Overlap, matching, or entropy weights: what are we weighting for?

Roland A. Matsouaka\textsuperscript{1,2,*}, Yi Liu\textsuperscript{1}, Yunji Zhou\textsuperscript{1}

\textsuperscript{1}Department of Biostatistics and Bioinformatics, Duke University, Durham, NC, USA
\textsuperscript{2}Program for Comparative Effectiveness Methodology, Duke Clinical Research Institute, Durham, NC, USA

Abstract

There has been a recent surge in statistical methods for handling the lack of adequate positivity when using inverse probability weights (IPW). However, these nascent developments have raised a number of questions. Thus, we demonstrate the ability of equipoise estimators (overlap, matching, and entropy weights) to handle the lack of positivity. Compared to IPW, the equipoise estimators have been shown to be flexible and easy to interpret. However, promoting their wide use requires that researchers know clearly why, when to apply them and what to expect.

In this paper, we provide the rationale to use these estimators to achieve robust results. We specifically look into the impact imbalances in treatment allocation can have on the positivity and, ultimately, on the estimates of the treatment effect. We zero into the typical pitfalls of the IPW estimator and its relationship with the estimators of the average treatment effect on the treated (ATT) and on the controls (ATC). Furthermore, we also compare IPW trimming to the equipoise estimators. We focus particularly on two key points: What fundamentally distinguishes their estimands? When should we expect similar results? Our findings are illustrated through Monte-Carlo simulation studies and a data example on healthcare expenditure.

Keywords: Positivity; propensity scores; equipoise; overlap weights; matching weights, entropy weights.
1 Introduction

To assess the effect of a new treatment regimen \((Z = 1)\) over a standard (or control) treatment \((Z = 0)\) based on data from an observational study, using causal identification a number of assumptions must be made, including the positivity assumption. For instance, to estimate the average treatment effect (ATE), this assumption requires \(0 < e(x) < 1\), where \(e(x) = P(Z = 1|X = x)\) is the propensity score (PS), i.e., the probability of treatment assignment, given the vector of baseline covariates \(X\) (Rosenbaum and Rubin, 1983; Rubin, 1997). The positivity assumption ensures that the distributions of the related baseline covariates have a good overlap and hence a good common support (Petersen et al., 2012; Li et al., 2018b).

The inverse probability weighting (IPW) estimator for ATE assigns to study participants weights that are inversely proportional to their respective PSs. Thus, IPW creates a pseudo-population of participants, corrects for observed covariates distributions imbalances between the treatment groups, and adjusts for (measured) confounding bias inherent to most non-randomized studies. Nevertheless, when PSs are equal to (or near) 0 or 1, there is violation (or near violation) of the positivity assumption, which we often refer to as lack of adequate positivity (Petersen et al., 2012). Violations (or near violations) of the positivity assumption occur either at random (or stochastically), i.e., by chance due the data (or underlying model) characteristics or when some subgroups of participants can never (or barely) receive one of the treatment options under study. This can lead to moderate or even poor overlap of the distributions of the PSs and may result in large IPW weights, especially when the ratio \([e(x)(1 - e(x))]^{-1}\) is highly variable (Li and Greene, 2013; Zhou et al., 2020b). As such, IPW may put a large amount of weights on a small number of observation, which can unduly influence the estimation of the treatment effect.

While violations of the positivity assumption can be remedied by either PS trimming or truncation, recent advancements have introduced methods that aim to overcome the limitations of these ad hoc solutions. Some of these novel methods propose bias-corrected estimators (Chaudhuri and Hill, 2014; Ma and Wang, 2020; Sasaki and Ura, 2022), while other reparametrize the PS estimation, by adding a priori covariate balancing constraints to modify the PS model (Graham et al., 2012; Imai and Ratkovic, 2014). Some consider direct optimization techniques to derive sample weights under covariate constraints (Hainmueller, 2012; Zubizarreta, 2015; Wong and Chan, 2017; Hirshberg and Zubizarreta, 2017) or redefine the target population altogether and bypass the need to account for the lack of positivity (Li et al., 2018a; Matsouaka and Zhou, 2020; Zhou et al., 2020b).

1.1 The positivity assumption and propensity score weighting methods

The literature defines two specific violations of the positivity assumption: random (i.e., by chance) and structural violations (Westreich, 2019; Petersen et al., 2012). Random (or stochastic) violation of the positivity assumptions arise by happenstance, e.g., when the sample size is small or the PS model is misspecified. In such cases, increased sample size, bias-corrected IPW trimming, PS reparameterization or direct optimization offer better alternatives to estimate ATE (Chaudhuri and Hill, 2014; Ma and Wang, 2020; Sasaki and Ura, 2022). Alternatively, methods for equipoise treatment effect, i.e., the overlap weight (OW), matching weight (MW), and Shannon’s entropy weight (EW) estimators (Matsouaka and Zhou, 2020; Li et al., 2018b), can also be considered. These estimators target treatment effects defined within the subgroup of participants for whom treatment equipoise exists.
As noted by Petersen et al. (2012), violations of the positivity assumption can lead to substantial bias and sometimes an increased variance of the causal effect estimator. While checking the PS distributions (or the PS weights) between treatment groups can help assess such violations, it is important to recognize that well-behaved weights alone may not guarantee the satisfaction of the positivity assumption (Ma and Wang, 2020; Petersen et al., 2012). A better investigation into violations of the positivity assumption must always be preceded by an expert-knowledge elicitation of the data at hand, the scientific questions as well as the source and the nature of the data at hand.

1.2 The positivity assumption and imbalance in treatment allocations

Correct estimation of the treatment effect is challenging when treatment (or exposure) allocation is rare (Pirracchio et al., 2012; Rudolph et al., 2022; Hajage et al., 2016). Nevertheless, assessment of treatment effects with small proportion of treated participants is a common occurrence, particularly in pharmacoepidemiologic observational studies of drugs (Hajage et al., 2016; Platt et al., 2012). The evaluation of the risk-benefit profile of a newly released drug is often conducted using observational studies where data on the effectiveness and safety of the drug are collected during routine care (Schneeweiss, 2007; Rassen and Schneeweiss, 2012). Schneeweiss et al. (2011) provide an example in the comparative effectiveness of newly marketed medications, which presents additional challenges. These challenges include potential bias due to patient channeling toward the newly marketed medication (due to patient, provider, and system related factors), shifts in the user population (due to varying background characteristics and comorbidities), timely data availability issues, and a smaller number of users in the initial months of marketing. As Schneeweiss et al. (2011) indicated, “Of these challenges, channeling is often the biggest threat to the validity of nonrandomized studies…” Therefore, there is a pressing need for the use and development of sound statistical methods that also aim at consistent and robust estimation of the treatment effects when the lack of positivity is expected or unavoidable.

While some authors have investigated the use of PS methods when the proportion of participant is small, their focus has primarily been on traditional PS methods, overlooking alternative methods that are well suited for lack of positivity. These alternatives go beyond the traditional use of PS matching, truncation, or trimming (Hajage et al., 2016; Franklin et al., 2017; Austin, 2011).

Causal inference, being inherently a missing data problem (Holland, 1986), we often overlook the fundamental task of any causal estimator: to use the available data to adequately input unknown potential outcome values. For IPW, this means weighting participants to create a pseudo-population where causal inference can be drawn. When the treatment allocation is imbalanced and extreme weights emerge, estimation and inference of the treatment effects relies heavily on a few participants with extremely large inverse probability weights, which can introduce severe bias due to data disparity. Therefore, regardless of whether violations of the positivity assumption are structural or not, it is crucial to ensure that the estimated treatment effects are disproportionately driven by a small number of outlying participants, especially if there is a substantial treatment allocation imbalance. For instance, in a tutorial for PS analysis, Austin uses a sample of current smokers discharged alive from a hospital following an acute myocardial infarction (Austin, 2011). What is remarkable in this paper are the small proportion (32.20%) of patients who did not benefit from in-patient smoking cessation counseling, the wide range of estimated
PSs, the presence of a few extreme weights, and the results on the 3-year survival outcomes (binary and time-to-event). Thus, reading this paper, we can’t help but raise some questions. Were the different conclusions drawn were solely due to methodological differences? In fact, some of these methods showed a significant reduced risk of mortality, while others just indicated that the treatment effect was not different from the null. Could the discrepancy in the study conclusion be solely driven by the differences in the selected methods and their underlying estimands? Did the imbalance in the number of participants between the two treatment groups play also a role? As we will demonstrate in this paper, we believe both the choice of specific methods and the imbalance in treatment allocation play a preeminent role.

1.3 How about trimming or truncating extreme weights?

The IPW estimator of the ATE targets \( E[B/A] \), with \( B = (Z - e(X))Y \) and \( A = e(X)(1 - e(X)) \). When there is a violation of the positivity assumption, some observations have \( A \approx 0 \), which can have undue influences on the naive sample mean of \( B/A \). The primary objective of trimming (i.e., dropping participants) and truncation (i.e., capping weights) is to curtail such undue influences and provide a stable estimator. Trimming (or truncating) participants with \( A \approx 0 \) (above given thresholds) is a common practice, as it effectively constraints the weights within reasonable bounds. However, the resulting estimate is often highly sensitive to the choice of on the threshold(s) (Chaudhuri and Hill, 2014; Ma and Wang, 2020; Sasaki and Ura, 2022). Unfortunately, the choice of a threshold is often ad hoc and subjective as they rest solely on the user’s discretion (Crump et al., 2006).

In many applications, a user-selected threshold can drastically change the number of participants we discard or for whom we curtail the weights (see, for instance, (Zhou et al., 2020b)). This tremendously affects the finite-sample performance of the estimator, influencing both its bias and efficiency. Moreover, the corresponding estimator may not target the ATE based on the original population since, for instance, under structural violation of the positivity assumption they both can alter their target estimands and the underlying populations of interest, depending on the threshold considered (Chaudhuri and Hill, 2014; Zhou et al., 2020b). For example, ATE trimming by a threshold \( \alpha \in (0,0.5) \) shifts its target population to the population of participants whose PS (of receiving either treatment or control) is inside the interval \((\alpha, 1-\alpha)\). Often, standard trimming and truncation method result to a non-negligible bias (even asymptotically) when estimating ATE, which may have some inference implications.

Landmark bias-correction strategies for trimming have been proposed. Chaudhuri and Hill (2014) proposed a bias-corrected, tailed-trimmed IPW estimator of the ATE, based on the tail behavior of \( |B/A| \). Their estimator is robust and asymptotically valid, even under substantial limited over of the PS distributions. Rather than trimming on the ratio \( B/A \), Ma and Wang (2020) and Sasaki and Ura (2022) considered trimming observations with \( A \approx 0 \) to build flexible, biased-corrected estimators that allow for larger trimming and hence smaller variances or faster rates of convergence. Robustness of the estimator by Ma and Wang (2020) is achieved by combining resampling with a local polynomial-based bias-correction technique, where a data-driven threshold is selected by minimizing the mean squared error. Sasaki and Ura (2022), on the other hand, leverage the smoothness of the conditional moment function \( a \mapsto E[B|A = a] \) to achieve more robust inference and faster convergence rate.

The above strategies (implicitly) assume random violation of the positivity assumption, under which true
ATE exist and can be point estimated; their respective trimming and bias correction solutions aim at improving inference. Unlike these papers, our proposed estimators do not even rely on the positivity assumption; thus, they are applicable to either random or structural violation of the positivity assumption. In addition, they automatically focus on the area of common support, identify a specific subgroup of participants where the estimate has a strong internal validity, without involving the outcome $Y$.

Furthermore, when there is an important imbalance in treatment allocation (i.e., the proportion of participants in one of the treatment groups is small), if often exacerbates the lack of adequate positivity, which can lead to more trimming or truncation in one group instead of both. Such practices not only reduce the number of participants in the final sample (after trimming), but also influence the contribution of those from whom extreme weights are capped (by truncation). This even further complicates point estimation and inference when structural violations of the positivity assumption are expected. Therefore, there is a growing interest for new practical methods that do not leave room to manually and subjectively pick a threshold; methods that can leverage inherent data-driven mechanisms to control the impact of extreme weights and provide robust assessments of treatment effects.

1.4 Are there better alternatives?

The overlap weight (OW), matching weight (MW), and Shannon’s entropy weight (EW) estimators (hereafter referred to as equipoise treatment effect estimators (Matsouaka and Zhou, 2020)) effectively circumvent the lack of positivity without specifying any user-driven threshold. Besides, they provide both better causal estimations and higher effective sample sizes (Li and Greene, 2013; Li et al., 2018a; Zhou et al., 2020b; Li and Li, 2021). However, it remains to see whether imbalances in treatment allocation can directly affect their estimations (compared to IPW estimation of ATE) and to what extent.

Therefore, the main objective of this paper is to provide a formal assessment of the impact of equipoise estimators (i.e., OW, MW, EW) on the treatment effect estimation in studies where there is a disproportionate distribution of treated or control participants in the population and how it relates to the lack of positivity. The title of our paper: “Overlap, matching and entropy weights: what are we weighting for?” is thus a call to action, i.e., to delve into the unique characteristics of these equipoise estimators, which grant them the flexibility to address the lack of positivity effectively (Zhou et al., 2020b). In the process, we showcase how OW, MW, and EW methods can be strategically used to estimate treatment effects and make asymptotically correct inferences of the corresponding estimators, when there is a violation of the positivity assumption.

The rest of the paper is organized as follows. We start in the next section with key questions that help define the purpose of our study and what we intend to accomplish. Then, in Section 3, we introduce notations and present the family of balancing weights. We specify their related estimands and define the corresponding estimators. Of particular interest are questions related to what is being estimated and what the target populations are when using these balancing weights? Next, we explore how the estimators are impacted by the proportion of treated participants and provide proper interpretations of their estimates. The illustrative example in Section 2.1 sets the scene for the main idea of this paper and informs the simulations in Section 4.

We evaluate the performance of the estimators using Monte-Carlo simulation studies in Section 4, covering three different treatment allocations, under various treatment effects and model specifications. The methods are
illustrated, in Section 5, to evaluate the impact of racial disparities on healthcare expenditure. Technical proofs, additional simulation details and data application results are presented in the Supplemental Material.

2 We do not always get what we expect

2.1 What are we trying to estimate?

Estimating ATE using IPW is problematic in the presence of observations with \( e(x)(1 - e(x)) \approx 0 \) as the method purposefully over-represents their contributions and can distort treatment effect estimation (Zhou et al., 2020b). In medical research, patients with with \( e(x)(1 - e(x)) \approx 0 \) are often those who exhibit distinct baseline characteristics, commorbidities, specific counterindications, restrictions, or outlying outcome measures.

On the other hand, imbalance in the treated allocation can also lead to \( e(x)(1 - e(x)) \approx 0 \). When the proportion of treated participant is small, many studies focus on the average treatment effect on the treated (ATT), i.e., the average effect in participants who ultimately received the active treatment. Nevertheless, the assessment of the average treatment effect (ATE) is de rigueur in some contexts, such as pharmacoepidemiologic observational studies of drugs (Hajage et al., 2016; Platt et al., 2012; Rassen and Schneeweiss, 2012; Schneeweiss, 2007). Unfortunately, a smaller proportion of treatment participants can lead to a lack of positivity and the presence of extreme PS weights, complicating the assessment of ATE. Furthermore, because ATE provides the treatment effect that we would have obtained if everyone (treated and controls alike) is assigned to one treatment option versus being everyone is assigned to the alternative treatment option, IPW plays an intriguing balancing act when the treatment groups are inadequately allocated, as we illustrate below.

2.2 Are we actually getting what we hope for?

2.2.1 An illustrative example

To illustrate the influence of \( p = P(Z = 1) \) in estimating different causal estimands, we conducted a simulation study as follows. For \( N = 10^4 \), we generated \( X = (1, X_1, X_2) \) such that \( X_1 \sim \mathcal{N}(6, 9) \), \( X_2 \sim \text{Bern}(0.75) \) and a treatment assignment \( Z = \text{Bern}(\text{expit}(X \beta)) \), with \( \beta \sim (\beta_0, 0.2, 0.8)' \). We chose \( \beta_0 = -3.5 \) and 0 to obtain \( p = 17.14\% \) (small) and 83.27\% (large), respectively. Next, we generated the outcome variable \( Y = ZY(1) + (1 - Z)Y(0) \), where \( Y(0) = -X_1 + 2X_2 + \varepsilon \) and \( Y(1) = Y(0) + (X_1 + X_2)^2 \), with \( \varepsilon \sim \mathcal{N}(0, 1) \).

