Machine learning and causal inference (observational studies)

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BSE
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Causal inference in observational studies

- In observational studies we have no control on the treatment/exposure of the units.
- Treatment assignment mechanism is not random, but some sort of selection made by the unit itself, the environment or any mechanism other than an experiment: no manipulation of causes.
- Approach: attempt to approximate a randomized experiment within the observational study.
- Note that treatment is not going to be independent from potential outcomes anymore. We need to work without strong ignorability $X \perp\!\!\!\perp Y(x)$ for $x = \{0, 1\}$.

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- Rewrite $\text{ATE} := \text{E}[Y(1)] - \text{E}[Y(0)]$ using the law of total probability:

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  but notice that $\text{E}[Y(1)|X=0]$ and $\text{E}[Y(0)|X=1]$ are not identifiable.

- It can be shown (Morgan and Winship, 2015, pg. 59) that the $\widehat{\text{ATE}}$ estimator converges in probability to

$$\text{E}[Y(1)|X=1] - \text{E}[Y(0)|X=0] = \text{ATE} + \text{bias},$$

  where

$$\text{bias} := \{\text{E}[Y(0)|X=1] - \text{E}[Y(0)|X=0]\} + \Pr(X=0)\{ATT - ATC\},$$

  and the first term is the *baseline bias* and the second is the *differential treatment effect bias*.

Causal inference in observational studies

- Example adapted from Morgan and Winship (2015, pg. 59-60): *the effect of obtaining a master's degree on labor market outcome*. We observe that individuals who have obtained a master's degree score higher than those who didn't.

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- **ATE:** *obtaining a master's degree makes individuals more successful in the labor market, reflected in some kind of numerical score, the higher the better.*

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- **Baseline bias** ($\mathrm{E}[Y(0)|X=1] - \mathrm{E}[Y(0)|X=0]$): *Individuals who obtain master's degrees would have been done better in the labor market than those who didn't obtain them, in the counterfactual state in which they did not in fact obtain master's degrees.*

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- **Differential treatment effect bias** ($\Pr(\mathrm{X}{=}0)\{\mathrm{ATT\text{-}ATC}\}$): *Those who didn't obtain master's degrees would not have done as well as those who did obtain them in the counterfactual state in which they did in fact obtain master's degrees.*

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- Suppose 30% of the people obtains a master's degree ($P(X = 1) = 0.3$) and we observe the labor market outcome scores specified below.

Group	$\mathrm{E}[Y(1)\|X]$	$\mathrm{E}[Y(0)\|X]$
Treatment $(X = 1)$	10	6
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- **DTE bias:=** $\Pr(X=0)(ATT-ATC)=0.7\cdot(10-6-8+5)=0.7$
- Note that $\widehat{\mathrm{ATE}}:=\mathrm{E}[Y(1)|X=1]-\mathrm{E}[Y(0)|X=0]=10-5=5$ is upwardaly biased from the actual ATE, 3.3, which follows also from substracting baseline and DTE bias from $\widehat{\mathrm{ATE}}$ (5-1-0.7=3.3).

Causal inference in observational studies: bounds

- Assuming SUTVA (consistency, no-interference) and observed data being a random sample, we can derive **bounds** on ATE for a bounded outcome Y. For instance, consider a binary outcome $Y = \{0, 1\}$, i.e., $-1 \leq \text{ATE} \leq 1$ is a risk difference (Robins[1], 1989; Manski[2], 1990).

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- Upper bound by making ATE as large as possible:

$$\Pr(X=0) + \text{E}[Y(1)|X=1]\Pr(X=1) - \text{E}[Y(0)|X=0]\Pr(X=0).$$

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- Lower bound by making ATE as small as possible:

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- These bounds are sharp, in the sense that narrower bounds are not possible without additional assumptions.

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- We have a random sample of 40 students, among which 25 obtained a master's degree, thus 15 did not. Among the 40 students, 20 found a job and 20 went unemployed. Among those 20 who found a job, 15 had obtained a master's degree and 5 didn't.

