DIFFERENCE-IN-DIFFERENCES WITH VARIATION IN TREATMENT TIMING*

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Abstract: The canonical difference-in-differences (DD) model contains two time periods, "pre" and "post", and two groups, "treatment" and "control". Most DD applications, however, exploit variation across groups of units that receive treatment at different times. This paper derives an expression for this general DD estimator, and shows that it is a weighted average of all possible two-group/two-period DD estimators in the data. This result provides detailed guidance about how to use regression DD in practice. I define the DD estimand and show how it averages treatment effect heterogeneity and that it is biased when effects change over time. I propose a new balance test derived from a unified definition of common trends. I show how to decompose the difference between two specifications, and I apply it to models that drop untreated units, weight, disaggregate time fixed effects, control for unit-specific time trends, or exploit a third difference.

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Difference-in-differences (DD) is both the most common and the oldest quasi-experimental research design, dating back to Snow's (1855) analysis of a London cholera outbreak. A DD estimate is the difference between the change in outcomes before and after a treatment (difference one) in a treatment versus control group (difference two): $(\overline{y}_{TREAT}^{POST} - \overline{y}_{TREAT}^{PRE}) - (\overline{y}_{CONTROL}^{POST} - \overline{y}_{CONTROL}^{POST})$. That simple quantity also equals the estimated coefficient on the interaction of a treatment group dummy and a post-treatment period dummy in the following regression:

$$y_{it} = \gamma + \gamma_i TREAT_i + \gamma_t POST_t + \beta^{2x2} TREAT_i \times POST_t + u_{it}$$
 (1)

The elegance of DD makes it clear which comparisons generate the estimate, what leads to bias, and how to test the design. The expression in terms of sample means connects the regression to potential outcomes and shows that, under a common trends assumption, a two-group/two-period (2x2) DD identifies the average treatment effect on the treated. All econometrics textbooks and survey articles describe this structure,² and recent methodological extensions build on it.³

Most DD applications diverge from this 2x2 set up though because treatments usually occur at different times.⁴ The processes that generate treatment variables naturally lead to variation in timing. Local governments change policy. Jurisdictions hand down legal rulings. Natural disasters strike across seasons. Firms lay off workers. In this case researchers estimate a regression with dummies for cross-sectional units (α_i) and time periods (α_t) , and a treatment dummy (D_{it}) :

$$y_{it} = \alpha_i + \alpha_t + \beta^{DD} D_{it} + e_{it}$$
 (2)

¹ A search from 2012 forward of nber.org, for example, yields 430 results for "difference-in-differences", 360 for "randomization" AND "experiment" AND "trial", and 277 for "regression discontinuity" OR "regression kink".

² This includes, but is not limited to, Angrist and Krueger (1999), Angrist and Pischke (2009), Heckman, Lalonde, and Smith (1999), Meyer (1995), Cameron and Trivedi (2005), Wooldridge (2010).

³ Inverse propensity score reweighting: Abadie (2005), synthetic control: Abadie, Diamond, and Hainmueller (2010), changes-in-changes: Athey and Imbens (2006), quantile treatment effects: Callaway, Li, and Oka (forthcoming).

⁴ Half of the 93 DD papers published in 2014/2015 in 5 general interest or field journals had variation in timing.

In contrast to our substantial understanding of the canonical 2x2 DD model, we know relatively little about the two-way fixed effects DD model when treatment timing varies. We do not know precisely how it compares mean outcomes across groups. We typically rely on general descriptions of the identifying assumption like "interventions must be as good as random, conditional on time and group fixed effects" (Bertrand, Duflo, and Mullainathan 2004, p. 250), and consequently lack well-defined strategies to test the validity of the DD design with timing. We have limited understanding of the treatment effect parameter that regression DD identifies. Finally, we often cannot evaluate when alternative specifications will work or why they change estimates.⁶

This paper shows that the two-way fixed effects DD estimator in (2) is a weighted average of all possible 2x2 DD estimators that compare timing groups to each other. Some use units treated at a particular time as the treatment group and untreated units as the control group. Some compare units treated at two different times, using the later group as a control before its treatment begins and then the earlier group as a control after its treatment begins. As in any least squares estimator, the weights on the 2x2 DD's are proportional to group sizes and the variance of the treatment dummy within each pair. Treatment variance is highest for groups treated in the middle of the panel and lowest for groups treated at the extremes. This result clarifies the theoretical interpretation and identifying assumptions of the general DD model and creates simple new tools for describing the design and analyzing problems that arise in practice.

By decomposing the DD estimator into its sources of variation (the 2x2 DD's) and providing an explicit interpretation of the weights in terms of treatment variances, my results

⁵ Imai, Kim, and Wang (2018) note "It is well known that the standard DiD estimator is numerically equivalent to the linear two-way fixed effects regression estimator if there are two time periods and the treatment is administered to some units only in the second time period. Unfortunately, this equivalence result does not generalize to the multiperiod DiD design...Nevertheless, researchers often motivate the use of the two-way fixed effects estimator by referring to the DiD design (e.g., Angrist and Pischke, 2009)."

⁶ This often leads to sharp disagreements. See Neumark, Salas, and Wascher (2014) on unit-specific linear trends, Lee and Solon (2011) on weighting and outcome transformations, and Shore-Sheppard (2009) on age time fixed effects.

extend recent research on DD models with heterogeneous effects. Assuming equal counterfactual trends, Abraham and Sun (2018), Borusyak and Jaravel (2017), and de Chaisemartin and D'HaultfŒuille (2018b) show that two-way fixed effects DD yields an average of treatment effects across all groups and times, some of which may have negative weights. My results show how these weights arise from differences in timing and thus treatment variances, facilitating a connection between models of treatment allocation and the interpretation of DD estimates. I also explain why the negative weights occur: when already-treated units act as controls, *changes* in their treatment effects over time get subtracted from the DD estimate. This negative weighting only arises when treatment effects vary over time, in which case it typically biases regression DD estimates away from the sign of the true treatment effect. This does not imply a failure of the underlying *design*, but it does caution against the use of a single-coefficient two-way fixed effects specification to summarize time-varying effects.

I also show that because regression DD uses group sizes and treatment variances to weight up simple estimates that each rely on common trends between two groups, its identifying assumption is a variance-weighted version of common trends between all groups. The extent to which a group's differential trend biases the overall estimate equals the difference between how much weight it gets when it acts as the treatment group and how much weight it gets when it acts as the control group. When the earliest and/or latest treated units have low treatment variance, they can get *more* weight as controls than treatments. In designs without untreated units they always

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⁷ Early research in this area made specific observations about stylized specifications such as models with no unit fixed effects (Bitler, Gelbach, and Hoynes 2003), or it provided simulation evidence (Meer and West 2013).

⁸ Related results on the weighting of heterogeneous treatment effects does not provide this intuition. Abraham and Sun (2018, p 9) describe the weights in a DD estimate with constant treatment effects as "residual[s] from predicting treatment status, $D_{i,t}$ with unit and time fixed effects." de Chaisemartin and D'HaultfŒuille (2018b, p 7) and Borusyak and Jaravel (2017) describe these same weights as coming from an auxiliary regression and Borusyak and Jaravel (2017, p 10-11) note that "a general characterization of [the weights] does not seem feasible." Athey and Imbens (2018) also decompose the DD estimator and develop design-based inference methods for this setting. Strezhnev (2018) expresses $\hat{\beta}^{DD}$ as an unweighted average of DD-type terms across pairs of observations and periods.

do. These weights, derived from the estimator itself, form the basis of a new balance test that generalizes the traditional notion of balance between treatment and control groups, and improves on existing strategies that test between treated/untreated units or early/later treated units.

Finally, I use the weighted average result to develop simple tools to describe the general DD design and evaluate why estimates change across specifications. Simply plotting the 2x2 DD's against their weight displays heterogeneity in the estimated components and shows which terms or groups matter most. Summing the weights on the timing comparisons versus treated/untreated comparisons quantifies "how much" of the variation comes from timing (a common question in practice). Additionally, the difference between DD estimates across specifications can often be written as a Oaxaca-Blinder-Kitagawa style decomposition allowing researchers to calculate how much comes from the 2x2 DD's, the weights, or the interaction of the two. The source of instability matters because changes due to different weighting reflect changes in the *estimand* (not bias), while changes in the 2x2 DD's suggest that covariates address confounding. Scatter plots of the 2x2 DD's (or the weights) from different specifications show which specific terms drive these differences. I develop this approach for models that weight, use triple-differences, control for unit-specific time trends (or pre-trends only), and control for disaggregated time fixed effects.

To demonstrate these methods I replicate Stevenson and Wolfers (2006) two-way fixed effects DD study of the effect of unilateral divorce laws on female suicide rates. The two-way fixed effects model suggest that unilateral divorce leads to 3 fewer suicides per million women. More than a third of the identifying variation comes from treatment timing and the rest comes from comparisons to states with no reforms during the sample period. Event-study estimates show that the treatment effects vary strongly over time, however, which biases many of the timing comparisons. The DD estimate (-3.08) is therefore a misleading summary estimate of the average

post-treatment effect, which is closer to -5. My proposed balance test detects significantly higher per-capita income and male/female sex ratios in reform states, in contrast to joint tests of covariate balance across timing groups, which cannot reject the null of balance. Finally, my results show that much of the sensitivity across specifications comes from changes in weights, or a small number of 2x2 DD's, and need not indicate bias.

I. THE DIFFERENCE-IN-DIFFERENCES DECOMPOSITION THEOREM

When units experience treatment at different times, one cannot estimate equation (1) because the post-period dummy is not defined for control observations. Nearly all work that exploits variation in treatment timing uses the two-way fixed effects model in equation (2) (Cameron and Trivedi 2005 pg. 738). Researchers clearly recognize that differences in *when* units received treatment contribute to identification, but have not been able to describe how these comparisons are made.⁹

The simplest way to illustrate how treatment timing works is to consider a balanced panel dataset with T periods (t) and N cross-sectional units (i) that belong to either an untreated group, U; an early treatment group, k, which receives a binary treatment at t_k^* ; and a late treatment group, ℓ , which receives the binary treatment at $t_\ell^* > t_k^*$. Figure 1 plots this structure.

Throughout the paper I use "group" or "timing group" to refer to collections of units either treated at the same time or not treated. I refer to units that do not receive treatment as "untreated" rather than "control" units because, while they obviously act as controls, treated units do, too. k will denote an earlier treated group and ℓ will denote a later treated group. Each group's sample share is n_k and the share of time it spends treated is \overline{D}_k . I use $\overline{y}_b^{POST(a)}$ to denote the sample mean of y_{it} for units in group b during group a's post period, $[t_a^*, T]$. $(\overline{y}_b^{PRE(a)})$ is defined similarly.)

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⁹ Angrist and Pischke (2015), for example, lay out the canonical DD model in terms of means, but discuss regression DD with timing in general terms only, noting that there is "more than one…experiment" in this setting.

The challenge in this setting has been to articulate how estimates of equation (2) compare the groups and times depicted in figure 1. We do, however, have clear intuition, for 2x2 designs in which one group's treatment status changes and another's does not. We could form several such designs, estimable by equation (1), in the three-group case. Figure 2 plots them.

