Difference-in-Differences with a Continuous Treatment*

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Abstract

This paper analyzes difference-in-differences designs with a continuous treatment. We show that treatment effect on the treated-type parameters can be identified under a generalized parallel trends assumption that is similar to the binary treatment setup. However, interpreting differences in these parameters across different values of the treatment can be particularly challenging due to selection bias that is not ruled out by the parallel trends assumption. We discuss alternative, typically stronger, assumptions that alleviate these challenges. We also provide a variety of treatment effect decomposition results, highlighting that parameters associated with popular linear two-way fixed-effect (TWFE) specifications can be hard to interpret, *even* when there are only two time periods. We introduce alternative estimation procedures that do not suffer from these drawbacks and show in an application that they can lead to different conclusions.

JEL Codes: C14, C21, C23

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1 Introduction

The canonical difference-in-differences (DiD) research design compares outcomes before and after treatment started (difference one), between treated and untreated groups (difference two). However, in many DiD applications, the treatment does not simply "turn on", it has a "dose" or operates with varying intensity. Pollution dissipates across space, affecting locations near its source more severely than faraway locations. Localities spend different amounts on public goods and services, or set different minimum wages. Students choose how long to stay in school.

Continuous treatments can offer advantages over binary ones.[1] Variation in intensity makes it possible to evaluate treatments that all units receive. A clear "dose-response" relationship between outcomes and treatment intensity can bolster the case for a causal interpretation or test a theoretical prediction.[2] Finally, we may care more about the effect of changes in treatment intensity, such as increased funding, pollution abatement, or expanded eligibility, than about the effect of the existence of a treatment that already exists.

Despite how conceptually useful and practically common continuous DiD designs are, currently available econometric results provide little guidance on applying and interpreting them, except in some specific cases. In this paper, we introduce a set of tools that are suitable for DiD setups with variation in treatment dosage. In particular, we (a) discuss how one can identify a variety of treatment effect parameters by exploiting parallel-trends-type assumptions, (b) provide several decompositions showing that the simple linear two-way-fixed-effects (TWFE) estimand is generally hard to motivate empirically, and (c) propose "forward-engineered" estimators that target ex-ante clearly defined causal parameters.

To foster intuition and simplify exposition, we start by discussing causal parameters in a two-period DiD design in which units move from no treatment to a non-zero dose. We call the difference between a unit's potential outcome under dose d and its untreated potential outcome a *level treatment effect*. We call the difference in a unit's potential outcome with a marginal increase in the dose a *causal response* (Angrist and Imbens, 1995). When treatment is binary, these two notions of treatment effects coincide, but they do not under a continuous treatment. Importantly, level treatment effects and causal responses can have meaningfully different interpretations, and we establish that they generally require different identifying assumptions as well.

Comparisons between treated and untreated units identify average (level) treatment effect parameters under a parallel trends assumption on untreated potential outcomes, similar to binary DiD designs. Comparisons between adjacent dose groups, however, do not identify average causal response parameters under the "standard" parallel trends assumption. We discuss an alternative, 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

[1] We generally use "continuous" treatments also to mean multi-valued ordered discrete treatments, but make the distinction explicit for certain results.

[2] In his 1965 presidential address to the Royal Society of Medicine, Sir Austin Bradford Hill, a pioneer in the study of smoking and cancer, included among his criteria for inferring causality from observational data, "a biological gradient, or dose-response curve" and argued that "we should look most carefully for such evidence" (Hill, 1965).

they instead experienced the lower dose. Thus, strong parallel trends restricts treatment effect heterogeneity and justifies comparing dose groups. Strong parallel trends may not be plausible in many applications. Currently, in empirical work, it is common for papers to write as if they have assumed standard parallel trends and interpret their results as if they have assumed strong parallel trends. Our results clarify what causal questions can be answered under standard parallel trends and what causal questions require stronger assumptions. 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.[3]

The ideas discussed above are in the spirit of what Mogstad and Torgovitsky (2024) call *forward engineering*, where the researcher clearly specifies target parameters and assumptions up front and builds estimators to implement the identification strategy. Our second main contribution is to *reverse engineer*[4] 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 suggest 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 provides 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.[5]

For instance, we show that β^{twfe} can be expressed as a weighted integral of average level treatment effect parameters but where the weights integrate to zero, indicating that β^{twfe} should not be interpreted as an average (level) treatment effect. Interestingly, however, TWFE puts negative weights on the below-average dose units and positive weights on above-average dose units, and, thus,

[3] In applications where units choose their amount of the treatment, it is natural to refer to this term as selection bias. In other applications where the dose measures a unit's amount of exposure to some treatment, a different term, such as "heterogeneity bias", could be more appropriate. For simplicity, throughout the paper, we simply refer to this term as selection bias.

[4] We again borrow the terminology from Mogstad and Torgovitsky (2024), who define reverse engineering as starting with an estimator/regression coefficient (e.g., the OLS estimator for β^{twfe} in a linear TWFE regression specification) and trying to understand how it behaves in the presence of treatment effect heterogeneity or other complications. That paper provides a sharp contrast between forward engineering and reverse engineering for instrumental variables applications in economics.

[5] The decompositions that we provide are specific to the particular TWFE regression specification in Equation (1.1), which we focus on due to its ubiquity in empirical work with a continuous treatment. Some of the drawbacks we discuss below, particularly regarding weighting schemes inherited from the TWFE regression, could be addressed by considering a more flexible specification. See also Wooldridge (2021) for related discussions considering more flexible TWFE specifications in staggered treatment setups.

after re-scaling by a weighted average of the difference between doses for high- and low-dose units, is equivalent to a weighted binary DiD using higher-dose units as the "treated" group and lower-dose units as the "comparison" group, with weights proportional to a unit's absolute distance from the mean dose. Our next decomposition, based on average level treatment effect parameters scaled by their dose, also displays negative weights, though their weights integrate up to one and not zero.

In contrast, a TWFE decomposition in terms of average causal response parameters has weights that integrate up to one and are non-negative, but also includes a selection bias term stemming from effect heterogeneity across doses. The strong parallel trends assumption eliminates this selection bias. The weights on causal responses at different doses, however, differ from the distribution of the dose, which creates a further challenge to interpreting β^{twfe} in the presence of treatment effect heterogeneity, even if strong parallel trends holds. This is particularly important when the magnitude of the causal effects is of interest, but also has a strong bite in setups with nonlinear average level treatment effects, as average causal responses may have different signs across the dosage distribution. We reach a similar conclusion when decomposing β^{twfe} using the scaled 2×2 average effects as building blocks.

Given these drawbacks, we discuss different DiD estimators that build on our identification results and recover interpretable causal parameters. When the treatment is discrete, this is as simple as running a linear regression with multiple treatment indicators, which is similar to staggered DiD setups (Callaway and Sant'Anna, 2021). When the treatment is continuous, there are several options, including adopting a parametric regression model or a nonparametric regression model—such as one based on kernels or sieves. In particular, we discuss how to adapt the data-driven sieve-based nonparametric regression proposed by Chen, Christensen, and Kankanala (2024) to our context, although we note that other nonparametric procedures are also possible. We also show how to construct causal summary measures of our average treatment effect functions that bypass the TWFE weighting problems by using the dose density as weights. Our results suggest that one can easily summarize average level treatment effects among treated units by comparing the average change in outcomes for all treated units to the average change in outcomes for untreated units. This can be estimated by running a binary DiD with a "treatment dummy" equal to one for any units with positive doses. Summarizing average causal responses using dose density weights involves estimating an average derivative, which is simple to compute using "flexible" linear regressions. We also discuss how to construct event-study results using these summary measures, which can then be used to assess the plausibility of the parallel trends assumptions.

To show how TWFE regressions perform in practice and to illustrate the benefits of our proposed estimators, we revisit Acemoglu and Finkelstein's 2008 study of a 1983 Medicare reform that eliminated labor subsidies for hospitals. The original paper uses a TWFE estimator to compare the change in capital-labor ratios between hospitals whose input prices were more or less affected by the end of the subsidy. It concludes that price regulations favoring capital significantly increase capital use. The distinction between level treatment effect parameters and causal responses is important in this example: a positive level treatment effect shows that the policy as a whole increased the use of capital, while causal responses describe which subsidy levels generated the largest responses. We

find that the reform raised capital-labor ratios by about 18 percent, which is 50 percent larger than the comparable TWFE estimate because of the weighting issues highlighted by our decompositions. We also estimate variable average causal response ($ACRT$) parameters that are quite large at low subsidy levels—implying elasticities of substitution greater than 2—yet slightly *negative* for most positive doses. These negative $ACRT$ estimates cast doubt on the strong parallel trends assumption, the simple two-factor model of hospital production, or both. Our results support Acemoglu and Finkelstein's 2008 conclusion that the 1983 Medicare reform led hospitals to favor capital over labor, but suggest caution in a policy interpretation about which subsidy levels have the largest effects or an economic interpretation in terms of production function parameters.

Related Literature: This paper contributes to the fast-growing literature on modern DiD methods; see, e.g., Roth, SantAnna, Bilinski, and Poe (2023), de Chaisemartin and dHaultfoeuille (2023), Callaway (2023), and Baker et al. (2025) for overviews. Most of this work focuses on binary treatment setups, with a few exceptions. de Chaisemartin and D'Haultfoeuille (2018) focuses on fuzzy designs, where a researcher is interested in individual-level effects of a binary treatment that has been aggregated across units into a continuous "treatment rate." In contrast, we study "sharp" designs where the treatment exposure is itself continuous or multi-valued discrete at the unit-level. The supplemental appendix of de Chaisemartin and D'Haultfoeuille (2020) considers the case with ordered multi-valued treatments and presents a decomposition of TWFE regressions using a scaled treatment effect measure as the "building block." Our decomposition differs from theirs in that we allow for continuous treatments and also consider different building blocks. The approach proposed in de Chaisemartin and D'Haultfoeuille (2024) can also accommodate continuous treatments, though they mainly focus on alternative, more aggregated target parameters than we do. See also D'Haultfoeuille, Hoderlein, and Sasaki (2023) for Changes-in-Changes-types of procedures with a continuous treatment in the spirit of Athey and Imbens (2006).

In work subsequent to ours, de Chaisemartin et al. (2025) consider a DiD setup with continuous treatments with potentially non-staggered (but static) treatments. Their paper and ours tackle related but different and complementary problems. For instance, their target parameters differ from ours, as they consider (distance-weighted) averages of what we refer to as 2×2 average effects. Unlike our $ACRT$, these parameters average effects of discrete rather than marginal changes of treatments. Furthermore, our estimation procedures greatly differ from theirs, as we consider both functional parameters (dose-response and $ACRT$ curves) and causal summary measures. On the other hand, they allow for units to already be exposed to the treatment in the first period and consider instrumental variable extensions, which we do not.

Our TWFE decompositions are related to a number of recent results on the limitations of TWFE regressions in the presence of treatment effect heterogeneity. For example, that some of our TWFE decompositions include negative weights is related to the negative weights that can arise for TWFE estimators with binary treatments (see, e.g., Goodman-Bacon, 2021, de Chaisemartin and D'Haultfoeuille, 2020, Sun and Abraham, 2021, and Borusyak, Jaravel, and Spiess, 2024). We add to this literature by highlighting that the same TWFE regression coefficient can have different interpretations depending on the "building blocks", and that new "bias" terms may appear, depending on

the type of parallel trends assumption being used. Although our results show that negative weights can show up even in two-period cases, which is not the case in the papers above, a perhaps more important lesson from our decompositions is that *even when all weights are non-negative*, TWFE can still provide an unappealing causal summary parameter with heterogeneous treatment effects. We also note that, as a by-product of our decomposition results, if one replaces our DiD setting with one with cross-sectional data and a randomly assigned dose, all four of our decomposition results would continue to go through (e.g., just take the pre-treatment outcome to be zero almost surely), highlighting that linear specifications may not be very attractive with continuous treatments, *even* when the dose is fully randomized. These results seem to be new to the literature.

Our results are also related to other branches of causal inference and econometrics. For instance, Goldsmith-Pinkham, Sorkin, and Swift (2020) connect Bartik instruments to DiD designs under an independence assumption. We complement this analysis by studying identification in a similar setup under different kinds of parallel trends assumptions. Our cautionary results about interpreting comparisons of ATT's at different doses echo related points on comparing "local" treatment effect parameters to each other. Some examples include Angrist and Fernandez-Val (2013), Mogstad, Santos, and Torgovitsky (2018), and Oreopoulos (2006) in the context of local average treatment effects; Cattaneo, Titiunik, Vazquez-Bare, and Keele (2016) and Cattaneo, Keele, Titiunik, and Vazquez-Bare (2021) in the context of regression discontinuity designs with multiple cutoffs; or Fricke (2017) in the context of difference-in-differences with two treatments. Our results highlighting limitations of linear regressions to approximate treatment effects are related to Aronow and Samii (2016), Sloczynski (2022, 2024), Blandhol, Bonney, Mogstad, and Torgovitsky (2025), and Goldsmith-Pinkham, Hull, and Kolesár (2024). In particular, our decomposition results about the importance of the building block parameters are related to Sloczynski (2022), which also discusses related points in binary cross-sectional designs based on unconfoundedness. Finally, we note that our causal response decomposition builds on Yitzhaki (1996, Proposition 2), which expresses the slope coefficient in a regression of an outcome on a continuous variable as a weighted average of underlying local slopes. Besides differences related to causal interpretations and panel data, we extend those results to allow for a mass of untreated units. See Ishimaru (2024) and Kolesár and Plagborg-Møller (2025) for other examples of recent work that builds on Yitzhaki (1996) in the context of causal inference.

2 Motivating Continuous DiD from an Empirical Perspective

To fix ideas and provide intuition for our results, we revisit Acemoglu and Finkelstein's 2008 (AF) study of how price regulations affect firms' input choices. When Medicare began in 1965, hospitals received reimbursements from the federal government for a share of their labor and capital expenditures proportional to the fraction of total patient days accounted for by Medicare recipients (m_i). Hospital i thus faced input prices equal to $(1 - s_L m_i)w$ for labor and $(1 - s_K m_i)r$ for capital, where s_L and s_K are the labor and capital subsidy rates and w and r are market wages and rental rates. In 1983, Medicare moved to the Prospective Payment System (PPS), which replaced the labor subsidy with a small payment per episode/diagnosis. This set $s_L = 0$ but left the capital subsidy unchanged.

Therefore, the price of labor for a given hospital rose from $(1 - s_L m_i)w$ to w, skewing relative factor prices.

The statutory relationship between a hospital's Medicare volume, m_i, and the change in its price of labor, $s_L m_i w$, motivates AF's use of a continuous DiD design comparing changes in capital/labor ratios before and after 1983 between hospitals with different pre-PPS Medicare inpatient shares.[6] AF's description, estimation, and interpretation of this empirical strategy touch on some of the most common ways of justifying and implementing continuous DiD designs.

One motivation for this design is practical: variation in a dose (or exposure) permits the evaluation of treatments for which binary DiD is either infeasible or undesirable. In AF's case, about 15 percent of hospitals were "untreated" by the change in Medicare's subsidy policy because they served non-Medicare-eligible populations, like children or psychiatric patients, so they may not constitute a valid comparison group. AF therefore describe m_i, which is the hospital's Medicare volume in 1983, as an "attractive source of variation" in the price of labor both because it varies substantially—the mean of m_i among treated hospitals is 0.45, and the standard deviation is 0.15—and because hospitals with $m_i > 0$ may be more comparable to each other than treated hospitals are to untreated hospitals.

Another common justification for continuous DiD designs is that a "dose-response" relationship between exposure and outcomes can support a causal interpretation or test a theoretical prediction. Meyer (1995, p. 158), for example, argues that "differences in the intensity of the treatment across different groups allow one to examine if the changes in outcomes differ across treatment levels in the expected direction."[7] AF lay out a simple theoretical framework in which the move to PPS should (i) raise capital/labor ratios and (ii) do so more strongly for hospitals with higher pre-PPS values of m_i. They view their continuous DiD design as a way to estimate a causal effect of PPS as a whole and test the theoretical predictions of their model.

Finally, researchers often advocate for continuous DiD designs because they can be used to estimate average causal effects of small changes in the dose. In many economic models, price and income elasticities determine optimal policies like tax rates, tax bases, subsidies, and regulations (Hendren, 2016), but these are continuous concepts that can be estimated accurately only with continuous variation. We discuss how AF's theoretical framework implies, under some assumptions, that DiD estimates provide information about hospitals' elasticity of substitution between capital and labor, although AF do not argue for this kind of "marginal" interpretation.

In terms of estimation, AF use the standard tool for continuous DiD designs: a TWFE regression with hospital and year fixed effects. They follow textbook advice. Wooldridge (2010, p. 132) observes that a two-period DiD regression estimator "can be easily modified to allow for continuous, or at

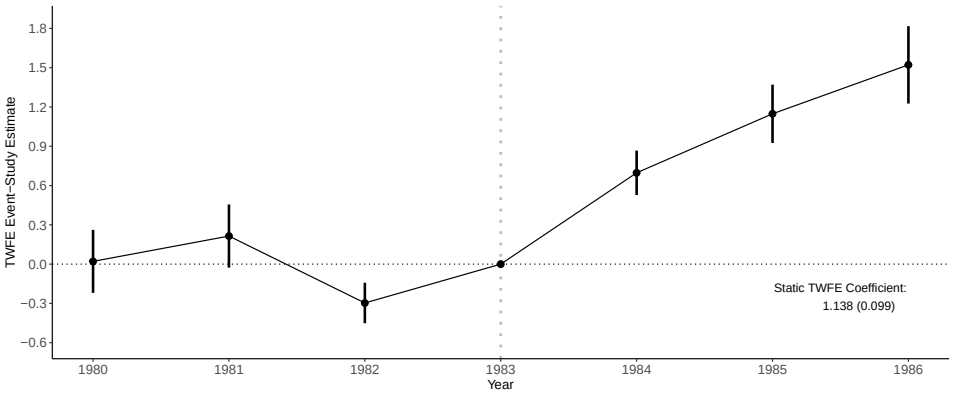
[6] AF use data reported by hospitals each year to the American Hospital Association from 1980 to 1986 (American Hospital Association, 1986). They proxy for the capital/labor ratio using the depreciation share of total operating expenses, which averages about 4.5 percent in their period.

[7] Hill (1965) makes this point in the context of smoking and cancer:

> "The fact that the death rate from cancer of the lung rises linearly with the number of cigarettes smoked daily, adds a very great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers."

He also notes that more deaths among light rather than heavy smokers would weaken the causal claim unless one could "envisage some much more complex relationship to satisfy the cause-and-effect hypothesis."

Figure 1: Two-Way Fixed Effects Event-Study Estimates of the Effect of Medicare's Reimbursement Reform on Hospital Input Mix



Notes: The figure plots TWFE event-study coefficients and their 95% confidence intervals from regressions with hospital fixed effects, year fixed effects, and the 1983 Medicare inpatient share (m_i) interacted with either a dummy for years after 1983 or the year dummies. The outcome variable is the depreciation share of total operating expenses, a measure of hospitals' capital/labor ratio. The data cover the years 1980-1986 and come from the American Hospital Association's annual survey (American Hospital Association, 1986). The results are not numerically identical to AF's because we drop 860 hospitals (out of 6,741) with missing outcomes for some years.

least non-binary, 'treatments'." Angrist and Pischke (2008, p. 234) emphasize "a second advantage of regression DD is that it facilitates the study of policies other than those that can be described by a dummy." They also follow common practice and describe their identifying assumption as an extension of the parallel trends assumption from binary designs: "*Without the introduction of PPS*, hospitals with different m_i's would not have experienced differential changes in their outcomes in the post-PPS period" (emphasis added).

Figure 1 reproduces AF's DiD event-study coefficients for each calendar year, relative to 1983, and the estimate of β^{twfe} from an equation like (1.1). AF interpret these results as indicative that after 1983, capital/labor ratios rose more strongly for hospitals with higher values of m_i, without a substantial differential change in input mix before PPS. Our impression is that event-study results like those in Figure 1 would usually be interpreted as strong causal evidence because there are (relatively) small pre-trend estimates, large estimates in post-treatment periods, and tight confidence intervals. What is missing from most continuous DiD analyses, however, is a specific statement about *what* causal parameters researchers would like to estimate, the assumptions under which they are identified, and a formal justification for a particular estimator. Our goal is to shed light on these three issues.

3 Baseline Case: A New Treatment with Two Periods

We illustrate our main points in a setup with two periods of panel data, $t = 1$ and $t = 2$. In the second period, some units receive a treatment "dose," denoted by D_i, and others remain untreated. Extensions to multiple periods and staggered setups are discussed in Section 5. We denote the support of D by $\mathcal{D}$. D_i can be (absolutely) continuous or can be multi-valued discrete, but to simplify the exposition, we refer to it as "continuous." We define potential outcomes for unit i in period t as

$Y_{i,t}(0,d)$, where potential outcomes are indexed by the treatment sequence (Robins, 1986). As we focus on the setup where all units have $d = 0$ in period $t = 1$, we simplify the potential outcome notation and henceforth write $Y_{i,t}(d)$. This is the outcome that unit i would experience in period t under (period-two) dose d. In each time period t, the observed outcome for unit i is $Y_{i,t} = Y_{i,t}(D_i)$. We assume that all expectations are finite and well-defined. Henceforth, we omit the unit index i to make the notation less cluttered and define $\Delta Y = Y_{t=2} - Y_{t=1}$.

3.1 Parameters of Interest with a Continuous Treatment

The potential outcomes notation $Y_t(d)$ reflects that treatment can take many values, and so each unit can experience many types of causal effects. The *level treatment effect* of dose d in time period t for a given unit is defined as its potential outcome when $D = d$ minus its untreated potential outcome: $Y_t(d) - Y_t(0)$. Level treatment effects measure the treatment effect at time t from switching treatment dosage from 0 to d. This is a straightforward extension of a binary "treatment effect" to a continuous "treatment effect function" or "dose-response function."

But zero-treatment is not the only relevant counterfactual. We define a unit's *causal response* at d as $Y_t'(d)$, the derivative of the potential outcome with respect to dose d (when the treatment is continuous),[8] or as the difference in potential outcomes between adjacent doses scaled by the difference in the doses, $(Y_t(d_j) - Y_t(d_{j-1}))/(d_j - d_{j-1})$ (when the treatment is discrete). Causal responses measure the treatment effect at time t of a marginal increment of dose d. These two types of treatment effects—the level of $Y_t(d) - Y_t(0)$ or its slope, $Y_t'(d)$—define unit-level causal parameters in continuous designs, and connect to results in the instrumental variables (IV) literature on multi-valued discrete or continuous endogenous variables (Angrist and Imbens, 1995, Angrist, Graddy, and Imbens, 2000).

We focus on "building block" parameters that are averages of these two kinds of causal effects in the post-treatment period, $t = 2$. Average level treatment effects (which we refer to as average treatment effects) extend definitions from the binary case:

$$ATT(d|d') = \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(0)|D = d'] \quad \text{and} \quad ATT(d) = \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(0)|D > 0],$$

where $ATT(d|d')$ is the average effect of dose d compared to zero dosage in the post treatment period $t = 2$ on units that actually experienced dose d'. When $d' = d$, this is the ATT local to units that received dose d. $ATT(d)$ is the average difference between potential outcomes under dose d relative to untreated potential outcomes across all treated units, not just those that experienced dose d, in time period $t = 2$.

