

On Event Studies and Distributed-Lags in Two-Way Fixed Effects Models: Identification, Equivalence, and Generalization





On Event Studies and Distributed-Lags in Two-Way Fixed Effects Models: Identification, Equivalence, and Generalization*

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March 2020

Abstract

We discuss important properties and pitfalls of panel-data event study designs. We derive three main results. First, binning of effect window endpoints is a practical necessity and key for identification of dynamic treatment effects. Second, event study designs with binned endpoints and distributed-lag models are numerically identical leading to the same parameter estimates after correct reparametrization. Third, classic dummy variable event study designs can be generalized to models that account for multiple events of different sign and intensity of the treatment, which are particularly interesting for research in labor economics and public finance. We show the practical relevance of our methodological points in a replication study.

Keywords: event study, distributed-lag, applied microeconomics, credibility revolution **JEL codes:** C23, C51, H00, J08

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1 Introduction

The credibility revolution in empirical economics has led researchers to set up more transparent (quasi-)experimental research designs. This shift has increased the policy relevance and the scientific impact of empirical work (Angrist and Pischke, 2010). An important element that enhanced transparency is the visualization of treatment effects and/or identifying assumptions. Differences-in-differences (DD) models are particularly popular in this respect since they are directly connected to the rationale of experiments and the underlying identifying assumptions are intuitive.

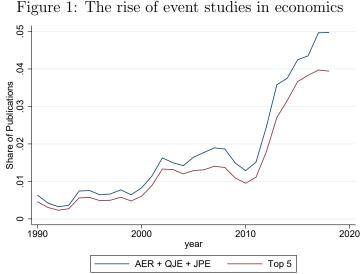
The event study (ES) design is the poster child of empirical methods in the DD family since (i) empirical estimates can be plotted, (ii) graphs are very intuitive and immediately show both dynamic post-treatment effects and the identifying assumption of "no pre-event trends", and (iii) the underlying econometrics are straightforward. The empirical specification usually boils down to a simple two-way fixed effects panel data model where the regressors of interest are a set of non-parametric event indicators which are defined relative to the event. Originating from the finance literature¹, event study designs are now widely used in applied economics, mostly public and labor economics, where an event is usually defined as a policy change whose effects are investigated. Figure 1 plots the use of event study designs in economics over time. We proxy the use by the share of studies mentioning the term "event study" in the Top Five economics journals.² While we see a steady increase since 1990, there is sharp increase since 2010. Moreover this increase is mostly driven by the three journals focusing on applied microeconomic work among the Top-Five, i.e. the American Economic Review (AER), the Quarterly Journal of Economics (QJE), and the Journal of Political Economy (JPE).

However, the intuitive appeal of event studies and its alleged simplicity entail a risk as it apparently leads researchers to refer to and model event study designs rather loosely. In more than one third of the event study papers published in the AER, QJE or JPE since 2010, no regression equation is specified. The purpose of this paper is to clarify the understanding of event study designs both in methodological and practical terms. We show below that the inherent imprecision in specifying event study designs is dangerous since important and nontrivial modeling decisions have to be made when specifying an event study model. These modeling choices have different and important implications for the underlying identifying assumptions of the model and eventually for treatment effect estimates.

We make three main methodological points that are important for applied researchers

 $^{^{1}}$ Dating back to Dolley (1933), see also MacKinlay (1997) for a survey on the financial literature.

 $^{^{2}}$ More than 80% of the studies mentioning event study designs actually implement one.



Notes: This graph plots the three-year moving average of the share of studies mentioning event study designs in top economics journals. We use a 3-year moving average to control for mean reversion. The Top 5 journals are the American Economic Review (AER), the Quarterly Journal of Economics (QJE), the Journal of Political Economy (JPE), Econometrica and the Review of Economic Studies. We report results for AER, QJE and JPE separately as these there journals are known to publish many applied microeconometric studies.

when setting up event studies. We derive these three points formally, but discuss practical implications and pitfalls along the way, making use of simple numerical examples to visualize our claims.

First, researchers need to define the window for which the dynamic effects are studied. We call this the *effect window*. While this choice is a practical necessity due to limited data availability, it is far from being innocuous. Limiting the number of leads and lags to a finite number requires the researcher to make assumptions about what happens beyond the endpoints of the effect window. The last lag (lead) is often defined as an open interval capturing all known events that (will) have happened in the past (future). We refer to this practice as *binning*. We show that binning allows identifying the model econometrically by separating the dynamic effects from the secular time trends even in the absence of nevertreated units. Intuitively, binning assigns the unit-year observations outside of the effect window to the control group which pins down the secular time trends.

The literature has so far put little emphasis on the treatment of endpoints of the effect window. Many studies have neglected the issue or discussed it not formally. Among the studies published in the AER, QJE or JPE since 2010 that used event studies and specified an empirical model, only 15% provide information on what has been done at the ends of the effect window. Among those 15%, researcher oftentimes discuss the treatment not formally, but only verbally, which aggravates reproducibility of results and might lead to mistakes when implementing the specific model in a different context. Moreover, different ways of treating endpoints of the effect window come along with different implicit parameter restrictions. These restrictions are important as they determine whether a model is identified econometrically and they affect the assignment of specific unit-year observations to treatment and control group. Hence, they directly affect the treatment effect estimates. Our first contribution is to highlight the importance of treating the endpoints of effect windows carefully and transparently. We make the case that binning of endpoints is a convincing practice in many institutional environments.

Second, we show that event study designs with binned endpoints and distributed-lag (DL) models are equivalent. To be precise, the DL model is just a reparametrization of the ES model with binned endpoints; event study estimates can be recovered from DL estimates by properly normalizing the DL model and cumulating the post-treatment and pre-treatment effects away from zero. This isomorphism provides an alternative, more transparent and intuitive way to understand the role of parameter restrictions for identification. The isomorphism also offers the distributed-lag model as an alternative implementation in statistical software which is less error-prone.

Third, we show that the simple event study can be generalized to account for multiple events and/or events of different signs and intensities of the treatment. Such institutional settings are common in public and labor economics. Consider, for instance, a sequence of state-level minimum wage or tax reforms of different sign and sizes. In the past, DL models were commonly recommended in such settings. We show that the equivalence result between DL and ES models also holds in the general case and that correct binning is of particular importance in such settings. Moreover, we discuss important modeling assumptions that are necessary when applying event studies in environments with multiple, heterogeneous events.

In the final part of the paper, we demonstrate the practical relevance of our three contributions, replicating and expanding the study by Baker and Fradkin (2017), henceforth BF2017. In the original paper, the authors suggest a neat way to measure worker search intensity based on Google search data. BF2017 apply their new measure to test whether search intensity responds negatively to increases in the potential benefits duration (PBD) induced by state-level reforms following the Great Recession. While their difference-in-difference estimates clearly show the expected negative relationship, the original event study results are inconclusive. We show that implementing the generalized event study design yields statistically highly significant dynamic effects, which are well in line with the difference-in-difference estimates. Hence, implementing our preferred specification of an event study design strengthens the credibility of the novel measure of search effort suggested by Baker and Fradkin (2017) and provides even stronger support for their key empirical finding that PBD has a negative effect on search effort.

Our paper is related to, but different from the recent methodological contributions focusing on event study designs. Current research can be loosely grouped into two strands. The first one deals with violations of the common trend assumption. Freyaldenhoven et al. (2019) shows how to extend the standard event study design to account for unobserved confounders generating a pre-treatment trend in the outcomes and still recover the causal effect of the event. Roth (2019) shows that treatment effects can be biased conditional on passing the flat pre-trend test. Malani and Reif (2015) point to the fact that non-flat pre-trends might also be due to anticipation rather than unobserved confounders. The second strand of the current methodological event study literature shows that standard event study specification do not produce average treatment effects if treatment effects are heterogeneous across cohorts (Abraham and Sun, 2018). This point is closely related to a current discussion on how to correctly estimate average treatment effects in difference-in-difference models when the treatment effects are heterogeneous (see Athey and Imbens, 2018; Borusyak and Jaravel, 2017; Callaway and Sant'Anna, 2018; de Chaisemartin and D'Haultfoeuille, 2019; Goodman-Bacon, 2018a; Gibbons et al., 2019). Importantly, and as discussed in more detail in Section 2.2, our paper abstracts from these two current debates. Throughout the paper, we assume that the common trend assumptions holds and that treatment effects are homogeneous across cohorts (and groups). The first methodological contribution of our study is more basic as we highlight practical pitfalls and potentially implausible implicit assumptions when setting up an event study model under ideal conditions. Secondly, we put forward a sensible specification that has been used in the form of distributed-lag models for a long time and, thirdly, we show that the proposed specification is generalizable to institutional set-ups with multiple event of various intensities.

The remainder of this paper is structured as follows. Section 2 introduces a standard version of an event study design in the simplest institutional environment and discusses how limiting the effect window and binning of endpoints imposes important parameter restrictions. In Section 3, we show that the event study model is equivalent to a standard distributed-lag model. In Section 4, we generalize the institutional environment and allow for multiple and heterogeneous events across and within units. We show that event study designs can also be used in such settings and discuss the additional adjustments and assumptions that need to be made in these cases. In Section 5, we demonstrate the relevance of our methodological points. We replicate and extend the study by Baker and Fradkin (2017) and further strengthen their results. Section 6 concludes.

2 Standard Event Study Design

In this section, we set up an event study model in the simplest institutional environment. We refer to this model as the standard event study set-up throughout the paper. We use the standard case to highlight the importance of introducing parameter restrictions to identify the model. In Subsection 2.1, we demonstrate that restricting the effect window is a practical necessity in applied work and advocate to bin the endpoints of effect windows. In Subsection 2.2, we show that binning of endpoints helps to overcome important underidentification problems recognized in the literature.

2.1 Restricting the Effect Window

We start our analysis with a standard event study set-up, where each unit i = 1, ..., N receives at most one single treatment at unit-specific time e_i . All units may receive treatment at the same time or treatment may be staggered over time with different units receiving treatment at different points in time. The treatment effect may unfold dynamically over time, but treatment effects are assumed to be homogeneous across cohorts unlike in Abraham and Sun (2018). We seek to estimate the dynamic effects of this treatment on our dependent variable y_{it} , which we observe in a balanced panel at different time periods $t = \underline{t}, ..., \overline{t}$. We call $[\underline{t}, ..., \overline{t}]$ the observation window for the dependent variable. Importantly, we do not consider cases in which we are faced with data gaps in our dependent or treatment variable.

In this set-up, the standard event study specification is given for all $t = \underline{t}, ..., \overline{t}$ by:

$$y_{it} = \sum_{j=-\infty}^{\infty} \beta_j d_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}$$
(1)

where $d_{it} = \mathbb{1}[e_i = t]$ is an event indicator that takes the value 1 in the year of the treatment, e_i , and zero otherwise. Unit fixed effects are denoted by μ_i and time fixed effects by θ_t . The parameter β_j is the dynamic treatment effect j time periods after $(j \ge 0)$ or before (j < 0)the event. All results derived in this paper also hold when the models include additional exogenous control variables X_{it} .

Remark 1 (Normalization).

The parameters β_j are only identified up to a constant due to the individual fixed effect μ_i . Treatment effects β_j are therefore typically expressed relative to some reference period, for example one period prior to the event. The corresponding coefficient is normalized to zero, e.g. $\beta_{-1} = 0$. In practice, the normalization is implemented by dropping the event indicator for the reference period, typically $d_{i,t-1}$. The intuition behind Remark 1 is straightforward: adding a constant to β_j for all $j = -\infty, ..., \infty$ and subtracting the same constant from the unit fixed effect μ_i for units with an event between $-\infty$ and ∞ does not alter equation (1).

