Misspecification and the Propensity Score: The Possibility of Overadjustment *

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Abstract

The popularity of propensity score matching has given rise to a robust, sometimes informal, debate concerning the number of pre-treatment variables that should be included in the propensity score. The standard practice when estimating a treatment effect is to include all available pre-treatment variables, and we demonstrate that this approach is not always optimal when the goal is bias reduction. We characterize the conditions under which including an additional relevant variable in the propensity score increases the bias on the effect of interest across a variety of different implementations of the propensity score methodology. Moreover, we find that balance tests and sensitivity analysis provide limited protection against overadjustment.

Keywords: Matching; Omitted variable bias; Conditioning

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Introduction

A recent debate featured Shrier (2008), Pearl (2009), and Sjölander (2009) responding to a paper by Rubin (2007). A chief concern of the participants was the question of whether conditioning on a new covariate can only decrease confounding bias when estimating a treatment effect. The three responses note an instance when the use of propensity score methods would increase the bias on estimates of the average treatment effect (ATE) above that of an unadjusted comparison between treated and untreated observations (Pearl, 2009, 1415). Rubin (2009) responds by arguing that the example amounts to a mere mathematical curiosity.[1]

The debate takes place within the context of the potential outcomes framework. Let Y_{i1} be the value of the response variable when unit i receives the treatment ($T_i = 1$), and let Y_{i0} be the value of the response variable when unit i does not receive the treatment ($T_i = 0$). Y_{i1} and Y_{i0} are potential outcomes as they cannot be observed simultaneously for unit i. The observed outcome is $Y_i = T_i Y_{i1} + (1 - T_i)Y_{i0}$, and the effect of the treatment for unit i is $\tau_i = Y_{i1} - Y_{i0}$. As the individual-level causal effects generally cannot be estimated, interest centers on the ATE:

$$E[\tau_i] = E[Y_{i1} - Y_{i0}].^2 \tag{1}$$

Equation (1) is estimable under the the stable unit treatment value assumption (SUTVA), which states if unit i receives treatment j, the observed value of Y is Y_{ij} (no interference between units and no variation in treatment) (Rubin, 1980, 591).

In observational studies, focus moves to the ATE conditional on a set of observed pretreatment covariates $\mathbf{X}$:

$$\tau = E[Y_i|\mathbf{X}_i, T_i = 1] - E[Y_i|\mathbf{X}_i, T_i = 0]. \tag{2}$$

Equation (2) can be used to consistently estimate the ATE when treatment is said to be strongly ignorable. That is, the potential outcomes and the treatment must be independent within levels of the covariates,

$$\{Y_{i1}, Y_{i0}\} \perp\!\!\!\perp T_i|\mathbf{X}_i, \tag{3}$$

and for every value of $\mathbf{X}$ there are treated and nontreated cases, $0 < \Pr(T_i = 1|\mathbf{X}_i) < 1$, for all $\mathbf{X}_i$. If unobserved confounding variables exist that are not included in $\mathbf{X}$, as is likely in an observational study, any estimator of the ATE will be biased.

Rosenbaum and Rubin (1983) show that if the set of covariates is of high dimension, thereby

[1] A similar debate began between Judea Pearl and Andrew Gelman on the latter's weblog (http://stat.columbia.edu/∼gelman/blog/). This exchange attracted the notice of other prominent Bayesian statisticians such as Philip Dawid.

[2] We will also be interested in the average treatment effect on the treated (ATT): $E[Y_{i1} - Y_{i0}|T_i = 1]$.

creating a problem for techniques such as matching, one can condition on the propensity score, which is the probability of assignment to treatment, conditional on the set of covariates, $e(\mathbf{X}_i) = \Pr(T_i = 1|\mathbf{X}_i)$. Free of behavioral assumptions, the propensity score is generally estimated with a simple logit model:

$$\Pr(T_i = 1|\mathbf{X}_i) = \frac{e^{\boldsymbol{\beta} h(\mathbf{X}_i)}}{1 + e^{\boldsymbol{\beta} h(\mathbf{X}_i)}},$$

where $h(\mathbf{X})$ comprises linear and higher order terms of the pretreatment covariates, and $\boldsymbol{\beta}$ is the set of parameters to be estimated.

The goals of this paper are threefold. First, assuming that we do not have access to the full list of covariates necessary for strong ignorability to hold, we characterize the conditions under which including an additional covariate in the propensity score, $e(\mathbf{X}_i)$, increases the bias on an estimator of the ATE. We find that these circumstances are not unusual. Second, we characterize these conditions across a variety of different implementations of the propensity score methodology— nearest neighbor matching, matching within calipers, stratification by blocks, weighting, and regression—to assess which, if any, of these implementations helps mitigate the problem. Our results show that differences across techniques are minor. Third, we assess the ability of post-estimation tools such as balance tests and sensitivity analysis to alert us to the problem of overconditioning. The results are not encouraging.

Our findings lend little credence to the claim that a researcher should condition on all available pretreatment covariates. Which variables should be included in a data analysis depends, as we demonstrate, on a number of factors that vary from situation to situation. In choosing covariates, researchers need to rely on theory, judgment, and common sense. The paper ends with a discussion of how our results can be helpful to applied researchers.

The debate and previous research

In responding to a paper by Rubin (2007) on the design of observational studies, Shrier (2008) raises the issue of selection bias caused by controlling for a covariate that is the common effect of two independent variables. He is interested in "M-structures," where a treatment X causes an outcome Y, an unmeasured covariate, U_1, causes both X and a measured covariate Z, and a second unmeasured covariate, U_2, causes both the measured covariate Z and Y (see Figure 1). In this situation, if a researcher controls for the measured covariate Z, a spurious dependence between X and Y is created that would bias the estimate of the causal effect of X on Y (because the value of Z predicts the value of $[X, Y]$).

Pearl (2009, 1415) expands on Shrier's (2008) point and argues that the use of propensity score techniques increases the bias on the estimated ATE whenever

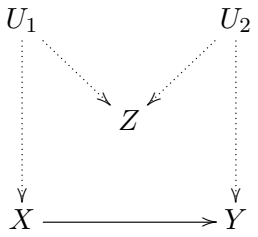


Figure 1: A causal directed acyclic graph of an M-structure. The dashed lines denote the effects of unmeasured covariates.

> ...treatment is strongly ignorable to begin with and becomes non-ignorable at some levels of e_i. In other words, although treated and untreated units are balanced in each stratum of e_i, the balance only holds relative to the covariates measured; unobserved confounders may be highly unbalanced in each stratum of e_i, capable of producing significant bias.

That is, the propensity score balances treated and untreated observations relative only to observed covariates. A new association is introduced between treatment and outcome by conditioning on a variable that is not causally related to either, but is an indicator of unobserved factors that are not balanced. This new association may increase or decrease the bias on the estimate of the ATE. Pearl (2009, 1416) concludes that the effectiveness of propensity score techniques depends "critically on the choice of covariates, and that choice cannot be left to guesswork."

Rubin's (2009) response (and to a lesser extent Gelman's blog response[3]) to these claims is that not controlling for an observed covariate is bad practical advice "in all but the most unusual circumstances." Furthermore, he argues that even if one were to condition on Z in Figure 1, the result would be inefficient, but not biased. In the end, he argues that not conditioning on an observed covariate because of fears of increasing bias "is neither Bayesian nor scientifically sound but rather it is distinctly frequentist and nonscientific ad hocery" (Rubin, 2009, 1421).

Our interest lies in assessing the risk of increasing bias through conditioning, and whether the choice of propensity score techniques makes a difference. Numerous articles have been published on misspecification of the propensity score, and we touch only on those studies that are particularly relevant to the discussion. Drake (1993), for example, finds that there is little difference between propensity score methods and prognostic (regression) models with regard to omitted confounders. The biases for both techniques are "large and of the same magnitude" (Drake, 1993, 1231). Additionally, she finds that misspecifications of the propensity score in terms of functional form have much smaller biases than similar misspecifications of the response model.

The simulation design of Augurzky and Schmidt (2001) includes three sets of variables:

[3] http://www.stat.columbia.edu/c̃ook/movabletype/archives/2009/07/disputes_about.html

- **Z**, which strongly influence exposure to treatment, but do not or only only weakly determine the outcome;
- **X**, which includes the strongest predictors of both the treatment and the outcome (the most effort should be spent balancing these confounders);
- **W**, which influence the outcome, but are irrelevant to exposure.