Average causal response parameters for absolutely continuous treatments are defined as

$$ACRT(d|d') = \left.\frac{\partial ATT(l|d')}{\partial l}\right|_{l=d} = \left.\frac{\partial \mathbb{E}[Y_{t=2}(l)|D = d']}{\partial l}\right|_{l=d} \text{ and } ACRT(d) = \frac{\partial ATT(d)}{\partial d} = \frac{\partial \mathbb{E}[Y_{t=2}(d)|D > 0]}{\partial d}.$$

$ACRT(d|d')$ is the average causal response to a marginal increase in dose from dose d for dose group d'. It equals the derivative of $ATT(l|d')$ with respect to l, evaluated at $l = d$, which is equivalent

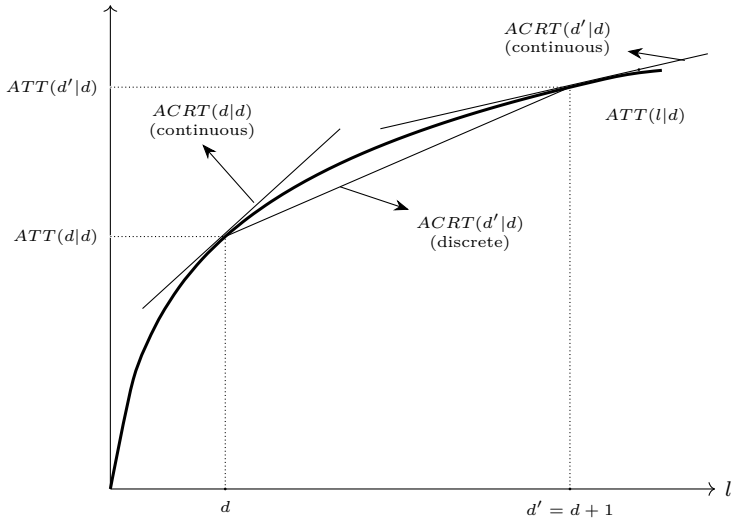
[8] This is a slight abuse of notation as we do not require $Y_t(d)$ to be differentiable (or even continuous), but rather we mean here the causal effect of a marginal increase in the dose on a unit's outcome: $\lim_{h \to 0^+} (Y_t(d + h) - Y_t(d))/h$.

to the derivative of the $t = 2$ average potential outcome of dose d for dose group d'. $ACRT(d)$ is the average causal response of dose d across all treated units. For discrete treatments, average causal responses are defined in a similar way but with slightly different notation to accommodate discreteness of d:

$$ACRT(d_j|d_k) = \frac{\mathbb{E}[Y_{t=2}(d_j) - Y_{t=2}(d_{j-1})|D = d_k]}{d_j - d_{j-1}} \quad \text{and} \quad ACRT(d_j) = \frac{\mathbb{E}[Y_{t=2}(d_j) - Y_{t=2}(d_{j-1})|D > 0]}{d_j - d_{j-1}}.$$

$ACRT(d_j|d_k)$ equals the difference in mean potential outcomes between dose level d_j and the next lowest dose d_{j-1} in period $t = 2$ for dose group d_k, scaled by the difference between the two doses. Similarly, $ACRT(d_j)$ gives the average causal response of dose d_j relative to dose d_{j-1}, but it is for the entire treated group.[9]

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 for dose group 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 the slope of a tangent line, and for a discrete dose when $ACRT$ is the slope of a line connecting two discrete points on $ATT(d|d)$.

Figure 2 illustrates these parameters graphically. The concave line plots an average treatment effect function against the dose for dose group d, $ATT(\cdot|d)$. If we consider dose levels d and d', there are two possible ATT parameters. The first, $ATT(d|d)$, the level of dose group d's average treatment effect function at d, is an average treatment effect that is "local" to units that experienced dose d. The second, $ATT(d'|d)$, is also "local" to dose group d, but refers to the effect they would experience at dose d' even though they did not actually receive that dose. The continuous-dose $ACRT$ parameters are the slopes of tangent lines to the $ATT(\cdot|d)$ function, and the discrete-dose $ACRT$ parameters are the slopes of lines connecting two points on the $ATT(\cdot|d)$ function. As with ATT's, our definitions encompass causal responses to doses other than the one a group actually receives (i.e., $ACRT(d'|d)$).

A proper interpretation of continuous DiD results hinges on which type of parameter one wants,

[9] We note that our definition of $ACRT(d_j|d_k)$ and $ACRT(d_j)$ differ from the definitions in Angrist and Imbens (1995), as it scales the changes in expected potential outcomes by the change in dosage.

and can identify and estimate. For instance, even if all $ATT(d|d)$ parameters are large and positive, some $ACRT(d|d)$ parameters could be zero or negative. A researcher misinterpreting a large ATT estimate as an $ACRT$, in this case, would mistakenly conclude that a policy to raise every unit's dose would have large effects. A researcher confusing a small $ACRT$ for an ATT would mistakenly conclude that an entire policy was ineffective, even though it actually just has small effects at the margin.

The above-mentioned causal parameters are functional parameters because they are allowed to vary arbitrarily across dose groups d and across (counterfactual) doses d'. This contrasts with β^{twfe} from (1.1), which is a single number. In many applications, it may be desirable to aggregate these functional parameters into lower-dimensional objects that are easier to report and may be more precisely estimated. We focus on aggregating the functional parameters discussed above by averaging them using the distribution of the dose among all treated units. We denote these summary parameters by

$$ATT^{\texttt{loc}} = \mathbb{E}[ATT(D|D)|D>0] \qquad \text{and} \qquad ATT^{\texttt{glob}} = \mathbb{E}[ATT(D)|D>0]$$
$$ACRT^{\texttt{loc}} = \mathbb{E}[ACRT(D|D)|D>0] \qquad \text{and} \qquad ACRT^{\texttt{glob}} = \mathbb{E}[ACRT(D)|D>0].$$

These provide natural ways to summarize the underlying parameters. We use the `loc` superscript to denote that $ATT^{\texttt{loc}}$ and $ACRT^{\texttt{loc}}$ summarize treatment effects that are local effects of particular doses, while we use the superscript `glob` to denote that $ATT^{\texttt{glob}}$ and $ACRT^{\texttt{glob}}$ summarize treatment effects of particular doses globally (i.e., across all treated units). All four of these parameters provide "best" approximations in the sense of minimizing the mean squared distance between the summary parameter and the functional parameters. Also, note that $ACRT^{\texttt{loc}}$ and $ACRT^{\texttt{glob}}$ are average derivative-type parameters, and average derivatives have been widely studied in econometrics, see, e.g., Newey and Stoker (1993), Ai and Chen (2007), Chen, Chen, and Tamer (2023), and references therein.

3.2 Identification with a Continuous Treatment

This section discusses the identification of average treatment effect and average causal response parameters. Toward this end, we make the following assumptions.

Assumption 1 (Random Sampling)**.** *The observed data consist of* $\{Y_{i,t=2}, Y_{i,t=1}, D_i\}_{i=1}^n$*, which is independent and identically distributed.*

Assumption 2 (Treatment)**.** *In period* $t=1$*, no unit is treated, while in period* $t=2$*, the treatment dosage* D *has support* $\mathcal{D} = \{0\} \cup \mathcal{D}_+$*, where* $\mathcal{D}_+ \subseteq (0,\infty)$*. In addition,* $\mathbb{P}(D=0) > 0$*.*

Assumption 3 (No-Anticipation and Observed Outcomes)**.** *For all units, and all* $d \in \mathcal{D}$*,*

$$Y_{i,t=1} = Y_{i,t=1}(d) = Y_{i,t=1}(0) \quad \textit{and} \quad Y_{i,t=2} = Y_{i,t=2}(D_i).$$

Assumption 1 says that we observe two periods of *iid* panel data. Assumption 2 formalizes that a mass of units do not participate in the treatment in either period (we discuss the case with no untreated units in more detail at the end of this section), and the rest receive a positive amount of the treatment that can vary in amount across units. Assumption 3 says that units do not anticipate

future treatments, so we observe untreated potential outcomes for all units in the first period. In the second period, we observe the potential outcome corresponding to the actual dose that unit i experienced.

3.2.1 Identification under parallel trends

Identification of average level treatment effects follows closely from the DiD setup with binary treatments. In particular, our results rely on an extension of the binary parallel trends assumption.

Assumption PT (Parallel Trends)**.** *For all $d \in \mathcal{D}_+$,*

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

Assumption PT says that the average evolution of outcomes that units with any dose d would have experienced without treatment is the same as the evolution of outcomes that units in the untreated group actually experienced. Binary DiD designs also rely on assumptions like this. To simplify the exposition below, we often simply refer to Assumption PT as *parallel trends* (PT). The following result shows that under Assumption PT, $ATT(d|d)$ is identified; all proofs are in Appendix A.

Theorem 3.1. *Under Assumptions 1, 2, 3, and PT, $ATT(d|d)$ is identified for all $d \in \mathcal{D}_+$, and it is given by*

$$ATT(d|d) = \mathbb{E}[\Delta Y|D = d] - \mathbb{E}[\Delta Y|D = 0].$$

Furthermore, $ATT^{loc} = \mathbb{E}[\Delta Y|D > 0] - \mathbb{E}[\Delta Y|D = 0]$.

The identification results for $ATT(d|d)$ in Theorem 3.1 hold by essentially the same arguments used for binary treatments. Because Assumption PT ensures that $\mathbb{E}[\Delta Y|D = 0]$ is the same as the evolution of outcomes that treated units would have experienced without the treatment, $ATT(d|d)$ equals the difference between the change in outcomes for dose group d and the untreated group. As a direct consequence, by averaging all the $ATT(d|d)$'s over the distribution of non-zero dosages, we have that the summary parameter $ATT^{\texttt{loc}}$ is identified by simply comparing units with a positive dose to untreated units.[10] On the other hand, parallel trends, as defined in Assumption PT, is *not* strong enough to guarantee the identification of $ATT(d)$.

The identification of average causal response parameters differs from the identification of ATT parameters because it requires comparisons between dose groups.

Assumption 4 (Continuous or Multi-Valued Discrete Treatment)**.** *The treatment is either continuous or multi-valued discrete. More precisely, one of the following is true:*

(a) Let $\mathcal{D}_+^c = (d_L, d_U) \subset \mathcal{D}_+$ with $f_{D|D>0}$ a Lebesgue density which satisfies $f_{D|D>0}(d) > 0$ for all $d \in \mathcal{D}_+^c$, and $\mathbb{E}[\Delta Y|D = d]$ is differentiable on $\mathcal{D}_+^c$.

[10] The result for $ATT^{\texttt{loc}}$ in Theorem 3.1 rationalizes the practice of "binarizing" a continuous treatment, that is, ignoring variation in the amount of the treatment across different units and simply categorizing units as being treated or untreated on the basis of whether or not they experience any positive amount of the treatment. Binarizing is fairly common in empirical work (for example, Bartik, Currie, Greenstone, and Knittel (2019), Cengiz, Dube, Lindner, and Zipperer (2019), Gentzkow, Shapiro, and Sinkinson (2011), Hoynes and Schanzenbach (2009), and Meyer, Viscusi, and Durbin (1995)). The expression for $ATT^{\texttt{loc}}$ also arises as a special case of the non-normalized event study estimator in de Chaisemartin and D'Haultfoeuille (2024).

(b) $\mathcal{D}_+ = \mathcal{D}_+^{mv}$ *where* $\mathcal{D}_+^{mv} \subset \mathbb{N}_+$*, where* $\mathbb{N}_+ = \{1, 2, 3, \ldots\}$ *denotes the strictly positive natural numbers. Let* d_j *denote the* j^{th} *element of* $\mathcal{D}_+^{mv}$*. In addition,* $\mathbb{P}(D = d) > 0$ *for all* $d \in \mathcal{D}_+^{mv}$*.*

Assumption 4 distinguishes between cases with a continuous 4(a) or discrete 4(b) treatment. Assumption 4(a) allows for the smallest value of the treatment to be arbitrarily close to zero or strictly larger than zero, both of which are common in applications.

Our central identification result is that causal response parameters are not identified under Assumption PT, because comparisons between different dose groups are biased when treatment effects (of the same dose) vary across dose groups, even when the average evolution of untreated potential outcomes is the same.[11]

Theorem 3.2. *Under Assumptions 1, 2, 3, and PT, comparisons of paths of outcomes among different dose groups recover a mix of causal effect parameters and selection bias terms. Specifically,*

(a) For $(h, l) \in \mathcal{D}_+ \times \mathcal{D}_+$*,*

$$\begin{aligned}\mathbb{E}[\Delta Y|D = h] - \mathbb{E}[\Delta Y|D = l] &= ATT(h|h) - ATT(l|l) \\ &= \underbrace{\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D = h]}_{\textit{causal effect}} + \underbrace{\Big(ATT(l|h) - ATT(l|l)\Big)}_{\textit{selection bias}}.\end{aligned}$$

(b) If Assumption 4(a) also holds, then, for $d \in \mathcal{D}_+^c$*,*

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

(c) Alternatively, if Assumption 4(b) also holds,

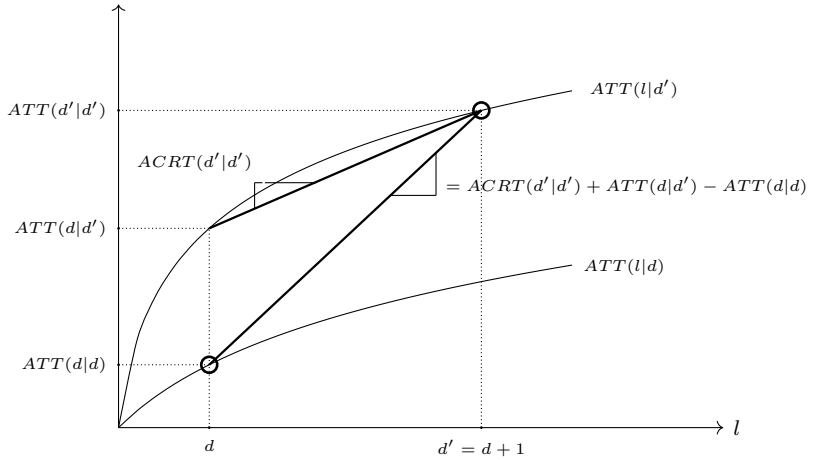
$$\frac{\mathbb{E}[\Delta Y|D = d_j] - \mathbb{E}[\Delta Y|D = d_{j-1}]}{d_j - d_{j-1}} = ACRT(d_j|d_j) + \underbrace{\frac{ATT(d_{j-1}|d_j) - ATT(d_{j-1}|d_{j-1})}{d_j - d_{j-1}}}_{\textit{scaled selection bias}}.$$

Theorem 3.2 says that under parallel trends, comparisons of outcome paths between higher- and lower-dose groups mix together (i) causal responses and (ii) a "selection bias" type of term that comes from differences in average treatment effects of the same dose for different dose groups. Intuitively, even if untreated potential outcomes evolve in the same way, observed paths of outcomes differ between dose groups for two reasons. One is the causal response itself, which comes from differences in doses (h versus l) causing differences in outcomes. The other is a selection bias type of contamination, which comes from differences across dose groups in the average level effect of the particular dose l—parallel trends does not rule out that different dose groups could experience different treatment effects of the same dose.

Figure 3 illustrates this result for an example with two dose groups and two doses: d and $d' = d+1$. The slope of the line that connects the points $(d, ATT(d|d))$ and $(d', ATT(d'|d'))$ is steeper than the average causal response of interest, $ACRT(d'|d')$, because it jumps from one ATT function to the other. This is captured by the selection bias term, a version of selection-on-gains that equals

[11] The canonical DiD comparison is $\mathbb{E}[\Delta Y|D = d] - \mathbb{E}[\Delta Y|D = 0]$. The results in this section instead involve comparing $\mathbb{E}[\Delta Y|D = h]$ to $\mathbb{E}[\Delta Y|D = l]$ for two different doses d and l. It is helpful to notice that $\mathbb{E}[\Delta Y|D = h] - \mathbb{E}[\Delta Y|D = l] = \big(\mathbb{E}[\Delta Y|D = h] - \mathbb{E}[\Delta Y|D = 0]\big) - \big(\mathbb{E}[\Delta Y|D = l] - \mathbb{E}[\Delta Y|D = 0]\big)$, where the term involving the untreated group cancels out.

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



Notes: The figure shows that comparing adjacent $ATT(d|d)$'s 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).

the difference in treatment effects at the lower dose: $ATT(d|d') - ATT(d|d)$. It breaks the causal interpretation because observed outcomes for lower-dose units are not a valid counterfactual for what higher-dose units would have experienced at a lower dose. The selection bias is not identified as we do not observe $Y_{t=2}(d)$ for units that experienced dose d'. Such a result precludes a causal interpretation of $ATT(d|d)$ differences across doses, at least when one is not willing to further strengthen parallel trends.

3.2.2 Identification under strong parallel trends

The fact that average causal responses are not identified under a traditional parallel trends assumption suggests that learning about this type of parameter with continuous DiD designs requires new assumptions as well. This section discusses an alternative, typically stronger assumption that allows for the identification of $ACRT(d)$ and $ATT(d)$ parameters, which we refer to as *strong parallel trends* (SPT).

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

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

Under Assumption 3, the right-hand side of the equation in Assumption SPT is the (observed) average evolution of outcomes for dose group d. Assumption SPT says that the average evolution of outcomes for the entire treated population if all experienced dose d (the left-hand side of the previous equation) is equal to the path of outcomes that dose group d actually experienced. In applications where the treatment is binary, Assumption SPT, like Assumption PT, reduces to the usual parallel trends assumption. Like the case with a binary treatment, it allows for treated units to select into being treated. Among treated units, though, it rules out selection into a particular amount of the treatment. With more complicated treatments, Assumption SPT notably differs from

Assumption PT because it involves potential outcomes under different doses, $Y_t(d)$, rather than only untreated potential outcomes, $Y_t(0)$. While Assumption SPT is not strictly stronger than Assumption PT (e.g., notice that it does not require parallel trends in untreated potential outcomes for all dose groups), we refer to it as *strong parallel trends* to indicate that in many applications it would be a stronger, perhaps much stronger, assumption. In an earlier working version of our paper, we provide a slightly stronger version of strong parallel trends that is strictly stronger than Assumption PT as well as a full comparison between several different varieties of parallel trends assumptions (Callaway, Goodman-Bacon, and Sant'Anna, 2025).

An alternative way to think about Assumption SPT is as an assumption that restricts treatment effect heterogeneity.[12] In an earlier working version of our paper (Callaway, Goodman-Bacon, and Sant'Anna, 2025), we show that if one maintains Assumption PT, Assumption SPT is equivalent to assuming that $ATT(d|d) = ATT(d)$ for all doses. While this condition does not impose full treatment effect homogeneity, it does rule out selection-on-gains into a particular dose group and ensures the observed outcome changes for every dose group reflect what would have happened if all other dose groups had received that dose. This condition can also be viewed as a structural assumption in the sense that it effectively allows one to extrapolate treatment effects of dose d among dose group d to treatment effects of dose d for the entire treated population.

In the remainder of this section, we show that Assumption SPT is useful for recovering "global" average causal effect parameters, which are straightforward to compare to each other, and, hence, sidestep the selection bias issues discussed above. Before doing that, it is worth mentioning that we are not proposing Assumption SPT as an assumption that empirical researchers should readily adopt; in fact, in many applications, Assumption SPT may be a strong or implausible assumption. Rather, our aim is to clarify that many natural target parameters in DiD applications with a continuous treatment require stronger assumptions than parallel trends as defined in Assumption PT.

Theorem 3.3. *Under Assumptions 1, 2, 3, and SPT,*

(a) For $d \in \mathcal{D}_+$, it follows that

$$ATT(d) = \mathbb{E}[\Delta Y|D = d] - \mathbb{E}[\Delta Y|D = 0].$$

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

$$\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D > 0] = ATT(h) - ATT(l) = \mathbb{E}[\Delta Y|D = h] - \mathbb{E}[\Delta Y|D = l]$$

(c) When Assumption 4(a) holds (i.e., treatment is continuous), it follows that, for $d \in \mathcal{D}_+^c$,

$$ACRT(d) = \frac{\partial \mathbb{E}[\Delta Y|D = d]}{\partial d}$$

[12] There are some instances of versions of strong parallel trends implicitly being discussed in empirical work. Chodorow-Reich, Nenov, and Simsek (2021, p. 1636)'s cross-region study of marginal propensities to consume (MPC) notes the possibility of finding a zero even when the MPC>0 in all areas: "if low wealth areas have high MPCs and high wealth areas have low MPCs, an increase in the stock market could induce the same change in spending in both low and high wealth areas." Similarly, Saez, Slemrod, and Giertz (2012, p. 25) discuss a version of strong parallel trends in the context of estimating the elasticity of taxable income for two groups facing different positive tax changes: "if the control group faces a tax change, difference-in-differences estimates will be consistent only if the elasticities are the same for the two groups."

(d) When Assumption 4(b) holds (i.e., treatment is discrete), it follows that

$$ACRT(d_j) = \frac{\mathbb{E}[\Delta Y|D = d_j] - \mathbb{E}[\Delta Y|D = d_{j-1}]}{d_j - d_{j-1}}$$

For part (a) of Theorem 3.3, recall that $ATT(d|d)$ and $ATT(d)$ differ when there is selection into dose group d on the basis of treatment effects. Strong parallel trends rules out that kind of selection, which means that comparing average outcome changes of dose group d to the untreated group identifies $ATT(d)$. Part (b) says that comparisons of the average change in outcomes over time for different dose groups has a causal interpretation under Assumption SPT. For parts (c) and (d), the same implication of strong parallel trends ensures that lower-dose groups are valid counterfactuals for higher-dose groups, and, hence, that causal response parameters are identified under Assumption SPT.

Strong parallel trends only changes the interpretation of the estimand, not its form. One important implication is that conventional pre-tests for differential changes across groups before treatment cannot distinguish between Assumption PT and Assumption SPT. That is, because only untreated potential outcomes are observed before treatment, these periods cannot test the additional content of an assumption like SPT that necessarily involves treated potential outcomes.[13]

Finally, the identification results in Theorem 3.3 immediately imply that averages of the $ATT(d)$ and $ACRT(d)$ building blocks are identified as well. The following corollary states these results.

Corollary 3.1. *Under Assumptions 1, 2, 3, and SPT,*

(a) For $d \in \mathcal{D}_+$, it follows that

$$ATT^{glob} = \mathbb{E}[\Delta Y|D > d] - \mathbb{E}[\Delta Y|D = 0].$$

(b) When Assumption 4(a) holds (i.e., treatment is continuous), it follows that, for $d \in \mathcal{D}_+^c$,

$$ACRT^{glob} = \mathbb{E}\left[\left.\frac{\partial \mathbb{E}[\Delta Y|D = d]}{\partial d}\right|_{d=D} \middle| D > 0\right] = \int_{d_L}^{d_U} \left.\frac{\partial \mathbb{E}[\Delta Y|D = d]}{\partial d}\right|_{d=s} f_{D|D>0}(s)ds.$$

(c) When Assumption 4(b) holds (i.e., treatment is multi-valued), it follows that, for $d_j \in \mathcal{D}_+^{mv}$,

$$ACRT^{glob} = \sum_{j=1}^{J} \left(\frac{\mathbb{E}[\Delta Y|D = d_j] - \mathbb{E}[\Delta Y|D = d_{j-1}]}{d_j - d_{j-1}}\right) \mathbb{P}(D = d_j|D > 0).$$

These results highlight how identification in continuous DiD designs is fundamentally a question about dose-specific building block parameters and the underlying parallel trends assumption, not the aggregation choices that lead to particular summary parameters.

