta}_t$ are unit and period fixed effects, respectively, and $\tilde{\varepsilon}_{it}$ is random noise. We call equation (1) the *fully dynamic specification*.

This formulation is consistent with a causal model in which each unit i for each period t has a set of potential outcomes $Y_{it}^{(k)}$ for each integer k, only one of which is realized. Treatment effects, expressed relative to one of them, e.g. $Y_{it}^{(-1)}$, are homogenous across units and calendar time periods, and depend only on k: $Y_{it}^{(k)} - Y_{it}^{(-1)} = \tilde{\gamma}_k$.[6] Furthermore, $Y_{it}^{(-1)} = \tilde{\alpha}_i + \tilde{\beta}_t + \tilde{\varepsilon}_{it}$, which is a standard assumption necessary for the validity of difference-in-difference approaches (Angrist and Pischke, 2008, p. 156). Together these assumptions deliver equation (1).

If the event is unpredictable, it is not known whether the current period corresponds to $K_{it} = -1, -2$, or any other negative number. As a consequence, $Y_{it}^{(-1)} = Y_{it}^{(-2)} = \ldots$, so $\tilde{\gamma}_k = 0$ for all $k < 0$. In that sense, random timing of the event implies that there cannot be any pre-trends.[7] Equation 1 reduces to the following specification, which we call *semi-dynamic* and take to be true if the empirical design is valid:

$$Y_{it} = \tilde{\alpha}_i + \tilde{\beta}_t + \sum_{k=0}^{\infty} \tilde{\gamma}_k \mathbf{1}\{K_{it} = k\} + \tilde{\varepsilon}_{it}. \tag{2}$$

2.2 Current Practice

In the current practice, it is prevalent to estimate models similar to (1) and (2) using OLS with two-way (unit and period) fixed effects. Different papers impose different restrictions on (1), but the following specification covers

[2] We consider settings with a control group in Section 5.1.1.

[3] We allow for more general models in the Extensions section, such as heterogeneity of treatment effects across units. All of our results also directly extend to adding time-varying controls.

[4] By $k = \pm\infty$ we mean the largest number possible given the sample.

[5] The notation for these estimands does not use tildes.

[6] Note that one cannot hope to estimate treatment effects relative to the situation in which the event never happens, simply because this circumstance is not observed in the data. Picking $k = -1$ as the omitted category is an innocent and standard normalization.

[7] In some settings anticipation of the event is possible but limited to a fixed number of A periods. In that case $\tilde{\gamma}_k = 0$ for $k < -A$, and any $k < -A$ can be chosen as the omitted category.

most of them:

$$Y_{it} = \alpha_i + \beta_t + \sum_{k=-A}^{B-1} \gamma_k \mathbf{1}\{K_{it} = k\} + \gamma_{B+} \mathbf{1}\{K_{it} \geq B\} + \varepsilon_{it}, \tag{3}$$

where $A \geq 0$ leads of treatment are included, together with $B \geq 0$ terms for specific short-term effects and a single last coefficient γ_{B+} for all longer-term effects.[8] Note the absence of tildes: unless $A = 0$ and $B = \infty$, this equation does not coincide with the true model (2). We will study where its coefficients converge to in large samples as functions of the true parameters. We will occasionally use hats to mark finite-sample objects.

The simplest and perhaps the most prevalent regression is (3) with $A = B = 0$, i.e.

$$Y_{it} = \alpha_i + \beta_t + \gamma_{0+} D_{it} + \varepsilon_{it}. \tag{4}$$

We refer to this specification as *static* or, following Allegretto et al. (2013), *canonical*, and will discuss it at great length later in the paper. Compared to the fully dynamic one, it imposes no pre-trends and constant treatment effects for all k. The other extreme is, of course, $A = B = \infty$, which is just the fully dynamic specification with no restrictions.

Often regressions are run using all available data, but sometimes the sample is balanced around the event time: only observations with $K_{it} \in [\underline{k}, \bar{k}]$, $\underline{k} < 0 \leq \bar{k}$, are included and only for units which are observed for all corresponding periods. We discuss pros and cons of this approach later on.

3 Underidentification of the Fully Dynamic Specification

3.1 Problem

In this section, we show that the fully dynamic specification, given by equation (1), suffers from a fundamental underidentification problem. The goal of such a specification is to recover the dynamic path of causal effects $\{\tilde{\gamma}_k\}_{k=-\infty}^{\infty}$. We show that this set of point estimates is in fact *identified only up to up to a linear trend.* A linear trend in the set of causal effects $\{\gamma_k\}_{k=-\infty}^{\infty}$ cannot be identified. In other words, one can start from any set of points estimates $\{\gamma_k\}_{k=-\infty}^{\infty}$, add a linear trend (in k) and adjust the sets of point estimates for the year fixed effects β_t and the individual fixed effects α_i to keep the same predicted value. Identification of the dynamic causal effects $\{\tilde{\gamma}_k\}_{k=-\infty}^{\infty}$ up to a linear trend is particularly problematic because researchers usually want to test for the absence of "pre-trends" prior to the event,[9] and more generally are hoping to document changes in the outcome variable after treatment in a non-parametric way.[10] In this section, we first illustrate the underidentification issue graphically to gain intuition. We then show mathematically where it stems from.

The intuition for why the fully dynamic specification is underidentified can easily be grasped with a few simple graphs. Consider a setting where the true parameters of the model are all zero: there is no year effect, no causal effect after the event, no pretrend before, no unit fixed effects, and no noise. In other words, the outcome variable is always equal to zero. Panel A of Figure 1 shows that in such a setting, we can easily have perfect fit with a set of coefficients $\{\gamma_k\}_{k=-\infty}^{\infty}$ that are linearly increasing in k, as if the treatment effect was growing over time. The data is equally consistent with this path of causal effects, which can be "undone" by carefully picking the period and unit fixed effects, as with a set of coefficients $\{\gamma_k\}_{k=-\infty}^{\infty}$ that are all equal to zero (in which case the period and unit

[8] One term, e.g. $\gamma_{-1}\mathbf{1}\{k_{it} = -1\}$, can be omitted as a normalization.

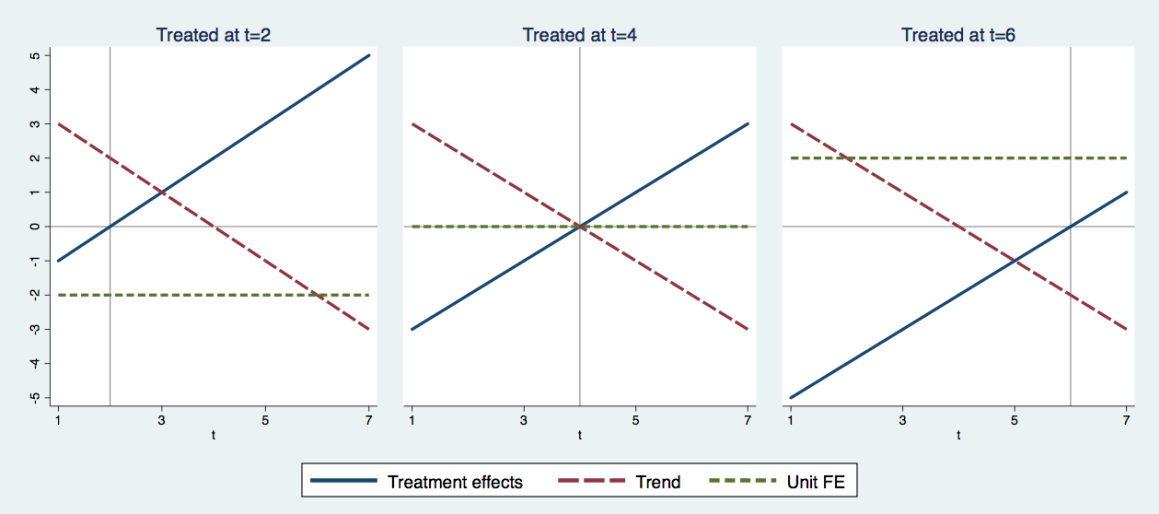
[9] In contrast, underidentification of the set of point estimates $\{\tilde{\gamma}_k\}_{k=-\infty}^{\infty}$ up to a constant, which is a well-known fact, is not problematic because it does not in itself prevent the study of dynamic effects and pre-trends.

[10] Alternatively, researchers could focus on testing for specific changes in the outcome variable after treatment that are invariant up to a linear transformation of the path of the dynamic causal effects $\{\gamma_k\}_{k=-\infty}^{\infty}$, i.e. they are identified. A change in the slope of the outcome variables or a sudden jump in the outcome variable after treatment are examples of parametric specifications that could be tested despite the underidentification problem.