Their results indicate that including **Z** and **W** in the propensity score has two effects. One, the inclusion of these variables balances **Z** at the expense of the variables most relevant to exposure and outcome, and two, unnecessary effort is used to remove small imbalances in **W** (Augurzky and Schmidt, 2001, 26). They find that leaving **Z** and **W** out of the propensity score equation produces average treatment effect estimates that are often better in terms of root mean squared error than including all the covariates. The authors recommend including only highly significant variables in the propensity score equation.

Brookhard et al. (2006) present two simulations that pick up on some of the same design features as Augurzky and Schmidt (2001). In their first simulation, they include three different types of covariates: one related to both the outcome and exposure X_1; one related to the outcome, but not the exposure X_2; and one related to the exposure, but not the outcome X_3. They find that the model that best predicts exposure does not yield the optimal propensity score model in terms of MSE; the optimal model included X_1 and X_2, but not X_3 (Brookhard et al., 2006, 6). Thus, one should include in the selection equation variables that are thought to be related to the outcome, whether or not they are related to the exposure. They also found that adding variables to the propensity score model that are unrelated to the outcome but related to the exposure increases the variance of an estimated exposure effect without decreasing its bias (Brookhard et al., 2006, 7).

In their second simulation, Brookhart *et al.* (2006) look at the addition of a covariate to a propensity score model when varying the strength of the covariate-outcome and covariate-exposure relations (Brookhard et al., 2006, 3). They find that in small studies situations exist where it would be better, in terms of MSE, to exclude a true confounder from the propensity score model (Brookhard et al., 2006, 7).

Millimet and Tchernis (2009) report a simulation that focuses on, essentially, the functional form of the propensity score. In particular, they look at the exclusion of relevant higher-order terms and the inclusion of irrelevant higher-order terms. Their results suggest that overfitting the propensity score model results in greater efficiency, and in the other cases, overfitting does no worse than the correctly specified model. They conclude that the penalty for overfitting is minimal. The experimental evidence, therefore, is mixed.

The experiments in the present paper are closely related to the experiments performed in Clarke (2005) and Clarke (2009). In those papers, Clarke revisits the omitted variable bias result familiar to most applied researchers. He assumes that the true specification is

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}\boldsymbol{\gamma} + \mathbf{W}\boldsymbol{\delta} + \boldsymbol{\epsilon},\ \boldsymbol{\epsilon} \sim N(\mathbf{0}, \sigma^2\mathbf{I}),$$

and then considers the following two misspecified models:

$$\begin{aligned} \mathbf{y} &= \mathbf{X}\boldsymbol{\beta} + \boldsymbol{\epsilon}_1 \\ \mathbf{y} &= \mathbf{X}\boldsymbol{\beta} + \mathbf{Z}\boldsymbol{\gamma} + \boldsymbol{\epsilon}_2 \end{aligned}$$

He finds that, in some circumstances, the inclusion of the additional covariates, $\mathbf{Z}$, increases the bias on the coefficient of interest, $\boldsymbol{\beta}$. Clarke (2009) extends the result to generalized linear models.

Motivating Example

We begin with a simple example that illustrates how conditioning on additional information may increase the bias of a treatment effect estimate. In the example, treatment assignment T_i and the observed outcome Y_i are functions of two covariates, W_i and Z_i, of which only W_i is observed by the analyst. We show that the naive difference of means estimator of the average treatment effect may be less biased than one that conditions on all available information, namely W_i.

The data-generating process of the example is as follows. The covariates W_i and Z_i are independent Bernoulli(0.5) random variables. The value of each potential outcome is increasing in W_i and decreasing in Z_i,

$$\begin{aligned} Y_{i0} &= 1 + W_i - 2Z_i, \\ Y_{i1} &= 2 + W_i - 2Z_i. \end{aligned}$$

From the above expressions, it is immediate that the true value of the average treatment effect, our estimand of interest, is $\tau = E[Y_{i1} - Y_{i0}] = 1$. The probability of receiving treatment is increasing in both covariates,

$$\Pr(T_i = 1 \,|\, W_i, Z_i) = \frac{1}{4} + \frac{1}{4}W_i + \frac{1}{4}Z_i.$$

This setup can be summarized by a pair of $2 \times 2 \times 2$ tables:

In this setup, treatment assignment is strongly ignorable conditional on W_i and Z_i. This allows us to write

$$\tau = E_{(W_i,Z_i)}\left[E[Y_{i1} \,|\, W_i, Z_i, T_i = 1] - E[Y_{i0} \,|\, W_i, Z_i, T_i = 0]\right], \tag{4}$$

where $E_{(W_i,Z_i)}$ denotes expectation with respect to the distribution of the covariates. If both covariates were observed, the average treatment effect could be estimated consistently via subclassification

	$T_i = 0$		$T_i = 1$	
	$Z_i = 0$	$Z_i = 1$	$Z_i = 0$	$Z_i = 1$
$W_i = 0$	1	-1	2	0
$W_i = 1$	2	0	3	1

(a) The observed value Y_i as a function of the covariates and treatment assignment.

	$T_i = 0$		$T_i = 1$	
	$Z_i = 0$	$Z_i = 1$	$Z_i = 0$	$Z_i = 1$
$W_i = 0$	$\frac{3}{16}$	$\frac{2}{16}$	$\frac{1}{16}$	$\frac{2}{16}$
$W_i = 1$	$\frac{2}{16}$	$\frac{1}{16}$	$\frac{2}{16}$	$\frac{3}{16}$

(b) The joint distribution of the covariates and treatment assignment, $\Pr(W_i, Z_i, T_i)$.

Table 1: Summary of the data-generating process for the motivating example.

(Rosenbaum and Rubin, 1983, Corollary 4.2):

$$\hat{\tau} = \frac{1}{N} \sum_{w=0}^{1} \sum_{z=0}^{1} \sum_{i=1}^{N} \delta_i(w, z)[Y_i T_i - Y_i(1 - T_i)],$$

where $\delta_i(w, z)$ is an indicator for $(W_i, Z_i) = (w, z)$. However, this first-best solution is not available because Z_i is unobserved. If one followed the standard advice of conditioning on all covariates, and subclassified just on W_i, the resulting estimator would have asymptotic mean

$$\begin{aligned}
&\sum_{w=0}^{1} \Pr(W_i = w) \left[E[Y_{i1} \,|\, T_i = 1, W_i = w] - E[Y_{i0} \,|\, T_i = 0, W_i = w]\right] \\
&\qquad = \frac{1}{2}\left(\frac{2}{3} - \frac{1}{5}\right) + \frac{1}{2}\left(\frac{9}{5} - \frac{4}{3}\right) \\
&\qquad = \frac{7}{15}.
\end{aligned}$$

Because the true treatment effect is $\tau = 1$, the asymptotic bias of this estimator is $-\frac{8}{15}$. Now consider estimating the average treatment effect via a naive difference of means in Y_i between the observed treated and untreated groups. The mean of this estimator is

$$E[Y_{i1} \,|\, T_i = 1] - E[Y_{i0} \,|\, T_i = 0] = \frac{11}{8} - \frac{5}{8} = \frac{3}{4},$$

giving it a bias of $-\frac{1}{4}$. Although both estimators are biased, the magnitude of the bias is lower under the one that does not condition on all available covariates.

The reason it is not optimal to condition on W_i in this scenario is that the unobserved variable

Z_i has countervailing effects. Recall that W_i affects both treatment assignment and the response positively. Therefore, failure to adjust for W_i causes the estimated average treatment effect to be biased upward. However, Z_i has a positive effect on treatment assignment and a negative effect on the response, so its exclusion causes the estimate to be biased downward. Because Z_i has a stronger effect on the response, the entirely unadjusted estimate of the treatment effect is biased downward. Balancing on W_i lowers the estimated treatment effect, which increases the magnitude of the bias if Z_i cannot also be balanced on. As such, balancing on a covariate that has countervailing effects with an unobserved confounder may actually worsen the quality of a treatment effect estimate. In the Monte Carlo simulations described in the following sections, we find that this is often the case.

Design of the Experiments

We now turn to a series of Monte Carlo experiments illustrating that conditioning on all observed variables may be suboptimal if some confounding variables are unobserved. The results of these simulations show that the result of our motivating example—that conditioning on all available pre-treatment variables may be suboptimal in the presence of unobserved confounders—also holds in a less stylized setting. In each simulation, we compare the performance of various ATE estimators (described below) under two specifications: when conditioning on all available covariates, and when conditioning on all except a single variable W_i. Our goal is to find sets of parameters, if any, under which the magnitude of the bias is lower when W_i is excluded from the specification.

