MY457/MY557: Causal Inference for Observational and Experimental Studies

Week 1: Causal Frameworks

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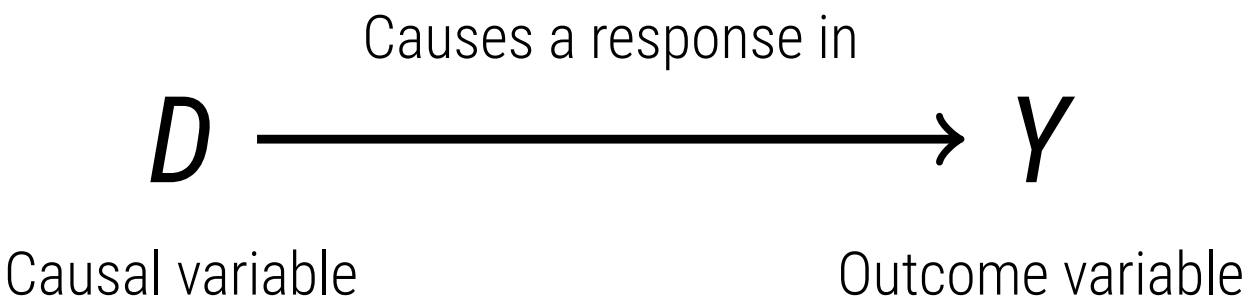
Lecture Roadmap

A (Very) Simple Causal Model



Effects of Causes

Causes and their effects have two properties: they are successive and can be reasoned about in counterfactual terms:

> *[...] We may define a cause to be an object followed by another, [...] where, if the first object had not been, the second never had existed.*
>
> *— Hume, 1748*

> *[...] would not have died if he had not eaten of it, people would be apt to say that eating of that dish was the cause of his death.*
>
> *— Mill, 1843*

One important implication is that causal variables must be manipulable:

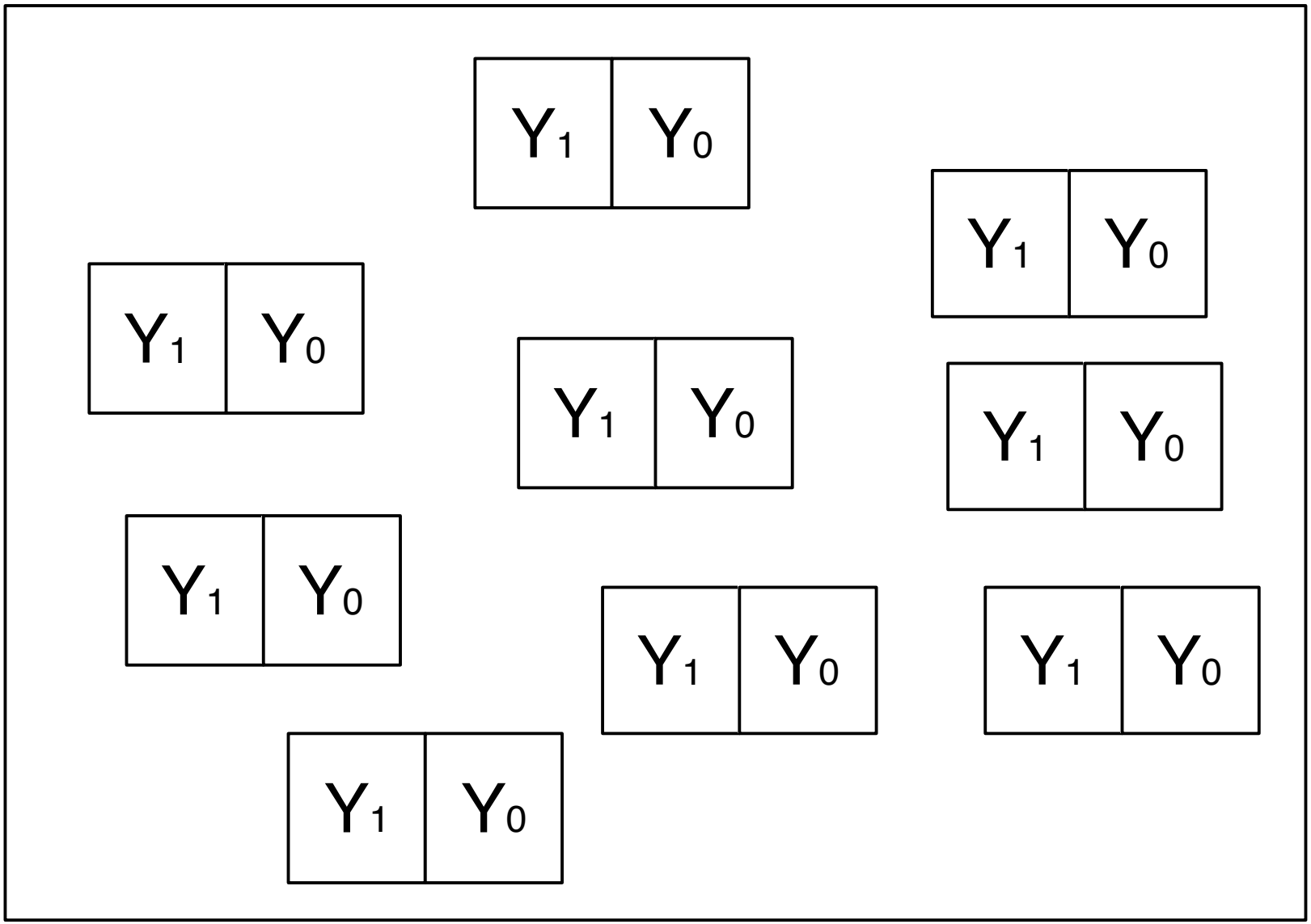
> *No causation without manipulation.*
>
> *— Holland, 1986*

