

# CAUSAL PANEL 2025

JULY



# Roadmap

Continuous DiD

Dx2 and DxT

Target Parameters

Identification

Selection bias

Interpreting TWFE

Estimation and an Example

# Continuous DiD

- It is very common for people to estimate difference-in-differences panel models where the treatment,  $D$ , is multi-valued or continuous, not binary:

$$Y_{it} = \alpha + \delta D_{it} + \tau_t + \sigma_s + \varepsilon_{it}$$

- Examples include minimum wage papers, my JHR on abortion clinic closures causing increased travel distance, vaccinations, price elasticity of demand etc.
- Variation is in “treatment intensity” and researchers typically use TWFE for estimation, or perhaps count models like Poisson

## Praise for OLS and Continuous Treatments

*"The two-period regression estimator can be easily modified to allow for continuous, or at least non-binary, treatments." (Wooldridge 2005)*

*"A second advantage of regression DiD is that it facilitates the study of policies other than those that can be described by a dummy."* (Angrist and Pischke 2008)

# New Continuous DiD

1. But new work suggests that the TWFE approach to continuous is problematic in light of unrestricted heterogenous treatment effects (Baker, et al. 2025)
2. What of the 2x2 and 2xT will be relevant for dosage designs ( $D \times 2$  and  $D \times T$ )?
3. What is the target parameter, what new assumptions, what estimation methods, what control group?

# Continuous Literature in Causal Inference

- Continuous treatments in instrumental variables (Angrist and Imbens 1995; Angrist, Graddy and Imbens 2000)
- Continuous instruments (Imbens and Angrist 1994; Heckman and Vytacil 2005)
- But the work on continuous diff-in-diff is newer (de Chaisemartin, et al. 2024; de Chaisemartin, et al. 2025; Callaway, Goodman-Bacon and Sant'Anna 2025)
- We will primarily focus on Callaway, Goodman-Bacon and Sant'Anna (2025) for the sake of time and focus

# Average causal response functions

Review of old terms, introducing new terms:

- **ATT**: Average effect for a binary treatment on a sub-population of treated units after they were treated (i.e.,  $E[\delta|D = 1]$  for binary treatment)
- **Dose**: Treatment is not binary but rather multi-valued or continuous. Represents either ATTs for groups at dosages, or movements along a dosage curve – not the same thing as it turns out

# Dosage Parameters give us a multitude of ATTs

Average treated on the treated for a given dose

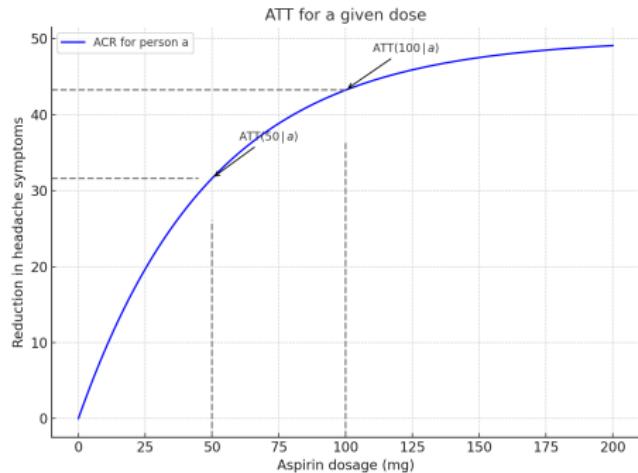
$$ATT(d|d) = E[Y_{it}^d - Y_{it}^0 | D_{it} = d]$$

- This is a restatement of our original ATT, except we are defining the binary treatment,  $D$ , at a given dosage  $d$ .
- “What is the average effect of being 100 kilometers from a hospital compared to being 0 kilometers from a hospital?”
  - Notice the counterfactual – “zero dose” (i.e.,  $Y^0$ )
- This is “the ATT of dose  $d = 100$  km for the groups that are at  $d = 100$  km” which uses as its comparison no dose (as opposed to a marginally smaller dose)

## Earlier dosage parameters in causal inference

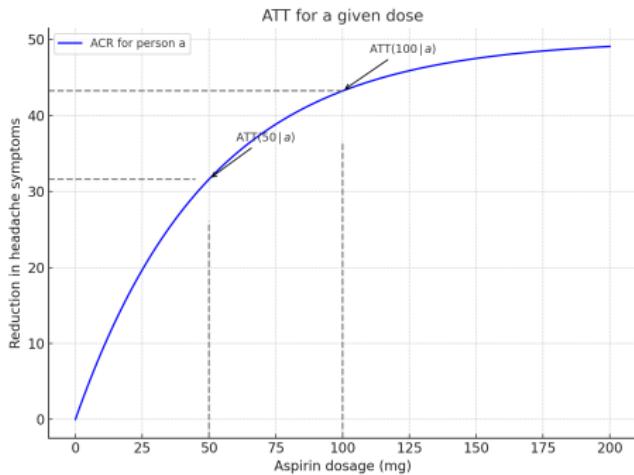
"We refer to the parameter  $\beta$  as the **average causal response (ACR)**. This parameter captures a weighed average causal responses to a unit change in treatment, for those whose treatment status is affected by the instrument. ..." (Angrist and Imbens 1995)