In practice, researchers have to impose restrictions on the effect window to implement the event study design since β_j can never be estimated from the infinite past to the infinite future. Commonly, the effect window is restricted to a finite number of leads and lags. This restriction goes along with an important, but often unstated assumption about the dynamic treatment effects, which is summarized in the following Remark 2.

Remark 2 (Restricted effect window).

Restricting the effect window to a finite number of leads, \underline{j} , and/or lags, \overline{j} , requires assumptions about the nature of the effect outside of the window. It is often economically plausible to assume that treatment effects stay constant before \underline{j} and/or after \overline{j} , i.e. $\beta_j = \beta_{\overline{j}}$ for all $j > \overline{j}$ and $\beta_j = \beta_{\underline{j}}$ for all $j < \underline{j}$. These assumptions should be explicitly stated and defended.

Importantly, restricting the effect window also affects identification of treatment effects β_j as discussed in Section 2.2 and Remark 4.

Applying Remark 2, we rewrite equation (1) as

$$y_{it} = \beta_{\underline{j}} \sum_{j=-\infty}^{\underline{j}} d_{i,t-j} + \sum_{j=\underline{j}+1}^{\overline{j}-1} \beta_j d_{i,t-j} + \beta_{\overline{j}} \sum_{j=\overline{j}}^{\infty} d_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}$$

which simplifies to our preferred standard event study specification:

$$y_{it} = \sum_{j=\underline{j}}^{\overline{j}} \beta_j b_{it}^j + \mu_i + \theta_t + \varepsilon_{it}$$
⁽²⁾

with

$$b_{it}^{j} = \begin{cases} \sum_{s=-\infty}^{j} d_{i,t-s} & \text{if } j = \underline{j} \\ d_{i,t-j} & \text{if } \underline{j} < j < \overline{j} \\ \sum_{s=\overline{j}}^{\infty} d_{i,t-s} & \text{if } j = \overline{j}. \end{cases}$$
(3)

We refer to coefficients b_{it}^j as binned event indicators, as the indicators at the endpoints, i.e. the maximum lag (lead) take into account all observable past (future) events going beyond the effect window. The definition of endpoints in equation (3) is for example used in Smith et al. (2017) and Fuest et al. (2018). Endpoints can be equivalently defined as $b_{it}^j = \sum_{s=t-j}^{\infty} d_{is}$ and $b_{it}^{\bar{j}} = \sum_{s=-\infty}^{t-\bar{j}} d_{is}$. Another commonly used, equivalent definition is given by

$$b_{it}^{j} = \begin{cases} \mathbb{1}[t \le e_{i} + j] & \text{if } j = \underline{j} \\ \mathbb{1}[t = e_{i} + j] & \text{if } \underline{j} < j < \overline{j} \\ \mathbb{1}[t \ge e_{i} + j] & \text{if } j = \overline{j} \end{cases}$$
(4)

By Remark 1, we drop the indicator for the period before the event, b_{it}^{-1} and normalize β_{-1} to zero.

2.1.1 Special cases of effect window restrictions.

As stated in Remark 2, it is possible to only restrict the effect window pre or post treatment. For example, one could only restrict the effects prior to the event and allow the treatment effect to continue indefinitely, i.e. $\bar{j} \to \infty$. In this case, and for a balanced panel in real time, the length of the effect window is determined by the units that were treated first. \bar{j} is equal to maximum number of periods between the observed outcome and an event. Clearly, not all lags will be identified by all units in such a case, which might be problematic in terms of statistical power and economic interpretation.

An extreme form of restricting the effect window is to assume that there are no effects prior to the event and the effect is constant at and after the event. Hence $y_{it} = \beta b_{it} + \mu_i + \theta_t + \varepsilon_{it}$ where b_{it} is a dummy variable which takes the value 1 at and after the event. This is a standard difference-in-differences model with staggered treatment, which may be written as $y_{it} = \beta Treat_i \cdot Post_{it} + \mu_i + \theta_t + \varepsilon_{it}$, where $Treat_i$ is a dummy variable indicating whether unit *i* was treated at some point, and $Post_{it}$ is a dummy variable indicating whether unit *i* was treated in or before period *t*. Then, β is the average treatment effect relative to the pre-treatment period under the assumption of homogeneous treatment effects across cohorts and groups.

Another type of restriction, which is sometimes seen in the literature, is to restrict the effect window but without binning of endpoints. Such a model implicitly assumes that treatment effects drop to zero outside of the effect window – an assumption which is typically hard to defend (cf. the replication exercise in Section 5).

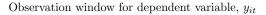
2.1.2 Data Requirements

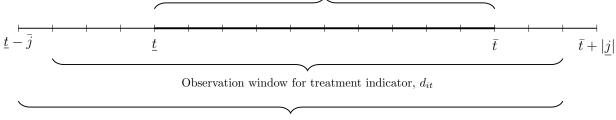
Due to the leads and lags of the effect window, we need to observe the treatment variable for a longer observation window than the dependent variable. Restrictions on the effect window determine the requirements on data availability for dependent variable y_{it} and treatment indicator d_{it} . In many applications, the treatment indicator d_{it} is generated from some variable that reports the status of the treatment, for example the level of tax rates or of minimum wages. We call such a policy variable the *treatment status* x_{it} . Hence $d_{it} = x_{it} - x_{i,t-1}$.

In the following remark, we summarize the data requirements for a given observation window of the dependent variable.

Remark 3 (Data requirements).

For a given balanced panel of the dependent variable from $[\underline{t}, \overline{t}]$ and a limited effect window $[\underline{j}, \overline{j}]$, we need to observe events from $\underline{t} - \overline{j} + 1$ to $\overline{t} + |\underline{j}| - 1$. If events are derived from changes in policy variables we need to observe treatment status from $\underline{t} - \overline{j}$ to $\overline{t} + |\underline{j}| - 1$. The following figure visualizes the required width of the observation window for a given limited effect window.





Observation window for treatment status, x_{it}

To understand the intuition behind Remark 3, it is first important to note that an event that happens before \underline{t} , i.e. the first data year of the dependent variable, can affect the outcome like any other event happening between \underline{t} and \overline{t} and needs to be taken into account. Likewise, we should account for events that happen after \overline{t} if we want to test for pre-trends. This rationale prompts the second question of how long before \underline{t} and after \overline{t} we need to observe events. By Remark 2, treatment effects are constant after \overline{j} and before \underline{j} periods. Hence, we need to observe events at least from $\underline{t} - \overline{j}$ to $\overline{t} + |\underline{j}|$. Remark 3 states that it suffices to observe events in one fewer year at each end of the data window, hence from $\underline{t} - \overline{j} + 1$ to $\overline{t} + |\underline{j}| - 1$. To see this, consider a case where the event takes place at $\underline{t} - \overline{j}$. Due to the binning, the treatment indicator $b_{it}^{\overline{j}}$ will be equal to one for this unit for all t. Conditional on unit fixed effects, this unit cannot be used to identify treatment effects. An analogous argument applies at the other end of the data window. If we generate event dummies from changes in policy states, the observation window for the treatment status needs to be observed for an additional period in the beginning, hence from $\underline{t} - \overline{j}$ onwards.

The top left panel of the numerical example shown in Section A.1 shows the construction of the binned treatment indicator in practice. The matrix immediately demonstrates the need

for normalization (cf. Remark 1) as all row sums in the left matrix are equal to one. Binning also implies that if the reform had happened on or before $t = t - \bar{j} = 2000 - 4 = 1996$ (rather than in t = 2005 as assumed in the example), $b_{it}^j = b_{it}^4 = 1$ for all t from 2000 to 2010. In this case of an always-treated unit, $b_{it}^{j} = b_{it}^{4}$ is a constant and its effect is absorbed in the unit fixed effect μ_i . By the analogous argument, events on or after $t = \bar{t} + |\bar{j}| = 2010 + 3 = 2013$ imply $b_{it}^{j} = b_{it}^{-3} = 1$ for all t from 2000 to 2010 whose effect is absorbed in μ_i . It therefore suffices to know all events from time period $\underline{t} - \overline{j} + 1 = 1997$ to $t = \overline{t} + |\overline{j}| - 1 = 2012$ to estimate the model, see Remark 3.

2.2Identification

It is important to assure that the model is econometrically identified such that the dynamic effects β_j are distinguished from secular time fixed effects θ_t . Throughout the paper, we understand identification as the purely mechanical recovery of the coefficients of interest β and not their causal interpretation. Borusyak and Jaravel (2017) nicely show that with an infinite effect window, $[j, \bar{j}] = [-\infty, \infty]$, the dynamic effects are only identified up to a linear trend. The argument can be easily replicated using our notation in equation (1): $y_{it} = \sum_{j=-\infty}^{\infty} \beta_j d_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it} = \sum_{j=-\infty}^{\infty} (\beta_j + \lambda \cdot j) d_{i,t-j} + (\theta_t - \lambda \cdot t) + \tilde{\mu}_i + \varepsilon_{it}$

where $\tilde{\mu}_i = \mu_i + \lambda \cdot e_i$.³ Hence, adding a linear trend $\lambda \cdot j$ to the dynamic treatment effects and adding the opposite linear trend $-\lambda \cdot t$ to the secular time fixed effects θ_t does not affect the estimates of the model. The underidentification arises because all units are treated at some point and $\sum_{j=-\infty}^{\infty} j d_{i,t-j} = \sum_{j=-\infty}^{\infty} j \mathbb{1}[t = e_i + j] = t - e_i$ for all units *i* and all time periods t.⁴ The presence of never-treated units would clearly solve the identification problem. However, *observing* a unit not be treated does not imply that this unit is never-treated.⁵

In practice, this underidentification can easily be overlooked as many statistical packages automatically drop regressors in the case of multicollinearity. A non-identified linear trends leads to dropping either one event dummy or one time dummy.

In the following section, we discuss how to deal with this underidentification problem in the absence of never-treated units. First, in Subsection 2.2.1, we show that restricting the effect window by Remark 2 gives rise to a natural solution. Second, in Subsection 2.2.2, we

³ Because $\sum_{j=-\infty}^{\infty} (\lambda \cdot j \cdot d_{i,t-j} - \lambda \cdot t + \lambda \cdot e_i) = \lambda \sum_{j=-\infty}^{\infty} (j \cdot \mathbb{1}[j = t - e_i] - t + e_i) = 0.$ ⁴ Assuming that all units are potentially treatable, each unit will be treated at some point with a infinite time horizon, i.e. $t = e_i + j$ for some t.

⁵ A unit could have been treated before or after the observed sample period or even in the yet unrealized future. When treatment effects are allowed to have effects into the infinite future (or affect the infinite past), no unit is known to be never-treated with certainty. If a unit cannot possibly be treated, it is unlikely to serve as a valid control group for causal inference.

discuss identification when the sample is also restricted in event time.

2.2.1 Restricted Effect Window

As introduced above, we are faced with a panel, in which units *i* receive a single treatment at unit-specific event time e_i . We argue that restricting the effect window as proposed in Remark 2 introduces restrictions that allow separately identifying dynamic effects, β_j , and secular time trends, θ_t .

Remark 4 (Identification and restricted effect window).

Unit-year observations outside of the effect window serve as control group observations. The length of the effect window hence directly affects identification and helps to separately identify dynamic treatment and secular time fixed effects.

