CISER Causal Inference Workshop

Session 1: Causal Inference Foundations

Chencheng Cai

Department of Mathematics and Statistics

Washington State University

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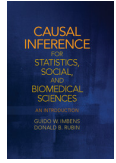
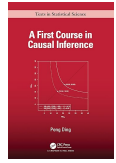
Resources for Causal Inference

- Book by Guido Imbens and Donald Rubin (2015): *Causal Inference for Statistics, Social, and Biomedical Sciences: An Introduction*.
- Book by Peng Ding (2024): *A First Course in Causal Inference*





- Soceity for Causal Inferene (est. 2020) hosts annual meeting called *American Causal Inference Conference* (ACIC).

  `https://sci-info.org/2025-meeting/`

- Stanford University initiated an weekly online seminar series called *Online Causal Inference Seminar* (OCIS).

  `https://sites.google.com/view/ocis/`

Causation and Correlation

"... we may attain the knowledge of a particular cause merely by one experiment, provided it be made with judgment, and after a careful removal of all foreign and superfluous circumstances ... the mind can draw an **inference concerning the existence of its correlative** *...*

— David Hume (1711-1776), A Treatise of Human Nature (1739)

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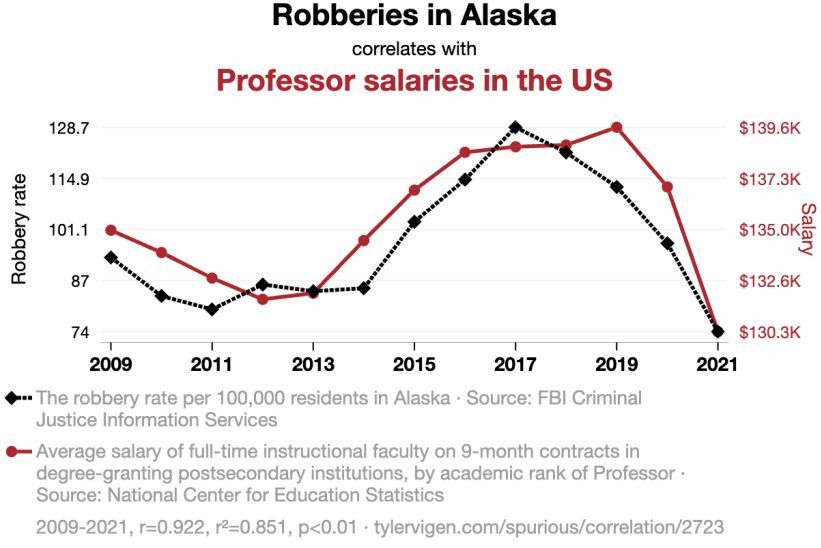
"Correlation does not imply causation."

— researchers nowadays

Spurious Correlations



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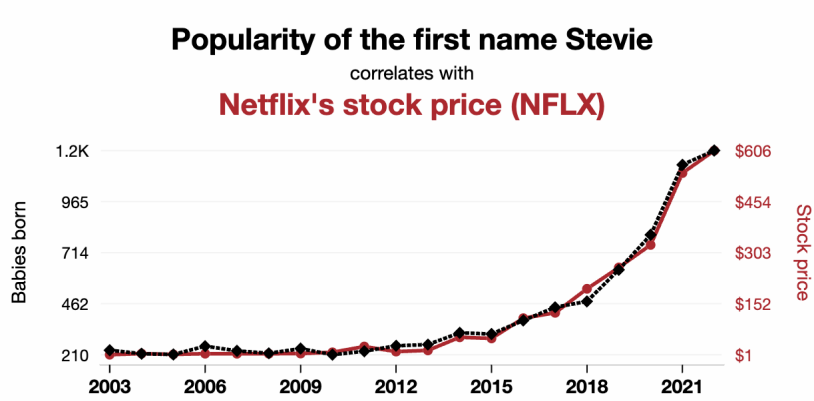

