CodeChella Madrid 2024

Roadmap

- Continuous DiD
  - Motivation
  - Misspecified Regressions
  - Average causal response functions
  - Identification
  - Selection bias
  - Interpreting TWFE

Continuous DiD

- A very common panel model will use a treatment variable which is continuous, not binary
- 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

Quotes

"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)

Continuous DiD

Hani Mansour @hnmansour · Dec 14, 2020
I remember seeing a paper about estimating an event study with a continuous variable but can't seem to track it. Any leads #EconTwitter @causalinf @agoodmanbacon?
7 8 27

David Burgherr @D_Burgherr · Mar 25, 2020
On this point, I am very curious how much the issues you point out with DD designs -- variance-weighting of treatment effects (TE) and bias in the case of time-varying TE -- matter for continuous treatment variables. Do you have any take or reference on that?

Thanks in advance!
2

Khoa Vu @KhoaVuUmn · Nov 16, 2020
#EconTwitter Question on DiD: I'm looking for a reference on what to do when the treatment variable is continuous and you suspect that the effect is nonlinear, e.g. medium exposure might have bigger effect than high exposure.
4 7 42

Ben Glasner @BenGlasner · Oct 29, 2020
Any recs on DID packages in r for multiple treatment periods with different timings and continuous treatment values? Think minimum wage changes over time? Something to compare TWFE against... #econtwitter
2

Michelle Spiegel @michspieg · Apr 22, 2020
I am writing a DiD paper with a continuous treatment. Any paper recommendations to help think about statistical power in this context? #EconTwitter #soctwitter #AcademicTwitter

Kait Sims @kaitmsims · Aug 25, 2020
#EconTwitter recommendations for event study/DiD papers with staggered treatment time, continuous treatment intensity, and where treatment can turn on and off more than once for the same individual?

3 4 4

Jason Baron @JasonBaron4 · Apr 21, 2020
I know there have been previous threads on the most recent DID papers, but does anyone know if there are any recent methodological papers specifically looking at DID implementation with a continuous treatment variable? @causalinf @jondr44
4 9 37

Adam Roberts @adamn_roberts · Mar 28
Question for DiD experts:
Is there a heterogenous treatment effects solution that works for continuous treatments? I'm specifically thinking about early childhood intervention papers that define treatment as "age 0-5" exposure to something like county food stamps availability.
1 7

Adam Roberts @adamn_roberts · Mar 28
This type of treatment has staggered timing and enough heterogeneity to make TWFE a poor approach but after diving into the new DiD literature I'm struggling to figure out the "correct" approach with a continuous treatment variable. Any thoughts @causalinf @Andrew__Baker ?
4

Nicholas Reynolds @nick_reynolds88 · Apr 28
Does anyone know of papers deriving what TWFE with continuous treatment and allowing for heterogenous treatment effects estimates?

My intuition is same "problems" found for staggered diff-in-diff would exist here ... but notation would explode so no one has written it out?
1 2 2

Peter Bergman @peterbergman_ · May 11, 2020
Seems like "dosage"/"intensity" diff-in-diff--where there aren't 2 groups but a continuous measure w/ varying intensity of treatment--requires potentially stronger identifying assumptions than DID for 2 groups. Is this discussed in any of the recent DID lit updates? cc @causalinf
6 5 47

Nick Hagerty @hagertynw · Mar 29
Conceptually it's not that distinct right? We're still trying to identify off similar shocks in different places at different times. I thought the main difference is that our variables are continuous treatments -- algebra is harder but papers prob. coming in next couple years
3 6

Michael Wiebe @michael_wiebe · Feb 9
Who's writing the @agoodmanbacon paper on diff-in-diff with a continuous treatment variable (instead of binary)?

#EconTwitter
1

Davide Proserpio @dade_us · Apr 12, 2020
Looking for recommendations about DD papers where the treatment is continuous, thanks! #EconTwitter
6 1 8

Overview

1. What of what we have learned carries forward to the continuous case?
2. Some of the problems with continuous (maybe most) don't even have to do with differential timing, so I'm not going to cover it

Recommended steps of causal projects

1. Define the parameter we want (“ATT”),
2. Ask what what beliefs do you need (“identification”), and
3. Build cranks that produce the correct numbers (“estimator”)

People often skip 1 and 2 and go straight to 3 and run regressions then go back and assume exogeneity (step 2), and hope that the estimates are weighted averages of individual treatment effects (1), but that is not guaranteed

Dangers of skipping 1 and 2

- TWFE was arguably a case where people skipped 1 and 2 and went straight to 3
- We now know that the “constant treatment effect” static specification does not recover the ATT under parallel trends, but the VWATT and requires no dynamics
- We can see this too in simulations even with matching and regression – defining the parameter ahead of time then clearly indicates what assumptions to make, pushing into specifications
- Let’s look at this now

Data Generating Process

- Covariate imbalance
- Heterogenous treatment effects with respect to covariates
- Linear data generating process
- Question: How do we estimate the ATE vs the ATT?