Using the potential outcomes, we calculated \( \text{ATE} = E[Y(1) - Y(0)] \) and estimated the PSs via a logistic regression model. Then, we determined ATT, ATC, ATO, ATM, and ATEN using formula (1) and those in Table 2. The results, presented in Table 1, indicate the the variances of PSs of the treated to the control groups are similar (\( r \in [0.5, 2] \), per the rule of thumb of Rubin (2001). Furthermore, when \( p \) is small (17.14\%), the ATE is closer to ATC whereas the equipoise estimands (ATO, ATM and ATEN) are closer to ATT. On the other hand, when \( p \) is large (83.27\%), ATE is closer to ATT where equipoise estimators (ATO, ATM and ATEN) are closer to ATC.
Table 1: Causal effects under two different treatment allocations $p$

| Scenario | $p$    | $r$  | IPW estimands | Equipoise estimands |
|----------|--------|------|---------------|---------------------|
|          | 17.14% | 1.29 | ATE | ATT | ATC | ATO | ATM | ATEN |
|          | 83.27% | 0.63 | 54.75 | 76.54 | 49.62 | 69.87 | 76.54 | 49.62 | 69.87 | 75.88 | 66.08 | 66.08 | 66.08 |

$r$: ratio of variances of the propensity scores (treatment vs. control)

2.2.2 Is it really a surprise?

In part, the above results on the ATE are not surprising. The ATE depends on how the treatment effects (ATT and ATC) vary given the treatment groups and, more importantly, it weighs more one or the other depending on which group has the higher proportion of participants. The relationship $ATE = pATT + (1 - p)ATC$, indicates what matters more when there is imbalance in treatment allocations. The influence of $p$ in estimating ATE is far from trivial; it can indeed have a substantial impact. When the treatment effect is heterogeneous and the proportion of treated participants $p$ is small (resp. large), ATE puts more emphasis on the subpopulation of control (resp. treated) participants. However, it is questionable that whether the ATC is the estimand that matters the most when dealing with a smaller number of treated participants. Usually, in this situation, most study designs and analytical methods target instead the ATT, which has a completely different interpretation than the ATC (Austin et al., 2021; Greifer and Stuart, 2021). In addition, in finite sample (as we will demonstrate in the simulations) and when $p$ is small, the estimate of the ATC is less precise, as it relies on outcomes from a small number of treated participants (comparators) to serve as counterfactuals for the larger percentage of controls participant. Similar observations can be made when $p$ is large, where ATE is now closer to ATT, even though the estimation of ATT relies on outcomes from a smaller fraction of control participants as counterfactuals for the treated participants.

This behavior of ATE is contrary to what we usually aim for, if we make an analogy with a rare-exposure study, where we use a large pool of controls to find better matches for the limited number of exposed cases. This is also counter-intuitive to our common practice when using matching (either on the PS or on some covariates) and our target is the ATT or an ATT-like estimator (Stuart, 2010). We match efficiently when we have at least as many controls as treated participants. Therefore, relying heavily on estimating the (population) ATE when we have a smaller percentage of treated participants may systematically fail to reach our target of inference. Fortunately, as indicated in Table 1, that goal can be achieved by purposefully targeting a specific subpopulation of patients using a different set of weights.

2.2.3 What if we target a different population of participants?

The results obtained from ATO, ATM, and ATEN are not really surprising either. Li et al. (2018a) alluded to similar results for overlap weights when explaining how flexible and data-adaptive they are vis-à-vis values of the PSs $e(X)$. They noted that the overlap weights $(w_1(x), w_0(x)) \propto (1 - e(x), e(x)) \approx \left(\frac{0.25}{1 - e(x)}, \frac{0.25}{1 - e(x)}\right)$ when $e(X) \approx 0.5$, in which case they are proportional to IPW weights for the ATE. For smaller $e(X)$, $(w_1(x), w_0(x)) \approx \left(1, \frac{e(x)}{1 - e(x)}\right)$ in which case $w_1(x)$ and $w_0(x)$ resemble ATT weights. Finally, for larger $e(X)$, the overlap weights
\((w_1(x), w_0(x)) \approx \left( \frac{1-e(x)}{e(x)} , 1 \right)\) in which case they resemble ATC weights. We naturally conjecture that, under some regularity conditions, \(p = P(Z = 1) = E[E(Z|X)] = E(e(X))\) (i.e., the first moment of the PSs \(e(X)\)) might be sufficient to reflect how overlap weights weigh ATT and ATC, thus extending the above observations from Li et al. (2018a).

Matsouaka and Zhou (2020) formalized and proved that asymptotically this phenomenon generalizes not only to ATO, but also to ATM and ATEN. To do so, they showed that ATO, ATM, and ATEN estimators are expected to yield similar estimated results. Besides, they demonstrated the direct links between the density distributions of PSs within each treatment group. Finally, they established the connections between the proportion of treated participants \(p = P(Z = 1)\), the ratio of variances of the PSs \(r\), and whether one might expect to obtain, in theory, a result close to ATT or ATC when using ATO, ATM, or ATEN. For instance, in the case of overlap weights (as well as all other equipoise estimators), Table 1 confirms that, when \(p\) is small, the emphasis is on the subpopulation of treated participants and the estimand is indeed close to ATT.

While the results from Table 1 align with the theoretical results in the literature (see, for instance, Matsouaka and Zhou (2020)), we still have a limited understanding of what ATO, ATM, ATEN truly identify and estimate in the presence of treatment allocation imbalances and under finite sample sizes. The ratio \(r\) also appears to play a role to determine whether these estimands lean heavily toward ATT or not. In addition, with finite sample sizes, estimation of ATE using IPW weights when the proportion of participants \(p\) is small (or large) is challenging by the issues of limited overlap and the influence of extreme weights. Hence, we also need to explore finite-sample statistical and numerical properties of estimating ATE via IPW weights as well as ATO, ATM, and ATEN. Therefore, we will need to run simulation studies to find out, whether ATE and equipoise weight estimators lead towards two different alternative estimands (ATT vs. ATC) in finite sample size when the proportion of treated participants \(p\) is small or large.

3 Balancing weights

3.1 Theoretical background, notation and assumptions

Let us denote \(Z = z\) the treatment indicator \((z = 1\) for treated and \(z = 0\) for control\), \(Y\) a continuous outcome, and \(X = (X_0, X_1, \ldots, X_p)\) the matrix of baseline covariates, where \(X_0 = (1, \ldots, 1)'\). The observed data \(O = \{(Z_i, X_i, Y_i) : i = 1 \ldots, N\}\) are a sample of \(N\) participants drawn independently from a large population of interest. We adopt the potential outcome framework (Neyman, 1923; Imbens and Rubin, 2015), and assume that for any randomly chosen participant in the population, there exists a pair of potential outcomes \((Y(0), Y(1))\), where \(Y(z)\) is the outcome that would been observed if, possibly contrary to fact, the individual were to receive treatment \(Z = z\). In addition, we make the following assumptions:

1. Consistency: \(Y = ZY(1) + (1 - Z)Y(0)\), i.e., for each individual, the observed outcome \(Y\) matches the potential outcome \(Y(z)\) for the treatment \(Z = z\) they received.

2. Stable-unit treatment value assumption (SUTVA): there is only one version of the treatment and the potential outcome \(Y(z)\) of an individual does not depend on another individual’s received treatment, as it is the case when participants’ outcomes interfere with one another (Rosenbaum and Rubin, 1983).
3. Unconfoundness: $E[Y(z)|X] = E[Y(z)|X, Z = z]$, $z = 0, 1$.

The PS, $e(x) = P(Z = 1|X = x)$, is the conditional probability of treatment assignment given the observed covariates. Under the unconfoundness assumption, the PS is a balancing score since $X \perp Z|e(X)$. This implies that participants with the same PS have similar distributions of their observed baseline covariates $X$ regardless of their treatment assignment (Rosenbaum and Rubin, 1983, 1984; Rubin, 1997). Therefore, instead of controlling for the whole vector of multiple covariates $X$ to estimate treatment effects, one can leverage this property of the PS $e(X)$ to derive unbiased estimators of the treatment effect. Since the PS $e(X)$ is usually unknown in non-randomized studies, we estimate it by postulating a model $e(X; \beta) = P(Z = 1|X; \beta)$, for some parameter vector $\beta$.

More often, the goal is to estimate the average treatment effect (ATE) $\tau = E[\tau(X)]$ from the data, where $\tau(x) = E[Y(1) - Y(0)|X = x]$ is the conditional average treatment effect (CATE). In this paper, we consider the weighted average treatment effect (WATE)

$$\tau_g = \frac{E[g(X)\tau(X)]}{E[g(X)]},$$

where $g(x)$ is a known selection (or tilting) function and $\tau$ corresponds to the identity function $g(x) = 1$.

The estimand $\tau_g$ encompasses a large class of causal estimands, depending of the function $g$ (Li et al., 2018a; Hirano et al., 2003; Crump et al., 2006). The selection function $g(x)$ delimits and specifies the target subpopulation defined in terms of the covariates $X$. Higher values of $g(x)$ indicates the regions of the covariates space with higher weights in the target subpopulation. In addition, the function $g$ helps with the interpretation of the corresponding treatment effect and characterizes the related weights (see Table 2).

The equipoise selection functions assign higher weights to observations around the middle of the PS spectrum, at $e(x) = 0.5$, and gradually and smoothly downweight the contributions of those at the tails of PS spectrum (as shown in Figure 1), which bypass the need to enforce positivity assumption. As demonstrated by Matsouaka and Zhou (2020), the selection functions for clinical equipoise are closely related. This explains why, in practice, one can expect to obtain statistically similar results with ATO, ATM, or ATEN, as they all target similar populations of participants, i.e., those for whom there is clinical equipoise. Additional insights on the equipoise estimators can be find in the references hereafter (Li et al., 2018b; Matsouaka and Zhou, 2020; Thomas et al., 2020).

Table 2: Examples of selection function, target population, causal estimand, and weights

| Target population | Selection function $g(x)$ | Estimand | Method |
|-------------------|---------------------------|----------|--------|
| overall           | 1                         | ATE      | IPW    |
| treated           | $e(x)$                    | ATT      | IPW Treated |
| control           | $1 - e(x)$                | ATC      | IPW Control |
| trimmed           | $I_{(\alpha_1, \alpha_2)}(x) = 1\{\{e(x) \leq 1 - \alpha_2\}\}$ | OSATE    | IPW Trimming |
| truncated         | $J_{(\alpha_1, \alpha_2)}(x) + J_{\alpha_1}(e(x)) + J_{1 - \alpha_2}(1 - e(x))t^{1 - z}$ | OWATE/ATO | OW |
| equipoise          | $e(x) (1 - e(x))$          | OWATE    | OW     |
| equipoise          | $\min\{e(x), 1 - e(x)\}$  | ATM      | MW     |
| equipoise          | $-e(x) \ln(e(x)) + (1 - e(x)) \ln(1 - e(x))$ | ATEN     | EW     |

$I(.)$ is the indicator function and $J_\alpha(e(x)) = \alpha^{-1}e(x)I(\{e(x) < \alpha\})$, with $\alpha, \alpha_1, 1 - \alpha_2 \in (0, 0.5)$, $z \in \{0, 1\}$.
The weighted treatment effect \( \tau_g \) can be estimated by the weighted, Hájek-type (Hájek, 1971) estimator

\[
\hat{\tau}_g = \frac{N}{N_{\hat{w}_1}} \left[ \sum_{i=1}^{N} \frac{Z_i \tilde{w}_1(x_i) \{ Y_i - \tilde{m}_1(x_i) \}}{N_{\tilde{w}_1(x_i)}} - \frac{(1 - Z_i) \tilde{w}_0(x_i) \{ Y_i - \tilde{m}_0(x_i) \}}{N_{\tilde{w}_0(x_i)}} \right], \quad \text{with } N_{\tilde{w}_k} = \sum_{i=1}^{N} Z_i^{1-k} \tilde{w}_k(x_i), \; k = 0, 1
\]

where \( \tilde{w}_k(x) = \tilde{g}(x)\tilde{e}(x)^{-k}(1 - \tilde{e}(x))^{k-1} \), with \( \tilde{g}(x) = g(x; \hat{\beta}), \; \tilde{e}(x) = e(x; \hat{\beta}) \), and \( \hat{\beta} \) an estimator of \( \beta \) (Li and Greene, 2013; Li et al., 2018a). The weights \( w_k(x) \) balance the weighted distributions of the covariates between the two treatment groups (Li et al., 2018a). Thus, the name balancing weights.

We also consider augmentations of the estimators \( \hat{\tau}_g \) based on outcome regression models. For ATE, ATT and ATC, they correspond to the following augmented estimators, with \( \tilde{w}_k(x) = \tilde{g}(x)\tilde{e}(x)^{-k}(1 - \tilde{e}(x))^{k-1}, \; k = 0, 1:\n
\[
\hat{\tau}_{\text{ATE}g} = \frac{N}{N_{\tilde{w}_1}} \left[ \sum_{i=1}^{N} \frac{Z_i \tilde{w}_1(x_i) \{ Y_i - \tilde{m}_1(x_i) \}}{N_{\tilde{w}_1(x_i)}} - \frac{(1 - Z_i) \tilde{w}_0(x_i) \{ Y_i - \tilde{m}_0(x_i) \}}{N_{\tilde{w}_0(x_i)}} \right], \quad g(x) = 1;
\]

\[
\hat{\tau}_{\text{ATT}g} = \frac{N}{N_{\tilde{w}_1}} \left[ \sum_{i=1}^{N} \frac{Z_i \{ Y_i - \tilde{m}_1(x_i) \}}{N_{\tilde{w}_1(x_i)}} - \frac{(1 - Z_i) \tilde{w}_0(x_i) \{ Y_i - \tilde{m}_0(x_i) \}}{N_{\tilde{w}_0(x_i)}} \right], \quad \text{with } g(x) = \tilde{e}(x);
\]

\[
\hat{\tau}_{\text{ATC}g} = \frac{N}{N_{\tilde{w}_1}} \left[ \sum_{i=1}^{N} \frac{Z_i \tilde{w}_1(x_i) \{ Y_i - \tilde{m}_1(x_i) \}}{N_{\tilde{w}_1(x_i)}} - \frac{(1 - Z_i) \tilde{w}_0(x_i) \{ Y_i - \tilde{m}_0(x_i) \}}{N_{\tilde{w}_0(x_i)}} \right], \quad \text{where } g(x) = (1 - \tilde{e}(x))
\]

where we postulate parametric models \( \tilde{m}_z(X) = m_z(X; \alpha_z) \), i.e., the outcome regression models, with parameter \( \alpha_z \) to estimate \( m_z(X) = E[Y(z)|X], \; z = 0, 1 \) (Matsouaka and Zhou, 2020; Matsouaka et al., 2023).

For equipoise estimands (ATO, ATM and ATEN), we consider the augmented estimators

\[
\hat{\tau}_{\text{aug}g} = \sum_{i=1}^{N} \left[ \frac{Z_i \tilde{w}_1(x_i) \{ Y_i - \tilde{m}_1(x_i) \}}{N_{\tilde{w}_1(x_i)}} - \frac{(1 - Z_i) \tilde{w}_0(x_i) \{ Y_i - \tilde{m}_0(x_i) \}}{N_{\tilde{w}_0(x_i)}} \right] + \sum_{i=1}^{N} g(x_i) \{ \tilde{m}_1(x_i) - \tilde{m}_0(x_i) \} / \sum_{i=1}^{N} g(x_i).
\]

While the estimators (3) are doubly-robust (i.e., they are consistent when at least one of the PS or the outcome regression models is correctly specified), the equipoise estimators \( \hat{\tau}_{\text{aug}g} \) are not. The latter are consistent only if the PS model is correctly specified (Matsouaka and Zhou, 2020; Mao et al., 2018).