Panels A and B show that with only one of the two treatment groups, an estimate from equation (2) reduces to the canonical case comparing a treated to an untreated group:

$$\widehat{\beta}_{jU}^{2x2} \equiv \left(\overline{y}_{j}^{POST(j)} - \overline{y}_{j}^{PRE(j)}\right) - \left(\overline{y}_{U}^{POST(j)} - \overline{y}_{U}^{PRE(j)}\right), \quad j = k, \ell. \tag{3}$$

If instead there were no untreated units, the two way fixed effects estimator would be identified only by the differential treatment timing between groups k and ℓ . For this case, panels C and D plot two clear 2x2 DD's based on sub-periods when only one group's treatment status changes. Before t_{ℓ}^* , the early units act as the treatment group because their treatment status changes, and later units act as controls during their pre-period. We compare outcomes between the window when treatment status varies, $MID(k, \ell)$, and group k's pre-period, PRE(k):

$$\widehat{\boldsymbol{\beta}}_{k\ell}^{2x2,k} \equiv \left(\overline{\boldsymbol{y}}_k^{MID(k,\ell)} - \overline{\boldsymbol{y}}_k^{PRE(k)} \right) - \left(\overline{\boldsymbol{y}}_\ell^{MID(k,\ell)} - \overline{\boldsymbol{y}}_\ell^{PRE(k)} \right) \tag{4}$$

The opposite situation, shown in panel D, arises after t_k^* when the later group changes treatment status but the early group does not. Later units act as the treatment group, early units act as controls, and we compare average outcomes between the periods $POST(\ell)$ and $MID(k, \ell)$:

$$\widehat{\beta}_{k\ell}^{2x2,\ell} \equiv \left(\overline{y}_{\ell}^{POST(\ell)} - \overline{y}_{\ell}^{MID(k,\ell)}\right) - \left(\overline{y}_{k}^{POST(\ell)} - \overline{y}_{k}^{MID(k,\ell)}\right) \tag{5}$$

The already-treated units in group k can serve as controls even though they are treated because treatment status does not change.

My central result is that any two-way fixed effects DD estimator is a weighted average of well-understood 2x2 DD estimators, like those plotted in figure 2. To see why, first assume a

balanced panel and partial out unit and time fixed effects from y_{it} and D_{it} using the Frisch-Waugh theorem (Frisch and Waugh 1933). Denote grand means by $\overline{\overline{x}} = \frac{1}{NT} \sum_i \sum_t x_{it}$, and adjusted variables by $\widetilde{x}_{it} = (x_{it} - \overline{\overline{x}}) - (\overline{x}_i - \overline{\overline{x}}) - (\overline{x}_t - \overline{\overline{x}})$. $\widehat{\beta}^{DD}$ then equals the univariate regression coefficient between adjusted outcome and treatment variables:

$$\frac{\widehat{cov}\left(\widetilde{y}_{it},\widetilde{D}_{it}\right)}{\widehat{var}\left(\widetilde{D}_{it}\right)} = \frac{\frac{1}{NT}\sum_{i}\sum_{t}\widetilde{y}_{it}\widetilde{D}_{it}}{\frac{1}{NT}\sum_{i}\sum_{t}\widetilde{D}_{it}^{2}}$$

The numerator equals the sample covariance between y_{it} and D_{it} minus the sample covariances between unit means and between time means:

$$\widehat{cov}(\widetilde{y}_{it},\widetilde{D}_{it}) = \frac{1}{NT} \sum_{i} \sum_{t} (y_{it} - \overline{\overline{y}})(D_{it} - \overline{\overline{D}}) - \frac{1}{N} \sum_{i} (\overline{y}_{i} - \overline{\overline{y}})(\overline{D}_{i} - \overline{\overline{D}}) - \frac{1}{T} \sum_{t} (\overline{y}_{t} - \overline{\overline{y}})(\overline{D}_{t} - \overline{\overline{D}})$$
(6)

The appendix shows how to simplify this covariance using the binary nature of D_{it} , and by replacing the \overline{y}_i and \overline{y}_t terms with weighted averages of pre- and post-treatment means or means in each group. ¹⁰ This gives the following theorem:

Theorem 1. Difference-in-Differences Decomposition Theorem

Assume that the data contain k = 1,...,K groups of units ordered by the time when they receive a binary treatment, $t_k^* \in (1,T]$. There may be one group, U, that never receives treatment. The OLS estimate, $\widehat{\beta}^{DD}$, in a two-way fixed-effects model (2) is a weighted average of all possible two-by-two DD estimators.

$$\hat{\beta}^{DD} = \sum_{k \neq U} s_{kU} \hat{\beta}_{kU}^{2x2} + \sum_{k \neq U} \sum_{\ell > k} s_{k\ell} \left[\mu_{k\ell} \hat{\beta}_{k\ell}^{2x2,k} + (1 - \mu_{k\ell}) \hat{\beta}_{k\ell}^{2x2,\ell} \right]$$
(7)

Where the two-by-two DD estimators are:

$$\widehat{\boldsymbol{\beta}}_{kU}^{2x2} \equiv \left(\overline{\boldsymbol{y}}_{k}^{POST(k)} - \overline{\boldsymbol{y}}_{k}^{PRE(k)}\right) - \left(\overline{\boldsymbol{y}}_{U}^{POST(j)} - \overline{\boldsymbol{y}}_{U}^{PRE(j)}\right)$$

Note that the first two terms in equation (6) collapse to a function of pre/post mean differences because $D_{it} = \overline{D}_i = 0$ in the untreated group, $D_{it} = 0$ in pre-treatment periods, and $\overline{y}_i = \overline{D}_k \overline{y}_i^{POST(k)} - (1 - \overline{D}_k) \overline{y}_i^{PRE(k)}$. The sums over i thus include only the treated units and the sum over t includes only post-periods $(t > t_k^*)$. Similarly, sum over time can be broken into pieces based on the different groups' treatment times, and the time means of y_{it} weight together each group's mean. $\widehat{var}(\widetilde{D}_{it})$ follows from replacing y_{it} with D_{it} in the expression for $\widehat{cov}(\widetilde{y}_{it}, \widetilde{D}_{it})$.

$$\begin{split} \widehat{\boldsymbol{\beta}}_{k\ell}^{2x2,k} &\equiv \left(\overline{\boldsymbol{y}}_k^{MID(k,\ell)} - \overline{\boldsymbol{y}}_k^{PRE(k)}\right) - \left(\overline{\boldsymbol{y}}_\ell^{MID(k,\ell)} - \overline{\boldsymbol{y}}_\ell^{PRE(k)}\right) \\ \widehat{\boldsymbol{\beta}}_{k\ell}^{2x2,\ell} &\equiv \left(\overline{\boldsymbol{y}}_\ell^{POST(\ell)} - \overline{\boldsymbol{y}}_\ell^{MID(k,\ell)}\right) - \left(\overline{\boldsymbol{y}}_k^{POST(\ell)} - \overline{\boldsymbol{y}}_k^{MID(k,\ell)}\right) \end{split}$$

the weights are:

$$s_{kU} = \frac{n_k n_U \overline{D}_k (1 - \overline{D}_k)}{\widehat{var}(\widetilde{D}_{it})}$$

$$s_{k\ell} = \frac{n_k n_\ell (\overline{D}_k - \overline{D}_\ell) (1 - (\overline{D}_k - \overline{D}_\ell))}{\widehat{var}(\widetilde{D}_{it})}$$

$$\mu_{k\ell} = \frac{1 - \overline{D}_k}{1 - (\overline{D}_k - \overline{D}_\ell)}$$

and $\sum_{k\neq U} s_{kU} + \sum_{k\neq U} \sum_{\ell>k} s_{k\ell} = 1$.

Proof: See appendix A.

Theorem 1 completely describes the sources of identifying variation in a general DD model and their importance. With K timing groups, one could form $K^2 - K$ "timing-only" estimates that either compare an earlier- to a later-treated timing group $(\widehat{\beta}_{k\ell}^{2x2,k})$ or a later- to earlier-treated timing group $(\widehat{\beta}_{k\ell}^{2x2,\ell})$. With an untreated group, one could form K 2x2 DD's that compare each timing group to the untreated group $(\widehat{\beta}_{k\ell}^{2x2})$. Therefore, with K timing groups and one untreated group, the DD estimator comes from K^2 distinct 2x2 DDs.

The weights come both from group sizes, via the n_j 's, and the treatment variance in each pair.¹¹ With one treated group, the variance of the treatment dummy is $\overline{D}_k(1-\overline{D}_k)$, and is highest

$$\hat{\alpha} = \frac{\sum_{i}(y_i - \bar{y})(x_i - \bar{x})}{\sum_{i}(x_i - \bar{x})^2} = \frac{\sum_{A}(y - \bar{y})(x - \bar{x}) + \sum_{B}(y - \bar{y})(x - \bar{x})}{\sum_{i}(x - \bar{x})^2} = \frac{n_A s_{xy}^A + n_B s_{xy}^B}{s_{xx}^2} = \frac{n_A s_{xx}^{2,A}}{s_{xx}^2} \hat{\alpha}_A + \frac{n_B s_{xx}^{2,B}}{s_{xx}^2} \hat{\alpha}_B$$

¹¹ Many other least-squares estimators weight heterogeneity this way. A univariate regression coefficient equals an average of coefficients in mutually exclusive (and demeaned) subsamples weighted by size and the subsample x -variance:

Similarly, the Wald/IV theorem (Angrist 1988) shows that any IV estimate is a linear combination of Wald estimators that compare two values of the instrument. Gibbons, Serrato, and Urbancic (2018) show a nearly identical weighting formula for one-way fixed effects. Panel data provide another well-known example: a pooled regression coefficients equals a variance-weighted average of two distinct estimators that each use less information: the between estimator for subsample means, and the within estimator for deviations from subsample means.

for units treated in the middle of the panel with $\overline{D}_k = 0.5$. With two treated groups, the variance of the difference in treatment dummies is $(\overline{D}_k - \overline{D}_\ell)(1 - (\overline{D}_k - \overline{D}_\ell))$, and is highest for pairs whose treatment shares differ by 0.5. Figure 2 sets t_k^* and t_ℓ^* so that $\overline{D}_k = 0.67$ and $\overline{D}_\ell = 0.15$, which means that $\overline{D}_k(1 - \overline{D}_k) = 0.22 > 0.13 = \overline{D}_\ell(1 - \overline{D}_\ell)$. Because it has higher treatment variance, group k's comparison to the untreated group, $\widehat{\beta}_{kU}^{2x2}$, gets more weight $(s_{kU} = 0.365)$ than the corresponding term for group ℓ , $\widehat{\beta}_{\ell U}^{2x2}$ ($s_{\ell U} = 0.202$). The difference in treated shares is $\overline{D}_k - \overline{D}_\ell = 0.52$, so $s_{k\ell} = 0.412$.

