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# CONTAMINATION BIAS IN LINEAR REGRESSIONS

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**ABSTRACT**

We study the interpretation of regressions with multiple treatments and flexible controls. Such regressions are often used to analyze stratified randomized control trials with multiple intervention arms, to estimate value-added (for, e.g., teachers) with observational data, and to leverage the quasi-random assignment of decision-makers (e.g. bail judges). We show that these regressions generally fail to estimate convex averages of heterogeneous treatment effects, even when the treatments are conditionally randomly assigned and the controls are sufficiently flexible to avoid omitted variables bias. Instead, estimates of each treatment's effects are generally contaminated by a non-convex average of the effects of other treatments. Thus, recent concerns about heterogeneity-induced bias in regressions leveraging potential outcome restrictions (e.g. parallel trends assumptions) also arise with "design-based" identification strategies. We discuss solutions to the contamination bias and propose a new class of efficient estimators of weighted average effects that avoid bias. In a re-analysis of the Project STAR trial, we find minimal bias because treatment effect heterogeneity is largely idiosyncratic. But sizeable contamination bias arises when effect heterogeneity becomes correlated with treatment propensity scores.

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A stata package for multiple treatment effect regression is available at
https://github.com/gphk-metrics/stata-multe/

# 1 Introduction

Consider a linear regression of an outcome $Y_i$ on a vector of mutually exclusive treatment indicators $X_i$ and a vector of flexible controls $W_i$. The treatments are assumed to be as good as randomly assigned, conditional on the controls. For example, $X_i$ may indicate the assignment of individuals $i$ to different interventions in a stratified randomized control trial (RCT), with the randomization protocol varying across some experimental strata indicators in $W_i$. Or, in an education value-added model (VAM), $X_i$ might indicate the matching of students $i$ to different teachers or schools with $W_i$ including measures of student demographics, lagged achievement, or other controls which give a credible selection-on-observables assumption. The regression might also be the first stage of an instrumental variables (IV) regression, perhaps leveraging the as-good-as-random assignment of multiple decision-makers (e.g. bail judges or benefit administrators) indicated in $X_i$, conditional on some controls $W_i$. These sorts of regressions are widely used across many fields in economics.[1]

This paper shows that such regressions generally fail to identify convex weighted averages of heterogeneous treatment effects, and discusses solutions to this problem. The problem may be surprising given an influential result in Angrist (1998), showing that regressions on a single binary treatment $D_i$ and flexible controls $W_i$ estimate a convex weighted average of treatment effects whenever $D_i$ is conditionally as good as randomly assigned. We show that this result does not generalize to multiple treatments. Despite a set of treatments being completely randomly assigned within groups, as in a stratified multi-armed RCT, a regression on treatment and strata indicators generally fails to yield causally interpretable regression coefficients. Instead, regression estimates of each treatment's effect are generally contaminated by a non-convex average of the effects of other treatments: the regression coefficient for a given RCT treatment arm generally incorporates the effects of *all* arms.

We first derive a general characterization of this "contamination bias" in multiple-treatment regressions. To separate the problem from the well-known challenge of omitted variables bias (OVB), we assume a best-case scenario where the covariate parametrization is flexible enough to include the treatment propensity scores (e.g., with a linear covariate adjustment, we assume that the propensity scores are linear in the covariates). This condition holds trivially if the only covariates are strata indicators. We show that the regression coefficient on each treatment identifies a convex weighted average of its causal effects, plus a contamination bias term that is generally non-zero. The bias term is given by a linear combination of the

[1] Prominent RCT examples include Project STAR (Krueger, 1999) and the RAND Health Insurance Experiment (Manning et al., 1987). Prominent VAM examples include studies of teachers (Kane & Staiger, 2008; Chetty et al., 2014), schools (Angrist et al., 2017; Angrist et al., 2021; Mountjoy & Hickman, 2020), and healthcare institutions (Hull, 2018a; Abaluck et al., 2021; Geruso et al., 2020). Prominent "judge IV" examples include Kling (2006), Maestas et al. (2013), and Dobbie and Song (2015).

causal effects of other treatments, with weights that sum to zero. As a result, each treatment effect estimate will generally incorporate the effects of other treatments, unless the effects are uncorrelated with the contamination weights. Since the contamination weights sum to zero, some are necessarily negative—further complicating the interpretation of the coefficients.

Contamination bias arises because regression adjustment for the confounders in $W_i$ is generally insufficient for making the other treatments ignorable when estimating a given treatment's effect, even when this adjustment is flexible enough to avoid OVB. To see this intuition clearly, consider the most flexible specification of controls as a set of strata indicators. OVB is avoided when the treatments are as good as randomly assigned within strata. But because the treatments enter the regression linearly, the Angrist (1998) result implies that the causal interpretation of a *given* treatment's coefficient is only guaranteed when its assignment depends linearly on both the strata indicators *and* the other treatment indicators. With mutually exclusive treatments, this condition fails because the dependence is inherently nonlinear—the probability of assignment to a given treatment is zero if an individual is assigned to one of the other treatments, regardless of their stratum, but strata indicators affect the treatment probability otherwise.

We show that contamination bias also arises under an alternative "model-based" identifying assumption that, instead of making assumptions on the treatment's "design" (i.e. propensity scores), assumes the regression parametrization of covariates is flexible enough to include the conditional mean of the potential outcome under no treatment, $Y_i(0)$. In a linear model with two-way unit and time fixed effects, this reduces to the parallel trends restriction used in difference-in-differences (DiD) and event study regressions. It is common for $X_i$ to include multiple indicators in such settings—for example, the leads and lags relative to a treatment adoption date used to support the parallel trends assumption or estimate treatment effect dynamics.[2] We show that replacing the restriction on propensity scores with an assumption on $Y_i(0)$ generates an additional source of bias: the own-treatment effect weights are no longer guaranteed to be positive. This result shows that the negative weighting and contamination bias issues documented previously in the context of two-way fixed effects regressions (e.g., Goodman-Bacon, 2021; Sun & Abraham, 2021; de Chaisemartin & D'Haultfœuille, 2020, 2022; Callaway & Sant'Anna, 2021; Borusyak et al., 2022; Wooldridge, 2021; Hull, 2018b) are more general—and conceptually distinct—problems.[3] Negative weighting arises because regressions leveraging model-based restrictions on $Y_i(0)$ are generally not robust to treatment effect heterogeneity. Contamination bias arises because linear regression fails to account for the non-linear dependence across multiple dependent treatments and controls.

[2] Alternatively $X_i$ may indicate multiple contemporaneous treatments, as in certain "mover" regressions.

[3] Our analysis also relates to issues with interpreting multiple-treatment IV estimates (Behaghel et al., 2013; Kirkeboen et al., 2016; Kline & Walters, 2016; Hull, 2018c; Lee & Salanié, 2018; Bhuller & Sigstad, 2022).

Even more broadly, contamination bias can arise in descriptive regressions which seek to estimate averages of certain conditional group contrasts without assuming a causal framework—as in studies of treatment or outcome disparities across multiple racial or ethnic groups, studies of regional variation in healthcare utilization or outcomes, or studies of industry wage gaps.[4] Our analysis shows that in such regressions the coefficient on a given group or region averages the conditional contrasts across all other groups or regions, with non-convex weights.

Our bias characterization also has implications for IV regressions leveraging multiple correlated instruments, such as indicators for as good as randomly assigned judges. Contamination bias in the first-stage regression of treatment on multiple instruments and flexible controls (e.g. courtroom fixed effects) can generate violations of the effective first-stage monotonicity restriction, even when conventional first-stage monotonicity is satisfied unconditionally. We show how this problem is distinct from previous concerns over the monotonicity assumption in judge IV designs (Mueller-Smith, 2015; Frandsen et al., 2019; Norris, 2019; Mogstad et al., 2021) and over insufficient flexibility in the control parameterization (Blandhol et al., 2022).[5]

We then discuss three solutions to the contamination bias problem, and their trade-offs, in the baseline case of conditionally ignorable treatments. One conceptually principled solution is to adapt approaches to estimating the average treatment effect (ATE) of a conditionally ignorable binary treatment (see, e.g. Imbens & Wooldridge, 2009, for a review) to the multiple treatment case (e.g. Cattaneo, 2010; Chernozhukov et al., 2021; Graham & Pinto, 2022). For example, one could run an expanded regression that includes interactions between the treatments and (demeaned) controls.[6] Such ATE estimators achieve the semiparametric efficiency bound under an assumption of strong overlap of the covariate distribution for units in each treatment arm. But this approach may be infeasible or yield imprecise estimates under limited overlap—a common scenario in practice (Crump et al., 2009).

This practical consideration motivates an alternative approach: estimating a convex weighed average of treatment effects, as regression does in the binary treatment case, while avoiding the contamination bias of multiple-treatment regressions. We derive the weights that are "easiest" to estimate, in that they minimize a semiparametric efficiency bound under homoskedasticity. These optimal weights coincide with the implicit linear regression weights when the treatment is binary (i.e. the Angrist (1998) case), formalizing a virtue of regression adjustment. In the multiple treatment case, the optimal weights for a given treatment-control contrast are similarly given by a linear regression which restricts estimation to the individuals who are either in the control group or the treatment group of interest. For estimating effects

[4]Prominent examples of such analyses respectively include Fryer and Levitt (2013), Skinner (2011), and Krueger and Summers (1988).

[5]The contamination bias issue is also distinct from the Freedman (2008a, 2008b) critique of regression to analyze randomized trials, which concerns estimation not identification.

[6]In the judge IV case, the analogous solution interacts judge indicators with courtroom fixed effects.

that are directly comparable across multiple treatments, our optimal weight characterization leads to a new estimator. We give guidance for how applied researchers can gauge the extent of contamination bias in practice and apply the different solutions with help from a new Stata package, `multe`.[7]

We illustrate the contamination bias problem and solutions in an application to the Project STAR trial, which randomized students within schools to either a small classroom treatment, a teaching aide treatment, or control conditions. We find the potential for sizeable bias in estimates of both treatment effects, from regressions with school fixed effects, due to significant treatment effect heterogeneity. Nevertheless, we show that the actual contamination is likely to be minimal, as the effect heterogeneity turns out to be largely uncorrelated with the contamination weights. The application thus highlights the importance of testing the empirical relevance of theoretical concerns with how regression combines heterogeneous effects.

We structure the rest of the paper as follows. Section 2 illustrates contamination bias in a simple example with two mutually exclusive treatment indicators and one binary control. Section 3 characterizes the general problem in regressions with multiple treatments and flexible controls, and discusses connections to previous analyses. Section 4 discusses the robustness and efficiency properties of three solutions. Section 5 gives guidance for measuring and avoiding contamination bias in practice, and illustrates these tools in the Project STAR experiment. Section 6 concludes. All proofs and extensions are given in Appendix A.

## 2 Motivating Example

We build intuition for the contamination bias problem in two simple examples. We first review how regressions on a single randomized binary treatment and binary controls identify a convex average of heterogeneous treatment effects. We then show how this result fails to generalize when we introduce an additional treatment arm. We base these examples on a stylized version of the Project STAR experiment, which we return to in our application in Section 5.2.

### 2.1 Convex Weights with One Randomized Treatment

Consider the regression of an outcome $Y_i$ on a single treatment indicator $D_i \in \{0, 1\}$, a single binary control $W_i \in \{0, 1\}$, and a constant:

$$Y_i = \alpha + \beta D_i + \gamma W_i + U_i. \tag{1}$$

By definition, $U_i$ is a mean-zero regression residual that is uncorrelated with $D_i$ and $W_i$. Krueger (1999), for example, primarily studied the effect of small class size $D_i$ on the test

[7] The Stata package is available at https://github.com/gphk-metrics/stata-multe.

scores $Y_i$ of middle school students indexed by $i$. Project STAR randomized students to classes within schools with at least three classes per grade. The number of students assigned to each intervention thus varied both by the number of students in a school and the relative classroom size. To account for this non-random treatment variation, Krueger (1999) followed earlier analyses of Project STAR in estimating regressions with school (and sometimes school-by-period) fixed effects as controls. Such specifications are often found in stratified RCTs with varying treatment assignment rates across a set of pre-treatment strata. If we imagine two such strata, demarcated by a binary indicator $W_i$, then eq. (1) corresponds to a stylized two-school version of a Project STAR regression.

We wish to interpret the regression coefficient $\beta$ in terms of the causal effects of $D_i$ on $Y_i$. For this we use potential outcome notation, letting $Y_i(d)$ denote the test score of student $i$ when $D_i = d$. Individual $i$'s treatment effect is then given by $\tau_i = Y_i(1) - Y_i(0)$, and we can write realized achievement as $Y_i = Y_i(0) + \tau_i D_i$. To formalize the random assignment of treatment within schools, we assume that $D_i$ is conditionally independent of potential outcomes given the control $W_i$:

$$(Y_i(0), Y_i(1)) \perp D_i \mid W_i. \tag{2}$$

Angrist (1998) showed that regression coefficients like $\beta$ identify a weighted average of within-strata ATEs, with convex weights.[8] In our stylized Project STAR regression, this result shows:

$$\beta = \phi\tau(0) + (1-\phi)\tau(1), \quad \text{where} \quad \phi = \frac{\text{var}(D_i \mid W_i = 0)\Pr(W_i = 0)}{\sum_{w=0}^{1}\text{var}(D_i \mid W_i = w)\Pr(W_i = w)} \in [0,1] \tag{3}$$

gives a convex weighting scheme and $\tau(w) = E[Y_i(1) - Y_i(0) \mid W_i = w]$ is the ATE in school $w \in \{0,1\}$. Thus, in our example the coefficient $\beta$ identifies a weighted average of school-specific small classroom effects $\tau(w)$ across the two schools.

Equation (3) can be derived by applying the Frisch-Waugh-Lovell (FWL) Theorem. The multivariate regression coefficient $\beta$ can be written as a univariate regression coefficient from regressing $Y_i$ onto the population residual $\tilde{D}_i$ from regressing $D_i$ onto $W_i$ and a constant:

$$\beta = \frac{E[\tilde{D}_i Y_i]}{E[\tilde{D}_i^2]} = \frac{E[\tilde{D}_i Y_i(0)]}{E[\tilde{D}_i^2]} + \frac{E[\tilde{D}_i D_i \tau_i]}{E[\tilde{D}_i^2]}, \tag{4}$$

where we substitute the potential outcome model for $Y_i$ in the second equality. Since $W_i$ is binary, the propensity score $E[D_i \mid W_i]$ is linear and the residual $\tilde{D}_i$ is mean independent of

[8]See Słoczyński (2022) for an alternative representation of this estimand, in terms of conditional average effects on the treated and untreated, under slightly different assumptions.

$W_i$ (not just uncorrelated with it): $E[\tilde{D}_i \mid W_i] = 0$. Therefore,

$$E[\tilde{D}_i Y_i(0)] = E[E[\tilde{D}_i Y_i(0) \mid W_i]] = E[E[\tilde{D}_i \mid W_i] E[Y_i(0) \mid W_i]] = 0. \tag{5}$$

The first equality in eq. (5) follows from the law of iterated expectations, the second equality follows by the conditional random assignment of $D_i$ and the third equality uses $E[\tilde{D}_i \mid W_i] = 0$. Hence, the first summand in eq. (4) is zero. Analogous arguments show that

$$E[\tilde{D}_i D_i \tau_i] = E[E[\tilde{D}_i D_i \tau_i \mid W_i]] = E[E[\tilde{D}_i D_i \mid W_i] E[\tau_i \mid W_i]] = E[\text{var}(D_i \mid W_i)\tau(W_i)],$$

where $\text{var}(D_i \mid W_i) = E[\tilde{D}_i^2 \mid W_i]$ gives the conditional variance of the small-class treatment within schools. Since $E[\text{var}(D_i \mid W_i)] = E[E[\tilde{D}_i^2 \mid W_i]] = E[\tilde{D}_i^2]$, it follows that we can write the second summand in eq. (4) as

$$\beta = \frac{E[\text{var}(D_i \mid W_i)\tau(W_i)]}{E[\text{var}(D_i \mid W_i)]} = \phi\tau(0) + (1 - \phi)\tau(1),$$

proving the representation of $\beta$ in eq. (3).

The key fact underlying this derivation is that the residual $\tilde{D}_i$ from the auxiliary regression of the treatment $D_i$ on the other regressors $W_i$ is mean-independent of $W_i$. By the FWL theorem, treatment coefficients like $\beta$ can always be represented as in eq. (4) even without this property. We next show, however, that the remaining steps in the derivation of eq. (3) fail when an additional treatment arm is included. This failure can be attributed to the fact that the auxiliary FWL regression delivers a treatment residual that is uncorrelated, but not mean-independent of the other regressors, leading to an additional bias term in the expression for the regression coefficient.