# ATT for a given dose



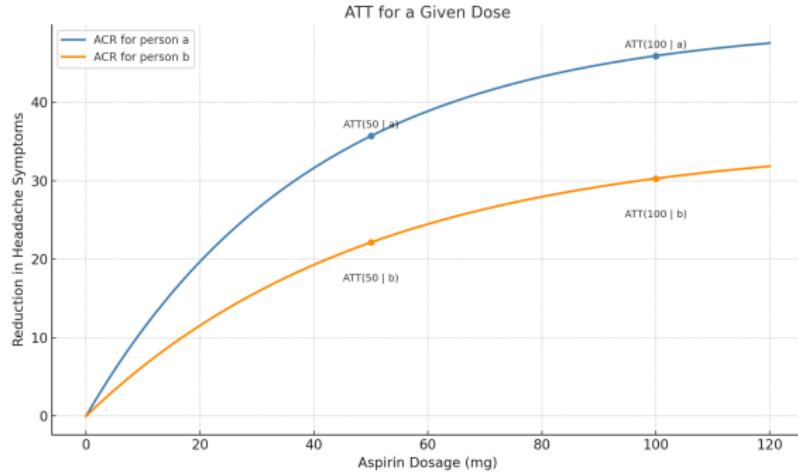
What is the effect of aspirin on headache symptoms for a single person? At 50 mg, the ATT is 31, but at 100mg, it's 44.

# ATT for a given dose



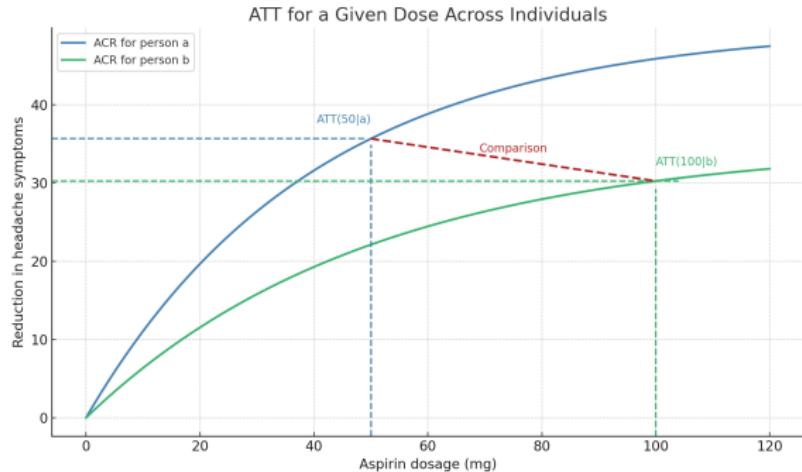
- Assume Giovanni is person  $a$  and he chose in reality  $d = 50$  mg of aspirin.
- Then  $ATT(100|a)$  is a counterfactual ATT because while it is the same person, it is a dosage that he has not yet taken.
- All ATTs have counterfactuals, but the curve maps out not just counterfactual causal effects, but also *counterfactual dosages*

# ATT for a given dose



What if everyone has different responses? In other words, Giovanni, person *a*, and Jessica, person *b*, have *difference* causal responses and different *response* at even the same dosage. Maybe Giovanni responds better to aspirin than Jessica, but Giovanni takes 50 mg, and Jessica takes 100. What then?

# ATT for a given dose

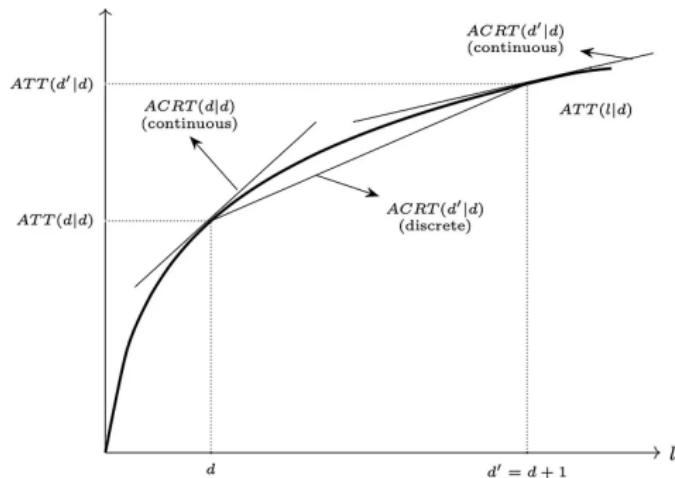


You might estimate negative effects, even though higher dosages actually improve health outcomes

## Moving along the dosage is not the ATT

- So, the vertical axis is a measure of the  $\text{ATT}(d-d)$ , similar to what we have been doing – but always the counterfactual is "zero dose"
- But when if we were to *move* between points?
- That's a different parameter called the average causal response for the treated group, or  $\text{ACRT}(d|d)$

Figure 2: Causal Parameters in a Continuous Difference-in-Differences Design



*Notes:* The figure plots  $ATT(\cdot|d)$  (the average effect of experiencing each dose among units that actually experienced dose  $d$ ). We highlight causal parameters for two doses,  $d$  and  $d'$ .  $ATT(d|d)$  and  $ATT(d'|d)$  are average treatment effect on the treated parameters and refer to the height of the curve.  $ACRT(d|d)$  and  $ACRT(d'|d)$  are average causal response parameters and refer to the slope of the curve. We show them for a continuous dose, when the  $ACRT$  is a tangent line, and for a discrete dose when  $ACRT$  is a line connecting two discrete points on  $ATT(D|d)$ .