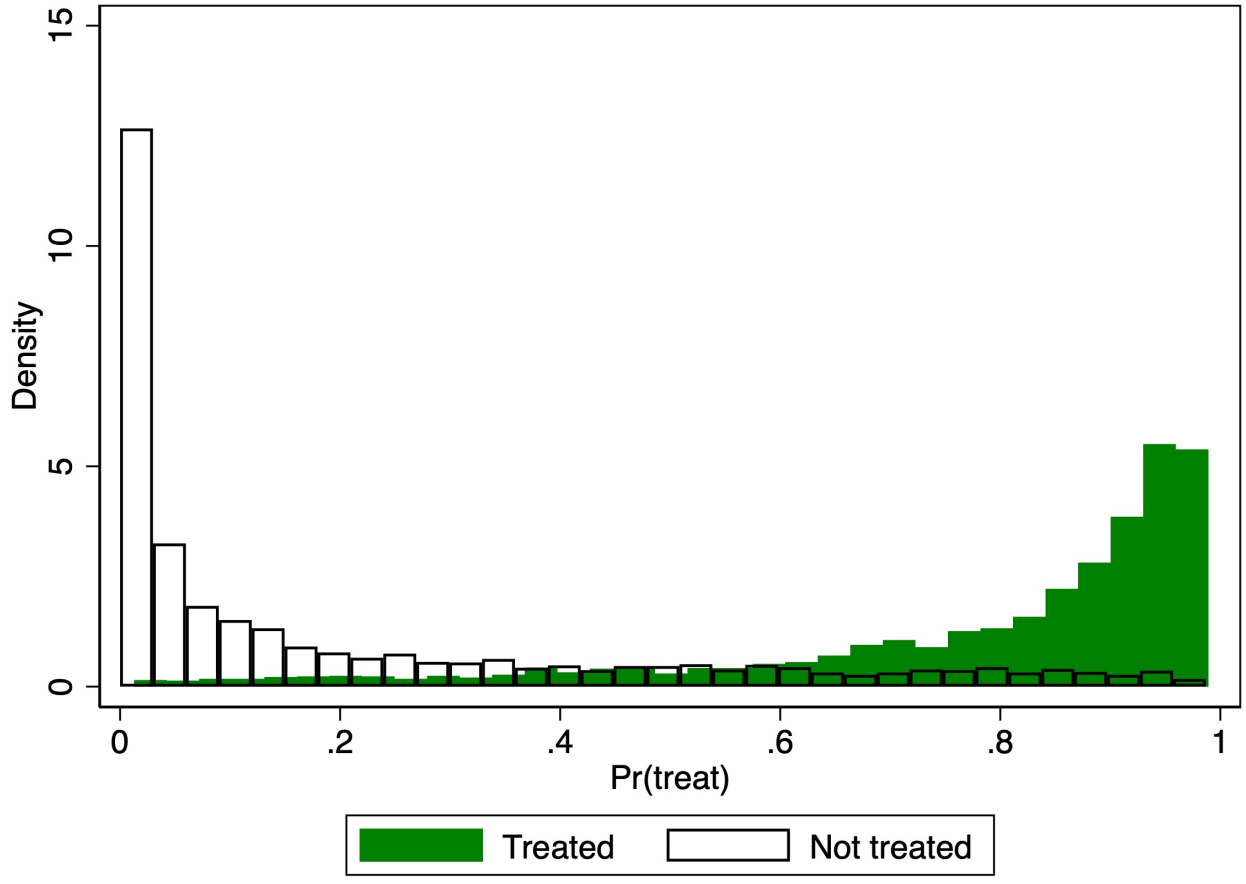
Data Generating Process

- Age is generated from a normal distribution:
  - → Treatment group: mean 25, standard deviation 2.5
  - → Control group: mean 30, standard deviation 3
- GPA is generated from a normal distribution:
  - → Treatment group: mean 1.76, standard deviation 0.5
  - → Control group: mean 2.3, standard deviation 0.75
- Age and GPA are centered around their respective means
- Squared terms and interaction terms are generated:
  - → Age squared (age˙sq), GPA squared (gpa˙sq)
  - → Interaction between age and GPA (interaction)

Data Generating Process

- Outcome variables are generated:
  - → No treatment (y0): $15000 + 10.25 \cdot$ age $- 10.5 \cdot$ age˙sq $+ 1000 \cdot$ gpa $- 10.5 \cdot$ gpa˙sq $+ 500 \cdot$ interaction $+ \epsilon$
  - → Treatment (y1): $y0 + 2500 + 100 \cdot$ age $+ 1000 \cdot$ gpa
  - → Treatment effect (delta): $y1 - y0$
- Average treatment effect (ATE) is estimated at 2500
- Average treatment effect on the treated (ATT) is estimated at 1971

Covariate imbalance (expressed as propensity score)



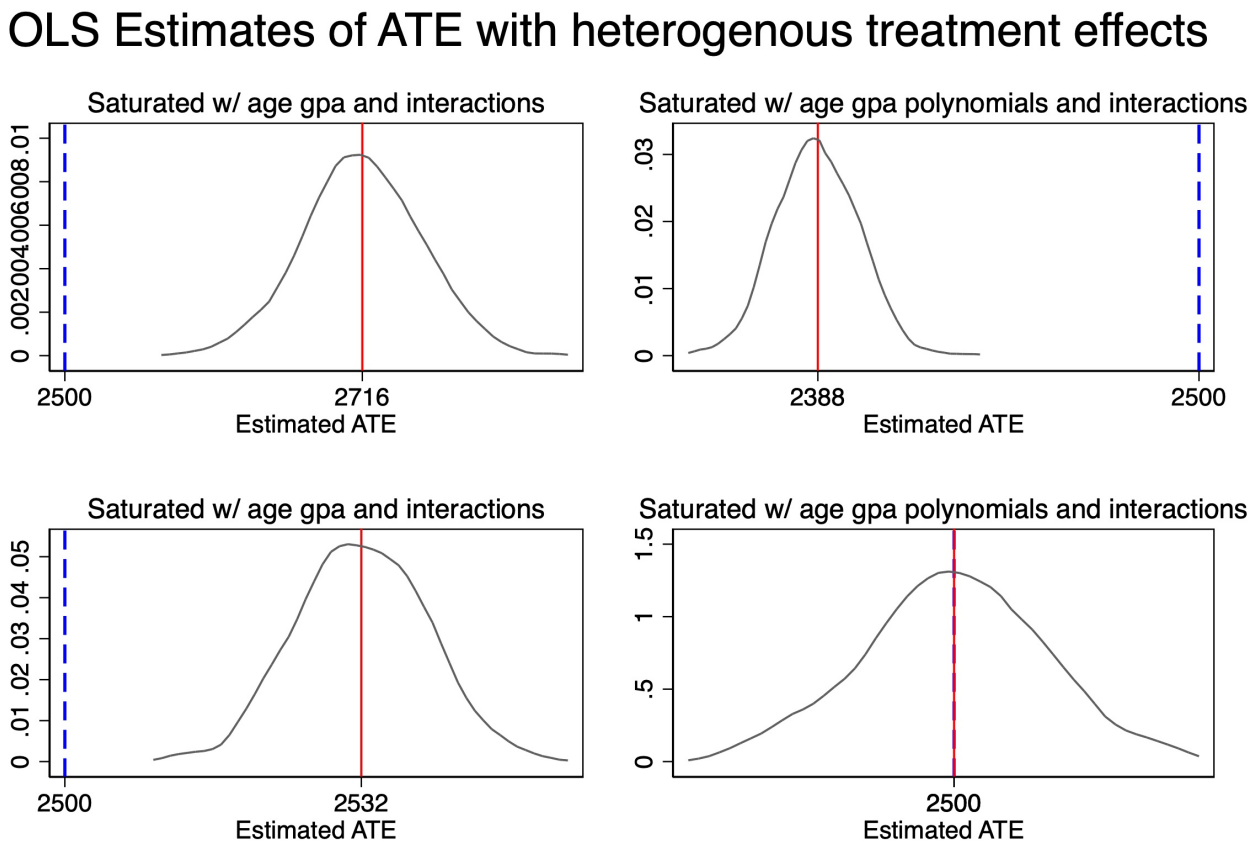
Regression specifications 1 and 2

We will look at several different specifications. These first two are standard ways of incorporating covariates. You enter them in linearly as controls.