Good Causal Questions

Manipulability means we must think very carefully about causal questions...

- (Largely) immutable characteristics?
  - Judges' sex assigned at birth $\rightarrow$ decision making
  - Race and ethnicity $\rightarrow$ employment outcomes
  - Country of origin $\rightarrow$ political beliefs
- Major global events?
  - Russian revolution $\rightarrow$ Karl Marx's intellectual popularity
  - 9/11 $\rightarrow$ Arab Spring
- Non-successive chains?
  - Monthly expenditure $\rightarrow$ monthly savings
  - Holocaust $\rightarrow$ modern AFD election support

Neyman Urn Model



Concepts: Treatment, Outcomes, and Potential Outcomes

Definition (Treatment)

D_i: Indicator of treatment intake for *unit* i

$$D_i = \begin{cases} 1 & \text{if unit } i \text{ received the treatment} \\ 0 & \text{otherwise.} \end{cases}$$

Definition (Observed outcome)

Y_i: Observed outcome variable of interest for unit i

Definition (Potential Outcome)

Y_{0i} and Y_{1i}: Potential outcomes for unit i:

$$\begin{array}{ll} Y_{1i} & \text{Outcome for unit } i \text{ when } D_i = 1 \\ Y_{0i} & \text{Outcome for unit } i \text{ when } D_i = 0 \end{array}$$

(Alternative notation: $Y_i(d)$, Y_i^d, etc.)

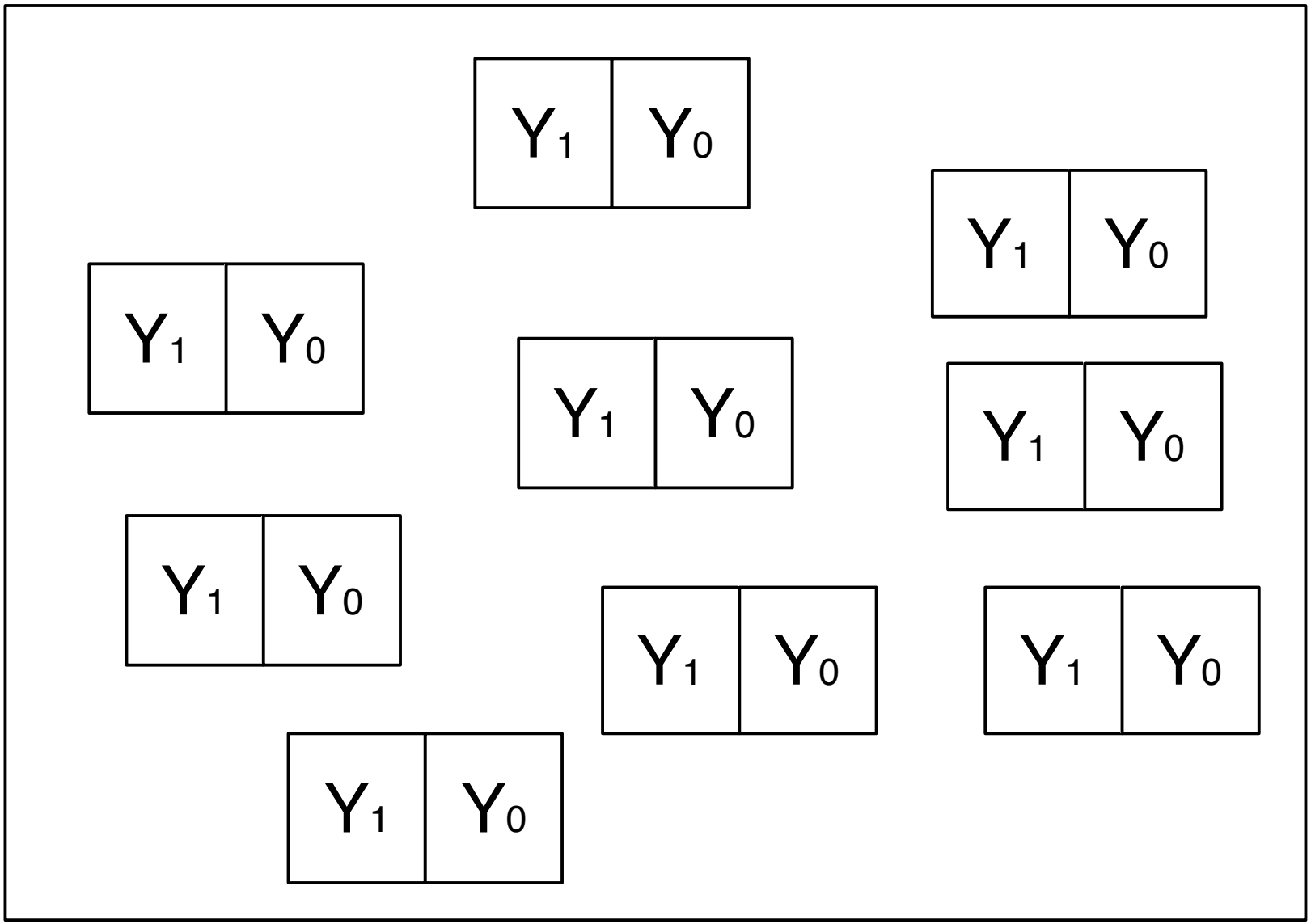
Concepts: Treatment, Outcomes, and Potential Outcomes

