MY457/MY557: Causal Inference for Observational and Experimental Studies

Week 4: Selection on Observables 2

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Topics of this lecture

Where Were We?

Observational settings where the assignment mechanism for D is either unknown or not under our control.

Problem: If Y_1, Y_0, and D are associated with observed pre-treatment X (a 'selection problem'), we cannot naïvely compare the group means of Y.

Solution: We make the (1) conditional independence assumption:

$$(Y_1, Y_0) \perp\!\!\!\perp D \mid X$$

And (2) common support assumption:

$$0 < \Pr(D_i = 1 \mid X_i = x) < 1 \quad \text{for any} \quad x \in \mathcal{X}$$

But even then, we still cannot naïvely compare the means of Y in different groups!

Where Were We?

Given our assumptions, the ATE is instead nonparametrically identified as the weighted difference in population regression functions:

$$\begin{aligned}\tau_{ATE} &= \mathbb{E}[\hat{\tau}_{CATE}(X_i)] \\ &= \int (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) f(x) dx\end{aligned}$$

The intuition easier to grasp if we consider a case in which all X_i is discrete...

Then we can rewrite the identification result (for both ATE and ATT) as:

$$\tau_{ATE} = \sum_{x \in \mathcal{X}} (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) \Pr(X_i = x)$$

$$\tau_{ATT} = \sum_{x \in \mathcal{X}} (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) \Pr(X_i = x \mid D_i = 1)$$

We calculate conditional ATEs (CATEs) for different levels of X, and re-weight them by the (conditional) prevalence of X in the data.

From Identification to Estimation

This gives us the identification result for selection on observables. But how should we estimate our estimands of interest?

There are four broad approaches for estimation under conditioning:

1. Subclassification
2. Matching
3. Weighting
4. Regression

Subclassification only works with discrete $\boldsymbol{X}$ variables, and is a sample analogue of the result we saw on the previous slide (consult last week's slides).

More general, and more frequently encountered solutions are matching, weighting, and regression. We turn to those now.

Matching

Matching seeks to impute missing potential outcomes using the observed outcomes of 'closest' units or nearest neighbors. Basic process:

1. For each observation in the treated group i, find an observation in the untreated group with the most similar values of X

2a. Estimate ATT with the average difference between the pairs:

$$\hat{\tau}_{ATT} = \frac{1}{n_1} \sum_{i:D_i=1} (Y_i - \tilde{Y}_i) \simeq \frac{1}{n_1} \sum_{i:D_i=1} (Y_{1i} - Y_{0i}) = \tau_{ATT}$$

where $\tilde{Y}_i$ is the observed outcome of i's untreated 'buddy'

2b. When there are multiple (M_i) 'close' units, their average can be used:

$$\hat{\tau}_{ATT} = \frac{1}{n_1} \sum_{i:D_i=1} (Y_i - \left(\frac{1}{M_i} \sum\nolimits_{m=1}^{M_i} \tilde{Y}_{i_m} \right))$$

where $\tilde{Y}_{i_m}$ is i's mth untreated buddy

Example with Single Pre-treatment Covariate

unit	Potential Outcome under Treatment	Potential Outcome under Control		
i	$Y_i(1)$	$Y_i(0)$	D_i	X_i
1	6	?	1	3
2	1	?	1	1
3	0	?	1	4
4		0	0	2
5		9	0	3
6		1	0	-2
7		1	0	-4

Match and plug in:

$$\hat{\tau}_{ATT} = \frac{1}{3}((6-9)+(1-0)+(0-9)) = -3.7$$

A Silver Bullet?

Matching looks like it is magic, but it's not.

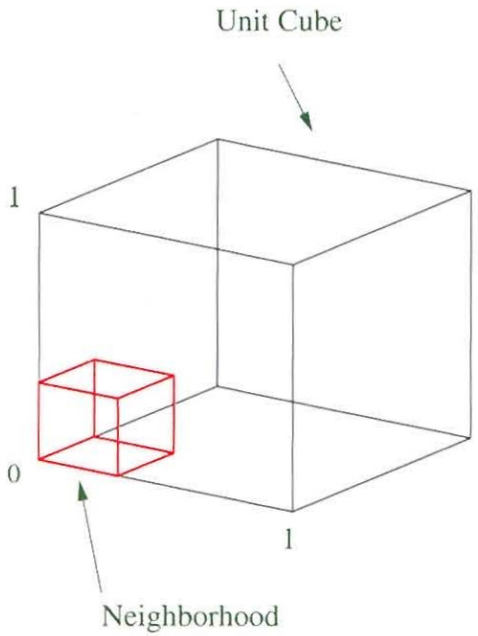
Matching is an approach to estimation (just like regression).