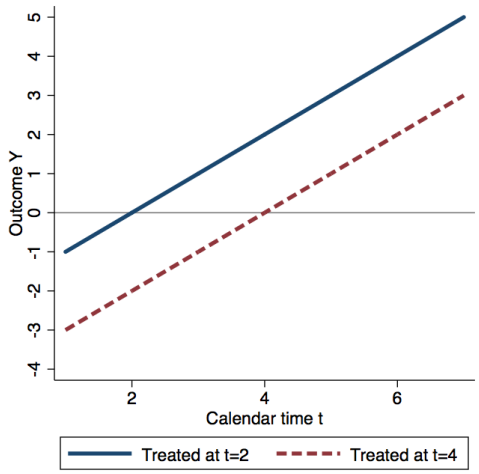
fixed effects are also all set equal to zero). Panel B shows this idea in a slightly different way, under another data generating process. This panel plots the path of outcomes for two cohorts treated at different points in time. The paths of these outcomes are equally consistent with *either* linearly growing causal effects/pretrends, no individual fixed effects and no calendar effects, *or* no causal effects, no pretrends but linearly growing calendar effects and unit fixed effects that are larger (by two units of the outcome) for the cohort treated earlier in the sample. Finally, Panel C shows that non-linearities break the underidentification: the change in slope around the time of the event can only be explained by the causal effect of treatment. We can now turn to a formal investigation of these issues.

Figure 1: Underidentification of Fully Dynamic Specification

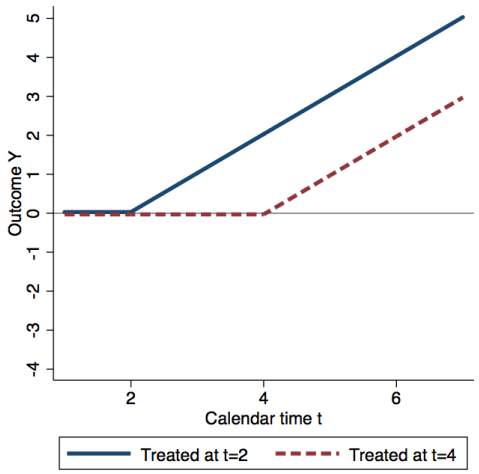
Panel A



Panel B



Panel C



Formally, note that for any constant h,

$$\begin{aligned} \hat{Y_{it}} &\equiv \alpha_i + \beta_t + \sum_{k=-\infty}^{\infty} \gamma_k \mathbf{1}\{K_{it} = k\} \\ &= (\alpha_i + h \cdot E_i) + (\beta_t - h \cdot t) + \sum_{k=-\infty}^{\infty} (\gamma_k + h \cdot k)\, \mathbf{1}\{K_{it} = k\} \end{aligned} \tag{5}$$

because by definition $t - E_i = K_{it}$. As a result, the dynamic causal effects $\{\gamma_k + h \cdot k\}$ fit the data just as well as the original $\{\gamma_k\}$ path, although these two sets of coefficients paint vastly different pictures about the causal effects.

To gain further intuition about the nature of the underidentification, we show that the empirical model above nests a specification with collinear terms. Specifically, replace the set of unit fixed effects $\{\alpha_i\}$ with a linear predictor in initial treatment period $\lambda + \alpha E_i$ (i.e. the outcomes of different "cohorts" of units experiencing the event at different times are allowed to differ in a linear way), the set of year fixed effects $\{\beta_t\}$ with a time trend βt, and the set of fully dynamic causal effects $\{\gamma_k\}$ with a trend in relative time γK_{it}. The fundamental underidentification problem described above can be seen immediately in the following regression:

$$Y_{it} = \lambda + \alpha E_i + \beta t + \gamma K_{it} + u_{it}$$

given that $t - E_i = K_{it}$. In other words, the presence of a linear term in the initial treatment period is necessary for the identification problem to arise. Individual fixed effects subsume such effects. In the presence of a control group, cohort fixed effects or individual fixed effects do not cause any identification problem because the control group pins down the year effects. The problem is effectively the same as the (well-known) age-cohort-time problem in the regression

$$Y_{it} = \underbrace{\alpha_{E_i}}_{\text{Cohort FE}} + \underbrace{\beta_t}_{\text{Time FE}} + \underbrace{\gamma_{t-E_i}}_{\text{Age FE}} + u_{it},$$

where E_i is the date of birth.

We want to stress that typically only a linear component of the $\{\gamma_k\}$ path is not identified.[11] It is not possible to reproduce a nonlinear $\{\gamma_k\}$ path perfectly with unit and period fixed effects. The reason is that by definition, such a path is a nonlinear function of $\gamma(t - E_i)$, and it cannot be represented as a sum of any functions $\alpha(E_i)$ and $\beta(t)$.[12]

In sum, the dynamic schedule of causal effects $\{\tilde{\gamma}_k\}_{k=-\infty}^{\infty}$ is identified only up to a linear trend because one cannot disentangle the effects of passing of absolute time t and relative time k when there is no control group and in presence of unit fixed effects. More specifically, unit fixed effects create an identification problem because they subsume "linear cohort effects" (i.e. linear predictors of the form $\lambda + \alpha E_i$). The calendar year (t) is equal to the year in which the event happens for unit i (E_i) plus the "relative time" (K_{it}): there is a perfect linear relationship between these effects and it is therefore impossible to observe independent variation in these variables.

3.2 Solutions

3.2.1 Overview

As we explained above, calendar time, relative time, and a linear term in the initial treatment period cannot be included together in the regression. To avoid this, additional restrictions on the statistical model have to be

[11] There are some exception to these rules, e.g. when treatment is staggered but happens at periodic intervals.

[12] To see why this is the case, imagine that t and E_i are continuous and take a cross-partial derivative $\partial^2 \gamma(t - E_i)/\partial t \partial E_i = -\gamma''(t - E_i) \neq 0$, whenever γ is nonlinear. In contrast, $\alpha(E_i) + \beta(t)$ always has zero cross-partial.

imposed. Dropping unit fixed effects is an immediate fix, with the advantage of being very easy to implement, but it suffers from two important drawbacks: it requires strengthening the identification assumption, and it reduces power. Although dropping unit fixed effects may be a reasonable approach in some settings, we develop two other approaches that address underidentification and do not suffer from these drawbacks. Our first strategy is to restrict the pre-trends in the fully dynamic specification, while keeping unit fixed effects, and we show how to test this restriction. Our second strategy is to replace unit fixed effects with unit random effects, which is also testable.

Both of these strategies can be justified by reasonable assumptions about the nature of the variation in the timing of the event across units. The restriction of pre-trends is justified when the event is *unpredictable* conditional on unit characteristics, while the random effects model is warranted when the timing of the event is *randomly assigned* across units.[13] Consider two examples to clarify this distinction. When the effect of health shocks on income is of interest (e.g. Dobkin et al., 2015), it is plausible that low-income individuals may be less healthy and get hospitalized earlier on average. Yet, conditional on permanent income level, individuals may not be able to predict when the health shock will happen. In contrast, in the case of tax rebates studied by Parker et al. (2013), people could know when they are getting the rebate. However, the date was assigned based on the last two digits of the Social Security Number, so it was uncorrelated with any relevant individual characteristics.

The following subsections develop each of these approaches. We also discuss two further issues: the role of unit fixed effects in balanced samples, and the possibility of adding a control group as a solution to the identification problem.

3.2.2 Restricting Pre-Trends

We begin with the situation where event timing is supposed to be randomly assigned *conditionally* on the fixed effect $\tilde{\alpha}_i$, and unpredictable. The former assumption justifies the use of difference-in-differences type approaches. And latter one means that the outcome cannot be adjusted based on anticipation of the event, so there can be no pre-trends, $\tilde{\gamma}_k = 0$ for $k < 0$. This assumption can be tested statistically and graphically, and then imposed for efficient estimation of causal effects. We discuss these issues in order.

Under the no pre-trends null hypothesis, the true model is semi-dynamic, which has no identification issues. The alternative allows for the fully dynamic specification, which is only set identified. Despite this, the F-test works and can be implemented in the following straightforward way. Start from the fully dynamic regression and drop *any* two terms corresponding to $k_1, k_2 < 0$. This is the minimum number of restrictions for point identification, to pin down a constant and a linear term in K_{it}. Then use the F-test on the pre-trends remaining in the model.[14] The F-test compares the residual sums of squares under the restricted and unrestricted specifications, where the former is always semi-dynamic, and the latter is fully dynamic with two restrictions. Precisely due to underidentification, the fully dynamic specification with two restrictions is effectively unrestricted and its fit is identical for any k_1 and k_2, so the F-statistic will be invariant to k_1 and k_2 even in finite samples.

This test has power only against non-linear pre-trends. Indeed, nothing in the data can point to a linear pre-trend—that is the essence of underidentification. However, if the empirical design is actually flawed, i.e. event timing is correlated with unobservables, there is no reason for pre-trends to be exactly linear, and the test will detect them.