For all the estimators, we derived their close-form sandwich variance estimators based on the asymptotic normal approximation and M-theory (see Supplemental Material A). The performance of the weights \( \tilde{w}_k(x), \; k = 0, 1 \), can be assessed through the effective sample size (ESS)

\[
\tilde{E}SS = \left( \frac{N}{\sum_{i=1}^{N} \tilde{w}(x_i)} \right)^{-1} \left( \frac{N}{\sum_{i=1}^{N} \tilde{w}(x_i)} \right)^2, \quad \text{where } \tilde{w}(x_i) = z_i \tilde{w}_1(x_i) + (1 - z_i) \tilde{w}_0(x_i), \; z_i = 0, 1.
\]
Our data generating process (DGP) is similar to that of Li and Li (2021). First, we simulated a superpopulation of 10^6 individuals under different scenarios for desired proportions p to determine the true values of the estimands under heterogeneous treatment effects. Then, to assess the finite-sample performance of the different estimators, we simulated M = 2000 independent data sets of size N = 1000 and allowed the p to vary, as specified below. Within each data set, we estimated ATE (with and without trimming) via IPW, ATT, ATC, and the treatment effects via the equipoise estimators (OW, MW, and EW). For ATE with trimming, we trimmed observations with the PSs that fall outside of the interval [α, 1 − α], with α = 0.05, 0.1, and 0.15, respectively.

We summarized and interpret the results based on the criteria laid out in Section 4.2. For Hájek-type estimators, we only report the results under correctly specified (PS) model. The results under misspecified PS models were similar to those in Zhou et al. (2020b) and were not reported here.

4.1 Data generating process

We first generated the covariates \(X = (X_0, X_1, \ldots, X_7)\) and the treatment assignment \(Z \sim \text{Bern}(\exp(-X'\beta))\). \(X_0 = (1, 1, \ldots, 1)\) and \(X_4 \sim \text{Bern}(0.5)\), \(X_3 \sim \text{Bern}(0.4 + 0.2X_4)\), \((X_1, X_2)' \sim N(\mu, \Sigma)\), \(X_5 = X_7^2\), \(X_6 = X_1X_2\), and \(X_7 = X_2^2\), where \(\mu = (X_4 - X_3 + 0.5X_3X_4, X_3 - X_4 + X_3X_4)'\), \(\Sigma = X_3\begin{pmatrix} 1 & 0.5 \\ 0.5 & 1 \end{pmatrix} + X_4\begin{pmatrix} 2 & 0.25 \\ 0.25 & 2 \end{pmatrix}\).

We selected different values of \(\beta = (\beta_0, \beta_1, \ldots, \beta_7)'\) to have a range of proportions of treated participants \(p\) and variances of PSs (treated vs. control), as shown in Table B.1.1 (see Supplemental Material B.1). The distributions of the estimated PSs are shown in Figure B.1.2. Then, we generated the outcomes \(Y = ZY(1) + (1 - Z)Y(0)\), with \(Y(0) = 0.5 + X_1 + 0.6X_2 + 2.2X_3 - 1.2X_4 + (X_1 + X_2)^2 + \varepsilon\) and \(Y(1) = Y(0) + \delta(X)\), with \(\varepsilon \sim N(0, 4)\). We considered both a constant \((\delta(X) = 4)\) and a heterogeneous \((\delta(X) = 4 + 3(X_1 + X_2)^2 + X_1X_3)\) treatment effect. The true average heterogeneous treatment effects are reported in Table B.1.2 (Supplemental Material B.1).

To assess the performance of the augmented estimators, we also analyzed the generated data under misspecified PS and outcome regression (OR) models. We considered 4 specific scenarios of model specification: (i) all the models are true, (ii) the PS model is true, (iii) the OR models are true, and (iv) all the models are misspecified. To misspecify the models, we removed \(X_1^2, X_2^2\) and \(X_1X_2\) from \(Z\) and \(Y\) to only use \((X_0, X_1, \ldots, X_4)\) instead.

4.2 Measures and performance criteria

To evaluate the performance of different estimators as well as their sensitivity to model misspecifications, we considered the following measures: the absolute relative percent bias, \(\text{ABias} = 100\% (\bar{e}_g - \tau_g)/\tau_g\); the relative root mean square error \(\text{RRMSE} = \left[(\bar{e}_g - \tau_g)/\tau_g\right]^2\); and the coverage probability (CP) for 95% Wald confidence.
intervals, i.e., the proportion of times $\tau_g$ was inside of its estimated confidence interval. For both ARBias and RRMSE, the smaller the measure, the better while the CP is considered significantly different from the nominal 95% coverage level if it is outside of the interval [0.94, 0.96].

We also reported, in Tables B.2.1–B.2.6 (Supplemental Material B.2), other measures such as the root mean square error \( \text{RMSE} = (\bar{\tau}_g - \tau_g)^2 \) and the relative efficiency \( \text{RE} = \text{SD}/\text{SE} \), where SD is the empirical standard deviation of $\bar{\tau}_g$ and SE is the estimated standard error of a $\bar{\tau}_g$ from its estimated sandwich variance. Finally, we considered how close estimates of ATE and equipoise estimates were from those of ATT and ATC, which allows us to answer our main question of interest: what are we weighting for?

### 4.3 Results

For brevity, we only present the results from a few models (Models 1, 2, 3, and 6) in this section. The complete and exhaustive simulation results for all the scenarios considered are provided in Supplemental Material B, including the average ESS (by treatment groups) we obtained from the different estimators (see B.1.3).

The results from Model 1 (constant treatment effect) are reported to showcase the variability in estimating both ATE and ATC under small $p$ (10.05%). We also show the results under heterogeneous treatment effects from Models 2, 3 and 6, where $0.5 \leq r \leq 2$ on average, but $p$ ranges from 20.77% to 79.59%.

![Figure 2: Relative bias under constant treatment effect (Model 1: proportion of treated = 10.05%)](image)

A: Hájek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both the PS and OR models correctly specified (resp. only the PS model correctly specified, only the OR model correctly specified, both the PS and OR models misspecified).

Figure 2 provides the ARBias for Model 1 (constant treatment effect), under different specifications of the PS and outcome models. Throughout the scenarios, the bias were lower for OW, MW and EW—except when both models were misspecified. The sheer number of outlying ARBias values for IPW without trimming in estimating the ATE and ATC clearly indicates how unstable and unreliable these estimators are whenever $p$ is small.

While the biases are smaller overall with true outcome regression models (scenarios B and D), they are higher
with the Hájek-type estimator (scenario A) and whenever the PS model is misspecified (scenarios C and E). The estimates of ATE and ATC via IPW without trimming are more biased and the magnitude of their related biases are highly variable. This is even more pronounced when both models are misspecified. The estimates under IPW trimming have also similar behavior, although to lesser extent.

Figure 3 gives the point estimates of all the WATEs under heterogeneous treatment effect of Models 2, 3 and 6. It can be seen that, more than often, when \( p \) is small and equal to 20.30% (resp. high and equal to 79.59%) and the variances of the PSs from the two treatment groups are roughly equal, the estimated ATEs via IPW, without trimming, are closer to ATC (resp. ATT) while the estimated equipoise estimands (ATO, ATM and ATEN) are closer to ATT (resp. ATC)—with both Hájek-type (scenario A) and augmented estimators (scenarios B, C, D, and E). When \( p \) is about 0.5, these estimands have similar results. This trend remains the same whether the PS and OR models are correctly specified or not.

The results from IPW trimming were intriguing and did not show any specific trend when we went from the 0.05 threshold to 0.15. When \( p = 20.77\% \), the point estimate from IPW trimming with the threshold of 0.05, i.e., ATE (0.05) is always between ATE (0.10) and ATE (0.15), except in the case where both PS and OR models are misspecified. For \( p = 49.72\% \), ATE (0.10) is between ATE (0.05) and ATE (0.15) as one would expect. Finally, when \( p = 79.59\% \), ATE (0.10) and ATE (0.15) are almost the same, while ATE (0.05) coincides with estimates from equipoise estimators in scenarios A, B, and C. In scenarios D and E, ATE and ATE (0.05) are very close, whereas ATE (0.10) and ATE (0.15) are far away from the ATT estimate they were expected to be close to.

In Figure 4, while trimming seems to have a good bias-variance trade-off when we look at their RRMSEs, this contrasts with their poor CPs. Trimming appears to systematically underestimate the targeted nominal 0.95 level, with some average CPs being as low as 0.76 for ATE (0.15), when both PS and OR models are correctly specified (scenario B) and \( p = 20.77\% \), or below 0.92 for ATE (0.05), ATE (0.10), and ATE (0.15) in all scenarios when \( p = 79.59\% \). In fact, ATE (0.10) and ATE (0.5) had the worst CPs throughout, with CPs close to 0.70 for ATE (0.15) under scenarios A, B, and C as well as for ATE (0.10) under scenario D—leaving a huge gap between the CPs and the targeted nominal 0.95 coverage level.

There are some nuances, depending on whether the values of \( p \) (small, large or in the vicinity of 50%).
A: Hájek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both the PS and OR models correctly specified (resp. only the PS model correctly specified, only the OR model correctly specified, both the PS and OR models misspecified).

Figure 4: Bias, mean-squared error, and coverage probability (under Models 2, 3, and 6).
Nevertheless, regardless of $p$, Hájek-type estimators as well as doubly robust ATE and augmented equipoise estimators (ATO, ATM and ATEN) tend to have small and similar ARBiases and RRMSEs when at least one model is correctly specified, i.e., under scenarios A, B, C, and D. The only exceptions are ATE (0.15), which have a higher ARBias under scenarios A, B, and D when $p = 20.77\%$; ATE (0.10), where ARBias is higher for $p = 79.59\%$; followed by ATE (0.05). When the two models are misspecified (scenario E), some estimators of ATE (with and without) have larger ARBiases and RRMSEs. Figure 4 also indicates that the estimates for ATC often have larger ARBiases (in scenarios E when $p < 50\%$) and RRMSEs (in most scenarios) than other estimates.

Furthermore, Figure 4 (bottom panel) show that CPs for equipoise estimators are closer to nominal 0.95 coverage level (i.e., within [0.94, 0.96]) than ATT, ATC and trimmed ATE estimates. This means that when constructing a 95% confidence interval, the close-form sandwich variance estimators lead to more efficient equipoise and ATE (without trimming) estimators than ATC, ATT and trimmed ATE, as expected (Li et al., 2018a). The confidence intervals for ATT and ATC are too conservative, while for trimmed ATE are extremely overoptimistic. As we alluded to in the previous paragraph, most of their average coverages are below 0.90–far away from the targeted nominal 0.95 coverage level.

Overall, these results suggest that the equipoise estimators (OW, ME, and EW) outperformed the IPW (with and without trimming). When there is no or few outlying observations, the former either weight more ATT or ATC proportional to $c_1(1 - p)ATT + c_2pATC$. The weight depends on which of the treatment groups has the smaller proportions of participants—and factors in attributes ($c_1, c_2$) of the ratio $r$ of variances of the PSs between the treatment groups. Such a weight assignment goes contrary to what IPW without trimming does. Our simulations also confirm the subjective results one can obtain with trimming: the more we trim the less sure we are as to whether we are improving the bias-variane trade-off or we are making it worse. Finally, the simulations also demonstrate that the augmented equipoise estimators $\tilde{\tau}_{\text{aug}}^g$ are more robust to model misSpecifications, whenever $p$ is extremely large or small and ratio of variances $r \approx 1$. We reached similar conclusions with the full set of our simulation results provided in Supplemental Material B.2.

In addition, the effective sample sizes in Table B.1.3 concur also with the overall conclusion of our simulations. Equipoise weights yield better effective sample sizes (to make up for the treatment allocation imbalances), when estimating treatment effects. For instance, in Model 1 (with about 10% treated participants in the original sample), the ESS was 52.35%, 69.12%, 60.32%, 45.73% with, respectively, IPW and IPW trimming at 0.05, 0.10, and 0.15, while the ESS was equal to 99.25%, 101.14%, and 94.15% with OW, MW and EW, respectively.

5 Data Application

We evaluate racial disparities in the health care expenditure using data from the Medical Expenditure Panel Survey (MEPS) (https://www.meps.ahrq.gov/mepsweb/). We focus on three specific 2-by-2 comparisons: White vs. Hispanic, White vs. Black, and White vs. Asian, with White as the reference group ($Z = 1$) and the minority racial or ethnic group as control ($Z = 0$). The proportion $p$ of White participants was, respectively, 65.06% (vs. Hispanic), 70.97% (vs. Black), and 87.18% (vs. Asian). We run separate logistic regression models to estimate PSs and linear regression models for the outcome models, with 31 covariates (4 continuous and 27 categorical
variables). Details on the data analysis are provided in Supplemental Material C.

| Comparison | N    | Minimum | 25-th Quantile | Median | Mean  | 75-th Quantile | Maximum |
|------------|------|---------|----------------|--------|-------|----------------|---------|
| White      | 9830 | 0.14    | 0.58           | 0.71   | 0.70  | 0.84           | 1.00    |
| Hispanic   | 5280 | 0.11    | 0.44           | 0.55   | 0.56  | 0.67           | 0.99    |
| White      | 9830 | 0.16    | 0.65           | 0.78   | 0.75  | 0.86           | 0.99    |
| Black      | 4020 | 0.04    | 0.51           | 0.62   | 0.62  | 0.75           | 0.99    |
| White      | 9830 | 0.30    | 0.83           | 0.92   | 0.89  | 0.97           | 1.00    |
| Asian      | 1446 | 0.22    | 0.68           | 0.78   | 0.77  | 0.87           | 1.00    |

Figure 5: MEPS Data: Propensity score estimates distributions and summary statistics.

Figure C.0.1 shows the estimated PS distributions for White ($Z = 1$) vs. a minority group ($Z = 0$), along with the summary statistics. Overall, the histograms of the PSs indicate good overlap. Nevertheless, a number of control participants have PSs near 1 in all three comparisons, especially for the White-Asian tandem. Most Asian participants have a PS greater than 0.6 and a substantial number of them have their PSs near 1. This indicates that some of participants in the minority groups (Hispanic, Black, and Asian) will have large weights in each of comparisons of interest.

Figure 6: Racial disparities in the health care expenditure

Figure 6 shows the Hájek-type and augmented estimates of ATE, ATT, ATC, ATO, ATM and ATEN from the three comparisons, with the point estimate and the corresponding 95% confidence interval. The results indicate significant health expenditure differences across racial and ethnic groups compare to White participants. The only exception is the ATT estimates in White vs. Hispanic: on average, White participants pay $699.12 (see Table C.0.2) more in health care expenditure than Hispanic participants.

As expected, the ATE estimates align with the ATT estimates while equipoise estimates (ATO, ATM and ATEN) are closer to ATC estimates. What is remarkable is the fact that the estimates of ATO, ATM, and ATM remained on the same ballpark (augmented or not) within each of the 2-by-2 comparisons. The estimates are in the range of $1202.48–1285.72 for White-Hispanic, $814.78–$841.77 for White-Black, and $1229.33–$1400.46 for White-Black (see Table C.0.2). In contrast, the estimates of ATE (IPW without trimming) was $699.12 using...
Hájek-type estimator and $1154.44 using the double-robust estimator when we compare White vs. Hispanic, $850.82 and $ 992.82 for White vs. Black, and finally $2253.00 and $4712.69 for White vs. Asian.

6 Discussion and conclusion

6.1 Summary

In studies where the lack of positivity is highly likely (or not), or there is an imbalance in the treatment allocation (i.e., proportion of treated participants is too small or too large), the standard inverse probability weighting (IPW) method can assign disproportionately extreme weights to a handful of participants and yield a less reliable treatment effect estimate with large variance. Practitioners of statistics often face a dilemma when deciding which path to follow: to weight or not to weight? And if they opt for weighting, which weights to use? Indeed, there is a confusion about how to weight, which weights to choose, and whether should this be done along with trimming. While weighting can be used to target different estimands, IPW is commonly used to target the average treatment effect (ATE), the average treatment effect on the treated (ATT), or the average treatment effect on the control (ATC). Both ATT or ATC are often estimated for policy evaluation, where the IPW is a specific type of weights to go after (sub)population treatment effects (Greifer and Stuart, 2021).