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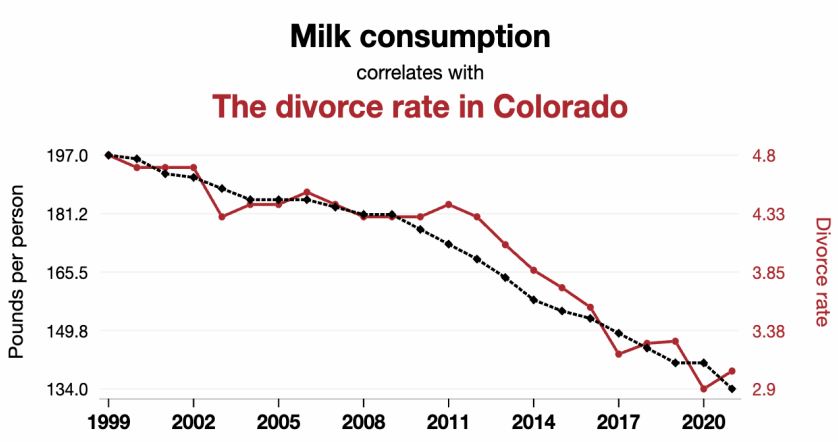

Spurious Correlations

All above examples of spurious correlations are credited to Tyler Vigen.
https://www.tylervigen.com/spurious-correlations

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Many of the spurious correlations are due to confounding variables.

- Robberies vs Salaries. Confounder could be economic condition.
- Stevie Babies vs NFLX price. Confounder could be the population.
- Milk vs Divorce rate. Confounder could be the generation.

Mill's Methods

John Stuart Mill (1806-1873) proposed a set of five methods to determine causation in his book *A System of Logic* (1843).

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- If a person who drinks milk with coffee and gets sick, and another person who drinks milk with tea and gets sick, then milk is the cause of sick.

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Example 2 (Method of Difference):

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Example 2 (Method of Difference):

- If a person who drinks milk with coffee and gets sick, and another person who drinks milk with tea and does not get sick, then coffee is the cause of sick.

- Not sufficient for causal inference.
- Lack of probabilistic reasoning.
- Prototype of randomized controlled trials.

Fisher's Randomized Clinical Trials

Ronald Fisher (1890-1962) proposed the idea of randomized controlled trials (RCT) in his book *Design of Experiments* (1935).

- The subjects are assigned to two groups: treatment and control.
- The assignment of treatment is **random**.
- Subjects in the treatment group receive the treatment, while subjects in the control group do not.
- The outcome of interest is compared between the two groups.

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Remarks:

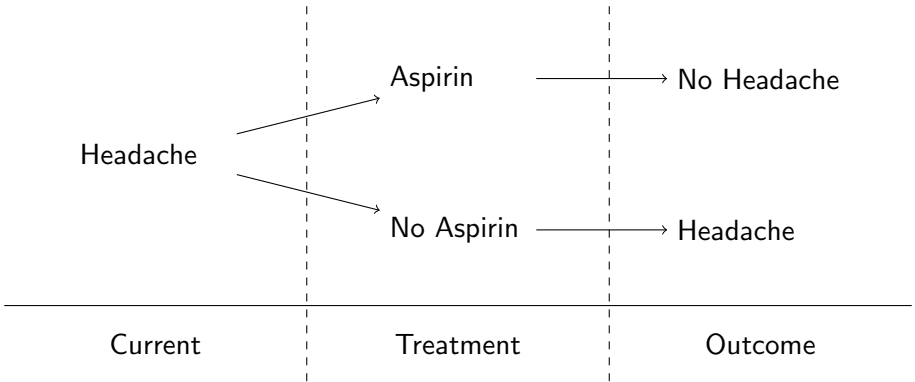
- Clinical trials have been used to evaluate the effectiveness of medical treatments in 1700s.
- Randomized experiments have been used in psychology and agriculture since 1880s.
- Fisher's work is the first to propose the necessity of physical randomization for assessing causal effects.

Neyman-Rubin Potential Outcome Framework

Neyman-Rubin potential outcome framework is a mathematical model for causal inference proposed by Jerzy Neyman (1894-1981) in 1920s and further developed by Donald Rubin.

Potential Outcome

One possibility of outcomes of Aspirin when having headache:



Potential Outcome

We define the **potential outcome** as the outcome that would have been observed if the treatment had been different. The notation is $Y(W)$, where W is the treatment.