This decomposition theorem also shows how DD compares groups treated at different times. A two-group "timing-only" estimator is itself a weighted average of the 2x2 DD's plotted in panels C and D of figure 2:

$$\widehat{\beta}_{k\ell}^{2x2} \equiv \mu_{k\ell} \widehat{\beta}_{k\ell}^{2x2,k} + (1 - \mu_{k\ell}) \widehat{\beta}_{k\ell}^{2x2,\ell}$$
(8)

Both groups serve as controls for each other during periods when their treatment status does not change, and the group with higher treatment variance (that is, treated closer to the middle the panel) gets more weight. In the three group example $\overline{D}_k(1-\overline{D}_k) > \overline{D}_\ell(1-\overline{D}_\ell)$ so $\mu_{k\ell} = .67$. Multiplying this by $s_{k\ell} = 0.412$, shows that $\widehat{\beta}_{k\ell}^{2x2,\ell}$ gets less weight than $\widehat{\beta}_{k\ell}^{2x2,k}$: $s_{k\ell}(1-\mu_{k\ell}) = 0.135 < 0.278 = s_{k\ell}\mu_{k\ell}$. ¹³

¹² Changing the number or spacing of time periods changes the weights. Imagine adding T periods to the end of the three-group panel. This would reduce $\widehat{var}(D_{kt})$ to 0.835(1-0.835)=0.138, but it would increase $\widehat{var}(D_{\ell t})$ to 0.58(1-0.58)=0.244. This puts less weight on terms where k is the treatment group ($s_{kU}=0.24$ and $s_{k\ell}\mu_{k\ell}=0.07$) and more weight on terms where ℓ is the treatment group ($s_{\ell U}=0.43$ and $s_{k\ell}(1-\mu_{k\ell})=0.26$).

¹³ Two recent papers present clear analyses using two-group timing-only estimators. Malkova (2017) studies a maternity benefit policy in the Soviet Union and Goodman (2017) studies high school math mandates. Both papers show differences between early and late groups before the reform, PRE(k), during the period when treatment status differs, $MID(k, \ell)$, and in the period after both have implemented reforms, $POST(\ell)$.

II. THEORY: WHAT PARAMETER DOES DD IDENTIFY AND UNDER WHAT ASSUMPTIONS?

Theorem 1 relates the regression DD coefficient to sample averages, which makes it simple to analyze its statistical properties by writing $\hat{\beta}^{DD}$ in terms of potential outcomes (Holland 1986, Rubin 1974). The outcome is $y_{it} = D_{it}Y_{it}^1 + (1 - D_{it})Y_{it}^0$, where Y_{it}^1 is unit i's treated outcome at time t, and Y_{it}^0 is the corresponding untreated outcome. Following Callaway and Sant'Anna (2018, p 7) define the ATT for timing group k at time τ (the "group-time average treatment effect"): $ATT_k(\tau) \equiv E[Y_{it}^1 - Y_{it}^0 | k, t = \tau]$. Because regression DD averages outcomes in pre- and post-periods, I define the average of the $ATT_k(\tau)$ in a date range, W:

$$ATT_k(W) \equiv E[Y_{it}^1 - Y_{it}^0 | k, t \in W]$$
 (9)

In practice, W will represent post-treatment windows that appear in the 2x2 components. Finally, define the difference over time in average potential outcomes (treated or untreated) as:

$$\Delta Y_k^h(W_1, W_0) \equiv E[Y_{it}^h | k, W_1] - E[Y_{it}^h | k, W_0], \quad h = 0, 1$$
 (10)

Applying this notation to the 2x2 DD's in equations (3)-(5), adding and subtracting post-period counterfactual outcomes for the treatment group yields the familiar result that (the probability limit of) each 2x2 DD equals an ATT plus bias from differential trends:

$$\beta_{kU}^{2x2} = ATT_k(POST(k)) + \Delta Y_k^0(POST(k), PRE(k)) - \Delta Y_U^0(POST(k), PRE(k))$$
(11a)

$$\beta_{k\ell}^{2x2,k} = ATT_k(MID(k,\ell)) + \Delta Y_k^0(MID(k,\ell), PRE(k)) - \Delta Y_\ell^0(MID(k,\ell), PRE(k))$$
(11b)

$$\beta_{k\ell}^{2x2,\ell} = ATT_\ell(POST(\ell)) + \Delta Y_\ell^0(POST(\ell), MID(k,\ell)) - \Delta Y_k^0(POST(\ell), MID(k,\ell))$$

$$- [ATT_k(POST(\ell)) - ATT_k(MID(k,\ell))]$$
(11c)

Note that the definition of common trends in (11a) and (11b) involves only counterfactual outcomes, but in (11c) identification of $ATT_{\ell}(POST(\ell))$ involves counterfactual outcomes and changes in treatment effects in the already-treated "control group".

Substituting equations (11a)-(11c) into the DD decomposition theorem expresses the probability limit of the two-way fixed effects DD estimator (assuming that T is fixed and N grows) in terms of potential outcomes and separates the estimand from the identifying assumptions:

$$\underset{N \to \infty}{plim} \, \hat{\beta}^{DD} = \beta^{DD} = VWATT + VWCT + \Delta ATT \tag{12}$$

The first term in (12) is the two-way fixed effects DD estimand, which I call the "variance-weighted average treatment effect on the treated" (VWATT):

$$VWATT \equiv \sum_{k \neq U} \sigma_{kU} ATT_k (POST(k))$$

$$+ \sum_{k \neq U} \sum_{\ell > k} \sigma_{k\ell} \left[\mu_{k\ell} ATT_k (MID(k, \ell)) + (1 - \mu_{k\ell}) ATT_\ell (POST(\ell)) \right] \quad (12a)$$

The σ terms correspond to the s terms in equation (7), but replace sample shares (n) with population shares (n_k^*). ¹⁴ VWATT is always a positively weighted average of ATTs for the units and periods that act as treatment groups across the 2x2 DD's that make up $\hat{\beta}^{DD}$. The weights come from the decomposition theorem and reflect group size and treatment variance.

The second term, which I call "variance-weighted common trends" (VWCT) generalizes common trends to a setting with timing variation:

$$VWCT \equiv \sum_{k \neq U} \sigma_{kU} \left[\Delta Y_k^0 \left(POST(k), PRE(k) \right) - \Delta Y_U^0 \left(POST(k), PRE(k) \right) \right]$$

$$+ \sum_{k \neq U} \sum_{\ell > k} \sigma_{k\ell} \left[\mu_{k\ell} \left\{ \Delta Y_k^0 \left(MID(k, \ell), PRE(k) \right) - \Delta Y_\ell^0 \left(MID(k, \ell), PRE(k) \right) \right\} \right]$$

$$+ (1 - \mu_{k\ell}) \left\{ \Delta Y_\ell^0 \left(POST(\ell), MID(k, \ell) \right) - \Delta Y_k^0 \left(POST(\ell), MID(k, \ell) \right) \right\} \right]$$
(12b)

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¹⁴ Note that a DD estimator is not consistent if T gets large because the permanently turned on treatment dummy becomes collinear with the unit fixed effects ($\frac{X'X}{T}$ does not converge to a positive definite matrix). Asymptotics with respect to T require the time dimension to grow in both directions (see Perron 2006).

Like VWATT, VWCT is also an average of the difference in untreated potential outcome trends between pair of groups (and over different time periods) using the weights from the decomposition theorem. VWCT is new, it defines internal validity for the DD design with timing, and it is weaker than the more commonly assumed *equal* counterfactual trends across groups.

The last term in (12) equals a weighted sum of the *change* in treatment effects within each unit's post-period:

$$\Delta ATT \equiv \sum_{k \neq U} \sum_{\ell > k} \sigma_{k\ell} (1 - \mu_{k\ell}) \left[ATT_k \left(POST(\ell) \right) - ATT_k \left(MID(k, \ell) \right) \right]$$
 (12c)

Because already-treated groups sometimes act as controls, the 2x2 estimators in equation (11c) subtract average changes in their untreated outcomes *and* their treatment effects. Equation (12c) defines the bias that comes from estimating a single-coefficient DD model when treatment effects vary over time. Note that this does not mean that the DD research design is invalid. In this case other specifications, such as an event-study model (Jacobson, LaLonde, and Sullivan 1993) or "stacked DD" (Abraham and Sun 2018, Deshpande and Li 2017, Fadlon and Nielsen 2015), or other estimators such as reweighting strategies (Callaway and Sant'Anna 2018, de Chaisemartin and D'HaultfŒuille 2018b) may be more appropriate.

Recent DD research comes to related conclusions about DD models with timing, but does not describe the full estimator as in equation (12). Abraham and Sun (2018), Borusyak and Jaravel (2017), and de Chaisemartin and D'HaultfŒuille (2018b) begin by imposing pairwise common trends (VWCT=0), and then incorporating ΔATT into the DD estimand. The structure of the decomposition theorem, however, suggests that we should think of ΔATT as a source of bias

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¹⁵ Abraham and Sun (2018, p 6) assume "parallel trends in baseline outcome"; Borusyak and Jaravel (2017, p 10) assume "no pre-trends"; and de Chaisemartin and D'HaultfŒuille (2018b, p 6) assume "common trends" in counterfactual outcomes. Two of these papers analyze common specifications that I do not consider. de Chaisemartin and D'HaultfŒuille (2018b) discuss designs where treatment evolves continuously within group-by-time cells and first-difference specifications. Abraham and Sun (2018) analyze the estimand for semi-parametric event-study models.

because it arises from the way equation (2) forms "the" control group. This distinction, made clear in equation (12), ensures an interpretable estimand (VWATT) and clearly defined identifying assumptions. ¹⁶ This follows from at least two related precedents. de Chaisemartin and D'HaultfŒuille (2018a, p. 5) prove identification of dose-response DD models under the assumption that "the average effect of going from 0 to d units of treatment among units with D(0)=d is stable over time." Treatment effect homogeneity ensures an estimand with no negative weights. Similarly, the monotonicity assumption in Imbens and Angrist (1994) ensures that the local average treatment effect does not have negative weights. In other words, negative weights are a failure of identification rather than a feature of the IV estimand.

A. Interpreting the DD Estimand

When the treatment effect is a constant, $ATT_k(W) = ATT$, $\Delta ATT = 0$, and VWATT = ATT. The rest of this section assumes that VWCT=0 and discusses how to interpret VWATT under different forms of treatment effect heterogeneity.

i. Effects that vary across units but not over time

If treatment effects are constant over time but vary across units, then $ATT_k(W) = ATT_k$ and we still have $\Delta ATT = 0$. In this case DD identifies:

$$VWATT = \sum_{k \neq U} ATT_k \left[\sigma_{kU} + \sum_{j=1}^{k-1} \sigma_{jk} (1 - \mu_{jk}) + \sum_{j=k+1}^{K} \sigma_{jk} \mu_{jk} \right]$$
(13)

-

¹⁶ Equation (12) shows that the negative weighting pointed out elsewhere only occurs when treatment effects vary over time. The mapping between (12) and existing decompositions can be made explicit by rewriting all the $ATT_k(W)$ terms (see equation 9) in terms of group-time effects, which are the object of, for example, Theorem 1 in de Chaisemartin and D'HaultfŒuille (2018b). In general, each group-time effect receives a total amount of weight that comes partly from strictly positive terms (equation 12b) and some from potentially negative terms (equation 12c). When treatment effects are constant, though, the negative weights in (12c) on the group-time effects in $ATT_k(MID(k,\ell))$ cancel with the positive weights on group-time effects in $ATT_k(POST(\ell))$. Then $\Delta ATT = 0$ and DD estimates VWATT, which has strictly positive weights.

VWATT weights together the group-specific ATTs not by sample shares, but by a function of sample shares and treatment variance. The weights in (13) equal the sum of the decomposition weights for all the terms in which group k acts as the treatment group, defined as w_k^T .

The parameter in (13) does not necessarily have a structural interpretation. In general, $w_k^T \neq n_k^*$, so the parameter does not equal the sample ATT.¹⁷ Neither are the weights proportional to the share of time each unit spends under treatment, so VWATT also does not equal the effect in the average treated period. The weights, specifically the central role of treatment variance, comes from the use of least squares. OLS combines 2x2 DD's efficiently by weighting them according to variances of the treatment dummy. WWATT lies along the bias/variance tradeoff: the weights deliver efficiency by potentially moving the point estimate away from, say, the sample ATT.