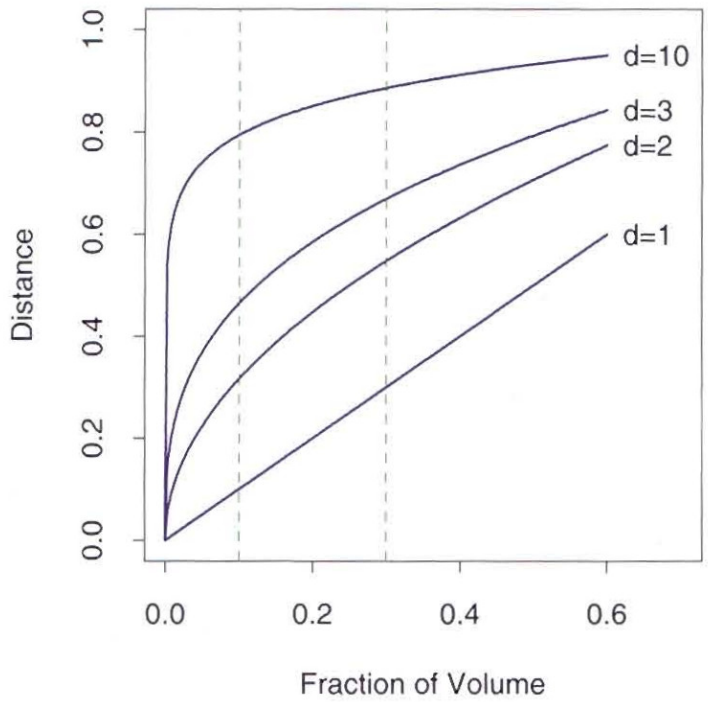
Always remember: "Design precedes estimation."

The Curse of Dimensionality

Consider a case where X_i contains > 1 variable? Can we hope to exactly match on every X_i, even if we have very large n? No!

We are struck by what is called the curse of dimensionality...





As number of dimensions in the covariate space increases, data sparsity exponentially increases for a given sample size.

"You must prepare to settle for a 60-70% match"

The Curse of Dimensionality and Bias

The curse of dimensionality implies a bias problem wherever we allow for non-exact matches.

Why? By tolerating not-quite-exact matches, we must (in expectation) inject 'error' into our estimates of missing potential outcomes (Abadie & Imbens, 2006).

The bias term is order $N^{(-1/k)}$, increasing in the number of dimensions k and implying no $\sqrt{n}$-consistency for $k > 2$.

If N_0 is much larger than N_1 (and there is common support), bias will typically be small. Generally wise to use Abadie & Imbens (2011) bias correction (more later).

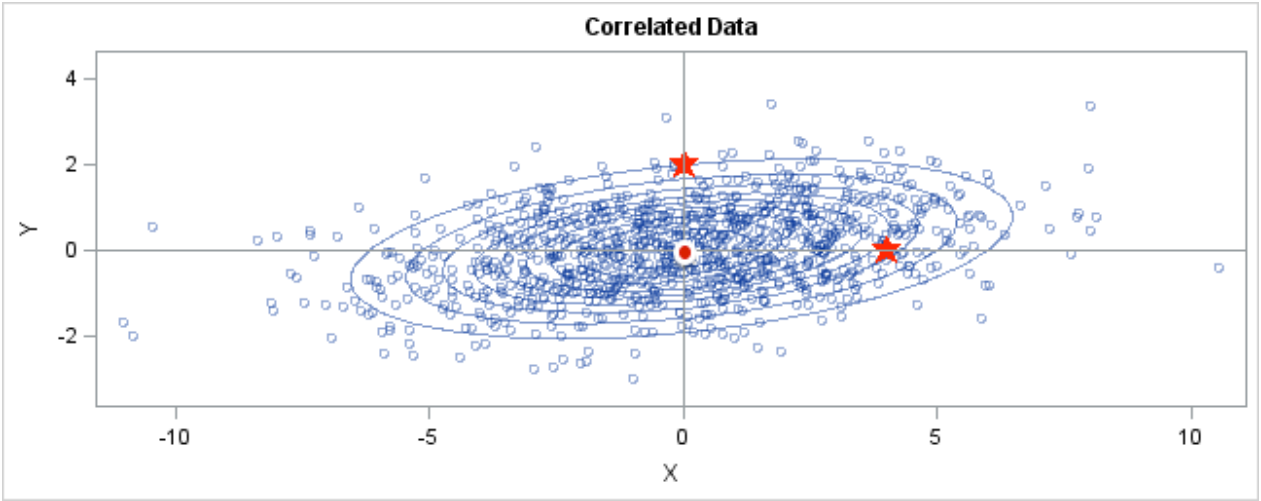
Matching as Dimension Reduction

How do we find the 'closest' match in multi-dimensional space?