To see how restricting the effect window can help identification, let us formally reconsider the underidentification results by Borusyak and Jaravel (2017). Restricting the effect window as in Remark 2 leads to our equation (2), $y_{it} = \sum_{j=j}^{\bar{j}} \beta_j b_{it}^j + \mu_i + \theta_t + \varepsilon_{it}$. Here, $\sum_{j=-\infty}^{\infty} j d_{i,t-j} =$ $\sum_{j=-\infty}^{\infty} j \mathbb{1}[t = e_i + j] = 0 \neq t - e_i$ for time periods t outside the effect window, i.e. if $t < e_i - |\underline{j}| + 1$ or $t > e_i + \overline{j}$. Adding a linear trend $\lambda \cdot j$ to the restricted number of dynamic treatment effects is therefore not offset by adding a linear trend to the secular time trend for the observations outside of the effect window. These observations serve as a control group and help to pin down secular time trends. Restricting the effect window may even produce units *i* for which all treatments happen outside of the observation window for the treatment variable (see Remark 3). Such units are never-treated in the *observed* sample and hence also serve as a control group. Hence, binning of endpoints allows for identification with or without the presence of never-treated units.

In Appendix B, we present intuitive and highly stylized examples demonstrating how identification is achieved technically. To summarize, the model is econometrically identified if two conditions are fulfilled: (i) for each lag/lead j, there is at least one unit i with an observation j periods after/before the event; (ii) for at least one endpoint (\underline{j} or \overline{j}) observed for some unit i in some period t, there is at least one other unit $\ell \neq i$, which is outside of its effect window in the same period t. Condition (ii) is automatically satisfied in the presence of at least one never-treated unit. Condition (i) identifies all other effects either from a direct comparison with a control group or from an iterative comparison of effects. The identified endpoint allows backing out all other treatment effects and all time fixed effects iteratively – akin to the econometric identification in staggered treatment difference-in-differences designs.

2.2.2 Restricted Sample in Event Time

A common alternative to binning the event dummies $d_{i,t-j}$ at the endpoints \underline{j} and \overline{j} , is to restrict the sample to observations of the dependent variable within the effect window. Hence, for each unit i, only observations in periods t with $e_i - |\underline{j}| \leq t \leq e_i + \overline{j}$ are used. In such a sample, $b_{it}^j = d_{i,t-j}$ for all j including the endpoints \underline{j} and \overline{j} . In our numerical example in Appendix Section A.1, the sample would only span the years 2002 to 2009. Hence, binning would have no effect on the estimates. However, in such a restricted sample, dynamic treatment effects are only identified up to a linear trend because $y_{it} = \sum_{j=\underline{j}}^{\underline{j}} \beta_j d_{i,t-j} + \mu_i +$ $\theta_t + \varepsilon_{it} = \sum_{\underline{j}=\underline{j}}^{\underline{j}} (\beta_j + \lambda \cdot \underline{j}) d_{i,t-j} + (\theta_t - \lambda \cdot t) + \tilde{\mu}_i + \varepsilon_{it}$ where $\tilde{\mu}_i = \mu_i + \lambda \cdot e_i$. The identity holds because $\sum_{\underline{j}=\underline{j}}^{\underline{j}} j d_{i,t-j} = \sum_{\underline{j}=\underline{j}}^{\underline{j}} j \mathbbm{1}[t = e_i + \underline{j}] = t - e_i$ for all i and all t which are here in the effect window, i.e. $t - |\underline{j}| \leq t \leq t + \overline{j}$.

There are three possibilities to overcome this inherent underidentification issue in a sample restricted in event time. First, never-treated units could be included. Second, the effect window could be restricted to be shorter than the observation window. Then identification is again achieved as described in Remarks 2 and 4. The third option is to sacrifice unit fixed effects.⁶ Clearly, this comes at the price of not controlling for unobserved time-constant individual factors otherwise absorbed in μ_i . Hence, this practice is akin to estimating a single-difference specification: $y_{it} = \beta Treat_i \cdot Post_{it} + \theta_t + \varepsilon_{it}$.

3 Event Studies and Distributed-lag Models

In this section, we show that event study (ES) models with binned endpoints and distributedlag (DL) models yield identical parameter estimates. In Subsection 3.1, we formally demonstrate under which restrictions ES and DL models yield equivalent dynamic treatment effects. In Subsection 3.2, we discuss the practical implications of this isomorphism and argue that DL models are easier to implement and less-error prone. In Appendix Section A.1, we illustrate all formal claims using a simple numerical example.

3.1 Equivalence

We start by showing the equivalence of event study and distributed-lag models in the general case without binning of endpoints. Taking first differences of the standard event study specifi-

⁶ In the absence of unit fixed effects μ_i , adding a linear trend to both dynamic effects and secular time trend changes the constant as as $y_{it} = \alpha + \sum_{j=\underline{j}}^{\underline{j}} \beta_j d_{i,t-j} + \theta_t + \varepsilon_{it} = (\alpha - \lambda \cdot e_i) + \sum_{j=\underline{j}}^{\underline{j}} (\beta_j + \lambda \cdot j) d_{i,t-j} + (\theta_t - \lambda \cdot t) + \varepsilon_{it}$. Dynamic effects and secular trends are therefore separately identified at the price of not controlling for unobserved time-constant individual factors.

cation given in equation (1), we can rewrite the event study specification into a distributed-lag model:

$$\Delta y_{it} = y_{it} - y_{i,t-1}$$

$$= \sum_{j=-\infty}^{\infty} \beta_j d_{i,t-j} - \sum_{j=-\infty}^{\infty} \beta_j d_{i,t-1-j} + \phi_t + \Delta \varepsilon_{it}$$

$$= \sum_{j=-\infty}^{\infty} \beta_j d_{i,t-j} - \sum_{j=-\infty}^{\infty} \beta_{j-1} d_{i,t-j} + \phi_t + \Delta \varepsilon_{it}$$

$$= \sum_{j=-\infty}^{\infty} \gamma_j d_{i,t-j} + \phi_t + \Delta \varepsilon_{it}$$

$$= \sum_{j=-\infty}^{\infty} \gamma_j \Delta x_{i,t-j} + \phi_t + \Delta \varepsilon_{it}$$
(5)

where $\gamma_j = \beta_j - \beta_{j-1}$ and $\phi_t = \theta_t - \theta_{t-1}$ are time fixed effects and the event indicator $d_{i,t-j}$ is the first difference $\Delta x_{i,t} = x_{it} - x_{i,t-1}$ of the treatment status x_{it} . In the standard case with a single binary treatment, the treatment status x_{it} is a dummy variable with an arbitrary constant as initial value, for example zero, that increases by 1 if an event occurred in period t. Parameters γ_j are the incremental changes of the treatment effects β_j , measuring the slope of treatment effects from one time period to the next. The distributed-lag specification in equation (5) is the first difference of the following distributed-lag specification in levels

$$y_{it} = \sum_{j=-\infty}^{\infty} \gamma_j x_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}$$
(6)

where μ_i denotes unit fixed effects. Note that the distributed-lag specification is either a regression of levels on levels (eq. 6) or of changes on changes (eq. 5) while the event-study specification is a regression of levels on (binned) changes (eq. 2).

We proved the equivalence between event study and distributed-lag models in the general case without restricting the effect window. Next, we show that the equivalence between ES and DL models for restricted effect windows holds *only* if endpoint are binned as in Remark 2. The distributed-lag parameters γ_j are related to the event study parameters β_j by $\gamma_j = \beta_j - \beta_{j-1}$. Binning the upper endpoint, $\beta_j = \beta_{\bar{j}}$ for all $j > \bar{j}$, is therefore equivalent to assuming that $\gamma_j = 0$ for all $j > \bar{j}$; for the lower endpoint $\beta_j = \beta_{\bar{j}}$ for all $j < \underline{j}$ is equivalent to $\gamma_j = 0$ for all $j \leq \underline{j}$. The event study model with restricted effect window between \underline{j} and \overline{j} and binned endpoints

$$y_{it} = \sum_{j=\underline{j}}^{\overline{j}} \beta_j b_{it}^j + \mu_i + \theta_t + \varepsilon_{it}$$
(7)

is therefore equivalent to a distributed-lag specification with \overline{j} lags and |j| - 1 leads

$$y_{it} = \sum_{j=\underline{j}+1}^{\overline{j}} \gamma_j x_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}$$
(8)

Without binning, ES and DL specifications are based on different parameter restrictions and yield different parameter estimates. We summarize this result in the following remark:

Remark 5 (Equivalence of Event Study and Distributed-Lag Model).

The event study specification with an effect window limited to \overline{j} periods after and $|\underline{j}|$ periods before the event given by equation (2) is equivalent to a distributed lag models with \overline{j} lags and $|\underline{j}| - 1$ leads as given by equation (8) if the endpoints of the effect window are binned according to equation (3).

It is important to note that distributed-lag coefficients measure treatment effect changes, such that one fewer lead has to be estimated: we include leads and lags running from γ_j from $\underline{j} + 1$ (not \underline{j} as in the event study design) to \overline{j} . Then event study parameters β_j can be calculated from the distributed-lag parameters γ_j by using the difference equation $\beta_j = \beta_{j-1} + \gamma_j$. The starting point for this difference equation is given by the normalization in Remark 1. Normalizing to one period prior to the effect, i.e. $\beta_{-1} = 0$, treatment effects β_j can be uniquely recovered as

$$\beta_{j} = \begin{cases} -\sum_{k=j+1}^{-1} \gamma_{k} & \text{if } j \leq -2 \\ 0 & \text{if } j = -1 \\ \sum_{k=0}^{j} \gamma_{k} & \text{if } j \geq 0. \end{cases}$$
(9)

We summarize this result in the following remark:

Remark 6 (Recovery of treatment effect from the distributed-lag model).

Dynamic event study treatment effects β_j are recovered from distributed-lag parameters γ_j as cumulative sums starting from a reference period, typically the period prior to the effect, according to equation (9).

As in the event study model, we need a normalization in the distributed-lag model since parameters β_j are only identified up to a constant due to the individual fixed effect μ_i (cf. Remark 1). Equation (9) shows how to recover the dynamic treatment effects β_j as the cumulative sums of distributed-lag parameters γ_j . Concretely, for post-treatment effects j > -1, we intuitively cumulate upwards: $\beta_j = \beta_{j-1} + \gamma_j$ with $\beta_{-1} = 0$. Importantly, for pre-treatment effect $j \leq -1$, we cumulate *downwards* with a *negative* sign: $\beta_j = \beta_{j-1} - \gamma_{j-1}$ with $\beta_{-1} = 0$. For instance, $\beta_{-2} = -\gamma_{-1}$; we *must not* assume $\gamma_{-1} = 0.7$

3.2 Practical Implications

The equivalence formally defined in Remark 5 has various implications when applying event study or distributed-lag models in practice. We briefly discuss the most important ones in the following subsection.

Model choice. The estimates of the event study parameters and corresponding cumulative distributed-lag parameters are numerically equivalent. The choice of the model is therefore purely a question of convenience, yet there are some practical (dis-)advantages for both models to be discussed below.