Under further assumptions ('SUTVA', more later), we can connect these three concepts mathematically:

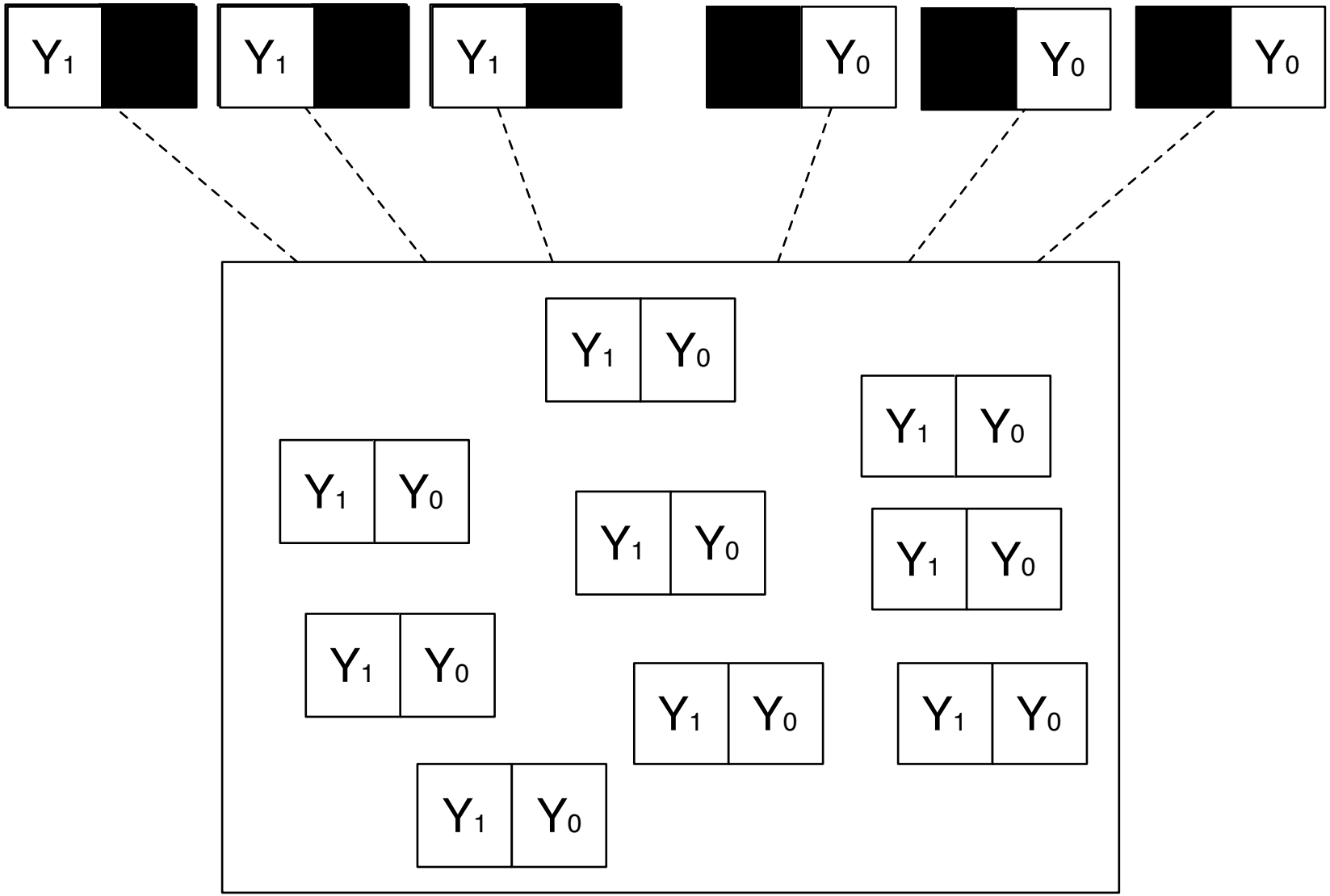
$$\begin{aligned} Y_i &= D_i \cdot Y_{1i} + (1 - D_i) \cdot Y_{0i} \\ \text{i.e.} \quad Y_i &= \begin{cases} Y_{1i} & \text{if } D_i = 1 \\ Y_{0i} & \text{if } D_i = 0 \end{cases} \end{aligned}$$