Remark 3.1 (No untreated units)**.** *Researchers often use continuous designs when all units in their sample receive some amount of the treatment having in mind comparing units that are "more treated" to units that are "less treated". Without untreated units, it is infeasible to compare dose group d to an untreated group, and, hence, it is infeasible to directly recover $ATT(d|d)$ or $ATT(d)$. However, a natural alternative is to compare dose group d to dose group d_L (the lowest possible amount of the*

[13] There are caveats to this argument, particularly in cases where the researcher targets an aggregated parameter such as ATT^{loc}. See the discussion in Section 5.4 for more details.

treatment). In Appendix SC.1 in the Supplementary Appendix, we show that, under parallel trends, when there are no untreated units,

$$\mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=d_L] = ATT(d|d) - ATT(d_L|d_L).$$

This shows that this comparison is related to underlying causal effect parameters under parallel trends; however, recall from Theorem 3.2 that the expression on the right-hand side mixes together the average causal response of moving from d_L to d with selection bias. Under strong parallel trends, we have instead that

$$\mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=d_L] = ATT(d) - ATT(d_L) = \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(d_L)|D>0],$$

which does not include selection bias terms. This discussion highlights that (unlike a setting with a binary treatment) continuous variation in the dose can be used to learn about causal effects even if there is no untreated comparison group, but interpreting these results as causal effects of the treatments requires strengthening Assumption PT.

Remark 3.2 (Comparison between different parallel trends assumptions)**.** *A researcher may be interested in comparing what is and what is not identified under different parallel trends assumptions, how these parallel trends assumptions restrict treatment effect heterogeneity, and how they compare to each other. In Appendix C of an earlier working version of our paper (Callaway, Goodman-Bacon, and Sant'Anna, 2025), we pursue this exercise and provide a Portmanteau-type theorem that allows us to better understand the "bite" of each assumption. Among other things, we show that, in general, Assumption PT and Assumption SPT are non-nested, though Assumption SPT will probably be stronger in most applications. We also introduce an aggregated parallel trends assumption that is useful for directly targeting ATT^{loc}, and an alternative strong parallel trends assumption that implies both Assumption PT and Assumption SPT but further restricts treatment effect heterogeneity.*

3.3 What Parameter Does TWFE Estimate?

In practice, empirical researchers using a continuous DiD design typically estimate a single summary parameter using a linear TWFE regression like Equation (1.1). This section links the TWFE estimand to our identification results for dose-specific parameters, describes the assumptions necessary to give TWFE *some* causal interpretation, and discusses what that interpretation is. We focus on continuous treatments and defer the discussion of multi-valued discrete treatments to Appendix SC.3 in the Supplementary Appendix.

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.

Table 1: TWFE Decomposition Weights

Decomposition	$D > 0$ Weights	$D = 0$ Weights
Causal response	$w_1^{\text{acrt}}(l) = \frac{(\mathbb{E}[D\|D \geq l] - \mathbb{E}[D])\mathbb{P}(D \geq l)}{\text{Var}(D)}$	$w_0^{\text{acrt}} = \frac{(\mathbb{E}[D\|D > 0] - \mathbb{E}[D])\mathbb{P}(D > 0)d_L}{\text{Var}(D)}$
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	$w_1^{2\times 2}(l, h) = \frac{(h - l)^2 f_D(h) f_D(l)}{\text{Var}(D)}$	$w_0^{2\times 2}(h) = \frac{h^2 f_D(h)\mathbb{P}(D = 0)}{\text{Var}(D)}$

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

The next proposition presents our decompositions of β^{twfe} under parallel trends (Assumption PT) and under strong parallel trends (Assumption SPT). The decompositions differ on the basis of the underlying building block parameters: causal response parameters ($ACRT(d|d)$ and $ACRT(d)$), level treatment effect parameters ($ATT(d|d)$ and $ATT(d)$), scaled level effects ($ATT(d|d)/d$ and $ATT(d)/d$), or scaled 2×2 effects ($\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D = h]/(h - l)$ and $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D > 0]/(h-l)$). These building blocks are connected with the dose-parameters discussed in Section 3.2 and how empirical researchers interpret β^{twfe}.[14] The weights attached to each of these decompositions are presented in Table 1.

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

(a) Causal Response Decomposition:

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

where the weights are always positive and integrate to 1.

(b) Levels Decomposition:

[14] 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.

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

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 SPT instead of Assumption PT, 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* $ACRT(l)$ *in Part (a),* $ATT(l|l)$ *with* $ATT(l)$ *in Parts (b), (c) and (d), and* $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D = h]$ *with* $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D > 0]$ *in Part (d).*

Heuristically, the proof of Theorem 3.4 builds on the fact that β^{twfe} equals the univariate slope coefficient from a regression of ΔY on an intercept and D: $\beta^{twfe} = \text{Cov}(\Delta Y, D)/\text{Var}(D)$. The covariance between outcome changes and the dose can be written in several different ways, each involving one type of comparison of paths of outcomes across different dose groups analyzed in Section 3.2. Upon imposing parallel trends (Assumption PT) or strong parallel trends (Assumption SPT), we can map these comparisons of means to causal estimands, allowing us to write these decompositions in terms of different causal building blocks. The weights show how TWFE then aggregates dose-specific estimands. The same TWFE coefficient β^{twfe} can, therefore, have different interpretations that depend on which building block parameter one has in mind. Unfortunately, Theorem 3.4 highlights that, in general, β^{twfe} does not have a clear causal interpretation: the weights are hard to interpret and can be negative, and/or selection-bias terms contaminate the interpretation of β^{twfe} as causal parameters. Despite the overall negative message, each decomposition provides interesting insights.

Theorem 3.4(a) shows that when causal responses are taken as the building blocks of the analysis, under Assumption PT, β^{twfe} is equal to a weighted average (the weights are all positive and integrate to 1) of $ACRT(d|d)$ and the same selection bias derived in Theorem 3.2.[15] The sign of this selection bias depends on how treatment effects vary across dose groups at a given dose. If units in higher dose groups would have had larger positive treatment effects at every dose, for example, then β^{twfe} will

[15] Part (a) is mechanically related to the results in Yitzhaki (1996) on interpreting linear projection coefficients with a continuous regressor when the conditional expectation may be nonlinear. Part (a) also includes a term that shows how TWFE handles a discrete jump from 0 to the minimum treated dose, d_L. Paths of outcomes are not observed for doses below d_L, but the scaled ATT for dose group d_L, $ATT(d_L|d_L)/d_L$, is averaged into β^{twfe}.

be larger than the weighted average of the $ACRT(d|d)$'s that appear in Theorem 3.4(a). Figure 3 illustrates this case for two groups. Invoking strong parallel trends eliminates the selection bias term.

The discussion above has important implications but does not come *from* TWFE itself. The weights, however, do inherit their form from ordinary least squares. Even under strong parallel trends, the particular interpretation of β^{twfe} in terms of $ACRT(d)$'s hinges on the aggregation embodied in the weights $w_1^{acrt}(d)$. Because $w_1^{acrt}(d)$ is positive and integrates to 1, β^{twfe} is *weakly causal* under Assumption SPT.[16] However, it does not estimate a natural target parameter like $ACRT^{\texttt{glob}}$ because the TWFE weights do not generally equal the dose distribution among treated, $f_{D|D>0}(d)$. Differentiating $w_1^{acrt}(d)$ shows that the weights are hump-shaped and centered around $\mathbb{E}[D]$, so causal responses around the average dose affect β^{twfe} the most (likewise, under parallel trends, selection bias around the average dose matters the most). Therefore, when $ACRT(d)$ varies across d, TWFE's weighting scheme can generate a hard-to-interpret summary parameter except for special dose distributions.[17] Instead of letting the estimation method implicitly summarize the $ACRT$'s, we recommend that researchers choose these aggregation schemes explicitly. In our view, a natural and econometrically-guided way to aggregate the $ACRT$'s into a summary parameter is given by $ACRT^{\texttt{glob}}$, which is identified (as indicated in Corollary 3.1) and can also be easily estimated.

Under linearity of realized outcomes, i.e., $\mathbb{E}[\Delta Y|D=d] = b_0 + b_1 d$, because the weights integrate to one, $\beta^{twfe} = b_1$. However, linearity alone does not imply that one necessarily recovers average causal responses. To see this, recall that $b_1 = \frac{\partial \mathbb{E}[\Delta Y|D=d]}{\partial d} = ACRT(d|d) + \frac{\partial ATT(d|h)}{\partial h}\Big|_{h=d}$, which is the sum of a causal response and a selection bias term. A leading example of linearity with selection bias would be when $\mathbb{E}[\Delta Y|D=d] = b_0 + (b_1^{acrt} + b_1^{sel})d$, where b_1^{acrt} is the causal response and b_1^{sel} is selection bias—under linearity, we would recover the sum of these two terms. In other words, in terms of $ACRT$'s, linearity gets rid of interpretation issues inherited from the weighting scheme but does not get rid of selection bias. Strong parallel trends, on the other hand, avoids selection bias, suggesting that SPT *and* linearity would restore a causal interpretation of β^{twfe} in terms of ACRT's.

Part (b) expresses β^{twfe} as a weighted integral of $ATT(d|d)$ under parallel trends with weights that integrate to zero rather than one. Therefore, some weights are negative, and, hence, β^{twfe} is not weakly causal when $ATT(d|d)$ is taken as the building block. More significantly, β^{twfe} puts the same amount of negative weight on $ATT(d|d)$'s for doses below $\mathbb{E}[D]$ as it does positive weight on $ATT(d|d)$'s for doses above $\mathbb{E}[D]$.[18] One way to view this result is that TWFE uses above-average

[16] We borrow the term *weakly causal* from Blandhol, Bonney, Mogstad, and Torgovitsky (2025), who call an estimand weakly causal if it rules out sign-reversal; i.e., an estimand is weakly causal if, when all of its underlying building blocks have the same sign, it is guaranteed to reflect this sign. This property is closely related to estimands that are weighted averages of underlying causal effect parameters whose weights are all non-negative and sum to one. They argue that this is a minimal requirement for an estimand to have a causal interpretation.

[17] Another difference between the weighting scheme of β^{twfe} and $ACRT^{\texttt{glob}}$ is that the weights underlying β^{twfe} depend on the entire distribution of the dose while the weights underlying $ACRT^{\texttt{glob}}$ only depend on the distribution of the dose among treated units. This means that β^{twfe} is sensitive to the size of the untreated group—this is hard to justify in most applications and is in contrast to DiD with a binary treatment. For example, in our application, if we drop the untreated group (dropping the untreated group does not change the underlying average causal responses), our estimate of β^{twfe} shrinks by 78%. This large difference in estimates is fully explained by how dropping the untreated group changes the weighting scheme inherited by β^{twfe}. In contrast, our estimate of $ACRT^{\texttt{glob}}$ is invariant to removing the untreated group.

[18] Unlike the other building block parameters considered in this section, even under versions of treatment effect

dose units as an "effective treated group" and below-average dose units as an "effective comparison group" that potentially includes some treated units. While the cumulative positive weights and negative weights are equal to each other, they do not generally integrate to one within these groups, which means that β^{twfe} does not equal the difference between a weighted average of outcome paths for the effective treated group relative to the effective comparison group. In Appendix SC.2 in the Supplementary Appendix, however, we derive a corollary of the result in Part (b), which shows that we can rewrite β^{twfe} as the following weighted Wald-estimand:

$$\beta^{twfe} = \frac{\mathbb{E}\Big[w_1^{bin}(D)\Delta Y\Big|D > \mathbb{E}[D]\Big] - \mathbb{E}\Big[w_0^{bin}(D)\Delta Y\Big|D < \mathbb{E}[D]\Big]}{\mathbb{E}\Big[w_1^{bin}(D)D\Big|D > \mathbb{E}[D]\Big] - \mathbb{E}\Big[w_0^{bin}(D)\Delta D\Big|D < \mathbb{E}[D]\Big]}. \tag{3.1}$$

The numerator of Equation (3.1) shows that β^{twfe} compares weighted average outcome changes above and below $\mathbb{E}[D]$ with weights proportional to how far a unit's dose is from $\mathbb{E}[D]$.[19] The denominator scales this comparison by the same weighted difference in D. This representation highlights some challenges of using β^{twfe} to summarize the average level-effect of a continuous treatment. First, while the numerator is (roughly) a weighted level-effect, the denominator shows that β^{twfe} additionally depends on a measure of the average distance between the effective treated and comparison group.[20] Second, the effective comparison group can include treated units. Third, β^{twfe} uses "distance" weights w^{bin}'s to aggregate across dosages. In contrast, $ATT^{\texttt{loc}}$ does not suffer from any of these issues. In applications where the researcher is targeting level-effect parameters, we recommend favoring $ATT^{\texttt{loc}}$ vis-a-vis β^{twfe}.

Parts (c) and (d) of Theorem 3.4 provide interpretations of β^{twfe} taking scaled paths of outcomes as building blocks. For part (c), $ATT(d|d)/d$ (under parallel trends) and $ATT(d)/d$ (under strong parallel trends) are "per-dosage" causal parameters. This part shows that the TWFE estimand includes negative weights under the same conditions as in part (b), though the weights integrate to one. Negative weights also appear in the TWFE estimand with a binary staggered treatment (Borusyak, Jaravel, and Spiess, 2024; de Chaisemartin and D'Haultfoeuille, 2020; Goodman-Bacon, 2021), and Theorem 3.4(c) shows that, with a continuous treatment, this drawback can arise even with two periods (i.e., no staggering).[21] The weights themselves equal $w^{lev}(d)$ weights times the dose, which creates two key differences. First, they integrate to one. Second, they weigh the building block parameters for the highest and lowest doses even more heavily than in part (a). We note that, in

homogeneity embedded in functional form restrictions, β^{twfe}, in general, will not recover $ATT(d|d)$ or $ATT(d)$.

[19] The exact expressions for the weights are $w_1^{bin}(d) = \frac{|d-\mathbb{E}[D]|}{\mathbb{E}\Big[|D-\mathbb{E}[D]|\Big|D>\mathbb{E}[D]\Big]}$ and $w_0^{bin}(d) = \frac{|d-\mathbb{E}[D]|}{\mathbb{E}\Big[|D-\mathbb{E}[D]|\Big|D\leq\mathbb{E}[D]\Big]}$. These are true weights in the sense that they additionally satisfy $\mathbb{E}[w_1^{bin}(D)|D > \mathbb{E}[D]] = \mathbb{E}[w_0^{bin}|D \leq \mathbb{E}[D]] = 1$. See Appendix SC.2 in the Supplementary Appendix for more details.

[20] To give an example of why this scaling term is undesirable in the context of summarizing level effects, suppose that a researcher rescales the dose by some constant, such as multiplying it by 100. This will not change the numerator in Equation (3.1), nor will it change the effective treated and comparison groups, nor will it change summary level effect parameters such as $ATT^{\texttt{loc}}$; however, it will change β^{twfe} through its effect on the denominator in Equation (3.1). At a higher level, all the other decompositions of β^{twfe} considered in this section (which all have weights that integrate to one) involve building blocks that reflect different notions of slopes (rather than level effects). The expression in Equation (3.1) also relates β^{twfe} to a binarized version of a slope effect.

[21] As in the binary staggered case, a larger untreated group reduces the influence of negative weights. In fact, here, if there are enough untreated observations to make $\mathbb{E}[D] < d_L$, then the weights are all positive.

the case of a discrete dose, this result corresponds to the one in Theorem S3 of the Supplementary Appendix of de Chaisemartin and D'Haultfoeuille (2020). Therefore, using "average slopes" as the underlying parameter of interest eliminates neither TWFE's potential for negative weights nor its non-intuitive weighting scheme. For part (d), when β^{twfe} is interpreted in terms of all possible 2×2 comparisons of changes of outcomes for higher dose groups relative to lower dose groups, the weights are all positive and integrate to 1, but, under parallel trends, these comparisons all mix together causal effects of the higher treatment with selection bias terms. Although strong parallel trends removes the selection bias, the weights attached to the causal parameters are still hard to interpret.

To conclude this section, it is worth pointing out the pattern that emerges from the decomposition results presented in this section. When the building block parameters are mainly level-effect parameters, as in parts (b) and (c), β^{twfe} is not affected by selection bias, but includes negative weights. Thus, β^{twfe} is not weakly causal when expressed in terms of either of these building blocks. On the other hand, when the building block parameters involve comparisons across different doses, as in parts (a) and (d), β^{twfe} has positive weights, but it includes selection bias terms under parallel trends alone. This implies that whether or not β^{twfe} is weakly causal changes depending on whether one assumes Assumption PT or Assumption SPT.

Remark 3.3 (Decomposition with no untreated units)**.** *It is straightforward to extend the TWFE decompositions discussed above to settings with no untreated units. For the causal response decomposition (part (a)), the exact same result applies with the exception that the second term involving w_0^{acrt} is equal to 0. Similarly, for the scaled 2×2 decomposition (part (d)), nothing changes except that the second term involving $w_0^{2\times 2}$ is equal to 0. For the levels decomposition and the scaled levels decomposition (parts (b) and (c)), with no untreated units, $ATT(d|d)$ (or $ATT(d)$) is not identified; instead, along the lines mentioned in Remark 3.1, instead of using the untreated comparison group, we can instead compare to the path of outcomes of the "least treated". Thus, the same decompositions continue to apply except that $ATT(l|l)$ should be replaced by $ATT(l|l) - ATT(d_L|d_L)$. This immediately means that these decompositions (in addition to negative weights) become complicated by issues related to selection bias.*

4 DiD estimators that can highlight or summarize heterogeneity

So far, we have discussed two types of average causal effects with continuous DiD designs (average level effects and average causal responses), described different assumptions to identify them (parallel trends and strong parallel trends), and shown that, as a summary of these effects, a linear TWFE coefficient suffers from at least one of three problems: negative weights, selection bias, or non-intuitive weighting schemes. In this section, we discuss how one can bypass the limitations of the TWFE regression specification in Equation (1.1) by proposing data-driven estimation procedures that target well-defined causal parameters. For simplicity, in this section, we rely on Assumption SPT so we can get all causal parameters under the same identification assumptions. If one is interested in $ATT(d|d)$ or their functionals, one can rely on Assumption PT and use the same estimation procedure for $ATT(d)$ that we discuss below.

4.1 Estimating average causal functions among the treated

We start with the estimation of the dose-specific functions, $ATT(d)$ and $ACRT(d)$ under Assumption SPT. First, recall that, from Theorem 3.3, we have that, for a positive dosage d,

$$ATT(d) = \mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=0],$$

as well as $ACRT(d) = \partial \mathbb{E}[\Delta Y|D=d]/\partial d$ when the treatment is continuous, and $ACRT(d_j) = (\mathbb{E}[\Delta Y|D=d_j] - \mathbb{E}[\Delta Y|D=d_{j-1}]) / (d_j - d_{j-1})$ when the treatment is multi-valued discrete. As $\mathbb{E}[\Delta Y|D=0]$ can be estimated using its sample analog, $\mathbb{E}_n[\Delta Y|D=0] = n_{D=0}^{-1} \sum_{i:D_i=0} \Delta Y_i$, with $n_{D=0} = \sum_{i=1}^n 1\{D_i = 0\}$, the main challenge in estimating all these functions resides in estimating $\mathbb{E}[\Delta Y|D=d]$ among treated units ($d > 0$) and its derivative.

Note that this is a standard regression problem, and, as such, researchers have different options on how to approach the problem. Examples include adopting a parametric model for $\mathbb{E}[\Delta Y|D=d]$ (e.g., assuming a quadratic model in dose among the treated), or pursuing nonparametric estimators using kernels or sieves/series. We discuss these considerations below.

For simplicity, we start with setups where the treatment is multi-valued discrete, and takes on a relatively few values. In this case, one can estimate $ATT(d_j)$ and $ACRT(d_j)$ for any positive treatment dose d_j in the dose support using a simple saturated regression

$$\Delta Y_i = \beta_0 + \sum_{j=1}^{J} 1\{D_i = d_j\}\beta_j + \varepsilon_i, \tag{4.1}$$

where we use the zero treatment dosage as the omitted category. It is straightforward to show that, under the identification assumptions in Theorem 3.3 and some weak regularity conditions, each OLS coefficient $\widehat{\beta}_j$ is a $\sqrt{n}$-consistent and asymptotically normal (nonparametric) estimator for the $ATT(d_j)$, and $\left(\widehat{\beta}_j - \widehat{\beta}_{j-1}\right) / (d_j - d_{j-1})$ is a consistent (nonparametric) estimator for $ACRT(d_j)$. Inference procedures are straightforward. Note that, in this setup, all that our regression (4.1) is doing is to automate the appropriate comparison of means justified under our identification assumptions.

When treatment doses among treated units are continuous, (4.1) becomes infeasible. Perhaps the most straightforward alternative approach is to impose a parametric functional form restriction on how ΔY varies with dosage D among treated. For instance, one can consider a quadratic model for $\Delta\widetilde{Y}_i = \Delta Y_i - \mathbb{E}_n[\Delta Y|D=0]$ with respect to treatment dose among treated units, i.e., for every observation with $D_i > 0$, we can consider the following regression specification[22]

$$\Delta\widetilde{Y}_i = \beta_0 + \beta_1 D_i + \beta_2 D_i^2 + \varepsilon_i. \tag{4.2}$$

When this putative regression specification is correctly specified, we have that a $\sqrt{n}$-consistent and asymptotically normal (CAN) parametric estimator for the $ATT(d)$ would be given by $\widehat{ATT}_{\text{par}}(d) = \widehat{\beta}_0 + \widehat{\beta}_1 d + \widehat{\beta}_2 d^2$, while a $\sqrt{n}$ CAN estimator for $ACRT(d)$ would be given by $\widehat{ACRT}_{\text{par}}(d) = \widehat{\beta}_1 + 2\widehat{\beta}_2 d$. Pointwise and uniform-in-d inference procedures are straightforward, though it is important to account for the estimation effect from estimating $\mathbb{E}[\Delta Y|D=0]$ when constructing $\Delta\widetilde{Y}_i$.[23] Of course, other parametric functional forms can also be adopted.

[22] One could also consider the regression $\Delta Y_i = \alpha + \mathbf{1}\{D>0\}\left(\beta_0 + \beta_1 D_i + \beta_2 D_i^2\right) + \varepsilon_i$ for all observations.

[23] This affects the standard error of estimators for $ATT(d)$, but not of estimators for $ACRT(d)$.

A limitation of parametric models like (4.2) is that we need to impose that it is correctly specified to guarantee reliable inference and interpretation about the causal effects of interest. Theorem 3.4 highlights potential consequences of misspecfication, suggesting that, provided that the sample size is large, researchers can use nonparametric procedures to avoid functional form restrictions. In practice, this would essentially entail considering a nonparametric regression model of $\Delta\widetilde{Y}_i$ on D_i among treated units,

$$\Delta\widetilde{Y}_i = ATT(D_i) + \varepsilon_i. \tag{4.3}$$

Here, one can adopt different nonparametric procedures, including kernel-based (Fan and Gijbels, 1996), and sieve-based estimators (Chen, 2007). As these nonparametric procedures have slower-than-$\sqrt{n}$ rates of convergence, there is no estimation effect from estimating $\mathbb{E}[\Delta Y|D=0]$. Given appropriate regularity conditions, it follows that the nonparametric estimator $\widehat{ATT}_{\text{np}}(d)$ and $\widehat{ACRT}_{\text{np}}(d) = \partial\widehat{ATT}_{\text{np}}(d)/\partial d$ are uniformly consistent and asymptotically normal (in an appropriate functional sense).

