# Causal Inference: Basic Concepts and Randomized Experiments

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# Concepts Covered Today

- Association versus causation
- Defining causal quantities with counterfactual/potential outcomes
- Connection to missing data
- Identification of the average treatment effect in a completely randomized experiment
- Covariate balance

# Does daily smoking cause a decrease in lung function?

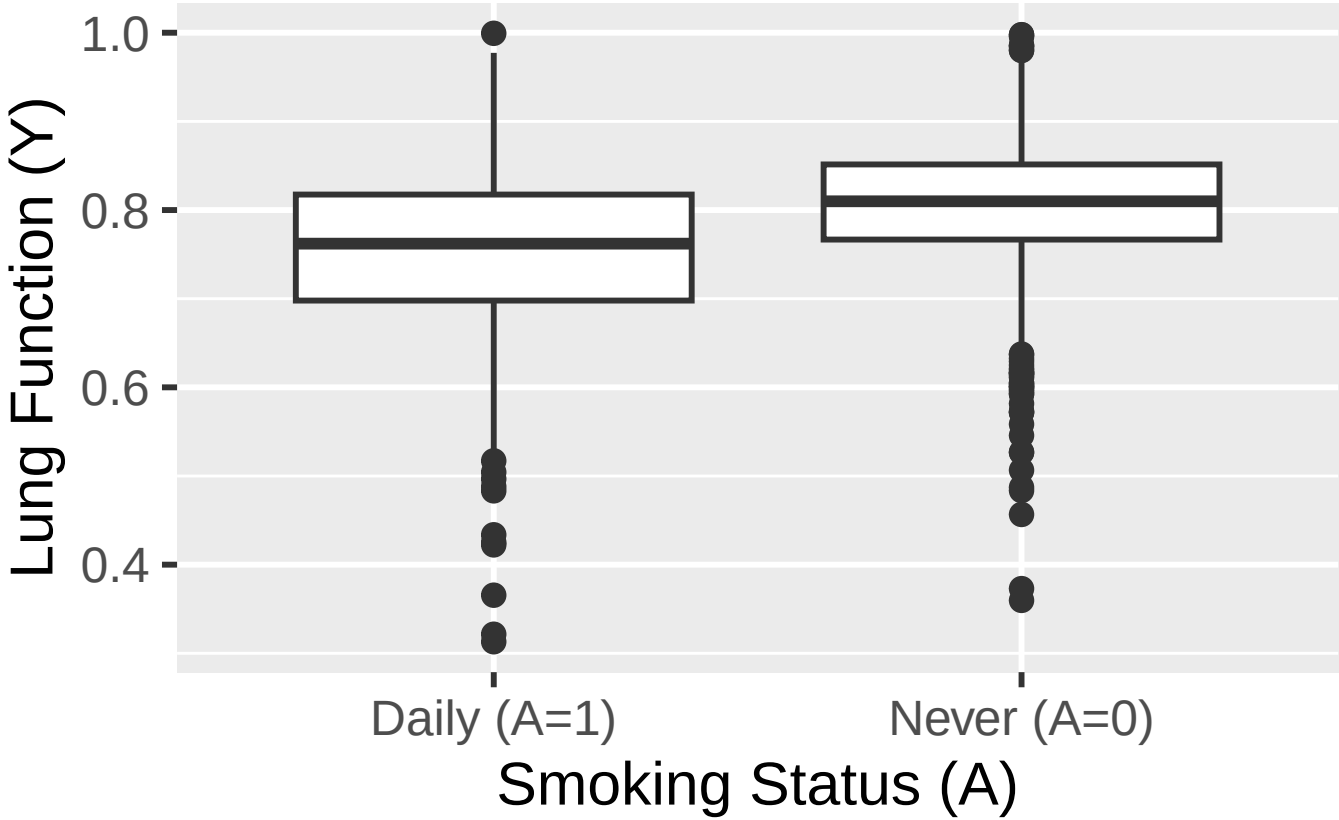
Data: 2009-2010 National Health and Nutrition Examination Survey (NHANES).