We typically use a low-dimensional representation or distance metric. One example is Mahalanobis distance:

$$D_M(X_i, X_j) = \sqrt{(X_i - X_j)^\top \Sigma_X^{-1} (X_i - X_j)}$$

where Σ_X is the (sample) variance-covariance matrix of X_i



Note: other variants and metrics are possible.

The Propensity Score and the Balancing Property

Definition (propensity score)

Probability of receiving the treatment given X_i

$$\pi(X_i) \equiv \Pr(D_i = 1 \mid X_i)$$

Assumptions: Suppose the following holds:

1. $(Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i \mid X_i$ (conditional ignorability)
2. $0 < \Pr(D_i = 1 \mid X_i = x) < 1$ for any x (common support)

Result: The propensity score has the balancing property (Rosenbaum & Rubin, 1983):

$$D_i \perp\!\!\!\perp X_i \mid \pi(X_i)$$

Read: Among those units with the same propensity score, X_i is independent of treatment assignment.

Identification with the Propensity Score

The balancing property implies that conditional ignorability holds, conditional on just the propensity score alone:

$$(Y_{1i}, Y_{0i}) \perp\!\!\!\perp D_i \mid \pi(X_i)$$

Implication: It is sufficient to condition on $\pi(X_i)$, instead of X_i

But there is a catch: $\pi(X_i)$ itself needs to be estimated!

Two-step procedure to estimate causal estimands:

(1) Estimate $\pi(X_i)$ with a model for a binary response (e.g. logit, probit)

(2) Do nearest neighbor matching on $\pi(X_i)$

Note: Need to allow some uncertainty from (1) to percolate through to (2) (this is an open area of study)

Estimating the Propensity Score

Estimation of propensity scores requires a correct specification of $\pi(X_i)$ (functional form, etc.).

Check balance:

- Ideally, want to compare the joint distribution of all X_i between the treated and untreated in the matched sample
- In practice, check various low-dimensional summaries of $F(x)$ (mean difference, variance ratio, etc.)
- Balance tests are often used (e.g. t-test, F-test, KS test) like the ones we saw for randomized experiments.
- Note that balance tests can be misleading in a matching context because "balance" often improves when you drop lots of observations – can think of this as a "balance-sample size frontier" (King et al., 2017)

Estimate $\rightarrow$ Check Balance $\rightarrow$ Re-estimate $\rightarrow$ Check Balance $\rightarrow \cdots$ (*ad infinitum* until you attain good balance)

Is this data snooping or p-hacking? No, as long as inference remains blind to Y and τ

Things to Consider as you Match

There is a plethora of choices to be made:

- One-to-one vs. Many-to-one matching
- Exact matching vs. non-exact matching
- Matching with or without replacement
- Calipar matching
- Propensity score matching
- Genetic matching
- Optimal matching
- Coarsened exact matching

...and more in the pipeline...

This creates many researcher degrees of freedom. Whatever you choose, do so for principled reasons (e.g. balance) and without 'snooping' (looking at $\hat{\tau}$).

Balance testing when matching is important, but can be misleading. If you only check things you matched on you will often see good balance. But what are you missing?

Consider the balance-sample size frontier: one way to achieve good balance is to heavily trim your sample. Is this a good idea? (e.g. King, Lucas, & Nielsen 2017)

Weighting on the Propensity Score

So far we have used the propensity score for matching.

