

MY457/MY557: Causal Inference for Observational and Experimental Studies

Week 4: Selection on Observables 2

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S00: Identification Assumptions (Review)

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 naively 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:

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

S00: Identification Result (Review)

Given our assumptions, the ATE is instead **non-parametrically identified** as the weighted difference in **population regression functions**:

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

Let's consider a simpler case in which all X_i is **discrete**...

We can **rewrite** the identification results (for both ATE and ATT) as:

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

Subclassification

Our SOO identification result when all X_i is **discrete**:

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

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

That is, the ATE is identified as:

1. Grouping units into strata (or cells) defined by the values of X_i .
2. For each stratum, calculating the difference in means of Y_i .
3. Taking weighted average of (2), where weights are the prop. of units per strata.

Similarly, the ATT is given by:

1 - 2. Same as for ATE.

3. Calculating the weighted average of (2), with weights equal to the proportions of units in the strata **within the treatment group**.

Subclassification Estimators

This can be translated into two **subclassification estimators** for a given sample:

$$\hat{\tau}_{ATE} = \sum_{j=1}^M (\bar{Y}_{1j} - \bar{Y}_{0j}) \frac{n_j}{n}$$
$$\hat{\tau}_{ATT} = \sum_{j=1}^M (\bar{Y}_{1j} - \bar{Y}_{0j}) \frac{n_{1j}}{n_1}$$

where

- M = # of strata
- n_j = # of units in cell j
- n_{1j} = # of treated units in cell j
- \bar{Y}_{dj} = mean outcome for units with $D_i = d$ in cell j

This estimator **only works** for **discrete** X covariates, such that the strata are well defined. For more complex cases we will need alternatives.

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Matching

Matching **imputes 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:

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

where \tilde{Y}_{i_m} is i 's m th 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.

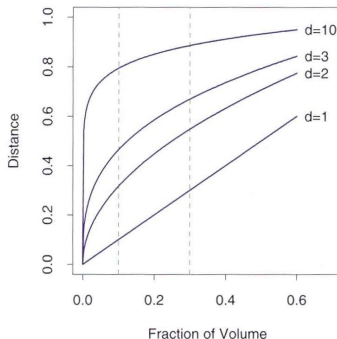
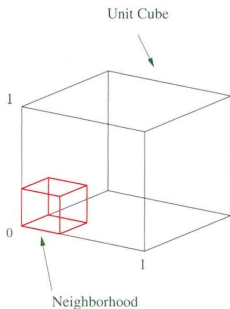
Matching is an approach to estimation (just like subclassification or regression).

Always remember: "Design precedes estimation."

The Curse of Dimensionality

Consider a case where \mathbf{X}_i contains > 1 variable? Can we hope to **exactly match** on every \mathbf{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).

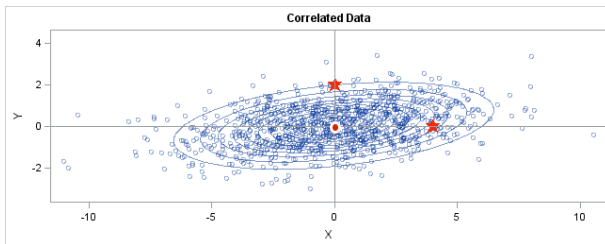
Matching as Dimension Reduction

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

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

Propensity score:

Probability of receiving the treatment given \mathbf{X}_i

$$\pi(\mathbf{X}_i) \equiv \Pr(D_i = 1 \mid \mathbf{X}_i)$$

Assumptions: Suppose the following holds:

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

Result: $\pi(\mathbf{X}_i)$ has the **balancing property** (Rosenbaum & Rubin, 1983):

$$D_i \perp\!\!\!\perp \mathbf{X}_i \mid \pi(\mathbf{X}_i)$$

Read: Among those units with the same propensity score $\pi(\mathbf{X}_i)$, \mathbf{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 binary response estimator (e.g. logit, probit)
- (2) Perform 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 requires a correct specification of $\pi(\mathbf{X}_i)$.

Intuitively we can check **empirical balance**:

- Ideally, compare the joint distribution of all \mathbf{X}_i between treated and control in the matched sample
- Practically, check low-dimensional summaries of $\mathbf{F}(\mathbf{x})$ (e.g. mean difference)
- **Balance tests** are often used (e.g. t-test, F-test, KS test) like the ones we saw for randomized experiments.

Estimate \rightarrow Check Balance \rightarrow Re-estimate \rightarrow Check Balance $\rightarrow \dots$

Is this p-hacking? No, as long as inference remains blind to \mathbf{Y} and $\hat{\tau}$.

Some Considerations

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, propensity score, genetic, optimal, coarsened exact
... and more...