$$\begin{aligned} \text{earnings} &= \beta_0 + \beta_1 \text{treat} + \beta_2 \text{age} + \beta_3 \text{gpa} + \epsilon && (1) \\ \text{earnings} &= \beta_0 + \beta_1 \text{treat} + \beta_2 \text{age} + \beta_3 \text{age\_sq} + \beta_4 \text{gpa} \\ &\quad + \beta_5 \text{gpa\_sq} + \beta_6 (\text{gpa} \times \text{age}) + \epsilon && (2) \end{aligned}$$

Interpretation focuses on $\widehat{\beta_1}$. But what does it mean? Look at top two. Remember ATE is 2500.

ATE estimates across different specifications



Four kernel density plots of estimated coefficients from 1000 simulations

ATT Calculation: Regression 3

Saturated regressions: interact treatment dummy with all covariates.

- Regression Specification:

$$\begin{aligned}\text{earnings} \quad = \quad & \beta_0 + \beta_{1t}\text{treat} + \beta_{2a}\text{age} + \beta_{3g}\text{gpa} + \beta_{4ta}(\text{treat} \times \text{age}) + \beta_{5tg}(\text{treat} \times \text{gpa}) + \beta_{6age}(\text{age} \times \text{gpa}) \\ & + \beta_{6tag}(\text{treat} \times \text{age} \times \text{gpa}) + \epsilon\end{aligned}$$

  Estimated ATE is the coefficient, $\widehat{\beta_{1t}}$, but how do we get the estimated ATT?

- Calculating ATT:

$$\text{ATT}_3 = \beta_{1t} + \beta_{4ta} \cdot \bar{\text{age}} + \beta_{5tg} \cdot \bar{\text{gpa}} + \beta_{6tag} \cdot \bar{\text{age}} \cdot \bar{\text{gpa}}$$

  using the means of all covariates

ATT Calculation: Regression 4

Saturated regressions: interact it with covariates, higher order polynomials, and all interactions

- Regression Specification:

$$\begin{aligned}\text{earnings} = \; & \beta_0 + \beta_{1t}\text{treat} + \beta_{2a}\text{age} + \beta_{3a_sq}\text{age}^{\cdot}\text{sq} + \beta_{4g}\text{gpa} + \beta_{5g_sq}\text{gpa}^{\cdot}\text{sq} + \\ & \beta_{6ta}(\text{treat} \times \text{age}) + \beta_{7ta_sq}(\text{treat} \times \text{age}^{\cdot}\text{sq}) + \beta_{8tg}(\text{treat} \times \text{gpa}) + \\ & \beta_{9tg_sq}(\text{treat} \times \text{gpa}^{\cdot}\text{sq}) + \beta_{10ag}(\text{age} \times \text{gpa}) + \beta_{11a_sqg}(\text{age}^{\cdot}\text{sq} \times \text{gpa}) + \\ & \beta_{12ag_sq}(\text{age} \times \text{gpa}^{\cdot}\text{sq}) + \beta_{13a_sqg_sq}(\text{age}^{\cdot}\text{sq} \times \text{gpa}^{\cdot}\text{sq}) + \epsilon\end{aligned}$$

Estimated ATE is the coefficient, $\widehat{\beta_{1t}}$, but how do we get the estimated ATT?

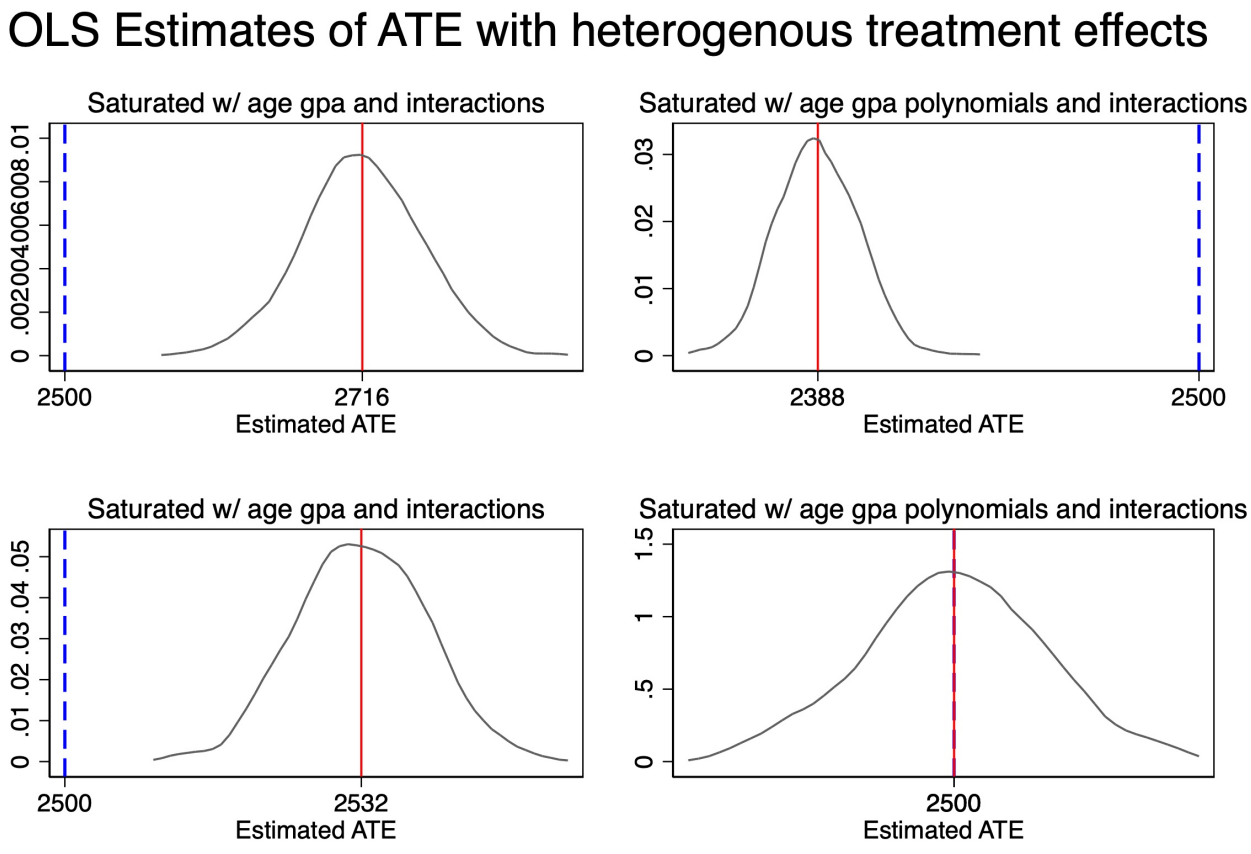
- Calculating ATT:

