MY457/MY557: Causal Inference for Observational and Experimental Studies

Week 1: Causal Frameworks

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LSE

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Lecture Roadmap

1. Potential outcomes
2. Causal estimands
3. Identification
4. Graphical Causal Framework
5. Assignment mechanisms
6. Summary

1 Potential outcomes

2 Causal estimands

3 Identification

4 Graphical Causal Framework

5 Assignment mechanisms

6 Summary

A (Very) Simple Causal Model

A (Very) Simple Causal Model

D

Causal variable

A (Very) Simple Causal Model

D

Causal variable

Y

A (Very) Simple Causal Model

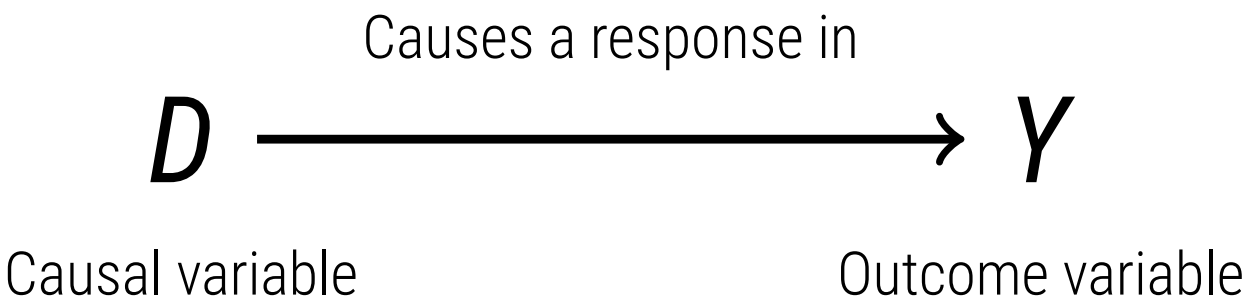
D

Causal variable

Y

Outcome variable

A (Very) Simple Causal Model



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One important implication is that causal variables must be manipulable:

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

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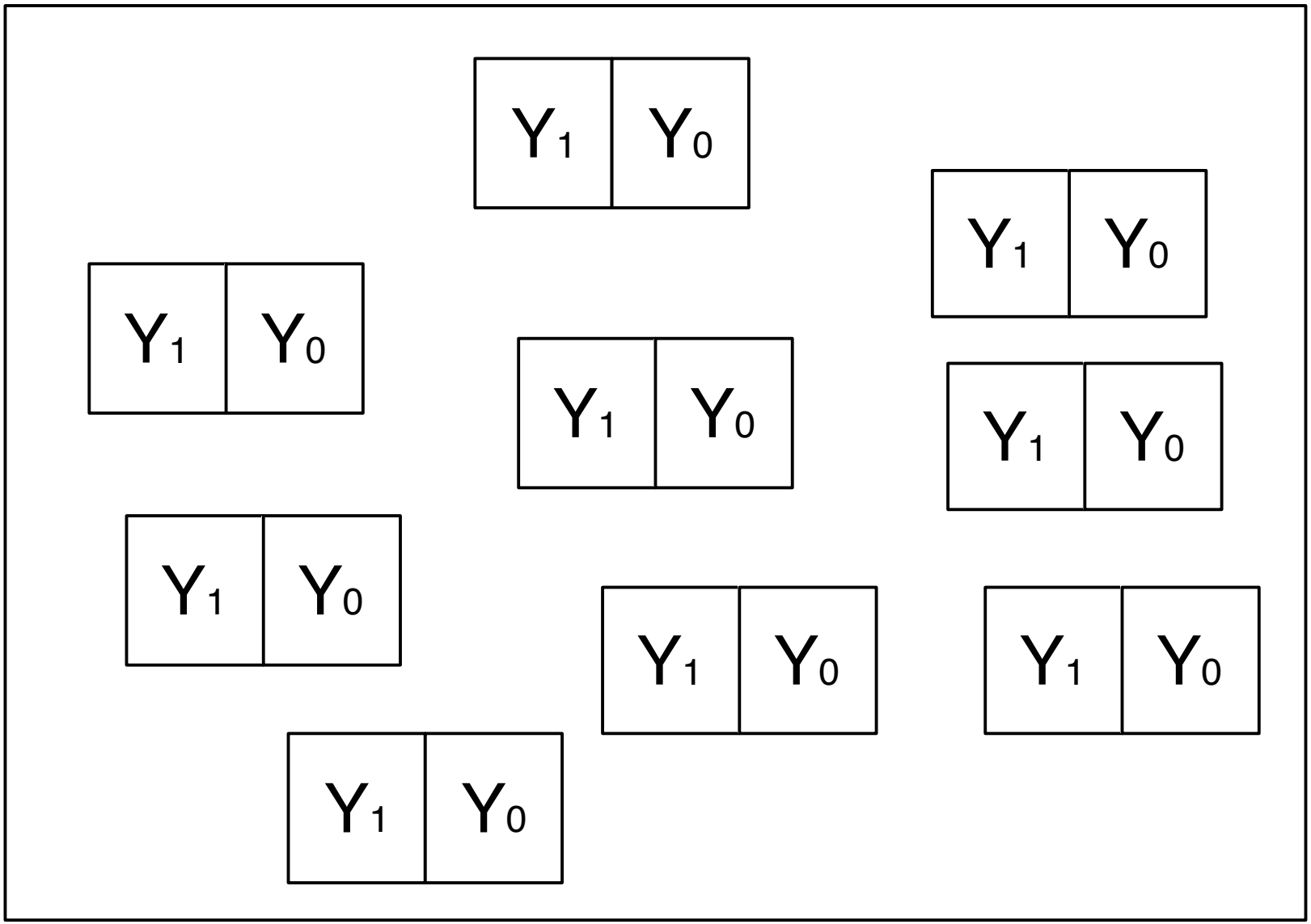
  - Holocaust $\rightarrow$ modern AFD election support