While we are not aware of any empirical papers implementing this F-test, a common way to check for pre-trends is to plot the path of $\hat{\gamma}_k$ before and after treatment. Sometimes this is called the event study approach. It originates from the standard difference-in-differences setup, where only an additive constant in $\tilde{\gamma}_k$ is not identified, and it is irrelevant for visual inspection; $\hat{\gamma}_{-1}$ is typically set to zero. In contrast, in the staggered design two restrictions

[13] These two senses of "randomness" of the event timing appear to have been conflated in the existing literature.

[14] Both the restricted and unrestricted specifications are identified now, so standard results about the F-test behavior apply.

have to be made. Different choices of the two restrictions, $\hat{\gamma}_{k_1} = \hat{\gamma}_{k_2} = 0$, will matter a lot for the whole picture: the whole path of estimated $\hat{\gamma}_k$ gets rotated by adding $\hat{h} \cdot k$ for some constant $\hat{h}$. If there are no pre-trends in the data-generating process, $\hat{h}$ asymptotically converges to zero for any k_1 and k_2 as the number of units grows. However, in finite samples difference may be large, particularly in longer panels, since $\hat{h}$ is multiplied by k.

So how does one pick the two omitted categories? While choosing $k_1 = -1$ and $k_2 = -2$ might seem natural, we propose setting the omitted categories far apart. Under the null hypothesis, this greatly reduces standard errors for most individual coefficients on the graph. To understand why, imagine that a line on a plane is drawn through two points with fixed x-coordinates $x_1 \neq x_2$, but stochastic y-coordinates, for simplicity with mean zero. The position of the line will be much more stable when x_1 and x_2 are far from each other. This is true both for the slope of the line (the analog of $\hat{h}$) and its value at a typical x (the analog of the $\hat{\gamma}_k$). The fully-dynamic specification with two restriction effectively draws a line and evaluates all dynamic effects relative to it. When k_1 is far from k_2, e.g. $k_1 = -1$ and k_2 close to the most negative value of K in the sample, it will be much less likely that a linear pre-trend (although perhaps statistically insignificant) will be visible. Remember that linear pre-trends are never possible to detect in the data, so choosing $k_2 = -2$ would only reduce the usefulness of the graph, distracting attention from nonlinearities in the pre-trends.

Even if the two restrictions are chosen well, this graph should only be used to evaluate pre-trends; it does not estimate the treatment effects efficiently. Once the researcher is comfortable with the assumption of no pre-trends, all γ_k, $k < 0$, should be set to zero. The semi-dynamic specification should be estimated and its coefficients plotted to provide a graphical illustration of the dynamics of causal effects.[15]

3.2.3 Unit Random Effects

We now consider a second sense in which the timing of the event is random: the treatment period E_i is independent of the relevant unit characteristics—in our model (1), the time-invariant unit intercept $\tilde{\alpha}_i$. In such a setting, the estimation can be carried out without unit fixed effects, which are no longer necessary for the research design. Dropping unit fixed effects immediately addresses the underidentification problem.[16] However, doing so reduces efficiency: instead, we propose to carry out estimation in a random effects model. In addition to increasing efficiency, another advantage of using a random effects model is that we can test the hypothesis that the treatment period is independent of the unit fixed effects. As in the case of the no pre-trends assumption, the random effects assumption can be tested against some, although not all, alternatives, and then imposed to regain identification.[17]

When we discussed underidentification of the fully dynamic specification, we emphasized that for any path of $\{\gamma_k\}$, identical fit of the model can be produced with the path $\{\gamma_k + h \cdot k\}$. But the same result holds for the unit fixed effects: $\{\alpha_i - h \cdot E_i\}$ and $\{\alpha_i\}$ fit the data equally well, as long as other coefficients in the model are adjusted appropriately (see equation (5)). As a consequence, it is impossible to test whether $\tilde{\alpha}_i$ is *uncorrelated* with E_i—the estimates can always be made uncorrelated by choosing h. Yet, *independence* is testable. Given the nature of $\tilde{\alpha}_i$ as an additive term, the most reasonable null hypothesis would be $\mathbb{E}[\alpha_i \mid E_i] = 0$.[18]

Since we are ultimately interested in the causal effects $\tilde{\gamma}_k$, our preferred approach for testing this null is based on the Hausman test. Under the null, the model can be efficiently estimated using random effects, imposing only one normalization on $\{\gamma_k\}$. As before, we choose $\gamma_{-1} = 0$. The unrestricted model is set identified, but we pick the unique solution for $\{\alpha_i\}$, $\{\beta_t\}$ and $\{\gamma_k\}$, which satisfies $\gamma_{-1} = 0$, plus two familiar conditions on the unit

[15] Note that in case there is truly a linear trend in the set of $\{\tilde{\gamma}_k\}_{-\infty}^{\infty}$, then the results from the fully dynamic specification can be interpreted as a test for any change relative to this linear trend around the time of the event.

[16] Recall that unit fixed effects create an identification problem because they subsume "linear cohort effects"

[17] For a reminder on fixed vs. random effects models, see appendix XX

[18] An example of a conceptually different moment restriction which could be tested but would not provide much intuition is $Cov\left(\alpha_i^2, E_i\right) = 0$.

effects: $\sum_i \alpha_i = 0$ and $\sum_i \alpha_i E_i = 0$. These conditions guarantee that under the null, the fixed and random effects estimands $\{\gamma_k\}$ will be identical.[19]

Now we have a classical Hausman testing setup: two estimators have the same probability limit under the null, but one of them—the random effects estimator—is efficient. The variance-covariance matrix of $\{\hat{\gamma}_k\}$ in the random effects model is standard. Our fixed effects estimator is a special case of OLS with a large number of covariates and certain linear restrictions, therefore it is theoretically straightforward. In ongoing work, we study how to implement it in a computationally efficient way (without inverting large matrices) using the standard fixed effects machinery.

When the researcher is comfortable to impose the independence assumption, they should use the random effects estimator as their preferred one for the full path of $\{\tilde{\gamma}_k\}$. Remember that in general, the setup does not imply there are no pre-trends. If the units' outcomes can adjust to the randomized, yet known in advance event timing, the pre-trends are part of the treatment effect.

3.2.4 Related Issues

Using a Balanced Sample: As previously discussed, without unit fixed effects there is no underidentification problem, but in some settings the research design requires the inclusion of unit fixed effects, for instance if treatment is unpredictable only conditional on some time-invariant characteristics. In such settings, a potential easy fix for the underidentification problem would be to drop fixed effects and balance the sample around the initial treatment period (i.e. restrict the sample such that each unit appears for the same number of periods before and after the initial treatment period). Indeed, there is a view that unit fixed effects are essentially irrelevant in event studies on a balanced panel.

The intuition underlying this view is that balancing the sample addresses one key issue that is handled by unit fixed effect in unbalanced panels: the changing composition of the sample. Omitting unit fixed effects when working with an unbalanced panel may be a big assumption because of selection into treatment. For instance, units that are treated earlier in the sample may be different from other units and because the sample is unbalanced they spend a bigger share of the sample under treated status. Therefore, in the absence of unit fixed effect one would worry that the estimated coefficient on the treatment dummy may partly reflect selection.

In fact, balancing the sample does *not* help address such selection effects. We show in Appendix D that omitting unit fixed effects when working with *balanced* panels is just as worrisome as when working with *unbalanced* panels (for the purpose of dealing with selection effects). A balanced panel appears to solve the "selection issue" that is salient in the case of unbalanced panels because each unit gets treated for the same number of years during the panel. However, in practice year fixed effects absorb all of the variation for years at the beginning (where all units are untreated) or end (where all units are treated) of the sample. For this reason, the point estimate we obtain for the treatment effect by running a regression in a balanced sample is exactly the same as the one obtained by running the same regression in this balanced sample further restricted to years in the middle of the sample (which means we are effectively running a regression on an *unbalanced* panel). Appendix D discusses this more formally and shows that endogenous selection effects in the data generating process affects the consistency of the point estimate in the same way under balanced and unbalanced panels.

Adding a Control Group: A number of empirical papers using random timing research designs start with a sample which includes units that are never treated, or could conceivably collect such a sample. Therefore, it would be possible to include a control group of units that never experience treatment in the estimation sample. In many instances, it is difficult to construct a control group that plausibly provides valid counterfactuals for the treatment group. But assuming that such a control group is available, then including it in the estimation sample solves the

[19] The same holds true for $\{\beta_t\}$, but we are not interested in estimating them.

underidentification problem because the control group can be used to estimate the year effects independently of the causal effect of teatment. However, the strategy of adding a control group has two important limitations. First, if the control group is small relative to the treatment group, important finite-sample issues can arise. Second, one cannot allow the control group to be on its own time trend, otherwise the underidentification problem is left intact. We discuss both of these issues in greated depth in Section 5.