$$\begin{aligned}\text{earnings} = \; & \beta_0 + \beta_{1t}\text{treat} + \beta_{2a}\text{age} + \beta_{3a\_sq}\text{age\_sq} + \beta_{4g}\text{gpa} + \beta_{5g\_sq}\text{gpa\_sq} + \beta_{6ta}(\text{treat} \times \text{age}) \\ & + \beta_{7ta\_sq}(\text{treat} \times \text{age\_sq}) + \beta_{8tg}(\text{treat} \times \text{gpa}) + \beta_{9tg\_sq}(\text{treat} \times \text{gpa\_sq}) + \beta_{10ag}(\text{age} \times \text{gpa}) \\ & + \beta_{11a\_sqg}(\text{age\_sq} \times \text{gpa}) + \beta_{12ag\_sq}(\text{age} \times \text{gpa\_sq}) + \beta_{13a\_sqg\_sq}(\text{age\_sq} \times \text{gpa\_sq}) + \epsilon\end{aligned}$$

Interpretations

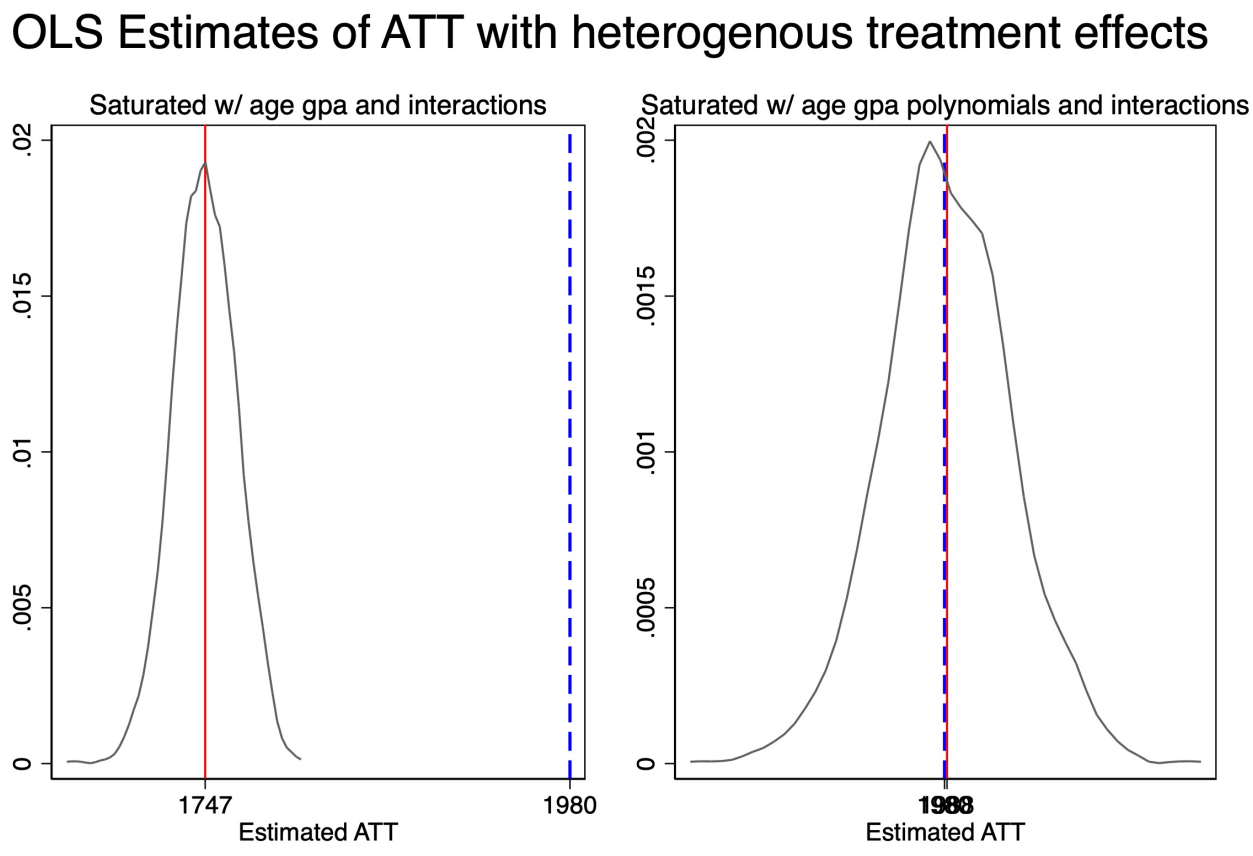
- ATE is 2500
- ATT is 1980
- Key point here: the same regression contains both parameters, but only when done correctly, and only when interpreted correctly