Binning vs. cumulating. In the event study design, treatment variables have to be binned at the endpoints of the effect window according to equation (3). Consequently, the event study model delivers direct estimates of the dynamic treatment effects and therefore readily interpretable parameters. In contrast, the coefficients from the distributed-lag model $\gamma = [\gamma_{\underline{j}+1}, ..., \gamma_{\overline{j}}]'$ have to be cumulated following equation (9) to obtain the event study parameters $\beta = [\beta_{\underline{j}}, ..., \beta_{-2}, \beta_0, ...\beta_{\overline{j}}]'$. This linear transformation transfers the statistical properties (consistency and asymptotic normality) of $\widehat{\gamma}$ to the calculated $\widehat{\beta}$. Standard errors of $\widehat{\beta}_j$ can be calculated from the variances and covariances of the vector $\widehat{\gamma}$ by the usual formula for linear combinations and are identical to the direct event study estimates.

Misspecification due to missing data. While the data requirements (cf. Remark 3) for the distributed-lag model are by Remark 5 identical to the ones in the event study model, they are more transparent in the DL specification. The distributed-lag model with restricted effect window given in equation (8) reveals immediately that treatment status x_{it} needs to be observed from period $(\underline{t} - \overline{j})$ to $(\overline{t} + |\underline{j}| - 1)$ and hence treatment dummy d_{it} from $(\underline{t} - \overline{j} + 1)$ to $(\overline{t} + |\underline{j}| - 1)$. In the distributed-lag model, a shorter observation window for the treatment variable will lead to missing values in leads and/or lag, which automatically reduces the estimation sample. In case the data contains event indicators $d_{i,t}$, the treatment status x_{it} has to be generated according to: $x_{it} = x_{i,t-1} + \Delta x_{i,t} = x_{i,t-1} + d_{i,t}$. The starting value for the treatment status $x_{i,\underline{t}-\overline{j}}$ will be absorbed by the individual fixed effects μ_i and can be set to an arbitrary number, typically zero. In contrast, in the event study model, a too

⁷ In our example 1 with effect window from $\underline{j} = -3$ to $\overline{j} = 4$, the coefficients are $\beta_{-3} = -(\gamma_{-1} + \gamma_{-2})$, $\beta_{-2} = -\gamma_{-1}, \beta_{-1} = 0, \beta_0 = \gamma_0, \beta_1 = \gamma_0 + \gamma_1, \beta_2 = \gamma_0 + \gamma_1 + \gamma_2, \beta_3 = \gamma_0 + \gamma_1 + \gamma_2 + \gamma_3, \beta_{-3} = \gamma_0 + \gamma_1 + \gamma_2 + \gamma_3 + \gamma_4$.

short observation window is easily overlooked when binning of endpoints, which might lead to a bias if events happened in the time interval between $[(\underline{t} - \overline{j}), \underline{t}]$ and/or $[\overline{t}, (\overline{t} + |\underline{j}| - 1)]$ (cf. Remark 3). For this reason, we argue that implementing the distributed-lag model is less error-prone.

Fixed effect vs. first difference estimator. Both the event study model in equation (2) and the distributed-lag model in equation (8) are panel data models including unit and time effects. The parameters β or γ can be estimated either with standard fixed effects estimation in levels or in first differences. Both estimators are consistent and asymptotically normal under standard assumptions for panel data models. In finite samples, the estimates obtained with the fixed effect estimator differ from the ones obtained with the first difference estimator.

The deviation between fixed effects and first difference estimation is small if the dynamic nature of the effect is modeled correctly, i.e. if the effect is truly constant \overline{j} periods after the event. If, however, the true treatment effect continues to unfold beyond \overline{j} , fixed effects and first difference estimates can differ strongly. As an example, assume that the true treatment effect is negligible at and shortly after treatment and only materializes after several posttreatment periods. Further assume, that the researchers includes too few post-treatment parameters to capture the full treatment effect. In this case, the first difference estimator will be close to zero while the fixed effect estimator will pick up some average of the delayed response. Both estimator will clearly underestimate the true long-term response. In contrast, if the number of lags is specified such that the treatment effect has fully materialized within the effect window (cf. Remark 2), both the fixed effects and the first difference estimator will correctly pick up the dynamic effects and correctly estimate the long-run effect.

4 Generalized Event Study Design

In many applications, treatment may occur repeatedly and be of different intensities across units and/or time. In this section, we show that the standard event study design can be generalized to accommodate institutional set-ups where multiple events with known but varying treatment intensity take place. We formally derive the generalized event study in Subsection 4.1. Subsection 4.2 discusses four typical institutional environments in which the generalized event study can be applied. Appendix Section A.2 provides an empirical numerical example visualizing such a case.

4.1 Set-up and Equivalence in the Generalized Design

In the following, we set up a generalized event study design that can be used in case of multiple events of identical intensity, single events with varying treatment intensity, and multiple events of different intensities. The set-up also nests the standard event study design set up in Section 2 as a special case.

In the generalized design, the treatment variable is defined as the change in the treatment status $\Delta x_{it} = x_{it} - x_{i,t-1}$. In other word, the treatment variable measures the exact size of a change in a certain policy variable (e.g. a tax rate) from unit t - 1 to unit t. In contrast, the treatment variable in the standard design is a dummy indicating that any change in the policy variable happened.

It is easy to see that the equivalence between event study designs and distributed-lag models shown in Section 3.1 also holds in the general case. The standard event study design with infinite event windows shown in equation (1) becomes:

$$y_{it} = \sum_{j=-\infty}^{\infty} \beta_j \Delta x_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}$$
(10)

where $\Delta x_{it} = x_{it} - x_{i,t-1}$.

Taking first differences of equation (10) and rewriting yields the distributed-lag model

$$\Delta y_{it} = \sum_{j=-\infty}^{\infty} \gamma_j \Delta x_{i,t-j} + \phi_t + \Delta \varepsilon_{it}$$
(11)

where $\gamma_j = \beta_j - \beta_{j-1}$. The distributed-lag model in levels is given by

$$y_{it} = \sum_{j=-\infty}^{\infty} \gamma_j x_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}.$$
 (12)

The event study specification given in equation (10) is a regression of levels (y_{it}) on changes (Δx_{it}) which may look disturbing. However, it is derived from the equivalent distributed-lag model in levels which is a completely intuitive regression of levels (y_{it}) on levels (x_{it}) . The event study specification just takes care of the re-parametrization and directly delivers the cumulative effects β_i rather than the incremental effects γ_i .

When restricting the effect window to \overline{j} periods after and \underline{j} before the event, the generalized event study in levels is given by:

$$y_{it} = \sum_{j=\underline{j}}^{\overline{j}} \beta_j c_{it}^j + \mu_i + \theta_t + \varepsilon_{it}$$
(13)

where binned treatment variables c_{it}^{j} are easily generated analogously to the definition for binned treatment dummies in (3):

$$c_{it}^{j} = \begin{cases} \sum_{s=-\infty}^{\underline{j}} \Delta x_{i,t-s} & \text{if } \underline{j} = \underline{j} \\ \Delta x_{i,t-j} & \text{if } \underline{j} < j < \overline{j} \\ \sum_{s=\overline{j}}^{\infty} \Delta x_{i,t-s} & \text{if } \underline{j} = \overline{j}. \end{cases}$$
(14)

Note that the more common definition in (4) cannot be generalized, which is why we prefer the more versatile event indicator definition given in equations (3) or 14. The analogous distributed lag model is

$$y_{it} = \sum_{j=-\underline{j}+1}^{\overline{j}} \gamma_j \, x_{i,t-j} + \mu_i + \theta_t + \varepsilon_{it}.$$
(15)

Remark 1 on normalization, Remarks 2 and 4 on restricting the event window, Remark 3 on data requirements and the practical implications in Section 3.2 on estimating the event study vs. the distributed-lag models also hold in the general case.

Importantly, estimating dynamic treatment effects using the generalized event study only produces unbiased estimates under a linearity and additivity assumption, which is summarized in the following remark.

Remark 7 (Applicability of the Generalized Event Study Design).

Assuming that the treatment effect is proportional to the observed treatment intensity, the generalized event study described by equations (13) and (14) delivers unbiased estimates of the dynamic treatment effect. Treatment effects can also be estimated using a distributed-lag models as specified in equation (12).

As the generalized event study specification incorporates the intensity of treatment, estimated effects can be interpreted as the effect of a one-unit increase akin to the interpretation in a generalized differences-in-differences model. This way, event study estimates can be used to infer long-term effects on an intuitive scale.

4.2 Typical Cases and Applications

In this subsection, we discuss typical cases of the generalized event study design and provide selected examples from recently published applications.

Case 1: Single Events of Identical Intensity. This is the standard case discussed in Section 2.

Case 2: Multiple Events of Identical Intensity. Consider the case in which events of identical intensity take place repeatedly for a unit. Using definition (14), this implies that $\Delta x_{it} = d_{it}$ is an event dummy that takes value 1 in *any* period where an event took place and 0 in other periods (see Appendix C.1 for a numerical example). Few analyses have applied event studies in such an institutional context (see Dube et al., 2011, for an exception). However, many institutional set-ups, such as hospital admissions or firm switches, fit the model. Sometimes, only the first of potentially many events is considered in a standard event study framework as developed in Section 2. This approach leads to biased estimates unless the second and subsequent events are known to have no additional effect at all.

Case 3: Single Events of Varying Treatment Intensity. Next, consider the case where each unit receives one treatment, but treatment intensity s_i differs across units, hence $\Delta x_{it} = d_{it} \cdot s_i$ in definition (14). A numerical example is given in Appendix C.2. This case is quite frequently applied as it fits an institutional setting where a shock at some aggregate level hits units at a disaggregate level with different intensities (see, e.g., Alsan and Wanamaker, 2018; Charles et al., 2018; Clemens et al., 2018; Goodman-Bacon, 2018b). Many applications of this type formally refer to the standard event study model but discuss generalization and treatment of endpoints only verbally if at all.

Case 4: Multiple Events of Different Intensities and Direction. Last, we consider the most general case, developed in Section 4.1, in which events may occur multiple times per unit and their treatment intensity differs both across individuals and across events. A numerical example is given in Appendix Section A.2. There are many settings that fit this model, such as multiple tax changes or minimum wage hikes, and correspondingly many applications. Traditionally, the respective models were framed as distributed-lag models rather than event study designs (Suárez Serrato and Zidar, 2016; Drechsler et al., 2017; Fuest et al., 2018).

A special case is when events have a different direction. Assume that d_{it} is a variable that takes the value 1 in periods with a "positive" treatment, value -1 in periods with a "negative" treatment and value 0 in periods without a treatment. The parameter β_j estimates the average effect j periods after the event of all "positive" treatments and – with reversed sign – all "negative" treatments. In other words, the effects of "positive" and "negative" treatments are assumed symmetric with opposing signs. A typical example would be the introduction of a new law in some period and the abolition of the law in some later period, or the opening and closing of plants across regions. We are not aware of any recent application of this case.

4.3 Dichotomizing treatment variables.

A common alternative empirical specification used when treatment effects are of different sizes is to dichotomize treatment variables and use a dummy variable that is only switched on for large events (see, e.g., Simon, 2016; Fuest et al., 2018). However, the parameter estimates of such a dichotomization are harder to interpret both in magnitude and direction. To see this, consider the following case: each unit is treated once, there are two types of treatment: a small reform $d_{it}^s = 1$ or a large reform $d_{it}^\ell = 2$; treatments are distributed randomly in time and treatment effects are linear in the intensities of the reform. Ignoring small events and applying the standard event dummy set-up yields $d_{it}^s = 0$, $d_{it}^{\ell} = 1$. In this case, units with small reforms become part of the the control group although they respond to the reform. This induces a bias in the time fixed effects and thereby also in the treatment coefficients. Depending on the elasticity of the treatment effect with respect to the reform intensity, the share of large vs. small reforms and the size of the effect window relative to the observation window, it is possible that estimates in the model only using the large reforms can be larger, smaller or identical to the model using all reforms. A possible fix for this ambiguity is to exclude units with small events from the sample, in which case, the model is, however, estimated on a different and possibly selective sample. Moreover, the dichotomization of the treatment variable eliminates valuable information which could otherwise be used to identify the magnitude of the effect.