This tradeoff may not be worthwhile, particularly when VWATT differs strongly from a given parameter of interest, which occurs when treatment effect heterogeneity is correlated with treatment timing. Therefore, the processes that determine treatment timing are central to the interpretation of VWATT (see Besley and Case 2002). For example, a Roy model of selection on gains (where the number of units treated in each period is constrained) implies that treatment rolls out first to units with the largest effects. Site selection in experimental evaluations of training programs (Joseph Hotz, Imbens, and Mortimer 2005) and energy conservation programs (Allcott 2015) matches this pattern. In this case, regression DD underestimates the sample-weighted ATT if t_1^* is early enough (or there are a lot of "post" periods) so that \overline{D}_1 is very small and $\overline{D}_K \approx 0.5$, and overestimates it if t_1^* is late enough (or there are a lot of "pre" periods) so that $\overline{D}_1 \approx 0.5$ and

¹⁷ Abraham and Sun (2018), Borusyak and Jaravel (2017), Chernozhukov et al. (2013), de Chaisemartin and D'HaultfŒuille (2018b), Gibbons, Serrato, and Urbancic (2018), Wooldridge (2005) all make a similar observation. The DD decomposition theorem, provides a new solution for the relevant weights.

¹⁸ This is exactly analogous to the result that two-stage least squares is the estimator that "efficient combines alternative Wald estimates" (Angrist 1991).

 \overline{D}_K is small. The opposite conclusions follow from "reverse Roy" selection where units with the smallest effects select into treatment first, which describes the take up of housing vouchers (Chyn forthcoming) and charter school applications (Walters forthcoming).

An easy way to gauge whether VWATT and a sample-weighted ATT is to scatter the weights from (13) against each group's sample share. These two may be close if there is little variation in treatment timing, if the untreated group is very large, or if some timing groups are very large. Conversely, weighting matters less if the ATT_k 's are similar, which one can evaluate by aggregating each group's 2x2 DD estimates from the decomposition theorem. Finally, one could directly compare VWATT to point estimates of a particular parameter of interest. Several alternative estimators can deliver differently weighted averages of ATT's (Abraham and Sun 2018, Callaway and Sant'Anna 2018, de Chaisemartin and D'HaultfŒuille 2018b).

ii. Effects that vary over time but not across units

Time-varying treatment effects (even if they are identical across units) generate cross-group heterogeneity in VWATT by averaging time-varying effects over different post-treatment windows, but more importantly they mean that $\Delta ATT \neq 0$. Equations (11b) and (11c) show that common trends in counterfactual outcomes leaves one set of timing terms biased $(\hat{\beta}_{k\ell}^{2x2,\ell})$, while common trends between counterfactual and treated outcomes leaves the other set biased $(\hat{\beta}_{k\ell}^{2x2,k})$.

To illustrate this point, figure 3 plots a case where counterfactual outcomes are identical, but the treatment effect is a linear trend-break, $Y_{it}^1 = Y_{it}^0 + \phi \cdot (t - t_i^* + 1)$ (see Meer and West 2013). $\hat{\beta}_{k\ell}^{2x^2,k}$ uses group ℓ as a control group during its pre-period and identifies the ATT during

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¹⁹ Relatedly, de Chaisemartin and D'HaultfŒuille (2018b) derive a statistic that gives the minimum variation in treatment effects that could lead to wrong-signed regression estimates when all treatment effects have the same sign.

the middle window in which treatment status varies: $\phi \frac{(t_{\ell}^* - t_k^* + 1)}{2} \cdot \hat{\beta}_{k\ell}^{2x^2,\ell}$, however, is biased because the control group (k) experiences a trend in outcomes due to the treatment effect:²⁰

$$\beta_{k\ell}^{2x2,\ell} = \overbrace{ATT_{\ell}(POST(\ell))}^{\frac{(T-(t_{\ell}^*-1))}{2}} - \phi \frac{(T-(t_{k}^*-1))}{2} = \phi \frac{(t_{k}^*-t_{\ell}^*)}{2} \le 0$$
 (14)

This bias feeds through to $\beta_{k\ell}^{2x^2}$ according to the size of $(1 - \mu_{k\ell})$:

$$\beta_{k\ell}^{2x^2} = \phi \frac{[(2\mu_{k\ell} - 1)(t_{\ell}^* - t_k^*) + 1]}{2} \tag{15}$$

The entire two-group timing estimate can be wrong signed if $\mu_{k\ell}$ is small (putting more weight on the downward biased term). In figure 3, for example, both units are treated equally close to the ends of the panel, so $\mu_{k\ell} = 0.5$ and the estimated DD effect equals $\frac{\phi}{2}$, even though both units experience treatment effects as large as $\phi \cdot [T - (t_k^* - 1)]$. This type of bias affects the overall DD estimate according to the weights in equation (7). It is smaller when there are untreated units, more so when these units are large.

Note that this bias is specific to the specification in equation (2). More flexible event-study specifications may not suffer from this problem (although see Proposition 2 in Abraham and Sun

$$ATT_{k}(MID(k,\ell)) = \phi \frac{(t_{\ell}^{*} - t_{k}^{*})(t_{\ell}^{*} - t_{k}^{*} + 1)}{2(t_{\ell}^{*} - t_{k}^{*})} = \phi \frac{t_{\ell}^{*} - t_{k}^{*} + 1}{2}$$
$$= \phi(t_{\ell}^{*} - t_{k}^{*}) + \phi \frac{T - t_{\ell}^{*} + 2}{2}$$

$$ATT_k\big(POST(\ell)\big) = \phi(t_\ell^* - t_k^*) + \phi\frac{T - t_\ell^* + 2}{2}$$
 And the difference, which appears in the identifying assumption in (11c) equals:
$$ATT_k\big(POST(\ell)\big) - ATT_k\big(MID(k,\ell)\big) = \phi(t_\ell^* - t_k^*) + \phi\frac{T - t_\ell^* + 2}{2} - \phi\frac{t_\ell^* - t_k^* + 1}{2} = \frac{\phi}{2}\big(T - (t_k^* - 1)\big)$$
 Another way to see this, as noted in figure 3, is that average outcomes in the treatment group are always below average

outcomes in the early group in the $POST(\ell)$ period and the difference equals the maximum size of the treatment effect in group k at the end of the $MID(k,\ell)$ period: $\phi \cdot (t_{\ell}^* - t_k^* + 1)$. Average outcomes for the late group are also below average outcomes in the early group in the $MID(k,\ell)$ period, but by the average amount of the treatment effect in group k during the $MID(k,\ell)$ period: $\phi(t_{\ell}^*-t_k^*+1)$. Outcomes in group ℓ actually fall on average relative to group k, which makes the DD estimate negative even when all treatment effects are positive.

²⁰ The average of the effects for group k during any set of positive event-times is just ϕ times the average event-time. The $MID(k,\ell)$ period contains event-times 0 through $t_{\ell}^* - t_k^* - 1$ and the $POST(\ell)$ period contains event-times $t_{\ell}^* - t_{\ell}^*$ $(t_k^* - 1)$ through $T - (t_k^* - 1)$, so we have:

2018). Fadlon and Nielsen (2015) and Deshpande and Li (2017) use a novel estimator that matches treated units with controls that receive treatment a given amount of time later. This specification does not use already treated units as controls, and yields an average of $\hat{\beta}_{k\ell}^{2x2,k}$ terms with a fixed post-period. Callaway and Sant'Anna (2018) discuss how to summarize heterogeneous treatment effects in this context and develop a reweighting estimator to do so. Summarizing time-varying effects using equation (2), however, yields estimates that are too small or even wrong-signed, and should not be used to judge the meaning or plausibility of effect sizes.²¹

B. What is the identifying assumption and how should we test it?

The preceding analysis maintained the assumption of *equal* counterfactual trends across groups, but (12) shows that (as long as treatment effects do not vary over time) identification of VWATT only requires VWCT to equal zero. The assumption itself is untestable because we cannot, for example, observe changes in Y^0 that occur after treatment or test for them using pre-treatment data.²² Assuming that differential counterfactual trends, ΔY_k^0 , are linear throughout the panel leads to a convenient approximation to VWCT:²³

$$\sum_{k \neq U} \Delta Y_k^0 \left[\sigma_{kU} + \sum_{j=1}^{k-1} \sigma_{jk} \left(1 - 2\mu_{jk} \right) + \sum_{j=k+1}^K \sigma_{kj} \left(2\mu_{kj} - 1 \right) \right] - \Delta Y_U^0 \sum_{k \neq U} \sigma_{kU} = 0 \quad (16)$$

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²¹ Borusyak and Jaravel (2017) show that that common, linear trends, in the post- *and* pre- periods cannot be estimated in this design. The decomposition theorem shows why: timing groups act as controls for each other, so permanent common trends difference out. This is not a meaningful limitation for treatment effect estimation, though, because "effects" must occur after treatment. Job displacement provides a clear example (Jacobson, LaLonde, and Sullivan 1993, Krolikowski 2017). Comparisons based on displacement timing cannot identify whether all displaced workers have a permanently different earnings trajectory than never displaced workers (the unidentified linear component), but they can identify changes in the time-path of earnings around the displacement event (the treatment effect).

²² One can use time-varying confounders as outcomes (Freyaldenhoven, Hansen, and Shapiro 2018, Pei, Pischke, and Schwandt 2017), but this does not test for balance in levels, nor can it be used for sparsely measured confounders.

²³ The path of counterfactual outcomes can affect each 2x2 DD's bias term in different ways. Linearly trending unobservables, for example, lead to larger bias in 2x2 DD's that use more periods. I show in the appendix that in the linear case differences in the magnitude of the bias cancel out across each group's "treatment" and "control" terms, and equation (16) holds.

Equation (16) generalizes the definition of common trends and collapses to the typical pairwise common trends assumption for any two-group estimator. It is immediately clear that *identical* trends across all groups satisfies (16), but this is not a necessary condition for it to hold since the bias induced by a given group's trend depends on the weights in brackets.²⁴

Fortunately, the weights have an intuitive interpretation. The weight on each group's counterfactual trend equals the difference between the total weight it gets when it acts as a treatment group— w_k^T from equation (13)—minus the total weight it gets when it acts as a control group— w_k^C . VWCT can therefore be written as:

$$\sum_{k} \Delta Y_{k}^{0} \left[w_{k}^{T} - w_{k}^{C} \right] = 0 \tag{17}$$

Figure 4 plots $w_k^T - w_k^C$ as a function of \overline{D} (assuming equal group sizes). Units treated in the middle of the panel have high treatment variance and get a lot of weight when they act as the treatment group, while units treated toward the ends of the panel get relatively more weight when they act as controls. As t^* moves closer to 1 or T, $w_k^T - w_k^C$ becomes negative, which shows that some timing groups effectively act as controls. This helps define "the" control group in timing-only designs (the dashed line in figure 6): all groups are controls in *some* terms, but the earliest and/or latest units necessarily get more weight as controls than treatments. The weights also map differential trends to bias.²⁵ A positive trend in group k induces positive bias when $w_k^T - w_k^C > 0$, negative bias when $w_k^T - w_k^C < 0$ (that is, if k is an effective control group), and no bias when $w_k^T - w_k^C = 0$. The size of the bias from a given trend is larger for groups with more weight.