Neyman Urn Model



Concepts: Treatment, Outcomes, and Potential Outcomes

Definition (Treatment)

D_i: Indicator of treatment intake for *unit* i

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

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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:

$$Y_i = D_i \cdot Y_{1i} + (1 - D_i) \cdot Y_{0i}$$

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

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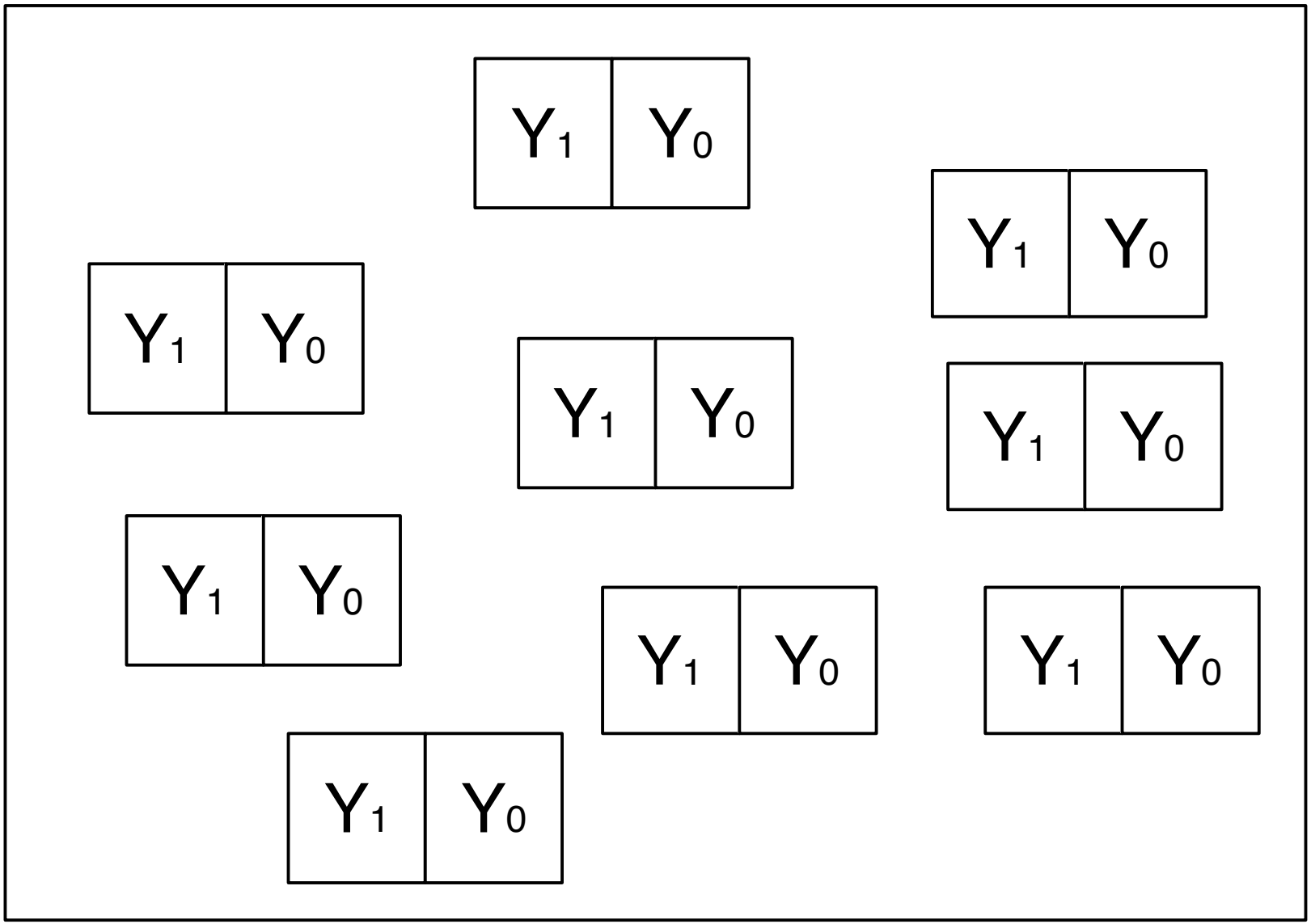