5 Replication

In this section, we demonstrate the relevance of the results derived in Sections 2 to 4 by replicating and extending the study by Baker and Fradkin (2017) (BF2017). We will particularly focus on the importance of restricting the effect window and on the power of the generalized event study design.

BF2017 makes an important contribution to the literature on search models and unemployment insurance (UI) by proposing a novel way to measure job search effort using Google Search data. Job search is a key parameter in theoretical search and matching models but it is notoriously difficult to quantify and measure precisely. The proposed Google Job Search Index (GJSI) is a convenient and broadly applicable way to operationalize job search in empirical studies. In the last part of the study, BF2017 apply their novel measure and test whether job search behavior responds to changes of potential benefit duration (PBD)). Theoretically, we would expect a negative effect of extended PBD on search behavior.

Empirically, the authors exploit variation in unemployment insurance generosity across

US states and time, and regress the Search Index on PBD in a state-month panel. They first estimates a simple differences-in-differences model (reported in Table 7 of their paper), in which they regress GJSI (in logs) on PBD (in weeks) controlling for state and time fixed effects, state-specific quadratic time trends, state-level total unemployment (second order polynomial) and the fraction of the population in the labor force. The results clearly indicate the expected negative effect of potential benefit duration on job search. In the preferred specification (4), they find a highly significant estimate of -0.00207, which implies that a ten week increase in UI benefits leads to 2.07 % drop in aggregate job search.

In a next step, the authors analyze the dynamics of the relationship by implementing an event study design. We recast their preferred event study model in our notation as:

$$\ln GJSI_{it} = \sum_{j=-3}^{4} \beta_j d_{i,t-j} + w'_{it}\xi + \mu_i + \theta_t + \varepsilon_{it}, \qquad (16)$$

where $GJSI_{st}$ is the the natural logarithm of the Google Job Search Index in state *i* and period *t* (year-month), $d_{i,t-j}$ is an indicator variable that indicates whether PBD in state *i* was changed $j \in [-3, 4]$ month before or after *t*. Parameter μ_i captures state fixed effects and θ_t denotes period fixed effects. The vector w_{it} captures state-year specific covariates. BF2017 control for the number of unemployment insurance claims in state *i* and period *t* (month-year) divided by state population.

Changes in PBD happen frequently and with different intensities across US states over time. The authors analyze PBD increases and decreases in separate regressions and for different time windows. Increases of PBD mainly occurred during the Great Recession up to 2011 while, decreases occurred thereafter. BF2017 consequently investigate the effects of PBD increases using data from January 2006 to December 2011 and the effects of PBD decreases using data from January 2012 to December 2015; we refer to the former as the "crisis sample" and the latter as the "recovery sample". For both increases and decreases, BF2017 only focus on large changes. For increases, d_{st} is equal to 1 if PBD in state *i* and period *t* (year-month) has increased by 13 weeks or more; for decreases, the dummy d_{it} is switched on for decreases of 7 weeks or more. In the respective models, the event indicator d_{it} is zero if (i) no change happened, (ii) a change of the same sign but with smaller absolute size occurred, or (iii) the state adjusted PBD in the respectively opposite direction. The results from these specifications are presented in specifications (3) and (5) of BF2017-Table 8 and BF2017-Figure 4.⁸

⁸ In columns (1), (2), (4) of Baker and Fradkin (2017)'s Table 8, the authors estimate different specifications, in which they focus on the largest single change observed within a state, exclude observations when other changes happen within this largest event's window and/or match control state-time-periods for the

In BF2017's sample, states experience up to five large increases in the crisis and seven large decreases in the recovery sample. In Panels A and B of Figure 2, we replicate the main event study results for large increases and large decreases on the two respective samples estimating equation (16). Our results are identical to the original version. Unlike the results from differences-in-differences model, the BF2017 event study estimates do not point to a strong negative relationship between search effort and PBD. However, the results depicted in Panel A of Figure 2 are based on strong implicit assumptions and parameter restrictions embodied in equation 16, which speak directly to our main points raised in the previous sections. While the empirical model looks like a classic event study design and therefore innocuous at first sight, event indicators d_{it} are not binned at the endpoints (cf Remark 2) and no coefficient is normalized to zero (cf Remark 1). This implies that treatment effects are implicitly normalized to be zero four and more periods before the event as well as five and more periods after the event, i.e. $\beta_j = 0$ for all $j \leq -4$ and for all $j \geq 5$. In particular, the assumption $\beta_5 = 0$ is very strong since it assumes that the effect builds up over 4 years and then immediately drops to zero (cf. Remark 4). In contrast, binning of endpoints assumes that the effect builds up over 4 years and stays constant thereafter which is an assumption more in line with the theoretical priors.

Next, we estimate equation (16) as an event study model with restrictions suggested in Sections 2.1 and 4. We bin endpoints according to Remark 2 and we normalized the pre-event coefficient $\beta_{-1} = 0$ according to Remark 1. As events can occur several times per state in our application, this leads to Case 2 "multiple events of identical intensity" in our Section 4.2. The β -coefficients can be estimated by creating binned treatment indicators at the endpoints j = -3 and j = 4 according to equation (3). Alternatively, γ -coefficients can be estimated in a distributed-lag model with 4 lags and 2 (not 3) leads and β -coefficients can be recovered according to equation (9). The two methods are equivalent and lead to identical parameter estimates and standard errors as shown in Remark 5. The choice of the estimation method is purely a question of convenience as explained in Section 3.2. Panel B of Figure 2 shows results with binned endpoints and normalized pre-event period. Different from the original results in Panel A, large increases of potential benefit duration (PBD) have a negative effect on job searches building up over 4 months and becoming statistically significant at the 5%level 3 and 4 months after the increase. The long-term effect is estimated as -0.036 (s.e. = (0.012), i.e. a fall in job searches by 3.6% for every large increase in potential benefit duration by 13 weeks or more. There are no significant effects prior to the large increase in PBD indicating that the parallel trends assumption is satisfied prior to the treatment. Hence,

respective largest changes without any PBD decrease. While we replicate the results in our programs posted online, we only focus on Baker and Fradkin (2017)'s preferred models here.

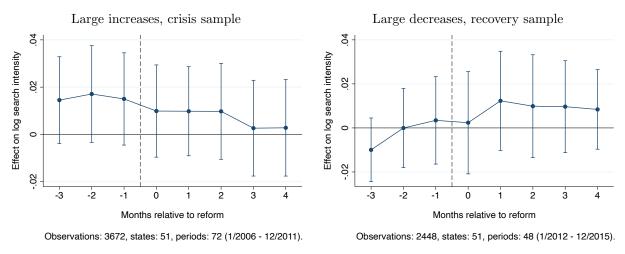
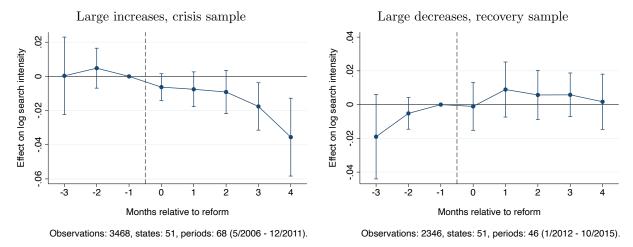


Figure 2: Baseline Results and the Role of Binning

Panel A: No binning and no normalization at -1 (Baker and Fradkin, 2017)

Panel B: Binning and normalization at -1 (own calculations)



Notes: The figure replicates and extends the main event study estimates reported in (Baker and Fradkin, 2017), BF2017. The graphs show point estimates and 95%-confidence intervals based on standard errors clustered by states. Graphs in Panel A replicate the estimates reported in specifications (3) and (5) of BF2017-Table 8 and plotted in the two panels of BF2017-Figure 4. The left graph in Panel A plots the dynamic effect of large increase (at least 13 weeks) in potential benefit duration (PBD) on log search intensity as measured with the newly proposed Google Job Search Index (GJSI). States that experiences no changes in a certain months or smaller changes, including negative ones are in the control group. The right graph in Panel A shows the analogous results for large PBD decreases (at least 7 weeks). Panel B extends the original specifications by binning endpoints of the effect window according to Remark 2 and by normalizing the effect at the pre-event period to zero according to Remark 1. All models are estimated in levels with state and time fixed effects.

the estimated dynamic treatment effects are fully consistent with the simple difference-indifferences estimation. In contrast, the large decreases occurring during the recovery period after the Great Recession do not seem to have a systematic effect on search intensity as shown in subgraph B2.

Note that the number of observations differs between Panels A and B of Figure 2. This is due to obeying the data requirements for observations windows for the dependent variable and the treatment status as stipulated in Remark 3. For increases, i.e. the crisis sample, the observation window for the dependent variable runs from 1/2006 to 12/2011. For the last month (12/2011), we are able to generate all leads up until j = -3 as we observe treatment status until 12/2015. However, at the beginning of the observation window, we can calculate the first binned endpoint for a specification with four lags b_{it}^4 in 5/2006. Consequently, our sample is j = 4 periods shorter and $4 \cdot 51 = 204$ observations smaller. An analogous argument applies for the decrease specification and the corresponding recovery sample. Here, BF2017 set the observation window for the dependent variable from 1/2012 to 12/2015. Given that we observe treatment status from 1/2006, we can generate all lags at time 1/2012. However, we cannot generate all leads in 12/2015. Following Remark 3, we have to shorten our estimation sample by $\overline{j} - 1 = 2$ periods. The sample is automatically reduced to the correctly shortened observation window when the distributed-lag model is estimated as we discuss in Section 3.2. By estimating the models on the respective larger samples, Baker and Fradkin (2017) implicitly assume that there are no changes in the PBS prior to 1/2006 and after 12/2015, which might be true, but would need to be demonstrated or at least explicitly assumed.

In their event study, BF2017 follow standard practice and dichotomize the changes in the PBD into a zero-one treatment dummy, which is only switched on for large reforms. While Panel B of Figure 2 shows that binning endpoints leads to convincing event study coefficients, which match the difference-in-differences estimates, the zero-one models do not use all available information. First, increases and decreases are estimated in two separate models (and samples). Second, smaller changes are ignored and used as control group observations, i.e. untreated observations. In the following, we therefore estimate a generalized event study design of Case 4 that exploits all available variation. Moreover, we estimate the model on the full sample, merging the "crisis" sample (1/2006 - 12/2011) and the "recovery" sample (1/2012 - 2015).