- *A priori* each potential outcome could be observed (manipulability!)
- After treatment assignment, one is observed, the other is counterfactual

Neyman Urn Model



Neyman Urn Model



Stable Unit Treatment Value Assumption (SUTVA)

- Recall: $Y_i = Y_{D_i i}$, or equivalently $Y_i = D_i Y_{1i} + (1 - D_i) Y_{0i}$
- This notation implicitly makes the following assumption:

Assumption (SUTVA)

$$Y_{(D_1, D_2, \ldots, D_N)i} = Y_{(D'_1, D'_2, \ldots, D'_N)i} \quad \text{if} \quad D_i = D'_i$$

SUTVA comprises two sub-assumptions:

1. No interference between units
   - Potential outcomes for a unit not affected by treatment status of other units
   - Violations: spill-over effects, contagion, dilution
2. No different versions of treatment (stability, consistency)
   - Nominally identical treatments are in fact identical
   - Violations: variable levels of treatment, technical errors

Causal Inference Without SUTVA

Let $\mathbf{D} = (D_1, D_2)$ be a vector of binary treatments for $N = 2$.

How many different values can $\mathbf{D}$ possibly take?

$$(D_1, D_2) = (0,0) \text{ or } (1,0) \text{ or } (0,1) \text{ or } (1,1)$$

How many potential outcomes for unit 1?

$$Y_{(0,0)1},\ Y_{(1,0)1},\ Y_{(0,1)1}\ Y_{(1,1)1}.$$

How many causal effects for unit 1?

$$\begin{aligned} &Y_{(1,1)1} - Y_{(0,0)1}, \quad Y_{(1,1)1} - Y_{(0,1)1}, \\ &Y_{(1,0)1} - Y_{(0,0)1}, \quad Y_{(1,0)1} - Y_{(0,1)1}, \\ &Y_{(1,1)1} - Y_{(1,0)1}, \quad Y_{(0,1)1} - Y_{(0,0)1}. \end{aligned}$$

How many observed outcomes for unit 1? Only one: $Y_1 = Y_{(D_1,D_2)1}$

Without SUTVA, causal inference is exponentially more difficult as $n \uparrow$.

Causal Inference as a Missing Data Problem

Imagine a population with 4 units:

i	D_i	Y_i	Y_{1i}	Y_{0i}
1	1	3	3	0
2	1	1	1	1
3	0	0	1	0
4	0	1	1	1

- We take the values of both Y_{1i} and Y_{0i} to be real and fixed for all i
- But we can only observe one of them for any i ...
- This is known as the fundamental problem of causal inference (FPCI)

Causal Inference as a Missing Data Problem

... because of the FPCI we see only this:

i	D_i	Y_i	Y_{1i}	Y_{0i}
1	1	3	3	?
2	1	1	1	?
3	0	0	?	0
4	0	1	?	1

Our goal:

- define causal estimands in terms of potential outcomes (previous table)
- estimate them using observable data on this slide (previous table)
- essentially: fill in the missing counterfactuals as best as possible!

Esti-what?

Estimand:

$\rightsquigarrow$ Unobserved population parameter or function.

Estimator:

$\rightsquigarrow$ A function that can be applied to observed data.

Estimate:

$\rightsquigarrow$ A specific output of said function.

Unit-Level Causal Estimands

Definition (Individual Treatment Effect)

Causal effect of the treatment on the outcome for unit i, defined by the comparison of two potential outcomes:

$$\tau_i = Y_{1i} - Y_{0i}$$

This cannot be observed, and is also very hard to estimate:

- We cannot observe both potential outcomes Y_{1i} and Y_{0i} for the same unit i.
- Hard to reliably fill in the missing potential outcome for any one unit i.

Group-Level Causal Estimands

- Consider a fixed group (population) of units $i = 1, \ldots, N$
- Values of the potential outcomes for this population can be represented as two vectors:

$$\mathbf{Y}_1 = (Y_{11}, Y_{12}, \ldots, Y_{1N})$$
$$\mathbf{Y}_0 = (Y_{01}, Y_{02}, \ldots, Y_{0N})$$

- A population causal estimand is a comparison of $\mathbf{Y}_1$ and $\mathbf{Y}_0$
- A common choice is a difference of their expected values (means).

Causal Estimand: The ATE

Definition (Average treatment effect, ATE)

$$\tau_{ATE} = \frac{1}{N}\sum_{i=1}^{N}(Y_{1i} - Y_{0i})$$

or equivalently

$$\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}]$$

- In the rest of this course, we will consider various assumptions under which τ_{ATE} can be identified from observed information
- Note on notation: We represent the estimand as a greek letter (in this case τ, but could be anything). We typically represent an estimator for that estimand as a greek letter with something on top (e.g. $\tilde{\tau}$ or $\hat{\tau}$). An estimate will be a realised number (interval, etc.).