An alternative approach is weighting

Result: Under the conditional ignorability and common support assumptions, we can identify the ATE and ATT (weakly assuming) as:

$$\tau_{ATE} = \mathbb{E}\left[Y_i \cdot \frac{D_i - \pi(X_i)}{\pi(X_i) \cdot (1 - \pi(X_i))}\right]$$

$$\tau_{ATT} = \frac{1}{\Pr(D = 1)} \cdot \mathbb{E}\left[Y_i \cdot \frac{D_i - \pi(X_i)}{1 - \pi(X_i)}\right]$$

These can be estimated using sample analogues called inverse probability weighting (IPW) estimators:

$$\widehat{\tau}_{ATE} = \frac{1}{N}\sum_{i=1}^{N}(Y_i \cdot \frac{D_i - \widehat{\pi}(X_i)}{\widehat{\pi}(X_i) \cdot (1 - \widehat{\pi}(X_i))}) = \frac{1}{N}\sum_{i=1}^{N}(\frac{D_i Y_i}{\widehat{\pi}(X_i)} - \frac{(1 - D_i)Y_i}{1 - \widehat{\pi}(X_i)})$$

$$\widehat{\tau}_{ATT} = \frac{1}{N_1}\sum_{i=1}^{N}(Y_i \cdot \frac{D_i - \widehat{\pi}(X_i)}{1 - \widehat{\pi}(X_i)}) = \frac{1}{N_1}\sum_{i=1}^{N}(D_i Y_i - (1 - D_i)Y_i \frac{\widehat{\pi}(X_i)}{1 - \widehat{\pi}(X_i)})$$

Performance of the IPW estimators

IPW estimators have poor small sample properties:

- They highly sensitive to extreme values of $\pi(X_i)$
- Tends to occur when there is a lack of overlap
- This generates high variance (inefficiency)
- Can also produce significant bias in certain settings (e.g. model misspecification)

A workaround is trim units with extreme weights. But this changes the estimand to a quantity that is still causal yet difficult to interpret.

Alternative weighting methods with preferable finite sample properties include:

- Augmented IPW estimators: e.g. doubly robust estimator (more later).
- Entropy balancing (Hainmueller 2012, `ebal`): choose weights that directly optimize balance in X_i.
- Covariate balancing propensity scores (Imai and Ratkovic 2014, `CBPS`): model $\pi(X_i)$ while optimizing balance in X_i.
- Kernel balancing (Hazlett, 2020, `kbal`): choose weights to balance an unspecified non-linear representation of X_i.

Model-based Estimation of Causal Effects

When we think of 'controlling for' variables, we usually think of regression. What role can it play in causal inference?

Recall that under conditional ignorability and common support, ATE/ATT equal weighted averages of the differences in population regression functions:

$$\hat{\tau}(x) \;=\; \mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]$$

where

$$\tau_{ATE} \;=\; \mathbb{E}[\hat{\tau}(x)] \quad \text{and} \quad \tau_{ATT} \;=\; \mathbb{E}[\hat{\tau}(x) \mid D_i = 1]$$

This suggests a model-based approach for estimating causal effects, where we use a regression model for $\mathbb{E}[Y_i \mid D_i, X_i]$, e.g.,

$$\mathbb{E}[Y_i \mid D_i, X_i] \;=\; \beta_0 + \beta_1 D_i + \mathbf{X_i}\gamma,$$

which is a linear regression, and we can estimate β_1 via OLS.

OLS as an Estimator of Causal Effects

Suppose we regressed Y_i on D_i and X_i, estimating the coefficient on D_i via OLS:

$$\hat{\beta}_{OLS} = \frac{Cov(Y_i, \tilde{D}_i)}{Var(\tilde{D}_i)},$$

where $\tilde{D}_i$ is the residual from the regression of D_i on X_i ("partialling out").

When is $\hat{\beta}_{OLS}$ a good estimator of τ_{ATE}?

The answer depends on whether these two assumptions hold:

(1) Constant treatment effect: $\tau = Y_{1i} - Y_{0i}$ for all i.

(2) Linearity: Potential outcomes can be written as

$$Y_i(d) = \beta_0 + d\beta_1 + \mathbf{X_i}\gamma + \varepsilon_i \quad \text{for} \quad d = 0, 1.$$

Noting that (2) implies (1) (such that $\beta_1 = \tau$), there are 3 possible scenarios:

1. Both (1) and (2) are true.
2. Only (1) is true.
3. Neither (1) nor (2) is true.

Case 1: Constant Effect & Linear Potential Outcomes

Result: If treatment effect is constant across units and potential outcomes are linear in X_i, then the OLS estimate of β_1 in the following regression model

$$Y_i = \beta_0 + \beta_1 D_i + \mathbf{X_i}\gamma + \varepsilon_i$$

is an unbiased and consistent estimator of τ_{ATE}.