Experiment 1: linear specification

In our baseline experiment, there are three covariates affecting treatment assignment and the outcome of interest. Two of these are observed, $X_i \sim N(1,1)$ and $W_i \sim N(1,1)$, while $Z_i \sim N(2,1)$ is unobserved to the analyst. In this set of simulations, the equations describing the data-generating process for the potential outcomes and the log odds of treatment assignment are linear in all covariates.[4] The equations describing the data-generating process are as follows:

$$\underline{\text{Outcome in treated state}}$$
$$Y_{i1} = \beta_{10} + \beta_{11}X_i + \beta_{12}Z_i + \beta_{13}W_i + \epsilon_{i1}$$
$$\underline{\text{Outcome in untreated state}}$$
$$Y_{i0} = \beta_{00} + \beta_{01}X_i + \beta_{02}Z_i + \beta_{13}W_i + \epsilon_{i0}$$
$$\underline{\text{Latent index function}}$$
$$T_i^* = \gamma_0 + \gamma_1 X_i + \gamma_2 Z_i + \gamma_3 W_i + u_i$$
$$\underline{\text{Treatment indicator}}$$
$$T_i = I(T_i^* > 0), \text{ where } I(\cdot) \text{ is the indicator function}$$

[4] We consider multiplicative and quadratic terms in experiment 2.

There are three moving parts in this set of simulations. The first is the coefficient on W_i in the propensity score equation, which varies between -0.5 and 0.5, $\gamma_3 \in \{-0.5, -0.25, 0, 0.25, 0.5\}$. The second is the coefficient on W_i in the outcome equations, which varies between -2 and 2, $\beta_{13} \in \{-2, 0, 2\}$. The third is the canonical correlation between X_i and $\mathbf{Z}_i = (W_i, Z_i)$, $cc_{XZ} \in \{0.2, 0.5, 0.8\}$. The sample size is $N = 1{,}000$ in each iteration. The error terms, ϵ_0 and ϵ_1, are normally distributed with mean 0 and variance 1 and are correlated at 0.25.[5] The error term u in the propensity score equation has a logistic distribution. The intercept γ_0 is chosen so that 25% of the observations are in the treatment group. The remaining coefficients are set at reasonable values.[6] We performed 500 replications of the experiment per combination of the three moving parameters, γ_3, β_{13}, and cc_{XZ}. For each combination, we confirmed that the estimators we examine recover the treatment effect without bias when all three covariates are included in the propensity score specification. The goal of the experiment is to compare the bias of each estimator when only X_i is included to the bias when both X_i and W_i are included.

An easy way to understand the various data generating processes (DGPs) used in the experiment is to look at the directed acyclic graphs (DAGs) in Figures 7–9 (see the Appendix). In each graph, an arrow indicates variables that affect one another; dashed arrows indicate unobserved variables. In Figure 7, W_i affects both the treatment T_i and the outcome Y_i. In Figure 8, W_i is related only to the treatment, which corresponds to the case where $\beta_{13} = 0$. In Figure 9, W_i is related only to the outcome, which corresponds to the case where $\gamma_3 = 0$.

The key to this experiment is the difference in the estimated ATE between the two misspecified models depicted in Figures 10 and 11. In the first misspecified model (Figure 10), both Z_i and W_i are unobserved. In the second misspecified model (Figure 11), W_i is included in the propensity score while Z_i remains unobserved. The question is whether the bias on the ATE in the second misspecified model is ever greater than the bias on the ATE in the first. If so, then there are cases where controlling for all observed covariates is incorrect advice.

Experiment 2: nonlinear specification

In the second experiment, we introduce multiplicative and quadratic terms into the outcome equations.

[5] We also ran the experiment with ϵ_0 and ϵ_1 uncorrelated; the results were essentially identical.

[6] $\gamma_1 = 0.5$, $\gamma_2 = 1$, $\beta_{00} = 1$, $\beta_{01} = 1$, $\beta_{02} = 1$, $\beta_{10} = 2.5$, $\beta_{11} = 1.5$, and $\beta_{12} = 0.5$. Extensive robustness checks were performed; see the Results section.

Outcome in treated state

$Y_{i1} = \beta_{10} + \beta_{11}X_i + \beta_{12}X_i^2 + \beta_{13}Z_i + \beta_{14}Z_i^2 + \beta_{15}X_iZ_i + \beta_{16}W_i + \epsilon_{i1}$

Outcome in untreated state

$Y_{i0} = \beta_{00} + \beta_{01}X_i + \beta_{02}X_i^2 + \beta_{03}Z_i + \beta_{04}Z_i^2 + \beta_{05}X_iZ_i + \beta_{16}W_i + \epsilon_{i0}$

Latent index function

$T_i^* = \gamma_0 + \gamma_1 X_i + \gamma_2 Z_i + \gamma_3 W_i + u_i$

Treatment indicator

$T_i = I(T_i^* > 0)$, where $I(\cdot)$ is the indicator function

Other than the inclusion of higher-order terms in the outcome equations, all elements of the simulation remain the same as in experiment 1. The moving parts of the experiment are still the coefficient on W_i in the propensity score, $\gamma_3 \in \{-0.5, -0.25, 0, 0.25, 0.5\}$; the coefficient on W_i in the outcome equations, $\beta_{16} \in \{-2, 0, 2\}$; and the canonical correlation between X_i and $\mathbf{Z}_i$, $cc_{XZ} \in \{0.2, 0.5, 0.8\}$. The error distributions are the same as in experiment 1, and the non-moving parameters are set at reasonable values.[7] We again performed 500 replications of the experiment per parameter combination.

Experiment 3: covariates from real data

In the first two experiments, all of the variables are drawn from a normal distribution—an ideal case for common matching methods (Rubin and Thomas, 1992), but one that hardly resembles a typical data set. To assess the relative performance of the propensity score estimators in a more realistic setting, we ran a third experiment with LaLonde's (1986) widely used data set.[8] As in the prior experiments, the treatment variable T_i and the potential outcomes (Y_{i0}, Y_{i1}) are simulated, as is the "new variable" W_i. However, all of the other observed and unobserved covariates are taken from LaLonde's data. The variables we use are *Age* (in years), *Education* (in years), ln(1+*Earnings 1974*) (in logged USD), and ln(1+*Earnings 1975*) (in logged USD). The last of these is assumed to be unobserved to the analyst, and thus always omitted from the propensity score specification, while the other three are always included. In each trial, we generate a variable W_i (mean 1, variance 1) that is correlated with the omitted variable, ln(1+*Earnings 1975*), at the level $\rho \in \{-0.75, -0.25, 0, 0.25, 0.75\}$. As in the previous experiments, we compare the bias in the estimated ATE when all observed covariates are included in the propensity score specification to the bias when all but W_i are included. The data generating process is as follows:

[7] $\gamma_1 = 0.5$, $\gamma_2 = 1$, $\beta_{00} = 1$, $\beta_{01} = 1$, $\beta_{02} = -2$, $\beta_{03} = 1$, $\beta_{04} = 0.2$, $\beta_{05} = 0.25$, $\beta_{10} = 2.5$, $\beta_{11} = 1.5$, $\beta_{12} = -1$, $\beta_{13} = 0.5$, $\beta_{14} = 0.4$, and $\beta_{15} = 0.5$.

[8] Following Dehejia and Wahba (1999), we use the subset of male participants for which 1974 earnings are available, giving us 445 observations.

Outcome in treated state

$Y_{i1} = 2 + 0.1\, Age_i + 0.15\, Educ_i + 0.4\, \ln(1 + Earn74)_i + 0.4\, \ln(1 + Earn75)_i + \beta W_i + \epsilon_{i1}$

Outcome in untreated state

$Y_{i0} = 1 + 0.05\, Age_i + 0.1\, Educ_i + 0.2\, \ln(1 + Earn74)_i + 0.2\, \ln(1 + Earn75)_i + \beta W_i + \epsilon_{i0}$

Latent index function

$T_i^* = \gamma_0 + 0.03\, Age_i + 0.14\, Educ_i + 0.12\, \ln(1 + Earn74)_i + 0.12\, \ln(1 + Earn75)_i + \gamma_1 W_i + u_i$

Treatment indicator

$T_i = I(T_i^* > 0)$, where $I(\cdot)$ is the indicator function

The other moving parts are once again the coefficient of W_i in the true propensity score, $\gamma_1 \in \{-0.3, -0.15, 0, 0.15, 0.3\}$, and its coefficient in the outcome equation for treated units, $\beta \in \{-0.5, -0.25, 0, 0.25, 0.5\}$. The intercept in the propensity score equation, γ_0, is chosen so that 25% of observations are treated. The error terms in the outcome equations, ϵ_{i0} and ϵ_{i1}, have a standard deviation of 3 and are correlated at 0.25. As in the prior experiments, our interest focuses on whether including W_i in the propensity score specification decreases the bias and mean squared error of various estimators of the average treatment effect.