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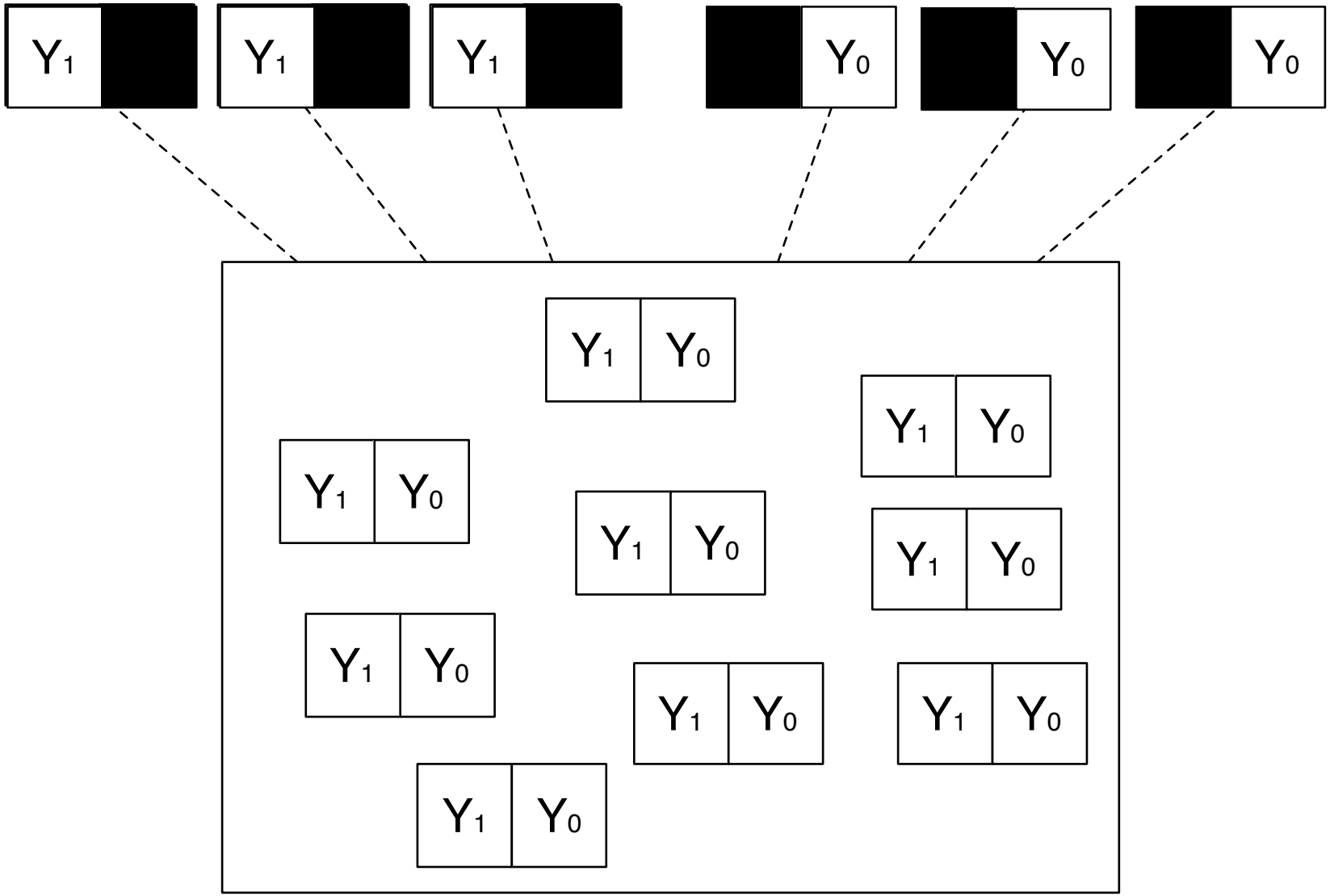

Stable Unit Treatment Value Assumption (SUTVA)

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Assumption (SUTVA)

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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

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

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

Causal Inference as a Missing Data Problem

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Imagine a population with 4 units:

i	D_i	Y_i	Y_{1i}	Y_{0i}
1	1	3	3	0
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- 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}
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Our goal:

- define causal estimands in terms of potential outcomes (previous table)

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- 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!

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Esti-what?

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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".)

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- 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.

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

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- 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
1	1	3
2	1	1
3	0	0
4	0	1

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

Illustration: Average Treatment Effect

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$\mathbb{E}[Y_i \mid D_i = 1]$		2
$\mathbb{E}[Y_i \mid D_i = 0]$		0.5
$\mathbb{E}[Y_i \mid D_i = 1] - \mathbb{E}[Y_i \mid D_i = 0]$		1.5

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

Naïve estimator:

$$\begin{aligned} \tilde{\tau} &= \mathbb{E}[Y_i \mid D_i = 1] - \mathbb{E}[Y_i \mid D_i = 0] \quad \text{(observed difference in means)} \\ &= \frac{3+1}{2} - \frac{0+1}{2} = 1.5 \quad \text{Could this be wrong?} \end{aligned}$$

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	?	?
2	1	1	1	?	?
3	0	0	?	0	?
4	0	1	?	1	?

What is $\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}]$? We need potential outcomes that we do not observe!

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2	1	1	1	1	?
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Suppose hypothetically: $Y_{01} = 0, Y_{02} = Y_{13} = Y_{14} = 1$.

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

Illustration: Average Treatment Effect on the Treated

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What is $\tau_{ATT} = \mathbb{E}[Y_{1i} - Y_{0i} \mid D_i = 1]$?