Causal Estimand: The ATT

Definition (Average treatment effect on the treated, ATT)

$$\tau_{ATT} = \frac{1}{N_1} \sum_{i=1}^{N} D_i (Y_{1i} - Y_{0i}) \quad \text{where} \quad N_1 = \sum_{i=1}^{N} D_i$$

or equivalently

$$\tau_{ATT} = \mathbb{E}[Y_{1i} - Y_{0i} | D_i = 1]$$

(Note: The mathematical symbol | means "conditional on".)

- In words, N_1 equals the number of treated units.
- When would $\tau_{ATT} \neq \tau_{ATE}$? When D_i and Y_{di} are associated.
- Exercise: Define τ_{ATU}, ATE on the untreated (control) units, also called the ATU. $\tau_{ATU} = \mathbb{E}[Y_{1i} - Y_{0i} | D_i = 0]$

Causal Estimand: The CATE

Definition (Conditional average treatment effects, CATE)

$$\tau_{CATE}(x) = \mathbb{E}[Y_{1i} - Y_{0i} | X_i = x]$$

where X_i is a pre-treatment covariate for unit i

- In words, $\tau_{CATE}(x)$ is a subgroup effect, treatment effect on units who have particular characteristics x.
- This estimand sometimes goes by other names (e.g. local average treatment effect or LATE).
- This is an increasing important area for causal inference (e.g. optimal policy targeting), and we will return to it later!

Illustration: Average Treatment Effect

Let's return to our population of 4 units:

i	D_i	Y_i	Y_{1i}	Y_{0i}	τ_i
1	1	3	3	0	3
2	1	1	1	1	0
3	0	0	1	0	1
4	0	1	1	1	0
$\mathbb{E}[Y_{1i}]$			1.5		
$\mathbb{E}[Y_{0i}]$				0.5	
$\mathbb{E}[Y_{1i} - Y_{0i}]$					1

$$\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}] = \mathbb{E}[\tau_i] = \frac{3+0+1+0}{4} = 1.$$

Why does $\tau_{ATE} \neq \tilde{\tau}$? When would they be equal?

Illustration: Average Treatment Effect on the Treated

Again suppose we observe a population of 4 units:

i	D_i	Y_i	Y_{1i}	Y_{0i}	τ_i
1	1	3	3	0	3
2	1	1	1	1	0
3	0	0	1	0	1
4	0	1	1	1	0
$\mathbb{E}[Y_{1i} \mid D_i = 1]$			2		
$\mathbb{E}[Y_{0i} \mid D_i = 1]$				0.5	
$\mathbb{E}[Y_{1i} - Y_{0i} \mid D_i = 1]$					1.5

$$\tau_{ATT} = \mathbb{E}[Y_{1i} - Y_{0i} \mid D_i = 1] = \mathbb{E}[\tau_i \mid D_i = 1] = \frac{3+0}{2} = 1.5.$$

Average Treatment Effect on the Treated

Why does $\tau_{ATT} \neq \tau_{ATE}$?

Because $\mathbb{E}[Y_{1i}] \neq \mathbb{E}[Y_{1i}|D_i = 1]$ (and likewise for $\mathbb{E}[Y_{0i}]$)

That is, D_i and Y_{di} are associated

Internal and External Validity

- Note that when we talk about the 'population' in causal inference settings we often mean *only* to the ***N*** units for whom we have observed data (i.e. what we would typically call the 'sample')
- The estimands considered on this course are defined and estimated for this population (not for some super-population from which a sample was drawn)
- Internal validity refers to the validity of our estimates of these effects. This class is focused only on internal validity.

Internal and External Validity

- External validity refers to the validity generalising our estimates of causal effects from the 'population' of N units to any other population (note, this could include generalising from a realised sample to a population)
- If claimed, external validity has to be justified by different kinds of arguments, e.g.
  - Representative sampling (ideally probability sampling) of the N units from a larger population. This is a population inference task, as in survey research (see MY456).)
  - Some re-weighting strategy designed to adjust the observed sample. Again, this is a population inference task.
  - Substantive theory / assumptions / wishful thinking about why a causal effect for these N units would also apply elsewhere

Identification Problem for Causal Inference

- In statistics, an estimand (parameter) is identified if its value can be uniquely estimated based on the observed data and unidentified if not
- Recall that in causal inference, estimands are population causal effects but the FPCI tells us that at least half of the potential outcomes are always missing
- An identification strategy is a combination of data and assumptions which allows us to identify a causal estimand by estimating ("filling in") the missing potential outcomes (usually at a group level)