Techniques

Because there are continuous covariates in each experiment, we focus on propensity score estimators of the average treatment effect. Each of the five estimators that we consider involves obtaining a first-stage estimate of the propensity score, $e_i(\mathbf{X}_i) = \Pr(T_i = 1 \,|\, \mathbf{X}_i)$, where $\mathbf{X}_i$ denotes the full set of confounding variables. In each Monte Carlo iteration, we obtain two distinct propensity score estimates, both using the fitted values from a logistic regression of T_i on observed covariates. The first estimate, $\hat{e}_{XWi}$, is obtained from a specification that includes all of the observed covariates. For experiments 1 and 2, this is

$$\hat{e}_{XWi} = \Lambda(\hat{\gamma}_0^{XW} + \hat{\gamma}_1^{XW} X_i + \hat{\gamma}_2^{XW} W_i),$$

where $\hat{\gamma}^{XW}$ represents the logistic regression coefficients. The second estimate, $\hat{e}_{Xi}$, comes from a specification that includes all of the observed variables except W_i. For experiments 1 and 2, this is

$$\hat{e}_{Xi} = \Lambda(\hat{\gamma}_0^{X} + \hat{\gamma}_1^{X} X_i).$$

After obtaining the two sets of propensity score estimates, we plug them into each of the following ATE estimators:[9]

[9] We implement the first two estimators using Sekhon's (2011) R package `Matching`.

- Nearest-neighbor matching (with replacement): In this method, all the units are ordered randomly, and the first treated unit is matched with the control unit having the nearest propensity score. The first treated unit is then removed from the data set while its matched control unit is kept to be used in future matches. The process is repeated for the second treated unit and so on. The causal effect is estimated by averaging the outcome differences between the matched treatment and control groups. Following Rosenbaum and Rubin (1985, 36), we use the linear predictor in place of the estimated probability to avoid compression of the propensity scores near 0 and 1.

- Caliper matching (with replacement): In this method, all the units are ordered randomly, and for the first treated unit, the control units with propensity scores (again the linear predictor) within a specified distance of the treated unit are gathered (0.25 standard deviations of the linear predictor), and the treated unit is matched with the closest control unit within the group in terms of Mahalanobis distance. The first treated unit is then removed from the data set while its matched control unit is kept to be used in future matches. The process is repeated for the second treated unit and so on. Again, the causal effect is estimated by averaging the outcome differences between the matched treatment and control groups.

- Blocked matching: In this method, all the observations are divided into blocks, or strata, based on the value of the propensity score, which should be approximately constant within the strata. (We use deciles.) The difference of means between the treated and the control is estimated within each block, and the estimated causal effect is the weighted mean of these differences.

- Weighting: Wooldridge (2002, 616) provides a consistent estimator of the ATE based on simple weighting that is identical to the Horvitz-Thompson estimator (Horvitz and Thompson, 1952),

$$\begin{aligned} \hat{\tau} &= N^{-1} \sum_{i=1}^{N} \left\{ \frac{[T_i - \hat{e}_i] Y_i}{\hat{e}_i (1 - \hat{e}_i)] } \right\} \\ &= N^{-1} \sum_{i=1}^{N} \left\{ \frac{T_i Y_i}{\hat{e}_i} - \frac{(1 - T_i) Y_i}{1 - \hat{e}_i} \right\} \end{aligned}$$

  where $\hat{e}_i$ is the estimated propensity score.

- Covariance adjustment: In this method, the ATE is estimated from a regression of the response variable on a constant, a variable denoting treatment assignment, the estimated propensity score, and a multiplicative term comprising the treatment variable and deviations

about the sample mean of the estimated propensity score. The regression equation is

$$Y_i = \beta_0 + \beta_1 T_i + \beta_2 \hat{e}_i + \beta_3 T_i \left[\hat{e}_i - \hat{\mu}_e\right] + \epsilon_i,$$

where $\hat{\mu}_e$ is the sample average of the estimated propensity score, $\hat{e}_i$. Rosenbaum and Rubin (1983, 46) demonstrate, assuming that $E[Y_{i0} \mid e(\mathbf{X}_i)]$ and $E[Y_{i1} \mid e(\mathbf{X}_i)]$ are linear in $e(\mathbf{X}_i)$, that $\hat{\beta}_3$ is a consistent estimator of the ATE:

In each Monte Carlo iteration, we yield ten estimates of the ATE: one for each method using the propensity score specification with all observed covariates, and one for each method using the propensity score specification from which W_i is excluded. We write $\hat{\tau}_{XW}$ to denote an estimator that uses the propensity score specification with W_i included and $\hat{\tau}_X$ for one that uses the specification where W_i is excluded. Our interest is in whether there are parameter combinations under which $\hat{\tau}_{XW}$ is more biased than $\hat{\tau}_X$ (for some or all of the five particular procedures), meaning it is better not to condition on all observed covariates.

Results

Experiment 1: linear specification

We first examine the results of the experiment in which all covariates enter the outcome equations linearly.[10] Figure 2 summarizes the main findings. Each point in a subplot represents the difference in absolute bias on the estimated ATE between the model that included W in the propensity score and the model that did not, as a function of γ_3 (W's coefficient in the true propensity score equation). Each subplot represents one combination of the other varying parameters: cc_{XZ}, the canonical correlation between $\mathbf{X}$ and $\mathbf{Z}$, and β_{13}, the coefficient on W in the outcome equation. Each line within a subplot represents the quantity of interest estimated with a specific method. Positive values indicate that the absolute value of the bias is greater when W is included in the estimated propensity score equation than when it is not. For example, the subplot in the top left corner presents the difference in absolute bias when $\beta_{13} = -2$ and $cc_{XZ} = 0.2$. In this example, intermediate values of γ_3 result in the bias increasing when W is included in the estimated propensity score, regardless of which method is used to estimate the ATE. We ran a similar set of experiments in which the estimand was the average treatment effect on the treated (ATT), and obtained substantively identical results.

Two observations are apparent. First, the absolute bias on the estimated ATE increases when W is included in the estimated propensity score equation for several parameter combinations. That is, it is *not* always optimal to condition on all available pre-treatment variables. Specifically, we find that when β_{13} is positive but γ_3 is equal to -0.5, it is often worse to include W in the propensity

[10] A replication file is available upon request.