### 2.2 Contamination Bias with Two Randomized Treatments

In reality, as noted above, Project STAR randomized two mutually exclusive interventions within schools: a reduction in class size ($D_i = 1$) and the introduction of full-time teaching aides ($D_i = 2$). We incorporate this extension of our stylized example by considering a regression of student achievement $Y_i$ on a vector of two treatment indicators, $X_i = (X_{i1}, X_{i2})'$, where the first element $X_{i1} = \mathbb{1}\{D_i = 1\}$ indicates assignment to a small class and the second element $X_{i2} = \mathbb{1}\{D_i = 2\}$ indicates assignment to a class with a full-time aide. We continue to include a constant and the school indicator $W_i$ as controls, yielding the regression

$$Y_i = \alpha + \beta_1 X_{i1} + \beta_2 X_{i2} + \gamma W_i + U_i. \tag{6}$$

To account for the second treatment, the observed outcome is now given by $Y_i = Y_i(0) + \tau_{i1} X_{i1} + \tau_{i2} X_{i2}$, with $\tau_{i1} = Y_i(1) - Y_i(0)$ and $\tau_{i2} = Y_i(2) - Y_i(0)$ denoting the potentially heterogeneous effects of a class size reduction and introduction of a teaching aide, respectively. As before, we analyze this regression by assuming $X_i$ is conditionally independent of the potential achievement outcomes $Y_i(d)$ given the school indicator $W_i$,

$$(Y_i(0), Y_i(1), Y_i(2)) \perp X_i \mid W_i.$$

To analyze the coefficient on $X_{i1}$, we again use the FWL theorem to write

$$\beta_1 = \frac{E[\tilde{\tilde{X}}_{i1} Y_i]}{E[\tilde{\tilde{X}}_{i1}^2]} = \frac{E[\tilde{\tilde{X}}_{i1} Y_i(0)]}{E[\tilde{\tilde{X}}_{i1}^2]} + \frac{E[\tilde{\tilde{X}}_{i1} X_{i1} \tau_{i1}]}{E[\tilde{\tilde{X}}_{i1}^2]} + \frac{E[\tilde{\tilde{X}}_{i1} X_{i2} \tau_{i2}]}{E[\tilde{\tilde{X}}_{i1}^2]}, \tag{7}$$

where $\tilde{\tilde{X}}_{i1}$ again denotes a population residual, but now from regressing $X_{i1}$ on $W_i$, a constant, *and* $X_{i2}$. Unlike before, this residual is not mean-independent of the remaining regressors $(W_i, X_{i2})$ because the dependence between $X_{i1}$ and $X_{i2}$ is non-linear. When $X_{i2} = 1$, $X_{i1}$ must be zero regardless of the value of $W_i$ (because they are mutually exclusive) while if $X_{i2} = 0$ the mean of $X_{i1}$ does depend on $W_i$ unless the treatment assignment is completely random. Thus, in general, $\tilde{\tilde{X}}_{i1} \neq X_{i1} - E[X_{i1} \mid W_i, X_{i2}]$.

Because $\tilde{\tilde{X}}_{i1}$ does not coincide with a conditionally de-meaned $X_{i1}$, we can not generally reduce eq. (7) to an expression involving only the effects of the first treatment arm, $\tau_{i1}$. It turns out that we nevertheless still have $E[\tilde{\tilde{X}}_{i1} Y_i(0)] = 0$, as in eq. (5), since the auxilliary regression residuals are still uncorrelated with any individual characteristic like $Y_i(0)$.[9] In this sense, the regression does not suffer from OVB. However, we do not generally have $E[\tilde{\tilde{X}}_{i1} X_{i2} \tau_{i2}] = 0$. Instead, simplifying eq. (7) by the same steps as before leads to the expression

$$\beta_1 = E[\lambda_{11}(W_i)\tau_1(W_i)] + E[\lambda_{12}(W_i)\tau_2(W_i)] \tag{8}$$

as a generalization of eq. (3). Here $\lambda_{11}(W_i) = E[\tilde{\tilde{X}}_{i1} X_{i1} \mid W_i]/E[\tilde{\tilde{X}}_{i1}^2]$ can be shown to be non-negative and to average to one, similar to the $\phi$ weight in eq. (3). Thus, if not for the second term in eq. (8), $\beta_1$ would similarly identify a convex average of the conditional ATEs $\tau_1(W_i) = E[Y_i(1) - Y_i(0) \mid W_i]$. But precisely because $\tilde{\tilde{X}}_{i1} \neq X_{i1} - E[X_{i1} \mid W_i, X_{i2}]$, this second term is generally present: $\lambda_{12}(W_i) = E[\tilde{\tilde{X}}_{i1} X_{i2} \mid W_i]/E[\tilde{\tilde{X}}_{i1}^2]$ is generally non-zero, complicating the interpretation of $\beta_1$ by including the conditional effects of the other treatment $\tau_2(W_i) = E[Y_i(2) - Y_i(0) \mid W_i]$.

[9] To see this, note that in the auxiliary regression $X_{i1} = \mu_0 + \mu_1 X_{i2} + \mu_2 W_i + \tilde{\tilde{X}}_{i1}$ we can partial out $W_i$ and the constant from both sides to write $\tilde{X}_{i1} = \mu_1 \tilde{X}_{i2} + \tilde{\tilde{X}}_{i1}$. Thus, $\tilde{\tilde{X}}_{i1} = \tilde{X}_{i1} - \mu_1 \tilde{X}_{i2}$ is a linear combination of residuals which, per eq. (5), are both uncorrelated with $Y_i(0)$. It follows that $E[\tilde{\tilde{X}}_{i1} Y_i(0)] = 0$.

The *contamination bias* term in eq. (8) arises because the residualized small class treatment $\tilde{\tilde{X}}_{i1}$ is not conditionally independent of the second full-time aide treatment $X_{i2}$ within schools, despite being uncorrelated with $X_{i2}$ by construction. This can be seen by viewing $\tilde{\tilde{X}}_{i1}$ as the result of an equivalent two-step residualization. First, both $X_{i1}$ and $X_{i2}$ are de-meaned within schools: $\tilde{X}_{i1} = X_{i1} - E[X_{i1} \mid W_i] = X_{i1} - p_1(W_i)$ and $\tilde{X}_{i2} = X_{i2} - E[X_{i2} \mid W_i] = X_{i2} - p_2(W_i)$ where $p_j(W_i) = E[X_{ij} \mid W_i]$ gives the propensity score for treatment $j$. Second, a bivariate regression of $\tilde{X}_{i1}$ on $\tilde{X}_{i2}$ is used to generate the residuals $\tilde{\tilde{X}}_{i1}$. When the propensity scores vary across the schools (i.e. $p_j(0) \neq p_j(1)$), the relationship between these residuals varies by school, and the line of best fit between $\tilde{X}_{i1}$ and $\tilde{X}_{i2}$ averages across this relationship. As a result, the line of best fit does not isolate the conditional (i.e. within-school) variation in $X_{i1}$: the remaining variation in $\tilde{\tilde{X}}_{i1}$ will tend to predict $X_{i2}$ within schools, making the *contamination weight* $\lambda_{12}(W_i)$ non-zero.

### 2.3 Illustration and Intuition

A simple numerical example helps make the contamination bias problem concrete. Suppose, in the previous setting, school 0 (indicated by $W_i = 0$) assigned only 5 percent of the students to the small classroom treatment, with 45 percent of the students assigned to a classroom with a full-time aide and the rest assigned to the control group. In school 1 (indicated by $W_i = 1$), there was a substantially larger push for students to be placed into treatment groups, such that 45 percent of students were assigned to a small classroom, 45 percent were assigned to a classroom with a full-time aide, and only 10 percent were assigned to the control group. Therefore, $p_1(0) = 0.05$, $p_2(0) = 0.45$, while $p_1(1) = p_2(1) = 0.45$. Suppose that the schools have the same number of students, so that $\Pr(W_i = 1) = 0.5$. It then follows from the above formulas that $\lambda_{12}(0) = 99/106$ and $\lambda_{12}(1) = -99/106$.

As reasoned above, the contamination weights are non-zero because the within-school correlation between the residualized treatments, $\tilde{X}_{i1}$ and $\tilde{X}_{i12}$, is heterogeneous: in school 0 it is about $-0.2$, while in school 1 it is $-0.8$.[10] The overall regression of $\tilde{X}_{i1}$ on $\tilde{X}_{i2}$ will average over these two correlations, leading to a misspecified residual $\tilde{\tilde{X}}_{i1}$ that is correlated with $X_{i2}$ within each school. Figure 1 illustrates this averaging by plotting the different potential pairs of the two demeaned treatments ($\tilde{X}_{i1}$, $\tilde{X}_{i2}$), with the two school strata in different colors and shapes. The figure shows how within the first school, the value of the demeaned class aide treatment is only weakly predictive of the small classroom treatment, while it is highly predictive in the second school. The overall regression line in black averages over these different relationships, leading to residuals that are predictive of the value of the class aide treatment.

[10] Here the conditional correlation is $\text{corr}(\tilde{X}_{i1}, \tilde{X}_{i12} \mid W_i) = -\sqrt{p_1(W_i)/(1-p_1(W_i))}\sqrt{p_2(W_i)/(1-p_2(W_i))}$.

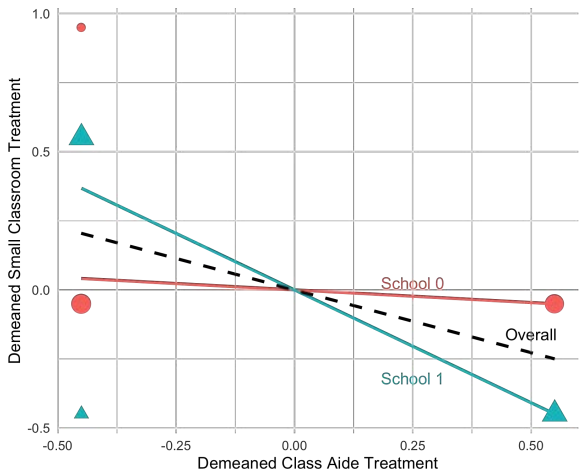


Figure 1: Regression of Small Classroom Treatment on Class Aide Treatment

*Notes:* This figure plots values of the demeaned class aide treatment ($\tilde{X}_{2i}$, the x-axis) against values of the demeaned small classroom treatment ($\tilde{X}_{1i}$, the y-axis) in our numerical example. The size of the points corresponds to the density of observations. The solid red and blue lines mark the within-school regression of the two residualized treatments, while the dashed black line is the overall regression line. The residuals from this line give $\tilde{\tilde{X}}_{i1}$.

To illustrate the potential magnitude of bias in this example, suppose that classroom reductions have no effect on student achievement (so $\tau_1(0) = \tau_1(1) = 0$), but that the effect of a teaching aide varies across schools. In the school 1 the aide is highly effective, $\tau_2(1) = 1$ (which may be the reason for the higher push in this school to place students into treatment groups), but in the school 0, the aide has no effect, $\tau_2(0) = 0$. Equation (8) then shows that the regression coefficient on the first treatment identifies

$$\beta_1 = E[\lambda_{11}(W_i) \cdot 0] + E[\lambda_{12}(W_i)\tau_2(W_i)] = 0 + (-99/106 \times 1 + 99/106 \times 0)/2 \approx -0.47.$$

Thus, in this example, a researcher would conclude that small classrooms have a sizeable negative effect on student achievement (equal in magnitude to around half of the true teaching aide effect in school 1), despite the true small-classroom effect being zero for all students. This treatment effect coefficient can be made arbitrarily large or small (and positive or negative), depending on the heterogeneity of the teaching aide effects across schools.

To build further intuition for eq. (8), it is useful to consider two cases where the contamination bias term is zero. First, suppose the average effects of the teaching aide treatment are constant across the two schools: $\tau_2(0) = \tau_2(1) \equiv \tau_2$. Since regression residuals are by construction uncorrelated with the included regressors, $E[\lambda_{12}(W_i)] = E[\tilde{\tilde{X}}_{i1} X_{i2}]/E[\tilde{\tilde{X}}_{i1}^2] = 0$. Thus, the contamination weights on the second treatment effects average to zero, and the contamination bias disappears: $E[\lambda_{12}(W_i)\tau_2(W_i)] = E[\lambda_{12}(W_i)]\tau_2 = 0$. More generally, the contamination bias will be small when the variation in average teacher's aide treatment effects across schools $\tau_2(W_i)$ is small, or when this treatment effect heterogeneity is only weakly correlated with the contamination weights across schools.

Second, consider the case where $X_{i1}$ and $X_{i2}$ are independent conditional on $W_i$, such as when the small classroom and teacher aid interventions are independently assigned within schools (in contrast to the previously assumed mutual exclusivity of these treatments). In this case the conditional expectation $E[X_{i1} \mid W_i, X_{i2}] = E[X_{i1} \mid W_i]$ will be linear, since $X_{i1}$ and $X_{i2}$ are unrelated given $W_i$, and will thus be identified by the auxiliary regression of $X_{i1}$ on $W_i$, $X_{i2}$, and a constant. Consequently, the $\tilde{\tilde{X}}_{i1}$ residuals will again coincide with $X_{i1} - E[X_{i1} \mid W_i]$. The coefficient on $X_{i1}$ in eq. (6) can therefore be shown to be equivalent to the previous eq. (3), identifying the same convex average of $\tau_1(w)$. This case highlights that dependence across treatments is necessary for the contamination bias to arise.

Before proceeding to a general characterization of contamination bias, we note that the above intuition about the non-linear conditional expectation $E[X_{i1} \mid W_i, X_{i2}]$ also suggests a simple solution to the problem. By including interactions of $W_i$ and $X_{i2}$ in eq. (6), the auxiliary regression of $X_{i1}$ on the other regressors will be saturated and thus will capture the inherent nonlinearity in $E[X_{i1} \mid W_i, X_{i2}]$. We show below how such interacted regressions can obviate contamination bias. In particular, we show how a particular interacted regression specification gives an efficient estimator of (unweighted) ATEs that is immune to the bias of the simpler specification. We then propose a new class of estimators which—as with the Angrist (1998) result for binary treatments—identify a convex average of conditional ATEs. These estimators may yield smaller standard errors, while still being free from bias.

## 3 General Problem

We now derive a general characterization of the contamination bias problem, in regressions of an outcome $Y_i$ on a $K$-dimensional treatment vector $X_i$ and flexible transformations of a control vector $W_i$. We focus on the case of mutually exclusive indicators $X_{ik} = \mathbb{1}\{D_i = k\}$ for values of an underlying treatment $D_i \in \{0, \dots, K\}$ (with the $\mathbb{1}\{D_i = 0\}$ indicator omitted). We extend the characterization to a general treatment vector in Appendix A.1.

We suppose the effects of $X_i$ on $Y_i$ are estimated by a partially linear model:

$$Y_i = X_i'\beta + g(W_i) + U_i, \tag{9}$$

where $\beta$ and $g$ are defined as the minimizers of expected squared residuals $E[U_i^2]$:

$$(\beta, g) = \operatorname*{argmin}_{\tilde{\beta} \in \mathbb{R}^K, \tilde{g} \in \mathcal{G}} E[(Y_i - X_i'\tilde{\beta} - \tilde{g}(W_i))^2] \tag{10}$$

for some linear space of functions $\mathcal{G}$. This setup nests linear covariate adjustment by setting $\mathcal{G} = \{\alpha + w'\gamma \colon [\alpha, \gamma']' \in \mathbb{R}^{1+\dim(W_i)}\}$, in which case eq. (9) gives a linear regression of $Y_i$ on $X_i$, $W_i$, and a constant. The setup also allows for more flexible covariate adjustments—such as by specifying $\mathcal{G}$ to be a large class of "nonparametric" functions (e.g. Robinson, 1988).

Two examples highlight the generality of this setup and are useful for developing our characterization of contamination bias below:

**Example 1** (*Multi-Armed RCT*)**.** $W_i$ is a vector of mutually-exclusive indicators for experimental strata, within which $X_i$ is randomly assigned to individuals $i$. $g$ is linear.

**Example 2** (*Two-Way Fixed Effects*)**.** $i = (j, t)$ indexes panel data, with units $j$ observed over a set of periods $t$, and $W_i$ contains unit and time indicators. $X_i$ contains indicators for leads and lags relative to a unit's deterministic adoption date $A(j)$, $\mathbb{1}\{A(j) = t - r\}$, with the indicator $\mathbb{1}\{A(j) = -1\}$ excluded to normalize the treatment effects. $g$ is linear. The expectation in eq. (10) is over the set of units and time periods in the population from which $i = (j, t)$ is drawn.

Example 1 nests the motivating RCT example in Section 2, allowing for an arbitrary number of experimental strata in $W_i$ and random treatment arms in $X_i$. Example 2 shows that our setup also nests the kind of regressions considered in an existing literature on various two-way fixed effect and difference-in-differences regressions (e.g. Goodman-Bacon, 2021; Sun & Abraham, 2021; de Chaisemartin & D'Haultfœuille, 2020, 2022; Callaway & Sant'Anna, 2021; Borusyak et al., 2022; Wooldridge, 2021).[11] Here $X_i$ is a non-random function of the unit and time indicators in $W_i$. Instead of (or in addition to) indicators for the leads and lags of each unit's adoption date $A(j, t)$ we might imagine $X_i$ including multiple static treatment indicators, as in "mover regressions" that leverage over-time transitions (Hull, 2018b).