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- By causal consistency we can write the previous upper bound as:

$$\underset{\frac{15}{40}}{\Pr(X=0)} + \underset{\frac{15}{25}\cdot\frac{25}{40}}{\mathrm{E}[Y|X=1]\Pr(X=1)} - \underset{\frac{5}{15}\cdot\frac{15}{40}}{\mathrm{E}[Y|X=0]\Pr(X=0)} = 0.625$$

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- Lower bound is one unit lower, thus bounds are [-0.375, 0.625].

Causal inference in observational studies: bounds

- Upper bound estimate on ATE derived by guessing numbers to best case:

	Group		Whole
	$X=0$	$X=1$	sample
$Y(1)=1$	15	15	30
$Y(1)=0$	0	10	10
Total	15	25	40
$Y(0)=1$	5	0	5
$Y(0)=0$	10	25	35
Total	15	25	40

- Upper bound estimate on ATE:

$$\text{ATE}^{\uparrow} := \text{E}[Y(1)] - \text{E}[Y(0)] = \frac{30}{40} - \frac{5}{40} = 0.625\,.$$

- Lower bound estimate on ATE. Guess numbers to worst case and then:

$$\text{ATE}^{\downarrow} := \text{E}[Y(1)] - \text{E}[Y(0)] = \frac{15}{40} - \frac{30}{40} = -0.375\,.$$

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- When treatment/exposure and potential outcomes share a common cause, a phenomenon known as **confounding**.

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- When treatment/exposure and potential outcomes share a common cause, a phenomenon known as **confounding**.

- For instance, a competitive admission process for a master's degree may be selecting students who would be successful in the job market anyway.

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Propensity scores

- Approach: if we knew the factors (covariates) Z that drive the confounding phenomenon, we could assume ignorability **conditional on baseline covariates** Z:

$$X \perp\!\!\!\perp \{Y(0), Y(1)\} | Z \,,$$

  also known in the literature as the *"conditional exchangeability"* or *"no unmeasured confounders"* assumption.

- Under this assumption, $\Pr(X = 1 | Y(0), Y(1), Z) = \Pr(X = 1 | Z)$. The term $\Pr(X = 1 | Z)$ is the probability of treatment given baseline covariates and it is also known as the **propensity score** (Rosenbaum and Rubin, 1983)[3].

[3] Rosenbaum, P.R. and Rubin, D.B. The central role of the propensity score in observational studies for causal effects. *Biometrika*, 1983. https://doi.org/10.1093/biomet/70.1.41.

Propensity scores

- Let $e(Z) := \Pr(X = 1|Z)$ denote the propensity score on covariates Z. If we assume that there are no unmeasured confounders other than Z, i.e., $\Pr(X = 1|\, Y(0), Y(1), Z) = \Pr(X = 1|Z)$, then

$$\Pr(X = 1|\, Y(0), Y(1), e(Z)) = \Pr(X = 1|e(Z))\,,$$

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- Given a sample of n units, we may have K different propensity scores with $K \leq n$. Let $\{\pi_1, \ldots, \pi_K\}$ be the set of those different propensity scores.

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- Given a sample of n units, we may have K different propensity scores with $K \leq n$. Let $\{\pi_1, \ldots, \pi_K\}$ be the set of those different propensity scores.

- If we stratify units according to their propensity score, the size n_k of stratum $k \in \{1 \ldots K\}$ corresponds to:

$$n_k := \sum_{i=1}^{n} \mathrm{I}[e(Z_i) = \pi_k]\,.$$

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- A consistent estimator of ATE is then

$$\widehat{\mathrm{ATE}}_{str} := \sum_k \left(\frac{n_k}{n}\right) \widehat{\mathrm{ATE}}_k \,,$$

where

$$\widehat{\mathrm{ATE}}_k := \frac{\sum_i^n Y_i \mathrm{I}[X_i = 1, e(Z_i) = \pi_k]}{\sum_i^n \mathrm{I}[X_i = 1, e(Z_i) = \pi_k]} - \frac{\sum_i^n Y_i \mathrm{I}[X_i = 0, e(Z_i) = \pi_k]}{\sum_i^n \mathrm{I}[X_i = 0, e(Z_i) = \pi_k]} \,.$$

Propensity scores: inverse probability weighting

- Consider the number m of individuals in stratum k that are treated:

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- Rewrite it as follows:

$$\widehat{\mathrm{ATE}}_k := \frac{\sum_i^n Y_i \mathrm{I}[X_i = 1, e(Z_i) = \pi_k]}{m_k} - \frac{\sum_i^n Y_i \mathrm{I}[X_i = 0, e(Z_i) = \pi_k]}{n_k - m_k} \,.$$

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- We can obtain the following equivalent form:

$$\widehat{\text{ATE}}_{str} := \frac{1}{n} \sum_k \left\{ \frac{\sum_i^n X_i \, Y_i \mathrm{I}[X_i = 1, e(Z_i) = \pi_k]}{m_k / n_k} - \frac{\sum_i^n (1 - X_i) \, Y_i \mathrm{I}[X_i = 0, e(Z_i) = \pi_k]}{(n_k - m_k)/n_k} \right.$$

- The previous expression is approximately equal to:

$$\widehat{\text{ATE}}_{ipw} := \frac{1}{n} \sum_i \left\{ \frac{X_i \, Y_i}{e(Z_i)} - \frac{(1 - X_i) \, Y_i}{1 - e(Z_i)} \right\} .$$

- We weight individuals by the inverse of the probability of being assigned the treatment actually received.

Propensity scores: regression

- Another approach is to use a regression model:

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- By consistency and ignorability within each stratum,

$$\mathrm{E}[\,Y(x)|e(Z) = e(z)] = \alpha_0 + \alpha_1 x + \alpha_2 e(z)\,,$$

Propensity scores: regression

- Another approach is to use a regression model:

$$\mathrm{E}[\,Y|X = x, e(Z) = e(z)] = \alpha_0 + \alpha_1 x + \alpha_2 e(z)\,.$$

- By consistency and ignorability within each stratum,

$$\mathrm{E}[\,Y(x)|e(Z) = e(z)] = \alpha_0 + \alpha_1 x + \alpha_2 e(z)\,,$$

- Consequently, for $\mathrm{ATE} := \mathrm{E}[\,Y(2)] - \mathrm{E}[\,Y(0)]$, $\alpha_1 = \mathrm{ATE}$ and thus we can define $\widehat{\mathrm{ATE}}_{reg} = \hat{\alpha}_1$.

Propensity scores: estimation

- Propensity scores are unknown in observational studies. They need to be estimated using logistic regression or some supervised machine learning method (Lee et al., 2010)[4].

- In the case of logistic regression, we consider the binary treatment variable X as response and the covariates $Z_1, \ldots, Z_k$ as explanatory variables:

$$\log \frac{e(Z)}{1-e(Z)} = \log \frac{\Pr(X=1|Z)}{1-\Pr(X=1|Z)} = \beta_0 + \beta_1 Z_1 + \cdots + \beta_k Z_k \,.$$

- Once the $\hat{\beta}_i$ cofficients have been estimated, we can obtain the propensity scores by using the model formula:

$$\hat{e}(Z) = \frac{e^{\hat{\beta}_0 + \cdots + \hat{\beta}_k}}{1 + e^{\hat{\beta}_0 + \cdots + \hat{\beta}_k}} \,.$$

[4] Lee et al. (2010). Improving propensity score weighting using machine learning. *Statistics in Medicine*, 29:337-346. https://doi.org/10.1002/sim.3782

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Graphical models !!!!!

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- In randomized studies association is causation.
- In observational studies we can work with bounds or ignorability given covariates (which covariates? do we measure all potential confounders?).
- Causal inference requires working with precise terminology, explicit assumptions and a strong subject-matter knowledge. Remember Rubin's quote: *"assumptions are the strands that link statistics to science"*.

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- In randomized studies association is causation.
- In observational studies we can work with bounds or ignorability given covariates (which covariates? do we measure all potential confounders?).
- Causal inference requires working with precise terminology, explicit assumptions and a strong subject-matter knowledge. Remember Rubin's quote: *"assumptions are the strands that link statistics to science"*.
- Causal inference is a vast field, many things we haven't seen: randomization-based inference using Fisher's exact test and permutation tests, causal inference with non-compliance and/or interference, Bayesian methods, etc.

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