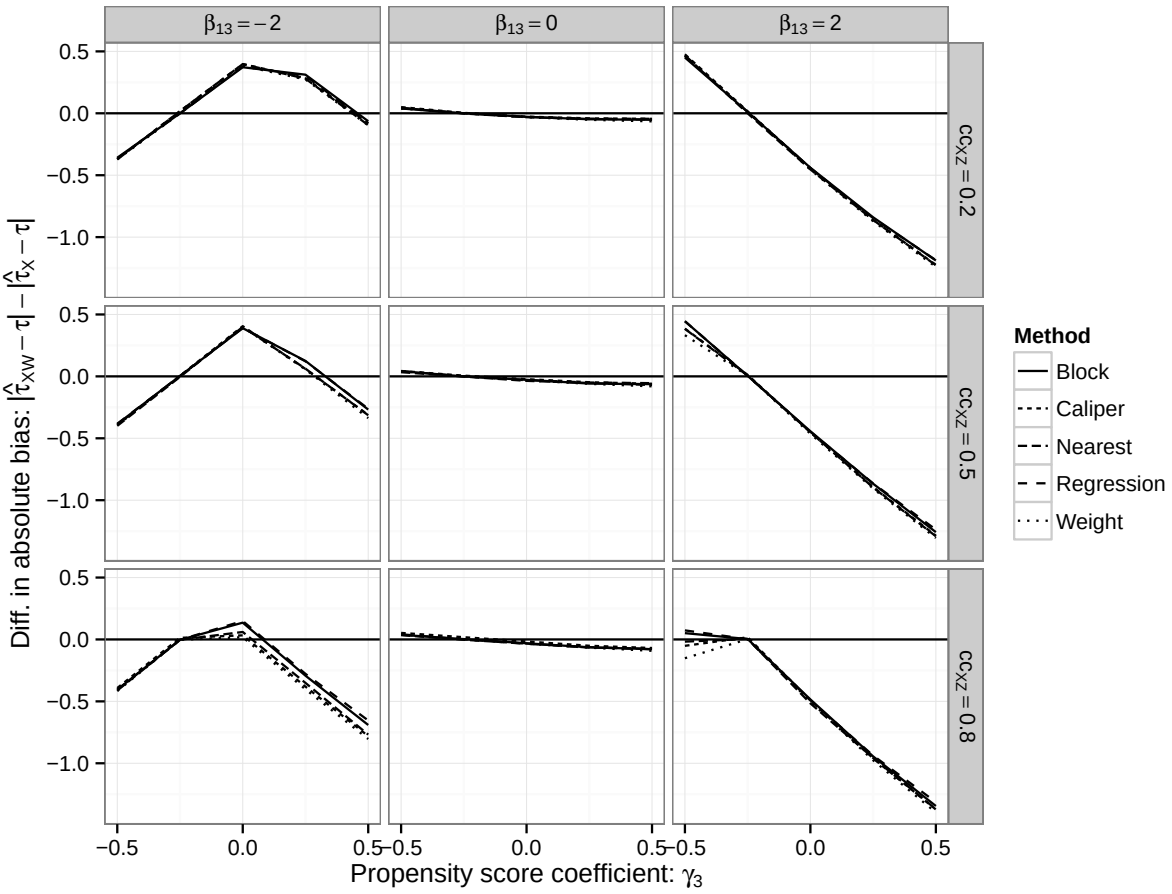
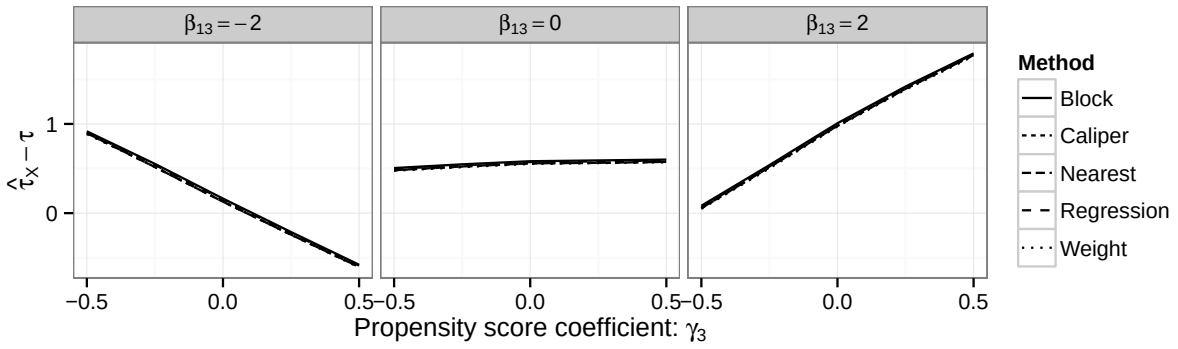


Figure 2: Difference in absolute bias with linear outcome equations.

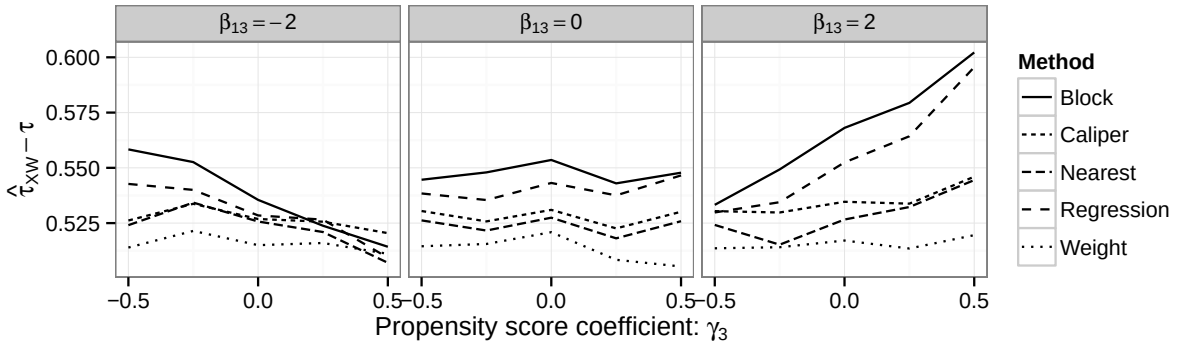
score. The same is true if W's effect on the outcome is negative but its effect on the probability of treatment is weakly positive ($\gamma_3 = 0.25$). This pattern is stronger when the canonical correlation between X and Z is low. Notice also that for all values of the canonical correlation, whenever there is no effect of W on the outcome, we see that there is not much difference between the bias when the propensity score model includes the variable and when it does not. If anything, when γ_3 is negative and $\beta_{13} = 0$, it is slightly worse to include the variable.

The second observation is that the five estimation procedures generally agree regarding whether including W in the propensity score equation worsens the bias on the ATE. Figure 3 shows the bias when W is excluded from the estimated propensity score and when it is not for a low canonical correlation between X and Z. When W is not included, all five methods return nearly identical estimates but the variation between methods is greater when W is included.[11] It appears, that weighting and nearest neighbor matching perform slightly better at reducing the bias when W is

[11]This is true accounting for the fact that the figure has different y scales for both specifications of the propensity score.



(a) Bias when W is excluded.



(b) Bias when W is included.

Figure 3: Biases in the experiment with linear outcome equations (the canonical correlation is held at 0.2, $cc_{XZ} = 0.2$).

included; however, as we will see later, this finding is not robust to changes implemented in other experiments.

We can also see how the inclusion vs. exclusion of W from the propensity score equation affects the root mean squared error of the ATE estimates.[12] Figure 4 presents plots for the difference in the root mean squared error (RMSE) of these experiments. The plots keep the same basic structure of the ones in Figure 2. We find similar results. When the coefficient of W in the treatment and outcome equations have opposite signs, the treatment effect is weak, and the canonical correlation is low, it is worse to include W. In addition, if the outcome effect is null and the treatment effect is strong and negative, including W slightly increases the RMSE.

If the magnitude of the increment in bias or RMSE were negligible for the conditions that we have characterized, we could be confident that adding all available pre-treatment variables would not seriously affect the conclusions derived by the results of our empirical analysis. Unfortunately,

[12]The estimate of the root mean squared error $RMSE_k$ with $k = X, XW$ is $\sqrt{\frac{\sum_{j=1}^{500}(\widehat{\tau}_{k,j}-\tau)^2}{500}}$, where $\widehat{\tau}_{k,j}$ is the estimate of the average treatment effect for a given parameter combination in one replication of the experiment.