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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)

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  - Substantive theory / assumptions / wishful thinking about why a causal effect for these N units would also apply elsewhere

1 Potential outcomes

2 Causal estimands

3 Identification

4 Graphical Causal Framework

5 Assignment mechanisms

6 Summary

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- 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:

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- Correlation [association, here observed mean difference] is not necessarily causation.

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& 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)]
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- 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 ($E[Y_0|D = 1] - E[Y_0|D = 0] > 0$)

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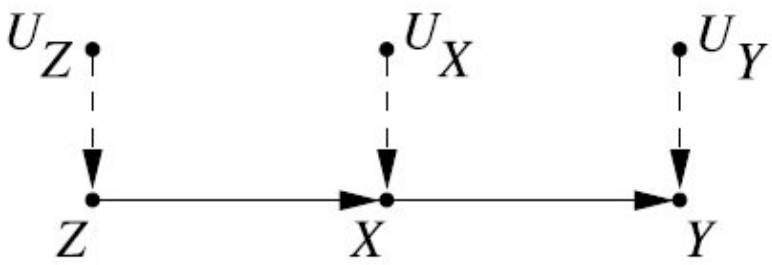
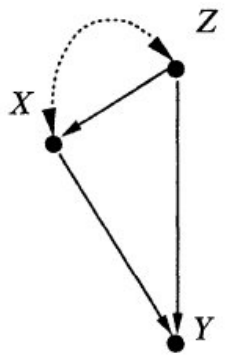
Causal Diagrams

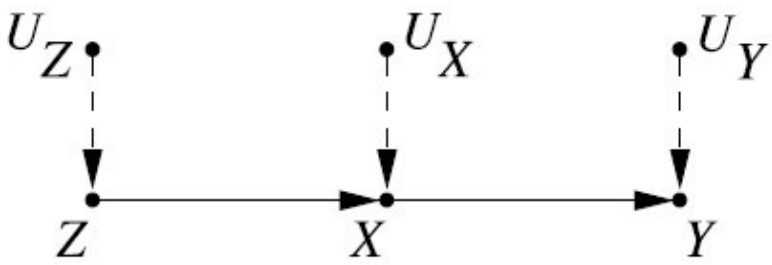


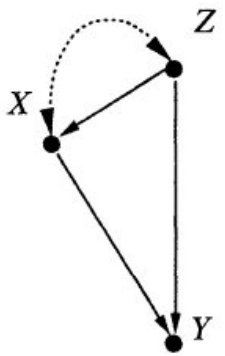


So far we have reasoned about causal effects using potential outcomes. An alternative (but intimately connected) framework is the graphical approach.

This uses causal diagrams, tools that allow us to:

Causal Diagrams





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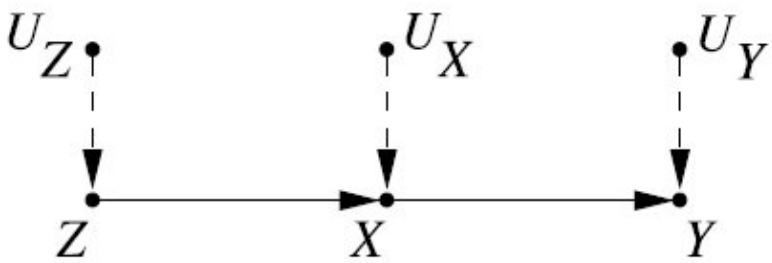

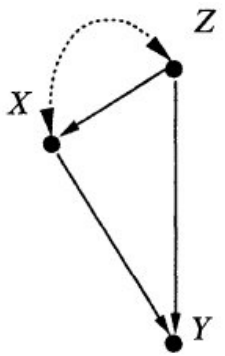

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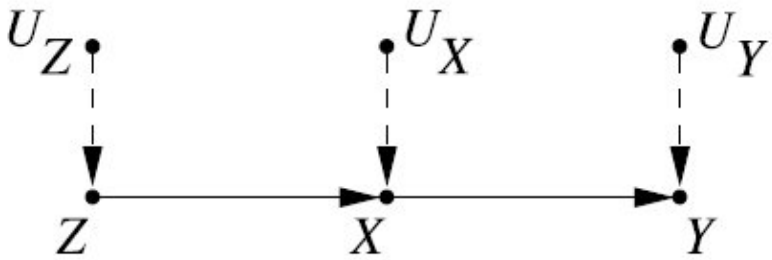

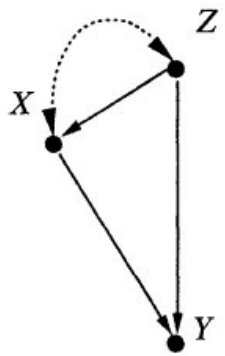

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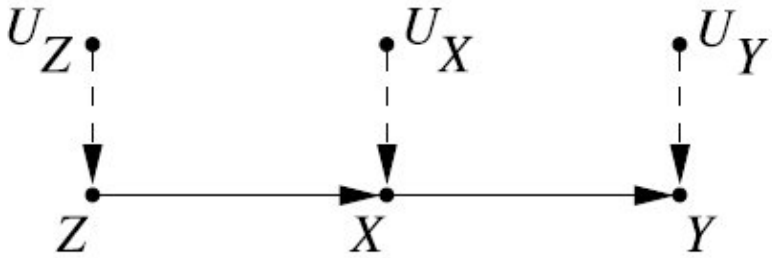