- Treatment ($A$): Daily smoker ($A = 1$) vs. never smoker ($A = 0$)
- Outcome ($Y$): ratio of forced expiratory volume in one second over forced vital capacity. $Y \geq 0.8$ is good lung function!
- Sample size is $n = 2360$.

Table 1: A Subset of the Observed Data

| Lung Function (Y) | Smoking Status (A) |
|---|---|
| 0.940 | Never |
| 0.918 | Never |
| 0.808 | Daily |
| 0.838 | Never |

# Association of Smoking and Lung Function




- $\overline{Y}_{\text{daily(A=1)}} = 0.75$ and $\overline{Y}_{\text{never(A=0)}} = 0.81$.
- $t$-stat = -11.8, two-sided p value: $\ll 10^{-16}$

Daily smoking is **strongly associated** with 0.06 reduction in lung function.

But, is the strong association evidence for **causality**?

# Definition of Association

**Association**: $A$ is associated with $Y$ if $A$ is *informative* about $Y$

- If you smoke daily $(A = 1)$, then it's likely that your lungs aren't functioning well $(Y)$.
- If smoking status doesn't provide any information about lung function, $A$ is not associated with $Y$.

Formally, $A$ is associated with $Y$ if $\mathbb{P}(Y|A) \neq \mathbb{P}(Y)$.

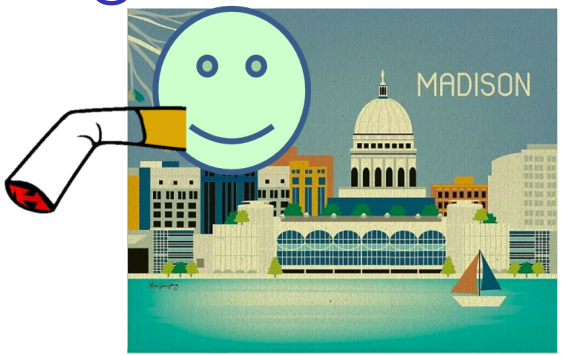
Some parameters that measure association:

- Population difference in means: $\mathbb{E}[Y|A = 1] - \mathbb{E}[Y|A = 0]$
- Population covariance: $\text{cov}(A, Y) = \mathbb{E}[(A - \mathbb{E}[A])(Y - \mathbb{E}[Y])]$

Estimators/tests that measure association:

- Sample difference in means, regression, etc.
- Two-sample t-tests, Wilcoxon signed-rank test, etc.

# Defining Causation: Parallel Universe Analogy




**John's Parallel Universe 1 (A=1)**


**John's Parallel Universe 2 (A=0)**

Suppose John's lung functions are different between the two universes.

- The difference in lung functions can **only be** attributed to the difference in smoking status.
- Why? All variables (except smoking status) are the same between the two parallel universes.

**Key Point**: comparing outcomes between parallel universes enable us to say *any difference* in the outcome must be due to a *difference in the treatment status.*

This provides a basis for defining a causal effect of $A$ on $Y$.

# Counterfactual/Potential Outcomes

Notation for outcomes in parallel universes:

- $Y(1)$: counterfactual/potential lung function if you smoked (i.e. parallel world where you smoked)
- $Y(0)$: counterfactual/potential lung function if you didn't smoke (i.e. parallel world where you didn't smoke)

Similar to the observed data table, we can create counterfactual/potential outcomes data table.

| | $Y(1)$ | $Y(0)$ |
|---|---|---|
| John | 0.5 | 0.9 |
| Sally | 0.8 | 0.8 |
| Kate | 0.9 | 0.6 |
| Jason | 0.6 | 0.9 |

For pedagogy, we'll assume that all data tables are an i.i.d. sample from some population (i.e. $Y_i(1), Y_i(0) \overset{\text{i.i.d.}}{\sim} \mathbb{P}\{Y(1), Y(0)\}$).