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 can be misleading. If you only check **things you matched on** 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)

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Weighting on the Propensity Score

An alternative use of the propensity score is **weighting**

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

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

The sample analogues are **inverse probability weighting (IPW)** estimators:

$$\begin{aligned}\hat{\tau}_{ATE} &= \frac{1}{N} \sum_{i=1}^N \left(Y_i \cdot \frac{D_i - \hat{\pi}(X_i)}{\hat{\pi}(X_i) \cdot (1 - \hat{\pi}(X_i))} \right) = \frac{1}{N} \sum_{i=1}^N \left(\frac{D_i Y_i}{\hat{\pi}(X_i)} - \frac{(1 - D_i) Y_i}{1 - \hat{\pi}(X_i)} \right) \\ \hat{\tau}_{ATT} &= \frac{1}{N_1} \sum_{i=1}^N \left(Y_i \cdot \frac{D_i - \hat{\pi}(X_i)}{1 - \hat{\pi}(X_i)} \right) = \frac{1}{N_1} \sum_{i=1}^N \left(D_i Y_i - (1 - D_i) Y_i \frac{\hat{\pi}(X_i)}{1 - \hat{\pi}(X_i)} \right)\end{aligned}$$

Performance of the IPW estimators

The default IPW estimators have poor **small sample properties**:

- Highly **sensitive to extreme values** of $\pi(\mathbf{X}_i)$, which 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)

Workaround: Trim units with extreme weights, but this changes the estimand to a quantity that while 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**): weights to optimize balance in \mathbf{X}_i .
- Covariate balancing propensity scores (Imai and Ratkovic 2014, **CBPS**): model $\pi(\mathbf{X}_i)$ while optimizing balance in \mathbf{X}_i .
- Kernel balancing (Hazlett, 2020, **kbal**): weights balance an unspecified non-linear representation of \mathbf{X}_i .

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Model-based Estimation of Causal Effects

When we think of ‘controlling for’ variables, we usually think of **regression**.

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 we might **assume 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:

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

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

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

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

- A. Both (1) and (2) are true.
- B. Only (1) is true.
- C. Neither (1) nor (2) is true.

Case A: Constant Effect & Linear Potential Outcomes

Result: If the treatment effect is constant across units and (potential) outcomes are linear in \mathbf{X}_i such that

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

then $\hat{\beta}_{OLS}$ 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 what conditional ignorability implies about D_i and ε_i :

$$(Y_{1i}, Y_{0i}) \perp\!\!\!\perp D_i \mid \mathbf{X}_i \implies \varepsilon_i \perp\!\!\!\perp D_i \mid \mathbf{X}_i \implies \mathbb{E}[\varepsilon_i \mid D_i, \mathbf{X}_i] = 0$$

This is the **zero conditional mean assumption**! So, $\hat{\beta}_{OLS}$ is an unbiased and consistent estimator of β_1 , which is equal to τ_{ATE} (and τ_i).



Case B: 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:

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

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

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

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 C: Heterogeneous Treatment Effects

Consider again our default OLS regression:

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

Remember, this is a **parametric model** of the underlying **data generating process** that produces Y_{1i} and Y_{0i} (and by implication Y_i).

By modeling the relationship between D_i and Y_i as a additive 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 individuals by chance
3. Effects vary over time (e.g. early vs. late)
4. Effects vary systematically by covariates (observed or unobserved)

Case C: Heterogeneous Treatment Effects

Now recall the subclassification estimator for the **ATE**:

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

where we weighted subgroup effects by the **marginal of X_i** .

Similarly, the subclassification estimator for the **ATT**:

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

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

Result: In the saturated discrete- \mathbf{X} case, the OLS estimator is itself a subclassification estimator, weighted by the **conditional variances of D_i** per subgroup (Angrist, 1998):

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

OLS as a Subclassification Estimator

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

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 $\text{Var}(D_i \mid \mathbf{X}_i = \mathbf{x}) = \pi(\mathbf{x})(1 - \pi(\mathbf{x}))$. 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 \mathbf{X} s, but intuition holds for continuous \mathbf{X} s.

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:

$$w_1 = \frac{(1 - \Pr(D=1)) \cdot \text{Var}[\pi(X)|D=0]}{\Pr(D=1) \cdot \text{Var}[\pi(X)|D=1] + (1 - \Pr(D=1)) \cdot \text{Var}[\pi(X)|D=0]},$$
$$w_0 = 1 - w_1$$

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 projects *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 (Lin, 2013):

$$Y_i = \hat{\alpha} + D_i \hat{\tau}_{int} + (X_i - \bar{X}) \hat{\beta} + D_i (X_i - \bar{X}) \hat{\gamma}$$

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)

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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!