# What is the ACRT?

- ACRT is the causal effect of dose  $D = d_j$  vs a different dose  $D = D_{j-1}$  for group  $d$ 
  - Easiest example is the demand function: at  $p = \$10$ , I buy 10 units, but at  $p = \$11$ , I buy 5 units.
  - Causal effect of that one dollar increase is  $-5$  units
  - Demand curves are pairs of potential outcomes and treatments and equilibrium “selects” one of them
- Discrete/multi-valued treatment is linear difference between two ATTs for the same city
- Continuous treatment is the derivative of the function itself

## Definition of the ACRT

$$ACRT(d|d') = \frac{\partial ATT(l|d')}{\partial l} \Big|_{l=d}$$

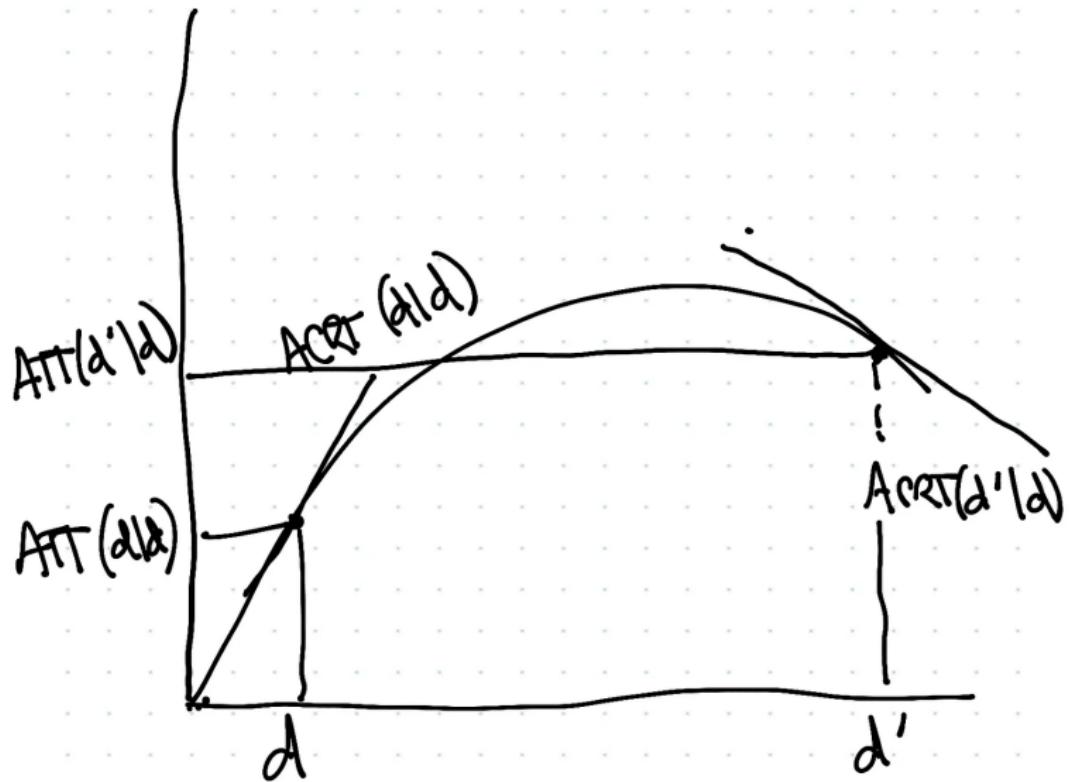
# Derivation of ACRT

Average causal response parameters for absolutely continuous treatments are defined as

$$ACRT(d|d') = \frac{\partial ATT(l|d')}{\partial l} \Big|_{l=d} = \frac{\partial \mathbb{E}[Y_{t=2}(l)|D = d']}{\partial l} \Big|_{l=d} \text{ and } ACR(d) = \frac{\partial ATE(d)}{\partial d} = \frac{\partial \mathbb{E}[Y_{t=2}(d)]}{\partial d}.$$

$ACRT(d|d')$  equals the derivative of the  $t = 2$  average potential outcome for units that received dose  $d$  evaluated at  $d'$ . This is equivalent to the derivative of  $ATT(l|d)$  with respect to  $l$ , evaluated at  $l = d$ . For discrete treatments, average causal responses are defined in a similar way but with slightly

# Heterogeneities



# Assumptions

The authors lay out 5 assumptions, but I'm going to focus on 4. They are:

1. Random sampling
2. Continuous (2a) and Multi-Valued Treatment (2b)
3. No Anticipation and Observed Outcomes
4. Parallel trends

## Identifying $ATT(d|d)$

We can estimate the  $ATT(d|d)$  using the simple DiD equation:

$$E[\Delta Y_{it}|D_i = d] - E[\Delta Y_{it}|D_i = 0]$$

No anticipation and parallel trends converts this comparison of before and after into the  $ATT(d|d)$

$ATT(d|d)$  is using as its counterfactual the “no treatment”, note. Treatment is a dosage compared to zero iow.