In this paper, we provided a coherent assessment of the different estimands of the PS weighting methods to dispel confusion we may have about their use, when there is a violation of the positivity assumption. We examined the analytic solutions that can be explore when there is a violation of the positivity assumption. Then, we demonstrated that when the proportion of participants is either too small or too large, commonly-sought-after estimands and treatment effects are inherently group-specific, aiming for specific subpopulations, which implicitly move the goalposts. Furthermore, we demonstrated why and how ATE estimators can fail to identify logical treatment effect estimands and why using IPW trimming is not always a good idea. Finally, we show that the equipoise estimators (matching, overlap, and matching weights) are more flexible and have the ability to deliver robust results, when there is a violation of the positivity or important imbalance in treatment allocations. We used a series of Monte-Carlo simulations to examine the performance of the different estimators and to highlight the shortcomings of the estimators of ATE based on IPW (with and without trimming), when there is violations of the positivity assumption or in the presence of treatment allocation imbalance. We also examine the performance of the corresponding augmented estimators, especially in the presence of underlying model misspecifications. Our simulations confirmed where ATE estimator via IPW falls in the ATT–ATC spectrum compared to equipoise estimators (OW, MW, and EW), when there is an important imbalance in treatment allocations.

When there are violations of the positivity assumption or when the proportion of participant in the treatment group, \( p \), is small (or large), one should use ATE with caution as it may yield an estimate that is substantially different from what we have in mind, unless we assume that the treatment effect is constant. However, even under this assumption, the ATE estimate can still be biased due to the influence of extreme weights. Our findings demonstrate that the equipoise estimators (OW, MW, and EW) have better performance when extreme weights are present due to the lack of adequate positivity or important imbalance in treatment allocation, which aligns with prior studies (Li et al., 2018a,b; Zhou et al., 2020b; Matsouaka and Zhou, 2020). The advantages of the
equipoise estimators is that they smoothly and gradually diminish the weights (i.e., the influence) of observations whose $e(x)(1 - e(x))$ is closer to 0. They do not require a user-specified threshold and target the subpopulation patients for whom there is equipoise. Nevertheless, in our study we found that IPW estimator of ATE without trimming and equipoise treatment effect estimators have comparable performance when the treatment allocation was nearly balanced ($p = 45.92\%$). However, when $p = 20.77\%$ and $p = 79.59\%$, we have noticed drastically different behaviors among all the IPW estimators of ATE (with or without trimming), those of ATT and ATC and the equipoise estimators (OW, MW, and EW). We concluded that the differences in estimating the treatment effects between the two methods were heightened by the presence of extreme weights as well as the tails of the treatment allocation proportions (i.e., small or large $p$). Although one may think of making their job easier by targeting ATT (or ATC), our study indicates that this might also be a bad idea. While choosing ATT (or ATC) results in relatively smaller absolute relative percentage bias, the root mean squared errors and more importantly, the coverage probability will be greatly and negatively affected whenever $p \leq 20.30\%$ or $p \geq 79.59\%$. This conundrum is well illustrated in our data application, particularly in the standard errors of ATE and ATT for the White-Asian comparison.

In practice, heterogeneity of treatment effects is the norm rather than the exception; we should estimate our treatment effect(s) under these premises. Thus, it is important to have a better understanding of our data and the target population to whom your results will be applied to. “What are we weighting for?” is thus a call to seriously examine the reasons we use specific weighting schemes and to make sure that (most of) the related assumptions are satisfied. In this context, it is worth asking questions that can shed light on the factors contributing to the lack of adequate positivity or a small $p$. For instance, does the lack of positivity or reason for a small $p$ is just related to our specific sample? In other word, do we have a case of random violation of the positivity assumptions? Does our sample happen to have an allocation imbalance, but not target population? Imbalance allocations from perfectly well balance target population are often used in education research interventions. Most studies tend to enroll a smaller number of participants than can be found in the general population (Terada, 2020; Bartlett et al., 2017). Imbalance allocation can just be a characteristic of the target population (e.g., unusual occupational exposures or exposure to toxic chemicals, see Hall et al. (2020); Checkoway et al. (2004)) or a dynamic phenomenon in the study of a drug (e.g., early users of a newly released drug—with the promised of improved effectiveness or safety—versus broaden user base after a longer period of time as mentioned by Schneeweiss et al. (2011))

Our work sheds light on some of the criticisms raised in the early days of PS methods, often caricatured through Basu’s elephant analogy (Basu, 2011), when applications of IPW led to extreme weights, unstable and often questionable causal estimates. These issues, as we now have demonstrated, go beyond the presence of extreme weights to also include the influence of treatment allocations imbalances and the mitigating role the ratio $r$ of the variances of PSs (between the treatment groups) can have. In addition, from a statistical standpoint, the question can be framed as: when $p$ is extremely small (or large), what comparisons ATE gives rise to? The ATE estimand and the related IPW estimation method(s) should not be the default approach, as if we have a hammer and view everything as a nail. Because ATE is the average treatment effect in the entire population if all participants were treated vs. if all participants were untreated, we need to ask ourselves probing and fundamental questions as the one above. We should be clear about our estimand of interest before we start analyzing the data,
based on subject matter knowledge and our scientific question. This allows us to be intentional and explicitly make meaningful and appropriate comparisons; do not compare “apples” to “oranges”.

Finally, each estimand that is estimated via any weighting method comes with a specific set of weights and targets a specific population. We must choose our estimand and corresponding weights wisely to recover the estimated or specific causal effects (or parameters) of interest that align with our scientific question(s). This will help better interpret the results we obtain. Similarly, we should be careful of using trimming blindly as it can be problematic. Ignorance in this context can be costly: trimming leads to biased estimation of the ATE and calls for a different estimand (Crump et al., 2006), unless one uses bias-corrected IPW trimming methods (Chaudhuri and Hill, 2014; Sasaki and Ura, 2022; Ma and Wang, 2020). We have indicated that the above biased-corrected ATE estimators are reliable only when there is random violation of the positivity assumption. When such a violation is structural, there is no guarantee that these methods would lead to consistent estimators of ATE. In addition, their asymptotic behaviors are unknown when there is a structural violation or when there is an important imbalance in treatment allocation.

6.2 Limitations and perspectives

The methods evaluated and compared in our study may have limitations, including but not limited to the following. First, to be concise, we did not elaborate on the impact of the ratio $r$. A thorough and granular investigation needs to be conducted to complement the work presented in this paper. Second, the sandwich variance estimation method is not always without drawbacks. It is possible to face challenges when there is “non-regularity”, whether due to chance or structural issues. Empirical studies, such as those discussed in Matsouaka et al. (2023), have demonstrated that when the sample size is limited and PS overlap between groups is poor, sandwich variance estimation may not be feasible. We also encountered this issue in our data analysis in Section 5. In practice, when sandwich variance estimation fails, people may turn to bootstrap for help. However, bootstrap approaches have their own potential drawbacks. For instance, when dealing with big data, bootstrapping can become computationally time-consuming. There can also be random violations of positivity in some bootstrap replications. Moreover, in cases where the tilting function contains non-smooth and jumping points, such as ATE trimming, the sandwich variance is expected to fail, even though it is still used in practice for this cases (e.g., the implementation of PSweight package (Zhou et al., 2020a)). Therefore, there is a need for an in-depth assessment of different variance estimation methods. We should also consider the development of alternative approaches that are flexible, computationally efficient, and robust for estimating WATE estimators. This could involve exploring ideas from other nonparametric resampling techniques, such as the wild bootstrap using semiparametric efficient influence functions (see Matsouaka et al. (2023)).

In addition, there is currently no doubly robust estimator proposed for WATE with a general tilting function. Double robustness is only applicable for the special cases of the tilting functions of ATE, ATT, and ATC. For the equipoise estimators (ATO, ATM, and ATEN), empirical studies suggest that the augmented estimators are still nearly double robust. However, the existing asymptotic analyses have not provide yet any theoretical justification of their double robustness. As part of our future research, we will explore the development of potentially doubly or multiply robust estimators while maintaining good efficiency, under certain regularity conditions.
Finally, although our study considered a number of useful alternatives for ATE when there is lack of positivity, we have not considered developing robust bias-corrected methods that still target ATE when positivity is violated, either randomly or structurally. Instead of just trimming observations or truncating their weights in regions of poor overlap, i.e., where \( e(x)(1 - e(x)) \approx 0 \) or use equipoise estimators, we may need to preserve an overall population level-inference (Nethery et al., 2019). In this case, one can first estimate the treatment effect in regions with good overlap, then in a principled manner extrapolate outside of these regions (if possible), and obtain an adequate estimate of a population level estimand, such as ATE, with less bias and better efficiency. Chaudhuri and Hill (2014), Nethery et al. (2019), Ma and Wang (2020), and Sasaki and Ura (2022) provided several strategies to better handle bias that may be induced by trimming. We can consider similar proposals, emulate their ideas and extend the methods to deal with both random and structural violations of the positivity assumption.

6.3 Conclusion

Several papers have investigated issues related to violations of positivity assumptions or imbalances in treatment allocation when using the inverse probability weighting methods. When using any weighting method, it is essential to ask yourself: what are you weighting for? You must be aware of what you ultimately get when using a specific weighting method and what estimand you are targeting. The ATE estimation via IPW, thanks to its many shortcomings, may not lead you where you expect to land. However, equipoise estimators (ATO, ATM, and ATEN) judiciously take you to the overlap/equipoise land, providing estimates of the treatment effect on the subgroup of participants for whom there is clinical equipoise (Matsouaka and Zhou, 2020).

References

Peter C Austin. A tutorial and case study in propensity score analysis: an application to estimating the effect of in-hospital smoking cessation counseling on mortality. Multivariate behavioral research, 46(1):119–151, 2011.

Peter C Austin, Amy Ying Xin Yu, Manav V Vyas, and Moira K Kapral. Applying propensity score methods in clinical research in neurology. Neurology, 97(18):856–863, 2021.

Robin Bartlett, Tiffany Wright, Tia Olarinde, Tara Holmes, Emily R Beamon, and Debra Wallace. Schools as sites for recruiting participants and implementing research. Journal of community health nursing, 34(2):80–88, 2017.

Debabrata Basu. An essay on the logical foundations of survey sampling, part one. Selected Works of Debabrata Basu, pages 167–206, 2011.

Saraswata Chaudhuri and Jonathan B Hill. Heavy tail robust estimation and inference for average treatment effects. Technical report, Working paper, 2014.

Harvey Checkoway, Neil Pearce, and David Kriebel. Research methods in occupational epidemiology, volume 34. Monographs in Epidemiology and, 2004.
Richard Crump, V Joseph Hotz, Guido Imbens, and Oscar Mitnik. Moving the goalposts: Addressing limited overlap in the estimation of average treatment effects by changing the estimand. *National Bureau of Economic Research*, 2006.

Jessica M Franklin, Wesley Eddings, Peter C Austin, Elizabeth A Stuart, and Sebastian Schneeweiss. Comparing the performance of propensity score methods in healthcare database studies with rare outcomes. *Statistics in medicine*, 36(12):1946–1963, 2017.

Bryan S Graham, Cristine Campos de Xavier Pinto, and Daniel Egel. Inverse probability tilting for moment condition models with missing data. *The Review of Economic Studies*, 79(3):1053–1079, 2012.

Noah Greifer and Elizabeth A Stuart. Choosing the estimand when matching or weighting in observational studies. *arXiv preprint arXiv:2106.10577*, 2021.

Jens Hainmueller. Entropy balancing for causal effects: A multivariate reweighting method to produce balanced samples in observational studies. *Political Analysis*, 20(1):25–46, 2012.

David Hajage, Florence Tubach, Philippe Gabriel Steg, Deepak L Bhatt, and Yann De Rycke. On the use of propensity scores in case of rare exposure. *BMC medical research methodology*, 16(1):38, 2016.

J Hájek. Comment on a paper by d. basu in: Godambe VP and sprott DA (eds) foundations of statistical inference, 1971.

Amy L Hall, Mary Beth MacLean, Linda VanTil, David Iain McBride, and Deborah C Glass. Considering exposure assessment in epidemiological studies of chronic health in military populations. *Frontiers in Public Health*, page 574, 2020.

Keisuke Hirano, Guido W Imbens, and Geert Ridder. Efficient estimation of average treatment effects using the estimated propensity score. *Econometrica*, 71(4):1161–1189, 2003.

David A Hirshberg and José R Zubizarreta. On two approaches to weighting in causal inference. *Epidemiology*, 28(6):812–816, 2017.

Paul W Holland. Statistics and causal inference. *Journal of the American statistical Association*, 81(396):945–960, 1986.

Kosuke Imai and Marc Ratkovic. Covariate balancing propensity score. *Journal of the Royal Statistical Society: Series B (Statistical Methodology)*, 76(1):243–263, 2014.

Guido W Imbens and Donald B Rubin. *Causal inference in statistics, social, and biomedical sciences*. Cambridge University Press, 2015.

Fan Li, Kari Lock Morgan, and Alan M Zaslavsky. Balancing covariates via propensity score weighting. *Journal of the American Statistical Association*, 113(521):390–400, 2018a.

Fan Li, Laine E Thomas, and Fan Li. Addressing extreme propensity scores via the overlap weights. *American journal of epidemiology*, 188(1):250–257, 2018b.
Liang Li and Tom Greene. A weighting analogue to pair matching in propensity score analysis. *The international journal of biostatistics*, 9(2):215–234, 2013.

Yan Li and Liang Li. Propensity score analysis methods with balancing constraints: A monte carlo study. *Statistical Methods in Medical Research*, 30(4):1119–1142, 2021.

Xinwei Ma and Jingshen Wang. Robust inference using inverse probability weighting. *Journal of the American Statistical Association*, 115(532):1851–1860, 2020.

Huzhang Mao, Liang Li, and Tom Greene. Propensity score weighting analysis and treatment effect discovery. *Statistical Methods in Medical Research*, page 0962280218781171, 2018.

Roland A Matsouaka and Yunji Zhou. A framework for causal inference in the presence of extreme inverse probability weights: the role of overlap weights. *arXiv preprint arXiv:2011.01388*, 2020.

Roland A Matsouaka, Yi Liu, and Yunji Zhou. Variance estimation for the average treatment effects on the treated and on the controls. *Statistical Methods in Medical Research*, 32(2):389–403, 2023.

Daniel F McCaffrey, Greg Ridgeway, and Andrew R Morral. Propensity score estimation with boosted regression for evaluating causal effects in observational studies. *Psychological methods*, 9(4):403, 2004.

Rachel C Nethery, Fabrizia Mealli, and Francesca Dominici. Estimating population average causal effects in the presence of non-overlap: The effect of natural gas compressor station exposure on cancer mortality. *The annals of applied statistics*, 13(2):1242, 2019.

Jersey Neyman. *Sur les applications de la théorie des probabilités aux experiences agricoles: Essai des principes*. *Roczniki Nauk Rolniczych*, 10:1–51, 1923.

Maya L Petersen, Kristin E Porter, Susan Gruber, Yue Wang, and Mark J van der Laan. Diagnosing and responding to violations in the positivity assumption. *Statistical methods in medical research*, 21(1):31–54, 2012.

Romain Pirracchio, Matthieu Resche-Rigon, and Sylvie Chevret. Evaluation of the propensity score methods for estimating marginal odds ratios in case of small sample size. *BMC medical research methodology*, 12(1):1–10, 2012.

Robert William Platt, Joseph Austin Christopher Delaney, and Samy Suissa. The positivity assumption and marginal structural models: the example of warfarin use and risk of bleeding. *European journal of epidemiology*, 27(2):77–83, 2012.

Jeremy A Rassen and Sebastian Schneeweiss. Newly marketed medications present unique challenges for non-randomized comparative effectiveness analyses. *Journal of Comparative Effectiveness Research*, 1(2):109–111, 2012.

Paul R Rosenbaum and Donald B Rubin. The central role of the propensity score in observational studies for causal effects. *Biometrika*, 70(1):41–55, 1983.
Paul R Rosenbaum and Donald B Rubin. Reducing bias in observational studies using subclassification on the propensity score. *Journal of the American statistical Association,* 79(387):516–524, 1984.

Donald B Rubin. Estimating causal effects from large data sets using propensity scores. *Annals of internal medicine,* 127(8_Part_2):757–763, 1997.

Donald B Rubin. Using propensity scores to help design observational studies: application to the tobacco litigation. *Health Services and Outcomes Research Methodology,* 2(3-4):169–188, 2001.