As with any nonparametric procedure, it is important to carefully choose tuning parameters—such as the bandwidth or the sieve dimension—so the resulting estimators have appealing and statistically justified guarantees. In this regard, a particularly attractive nonparametric procedure for estimating the $ATT(d)$ and $ACRT(d)$ curves is the data-driven sieve-based estimator proposed by Chen, Christensen, and Kankanala (2024). When adapted to our DiD with continuous treatment context, Chen, Christensen, and Kankanala (2024)'s results imply that the nonparametric estimators for $ATT(d)$ and $ACRT(d)$ curves converge at the fastest possible (i.e., minimax) rate in sup-norm, and lead to uniform confidence bands that are asymptotically narrower (more precise) than those based on undersmoothing, and yet have correct asymptotic coverage and contract at, or within a $\log\log n$ factor of, the minimax rate. Importantly, the data-driven procedures in Chen, Christensen, and Kankanala (2024) do not require additional knowledge of model structure, such as the smoothness of $ATT(d)$, which, in practice, is ex-ante unknown. This approach adapts to these unknown model regularities, yields estimators and confidence bands with strong statistical guarantees, and, at the same time, is easy to implement.

Implementing Chen, Christensen, and Kankanala (2024) is indeed very simple. In fact, it resembles (4.2) in the sense that, upon computing the optimal sieve-dimension $\widehat{K}$, it involves running a linear regression of ΔY on flexible $\widehat{K}$-dimensional transformations of D (cubic B-splines), $\psi^{\widehat{K}}(D)$, in the subsample of units with $D_i > 0$,

$$\Delta\widetilde{Y}_i = \psi^{\widehat{K}}(D)'\beta_{\widehat{K}} + \varepsilon_i, \tag{4.4}$$

and then forming the nonparametric estimators for $ATT(d)$ and $ACRT(d)$ as

$$\widehat{ATT}_{\text{cck}}(d) = \left(\psi^{\widehat{K}}(d)\right)'\widehat{\beta}_{\widehat{K}}, \qquad \widehat{ACRT}_{\text{cck}}(d) = \left(\partial\psi^{\widehat{K}}(d)\right)'\widehat{\beta}_{\widehat{K}}, \tag{4.5}$$

where $\partial\psi^K(s) = (d\psi_{K1}(s)/\,ds, \ldots, d\psi_{KK}(s)/\,ds)'$, and $\widehat{\beta}_{\widehat{K}}$ is the $\widehat{K}$-dimension vector of OLS estimators for $\beta_{\widehat{K}}$. In Appendix B, we describe how to compute $\widehat{K}$, as well as how to construct uniform confidence bands based on $\widehat{ATT}_{\text{cck}}(d)$ and $\widehat{ACRT}_{\text{cck}}(d)$ that have asymptotically corrected cover-

age over a large (and generic) class of data-generating processes. We adopt this procedure in our application.

One can also use alternative nonparametric estimators—such as the bias-corrected local-polynomial estimators proposed by Calonico, Cattaneo, and Farrell (2018)—and choose tuning parameters using alternative criteria functions, e.g., using a cross-validation or a penalization procedure (Chetverikov, 2024). These procedures, however, may have different statistical guarantees.

4.2 Estimating summary measures of treatment effects

Researchers frequently want to report summary estimates to enhance interpretability and/or statistical precision, or because a lower-dimensional parameter is an input into some model or post-estimation calculation. As we showed in Section 3, however, the predominant method for estimating such summary estimates, a linear TWFE regression, generally does not average across dose-specific parameters with intuitive weights. In this section, we discuss estimation of $ATT^{\texttt{glob}}$ and $ACRT^{\texttt{glob}}$, which are summary causal effect parameters that have a clear interpretation. As above, if one maintains PT rather than SPT, then the same estimation procedures discussed belows for $ATT^{\texttt{glob}}$ can be used for $ATT^{\texttt{loc}}$.

When there are untreated units, part (a) of Corollary 3.1 suggests an extremely simple and familiar estimator of $ATT^{\texttt{glob}}$: the difference between the average change in outcomes among treated units minus the average outcome change for untreated units. This "binarized" DiD estimator can be obtained from the following simple linear regression specification:

$$\Delta Y_i = \beta_0^{bin} + D_i^{>0}\beta^{bin} + \epsilon_i, \tag{4.6}$$

where $D_i^{>0} = 1\{D_i > 0\}$ is a dummy variable for the dose being greater than zero, β_0^{bin} and β^{bin} are (unknown) finite-dimensional parameters, and ϵ_i is the error term. It is straightforward to show that under the identification assumptions in Corollary 3.1, $\beta^{bin} = ATT^{\texttt{glob}}$, implying that, under some standard regularity conditions, one can estimate and make (asymptotically valid) inferences about $ATT^{\texttt{glob}}$.[24] Note that this estimator applies in the same way to continuous and multi-valued discrete treatments.

Aggregated average causal response parameters can be constructed easily by weighting the estimated average causal functions across doses using the dose distribution itself. This solves the problem with TWFE's weighting scheme. For discrete treatments, it is straightforward to aggregate these $ACRT(d)$'s based on the coefficients from (4.1) to form a plug-in estimator for the $ACRT^{\texttt{glob}}$, using the identification formula in Corollary 3.1(c),[25] i.e.,

$$\widehat{ACRT}^{\texttt{glob}} = \sum_{j=1}^{J} \frac{\widehat{\beta}_j - \widehat{\beta}_{j-1}}{d_j - d_{j-1}} \widehat{\mathbb{P}}(D = d_j | D > 0), \tag{4.7}$$

[24] Regularity conditions include bounded second moments, and $\mathbb{P}(D = 0)$ and $\mathbb{P}(D > 0)$ being uniformly bounded away from zero. If one wishes to cluster the standard errors at a higher level than i, there should also be sufficiently many treated ($D > 0$) and untreated ($D = 0$) clusters to justify the application of a Central Limit Theorem; see Roth, SantAnna, Bilinski, and Poe (2023) for a discussion.

[25] When one imposes the PT Assumption PT instead of the SPT assumption SPT, each $\widehat{\beta}_j$ is a consistent estimator for the $ATT(d_j|d_j)$. However, comparison across $\widehat{\beta}_j$ does not give an $ACRT$-type parameter, as indicated in Theorem 3.2.

where $\widehat{\mathbb{P}}(D = d_j|D > 0) = \sum_{i=1}^{n} 1\{D_i = d_j\}/\sum_{i=1}^{n} 1\{D_i > 0\}$. It follows from the delta method, our identification assumptions, and some weak regularity conditions that, as the sample size increases, $\sqrt{n}\left(\widehat{ACRT}^{\texttt{glob}} - ACRT^{\texttt{glob}}\right)$ converges to a normal distribution with mean zero and estimable asymptotic variance, implying that standard inference procedures can be reliably used when treatments are multi-valued discrete. One can follow a similar strategy when using the scaled $ATT(d)$ as the "building block" of the aggregation.

A similar approach applies to estimating $ACRT^{\texttt{glob}}$ with a continuous dose. Our proposed estimator is simple to compute as it is based on the plug-in principle, i.e.,

$$\widehat{ACRT}^{\texttt{glob}} = \mathbb{E}_n\left[\widehat{ACRT}(D)\Big| D > 0\right] = \frac{1}{n_{D>0}} \sum_{i:D_i>0} \widehat{ACRT}(D_i),$$

with $n_{D>0} = \sum_{i=1}^{n} 1\{D_i > 0\}$ denoting the sample size with a positive dose, and $\widehat{ACRT}(D)$ being a parametric or nonparametric estimator. Under some regularity conditions,[26] one can show that $\widehat{ACRT}^{\texttt{glob}}$ is $\sqrt{n_{D>0}}$ consistent and asymptotically normal; see, e.g., Section 4.1 of Ai and Chen (2007) and Theorem 3.1 of Newey and Stoker (1993) for a discussion involving nonparametric estimators.[27]

We close this section by noticing that it is also possible to consider alternative estimators for $ACRT^{\texttt{glob}}$ using a so-called Neyman-Orthogonal moment representation. More precisely, by exploring the efficient influence function for $ACRT^{\texttt{glob}}$ implied by Theorem 3.1 of Newey and Stoker (1993), it is straightforward to show that

$$ACRT^{\text{glob}} = \mathbb{E}\left[ACRT(D) - (\Delta Y - \mathbb{E}[\Delta Y|D, D > 0])\,\frac{f'_{D|D>0}(D)}{f_{D|D>0}(D)}\Big| D > 0\right]. \tag{4.8}$$

Based on this representation, one can then use flexible nonparametric or machine-learning-based estimators for the nuisance functions and still conduct asymptotically valid inference procedures. This opens the door for leveraging double machine learning procedures to estimate $ACRT^{\text{glob}}$ in DiD contexts; see Ahrens et al. (2025) for a discussion. We leave this topic for future research.

5 Extensions

In this section, we briefly summarize several extensions of our main results that are further discussed in the Appendix and Supplementary Appendix.

[26] With nonparametric methods, these conditions involve deterministic choices of tuning parameters such as bandwidths and sieve dimensions. An interesting topic for future research is to assess the potential impact of data-driven tuning parameters when studying the asymptotic properties of $\widehat{ACRT}^{\texttt{glob}}$. In our application, we abstract from this.

[27] For nonparametric estimation, it is common to require that the density of the treatment dosage among the treated units is uniformly bounded away from zero. If such a condition is not satisfied, one may not guarantee parametric rates of convergence. In such cases, a possibility is to use self-normalizing inference procedures similar to Khan and Tamer (2010), Chen and Liao (2014), and Chen, Liao, and Sun (2014). See also de Chaisemartin, D'Haultfoeuille, and Vazquez-Bare (2024) for a related discussion involving "quasi-stayers". We leave a formal treatment of this topic for future research.

5.1 Relaxing Strong Parallel Trends

Under traditional DiD assumptions, Assumption PT led to the identification of local $ATT(d|d)$ parameters that are difficult to compare across dosages. On the other hand, the strong parallel trends assumption led to $ATT(d)$ parameters. These can be seen as extreme cases, and it is possible to trade off the strength of assumptions with the type of parameters that can be identified in different ways. The number of these intermediate possibilities is large, however. Here, we sketch what we consider to be three main ideas to relax strong parallel trends. Appendix SD of the Supplementary Appendix provides substantially more detail.

First, in many cases, researchers may be willing to assume that they know the direction of the selection bias. For example, suppose that a researcher is willing to assume that, for all d and any dose groups $l < h$, $ATT(d|l) \leq ATT(d|h)$, i.e., that higher dose groups would experience larger treatment effects at any value of the dose. In the Supplementary Appendix, we show that this type of assumption leads to bounds on causal effect parameters without requiring strong parallel trends. For example, it implies that, for all d

$$ACRT(d|d) \leq \frac{\partial \mathbb{E}[\Delta Y|D=d]}{\partial d},$$

which provides a bound on $ACRT(d|d)$. See Proposition S7 in the Supplementary Appendix for more details.

A second possibility for relaxing strong parallel trends is to define a sub-region $\mathcal{D}_s \subseteq \mathcal{D}_+$ for which strong parallel trends holds, i.e., to assume that

$$\mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D \in \mathcal{D}_s] = \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D=d] \tag{5.1}$$

holds for all $d \in \mathcal{D}_s$. Under this assumption, we show in Proposition S8 in the Supplementary Appendix that, for $h, l \in \mathcal{D}_s$,

$$\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=l] = \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D \in \mathcal{D}_s].$$

In other words, comparing the trends in outcomes over time for dose group h to dose group l delivers the average causal effect of dose h relative to dose l among those dose groups in $\mathcal{D}_s$. Under PT, the same comparison would include selection bias terms. While the assumption in Equation (5.1) is weaker than SPT, the tradeoff is that now the only comparisons that have a causal interpretation are for dose groups within the set $\mathcal{D}_s$. In some applications, this assumption could be notably weaker than Assumption SPT—in fact, this assumption should, at least arguably, no longer be called "strong parallel trends" because it is non-trivially non-nested with Assumption PT. It could also be more plausible in some applications than PT. For example, suppose that $\mathcal{D}_s$ contains dose groups that experienced large values of the treatment. The assumption above says that, for units that experience similar doses, we could learn about the trend in outcomes over time at a counterfactual dose by looking at the trend in outcomes for the dose group that experienced this dose. This is a different type of assumption, which could be more plausible than assuming that these dose groups would have experienced the same trend in untreated potential outcomes as the untreated group did (where untreated units may be substantially different from units that experience large values of the

treatment). This local version of the SPT assumption might be appealing in applications where there is substantial variation in the dose and the researcher is willing to assume that there is no selection bias among units that selected similar doses, but the researcher is unwilling to assume that there is no selection bias among units that select substantially different doses.[28]

Finally, in some applications, strong parallel trends may be more plausible after conditioning on some observed covariates X. Under a version of strong parallel trends conditional on covariates, one can show that the conditional average treatment effect, $ATT_x(d) = \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(0)|X = x, D > 0]$, is identified. Since this parameter is not local to dose group d, conditional on $X = x$, one can compare $ATT_x(d)$ across different values of the dose without inducing selection bias terms. This is an intermediate case, however, in that these are more local parameters than $ATT(d)$ because they are local to the particular value of the covariates x. See the discussion in Appendix SD in the Supplementary Appendix for more details.

5.2 Multiple time periods and variation in treatment timing

Although our results so far focus on two-period cases, it is straightforward to extend them to setups with multiple time periods and variation in treatment timing across units by combining the ideas discussed in Section 3.2 with those in Callaway and Sant'Anna (2021). We consider this setting in detail in Appendix C.

In a setting with staggered treatment adoption (i.e., where once a unit becomes treated with dose d, that unit remains treated with dose d in subsequent periods), knowing the time period that a unit becomes treated with a positive dose (which we denote by G_i and refer to as a unit's *timing group*) and dose D_i (i.e., dose group) fully characterizes a unit's sequence of treatments across all periods. In this context, we need to augment our potential outcomes terminology and write $Y_{i,t}(g, d)$ as the potential outcome of unit i at time t if it were first treated in period g, with dose d; we write $Y_{i,t}(0) = Y_{i,t}(\infty, 0)$ to denote a unit's untreated potential outcome—the potential outcome in time period t if that unit did not participate in the treatment in any available period. With this notation at hand, we can define a multi-period analog of $ATT(d|d)$ as

$$ATT(g, t, d|g, d) = \mathbb{E}[Y_t(g, d) - Y_t(0)|G = g, D = d] \quad \text{and} \quad ACRT(g, t, d|g, d) = \left.\frac{\partial ATT(g, t, l|g, d)}{\partial l}\right|_{l=d}$$

which are the average treatment effect and average causal response in period t of (i) becoming treated in period g and (ii) experiencing dose d among those in timing group g and dose group d.

Under no anticipation and a multiple-period version of the parallel trends assumption, we show in Appendix C that, in post-treatment periods (i.e., periods where $t \geq g$)

$$ATT(g, t, d|g, d) = \mathbb{E}[Y_t - Y_{g-1}|G = g, D = d] - \mathbb{E}[Y_t - Y_{g-1}|G = \infty, D = 0]. \tag{5.2}$$

[28] A related intermediate assumption between Assumption PT and Assumption SPT would be to directly assume that the selection bias term in Theorem 3.2 (i.e., $\partial ATT(d|l)/\partial l|_{l=d}$) is equal to 0. This would imply that $ACRT(d|d)$ is identified. In applications where the target parameter is $ACRT(d|d)$ for a particular d, this is a notably weaker assumption than Assumption SPT. In applications where the target parameter is $ACRT(d|d)$ for all $d \in \mathcal{D}_+$, this assumption is still mechanically weaker than Assumption SPT though, to our knowledge, economic models that imply this condition (across all values of d) typically also imply strong parallel trends.

The argument is similar to the two-period case discussed earlier. The main difference is that the expression above involves the "long difference" in changes in outcomes over time, i.e., from period $g-1$ to t. The reason for this difference is that $g-1$ is the most recent period for which units in group g were untreated. The expression above uses the never-treated group ($G = \infty$) as the comparison group, but, like the case with a binary treatment, one can use alternative comparison groups such as the not-yet-treated. Under a multiple-period version of the strong parallel trends assumption, one can take the derivative of the right-hand side of Equation (5.2) with respect to d to identify $ACRT(g, t, d|g, d)$.

One complication that arises in the staggered case is that $ATT(g, t, d|g, d)$ and $ACRT(g, t, d|g, d)$ are often relatively high-dimensional objects that can be hard to report (and perhaps hard to estimate precisely). In Appendix C, we discuss two main strategies for aggregating these parameters into lower-dimensional objects. First, we average across timing groups and time periods to target causal effect parameters that are a function of only the dose: $ATT^{dose}(d|d)$, and $ACRT^{dose}(d|d)$—these parameters highlight heterogeneous effects across different doses and are analogous to $ATT(d|d)$ and $ACRT(d|d)$ in the two-period case that we have emphasized above. They can be averaged across the dose to deliver scalar summary parameters. Second, we consider event-study parameters: $ATT^{es}_{\texttt{loc}}(e)$, and $ACRT^{es}_{\texttt{loc}}(e)$ that average across the dose and highlight how treatment effects and/or causal responses vary with the length of exposure to the treatment—these parameters are the event study analog of $ATT^{\texttt{loc}}$ and $ACRT^{\texttt{loc}}$ in the two period case above. See Callaway, Goodman-Bacon, and Sant'Anna (2024) for alternative, intermediate aggregations. The discussion here focuses on causal effect parameters that are local to a specific dose group and timing group, but, like the two-period case discussed above, it is also possible to recover causal effect parameters across all treated units under strong parallel trends; see Appendix SA in the Supplementary Appendix for more details.

5.3 Interpreting TWFE Regressions with Multiple Periods/Groups

In Appendix SA.3 of the Supplementary Appendix, we also extend our TWFE decomposition results from Theorem 3.4 to cover setups beyond the two-period case, including setups with staggered treatment adoptions with continuous or multi-valued discrete treatments. These results generalize the decompositions in de Chaisemartin and D'Haultfoeuille (2020) and Goodman-Bacon (2021) to the case of a continuous treatment. Those results demonstrate that TWFE regressions with multiple periods and variation in treatment timing (i) continue to suffer from the weighting and selection bias issues that we highlighted in Theorem 3.4, (ii) inherit weighting issues (including possible negative weights) that are prevalent in TWFE regressions with binary, staggered treatment adoption, and (iii) are affected by violations of parallel trends in pre-treatment periods.

5.4 Event-Study and Pre-Treatment Differences

When there are multiple periods of data available, DiD applications typically assess the plausibility of the parallel trends assumption by checking whether or not parallel trends holds in pre-treatment periods. In a setting with a continuous treatment, one can check whether or not $\mathbb{E}[\Delta Y_t|D = d] =$

$\mathbb{E}[\Delta Y_t|D=0]$ holds for all pre-treatment time periods t and all d. Implementing this test, however, can be complicated because it involves multiple dose-response nonparametric estimates. A convenient alternative is to report aggregated event study parameters such as $ATT^{es}_{\texttt{loc}}(e)$ or $ACRT^{es}_{\texttt{loc}}(e)$ in pre-treatment periods (i.e., $e<0$). Plotting estimates of $ATT^{es}_{\texttt{loc}}(e)$ and $ACRT^{es}_{\texttt{loc}}(e)$ for pre-treatment periods ($e<0$) can be used to assess the plausibility of parallel trends.[29] We report these for our empirical application in Figures 8 and 10.

Assessing the plausibility of parallel trends using these event-study-type aggregations is probably a good default option for empirical work, though we note that one possible drawback of this test is that there are violations of the parallel trends assumption that these event-study versions of the test would not detect.[30] Another possible drawback is related to lack of power. See, e.g., Roth (2022).

6 Continuous DiD in Practice: Causal Effects of Medicare PPS

We have so far shown that the causal question of interest shapes identification in a continuous DiD design and argued that it should guide the estimation approach, too. We now apply our preferred average level treatment effect and average causal response estimators to Acemoglu and Finkelstein (2008)'s study of Medicare PPS, discuss their interpretation, and contrast them with TWFE estimates. To start our discussion and map it into our baseline results, we consider the balanced panel data component of Acemoglu and Finkelstein (2008), which is comprised of 5881 hospitals, and also (time) average all pre-treatment outcomes and post-treatment outcomes to map into our two-period setup. Thus, we use $t=1$ to denote the average of pre-treatment periods (1980-1983), and $t=2$ to denote the average of post-treatment periods (1984-1986). Later, we discuss how one can leverage the time dimension further to assess the plausibility of the identification assumptions and highlight treatment effect dynamics. We also denote treatment dose here by m instead of d, as m is a short-hand notation for Medicare inpatient share that determines treatment exposure in the AF application.

To begin, consider the profit maximization problem for a hospital with Medicare inpatient share M. We follow AF and assume a production function, $F_t(L,K)$, that is homothetic in labor (L) and capital (K). Market wages and rental rates are normalized by the output price, and Medicare subsidies mean that net input prices are $(1-s_{L,t}M)w$ and $(1-s_{K,t}M)r$. Firms consider the following profit maximization problem:

[29] An interesting (though subtle) point is that in cases where an aggregate level effect such as $ATT^{\texttt{loc}}$ or its event study version $ATT^{es}_{\texttt{loc}}(e)$ is the target parameter of the analysis, it is possible to recover it under "weaker" parallel trends assumptions that allow for violations of parallel trends where the *average* violation of parallel trends across dose groups is equal to zero (rather than the violation of parallel trends being equal to zero for all dose groups)—we refer to the corresponding averaged version of parallel trends as aggregate parallel trends. If one maintains aggregate parallel trends, then only $ATT^{es}_{\texttt{loc}}(e)$ (and not, e.g., $ACRT^{es}_{\texttt{glob}}(e)$) is relevant for assessing its plausibility using pre-treatment periods. That being said, it is debatable whether or not the violations of parallel trends that can be allowed for under aggregate parallel trends should be counted as evidence against the design. See an earlier working version of our paper (Callaway, Goodman-Bacon, and Sant'Anna, 2025) for a more detailed discussion of this point.

[30] This approach does have advantages over TWFE alternatives. If one runs a sequence of placebo regressions in pre-treatment periods, the weighting issues in Section 3.3 apply. Alternatively, relative to the common empirical practice of estimating an event-study version of the TWFE regression in Equation (1.1), in light of the results in Sun and Abraham (2021) in a setting with a binary treatment, we conjecture that the event-study coefficients could additionally include effects at different lengths of exposure to the treatment. See also Goldsmith-Pinkham, Hull, and Kolesár (2024).

$$\max_{L,K} F_t(L,K) - (1 - s_{L,t}M)wL - (1 - s_{K,t}M)rK.$$

The solution to this problem generates factor demands and a capital-labor ratio that is only a function of the input price ratio, $k_t^*\left(\frac{(1-s_{L,t}M)w}{(1-s_{K,t}M)r}\right)$. We write the subsidy ratio, $\frac{(1-s_{L,t}M)}{(1-s_{K,t}M)}$ as $1 + S_t(M) = 1 + \frac{(s_{K,t}-s_{L,t})M}{1-s_{K,t}M}$. This reflects the fact that hospitals with no Medicare patients ($M = 0$), and all hospitals before PPS (when $s_{K,t=1} = s_{L,t=1} = s$) face no relative price distortion. PPS set $s_{L,t} = 0$ in 1983, making $S_{t=2}(M) = \frac{s_{K,t=2}M}{1-s_{K,t=2}M}$.

This structure allows us to define the capital-labor ratio potential outcomes in terms of Medicare inpatient share M:

$$Y_{t=1} = Y_{t=1}(0) = k_{t=1}^*\left(\frac{w}{r}\right)$$

$$Y_{t=2} = Y_{t=2}(M) = k_{t=2}^*\left((1 + S_{t=2}(M))\frac{w}{r}\right)$$

Three details of the theoretical setup are worth noting. First, homotheticity allows us to connect potential outcomes as a function of M to a firm's optimal capital-labor ratio as a function of relative prices (as a function of M). Without this assumption, a hospital's scale affects its input mix, and capital-labor ratios are a function of net labor and capital prices separately, complicating the theoretical interpretation of causal parameters. Second, we define our parameters of interest in terms of causal effects of M on Y. A structural interpretation of those parameters in terms of k^* necessarily involves the non-linear way in which M changes the subsidy ratio, $S_t(M)$ (as well as a kind of exclusion restriction that rules out direct effects of M on outcomes). Third, we use time subscripts to match the fact that PPS changed over time, but this is not a dynamic model. The assumed lack of forward-looking behavior implies the no anticipation assumption (Assumption 3) and allows us to write $Y_{t=1} = Y_{t=1}(0)$. All these details are in line with AF's theoretical model.