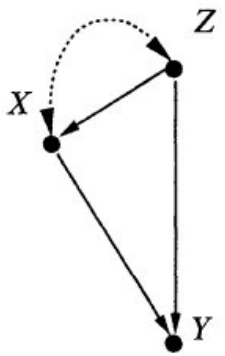

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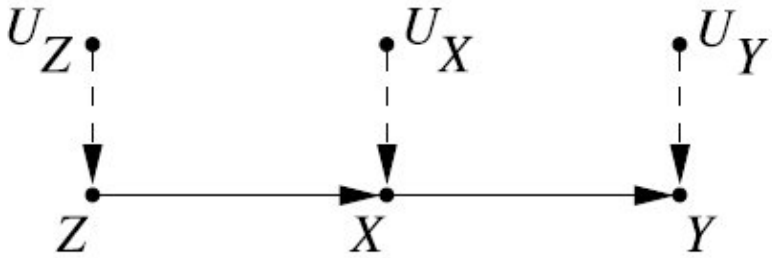

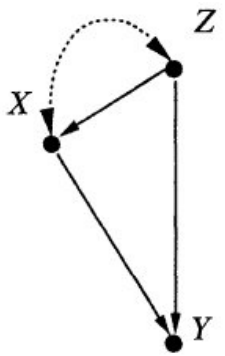

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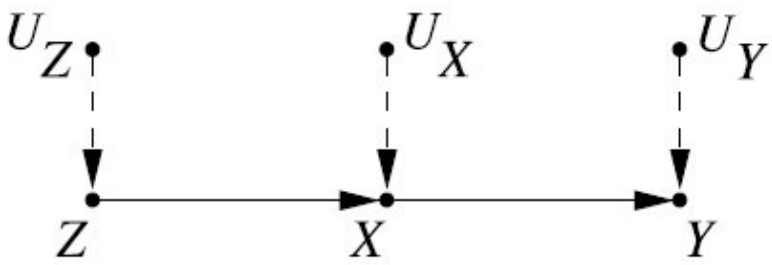

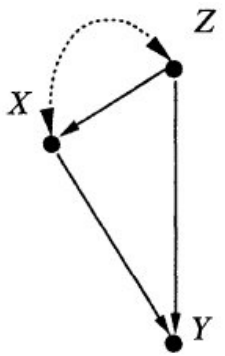

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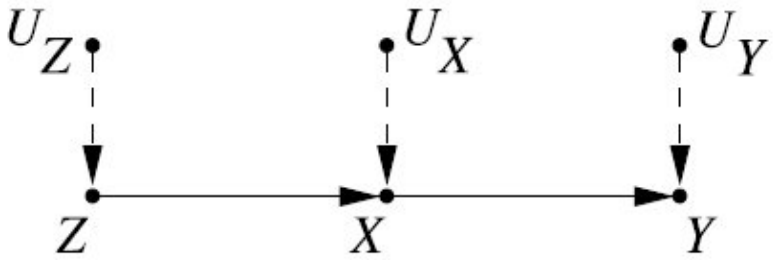
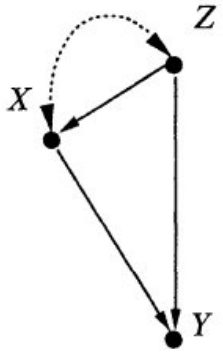
This can help us to:

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

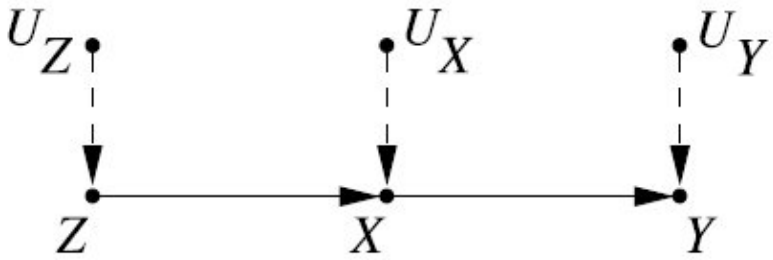
Causal Diagrams as Directed Acyclic Graphs

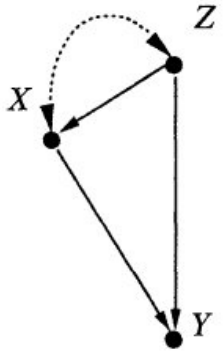




Components of a causal diagram as a Directed Acyclic Graphs (DAG):

Causal Diagrams as Directed Acyclic Graphs





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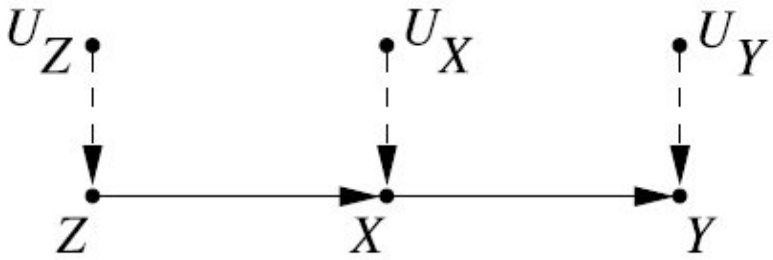