As a first step towards characterizing the $\beta$ treatment coefficient vector, we solve the minimization problem in eq. (10). Let $\tilde{X}_i$ denote the residuals from projecting $X_i$ onto the control specification, with elements $\tilde{X}_{ik} = X_{ik} - \operatorname{argmin}_{\tilde{g} \in \mathcal{G}} E[(X_{ik} - \tilde{g}(W_i))^2]$. It follows from the projection theorem (e.g. van der Vaart, 1998, Theorem 11.1) that

$$\beta = E[\tilde{X}_i \tilde{X}_i']^{-1} E[\tilde{X}_i Y_i]. \tag{11}$$

[11] Some papers in this literature study issues we do not consider, such as when researchers fail to include indicators for all relevant treatment states. This specification of $X_i$ will generally add bias terms to our decomposition of $\beta$, below. Similarly, we do not consider multicollinearity issues like in Borusyak et al. (2022), by implicitly assuming a unique solution to eq. (10). In Example 2 this means we assume some units are "never treated," with $A(j) = \infty$. See Roth et al. (2022) for a recent review of these and other issues.

A further application of the FWL theorem allows us to write each treatment coefficient as

$$\beta_k = \frac{E[\tilde{\tilde{X}}_{ik} Y_i]}{E[\tilde{\tilde{X}}_{ik}^2]},$$

where $\tilde{\tilde{X}}_{ik}$ is the residual from regressing $\tilde{X}_{ik}$ on $\tilde{X}_{i,-k} = (\tilde{X}_{i1}, \ldots, \tilde{X}_{i,k-1}, \tilde{X}_{i,k+1}, \ldots, \tilde{X}_{iK})'$.

### 3.1 Causal Interpretation

We now consider the interpretation of each treatment coefficient $\beta_k$ in terms of causal effects. Let $Y_i(k)$ denote the potential outcome of unit $i$ when $D_i = k$. Observed outcomes are given by $Y_i = Y_i(D_i) = Y_i(0) + X_i'\tau_i$ where $\tau_i$ is a vector of treatment effects with elements $\tau_{ik} = Y_i(k) - Y_i(0)$. We denote the conditional expectation of the vector of treatment effects given the controls by $\tau(W_i) = E[\tau_i \mid W_i]$, so that $\tau_k(W_i)$ is the conditional ATE for the $k$th treatment. We let $p(W_i) = E[X_i \mid W_i]$ denote the vector of propensity scores, so that $p_k(W_i) = \Pr(D_i = k \mid W_i)$. Our characterization of contamination bias doesn't require the propensity scores to be bounded away from 0 and 1 and in fact allows them to be degenerate, i.e. $p_k(w) \in \{0, 1\}$ for all $w$. This is the case in Example 2, since $X_i$ is a non-random function of $W_i$. We return to practical questions of propensity score support in Section 4.

We make two assumptions to interpret $\beta_k$ in terms of the effects $\tau_i$. First, we assume mean-independence of the potential outcomes and treatment, conditional on the controls:

**Assumption 1.** *$E[Y_i(k) \mid D_i, W_i] = E[Y_i(k) \mid W_i]$ for all $k$.*

A sufficient condition for this assumption is that the treatment is randomly assigned conditional on the controls, making it conditionally independent of the potential outcomes:

$$(Y_i(0), \ldots, Y_i(K)) \perp D_i \mid W_i. \tag{12}$$

Such conditional random assignment appears in Example 1. In Example 2, where treatment is a non-random function of the two-way fixed effects in $W_i$, Assumption 1 holds trivially.

Second, we assume $\mathcal{G}$ is specified such that that one of two conditions holds:

**Assumption 2.** *Let $\mu_0(w) = E[Y_i(0) \mid W_i = w]$ and recall $p_k(w) = E[X_{ik} \mid W_i = w]$. Either*

$$p_k \in \mathcal{G} \tag{13}$$

*for all $k$, or*

$$\mu_0 \in \mathcal{G}. \tag{14}$$

The first condition requires the covariate adjustment to be flexible enough to capture each treatment's propensity score. For example, with a linear specification for $g$, eq. (13) requires the propensity scores to be linear in $W_i$ (cf. eq. (30) in Angrist & Krueger, 1999). This condition holds trivially in Example 1, since $W_i$ is a vector of indicators for groups within which $X_i$ is randomly assigned. When this condition holds, the projection of the treatment onto the covariates coincides with the vector of propensity scores, and the projection residuals are given by $\tilde{X}_i = X_i - p(W_i)$.

In Example 2, with $X_i$ being a deterministic function of unit and time indices and a linear specification for $g$, eq. (13) fails because the propensity scores are binary—they cannot be captured by a linear function of the unit and time indicators in $W_i$. However, eq. (14) can still be satisfied by a parallel trends assumption: that the average untreated potential outcomes $Y_i(0)$ are linear in the unit and time effects.[12]

Under either condition in Assumption 2, the specification of controls is flexible enough to avoid OVB. To see this formally, suppose all treatment effects are constant: $\tau_{ik} = \tau_k$ for all $k$. This restriction lets us write $Y_i = Y_i(0) + X_i'\tau$, where $\tau$ is a vector collecting the constant effects. The only source of bias when regressing $Y_i$ on $X_i$ and controls is then the unobserved variation in the untreated potential outcomes $Y_i(0)$. But it follows from the definition of $\beta$ in eq. (11) that there is no such OVB when Assumption 2 holds; the coefficient vector identifies the constant effects:

$$\begin{aligned}\beta = E[\tilde{X}_i\tilde{X}_i']^{-1}E[\tilde{X}_iY_i] &= E[\tilde{X}_i\tilde{X}_i']^{-1}(E[\tilde{X}_iY_i(0)] + E[\tilde{X}_i\tilde{X}_i']\tau) \\ &= E[\tilde{X}_i\tilde{X}_i']^{-1}\underbrace{E[\tilde{X}_iE[Y_i(0) \mid W_i]]}_{=0} + \tau = \tau.\end{aligned}$$

Here the first line uses the fact that $E[\tilde{X}_iX_i'] = E[\tilde{X}_i\tilde{X}_i']$ because $\tilde{X}_i$ is a vector of projection residuals, and the second line uses the law of iterated expectations and Assumption 1. Under eq. (13), $E[\tilde{X}_i \mid W_i] = 0$, so that the term in braces is zero by another application of the law of iterated expectations: $E[\tilde{X}_iE[Y_i(0) \mid W_i]] = E[E[\tilde{X}_i \mid W_i]E[Y_i(0) \mid W_i]] = 0$. It is likewise zero under eq. (14) since $\tilde{X}_i$ is by definition of projection orthogonal to any function in $\mathcal{G}$ such that $E[\tilde{X}_iE[Y_i(0) \mid W_i]] = E[\tilde{X}_i\mu_0(W_i)] = 0$. Hence, OVB is avoided in the constant-effects case so long as either the propensity scores or the untreated potential outcomes are spanned by the control specification. Versions of this robustness property have been previously observed in, for instance, Robins et al. (1992).

When treatment effects are heterogeneous but $X_i$ contains a *single* treatment indicator, $\beta$

[12] Identification based on eq. (13) can be seen as "design-based" in that it leverages only the conditional random assignment of $D_i$ and specifies the treatment assignment process. Identification based on eq. (14) can be seen as "model-based" in that it makes no assumptions on the treatment assignment process but specifies a model for the unobserved untreated potential outcomes.

identifies a weighted average of the conditional effects $\tau(W_i)$. Specifically, since we still have $E[\tilde{X}_i Y_i(0)] = 0$ under Assumptions 1 and 2, it follows from eq. (11) that

$$\beta = \frac{E[\tilde{X}_i X_i \tau_i]}{E[\tilde{X}_i^2]} = E[\lambda_{11}(W_i)\tau(W_i)], \quad \text{with} \quad \lambda_{11}(W_i) = \frac{E[\tilde{X}_i X_i \mid W_i]}{E[\tilde{X}_i X_i]}, \tag{15}$$

where the second equality uses iterated expectations and the fact that $E[\tilde{X}_i^2] = E[\tilde{X}_i X_i]$. Under eq. (13), $E[\tilde{X}_i X_i \mid W_i] = E[\tilde{X}_i^2 \mid W_i] = \text{var}(X_i \mid W_i)$, so the weights further simplify to $\lambda_{11}(W_i) = \frac{\text{var}(X_i|W_i)}{E[\text{var}(X_i|W_i)]} \geq 0$. This extends the Angrist (1998) result to a general control specification; versions of this extension appear in, for instance, Angrist and Krueger (1999), Angrist and Pischke (2009, Chapter 3.3), and Aronow and Samii (2016). The result provides a rationale for estimating the effect of a scalar as good as randomly assigned treatment using a partially linear model: so long as the specification of $\mathcal{G}$ is rich enough so that eq. (13) holds, this model will identify a convex average of heterogeneous treatment effects. Moreover, as we will show in Section 4, the weights $\lambda_{11}(W_i)$ are efficient in that they minimize the semiparametric efficiency bound (conditional on the controls) for estimating a weighted-average treatment effect. This result makes the partially linear specification (9) especially appealing with a single binary treatment. On the other hand, when eq. (14) holds but eq. (13) does not, the weights $\lambda_{11}(W_i)$ need not be positive. We return to this point in Section 3.2.

The next proposition shows that with multiple treatments, the interpretation of $\beta$ becomes more complicated because of contamination bias:

**Proposition 1.** *Under Assumptions 1 and 2, the treatment coefficients in the partially linear model (9) identify*

$$\beta_k = E[\lambda_{kk}(W_i)\tau_k(W_i)] + \sum_{\ell \neq k} E[\lambda_{k\ell}(W_i)\tau_\ell(W_i)], \tag{16}$$

*where*

$$\lambda_{kk}(W_i) = \frac{E[\tilde{\tilde{X}}_{ik} X_{ik} \mid W_i]}{E[\tilde{\tilde{X}}_{ik}^2]} \qquad \text{and} \qquad \lambda_{k\ell}(W_i) = \frac{E[\tilde{\tilde{X}}_{ik} X_{i\ell} \mid W_i]}{E[\tilde{\tilde{X}}_{ik}^2]}.$$

*These weights satisfy $E[\lambda_{kk}(W_i)] = 1$ and $E[\lambda_{k\ell}(W_i)] = 0$. Furthermore, if eq. (13) holds, $\lambda_{kk}(W_i) \geq 0$ for each $k$.*

Proposition 1 shows that the coefficient on $X_{ik}$ in eq. (9) is a sum of two terms. The first term is a weighted average of conditional ATEs $\tau_k(W_i)$, with weights $\lambda_{kk}(W_i)$ that average to one and are guaranteed to be convex when eq. (13) holds. This term generalizes the characterization of the single-treatment case, eq. (15). The second term is a weighted average of treatment effects for *other* treatments $\tau_\ell(W_i)$, with weights $\lambda_{k\ell}(W_i)$ that average to zero.

Because these contamination weights are zero on average, they must be negative for some values of the controls unless they are all identically zero.

Each treatment coefficient $\beta_k$ thus generally suffers from contamination bias. Two exceptions are when $\lambda_{k\ell}(W_i) = 0$ almost-surely for all $\ell \neq k$, and when the conditional effects of these other treatments are homogeneous such that $\tau_\ell(W_i) = \tau_\ell$. In the second case $E[\lambda_{k\ell}(W_i)\tau_\ell(W_i)] = \tau_\ell E[\lambda_{k\ell}(W_i)] = 0$, so there is no contamination bias term. By the law of iterated expectations the first case holds if $E[\tilde{\tilde{X}}_{ik} \mid X_{i,-k}, W_i] = 0$, or, equivalently, if the conditional expectation of $X_{ik}$ given $X_{i,-k}$ and $W_i$ is partially linear (i.e. $E[X_{ik} \mid X_{i,-k}, W_i] = X_{i,-k}'\alpha + g_k(W_i)$ for some vector $\alpha$ and $g_k \in \mathcal{G}$). In other words, it holds when the assignment of treatment $k$ depends linearly on the other treatment indicators and a flexible function of the controls. This condition is the analog of condition (13) if we interpret $X_{ik}$ as a binary treatment of interest, and $X_{i,-k}'\alpha + g_k(W_i)$ as a specification for the controls. However, with mutually exclusive treatments, it cannot hold unless treatment assignment is unconditionally random. In particular, since $X_{ik}$ must equal zero if the unit is assigned to one of the other treatments regardless of the value of $W_i$, we have $\alpha_\ell = -g_k(W_i)$ for all elements $\alpha_\ell$ of $\alpha$. This in turn implies the assignment of treatment $k$ doesn't depend on $W_i$, which can't be the case unless the propensity score $p_k(W_i)$ is constant.

A third weaker case of no contamination bias is when the weights $\lambda_{k\ell}(W_i)$ and conditional ATEs $\tau_\ell(W_i)$ vary, but are uncorrelated with each other. More generally, contamination bias may be minimal when the contamination weights $\lambda_{k\ell}(W_i)$ and the conditional ATEs $\tau_\ell(W_i)$ are weakly correlated: that is, when the factors influencing treatment effect heterogeneity are largely unrelated to the factors influencing the treatment assignment process. We return to this possibility in our empirical application (Section 5.2).

We make three further remarks on our general characterization of contamination bias:

*Remark* 1. Since the weights in eq. (16) are functions of the variances $E[\tilde{\tilde{X}}_{ik}^2]$ and covariances $E[\tilde{\tilde{X}}_{ik}X_{i\ell}]$ and $E[\tilde{\tilde{X}}_{ik}X_{ik}]$, they are identified and can be used to further characterize each $\beta_k$ coefficient. For example, the contamination bias term can be bounded by the identified contamination weights $\lambda_{k\ell}(W_i)$ and bounds on the heterogeneity in conditional ATEs $\tau_\ell(W_i)$. We illustrate such an approach in our empirical application.

*Remark* 2. The results in Proposition 1 are stated for the case when $X_i$ are mutually exclusive treatment indicators. In Appendix A.1 we relax this assumption to allow for combinations of non-mutually exclusive treatments (either discrete or continuous). In this case, the own-treatment weights $\lambda_{kk}(W_i)$ may be negative even if eq. (13) holds.

*Remark* 3. While we derived Proposition 1 in the context of a causal model, analogous results follow for descriptive regressions that do not assume the existence of potential outcomes or impose Assumption 1. Consider, specifically, the goal of estimating a convex average of conditional group contrasts $E[Y_i \mid D_i = k, W_i = w] - E[Y_i \mid D_i = 0, W_i = w]$ with a partially linear model eq. (9) and replace condition (14) with an assumption that $E[Y_i \mid D_i = 0, W_i = w] \in \mathcal{G}$. The same steps that lead to Proposition 1 then show that such regressions also generally suffer from contamination bias: the coefficient on a given group indicator averages the conditional contrasts across all other groups, with non-convex weights. Furthermore, the weights on own-group conditional contrasts are not necessarily positive. These sorts of conditional contrast comparisons are therefore not generally robust to misspecification of the conditional mean, $E[Y_i \mid D_i, W_i]$.

### 3.2 Implications

Proposition 1 shows that treatment effect heterogeneity can induce two conceptually distinct issues in flexible regression estimates of treatment effects. First, with either single or multiple treatments, there is a potential for negative weighting of a treatment's *own* effects when condition (14) holds but condition (13) fails. This negative weighting issue is relevant in various DiD regressions and related estimators which rely on such models for untreated potential outcomes (via parallel trends assumptions) while conditioning on treatment assignment. Although the recent DiD literature focuses on two-way fixed effect regressions, Proposition 1 shows such negative weighing can arise more generally—such as when researchers allow for linear trends, interacted fixed effects, or other extensions of the basic parallel trends model. None of these alternative specifications for $g$ are in general flexible enough to capture the degenerate propensity scores and hence ensure that eq. (13) holds.[13]

Second, in the multiple treatment case, there is a potential for contamination bias from *other* treatment effects regardless of which condition in Assumption 2 holds. This form of bias is thus relevant whenever one uses an additive covariate adjustment, regardless of how flexibly the covariates are specified. Versions of this problem have been noted in, for example, the Sun and Abraham (2021) analysis of DiD regressions with treatment leads and lags or the Hull (2018b) analysis of mover regressions.[14]

The characterization in Proposition 1 also relates to concerns in interpreting multiple-

[13] More broadly, negative weighting issues arise whenever the covariate specification is not flexible enough for eq. (13) to hold. For example, consider a scalar covariate $W_i$ with a uniform distribution on $[0, 1]$, and a binary treatment with non-linear propensity score $p(W_i) = \min(2W_i, 0.9)$. Suppose that $Y_i(0) = 0$, so that (14) holds. Then $\lambda_{11}(W_i)$ is negative for $W_i \geq 2911/3402 \approx 0.855$. If, say $Y_i(1) = W_i$, so that $\tau(W_i) = W_i \geq 0$, the regression coefficient on the treatment is negative despite uniformly non-negative treatment effects.

[14] The negative weights issue raised in de Chaisemartin and D'Haultfœuille (2020) (when $K = 1$), and the related issue that own-treatment weights may be negative in Sun and Abraham (2021) and de Chaisemartin and D'Haultfœuille (2022) (when $K > 1$), arise because the treatment probability is not linear in the unit and time effects. If eq. (13) holds with $K = 1$, Proposition 1 shows $\beta$ estimates a convex combination of treatment effects. This covers the setting considered in Theorem 1(iv) in Athey and Imbens (2022). In their Comment 2, Athey and Imbens (2022) say that "the sum of the weights [used in Theorem 1(iv)] is one, although some of the weights may be negative." Proposition 1 shows these weights are, in fact, non-negative.

treatment IV estimates with heterogeneous treatment effects (Behaghel et al., 2013; Kirkeboen et al., 2016; Kline & Walters, 2016; Hull, 2018c; Lee & Salanié, 2018; Bhuller & Sigstad, 2022). This connection comes from viewing eq. (9) as the second stage of an IV model estimated by a control function approach; in the linear IV case, for example, $g(W_i)$ can be interpreted as giving the residuals from a first-stage regression of $X_i$ on a vector of valid instruments $Z_i$. In the single-treatment case, the resulting $\beta$ coefficient has an interpretation of a weighted average of conditional local average treatment effects under the appropriate first-stage monotonicity condition (Imbens & Angrist, 1994). But as in Proposition 1 this interpretation fails to generalize when $X_i$ includes multiple mutually-exclusive treatment indicators: each $\beta_k$ combines the local effects of treatment $k$ with a non-convex average of the effects of other treatments.