6.1 Causal Questions Around Medicare PPS

AF is primarily interested in the question: did PPS raise capital-labor ratios? PPS sought to help hospitals invest in new medical technologies with the aim of improving patient outcomes (Office of Technology Assessment, 1984). But regulators also worried about the "incentive for hospitals to adopt expensive capital equipment that reduces operating costs but raises total costs per case" (Office of Technology Assessment, 1984, p. 14). Thus, Medicare's role in technology investments has major policy implications. Moreover, the theoretical model predicts that PPS would raise capital-labor ratios for all treated hospitals, so the sign of its effects is a test of a simple neoclassical production theory. The building block parameters that answer these questions are the average treatment effect of PPS on hospitals with $M = m$:

$$ATT(m|m) = \mathbb{E}[Y_{t=2}(m) - Y_{t=2}(0)|M = m] = \mathbb{E}\left[k_{t=2}^*\left((1 + S_{t=2}(m))\frac{w}{r}\right) - k_{t=2}^*\left(\frac{w}{r}\right)\middle| M = m\right].$$

Estimating and plotting the entire $ATT(m|m)$ function shows which hospitals responded most to PPS and tests the prediction that *all* treated hospitals increase their capital intensity. Under parallel trends alone, it is not possible to discern whether that heterogeneity comes directly from subsidy

differences or from treatment effect heterogeneity, i.e., one cannot compare across $ATT(m|m)$'s. Averaging this function across treated hospitals yields $ATT^{\texttt{loc}} = \mathbb{E}[ATT(M|M)|M > 0]$, a summary parameter that directly answers the question "did PPS raise capital-labor ratios on average?"

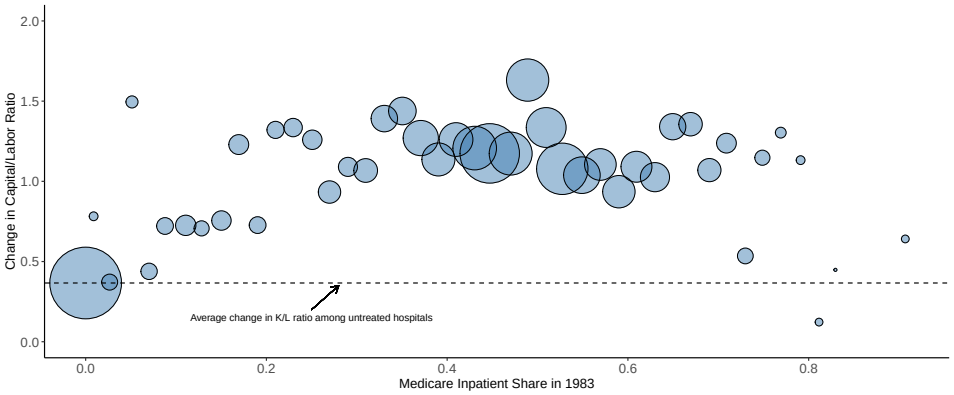
One may also be interested in which subsidy levels have larger causal effects. For example, if technologies are "lumpy", then hospitals may not respond to subsidies too small to cover the minimum investment costs. Improving the design of input subsidies thus requires causal estimates of the responsiveness to different subsidy levels. The causal effects of marginal changes in the subsidy ratio also represent another test of the theoretical model because they are proportional to a hospital's elasticity of substitution, $\sigma_{i,t}(m) = \frac{k^{*\prime}_{i,t}}{k^{*}_{i,t}} \times (1 + S_t(m)) \times \frac{w}{r}$, which, with two inputs, must be positive. The building block parameters that answer these questions are the average causal responses of PPS:[31]

$$\begin{aligned} ACRT(m) = \mathbb{E}\big[Y'_{t=2}(m)\big|M > 0\big] &= \mathbb{E}\left[k^{*\prime}_{t=2}\left((1 + S_{t=2}(m))\frac{w}{r}\right)S'_{t=2}(m)\frac{w}{r}\middle|M > 0\right] \\ &= \mathbb{E}\left[\sigma_{t=2}(m)k^{*}_{t=2}\left((1 + S_{t=2}(m))\frac{w}{r}\right)\frac{s_k}{1 - s_k m}\middle|M > 0\right] \end{aligned} \tag{6.1}$$

Again, reporting estimates of the entire $ACRT(m)$ function highlights heterogeneity in how hospitals respond to subsidies, and the summary parameter $ACRT^{\texttt{glob}}$ provides a single measure of how much hospitals respond on average to small subsidy differences.

Before turning to our formal estimates, Figure 4 presents a binned scatter plot of the change in mean capital-labor ratios before (1980-1983) and after (1984-1986) PPS against the Medicare share of inpatient days in 1983, m. Following AF, we measure the capital-labor ratio using the depreciation share of total costs.

Figure 4: Changes in Capital-Labor Ratios before and after 1983 versus the Medicare Inpatient Share



Notes: The figure presents a binned scatter plot of the change in the average depreciation share (capital-labor ratio) between the periods 1980-1983 and 1984-1986 for hospitals in 2-percentage-point bins of the 1983 Medicare inpatient share, M. In the lowest bin, hospitals with $M = 0$ are plotted separately from hospitals with $M \in (0, 0.02]$. We also consider a single bin for all hospitals with $M > 0.84$.

The horizontal line equals the mean change in capital-labor ratio for untreated hospitals (0.37). Each circle is the mean outcome change for a given bin of the Medicare inpatient share, with their size proportional to the number of hospitals in that bin. Almost all groups of treated hospitals had stronger growth in capital intensity than untreated hospitals, consistent with the theoretical prediction. The

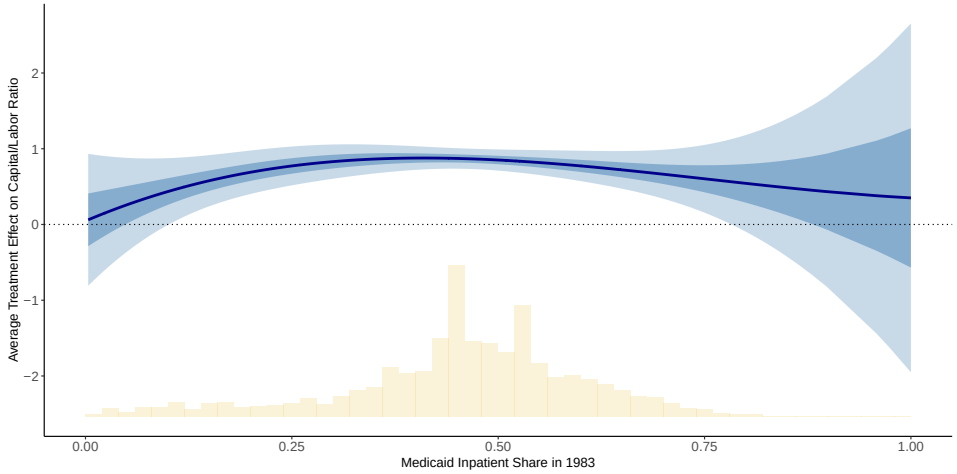
[31] Equation (6.1) follows from the definition of $\sigma_{i,t}(m)$ and the fact that $\frac{S'_t(m)}{1+S_t(m)} = \frac{s_k}{1-s_k m}$.

relationship is nonlinear, however, which indicates heterogeneity in average treatment effects, at least, and perhaps heterogeneity in the sign of average causal responses.

6.2 Average Treatment Effects of PPS

Figure 5 presents our proposed data-adaptive nonparametric estimates of $ATT(m|m)$ based on (4.5). For inference, we cluster at the hospital-level. Our data-driven procedure to optimally choose the sieve dimension selected $\widehat{K} = 4$. These estimates formalize what the scatter plot suggests: that $ATT(m|m)$ is positive. We plot pointwise 95% confidence intervals in the dark-shaded region and the wider (honest) 95% uniform confidence bands in the light-shaded region. We do not detect an effect for values of m below 5 percent, but we reject zero for doses between 0.05 and 0.78, which contains 96 percent of treated hospitals. Significant values of $\widehat{ATT}(m|m)$ range from about 0.44 percentage points at $m = 0.1$ to 0.88 percentage points at $m = 0.41$. The average across all doses ($\widehat{ATT}^{\texttt{loc}}$) is 0.80 (s.e. = 0.05), or about 18 percent of the 1983 mean outcome (measured by the depreciation share) of 4.5. This evidence suggests that PPS substantially raised capital-labor ratios.

Figure 5: Nonparametric Estimates of $ATT(m|m)$ for Medicare PPS



Notes: The figure plots nonparametric estimate of $ATT(m|m)$ that adapts the Chen, Christensen, and Kankanala (2024) data-driven estimator to our context, as discussed in Section 4.1 and Appendix B. The dark-shaded region is the 95-percent point-wise confidence interval, and the lighter-shaded region is the 95-percent honest and sup-norm rate-adaptive uniform confidence band. We display the histogram of the treatment dose among the treated in yellow.

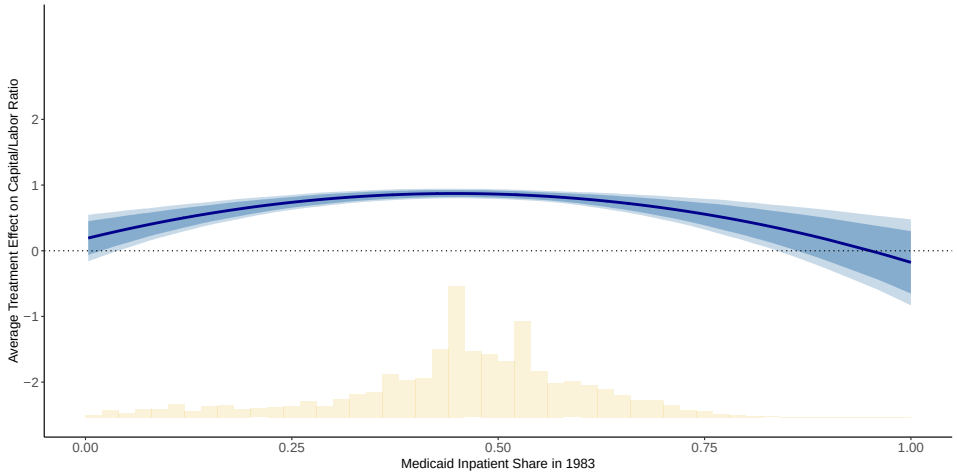
For comparison, we report in Figure 6 parametric estimates for $ATT(m|m)$ that use the quadratic regression specification in Equation 4.2. Different from Figure 5, the interpretability of $\widehat{ATT}_{\text{par}}(m|m)$ in Figure 6 depends on the quadratic specification being correctly specified. When we know that is the case, it is clear from Figure 6 that this results in substantially more precise estimates, as they now fully leverage the functional form. Importantly, these gains in precision are more substantial in the regions where data for particular treatment doses are scarce, e.g., for treatment doses above 0.75.[32] The rationale for this is very simple: parametric models are good at extrapolating, whereas

[32]Overall, we have 4987 observations with a positive treatment dose. Among these, only 57 have a treatment dose above 0.75, 20 above 0.80, and 3 above 0.90.

nonparametric procedures are more cautious about it. The reliability of the extrapolation, once again, crucially depends on the parametric model for $ATT(m|m)$ being correctly specified.

Although gains in precision are desirable, we caution against using nonparametric results to pick a parametric specification. This, to some extent, resembles a pre-testing problem, and inference based on the parametric model could be misleading. In fact, the appeal of the uniform confidence bands from Chen, Christensen, and Kankanala (2024) that we report in light-shaded blue in Figure 5 is that they account for this type of pre-testing issue and are honest, i.e., they are guaranteed to have asymptotically correct coverage over a large (and generic) class of data-generating processes. The uniform confidence bands in Figure 6 are uniform only in treatment dosage, highlighting that it reflects a narrower type of uncertainty than those in Figure 5. Henceforth, as we find it challenging to ex ante motivate a parametric functional form for $ATT(m|m)$ using arguments grounded in economic theory, we focus our attention on our nonparametric estimators.

Figure 6: Parametric Estimates of $ATT(m|m)$ for Medicare PPS using quadratic specification



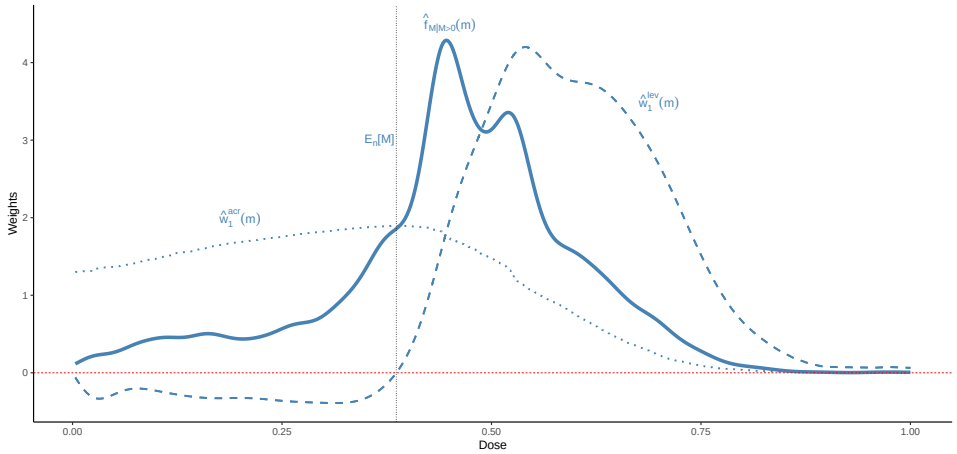
Notes: The figure plots parametric estimate of $ATT(m|m)$ that use the quadratic regression specification in Equation 4.2. The dark-shaded region is the 95-percent point-wise confidence interval, and the lighter-shaded region is the 95-percent uniform-in-treatment-dose confidence band. We display the histogram of the treatment dose among the treated in yellow. We use the same y-scale as in Figure 5.

In Section 3.3, we argued that β^{twfe} should not be relied upon to summarize level effects. However, the TWFE coefficient is 1.14—fairly similar to our estimate of $ATT^{\texttt{loc}}$. What accounts for their similarity? One explanation comes from Equation (3.1). The numerator compares weighted averages of the paths of outcomes for the "effective" treated group (those with above-average doses) to the "effective" comparison group (those with below-average doses). However, in our example, slightly more than half of the weight on paths of outcomes in the effective comparison group falls on hospitals with a positive dose. That these treated hospitals show up in the effective comparison group biases β^{twfe} downward relative to $ATT^{\texttt{loc}}$—our estimate of the numerator in Equation (3.1) is 0.60. In contrast, the "weighted distance" between the effective treated and comparison groups in the denominator of Equation (3.1) is estimated to be 0.53—that this is less than 1 is a byproduct of our setting where we measure a hospital's dose on a scale of 0 to 1.[33] Dividing by 0.53 results in our estimate of β^{twfe}

[33] If we instead were to code a hospital's dose on a scale of 0 to 100, our estimate of β^{twfe} shrinks to $0.0114 = 1.14/100$

being upward biased. That these two biases work in opposite directions and have similar magnitudes in our particular application happens to result in $\widehat{\beta}^{twfe}$ being fairly close to $\widehat{ATT}^{\texttt{loc}}$.[34]

Figure 7: Weighting Schemes for TWFE and Dose Distribution Among Treated



Notes: The dashed lines are the weights that TWFE puts on $ATT(m|m)$ and $ACRT(m)$ parameters, as in Theorem 3.4. The solid line is a smoothed estimate of the density of the Medicare inpatient share, M.

Figure 5 abstracts from dynamics since it is based on average outcomes in the pre- and post-treatment periods. As an alternative, Figure 8 plots estimates of event-study summary parameters, $ATT^{es}_{\texttt{loc}}(e) = \mathbb{E}[Y_{t=e} - Y_{t=1983}|D > 0] - \mathbb{E}[Y_{t=e} - Y_{t=1983}|D = 0]$, using 1983 as the baseline year. The patterns are similar to the TWFE event-study in Figure 1, but their magnitudes reflect proper averages of year-specific $ATT(m|m)$ parameters.[35]

6.3 Average Causal Responses to PPS

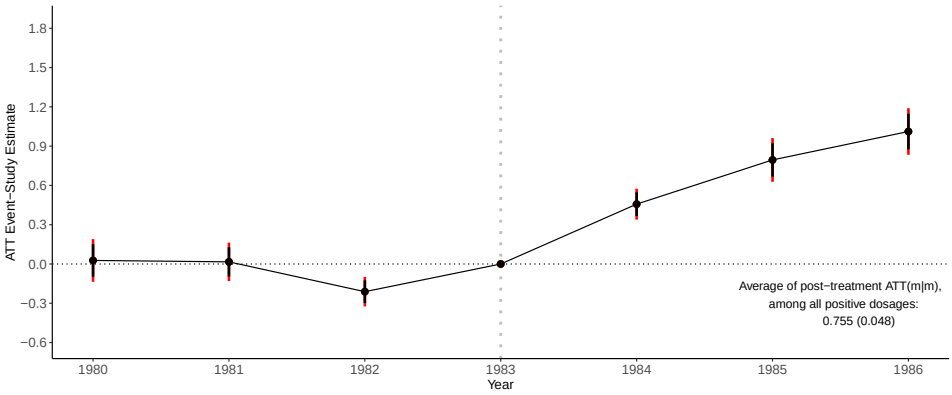
Figure 9 plots our proposed data-adaptive nonparametric estimate of the slope of the function estimated in Figure 5. Under Assumption SPT, the function in Figure 5 is the $ATT(m)$ and its slope in Figure 9 equals the $ACRT(m)$. The hump shape in Figure 5 is reflected in an $ACRT(m)$ function that starts positive, and declines through most of its support. We estimate negative $ACRT(m)$ parameters for doses above $m = 0.41$, a range that includes 71 percent of treated hospitals. The 95% uniform confidence band covers zero everywhere, although we are able to detect positive $ACRT(m)$

while our estimate of $ATT^{\texttt{loc}}$ remains unchanged.

[34] Another way to think through the difference between $\widehat{\beta}^{twfe}$ and $\widehat{ATT}^{\texttt{loc}}$ comes from mapping the estimates of $ATT(m|m)$ in Figure 5 to the level weights, $\widehat{w}_1^{lev}(m)$, provided in Figure 7. The negative weights reflect the same issue as using units actually treated in the effective *comparison* group discussed above. The scaling issues (from the denominator in Equation (3.1)) are more subtle. In Corollary S2 in the Supplementary Appendix, we show that the positive weights do not integrate to one (neither do the negative weights integrate to negative one), rather they integrate to the reciprocal of the denominator in Equation (3.1). This results in an analogous scaling effect that, in this particular application, contributes to $\widehat{\beta}^{twfe}$ being upward biased for $ATT^{\texttt{loc}}$.

[35] The negative pre-PPS coefficient may reflect the fact that PPS was passed in April 1983 and partially took effect in that calendar year, and also that hospitals report labor and capital costs for different fiscal years. Therefore, some 1983 outcomes may include post-treatment months. The results also show that the $ATT^{es}_{\texttt{loc}}(e)$ grows each year following PPS, which matches the fact that PPS' subsidy reforms actually phased in over three years. We also note, however, that these can represent other types of violations of parallel trends.

Figure 8: Event-Study Estimates of ATT



Notes: The figure plots the event-study estimates of $ATT^{es}_{\texttt{loc}}(e)$, with their 95% pointwise confidence intervals reported in black, and the 95% uniform confidence bands reported in red.

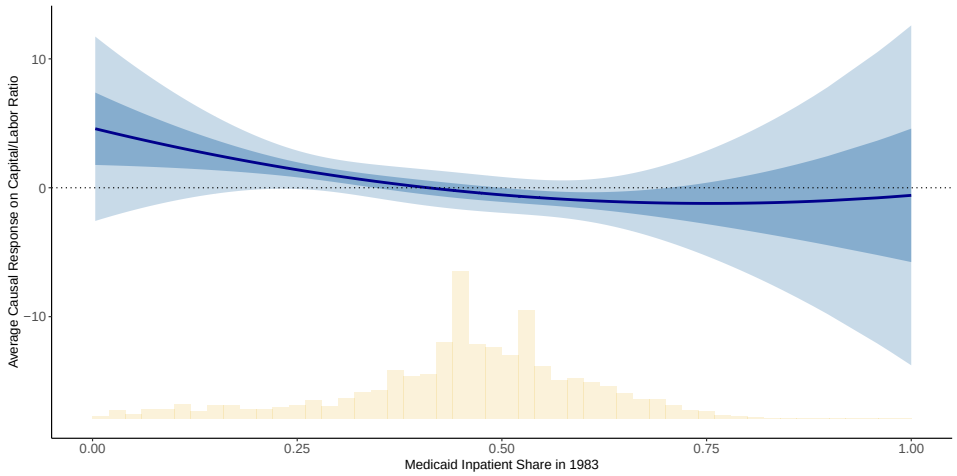
values for doses below the mean as well as negative $ACRT(m)$ values for doses between about 0.5 and 0.7 using pointwise confidence intervals.

PPS' average causal response parameter weighted by the actual dose distribution of treated hospitals is $\widehat{ACRT}^{\texttt{glob}} = -0.08$ (s.e. $= 0.19$) and is not significantly different from zero.[36] This differs substantially from the TWFE coefficient, $\widehat{\beta}^{twfe} = 1.14$. From Theorem 3.4(a), the difference between these estimates is fully driven by differences in the weighting scheme. Our estimate of $ACRT^{\texttt{glob}}$ comes from mapping the estimates of $ACRT(m)$ in Figure 9 to the dose distribution weights, $\widehat{f}_{M|M>0}(m)$, in Figure 7; our estimate of β^{twfe} comes from mapping the estimates of $ACRT(m)$ to the TWFE causal response weights, $\widehat{w}_1^{acrt}(m)$, in Figure 7. As discussed in Theorem 3.4(a), the TWFE causal response weights are positive for all values of the dose and integrate to one, providing a reason to hope that estimates of $ACRT^{\texttt{glob}}$ and β^{twfe} would be similar. However, the TWFE weighting scheme turns out to be much different from the dose distribution weighting scheme. Combining these differences with the high degree of heterogeneity in $ACRT(m)$ across m is what leads to the sharp differences in the estimates. Another reason to emphasize the large difference between these estimates is that the literature has often viewed negative weights as a dividing line between an "unreasonable" or "reasonable" weighting scheme (see, e.g., Angrist (1998), de Chaisemartin and D'Haultfoeuille (2020), and Blandhol, Bonney, Mogstad, and Torgovitsky (2025) for related discussions of this point in different contexts). The results here suggest that, at least in our context, articulating a well-defined causal effect parameter and targeting that parameter directly is likely to be more important than checking that weights are all positive and integrate to one.

Under Assumption SPT, one policy implication of these estimates is that Medicare could have achieved similar, if not greater, capital investments while providing lower capital subsidies. Figure 9 shows that marginal increments in the subsidy ratio increase capital intensity only for those with low subsidy levels. Hospitals that received large capital subsidies under PPS responded with smaller

[36] We treat the sieve dimension used to compute $\widehat{ACRT}^{\texttt{glob}}$ as a non-random sequence, which is in line with the theoretical justification in Ai and Chen (2007). A formal theoretical treatment that accounts for the stochastic nature of our Lepski-type selection is interesting but left for future research.