For the previous example, we have two potential outcomes:

$$Y(\text{Aspirin}) = \text{No Headache}, \quad Y(\text{No Aspirin}) = \text{Headache}.$$

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For the previous example, we have two potential outcomes:

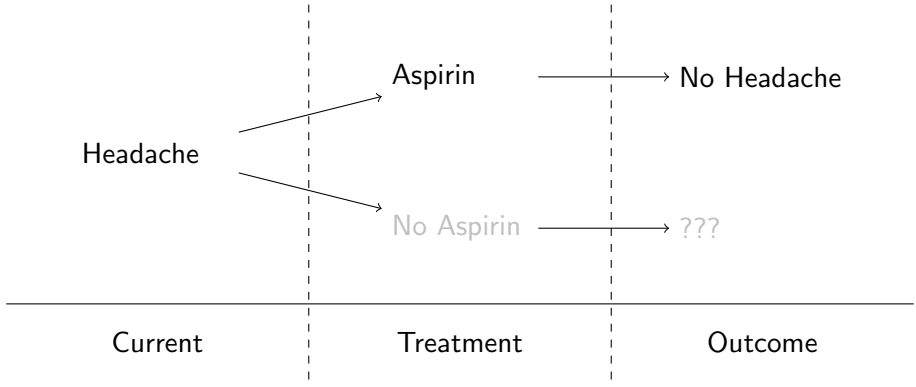
$$Y(\text{Aspirin}) = \text{No Headache}, \quad Y(\text{No Aspirin}) = \text{Headache}.$$

The difference between the two potential outcomes is the **causal effect** of the treatment.

$$\begin{aligned}\text{Causal Effect of Aspirin} = &\text{ Potential Outcome if I took Aspirin} \\ &- \text{ Potential Outcome if I did not take Aspirin}\end{aligned}$$

Potential Outcome

In reality, we can only observe **one** of the two arms.
If taking Aspirin, we observe:



The observed outcome is $Y^{obs} = Y(\text{Aspirin}) = \text{No Headache}$.

Potential Outcome

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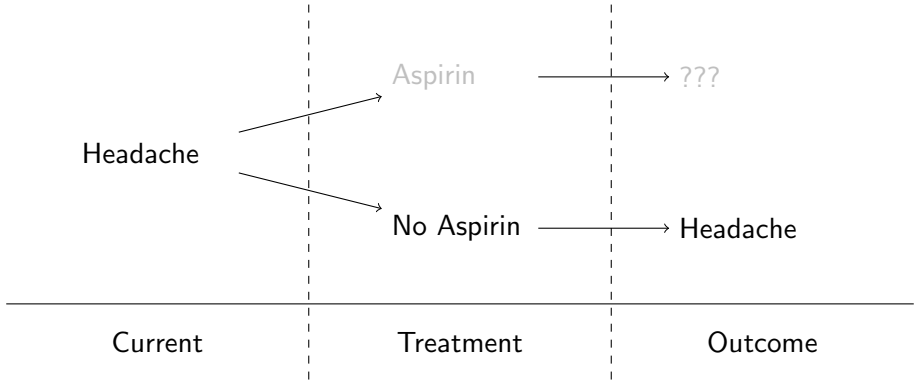
If not taking Aspirin, we observe:



The observed outcome is $Y^{obs} = Y(\text{No Aspirin}) = \text{Headache}$.

Potential Outcome

Because of the fact that we can only observe one of the two potential outcomes, in order to estimate the causal effect, we need to have multiple units.

Unit	Y_i(Aspirin)	Y_i(No Aspirin)
1	No Headache	Headache
2	No Headache	Headache
3	Headache	Headache
4	No Headache	Headache
5	Headache	Headache
6	No Headache	Headache

or

Unit	$Y_i(1)$	$Y_i(0)$
1	1	0
2	1	0
3	0	0
4	1	0
5	0	0
6	1	0

Above is the table of all potential outcomes for six units.

Potential Outcome

Unit	$Y_i(1)$	$Y_i(0)$
1	1	0
2	1	0
3	0	0
4	1	0
5	0	0
6	1	0

- Causal effect is defined as the difference between potential outcomes.
- The unit-level causal effect is

$$\tau_i = Y_i(1) - Y_i(0)$$

- The average causal effect is

$$\tau = \frac{1}{N} \sum_{i=1}^{N} \tau_i$$

Assigning Mechanism

The units are further assigned to treatment and control groups. Their outcomes are observed.