ATE estimates across different specifications



Four kernel density plots of estimated coefficients from 1000 simulations

ATT estimates across different specifications

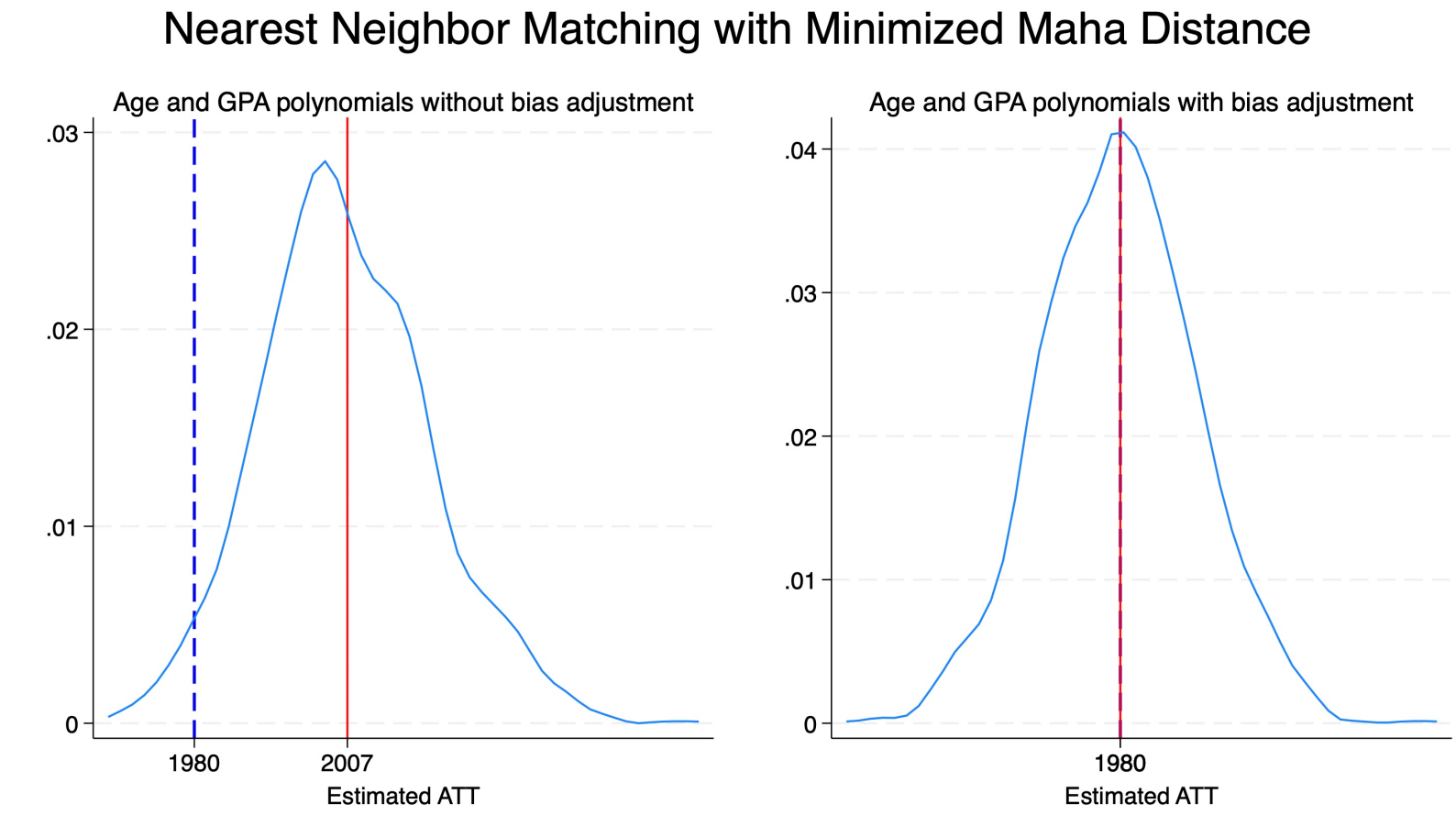


Two kernel density plots of estimated coefficients from two regressions and 1000 simulations

Matching by minimizing Maha distance metric

- Next I just estimated the ATT using a simpler model – nonparametric matching using Abadie and Imbens (2006;2011)
- It's biased when the matches aren't exact, but you can use regression adjustment to estimate the selection bias
- Similar to what augmented synth does
- Much less difficult syntax

ATT estimates across different matching specifications



Estimated ATT from 1000 simulations using nearest neighbor matching

Recommended steps of causal projects

1. Define the parameter we want (“ATT”),
2. Ask what what beliefs do you need (“identification”), and
3. Build cranks that produce the correct numbers (“estimator”)

See how when we skep 1 and 2 and go straight to 3, heterogenous treatment effects makes major problems for interpretation? It isn’t that regressions can’t recover parameters, but you have to saturate when you’re attempting to recover ATE or ATT, and even then it’s challenging to interpret – and programming, you often don’t have code that will do it for you.

Introducing a new causal parameter

- **ATT**: Extensive margin causal parameter. Do this versus don't do this.
- **Dose**: Intensive margin causal parameter. Do this much versus this much.

The dose causal parameter will be based on Angrist and Imbens (1995)

Parameters

Average treated on the treated

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

while the treatment, D, can be any amount, d, that amount is technically a particular dose. We raised the minimum wage, but we raised it to a particular wage.

Parameters

Average treated on the treated

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

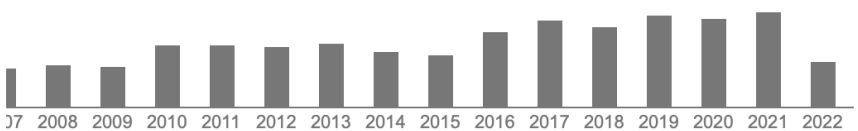
This is “the ATT of d for the groups that chose d dosage” which uses as its comparison no dose.

Average causal response function

guido imbens

Two-stage least squares estimation of average causal effects in models with variable treatment intensity

Authors	Joshua D Angrist, Guido W Imbens
Publication date	1995/6/1
Journal	Journal of the American statistical Association
Volume	90
Issue	430
Pages	431-442
Publisher	Taylor & Francis Group
Description	Two-stage least squares (TSLS) is widely used in econometrics to estimate parameters in systems of linear simultaneous equations and to solve problems of omitted-variables bias in single-equation estimation. We show here that TSLS can also be used to estimate the average causal effect of variable treatments such as drug dosage, hours of exam preparation, cigarette smoking, and years of schooling. The average causal effect in which we are interested is a conditional expectation of the difference between the outcomes of the treated and what these outcomes would have been in the absence of treatment. Given mild regularity assumptions, the probability limit of TSLS is a weighted average of per-unit average causal effects along the length of an appropriately defined causal response function. The weighting function is illustrated in an empirical example based on the relationship between schooling and earnings.
Total citations	Cited by 1372