# Causal Estimands

| | $Y(1)$ | $Y(0)$ |
|---|---|---|
| John | 0.5 | 0.9 |
| Sally | 0.8 | 0.8 |
| Kate | 0.9 | 0.6 |
| Jason | 0.6 | 0.9 |

Some quantities/parameters from the counterfactual outcomes:

- $Y_{\text{John}}(1) - Y_{\text{John}}(0) = -0.4$: Causal effect of John smoking versus not smoking (i.e. *individual treatment effect*)
- $\mathbb{E}[Y(1)]$: Average of counterfactual outcomes when everyone is a daily smoker.
- $\mathbb{E}[Y(1) - Y(0)]$: Difference in the average counterfactual outcomes when everyone is smoking versus when everyone is not smoking (i.e. *average treatment effect*, *ATE*)

A **causal estimand/parameter** is a function of the counterfactual outcomes.

# Counterfactual Data Versus Observed Data

Table 4: Comparison of tables.

(a) Counterfactual table

| | $Y(1)$ | $Y(0)$ |
|---|---|---|
| John | 0.5 | 0.9 |
| Sally | 0.8 | 0.8 |
| Kate | 0.9 | 0.6 |
| Jason | 0.6 | 0.9 |

(b) Observed table

| | $Y$ | $A$ |
|---|---|---|
| John | 0.9 | 0 |
| Sally | 0.8 | 1 |
| Kate | 0.6 | 0 |
| Jason | 0.6 | 1 |

For both, we can define parameters (i.e. $\mathbb{E}[Y]$ or $\mathbb{E}[Y(1)]$) and take i.i.d. samples from their respective populations to learn them.

- $Y_i(1), Y_i(0) \overset{\text{i.i.d.}}{\sim} \mathbb{P}\{Y(1), Y(0)\}$ and $\mathbb{P}$ is Uniform, etc.

**If** we can observe the counterfactual table, we can run your favorite statistical methods and estimate/test causal estimands.

# Fundamental Problem of Causal Inference

**If** we can observe all counterfactual outcomes, causal inference reduces to doing usual statistical analysis with $Y(0), Y(1)$

But, in many cases, we don't get to observe all counterfactual outcomes.

A key goal in causal inference is to learn about the counterfactual outcomes $Y(1), Y(0)$ from the observed data $(Y, A)$.

- How do we learn about causal parameters (e.g. $\mathbb{E}[Y(1)]$) from the observed data $(Y, A)$
- What causal parameters are impossible to learn from the observed data?

Addressing this type of question is referred to as **causal identification**.

# Causal Identification: SUTVA or Causal Consistency

First, let's make the following assumption known as stable unit treatment value assumption (SUTVA) or causal consistency (Rubin (1980), page 4 of Herna'n MA (2020)).

$$Y = AY(1) + (1 - A)Y(0)$$

Equivalently,

$$Y = Y(A) \text{ or if } A = a, Y = Y(a)$$

The assumption states the observed outcome is one realizaton of the counterfactual outcomes.

- It also states that there are **no multiple versions of treatment.**
- It also states that there is **no interference**, a term coined by Cox (1958).

# No Multiple Versions of Treatment

Daily smoking (i.e. $A = 1$) can include different type of smokers

- Daily smoker who smokes one pack of cigarettes per day
- Daily smoker who smokes one cigarette per day
- Daily smoker who vapes per day

The current $Y(1)$ does not distinguish outcomes between different types of smokers.

We can define counterfactual outcomes for all kinds of daily smokers, say $Y(k)$ for $k = 1, ..., K$ type of daily smokers. But, if $A = 1$, which counterfactual outcome should this correspond to?

SUTVA eliminates these variations in the counterfactuals. Or, if $Y(k)$ exists, it assumes that these variations $Y(1) = Y(2) = ... = Y(K)$.