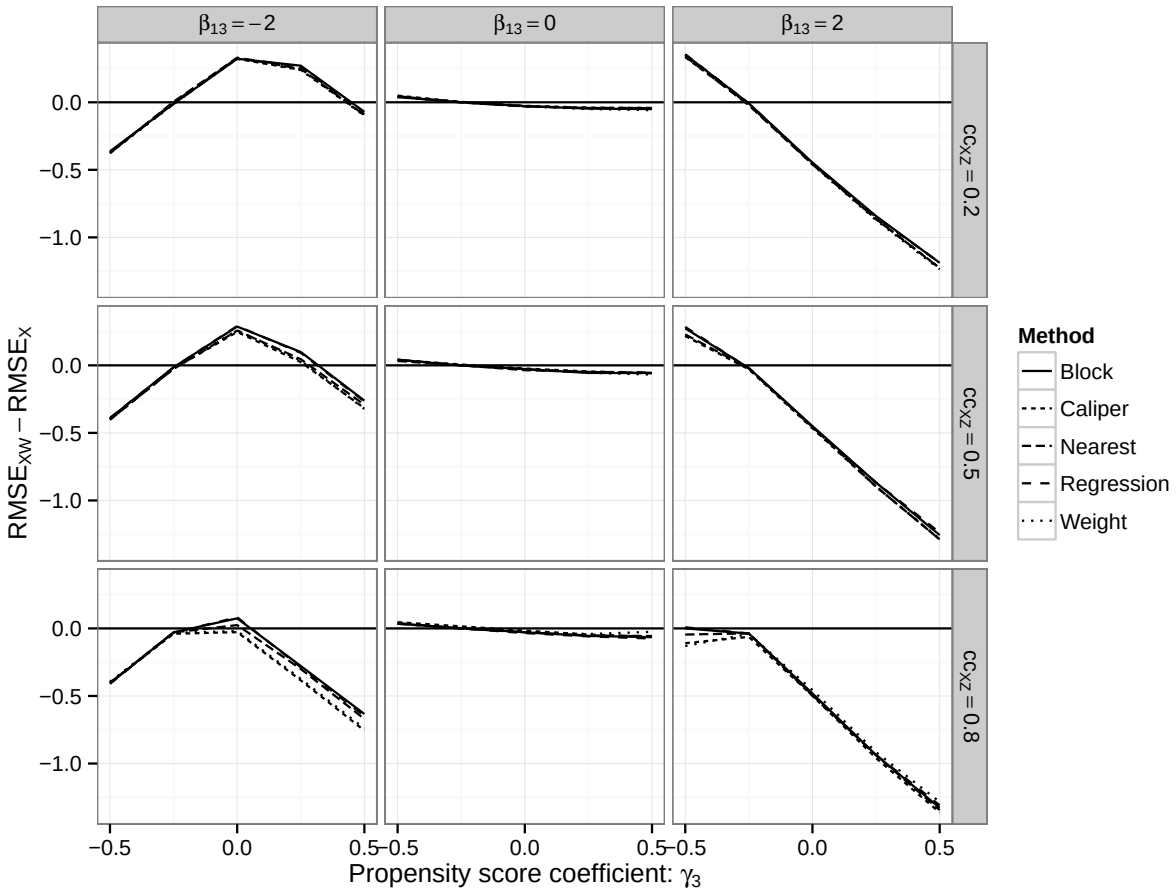


Figure 4: Difference in root mean squared error with linear outcome equations.

this is not always the case. If we consider the parameter combination of $\beta_{13} = 2$, $\gamma_3 = -0.5$ and $cc_{XZ} = 0.2$, the bias when W is included is approximately 0.53, compared to 0.05 when it is excluded. Including W increases by more than 10 times the bias for this parameter combination. Moreover, the increment in the bias represents 16% of the total ATE (which is 3 for this parameter combination).

To confirm that the relationships between the sign and magnitude of the coefficients in the propensity and outcome equations are indeed driving the results, we ran a number of additional Monte Carlo experiments. We ran the baseline model with each of the following changes (separately):

- Coefficient on Z in the propensity score equation reversed to -1;
- Coefficients on Z in the outcome equations, Y_0 and Y_1, reversed to -1 and -0.5 respectively;
- Correlation between W and Z reversed to -0.25.

Pattern	$\gamma_2 \cdot \beta_{12}$	$\gamma_3 \cdot \beta_{13}$	Including W increases bias?
1	+	+	no
2	+	-	yes
3	-	+	yes
4	-	-	no

Table 2: When does including a new variable W in the propensity score equation increase the bias of the estimated average treatment effect? (γ_2 and β_{12} are the coefficients on Z in the propensity and outcome equations, respectively. γ_3 and β_{13} are the coefficients on W in the propensity and outcome equations, respectively.)

In the first two cases, the results were nearly a mirror image of those shown in Figure 2: adding the new variable W increased the bias mainly in cases where it had the same effect on the treatment assignment and outcome.[13] In the third case, with the correlation between Z and W reversed, the results were substantively the same as in the original experiment. Our findings are summarized in Table 2, which describes the conditions or patterns of signs of effects under which adding a variable W to the propensity score equation increases the bias in the ATE when another variable Z is unavailable.

The cases where including W in the estimated propensity score equation increases the bias are those where balancing on W and balancing on Z have countervailing effects (patterns 2 and 3 in Table 2), and the effect of Z's confounding is stronger than that of W's. In this experiment, Z has a positive effect on the probability of treatment and a positive direct effect on the outcome, meaning the ATE would be overstated if only Z were omitted. If W has a negative effect on treatment assignment but a positive direct effect on the outcome, then W's omission causes the ATE to be understated. In this situation, balancing on Z causes the estimated ATE to decrease, and balancing on W causes an increase. Suppose the confounding effect of Z is larger than that of W, so the estimated ATE is too high when both are omitted. Including W in the propensity score specification would further increase the estimated ATE, exacerbating the bias due to the omission of Z.[14] This is precisely what we observe in Figure 2 when $\beta_{13} = 2$ and $\gamma_3 = -0.5$.

The patterns that we have consistently found in which adding a relevant pre-treatment variable increases the bias of the ATE could easily occur in empirical applications. Imagine an observational study on whether methamphetamine use T increases an individual's risk of heart disease Y. Suppose data are available on whether each individual is white W, but not on their household income Z. Compared to racial minorities, whites are more likely to be methamphetamine users and less likely to have heart disease, so the ATE would be underestimated if only W were left out. Conversely,

[13] In particular, these were $(\gamma_3, \beta_{13}) = (-0.5, -2)$ and $(0.5, 2)$, whereas in the original model the cases where including W increased bias were $(0.5, -2)$ and $(-0.5, 2)$.

[14] The only exception is if W and Z are so strongly positively correlated that balancing on W substantially improves balance on Z.

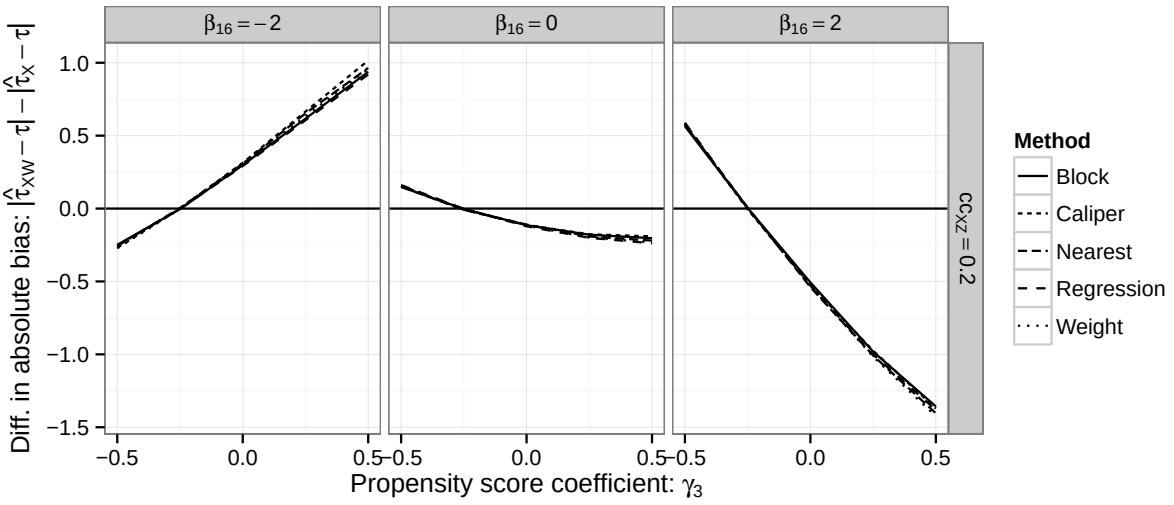


Figure 5: Difference in absolute bias with nonlinear outcome equations.

high-income individuals are both less likely to use methamphetamines and to have heart disease, so the omission of Z causes the ATE to be overestimated. This corresponds to pattern 2 in Table 2: If the confounding effect of income is stronger than that of race, controlling for race when income data are unavailable will likely increase the bias.

Experiment 2: nonlinear specification

Figure 5 presents the difference in absolute biases as a function of W's coefficient in the true propensity score when we add interactions and quadratic terms to the true outcome equation as specified in the previous section.[15] We again find that including W is worse in terms of bias when the signs of β_{13} and γ_3 are different, but this time the result holds regardless of the value of the canonical correlation. The second pattern found in the linear outcome equation case is still present: when γ_3 is negative and $\beta_{16} = 0$, including W increases the ATE's bias. The most noticeable differences between these results and those from the first experiment are that including W in the propensity score equation increases the bias in more cases, and that the magnitude of these increases is higher than those from before.