Jacqueline E Rudolph, David Benkeser, Edward H Kennedy, Enrique F Schisterman, and Ashley I Naimi. Estimation of the average causal effect in longitudinal data with time-varying exposures: The challenge of non-positivity and the impact of model flexibility. *American Journal of Epidemiology,* 2022.

Yuya Sasaki and Takuya Ura. Estimation and inference for moments of ratios with robustness against large trimming bias. *Econometric Theory,* 38(1):66–112, 2022.

S Schneeweiss. Developments in post-marketing comparative effectiveness research. *Clinical Pharmacology & Therapeutics,* 82(2):143–156, 2007.

Sebastian Schneeweiss, JJ Gagne, RJ Glynn, M Ruhl, and JA Rassen. Assessing the comparative effectiveness of newly marketed medications: methodological challenges and implications for drug development. *Clinical Pharmacology & Therapeutics,* 90(6):777–790, 2011.

Leonard A Stefanski and Dennis D Boos. The calculus of m-estimation. *The American Statistician,* 56(1):29–38, 2002.

Elizabeth A Stuart. Matching methods for causal inference: A review and a look forward. *Statistical science: a review journal of the Institute of Mathematical Statistics,* 25(1):1, 2010.

Youki Terada. The 10 most significant education studies of 2020, Dec 2020. URL https://www.edutopia.org/article/10-most-significant-education-studies-2020.

Laine E Thomas, Fan Li, and Michael J Pencina. Overlap weighting: A propensity score method that mimics attributes of a randomized clinical trial. *Jama,* 2020.

Daniel Westreich. *Epidemiology by design: a causal approach to the health sciences.* Oxford University Press, 2019.

Raymond KW Wong and Kwun Chuen Gary Chan. Kernel-based covariate functional balancing for observational studies. *Biometrika,* 105(1):199–213, 2017.

Tianhui Zhou, Guangyu Tong, Fan Li, and Laine E Thomas. Psweight: An r package for propensity score weighting analysis. *arXiv preprint arXiv:2010.08893,* 2020a.

Yunjie Zhou, Roland A Matsouaka, and Laine Thomas. Propensity score weighting under limited overlap and model misspecification. *Statistical Methods in Medical Research,* 29(12):3721–3756, 2020b.
José R Zubizarreta. Stable weights that balance covariates for estimation with incomplete outcome data. *Journal of the American Statistical Association*, 110(511):910–922, 2015.
### A Appendix: Technical Proofs

#### A.1 Sandwich variance estimation

In this section we calculate the variance estimation of the ATE, ATT, and ATC using M-theory (see Stefanski and Boos (2002)). The estimator \( \hat{\tau}_g \) is derived using an estimating equation of the form 
\[
0 = \sum_{i=1}^{N} \Psi_\theta(X_i, Z_i, Y_i)
\]
for which \( \hat{\theta} \) is solution to the equation and the estimator \( \hat{\tau}_g \) is a linear combination of the components of the estimator \( \hat{\theta} \), for some matrix \( \Psi_\theta(X_i, Z_i, Y_i) \).

Using \( E[\Psi_\theta(X_i, Z_i, Y_i)] = 0 \), we have \( \hat{\theta} \xrightarrow{p} \theta \) when \( N \rightarrow \infty \) under some regularity conditions (Stefanski and Boos, 2002), and then we can conclude that the estimator \( \hat{\tau}_g \) is consistent by Slutsky’s theorem. In addition, 
\[
\sqrt{N}(\hat{\theta} - \theta) \xrightarrow{d} N(0, \Sigma(\theta)),
\]
with \( \Sigma(\theta) = A(\theta)^{-1}B(\theta)[A(\theta)^{\prime}]^{-1} \).
A consistent estimator of the asymptotic covariance matrix \( \Sigma(\theta) \) is \( \hat{\Sigma}(\hat{\theta}) = A_N(\hat{\theta})^{-1}B_N(\hat{\theta})[A_N(\hat{\theta})^{\prime}]^{-1} \), where \( A(\theta), B(\theta), A_N(\hat{\theta}) \) and \( B_N(\hat{\theta}) \) are the following matrices:

\[
A_N(\hat{\theta}) = -\frac{1}{N} \sum_{i=1}^{N} \frac{\partial \Psi_\theta(X_i, Z_i, Y_i)}{\partial \theta} : B_N(\hat{\theta}) = \frac{1}{N} \sum_{i=1}^{N} \Psi_\theta(X_i, Z_i, Y_i)\Psi_\theta(X_i, Z_i, Y_i)^{\prime} \bigg|_{\theta = \hat{\theta}},
\]

where \( A_N(\hat{\theta}) \xrightarrow{p} A(\theta) = -E_0 \left[ \frac{\partial \Psi_\theta(X_i, Z_i, Y_i)}{\partial \theta} \right] \) and \( B_N(\hat{\theta}) \xrightarrow{p} B(\theta) = E_0[\Psi_\theta(X_i, Z_i, Y_i)\Psi_\theta(X_i, Z_i, Y_i)^{\prime}] \), as \( N \rightarrow \infty \), by the weak law of large numbers and the consistency of \( \hat{\theta} \).

As an illustrative example, we provide the matrices \( A_N(\hat{\theta}) \) and \( B_N(\hat{\theta}) \) when the propensity score model \( \epsilon(x_i; \hat{\beta}) = P(Z = 1|X_i = x_i; \hat{\beta}) \) and the regression models \( m_z(x_i) = m(x_i; \hat{\alpha}_z) \) for \( z = 0 \) and \( z = 1 \) are estimated by maximum likelihood using, respectively, the logistic and linear regression models. We consider different combinations (or subsets) of covariates \( X \) that are entered into the logistic and regression models, which we denote them by \( V \) and \( W \), respectively.

#### A.1.1 Variance for the Hájek-type weighted estimator

For the weighted average treatment effect (WATE) estimator, we have

\[
\hat{\tau}_g = \sum_{i=1}^{N} \left[ \frac{Z_i \tilde{w}_1(x_i)}{N \tilde{w}_1} - \frac{(1 - Z_i) \tilde{w}_0(x)}{N \tilde{w}_0} \right] Y_i,
\]

with \( N \tilde{w}_k = \sum_{i=1}^{N} Z_i^k (1 - Z_i)^{1-k} \tilde{w}_k(x_i) \), \( k = 0, 1 \).

where \( \tilde{w}_k(x) = \tilde{g}(x) \tilde{c}(x)^{-k} (1 - \tilde{c}(x))^{k-1} \).

The estimated propensity score parameter vector \( \hat{\beta} \) and the estimator \( (\hat{\mu}_{1g}, \hat{\mu}_{0g}) \) are derived as solutions to the estimating equation

\[
0 = \sum_{i=1}^{N} \Psi_\theta(X_i, Z_i, Y_i) = \sum_{i=1}^{N} \begin{bmatrix} \psi_{\beta}(X_i, Z_i) \\ \psi_{\mu_{1g}}(X_i, Z_i, Y_i) \\ \psi_{\mu_{0g}}(X_i, Z_i, Y_i) \end{bmatrix} = \sum_{i=1}^{N} \begin{bmatrix} \psi_{\beta}(X_i, Z_i) \\ Z_i w_1(X_i)(Y_i - \mu_{1g}) \\ (1 - Z_i) w_0(X_i)(Y_i - \mu_{0g}) \end{bmatrix}
\]

with respect to \( \theta = (\beta', \mu_{1g}, \mu_{0g})' \) where \( \tilde{\tau}_g = c_0' \theta = \tilde{\mu}_{1g} - \tilde{\mu}_{0g} \) and \( c_0 = (0, 1, -1)' \).
The matrices $A(\theta)$, $A_N(\hat{\theta})$, $B(\theta)$ and $B_N(\hat{\theta})$ are

$$A_N(\hat{\theta}) = N^{-1}\sum_{i=1}^{N} \left[ -\frac{\partial}{\partial \theta} \Psi_\theta(X_i, Z_i, Y_i) \right]_{\theta = \hat{\theta}} = \begin{bmatrix} \hat{A}_{11} & 0 & 0 \\ \hat{A}_{21} & \hat{A}_{22} & 0 \\ \hat{A}_{31} & 0 & \hat{A}_{33} \end{bmatrix}.$$ 

If we estimate the propensity scores via a logistic regression model $e(X_i) = [1 + \exp(-V_i'\beta)]^{-1}$, we have $\hat{\psi}_\theta(X_i, Z_i) = [Z_i - e(V_i; \beta)]V_i$. The components of the matrix $A_N$ are given by

$$\hat{A}_{11} = N^{-1} \sum_{i=1}^{N} \hat{e}(v)(1 - \hat{e}(v))V_i'$$

$$\hat{A}_{21} = -N^{-1} \sum_{i=1}^{N} Z_i \left[ \left( \frac{\partial g(V_i)}{\partial \beta} \right)_{\beta = \hat{\beta}} - (1 - \hat{e}(v))\hat{g}(V_i)V_i' \right] \hat{e}(v)^{-1}(Y_i - \hat{\mu}_{1\theta});$$

$$\hat{A}_{31} = -N^{-1} \sum_{i=1}^{N} (1 - Z_i) \left[ \left( \frac{\partial g(V_i)}{\partial \beta} \right)_{\beta = \hat{\beta}} + \hat{e}(v)\hat{g}(V_i)V_i' \right] (1 - \hat{e}(v))^{-1}(Y_i - \hat{\mu}_{0\theta});$$

$$\hat{A}_{22} = N^{-1} \sum_{i=1}^{N} Z_i\hat{e}(v)^{-1}\hat{g}(V_i); \quad \hat{A}_{33} = N^{-1} \sum_{i=1}^{N} (1 - Z_i)(1 - \hat{e}(v))^{-1}\hat{g}(V_i).$$

Therefore, an estimator of the variance of $\tau_\theta$ is $\sqrt{\text{Var}(\hat{\tau}_\theta)} = N^{-1} \hat{c}_0 \hat{\Sigma}(\hat{\theta})c_0$.

Note that $\left[ \frac{\partial g(V_i)}{\partial \beta} \right]_{\beta = \hat{\beta}}$ is equal to 0 for ATE, equal to $e(v)[1 - e(v)]V_i'$ for ATT and to $-e(v)[1 - e(v)]V_i'$ for ATC.

### A.1.2 Variance for the doubly robust ATE, ATT, and ATC

The doubly robust estimators for ATE, ATT, and ATC are given, respectively, by

$$\hat{\tau}_{ATE}^d = \sum_{i=1}^{N} Z_i \hat{e}(x_i)^{-1} \{ Y_i - \hat{m}_1(x_i) \} - \sum_{i=1}^{N} \{ 1 - Z_i \}(1 - \hat{e}(x_i))^{-1}\{ Y_i - \hat{m}_0(x_i) \}$$

$$+ \frac{1}{N} \sum_{i=1}^{N} \{ \hat{m}_1(x_i) - \hat{m}_0(x_i) \}; \quad (A.1.1)$$

$$\hat{\tau}_{ATT}^d = \sum_{i=1}^{N} Z_i \{ Y_i - \hat{m}_0(x_i) \} - \sum_{i=1}^{N} \{ 1 - Z_i \}\hat{e}(x_i)(1 - \hat{e}(x_i))^{-1}\{ Y_i - \hat{m}_0(x_i) \}; \quad (A.1.2)$$

$$\hat{\tau}_{ATC}^d = \sum_{i=1}^{N} Z_i \hat{e}(x_i)^{-1}(1 - \hat{e}(x_i)) \{ Y_i - \hat{m}_1(x_i) \} - \sum_{i=1}^{N} \{ 1 - Z_i \} \{ Y_i - \hat{m}_1(x_i) \}$$

$$\sum_{i=1}^{N} (1 - Z_i); \quad (A.1.3)$$

In addition to the above function $\psi_\theta(X_i, Z_i)$, we also consider the estimating functions $\psi_{\alpha_z}(X)$ for the regression models $m_z(X) = m_z(X; \alpha_z)$, $z = 0, 1$. Let $c_1 = (0, 0, 0, 1, -1, 1, -1)'$; we can derive the estimator $\hat{\tau}_{ATE} = c_1 \hat{\theta}_{ate} = \ldots$
\[ \tau_{1g}^m - \tau_{0g}^m + \mu_{1g} - \mu_{0g} \text{ through } \hat{\theta}_{atc} = (\hat{\beta}', \hat{\alpha}', \hat{\alpha}_0', \tau_{1g}^m, \tau_{0g}^m, \hat{\mu}_{1g}, \hat{\mu}_{0g})' \],

the solution to the estimating equation

\[
\sum_{i=1}^{N} \Psi_{\theta_{atc}}(X_i, Z_i, Y_i) = \sum_{i=1}^{N} \begin{bmatrix}
\psi_\beta(X_i, Z_i) \\
Z_i \psi_{\alpha_1}(X_i, Y_i) \\
(1 - Z_i) \psi_{\alpha_0}(X_i, Y_i) \\
m_1(X_i) - \tau_1^m \\
m_0(X_i) - \tau_0^m \\
Z_i e(x_i)^{-1} (Y_i - m_1(X_i) - \mu_{1g}) \\
(1 - Z_i)(1 - e(x_i))^{-1} (Y_i - m_0(X_i) - \mu_{0g})
\end{bmatrix} = 0
\]

with respect to \( \theta_{atc} = (\beta', \alpha', \alpha'_0, \tau_{1g}^m, \tau_{0g}^m, \mu_{1g}, \mu_{0g})' \).

If we estimate \( e(X) \) and \( m_s(X) \) using logistic and linear regression models \( e(V_i) = [1 + \exp(-V_i'\beta)]^{-1} \) and \( m_s(W_i) = W_i'\alpha_s, \) \( z = 0, 1, \) then \( \psi_\beta(X_i, Z_i) = [Z_i - e(V_i; \beta)]V_i \) and \( \psi_{\alpha_s}(X_i, Z_i) = W_i(Y_i - W_i'\alpha_s). \) Assuming that the same covariates appear as predictors in the regression models \( m_s(W) \), the non-zero components \( \hat{A}_{ij} \) of the matrix \( A_N \) are given by

\[
\begin{align*}
\hat{A}_{11} &= N^{-1} \sum_{i=1}^{N} \hat{e}_i(v)(1 - \hat{e}_i(v))V_i'V_i' \\
\hat{A}_{22} &= N^{-1} \sum_{i=1}^{N} Z_i W_i W_i' \\
\hat{A}_{33} &= N^{-1} \sum_{i=1}^{N} (1 - Z_i) W_i W_i'
\end{align*}
\]

An estimator of \( \Sigma(\theta_{atc}) \) is then \( \hat{\Sigma}(\hat{\theta}_{atc}) = A_N(\hat{\theta}_{atc})^{-1}B(\hat{\theta}_{atc})\{A(\hat{\theta}_{atc})'\}^{-1} \), from which we can derive the variance of \( \tau_{ATE}^{e_{1x}} \) as \( \text{Var}(\tau_{ATE}^{e_{1x}}) = N^{-1}c_1\hat{\Sigma}(\hat{\theta}_{atc})c_1 \).

For the ATT estimator \( \tau_{ATE}^{m_{1x}} \), we can use the solution to the estimating equation

\[
\sum_{i=1}^{N} \Psi_{\theta_{att}}(X_i, Z_i, Y_i) = \sum_{i=1}^{N} \begin{bmatrix}
\psi_\beta(X_i, Z_i) \\
(1 - Z_i) \psi_{\alpha_0}(X_i, Y_i) \\
Z_i (Y_i - m_0(X_i) - \mu_{0g}) \\
(1 - Z_i)e(x_i)(1 - e(x_i))^{-1} (Y_i - m_0(X_i) - \mu_{0g})
\end{bmatrix} = 0
\]

with respect to \( \theta_{att} = (\beta', \alpha'_0, \mu_{1g}, \mu_{0g})' \), to calculate \( \tau_{ATE}^{m_{1x}} = c_2\hat{\theta}_{att} = \hat{\mu}_{1g} - \hat{\mu}_{0g} \) where \( c_2 = (0, 0, 1, -1) \) and \( \hat{\theta}_{att} = (\hat{\beta}', \hat{\alpha}_0', \hat{\mu}_{1g}, \hat{\mu}_{0g})' \).