Figure 9: Nonparametric Estimates of $ACRT(m)$ for Medicare PPS



Notes: The figure plots nonparametric estimate of $ACRT(m)$ that adapts the Chen, Christensen, and Kankanala (2024) data-driven estimator to our context, as discussed in Section 4.1 and Appendix B. The dark-shaded region is the 95-percent point-wise confidence interval, and the lighter-shaded region is the 95-percent honest and sup-norm rate-adaptive uniform confidence band. We display the histogram of the treatment dose among the treated in yellow.

increases in capital intensity than hospitals with slightly smaller subsidies, a fact easily seen in the binned scatter plot in Figure 4. The strong parallel trends assumption means that these estimated responses are "externally valid" for all treated hospitals, which means that only low subsidies matter for hospitals' input choices. Because higher subsidy ratios do not create further investments in capital, capping capital subsidies may not affect input choices very much.

An important economic implication, however, is that negative $ACRT(m)$ estimates contradict AF's two-factor economic model. $ACRT(m)$ is proportional to the average derivative of the optimal capital-labor ratio for hospitals with Medicare share equal to m, and Equation (6.1) shows specifically how it relates to the elasticity of substitution, $\sigma_{i,t}(m)$. To approximate $\mathbb{E}[\sigma_{i,t=2}(m)|M > 0]$, we separate out the two terms in (6.1) and construct $\frac{ACRT(m)}{\mathbb{E}[Y_{i,t=2}|M=m]}\frac{1-s_k m}{s_k}$ assuming that $s_k = 0.75$.[37] With only two inputs, a rise in the relative price of one must lead to a reduction in its relative use: the elasticity of substitution must be positive. The point estimates of $\mathbb{E}[\sigma_{i,t=2}(m)|M > 0]$ do not fit that prediction, although our uniform confidence bands do not reject an average elasticity of substitution of zero. Alternative models, such as a three-factor production function (which AF

[37] To see why this is an approximation and to understand the bias, add and subtract $\frac{s_k}{1-s_k m}\mathbb{E}\left[k^*_{i,t}\middle|M=m\right]$ in equation (6.1). Then $\mathbb{E}\left[\sigma_{i,t=2}(m)k^*_{i,t=2}\frac{s_k}{1-s_k m}\middle|M=m\right]$ equals:

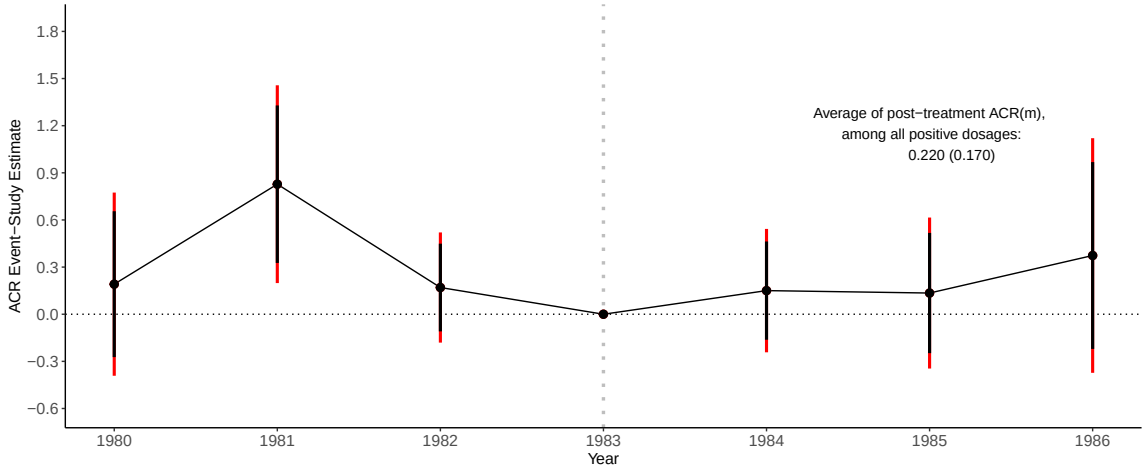
$$= \frac{s_k}{1-s_k m}\Big(\overbrace{\mathbb{E}\left[\sigma_{i,t=2}(m)(k^*_{i,t=2}-\mathbb{E}\left[k^*_{i,t=2}\middle|M=m\right])\middle|M=m\right]}^{\mathrm{Cov}(\sigma_{i,t=2}(m),k^*_{i,t=2}|M=m)} + \mathbb{E}\left[\sigma_{i,t=2}(m)|M=m\right]\mathbb{E}\left[k^*_{i,t=2}\middle|M=m\right]\Big)$$

We ignore the covariance between the elasticity of substitution and post-treatment capital-labor ratios among hospitals with the same value of m when we calculate $\mathbb{E}[\sigma_{i,t=2}(m)|M > 0]$. The theoretical model implies that this covariance is zero since identical production functions mean that all hospitals choose the same inputs given m. Our qualitative conclusions also do not depend strongly on the value we assume for s_k, the marginal capital subsidy rate. Medicare actually subsidized capital by reimbursing hospitals at "reasonable cost" for depreciation and interest on capital, so a specific subsidy rate was not defined. In a working paper version, Acemoglu and Finkelstein (2008) use $s_k = 1$ when calculating an elasticity of substitution. Finally, we divided our estimated $ACRT(m)$ curve by a smoothed estimate of $\mathbb{E}[Y_{i,t=2}|M=m]$ during the post-PPS years.

consider in their working paper) or non-homothetic production, could potentially rationalize this finding.

Finally, both the policy and structural interpretations of Figure 9 depend on the strong parallel trends assumption. Without SPT, the slope of $ATT(m|m)$ may be negative for higher-Medicare-share hospitals simply because their treatment effect functions are systematically lower. Medicare might not have been able to achieve similar capital increases with lower subsidy rates if high-subsidy hospitals just responded differently to low subsidy levels than low-subsidy hospitals did. A negative slope also does not necessarily reject a two-factor production model; just a constant-coefficient model with homogeneous firms, as considered by AF.

Figure 10: Event-Study Estimates of $ACRT$



Notes: The figure plots the event-study estimates of $ACRT^{es}_{\texttt{glob}}(e)$, with their 95% pointwise confidence intervals reported in black, and the 95% uniform confidence bands reported in red

Another way to assess the plausibility of SPT that justifies a causal interpretation of $ACRT^{\texttt{glob}}$ is to compute $ACRT^{es}_{\texttt{glob}}(e)$, the event-study version of $ACRT^{\texttt{glob}}$. These parameters can be estimated using the same procedure discussed in Section 4, and we plot these in Figure 10. The no-anticipation assumption means that prior to treatment, when all observed outcomes are untreated potential outcomes, both Assumptions PT and SPT have the same implication: that the average relationship between outcome changes for adjacent dose groups should be zero. Our estimates of these pre-trends reject this in 1981, which is a pre-treatment period. Interpreting that violations of strong parallel pre-trends as informative as violations of SPT in post-treatment periods, Figure 10 corroborates our conclusions about the implausibility of SPT based on implausibly high implied elasticities of substitution.[38]

In summary, our empirical results align with AF's conclusion that the 1983 Medicare reform led hospitals to favor capital over labor. We find evidence against parallel trends in pre-treatment periods, though the magnitudes of these violations are small relative to estimated effects in post-treatment

[38] As noted in Section 5.4, whether or not the figure also provides a piece of evidence against causally interpreting $ATT^{es}_{\texttt{loc}}(e)$ in Figure 8 depends on whether the event study is rationalized under an aggregate parallel trends assumption or the parallel trends assumption in Assumption PT.

periods. Finally, our negative estimates of $ACRT(m)$ at high values of m cut against the theoretical predictions of the model discussed above; this provides a piece of evidence that casts doubt on the plausibility of strong parallel trends in this application, indicating that one should be cautious when interpreting $ACRT$ parameters.

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A Proofs of Main Results

A.1 Proofs of Results in Section 3.2

This section contains the proofs of the results in Section 3.2 on identifying causal effect parameters such as $ATT(d|d)$ and $ATT(d)$ under parallel trends assumptions and with a continuous treatment or multi-valued discrete treatment.

Proof of Theorem 3.1

Proof. To show the result, notice that

$$\begin{aligned} ATT(d|d) &= \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(0)|D=d] \\ &= \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D=d] - \mathbb{E}[Y_{t=2}(0) - Y_{t=1}(0)|D=d] \\ &= \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D=d] - \mathbb{E}[Y_{t=2}(0) - Y_{t=1}(0)|D=0] \\ &= \mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=0] \end{aligned} \tag{A.1}$$

where the second equality holds by adding and subtracting $\mathbb{E}[Y_{t=1}(0)|D=d]$, the third equality holds by Assumption PT, and the last equality holds because $Y_{t=2}(d)$ and $Y_{t=1}(0)$ are observed potential outcomes when $D=d$ and $Y_{t=2}(0)$ and $Y_{t=1}(0)$ are observed potential outcomes when $D=0$. That $ATT^{\texttt{loc}}$ is identified holds immediately given its definition and that $ATT(d|d)$ is identified. To derive the particular expression for $ATT^{\texttt{loc}}$, notice that

$$\begin{aligned} ATT^{\texttt{loc}} &= \mathbb{E}\Big[ATT(D|D)\Big|D>0\Big] \\ &= \mathbb{E}\Big[\Big(\mathbb{E}[\Delta Y|D] - \mathbb{E}[\Delta Y|D=0]\Big)\Big|D>0\Big] \\ &= \mathbb{E}[\Delta Y|D>0] - \mathbb{E}[\Delta Y|D=0] \end{aligned}$$

where the first equality is the definition of $ATT^{\texttt{loc}}$, the second equality holds from Equation (A.1), the first part of the third equality holds by an implication of the law of iterated expectations, and the second part of the third equality holds because $\mathbb{E}[\Delta Y|D=0]$ is non-random. □

Proof of Theorem 3.2

Proof. To prove part (a), notice that

$$\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=l] = \Big(\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=0]\Big) - \Big(\mathbb{E}[\Delta Y|D=l] - \mathbb{E}[\Delta Y|D=0]\Big)$$

$$= ATT(h|h) - ATT(l|l) \tag{A.2}$$

where the first equality holds by adding and subtracting $\mathbb{E}[\Delta Y|D=0]$, and the second equality holds by Theorem 3.1. Next,

$$\begin{aligned} ATT(h|h) - ATT(l|l) &= \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(0)|D=h] - \mathbb{E}[Y_{t=2}(l) - Y_{t=2}(0)|D=l] \\ &= \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h] \\ &\quad + \mathbb{E}[Y_{t=2}(l) - Y_{t=2}(0)|D=h] - \mathbb{E}[Y_{t=2}(l) - Y_{t=2}(0)|D=l] \\ &= \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h] + \Big(ATT(l|h) - ATT(l|l)\Big) \end{aligned} \tag{A.3}$$

where the first equality holds by the definition of $ATT(d|d)$, the second equality holds by adding and subtracting $\mathbb{E}[Y_{t=2}(l)|D=h]$, and the third equality holds by the definition of $ATT(l|h)$ and $ATT(l|l)$. Notice that $\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h]$ is a causal response of going from dose l to dose h for dose group h. An alternative expression for this term is

$$\mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D=h] = ATT(h|h) - ATT(l|h) \tag{A.4}$$

Next, we prove part (b). Using a similar argument as above, notice that, for $d \in \mathcal{D}_+^c$ and $(d+h) \in \mathcal{D}_+^c$,

$$\begin{aligned} \frac{\mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=d+h]}{h} &= \frac{ATT(d|d) - ATT(d+h|d+h)}{h} \\ &= \frac{ATT(d|d) - ATT(d+h|d)}{h} + \frac{ATT(d+h|d) - ATT(d+h|d+h)}{h} \end{aligned}$$

where the first equality holds using the same argument as for Equation (A.2), and the second equality holds by using the arguments in Equations (A.3) and (A.4). The result holds by taking the limit as $h \to 0$ and the definition of $ACRT(d|d)$.

Finally, the second result in part (c) involving a discrete treatment holds by taking $h = d_j$ and $l = d_{j-1}$ in Equations (A.2) and (A.3) and by the definition of $ACRT(d_j|d_j)$. □

Proof of Theorem 3.3

Proof. For part (a), notice that

$$\begin{aligned} ATT(d) &= \mathbb{E}[Y_{t=2}(d) - Y_{t=2}(0)|D>0] \\ &= \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D>0] - \mathbb{E}[Y_{t=2}(0) - Y_{t=1}(0)|D>0] \\ &= \mathbb{E}[Y_{t=2}(d) - Y_{t=1}(0)|D=d] - \mathbb{E}[Y_{t=2}(0) - Y_{t=1}(0)|D=0] \\ &= \mathbb{E}[\Delta Y|D=d] - \mathbb{E}[\Delta Y|D=0] \end{aligned}$$

where the second equality holds by adding and subtracting $\mathbb{E}[Y_{t=1}(0)|D>0]$, the third equality holds by Assumption SPT, and the fourth equality holds because $Y_{t=2}(d)$ and $Y_{t=1}(0)$ are observed outcomes when $D=d$.

Next, we prove the first part of part (b). First, notice that

$$\begin{aligned} ATT(h) - ATT(l) &= \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(0)|D>0] - \mathbb{E}[Y_{t=2}(l) - Y_{t=2}(0)|D>0] \\ &= \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D>0] \end{aligned}$$

where the first equality holds by the definition of $ATT(d)$, and the second equality holds by cancelling the terms involving $Y_{t=2}(0)$. For the second part, notice that, from part (a), we have that

$$\begin{aligned} ATT(h) - ATT(l) &= \Big(\mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=0]\Big) - \Big(\mathbb{E}[\Delta Y|D=l] - \mathbb{E}[\Delta Y|D=0]\Big) \\ &= \mathbb{E}[\Delta Y|D=h] - \mathbb{E}[\Delta Y|D=l] \end{aligned}$$

Now, for part (c), notice that for $d \in \mathcal{D}_+^c$ and $(d+h) \in \mathcal{D}_+^c$,

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

which follows from part (b). The result holds by taking the limit as $h \to 0$ and from the definition of $ACRT(d)$. Finally, the result in part (d) involving a discrete treatment holds from part (b) by taking $h = d_j$ and $l = d_{j-1}$ and by the definition of $ACRT(d_j)$. □

Proof of Corollary 3.1

Proof. The result holds immediately by averaging the results in Theorem 3.1 over the distribution of the dose among dose groups that experienced any positive amount of the treatment. □

A.2 Proofs of Results from Section 3.3

This section contains the proofs of the results in Theorem 3.4 in Section 3.3 on interpreting TWFE regressions with a continuous treatment. To conserve on notation, we define

$$m_\Delta(d) = \mathbb{E}[\Delta Y|D=d],$$

We divide the proofs according to each part of the theorem. In the proof, we derive all the results in terms of $m_\Delta(d)$. This results in a mechanical decomposition in the sense that $\widehat{\beta}^{twfe}$ is equal to the sample analog of each derived quantity below. The result in Theorem 3.4 is stated in terms of various causal building block parameters. Those results follow immediately from the ones below by noting that, under Assumption PT,

- $m_\Delta(d) - m_\Delta(0) = ATT(d|d)$
- $m'_\Delta(d) = ACRT(d|d) + \underbrace{\frac{\partial ATT(d|h)}{\partial h}\Big|_{h=d}}_{\text{selection bias}}$
- $m_\Delta(h) - m_\Delta(l) = ATT(h|h) - ATT(l|l) = \mathbb{E}[Y_t(h) - Y_t(l)|D=h] + \underbrace{\Big(ATT(l|h) - ATT(l|l)\Big)}_{\text{selection bias}}$

or, when Assumption SPT holds,

- $m_\Delta(d) - m_\Delta(0) = ATT(d)$
- $m'_\Delta(d) = ACRT(d)$
- $m_\Delta(h) - m_\Delta(l) = ATT(h) - ATT(l) = \mathbb{E}[Y_{t=2}(h) - Y_{t=2}(l)|D>0]$

Proof of Theorem 3.4(a)

Proof. First, notice that Equation (1.1) is equivalent to

$$\Delta Y_i = (\theta_{t=2} - \theta_{t=1}) + \beta^{twfe} D_i + \Delta v_{i,t} \tag{A.5}$$

which holds by taking first differences and because all units are untreated in the first period. Therefore, it immediately follows that

$$\begin{aligned}
\beta^{twfe} &= \frac{\mathbb{E}[(D - \mathbb{E}[D])\Delta Y]}{\text{Var}(D)} \\
&= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(D) - m_\Delta(0))\right] \\
&= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(D) - m_\Delta(0))\Big| D > 0\right]\mathbb{P}(D > 0) \\
&= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(D) - m_\Delta(d_L))\Big| D > 0\right]\mathbb{P}(D > 0) \\
&\quad + \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(d_L) - m_\Delta(0))\Big| D > 0\right]\mathbb{P}(D > 0) \\
&= A_1 + A_2
\end{aligned} \tag{A.6}$$

where the first equality holds because Equation (A.5) is a simple linear regression of ΔY on an intercept and D, the second equality holds by the law of iterated expectations and because $\mathbb{E}[(D - \mathbb{E}[D])m_\Delta(0)] = 0$, the third equality holds because $\mathbb{E}[m_\Delta(D) - m_\Delta(0)|D = 0] = 0$, and the fourth equality holds by adding and subtracting $m_\Delta(d_L)$ inside the expectation.

We consider A_1 and A_2 separately next. First, for A_1,

$$\begin{aligned}
A_1 &= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(D) - m_\Delta(d_L))\Big| D > 0\right]\mathbb{P}(D > 0) \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U}(k - \mathbb{E}[D])(m_\Delta(k) - m_\Delta(d_L))\, dF_{D|D>0}(k) \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U}(k - \mathbb{E}[D])\int_{d_L}^{k} m'_\Delta(l)\, dl\, dF_{D|D>0}(k) \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U}(k - \mathbb{E}[D])\int_{d_L}^{d_U} \mathbf{1}\{l \le k\} m'_\Delta(l)\, dl\, dF_{D|D>0}(k) \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U} m'_\Delta(l)\int_{d_L}^{d_U}(k - \mathbb{E}[D])\mathbf{1}\{l \le k\}\, dF_{D|D>0}(k)\, dl \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U} m'_\Delta(l)\mathbb{E}[(D - \mathbb{E}[D])\mathbf{1}\{l \le D\}|D > 0]\, dl \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)}\int_{d_L}^{d_U} m'_\Delta(l)\mathbb{E}[(D - \mathbb{E}[D])|D \ge l]\mathbb{P}(D \ge l|D > 0)\, dl \\
&= \int_{d_L}^{d_U} m'_\Delta(l)\frac{(\mathbb{E}[D|D \ge l] - \mathbb{E}[D])\mathbb{P}(D \ge l)}{\text{Var}(D)}\, dl
\end{aligned} \tag{A.7}$$

where the first equality is the definition of A_1, the second equality holds by rearranging terms and writing the expectation as an integral, the third equality holds by the fundamental theorem of calculus, the fourth equality rewrites the inner integral so that it is over d_L to d_U, the fifth equality holds by

changing the order of integration and rearranging terms, the sixth equality holds by rewriting the inner integral as an expectation, the seventh equality holds by the law of iterated expectations (and since $D \geq l \implies D > 0$), and the last equality holds by combining terms.

Next, for A_2, it immediately holds that

$$\begin{aligned} A_2 &= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\text{Var}(D)}(m_\Delta(d_L) - m_\Delta(0))\Big| D > 0\right]\mathbb{P}(D > 0) \\ &= \frac{(\mathbb{E}[D|D > 0] - \mathbb{E}[D])\mathbb{P}(D > 0)d_L}{\text{Var}(D)}\frac{(m_\Delta(d_L) - m_\Delta(0))}{d_L} \end{aligned} \tag{A.8}$$

where the first equality is the definition of A_2, and the second equality holds by multiplying and dividing by d_L.

Then, the first result in Part (a) holds by combining Equations (A.7) and (A.8). That the weights are all positive holds immediately since $(\mathbb{E}[D|D \geq l] - \mathbb{E}[D]) > 0$ for all $l \geq d_L$, $\mathbb{P}(D \geq l) > 0$ for all $l \geq d_L$, $(\mathbb{E}[D|D > 0] - \mathbb{E}[D]) > 0$, $\mathbb{P}(D > 0) > 0$, and $\text{Var}(D) > 0$.

Next, we next show that $\int_{d_L}^{d_U} w_1^{acrt}(l)\, dl + w_0^{acrt} = 1$. First, notice that

$$\begin{aligned} \int_{d_L}^{d_U} w_1^{acrt}(l)\, dl + w_0^{acrt} = \frac{1}{\text{Var}(D)}\Bigg\{ &\int_{d_L}^{d_U} \mathbb{E}[D|D \geq l]\mathbb{P}(D \geq l)\, dl \\ &- \mathbb{E}[D]\int_{d_L}^{d_U} \mathbb{P}(D \geq l)\, dl \\ &+ \mathbb{E}[D|D > 0]\mathbb{P}(D > 0)d_L \\ &- \mathbb{E}[D]\mathbb{P}(D > 0)d_L \Bigg\} \\ = \frac{1}{\text{Var}(D)}\Big\{B_1 - B_2 + B_3 - B_4\Big\} \end{aligned}$$

and we consider B_1, B_2, B_3, and B_4 in turn.

For B_1, first notice that for all $l \in \mathcal{D}_+^c$,

$$\begin{aligned} \mathbb{E}[D|D \geq l]\mathbb{P}(D \geq l) &= \mathbb{E}[D\mathbf{1}\{D \geq l\}|D \geq l]\mathbb{P}(D \geq l) \\ &= \mathbb{E}[D\mathbf{1}\{D \geq l\}] \end{aligned} \tag{A.9}$$

which holds by the law of iterated expectations and implies that

$$\begin{aligned} B_1 &= \int_{d_L}^{d_U} \mathbb{E}[D|D \geq l]\mathbb{P}(D \geq l)\, dl \\ &= \int_{d_L}^{d_U} \int_{\mathcal{D}} d\mathbf{1}\{d \geq l\}\, dF_D(d)\, dl \\ &= \int_{\mathcal{D}} d\left(\int_{d_L}^{d_U} \mathbf{1}\{l \leq d\}\, dl\right) dF_D(d) \\ &= \int_{\mathcal{D}} d(d - d_L)\, dF_D(d) \\ &= \mathbb{E}[D^2] - \mathbb{E}[D]d_L \end{aligned} \tag{A.10}$$

where the first line is the definition of B_1, the second equality holds by Equation (A.9), the third

equality holds by changing the order of integration, the fourth equality holds by carrying out the inner integration, and the last equality holds by rewriting the integral as an expectation.

Next, for term B_2,

$$\begin{aligned}
B_2 &= \mathbb{E}[D] \int_{d_L}^{d_U} \mathbb{P}(D \geq l)\, dl \\
&= \mathbb{E}[D]\mathbb{P}(D > 0) \int_{d_L}^{d_U} \mathbb{P}(D \geq l | D > 0)\, dl \\
&= \mathbb{E}[D]\mathbb{P}(D > 0) \int_{d_L}^{d_U} \int_{d_L}^{d_U} \mathbf{1}\{d \geq l\}\, dF_{D|D>0}(d)\, dl \\
&= \mathbb{E}[D]\mathbb{P}(D > 0) \int_{d_L}^{d_U} \left(\int_{d_L}^{d_U} \mathbf{1}\{l \leq d\}\, dl \right) dF_{D|D>0}(d) \\
&= \mathbb{E}[D]\mathbb{P}(D > 0) \int_{d_L}^{d_U} (d - d_L)\, dF_{D|D>0}(d) \\
&= \mathbb{E}[D]\mathbb{P}(D > 0) \Big(\mathbb{E}[D|D > 0] - d_L \Big) \\
&= \mathbb{E}[D]^2 - \mathbb{E}[D]\mathbb{P}(D > 0) d_L
\end{aligned} \tag{A.11}$$

where the first equality is the definition of B_2, the second equality holds by the law of iterated expectations, the third equality holds by writing $\mathbb{P}(D \geq l | D > 0)$ as an integral, the fourth equality changes the order of integration, the fifth equality carries out the inside integration, the sixth equality rewrites the integral as an expectation, and the last equality holds by combining terms and by the law of iterated expectations.