As in the previous experiment, the differences across estimation methods are slight. Figure 5 shows that there is no case where including W in the propensity score equation increases the bias when one method is used but decreases it under other methods. Finally, the results for the RMSE in the nonlinear case are almost identical to those for the difference in absolute bias, and thus are omitted.

[15] We present results only for a canonical correlation of 0.2, cc_{XZ}=0.2. The results for the other correlations are nearly identical.

Experiment 3: real data

Results from the final experiment, in which most of the covariates are taken from LaLonde's data rather than being simulated, are summarized in Figure 6. These results are similar to those of the first two experiments, though the pattern identified in Table 2 is somewhat weaker. For example, take the fourth column of Figure 6, where $\beta = 0.25$ (i.e., the new variable has a positive effect on the outcome). When the correlation between W and the omitted variable ($\ln(1+Earn75)$) is 0.25, including W in the estimated propensity score equation increases the bias when W's true effect on receiving the treatment is negative. Since the omitted variable has a positive effect on both assignment and outcome, this corresponds to the second row of Table 2. There are some anomalies when the magnitude of the correlation between W and Z is high. For example, when $\beta = -0.25$, inclusion of W in the propensity score specification when γ_1 is positive exacerbates bias for all ρ except 0.75. In this case, conditioning on W increases balance on Z enough to offset any potential exacerbation of the bias.

The differences between estimation methods are still relatively small in this experiment, but they are more noticeable than in the first two experiments. We find that including W in the propensity score equation is less likely to increase bias under caliper matching than under other methods. Covariance adjustment (regression on the propensity score) also seems to perform well with the LaLonde data, which was not the case in the fully simulated experiments. When using covariance adjustment to estimate the ATE, including W in the estimated propensity score equation increases the magnitude of bias in 40 cases, out of 125 total combinations of the parameters (β, γ, and ρ). That figure increases to 58 cases under nearest-neighbor matching; in between are caliper matching (46), weighting (53), and blocking (55).

Balance tests

The previous experiments identified situations where conditioning on all available pre-treatment variables could lead to an increase in bias on the estimated ATE (or ATT). The results show that when considering whether to include a pre-treatment variable in the propensity score equation a researcher should take into account the potential effect of unobservables on treatment and outcomes, as well as their relation with the variable in question. At this point it is natural to ask whether a post-matching balance test on treated and untreated units could be thought of as an alternative way to identify whether a covariate should be included in the propensity score equation. If balance on matched units improves once the variable is included in the propensity score, the inclusion might be justified. Improving balance, however, does not necessarily mean reducing bias. Adopting this practice could lead to worse estimation results. Using the LaLonde data, we show that it is not uncommon to find situations where a post-matching test indicates that a candidate variable should be included, when in fact its inclusion worsens the bias. What this result suggests is that balance tests are not substitutes for careful consideration of the potential effects of unobservables when

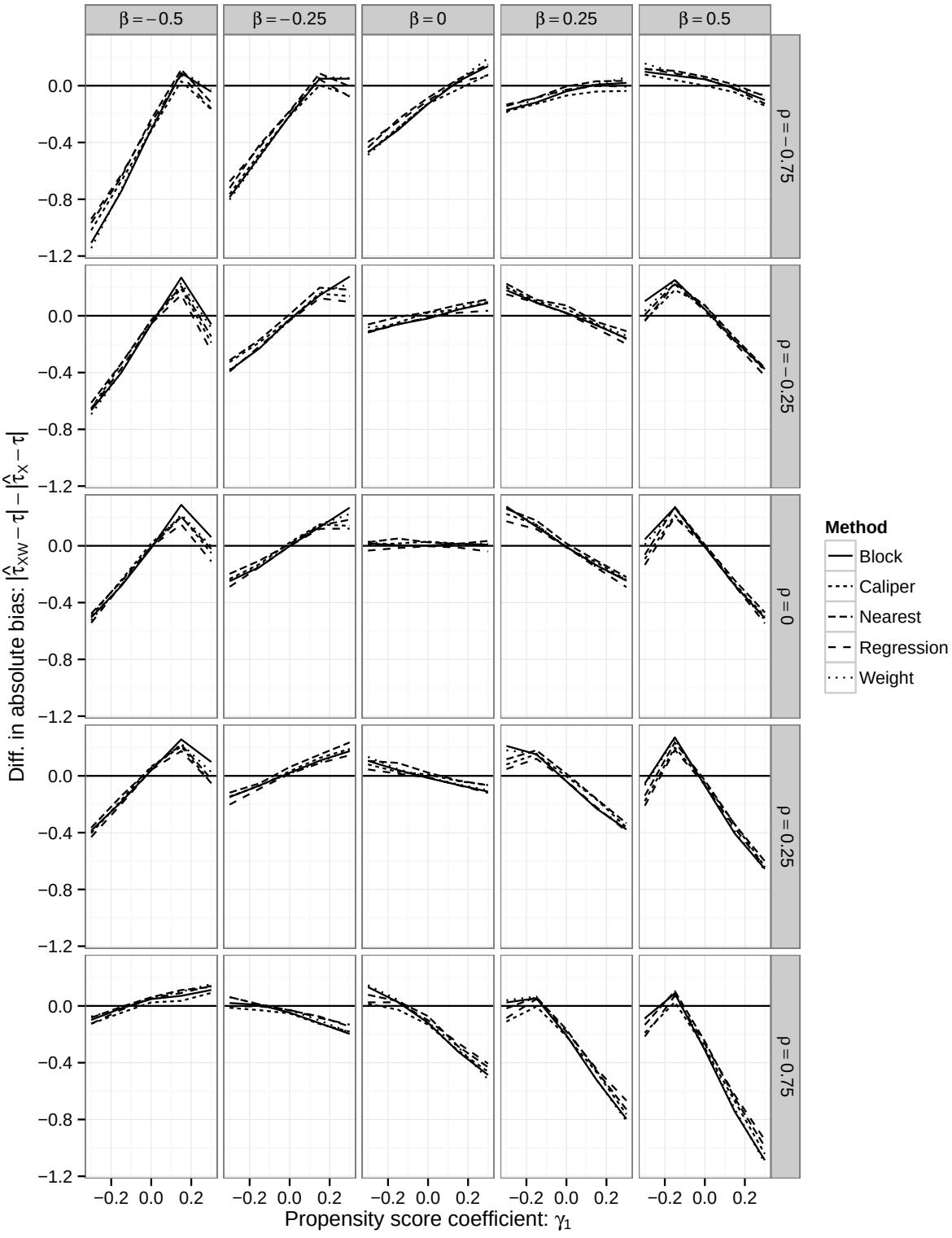


Figure 6: Difference in absolute bias with regressors taken from the LaLonde (1986) data.

choosing a specification.

For the following exercises, we use LaLonde's subset of treated units and the CPS (Current Population Survey) control individuals from the well-known study of the impact of the NSW labor training program on post-intervention income (LaLonde, 1986).[16] This data set has been used to evaluate the performance of treatment effect estimators on observational data by comparing them with an experimental benchmark. We are interested in finding pairs of variables from this data set that would play the role of Z and W in our previous experiments and that simultaneously satisfy three conditions: 1) They have one of the countervailing effects identified in Table 2, 2) balance seems to improve with W's inclusion in the propensity score according to a post-matching balance test, and 3) the estimated ATT is more biased when W is included. Identifying such cases gives evidence of the risks of relying exclusively on balance tests, and the potential benefit of using the identified countervailing patterns in justifying the inclusion of additional variables on the propensity score. Given that we are using observational data, we do not know the true effects of W and Z on the outcome and treatment. We generate estimates of these effects by relying on the propensity score specification that in Dehejia and Wahba (1999) gave the authors the closest ATT estimate to the experimental benchmark when using the CPS control individuals.[17]

To further clarify the exercise, consider the pair of variables $re75$ (real earnings in 1975) and *nodegree* (indicator variable of not possessing a degree) and suppose that the first one takes the role of Z (the unobserved variable) and the second one is the candidate variable to be included W. This would represent a situation where a researcher interested in finding the effect of the training program on income is considering to include the indicator of no degree in the propensity score equation when past earnings are not available. Following Dehejia and Wahba's specification that gives the best estimate of the ATT with this data, it is found that the effect of $re75$ and *nodegree* on income are both positive and that the effect of $re75$ on treatment assignment is negative while this same effect for *nodegree* is positive. This example follows pattern 3 in Table 2. For this specific case, and using as a benchmark the experimental ATT, we calculate that the bias after including *nodegree* (while leaving out $re75$) increases by 150.71, which is approximately 8.5% of the experimental ATT.[18]

We also compared the results of a balance test after matching when *nodegree* was not included in the treatment equation with the results of the same test when it was. We found that after including it, the p-value of a t-test between the treated and untreated matched units increased for 6 variables (out of a total of 11 covariates) compared to the p-value of the same test when it

[16]The data set is included in the R package of Random Recursive Partitioning (Iacus, 2007). It has a total of 16177 observations with no missingness on the variables from the original study. For a description of the data set see Dehejia and Wahba (1999, 1054)

[17]The specification includes the following variables: *age, age*^{2}*, education, education*^{2}*, no degree, married, black, hispanic, real earnings in 1974 and 1975 (re74 and re75), indicators of zero earnings in 1974 and 1975 (u74 and u75), education* $\times$ $re74$ *and age*^{3}.