Proof: First, note that $\beta_1 = \tau_i$ for every i under these assumptions:

$$\begin{aligned} \tau_i &= Y_{1i} - Y_{0i} \\ &= (\beta_0 + \beta_1 + \mathbf{X_i}\gamma + \varepsilon_i) - (\beta_0 + \mathbf{X_i}\gamma + \varepsilon_i) \\ &= \beta_1 \end{aligned}$$

Next, note that conditional ignorability implies the conditional independence between D_i and ε_i:

$$(Y_{1i}, Y_{0i}) \perp\!\!\!\perp D_i \mid X_i \quad \Longrightarrow \quad \varepsilon_i \perp\!\!\!\perp D_i \mid X_i$$

Because this implies the zero conditional mean assumption, $\hat{\beta}_{OLS}$ is an unbiased and consistent estimator of β_1, which is equal to τ_{ATE} (and τ_i). □

Case 2: Constant Effect & Unknown Functional Form

What happens if $Y_i(d)$ is an unknown, nonlinear function of d and X_i, and yet we used $\hat{\beta}_{OLS}$ as an estimator of $\hat{\tau}_{ATE}$ anyway?

Recall that OLS is the best linear predictor in terms of MSE:

$$\hat{\beta}_{OLS} = \underset{\hat{\beta}_1}{\text{argmin}}\ \mathbb{E}[(Y_i - \hat{\beta}_0 - \hat{\beta}_1 D_i - \mathbf{X_i}\hat{\gamma})^2]$$

This, it turns out, also implies that $\hat{\beta}_{OLS}$ provides the best linear approximation to the population regression function:

$$\hat{\beta}_{OLS} = \underset{\hat{\beta}_1}{\text{argmin}}\ \mathbb{E}[(\mathbb{E}[Y_i \mid D_i, X_i] - \hat{\beta}_0 - \hat{\beta}_1 D_i - \mathbf{X_i}\hat{\gamma})^2]$$

Result:

- $\hat{\beta}_{OLS}$ can be interpreted as the best linear approximation to the true treatment effect, whatever the true functional form is.
- This approximation may or may not be good in absolute terms.
- More flexible models (nonlinear, semi-/non-parametric, etc.) may provide a better performing approximation.

Case 3: Heterogeneous Treatment Effects

Consider again our default OLS specification:

$$Y_i = \beta_0 + \beta_1 D_i + \mathbf{X_i}\gamma + \varepsilon_i$$

This can be thought of as a parametric model of the underlying data generating process that produces Y_i (and by implication, Y_{1i} and Y_{0i}).

By modeling the relationship between D_i and Y_i as a multiplicative function of just β_1, we assert that the effect of D_i is fixed and homogeneous.

Treatment effect heterogeneity is any real deviation from that assumed model, for example:

1. SUTVA violations generate variation in treatment effects
2. Effects vary across individual by chance
3. Effects vary over time (e.g. early vs. late)
4. Effects vary systematically by covariates (observed or unobserved)

Case 3: Heterogeneous Treatment Effects

Now recall the subclassification estimator for the ATE:

$$\hat{\tau}_{ATE} = \sum_{x \in \mathcal{X}} (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) \Pr(X_i = x),$$

where we weighted subgroup effects by the marginal of X_i.

Similarly, the subclassification estimator for the ATT:

$$\hat{\tau}_{ATT} = \sum_{x \in \mathcal{X}} (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) \Pr(X_i = x \mid D_i = 1),$$

where we weighted subgroup effects by the conditional of X_i given $D_i = 1$.

Result: The OLS estimator can be written as a subclassification estimator, weighted by the conditional variances of D_i in each subgroup (Angrist, 1998):

$$\hat{\beta}_{OLS} = \sum_{x \in \mathcal{X}} (\mathbb{E}[Y_i \mid D_i = 1, X_i = x] - \mathbb{E}[Y_i \mid D_i = 0, X_i = x]) \frac{\mathrm{Var}(D_i \mid X_i = x) \Pr(X_i = x)}{\sum_{x'} \mathrm{Var}(D_i \mid X_i = x') \Pr(X_i = x')}$$

OLS as a Subclassification Estimator

Estimator	Weights for Subgroups	Unbiased for
$\hat{\tau}_{ATE}$	$\Pr(X_i = x)$	τ_{ATE}
$\hat{\tau}_{ATT}$	$\Pr(X_i = x \mid D_i = 1)$	τ_{ATT}
$\hat{\beta}_{OLS}$	$\frac{\mathrm{Var}(D_i \mid X_i = x)\Pr(X_i = x)}{\sum_{x'} \mathrm{Var}(D_i \mid X_i = x')\Pr(X_i = x')}$	"$\tau_{\text{CVW-ATE}}$"

With non-constant treatment effects, OLS provides an unbiased estimator for a conditional-variance-weighted average treatment effect.