4 Negative Weighting in Canonical Regression

4.1 Problem

In this section we show that fundamental underidentification discussed above creates problems for more restricted specifications. Our flagship case will be the canonical regression (4), but the argument extends to all specifications with two-way fixed effects, which do not allow for flexible dynamic treatment effects. We show that these regressions estimate an average of treatment effects that severely overweighs short-term effects and weighs long-term effects negatively. That is, if programs P1 and P2 have similar short-term effects but P1 is uniformly more efficient in the long-term, the canonical regression will show that P1 has *lower* average effect.

Assume that the design is valid in the sense that there are no pre-trends, so the true model is semi-dynamic (2). People often summarize treatment effects by the γ coefficient from the canonical regression,

$$Y_{it} = \alpha_i + \beta_t + \gamma D_{it} + \varepsilon_{it}.$$

This specification is valid under a restriction that $\tilde{\gamma}_k$ are equal for all $k \geq 0$, i.e. that treatment leads to an immediate and permanent jump in the outcome variable and no further effects. This restriction should not hold in most applications, in our view. Quite often treatment effects are growing or decaying over time, may kick in with a delay, etc. However, there is a perception that γ should estimate average treatment effects with some reasonable weights. While true in some other contexts (e.g. []), we show that this logic does not apply to the canonical regression estimand—the weights are not even necessarily positive.

Our first argument is that γ is indeed a weighted average of $\{\tilde{\gamma}_k\}$ with weights which can be easily estimated from the data and solely depend on the *grid*—the distribution of treatment periods E_i and sample selection.

Lemma 1. *The canonical regression OLS estimand can be expressed as a weighted average of dynamic treatment effects,*

$$\gamma = \sum_{k=0}^{\infty} \omega_k \tilde{\gamma}_k$$

with weights ω_k that sum up to one and equal the coefficients for D_{it} from in the following regressions:

$$\mathbf{1}\{K_{it} = k\} = FE_i + FE_t + \omega_k D_{it} + noise, \qquad k \geq 0. \tag{6}$$

where FE_i denotes unit fixed effects and FE_t time fixed effects.

To gain intuition for this lemma, note the following: by linearity of OLS, one can recover the coefficient for D_{it} in the canonical regression with Y_{it} as the outcome by instead running two canonical regressions with subcomponents of Y_{it} as the outcome variable and then summing up the coefficients on D_{it} obtained in each of these regressions.[20]

[20] To be explicit, if $Y = A + B$, then the specifications

$$Y = \beta^Y X + \epsilon$$
$$A = \beta^A X + \eta$$

Consider first running a canonical regression with $\left(\tilde{\alpha}_i + \tilde{\beta}_t + \tilde{\varepsilon}_{it}\right)$ as the outcome (one could do this in theory if the true parameters of the model were known), and then another canonical regression with $\sum_{k=0}^{\infty} \tilde{\gamma}_k \mathbf{1}\{K_{it} = k\}$ as the outcome, and finally sum up the coefficients for D_{it} obtained in each of these two regressions to recover γ. The first regression will load on the fixed effects and not on D_{it}, so γ comes solely from the second regression. By the same logic, one further notes that γ can be recovered by running a series of canonical regressions with $\tilde{\gamma}_k \mathbf{1}\{K_{it} = k\}$ as the outcomes (repeating for all $k \geq 0$) and summing up the coefficients. Since $\tilde{\gamma}_k$ is a multiplicative constant in $\tilde{\gamma}_k \mathbf{1}\{K_{it} = k\}$, each of these canonical regressions generates coefficients for D_{it} that can be written $\tilde{\gamma}_k \cdot \omega_k$, for ω_k determined by specification (6). Importantly, the only variables required by (6) are the unit identifier, calendar time, and relative time—what we call the grid.[21]

Now the question is whether these ω_k weights are "reasonable" in some sense, and our answer is strongly negative. Although a general characterization of ω_k does not seem feasible, we demonstrate our result in three ways: by proving and applying a very general result on negative weighting by OLS, then by means of a special case where we get a striking closed-form solution for ω_k, and then by solving for them in the general case for a simpler specification.

We show in Appendix C that the weighting scheme implicitly used by OLS is determined by the residuals from the linear propensity score regression. That is, suppose treatment indicator D_{it} is regressed on all other right-hand side variables—unit and period fixed effects in our case. Then observations it which have fitted values above D_{it} (and hence negative residuals) will have a negative weight in the original canonical regression—larger Y_{it} produces smaller γ. Although D_{it} is a dummy variable, the linear probability model can easily generate fitted values $\hat{D}_{it} > D_{it} = 1$ for some post-treatment observations. The outcome variable Y_{it} contains treatment effects for these observations, and those will be weighted negatively.

Which observations could suffer from this problem? As once-treated units stay treated forever, the probability of being treated increases over time, so time fixed effects in the probability score regression should be increasing in t. Similarly, units that are treated earlier (with small E_i) are treated for a larger fraction of the periods. Therefore, fitted values will be particularly large for observations corresponding to early treated units at the end of the sample—precisely those which identify long-term treatment effects. They will be negatively weighted, or at least underweighted, by the canonical regression.

This argument also implies that negative weighting will not happen in models without individual fixed effects, such as

$$Y_{it} = FE_t + \gamma D_{it} + \text{noise}.$$

Fitted values from the simple propensity score regression of D_{it} on all calendar time dummies are fractions of treated units in each year, which always lie between zero and one. This result is consistent with Angrist (1998) who show that when the regression is *saturated*, i.e. solely includes dummies for all levels of a single categorical variable, OLS weights treatment effects by the variance of treatment conditional on the controls (see also Angrist and Pischke, 2008, sec. 3.3.1). Such variance is of course non-negative.

Now consider a specific, very reasonable grid.

Proposition 1. *Suppose there are $T \geq 2$ time periods, E_i is distributed uniformly among them, and for each unit*

$$B = \beta^B X + \zeta$$

yield

$$\beta^Y = \frac{Cov(Y, X)}{Var(X)} = \frac{Cov(A, X) + Cov(B, X)}{Var(X)} = \beta^A + \beta^B.$$

The result holds for multivariate X.

[21] To show that weights always add up to one, imagine that $\tilde{\gamma}_k = \tilde{\gamma} \neq 0$ for all $k \geq 0$. The canonical regression is then correctly specified and provides consistent estimate $\gamma = \tilde{\gamma}$. But $\gamma = \sum_{k \geq 0} \omega_k \tilde{\gamma}$, so $\sum_{k \geq 0} \omega_k = 1$. Since weights do not depend on the true outcomes, this result holds generally.

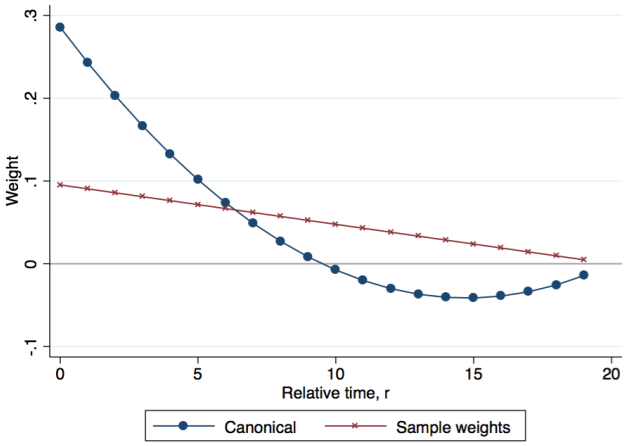
i, the outcome is observed for all periods. Then,

$$\omega_k = \frac{(T-k)(T-2k-1)}{T(T^2-1)/6}, \quad k = 0, \ldots, T-1.$$

Strikingly, $\omega_k < 0$ *for* $k > (T-1)/2$.[22]

Figure 2 illustrates this proposition by plotting the weights ω_k for $T = 20$. For comparison, it also shows the fraction s_k of observations with each k in the post-treatment sample, which could be a possible definition of a reasonable weighting scheme. It is clear that short-term effects are severely overweighted, whereas long-term effects enter negatively. That means that a program that produces uniformly larger effects may look worse in the canonical regression.