Unit	$Y_i(1)$	$Y_i(0)$	W_i	Y_i^{obs}
1	1	0	1	1
2	1	0	0	0
3	0	0	1	0
4	1	0	0	0
5	0	0	1	0
6	1	0	0	0

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6	1	0	0	0

- In **observational studies**, the assignment mechanism is often determined by the units themselves.
- In **experimental studies**, the assignment mechanism is determined by the researcher and is often random.

Causal Inference

Once the treatments have been assigned and the outcomes have been observed, the rest is an inference problem.

- Estimand: causal effect related to the potential outcomes.

$$\tau = \frac{1}{N}\sum_{i=1}^{N}(Y_i(1) - Y_i(0))$$

- Estimator: constructed from the observed data

$$\hat{\tau} = \frac{1}{N_1}\sum_{i:W_i=1} Y_i^{obs} - \frac{1}{N_0}\sum_{i:W_i=0} Y_i^{obs}$$

  where N_1 and N_0 are the number of units in treatment and control groups.

- Hypothesis testing, confidence interval, and p-value.

Potential Outcome Framework for Causal Inference

- Sampling units.
  For now: N units randomly sampled from the population.
- Potential outcomes model.
  For now: $Y_i(W_i)$.
- Assignment mechanism.
  Observational study or experimental study.
- Data collection.
  For now: $Y_i^{obs} = Y_i(W_i)$.
- Estimand and estimator.
  For now: average causal effect.
- Hypothesis testing.
  Later in the workshop.

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Assumptions

Stable Unit Treatment Value Assumption (SUTVA) (Rubin, 1980): The potential outcomes for any unit do not vary with the treatments assigned to other units, and, for each unit, there are no different forms or versions of each treatment level, which lead to different potential outcomes.

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- No interference between units. (counterexample: network data)
  - So we can write $Y_i(W_1, W_2, \ldots, W_N)$ as $Y_i(W_i)$.
- No hidden versions of treatment. (counterexample: non-compliance)
  - So we can write $Y_i^{(obs)} = Y_i(W_i)$.

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Ignorance Assumption with Confounders (Rubin, 1974): The assignment mechanism is independent of the potential outcomes conditional on the observed confounders.

- In observational studies, the ignorance assumption with confounders is often satisfied if all the confounders are measured.
- It is also known as no unmeasured confounders assumption.

Assigning Mechanism

Denote

- $\boldsymbol{Y}(0)$: vector of potential outcomes of control for all units.
- $\boldsymbol{Y}(1)$: vector of potential outcomes of treatment for all units.
- $\boldsymbol{W}$: vector of treatment assignment for all units.
- $\boldsymbol{X}$: vector of covariates for all units.

An **assigning mechanism** is a row-exchangeable function $P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1))$ satisfying

$$\sum_{\boldsymbol{W} \in \{0,1\}^N} P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = 1.$$

- Row-exchangeable: the order of the units is irrelevant.

Assigning Mechanism

The **unit assignment probability** is defined as

$$p_i(\boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = \sum_{\boldsymbol{W}: W_i = 1} P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)).$$

The **propensity score** is defined as

$$e(x) = \frac{1}{N(x)} \sum_{i: X_i = x} p_i(\boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1))$$

where $N(x) = \sum_{i=1}^{N} I(X_i = x)$ is the number of units with $X_i = x$.

Assigning Mechanism

A **probabilistic assignment mechanism** is an assigning mechanism that satisfies

$$0 < p_i(\boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) < 1 \quad \text{for all } i = 1, \ldots, N.$$

An **unconfounded assignment mechanism** is an assignment mechanism that satisfies

$$P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}'(0), \boldsymbol{Y}'(1)),$$

for all $\boldsymbol{W}, \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1), \boldsymbol{Y}'(0), \boldsymbol{Y}'(1)$.

Assigning Mechanism

An **individualistic assignment mechanism** is an assignment mechanism that if, for some function $q(\cdot)$,

$$p_i(\boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = q(X_i, Y_i(0), Y_i(1)), \quad \text{for all } i = 1, \ldots, N,$$

and

$$P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = c \cdot \prod_{i=1}^{N} q(X_i, Y_i(0), Y_i(1))^{W_i} (1 - q(X_i, Y_i(0), Y_i(1)))^{1-W_i}$$

for some constant c.