Scholar articles

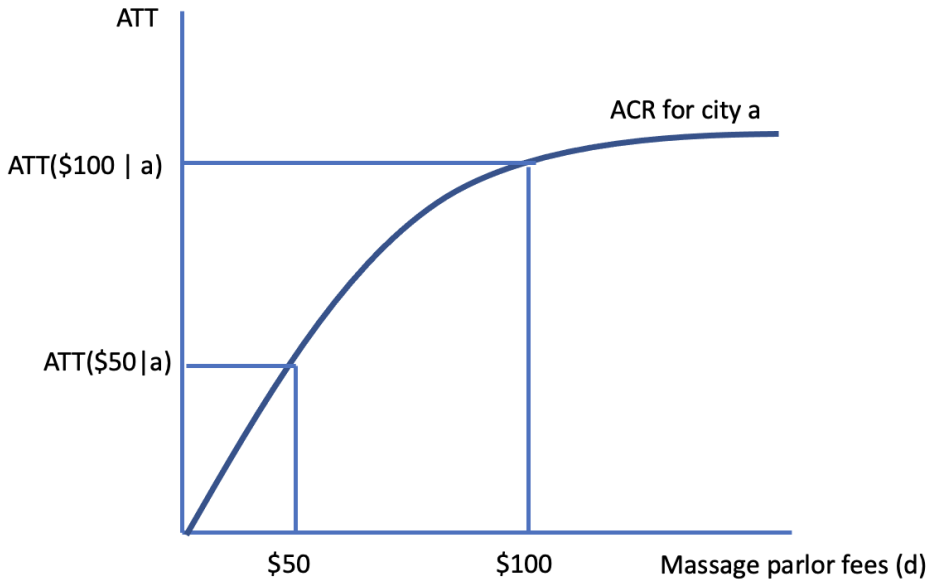
Two-stage least squares estimation of average causal effects in models with variable treatment intensity
JD Angrist, GW Imbens - Journal of the American statistical Association, 1995
Cited by 1358 Related articles All 14 versions

Average causal response with variable treatment intensity *
J Angrist, G Imbens - 1995
Cited by 16 Related articles All 10 versions

Angrist and Imbens 1995

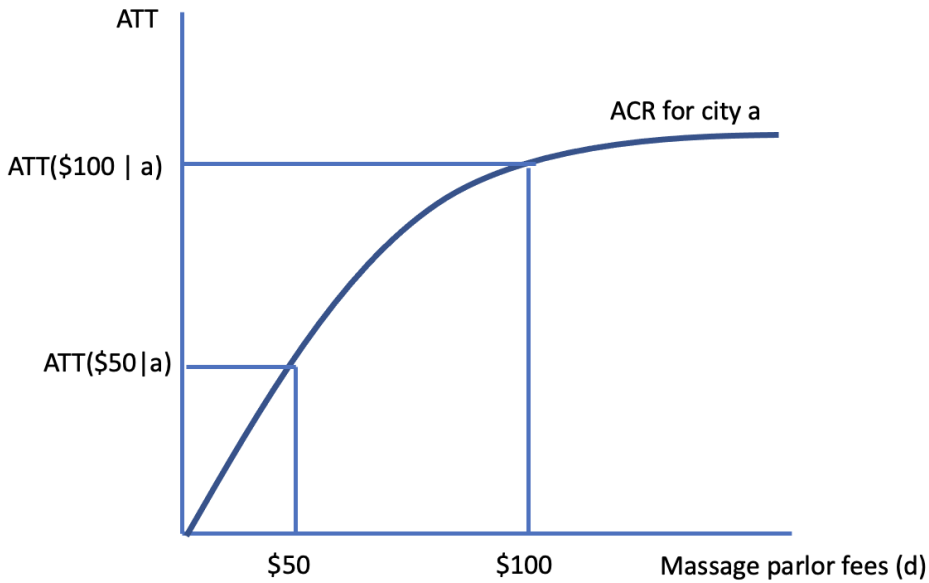
*"We refer to the parameter β as the **average causal response (ACR)**. This parameter captures a weighed average causal responses to a unit change in treatment, for those whose treatment status is affected by the instrument. …"*

ATT for a given dose



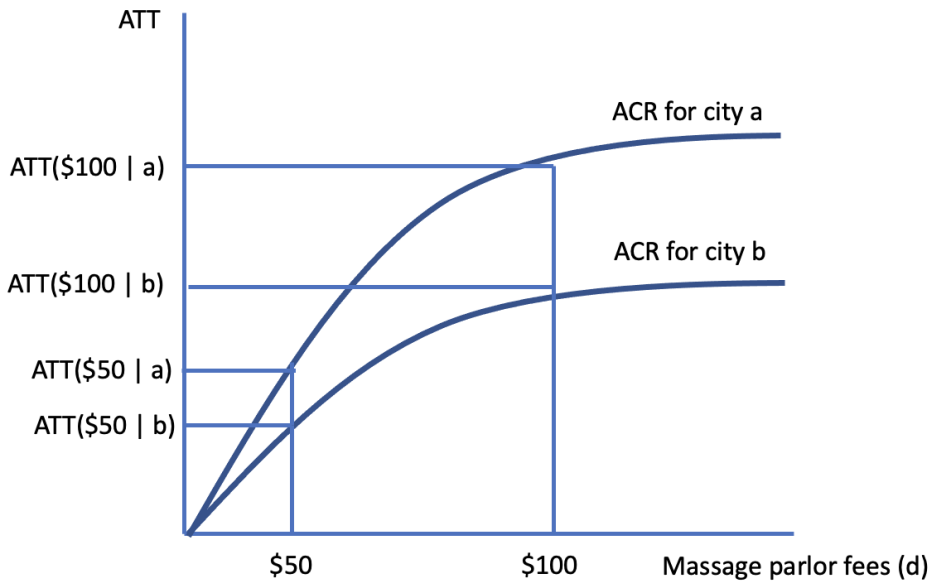
What is the effect of setting fees to $100 versus nothing at all? It's ATT($100|a) for this city.

ATT for a given dose



Assume city a did choose $d = \$100$. Then ATT($50|a) just means that that is its ATT *had* it chosen the lower level. The curve, in other words, is tracing out all average causal response for this city.

ATT for a given dose



What if everyone has different responses? In other words, city a has the higher curve than city b. Then there are several comparisons possible. What is the effect of $50 on outcomes for cities that actually chose $50 versus those than actually chose $100?