Figure 2: Weights in Canonical Regressions



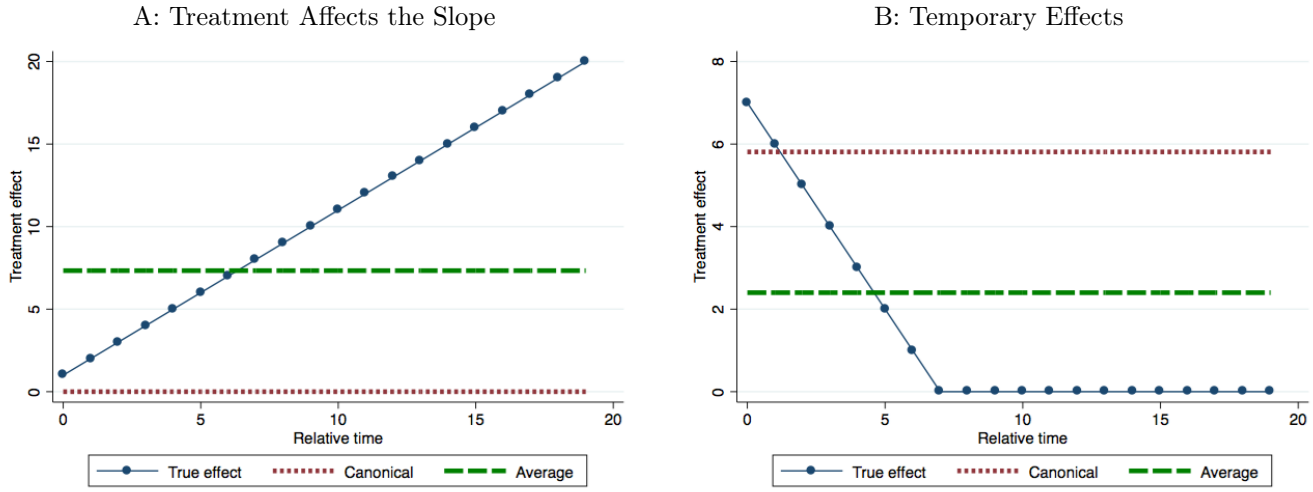
When treatment effects have strong dynamics, there will be a wide discrepancy between the sample size-weighted average treatment effects and the canonical regression estimand. Figure 3 shows two examples of this. Panel A corresponds to the case treatment permanently changes the *slope* (growth rate) rather than the level of the outcome, i.e. $\tilde{\gamma}_k = k+1$ for $k \geq 0$. Canonical regression completely misses the effects, estimating γ to be zero! The following corollary formalizes the result:

Corollary 1. *Suppose the assumptions of Proposition 1 hold. Then, when treatment changes the slope of the outcome's growth, i.e.* $\tilde{\gamma}_k = \varphi(k+1)$ *for* $k \geq 0$ *and some constant* φ*, then the canonical regression OLS estimand* $\gamma = 0$*, regardless of* φ *and the panel length* T*.*

The canonical regression coefficient in this case lies outside of the convex hull of the effects at each time horizon. We show later that this is not just a theoretical possibility but happens in applications. Although negative weights in Figure 2 are not very large, they are multiplied by the large treatment effects at longer horizons in this example.

[22] All proofs are given in Appendix B.

Figure 3: Biases of Canonical Regressions



Panel B of Figure 3 considers a situation when treatment effects are temporary, and the outcome gradually (in seven periods) reverts back to the original trajectory. Canonical regression will produce a large coefficient close to the largest, very short-term effect, and does not characterize the average well.

How is the weighting problem related to the underidentification of the fully dynamic regression? Recall that two-way fixed effects can reproduce a linear trend in the relative time K_{it}, which is at the core of the underidentification problem. Consider the following regression that is nested within the canonical one:[23]

$$Y_{it} = \mu + hK_{it} + \gamma^R D_{it} + \varepsilon_{it}. \tag{7}$$

As in Lemma 1, $\gamma^R = \sum_{k\geq 0} \omega_k^R \tilde{\gamma}_k$ with weights that can be estimated from a series of regressions

$$\mathbf{1}\{K_{it} = k\} = \mu + h_k^R K_{it} + \omega_k^R \mathbf{1}\{K_{it} \geq 0\} + \text{noise}, \quad k \geq 0. \tag{8}$$

This regression (8) is defined solely in terms of a single variable K_{it}. Figure 4 illustrates it in the context of Proposition 1 for two values of k: small ($k = 3 \ll T = 20$) and large ($k = 12$). Specification (8) fits the left-hand side variable with two parallel lines of any location and slope, and ω_k^R is the shift between them. When k is large, this shift can be negative (see panel B). Moreover, short-term effects are always overweighted, as formalized by the following proposition:[24]

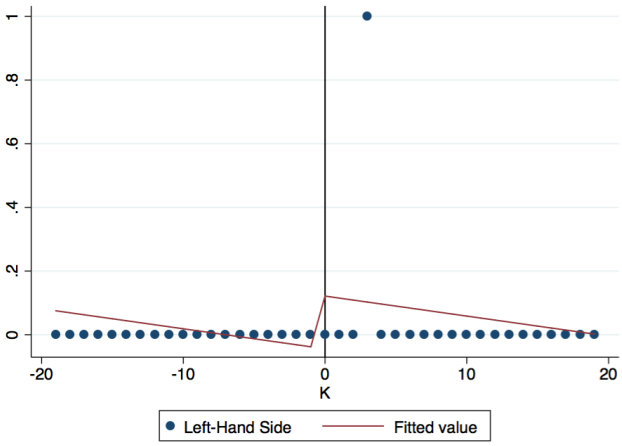
Proposition 2. *The weight schedule implied by regression (7) is always short-term biased. That is, $\omega_k^{Restricted}/s_k$ is decreasing in k. It is greater than 1 if and only if k is smaller than the average K_{it} post-treatment, i.e. $k < \sum_{l=0}^{\infty} l s_l$.*

[23] To get it, plug in $\alpha_i = -hE_i$ and $\beta_t = \mu + ht$.

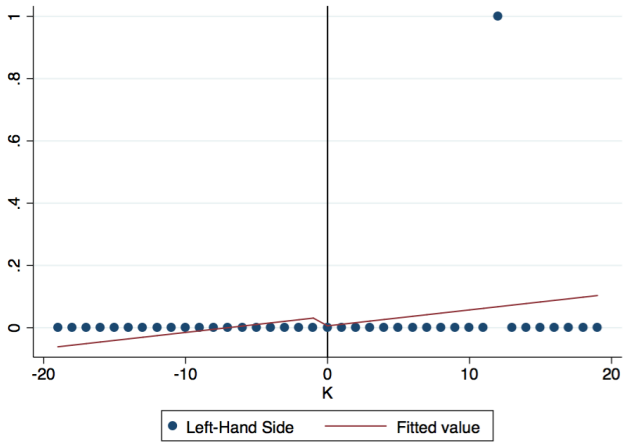
[24] It should be primarily viewed as providing intuition because $\omega_k \neq \omega_k^R$ in general.

Figure 4: Estimation of Specification (8)

A: For Small $k = 3$



B: For Large $k = 12$



The weighting problem extends to more flexible specifications with two-way fixed effects. Consider for example, a "capped" specification

$$Y_{it} = \alpha_i + \beta_t + \sum_{k=0}^{B-1} \gamma_k \mathbf{1}\{K_{it} = k\} + \gamma_{B+} \mathbf{1}\{K_{it} \geq B\} + \varepsilon_{it}.$$

If treatment effects after $K_{it} = B$ are not constant, as assumed by this specification, this will make the estimate of γ_{B+} unreliable. But also, through the wrong choice of individual and time fixed effects, short-term effects will be biased. Figure 5 provides an illustration for $B = 6$ when the true effects are growing linearly, as in Figure 3A. The long-term effect is outside of the convex hull of the true effects for $k \geq 6$, and short-term effects are downward biased.

4.2 Solutions

Unlike with the underidentification issue, the solution for the weighting problem is quite straightforward. Researchers should never run regressions which impose any restrictions on the dynamics of treatment effects post-treatment. They should estimate them flexibly and average the coefficients manually with some weights (e.g. proportionately to the sample size).[25] When pre-trends can be assumed away, that amounts to fitting the semi-dynamic regression (2). If some anticipation effects are possible, a more general specification (3) should be employed with the number of leads A reflecting the horizon of anticipation. Here $A < \infty$ is required for identification, and $B = \infty$ is necessary to avoid weighting problems.

Another solution is to find a valid control group that never experiences treatment, yet faces the same time effects $\tilde{\beta}_t$. The control group helps identify the time effects, alleviating the problem. Importantly, the control group should not be allowed to be on a separate time trend (see Section 5.1.1).

Note, however, that if the control group is small *relative to the treatment group*, it would not help. For the problem to disappear, time effects should be identified *solely* from the control group. Figure 6 shows, in the context of Proposition 1, how weights ω_k vary with the fraction of units (and, equivalently, observations) in the control group. Having a 10% control group does almost nothing, and even with equally sized groups, the weighting scheme

[25] We provide the Stata code for this in Appendix **??**.

is still quite far from s_k. Running a flexible specification and averaging the effects manually seems worthwhile even in presence of a control group.[26]

Although this approach solves the problem when the only type of treatment effect heterogeneity is across the time horizon, averaging of the effects across individuals or calendar times, that is implied by the semi-dynamic specification, may not be perfect. We return to this problem in Extensions, proposing a matching procedure that works more generally.

5 Extensions

It should by now be clear to the reader that both problems—underidentification and negative weighting—stem from the ability of two-way fixed effects to recover the relative time variable K_{it} in our staggered design without the control group. Here we demonstrate that equivalent problems may arise in a variety of different empirical designs.

We also extend our setting to the case with more general treatment effect heterogeneity and show that OLS for the semi-dynamic specification has undesirable extrapolation properties. We propose a matching scheme robust to this type of heterogeneity.