Assigning Mechanism

A **randomized experiment** is an assigning mechanism that

1. is probabilistic, and
2. has a known functional form that is controlled by the researcher.

A **classical randomized experiment** is a randomized experiment with an assigning mechanism that is

1. individualistic, and
2. unconfounded.

Assigning Mechanism

Bernoulli Trials:

$$P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = \prod_{i=1}^{N} \left[e(X_i)^{W_i} (1 - e(X_i))^{1-W_i} \right],$$

where $e(x)$ is the propensity score.

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where $e(x)$ is the propensity score.

- It is a classical randomized experiment.
- It is usually the case for observational studies.
- Usually, $e(X) > 0.5$ is more practical. (e.g. disease treatment)
- Advantage: independent assignments.
- Disadvantage: potentially highly-imbalanced treatment groups.

Assigning Mechanism

Completely Randomized Design:

$$P(\boldsymbol{W} \mid \boldsymbol{X}, \boldsymbol{Y}(0), \boldsymbol{Y}(1)) = \begin{cases} \binom{N}{N_t}^{-1} & \text{if } \sum_{i=1}^{N} W_i = N_t, \\ 0 & \text{otherwise,} \end{cases}.$$

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- It is a randomized experiment.
- The process is same as "randomly assign N_t units to treatment group".
- Advantage: balanced treatment groups.
- Disadvantage: no control over the balance of covariates.
- Disadvantage: weak negative correlation between assignments.

Assigning Mechanism

Stratified/Blocked Randomized Experiment:
All units are divided into J blocks. Within each block, the units are randomly assigned according to the compeltely randomized design.

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- The blocks are usually determined by the covariates that are believed to be important for potential outcomes.
- Advantage: better control over the balance of covariates.
- Advantage: accuracy gain from more balanced units.
- Disadvantage: potential accuracy loss from stratification, especially for small stratas.

More discussion in Imbens (2011), *On the Finite Sample Benefits of Stratification, Blocking and Pairing in Randomized Experiments*.

Assigning Mechanism

Paired Randomized Experiment:
Each unit is paired with another unit based on the covariates. Within each pair, one of them is randomly assigned to treatment.

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- Advantage: accuracy gain from more balanced units.
- Disadvantage: accuracy depends on pairing quality.

Potential Outcome Framework for Causal Inference

- Sampling units.
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- Potential outcomes model.
  For now: SUTVA.
- Assignment mechanism.
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- Data collection.
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Fisher's Exact P-value

The Fisher's method is to test the following **sharp null** hypotheis:

$$H_0 : Y_i(1) = Y_i(0) \quad \text{for all } i = 1, \ldots, N.$$

Recall the major problem of causal inference is only one of the two potential outcomes is observed.

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- Under Fisher's sharp null hypothesis, the unobserved counterfactuals are the same as the observed outcomes:

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- After the imputation of the counterfactuals, we can repeat the randomization and re-estimate the causal effect.
- The p-value can be estimated empirically comparing the re-estimated causal effect with the original one.

Fisher's Exact P-value

A study of 6 children on their cough frequency prior and after a honey/placebo treatment. (see Imbens and Rubin, Chapter 5.2)

Unit	Potential Outcomes				
	Cough Frequency (cfa)		Observed Variables		
	$Y_i(0)$	$Y_i(1)$	W_i	X_i (cfp)	Y_i^{obs} (cfa)
1	?	3	1	4	3
2	?	5	1	6	5
3	?	0	1	4	0
4	4	?	0	4	4
5	0	?	0	1	0
6	1	?	0	5	1

The estimated effect is 1.0. The rank difference is 0.67.

Fisher's Exact P-value

Under Fisher's sharp null hypothesis, the unobserved counterfactuals are the same as the observed outcomes:

Unit	Potential Outcomes					
	Cough Frequency (`cfa`)		Observed Variables			
	$Y_i(0)$	$Y_i(1)$	Treatment	X_i	Y_i^{obs}	$\text{rank}(Y_i^{\text{obs}})$
1	(3)	3	1	4	3	4
2	(5)	5	1	6	5	6
3	(0)	0	1	4	0	1.5
4	4	(4)	0	4	4	5
5	0	(0)	0	1	0	1.5
6	1	(1)	0	5	1	3

Fisher's Exact P-value

Given the imputed counterfactuals, we can repeat the randomization and re-estimate the causal effect. (Last line is the realized one.)