Which means we will need as our controls units whose potential outcome was *always* zero in the long difference

# Identifying ACRT

$$\begin{aligned}ATT(b|b) - ATT(a|a) &= (E[\Delta Y_{it}|D_i = a] - E[\Delta Y_{it}|D_i = 0]) \\&\quad - (E[\Delta Y_{it}|D_i = b] - E[\Delta Y_{it}|D_i = 0]) \\&= E[\Delta Y_{it}|D_i = a] - E[\Delta Y_{it}|D_i = b]\end{aligned}$$

Comparing high and low dose groups.

# Identifying ACRT

$$\begin{aligned} ATT(d_j|d_j) - ATT(d_{j-1}|d_{j-1}) &= \\ (ATT(d_j|d_j) - ATT(d_{j-1}|d_j)) + (ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})) &= \\ (\textcolor{blue}{ACRT(d_j|d_j)}) + (\textcolor{red}{ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})}) &= \end{aligned}$$

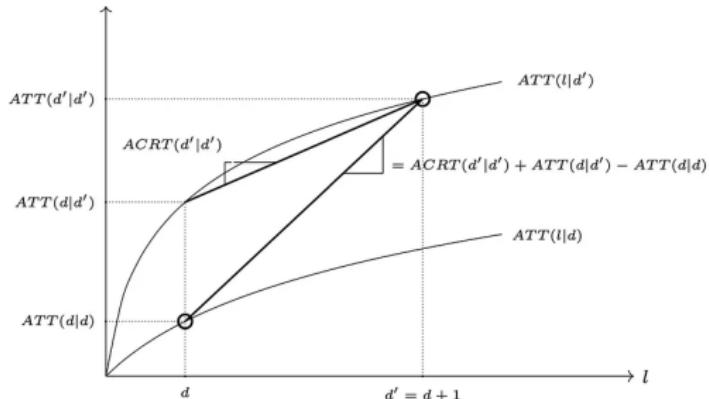
Part in blue is the movement along the average causal response function, the ACRT, and is causal. The part in red is selection bias.

# Identifying ACRT

$$\begin{aligned} ATT(d_j|d_j) - ATT(d_{j-1}|d_{j-1}) &= \\ (ATT(d_j|d_j) - ATT(d_{j-1}|d_j)) + (ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})) &= \\ (\textcolor{blue}{ACRT(d_j|d_j)}) + (\textcolor{red}{ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})}) &= \end{aligned}$$

Notice parallel trends allows to identify ATT terms but we need additional assumptions for this red part to vanish. We must assume that the ATT for cities that chose  $d_j$  and cities that chose  $d_{j-1}$  are the same had they both chose  $d_{j-1}$ .

Figure 3: Non-identification of Average Causal Response with Treatment Effect Heterogeneity, Two Discrete Doses



*Notes:* The figure shows that comparing adjacent  $ATT(d|d)$  estimates equals an  $ACRT$  parameter (the slope of the higher-dose group's  $ATT$  function) and selection bias (the difference between the two groups'  $ATT$  functions at the lower dose).

**Theorem 3.2.** Under Assumptions 1 to 4, causal response parameters are not identified. Specifically,

(a) Under Assumption 2(a), for  $d \in \mathcal{D}_+^c$ ,

$$\frac{\partial \mathbb{E}[\Delta Y|D=d]}{\partial d} = \frac{\partial ATT(d|d)}{\partial d} = ACRT(d|d) + \underbrace{\frac{\partial ATT(d|l)}{\partial l}}_{\text{selection bias}} \Big|_{l=d};$$

(b) For  $(h, l) \in \mathcal{D} \times \mathcal{D}$ ,

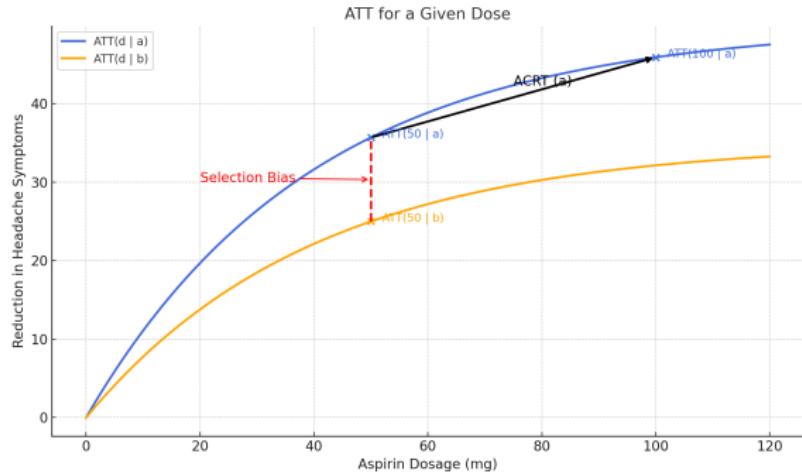
$$\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=l] = ATT(h|h) - ATT(l|l)$$

$$= \underbrace{\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h]}_{\text{causal response}} + \underbrace{\left(ATT(l|h) - ATT(l|l)\right)}_{\text{selection bias}}.$$

When Assumption 2(b) holds, taking  $h = d_j$  and  $l = d_{j-1}$  implies that

$$\mathbb{E}[\Delta Y|D=d_j] - \mathbb{E}[\Delta Y|D=d_{j-1}] = ACRT(d_j|d_j) + \underbrace{ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})}_{\text{selection bias}}.$$

# Causality and selection bias



Draw the ACET for top curve and the selection bias from estimation under assumptions 1 to 4.