5.1 Related Problems in Other Empirical Designs

5.1.1 Difference-in-differences with Group-specific Time Trend

Consider first the standard difference-in-difference setup where all units in the treatment group ($G_i = 1$) are treated in same period E, whereas control group units ($G_i = 0$) are never treated. The treatment indicator is $D_{it} = Post_t \cdot G_i$, where $Post_t = \mathbf{1}\{t \geq E\}$. While the plain vanilla event study specification

$$Y_{it} = \alpha_i + \beta_t + \sum_{k=-\infty}^{\infty} \gamma_k \mathbf{1}\{t - E = k\} \cdot G_i + \text{noise}$$

does not suffer from any identification problems, sometimes researchers are not confident in the quality of their control group and would like to include a group-specific trend,

$$Y_{it} = \alpha_i + \beta_t + \mu t \cdot G_i + \sum_{k=-\infty}^{\infty} \gamma_k \mathbf{1}\{t - E = k\} \cdot G_i + \text{noise}.$$

Because E is the same for all units, the group-specific time trend is collinear with $(t - E)\, G_i$ in presence of individual fixed effects.

As a consequence, restricted specifications, such as

$$Y_{it} = \alpha_i + \beta_t + \mu t \cdot G_i + \gamma D_{it} + \text{noise}, \tag{9}$$

suffer from negative weighting of dynamic effects, *regardless* of the relative size of the control group. Figure 7 illustrates this point for the case when there are $t = 1, \ldots, 20$ periods, and treatment happens in the middle, $E = 11$.

[26] A lazy man's alternative is to "boost" the control group: use weighted OLS where all control group observations are weighted by a very large number, e.g. 1,000 times more than the control group. This may increase standard errors but from the identification point of view, it is equivalent to having a very large control group. This weighted OLS can be viewed as a two-stage procedure: time effects are first identified from a regression $Y_{it} = \alpha_i + \beta_t + \text{noise}$ on the control group only, and then used in the canonical regression for the treatment group.

One can replace individual fixed effects with group-specific intercepts in (9),

$$Y_{it} = \beta_t + (\alpha + \mu t) \cdot G_i + \gamma D_{it} + \text{noise},$$

or include unit-specific trends instead,

$$Y_{it} = \alpha_i + \beta_t + \mu_i \cdot t + \gamma D_{it} + \text{noise}.$$

All of these specifications are affected by the same problem.[27]

As we mentioned in the previous sections, the issues equally arise in *staggered* designs with a control group if either group- or unit-specific time trend is included. Indeed, the relative time in the treatment group, $(t - E_i) G_i$, can be recovered from $\alpha_i + \mu t \cdot G_i$.

5.1.2 Unit-Specific Trends without Control Group

Return now to the staggered design without a control group. When the empirical strategy is not perfectly convincing, it is sometimes recommended to check robustness to including unit-specific (e.g., state-specific) time trends:

$$Y_{it} = \alpha_i + \beta_t + \mu_i \cdot t + \sum_{k=-\infty}^{\infty} \gamma_k \mathbf{1}\{K_{it} = k\} + \text{noise}.$$

Without the control group, this creates an additional problem. Not only a linear term K_{it} can now be recovered by fixed effects, but also a quadratic term $(K_{it})^2$. Indeed,

$$(K_{it})^2 = (E_i)^2 + t^2 - 2E_i t.$$

These three components are nested by α_i, β_t, and $\mu_i \cdot t$, respectively. As a result, the fully dynamic path of treatment effects is identified only up to a quadratic polynomial. Three restrictions instead of two must be imposed to regain identification, and the F-test described above is only powerful against pre-trends which have more complicated shape.

Correspondingly, weighting in the canonical-type regression

$$Y_{it} = \alpha_i + \beta_t + \mu_i \cdot t + \gamma D_{it} + \text{noise}$$

becomes even worse than before.[28]

Given our results in the previous subsection and here, we do not recommend including unit-specific time trends in any difference-in-difference or event study specifications (except for the case discussed in footnote 27).

5.2 Treatment Effect Heterogeneity

Throughout the paper, we imposed a strong assumption that the fully dynamic specification characterizes the true data generating process. Treatment effects are required to depend only on the time relative to treatment, K_{it}, but otherwise are homogenous across units and calendar time periods. Formally, we define treatment effects in terms of potential outcomes as $\tau_{itk} = Y_{it}^{(k)} - Y_{it}^{(-1)}$, and the fully dynamic specification requires $\tau_{itk} \equiv \gamma_k$.

[27] In all of these cases, semi-dynamic specifications, e.g. $Y_{it} = \alpha_i + \beta_t + \mu t \cdot G_i + \sum_{k=0}^{\infty} \gamma_k \mathbf{1}\{t - E = k\} \cdot G_i + \text{noise}$, are fine. In this regression μ is estimated only using non-treated observations, while in restricted specifications treatment effects influence the estimate of μ, biasing γ.

[28] Illustrative simulations are available from the authors upon request.

Such homogeneity, particularly across time periods, is very restrictive and difficult to reconcile with economic models. To fix ideas, we consider the simplest setting where event timing is fully random and unpredictable. All units (for simplicity, let us think of them as people for now) have the same prior belief about when the event may happen, and they update it over time using the Bayes rule. If a person does not receive treatment at the beginning of period t, this may be a minor shock if she thinks she is likely to get it next period, or a large shock if the expected date of the event moves far into the future, and behavior will respond accordingly. Only under very specific prior distributions can the treatment effect be independent of t.

For a specific example, consider an agent who lives for $t = 1, \dots, T$ periods and is expecting to get a permanent raise of R from her baseline wage normalized to zero. The raise will happen at the beginning of a random period drawn from some distribution, and she does not get any information until the date of the raise. Her consumption follows the permanent income hypothesis, and there is no discounting. What is the effect of getting a raise on consumption at impact, τ_{it0}? If she gets the raise at time t, she is certain that the permanent income per period is R. If the event does not happen at t, it equals $R \cdot \mathbb{E}\left[T - E_i + 1 \mid E_i > t\right] / \left(T - t + 1\right)$. The treatment effect equals the difference between the two:

$$\tau_{ito} = R \cdot \frac{\mathbb{E}\left[E_i - t \mid E_i > t\right]}{T - t + 1}.$$

It is independent of t only if E_i is uniformly distributed across periods.

What is the estimand of the semi-dynamic regression when heterogeneity is allowed for? To understand this, we again invoke the result on OLS as a weighting estimator, which weights each observation by the residual from the linear propensity score regression (see Appendix C). The propensity score regression behind γ_k, $k \geq 0$, in the semi-dynamic specification is the following one:

$$\mathbf{1}\left\{K_{it} = k\right\} = FE_i + FE_t + \sum_{\substack{l=0 \\ l \neq k}}^{\infty} \rho_{kl} \mathbf{1}\left\{K_{it} = l\right\} + \text{noise}. \tag{10}$$

An observation is weighted negatively in two cases: if $K_{it} = k$ and the fitted value in (10) is above one, or if $K_{it} \neq 0$ and the fitted value is any positive number. While we did not observe the former situation in simulations, the latter was quite prevalent among treated observations. The semi-dynamic estimand γ_k is a positively-weighted average of observations with $K_{it} = k$, minus a weighted average of control observations,, plus additional terms for treated observations with $K_{it} = l \neq k$ which have both positive and negative weights summing up to zero.

The intuition for having these additional terms is that the semi-dynamic specification imposes $\tau_{itk} \equiv \gamma_k$, allowing for substantial extrapolation. For instance, γ_1 can be estimated by comparing units treated at $E_i = 1$ and 2, both observed at periods $t = 1$ and 3. Alternatively, it can be estimated from units treated at $E_i = 5$ and 6 observed at $t = 5$ and 7. The difference between the resulting estimates is consistent for *zero* when the semi-dynamic specification is true, so an estimator for γ_0 can add or subtract this difference multiplied by any constant. Such extrapolation can improve efficiency, but also makes OLS non-robust to heterogeneity.

Severe extrapolation is inevitable in some cases. Suppose for example that treatment happens to all units between periods t_F and $t_F + 3$, but we want to estimate γ_7—the effect of being treated seven periods ago compared to not having been treated yet. There is no direct difference-in-differences quasi-experiment that would help for this task, because in calendar periods where some unit has been treated for 7 periods, all units have already been treated.

However, for k smaller than the range of treatment periods, $\tilde{\gamma}_k$ can be estimated without extrapolation. As long as individual heterogeneity is captured by unit fixed effects, as we have always assumed, one can find periods $t' < t$, as well as units i and j treated at $E_i < E_j$ and observed in these periods, which satisfy $t' < E_i < t < E_j$ and $t = E_i + k$. That is, unit i has been treated at t for k periods, but not treated yet at t', whereas unit j has not

been treated in either period. When there are no pre-trends, j provides a valid counterfactual to i.