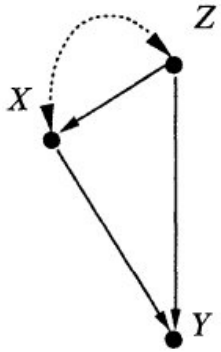

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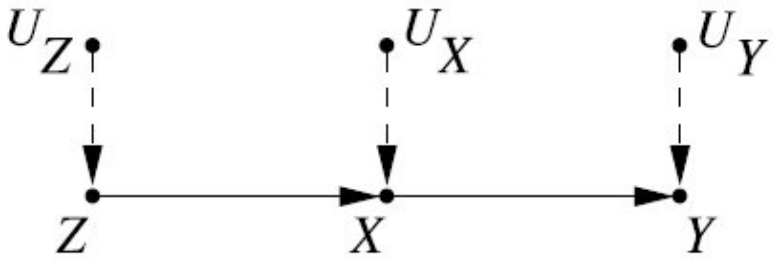

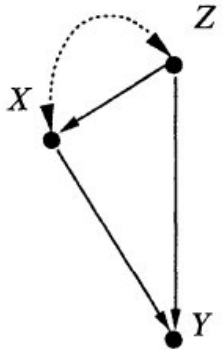

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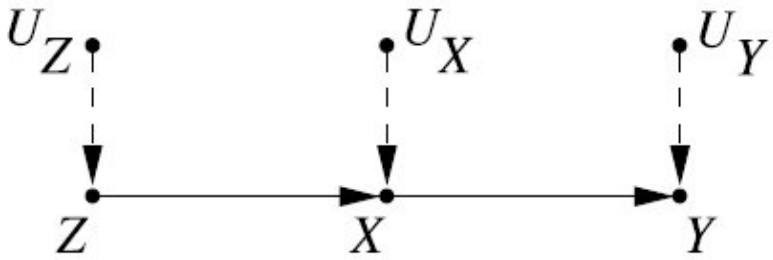

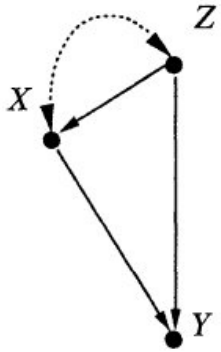

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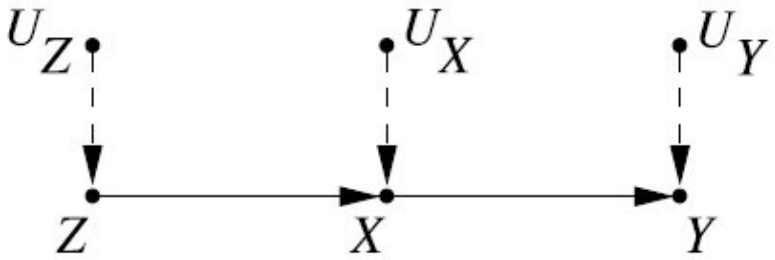

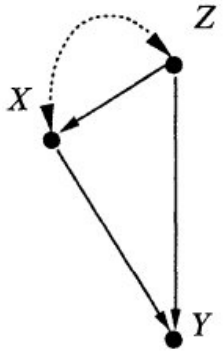

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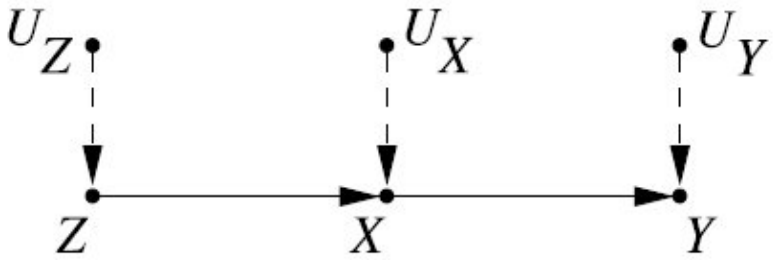

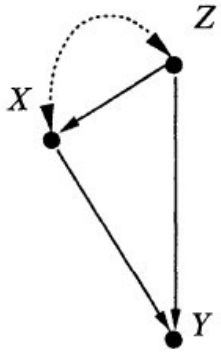

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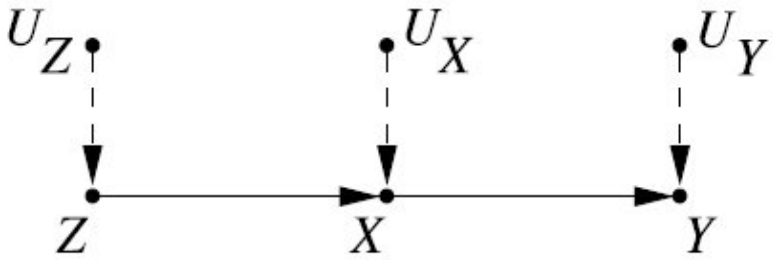

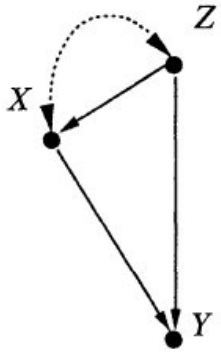

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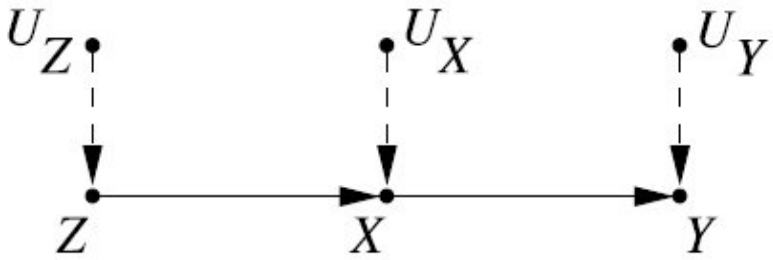