## Interpreting this

- Unrestricted heterogenous treatment effects (across dosage levels and across units with difference dose response functions) is not itself the problem
- If we randomized dosages, then
$$ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1}) = 0$$
- Why? Because then there is no selection on gains from dosages, and average causal response functions are the same for all dosage groups
- So then when is this a problem? Sorting on gains

# Interpreting this

- When estimating treatment effects using continuous DiD, you will need to make one of two assumptions
  1. Strong parallel trends: That selection bias isn't there because everyone has the same ACR
  2. Parallel trends plus homogenous treatment effect functions
- Roy model like sorting on gains typically lead to violations of the second condition insofar as there is heterogenous returns to dosages across units
- So the question you have to ask yourself is do you think that cities are “optimally setting the minimum wage” around some given minimum wage?

## Stronger assumption

- I'm really not so sure I think that when it comes to state legislation that I think a Roy model is likely responsible for the equilibrium
- Solving constrained optimization problems is hard and unlikely is it the case that Florida's ATT and Georgia's ATT are terribly different from one another had both chosen the same minimum wage (but that is the bias)
- Authors introduce a fifth assumption that will eliminate selection bias, but at the price of restricting heterogeneity

## Discussion of strong parallel trends

We discuss an alternative but typically stronger assumption, which we call *strong parallel trends*, that says that the path of outcomes for lower-dose units must reflect how higher-dose units' outcomes would have changed had they instead experienced the lower dose. Thus, *strong parallel trends* restricts treatment effect heterogeneity and justifies comparing dose groups. Absent this type of condition, comparisons across dose groups include causal responses but are "contaminated" by an additional term involving possibly different treatment effects of the same dose for different dose groups—we refer to this additional term as *selection bias*.

## A5: Strong parallel trends

**Assumption 5** (Strong Parallel Trends). *For all  $d \in \mathcal{D}$ ,*

$$\mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)] = \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D = d].$$

## Randomization and strong parallel trends

- Randomized dosages guarantees that the ACRT are the same across all dosage groups
- In this situation, strong parallel trends holds because all dosages have the same ATE and ACRT
- Roy like sorting on dosage may be the biggest challenge you'll face – schooling stops, family size may not satisfy strong parallel trends

# Interpreting TWFE results

We next use the identification results to evaluate the most common way that practitioners estimate continuous DiD designs, which is to run a TWFE regression that includes time fixed effects ( $\theta_t$ ), unit fixed effects ( $\eta_i$ ), and the interaction of a dummy for the post-treatment period ( $Post_t$ ) with a variable that measures unit  $i$ 's dose or treatment intensity,  $D_i$ :

$$Y_{i,t} = \theta_t + \eta_i + \beta^{twfe} D_i \cdot Post_t + v_{i,t}. \quad (1.1)$$

This TWFE specification is clearly motivated by DiD setups with two periods and two treatment groups, though many prominent textbooks recommend using it in more general setups (e.g., Cameron and Trivedi, 2005, Angrist and Pischke, 2008, and Wooldridge, 2010). There are several ways to interpret  $\beta^{twfe}$ , each corresponding to a different type of causal parameter. We decompose it in terms of level effects, scaled level effects, causal responses, and scaled high-versus-low ( $2 \times 2$ ) effects. Each decomposition is a weighted integral of dose-specific causal parameters, and none provide a clear causal and policy-relevant interpretation of  $\beta^{twfe}$ , at least not when treatment effects are allowed to vary across doses and/or groups.

Our impression is that empirical researchers typically interpret  $\beta^{twfe}$  in three main (and related) ways, implicitly relying on different building blocks. First,  $\beta^{twfe}$  is often directly interpreted as a causal response parameter; that is, how much the outcome causally increases on average when the treatment increases by one unit. This is the causal version of how regression coefficients are often taught to be interpreted in introductory econometrics classes. Second, it is common to pick a representative value for  $d$ , to report  $d \times \beta^{twfe}$ , and interpret this quantity as  $ATT(d)$ . This is the main interpretation provided in Acemoglu and Finkelstein (2008): “Given that the average hospital has a



38 percent Medicare share prior to PPS, this estimate [i.e., of  $\beta^{twfe}$ , here equal to 1.129] suggests that in its first 3 years, the introduction of PPS was associated with an increase in the depreciation share of about 0.42 ( $\approx 1.129 \times 0.38$ ) for the average hospital.” Rearranging this expression shows that under this interpretation  $\beta^{twfe} = ATT(d|d)/d$ , which relates  $\beta^{twfe}$  to a scaled level effect. Third, it is common to take two different representative values of the dose,  $d_1$  and  $d_2$ —a common choice is the 25th percentiles and 75th percentiles of the dose—and interpret  $\beta^{twfe}$  as the average causal response of moving from dose  $d_1$  to dose  $d_2$  scaled by the distance between  $d_1$  and  $d_2$ ; this is a scaled  $2 \times 2$  effect. We aim to assess whether such types of interpretations are justified and under which conditions.