As described in equations (13) and (14), all events are scaled with the respective treatment intensities, i.e. the changes in PBD of different magnitudes. The resulting left graph in Figure 3 shows a strong and more precisely estimated negative effect of potential benefit duration (PBD) on job search effort (GJSI). Pre-trends are reasonably flat and never significantly

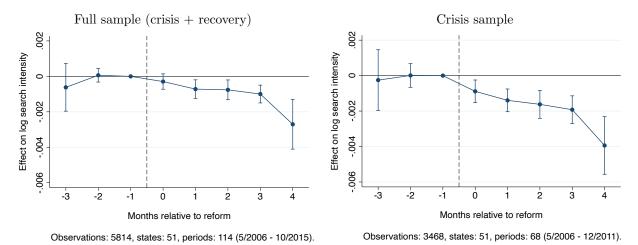
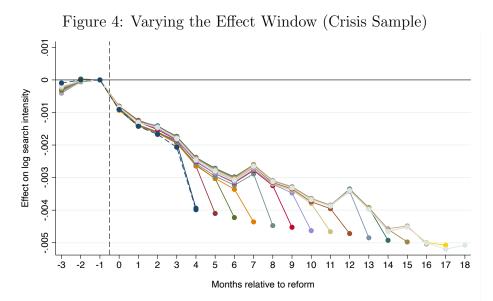


Figure 3: Generalized Event Study Design

Notes: The figure plots the results when applying the generalized event study as defined by equations (13) and (14) to the setting in (Baker and Fradkin, 2017). The graphs shows the dynamic effect of an increase the potential benefit duration by one week on log search intensity as measured by Google Job Search Index (GJSI). 95% confidence intervals are plotted.

different from zero, which corroborates the parallel trend assumption of the research design. As expected confidence bands are much tighter as this specification uses all available variation in the data to identify the policy effects. In terms of magnitude, a 10-week increase in potential benefit duration leads to a decrease in log job search activity of -0.027 (s.e. = 0.007), i.e. 2.7%, after 4 months. Conventionally, the estimates of the generalized event study design are measured on the same scale as simple difference-in-differences model and can be readily compared (see below for more details).

Merging crisis and the recovery samples is not per se the right thing to do. The generalized event study relies on the assumption that treatment effects are proportional to observed treatment intensities as stated in Remark 7. In the context of the replication, the remark implies symmetry between increases and decreases. It is crucial to test these assumptions, e.g. by separating between treatments of different signs (see, e.g., Fuest et al., 2018; Benzarti et al., 2019) and/or splitting by clear-cut time periods as done by BF2017. Panel B of Figure 2 has already pointed to asymmetric effects, with increases in PBD leading to a strong and significant negative effect in search intensity, while decreases in PBD show no effect. For this reason, we also estimate the generalized event study model on the crisis sample only, where mainly increases occurred. The right graph in Figure 3 shows that effects are stronger when focusing only on the crisis sample and pre-trends become even flatter. Hence, there are good reasons to follow BF2017 and analyze the crisis and the recovery sample separately –



Notes: The figure plots the results when applying the generalized event study as defined by equations (13) and (14) to the setting in (Baker and Fradkin, 2017). The graphs shows the dynamic effect of an increase the potential benefit duration by one week on log search intensity as measured by Google Job Search Index (GJSI) for specifications with a varying number of lags. The dashed blue estimate the standard effect window with four lags in the estimation sample of the specification with the longest effect window of 18 lags. Confidence intervals are omitted.

either because increases and decreases of PBD have asymmetric effects or because treatment effects are different during crisis and recovery period or both. We make the crisis sample our baseline sample for the remainder of the analysis.

Next, we study the role of determining the size of the effect window. By Remark 2, binning of endpoints comes along with the assumption that treatment effects have fully materialized after \bar{j} periods. In Figure 3, we see that treatment effects are still on the decline four months after the reform. Moreover, the slope of the event study graph becomes steeper between lag 3 and lag 4. This is an indication that treatment effects have not fully materialized within the effect window and that the assumption of Remark 2 might not hold. We explore this in the following.

One procedure to determine the length of the effect window is to simply increase the number of lags until the treatment effect flattens out. However, this approach comes at a cost as it will often reduce sample size and precision. Nonetheless, we re-estimate the generalized event study design given in equations (13) and (14) gradually increase \bar{j} to one and a half years (18 months). Results are presented in Figure 4. The figure suggests that treatment effects have fully materialized approximately after 16 months. As a result, the long-run effect of PBD on search intensity is around -0.005 (0.001). This effect is higher than the DiD estimate of -0.002 because DiD is an average of the smaller short-run effects

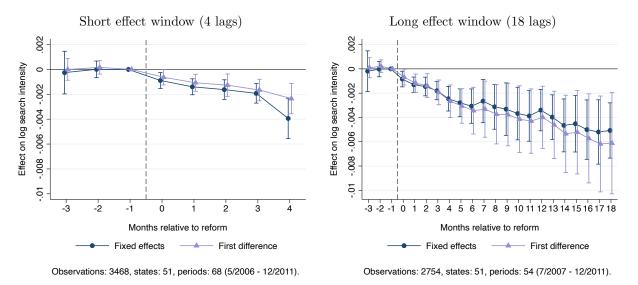


Figure 5: Fixed Effects vs. First Differences (Crisis Sample)

Notes: The figure plots the results when applying the generalized event study as defined by equations (13) and (14) to the setting in (Baker and Fradkin, 2017). The graphs shows the dynamic effect of an increase the potential benefit duration by one week on log search intensity as measured by Google Job Search Index (GJSI) for specifications estimated in levels with a fixed effects model (circle) and in first differences (triangle). 95% confidence intervals are plotted.

and the larger long-run effects.

While increasing the length of the effect window may be possible in some applications, data restrictions and sample size might prevent researchers from reaching the point at which treatment effects have fully materialized. An alternative check to assess whether the effect window is long enough is to compare estimates from a model specified in levels and estimated with unit fixed effects with estimates from a model estimated in first differences. At the endpoint of the effect window, the first difference model only accounts for the change happening from $\overline{j} - 1$ to \overline{j} , while the fixed effects model takes into account a weighted average of the remaining changes. As a result, coefficients from the fixed effects and the first difference specification will deviate if the effect has not fully materialized within the given effect window. This pattern is nicely demonstrated in Figure 5, which shows a clear deviation between first difference and fixed-effects estimates for a short (Panel A) but smaller differences for a longer effect window (Panel B). Clearly, in case the effect window is too short and treatment effects unfold monotonically, the long-run estimates will be biased toward zero.

6 Conclusion

This paper makes three interrelated methodological points, which are important to bear in mind when setting up event study designs in economics. The points are valid in general, and might be particularly helpful when applying the event study technique to settings in public and labor economics with multiple policy shocks of different intensities.

First, researchers need to define an effect window, i.e. the window within which the effect is studied. While this choice is a practical necessity due to limited data availability, it is far from being innocuous. Setting the number of leads and lags to a finite number, practically requires to define the last lag (lead) as an open interval capturing all known events that (will) have happened in the past (future). We refer to this practice as binning. We show that binning affects which unit-year observations are assigned to treatment or control group and thus directly affects the identifying assumption. At the same time, binning introduces important parameter restrictions, which help to identify the model econometrically.

Second, we demonstrate that event study designs and distributed-lag models are equivalent. To be precise, the distributed-lag model is a reparametrization of an event study *with binned endpoints*. Event study estimates can be recovered from distributed-lag models by cumulating the post-treatment and pre-treatment effects away from zero. We use this isomorphism to reinforce the necessity and importance of limiting the effect window properly and critically discuss the plausibility of alternative parameter restrictions used in the literature. The distributed-lag model is in our view also less error-prone in the practical implementation.

Third, we generalize the simple event study with single event dummy events to account for multiple events and/or events of different sign and intensity of the treatment. We show that the event study methodology is perfectly applicable to such environments and that the equivalence between event study and distributed-lag models also holds in the general case. We point to the necessary underlying assumptions and briefly discuss where generalized event study designs could be implemented in light of current empirical research.

In a final part of the paper, we demonstrate the practical relevance of our three methodological points replicating and discussing the event study in Baker and Fradkin (2017).

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Appendix A Numerical Examples

A.1 The Standard Case

In the following, we illustrate the standard event study design set-up, discussed in Section 2 using a simple numerical example. The example also demonstrates the equivalence result between event study and distributed lag models summarized in Remark 5.

Example A.1. We assume a panel that runs from $\underline{t} = 2000$ to $\overline{t} = 2010$ and an effect window from $\underline{j} = -3$ to $\overline{j} = 4$. For unit *i*, the single event takes place at $e_i = 2005$.

In example A.1, the explanatory variables of the event study model in levels (equation 7) and in first differences are visualized by the following matrices, respectively.

t	b_{it}^{-3}	b_{it}^{-2}	b_{it}^{-1}	b_{it}^0	b_{it}^1	b_{it}^2	b_{it}^3	b_{it}^4	Δb_{it}^{-3}	Δb_{it}^{-2}	Δb_{it}^{-1}	Δb_{it}^0	Δb^1_{it}	Δb_{it}^2	Δb_{it}^3	Δb_{it}^4
2000	1	0	0	0	0	0	0	0								
2001	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2002	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2003	0	1	0	0	0	0	0	0	-1	1	0	0	0	0	0	0
2004	0	0	1	0	0	0	0	0	0	-1	1	0	0	0	0	0
2005	0	0	0	1	0	0	0	0	0	0	-1	1	0	0	0	0
2006	0	0	0	0	1	0	0	0	0	0	0	-1	1	0	0	0
2007	0	0	0	0	0	1	0	0	0	0	0	0	-1	1	0	0
2008	0	0	0	0	0	0	1	0	0	0	0	0	0	-1	1	0
2009	0	0	0	0	0	0	0	1	0	0	0	0	0	0	-1	1
2010	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0

The following matrices visualize the explanatory variables of the distributed-lag model (eq. 8) applied to Example A.1, again in levels and first-differences respectively.

t	$x_{i,t+2}$	$x_{i,t+1}$	x_{it}	$x_{i,t-1}$	$x_{i,t-2}$	$x_{i,t-3}$	$x_{i,t-4}$	$\Delta x_{i,t+2}$	$\Delta x_{i,t+1}$	Δx_{it}	$\Delta x_{i,t-1}$	$\Delta x_{i,t-2}$	$\Delta x_{i,t-3}$	$\Delta x_{i,t-4}$
2000	0	0	0	0	0	0	0							
2001	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2002	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2003	1	0	0	0	0	0	0	1	0	0	0	0	0	0
2004	1	1	0	0	0	0	0	0	1	0	0	0	0	0
2005	1	1	1	0	0	0	0	0	0	1	0	0	0	0
2006	1	1	1	1	0	0	0	0	0	0	1	0	0	0
2007	1	1	1	1	1	0	0	0	0	0	0	1	0	0
2008	1	1	1	1	1	1	0	0	0	0	0	0	1	0
2009	1	1	1	1	1	1	1	0	0	0	0	0	0	1
2010	1	1	1	1	1	1	1	0	0	0	0	0	0	0

Note how the event study model with effects up to $\overline{j} = 4$ years after event and $|\underline{j}| = 3$ years before the event corresponds to a distributed-lag model with $\overline{j} = 4$ lags and $|\underline{j}| - 1 = 2$ leads. Also notice that the right matrix becomes a zero matrix if the event takes place on or before 1996 and on or after 2013. Hence, again only information of events between 1997 and 2012 is necessary to estimate the model. The four matrices can also be used to verify the condition that allow deriving our Result 5: $b_{it}^j = d_{i,t-j} = \Delta x_{i,t-j}$ and $b_{i,t-1}^j = d_{i,t-j-1} = \Delta x_{i,t-j-1}$ for $\underline{j} = -3 < j < \overline{j} = 4$ as well as $\Delta b_{it}^j = \Delta b_{it}^{-3} = -d_{i,t-j-1} = -d_{i,t-2} = -\Delta x_{i,t-2}$ and $\Delta b_{it}^{\overline{j}} = \Delta b_{it}^4 = d_{i,t-\overline{j}} = d_{i,t-4} = \Delta x_{i,t-4}$.