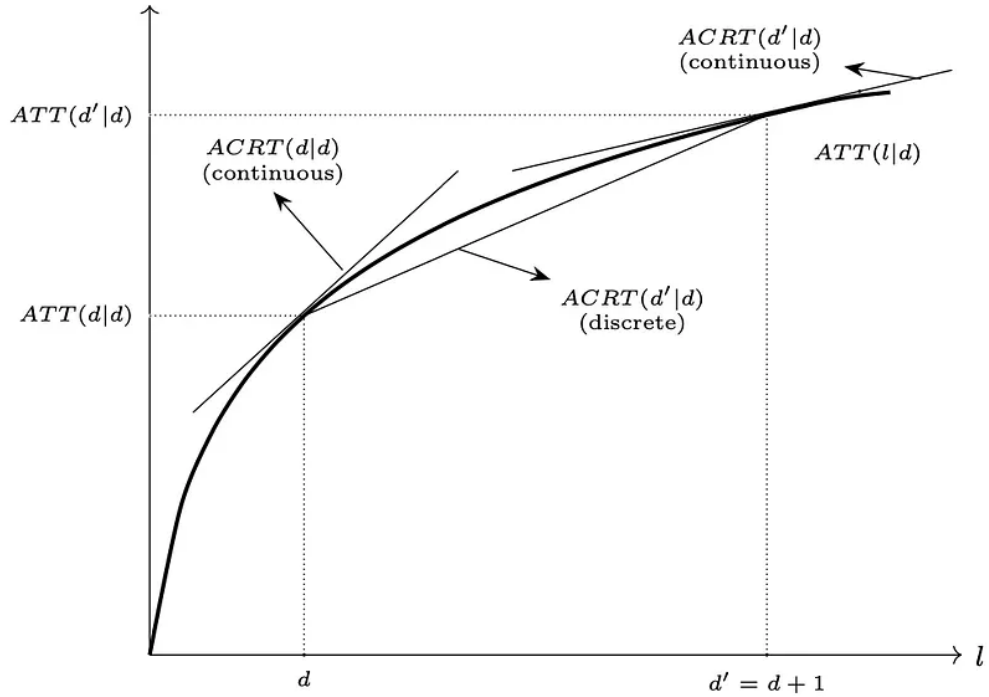
Parameters

Average treated on the treated

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

Notice that you are comparing any dose d to no treatment at all – sort of an extensive margin causal response, but that isn't the only causal concept we have. Elasticities are causal, demand curves are causal, but they aren't based on comparisons to nothing – they are intensive margin comparisons, local comparisons, adjacencies. Zero isn't the only counterfactual in other words.

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

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

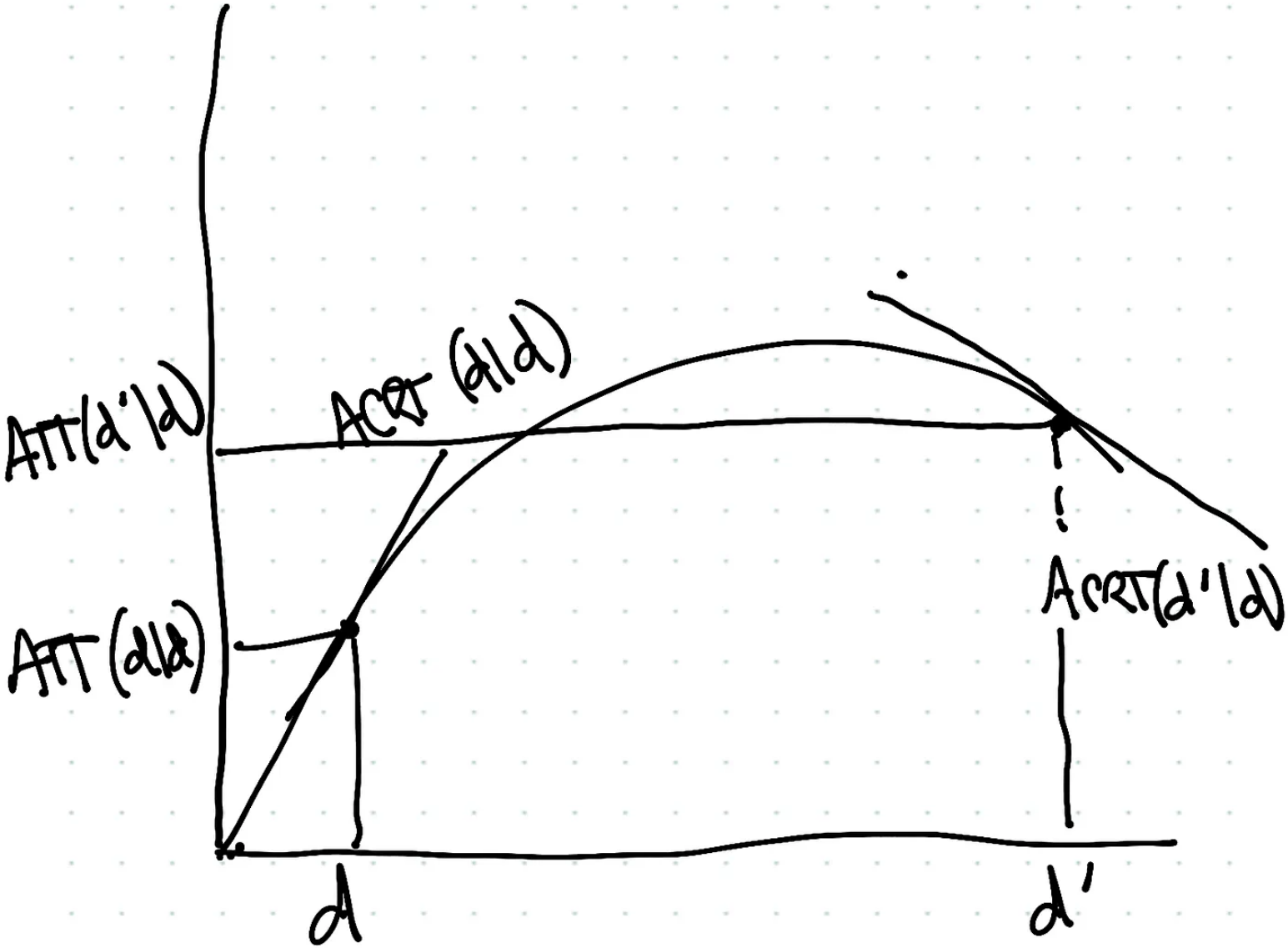
Derivation of ACRT

Average causal response parameters for absolutely continuous treatments are defined as

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

$ACRT(d|d)$ 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.