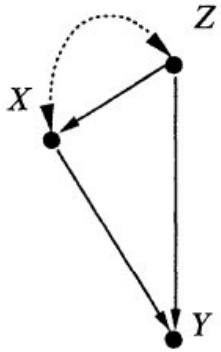

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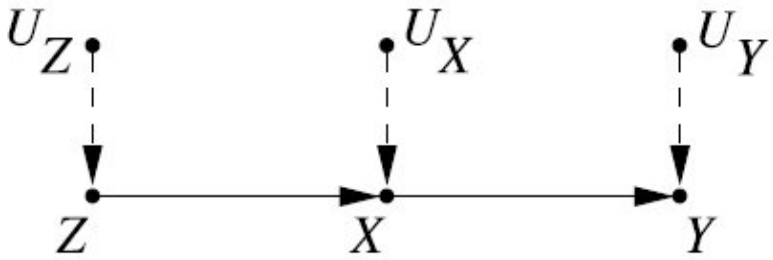

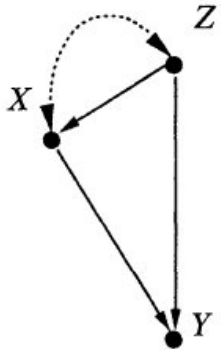

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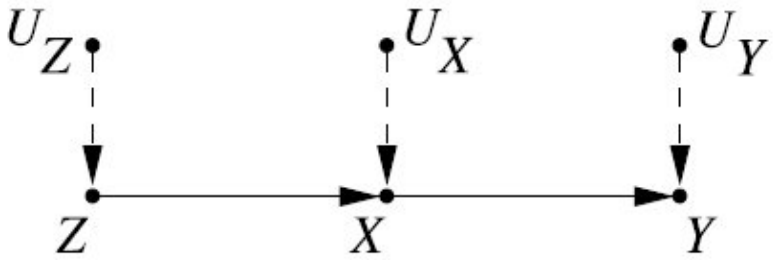

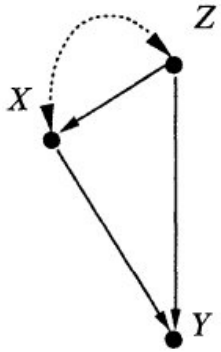

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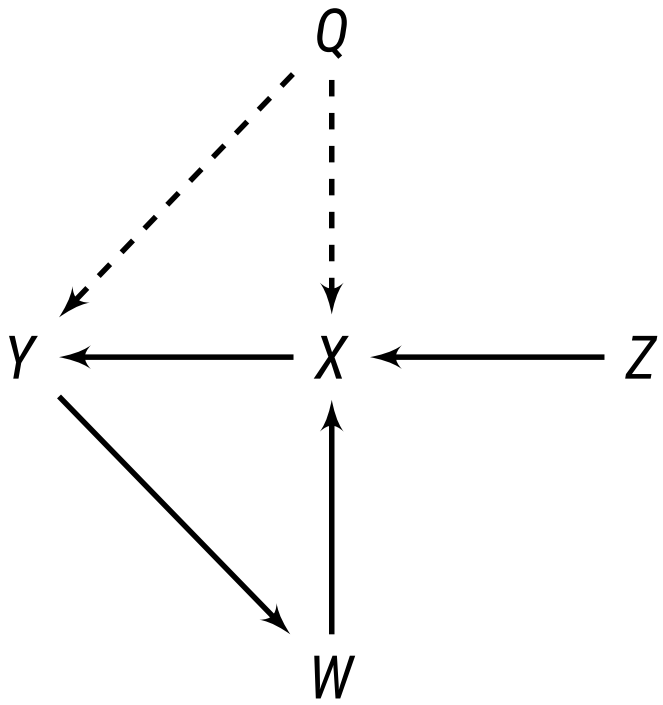
Directed Acyclic Graphs: Example



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

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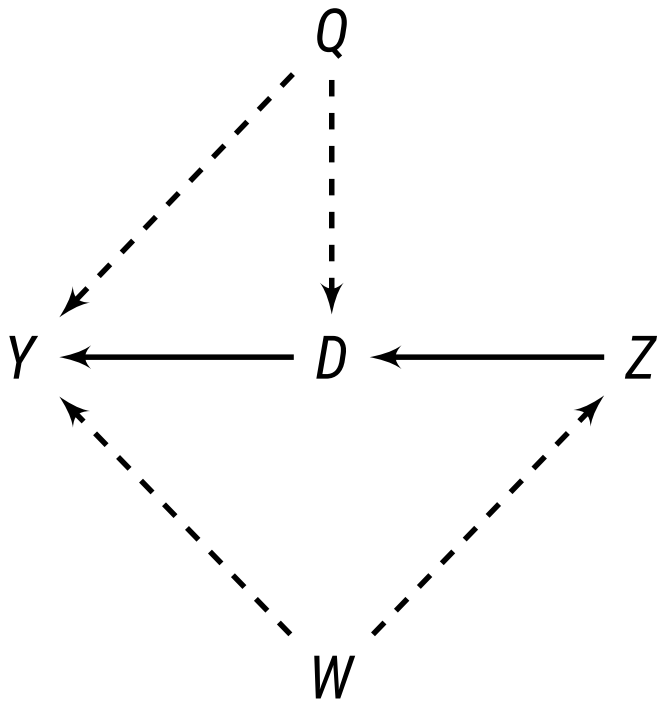