Of course, for a treated observation (i,t) there are multiple t' and potentially many units j that i can be matched with. For efficiency reasons, all of them should be used with some weights $w_{it,jt'}$, and the resulting matching estimator can be written as

$$\hat{\gamma}_k = \frac{\sum_{i,t} \left(Y_{it} - Y_{it}^{CF}\right) \mathbf{1}\left\{K_{it} = k\right\}}{\sum_{i,t} \mathbf{1}\left\{K_{it} = k\right\}}$$

where $Y_{it}^{CF} = \sum_{j,t'} w_{it,jt'} \cdot (Y_{it'} - Y_{jt'} + Y_{jt})$ is the counterfactual outcome and $\sum_{j,t'} w_{it,jt'} = 1$. Although we have not solved for the optimal weighting scheme, equal weighting of all pre-periods t', and for each t' equal weighting of all permissible units j, seems reasonable. Averaging over all observations rather than only those with $K_{it} = k$ provides a nonparametric analog to the canonical regression, which also involves no extrapolation for treated observations.

Importantly, $\hat{\gamma}_k$ constructed this way weights treated observations positively, in fact equally, to obtain the average treatment effect on the treated. This matching estimator can be rewritten as a weighting estimator, which extends Hirano et al. (2003) on regressions with controls and Abadie (2005) on difference-in-differences with a single pre-period, to our event study design. We have not derived inference for this estimator yet.

The only paper known to us which uses a similar estimator is Fadlon and Nielsen (2015), except that they require $E_j = E_i + 5$ and $t' = E_i - 2$. That reduces efficiency without relaxing any assumptions.[29]

6 Empirical Relevance

This section is available from the authors upon request.

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[29] Fadlon and Nielsen (2015) also test the identification assumptions by comparing 5-year pre-trends of treated observations and their counterfactuals. This is a nonparametric version of testing for $\tilde{\gamma}_{-1} - \tilde{\gamma}_{-6} = \tilde{\gamma}_{-2} - \tilde{\gamma}_{-7} = \ldots$, which is less powerful than what we proposed in Section 3.2.2. A nonparametric version of our test could be developed in a similar way.

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A Additional Figures and Tables

Figure 5: Biases in the Capped Regression

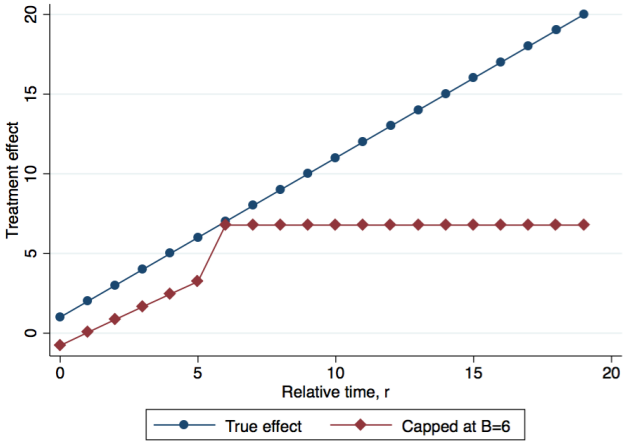


Figure 6: Canonical Weights with Control Group

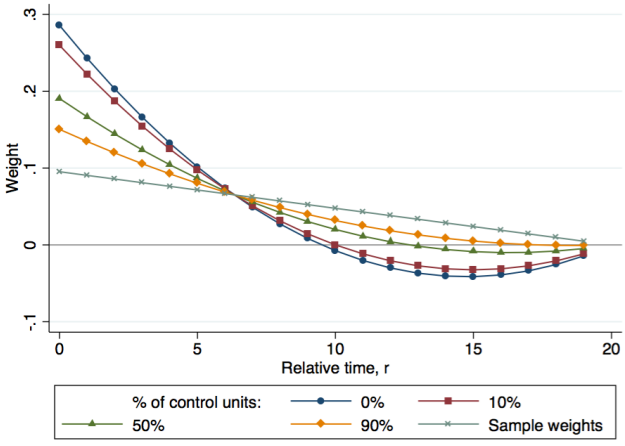
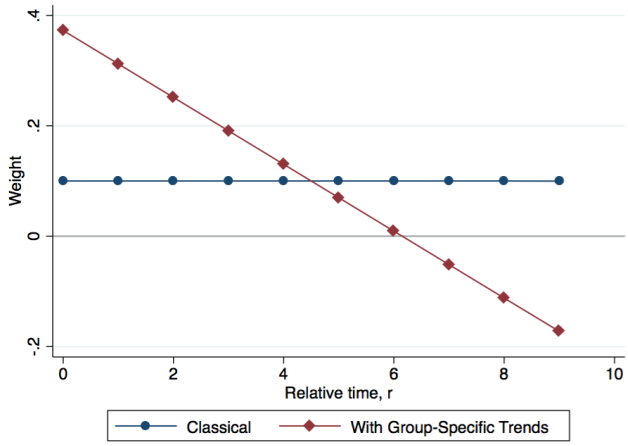


Figure 7: Weights in Diff-in-Diff Specifications



Notes: "Classical" refers to specification $Y_{it} = \alpha_i + \beta_t + \gamma D_{it}$. The specification with group-specific trends is given by (9).

B Proofs

Proof of Proposition 1. [TBA]

Proof of Corollary 1. It is straightforward to verify that weights from Proposition 1 satisfy

$$(k+1)\omega_k = -(l+1)\omega_l$$

whenever $k + l = T - 1$. Using this fact and Proposition 1, we can write

$$\gamma = \sum_{k=0}^{T-1} (k+1)\,\omega_k = \frac{1}{2}\left(\sum_{k=0}^{T-1} (k+1)\,\omega_k + \sum_{l=0}^{T-1} (l+1)\,\omega_l\right) = 0.$$

Proof of Proposition 2. [TBA]

C Negative Weighting

The derivation below illustrates why in regressions with non-saturated controls OLS may assign "negative weights" to treated observations, although one wishes to have positive weights for treated observations and negative weights for control observations. We start from a simple setup with treatment indicator D and control variables X.

$$Y = \beta D + \alpha X + \varepsilon$$

By the Frisch-Waugh-Lovell theorem,

$$Y - f(X) = \beta\,(D - p(X)) + \varepsilon^*$$

where $f(X)$ and $p(X)$ are linear projections of Y and D on X, respectively. The latter is the propensity score estimated using the linear probability model. Then,

$$\beta = \frac{Cov(Y - f(X), D - p(X))}{Var(D - p(X))}$$

We can write this as a sum of Y_is multiplied by weights, or using the population notation,

$$\beta = \mathbb{E}\left[(Y - f(X))(D - p(X))\right] \frac{1}{Var(D - p(X))} = \mathbb{E}\left[Y \cdot \omega(X)\right], \qquad \text{for } \omega(X) = \frac{D - p(X)}{Var(D - p(X))},$$

where the second equality uses the fact that $f(X)$ is a linear function of X, hence must be uncorrelated with $(D - p(X))$. It is clear that the OLS-implied weights are proportionate to the residual in the propensity score regression.

It is straightforward that $\mathbb{E}\left[\omega(X) \cdot D\right] = 1$ and $\mathbb{E}\left[\omega(X) \cdot (1 - D)\right] = -1$. That is, β is an average of the outcomes in the treatment group *minus* an average of outcomes in the control group, with weights adding up to one in both cases. In the standard Rubin causal model, one can write $Y = Y_0 + \tau D$, where Y_0 is the no-treatment potential outcome, and τ is the (heterogenous) treatment effect. Therefore, OLS estimates

$$\beta = \mathbb{E}\left[Y_0 \cdot \omega(X)\right] + \mathbb{E}\left[\tau \cdot (\omega(X) \cdot D)\right].$$

The first term (with the weights adding up to zero) represents selection bias, and the second one—the average of treatment effects. Even if selection is not a problem, the second term is problematic when $\omega(X) < 0$, i.e. $p(X) > D = 1$. This can never happen in a saturated regression, but is easily very likely for at least some observations in regressions with continuous controls or multiple sets of fixed effects.

As simple as the result is, we are unaware of any other paper showing it. Abadie et al. (2015) note for the setting with one treated and many untreated observations that the untreated ones can be weighted negatively. However, they do not connect this to the propensity score regression or, since they do not allow for multiple treated observations, to the averaging of treatment effects.

In the setting discussed in Section 4, the X is a set of individual and time dummies. $P(X)$ is the fitted values. Note that people who are treated earlier in the sample have more observations with treated status $D = 1$, and there are also more treated observations later in the sample because treatment status doesn't revert to 0. Therefore, the largest treated values are for the long-term treatment effects (high K_{it}) and we can get negative weights. These weights just depend on the "grid"—the distribution of calendar time t and initial treatment periods E_i in the sample—because there is no other variable in the propensity score regression.