Next,

$$\begin{aligned}
B_3 &= \mathbb{E}[D|D > 0]\mathbb{P}(D > 0) d_L \\
&= \mathbb{E}[D] d_L
\end{aligned} \tag{A.12}$$

which holds by the law of iterated expectations. And finally, recall that

$$B_4 = \mathbb{E}[D]\mathbb{P}(D > 0) d_L \tag{A.13}$$

Thus, from Equations (A.10) to (A.13), it follows that

$$B_1 - B_2 + B_3 - B_4 = \mathbb{E}[D^2] - \mathbb{E}[D]^2 = \mathrm{Var}(D)$$

which implies the result.

□

Proof of Theorem 3.4(b)

Proof. From the proof of Part (a), we have that

$$\begin{aligned}
\beta^{twfe} &= \frac{\mathbb{P}(D > 0)}{\mathrm{Var}(D)} \mathbb{E}\Big[(D - \mathbb{E}[D])(m_\Delta(D) - m_\Delta(0)) \Big| D > 0 \Big] \\
&= \frac{\mathbb{P}(D > 0)}{\mathrm{Var}(D)} \int_{d_L}^{d_U} (l - \mathbb{E}[D])(m_\Delta(l) - m_\Delta(0))\, dF_{D|D>0}(l)
\end{aligned}$$

$$= \frac{1}{\text{Var}(D)} \int_{d_L}^{d_U} (l - \mathbb{E}[D])(m_\Delta(l) - m_\Delta(0)) f_D(l)\, dl$$
$$= \int_{d_L}^{d_U} w_1^{lev}(l)(m_\Delta(l) - m_\Delta(0))\, dl$$

where the second equality holds by writing the expectation as an integral, the third equality holds under Assumption 4(a), and the last equality holds by the definition of w_1^{lev}.

Next, we show the properties of the weights for this part of the theorem. The weights can be negative since l can be less than $\mathbb{E}[D]$. To see that the weights integrate to zero, first note that $w_0^{lev}(m_\Delta(0) - m_\Delta(0)) = 0$, so that the previous expression for β^{twfe} can equivalently be written as

$$\beta^{twfe} = \int_{d_L}^{d_U} w_1^{lev}(l)(m_\Delta(l) - m_\Delta(0))\, dl + w_0^{lev}(m_\Delta(0) - m_\Delta(0))$$

Then, notice that

$$\begin{aligned}
\int_{d_L}^{d_U} w_1^{lev}(l)\, dl + w_0^{lev} &= \left(\int_{d_L}^{d_U} (l - \mathbb{E}[D])\, dF_D(l) + (0 - \mathbb{E}[D])\mathbb{P}(D = 0) \right) \Big/ \text{Var}(D) \\
&= \left(\int_{\mathcal{D}} (l - \mathbb{E}[D])\, dF_D(l) \right) \Big/ \text{Var}(D) \\
&= (\mathbb{E}[D] - \mathbb{E}[D])/\text{Var}(D) \\
&= 0
\end{aligned}$$

where the first equality holds by the definitions of w_1^{lev} and w_0^{lev}, the second equality combines terms, and the third and fourth equalities hold immediately. This completes the proof. □

Proof of Theorem 3.4(c)

Proof. From the proof of Theorem 3.4(a), we have that

$$\begin{aligned}
\beta^{twfe} &= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)} \mathbb{E}\left[(D - \mathbb{E}[D])(m_\Delta(D) - m_\Delta(0)) \Big| D > 0 \right] \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)} \int_{d_L}^{d_U} (l - \mathbb{E}[D])(m_\Delta(l) - m_\Delta(0))\, dF_{D|D>0}(l) \\
&= \frac{\mathbb{P}(D > 0)}{\text{Var}(D)} \int_{d_L}^{d_U} (l - \mathbb{E}[D]) l \frac{(m_\Delta(l) - m_\Delta(0))}{l}\, dF_{D|D>0}(l) \\
&= \frac{1}{\text{Var}(D)} \int_{d_L}^{d_U} (l - \mathbb{E}[D]) l \frac{(m_\Delta(l) - m_\Delta(0))}{l} f_D(l)\, dl \\
&= \int_{d_L}^{d_U} w^s(l) \frac{(m_\Delta(l) - m_\Delta(0))}{l}\, dl
\end{aligned}$$

where the second equality holds by writing the expectation as an integral, the third equality holds by multiplying and dividing by l, the fourth equality holds under Assumption 4(a), and the last equality holds by the definition of w^s.

The weights can be negative because it is possible that $l < \mathbb{E}[D]$ for some values of $l \in \mathcal{D}_+^c$. That the weights integrate to 1 holds because

$$\int_{d_L}^{d_U} w^s(l)\, dl = \left(\int_{d_L}^{d_U} (l - \mathbb{E}[D]) l\, dF_D(l) + (0 - \mathbb{E}[D])\, 0\, \mathbb{P}(D = 0) \right) \Big/ \text{Var}(D)$$

$$= \left(\int_{\mathcal{D}} (l - \mathbb{E}[D]) l \, dF_D(l) \right) \Big/ \mathrm{Var}(D)$$
$$= (\mathbb{E}[D^2] - \mathbb{E}[D]^2) \Big/ \mathrm{Var}(D) = 1$$

where the first equality uses the definition of the weights and that $(0 - \mathbb{E}[D])\, 0\, \mathbb{P}(D = 0) = 0$, the second equality comes from combining terms, and the last line holds immediately. □

Proof of Theorem 3.4(d)

Proof. From the proof of part (a), we have that

$$\begin{aligned}
\beta &= \mathbb{E}\left[\frac{(D - \mathbb{E}[D])}{\mathrm{Var}(D)} m_\Delta(D) \right] \\
&= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} (h - \mathbb{E}[D]) m_\Delta(h) \, dF_D(h) \\
&= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} \left(h - \int_{\mathcal{D}} l \, dF_D(l) \right) m_\Delta(h) \, dF_D(h) \\
&= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} \int_{\mathcal{D}} (h - l) m_\Delta(h) \, dF_D(h) \, dF_D(l) \\
&= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} \int_{\mathcal{D}, h > l} (h - l)(m_\Delta(h) - m_\Delta(l)) \, dF_D(h) \, dF_D(l) \\
&= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} \int_{\mathcal{D}, h > l} (h - l)^2 \frac{(m_\Delta(h) - m_\Delta(l))}{(h - l)} \, dF_D(h) \, dF_D(l) \qquad \text{(A.14)}
\end{aligned}$$

where the second equality holds by writing the expectation as an integral, the third equality by writing $\mathbb{E}[D]$ as an integral, the fourth equality rearranges terms, the fifth equality holds because the integrations are symmetric, and the last equality holds by multiplying and dividing by $(h - l)$.

The above arguments hold if the treatment is continuous or discrete. Under Assumption 4(a),

$$\begin{aligned}
\text{Equation (A.14)} = &\frac{1}{\mathrm{Var}(D)} \int_{d_L}^{d_U} \int_{\mathcal{D}, h > l} (h - l)^2 \frac{(m_\Delta(h) - m_\Delta(l))}{(h - l)} f_D(h) f_D(l) \, dh \, dl \\
&+ \frac{1}{\mathrm{Var}(D)} \int_{d_L}^{d_U} h^2 \frac{m_\Delta(h) - m_\Delta(0)}{h} f_D(h) \mathbb{P}(D = 0) \, dh
\end{aligned}$$

which holds by splitting up the first integral in Equation (A.14) by whether $l \in \mathcal{D}_+^c$ or $l = 0$. Then, the first part of this result holds by the definition of $w_1^{2\times2}$ and $w_0^{2\times2}$.

That the weights are all positive holds immediately by their definitions. That the weights integrate to one holds because

$$\begin{aligned}
\int_{d_L}^{d_U} \int_{\mathcal{D}, h > l} w_1^{2\times2, cont}(l, h) \, dh \, dl + \int_{d_L}^{d_U} w_0^{2\times2, cont}(h) \, dh &= \frac{1}{\mathrm{Var}(D)} \int_{\mathcal{D}} \int_{\mathcal{D}} \mathbf{1}\{h > l\} (h - l)^2 \, dF_D(h) \, dF_D(l) \\
&= \frac{1}{2} \int_{\mathcal{D}} \int_{\mathcal{D}} (h - l)^2 \, dF_D(h) \, dF_D(l) \Big/ \mathrm{Var}(D) \\
&= 1
\end{aligned}$$

where the first equality holds by combining the integrals and the definition of the weights (it amounts to re-writing the integrals as in Equation (A.14)), the second equality holds because $\int_{\mathcal{D}} \int_{\mathcal{D}} \mathbf{1}\{h > l\}(h - l)^2 \, dF_D(h) \, dF_D(l) = \int_{\mathcal{D}} \int_{\mathcal{D}} \mathbf{1}\{h \leq l\}(h - l)^2 \, dF_D(h) \, dF_D(l)$ (and these two terms add up to

the expression on the next line), and the third equality holds because the double integral is equal to $2\text{Var}(D)$. This completes the proof. □

B Adapting CCK to DiD Contexts

In this Appendix, we provide more details on how to adapt the Chen, Christensen, and Kankanala (2024) (henceforth, CCK) data-driven nonparametric estimation and inference procedures in our DiD context. As discussed in Section 4.1, the CCK estimator for $ATT(d)$ and $ACRT(d)$ are given by

$$\widehat{ATT}_{\text{cck}}(d) = \left(\psi^{\widehat{K}}(d)\right)' \widehat{\beta}_{\widehat{K}}, \qquad \widehat{ACRT}_{\text{cck}}(d) = \left(\partial\psi^{\widehat{K}}(d)\right)' \widehat{\beta}_{\widehat{K}},$$

where $\psi^K(d)$ is a K-dimensional vector of cubic B-splines basis functions, $\partial\psi^K(s) = (d\psi_{K1}(s)/\,ds, \ldots, d\psi_{KK}(s)/\,ds)'$, $\widehat{\beta}_{\widehat{K}}$ is the $\widehat{K}$-dimension vector of OLS estimators for $\beta_{\widehat{K}}$, and $\widehat{K}$ is the CCK data-driven estimator for the optimal sieve dimension.

To discuss the optimal choice of the sieve dimension K derived in CCK, we need to add more notation. Let $\mathcal{K} = \left\{\left(2^k + 3\right) : k \in \mathbb{N}_+ \cup 0\right\}$ be the set of possible sieve dimensions for the cubic B-splines. Let $K^+ = \min\{k \in \mathcal{K} : k > K\}$ be the smallest sieve dimension in $\mathcal{K}$ exceeding K, and $v_n = \max\{1, (0.1 \log n)^4\}$. Let $\{\omega_i\}_{i=1}^n$ be iid standard normal draws independent of the data $\{W_i\}_{i=1}^n = \{Y_{i,t=2}, Y_{i,t=1}, D_i\}_{i=1}^n$. In addition, let

$$\widehat{\varphi}_K(W_i, d) = \left(\psi^K(d)\right)' \widehat{\phi}_K(W_i),$$

with

$$\widehat{\phi}_K(W_i) = \mathbb{E}_n\left[1\{D > 0\} \cdot \psi^K(D)\psi^K(D)'\right]^- 1\{D_i > 0\}\psi^K(D_i)\widehat{u}_{i,K},$$

and $\widehat{u}_{i,K} = \Delta Y_i - \mathbb{E}_n[\Delta Y | D > 0] - \left(\psi^K(D_i)\right)' \widehat{\beta}_K$. Finally, for a given K and K_2, let

$$\widehat{\sigma}^2_{K,K_2}(d) = \frac{1}{n}\sum_{i=1}^n \left(\widehat{\varphi}_K(W_i, d) - \widehat{\varphi}_{K_2}(W_i, d)\right)^2$$

be an estimator of the (asymptotic) variance of the contrast $\sqrt{n}\left(\widehat{ATT}_K(d) - \widehat{ATT}_{K_2}(d)\right)$, and consider the bootstrap process

$$\mathbb{Z}^*_n(d, K, K_2) = \frac{1}{\widehat{\sigma}_{K,K_2}(d)}\left(\frac{1}{\sqrt{n}}\sum_{i=1}^n \left(\widehat{\varphi}_K(W_i, d) - \widehat{\varphi}_{K_2}(W_i, d)\right) \cdot \omega_i\right).$$

For a given sieve dimension $K \in \mathcal{K}$, let

$$\widehat{ATT}_K(d) = \left(\psi^K(d)\right)' \widehat{\beta}_K, \qquad \widehat{ACRT}_K(d) = \left(\partial\psi^K(d)\right)' \widehat{\beta}_K, \tag{B.1}$$

where $\partial\psi^K(s) = (d\psi_{K1}(s)/\,ds, \ldots, d\psi_{KK}(s)/\,ds)'$,

$$\begin{aligned}\widehat{\beta}_K &= \arg\min_{b_K \in \Theta_K} \mathbb{E}_n\left[\left(\Delta Y - \mathbb{E}_n\left[\Delta Y | D = 0\right] - \psi^K(D)'b_K\right)^2 \middle| D > 0\right] \\ &= \mathbb{E}_n\left[1\{D > 0\}\psi^K(D)\psi^K(D)'\right]^- \mathbb{E}_n\left[1\{D > 0\}\psi^K(D)\left(\Delta Y - \mathbb{E}_n\left[\Delta Y | D = 0\right]\right)\right],\end{aligned} \tag{B.2}$$

and A^- denote the Moore-Penrose inverse of a generic matrix A, and for a generic variable B,

$$\mathbb{E}_n[B|D>0] = \frac{\sum_{i=1}^n 1\{D_i>0\}B_i}{\sum_{i=1}^n 1\{D_i>0\}}.$$

The next algorithm adapts Procedure 1 of CCK to our DiD context and provides the Lepski-type data-driven selection $\widehat{K}$ of the sieve dimension K.

Algorithm 1 (Computation of data-driven choice of sieve-dimension K based on CCK.)**.**

1. Compute the data-driven index set of sieve dimensions

$$\widehat{\mathcal{K}} = \left\{K \in \mathcal{K} : 0.1\left(\log \widehat{K}_{max}\right)^2 \leq K \leq \widehat{K}_{max}\right\} \tag{B.3}$$

where

$$\widehat{K}_{max} = \min\left\{K \in \mathcal{K} : K\sqrt{\log K}v_n \leq 10\sqrt{n} < K^+\sqrt{\log K^+}v_n\right\} \tag{B.4}$$

2. Let $\widehat{\alpha} = \min\left\{0.5, \sqrt{\log \widehat{K}_{max}/\widehat{K}_{max}}\right\}$*. For each independent draw of* $\{\omega_i\}_{i=1}^n$*, compute*

$$\sup_{(d,K,K_2)\in\mathcal{D}_+^c\times\widehat{\mathcal{K}}\times\widehat{\mathcal{K}}:K_2>K} |\mathbb{Z}_n^*(d,K,K_2)|\,. \tag{B.5}$$

Let $\gamma^*_{1-\widehat{\alpha}}$ *denote the* $(1-\widehat{\alpha})$ *quantile of the sup-t statistic* (B.5) *across a large number of independent draws of* $\{\omega_i\}_{i=1}^n$*, say, 1,000.*

3. The data-driven choice of the sieve dimension is

$$\widehat{K} = \inf\left\{K \in \widehat{\mathcal{K}} \;:\; \sup_{(d,K_2)\in\mathcal{D}_+^c\times\widehat{\mathcal{K}}:K_2>K} \frac{\sqrt{n}\left|\widehat{ATT}_K(d) - \widehat{ATT}_{K_2}(d)\right|}{\widehat{\sigma}_{K,K_2}(d)} \leq 1.1\gamma^*_{1-\widehat{\alpha}}\right\}. \tag{B.6}$$

The intuition behind Algorithm 1 is that it selects the most parsimonious specification across all considered ones, provided that the estimated $ATT_K(d)$ curves are not "statistically different" from each other. If increasing K leads to a statistically different estimate of $ATT_K(d)$, then it is "worth it" to increase the dimension. Heuristically, this is how Algorithm 1 trades off "bias" and "variance" in a sup-norm sense.

Next, we show how one can form data-driven uniform confidence bands (UCBs) for both $ATT(d)$ and $ACRT(d)$ by adapting Procedure 2 of CCK to our DiD context. Toward this end, let $\widehat{A} = \log\log\widehat{K}$ and set $\widehat{\mathcal{K}}_- = \{K \in \widehat{\mathcal{K}} : J < \widehat{K}\}$. Define the bootstrap processes

$$\mathbb{Z}_n^*(d,K) = \frac{1}{\widehat{\sigma}_K(d)}\frac{1}{\sqrt{n}}\sum_{i=1}^n \widehat{\varphi}_K(W_i,d)\cdot\omega_i, \quad \text{and} \quad \mathbb{Z}_n^{*,acr}(d,K) = \frac{1}{\widehat{\sigma}_K^{acrt}(d)}\frac{1}{\sqrt{n}}\sum_{i=1}^n \widehat{\varphi}_K^{acrt}(W_i,d)\cdot\omega_i.$$

where $\widehat{\varphi}_K^{acrt}(W_i,d) = \left(\partial\psi^K(d)\right)'\widehat{\phi}_K(W_i)$,

$$\widehat{\sigma}_K^2(d) = \frac{1}{n}\sum_{i=1}^n \widehat{\varphi}_K(W_i,d)^2, \qquad \text{and} \qquad \widehat{\sigma}_K^{acrt,2}(d) = \frac{1}{n}\sum_{i=1}^n \widehat{\varphi}_K^{acrt}(W_i,d)^2.$$

Algorithm 2 (Computation of UCBs for $ATT(\cdot)$ and $ACRT(d)$ based on CCK.)**.**

4. For each independent draw of $\{\omega_i\}_{i=1}^n$*, compute*

$$t^* = \sup_{(d,K)\in\mathcal{D}_+^c\times\widehat{\mathcal{K}}_-} |\mathbb{Z}_n^*(d,K)|\,, \quad \textit{and} \quad t^{*,acr} = \sup_{(d,K)\in\mathcal{D}_+^c\times\widehat{\mathcal{K}}_-} |\mathbb{Z}_n^{*,acr}(d,K)|\,. \tag{B.7}$$

Let $z^*_{1-\alpha}$ *and* $z^{*,acr}_{1-\alpha}$ *denote the* $(1-\alpha)$ *quantile of the sup-t statistic* t^* *and* $t^{*,acr}$*, respectively, across a large number of independent draws of* $\{\omega_i\}_{i=1}^n$*, say, 1,000.*

5. *The data-driven* $100(1-\alpha)\%$ *UCB for* $ATT(d)$ *and* $ACRT(d)$, $d \in \mathcal{D}_+^c$, *are respectively given by*

$$C_n(d) = \left[\widehat{ATT}_{\widehat{K}}(d) - \left(z^*_{1-\alpha} + \widehat{A}\,\gamma^*_{1-\widehat{\alpha}}\right)\frac{\widehat{\sigma}_{\widehat{K}}(d)}{\sqrt{n}},\ \widehat{ATT}_{\widehat{K}}(d) + \left(z^*_{1-\alpha} + \widehat{A}\,\gamma^*_{1-\widehat{\alpha}}\right)\frac{\widehat{\sigma}_{\widehat{K}}(d)}{\sqrt{n}}\right] \tag{B.8}$$

$$C^{acrt}_n(d) = \left[\widehat{ACRT}_{\widehat{K}}(d) - \left(z^{*,acr}_{1-\alpha} + \widehat{A}\,\gamma^*_{1-\widehat{\alpha}}\right)\frac{\widehat{\sigma}^{acrt}_{\widehat{K}}(d)}{\sqrt{n}},\ \widehat{ACRT}_{\widehat{K}}(d) + \left(z^{*,acr}_{1-\alpha} + \widehat{A}\,\gamma^*_{1-\widehat{\alpha}}\right)\frac{\widehat{\sigma}^{acrt}_{\widehat{K}}(d)}{\sqrt{n}}\right] \tag{B.9}$$

Heuristically, Algorithm 2 is essentially describing that one can compute uniform confidence bands in a traditional way, except that we "inflate" critical values to account for potential "biases" that could be proportional to the "standard deviation". The critical values also account for the model-selection uncertainty. Importantly, the UCBs described in Algorithm 2 enjoy attractive statistical guarantees such as *honesty* and *adaptivity*. In practice, these mean that these UCBs are guaranteed to have asymptotically corrected coverage over a large (and generic) class of data-generating processes (honesty), and contract at the minimax sup-norm rate (adaptivity). We refer the reader to Chen, Christensen, and Kankanala (2024) for additional discussions.

We close this section by discussing the required assumptions such that one can reliably leverage the procedures in Chen, Christensen, and Kankanala (2024). Let $\Delta Y - \mathbb{E}[\Delta Y|D=0] = h(D) + u$. Under Assumption PT, $h(d) = ATT(d|d)$, whereas under Assumption SPT, $h(d) = ATT(d)$. Let $\overline{\sigma}, \underline{\sigma}, \overline{C}, \underline{c}$ be some finite, positive constants, and $\rho \in (0,1)$. Finally, let

$$\sigma^2_K(d) = \psi^K(d)'\mathbb{E}\left[\psi^K(D)\psi^K(D)'\middle|\, D>0\right]^- \mathbb{E}\left[u^2\psi^K(D)\psi^K(D)'\middle|\, D>0\right]\mathbb{E}\left[\psi^K(D)\psi^K(D)'\middle|\, D>0\right]^-\psi^K(d),$$

and $||\sigma_{d,K}||^2 = \psi^K(d)'\mathbb{E}\left[\psi^K(D)\psi^K(D)'\middle| D>0\right]^-\psi^K(d)$, which satisfies $||\sigma_{d,K}|| \asymp \sigma_K(d)$ under Assumption 5(ii) below. Let $\left|\left|\sigma^{acrt}_{d,K}\right|\right|^2 = (\partial\psi^K(d))'\mathbb{E}\left[\psi^K(D)\psi^K(D)'\middle| D>0\right]^-(\partial\psi^K(d))$.

Assumption 5 (Additional regularity conditions)**.**

(i) For all $d \in \mathcal{D}_+^c$, $a_f^{-1} < f_{D|D>0}(d) < a_f$ *for some positive constant* $a_f < \infty$, *and* $\mathbb{E}[\Delta Y|D=d]$ *is continuously differentiable on* $\mathcal{D}_+^c$.

(ii) $\mathbb{P}\left(\mathbb{E}\left[u^4|D, D>0\right] \le \overline{\sigma}^2\right) = 1$, *and* $\mathbb{P}\left(\mathbb{E}\left[u^2|D, D>0\right] \ge \underline{\sigma}^2\right) = 1$.

(iii) $\underline{c}K \le \inf_{d\in\mathcal{D}_+^c}||\sigma_{d,K}||^2 \le \sup_{d\in\mathcal{D}_+^c}||\sigma_{d,K}||^2 \le \overline{C}K$ *for all* $K \in \mathcal{K}$;

(iv) $\limsup_{K\to\infty}\sup_{d\in\mathcal{D}_+^c, K_2\in\mathcal{K}:K_2>K}(\sigma^2_K(d)/\sigma^2_{K_2}(d)) < \rho$;

(v) $\underline{c}K^3 \le \inf_{d\in\mathcal{D}_+^c}\left|\left|\sigma^{acrt}_{d,K}\right|\right|^2 \le \sup_{d\in\mathcal{D}_+^c}\left|\left|\sigma^{acrt}_{d,K}\right|\right|^2 \le \overline{C}K^3$ *for all* $K \in \mathcal{K}$.