In example A.1, the event study effects are calculated according to equation (9) from the distributed-lag/lead coefficients as $\beta_{-3} = -(\gamma_{-1} + \gamma_{-2}), \ \beta_{-2} = -\gamma_{-1}, \ \beta_{-1} = 0, \ \beta_0 = \gamma_0, \ \beta_1 = \gamma_0 + \gamma_1, \ \beta_2 = \gamma_0 + \gamma_1 + \gamma_2, \ \beta_3 = \gamma_0 + \gamma_1 + \gamma_2 + \gamma_3, \ \beta_4 = \gamma_0 + \gamma_1 + \gamma_2 + \gamma_3 + \gamma_4.$

A.2 The General Case

In the following subsection, we present a brief generic numerical example that features the general case derived in Section 4.

Example A.2. We assume a panel that runs from $\underline{t} = 2000$ to $\overline{t} = 2010$ and an effect window from $\underline{j} = -3$ to $\overline{j} = 4$. For individual *i*, one event of intensity $d_{i,2003} = 0.2$ takes place in 2003, another event of intensity $d_{i,2004} = -0.1$ in 2004 and yet another event of intensity $d_{i,2006} = 0.3$ in 2006; there are no events in the other years.

The following four matrices show the explanatory variables for the event study in levels b_{it}^{j} and in first differences Δb_{it}^{j} , as well as for the distributed-lag model in levels, $x_{it} = x_{it} + \Delta x_{i,t-1}$ with initial value $x_{i,t-\bar{j}} = 0$, and in first differences, $\Delta x_{it} = d_{it}$:

t	c_{it}^{-3}	c_{it}^{-2}	c_{it}^{-1}	c_{it}^0	c_{it}^1	c_{it}^2	c_{it}^3	c_{it}^4	Δc_{it}^{-3}	Δc_{it}^{-2}	Δc_{it}^{-1}	Δc_{it}^0	Δc_{it}^1	Δc_{it}^2	Δc_{it}^3	Δc_{it}^4
2000	0.4	0	0	0	0	0	0	0								
2001	0.2	0.2	0	0	0	0	0	0	-0.2	0.2	0	0	0	0	0	0
2002	0.3	-0.1	0.2	0	0	0	0	0	0.1	-0.3	0.2	0	0	0	0	0
2003	0.3	0	-0.1	0.2	0	0	0	0	0	0.1	-0.3	0.2	0	0	0	0
2004	0	0.3	0	-0.1	0.2	0	0	0	-0.3	0.3	0.1	-0.3	0.2	0	0	0
2005	0	0	0.3	0	-0.1	0.2	0	0	0	-0.3	0.3	0.1	-0.3	0.2	0	0
2006	0	0	0	0.3	0	-0.1	0.2	0	0	0	-0.3	0.3	0.1	-0.3	0.2	0
2007	0	0	0	0	0.3	0	-0.1	0.2	0	0	0	-0.3	0.3	0.1	-0.3	0.2
2008	0	0	0	0	0	0.3	0	0.1	0	0	0	0	-0.3	0.3	0.1	-0.1
2009	0	0	0	0	0	0	0.3	0.1	0	0	0	0	0	-0.3	0.3	0
2010	0	0	0	0	0	0	0	0.4	0	0	0	0	0	0	-0.3	0.3
+	~	~	~	~	~	~		2	Λm	Λm	Δ	Δ	Δ	Δ	~ /	1.00
$\frac{t}{2000}$		$\frac{2x_{i,t-1}}{2}$							$\Delta x_{i,t+}$	$-2\Delta x_{i,t}$	$+1\Delta x_{ii}$	$\Delta x_{i,t}$	$-1\Delta x_{1}$	$_{i,t-2}\Delta$	$x_{i,t-3}$	$\Delta x_{i,t-4}$
2000	0	0	0) (0	0	0	0								
2000 2001	0	0	0 0) (0 0	0 0	0 0	0 0	0.2	0	0	0	()	0	0
2000 2001 2002	0 0.2 0.1	0 0 0.2	0 0 2 0)))	0 0 0	0 0 0	0 0 0	0.2	0 0.2	0 0	0 0	()	0 0	0 0
2000 2001 2002 2003	0 0.2 0.1 0.1	0 0 0.2 0.1	0 0 2 0 1 0.))))	0 0 0 0	0 0 0 0	0 0 0 0	0.2 -0.1 0	0 0.2 -0.1	0 0 0.2	0 0 0	(()))	0 0 0	0 0 0
2000 2001 2002 2003 2004	0 0.2 0.1 0.1 0.4	0 0.2 0.1 0.1	0 0 2 0 1 0. 1 0.	2 (1 0)))) .2	0 0 0 0 0	0 0 0 0 0	0 0 0 0 0	0.2 -0.1 0 0.3	0 0.2 -0.1 0	0 0 0.2 -0.1	$0 \\ 0 \\ 0 \\ 0.2$	((()))	0 0 0 0	0 0 0 0
2000 2001 2002 2003 2004 2005	0 0.2 0.1 0.1 0.4 0.4	0 0.2 0.1 0.1 0.4	0 0 2 0 1 0. 1 0. 4 0.	$ \begin{array}{c} $))) .2 .1 (0 0 0 0 0 0 0 0.2	0 0 0 0 0 0	0 0 0 0 0 0	0.2 -0.1 0 0.3 0	0 0.2 -0.1 0 0.3	0 0 0.2 -0.1 0	0 0 0.2 -0.1	((((((())) .2	0 0 0 0 0	0 0 0 0 0
2000 2001 2002 2003 2004 2005 2006	0 0.2 0.1 0.1 0.4 0.4 0.4	0 0.2 0.1 0.1 0.4	0 0 2 0 1 0. 1 0. 4 0.	2 (0) 2 (0) 1 (0) 1 (0) 4 (0)))) .2 .1 () .1 ()	0 0 0 0 0 0.2 0.1	0 0 0 0 0 0 0.2	0 0 0 0 0 0 0	0.2 -0.1 0 0.3 0 0	0 0.2 -0.1 0 0.3 0	0 0 -0.2 -0.1 0 0.3	0 0 0.2 -0.1 0) ((((0. -0))) .2	$egin{array}{ccc} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0.2 \end{array}$	0 0 0 0 0 0
2000 2001 2002 2003 2004 2005 2006 2007	$0 \\ 0.2 \\ 0.1 \\ 0.1 \\ 0.4 \\ 0.4 \\ 0.4 \\ 0.4$	0 0.2 0.1 0.1 0.4 0.4	0 0 2 0 1 0. 1 0. 1 0. 4 0. 4 0.	$\begin{array}{cccccccccccccccccccccccccccccccccccc$))) .2 .1 () .4 ()	0 0 0 0 0.2 0.1 0.1	0 0 0 0 0 0 0.2 0.1	0 0 0 0 0 0 0 0 0 0.2	$0.2 \\ -0.1 \\ 0 \\ 0.3 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ $	0 0.2 -0.1 0 0.3 0 0	0 0.2 -0.1 0 0.3 0	0 0 0.2 -0.1 0 0.3	(((((((((())) .2 .1	0 0 0 0 0 0.2 -0.1	$0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0.2$
2000 2001 2002 2003 2004 2005 2006 2007 2008	$\begin{array}{c} 0\\ 0.2\\ 0.1\\ 0.1\\ 0.4\\ 0.4\\ 0.4\\ 0.4\\ 0.4\\ 0.4\\ 0.4\\ \end{array}$	0 0.2 0.1 0.1 0.4 0.4 0.4	0 0 2 0 1 0. 1 0. 1 0. 1 0. 1 0. 1 0. 1 0.	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$))) .2 .1 () .4 () .4 ()	0 0 0 0 0 0 0 0 0 0 0 0 0 0	0 0 0 0 0 0 0.2 0.1 0.1	0 0 0 0 0 0 0 0.2 0.1	$0.2 \\ -0.1 \\ 0 \\ 0.3 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ $	0 0.2 -0.1 0 0.3 0 0 0	0 0.2 -0.1 0 0.3 0 0	0 0 0.2 -0.1 0 0.3 0) ((((-0 ((0,))) .2 .1 .3	0 0 0 0 0.2 0.1 0	0 0 0 0 0 0 0.2 -0.1
2000 2001 2002 2003 2004 2005 2006 2007	$0 \\ 0.2 \\ 0.1 \\ 0.1 \\ 0.4 \\ 0.4 \\ 0.4 \\ 0.4$	0 0.2 0.1 0.1 0.4 0.4 0.4 0.4	0 0 2 0 1 0. 1	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	0 0 0 0 .2 .1 .4 .4 .4 .4	0 0 0 0 0 0.2 0.1 0.1 0.4 0.4	$\begin{array}{c} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0.2 \\ 0.1 \\ 0.1 \\ 0.4 \end{array}$	0 0 0 0 0 0 0 0 0 0.2	$0.2 \\ -0.1 \\ 0 \\ 0.3 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ $	0 0.2 -0.1 0 0.3 0 0	0 0.2 -0.1 0 0.3 0	0 0 0.2 -0.1 0 0.3	(((((((((())) .1 .3)	0 0 0 0 0 0.2 -0.1	$0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0.2$

Appendix B Identification

In the following, we present intuitive examples that demonstrate how identification is achieved. The empirical model is described by equation (5), hence

$$\Delta y_{it} = \gamma_{-1} d_{i,t-1} + \gamma_0 d_{i,t} + \gamma_1 d_{i,t+1} + \theta_t + \Delta \varepsilon_{it}.$$

There are no unit fixed effects and there is no constant in this regression, so no time fixed effect has to be dropped for identification. Moreover, the examples in this appendix reveal that identification is most easily studied in the first difference version of the distributed-lag specification.

Consider the following seven examples with an effect window from $\underline{j} = -2$ to $\overline{j} = 1$.

Example B.1 (identified). Unit 1 is treated in t = 2, unit 2 is not treated, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

The matrix of explanatory variables in Example B.1 is given by

t	i	t1	t2	t3	d_{t+1}	d_t	d_{t-1}	
0	1						•	
1	1	1	0	0	1	0	0	\leftarrow observation of Δy_1 one period before event
2	1	0	1	0	0	1	0	\leftarrow observation of Δy_1 at event
3	1	0	0	1	0	0	1	\leftarrow observation of Δy_1 one period after event
0	2							
1	2	1	0	0	0	0	0	\leftarrow control for Δy_1 one period before event
2	2	0	1	0	0	0	0	\leftarrow control for Δy_1 at event
3	2	0	0	1	0	0	0	\leftarrow control for Δy_1 after period before event

This is the example given in Borusyak and Jaravel (2017). The non-treated unit pins down the time fixed effects, which thereby can be separated from the dynamic treatment effects. The matrix of explanatory variables has full rank.

Example B.2 (not identified). Both units are treated in t = 2, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

t	i	t1	t2	t3	d_{t+1}	d_t	d_{t-1}	
0	1							
1	1	1	0	0	1	0	0	\leftarrow observation of Δy_1 one period before event
2	1	0	1	0	0	1	0	\leftarrow observation of Δy_1 at event
3	1	0	0	1	0	0	1	\leftarrow observation of Δy_1 one period after event
0	2							
1	2	1	0	0	1	0	0	\leftarrow observation of Δy_1 one period before event
2	2	0	1	0	0	1	0	\leftarrow observation of Δy_1 at event
3	2	0	0	1	0	0	1	\leftarrow observation of Δy_1 one period after event

Clearly, the model in Example B.2 is not identified. Treatment and time effects cannot be separated. This can be remedied if we shift the treatment of one unit by one year.