Implicitly, SUTVA forces you to define meaningful $Y(a)$. Some authors restrict counterfactual outcomes to be based on well-defined interventions or "no causation without manipulation" (Holland (1986) Hernán and Taubman (2008) Cole and Frangakis

# No Interference

Suppose we want to study the causal effect of getting the measles vaccine on getting the measles. Let's define the following counterfactual outcomes:

- $Y(0)$: Jamie's counterfactual measles status when Jamie is not vaccinated
- $Y(1)$: Jamie's counterfactual measles status when Jamie is vaccinated

Suppose Jamie has a sibling Alex and let's entertain the possible values of Jamie's $Y(0)$ based on Alex's vaccination status.

- Jamie's counterfactual measles status when Alex is vaccinated.
- Jamie's counterfactual measles status when Alexis not vaccinated.

The current $Y(0)$ does not distinguish between the two counterfactual outcomes.

# No Interference

We can again define counterfactual outcomes to incorporate this scenario, say $Y(a, b)$ where $a$ refers to Jamie's vaccination status and $b$ refers to Alex's vaccination status.

SUTVA states that Jamie's outcome only depends on Jamie's vaccination status, not Alex's vaccination status. Or, more precisely $Y(a, b) = Y(a, b')$ for all $a, b, b'$.

In some studies, the no interference assumption is not plausible (e.g. vaccine studies, peer effects in classrooms/neighborhoods, air pollutions). Rosenbaum (2007) has a nice set of examples of when the no interference assumption is not plausible.

There is **a lot** of ongoing work on this topic (Rosenbaum (2007),Hudgens and Halloran (2008), Tchetgen and VanderWeele (2012)). I am interested in in this area as well and let me know if you want to learn more.

# Causal Identification and Missing Data

Once we assume SUTVA (i.e. $Y = AY(1) + (1 - A)Y(0)$), causal identification can be seen as a problem in missing data.

| | $Y(1)$ | $Y(0)$ | $Y$ | $A$ |
|---|---|---|---|---|
| John | NA | 0.9 | 0.9 | 0 |
| Sally | 0.8 | NA | 0.8 | 1 |
| Kate | NA | 0.6 | 0.6 | 0 |
| Jason | 0.6 | NA | 0.6 | 1. |

Under SUTVA, we only see one of the two counterfactual outcomes based on $A$.

- $A$ serves as the "missingness" indicator where $A = 1$ implies $Y(1)$ is observed and $A = 0$ implies $Y(0)$ is observed.
- $Y$ is the "observed" value.
- Being able to only observe one counterfactual outcome in the observed data is known as the "fundamental problem of causal inference'' (page 476 of Holland (1988)).

# Assumption on Missingness Pattern

| | $Y(1)$ | $Y(0)$ | $Y$ | $A$ |
|---|---|---|---|---|
| John | NA | 0.9 | 0.9 | 0 |
| Sally | 0.8 | NA | 0.8 | 1 |
| Kate | NA | 0.6 | 0.6 | 0 |
| Jason | 0.6 | NA | 0.6 | 1. |

Suppose we are interested in learning the causal estimand $\mathbb{E}[Y(1)]$ (i.e. the mean of the first column).

One approach would be to take the average of the "complete cases" (i.e. Sally's 0.8 and Jason's 0.6).

- Formally, we would use $\mathbb{E}[Y|A=1]$, the mean of the observed outcome $Y$ among $A=1$.
- This approach is valid if the entries of the first column are **missing completely at random** (MCAR)
- In other words, the missingness indicator $A$ flips a random coin per each individual and decides whether its $Y(1)$ is

# Formal Statement of MCAR

Formally, MCAR can be stated as

$$A \perp Y(1) \text{ and } 0 < \mathbb{P}(A = 1)$$

- $A \perp Y(1)$ states that missingness is independent of $Y(1)$
  i. Missingness occurs completely at random in the rows of the first column, say by a flip of a random coin.
  ii. Missingness doesn't occur more frequently for higher values of $Y(1)$; this would violate $A \perp Y(1)$.
- $0 < \mathbb{P}(A = 1) < 1$ states that you have a non-zero probability of observing some entries of the column $Y(1)$
  i. If $\mathbb{P}(A = 1) = 0$, then all entries of the column $Y(1)$ are missing and we can't learn anything about its column mean.