²⁴ Callaway and Sant'Anna (2018) provide alternative definitions of common trends and tests not based on linearity.

²⁵ Applications typically discuss bias in general terms, arguing that unobservables must be "uncorrelated" with timing, but have not been able to specify *how* counterfactual trends would bias a two-way fixed effects estimate. For example, Almond, Hoynes, and Schanzenbach (2011, p 389-190) argue: "Counties with strong support for the low-income population (such as northern, urban counties with large populations of poor) may adopt FSP earlier in the period. This systematic variation in food stamp adoption could lead to spurious estimates of the program impact if those same county characteristics are associated with differential trends in the outcome variables."

Equation (17) also shows exactly how to weight together averages of x_{it} and perform a single t-test that directly captures the identifying assumption. Generate a dummy for the effective treatment group, $B_k = w_k^T - w_k^C > 0$, then regress timing-group means, \overline{x}_k , on B_k weighting by $|w_k^T - w_k^C|$. The coefficient on B_k equals covariate differences weighted by the actual identifying variation, and its t-statistic tests the null of reweighted balance in (17). One can also use this strategy to test for pre-treatment trends in confounders (or the outcome) by regressing \overline{x}_{kt} on B_k , year dummies, and their interaction, or the interaction of B_k with a linear trend using dates before any treatment starts.²⁶

The reweighted balance test has advantages over other strategies for testing balance in this setting. Regressing x_{it} on a constant and dummies for timing groups allows a test of the null of joint balance across groups $(H_0: \overline{x}_k - \overline{x}_U = 0, \ \forall k \in K)$. With many timing groups, however, this F-test will have low power, it does not reflect the importance of each group $(w_k^T - w_k^C)$, and does not show the sign or magnitude of any imbalance. The reweighted test, on the other hand, has higher power and describes the sign and magnitude of imbalance. It is also better than a test for linear relationships between x_{it} and t_k^* or tests for balance between "early" and "late" treated units (Almond, Hoynes, and Schanzenbach 2011, Bailey and Goodman-Bacon 2015). Because the effective control group can include both the earliest and latest treated units, failing to find a linear relationship between \overline{x}_k and t_k^* can miss the relevant imbalance between the most important "middle" units and the end points.

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²⁶ Plotting confounders or pre-trends across groups, however, is important to ensure that a failure to reject does not reflect offsetting trends or covariate means across timing groups.

²⁷ Note that using an event-study specification to evaluate pre-trends does not test for joint equality of pre-trends either. It tests for common trends using the (heretofore unarticulated) estimator itself.

III. DD DECOMPOSITION IN PRACTICE: UNILATERAL DIVORCE AND FEMALE SUICIDE

To illustrate how to use DD decomposition theorem in practice, I replicate Stevenson and Wolfers' (2006) analysis of no-fault divorce reforms and female suicide. Unilateral (or no-fault) divorce allowed either spouse to end a marriage, redistributing property rights and bargaining power relative to fault-based divorce regimes. Stevenson and Wolfers exploit "the natural variation resulting from the different timing of the adoption of unilateral divorce laws" in 37 states from 1969-1985 (see table 1) using the "remaining fourteen states as controls" to evaluate the effect of these reforms on female suicide rates. Figure 5 replicates their event-study result for female suicide using an unweighted model with no covariates. Our results match closely: suicide rates display no clear trend before the implementation of unilateral divorce laws, but begin falling soon after. They report a DD coefficient in logs of -9.7 (s.e. = 2.3). I find a DD coefficient in levels of -3.08 (s.e. = 1.13), or a proportional reduction of 6 percent.²⁹

A. Describing the design

Figure 6 uses the DD decomposition theorem to illustrate the sources of variation. I plot each 2x2 DD against its weight and calculate the average effect and total weight for the three types of 2x2 comparisons: treated/untreated, early/late, late/early.³⁰ As theorem 1 states, the two-way fixed effects estimate, -3.08, is an average of the y-axis values weighted by their x-axis values. Summing the weights on timing terms $(s_{k\ell})$ shows exactly how much of $\hat{\beta}^{DD}$ comes from timing variation (37 percent). The large untreated group puts a lot of weight on $\hat{\beta}_{kU}^{2x2}$ terms, but more on those

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²⁸ Data on suicides by age, sex, state, and year come from the National Center for Health Statistics' Multiple Cause of Death files from 1964-1996, and population denominators come from the 1960 Census (Haines and ICPSR 2010) and the Surveillance, Epidemiology, and End Results data (SEER 2013). The outcome is the age-adjusted (using the national female age distribution in 1964) suicide mortality rate per million women. The average suicide rate in my data is 52 deaths per million women versus 54 in Stevenson and Wolfers (2006). My replication analysis uses levels to match their figure, but the conclusions all follow from a log specification as well.

²⁹ The differences in the magnitudes likely come from three sources: age-adjustment (the original paper does not describe an age-adjusting procedure); data on population denominators; and my omission of Alaska and Hawaii.

³⁰ There are 156 distinct DD components: 12 comparisons between timing groups and pre-reform states, 12 comparisons between timing groups and non-reform states, and $(12^2 - 12)/2 = 66$ comparisons between an earlier switcher and a later non-switcher, and 66 comparisons between a later switcher and an earlier non-switcher

involving pre-1964 reform states (38.4 percent) than non-reform states (24 percent). Figure 6 also highlights the role of a few influential 2x2 terms—comparisons between the 1973 states and nonreform or pre-1964 reform states account for 18 percent of the estimate, and the ten highest-weight 2x2 DD's account for over half.

The bias resulting from time-varying effects is also apparent in figure 6. The average of these post-treatment event-study estimates in figure 5 is -4.92, while the DD estimate is just 60 percent as large. The difference stems from the comparisons of later- to earlier-treated groups. The average treated/untreated estimates are negative (-5.33 and -7.04) as are the comparisons of earlierto later-treated states (although less so: -0.19). ³¹ The comparisons of later- to earlier-treated states, however, are *positive* on average (3.51) and account for the bias in the overall DD estimate. Using the decomposition theorem to take these terms out of the weighted average yields an effect of -5.44—close to the average of the event-study coefficients. The DD decomposition theorem shows that one way to summarize effects in the presence of time-varying heterogeneity is simply to subtract the components of the DD estimate that are biased using the weights in equation (7).

B. Testing the design

Figures 7 and 8 test for covariate balance in the unilateral divorce analysis. Figure 6 plots the reweighted balance test weights, $w_k^T - w_k^C$, from equation (17), the corresponding weights from a timing-only design, and each group's sample share. Larger groups have larger weight, but because they have relatively low treatment variance, the earliest timing groups are downweighted relative to their sample shares.³² In fact, the 1969 states effectively act as controls because $w_k^T - w_k^C < 0$.

³¹ This point also applies to units that are already treated at the beginning of the panel, like the pre-1964 reform states

in the unilateral divorce analysis. Since their $\overline{D}_k = 1$ they can only act as an already-treated control group. If the effects for pre-1964 reform states were constant they would not cause bias.

32 Adding $5 \times year$ to the suicide rate for the 1970 states ($w_k^T - w_k^C = 0.0039$) changes the DD estimate from -3.08 to -2.75, but adding it to the 1973 group ($w_k^T - w_k^C = 0.18$) yields a very biased DD estimate of 12.28.

Figure 8 implements both a joint balance test and the reweighted test using two potential determinants of marriage market equilibria in 1960: per-capita income and the male/female sex ratio. Panel A shows that average per-capita income in untreated states (\$13,431) is lower than the average in every timing group except for those that implemented unilateral divorce in 1969 (which actually get more weight as controls) or 1985. The joint F-test, however, fails to reject the null hypothesis that these means are the same. It is not surprising that such a low power test (12 restrictions on 48 states observations) fails to generate strong evidence against the null. The reweighed test, on the other hand, does detect a difference in per-capita income of \$2,285 between effective treatment states—those that implemented unilateral divorce in 1970 or later—and effective control states—pre-1964 reform states, non-reform states, and the 1969 states. Panel B shows that the 1960 sex ratio is higher in almost all treatment states than in the control states. While the joint test cannot reject the null of equal means, the reweighted test does reject reweighted balance (p = 0.06). 33

C. Evaluating alternative specifications

Researchers almost always estimate models other than (2) and use differences across specifications to evaluate internal validity (Oster 2016) or choose projects in the first place. The DD decomposition theorem suggests a simple way to understand why estimates change. Stacking the 2x2 DD's and weights from (7) into vectors, we can write $\hat{\beta}^{DD} = s'\hat{\beta}^{2x2}$. Any alternative specification that equals a weighted average can be written similarly, and the difference between

³³ One can run a joint test of balance across covariates using a seemingly unrelated regressions (SUR), as suggested by Lee and Lemieux (2010) in the regression discontinuity context. The results of these χ^2 tests are displayed at the top of figure 6. As with the separate balance tests, I fail to reject the null of equal means across groups and covariates. The joint reweighted balance test, however, does reject the null of equal weighted means between effective treatment and control groups. With 48 states and 12 timing groups, there are not sufficient degrees of freedom to implement a full joint test across many covariates. This is an additional rationale for the reweighted test.

the two estimates has the form of a Oaxaca-Blinder-Kitagawa decomposition (Blinder 1973, Oaxaca 1973, Kitagawa 1955):

$$\hat{\beta}_{alt}^{DD} - \hat{\beta}^{DD} = \underbrace{s'(\hat{\beta}_{alt}^{2x2} - \hat{\beta}^{2x2})}^{Due \ to \ 2x2 \ DDs} + \underbrace{(s'_{alt} - s')\hat{\beta}^{2x2}}^{Due \ to \ weights} + \underbrace{(s'_{alt} - s')(\hat{\beta}_{alt}^{2x2} - \hat{\beta}^{2x2})}^{Due \ to \ interaction}$$
(18)

Dividing each term on the right side of (18) by $\hat{\beta}_{alt}^{DD} - \hat{\beta}^{DD}$ shows the proportional contribution of changes in the 2x2 DD's, changes in the weights, and the interaction of the two.³⁴ Differences arising from the 2x2 DD's show that control variables are correlated with treatment and the outcome, often pointing to a real sources of bias. Differences arising because of the weights, though, change the way the DD deals with heterogeneity and do not indicate that the 2x2 DD's are confounded. It is also simple to learn which terms drive each kind of difference by plotting $\hat{\beta}_{alt}^{2x2}$ and s against s_{alt} . One of the most valuable contributions of the DD decomposition theorem is to provide simple new tools for learning why estimates change across specifications.

i. Dropping Untreated Units

Papers commonly estimate models with and without untreated units, and the decomposition theorem shows that this is equivalent to setting all $s_{kU} = 0$ and rescaling the $s_{k\ell}$ to sum to one. Table 2 shows that this changes the unilateral divorce estimate so much that it becomes positive (2.42, s.e. = 1.81), but figure 6 suggests that this occurs not necessarily because of a problem with the design, but because *half* of the timing terms are biased by time-varying treatment effects.

ii. Population Weighting

Solon, Haider, and Wooldridge (2015) show that differences between population-weighted (WLS) and unweighted (OLS) estimates can arise in the presence of unmodeled heterogeneity, and suggest comparing the two estimators (Deaton 1997, Wooldridge 2001). WLS increases the

³⁴ Grosz, Miller, and Shenhav (2018) propose a similar decomposition for family fixed effects estimates.

influence of large units by weighting the means of y that make up each 2x2 DD, and it increases the influence of terms involving large groups by basing the decomposition weights on population rather than sample shares.