[18]The ATT was calculated using caliper matching using the same estimation setup as in the Monte Carlo experiments.

was left out of the propensity score. This result suggests that in more than half of the rest of the covariates, balance seems to have improved after matching when *nodegree* was included. In this example, a researcher expecting a positive relation between past earnings and income, could infer with the help of Table 2 that the inclusion of this variable could bring an increase in bias if there is a positive relation between *nodegree* and income once other variables are controled for in the outcome equation.

The case of $re75$ and *nodegree* is not the only one. Table 3 shows 14 other cases where a countervailing effect is present and including W improves balance for a given number of covariates, but increases bias. These results are not definitive as we have relied on a particular specification from Dehejia and Wahba to find estimates of the true effects on income and participation on the program. However, given that the bias increased in the situations where we expected it to happen, based on our Monte Carlo findings, we can be more confident that the results are not unique to this data set or this particular specification.

Countervailing pattern	Z	W	Δ Bias	$\frac{\text{Vars. p-val increased}}{\text{Total}}$
2	$nodegree$	age^2	27.13	7/12
2	$education$	age	171.64	4/10
3	$black$	$nodegree$	112.75	8/12
3	$black$	$u74$	690.72	7/12
3	$u75$	$nodegree$	150.70	6/11
3	age	$education$	104.99	8/10
3	age	$u74$	94.37	8/10
3	age^2	$education$	104.99	8/10
3	age^2	$u74$	94.37	8/10
3	$black$	$education$	11.85	8/12
3	$married$	$nodegree$	107.37	9/12
3	$married$	$u74$	24.87	9/12
3	$re75$	$nodegree$	150.70	6/11
3	$education^3$	$education$	104.99	8/10
3	$education^3$	$u74$	94.37	8/10

Notes: The column 'Countervailing patterns' refers to the patterns defined in Table 2. $\frac{\text{Vars. p-val increased}}{\text{Total}}$ is the number of control variables that had an increase in the p-value of the t-test after matching when W was included over the number of common controls between the specifications with and without W.

Table 3: Balance test and propensity score selection of variables

Sensitivity analysis

We ran additional simulations to determine whether researchers can use sensitivity analysis to inform their decisions on whether a covariate should be included in the propensity score. The logic of using sensitivity analysis in this situation is as follows: If after including a variable in the

propensity score the results of the ATE estimation are less sensitive to unobserved factors than when the variable is left out, this could be used to justify its inclusion.

We use Rosenbaum's (2002) method for calculating bounds on the ATE estimate when the log odds of treatment assignment differ by up to Γ due to unobserved confounding.[19] Using the same parameters as in the simulations described above (with both the fully simulated data and the LaLonde data), we calculated the average lowest level of Γ at which the bounds on the treatment effect contained 0, under both inclusion and exclusion of W from the propensity score equation. If the level of Γ was larger when including W than when it was left out, this would suggest that the results are less sensitive to unobservables, and that therefore W should be added to the propensity score. However, what we found is that in all cases, this level is almost entirely determined by the magnitude of the estimated ATE and that there are no significant differences between including and excluding W on the calculated Γ. Therefore, this kind of sensitivity analysis is not useful for determining whether it is worse to include an observed pre-treatment variable because of its interaction with an unobserved confounder.

Discussion

This paper investigates claims made by both sides in recent debates regarding conditioning and matching using propensity scores. The results of our experiments suggest that conditioning on all available pre-treatment variables is not always optimal. In every case, the researcher must consider the effects of unobserved pre-treatment variables and their relationships with observed pre-treatment variables. Whether conditioning on an additional observed pre-treatment variable increases or decreases the bias on the ATE depends on these relationships. Specifically, in the linear case, we show that when the newly included covariate has a positive effect on the outcome and a negative effect on the propensity (and when there is an unobserved covariate whose effects on the outcome and treatment have the same sign), it is often worse to include the covariate. This basic pattern also holds in nonlinear specifications and in simulations using real data.

We have yet to address how researchers can best make use of our findings. Our results suggest that researchers cannot rely on advice such as condition on all pre-treatment covariates or on balance and sensitivity tests. Some progress can be made if we consider the two kinds of unobserved covariates that plague empirical analyses. To paraphrase Donald Rumsfeld (Morris, 2010), there are known unknowns and unknown unknowns. That is, there are covariates, perhaps suggested by theory, that cannot be measured or perhaps measurement is infeasible. These are the known unknown covariates. A researcher can hypothesize about the relationships of such a covariate with previously included variables and any variables that are candidates for inclusion. Our results provide some guidance in such a situation. If the candidate covariate and the unobserved covariate

[19] To calculate the bounds, we used the R package `rbounds` (Keele, 2011).

have countervailing effects, a case can be made for leaving the candidate covariate unadjusted.

On the other hand, there exist, in Rumsfeldian terms, unknown unknown covariates. These are variables that have not been suggested by theory and have not crossed the mind of the researcher in question (or anyone else). In such a case, no theorizing can take place, and our results demonstrate that including a new covariate in a propensity score equation may increase or decrease the bias on the estimated ATE. Sensitivity analysis that explicitly takes unobserved covariates into account, e.g. Rosenbaum (2002), seems to be of little use. The only surefire response a researcher has to the problem discussed in this paper is to be modest in the claims she makes based on her results. Scientific progress is rarely the result of a single study, and empirical generalizations are accepted only after many repeated demonstrations across varying spatial and temporal domains.

Appendix

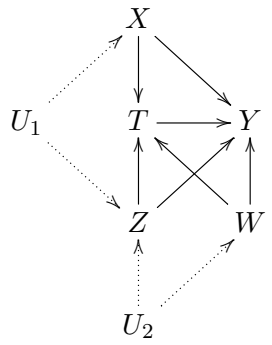


Figure 7: DGP 1 — W is related to both treatment and outcome.

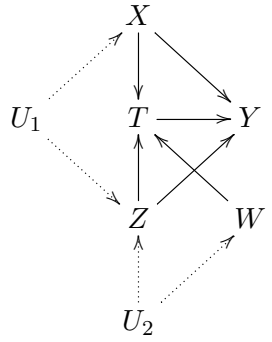


Figure 8: DGP 2 — W is related only to the treatment.

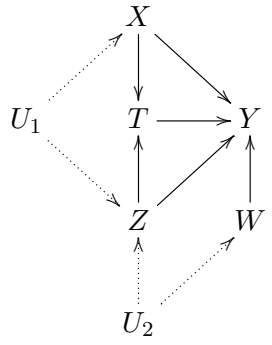


Figure 9: DGP 3 — W is related to the outcome.

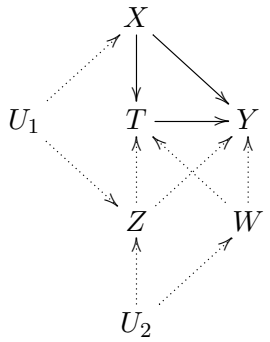


Figure 10: Misspecified model 1 — Z and W are unobserved.

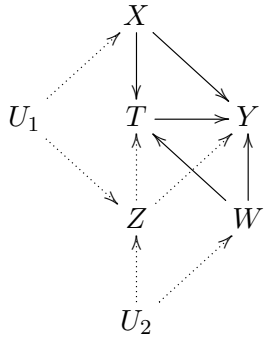


Figure 11: Misspecified model 2 — Z is unobserved, and W is assumed related to treatment and outcome.

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