Finally, Proposition 1 has implications for single-treatment IV estimation with multiple instruments and flexible controls. The first stage of such IV regressions will tend to have the form of eq. (9), where now $Y_i$ is interpreted as the treatment and $X_i$ gives the vector of instruments. Proposition 1 shows that the first-stage coefficients on the instruments $\beta_k$ will not generally be convex weighted average of the true first-stage effects $\tau_{ik}$. Because of this non-convexity, the regression specification may fail to satisfy the effective monotonicity condition even when the true effects are always positive. In other words, the cross-instrument contamination of causal effects may cause monotonicity violations, even when specifications with individual instruments would be appropriate. This issue is distinct from previous concerns over monotonicity failures in multiple-instrument designs (Mueller-Smith, 2015; Frandsen et al., 2019; Norris, 2019; Mogstad et al., 2021), which are generally also present in such just-identified specifications. It is also distinct from some concerns about insufficient flexibility in the control specification when monotonicity holds unconditionally (Blandhol et al., 2022).

This new monotonicity concern may be especially important in "judge" IV designs, which exploit the conditional random assignment to multiple decision-makers. Many studies leverage such variation by computing average examiner decision rates, often with a leave-one-out correction, and use this "leniency" measure as a single instrument with linear controls. These IV estimators can be thought of as implementing versions of a jackknife IV estimator (Angrist et al., 1999), based on a first stage that uses examiner indicators as instruments, similar to eq. (9). Proposition 1 thus raises a new concern with these IV analyses when the controls (such as courtroom or time fixed effects) are needed to ensure ignorable treatment assignment.[15]

[15] As we discuss in Section 4, one solution to this problem is to interact the examiner instruments with the controls, which would amount to computing "leniency" separately within location and time cells. This may greatly increase the effective number of instruments, heightening concerns of many-instrument bias in finite samples as well as the importance of appropriate leave-one-out corrections (e.g., Kolesár, 2013).

# 4 Solutions

We now discuss solutions to the contamination bias problem raised by Proposition 1. We focus on the case of conditionally ignorable treatment assignment (in the sense that eq. (12) holds, and the propensity scores are not degenerate) since solutions that allow for degenerate propensity scores are generally different and have been previously explored in the literature in the context of DiD regressions. We refer readers to de Chaisemartin and D'Haultfœuille (2022), Sun and Abraham (2021), Callaway and Sant'Anna (2021), Borusyak et al. (2022), and Wooldridge (2021) for such solutions.

We propose three solutions, each identifying a distinct causal parameter. First, in Section 4.1, we discuss estimation of ATEs. The other two solutions, discussed in Sections 4.2 and 4.3, estimate weighted averages of individual treatment effects using weights that are easiest to estimate in that they minimize the semiparametric efficiency bound for estimating weighted ATEs under homoskedasticity. If the weights are allowed to vary across treatments, it is optimal to estimate the effect of each $k$ using the partially linear model in eq. (9), but in a sample restricted to individuals in the control group and to those receiving treatment $k$. If the weights are constrained to be common across treatments, this leads to a new weighted regression estimator.

## 4.1 Estimating Average Treatment Effects

Many estimators exist for the ATE of binary treatments (see Imbens and Wooldridge (2009) for a review). A number of these approaches extend naturally to multiple treatments, including matching, inverse propensity score weighting, regressions with interactions, or doubly-robust combinations of these methods (see, among others, Cattaneo (2010), Chernozhukov et al. (2021), and Graham and Pinto (2022)).

Rather than reviewing all of these approaches, we briefly outline a simple implementation of one method which follows the intuition given at the end of Section 2. Namely, one may estimate the ATE vector $\tau$ by expanding the partially linear model in eq. (9) to include treatment interaction terms. This generalizes the implementation in the binary treatment case discussed in Imbens and Wooldridge (2009, Section 5.3). Consider the expanded model

$$Y_i = X_i'\beta + q_0(W_i) + \sum_{k=1}^{K} X_{ik}\left(q_k(W_i) - E[q_k(W_i)]\right) + \dot{U}_i, \tag{17}$$

where $q_k \in \mathcal{G}$, $k = 0, \ldots, K$ and we continue to define $\beta$ and the functions $q_k$ as minimizers of $E[\dot{U}_i^2]$. When $\mathcal{G}$ consists of linear functions, eq. (17) specifies a linear regression of $Y_i$ on $X_i$, $W_i$, a constant, and the interactions between each treatment indicator $X_{ik}$ and the demeaned control vector $W_i - E[W_i]$. Define $\mu_k(w) = E[Y_i(k) \mid W_i = w]$ for $k = 0, \ldots, K$, so that $\tau_k(w) = \mu_k(w) - \mu_0(w)$. When Assumption 1 holds, and $\mathcal{G}$ is furthermore rich enough to ensure $\mu_k \in \mathcal{G}$ for $k = 0, \ldots, K$, then $\beta = \tau$. Moreover $q_k(w) = \tau_k(w)$ for $k = 1, \ldots, K$, such that the regression identifies both the unconditional and conditional ATEs.

Following the intuition at the end of Section 2, the added interactions in eq. (17) ensure that each treatment coefficients $\beta_k$ is determined only by the outcomes in treatment arms with $D_i = 0$ and $D_i = k$, avoiding the other-treatment contamination bias in Proposition 1. Demeaning the $q_k(W_i)$ in the interactions ensures they are appropriately centered to interpret the coefficients on the uninteracted $X_{ik}$ as ATEs.

Estimation of eq. (17) by least squares is conceptually straightforward, with sample averages replacing expectations. Furthermore, it can be shown that the resulting estimator achieves the semiparametric efficiency bound under strong overlap (i.e. when the propensity score is bounded away from zero and one) when implemented as a series estimator: it is impossible to construct another regular estimator of the ATE with smaller asymptotic variance.

Nonetheless, under weak overlap, the estimator may be imprecise, with poor finite-sample behavior. This is not a shortcoming of the specific estimator: Khan and Tamer (2010) shows that identification of the ATE is irregular under weak overlap, and it is not possible to estimate it at a $\sqrt{N}$-rate. These results formalize the intuition that it is difficult to reliably estimate the counterfactual outcomes for observations with extreme propensity scores. Overlap concerns tend to be more severe with multiple treatments, because some propensity scores necessarily become closer to zero or one as more treatment arms are added. We next turn to the problem of estimating weighted averages of conditional ATEs that downweight these difficult-to-estimate counterfactuals.

### 4.2 Efficient Weighted Averages of Treatment Effects

Suppose in a sample of observations $i = 1, \ldots, N$ we wish to estimate a weighted average of conditional potential outcome contrasts $\sum_{i=1}^{N} \lambda(W_i) \sum_{k=0}^{K} c_k \mu_k(W_i) / \sum_{i=1}^{N} \lambda(W_i)$, where $\mu_k(W_i) = E[Y_i(k) \mid W_i]$, $c$ is a $(K+1)$-dimensional contrast vector with elements $c_k$, and $\lambda(W_i)$ is some weighting scheme.[16] We focus on two specifications for the contrast vector, leading to two alternatives to estimating the ATE based on eq. (17). First, for separately estimating the effect of each treatment $k$, we set $c_k = 1$, $c_0 = -1$, and set the remaining entries of $c$ to 0. The contrast of interest then becomes $\sum_{i=1}^{N} \lambda(W_i) \sum_{k=0}^{K} \tau_k(W_i) / \sum_{i=1}^{N} \lambda(W_i)$, the weighted ATE of treatment $k$ across different strata. Second, we specify $c$ so as to allow us to simultaneously contrast the effects of all $K$ treatments—we discuss this further below.

[16] In a slight abuse of notation relative to Section 3, the weights $\lambda$ here are not required to average to one. Instead, we scale the estimand by the sum of the weights, $\sum_{i=1}^{N} \lambda(W_i)$.

Given the contrast vector $c$, we consider the problem of finding the weighting scheme $\lambda(W_i)$ that is the "easiest" to estimate in that it leads to the smallest possible standard errors. This objective has two motivations. First is a robustness concern: a researcher would like to estimate a given contrast as efficiently as possible, at least under the benchmark of constant treatment effects, while being robust to the possibility that the effects are heterogeneous. Under constant effects, the weighting $\lambda(W_i)$ is immaterial. But the robustness property ensures that the estimand retains a causal interpretation as a convex average of conditional contrasts under weak conditions, avoiding the contamination bias displayed by the regression estimator per Proposition 1. Such a motivation presumably underlies the popularity of regression for estimating the effect of a binary treatment: the regression estimator is efficient under homoskedasticity and constant treatment effects, while, by the Angrist (1998) result, retaining a causal interpretation under heterogeneous effects.

The second motivation is that the easiest-to-estimate weighting scheme gives a bound on the information available in the data: if these weights nonetheless yield overly large standard errors, inference on other treatment effects (such as the unweighted ATE) will be at least as uninformative. Computing standard errors for this efficient weighted average of treatment effects can be useful as it reveals whether informative conclusions—regardless of how one specifies the treatment effect of interest—are only possible under additional assumptions or with the aid of additional data. If the easiest-to-estimate weighting scheme yields small standard errors even though the standard errors for the unweighted ATE are large, it can be concluded that the data is informative about *some* treatment effects—even if it is not informative about the unweighted average.

In fact, our solution below shows that in the binary treatment case the easiest-to-estimate weighting scheme is exactly the same as the weights used by regression. This special case illustrates the second motivation: the optimal binary treatment weights are proportional to the conditional variance of treatment, $\text{var}(D_i \mid W_i) = p_1(W_i)(1 - p_1(W_i))$, which tend to zero as $p_1(W_i)$ tends to zero or one. Regression thus downweights observations with extreme propensity scores where the estimation of counterfactual outcomes is difficult, avoiding the poor finite-sample behavior of ATE estimators under weak overlap and allowing regression to be informative even in cases when it is not possible to precisely estimate the unweighted ATE. More generally, since regression solves the efficient binary treatment weighting scheme, regression estimates establish the extent to which internally valid and informative inference for *any* causal effect are possible with the data at hand.

We derive the easiest-to-estimate weighting scheme for multiple treatments in two steps. First, we establish an efficiency benchmark—a semiparametric efficiency bound—for estimation of a given weighted average of treatment effects under the idealized scenario that the propensity score is known. Second, we determine which weighted average is the "easiest" to estimate, in that it minimizes the semiparametric efficiency bound over the choice of $\lambda(W_i)$. When the contrast vector is specified to allow simultaneous comparison of all treatments, estimation of this efficient weighted average leads to a new estimator; we discuss its implementation when the propensity score is not known in Section 4.3.

The following proposition establishes the first step of our derivation:

**Proposition 2.** *Suppose eq. (12) holds in an i.i.d. sample of size $N$, with known non-degenerate propensity scores $p_k(W_i)$. Let $\sigma_k^2(W_i) = \operatorname{var}(Y_i(k) \mid W_i)$. Consider the problem of estimating the weighted average of contrasts*

$$\theta_{\lambda,c} = \frac{1}{\sum_{i=1}^N \lambda(W_i)} \sum_{i=1}^N \lambda(W_i) \sum_{k=0}^K c_k \mu_k(W_i),$$

*where the weighting function $\lambda$ and contrast vector $c$ are both known. Suppose the weighting function satisfies $E[\lambda(W_i)] \neq 0$, and that the second moments of $\lambda(W_i)$ and $\mu(W_i)$ are bounded. Then, conditional on the controls $W_1, \dots, W_N$, the semiparametric efficiency bound is almost surely given by*

$$\mathcal{V}_{\lambda,c} = \frac{1}{E[\lambda(W_i)]^2} E\left[\sum_{k=0}^K \frac{\lambda(W_i)^2 c_k^2 \sigma_k^2(W_i)}{p_k(W_i)}\right]. \tag{18}$$

As formalized in the proof (see Appendix A.2), the efficiency bound $\mathcal{V}_{\lambda,c}$ establishes a lower bound on the asymptotic variance of any regular estimator of $\theta_{\lambda,c}$ under the idealized situation of known propensity scores.[17]

To establish the second step, we choose $\lambda$ to minimize eq. (18) subject to the constraint that these weights are non-negative and average to one: $E[\lambda(W_i)] = 1$. Simple algebra shows that this variance-minimizing weighting scheme is given by

$$\lambda_c^*(W_i) = \left(\sum_{k=0}^K \frac{c_k^2 \sigma_k^2(W_i)}{p_k(W_i)}\right)^{-1}. \tag{19}$$

The asymptotic variance of this easiest-to-estimate weighting,

$$\mathcal{V}_{\lambda_c^*,c} = E\left[\left(\sum_{k=0}^K \frac{c_k^2 \sigma_k^2(W_i)}{p_k(W_i)}\right)^{-1}\right]^{-1},$$

[17] The efficiency bound for the population analog $\theta^*_{\lambda,c} = E[\lambda(W_i)\sum_{k=0}^K c_k \mu_k(W_i)]/E[\lambda(W_i)]$ has an additional term, $E[\lambda(W_i)^2(\sum_{k=0}^K c_k\mu_k(W_i) - \theta^*_{\lambda,c})^2]/E[\lambda(W_i)]^2$, reflecting the variability of the conditional average contrast. The optimal weights for $\theta^*_{\lambda,c}$ thus depend on the nature of treatment effect heterogeneity. By focusing on $\theta_{\lambda,c}$, we avoid this term, which allows us to characterize the optimal weights in eq. (19) while remaining completely agnostic about heterogeneity in treatment effects. See Crump et al. (2006) for additional discussion in the context of a binary treatment.

is the harmonic mean of $\sum_{k=0}^{K} \frac{c_k^2 \sigma_k^2(W_i)}{p_k(W_i)}$. In contrast, the efficiency bound for the unweighted contrast is given by the arithmetic mean $E\left[\left(\sum_{k=0}^{K} \frac{c_k^2 \sigma_k^2(W_i)}{p_k(W_i)}\right)\right]$, which can be considerably bigger when the propensity scores are not bounded away from zero or one.

When the contrast vector $c$ is selected to estimate the weighted average effect of a particular treatment $k$, Proposition 2 implies that the regression weights are efficient:

**Corollary 1.** *For some $k \geq 1$, let $c^k$ be a vector with elements $c_j^k = 1$ if $j = k$, $c_j^k = -1$ if $j = 0$, and $c_j^k = 0$ otherwise. Suppose that the conditional variance of relevant potential outcomes is homoskedastic: $\sigma_k^2(W_i) = \sigma_0^2(W_i) = \sigma^2$. Then the variance-minimizing weighting scheme is given by $\lambda_{c^k}^* = \lambda^k$, where*

$$\lambda^k(W_i) = \frac{p_0(W_i)p_k(W_i)}{p_0(W_i) + p_k(W_i)}, \tag{20}$$

*with the semiparametric efficiency bound given by*

$$\mathcal{V}_{\lambda^k, c^k} = \sigma^2 E\left[\frac{p_0(W_i)p_k(W_i)}{p_0(W_i) + p_k(W_i)}\right]^{-1}, \tag{21}$$

*where $p_0(W_i) = \Pr(D_i = 0 \mid W_i) = 1 - \sum_{k=1}^{K} p_k(W_i)$.*

Per eq. (15), the optimal weighting $\lambda^k$ coincides with the implicit weighting of conditional ATEs from the partially linear model (9) when it is fit only on observations with $D_i \in \{0, k\}$. This follows since the propensity score in the subsample is given by $\Pr(D_i = k \mid W_i, D_i \in \{0, k\}) = \frac{p_k(W_i)}{p_0(W_i)+p_k(W_i)}$, making $\lambda^k(W_i)$ in eq. (20) the conditional variance of the treatment indicator. Moreover, it follows by standard arguments that regressing $Y_i$ onto $X_{ik}$ and $g(W_i)$ in the subsample with $D_i \in \{0, k\}$ efficiently estimates this weighted average effect provided $g$ is sufficiently flexible (such as when $g$ is linear and $W_i$ consists of group indicators).[18] When the treatment $D_i$ is binary, this simply amounts to running a regression on the binary treatment indicator, with an additive covariate adjustment.