When \( e(X) \) and \( m_s(X) \) are estimated via maximum likelihood based on logistic and linear regression models \( e(V_i) = [1 + \exp(-V_i'\beta)]^{-1} \) and \( m_s(W_i) = W_i'\alpha_s, \) \( z = 0, 1, \) then \( \psi_\beta(X_i, Z_i) = [Z_i - e(V_i; \beta)]V_i \) and \( \psi_{\alpha_s}(X_i, Z_i) = W_i(Y_i - W_i'\alpha_s) \).
\[ W_i(Y_i - W_0') \]. Assuming that the same covariates appear as predictors in the regression models \( m_z(W) \), the non-zero components \( \hat{A}_{ij} \) of the matrix \( A_N \) are given by

\[
\hat{A}_{11} = N^{-1} \sum_{i=1}^{N} \hat{e}_i(v)(1 - \hat{e}_i(v))V_i V_i'; \\
\hat{A}_{22} = N^{-1} \sum_{i=1}^{N} Z_i W_i W_i'; \\
\hat{A}_{32} = N^{-1} \sum_{i=1}^{N} Z_i W_i'; \\
\hat{A}_{44} = N^{-1} \sum_{i=1}^{N} Z_i.
\]

The variance of \( \tau_{ATE}^{st} \) is then estimated via \( \bar{V}ar(\tau_{ATE}^{st}) = N^{-1}c_2^2\hat{\Sigma}(\hat{\theta}_{atc})c_2 \).

Finally, for the ATC, the estimators \( \hat{\tau}_{ATC}^{st} \) can be derived by using the solution \( \hat{\theta}_{atc} = (\beta', \alpha'_1, \mu_{1g}, \mu_{0g})' \), to the estimating equation

\[
\sum_{i=1}^{N} \Psi_{\theta_{atc}}(X_i, Z_i, Y_i) = \sum_{i=1}^{N} \begin{bmatrix}
\psi_\beta(X_i, Z_i) \\
Z_i \psi_{\alpha_1}(X_i, Y_i) \\
Z_i e(X_i)^{-1}(1 - e(X_i))(Y_i - m_1(X_i) - \mu_{1g}) \\
(1 - Z_i)(Y_i - m_1(X_i) - \mu_{0g})
\end{bmatrix} = 0
\]

where \( \hat{\tau}_{ATC}^{st} = c_2'\hat{\theta}_{atc} = \tilde{\mu}_{1g} - \tilde{\mu}_{0g} \), with \( \hat{\theta}_{atc} = (\tilde{\beta}', \tilde{\alpha}_1', \tilde{\alpha}_0', \tilde{\mu}_{1g}, \tilde{\mu}_{0g})' \).

In this case, the non-zero components of the matrix \( A_N \) are then

\[
\hat{A}_{11} = N^{-1} \sum_{i=1}^{N} \hat{e}_i(v)(1 - \hat{e}_i(v))V_i V_i'; \\
\hat{A}_{22} = N^{-1} \sum_{i=1}^{N} Z_i W_i W_i'; \\
\hat{A}_{31} = N^{-1} \sum_{i=1}^{N} Z_i \hat{e}_i(v)(1 - \hat{e}_i(v))(Y_i - \tilde{m}_0(W_i) - \tilde{\mu}_{0g})V_i'; \\
\hat{A}_{32} = N^{-1} \sum_{i=1}^{N} Z_i \hat{e}_i(v)(1 - \hat{e}_i(v))W_i; \\
\hat{A}_{33} = N^{-1} \sum_{i=1}^{N} Z_i \hat{e}_i(v)(1 - \hat{e}_i(v)); \\
\hat{A}_{42} = N^{-1} \sum_{i=1}^{N} (1 - Z_i)W_i'; \\
\hat{A}_{44} = N^{-1} \sum_{i=1}^{N} (1 - Z_i).
\]
A.1.3 Sandwich variance for the augmented estimator

For the estimator \( \hat{\tau}_g^{aug} \), we also consider \( c = (0, 0, 0, 1, -1, -1)' \) such that \( \hat{\tau}_g^{aug} = c'\hat{\theta}_g = \tau^m_{1g} - \tau^m_{0g} + \mu_{1g} - \mu_{0g} \), where \( \hat{\theta}_g = (\hat{\beta}', \hat{\alpha}_1', \hat{\alpha}_0', \tau^m_{1g}, \tau^m_{0g}, \mu_{1g}, \mu_{0g})' \) is the solution to the estimating equation

\[
\sum_{i=1}^{N} \Psi_{aug}(X_i, Z_i, Y_i) = \sum_{i=1}^{N} \begin{pmatrix}
\psi(X_i, Z_i) \\
Z_i\psi_{e1}(X_i, Y_i) \\
(1 - Z_i)\psi_{e0}(X_i, Y_i) \\
g(X_i)\{m_1(X_i) - \tau^m_{1g}\} \\
g(X_i)\{m_0(X_i) - \tau^m_{0g}\} \\
Z_iw_1(X_i)(Y_i - m_1(X_i) - \mu_{1g}) \\
(1 - Z_i)w_0(X_i)(Y_i - m_0(X_i) - \mu_{0g})
\end{pmatrix} = 0
\]

with respect to \( \hat{\theta}_g = (\beta', \alpha_1', \alpha_0', \tau^m_{1g}, \tau^m_{0g}, \mu_{1g}, \mu_{0g})' \).

When we estimate the propensity score \( e(X) \) and the regression models \( m_z(X) \) using, respectively, a logistic regression model and a linear regression model, i.e., \( e(V_i) = [1 + \exp(-V_i'\beta)]^{-1} \) and the regression models \( m_z(W_i) = W_i'\alpha_z, z = 0, 1 \). Hence, \( \psi_{e1}(X_i, Z_i) = [Z_i - e(V_i; \beta)]V_i \) and \( \psi_{e0}(X_i, Z_i) = W_i(Y_i - W_i'\alpha_z) \). Assuming that the same covariates appear as predictors in the regression models \( m_z(W) \), the non-zero components \( \hat{A}_{ij} \) of the matrix \( A_N \) are given by

\[
\begin{align*}
\hat{A}_{11} &= N^{-1} \sum_{i=1}^{N} (1 - \hat{e}_i(v))V_i'V_i' \\
\hat{A}_{22} &= N^{-1} \sum_{i=1}^{N} Z_iW_iW_i' \\
\hat{A}_{33} &= N^{-1} \sum_{i=1}^{N} (1 - Z_i)W_iW_i'; \\
\hat{A}_{41} &= -N^{-1} \sum_{i=1}^{N} \left[ \frac{\partial g(V_i)}{\partial \beta} \right]_{\beta = \hat{\beta}} \{\hat{m}_1(W_i) - \tau^m_{1g}\}; \\
\hat{A}_{42} &= \hat{A}_{53} = -N^{-1} \sum_{i=1}^{N} \hat{g}(V_i)W_i'; \\
\hat{A}_{44} &= \hat{A}_{55} = N^{-1} \sum_{i=1}^{N} \hat{g}(V_i); \\
\hat{A}_{51} &= -N^{-1} \sum_{i=1}^{N} \left[ \frac{\partial g(V_i)}{\partial \beta} \right]_{\beta = \hat{\beta}} \{\hat{m}_0(W_i) - \tau^m_{0g}\}; \\
\hat{A}_{61} &= -N^{-1} \sum_{i=1}^{N} Z_i \left[ \frac{\partial g(V_i)}{\partial \beta} \right]_{\beta = \hat{\beta}} \{\hat{m}_1(W_i) - \hat{\mu}_{1g}\}; \\
\hat{A}_{62} &= N^{-1} \sum_{i=1}^{N} Z_iw_1(v)W_i'; \\
\hat{A}_{66} &= -N^{-1} \sum_{i=1}^{N} Z_iw_1(v); \\
\hat{A}_{71} &= -N^{-1} \sum_{i=1}^{N} (1 - Z_i) \left[ \frac{\partial g(V_i)}{\partial \beta} \right]_{\beta = \hat{\beta}} \{\hat{e}_i(v)\hat{g}(V_i)V_i' + (1 - \hat{e}_i(v))\hat{g}(V_i)V_i'\} (1 - \hat{e}_i(v))^{-1} (\hat{Y}_i - \hat{m}_0(W_i) - \hat{\mu}_{0g}); \\
\hat{A}_{73} &= \hat{A}_{77} = -N^{-1} \sum_{i=1}^{N} (1 - Z_i)\hat{w}_0(v).
\end{align*}
\]

An estimator of \( \Sigma(\hat{\theta}_g) \) is then \( \hat{\Sigma}(\hat{\theta}_g) = A_N(\hat{\theta}_g^{-1})B(\hat{\theta}_g)A(\hat{\theta}_g')^{-1} \), from which we can derive the variance of \( \hat{\tau}_g^{aug} = c'\hat{\theta}_g \) as \( \sqrt{\text{det}(\hat{\Sigma}(\hat{\theta}_g))} = N^{-1}c'\hat{\Sigma}(\hat{\theta}_g)c \).
B Appendix: Additional Simulation Details

B.1 Propensity score analysis

We considered 6 propensity score (PS) models for different proportions \((p = P(Z = 1))\) and ratio of variances \((r)\). For each correctly specified PS model, the coefficients \((\beta_0 \text{ to } \beta_7)\) of the PS model via logistic regression, proportion \(p\) as well as the ratio \((r)\) of estimated variance of propensity score of treatment group to control group are provided in table B.1.1.

| Model | \(\beta_0\) | \(\beta_1\) | \(\beta_2\) | \(\beta_3\) | \(\beta_4\) | \(\beta_5\) | \(\beta_6\) | \(\beta_7\) | \(p\) | \(r\) |
|-------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------|-------|
| 1     | -3.07       | 0.3         | 0.4         | 0.4         | -0.1        | -0.1        | 0.1         | 10.05%     | 2.54  |
| 2     | -1.82       | -0.25       | 0.45        | -0.3        | 0.65        | -0.03       | -0.03       | 0.07        | 20.77%| 1.80  |
| 3     | -0.37       | -0.25       | 0.45        | -0.3        | 0.65        | -0.03       | -0.03       | 0.07        | 49.72%| 1.13  |
| 4     | 0.98        | 0.3         | 0.4         | 0.4         | 0.4         | -0.1        | -0.1        | 0.1         | 79.22%| 0.42  |
| 5     | 1.86        | 0.3         | 0.4         | 0.4         | 0.4         | -0.1        | -0.1        | 0.1         | 89.18%| 0.26  |
| 6     | 1.12        | -0.25       | 0.45        | -0.3        | 0.65        | -0.03       | -0.03       | 0.07        | 79.59%| 0.75  |

\(p\): proportion of treated participants; \(r\): ratio of variances of propensity scores (treatment vs. control group).

Figure B.1.1 shows the boxplot of \(r\)'s of the 6 models over the 2000 simulation replicates.

![Figure B.1.1: Ratio of estimated variance of propensity score in treatment group to control group](image)

Figure B.1.1: Ratio of estimated variance of propensity score in treatment group to control group

We also provide the estimated propensity score (PS) distributions of all 6 models in Figure B.1.2. To do so, we randomly pick a sample out of the 2000 simulation replicates and plot the estimated PS histograms using the correctly specified PS model. Table B.1.3 shows the effective sample sizes (ESS) by group of the 6 models, which reflects the performance of different PS weights.

Finally, we give the true values of the heterogeneous treatment effects of all models in table B.1.2.

B.2 Full simulation results

We provide the full set of simulation results of all the models we considered in Tables B.2.1– B.2.6. In each model, the results by Hájek-type (weighted) estimator and the augmented estimator of all weighted average treatment effects (WATE), including ATE with and without trimming (at 0.05, 0.1, 0.15), ATO, ATM, ATEN, ATT and ATC, are reported. For augmented estimators, we considered 4 cases of model specifications: (i) both propensity
### Table B.1.2: True heterogeneous treatment effects by different weighted methods

| Model | \(p\) | \(r\) | ATE | ATE (0.05) | ATE (0.1) | ATE (0.15) | ATO | ATM | ATEN | ATC | ATT |
|-------|------|------|-----|----------|----------|----------|-----|-----|------|-----|-----|
| 1     | 10.05% | 2.54 | 17.22 | 16.61 | 22.79 | 30.46 | 20.53 | 22.28 | 19.26 | 16.62 | 22.59 |
| 2     | 20.77% | 1.80 | 17.22 | 17.15 | 16.90 | 17.08 | 17.72 | 18.33 | 17.55 | 16.81 | 17.83 |
| 3     | 49.72% | 0.42 | 17.22 | 17.20 | 17.12 | 16.91 | 16.69 | 16.30 | 16.81 | 16.61 | 17.83 |
| 4     | 79.22% | 0.26 | 17.22 | 15.28 | 13.48 | 13.87 | 15.39 | 15.81 | 15.55 | 18.58 | 16.86 |
| 5     | 79.59% | 0.75 | 17.22 | 16.75 | 15.78 | 15.77 | 16.56 | 16.63 | 16.63 | 16.70 | 17.35 |

\(p\): percentage of treated participants; \(r\): ratio of variances of propensity scores (treatment vs. control group); ATE: average treatment effect; ATE \((\alpha)\) by trimming PS outside of \(\alpha < x(X) < 1 - \alpha, \alpha = 0.05, 0.1, 0.15\); ATO (resp. ATM, ATEN, ATC, ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated).

### Figure B.1.2: Estimated propensity score histograms by treatment group of models 1–6

### Table B.1.3: Effective sample sizes (ESS) for all propensity score weights (Simulation models 1–6)

| Group | IPW | ATE (0.05) | ATE (0.1) | ATE (0.15) | OW | MW | EW | ATC | ATT |
|-------|-----|-----------|----------|----------|----|----|----|-----|-----|
| **Model 1, \(p = 10.05\%, r = 2.54\)** | | | | | | | | | |
| Control | 886.28 | 580.09 | 297.93 | 159.48 | 530.09 | 437.23 | 620.19 | 898.41 | 410.27 |
| Treated | 52.35 | 69.12 | 60.32 | 45.73 | 99.25 | 101.14 | 94.15 | 101.59 | |
| **Model 2, \(p = 20.77\%, r = 1.80\)** | | | | | | | | | |
| Control | 767.65 | 748.33 | 635.68 | 458.58 | 617.22 | 528.77 | 661.71 | 792.37 | 471.82 |
| Treated | 152.23 | 158.77 | 164.76 | 151.44 | 200.46 | 205.42 | 194.03 | 132.09 | 207.63 |
| **Model 3, \(p = 49.72\%, r = 1.13\)** | | | | | | | | | |
| Control | 441.66 | 438.16 | 432.71 | 421.32 | 444.86 | 429.45 | 447.79 | 541.46 | 265.54 |
| Treated | 333.81 | 350.74 | 367.48 | 372.51 | 395.51 | 392.80 | 390.76 | 207.89 | 458.54 |
| **Model 4, \(p = 79.22\%, r = 0.42\)** | | | | | | | | | |
| Control | 121.82 | 140.07 | 150.83 | 143.26 | 192.69 | 198.93 | 183.87 | 207.96 | 98.52 |
| Treated | 732.93 | 672.33 | 517.12 | 381.13 | 539.84 | 451.14 | 590.98 | 309.95 | 792.04 |
| **Model 5, \(p = 79.18\%, r = 0.26\)** | | | | | | | | | |
| Control | 55.09 | 70.94 | 61.35 | 46.07 | 102.73 | 105.52 | 96.91 | 107.92 | 49.13 |
| Treated | 867.74 | 570.55 | 294.78 | 144.05 | 500.88 | 392.97 | 595.16 | 308.56 | 892.08 |
| **Model 6, \(p = 79.59\%, r = 0.75\)** | | | | | | | | | |
| Control | 148.63 | 158.37 | 165.38 | 153.22 | 199.45 | 203.66 | 193.31 | 204.42 | 129.12 |
| Treated | 778.19 | 750.15 | 640.26 | 473.07 | 628.90 | 547.46 | 672.03 | 525.07 | 795.58 |

ATE: average treatment effect; ATE \((\alpha)\): ATE by trimming outside of \(\alpha < x(X) < 1 - \alpha, \alpha = 0.05, 0.1, 0.15\); OW: overlap weight; MW: matching weight; EW: entropy weight; IPWC: IPW on the controls; IPWT: IPW on the treated; \(p\): proportion of treated participants; \(r\): ratio of variances of propensity scores (treatment vs. control group).
score (PS) and outcome (OR) models are correctly specified; (ii) only PS model is correctly specified; (iii) only OR model is correctly specified; (iv) both PS and OR models are misspecified.