Weighting in the unilateral divorce analysis changes the DD estimate from -3.08 to -0.35. Table 2 indicates that just over half of the difference comes from changes in the 2x2 DD terms, 38 percent from changes in the weights, and 9 percent from the interaction of the two. Figure 9 scatters the weighted 2x2 DD's versus the unweighted ones. Most components do not change and lie along the 45-degree line, but large differences emerge for terms involving the 1970 states: Iowa and California. Weighting, which obviously gives more influence to California, makes the terms that use 1970 states as treatments more negative, while it makes terms that use them as controls more positive. This is consistent either with an ongoing downward trend in suicides in California or, as discussed above, strongly time-varying treatment effects. 36

iii. Triple-Difference Estimator

When some units should not be (as) affected by a given treatment, they can be used as a falsification test. Assume that units belong to either an affected group ($G_i = 1$) or an unaffected group ($G_i = 0$). The simplest way to incorporate the "third difference", G_i , would be to estimate separate DD coefficients in each sub-sample: $\widehat{\beta}_0^{DD}$ and $\widehat{\beta}_1^{DD}$. One could equivalently estimate the

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³⁵ Lee and Solon (2011) observe that California drives the divergence between OLS and WLS estimate in analyses of no-fault divorce on divorce rates (Wolfers 2006).

³⁶ Weighting by (a function of) the estimated propensity score (Abadie 2005) is often used to impose covariate balance between treated and untreated units (see Bailey and Goodman-Bacon 2015). The decomposition theorem points to two limitations of this approach. First, reweighting untreated observations has no effect on the timing terms. Second, reweighting untreated observations using the estimated probability of being in *any* treatment group does not impose covariate balance within each pair. By changing the relative weight on different untreated units but leaving their total weight the same, this strategy does not change s, so all differences stem from the way reweighting affects the $\hat{\beta}_{kU}^{2x2}$ terms. Table 2 estimates reweighted models based on a propensity score equation that contains the 1960 sex ratio and per-capita income from figure 8, as well as the 1960 general fertility rate and infant mortality rate. This puts much more weight on Delaware and less weight on New York, and makes almost all $\hat{\beta}_{kU}^{2x2}$ much less negative, changing the overall DD estimate to 1.04. Callaway and Sant'Anna (2018) propose a generalized propensity score reweighted estimator for DD models with timing variation.

following triple-difference model (DDD) on the pooled sample including interactions of G_i with all variables from equation (2):³⁷

$$y_{it} = \alpha_i + \alpha_t + \beta_0^{DD} D_{it} + \alpha_t G_i + \beta^{DDD} D_{it} G_i + e_{it}$$
(19)

 $\widehat{\boldsymbol{\beta}}_0^{DD}$ is the two-way fixed effects DD estimate for the $G_i=0$ sample and $\widehat{\boldsymbol{\beta}}^{DDD}$ equals the difference between the sub-sample DD coefficients: $\widehat{\boldsymbol{\beta}}_1^{DD}-\widehat{\boldsymbol{\beta}}_0^{DD}$.

One problem with this estimator is that $\widehat{\beta}_1^{DD}$ equals an average weighted by the cross-sectional distribution in the in the $G_i=1$ sample, but $\widehat{\beta}_0^{DD}$ uses the cross-sectional distribution of the $G_i=0$ sample. If G_i were an indicator for black respondents, then 2x2 DD's that included Southern states would get more weight in the black than the white sample while the opposite would be true for Vermont and New Hampshire. Estimates of (19) difference out cross-state/cross-year changes in white outcomes weighted by white populations, and so may not capture relative trends by race *within* states. A null result for $\widehat{\beta}_0^{DD}$, which is typically reassuring, could be driven by completely different 2x2 DD's than the ones that matter most for $\widehat{\beta}_1^{DD}$.

DDD specifications that include a more saturated set of fixed effects overcome this problem. If treatment rolled out by state (s), for example, a DDD model can include state-by-time fixed effects ($\alpha_s \alpha_t$):

$$y_{it} = \alpha_i + G_i \alpha_s + G_i \alpha_t + \alpha_s \alpha_t + \beta^{DDD} D_{it} G_i + e_{it}$$
 (20)

The DDD estimate from (20) does equal a weighted average of 2x2 DD's. $\hat{\beta}^{DDD}$ is equivalent to first collapsing the data to mean differences between G-groups within (s,t) cells, $\bar{y}_{st}^{G=1} - \bar{y}_{st}^{G=0}$, then estimating a DD model weighted by cell sizes times the $\hat{var}(G_i|s,t) = g_{st}(1-g_{st})$, where

³⁷ In this set up the third difference partitions units, so $\alpha_i G_i$ is collinear with α_i .

 g_{st} is the mean of G_i by s and t. Unlike (19), estimates from (20) do net out changes across G_i within state/year cells. This changes the weights, though, because the introduction of variation across the third difference within a cell leads to the typical OLS result that cells with more variation get more weight. In this case, "more variation" in sample membership within a cell means approximately equal numbers of units with $G_i = 1$ and $G_i = 0$.

Recasting this version of a DDD model as a DD on differences by G_i implies that the DD decomposition theorem holds, albeit with a slight change to the calculation of the weights. All the results and diagnostic tools derived above apply to specifications like (20) by defining the outcome as $\bar{y}_{st}^{G=1} - \bar{y}_{st}^{G=0}$ and using the proper weights.

iv. Unit-specific linear time trends

Researchers control for unit-specific linear time trends to allow "treatment and control states to follow different trends" (Angrist and Pischke 2009, p 238), and view it as "an important check on the causal interpretation of any set of regression DD estimates" (Angrist and Pischke 2015, p. 199). Appendix B derives a closed-form solution for the detrended estimator and shows that a version of the DD decomposition theorem applies to it. The specific way that unit-specific trends change each 2x2 component fit with previous intuition. They essentially subtract the cross-group difference in averages of y before and after the middle of the panel, \bar{t} , but these differences are weighted by absolute distance to \bar{t} : $|t - \bar{t}|$. This is, as Lee and Solon (2011) point out, akin to a regression discontinuity design in that the estimator relies less on variation at the beginning or end of the panel because this variation is absorbed by the trends. Unfortunately, trends tend to absorb time-varying treatment effects that are necessarily larger at the end of the panel, and in these cases

³⁸ Collapsing the data to the within-cell mean differences $\bar{y}_{st}^{G=1} - \bar{y}_{st}^{G=0}$ and running OLS on the aggregated data (or WLS using cell populations) would eliminate the $g_{st}(1 - g_{st})$ from the decomposition weights.

they over control. Unit-specific trends also increase the weight on units treated at the extremes of the panel, changing estimates for this reason as well.

The unilateral divorce analysis provides a striking illustration of how unit-specific trends can fail because figure 5 shows no pre-trends but strongly time-varying treatment effects. Trends shrink the estimate so much that its sign changes (0.59, s.e.=1.35). Table 2 shows that changes in 2x2 DD's account for 90 percent of the difference between estimates with and without trends, and panel A of figure 10 shows that most of the treated/untreated components are much less negative with trends, with especially large differences for the terms involving the 1970 states. Panel B of figure 10 shows that trends reduce the weight on β_{kU}^{2x2} terms, and increase the importance of timing-only comparisons. Since equation (11c) shows that the timing-only terms are already biased by the time-varying treatment effects, the change in weighting induced by unit-specific trends exacerbates this bias, accounting for 47 percent of the coefficient difference. Because unit-specific trends change the treated/untreated terms *and* give them less weight, the interaction of those two factors accounts for -36 percent of the overall change in the estimates.

v. Group-specific linear pre-trends

A simple strategy to address counterfactual trends is to estimate pre-treatment trends in Y_k^0 directly and partial them out of the full panel (cf. Bhuller et al. 2013, Goodman-Bacon 2018). This is what we hope that unit-specific trends do, it does not depend on the treatment effect pattern, and it does not change the weighting of the 2x2 DD's. Specifically, using data from before t_1^* , one can estimate a pre-trend in y_{it} for each timing group.³⁹ The slope will equal the linear component of

-

³⁹ One could estimate pre-trends for each *unit*, but this yields identical point estimates to the group-specific pre-trends. Moreover, group-specific trends reduce the variability of the estimator because trend deviations that would bias *unit* specific pre-trends cancel out to some extent when averaged by timing group. This matters because this strategy extrapolates potentially many periods into the future, magnifying specification error in the pre-trends. This point applies to unit-specific linear trend specifications as well. Point estimates are identical when this specification includes linear trends for each group rather than each unit.

unobservables plus a linear approximation to trend deviations before before t_1^* . Removing this trend from the full panel yields an outcome variable that is robust to linear trends, unaffected by time-varying treatment effects, and weights each 2x2 DD component in the same way as the unadjusted estimator. Like the unit-specific time trend control strategy, group-specific pre-trends are sensitive to non-linear unobservables in the pre-period. Appendix C analyzes this estimator in detail. Partialling out pre-trends yields a unilateral divorce effect of -6.52 (s.e. = 1.7); close to the average post-treatment effect of -4.92. While inference is outside the scope of this paper (see Athey and Imbens 2018), note that this two-step strategy necessarily involves a partly estimated outcome variable so second-stage standard errors are incorrect.

vi. Disaggregated time effects

If counterfactual outcomes evolve differently by a category, R, to which units belong, one can model these changes flexibly by including separate time fixed effects for each category:

$$y_{it} = \alpha_i + \alpha_t^{R(i)} + \beta_{R \times t}^{DD} D_{it} + e_{it}$$
 (21)

The coefficient $\widehat{\beta}_{R\times t}^{DD}$ equals an average of two-way fixed effects estimates by values of R weighted by the share of units in each R (n^R) and the within-R variance of \widetilde{D}_{it} . For simplicity, I refer to R as "region", but the analysis is not specific to region-by-time fixed effects. When treatments vary by county or city, for example, studies include state-by-time fixed effects (e.g. Almond, Hoynes, and Schanzenbach 2011, Bailey and Goodman-Bacon 2015); when treatments vary by firm studies include industry-by-year fixed effects (e.g. Kovak, Oldenski, and Sly 2018).

Since the decomposition theorem holds for each within-region DD estimate, it also holds for $\widehat{\beta}_{R\times t}^{DD}$. Each 2x2 DD averages the region-specific 2x2 DD's $(\widehat{\beta}_{kU,R}^{2x2})$ and $\widehat{\beta}_{k\ell,R}^{2x2}$ but the weights reflect region size and the within-region distribution of timing groups:

$$\widehat{\beta}_{kU,R\times t}^{2x2} = \sum_{R} \frac{n^R n_k^R n_U^R}{\sum_{R} n^R n_k^R n_U^R} \widehat{\beta}_{kU,R}^{2x2}$$
(22)

$$\widehat{\beta}_{k\ell,R\times t}^{2x2} = \sum_{R} \frac{n^R n_k^R n_\ell^R}{\sum_{R} n^R n_k^R n_\ell^R} \left[\mu_{k\ell} \, \widehat{\beta}_{k\ell,R\times t}^{2x2,k} + (1 - \mu_{k\ell}) \, \widehat{\beta}_{k\ell,R\times t}^{2x2,\ell} \right]$$
(23)

2x2 DD's from large regions get more weight (via the n^R), but the distribution of timing groups within region (the $n_a^R n_b^R$ terms) also determine the importance of each region. Regions with no units in group k contribute nothing to 2x2 DD's involving that group, in contrast to the simpler estimator where that region would contribute controls for group k. If no region contains units from a given pair of groups that pair drops out of the disaggregated time effects specification. These within-region 2x2 DD's in (22) and (23) are weighted together using the cross-region average of the sample share products, $\sum_R n^R n_k^R n_k^R$ as well as treatment variances.