# Interpreting TWFE

**Theorem 3.4.** Under Assumptions 1, 2(a), 3, and 4,  $\beta^{twfe}$  can be decomposed in the following ways:

(a) Causal Response Decomposition:

$$\beta^{twfe} = \int_{d_L}^{d_U} w_1^{acr}(l) \left( ACRT(l|l) + \underbrace{\frac{\partial ATT(l|h)}{\partial h} \Big|_{h=l}}_{\text{selection bias}} \right) dl + w_0^{acr} \frac{ATT(d_L|d_L)}{d_L}$$

where the weights are always positive and integrate to 1.

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<sup>10</sup>The decompositions in the main text integrate over all possible doses. In Appendix SC.2 in the Supplementary Appendix, we additionally consider scaled level and scaled  $2 \times 2$  decompositions for particular, fixed values of the dose. There we show that, even under strong parallel trends,  $\beta^{twfe}$  can be (possibly much) different from these parameters when there is treatment effect heterogeneity due to (i) different weighting schemes (similar to the differences that we point out in this section) and (ii)  $\beta^{twfe}$  being dependent on causal responses at other doses.

(b) *Levels Decomposition:*

$$\beta^{twfe} = \int_{d_L}^{d_U} w_1^{lev}(l) ATT(l|l) dl,$$

where  $w_1^{lev}(l) \leq 0$  for  $l \leq \mathbb{E}[D]$ , and  $\int_{d_L}^{d_U} w_1^{lev}(l) dl + w_0^{lev} = 0$ .

(c) *Scaled Levels Decomposition:*

$$\beta^{twfe} = \int_{d_L}^{d_U} w^s(l) \frac{ATT(l|l)}{l} dl,$$

where  $w^s(l) \leq 0$  for  $l \leq \mathbb{E}[D]$ , and  $\int_{d_L}^{d_U} w^s(l) dl = 1$ .

(d) *Scaled  $2 \times 2$  Decomposition*

$$\begin{aligned} \beta^{twfe} = & \int_{d_L}^{d_U} \int_{\mathcal{D}, h>l} w_1^{2 \times 2}(l, h) \left( \underbrace{\frac{\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h]}{h-l}}_{causal\ response} + \underbrace{\frac{ATT(h|h) - ATT(l|h)}{h-l}}_{selection\ bias} \right) dh dl \\ & + \int_{d_L}^{d_U} w_0^{2 \times 2}(h) \frac{ATT(h|h)}{h} dl, \end{aligned}$$

where the weights  $w_1^{2 \times 2}$  and  $w_0^{2 \times 2}$  are always positive and integrate to 1.

If one imposes Assumption 5 instead of Assumption 4, then the selection bias terms from Part (a) and Part (d) become zero, and the remainder of the decompositions remain true, except one needs to replace  $ACRT(l|h)$  with  $ACR(l)$  in Part (a),  $ATT(l|h)$  with  $ATE(l)$  in Parts (b), (c) and (d), and  $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h]$  with  $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)]$  in Part (d).

Table 1: TWFE Decomposition Weights

Decomposition	$D > 0$ Weights	$D = 0$ Weights
Causal response	$w_1^{\text{acr}}(l) = \frac{(\mathbb{E}[D D \geq l] - \mathbb{E}[D])\mathbb{P}(D \geq l)}{\text{Var}(D)}$	$w_0^{\text{acr}} = \frac{(\mathbb{E}[D D > 0] - \mathbb{E}[D])\mathbb{P}(D > 0)d_L}{\text{Var}(D)}$
Levels	$w_1^{\text{lev}}(l) = \frac{(l - \mathbb{E}[D])}{\text{Var}(D)} f_D(l)$	$w_0^{\text{lev}} = -\frac{\mathbb{E}[D]\mathbb{P}(D = 0)}{\text{Var}(D)}$
Scaled levels	$w^*(l) = l \frac{(l - \mathbb{E}[D])}{\text{Var}(D)} f_D(l)$	
Scaled $2 \times 2$	$w_1^{2 \times 2}(l, h) = \frac{(h - l)^2 f_D(h)f_D(l)}{\text{Var}(D)}$	$w_0^{2 \times 2}(h) = \frac{h^2 f_D(h)\mathbb{P}(D = 0)}{\text{Var}(D)}$

Notes: The table provides the formulas for the weights used in the decompositions of  $\beta^{\text{twfe}}$  provided in this section.

# Understanding Decomposition Results

- The pattern from decomposition shows distinct impacts of parameter types.
- **Level-effect parameters** (parts b and c):
  - $\beta_{twfe}$  is not influenced by selection bias.
  - Includes negative weights.
- **Comparative doses parameters** (parts a and d):
  - $\beta_{twfe}$  carries positive weights.
  - Encounters selection bias under parallel trends.

# Addressing Selection Bias and Weighting Schemes

- Parametric linearity restrictions may overlook weighting scheme issues inherent in TWFE regression.
- These restrictions do not resolve selection bias problems.
- Next, we explore:
  - Alternative estimators to TWFE that adjust the weighting scheme.
  - These alternatives do not rely on the stringent linearity assumption.
  - Selection bias issues persist and require different solutions.