Selection Bias

Consider again the naïve difference of observed means in the treatment groups:

$$\underbrace{E(Y_i|D_i=1) - E(Y_i|D_i=0)}_{\text{Observed difference in average outcome measures}} = E(Y_{1i}|D_i=1) - E(Y_{0i}|D_i=0)$$

$$= \underbrace{E(Y_{1i}|D_i=1) - E(Y_{0i}|D_i=1)}_{\text{ATT}} + \underbrace{E(Y_{0i}|D_i=1) - E(Y_{0i}|D_i=0)}_{\text{Selection bias}}$$

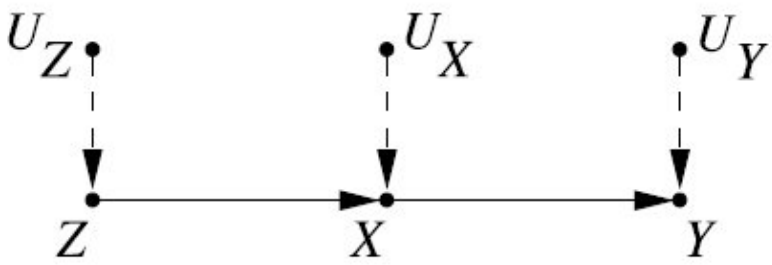
- The same observed mean difference could be due to different combinations of the ATT (estimand!) and selection bias terms. We might say the causal effect of D on Y is confounded.
- Thus ATT is not identified from the naïve observed mean difference: it is not uniquely mapped from the observed data. We need more assumptions.
- Correlation [association, here observed mean difference] is not necessarily causation.

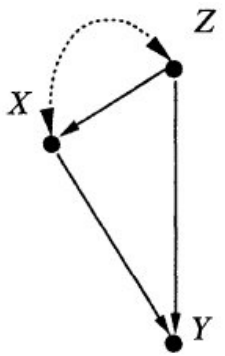
Selection Bias

$$
\begin{aligned}
& E(Y_i|D_i = 1) - E(Y_i|D_i = 0) \\
&= \quad [E(Y_{1i}|D_i = 1) - E(Y_{0i}|D_i = 1)] + [E(Y_{0i}|D_i = 1) - E(Y_{0i}|D_i = 0)]
\end{aligned}
$$

- $E(Y_{0i}|D_i = 1) - E(Y_{0i}|D_i = 0)$ is referred to as selection bias because if it is not 0, it implies treatment and control groups are systematically different in potential outcome Y_{0i}.
- Canonical example: Job training program
  - participants are self-selected from a population of individuals in difficult labor situations
  - perhaps better resourced or more motivated individuals decide to take part
  - even in the absence of the program, post-training period earnings for those people would then have been higher than those for those who did not opt in ($E[Y_0|D = 1] - E[Y_0|D = 0] > 0$)

Causal Diagrams





So far we have reasoned about causal effects using potential outcomes. An alternative (but intimately connected) framework is the graphical approach.

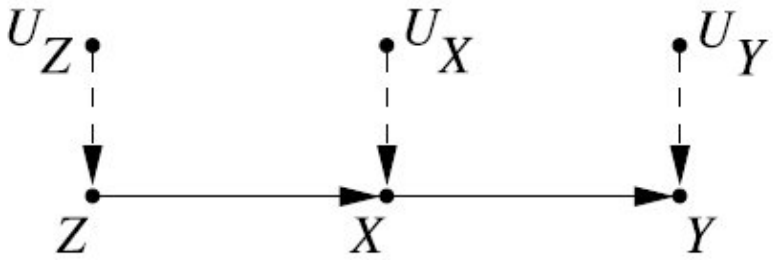
This uses causal diagrams, tools that allow us to:

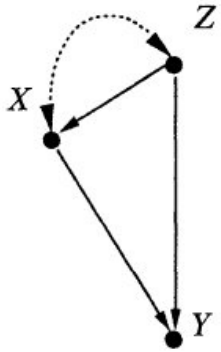
1. Specify the variables (observed and unobserved) we care about
2. Specify how those variables are connected
3. See what we can learn about causal effects, and with what assumptions.

This can help us to:

1. Study how conditioning affects our research designs
2. Create new research designs and methodologies.

Causal Diagrams as Directed Acyclic Graphs





Components of a causal diagram as a Directed Acyclic Graphs (DAG):

- Nodes: Representing “variables" (also called vertices)
- Directed Edges: Encoding one-way (causal) relationships