Adding region-by-year effects to the unilateral divorce analysis cuts the estimated effect by a factor of three (-1.16). Figure 11 plots the 2x2 DD's and the weights from this specification against those from the baseline model. 43 of the 156 2x2 terms in the baseline model drop out of the within-region specification, and table 2 shows that about three quarters of the difference in the estimates comes from the way these fixed effects change the weights.

IV. CONCLUSION

Difference-in-differences is perhaps the most widely applicable quasi-experimental research design. Its transparency makes it simple to describe, test, interpret, and teach. This paper extends all of these advantages from canonical two-by-two DD models to general and much more common DD models with variation in the timing of treatment.

My central result, the DD decomposition theorem, shows that a two-way fixed effects DD coefficient equals a weighted average of all possible simple 2x2 DD's that compare one group that changes treatment status to another group that does not. Many ways in which the theoretical

interpretation of regression DD differs from the canonical model stem from the fact that these simple components are weighted together based both on sample sizes *and* the variance of their treatment dummy. This defines the DD estimand, the variance-weighted average treatment effect on the treated (VWATT), and generalizes the identifying assumption on counterfactual outcomes to variance-weighted common trends (VWCT). Moreover, I show that because already-treated units act as controls in some 2x2 DD's, the two-way fixed effects model requires an additional identifying assumption of time-invariant treatment effects.

The DD decomposition theorem also leads to several new tools for practitioners. Graphing the 2x2 DD's against their weight displays all the identifying variation in any DD application, and summing weights across types of comparisons quantifies "how much" of a given estimate comes from different sources of variation. I use the DD decomposition theorem to propose a reweighted balance test that reflects this identifying variation, is easy to implement, has higher power than tests of joint balance across groups, and shows how large and in what direction any imbalance occurs. I suggest several simple methods to learn why estimates differ across alternative specifications. The weighted average representation leads to a Oaxaca-Blinder-Kitagawa-style decomposition that quantifies how much of the difference in estimates comes from changes in the 2x2 DD's, the weights, or both. Plots of the components or the weights across specifications show clearly where differences come from and can help researchers understand why their estimates changes and whether or not it is a problem.

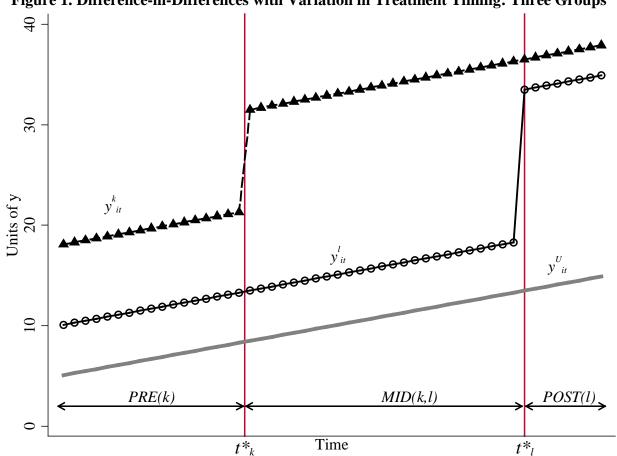
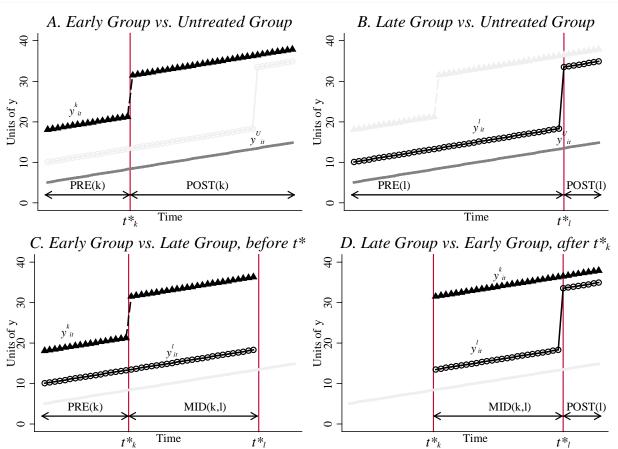


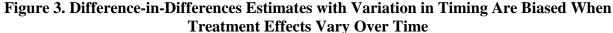
Figure 1. Difference-in-Differences with Variation in Treatment Timing: Three Groups

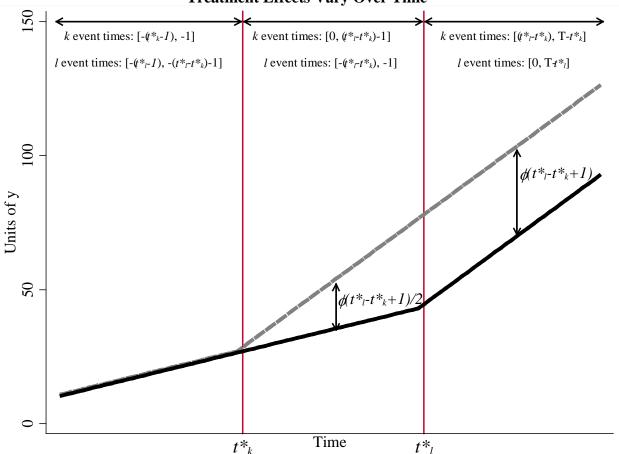
Notes: The figure plots outcomes in three groups: a control group, U, which is never treated; an early treatment group, E, which receives a binary treatment at $t_k^* = \frac{34}{100}T$; and a late treatment group, ℓ , which receives the binary treatment at $t_\ell^* = \frac{85}{100}T$. The x-axis notes the three sub-periods: the pre-period for group k, $[1, t_k^* - 1]$, denoted by PRE(k); the middle period when group E is treated and group E is not, $[t_k^*, t_\ell^* - 1]$, denoted by E is and the post-period for group E, E is group E. I set the treatment effect to 10 in group E and 15 in group E.

Figure 2. The Four Simple (2x2) Difference-in-Differences Estimates from the Three Group Case



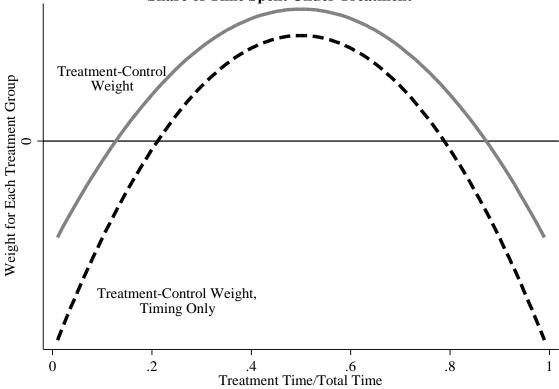
Notes: The figure plots the groups and time periods that generate the four simple 2x2 difference-in-difference estimates in the case with an early treatment group, a late treatment group, and an untreated group from Figure 1. Each panel plots the data structure for one 2x2 DD. Panel A compares early treated units to untreated units ($\hat{\beta}_{kU}^{DD}$); panel B compares late treated units to untreated units ($\hat{\beta}_{kU}^{DD}$); panel C compares early treated units to late treated units during the late group's pre-period ($\hat{\beta}_{k\ell}^{DD,k}$); panel D compares late treated units to early treated units during the early group's post-period ($\hat{\beta}_{k\ell}^{DD,\ell}$). The treatment times mean that $\overline{D}_k = 0.67$ and $\overline{D}_\ell = 0.16$, so with equal group sizes, the decomposition weights on the 2x2 estimate from each panel are 0.365 for panel A, 0.222 for panel B, 0.278 for panel C, and 0.135 for panel D.





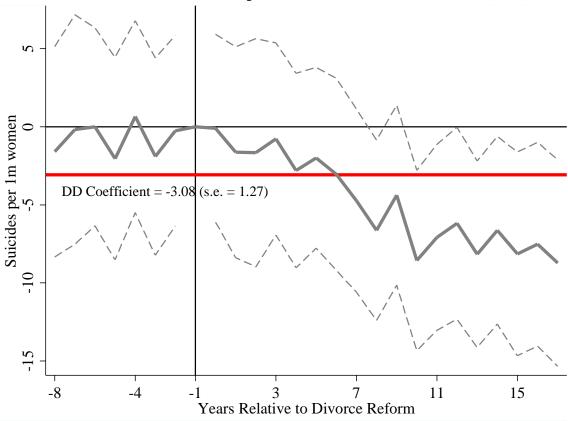
Notes: The figure plots a stylized example of a timing-only DD set up with a treatment effect that is a trend-break rather than a level shift (see Meer and West 2013). Following section II.A.ii, the trend-break effect equals $\phi \cdot (t - t^* + 1)$. The top of the figure notes which event-times lie in the PRE(k), $MID(k,\ell)$, and $POST(\ell)$ periods for each unit. The figure also notes the average difference between groups in each of these periods. In the $MID(k,\ell)$ period, outcomes differ by $\frac{\phi}{2}(t^*_{\ell} - t^*_k + 1)$ on average. In the $POST(\ell)$ period, however, outcomes had already been growing in the early group for $t^*_{\ell} - t^*_k$ periods, and so they differ by $\phi(t^*_{\ell} - t^*_k + 1)$ on average. The 2x2 DD that compares the later group to the earlier group is biased and, in the linear trend-break case, weakly negative despite a positive and growing treatment effect.

Figure 4. Weighted Common Trends: The Treatment/Control Weights as a Function of the Share of Time Spent Under Treatment



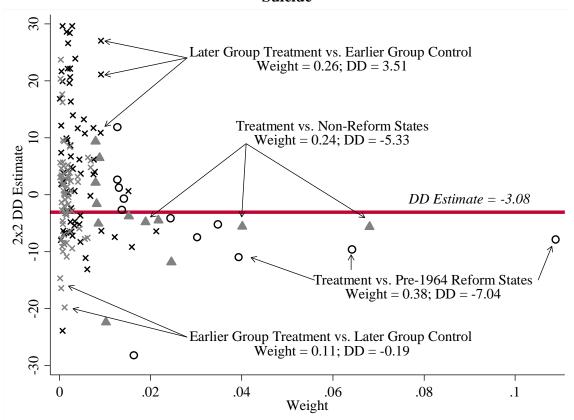
Notes: The figure plots the weights that determine each timing group's importance in the weighted common trends expression in equations (16) and (17).

Figure 5. Event-Study and Difference-in-Differences Estimates of the Effect of No-Fault Divorce on Female Suicide: Replication of Stevenson and Wolfers (2006)



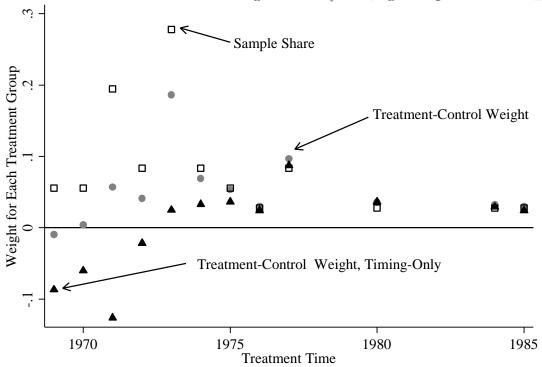
Notes: The figure plots event-study estimates from the two-way fixed effects model on page 276 and plotted in figure 1 of Stevenson and Wolfers (2006), along with the DD coefficient. The specification does not include other controls and does not weight by population. Standard errors are robust to heteroskedasticity.