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

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

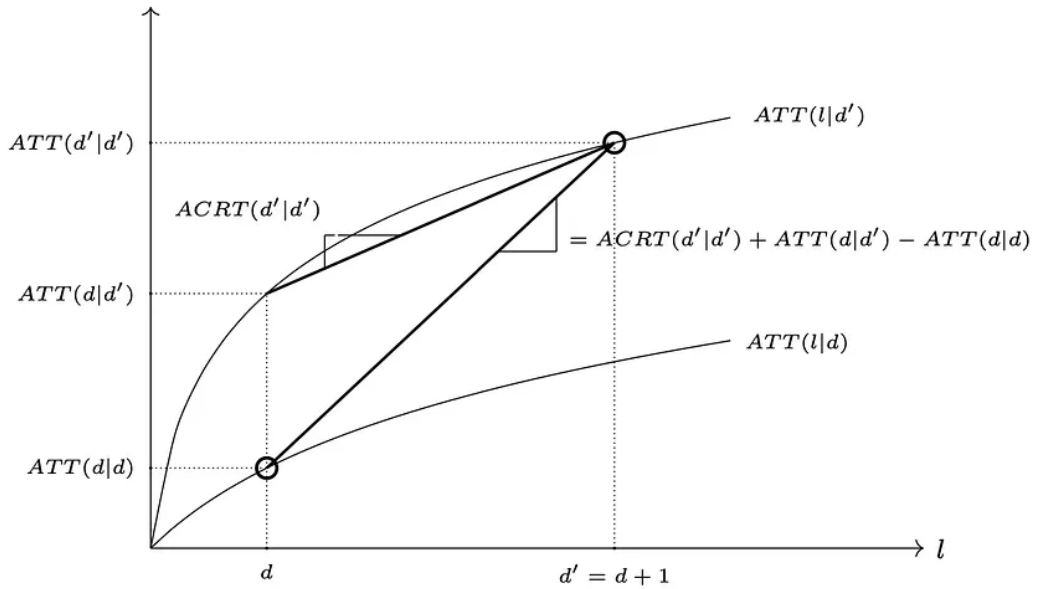
Part in blue is the movement along the average causal response function, the ACRT, and is causal. The part in red is selection bias.

Identifying ACRT

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

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 $d \in \mathcal{D}_+^c$,

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

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

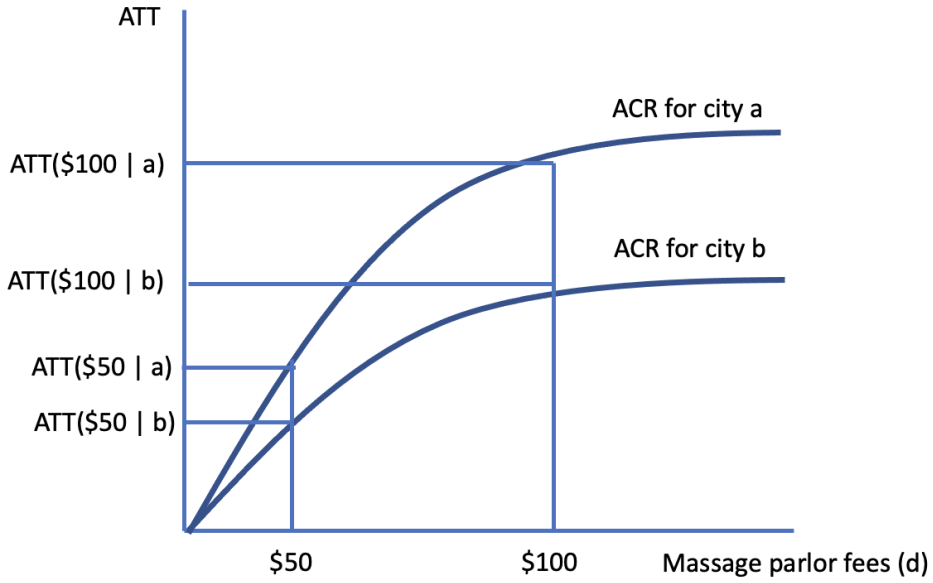
$$\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=l] = ATT(h|h) - ATT(l|l)$$

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

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

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

Causality and selection bias



Draw the ACRT 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: Average change in $E[Y^0]$ for the entire sample is the same as the d group
  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 (θ_t), unit fixed effects (η_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:

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

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 β^{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 (2×2) effects. Each decomposition is a weighted integral of dose-specific causal parameters, and none provide a clear causal and policy-relevant interpretation of β^{twfe}, at least not when treatment effects are allowed to vary across doses and/or groups.

Our impression is that empirical researchers typically interpret β^{twfe} in three main (and related) ways, implicitly relying on different building blocks. First, β^{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 β^{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 β^{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 β^{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 2×2 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, β^{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.

[10]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 2×2 decompositions for particular, fixed values of the dose. There we show that, even under strong parallel trends, β^{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) β^{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 $w_1^{lev}(l) \lesseqgtr 0$ *for* $l \lesseqgtr \mathbb{E}[D]$, *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 $w^{s}(l) \lesseqgtr 0$ *for* $l \lesseqgtr \mathbb{E}[D]$, *and* $\int_{d_L}^{d_U} w^{s}(l)\, dl = 1$.

(d) Scaled 2×2 *Decomposition*

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

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 $ACRT(l|l)$ *with* $ACR(l)$ *in Part (a),* $ATT(l|l)$ *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	$w_1^{\text{lev}}(l) = \frac{(l - \mathbb{E}[D])}{\text{Var}(D)} f_D(l)$	$w_0^{lev} = -\frac{\mathbb{E}[D]\mathbb{P}(D = 0)}{\text{Var}(D)}$
Scaled levels	$w^{\text{s}}(l) = l\frac{(l - \mathbb{E}[D])}{\text{Var}(D)} f_D(l)$	
Scaled 2×2	$w_1^{2\times2}(l, h) = \frac{(h - l)^2 f_D(h) f_D(l)}{\text{Var}(D)}$	$w_0^{2\times2}(h) = \frac{h^2 f_D(h)\mathbb{P}(D = 0)}{\text{Var}(D)}$

Notes: The table provides the formulas for the weights used in the decompositions of β^{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):
  - → β_{twfe} is not influenced by selection bias.
  - → Includes negative weights.
- **Comparative doses parameters** (parts a and d):
  - → β_{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.

TWFE has many meanings

- Today I just wanted to emphasize the basic principles though – define the parameter, figure out the assumptions, then build the estimators
- Just running regressions doesn't work once we have heterogenous treatment effects
- "Although strong parallel trends removes the selection bias, the weights attached to the causal parameters are still hard to interpret"
- New papers have developed new methods experimenting with different assumptions, but still waiting on the software