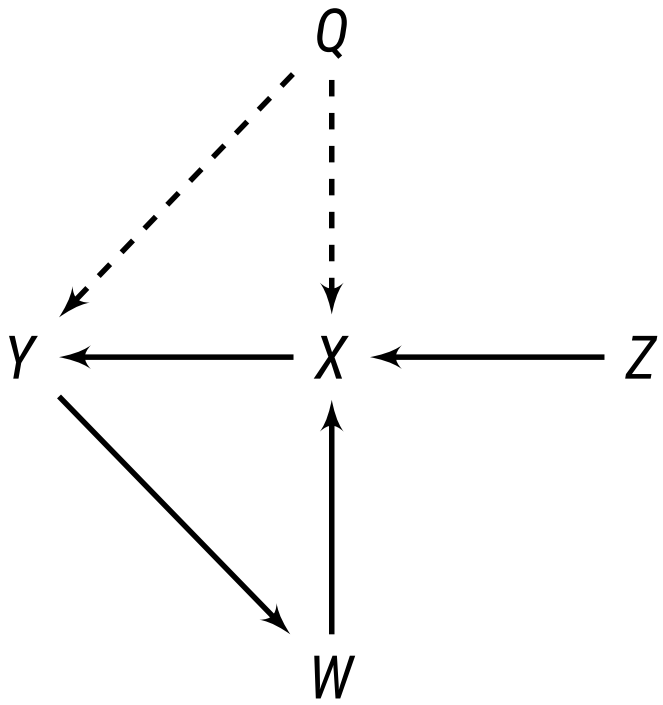
  $\rightarrow$ This implies nodes are ordered (each pair: head and tail)

  $\rightarrow$ These connections can be observed (solid) or unobserved (dashed)

Features of a DAG:

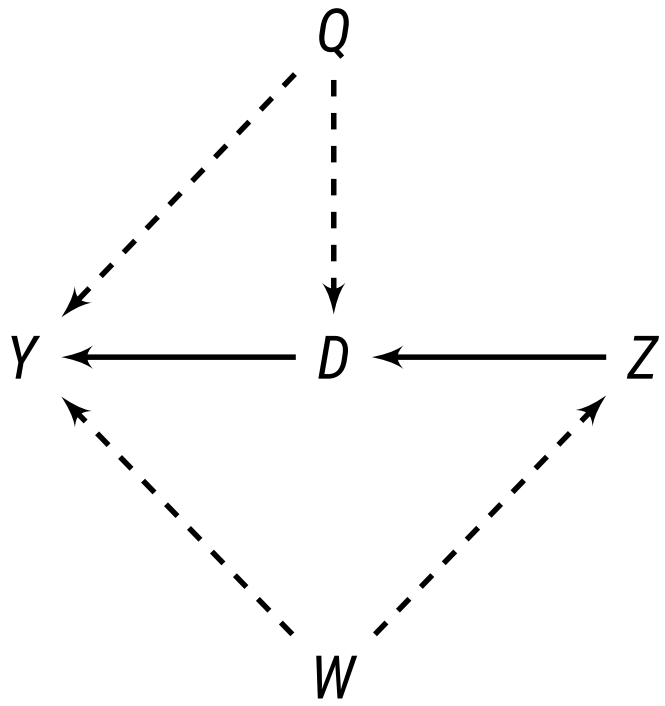
- Acyclic: No directed cycles (e.g. A does not terminate A)
- Non-connections: The absence of relationships between variables

Directed Acyclic Graphs: Example



"These aren't the DAGs you're looking for"

Directed Acyclic Graphs: Example

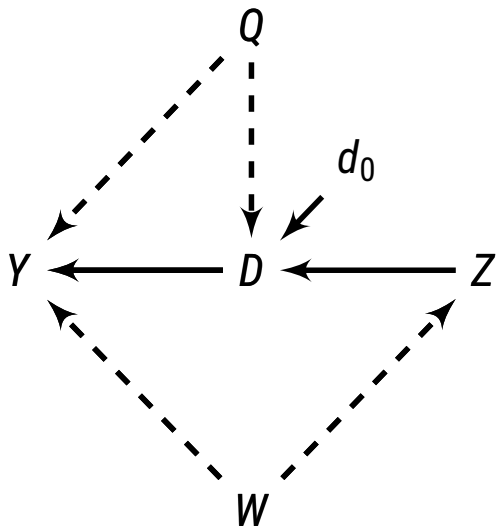


What can we learn from this DAG?

- $Z \rightarrow Y$ is confounded by W
- $D \rightarrow Y$ is confounded by Q
- $Z \rightarrow D$ is identified

But only if our DAG is correct!

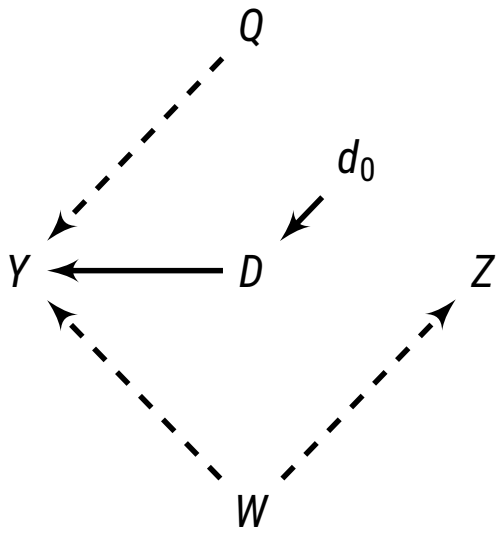
Representing Interventions



Treatments (interventions) are represented by the ***do***() operator.

For example, $do(d_0)$ holds $D = d_0$ exogenously.

