The Potential Outcome Framework

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Linear Methods in Causal Inference
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Review

- We discussed basic concepts in statistical analysis.
- The estimand is the theoretical quantity we want to estimate.
- The estimator is a mapping from data to a number (the estimate).
- We hope that the estimator is well behaved.
- Desirable properties include unbiasedness, consistency, efficiency, and asymptotic normality.
- We also want to quantify the uncertainty around our estimate.
- This process is known as statistical inference.

Review

- Typically, we first derive the variance of the estimator.
- Then, we use its sample analogue to estimate the variance.
- It is acceptable if the variance estimate is conservative.
- If the estimator converges to a normal distribution with the root-N rate, we can construct confidence intervals using normal critical values.

Why do we care about causality?

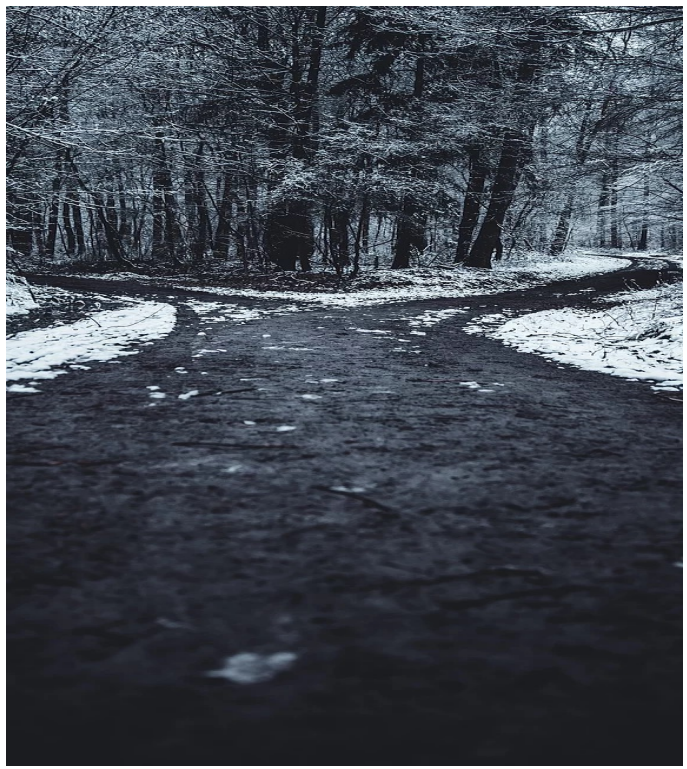
- This lecture introduces basic concepts in causal inference.
- Most theories we are interested in take the form of causal relationships.
- What would happen to Y if D changes?
  - Does economic growth cause democratization?
  - Do political ads change viewers' political preference?
  - Can trade reduce the probability of war?
- We call Y the outcome and D the treatment.
- The better we understand causal relationships, the better we can design policy interventions.

How do we define causality?

- There has been a long history of defining causality.
- Aristotle (four causes), Hume (does it exist?), Mill (the method of agreement/difference)...
- We follow the current practice and define causality using counterfactual.
- Ideally, we travel back to the past with a time machine and alter the value of D.
- We then observe what would happen to Y.

How do we define causality?

- "Two roads diverged in a wood, and I—
  I took the one less traveled by,
  And that has made all the difference."
  — Robert Frost, *The Road Not Taken*



- This simple idea is captured by the Neyman-Rubin model.

The Neyman-Rubin model

- We possess a sample of N units.
- Denote the outcome of interest for unit i as Y_i and the treatment as $D_i \in \{0, 1\}$.
- We may also have some pre-treatment covariates $\mathbf{X}_i$.
- Then, we have

$$Y_i = \begin{cases} Y_i(0), \ D_i = 0 \\ Y_i(1), \ D_i = 1. \end{cases}$$

- $Y_i(d)$ is called the "potential outcome."
- $\tau_i = Y_i(1) - Y_i(0)$ is the individualistic treatment effect.

The Neyman-Rubin model

- We call the average of τ_i, $\tau = \frac{1}{N}\sum_{i=1}^{N}\tau_i$, the average treatment effect (ATE).
- When N is sufficiently large, we can also write the ATE as $E[\tau_i]$.
- Obviously,

$$\tau = E[\tau_i] = E[Y_i(1)] - E[Y_i(0)].$$

- Note that $Y_i(d)$ could be a complex function of both observable and unobservable factors:

$$Y_i(d) = f_d(\mathbf{X}_i, U_i),$$

  where U_i represents unobservable factors.
- τ is a quantity that marginalizes over all these factors.