This result is particularly worrisome if the treatment effect is dynamic because large treatment effects in the long run get assigned a weight of the wrong sign. Because of this, the γ estimated in the canonical regression could be outside of the convex hull of the true treatment effects $\tilde{\gamma}_k$. For instance if the treatment effect is positive and growing over time, the estimated γ could be *negative* although we were hoping it would be a weighted average of $\tilde{\gamma}_k$ (we show in Section 6 that this in fact happens in several important empirical applications).

D Unbalanced vs. Balanced Panels and Individual Fixed Effects

As discussed in Section 3.2.3, one approach to regain identification is to do away with unit fixed effects and hope that this does not pose a threat to identification. In this section, we clarify the nature of the potential threats to identification when excluding unit fixed effects for both unbalanced and balanced panels. For this section, assume throughout that there are no year effects to simplify the analysis (also note that since we are considering specifications without individual fixed effects, the other concerns with the canonical regression discussed in 4 do not

apply). We believe there is a conventional wisdom in applied work that omitting unit fixed effects when working with *unbalanced* panels is a big assumption because of selection into treatment, while omitting unit fixed effects in the case of *balanced* panels is much less problematic. We discuss below why the two settings in fact pose similar issues (as in the rest of this note, the discussion considers setting without a control group of units that never experience treatment).

Unbalanced panels. It is well understood that when panels are unbalanced, if there is a correlation between the time of treatment and the unit fixed effects (in our notation, $Cov(\alpha_i, E_i) \neq 0$), then including individual fixed effects is key. Such a correlation could result from intuitive patterns of endogenous selection into treatment. Consider for instance a setting where i) treatment has a positive and constant effect, ii) unit fixed effects α_i reflect the bargaining power of the unit, which allows for better outcomes Y_{it} in general and also for earlier selection into treatment (e.g. $Cov(\alpha_i, E_i) < 0$). In this setting, running a regression of the form $Y_{it} = \beta_t + \gamma T_{it} + u_{it}$ (without unit fixed effects) yields an upwardly biased estimate of the true constant treatment effect. Intuitively, T_{it} conveys information about the "type" of the unit: units with higher individual fixed effects are treated earlier in the sample, i.e. for a longer period of time in the sample, and the estimated treatment effect coefficient partly captures these higher fixed effects. In simulated data, this can be checked by running a regression of the form $T_{it} = \lambda\alpha_i + u_{it}$,which yields $\lambda < 0$. Balanced panel may at first glance appear to be impervious to this issue.

Balanced panels. By construction, in a balanced sample each unit gets treated for the same number of periods of the observed sample. Therefore, in simulated data with the same data generating process as discussed for unbalanced panels, running a regression of the form $T_{it} = \lambda\alpha_i + u_{it}$ yields $\lambda \approx 0$. Is it sufficient to restrict the sample to a balanced panel to address the concerns resulting from endogenous selection into treatment discussed in the case of unbalanced panels? And if not, why not and what is the link with unbalanced panels? We have verified in simulations that balancing the sample does not solve the problem, and here we provide intuition for why selection into treatment correlated with individual fixed effects in fact poses exactly the same problem in balanced and unbalanced panels. The intuition can be best seen based on the following figure:

Figure 8: Share of Treated Units and Unit Fixed Effects over Time in Balanced Sample

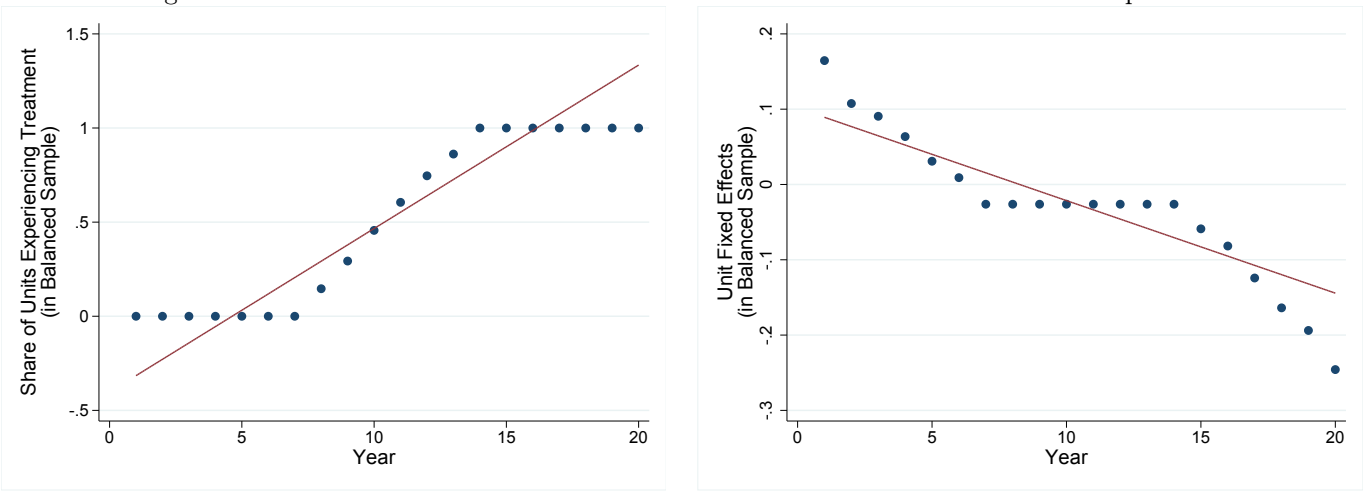


Figure 8 is based on a simulation with $Cov(\alpha_i, E_i) < 0$ ("better" units get treated earlier in calendar time), where the sample used for regressions is restricted such that each unit is observed for k years before the first year of treatment, as well as during the year of treatment and for $k-1$ years after. In other words, each unit is observed for k years under treated status and for k years under untreated status (in our simulation, $k = 7$). Figure 8 illustrates two points. First, in the first k years of the balanced sample none of the units are treated, and in the last k years

of the sample all units are treated. This means that the year fixed effects for years that are early and late in the sample will apply to a homogeneous group of units, which are all either treated or untreated - in other words, they will absorb the treatment effect in those years. This means that identification of the treatment effect coefficient will come entirely from observations in the interval of time between those cutoff dates (i.e. with t such that $k < t < \bar{T} - k$, which in our simulation amounts to 6 years, with a total number of years given by $\bar{T} = 20$). Second, there are composition effects over the years in terms of the unit fixed effects: units that show up earlier in the sample tend to have higher fixed effects (because $Cov(\alpha_i, E_i) < 0$). The composition of unit fixed effects is stable in a balanced sample for years t such that $k \leq t \leq \bar{T} - k$, which in our simulation amount to 8 years. Intuitively, a balanced sample appears to solve the "selection issue" discussed in the setting of unbalanced samples because each unit gets treated for the same number of years during the sample: however, in practice year fixed effects absorb all of the variation for years t that are not such that $k < t < \bar{T} - k$ (i.e. that are at the beginning or end of the sample). For this reason, the point estimate we obtain for the treatment effect by running $Y_{it} = \beta_t + \gamma T_{it} + u_{it}$ in the balanced sample is exactly the same as the one we obtain by running the same specification in the balanced sample further restricted to t such that $k < t < \bar{T} - k$ (the middle of the sample).[30] In this restricted sample (which is *unbalanced*!) we have $Cov(\alpha_i, E_i) < 0$.[31] Intuitively, once we eliminate the observations that are at the very beginning and the very end of the sample, we are left with a sample where units with higher fixed effects spend a bigger share of the sample under treated status.[32] This shows that endogenous selection effects in the data generating process affects the consistency of the point estimate in the same way under balanced and unbalanced panels.

Another way to summarize the intuition is as follows: the idea of irrelevance of unit fixed effects in balanced panel is coming from the observation that T_{it} is orthogonal to the unit dummies in the balanced sample. This holds unconditionally but fails conditionally on the time dummies, which are always included in these regressions. This failure is obvious: if ones fixes t, the dependence between T_{it} (equivalently, E_i) and the true unit fixed effects is precisely the problem we were hoping to address - and it exists even restricting the sample to individuals observed at t in the balanced sample.

[30] We have verified in simulation that the point estimate and standard errors for the treatment effect are indeed exactly the same in the balanced sample and the balanced sample with the further restriction that $k < t < \bar{T} - k$.

[31] We have verified this in the simulation by running $T_{it} = \lambda \alpha_i + u_{it}$ in the balanced sample restricted to t such that $k < t < T - k$, which indeed yields $\lambda < 0$

[32] For instance, the units that were treated at the earliest possible time, i.e. with $T_i = k + 1$, are now treated in 100% of observations. Before further restricting the balanced sample, they were (by definition!) treated in 50% of observations. Conversely, the units that were treated at the latest possible time, i.e. with $T_i = \bar{T} - k - 1$, appear as treated in $\frac{1}{\bar{T} - 2k}$% of observation, or 16.66% of observations in our simulation.