A: H˘ajek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both models correctly specified (resp. PS model correctly specified, OR model correctly specified, both PS and OR models misspecified).

Figure B.2.1: Point estimates under constant (top) and heterogeneous (bottom) treatment effects
A: Hájek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both models correctly specified (resp. PS model correctly specified, OR model correctly specified, both PS and OR models misspecified); the “Heterogeneous” (resp. “Constant”) in the green boxes means the window is under heterogeneous (resp. constant) treatment effect.

Figure B.2.2: Relative bias boxplots of point estimates (for models 1–3)
### Estimator and model specification

| Method       | Relative Bias |
|--------------|---------------|
| ATE          | Model 4, p = 79.22%, r = 0.42 |
| ATE 0.05     | Model 5, p = 89.18%, r = 0.26 |
| ATE 0.1      | Model 6, p = 79.59%, r = 0.75 |

A: Hájek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both models correctly specified (resp. PS model correctly specified, OR model correctly specified, both PS and OR models misspecified); the “Heterogeneous” (resp. “Constant”) in the green boxes means the window is under heterogeneous (resp. constant) treatment effect.

**Figure B.2.3:** Relative bias boxplots of point estimates (for models 4–6)
The red dotted line indicates the relative efficiency of 1; A: Hájek-type (weighted) estimator; B (resp. C, D, and E): augmented estimator, with both models correctly specified (resp. PS model correctly specified, OR model correctly specified, both PS and OR models misspecified); the “Heterogeneous” (resp. “Constant”) in the green boxes means the window is under heterogeneous (resp. constant) treatment effect.
### Table B.2.1: Model 1, with $p = 10.05\%$, $r = 2.54$

| Estimand | H\'ajek-type estimator | Aug. both models correctly specified | Aug. PS model correctly specified |
|----------|------------------------|-------------------------------------|----------------------------------|
|          | PE Bias RMSE RE CP     | PE Bias RMSE RE CP                 |                                  |
| ATE      | 3.57 15.84 2.33 1.40 0.60 | 4.00 0.09 0.31 1.20 0.91 | 2.56 35.88 4.53 1.82 0.68 |
|          | ATE (0.05) 3.51 2.92 0.84 0.94 | 4.00 0.12 0.26 1.12 0.93 | 3.52 1.91 0.82 0.65 0.95 |
|          | ATE (0.1) 3.53 1.05 1.05 0.98 | 4.00 0.04 0.32 1.21 0.92 | 3.86 3.43 0.81 0.73 0.94 |
|          | ATE (0.15) 3.59 1.50 0.32 0.98 | 4.00 0.12 0.48 1.33 0.94 | 3.78 5.39 1.05 0.71 0.94 |
| ATO      | 4.00 0.04 0.22 1.00 0.95 | 4.00 0.04 0.22 1.00 0.95 | 4.03 0.80 0.26 0.96 0.95 |
| ATM      | 4.04 1.00 0.49 0.64 0.98 | 4.00 0.04 0.22 0.99 0.95 | 4.06 1.52 0.47 0.75 0.97 |
| ATC      | 3.26 18.46 2.61 1.52 0.69 | 4.00 0.10 0.33 1.01 0.94 | 2.37 40.71 4.96 1.88 0.84 |
| ATT      | 4.14 3.48 1.00 0.91 0.98 | 4.00 0.04 0.22 0.23 1.00 | 4.13 3.35 0.91 0.51 1.00 |
|          | 4.00 0.10 0.30 1.16 0.92 | 4.04 1.06 6.65 1.83 0.80 |
|          | 4.00 0.12 0.28 1.15 0.93 | 2.34 41.58 2.29 1.16 0.74 |
|          | 4.00 0.05 0.32 1.23 0.92 | 3.09 22.77 1.50 1.23 0.80 |
|          | 4.00 0.03 0.43 1.41 0.92 | 3.38 15.41 1.28 1.27 0.85 |
|          | 4.00 0.11 0.23 1.06 0.93 | 2.92 27.11 2.07 1.02 0.86 |
|          | 4.00 0.11 0.23 1.05 0.94 | 2.87 28.29 2.24 1.01 0.88 |
|          | 4.00 0.10 0.24 1.07 0.93 | 2.97 25.68 2.19 1.08 0.85 |
|          | 4.00 0.11 0.32 0.98 0.94 | 4.10 2.46 7.11 1.92 0.82 |
|          | 4.00 0.04 0.22 0.23 1.00 | 2.88 28.11 2.30 0.86 0.90 |
| ATO      | 20.46 0.34 1.55 1.00 0.95 | 20.51 0.18 1.52 0.98 0.95 | 20.46 0.35 1.60 0.98 0.95 |
| ATM      | 22.18 0.45 1.98 0.94 0.94 | 22.18 0.42 1.96 0.97 0.94 | 22.16 0.55 2.01 0.95 0.94 |
| ATC      | 16.61 6.08 3.55 1.54 0.69 | 16.60 0.13 0.70 0.65 0.98 | 14.20 14.54 5.90 1.12 0.82 |
| ATT      | 22.64 0.45 2.31 0.96 0.94 | 22.64 0.23 2.26 0.49 0.99 | 22.69 0.46 2.30 0.47 0.99 |

Heterogeneous treatment effect

| Estimand | H\'ajek-type estimator | Aug. both models correctly specified | Aug. PS model correctly specified |
|----------|------------------------|-------------------------------------|----------------------------------|
|          | PE Bias RMSE RE CP     | PE Bias RMSE RE CP                 |                                  |
| ATE      | 16.35 5.04 3.22 1.54 0.70 | 17.21 0.94 0.68 1.08 0.94 | 15.08 12.43 5.37 1.53 0.67 |
|          | ATE (0.05) 16.80 1.14 1.72 1.33 0.89 | 18.94 1.96 1.25 2.52 0.78 | 16.79 3.10 1.66 1.24 0.90 |
|          | ATE (0.1) 22.47 1.39 2.81 2.11 0.72 | 22.61 0.79 2.45 4.63 0.62 | 22.37 1.82 2.67 2.61 0.70 |
|          | ATE (0.15) 20.50 3.16 4.34 1.87 0.65 | 20.64 2.70 4.41 6.69 0.54 | 20.38 3.55 8.68 0.22 0.58 |
| ATO      | 20.46 0.34 1.55 1.00 0.95 | 20.51 0.18 1.52 0.98 0.95 | 20.46 0.35 1.60 0.98 0.95 |
| ATM      | 22.18 0.45 1.98 0.94 0.94 | 22.18 0.42 1.96 0.97 0.94 | 22.16 0.55 2.01 0.95 0.94 |
| ATC      | 19.17 0.45 1.36 1.07 0.94 | 19.27 0.06 1.23 0.99 0.95 | 19.12 0.75 1.43 1.02 0.94 |
| ATT      | 15.61 6.08 3.55 1.54 0.69 | 16.60 0.13 0.70 0.65 0.98 | 14.20 14.54 5.90 1.12 0.82 |
|          | 22.69 0.45 2.31 0.96 0.94 | 22.64 0.23 2.26 0.49 0.99 | 22.69 0.46 2.30 0.47 0.99 |

PE: point estimation; Bias: absolute relative bias × 100; RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability; Aug: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE (α): ATE by trimming PS > 1 − α or PS < α, α = 0.05, 0.1, 0.15; ATO (resp. ATM, ATC, ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated)
### Table B.2.2: Model 2, with $p = 20.77\%$, $r = 1.80$

| Estimand | Constant treatment effect | Heterogeneous treatment effect |
|----------|---------------------------|-------------------------------|
|          | Aug: both models correctly specified | Aug: PS model correctly specified |
|          | PE Bias RMSE RE CP | PE Bias RMSE RE CP |
| ATE      | 3.93 0.82 1.33 0.94 | 3.93 0.82 1.33 0.94 |
| ATE (0.05) | 3.97 0.86 1.37 0.94 | 3.97 0.86 1.37 0.94 |
| ATE (0.1)  | 3.97 0.86 1.37 0.94 | 3.97 0.86 1.37 0.94 |
| ATE (0.15) | 3.97 0.86 1.37 0.94 | 3.97 0.86 1.37 0.94 |
| ATO      | 4.00 0.81 1.80 0.94 | 4.00 0.81 1.80 0.94 |
| ATM      | 3.90 0.85 1.87 0.94 | 3.90 0.85 1.87 0.94 |
| ATEN     | 4.00 0.83 1.90 0.94 | 4.00 0.83 1.90 0.94 |
| ATT      | 4.03 0.87 1.94 0.94 | 4.03 0.87 1.94 0.94 |

PE: point estimation; Bias: absolute relative bias $\times 100$; RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability; Aug: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE ($\alpha$): ATE by trimming PS $> 1 - \alpha$ or PS $< \alpha$, $\alpha = 0.05, 0.1, 0.15$; ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated).
Table B.2.3: Model 3, with \( p = 49.72\% \), \( r = 1.13 \)

| Estimand | Constant treatment effect | Augmented model correctly specified | Aug PS model correctly specified |
|----------|---------------------------|------------------------------------|---------------------------------|
|          | H"ajek-type estimator     | PE Bias RMSE RE CP                 | PE Bias RMSE RE CP               |
| ATE      | 4.02 0.41 0.43 0.83 0.96  | 4.00 0.08 0.13 1.00 0.95           | 4.02 0.50 0.37 0.78 0.96         |
|          | (0.00)                    |                                    |                                 |
| ATE (0.1)| 4.01 0.24 0.37 0.87 0.97  | 4.00 0.08 0.13 1.00 0.95           | 4.01 0.32 0.34 0.83 0.97         |
| ATE (0.5)| 4.00 0.00 0.24 0.74 0.98  | 4.00 0.07 0.13 1.00 0.95           | 4.00 0.10 0.23 0.71 0.98         |
| ATO      | 3.99 0.15 0.21 0.73 0.97  | 4.00 0.09 0.13 0.99 0.96           | 4.00 0.12 0.20 0.71 0.97         |
| ATEN     | 3.99 0.15 0.21 0.73 0.97  | 4.00 0.09 0.13 0.99 0.96           | 4.00 0.12 0.20 0.71 0.97         |
| ATM      | 3.95 1.32 0.57 0.98 0.95  | 4.00 0.09 0.14 0.39 1.00           | 3.95 1.13 0.54 0.14 0.90         |
| ATT      | 3.99 1.32 0.57 0.98 0.95  | 4.00 0.09 0.14 0.39 1.00           | 3.95 1.13 0.54 0.14 0.90         |
|          | Heterogeneous treatment effect | Augmented model correctly specified | Aug PS model correctly specified |
|          | H"ajek-type estimator     | PE Bias RMSE RE CP                 | PE Bias RMSE RE CP               |
| ATE      | 17.19 0.14 0.71 1.07 0.94  | 17.21 0.04 0.63 1.06 0.94           | 17.18 0.22 0.74 1.05 0.94         |
|          | (0.05)                    |                                    |                                 |
| ATE (0.1)| 17.06 0.41 0.70 1.11 0.93  | 17.06 0.33 0.63 1.10 0.93           | 17.03 0.48 0.73 1.07 0.93         |
| ATE (0.5)| 17.15 0.41 0.70 1.11 0.93  | 17.15 0.41 0.68 1.09 0.93           | 17.15 0.43 0.74 1.06 0.93         |
| ATO      | 16.82 0.56 0.71 1.19 0.91  | 16.82 0.47 0.66 1.20 0.92           | 16.61 0.63 0.74 1.14 0.92         |
| ATM      | 17.19 0.41 0.70 1.11 0.93  | 17.19 0.41 0.68 1.09 0.93           | 17.15 0.43 0.74 1.06 0.93         |
| ATEN     | 16.83 0.56 0.71 1.19 0.91  | 16.83 0.47 0.66 1.20 0.92           | 16.61 0.63 0.74 1.14 0.92         |
| ATM      | 17.19 0.41 0.70 1.11 0.93  | 17.19 0.41 0.68 1.09 0.93           | 17.15 0.43 0.74 1.06 0.93         |
| ATT      | 17.19 0.41 0.70 1.11 0.93  | 17.19 0.41 0.68 1.09 0.93           | 17.15 0.43 0.74 1.06 0.93         |

PE: point estimation; Bias: absolute relative bias \times 100; RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability; Aug: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE (\( \alpha \)): ATE by trimming PS > 1 - \( \alpha \) or PS < \( \alpha \); ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated).
Table B.2.4: Model 4, with \( p = 79.22\% \), \( r = 0.42 \)

### Constant treatment effect

| Estimand | PE Bias | RMSE | RE CP | PE Bias | RMSE | RE CP | PE Bias | RMSE | RE CP |
|----------|---------|------|-------|---------|------|-------|---------|------|-------|
| ATE      | 4.00    | 0.04 | 0.20  | 1.11    | 0.94 | -0.32 | 108.05  | 5.85 | 1.15  | 0.80 |
| ATE (0.05) | 4.00    | 0.03 | 0.19  | 1.08    | 0.94 | 1.55  | 61.23   | 3.20 | 1.11  | 0.77 |
| ATE (0.1) | 4.00    | 0.05 | 0.20  | 1.09    | 0.94 | 2.79  | 30.37   | 1.90 | 1.15  | 0.85 |
| ATE (0.15) | 4.00    | 0.05 | 0.20  | 1.07    | 0.94 | 3.29  | 17.65   | 1.45 | 1.20  | 0.88 |
| ATO      | 4.00    | 0.04 | 0.18  | 1.07    | 0.94 | 1.53  | 61.71   | 2.96 | 1.05  | 0.67 |
| ATM      | 4.00    | 0.04 | 0.18  | 1.06    | 0.94 | 1.59  | 60.20   | 2.92 | 1.04  | 0.69 |
| ATEN     | 4.00    | 0.03 | 0.18  | 1.07    | 0.94 | 1.29  | 67.76   | 3.26 | 1.06  | 0.67 |
| ATT      | 4.00    | 0.04 | 0.22  | 0.75    | 0.97 | -0.60 | 114.96  | 6.46 | 1.15  | 0.83 |

### Heterogeneous treatment effect

| Estimand | PE Bias | RMSE | RE CP | PE Bias | RMSE | RE CP | PE Bias | RMSE | RE CP |
|----------|---------|------|-------|---------|------|-------|---------|------|-------|
| ATE      | 17.24   | 0.14 | 1.07  | 1.09    | 0.95 | 17.22 | 0.01   | 0.63 | 1.00  | 0.95 |
| ATE (0.05) | 15.20   | 0.58 | 1.09  | 2.17    | 0.79 | 15.24 | 0.31   | 0.98 | 2.03  | 0.75 |
| ATE (0.1) | 13.60   | 0.89 | 0.92  | 1.61    | 0.87 | 13.63 | 1.16   | 0.83 | 1.87  | 0.85 |
| ATE (0.15) | 13.89   | 0.11 | 1.09  | 1.92    | 0.83 | 13.92 | 0.36   | 1.02 | 2.28  | 0.82 |
| ATO      | 15.33   | 0.41 | 0.86  | 1.00    | 0.94 | 15.37 | 0.15   | 0.81 | 0.96  | 0.95 |
| ATM      | 15.75   | 0.55 | 0.98  | 0.96    | 0.94 | 15.76 | 0.32   | 0.91 | 0.94  | 0.94 |
| ATEN     | 15.49   | 0.39 | 0.82  | 1.01    | 0.94 | 15.53 | 0.16   | 0.76 | 0.97  | 0.94 |
| ATT      | 16.96   | 0.59 | 1.06  | 1.18    | 0.93 | 16.85 | 0.04   | 0.70 | 0.59  | 0.99 |

**Note:** PE: point estimation; Bias: absolute relative bias\(\times 100\); RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability; Aug: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE (\(\alpha\)): ATE by trimming PS \(> 1 - \alpha \) or PS \(< \alpha\), \(\alpha = 0.05, 0.1, 0.15\); ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated)
Table B.2.5: Model 5, with $p = 89.18\%$, $r = 0.26$