Corollary 1 thus gives justification for estimating the effect of any given treatment $k$ by a partially linear regression with an additive covariate adjustment in the subsample with $D_i \in \{0, k\}$. To estimate the effects of all treatments, one runs $K$ such regressions, restricting the sample to one treatment arm and the control group. Such one-treatment-at-a-time regressions are simple to implement and do not require explicitly estimating the propensity score. The regression coefficients are causally interpretable as weighted averages of conditional treatment

[18] As we discuss in the next subsection, when the propensity score is unknown, the semiparametric efficiency bound for estimating $\theta_{\lambda^k, c^k}$ has an additional term relative to eq. (21) arising from the estimation of the optimal weights eq. (20). Thus, while the regression estimator is semiparametrically efficient, it does not generally attain the efficiency bound derived in Proposition 2 that assumes a known propensity score.

effects $\tau_k(W_i)$, so long as $p_k/(p_0+p_k) \in \mathcal{G}$. Moreover, the weighted averages are locally efficient in the sense of Corollary 1.[19]

While the robustness property of the one-treatment-at-a-time regression is well-established, by Angrist (1998) and subsequent extensions, our efficiency characterization appears novel. It builds on earlier results in Crump et al. (2006, Corollary 5.2) (a working paper version of Crump et al., 2009), and Li et al. (2018, Corollary 1), who show that the weighting $p_1(W_i)(1-p_1(W_i))$ is optimal for estimating the effect of a binary treatment in that the weighting minimizes the asymptotic variance of a particular class of inverse propensity score weighted estimators. Corollary 1 above extends the optimality of this weighting to all regular estimators, and to multiple treatments. Importantly, this result formalizes a common motivation for using regression to estimate the effects of a single treatment instead of more involved unconditional ATE estimators: when treatment effect heterogeneity is minimal or only weakly correlated with the $\lambda_k^*(W_i)$ weights, the regression's weighted-average effect will be close to the ATE while being more precisely estimated.

A shortcoming of the optimal weighting scheme in Corollary 1 is that it is treatment-specific, so comparisons of the "one-at-a-time" weighted-average effects across treatments are generally not causally interpretable.[20] This issue is especially salient when the control group is arbitrarily chosen, such as in teacher VAM regressions which omit an arbitrary teacher from estimation and seek to make causal comparisons across all teachers.[21]

We thus turn to the question of how Proposition 2 can be used to select an efficient weighting scheme that allows for simultaneous comparisons across all treatment arms. We are interested in reporting estimates of a vector $\beta_{\lambda^{\mathrm{C}}}$ of $K$ coefficients with elements $\beta_{\lambda^{\mathrm{C}},k} = \sum_{i=1}^N \lambda^{\mathrm{C}}(W_i)\tau_k(W_i)/\sum_{i=1}^N \lambda^{\mathrm{C}}(W_i)$, where the weights $\lambda^{\mathrm{C}}$ are common across treatment arms. If we are equally interested in all $K(K+1)$ contrasts (that is, weighted averages $\mu_j(W_i) - \mu_k(W_i)$, for all $j \neq k$, $j,k = 0,\dots,K$), a natural approach is to choose the weighting scheme $\lambda^C$ that minimizes the average variance across all contrasts:

$$\int \mathcal{V}_{\lambda,c}\, dF(c) = \frac{1}{E[\lambda(W_i)]^2} \sum_{k=0}^{K} \frac{2}{K+1} E\left[\frac{\lambda(W_i)^2 \sigma_k^2(W_i)}{p_k(W_i)}\right],$$

where $F$ gives the uniform distribution over the possible (now random) contrasts $c$, so that

[19] As usual, homoskedasticity is a tractable baseline: the arguments in favor of ordinary least squares regression following Corollary 1 can be extended to favor a (feasible) generalized least squares regression when $\sigma^2(W_i)$ is known or consistently estimable.

[20] Formally, for treatments 1 and 2, we estimate the weighted averages $\sum_i \lambda^1(W_i)\tau_1(W_i)/\sum_i \lambda^1(W_i)$ and $\sum_i \lambda^2(W_i)\tau_2(W_i)/\sum_i \lambda^2(W_i)$. Because the weights $\lambda^1$ and $\lambda^2$ differ, the difference between these estimands cannot generally be written as a convex combination of conditional treatment effects $\tau_1(W_i) - \tau_2(W_i)$.

[21] Note that this critique also applies to the own-treatment weights in Proposition 1. Thus even without contamination bias one may find the implicit multiple-treatment regression weighting unsatisfying.

$c_j = 1$ with probability $1/(K+1)$ and $-1$ with probability $1/(K+1)$. Minimizing this expression over $\lambda$ is equivalent to minimizing eq. (18) with $c_k^2 = 2/(K+1)$, which leads to the following result:

**Corollary 2.** *Let $F$ denote the uniform distribution over the possible contrast vectors. Suppose that $\sigma_k^2(W_i) = \sigma^2$ for all $k$. Then the weighting scheme minimizing the average variance bound $\int \mathcal{V}_{\lambda,c} dF(c)$ is given by*

$$\lambda^C(W_i) = 1 \Big/ \sum_{k=0}^{K} p_k(W_i)^{-1}. \tag{22}$$

The weights $\lambda^{\mathrm{C}}$ generalize the intuition behind the single binary treatment (Corollary 1), placing higher weight on covariate strata where the treatments are evenly distributed, and putting less weight on strata with limited overlap. When the treatment is binary, $K = 1$, the weights reduce to the one-at-a-time weights in Corollary 1, $\lambda^C(W_i) = \lambda^1(W_i) = \lambda^0(W_i) = p_1(W_i)p_0(W_i)$. With multiple treatments, however, the weights $\lambda^C$ remain the same for every treatment, allowing for simultaneous comparisons across all treatment pairs $(k, \ell)$. We next consider estimating $\beta_{\lambda^C}$ using a weighted regression approach.

### 4.3 Estimating Efficiently Weighted Average Effects

If the propensity scores $p(W_i)$ were known, one could estimate $\beta_{\lambda^{\mathrm{C}}}$ by a weighted regression of $Y_i$ onto $X_i$ and a constant, with each observation weighted by $\lambda^{\mathrm{C}}(W_i)/p_{D_i}(W_i)$. When the treatment is binary, this estimator reduces to the estimator studied in Li et al. (2018). Since the propensity score is unknown, we replace the infeasible weights with feasible weights $\hat{\lambda}^{\mathrm{C}}(W_i)/\hat{p}_{D_i}(W_i)$, where $\hat{p}_k(W_i)$ is a feasible estimate of the propensity score and $\hat{\lambda}^{\mathrm{C}}(W_i) = 1/\sum_{k=0}^{K} 1/\hat{p}_k(W_i)$. When $\mathcal{G}$ is finite-dimensional, we may use the regression estimator that projects $X_{ik}$ onto $g(W_i)$:

$$\hat{p}_k(W_i) = \arg\min_{\tilde{p}\in\mathcal{G}} \sum_{i=1}^{N} \left(X_{ik} - \tilde{p}(W_i)\right)^2.$$

With linear $\mathcal{G}$, for example, $\hat{p}_k(W_i)$ is simply the fitted value from a linear regression of $X_{ik}$ on $W_i$ and a constant. The resulting estimator can be written as

$$\hat{\beta}_{\hat{\lambda}^{\mathrm{C}},k} = \frac{1}{\sum_{i=1}^{N} \frac{\hat{\lambda}^{\mathrm{C}}(W_i)}{\hat{p}_k(W_i)} X_{ik}} \sum_{i=1}^{N} \frac{\hat{\lambda}^{\mathrm{C}}(W_i)}{\hat{p}_k(W_i)} X_{ik} Y_i - \frac{1}{\sum_{i=1}^{N} \frac{\hat{\lambda}^{\mathrm{C}}(W_i)}{\hat{p}_0(W_i)} X_{i0}} \sum_{i=1}^{N} \frac{\hat{\lambda}^{\mathrm{C}}(W_i)}{\hat{p}_0(W_i)} X_{i0} Y_i. \tag{23}$$

When the treatment is binary and $\mathcal{G}$ is linear, this weighted regression estimator coincides with the usual (unweighted) regression estimator that regresses $Y_i$ onto $D_i$ and $W_i$.[22]

We now show that the estimator $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}}$ is efficient in the sense that is achieves the semiparametric efficiency bound for estimating $\beta_{\lambda^{\mathrm{C}}}$:

**Proposition 3.** *Suppose eq. (12) holds in an i.i.d. sample of size $N$, with known non-degenerate propensity scores $p_k(W_i)$. Let $\beta^*_{\lambda^C,k} = E[\lambda^C(W_i)\tau_k(W_i)]/E[\lambda^C(W_i)]$, and $\alpha^*_k = \beta^*_{\lambda^C,k} + E[\lambda^C(W_i)\mu_0(W_i)]/E[\lambda^C(W_i)]$. Suppose that the fourth moments of $\lambda^C(W_i)$ and $\mu(W_i)$ are bounded, and that $p_k \in \mathcal{G}$, $(\mu_k(W_i) - \alpha^*_k)\frac{\lambda^C(W_i)^2}{p_{k'}(W_i)^2} \in \mathcal{G}$, and $(\mu_k(W_i) - \alpha^*_k)\frac{\lambda^C(W_i)}{p_k(W_i)} \in \mathcal{G}$ for all $k, k'$. Then, provided it is asymptotically linear and regular, $\hat{\beta}_{\hat{\lambda}^C}$ achieves the semiparametric efficiency bound for estimating $\beta_{\lambda^C}$, with diagonal elements of its asymptotic variance of:*

$$\frac{1}{E[\lambda^C(W_i)]^2} E\left[\frac{\lambda^C(W_i)^2\sigma_0^2(W_i)}{p_0(W_i)} + \frac{\lambda^C(W_i)^2\sigma_k^2(W_i)}{p_k(W_i)} \right.$$
$$\left. + \lambda^C(W_i)^2(\tau_k(W_i) - \beta^*_{\lambda^C,k})^2 \left(\sum_{k'=0}^{K} \frac{\lambda^C(W_i)^2}{p_k(W_i)^3} - 1\right)\right].$$

This efficiency result doesn't rely on homoskedasticity: under heteroskedasticity, the estimator $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}}$ is still efficient for $\beta_{\lambda^{\mathrm{C}}}$ (although the weighting $\lambda^{\mathrm{C}}(W_i)$ is itself not optimal under heteroskedasticity). Proposition 3 is stated under the high-level condition that $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}}$ is regular; the proof uses the calculations in Newey (1994) to verify that the estimator achieves the efficiency bound. Primitive regularity conditions will depend on the form of $\mathcal{G}$ and are omitted for brevity.

*Remark* 4. The asymptotic variance of the estimator $\hat{\beta}_{\lambda^{\mathrm{C}}}$ is larger than the asymptotic variance of the infeasible estimator that replaces the estimated weights $\hat{\lambda}^{\mathrm{C}}(W_i)/\hat{p}_{D_i}(W_i)$ in eq. (23) with the infeasible weights $\lambda^{\mathrm{C}}(W_i)/p_{D_i}(W_i)$. The latter achieves the asymptotic variance implied by Corollary 2,

$$\frac{1}{E[\lambda^{\mathrm{C}}(W_i)]^2} E\left[\frac{\lambda^{\mathrm{C}}(W_i)^2\sigma_0^2(W_i)}{p_0(W_i)} + \frac{\lambda^{\mathrm{C}}(W_i)^2\sigma_k^2(W_i)}{p_k(W_i)}\right]. \tag{24}$$

The extra term of the asymptotic variance in Proposition 3 relative to eq. (24) reflects the cost of having to estimate the weights.[23]

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[22] To see this, note that in this case $\hat{\lambda}(W_i) = \hat{p}_1(W_i)\hat{p}_0(W_i)$, so that $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}},1} = \frac{\sum_{i=1}^N (1-\hat{p}_1(W_i))D_iY_i}{\sum_{i=1}^N (1-\hat{p}_1(W_i))D_i} - \frac{\sum_{i=1}^N \hat{p}_1(W_i)(1-D_i)Y_i}{\sum_{i=1}^N \hat{p}_1(W_i)(1-D_i)} = \frac{\sum_{i=1}^N (D_i - \hat{p}_1(W_i))Y_i}{\sum_{i=1}^N (D_i-\hat{p}_1(W_i))^2}$, where the second equality uses the least-squares normal equations $\sum_{i=1}^N X_{i1} = \sum_{i=1}^N \hat{p}_1(W_i)$ and $\sum_i X_{i1}\hat{p}_1(W_i) = \sum_{i=1}^N \hat{p}_1(W_i)^2$.

[23] The extra term shows this cost is zero if either there is no treatment effect heterogeneity, so that $\tau_k(W_i) = \beta^*_{\lambda^C,k}$, or if the treatment assignment is completely randomized so that $p_k(W_i) = 1/(K+1)$. In the latter case $\lambda^*(W_i) = 1/(K+1)^2$ so $\sum_{k=0}^K \lambda^{\mathrm{C}}(W_i)^2/p(W_i)^3 = 1$. The extra term can be avoided altogether if we interpret

## 5 Practical Guidance and Application

### 5.1 Measuring and Avoiding Contamination Bias

A researcher interested in estimating the effects of multiple dependent treatments with regression can use Proposition 1 to measure the extent of contamination bias in her estimates. When the treatment assignment is conditionally ignorable, she can further compute one of the three alternative estimators discussed in Section 4.[24] Here we provide practical guidance on both procedures, which we illustrate in an application in the next subsection.

For simplicity, we focus on the case where $g$ is linear and eq. (9) is estimated by ordinary least squares (OLS). We assume Assumption 1 and both conditions in Assumption 2 hold, such that all propensity scores $p_k$ and potential outcome conditional expectation functions $\mu_k$ are linearly spanned by the controls $W_i$. These conditions hold, for example, when $W_i$ contains a set of mutually exclusive group indicators.

Under this setup, we can decompose the OLS estimator $\hat{\beta}$ from the uninteracted regression

$$Y_i = \alpha + \sum_{k=1}^{K} X_{ik}\beta_k + W_i'\gamma + U_i, \tag{25}$$

and obtain a sample analog of the decomposition in Proposition 1. To this end, note that the own-treatment and contamination bias weights in Proposition 1 are identified by the linear regression of $X_i$ on the residuals $\tilde{X}_i$. Specifically, $\lambda_{k\ell}(W_i)$ is given by the $(k,\ell)$th element of the $K \times K$ matrix

$$\Lambda(W_i) = E[\tilde{X}_i\tilde{X}_i']^{-1}E[\tilde{X}_iX_i' \mid W_i].$$

An estimate of this weight matrix is given by the sample analog:

$$\hat{\Lambda}_i = (\dot{X}'\dot{X})^{-1}\dot{X}_iX_i',$$

where $\dot{X}_i$ is the sample residual from an OLS regression of $X_i$ on $W_i$ and a constant, and $\dot{X}$ is a matrix collecting these sample residuals. The $(k,\ell)$th element of $\hat{\Lambda}_i$ estimates the weight that observation $i$ puts on the $\ell$th treatment effect in the $k$th treatment coefficient. For $k = \ell$ this is an estimate of the own-treatment weight in Proposition 1; for $k \neq \ell$ this is an estimate of a contamination weight.

Under linearity, the $k$th conditional ATEs may be written as $\tau_k(W_i) = \gamma_{0,k} + W_i'\gamma_{W,k}$,

$\hat{\beta}_{\hat{\lambda}_C}$ as an estimator of $\beta_{\hat{\lambda}_C}$. This follows from arguments in Crump et al. (2006, Lemma B.6).

[24] We again refer readers to de Chaisemartin and D'Haultfœuille (2022), Sun and Abraham (2021), Callaway and Sant'Anna (2021), Borusyak et al. (2022), and Wooldridge (2021) for solutions under a parallel trends assumption.

where $\gamma_{0,k}$ and $\gamma_{W,k}$ are coefficients in the interacted regression specification

$$Y_i = \alpha_0 + \sum_{k=1}^{K} X_{ik}\gamma_{0,k} + W_i'\alpha_{W,0} + \sum_{k=1}^{K} X_{ik}W_i'\gamma_{W,k} + \dot{U}_i. \tag{26}$$

Estimating eq. (26) by OLS yields estimates $\hat{\tau}_k(W_i) = \hat{\gamma}_{0,k} + W_i'\hat{\gamma}_{W,k}$. For each observation $i$, we stack the set of conditional ATE estimates in a $K \times 1$ vector $\hat{\tau}(W_i)$.

Using OLS normal equations, we then obtain the exact decomposition

$$\hat{\beta} = \sum_{i=1}^{N} \operatorname{diag}(\hat{\Lambda}_i)\hat{\tau}(W_i) + \sum_{i=1}^{N} [\hat{\Lambda}_i - \operatorname{diag}(\hat{\Lambda}_i)]\hat{\tau}(W_i), \tag{27}$$

which is the sample analog of the population decomposition in Proposition 1. The first term in this decomposition estimates the own-treatment effect components, $E[\lambda_{kk}(W_i)\tau_k(W_i)]$, while the second term estimates the contamination bias components, $\sum_{\ell \neq k} E[\lambda_{k\ell}(W_i)\tau_\ell(W_i)]$. If the contamination bias term is large for some $\hat{\beta}_k$, it suggests the estimate of the $k$th treatment effect is substantially impacted by the effects of other treatments. Researchers can also compare the first term of eq. (27) to other weighted averages of own-treatment effects, including the ones discussed next, to gauge the impact of the regression weighting $\operatorname{diag}(\hat{\Lambda}_i)$.[25]

Further analysis of the estimated weights $\hat{\lambda}_{k\ell}(w) = \frac{\sum_{i=1}^{N} \mathbb{1}\{W_i=w\}\hat{\Lambda}_{i,k\ell}}{\sum_{i=1}^{N} \mathbb{1}\{W_i=w\}}$ can shed more light on the regression estimates in $\hat{\beta}$. For example, the contamination weights for $\ell \neq k$ can be plotted against the treatment effect estimates $\hat{\tau}_\ell(W_i)$ to visually assess the sources of contamination bias. Low bias may arise from limited treatment effect heterogeneity or a low correlation between such heterogeneity and the contamination weights.