Figure 6. Difference-in-Differences Decomposition for Unilateral Divorce and Female Suicide



Notes: Notes: The figure plots each 2x2 DD components from the decomposition theorem against their weight for the unilateral divorce analysis. The open circles are terms in which one timing group acts as the treatment group and the pre-1964 reform states act as the control group. The closed triangles are terms in which one timing group acts as the treatment group and the non-reform states act as the control group. The *x*'s are the timing-only terms. The figure notes the average DD estimate and total weight on each type of comparison. The two-way fixed effects estimate, -3.08, equals the average of the *y*-axis values weighted by their *x*-axis value.

Figure 7. Weighted Common Trends in the Unilateral Divorce Analysis: The Treatment/Control Weights on Each Timing Group

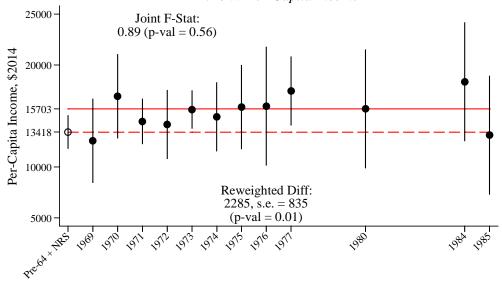


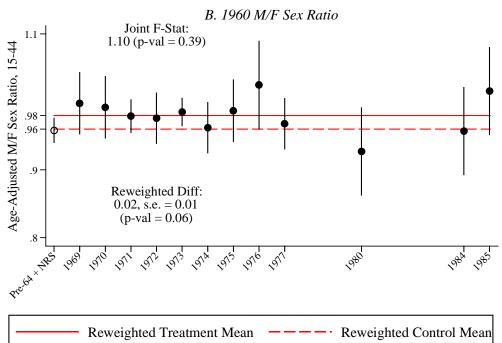
Notes: The figure plots the weights that determine each timing group's role in the weighted common trends expression. These are show in solid triangles and equal the difference between the total weight each treatment timing group receives in terms where it is the treatment group (w_k^T) and terms where it is the control group (w_k^C) : $w_k^T - w_k^C$. The solid circles show the same weights but for versions of each estimator that exclude the untreated (or already-treated) units and, therefore, are identified only by treatment timing. The open squares plot each group's sample share.

Figure 8. Testing for Balance in a Difference-in-Differences Model with Timing:
Reweighted Test versus Joint Test

Joint Test by Group: $\chi_2^2(24) = 23.8 \ (p\text{-val} = 0.47)$ Joint Test of Reweighted Differences: $\chi(2) = 11.1 \ (p\text{-val} = 0.00)$

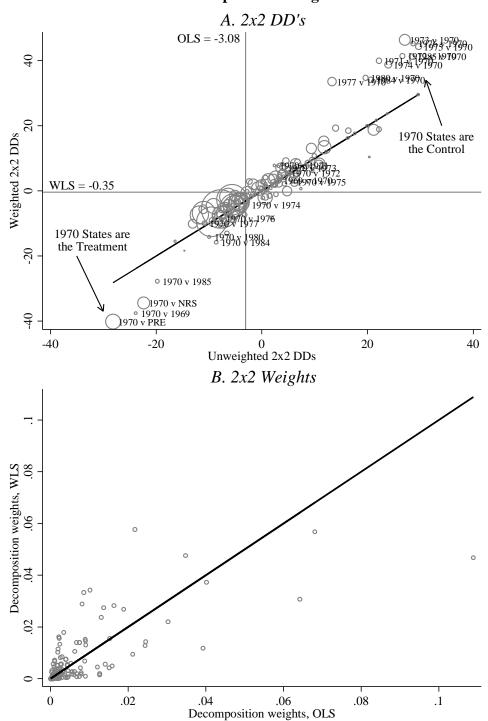
A. 1960 Per-Capita Income





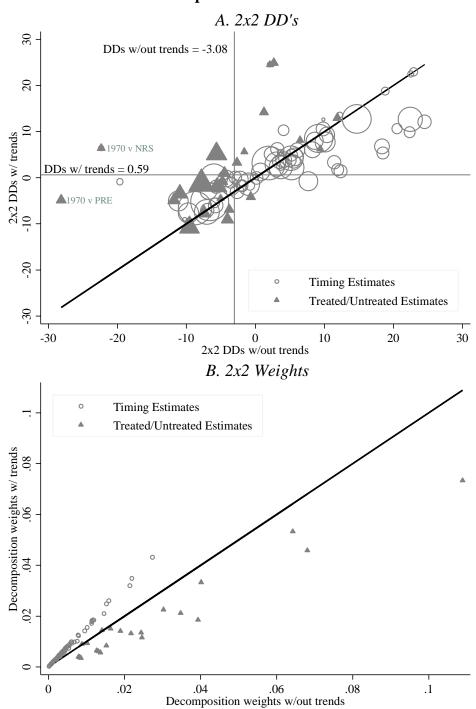
Notes: The figure plots average per-capita income and male/female sex ratio in 1960 for each timing group in the unilateral divorce analysis (combining the non-reform and pre-1964 reform states into the group labeled "Pre-64 + NRS"). The horizontal lines equal the average of these variables using the weights from figure 6 ($w_k^T - w_k^C$). Note that the 1969 states get more weight as a control group, so they are part of the reweighted control mean. Each panel reports the *F*-statistic and *p*-value from a joint test of equality across the means, and the reweighted difference, standard error, and *p*-value from the re-weighted balance test. The top of the figure reports $\chi^2(df)$ test-statistics for both covariates estimated using seemingly unrelated regressions.

Figure 9. Comparison of 2x2 DD Components and Decomposition Weights with and without Population Weights



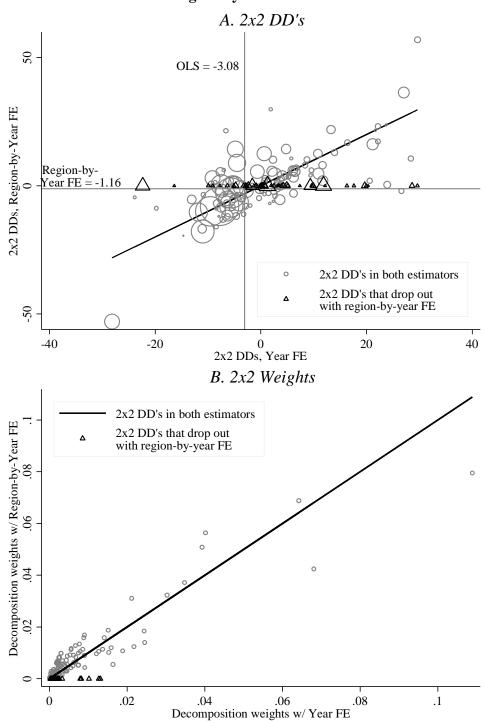
Notes: Panel A plots the 2x2 DD components from two-way fixed effects estimates that use population weights (*y*-axis) and do not (*x*-axis). The size of each point is proportional to its weight in an OLS version of equation (7). WLS estimates are much smaller than OLS estimates, and this figure shows that the source of this discrepancy is the 1970 no-fault divorce states, which include only Iowa and California. Weighting puts much more emphasis on California and, therefore, every 2x2 DD component involving the 1970 states. Dropping California changes yields an OLS estimate of -3.32 and a WLS estimate of -1.43.

Figure 10. Comparison of 2x2 DD Components and Decomposition Weights with and without Unit-Specific Linear Time Trends



Notes: Panel A plots the two-group DD's from a model with unit-specific time trends (y-axis) against those from a model without trends (x-axis). The size of each point is proportional to its weight in a model without trends. The trendadjusted estimate is small and positive compared to a negative DD estimate without them. Much of the change comes from the effect of trends on each 2x2 estimate, especially for treated/untreated comparisons (closed triangles). Panel B is the same except that it plots the weights. Treated/untreated terms that received the most weight in the unadjusted model get relatively less in a trend-adjusted model. Timing terms that received more weight get relatively more in a trend-adjusted model.

Figure 11. Comparison of 2x2 DD Components and Decomposition Weights with and without Region-by-Year Fixed Effects



Notes: Panel A plots the two-group DD's from a model with region-by-year fixed effects (*y*-axis) against those from a model without them (*x*-axis). The size of each point is proportional to its weight in the sparser model with only year fixed effects. 2x2 DD's that drop out of the region-by-year FE model are shown in triangles and appear at "0" on the *y*-axis, although they do not enter the decomposition as zeros. Panel B plots the decomposition weights from the two models. Some 2x2 comparisons drop out in the model with region-by-year effects (when no region has states from both timing groups) and the figures plot these components using triangles.

Table 1. The No-Fault Divorce Rollout: Treatment Times, Group Sizes, and Treatment Shares

No-Fault Divorce	Number of	Share of	Treatment Share	
$\operatorname{Year}(t_k^*)$	States	States (n_k)	(\overline{D}_k)	
Non-Reform States	5	0.10	•	
Pre-1964 Reform States	8	0.16	•	
1969	2	0.04	0.85	
1970	2	0.04	0.82	
1971	7	0.14	0.79	
1972	3	0.06	0.76	
1973	10	0.20	0.73	
1974	3	0.06	0.70	
1975	2	0.04	0.67	
1976	1	0.02	0.64	
1977	3	0.06	0.61	
1980	1	0.02	0.52	
1984	1	0.02	0.39	
1985	1	0.02	0.36	

Notes: The table lists the dates of no-fault divorce reforms from Stevenson and Wolfers (2006), the number and share of states that adopt in each year, and the share of periods each treatment timing group spends treated in the estimation sample from 1964-1996.

Table 2. DD Estimates of the Effect of Unilateral Divorce Analysis on Female Suicide using Alternative Specifications

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Baseline	No Untreated States	WLS	Propensity Score Weighting	Unit- Specific Trends	Group- Specific Pre- Trends	Region- by-Year Fixed Effects
Unilateral Divorce	-3.08	2.42	-0.35	1.04	0.59	-6.52	-1.16
	[1.27]	[1.81]	[1.97]	[1.78]	[1.35]	[2.98]	[1.37]
Difference from baseline specification Share due to:		5.50	2.73	4.12	3.67	-3.44	1.92
2x2 DDs		0	0.52	1	0.90	1	0.37
Weights		1	0.39	0	0.47	0	0.76
Interaction		0	0.09	0	-0.36	0	-0.13

Notes: The table presents DD estimates from the alternative specifications discussed in section III. Column (1) is the two-way fixed effects estimate from equation (2). Column (2) drops the pre-1964 reform and non-reform states. Column (3) weights by state adult populations in 1964. Column (4) weights by the inverse propensity score estimated from a probit model that contains the sex ratio, per-capita income, the general fertility rate, and the infant mortality rate all measured in 1960. Column (5) includes state-specific linear time trends. Column (6) comes from a two-step procedure that first estimates group-specific trends from 1964-1968, subtracts them from the suicide rate, and estimates equation (2) on the transformed outcome variable. Column (7) includes region-by-year fixed effects. Below the standard errors I show the difference between each estimate and the baseline result, an the last three rows show the share of this difference that comes from changes in the 2x2 DD's, the weights, or their interaction as shown in equation (18).

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