# Roadmap

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Dx2 and DxT

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Selection bias

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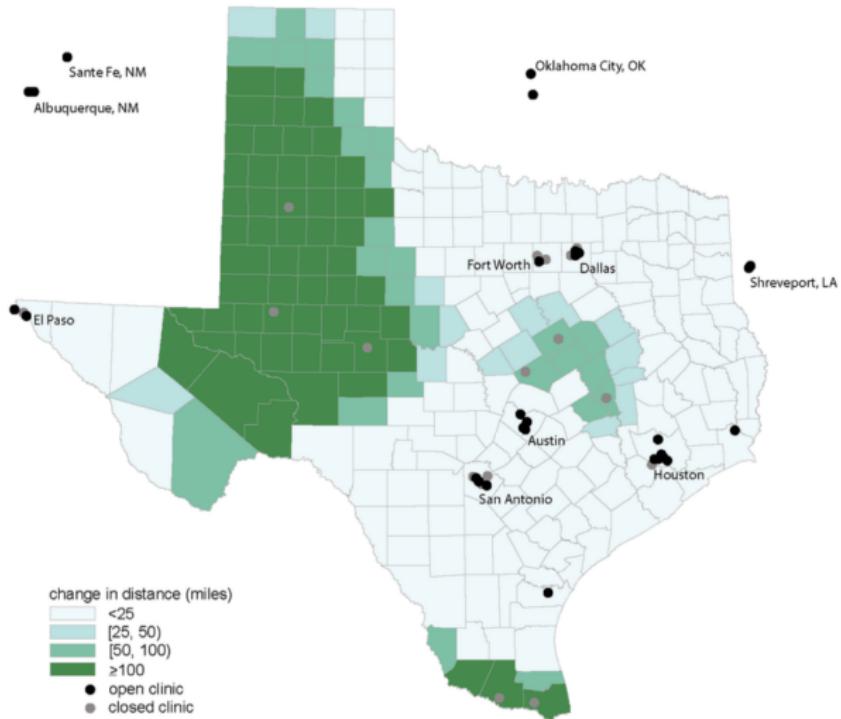
Estimation and an Example

## Institutional Background – Texas House Bill 2 (HB2)

- In July 2013, Texas passed HB2, imposing new regulations on abortion providers.
- Two provisions had major impact:
  1. Required physicians to have admitting privileges at a hospital within 30 miles of the clinic.
  2. Required clinics to meet ambulatory surgical center (ASC) standards.
- The admitting privileges rule went into effect on November 1, 2013, forcing nearly half of the state's clinics to close.
- This caused large increases in travel distance and congestion at remaining clinics.

# Clinic Access Map

Figure 2: Change in distance to the nearest abortion clinic, Q2 2013 to Q4 2013



# Our Poisson Fixed Effects Model

We estimate a generalized DiD using Poisson pseudo-likelihood:

$$\mathbb{E}[Y_{ct} \mid D_{ct}, X_{ct}, \alpha_c, \lambda_t] = \exp(\beta D_{ct} + \alpha_c + \lambda_t + \gamma X_{ct})$$

- $Y_{ct}$ : Count of abortions per county-year (age-standardized).
- $D_{ct}$ : Distance bin indicators and average service population.
- $\alpha_c, \lambda_t$ : County and year fixed effects.
- $X_{ct}$ : Controls (demographics, unemployment, family planning access).

# Main Results

## **Key Findings (Table 2, Column 1):**

- 50–100 miles → **16% reduction** in abortion rates
- 100–150 miles → **28% reduction**
- >200 miles → **44% reduction**
- +100,000 in service population → **7% reduction**

## **Interpretation:**

- Both distance and clinic congestion significantly reduce access.
- Effects are non-linear—biggest impact occurs at shorter distances.
- Congestion explains more than half the overall effect of clinic closures.

## Estimating $\text{ATT}(d \mid d)$ from Clinic Closures

Now we want to estimate the causal effect of clinic closures on abortion rates, allowing that effect to vary with dose  $d$  – where dose = distance to nearest clinic.

We'll estimate a **dose-specific DiD**, defined as:

$$\widehat{\text{ATT}}(d \mid d) = \mathbb{E}[\Delta Y_i \mid D_i = d] - \mathbb{E}[\Delta Y_i \mid D_i = 0]$$

- The second term is the average change in abortion rates in counties with no clinic closures.
- The first term is the average change in counties with exposure  $D_i = d$ .
- We're not using Poisson here – we'll estimate this directly using nonparametric methods from CGBS (2024).

## Step 1: Binning the Dose (Distance)

A simple approach is to group counties by their post-HB2 distance to the nearest open clinic.

Within each bin, we compute the average change in abortion rates:

$$\widehat{\text{ATT}}(\text{bin}) = \mathbb{E}[\Delta Y_i \mid D_i \in \text{bin}] - \mathbb{E}[\Delta Y_i \mid D_i = 0]$$

- This gives us a discrete approximation of  $\text{ATT}(d-d)$  at several key exposure levels.
- We would then show those visually

## Estimating ATT( $d \mid d$ ) via Binned DiD

Let's start with a simple idea: divide counties by their **dose** – i.e., the post-HB2 distance to the nearest clinic.

For each bin of distance  $B$ , estimate the causal effect of clinic closure exposure on abortion rates:

$$\widehat{\text{ATT}}(B \mid B) = \mathbb{E}[\Delta Y_i \mid D_i \in B] - \mathbb{E}[\Delta Y_i \mid D_i = 0]$$

- $\Delta Y_i = Y_{i1} - Y_{i0}$  is the change in abortion rates in county  $i$ .
- Control group: counties with  $D_i = 0$  (no change in access).
- Treated group: counties with  $D_i$  in bin  $B$ .