						Statistic: Absolute Value of Difference in Average	
W_1	W_2	W_3	W_4	W_5	W_6	Levels (Y_i)	Ranks (R_i)
0	0	0	1	1	1	−1.00	−0.67
0	0	1	0	1	1	−3.67	−3.00
0	0	1	1	0	1	−1.00	−0.67
0	0	1	1	1	0	−1.67	−1.67
0	1	0	0	1	1	−0.33	0.00
0	1	0	1	0	1	2.33	2.33
0	1	0	1	1	0	1.67	1.33
0	1	1	0	0	1	−0.33	0.00
0	1	1	0	1	0	−1.00	−1.00
0	1	1	1	0	0	1.67	1.33
1	0	0	0	1	1	−1.67	−1.33
1	0	0	1	0	1	1.00	1.00
1	0	0	1	1	0	0.33	0.00
1	0	1	0	0	1	−1.67	−1.33
1	0	1	0	1	0	−2.33	−2.33
1	0	1	1	0	0	0.33	0.00
1	1	0	0	0	1	1.67	1.67
1	1	0	0	1	0	1.00	0.67
1	1	0	1	0	0	3.67	3.00
1	1	1	0	0	0	**1.00**	**0.67**

- The Fisher's exact p-value can be computed using the empirical distribution of the test statistics.

Neyman's Method

Neyman's method is to test the following null hypothesis:

$$H_0 : \bar{Y}(1) = \bar{Y}(0)$$

where $\bar{Y}(1)$ and $\bar{Y}(0)$ are the average potential outcomes for treated and control. The hypothesis is equivalent to

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$$H_0 : \tau = 0.$$

We consider the **difference-in-means** estimator:

$$\hat{\tau} = \bar{Y}_t^{obs} - \bar{Y}_c^{obs} = \frac{1}{N_t} \sum_{i:W_i=1} Y_i^{obs} - \frac{1}{N_c} \sum_{i:W_i=0} Y_i^{obs}$$

Neyman's Method

The bias and variance of the estimator is given by

$$\mathbb{E}(\hat{\tau}) = \tau,$$
$$\mathrm{Var}(\hat{\tau}) = \frac{S_c^2}{N_c} + \frac{S_t^2}{N_t} - \frac{S_{ct}^2}{N},$$

where

$$S_c^2 = \frac{1}{N-1}\sum_{i=1}^{N}(Y_i(0) - \bar{Y}(0))^2,$$
$$S_t^2 = \frac{1}{N-1}\sum_{i=1}^{N}(Y_i(1) - \bar{Y}(1))^2,$$
$$S_{ct}^2 = \frac{1}{N-1}\sum_{i=1}^{N}(Y_i(1) - Y_i(0) - \tau)^2.$$

Neyman's Method

- The first two variances can be estiamted from the observed data:

$$s_c^2 = \frac{1}{N_c - 1} \sum_{i:W_i=0} (Y_i(0) - \bar{Y}_c^{obs})^2,$$

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- The above estimation is unbiased only when τ_i is constant across units, i.e. Fisher's sharp null.
- The rest follows a standard Z-test.

Fisher v.s. Neyman

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- Logically, rejecting Neyman's null should also reject Fisher's null.
- That is it should reject the Fisher's null more often than the Neyman's.
- Not even mention that Neyman's method is more conservative.
- But in many realized cases, the Neyman's method rejects more often than Fisher's.
- See Peng Ding's paper:

  A Paradox from Randomization-Based Causal Inference

Beyond the Simplest Case

Beyond the simplest case with completely randomized design and few covariates, we will discuss the following extension in the upcoming sesions.

- Voilation of SUTVA: interference, network data, spillover effects.
- Voilation of ignorability: confounders, selection bias
- Voilation of consistency: non-compliance, instrumental variables.
- Other types of estimand and models: discountinuity design, regression-based methods, etc..

Thank you for joining the workshop!

Contact:

Me	chencheng.cai@wsu.edu
CISER	ciser.info@wsu.edu