Assumption 5(i) imposes that the density of the treatment dose among treated units is uniformly bounded away from zero, which may rule out "quasi-stayers".

The next assumption can be used to establish the large sample properties of our average derivative estimator based on $\widehat{ACRT}_{\text{cck}}(d)$ when taking the sieve dimension as non-stochastic. When applied together with Assumption 5, these assumptions imply Condition 2.1.1 and 2.1.2 of Ai and Chen (2007), with $s=1$.

Assumption 6 (Regularity conditions for average derivatives)**.**

(i) $f_{D|D>0}(d)$ *is continuously differentiable and is zero in the boundary of* $\mathcal{D}_+^c$.

(ii) $\mathbb{E}\left[\left(\dfrac{f'_{D|D>0}(D)}{f_{D|D>0}(D)}\right)^2 \middle| D>0\right] < \infty$, *where* $f'_{D|D>0}(d) = \left.\dfrac{df_{D|D>0}(a)}{da}\right|_{a=d}$.

Assumption 6(i) should be understood as imposing stronger conditions than Assumption 4(a), as it requires additional smoothness conditions on the density of the dosage. It also requires the density to go to zero in the boundary of the dose. When applied together with Assumption 4(a) and Assumption 5(i), Assumption 6(i) should be understood as ruling out positive density in the boundary of treatment dose.

C Multiple Periods and Variation in Treatment Timing and Dose

DiD applications often use more than two time periods, wherein treatments, whether binary or not, can turn on at different times for different units. This section extends the results from the main text to allow for multiple time periods ($t = 1, ..., T$) with variation in the time when units become treated. We refer to the time period when a unit becomes treated as a unit's *timing group*, which we denote by G_i, which takes values in the set $\mathcal{G}$. By convention, we set $G = \infty$ for units that remain untreated across all time periods, and we exclude units that are treated in the first period so that $\mathcal{G} \subseteq \{2, \ldots, T, \infty\}$; we also set $\bar{\mathcal{G}} = \mathcal{G} \setminus \{\infty\}$ to be the set of all timing groups that ever participate in the treatment. Treated units receive dose $D = d \in \mathcal{D}_+$. As in the two-period case, the dose actually experienced, D, also defines a unit's *dose group*. We focus on the case where treatment is an absorbing state, so that the amount of the treatment remains constant in post-treatment periods. Note that treated potential outcomes at time t depend on when a unit first becomes treated—i.e., $Y_{i,t}(g, d)$ may not equal $Y_{i,t}(g', d)$ for $g \neq g'$—which allows for general treatment effect dynamics. We also define the variable $W_{i,t} = D_i \mathbf{1}\{t \geq G_i\}$. which is the amount of dose that unit i experiences in time period t; $W_{i,t} = 0$ for all units that are not yet treated by time period t.

Throughout this section, we make the following assumptions.

Assumption 1-MP (Random Sampling)**.** *The observed data consists of* $\{Y_{i1}, \ldots, Y_{iT}, D_i, G_i\}_{i=1}^n$ *which is independent and identically distributed.*

Assumption 2-MP (Support)**.**

(a) The support of D, $\mathcal{D} = \{0\} \cup \mathcal{D}_+$ *where* $\mathcal{D}_+ \subseteq (0, \infty)$. *In addition,* $\mathbb{P}(D = 0) > 0$ *and* $dF_{D|G}(d|g) > 0$ *for all* $(g, d) \in \bar{\mathcal{G}} \times \mathcal{D}_+$.

(b) $\mathcal{D}_+^c = (d_L, d_U) \subset \mathcal{D}_+$. *In addition, for all* $g \in \bar{\mathcal{G}}$ *and* $t = 2, \ldots, T$, $\mathbb{E}[\Delta Y_t | G = g, D = d]$ *is continuously differentiable in* d *on* $\mathcal{D}_+^c$.

Assumption 3-MP (No Anticipation / Staggered Adoption)**.**

(a) For all $g \in \mathcal{G}$ *and* $t = 1, \ldots, T$ *with* $t < g$ *(i.e., in pre-treatment periods),* $Y_{i,t}(g, d) = Y_{i,t}(0)$.

(b) $W_{i,1} = 0$ *almost surely, and, for* $t = 2, \ldots, T$, $W_{i,t-1} = d$ *implies that* $W_{i,t} = d$.

Assumption 1-MP says that we have access to T periods of panel data and observe each unit's dose and treatment timing. Assumption 2-MP extends our definitions of the support of D to the case with multiple periods and variation in treatment timing. As in earlier sections, many of our identification results only require part (a) (which allows for very general treatment regimes) while some of our results are specialized to the continuous case as in part (b).[39] Assumption 2-MP also imposes a kind of common support of the dose across timing groups, though it allows for the distribution of the dose to vary across timing groups in otherwise unrestricted ways; that said, it appears to be straightforward to relax this part of the assumption at the cost of additional notation.

Assumption 3-MP(a) rules out that units anticipate experiencing the treatment in ways that affect their outcomes before they actually participate in the treatment. It would be relatively straightforward to extend our arguments in this section to allow for anticipation along the lines of Callaway and Sant'Anna (2021) (in the case of a binary treatment). Assumption 3-MP(b) implies that we consider the case with staggered adoption which means that, once a unit becomes treated with dose d, it remains treated with dose d in all subsequent periods. This allows us to fully categorize a unit by the timing of their treatment adoption and the amount of dose that they experience.

For each unit, we observe their outcome in period t, $Y_{i,t}$, which is given by

$$Y_{i,t} = Y_{i,t}(0)\mathbf{1}\{t < G_i\} + Y_{i,t}(G_i, D_i)\mathbf{1}\{t \geq G_i\}.$$

In other words, we observe a unit's untreated potential outcomes in time periods before they participate in the treatment, and we observe treated potential outcomes in post-treatment time periods corresponding to the timing of the treatment and the amount of the dose experienced.

C.1 Parameters of Interest with a Staggered Continuous Treatment

The causal parameters of interest are the same as in our baseline case, except that they are separately defined for each timing group and in each post-treatment time period:

$$ATT(g, t, d|g, d) = \mathbb{E}[Y_t(g, d) - Y_t(0)|G = g, D = d],$$

which is the average treatment effect of dose d, for timing group g, in time period t, among units in group g that experienced dose d. $ATT(g, t, d|g, d)$ generalizes $ATT(d|d)$ from the setting with two time periods to the setting with multiple periods and variation in treatment timing; e.g., specialized to the case with two time periods, $ATT(2, 2, d|2, d)$ is what we called $ATT(2|2)$ in the main text. $ATT(g, t, d|g, d)$ is also related to the group-time average treatment effects discussed in Callaway and Sant'Anna (2021) except for also being specific to a particular dose, and allows for the effect of dose to vary arbitrarily across timing groups, time periods, and dose groups.

Causal response parameters are similarly defined as the effect of a marginal change in the dose on the outcomes of timing group g in period t. For continuous treatments, these are defined as

$$ACRT(g, t, d|g, d) = \left.\frac{\partial ATT(g, t, l|g, d)}{\partial l}\right|_{l=d} = \left.\frac{\partial \mathbb{E}\left[Y_t(g, l)|G = g, D = d\right]}{\partial l}\right|_{l=d}.$$

[39] For the results in this section that are specialized to the case where the treatment is continuous, it is straightforward to adjust them to allow for a multi-valued discrete treatment along the same lines as in the main text.

For discrete treatments, these are defined as

$$ACRT(g,t,d_j|g,d_j) = \mathbb{E}[Y_t(g,d_j) - Y_t(g,d_{j-1})|D = d_j, G = g] \Big/ (d_j - d_{j-1}).$$

$ACRT(g,t,d|g,d)$ is the causal response version of $ATT(g,t,d|g,d)$. It is a causal response that is local to a specific timing group, time period, and dose group.

For brevity and clarity, in this section we focus on the "local" causal effect parameters $ATT(g,t,d|g,d)$ and $ACRT(g,t,d|g,d)$, which are analogous to the local causal effect parameters $ATT(d|d)$ and $ACRT(d|d)$ in the two-period case that we emphasized in the main text. In an earlier working version of our paper (Callaway, Goodman-Bacon, and Sant'Anna, 2025), we additionally consider in more detail "global" parameters $ATT(g,t,d) := \mathbb{E}[Y_t(g,d) - Y_t(0)|G = g]$[40] and $ACRT(g,t,d) := \frac{\partial ATT(g,t,d)}{\partial d}$. These parameters are analogous to the global causal effect parameters $ATT(d)$ and $ACRT(d)$ in the two-period case and can be identified under a multiple periods version of strong parallel trends.

One important new issue that arises with multiple periods and variation in treatment timing is that, in many applications, $ATT(g,t,d|g,d)$ and $ACRT(g,t,d|g,d)$ are relatively high-dimensional and challenging to report. Therefore, in the next section, we provide several ways to aggregate these parameters into lower-dimensional causal parameters that are easier to report/estimate. We focus on two sorts of aggregations. The first is to aggregate across timing groups and time periods into causal parameters $ATT^{dose}(d|d)$ and $ACRT^{dose}(d|d)$ that are functions of only the dose. The second aggregation averages across the dose (and combines timing groups and calendar time into event time) to deliver the event study parameters $ATT^{es}_{\texttt{loc}}(e)$ and $ACRT^{es}_{\texttt{loc}}(e)$.

Aggregations highlighting dose-specific effects

In this section, we propose aggregated causal effect parameters that average over timing groups and time periods to highlight how treatment effects vary across different doses. Toward this end, among units that ever participate in the treatment (i.e., $G_i \neq \infty$), define

$$\overline{TE}_i(d) = \frac{1}{T - G_i + 1} \sum_{t=G_i}^{T} \Big(Y_{i,t}(G_i, d) - Y_{i,t}(0) \Big),$$

which, for a particular unit i, is its average treatment effect of dose d across all of its post-treatment periods. We define the following aggregated treatment effect parameter:

$$ATT^{dose}(d|d) = \mathbb{E}\Big[\overline{TE}(d) \Big| D = d, G \leq T\Big],$$

which is the average treatment effect of dose d across post-treatment periods for dose group d. We can likewise define a causal response parameter

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

$ATT^{dose}(d|d)$ and $ACRT^{dose}(d|d)$ average out time periods and timing groups, resulting in a summary parameter that is a function of the dose only. In this way, they are similar to $ATT(d|d)$ and

[40] $ATT(g,t,d)$ is global in the sense that it is across all treated dose groups.

$ACRT(d|d)$ considered in the main text in the setting with only two time periods. As in the main text, we can further aggregate these parameters into scalar summary parameters:

$$ATT^{\texttt{loc}} = \mathbb{E}\Big[ATT^{dose}(D|D)\Big|G \leq T\Big] \quad \text{and} \quad ACRT^{\texttt{loc}} = \mathbb{E}\Big[ACRT^{dose}(D|D)\Big|G \leq T\Big]$$

$ATT^{\texttt{loc}}$ and $ACRT^{\texttt{loc}}$ are fully aggregated parameters that can summarize "on-the-treated" level effects and causal responses, respectively; they are analogous to the homonymous parameters discussed in the two-period case considered in the main text. Next, we argue that $ATT^{dose}(d|d)$ (and, hence, $ATT^{\texttt{loc}}$) is identified if $ATT(g,t,d|g,d)$ is identified—therefore, the identification results in the next section can target those more disaggregated parameters. To see this, notice that

$$\begin{aligned}
ATT^{dose}(d|d) &= \mathbb{E}\Big[\overline{TE}(d)\Big|D=d, G\leq T\Big] \\
&= \sum_{g\in\bar{\mathcal{G}}} \frac{1}{T-g+1}\sum_{t=2}^{T} \mathbf{1}\{t\geq g\}\mathbb{E}\Big[Y_{i,t}(g,d) - Y_{i,t}(0)\Big|G=g, D=d\Big]\mathbb{P}(G=g|D=d, G\leq T) \\
&= \sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T} \omega^{dose}(g,t,d)ATT(g,t,d|g,d)
\end{aligned}$$

where $\omega^{dose}(g,t,d) = \frac{\mathbf{1}\{t\geq g\}}{T-g+1}\mathbb{P}(G=g|D=d, G\leq T)$ and where the first equality comes from the definition of $ATT^{dose}(d|d)$, the second equality holds by the definition of $\overline{TE}_i(d)$ and by the law of iterated expectations, and the third equality holds by the definition of $\omega^{dose}(g,t,d)$. It is also straightforward to see that, given some value of d, $\omega^{dose}(g,t,d)$ is non-negative for all values of (g,t,d) and that $\sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T}\omega^{dose}(g,t,d) = 1$.

From the same sort of argument, it is straightforward to show that

$$ACRT^{dose}(d|d) = \sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T} \omega^{dose}(g,t,d)ACRT(g,t,d|g,d)$$

which shows that $ACRT^{dose}(d|d)$ and $ACRT^{loc}$ will be identified if $ACRT(g,t,d|g,d)$ is identified.

Event-study aggregations

Next, we consider event-study aggregations that highlight how treatment effects and causal responses vary with length of exposure to the treatment. Toward this end, among units that are ever observed to participate in the treatment for e periods (i.e., these are units for which $G_i + e \in \{2,\ldots,T\}$), define $TE_i(d|e) = Y_{i,G_i+e}(G_i,d) - Y_{i,G_i+e}(0)$ which is the treatment effect of dose d for unit i when it has been exposed to the treatment for e periods. Next, we define two intermediate parameters

$$\begin{aligned}
\widetilde{ATT}^{dose,es}(d|d,e) &= \mathbb{E}\Big[TE(d|e)\Big|D=d, G+e\in[2,T], G\leq T\Big], \\
\widetilde{ACRT}^{dose,es}(d|d,e) &= \left.\frac{\partial\widetilde{ATT}^{dose,es}(l|d,e)}{\partial l}\right|_{l=d}
\end{aligned}$$

where $\widetilde{ATT}^{dose,es}(d|d,e)$ and $\widetilde{ACRT}^{dose,es}(d|d,e)$ are the average treatment effect of dose d and average causal response of dose d among those in dose group d for those that have been exposed to the

treatment for e periods. If there is a particularly interesting value of the dose d, then it is possible to fix that value of d and report an event study that varies e using either of these parameters. The other leading approach is to average these parameters over the distribution of the dose. Consider

$$ATT^{es}_{\texttt{loc}}(e) = \mathbb{E}\Big[\widetilde{ATT}^{dose,es}(D|D,e)\Big|G+e\in[2,T],G\leq T\Big]$$
$$ACRT^{es}_{\texttt{loc}}(e) = \mathbb{E}\left[\widetilde{ACRT}^{dose,es}(D|D,e)\Big|G+e\in[2,T],G\leq T\right]$$

which provide event study versions of average treatment effects and average causal responses across different lengths of exposure to the treatment. For values of $e\geq 0$, $ATT^{es}_{\texttt{loc}}(e)$ and $ACRT^{es}_{\texttt{loc}}(e)$ are related to treatment effect dynamics. It is also interesting to consider cases where $e<0$, which can be interpreted as a pre-test of the parallel trends assumption. Next we show that $\widetilde{ATT}^{dose,es}(d|d,e)$ and $\widetilde{ACRT}^{dose,es}(d|d,e)$ can be related to the corresponding underlying, disaggregated parameters $ATT(g,t,d|g,d)$ and $ACRT(g,t,d|g,d)$, implying that the more aggregated parameters are identified if the less aggregated parameters are identified. Toward this end, let $\pi_g(e,d)=\mathbb{P}(G=g|D=d,G+e\in[2,T],G\leq T)$, and notice that

$$\begin{aligned}
\widetilde{ATT}^{dose,es}(d|d,e) &= \mathbb{E}\Big[TE(d|e)|D=d,G+e\in[2,T],G\leq T\Big] \\
&= \sum_{g\in\bar{\mathcal{G}}}\mathbf{1}\{g+e\in[2,T]\}\mathbb{E}[Y_{g+e}(g,d)-Y_{g+e}(0)|G=g,D=d]\pi_g(e,d) \\
&= \sum_{g\in\bar{\mathcal{G}}}\left\{\mathbf{1}\{g+e\in[2,T]\}\mathbb{E}[Y_{g+e}(g,d)-Y_{g+e}(0)|G=g,D=d]\pi_g(e,d)\sum_{t=2}^{T}\mathbf{1}\{g+e=t\}\right\} \\
&= \sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T}\mathbf{1}\{g+e\in[2,T]\}\mathbf{1}\{g+e=t\}\mathbb{E}[Y_t(g,d)-Y_t(0)|G=g,D=d]\pi_g(e,d) \\
&= \sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T}\omega^{dose,es}(g,t,d|e)ATT(g,t,d|g,d)
\end{aligned}$$

where $\omega^{dose,es}(g,t,d|e)=\mathbf{1}\{g+e\in[2,T]\}\mathbf{1}\{g+e=t\}\pi_g(e,d)$ and where the first equality holds by the definition of $\widetilde{ATT}^{dose,es}(d|d,e)$, the second equality holds by the law of iterated expectations, the third equality holds because $\sum_{t=2}^{T}\mathbf{1}\{g+e=t\}=1$ among groups that are observed to participate in the treatment for e periods, the fourth equality holds by combining the summations, and the last equality holds by the definitions of $\omega^{dose,es}$ and $ATT(g,t,d|g,d)$. The same sort of argument also implies that

$$\widetilde{ACRT}^{dose,es}(d|d,e)=\sum_{g\in\bar{\mathcal{G}}}\sum_{t=2}^{T}\omega^{dose,es}(g,t,d|e)ACRT(g,t,d|g,d)$$

This discussion highlights that if $ATT(g,t,d|g,d)$ and/or $ACRT(g,t,d|g,d)$ are identified, then we can recover the more aggregated summary parameters that have been discussed in this section.

C.2 Identification with a Continuous Treatment and Staggered Timing

As emphasized above, with multiple periods and variation in treatment timing, identification of a large number of causal effect parameters comes down to identifying $ATT(g,t,d|g,d)$ and $ACRT(g,t,d|g,d)$. With multiple time periods and variation in treatment timing, there are several possible versions of parallel trends and strong parallel trends assumptions that one could make. In this section, we focus on leading versions and representative arguments; see an earlier working version of our paper (Callaway, Goodman-Bacon, and Sant'Anna, 2025) for more details regarding different varieties of parallel trends assumptions that could be used in this context. We next introduce versions of Assumption PT and SPT that are suitable for the setting with multiple periods and variation in treatment timing.

Assumption PT-MP (Parallel Trends with Multiple Periods and Variation in Treatment Timing)**.** *For all $g \in \bar{\mathcal{G}}$, $t = 2,\ldots,T$, $d \in \mathcal{D}_+$, $\mathbb{E}[\Delta Y_t(0)|G=g,D=d] = \mathbb{E}[\Delta Y_t(0)|G=\infty,D=0]$.*

Assumption SPT-MP (Strong Parallel Trends with Multiple Periods and Variation in Treatment Timing)**.** *For all $g \in \bar{\mathcal{G}}$, $t = 2,\ldots,T$, and $l,d \in \mathcal{D}_+$, $\mathbb{E}[Y_t(g,d) - Y_{t-1}(g,d)|G=g,D=l] = \mathbb{E}[Y_t(g,d) - Y_{t-1}(g,d)|G=g,D=d]$ and $\mathbb{E}[\Delta Y_t(0)|G=g,D=d] = \mathbb{E}[\Delta Y_t(0)|G=\infty,D=0]$.*

Assumption PT-MP says that the average paths of untreated potential outcomes are the same for all groups and for all doses across all time periods. Assumption SPT-MP strengthens PT-MP as it additionally restricts paths of treated potential outcomes (not just paths of untreated potential outcomes) so that all dose groups treated at time g would have had the same average path of potential outcomes under dose d as those in group g that actually experienced dose d (and that this holds for all doses).[41]

Theorem C.1. *Under Assumptions 1-MP, 2-MP(a), 3-MP, and PT-MP, and for all $g \in \bar{\mathcal{G}}$, $t = 2,\ldots,T$ such that $t \geq g$, and for all $d \in \mathcal{D}_+$,*

$$ATT(g,t,d|g,d) = \mathbb{E}[Y_t - Y_{g-1}|G=g,D=d] - \mathbb{E}[Y_t - Y_{g-1}|W_t=0].$$

If, in addition, Assumptions 2-MP(b) and SPT-MP hold, then, for all $d \in \mathcal{D}_+^c$,

$$ACRT(g,t,d|g,d) = \frac{\partial \mathbb{E}[Y_t - Y_{g-1}|G=g,D=d]}{\partial d}.$$

The proof of Theorem C.1 is provided in Appendix SB in the Supplementary Appendix. The result is broadly similar to the one in the case with two periods. The first part says that, under Assumption PT-MP, $ATT(g,t,d|g,d)$ can be recovered by a DiD comparison between the path of outcomes from period $g-1$ to period t for units in group g treated with dose d and the path of

[41] Besides differences related to multiple periods and variation in treatment timing, the version of strong parallel trends made here is slightly different from Assumption SPT in the main text. Part of the difference comes from there being no untreated units in group $g \in \bar{\mathcal{G}}$, which is why there is a separate part of the assumption for untreated potential outcomes. The other difference is that both parts of the assumption hold for all dose groups rather than on average (i.e., we condition on dose group l in the first part and on dose group d in the part for untreated potential outcomes). The version here is stronger, though only slightly, and is made for clarity and because we target $ACRT(g,t,d|g,d)$ rather than $ACRT(g,t,d)$ in this part of the paper. See Callaway, Goodman-Bacon, and Sant'Anna (2025) for more details and for other combinations of target parameters and identifying assumptions.

outcomes among units that have not participated in the treatment yet (the setup in this section also rationalizes using the never-treated group, $G = \infty$, as the comparison group as was mentioned in Section 5). Relative to the case with two time periods, the main difference is that the "base period" is $g-1$. The reason for using the base period $g-1$ is that it is the most recent time period when the researcher observes untreated potential outcomes for units in group g. Thus, the result is very much like the case with two time periods: take the most recent untreated potential outcomes for units in a particular group, impute the path of outcomes that they would have experienced in the absence of participating in the treatment from the group of not-yet-treated units (these steps yield mean untreated potential outcomes that units in group g would have experienced in time period t) and compare this to the outcomes that are actually observed for units in group g that experienced dose d. The second part says that, under Assumption SPT-MP, $ACRT(g,t,d|g,d)$ can be recovered by taking the derivative of the average path of outcomes from period $g-1$ to period t among timing group g that experienced dose d. Similarly to the arguments in the main text, if Assumption PT-MP held rather than SPT-MP, then the same derivative term would additionally include selection bias terms.

Remark C.1. *We do not provide formal estimation results for the setting with multiple periods and variation in treatment timing. However, we note that, if one bases estimation on the sample analog of the results in Theorem C.1, then the results in the main text for the case with two periods apply directly to the disaggregated parameters* $ATT(g,t,d|g,d)$ *and* $ACRT(g,t,d|g,d)$*. Then, one can estimate any of the aggregated parameters discussed above as the appropriate weighted average of* $ATT(g,t,d|g,d)$ *or* $ACRT(g,t,d|g,d)$*. Interestingly, given the results in Corollary 3.1 and Callaway and Sant'Anna (2021), when* $ATT^{es}_{loc}(e)$ *is the target parameter, one can binarize the treatment (i.e., classify units as being treated if they experience any positive amount of the treatment) and simply rely on the event-study procedures proposed by Callaway and Sant'Anna (2021).*