The Neyman-Rubin model

- The model includes several implicit assumptions:
  1. Consistency,
  2. Manipulable treatment,
  3. Stable Unit Treatment Value Assumption (SUTVA).

The Neyman-Rubin model

- Consistency is a philosophical concern on the interpretation of potential outcomes.
- Manipulable treatment restricts the scope of problems we could study.
- It forces us to focus on the "effects of causes" rather than "causes of effects."
- SUTVA can be relaxed in many cases.

The Neyman-Rubin model

- The idea of potential outcomes was first established by Neyman when analyzing agricultural experiments (Neyman 1923).
- It was formalized by Rubin in his 1974 paper ("the science").
- One motivation of the model is Heisenberg's uncertainty principle.
- It was independently developed in other disciplines (Roy model, Pearl's DAG, etc.).

The fundamental problem of causal inference

- We observe either $Y_i(0)$ or $Y_i(1)$ in practice, but never both.
- "The fundamental problem of causal inference" (Holland 1986)
- The unobserved potential outcome is called the counterfactual.
- Causal inference aims to impute the counterfactual based on assumptions.
- "Causal inference is a missing data problem." —Donald Rubin
- Qualitative studies can be similarly understood (Coppock and Kaur 2022)

The fundamental problem of causal inference

Unit	$Y_i(1)$	$Y_i(0)$	D_i
1	3	2	
2	5	3	
3	4	5	

- The ATE equals to $(1 + 2 - 1)/3 = 2/3$.

The fundamental problem of causal inference

Unit	$Y_i(1)$	$Y_i(0)$	D_i
1	3	NA	1
2	NA	3	0
3	4	NA	1

The scientific solution

- How do we test Newton's second law of motion?
- "When a constant force acts on a massive body, it causes it to accelerate."
- We need two assumptions: temporal stability and unit homogeneity.
- Neither is credible in social science.

The statistical solution

- The statistical solution relies on a large sample.
- We divide the sample into the treatment group and the control group.
- Idea: John Mill's method of difference.
- If the two groups are the same in all the aspects, their difference in the average outcome could be attributed to the treatment.
- Yet this does not work in practice.

Treatment assignment

- Suppose there are 20 binary covariates that affect the outcome variable.
- To apply the method of difference, we need a sample of $2^{20} \approx 1$ million units.
- Instead, we rely on randomization of treatment assignment.
- Suppose there exists a probability $0 < p_i < 1$ for each unit i, such that

$$P(D_i = 1) = p_i.$$

- This is an individualistic and probabilistic assignment mechanism (Imbens and Rubin 2015).

Treatment assignment

- In theory, we can assign the treatment vector $\mathbf{d} = (d_1, d_2, \ldots, d_N)$ altogether.

Assignment	Probability
$(1, 0, 1)$	0.4
$(0, 0, 1)$	0.6

- There are at most 2^N possibilities.
- We can assign each possibility a probability such that these probabilities sum up to one.
- These probabilities are known as an assignment mechanism.
- But we usually assume that treatment assignment is decided by one's own attributes (individualistic) and the probability is strictly between 0 and 1 (probabilistic).
- Individualistic assignment does not mean that the probabilities are independent across units.

Treatment assignment

- In this case, p_i may still be a function of all the variables in sample:

$$p_i = g(\mathbf{X}_i, U_i, Y_i(1), Y_i(0)).$$

- This assignment mechanism is unconfounded if $p_i = p(\mathbf{X}_i)$.
- If the assignment mechanism is individualistic, probabilistic, and unconfounded, we have a **classical randomized experiment**.
- From now on, we further assume that p_i does not depend on $\mathbf{X}_i$.
- There are two common assignment mechanisms in practice.
- Bernoulli trial: $p_i = p$ for any i.
- Complete randomization:

$$P(\mathbf{d}) = \begin{cases} \frac{1}{\binom{N}{N_1}}, & \text{if } \sum_{i=1}^{N} d_i = N_1 \\ 0, & \text{otherwise.} \end{cases}$$

Treatment assignment

- Let's define $N_1 = \sum_{i=1}^{N} D_i$ and $N_0 = \sum_{i=1}^{N}(1 - D_i)$.
- Obviously, $N = N_1 + N_0$.
- They are random variables under Bernoulli trial.
- If $p = 0.5$, N_1 can be either 60 or 40 in one assignment.
- Under complete randomization, N_1 and N_0 are pre-fixed numbers.
- Complete randomization gives you the group size you want.
- It is like a lottery.