Identification



ATE of D on Y defined as the average difference in Y between two interventions:

$$\mathbb{E}[Y \mid do(d_1)] - \mathbb{E}[Y \mid do(d_0)]$$

Problem: Can this be estimated without an explicit intervention (identification)?

Insight: If the DAG is equivalent with and without $do()$, yes.

Generally: We can identify the effect of D on Y if all back-door paths are blocked.

Assignment Mechanism

Definition (Assignment Mechanism)

The assignment mechanism is the procedure that determines the treatment status of each unit.

- Most causal inference methods achieve identification by restricting the (assumed) assignment mechanism
- For example, if we are willing to assume that treatment assignment is independent of potential outcomes under no treatment, then:

$$E(Y_{0i}|D_i = 1) - E(Y_{0i}|D_i = 0) = 0$$

i.e. selection bias is zero and the observed mean difference is (in expectation) equal to ATT (and also ATE in that case)

Different Assignment Mechanisms

Imbens and Rubin (2015, Ch. 3) present three assumptions about assignment mechanisms (for each unit) that provide the grounds for identification:

1. *Individualistic*: Assignment does not depend on the covariates or potential outcomes for other units.
2. *Probabilistic*: There is a nonzero probability of each treatment value, for every unit.
3. *Unconfounded*: Assignment does not depend on potential outcomes.

Assuming the above, we can distinguish:

- Experiments: The assignment mechanism is both known and controlled by the researcher, and
- Observational studies: The assignment mechanism is not known to, or not under the control of, the researcher

Our Key Assignment Mechanisms

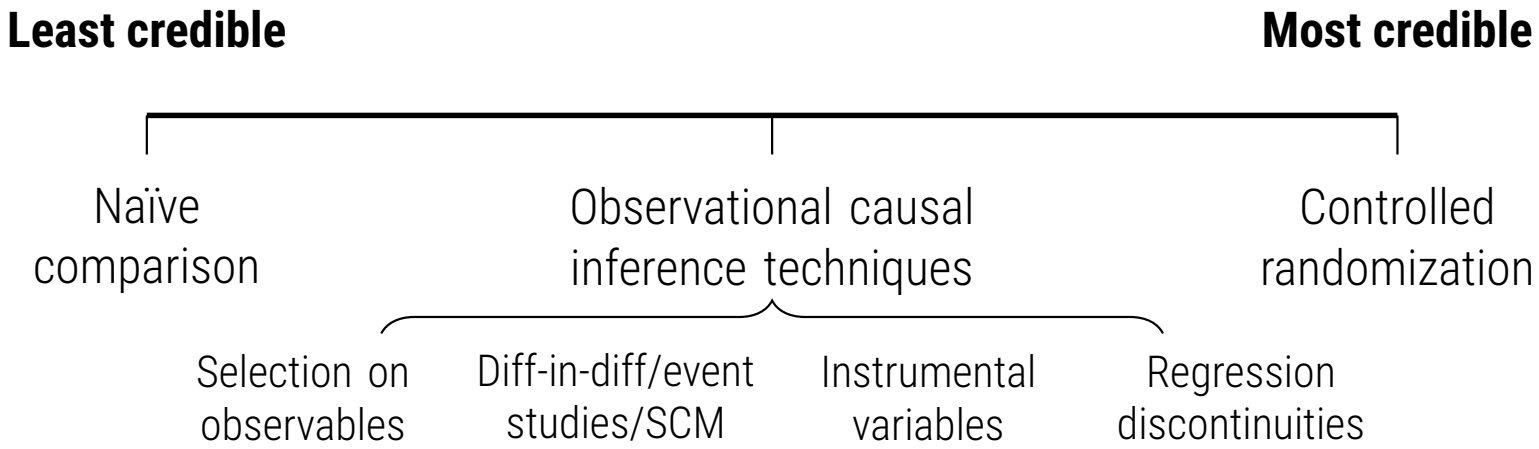
Randomised Experiments:

- These come in many flavours, only a few of which we will discuss!
  - ⇝ Within designs, between designs
  - ⇝ Unit-randomized, cluster-randomized, dynamic randomization
  - ⇝ Crossover designs, stepped-wedge designs, etc. etc. etc.

Observational Studies:

- Adjustment: Selection on observables with regression, matching, etc.
- Temporal: Diff-in-diff, event studies, synthetic control methods
- Instrumental variables, shift-share designs, etc.
- Sharp and fuzzy regression discontinuity designs

The Continuum of Credibility™



Key point: The art (and science) of applied causal inference is making defensible assumptions. There is no 'magic' solution to the fundamental problem of causal inference, only assumptions all the way down!

Key ideas from this week

- Learned to think about causal effects in terms of potential outcomes, not realized (observed) outcomes
- Observed association is neither necessary nor sufficient for causality – focused on one big problem, selection bias
- Introduced an alternative framework for thinking about causal models – the graphical approach
- Learning about causal effects should start from understanding the assignment mechanism for treatment
- Evaluate the plausibility of your assumptions to understand the credibility of your conclusions