This is a causal quantity, but hard to interpret. It is not generally equal to the ATT or ATE (more in a moment).

Recall $\mathrm{Var}(D_i \mid X_i = x) = \pi(x)(1 - \pi(x))$. Therefore:

- Weights are high for groups with propensity scores close to 0.5.
- Weights are low for groups with propensity scores close to 0 or 1.
- OLS minimizes estimation uncertainty by downweighting groups where group-specific ATEs are less precisely estimated.

This result assumes discrete Xs, but intuition holds for continuous Xs.

OLS as a Weighted Average of Estimands

Given heterogeneous treatment effects (and some linearity assumptions), the causal estimand targeted by OLS can be decomposed as:

$$\tau_{OLS} = w_1 \cdot \tau_{ATT} + w_0 \cdot \tau_{ATU}$$

where:

$$w_1 = \frac{(1-P(D=1)) \cdot \mathrm{Var}[\pi(X)|D=0]}{P(D=1) \cdot \mathrm{Var}[\pi(X)|D=1] + (1-P(D=1)) \cdot \mathrm{Var}[\pi(X)|D=0]}, \text{ and}$$

$$w_0 = 1 - w_1$$

With heterogeneous treatment effects, OLS can be an unbiased estimator for a weighted average of the ATT and ATU (Słoczyński, 2022).

This can admit a strange interpretation:

- Weights w_j are inversely proportional to the share of units in j.
- This is weird: if you have a lot of treated units, ATU will be upweighted, and ATT downweighted. Why?
- For the ATT, OLS is predicting the *missing potential outcomes* for the treated – those come from the coefficients for the control, so these are upweighted.

Solutions: weighting, matching, and fully interacting de-meaned X and D.

The Fully-Interacted Estimator

One well regarded large-sample linear regression estimator is as follows:

$$Y_i = \hat{\alpha} + \hat{\tau_{int}} D_i + \hat{\beta}^T (X_i - \bar{X}) + \hat{\gamma}^T D_i (X_i - \bar{X})$$

where:

X_i are covariates sufficient to satisfy the conditional independence assumption

$\bar{X}$ is the sample mean of X_i

This estimator has numerous desirable properties:

- The bias in $\hat{\tau_{int}}$ as an estimator for τ_{ATE} is arbitrarily small in large samples under only conditional independence.
- Huber-White robust standard errors are sufficient for hypothesis testing.
- Mitigates small sample biases and inefficiency (Freedman, 2008).
- Resolves the weighted average of estimands problem (Słoczyński, 2022).
- Robust to contamination bias (Goldsmith-Pinkham et al, 2022)

Matching or Regression?

Regression:

+ Regression is simple.
- In SOO world, simple regression relies on a number of strong assumptions to admit a readily interpretable estimate. More complex specifications can help.
- Regression is prone to extrapolation beyond common support.

Matching:

+ Non-parametric (no model dependence)
+ Can be a transparent way to move from data/design to an estimate
- Can be rather non-transparent if implemented in certain ways
- Recall that because we can very rarely ever exactly match, matching usually induces bias by pulling our estimate slightly away from the estimand. This becomes more severe:
  - the more matches for each treated unit (as in, M=2 or 3 or 10); and
  - the more covariates we match on

Combining Regression, Matching and Weighting

Some approaches combine regression with matching or weighting for better finite-sample performance and/or robustness properties.

- Bias-corrected matching (Abadie and Imbens 2005):
  - Estimate bias inherent to matching estimators via regression
  - Subtract it off from the matching estimate for correction
  - → In **R**, can e.g. use `BiasAdjust = TRUE` in the `Matching` package.
- Doubly-robust estimation (Robins and Rotnitzky 2001):
  - Use a weighted average of regression and IPW estimators
  - The estimator will be consistent as long as either the regression model or PS model is correct
  - → In **R**, see e.g. `tmle` or `drgee` packages.
- Matching as nonparametric data preprocessing (Ho, Imai, King, & Stuart 2007):
  - Model-based estimation of causal effect is most likely to go wrong when it involves extrapolation due to poor overlap in covariates
  - Use matching to make treatment and control groups similar
  - Then run regression models to estimate causal effects
  - → In **R**, use whatever matching tool then whatever parametric tool!