Implementing the alternative estimators from Section 4 is also straightforward under the linearity assumptions. For the first solution, estimating the interacted regression

$$Y_i = \alpha_0 + \sum_{k=1}^{K} X_{ik}\tau_k + W_i'\alpha_{W,0} + \sum_{k=1}^{K} X_{ik}(W_i - \overline{W})'\gamma_{W,k} + \dot{U}_i. \tag{28}$$

by OLS yields estimates of the unweighted ATEs $\tau_k = E[\tau_k(W_i)]$. Here $\overline{W} = \frac{1}{N}\sum_i W_i$ is the sample average of the covariate vector. The estimates are numerically equivalent to $\hat{\tau}_k = \hat{\gamma}_{0,k} + \overline{W}'\hat{\gamma}_{W,k}$, where $\hat{\gamma}_{0,k}$ and $\hat{\gamma}_{W,k}$ are OLS estimates of eq. (26).

[25] When the covariates are not saturated, it is possible that the estimated weighting function $\hat{\Lambda}(w) = \frac{1}{N}\sum_{i=1}^{N} \mathbb{1}\{W_i = w\}\hat{\Lambda}_i$ is not positive-definite for some or all $w$. In particular, the diagonal elements of $\hat{\Lambda}(w)$ need not all be positive. However, it is guaranteed that the diagonal of $\hat{\Lambda}(w)$ sums to one and the non-diagonal weights sum to zero, since $\sum_{i=1}^{N} \hat{\Lambda}_i = I_k$.

The second solution is to estimate the uninteracted regression,

$$Y_i = \ddot{\alpha}_k + X_{ik}\ddot{\beta}_k + W_i'\ddot{\gamma}_k + \ddot{U}_{ik} \tag{29}$$

among observations assigned either to treatment $k$ or the control group, $D_i \in \{0, k\}$, for each of the treatments $k = 1, \ldots, K$. These one-treatment-at-a-time regressions estimate convex weighted averages of treatment effects, with weights that are efficient under homoskedasticity (in the sense of corollary 1) but which will generally vary across the different treatments. This can make comparisons across treatment arms difficult.

The third solution is to estimate an efficiently weighted average of the conditional ATEs, with weights that are constrained to be common across treatments. Under linearity, we can estimate the common weights $\lambda^{\mathrm{C}}$ as

$$\hat{\lambda}^{\mathrm{C}}(W_i) = \left(\sum_{k=0}^{K} \hat{p}_k(W_i)^{-1}\right)^{-1}, \tag{30}$$

where $\hat{p}_k(W_i) = X_{ik} - \dot{X}_{ik}$ denote estimated propensity scores. We then regress $Y_i$ on $X_i$, weighting each observation by $\hat{\lambda}^C(W_i)/\hat{p}_{D_i}(W_i)$.

While the second and third solutions may yield more precise estimates than the equal-weighted ATE estimates, the gains in precision are achieved by changing the estimand to a different convex average of conditional treatment effects. In particular, covariate values $w$ where the propensity score $p_k(w)$ is close to zero for some $k$ will be effectively discarded.

On the other hand, if the conditional treatment effects $\tau(W_i)$ are approximately independent of the propensity scores $p(W_i)$, the weighting scheme may have little effect on the estimands, even if the treatment effect heterogeneity is substantial. In such cases, we also expect the contamination bias to be small, since the contamination weights are a function of the propensity scores. We next investigate this possibility in our application.

## 5.2 Application

We illustrate the potential for contamination bias using data from Project STAR, as analyzed in Krueger (1999). The Project STAR RCT randomized 11,600 students in 79 public Tennessee elementary schools to one of three types of classes: regular-sized (20–25 students), small (target size 13–17 students), or regular-sized with a teaching aide. The proportion of students randomized to the small class size and teaching aide treatment varied over schools, due to school size and other constraints on classroom organization. Students entering kindergarten in the 1985–1986 school year participated in the experiment through the third grade. Other students entering a participating school in grades 1–3 during these years were similarly randomized between the three class types. We focus on kindergarten effects, where differential attrition and other complications with the experimental analysis are minimal.[26] All analyses in this section are conducted with our Stata package, `multe`, which researchers can use to gauge the extent of contamination bias in similar applications.

Column 1 of Panel A in Table 1 reports estimates of kindergarten treatment effects in a sample of 5,868 students initially randomized to the small class size and teaching aide treatments. Specifically, we estimate the uninteracted regression in eq. (25), where $Y_i$ is student $i$'s test score achievement at the end of kindergarten, $X_i = (X_{i1}, X_{i2})$ are indicators for the initial experimental assignment to a small kindergarten class and a regular-sized class with a teaching aide, respectively, and $W_i$ is a vector of school fixed effects. We follow Krueger (1999) in computing $Y_i$ as the average percentile of student $i$'s math, reading, and word recognition score on the Stanford Achievement Test in the experimental sample. As in the original analysis (Krueger, 1999, column 6 of Table V, panel A), we obtain a small class size effect of 5.36, with a heteroskedasticity-robust standard error of 0.78, and a teaching aide effect of 0.18 (standard error 0.72).[27]

As discussed in Section 2, treatment assignment probabilities vary across the schools, indicated by the fixed effects in $W_i$. If treatment effects also vary across schools, and if this variation is correlated with the contamination weights $\lambda_{k\ell}(W_i)$, we expect the estimated effect of small class sizes to be partly contaminated by the effect of teaching aides (and vice versa). Net of any contamination bias, we expect each $\beta_k$ to identify a weighted average of own treatment effects $\tau_k(W_i)$, with convex weights given by $\lambda_{kk}(W_i)$.

Columns 2 and 3 of Table 1 apply the decomposition in eq. (27) to the regression coefficients in column 1. The contamination bias appears to be minimal. The small class size regression estimate of 5.36 is composed of a weighted average of small class size treatment effects equalling 5.20 and a weighted average of teaching aide treatment effects equalling 0.16. Similarly, the teaching aide regression coefficient of 0.18 decomposes into a weighted average of teaching aide treatment effects equalling 0.36 and a weighted average of small class size effects equalling $-0.18$. Netting out the contamination bias estimate doubles the teaching aide effect estimate, from 0.18 to 0.36, but the estimate remains statistically insignificant with standard errors of around 0.71.

The lack of meaningful bias in the regression estimates of Project STAR effects is due to

[26] Students in regular-sized classes were randomly reassigned between classrooms with and without a teaching aide after kindergarten, complicating the interpretation of the aide effect in later grades. The randomization of students entering the sample after kindergarten was also complicated by the uneven availability of slots in small and regular-sized classes (Krueger, 1999).

[27] Our sample and estimates are very similar to—but not exactly the same as—those in Krueger (1999). We use robust (non-clustered) standard errors throughout this analysis, since the randomization of students to classrooms is at the individual level (Abadie et al., 2017). Results are similar when we cluster by classroom.

Table 1: Project STAR Contamination Bias and Treatment Effect Estimates

| | A. Contamination Bias Estimates | | | | |
|---|---|---|---|---|---|
| | Regression Coefficient | Own Effect | Bias | Worst-Case Bias: Negative | Worst-Case Bias: Positive |
| | (1) | (2) | (3) | (4) | (5) |
| Small Class Size | 5.357 | 5.202 | 0.155 | -1.654 | 1.670 |
| | (0.778) | (0.778) | (0.160) | (0.185) | (0.187) |
| Teaching Aide | 0.177 | 0.360 | -0.183 | -1.529 | 1.530 |
| | (0.720) | (0.714) | (0.149) | (0.176) | (0.177) |

| | B. Treatment Effect Estimates | | |
|---|---|---|---|
| | Unweighted (ATE) | Efficiently-Weighted: One-at-a-time | Efficiently-Weighted: Common |
| | (1) | (2) | (3) |
| Small Class Size | 5.561 | 5.295 | 5.563 |
| | (0.763) | (0.775) | (0.764) |
| | [0.744] | [0.743] | [0.742] |
| Teaching Aide | 0.070 | 0.263 | -0.003 |
| | (0.708) | (0.715) | (0.712) |
| | [0.694] | [0.691] | [0.695] |

*Notes:* Panel A estimates the contamination bias and range of potential contamination bias in regression estimates of small class and teaching aide treatment effects for the Project STAR kindergarten analysis. The analysis sample includes 5,868 students. Column 1 reports estimates from a partially linear model in eq. (25). Columns 2 and 3 estimate the own- and cross-treatment decomposition of this estimate in eq. (27). Columns 4 and 5 reports the smallest (largest) possible contamination bias from reordering the conditional ATEs to be as negatively (positively) correlated with the cross-treatment weights as possible. Panel B summarizes estimates of small class and teaching aide treatment effects from different specifications in the kindergarten sample of Project STAR. Column 1 reports estimates of small class and teaching aide treatment effects from the interacted model in eq. (28). Column 2 reports estimates from the treatment-specific regressions in eq. (29). Column 3 reports estimates from the efficiently weighted specification, using the estimated weights in eq. (30). Robust standard errors are reported in parentheses. Standard errors that assume the propensity scores are known are reported in square brackets.

a weak correlation between the conditional treatment effects $\tau(W_i)$ and the contamination weights. These correlations are shown in Figure 2, which plots estimates of the school-specific treatment effects $\tau_k(W_i)$ against the own-treatment and contamination weights $\lambda_{kk}(W_i)$ and $\lambda_{k\ell}(W_i)$ for $\ell \neq k$. Panels A and C show that the own-treatment weight correlation is negative for the small class size treatment ($-0.19$) and positive for the aide treatment ($0.25$). The partially linear regression model's estimate of own-treatment effects (column 2 of Table 1) thus understates the average small class size effect and overstates the average aide effect, relative to the ATE. Panels B and D further show that correlation between estimated cross-treatment effects and weights is positive for the small class effect estimate ($0.10$) and negative for the aide effect estimate ($-0.13$). There is thus positive contamination bias in the partially linear regression model's estimate of small class size effects and negative contamination bias in the regression's aide effect estimate, as shown in column 2 of Table 1. But neither set of correlations is strong enough to meaningfully bias the estimates.

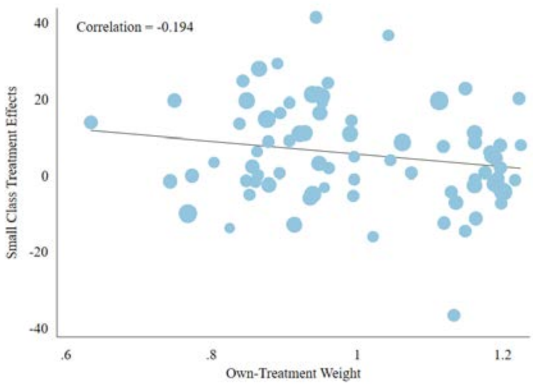
Importantly, Figure 2 shows that the lack of contamination bias is not due to a lack of treatment effect heterogeneity across schools. There is considerable variation along the $y$-axis of each plot. Adjusting for estimation error, we find a standard deviation of $\tau_k(W_i)$ across the schools indexed by $W_i$ of 12.7 for the small class treatment and of 10.9 for the aide treatment.[28] Both standard deviations are an order of magnitude larger than the standard errors in Table 1. Thus, had the experimental design been such that the contamination weights strongly correlate with this variation, sizeable contamination bias could have resulted. In practice, the variation in school-specific propensity scores $p_k(W_i)$ appears to have been largely unrelated to school-specific treatment effects.[29]

To illustrate the potential for contamination bias in this setting, we compute worst-case (positive and negative) weighted averages of the estimated $\tau_k(W_i)$ by re-ordering them across the computed cross-treatment weights $\lambda_{k\ell}(W_i)$. This exercise highlights potential scenarios in which the randomization strata happened to have been highly correlated with the heterogeneity in treatment effects. Columns 4 and 5 in Table 1 shows that both bounds on possible contamination bias are large relative to the effect estimates: $[-1.654, 1.670]$ for the small class size treatment and $[-1.529, 1.530]$ for the teaching aide treatment. Thus, while actual contamination bias for both treatments is an order of magnitude smaller, the underlying heterogeneity in this setting makes severe contamination bias possible.

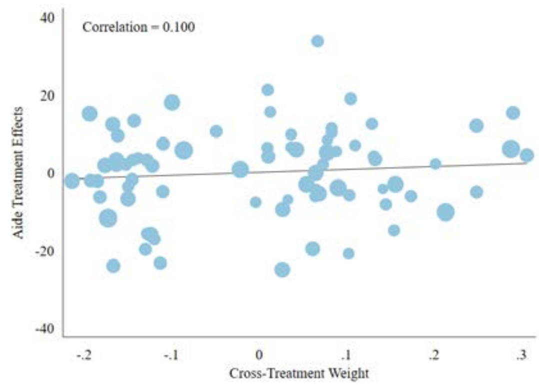
Panel B of Table 1 illustrates the three solutions to the contamination bias problem discussed in Section 4. Column 1 estimates the unweighted ATEs of the small class size and

[28] We adjust for estimation error by subtracting the average squared standard error from the empirical variance of the treatment effect estimates and taking the square root.

[29] The own-treatment weights in Figure 2 are highly correlated with the respective treatment propensity score. For the small class size (teaching aide) treatment this correlation is 0.92 (0.73).

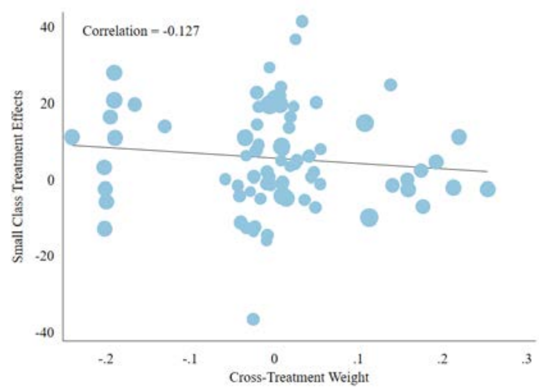



Panel A: Small Class
Own-Treatment Weight




Panel B: Aide
Cross-Treatment Weight


Panel C: Aide
Own-Treatment Weight




Panel D: Small Class
Cross-Treatment Weight

Figure 2: Project STAR Treatment Effects and Regression Weights

*Note:* This figure shows correlations between estimated school-specific treatment effects and the implicit school-specific regression weights in column 1 of Table 1. Panels A and B show correlations for the decomposition of the small class treatment effect estimate in columns 2 and 3 of Table 1. Panel A plots the estimated small class treatment effects by school against the estimated own-treatment weights, while Panel B plots the estimated teaching aide treatment effects by school against the estimated cross-treatment weights. Panels C and D show analogous correlations for the decomposition of the teaching aide treatment effect estimate in columns 2 and 3 of Table 1. Panel C plots the estimated teaching aide treatment effects by school against the estimated own-treatment weights, while Panel D plots the estimated small class treatment effects by school against the estimated cross-treatment weights. Correlations and lines of best fit are reported on each panel. The size of the points is proportional to the number of students enrolled in each school.

teaching aide treatment, by estimating the interacted regression specification in eq. (28). Column 2 estimates the one-treatment-at-a-time regressions in eq. (29) for $k = 1, 2$. Finally, column 3 estimates the efficiently-weighted ATEs of each treatment, by running a weighted regression of $Y_i$ onto $X_i$, using the common weight estimates in eq. (30).

As discussed in Remark 4, the optimal weighting schemes underlying the estimates in columns 2 and 3 of Panel B are derived under the assumption that the propensity scores are known. To gauge the relative importance of this assumption, Panel B also reports a version of the standard errors computed under the assumption that the sample treatment probabilities in each school match the true propensity scores.[30] This changes the standard errors little, showing that there is minimal cost to estimating the optimal weights.[31]

There turns out to be little difference between the partially linear model estimates of Project STAR treatment effects and these alternative estimates. In columns 1 and 2 of Panel B we estimate a small class size effect of 5.56 and 5.30, which are close to the 5.36 estimate in column 1 of Panel A. Teaching aide effect estimates are also similar: 0.07 and 0.26 in columns 1 and 2 of Panel B, compared to 0.18 in column 1 of Panel A. The efficiently weighted estimates in column 3 of Panel B are again similar: 5.56 for the small class size treatment and 0.00 for the teaching aide treatment. Interestingly, the standard errors are roughly constant across the columns, regardless of whether the propensity score is treated as known.

# 6 Conclusion

Regressions with multiple treatments and flexible controls are common across a wide range of empirical settings in economics. We show that such regressions generally fail to estimate a convex weighted average of heterogeneous effects, with coefficients on each treatment generally contaminated by the effects of other treatments. We provide intuition for why the influential result of Angrist (1998) fails to generalize to multiple treatments, and show how the contamination bias problem connects to a recent literature studying DiD regressions and related estimators. We discuss three alternative estimators that are free of this bias, including a new estimator that efficiently weights conditional average treatment effects. The analysis underling this estimator also formalizes a virtue of regression adjustment in the binary treatment case: the weighting that it implicitly uses to combine heterogeneous treatment effects minimizes the semiparametric efficiency bound for convex weighted averages of ATEs.