Example B.3 (identified). Unit 1 treated in t = 2, unit 2 treated in t = 3, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

t	i		t1	t2	t3	d_{t+1}	d_t	d_{t-1}	
0	1	_			•				
1	1		1	0	0	1	0	0	\leftarrow observation of Δy_1 one period before eve
2	1		0	1	0	0	1	0	\leftarrow observation of Δy_1 at event
3	1		0	0	1	0	0	1	\leftarrow observation of Δy_1 one period after event
0	2								
1	2		1	0	0	0	0	0	\leftarrow control for Δy_1 one period before event
2	2		0	1	0	1	0	0	
3	2		0	0	1	0	1	0	

Example B.3 demonstrates the main intuition behind the identification when binning endpoints. The staggered treatment enables to pin down one time fixed effects for unit 2 and t = 1. If t1 is identified, we can back out d_{t-1} for unit 1, then t2 for unit 2, and so on. For such an iterative procedure it is necessary that we observe all event indicators in the data window, they do not have to observable completely for one unit.

Example B.4 (identified). Unit 1 treated in t = 2, unit 2 treated in t = 4, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

t i	$t1 t2 t3 d_{t+1} d_t d_{t-1}$	
0 1		
1 1	1 0 0 0 1 0	\leftarrow observation of Δy_1 at event
$2 \ 1$	0 1 0 0 0 1	\leftarrow observation of Δy_1 one period after event
$3 \ 1$	0 0 1 0 0 0	\leftarrow control for Δy_2 one period before event
$0 \ 2$		
$1 \ 2$	1 0 0 0 0 0	$\leftarrow \text{ control for } \Delta y_1 \text{ at event}$
2 2	0 1 0 0 0 0	\leftarrow control for Δy_1 one period after event
$3 \ 2$	0 0 1 1 0 0	\leftarrow observation of Δy_2 one period before event

Again, we can iteratively separate event from time effects even though we do not observe a full set of event effects for a single unit. However, it is important that we observe at least one endpoint in a year t where the other unit is not treated.

Example B.5 (not identified). Unit 1 treated in t = -1, unit 2 treated in t = 4, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

t	i	t1	t2	t3	d_{t+1}	d_t	d_{t-1}	
0	1							
1	1	1	0	0	0	0	1	$\leftarrow \text{ observation of } \Delta y_1 \text{ one period after event}$
2	1	0	1	0	0	0	0	
3	1	0	0	1	0	0	0	\leftarrow control for Δy_2 one period before event
0	2							
1	2	1	0	0	0	0	0	\leftarrow control for Δy_1 one period after event
2	2	0	1	0	0	0	0	
3	2	0	0	1	1	0	0	\leftarrow observation of Δy_2 one period before event

Here, identification is not achieved. The matrix of explanatory variables has rank 5, as e.g., $d_{t+1} = t_1 - t_3 - d_{t-1}$. The effect one period before and one period after the event are identified but the effect at the event is not observed for any unit.

Example B.6 (not identified). Unit 1 treated in t = 1, unit 2 treated in t = 3, panel from $\underline{t} = 0$ to $\overline{t} = 3$.

t	i	t1	t2	t3	d_{t+1}	d_t	d_{t-1}	
0	1							
1	1	1	0	0	0	1	0	\leftarrow observation of Δy_1 at event
2	1	0	1	0	0	0	1	\leftarrow observation of Δy_1 one period after event
3	1	0	0	1	0	0	0	\leftarrow control for Δy_2 at event
0	2							
1	2	1	0	0	0	0	0	\leftarrow control for Δy_1 at event
2	2	0	1	0	1	0	0	\leftarrow observation of Δy_2 one period before event
3	2	0	0	1	0	1	0	\leftarrow observation of Δy_2 at event

Here, identification is not achieved. The matrix of explanatory variables has rank 5, as e.g., $d_{t-1} = t2 - d_{t+1}$. Iterative identification is not possible. The reason is that only two endpoints in the data window are observed in the same year (t = 2).

Example B.7 (identified). Unit 1 treated in t = 0, unit 2 treated in t = 1, unit 3 treated in t = 2, unit 4 not treated, panel from $\underline{t} = 0$ to $\overline{t} = 1$.

t i	t1	d_{t+1}	d_t	d_{t-1}	
0 1					
1 1	1	1	0	0	\leftarrow observation of Δy_1 one period before event
$0 \ 2$					
1 2	1	0	1	0	\leftarrow observation of Δy_2 at event
0 3					
1 3	1	0	0	1	\leftarrow observation of Δy_3 one period after event
0 4					
1 4	1	0	0	0	$\leftarrow \text{ control for } \Delta y_{1,t-1}, \Delta y_{2,t}, \Delta y_{3,t+1}$

All three dynamic effects are directly identified in direct comparison to a never-treated unit. The matrix of explanatory variables is full rank. This example shows that the observation window for the dependent variable can be shorter than the effect window.

Appendix C More Numerical Examples

C.1 Multiple Events of Identical Intensity

Example C.2. We assume a panel that runs from $\underline{t} = 2000$ to $\overline{t} = 2010$ and an effect window from $\underline{j} = -3$ to $\overline{j} = 4$. For individual *i*, a first event takes place at 2004 and a second at 2006.

The explanatory variables for the event study in levels, $c_{it}^j = b_{it}^j$, and in first differences, $\Delta c_{it}^j = \Delta b_{it}^j$, are

t	c_{it}^{-3}	c_{it}^{-2}	c_{it}^{-1}	c_{it}^0	c_{it}^1	c_{it}^2	c_{it}^3	c_{it}^4	Δc_{it}^{-3}	Δc_{it}^{-2}	Δc_{it}^{-1}	Δc_{it}^0	Δc_{it}^1	Δc_{it}^2	Δc_{it}^3	Δc_{it}^4
2000	2	0	0	0	0	0	0	0								
2001	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2002	1	1	0	0	0	0	0	0	-1	1	0	0	0	0	0	0
2003	1	0	1	0	0	0	0	0	0	-1	1	0	0	0	0	0
2004	0	1	0	1	0	0	0	0	-1	1	-1	1	0	0	0	0
2005	0	0	1	0	1	0	0	0	0	-1	1	-1	1	0	0	0
2006	0	0	0	1	0	1	0	0	0	0	-1	1	-1	1	0	0
2007	0	0	0	0	1	0	1	0	0	0	0	-1	1	-1	1	0
2008	0	0	0	0	0	1	0	1	0	0	0	0	-1	1	-1	1
2009	0	0	0	0	0	0	1	1	0	0	0	0	0	-1	1	0
2010	0	0	0	0	0	0	0	2	0	0	0	0	0	0	-1	1

The explanatory variables of the distributed-lag model in levels, $x_{it} = x_{it} + \Delta x_{i,t-1} = x_{it} + d_{i,t-1}$ with $x_{i,t-\bar{j}} = 0$, and in first differences, $\Delta x_{it} = d_{it}$, are

t	$x_{i,t+2}$	$x_{i,t+1}$	x_{it}	$x_{i,t-1}$	$x_{i,t-2}$	$x_{i,t-3}$	$x_{i,t-4}$	$\Delta x_{i,t+2}$	$\Delta x_{i,t+1}$	Δx_{it}	$\Delta x_{i,t-1}$	$\Delta x_{i,t-2}$	$\Delta x_{i,t-3}$	$\Delta x_{i,t-4}$
2000	0	0	0	0	0	0	0							
2001	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2002	1	0	0	0	0	0	0	1	0	0	0	0	0	0
2003	1	1	0	0	0	0	0	0	1	0	0	0	0	0
2004	2	1	1	0	0	0	0	1	0	1	0	0	0	0
2005	2	2	1	1	0	0	0	0	1	0	1	0	0	0
2006	2	2	2	1	1	0	0	0	0	1	0	1	0	0
2007	2	2	2	2	1	1	0	0	0	0	1	0	1	0
2008	2	2	2	2	2	1	1	0	0	0	0	1	0	1
2009	2	2	2	2	2	2	1	0	0	0	0	0	1	0
2010	2	2	2	2	2	2	2	0	0	0	0	0	0	1

C.2 Single Events of Varying Treatment Intensity

Example C.3. We assume a panel that runs from $\underline{t} = 2000$ to $\overline{t} = 2010$ and an effect window from $\underline{j} = -3$ to $\overline{j} = 4$. For individual *i*, the single event of intensity $d_i = 0.1$ takes place at $e_i = 2005$.

The explanatory variables for the event study in levels, $c_{it}^j = b_{it}^j \times s_i$, and in first differences, $\Delta c_{it}^j = \Delta b_{it}^j \times \Delta s_i$, are

t	c_{it}^{-3}	c_{it}^{-2}	c_{it}^{-1}	c_{it}^0	c_{it}^1	c_{it}^2	c_{it}^3	c_{it}^4	Δc_{it}^{-3}	Δc_{it}^{-2}	Δc_{it}^{-1}	Δc_{it}^0	Δc_{it}^1	Δc_{it}^2	Δc_{it}^3	Δc_{it}^4
$\overline{2000}$	0.1	0	0	0	0	0	0	0								
2001	0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2002	0.1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2003	0	0.1	0	0	0	0	0	0	0.1	0.1	0	0	0	0	0	0
2004	0	0	0.1	0	0	0	0	0	0	-0.1	0.1	0	0	0	0	0
2005	0	0	0	0.1	0	0	0	0	0	0	-0.1	0.1	0	0	0	0
2006	0	0	0	0	0.1	0	0	0	0	0	0	-0.1	0.1	0	0	0
2007	0	0	0	0	0	0.1	0	0	0	0	0	0	-0.1	0.1	0	0
2008	0	0	0	0	0	0	0.1	0	0	0	0	0	0	-0.1	0.1	0
2009	0	0	0	0	0	0	0	0.1	0	0	0	0	0	0	-0.1	0.1
2010	0	0	0	0	0	0	0	0.1	0	0	0	0	0	0	0	0

The corresponding explanatory variables of the distributed-lag model in levels, $x_{it} = x_{it} + \Delta x_{i,t-1} = x_{it} + d_{it} \times s_i$ with $x_{i,t-\bar{j}} = 0$, and in first differences $\Delta x_{it} = d_{it} \times s_i$, are

	$x_{i,t+2}$	$x_{i,t+1}$	x_{it}	$x_{i,t-1}$	$x_{i,t-2}$	$x_{i,t-3}$	$x_{i,t-4}$	$\Delta x_{i,t+2}$	$\Delta x_{i,t+1}$	$\Delta x_{it} \ \Delta x_{i,t-1} \Delta x_{i,t-2} \Delta x_{i,t-3} \Delta x_{i,t-4}$					
2000	0	0	0	0	0	0	0								
2001	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
2002	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
2003	0.1	0	0	0	0	0	0	0.1	0	0	0	0	0	0	
2004	0.1	0.1	0	0	0	0	0	0	0.1	0	0	0	0	0	
2005	0.1	0.1	0.1	0	0	0	0	0	0	0.1	0	0	0	0	
2006	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0.1	0	0	0	
2007	0.1	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0.1	0	0	
2008	0.1	0.1	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0.1	0	
2009	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0.1	
2010	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0	0	0	0	0	0	0	



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