### Constant treatment effect

| Estimand | PE Bias RMSE RE CP | PE Bias RMSE RE CP | PE Bias RMSE RE CP |
|----------|---------------------|---------------------|---------------------|
| ATE      | 4.34 8.48 2.84 1.39 0.79 | 4.00 0.06 0.29 1.16 0.92 | 4.52 13.11 3.69 1.44 0.82 |
| ATE (0.05) | 4.16 2.38 0.90 0.77 0.94 | 4.00 0.10 0.25 1.05 0.94 | 4.04 1.01 1.06 0.76 0.98 |
| ATE (0.1) | 4.13 3.20 0.90 0.34 0.96 | 4.00 0.08 0.29 1.14 0.93 | 4.06 1.40 1.07 0.35 0.87 |
| ATE (0.15) | 4.24 6.08 0.13 0.45 0.97 | 4.01 0.18 0.39 1.21 0.92 | 4.31 7.71 1.53 0.75 0.96 |
| ATT      | 3.97 3.27 0.43 0.60 0.98 | 4.00 0.02 0.22 0.89 0.85 | 3.91 2.15 0.55 0.70 0.97 |
| ATTN     | 4.03 0.73 0.39 1.05 0.92 | 4.00 0.07 0.31 1.21 0.93 | 4.06 1.80 1.01 1.06 0.90 |
| ATM      | 3.79 7.45 0.82 1.47 0.94 | 4.00 0.05 0.23 0.26 1.00 | 3.61 9.80 3.51 0.60 1.00 |
| ATEN     | 4.42 10.51 3.18 1.44 0.78 | 4.00 0.06 0.31 0.97 0.95 | 4.64 16.02 4.08 1.48 0.81 |

| Aug: both models correctly specified | Aug: PS model correctly specified |
|-------------------------------------|----------------------------------|
| PE Bias RMSE RE CP                  | PE Bias RMSE RE CP               |
| ATE                                 | 4.00 0.06 0.28 1.11 0.93         | 1.88 1.47 0.32 0.84 |
| ATE (0.05)                          | 4.00 0.01 0.27 1.11 0.93         | 2.84 2.93 2.36 1.20 0.90 |
| ATE (0.1)                           | 4.00 0.04 0.30 1.10 0.93         | 3.60 10.64 1.58 1.28 0.91 |
| ATE (0.15)                          | 4.00 0.01 0.37 1.19 0.93         | 3.85 3.86 1.38 1.27 0.92 |
| ATO                                 | 4.00 0.03 0.23 1.02 0.94         | 1.08 7.24 0.36 1.08 0.74 |
| ATM                                 | 4.00 0.03 0.23 1.02 0.94         | 0.90 7.69 3.96 1.05 0.76 |
| ATEN                                | 4.00 0.04 0.23 1.03 0.94         | 0.81 7.94 4.04 1.13 0.74 |
| ATC                                 | 4.00 0.01 0.22 0.25 1.00         | 0.62 8.59 4.37 0.69 0.87 |
| ATT                                 | 4.00 0.07 0.30 0.91 0.96         | -2.07 151.68 9.81 1.51 0.85 |

### Heterogeneous treatment effect

| Estimand | PE Bias RMSE RE CP | PE Bias RMSE RE CP | PE Bias RMSE RE CP |
|----------|---------------------|---------------------|---------------------|
| ATE      | 17.30 8.46 1.31 1.17 0.92 | 17.20 0.08 0.68 1.08 0.94 | 17.42 1.20 1.45 1.20 0.82 |
| ATE (0.05) | 13.93 1.83 1.24 2.07 0.86 | 13.94 1.91 1.17 2.84 0.76 | 13.94 1.91 1.27 1.97 0.81 |
| ATE (0.1) | 16.47 0.69 1.97 2.30 0.72 | 16.50 0.52 1.89 3.82 0.69 | 16.45 0.83 1.95 2.36 0.73 |
| ATE (0.15) | 22.87 3.14 3.44 3.08 0.60 | 22.13 2.86 3.34 5.09 0.57 | 22.08 3.09 3.43 3.53 0.61 |
| ATO      | 17.23 0.74 1.29 1.04 0.93 | 17.29 0.40 1.23 1.00 0.94 | 17.21 0.87 1.33 1.02 0.93 |
| ATM      | 18.63 1.12 1.54 0.98 0.93 | 18.71 0.69 1.42 0.97 0.95 | 18.60 1.27 1.61 0.98 0.93 |
| ATEN     | 16.67 0.64 1.12 1.03 0.94 | 16.71 0.39 1.08 1.02 0.94 | 16.66 0.67 1.15 1.00 0.94 |
| ATC      | 20.37 2.03 4.23 1.35 0.84 | 20.77 0.08 2.22 0.56 0.88 | 20.23 2.69 4.67 0.96 0.95 |
| ATT      | 16.92 0.80 1.36 1.29 0.90 | 16.77 0.08 0.70 0.64 0.99 | 17.08 1.75 1.49 1.10 0.94 |

| Aug: both models correctly specified | Aug: PS model correctly specified |
|-------------------------------------|----------------------------------|
| PE Bias RMSE RE CP                  | PE Bias RMSE RE CP               |
| ATE                                 | 17.20 0.08 0.68 1.08 0.94         | 15.01 12.82 3.30 1.52 0.79 |
| ATE (0.05)                          | 13.93 1.83 1.24 2.07 0.86         | 11.80 13.70 2.19 1.30 0.79 |
| ATE (0.1)                           | 14.79 10.85 2.27 2.49 0.44         | 14.56 12.23 2.60 2.15 0.48 |
| ATE (0.15)                          | 21.08 7.49 3.14 3.61 0.54         | 20.96 8.01 3.35 3.52 0.56 |
| ATO                                 | 16.56 4.59 1.19 1.01 0.80         | 14.81 14.69 2.99 1.10 0.54 |
| ATM                                 | 18.19 3.43 1.36 1.00 0.87         | 16.15 14.25 3.55 1.08 0.64 |
| ATEN                                | 16.01 4.55 1.07 1.01 0.80         | 14.30 14.75 2.86 1.12 0.52 |
| ATC                                 | 20.77 0.08 2.22 0.56 0.98         | 16.06 22.72 5.31 0.66 0.55 |
| ATT                                 | 16.77 0.08 0.70 0.64 0.96         | 14.93 11.07 3.19 1.43 0.89 |

PE: point estimation; Bias: absolute relative bias x 100; RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability; Aug: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE (α): ATE by trimming PS > 1 - α or PS < α, α = 0.05, 0.1, 0.15; ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated)
| Estimand     | Constant treatment effect | Heterogeneous treatment effect |
|-------------|---------------------------|--------------------------------|
|              | PE Bias RMSE RE CP        | PE Bias RMSE RE CP             |
| ATE         | 4.05 1.33 1.21 1.51 0.90  | 17.23 0.05 0.74 0.98 0.95      |
| ATE (0.05)  | 4.04 0.97 0.67 0.92 0.94  | 15.74 0.23 1.02 2.55 0.77      |
| ATE (0.1)   | 4.02 0.57 0.45 0.76 0.97  | 15.66 0.70 1.31 3.24 0.71      |
| ATE (0.15)  | 4.02 0.50 0.39 0.61 0.98  | 15.44 0.75 1.01 1.07 0.93      |
| ATO         | 4.00 0.11 0.16 1.00 0.95   | 16.52 0.64 0.89 1.07 0.94      |
| ATM         | 3.99 0.15 0.28 0.68 0.97   | 16.52 0.63 1.25 1.04 0.92      |
| ATEN        | 4.01 0.30 0.23 0.98 0.95   | 16.52 0.63 1.25 1.04 0.92      |
| ATC         | 3.98 0.56 0.42 0.61 0.98   | 16.52 0.63 1.25 1.04 0.92      |
| ATT         | 4.08 1.90 1.53 1.12 0.90   | 17.38 0.17 0.88 1.01 0.95      |

**Notes:**
- PE: point estimation; Bias: absolute relative bias × 100; RMSE: root mean squared error; RE: relative efficiency; CP: coverage probability.
- Aug.: augmented estimator; PS: propensity score; OR: outcome regression; ATE: average treatment effect; ATE (α): ATE by trimming PS > 1 − α or PS < α, α = 0.05, 0.1, 0.15; ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated).
C Appendix: Additional Data Analysis Results

In this section, we show additional details of the MEPS data analysis in Section 5. The love plots in Figure C.0.2 display the standardized mean differences (SMD) of covariates between the 2-by-2 groups. Most SMDs in the first two sub-population are within the 0.1 threshold, but those in White-Asian group by ATT and ATC exceed 0.1 a lot. At the same time, in general the equipoise estimands (ATO, ATM and ATEN) balance the covariates the best.

Comparison

| Comparison | N    | Minimum | 25-th Quantile | Median | Mean | 75-th Quantile | Maximum |
|------------|------|---------|----------------|--------|------|----------------|---------|
| White      | 9830 | 0.14    | 0.58           | 0.71   | 0.70 | 0.84           | 1.00    |
| Hispanic   | 5280 | 0.11    | 0.44           | 0.55   | 0.56 | 0.67           | 0.99    |
| White      | 9830 | 0.16    | 0.65           | 0.78   | 0.75 | 0.86           | 0.99    |
| Black      | 4020 | 0.04    | 0.51           | 0.62   | 0.62 | 0.75           | 0.99    |
| White      | 9830 | 0.30    | 0.83           | 0.92   | 0.89 | 0.97           | 1.00    |
| Asian      | 1446 | 0.22    | 0.68           | 0.78   | 0.77 | 0.87           | 1.00    |

Figure C.0.1: Propensity score distributions and summary statistics of the three comparison groups of the medical expenditure data

![Propensity score distributions and summary statistics of the three comparison groups](image)

Table C.0.2 shows all the estimated causal effects from the estimators we considered to evaluate racial disparities of the health care expenditure between the Whites participants and each of three comparison groups in the MEPS data. We need to point out the reason that the NA’s shown in the standard errors and p-values of augmented ATE (0.1) and ATE (0.15) in the White-Asian comparison group. This is potentially due to the trimming. As can be seen in the Figure C.0.1, the propensity scores of White group in White-Asian figure has

![Comparison of covariates](image)

ATE: average treatment effect; ATE (α): ATE by trimming PS > 1 − α or PS < α, α = 0, 0.05, 0.1, 0.15; ATO (resp. ATM, ATEN, ATC, and ATT): average treatment effect on overlap (resp. matching, entropy, controls, and treated)

Figure C.0.2: MEPS Data: covariates balance by the three sub-population of race comparisons
Table C.0.1: Effective sample sizes (ESS) for the medical expenditure data

| Group       | N    | ATE  | ATE (0.05) | ATE (0.1) | ATE (0.15) | OW | MW | EW | ATT | ATC |
|-------------|------|------|------------|-----------|------------|----|----|----|-----|-----|
| Hispanic    | 5280 | 2018.91 | 3584.18 | 4078.39 | 4284.12 | 4843.23 | 4987.47 | 4636.77 | 1124.28 | 5280 |
| White       | 9830 | 8802.92 | 8420.37 | 7707.37 | 6952.06 | 7481.73 | 6773.92 | 7787.66 | 9830 | 5029.29 |

**White-Hispanic:** $p = 65.06\%, r = 1.00$

**White-Black:** $p = 70.97\%, r = 0.81$

**White-Asian:** $p = 87.18\%, r = 0.60$

Table C.0.2: Disparities in the health care expenditure of the three racial comparison groups of the MEPS data

| Estimand   | Hájek-type Estimator | Augmented Estimator |
|------------|----------------------|---------------------|
|            | Estimation           | Standard error      | p-value  | Estimation | Standard error | p-value  |
| **White-Hispanic:** $p = 65.06\%, r = 1.00$ | | | | | | |
| ATE        | 699.12               | 304.77              | 0.022    | 1154.44  | 274.31        | <0.001  |
| ATE (0.05) | 1326.22              | 198.59              | <0.001   | 1448.01  | 189.55        | <0.001  |
| ATE (0.1)  | 1170.73              | 198.52              | <0.001   | 1234.85  | 188.67        | <0.001  |
| ATE (0.15) | 1240.09              | 188.38              | <0.001   | 1284.77  | 181.44        | <0.001  |
| ATO        | 1264.21              | 165.61              | <0.001   | 1282.59  | 166.30        | <0.001  |
| ATM        | 1306.46              | 158.35              | <0.001   | 1285.72  | 161.91        | <0.001  |
| ATEN       | 1202.48              | 173.40              | <0.001   | 1260.32  | 170.76        | <0.001  |
| ATT        | 345.26               | 419.58              | 0.411    | 1080.89  | 374.99        | 0.004   |
| ATC        | 1426.19              | 171.75              | <0.001   | 1289.21  | 170.10        | <0.001  |

**White-Black:** $p = 70.97\%, r = 0.81$

| ATE        | 850.82               | 234.56              | <0.001   | 992.82   | 235.79        | <0.001  |
| ATE (0.05) | 738.62               | 238.16              | <0.002   | 764.25   | 235.05        | 0.001   |
| ATE (0.1)  | 802.82               | 223.56              | <0.001   | 836.40   | 219.82        | <0.001  |
| ATE (0.15) | 846.31               | 228.58              | <0.001   | 868.74   | 223.59        | <0.001  |
| ATO        | 818.97               | 210.55              | <0.001   | 834.23   | 210.79        | <0.001  |
| ATM        | 824.31               | 213.90              | <0.001   | 814.78   | 215.29        | <0.001  |
| ATEN       | 823.11               | 212.55              | <0.001   | 841.77   | 212.56        | <0.001  |
| ATT        | 850.92               | 261.23              | 0.001    | 1088.15  | 264.84        | <0.001  |
| ATC        | 850.42               | 244.03              | <0.001   | 760.23   | 239.60        | 0.002   |

**White-Asian:** $p = 87.18\%, r = 0.60$

| ATE        | 2253.00              | 653.03              | <0.001   | 4712.69  | 2143.97       | 0.028   |
| ATE (0.05) | 1248.64              | 256.82              | <0.001   | 1244.45  | 252.67        | <0.001  |
| ATE (0.1)  | 1293.97              | 216.63              | <0.001   | 1279.91  | NA            | NA      |
| ATE (0.15) | 1456.62              | 241.66              | <0.001   | 1442.50  | NA            | NA      |
| ATO        | 1273.73              | 224.80              | <0.001   | 1303.53  | 227.28        | <0.001  |
| ATM        | 1391.96              | 219.19              | <0.001   | 1400.46  | 223.57        | <0.001  |
| ATEN       | 1231.66              | 243.22              | <0.001   | 1229.33  | 245.26        | <0.001  |
| ATT        | 2399.32              | 711.52              | <0.001   | 4960.53  | 2224.62       | 0.026   |
| ATC        | 1392.45              | 220.43              | <0.001   | 1388.95  | 224.85        | <0.001  |

ATE: average treatment effect; ATE (α): ATE with trimming outside of $α < ε(α) \leq 1 - α$, $α = 0.05, 0.1, 0.15$; OW: overlap weight; MW: matching weight; EW: entropy weight; IPW: inverse probability weight on control; IPWT: inverse probability weight on treated; p: proportion of participants in the treatment group; r: ratio of variance of propensity scores in treatment group to control group.

- **White-Hispanic:** $p = 65.06\%, r = 1.00$
- **White-Black:** $p = 70.97\%, r = 0.81$
- **White-Asian:** $p = 87.18\%, r = 0.60$

Additional notes:
- $\text{ATE} (\alpha)$: ATE with trimming outside of $\alpha < \varepsilon(\alpha) \leq 1 - \alpha$, $\alpha = 0.05, 0.1, 0.15$.
- OW: overlap weight; MW: matching weight; EW: entropy weight; IPW: inverse probability weight on control; IPWT: inverse probability weight on treated.
- p: proportion of participants in the treatment group; r: ratio of variance of propensity scores in treatment group to control group.

43
many values close to 1, which means when we trim those who have extreme propensity score, there is a large loss in sample size. In fact, after trimming by 0.1, the sample size reduces to only 5458 (48.40%), and after trimming by 0.15, only 3905 (34.63%) remains in the whole White-Asian group ($N = 11276$). When we lose these many participants (which implies a substantial information loss), the matrix $A_N(\tilde{\theta})$ (estimated information matrix of the parameter vector, see Section A.1.1 for the details) used in the sandwich variance estimator becomes singular. This makes it difficult to invert the matrix and thus disrupts the calculation of the sandwich variance estimate.