[30] This is the case under stratified block randomization, where a fixed proportion of students is assigned to the two treatments. In contrast, sample treatment proportions need not match the true propensity scores under a Bernoulli trial where each student is assigned to treatments according to a coin toss.

[31] The standard errors reported in parentheses in Panel B are valid for the population analogs $\beta_k$ and $\beta_{\lambda^{\text{C}}}$, i.e. $E[\lambda^k(W_i)\tau_k(W_i)]/E[\lambda^k(W_i)]$ and $E[\lambda^{\text{C}}(W_i)\tau_k(W_i)]/E[\lambda^{\text{C}}(W_i)]$. Since these standard errors are potentially conservative when viewed as standard errors for $\beta_k$ and $\beta_{\lambda^{\text{C}}}$, the standard error comparison gives an upper bound on the cost to estimating the optimal weights.

Our application to Project STAR shows that significant contamination bias could arise in RCTs when there is significant treatment effect heterogeneity. Whether the bias *does* arise, however, depends on the correlation between effect heterogeneity and the contamination weights we derive in our theoretical analysis. Researchers can estimate this correlation, and report it alongside the alternative estimates that are free of contamination bias. Such investigation reveals whether, as in our application, the results based on alternative estimators are more similar than the worst-case bounds implied by the theory. Broadly, our analysis highlights the importance of testing the empirical relevance of theoretical concerns with how regression combines heterogeneous effects.

We expect the tools in this paper to be especially relevant in modern RCT designs that generate substantial variation in treatment propensity scores to maximize efficiency (e.g. Tabord-Meehan, 2021). Propensity scores are also likely to vary dramatically in quasi-experimental analyses, such as with teacher VAMs, where a large number of covariates are needed to make the conditionally ignorability of treatment plausible. Contamination bias diagnostics can be a useful tool for ensuring the reliability and robustness of regression estimates in such settings.

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# Appendix A Proofs

## A.1 Proof of Proposition 1

We prove a generalization of the Proposition 1 which allows any vector of treatments $X_i$ (which may not be binary or mutually exclusive). We continue to consider the partially linear model in eq. (9), and maintain Assumption 2, as well as conditional mean-independence of the potential outcomes $E[Y_i(x) \mid X_i, W_i] = E[Y_i(x) \mid W_i]$, which extends Assumption 1. We also assume that the potential outcomes $Y_i(x)$ are linear in $x$, conditional on $W_i$:

$$E[Y_i(x) \mid W_i = w] = E[Y_i(0) \mid W_i = w] + x'\tau(w),$$

for some function $\tau$. This condition holds trivially in the main-text discussion of mutually exclusive binary treatments. More generally, $\tau_k(w)$ corresponds to the conditional average effect of increasing $X_{ik}$ by one unit among observations with $W_i = w$. Although this assumption is not essential, it considerably simplifies the derivations. We continue to define $\tau = E[\tau(W_i)]$ as the average vector of per-unit effects.

We now prove that under these assumptions $\beta_k$ is given by the expression in eq. (16). We further prove that $E[\lambda_{kk}(W_i)] = 1$ and $E[\lambda_{k\ell}(W_i)] = 0$ for $\ell \neq k$ in general, and that $\lambda_{kk}(W_i) \geq 0$ in the case of mutually exclusive treatment indicators.

First note that by iterated expectations and conditional mean-independence, $E[\tilde{\tilde{X}}_{ik} Y_i] = E[E[\tilde{\tilde{X}}_{ik} Y_i \mid X_i, W_i]] = E[\tilde{\tilde{X}}_{ik} E[Y_i(0) \mid W_i]] + E[\tilde{\tilde{X}}_{ik} X_i' \tau(W_i)]$. By definition of projection, $E[\tilde{X}_i g(W_i)] = 0$ for all $g \in \mathcal{G}$ (van der Vaart, 1998, Theorem 11.1); thus if eq. (14) holds $E[\tilde{\tilde{X}}_{ik} E[Y_i(0) \mid W_i]] = 0$. Similarly, under eq. (13), $E[\tilde{\tilde{X}}_{ik} \mid W_i] = 0$, so by iterated expectations, $E[\tilde{\tilde{X}}_{ik} E[Y_i(0) \mid W_i]] = E[E[\tilde{\tilde{X}}_{ik} \mid W_i] E[Y_i(0) \mid W_i]] = 0$. Thus,

$$\beta_k = \frac{E[\tilde{\tilde{X}}_{ik} X_i' \tau(W_i)]}{E[\tilde{\tilde{X}}_{ik}^2]} = \frac{E[\tilde{\tilde{X}}_{ik} X_{ik} \tau_k(W_i)]}{E[\tilde{\tilde{X}}_{ik}^2]} + \frac{\sum_{\ell \neq k} E[\tilde{\tilde{X}}_{ik} X_{i\ell} \tau_\ell(W_i)]}{E[\tilde{\tilde{X}}_{ik}^2]}.$$

This proves eq. (16).

To show that $E[\lambda_{kk}(W_i)] = 1$ and $E[\lambda_{k\ell}(W_i)] = 0$ for $\ell \neq k$ in general, note that

$$E[\lambda_{kk}(W_i)] = \frac{E[\tilde{\tilde{X}}_{ik} X_{ik}]}{E[\tilde{\tilde{X}}_{ik}^2]} = 1,$$

since $\tilde{\tilde{X}}_{i,k}$ is a residual from projecting $X_{ik}$ onto the space spanned by functions of the form $\tilde{g}(W_i) + X_{i,-k}' \tilde{\beta}_{-k}$, so that $E[\tilde{\tilde{X}}_{ik} X_{ik}] = E[\tilde{\tilde{X}}_{ik}^2]$. Furthermore, $\tilde{\tilde{X}}_{i,k}$ must also be orthogonal to $X_{i,-k}$ by definition of projection, so that $E[\lambda_{k\ell}(W_i)] = E[\tilde{\tilde{X}}_{ik} X_{i\ell}] / E[\tilde{\tilde{X}}_{ik}^2] = 0$.

Finally, we show that $\lambda_{kk}(W_i) \geq 0$ if eq. (13) holds and $X_i$ consists of mutually exclusive indicators. To that end, observe that $\lambda_{k\ell}(W_i)$ is given by the $(k, \ell)$ element of

$$\Lambda(W_i) = E[\tilde{X}_i \tilde{X}_i']^{-1} E[\tilde{X}_i X_i' \mid W_i]$$

If Equation (13) holds, then we can write this as $\Lambda(W_i) = E[v(W_i)]^{-1} v(W_i)$ where $v(W_i) = E[\tilde{X}_i \tilde{X}_i' \mid W_i]$. If $X$ is a vector of mutually exclusive indicators, then $v(W_i) = \operatorname{diag}(p(W_i)) - p(W_i)p(W_i)'$. Let $v_{-k}(W_i)$ denote the submatrix with the $k$th row and column removed, and for any vector $a$, let $a_{-k}$ denote subvector with the $k$th column removed. Then by the block matrix inverse formula,

$$\lambda_{kk}(W_i) = \frac{p_k(W_i)(1 - p_k(W_i)) - E[p_k(W_i)p(W_i)'_{-k}]E[v_{-k}(W_i)]^{-1}p(W_i)_{-k}p_k(W_i)}{E[p_k(W_i)(1 - p_k(W_i))] - E[p_k(W_i)p_{-k}(W_i)']E[v_{-k}(W_i)]^{-1}E[p_k(W_i)p_{-k}(W_i)]}$$

Note $p_0(W_i) = 1 - \sum_{k=1}^{K} p_k(W_i)$ and $p_k(W_i)p(W_i)_{-k} = v_{-k}(W_i)\iota - p_0(W_i)p(W_i)_{-k}$, where $\iota$ denotes a $(K-1)$-vector of ones. Thus the numerator can be written as

$$\begin{aligned} p_k(W_i)&(1 - p_k(W_i)) - \iota' p(W_i)_{-k} p_k(W_i) \\ &\qquad + E[p_0(W_i)p(W_i)'_{-k}]E[v_{-k}(W_i)]^{-1}p(W_i)_{-k}p_k(W_i) \\ &\qquad\quad = p_k(W_i)p_0(W_i) + E[p_0(W_i)p(W_i)'_{-k}]E[v_{-k}(W_i)]^{-1}p(W_i)_{-k}p_k(W_i). \end{aligned}$$

The eigenvalues of $E[v_{-k}(W_i)]$ are positive because it is a covariance matrix. Furthermore, since the off-diagonal elements of $E[v(W_i)]$ are negative, the off-diagonal elements of $E[v_{-k}(W_i)]$ are also negative. It therefore follows that $E[v_{-k}(W_i)]$ is an $M$-matrix (Berman & Plemmons, 1994, property $D_{16}$, p. 135). Hence, all elements of $E[v_{-k}(W_i)]^{-1}$ are positive (Berman & Plemmons, 1994, property $N_{38}$, p. 137). Thus, both summands in the above expression are positive, so that $\lambda_{kk}(W_i) \geq 0$.

### A.2 Proof of Proposition 2

The parameter of interest $\theta_{\lambda,c}$ depends on the realizations of the controls. We therefore derive the semiparametric efficiency bound conditional on the controls; i.e. we show that eq. (18) is almost surely the variance bound for estimators that are regular conditional on the controls. Relative to the earlier results in Hahn (1998) and Hirano et al. (2003), we need account for the fact that the data are no longer i.i.d. once we condition on the controls.

To that end, we use the notion of semiparametric efficiency based on the convolution theorem of van der Vaart and Wellner (1989, Theorem 2.1) (see also van der Vaart & Wellner, 1996, Chapter 3.11). We first review the result for convenience. Consider a model $\{P_{n,\theta} \colon \theta \in \Theta\}$ parametrized by (a possibly infinite-dimensional) parameter $\theta$. Let $\dot{\mathcal{P}}$ denote a tangent space, a linear subspace of some Hilbert space with an inner product $\langle \cdot, \cdot \rangle$. Suppose that the model is locally asymptotically normal (LAN) at $\theta$ relative to a tangent space $\dot{\mathcal{P}}$: for each $g \in \dot{\mathcal{P}}$, there exists a sequence $\theta_n(g)$ such that the likelihood ratios are asymptotically quadratic, $dP_{n,\theta_n(g)}/dP_{n,\theta} = \Delta_{n,g} - \langle g, g \rangle/2 + o_{P_{n,\theta}}(1)$, where $(\Delta_{n,g})_{g \in \dot{\mathcal{P}}}$ converges under $P_{n,\theta}$ to a Gaussian process with covariance kernel $\langle g_1, g_2 \rangle$. Suppose also that the parameter $\beta_n(P_{n,\theta})$ is differentiable: for each $g$, $\sqrt{n}(\beta_n(P_{n,\theta_n(g)}) - \beta_n(P_{n,\theta})) \to \langle \psi, g \rangle$ for some $\psi$ that lies in the completion of $\dot{\mathcal{P}}$. Then the semiparametric efficiency bound is given by $\langle \psi, \psi \rangle$: the asymptotic distribution of any regular estimator of this parameter, based on a sample $\mathbf{S}_n \sim P_{n,\theta}$, is given by the convolution of a random variable $Z \sim \mathcal{N}(0, \langle \psi, \psi \rangle)$ and some other random variable $U$ that is independent of $Z$.

To apply this result in our setting, we proceed in three steps. First, we define the tangent space and the probability-one set over which we will prove the efficiency bound. Next, we verify that the model is LAN. Finally, we verify differentiability and derive the efficient influence function $\psi$.

**Step 1** By the conditional independence assumption in eq. (12), we can write the density of the vector $(Y_i(0), \dots, Y_i(K), D_i)$ (with respect to some $\sigma$-finite measure) conditional on $W_i = w$ as $f(y_0, \dots, y_K \mid w) \cdot \prod_{k=0}^{K} p_k(w)^{\mathbb{1}\{d=k\}}$, where $f$ denotes the conditional density of the potential outcomes, conditional on the controls. The density of the observed data $\mathbf{S}_N = \{(Y_i, D_i)\}_{i=1}^{N}$ conditional on $(W_1, \dots, W_N) = (w_1, \dots, w_N)$ is given by $\prod_{i=1}^{N} \prod_{k=0}^{K} (f_k(y_i \mid w_i) p_k(w_i))^{\mathbb{1}\{d_i=k\}}$, where $f_k(y \mid w) = \int f(y_k, y_{-k} \mid w) dy_{-k}$.

Since the propensity scores are known, the model is parametrized by $\theta = f$. Consider one-dimensional submodels of the form $f_k(y \mid w; t) = f_k(y \mid w)(1 + t s_k(y \mid w))$, where the function $s_k$ is bounded and satisfies $\int s_k(y \mid w) f_k(y \mid w) dy = 0$ for all $w \in \mathcal{W}$ with $\mathcal{W}$ denoting the support of $W_i$. For small enough $t$, we have $f_k(y \mid w; t) \geq 0$ by boundedness of $s_k$; hence $f_k(y \mid w; t)$ is a well-defined density for $t$ small enough. The joint log-likelihood, conditional on the controls, is given by

$$\sum_{i=1}^{N} \sum_{k=0}^{K} \mathbb{1}\{D_i = k\} (\log f_k(Y_i \mid w_i; t) + \log p_k(w_i)).$$

The score at $t = 0$ is $\sum_{i=1}^{N} s(Y_i, D_i \mid w_i)$, with $s(Y_i, D_i \mid w_i) = \sum_{k=0}^{K} \mathbb{1}\{D_i = k\} s_k(Y_i \mid w_i)$.

This result suggests defining the tangent space to consist of functions $s(y, d \mid w) = \sum_{k=0}^{K} \mathbb{1}\{d = k\} s_k(y \mid W_i = w)$, such that $s_k$ is bounded and satisfies $\int s_k(y \mid w) f_k(y \mid w) dy = 0$ for all $w \in \mathcal{W}$. Define the inner product on this space by $\langle s_1, s_2 \rangle = E[s_1(Y_i, D_i \mid W_i) s_2(Y_i, D_i \mid W_i)]$. Note this is a marginal (rather than a conditional) expectation, over the unconditional distribution $(Y_i, D_i, W_i)$ of the observed data.

We will prove the efficiency bound on the event $\mathcal{E}$ that (i) $\frac{1}{N}\sum_{i=1}^{N} E[s(Y_i, D_i \mid W_i)^2 \mid W_i] \to E[s(Y, D_i \mid W_i)^2]$, (ii) $\frac{1}{N}\sum_{i=1}^{N}\lambda(W_i) \to E[\lambda(W_i)]$, and (iii) $\frac{1}{N}\sum_{i=1}^{N}\lambda(W_i)\sum_{k=0}^{K} c_k \cdot E[Y_i(k)s_k(Y_i(k) \mid W_i) \mid W_i] \to \sum_{k=0}^{K} c_k E[\lambda(W_i)Y_i(k)s_k(Y_i(k) \mid W_i)]$. By assumptions of the proposition, these are all averages of functions of $W_i$ with finite absolute moments. Hence, by the law of large numbers, $\mathcal{E}$ is a probability one set.

**Step 2** We verify that the conditions (3.7–12) of Theorem 3.1 in McNeney and Wellner (2000) hold on the set $\mathcal{E}$ conditional on the controls, with $\theta_N(s) = f(\cdot \mid \cdot; 1/\sqrt{N})$. Let $\alpha_{Ni} = \prod_{k=0}^{K}(f_k(Y_i \mid w_i; 1/\sqrt{N})/f_k(Y_i \mid w_i))^{\mathbb{1}\{D_i=k\}} = \prod_{k=0}^{K}(1 + s_k(Y_i \mid w)/\sqrt{N})^{\mathbb{1}\{D_i=k\}}$ denote the likelihood ratio associated with the $i$th observation. Since this is bounded by the boundedness of $s_k$, condition (3.7) holds. Also since $(1+ts_k)^{1/2}$ is continuously differentiable for $t$ small enough, with derivative $s_k/2\sqrt{1+ts_k}$, it follows from Lemma 7.6 in van der Vaart (1998) that $N^{-1}\sum_{i=1}^{N} E[\sqrt{N}(\alpha_{Ni}^{1/2} - 1) - s(Y_i, D_i \mid w_i)/2 \mid W_i = w_i]^2 \to 0$ such that the quadratic mean differentiability condition (3.8) holds. Since $s_k$ is bounded, the Lindeberg condition (3.9) also holds. Next, $\frac{1}{N}\sum_{i=1}^{N} E[s(Y_i, D_i \mid W_i)^2 \mid W_i]$ converges to $E[s(Y, D_i \mid W_i)^2] = \langle s, s\rangle$ on $\mathcal{E}$ by assumption. Hence, conditions (3.10) and (3.11) also hold. Since the scores $\Delta_{N,s} = \frac{1}{\sqrt{N}}\sum_{i=1}^{N} s(Y_i, D_i \mid w_i)$ are exactly linear in $s$, condition (3.12) also holds. It follows that the model is LAN on $\mathcal{E}$.