# Formal Proof of Causal Identification of $\mathbb{E}[Y(1)]$

Suppose SUTVA and MCAR hold:

- (A1): $Y = AY(1) + (1-A)Y(0)$
- (A2): $A \perp Y(1)$
- (A3): $0 < \mathbb{P}(A = 1)$

Then, we can identify the causal estimand $\mathbb{E}[Y(1)]$ by writing it as the following function of the observed data $\mathbb{E}[Y|A=1]$:

$$\begin{aligned}
\mathbb{E}[Y|A=1] &= \mathbb{E}[AY(1) + (1-A)Y(0)|A=1] && \text{(A1)} \\
&= \mathbb{E}[Y(1)|A=1] && \text{Definition of conditional} \\
&= \mathbb{E}[Y(1)] && \text{(A2)}
\end{aligned}$$

(A3) is used to ensure that $\mathbb{E}[Y|A=1]$ is a well-defined quantity.

# Causal Identification of the ATE

In a similar vein, to identify the ATE $\mathbb{E}[Y(1) - Y(0)]$, a natural approach would be to use $\mathbb{E}[Y|A = 1] - \mathbb{E}[Y|A = 0]$, respectively.

This approach would be valid under the following variation of the MCAR assumption:

$$A \perp Y(0), Y(1), \quad 0 < \mathbb{P}(A = 1) < 1$$

- The first part states that the treatment $A$ is independent of $Y(1), Y(0)$. This is called **exchangeability** or **ignorability** in causal inference.
- $0 < \mathbb{P}(A = 1) < 1$ states that there is a non-zero probability of observing some entries from the column $Y(1)$ and from the column $Y(0)$. This is called **positivity** or **overlap** in causal inference.

# Formal Proof of Causal Identification of the ATE

Suppose SUTVA and MCAR hold:

- (A1): $Y = AY(1) + (1-A)Y(0)$
- (A2): $A \perp Y(1), Y(0)$
- (A3): $0 < \mathbb{P}(A = 1) < 1$

Then, we can identify the ATE from the observed data via:

$$
\begin{aligned}
&\mathbb{E}[Y|A=1] - \mathbb{E}[Y|A=0] \\
=&\mathbb{E}[AY(1) + (1-A)Y(0)|A=1] \\
&\quad - \mathbb{E}[AY(1) + (1-A)Y(0)|A=0] && \text{(A1)} \\
=&\mathbb{E}[Y(1)|A=1] - \mathbb{E}[Y(0)|A=0] && \text{Definition of conditional expecta} \\
=&\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] && \text{(A2)}
\end{aligned}
$$

(A3) ensures that the conditioning events in $\mathbb{E}[|A=0]$ and $\mathbb{E}[|A=1]$ are well-defined.

# Why Randomized Experiments Identify Causal Effects

Consider an ideal, completely randomized experiment (RCT):

1. Treatment & control are well-defined (e.g. take new drug or placebo)
2. Counterfactual outcomes do not depend on others' treatment (e.g. taking the drug/placebo only impacts my own outcome)
3. Assignment to treatment or control is completely randomized
4. There is a non-zero probability of receiving treatment and control (e.g. some get drug while others get placebo)

Assumptions (A1)-(A3) are satisfied because

- From 1 and 2, SUTVA holds.
- From 3, treatment assignment $A$ is completely random, i.e. $A \perp Y(1), Y(0)$
- From 4, $0 < P(A = 1) < 1$

This is why RCTs are considered the **gold standard** for identifying causal effects as all assumptions for causal identification are satisfied by the experimental design.