Treatment assignment

- But complete randomization is not possible in certain contexts.
- E.g., decide whether a patient is treated or not upon their arrival.
- It is easier to analyze the Bernoulli trial as probabilities are independent to each other.
- When $N \to \infty$, the difference between the two mechanisms disappears.
- Therefore, we use the Bernoulli trial as the benchmark.

Bernoulli trial vs. complete randomization

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## Under Bernoulli trial, we have 103 treated units, and
##  97 untreated units.

## Under complete randomization, we have 100
##  treated units, and  100 untreated units.
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The statistical solution (continued)

- Treatment assignment is randomized in a classical randomized experiment, hence

$$D_i \perp \{Y_i(0), Y_i(1)\},$$

and

$$E[Y_i|D_i = 1] = E[Y_i(1)|D_i = 1] = E[Y_i(1)],$$
$$E[Y_i|D_i = 0] = E[Y_i(0)|D_i = 0] = E[Y_i(0)].$$

- Hence,

$$E[Y_i|D_i = 1] - E[Y_i|D_i = 0] = E[Y_i(1)] - E[Y_i(0)] = E[\tau_i].$$

- Remember that $E[\tau_i] = \tau$ is the ATE.

The statistical solution (continued)

- The power of randomization was first recognized by Ronald Fisher.
- Randomization creates an exogenous variation so that causal identification becomes possible.
- Due to randomization, all the other factors that affect Y_i are balanced in expectation: $E[\mathbf{X}_i|D_i = 1] = E[\mathbf{X}_i|D_i = 0]$.
- This is no guarantee that $\frac{1}{N_1}\sum_{i:D_i=1} X_i = \frac{1}{N_0}\sum_{i:D_i=0} X_i$.
- As N grows, the probability for $\sum_{i:D_i=1} X_i$ to be significantly different from $\sum_{i:D_i=0} X_i$ will get smaller.
- People typically test the null hypothesis that $\frac{1}{N_1}\sum_{i:D_i=1} X_i = \frac{1}{N_0}\sum_{i:D_i=0} X_i$ for each X_i.
- Rejection of the null implies the failure of randomization.

Balance of covariates

- Suppose the null is not rejected, what have we learned?
- What is the right null hypothesis?
- Should we use the t-test or the F-test?
- It is challenging when the covariates are high-dimensional.
- We may still have imbalance in each assignment.
- If possible, you may rerandomize, until $\left|\left| \frac{1}{N_1} \sum_{i:D_i=1} \mathbf{X}_i - \frac{1}{N_0} \sum_{i:D_i=0} \mathbf{X}_i \right|\right| \leq \delta$.
- By doing so, you are truncating the distribution of potential estimates.
- Some adjustments might be necessary (Li, Ding, and Rubin 2018).

The statistical solution (continued)

- If D_i is not randomly assigned, there will be $\mathbf{X}_i$ affecting both Y_i and D_i.
- The causal relationship between Y_i and D_i will be confounded by $\mathbf{X}_i$, hence we call them confounders.
- Causal inference studies how to utilize existing randomization from either experiments or hypothetical experiments to identify causal relationships.
- It is about inference rather than creating causality from nowhere.

Estimand vs. estimator

- No individualistic treatment effect is identifiable under statistical solutions.
- We focus on the average effect over a fixed population.
- These average effects are our estimands.
- It could be the ATE, the ATT ($\tau_{ATT} = E[Y_i(1) - Y_i(0)|D_i = 1]$), or the CATE ($\tau(\mathbf{x}) = E[Y_i(1) - Y_i(0)|\mathbf{X}_i = \mathbf{x}]$).
- We sometimes differentiate these estimands in the sample and them in the population.
- E.g., the SATE ($\frac{1}{N}\sum_{i=1}^{N}[Y_i(1) - Y_i(0)]$) vs. the PATE ($E[Y_i(1) - Y_i(0)]$).

Estimand vs. estimator

- Our estimands are functionals of the joint distribution of $\{Y_i(1), Y_i(0)\}$, $F(y_1, y_0)$.
- Such a distribution is unknown to the researcher.
- For example, the population average treatment effect (PATE) equals to

$$\tau_{PATE} = E[Y_i(1) - Y_i(0)] = \int (y_1 - y_0) dF(y_1, y_0)$$

- In the sample, we only have access to the observed outcome: $Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0)$.
- Denote the joint distribution of $\{Y_i, D_i, \mathbf{X}_i\}, i \in \{1, 2, \ldots, N\}$ as $G(y, d, \mathbf{x})$.
- Our estimator $\hat{\tau}$ is a functional of $G(y, d, \mathbf{x})$.
- Causal identification means that there exists a $\hat{\tau}$ such that $\hat{\tau}(G) = \tau(F)$ when N is infinite.

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