**Step 3** Write the parameter of interest $\theta_{\lambda,c}$ as $\beta_N(f) = \sum_{i=1}^{N}\lambda(w_i)\int y\sum_{k=0}^{K} c_k f_k(y \mid w_i)dy/\sum_{i=1}^{N}\lambda(w_i)$. It follows that

$$
\begin{aligned}
&\sqrt{N}(\beta_N(f(\cdot \mid \cdot; 1/\sqrt{N})) - \beta_N(f)) \\
&\quad = \frac{1}{N^{-1}\sum_{i=1}^{N}\lambda(w_i)}\frac{1}{\sqrt{N}}\sum_{i=1}^{N}\lambda(w_i)\int y\sum_{k=0}^{K} c_k(f_k(y \mid w_i; 1/\sqrt{N}) - f_k(y \mid w_i))dy \\
&\qquad\qquad = \frac{1}{N^{-1}\sum_{i=1}^{N}\lambda(w_i)}\frac{1}{N}\sum_{i=1}^{N}\lambda(w_i)\sum_{k=0}^{K} c_k\int y s_k(y \mid w_i) f_k(y \mid w_i)dy,
\end{aligned}
$$

which converges to $\sum_{k=0}^{K} c_k E[\lambda(W_i)Y_i(k)s_k(Y_i(k) \mid W_i)]/E[\lambda(W_i)]$ on $\mathcal{E}$ by assumption. We can write this as $\langle \psi, s\rangle$, where

$$
\psi(Y_i, D_i, W_i) = \sum_{k=0}^{K}\mathbb{1}\{D_i = k\}\lambda(W_i)c_k\frac{(Y_i - \mu_k(W_i)).}{p_k(W_i)E[\lambda(W_i)]}.
$$

Observe that $\psi$ is in the model tangent space, with the summands playing the role of $s_k(y \mid w)$ (more precisely, since $\psi$ is unbounded, it lies in the completion of the tangent space). Hence, the semiparametric efficiency bound is given by $E[\psi^2]$.

### A.3 Proof of Proposition 3

We first derive the semiparametric efficiency bound for estimating $\beta_{\lambda^{\mathrm{C}}}$ when the propensity scores are not known, using the same steps, notation, and setup as in the proof of Proposition 1. We then verify that the estimator $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}}$ achieves this bound.

**Step 1** Since the propensity scores are not known, the model is now parametrized by $\theta = (f, p)$. Consider one-dimensional submodels of the form $f_k(y \mid w; t) = f_k(y \mid w)(1 + ts_{y,k}(y \mid w))$, and $p_k(w; t) = p_k(w)(1 + ts_{p,k}(x))$, where the functions $s_{y,k}, s_{p,k}$ are bounded and satisfy $\int s_{y,k}(y \mid w) f_k(y \mid w)dy = 0$ and $\sum_{k=0}^{K} p_k(w)s_{p,k}(w) = 0$ for all $w \in \mathcal{W}$. These conditions ensure that $f_k(y \mid w; t)$ and $p_k(w; t)$ are positive for $t$ small enough and that $\sum_{k=0}^{K} p_k(w; t) = \sum_{k=0}^{K} p_k(w) = 1$, so that the submodel is well-defined. The joint log-likelihood, conditional on the controls, is given by

$$\sum_{i=1}^{N}\sum_{k=0}^{K} \mathbb{1}\{D_i = k\}(\log f_k(Y_i \mid w_i; t) + \log p_k(w_i; t)).$$

The score at $t = 0$ is given by $\sum_{i=1}^{N} s(Y_i, D_i \mid w_i)$, with $s(Y_i, D_i \mid w_i) = \sum_{k=0}^{K} \mathbb{1}\{D_i = k\}(s_{y,k}(Y_i \mid w_i) + s_{p,k}(w_i))$.

In line with this result, we define the tangent space to consist of all functions $s(y, d \mid w) = \sum_{k=0}^{K} \mathbb{1}\{d = k\}(s_{y,k}(y \mid w) + s_{p,k}(w))$ such that $s_{y,k}$ and $s_{p,k}$ satisfy the above restrictions. Define the inner product on this space by the marginal expectation $\langle s_1, s_2\rangle = E[s_1(Y_i, D_i \mid W_i)s_2(Y_i, D_i \mid W_i)]$. We will prove the efficiency bound on the event $\mathcal{E}$ that (i) $\frac{1}{N}\sum_{i=1}^{N} E[s(Y_i, D_i \mid W_i)^2 \mid W_i] \to E[s(Y, D_i \mid W_i)^2]$; (ii) $N^{-1}\sum_i \lambda^{\mathrm{C}}(W_i) \to E[\lambda^{\mathrm{C}}(W_i)]$; (iii) $N^{-1}\sum_i \lambda^{\mathrm{C}}(W_i)\sum_{k=0}^{K} c_k E[Y_i(k)s_{y,k}(Y_i \mid W_i) \mid W_i] \to \sum_{k=0}^{K} c_k E[\lambda^{\mathrm{C}}(W_i)Y_i(k)s_{y,k}(Y_i(k) \mid W_i)]$; (iv) $N^{-1}\sum_{i=1}^{N} \lambda^{\mathrm{C}}(W_i)^2 \sum_{k,k'} \frac{s_{p,k}(W_i)}{p_k(W_i)} c_{k'}\mu_{k'}(W_i) \to E[\lambda^{\mathrm{C}}(W_i)^2 \sum_{k,k'} \frac{s_{p,k}(W_i)}{p_k(W_i)} c_{k'}\mu_{k'}(W_i)]$; (v) $N^{-1}\sum_{i=1}^{N} \lambda^{\mathrm{C}}(W_i)^2 \sum_{k=0}^{K} \frac{s_{p,k}(W_i)}{p_k(W_i)} \to E[\lambda^{\mathrm{C}}(W_i)^2 \sum_{k=0}^{K} \frac{s_{p,k}(W_i)}{p_k(W_i)}]$; and (vi) $\beta_{\lambda^C} \to \beta^*_{\lambda^C}$. Under the proposition assumptions and the law of large numbers, $\mathcal{E}$ is a probability-one set.

**Step 2** We verify that the conditions (3.7–3.12) of Theorem 3.1 in McNeney and Wellner (2000) hold on the set $\mathcal{E}$ conditional on the controls, with $\theta_N(s) = (f(\cdot \mid \cdot; 1/\sqrt{N}), p(\cdot; 1/\sqrt{N}))$. Let $\alpha_{Ni} = \prod_{k=0}^{K}(f_k(Y_i \mid w_i; 1/\sqrt{N})p_k(w_i; 1/\sqrt{N})/f_k(Y_i \mid w_i)p_k(w_i))^{\mathbb{1}\{D_i=k\}} = \prod_{k=0}^{K}((1 + N^{-1/2}s_{y,k}(Y_i \mid W_i; N^{-1/2}))(1 + N^{-1/2}s_{p,k}(w_i; 1/\sqrt{N})))^{\mathbb{1}\{D_i=k\}}$ denote the likelihood ratio associated with the $i$th observation. Since this is bounded by the boundedness of $s_{y,k}, s_{p,k}$, condition (3.7) holds. Also, since $(1+ts_{p,k})^{1/2}$ and $(1+ts_{y,k})^{1/2}$ are continuously differentiable for $t$ small enough, it follows from Lemma 7.6 in van der Vaart (1998) that the quadratic mean differentiability condition (3.8) holds. Since $s_k$ is bounded, the Lindeberg condition (3.9) also holds. Next, $\frac{1}{N}\sum_{i=1}^{N} E[s(Y_i, D_i \mid W_i)^2 \mid W_i]$ converges to $E[s(Y, D_i \mid W_i)^2] = \langle s, s\rangle$ on

$\mathcal{E}$ by assumption. Hence, conditions (3.10) and (3.11) also hold. Since the scores $\Delta_{N,s} = \frac{1}{\sqrt{N}}\sum_{i=1}^N s(Y_i, D_i \mid w_i)$ are exactly linear in $s$, condition (3.12) also holds. It follows that the model is LAN on $\mathcal{E}$.

**Step 3** Write the parameter of interest, $\beta_{\lambda^{\mathrm{C}}}$, as $\beta_N(\theta) = \sum_{i=1}^N \lambda^{\mathrm{C}}(w_i)\int y \sum_{k=0}^K c_k f_k(y \mid w_i)dy / \sum_{i=1}^N \lambda^{\mathrm{C}}(w_i)$, where $\lambda^{\mathrm{C}}(w_i) = 1/\sum_{k=0}^K p_k(w_i)^{-1}$. Letting $\dot{\beta}_N(\theta)$ denote the derivative of $\beta_N(\theta(\cdot \mid \cdot; t))$ at $t = 0$, we have

$$\sqrt{N}(\beta_N(\theta(\cdot \mid \cdot; 1/\sqrt{N})) - \beta_N(\theta)) = \dot{\beta}_N(\theta) + o(1).$$

Let $h(w) = \lambda^{\mathrm{C}}(w)\sum_{k=0}^K c_k \int y s_{y,k}(y \mid w) f_k(y \mid w)dy$, and $\tilde{h}(W_i) = \sum_{k'=0}^K c_{k'}\mu_{k'}(W_i) - \beta^*_{\lambda^C}$. The derivative may then be written as

$$\begin{aligned}
\dot{\beta}_N(\theta) &= \frac{1}{\sum_{i=1}^N \lambda^{\mathrm{C}}(w_i)} \sum_{i=1}^N \left( h(w_i) + \lambda^{\mathrm{C}}(w_i)^2 \sum_{k=0}^K \frac{s_{p,k}(w_i)}{p_k(w_i)} \left( \sum_{k'=0}^K c_{k'}\mu_{k'}(w_i) - \beta_N(\theta) \right) \right) \\
&\to \frac{1}{E[\lambda^{\mathrm{C}}(W_i)]} E\left[ h(W_i) + \lambda^{\mathrm{C}}(W_i)^2 \sum_{k=0}^K \frac{s_{p,k}(W_i)}{p_k(W_i)} \left( \sum_{k'=0}^K c_{k'}\mu_{k'}(W_i) - \beta^*_{\lambda^C} \right) \right] \\
&= \frac{1}{E[\lambda^{\mathrm{C}}(W_i)]} E\left[ \lambda^{\mathrm{C}}(W_i) \sum_{k=0}^K X_{ki} \left( c_k \frac{Y_i - \mu_k(W_i)}{p_k(W_i)} + \lambda^{\mathrm{C}}(W_i) \frac{\tilde{h}(W_i)}{p_k(W_i)^2} \right) s(Y_i, D_i \mid W_i) \right],
\end{aligned}$$

where the limit on the second line holds on the event $\mathcal{E}$, and the third line uses $E[X_{ki}(Y_i - \mu_k(W_i))s(Y_i, D_i \mid W_i) \mid W_i] = p_k(W_i)E[Y_i(k)s_{y,k}(Y_i(k) \mid W_i) \mid W_i]$ and $E[X_{ki}s(Y_i, D_i \mid W_i) \mid W_i] = p_k(W_i)s_{p,k}(W_i)$. Since for any function $a(W_i)$, $E[a(W_i)s(Y_i, D_i \mid W_i)] = 0$, subtracting $\frac{1}{E[\lambda^{\mathrm{C}}(W_i)]}\sum_{k=0}^K E[\lambda^{\mathrm{C}}(W_i)^2 \frac{\tilde{h}(W_i)}{p_k(W_i)} s(Y_i, D_i \mid W_i)] = 0$ from the preceding display implies $\sqrt{N}(\beta_N(\theta(\cdot \mid \cdot; 1/\sqrt{N})) - \beta_N(\theta)) = E[\psi(Y_i, D_i, W_i)s(Y_i, D_i \mid W_i)] + o(1)$, where

$$\psi(Y_i, D_i, W_i) = \sum_{k=0}^K X_{ki} \cdot \left( \frac{\lambda^{\mathrm{C}}(W_i)}{E[\lambda^{\mathrm{C}}(W_i)]} c_k \frac{Y_i - \mu_k(W_i)}{p_k(W_i)} + \frac{\lambda^{\mathrm{C}}(W_i)}{E[\lambda^{\mathrm{C}}(W_i)]} \tilde{h}(W_i) \left( \frac{\lambda^{\mathrm{C}}(W_i)}{p_k^2} - 1 \right) \right).$$

Observe that $\psi$ lies in the completion of the tangent space, with the expression in parentheses playing the role of $s_{y,k}(Y_i \mid W_i) + s_{p,k}(W_i)$. Hence, the semiparametric efficiency bound is given by $E[\psi^2]$, which yields the expression in the statement of the Proposition.

**Attainment of the bound** We derive the result in two steps. First, we show that

$$\sqrt{N}(\beta_{\lambda^{\mathrm{C}}} - \beta^*_{\lambda^C}) = \frac{1}{\sqrt{N}}\sum_{i=1}^N \psi^*(W_i) + o_p(1) \qquad \text{and} \qquad \psi^*(W_i) = \frac{\lambda^{\mathrm{C}}(W_i)}{E[\lambda^{\mathrm{C}}(W_i)]}(\tau(W_i) - \beta^*_{\lambda^{\mathrm{C}}}). \tag{31}$$

Second, we show that

$$\sqrt{N}(\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}} - \beta^*_{\lambda^C}) = \frac{1}{\sqrt{N}} \sum_{i=1}^{N} \psi(Y_i, D_i, W_i) + o_p(1), \tag{32}$$

where, letting $\epsilon_{ki} = Y_i - \mu_k(W_i)$,

$$\psi_k(Y_i, D_i, W_i) = \frac{\lambda^{\mathrm{C}}(W_i)}{E[\lambda^{\mathrm{C}}(W_i)]} \left( \frac{X_{ki}\epsilon_{ki}}{p_k(W_i)} - \frac{X_{0i}\epsilon_{0i}}{p_k(W_i)} + (\tau_k(W_i) - \beta^*_{\lambda^{\mathrm{C}},k})\lambda^{\mathrm{C}}(W_i) \sum_{k'} \frac{X_{k'i}}{p_{k'}(W_i)^2} \right).$$

Together, these results imply that the asymptotic variance of $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}}}$ as an estimator of $\beta_{\lambda^{\mathrm{C}}}$ is given by $\mathrm{var}(\psi - \psi^*)$, which coincides with the semiparametric efficiency bound.

Equation (31) follows directly under the assumptions of the proposition by the law of large numbers and the fact that the variance of $\lambda^{\mathrm{C}}(W_i)(\tau(W_i) - \beta^*_{\lambda^{\mathrm{C}}})$ is bounded. To show eq. (32), write $\hat{\beta}_{\hat{\lambda}^{\mathrm{C}},k} = \hat{\alpha}_k - \hat{\alpha}_0$, where $\hat{\alpha}$ is a two-step method of moments estimator based on the $(K+1)$ dimensional moment condition $E[m(Y_i, D_i, W_i, \alpha^*, p)] = 0$ with elements $m_k(Y_i, D_i, W_i, \alpha^*, p) = \lambda^{\mathrm{C}}(W_i)\frac{X_{ki}}{p_k(W_i)}(Y_i - \alpha^*_k)$, and $\alpha^*$ is a $(K+1)$ dimensional vector with elements $\alpha^*_k = E[\lambda^{\mathrm{C}}(W_i)\mu_k(W_i)]/E[\lambda^{\mathrm{C}}(W_i)]$.

Consider a one-dimensional path $F_t$ such that the distribution of the data is given by $F_0$. Let $p_{k,t}(W_i) = E_{F_t}[X_{ki} \mid W_i]$ denote the propensity score along this path. The derivative of $E[m_k(Y_i, D_i, W_i, \alpha^*, p_t)]$ with respect to $t$ evaluated at $t = 0$ is

$$E\left[ \frac{\lambda^{\mathrm{C}}(W_i)X_{ki}}{p_k(W_i)}(Y_i - \alpha^*_k) \left( \lambda^{\mathrm{C}}(W_i) \sum_{k'=0}^{K} \frac{\dot{p}_{k'}(W_i)}{p_{k'}(W_i)^2} - \frac{\dot{p}_k(W_i)}{p_k(W_i)} \right) \right] = \sum_{k'=0}^{K} E[\delta_{kk'}(W_i)'\dot{p}_{k'}(W_i)],$$

where $\dot{p}_k$ denotes the derivative of $p_{k,t}$ at $t = 0$, and

$$\delta_{k,k'}(W_i) = \lambda^{\mathrm{C}}(W_i)(\mu_k(W_i) - \alpha^*_k) \left( \frac{\lambda^{\mathrm{C}}(W_i)}{p_{k'}(W_i)^2} - \frac{\mathbb{1}\{k = k'\}}{p_k(W_i)} \right).$$

Under the assumptions of the proposition, $\delta_{k,k'} \in \mathcal{G}$. It therefore follows by Proposition 4 in Newey (1994) that the influence function for $\hat{\alpha}_k$ is given by

$$\begin{aligned} &\frac{1}{E[\lambda^{\mathrm{C}}(W_i)]} \left( \frac{\lambda^{\mathrm{C}}(W_i)X_{ki}}{p_k(W_i)}(Y_i - \alpha^*_k) + \sum_{k'} \delta_{kk'}(W_i)(X_{k'i} - p_{k'}(W_i)) \right) \\ &\qquad = \frac{\lambda^{\mathrm{C}}(W_i)}{E[\lambda^{\mathrm{C}}(W_i)]} \left( \frac{X_{ki}\epsilon_{ki}}{p_k(W_i)} + (\mu_k(W_i) - \alpha^*_k)\lambda^{\mathrm{C}}(W_i) \sum_{k'} \frac{X_{k'i}}{p_{k'}(W_i)^2} \right), \end{aligned}$$

which yields eq. (32).