# RCTs with Covariates

In addition to $Y$ and $A$, we often collect pre-treatment covariates $X$.

| | $Y(1)$ | $Y(0)$ | $Y$ | $A$ | X (Age) |
|---|---|---|---|---|---|
| John | NA | 0.9 | 0.9 | 0 | 38 |
| Sally | 0.8 | NA | 0.8 | 1 | 30 |
| Kate | NA | 0.6 | 0.6 | 0 | 23 |
| Jason | 0.6 | NA | 0.6 | 1 | 26 |

If the treatment $A$ is completely randomized (as in an RCT), we would also have $A \perp X$.

Note that we can then combine this into the existing (A2) as (A2):

$$A \perp XY(1), Y(0)$$

Other assumptions, (A1) and (A3), remain the same.

# Causal Identification of The ATE with Covariates

Even with the change in (A2), the proof to identify the ATE in an RCT remains the same as before.

- (A1): $Y = AY(1) + (1 - A)Y(0)$
- (A2): $A \perp Y(1), Y(0), X$
- (A3): $0 < \mathbb{P}(A = 1) < 1$

Then, we can identify the ATE from the observed data via:

$$\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] = \mathbb{E}[Y|A = 1] - \mathbb{E}[Y|A = 0]$$

However, we can also identify the ATE via

$$\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] = \mathbb{E}[\mathbb{E}[Y|X, A = 1]|A = 1] - \mathbb{E}[\mathbb{E}[Y|X, A = 0]|A = 0]$$

The new equality simply uses the law of total expectation, i.e. $\mathbb{E}[Y|A = 1] = \mathbb{E}[\mathbb{E}[Y|X, A = 1]|A = 1]$. However, this new equality requires modeling $\mathbb{E}[Y|X, A = a]$ correctly. We'll discuss more about this in later lectures.

# Covariate Balance

An important, conceptual implication of complete randomization of the treatment (i.e. $A \perp X$) is that

$$\mathbb{P}(X|A = 1) = \mathbb{P}(X|A = 0)$$

This concept is known as **covariate balance** where the distribution of covariates are balanced between treated units and control units.

Often in RCTs (and non-RCTs), we check for covariate balance by comparing the means of $X$s among treated and control units (e.g. two-sample t-test of the mean of $X$). This is to ensure that randomization was actually carried out properly.

# RCT Balances Measured and Unmeasured Covariates

Critically, the above equality would hold even if some characteristics of the person are unmeasured (e.g. everyone's precise health status).

- Formally, let $U$ be unmeasured variables and $X$ be measured variables.
- Because $A$ is completely randomized in an RCT, we have $A \perp X, U$ and

$$\mathbb{P}(X, U|A = 1) = \mathbb{P}(X, U|A = 0)$$

Complete randomization ensures that the distribution of both **measured** and **unmeasured** characteristics of individuals are the same between the treated and control groups.

# Randomization Creates Comparable Groups

Roughly speaking, completely randomization creates two synthetic, parallel universes where, on average, the characteristics between universe $A = 0$ and universe $A = 1$ are identical.

Thus, in an RCT, any difference in $Y$ can **only be attributed** a difference in the group label (i.e. $A$) since all measured and unmeasured characteristics between the two universes are distributionally identical.

This was essentially the "big" idea from Fisher in 1935 where he used randomization as the "reasoned basis' ' for causal inference from RCTs. Paul Rosenbaum explains this more beautifully than I do in Chapter 2.3 of Rosenbaum (2020).

# Note About Pre-treatment Covariates

We briefly mentioned that covariates $X$ must precede treatment assignment, i.e.

1. We collect $X$ (i.e. baseline covariates)
2. We assign treatment/control $A$
3. We observe outcome $Y$

If they are post-treatment covariates, then the treatment can have a causal effect on both the outcome $Y$ and the covariates $X$.

In this case, it's not unclear whether $Y$ has a causal effect because of a causal effect in $X$. Studying this type of question is called **causal mediation analysis.**

In general, we don't want to condition on post-treatment covariates $X$ when the goal is to estimate the average treatment effect of $A$ on $Y$.

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