# Step-by-Step: What Do We Do?

## **Step 1: Define bins of distance**

- Bin 1: 0–50 miles
- Bin 2: 50–100 miles
- Bin 3: 100–150 miles
- Bin 4: 150–200 miles
- Bin 5: >200 miles

## **Step 2: For each bin, construct a dummy:**

$$\text{Treated}_i = \mathbf{1}\{D_i \in \text{Bin } B\}$$

## **Step 3: Subset the data to:**

- Units with  $D_i \in B$  (treated) and
- Units with  $D_i = 0$  (control)

# Step-by-Step: Estimation per Bin

## Step 4: Estimate the DiD

$$\Delta Y_i = \alpha + \beta_B \cdot \text{Treated}_i + \varepsilon_i$$

- $\Delta Y_i$  is the change in outcome (e.g., abortion rate) from pre- to post-period.
- $\text{Treated}_i = 1$  if  $D_i$  is in bin  $B$ , 0 if  $D_i = 0$ .
- $\beta_B$  estimates  $\text{ATT}(B | B)$  for bin  $B$ .

**Repeat this process** for each bin to get a full set of ATT estimates across the dose distribution.

# Why This Works (and When It Doesn't)

## **Why this is valid:**

- Relies only on standard parallel trends: untreated units represent the counterfactual trend.
- No functional form assumptions on dose response.

## **Limitations:**

- Requires sufficient untreated and treated units per bin.
- Bin definitions can affect results.
- Flat effects within bins – no curvature captured.

# Binned Estimation: Pros and Cons

## **Advantages:**

- Easy to explain and plot: good for papers and policy presentations.
- Lets us see effect heterogeneity across dose levels.

## **Disadvantages:**

- Estimates are “flat” within each bin.
- Shape depends on bin cutpoints – can be misleading if dose-response is smooth.

## Alternative Method: Smoothed Estimation of $\text{ATT}(d \mid d)$

As an alternative to binning, we use a smoothed estimator of  $\text{ATT}(d \mid d)$ .

This method is based on work by Chen, Christensen, and Kankanala (2023), and adapted to the difference-in-differences setting by Callaway, Goodman-Bacon, and Sant'Anna (2024).

## Alternative Method: Smoothed Estimation of ATT( $d \mid d$ )

### Key idea:

- Smoothly estimate  $\mathbb{E}[\Delta Y_i \mid D_i = d]$  across dose levels using only treated units ( $D_i > 0$ )
- Subtract off  $\mathbb{E}[\Delta Y_i \mid D_i = 0]$  from untreated counties

$$\widehat{\text{ATT}}(d \mid d) = \widehat{\mathbb{E}}[\Delta Y_i \mid D_i = d] - \widehat{\mathbb{E}}[\Delta Y_i \mid D_i = 0]$$

This gives a continuous estimate of treatment effects across the dose distribution.

# Intuition and Implementation with condid

## What the method does (intuitively):

1. Calculate  $\Delta Y_i = Y_{i,\text{post}} - Y_{i,\text{pre}}$  for each county
2. For treated units ( $D_i > 0$ ), regress  $\Delta Y_i$  on a flexible function of  $D_i$  (e.g., B-splines)
3. Predict  $\mathbb{E}[\Delta Y_i | D_i = d]$  across the observed dose range
4. Subtract the average change in untreated units ( $D_i = 0$ )

## Intuition and Implementation with `condid`

We'll implement this using the `condid` package in R, developed by Brantly Callaway.

- Includes built-in routines for smoothed ATT( $d | d$ ) estimation
- **Currently does not support covariates**, but is actively being expanded

# Implementation and Parameters

## Treatment Assignment:

- $D_i$  = distance increase from 2013 to 2014 if  $> 10$  miles, 0 otherwise
- $G_i$  = 2014 if treated, 0 if never-treated (control)
- 126 counties treated, 128 counties control

# Implementation and Parameters

## Key Parameters:

$$ATT(d|d) = \mathbb{E}[Y_t(d) - Y_t(0)|D = d] \quad (1)$$

$$ACRT(d|d) = \frac{\partial ATT(l|d)}{\partial l} \Big|_{l=d} \quad (2)$$

$$ACRT^o = \mathbb{E}[ACRT(D|D)|D > 0] \quad (3)$$

# Implementation and Parameters

## **Interpretation:**

- $ATT(d|d)$ : Effect of dose  $d$  vs. no treatment for counties that got dose  $d$
- $ACRT(d|d)$ : Dose-response curve connecting all ATT points (under strong PT)
- $ACRT^o$ : Overall average marginal effect (single coefficient in output)
- Allows heterogeneous effects across dose levels

**Identification:** Parallel trends between treated and never-treated counties

## Summary and Thank you!

- Remember: it's very helpful to think of diff-in-diff as  $2 \times 2$  and then aggregations – even differential timing
- Think about who you're matching the treatment group too (both which units and covariates)
- Follow checklists! Focus on designing this before moving into economic narratives
- And if you in the end find parallel trends to be implausible consider synthetic control methods!