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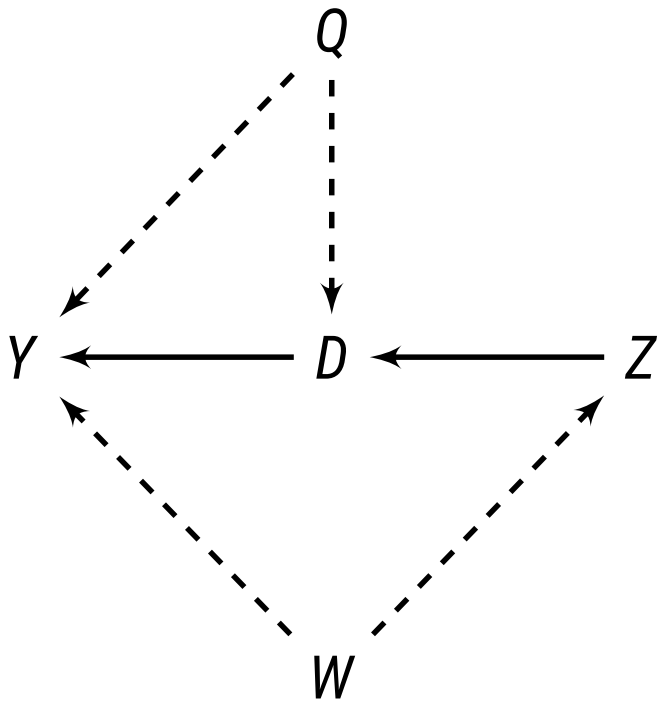

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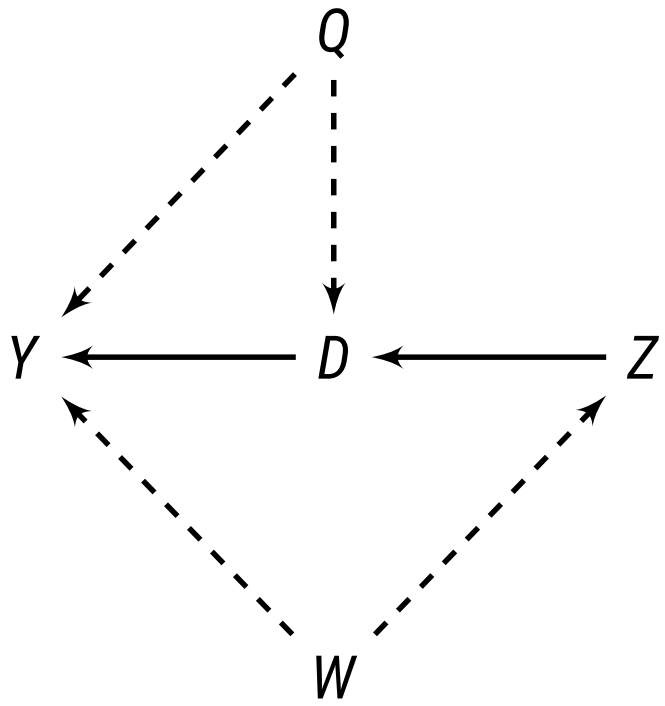

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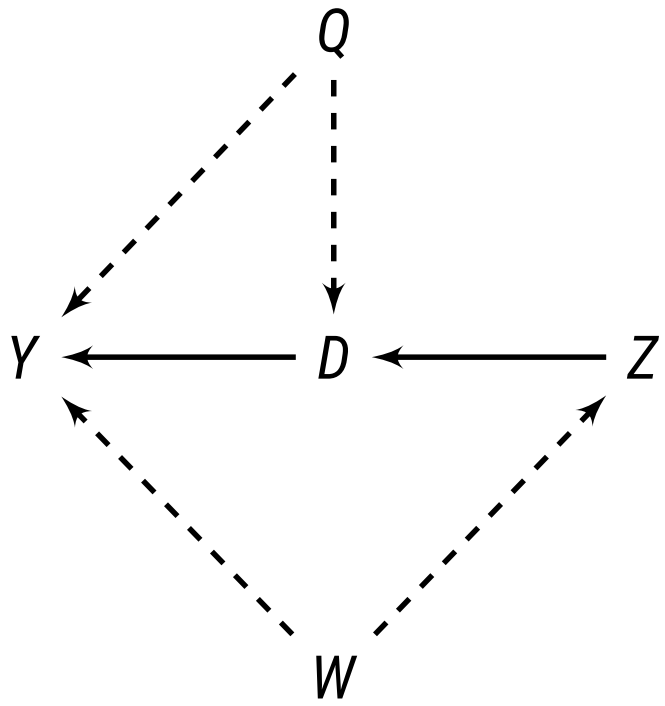

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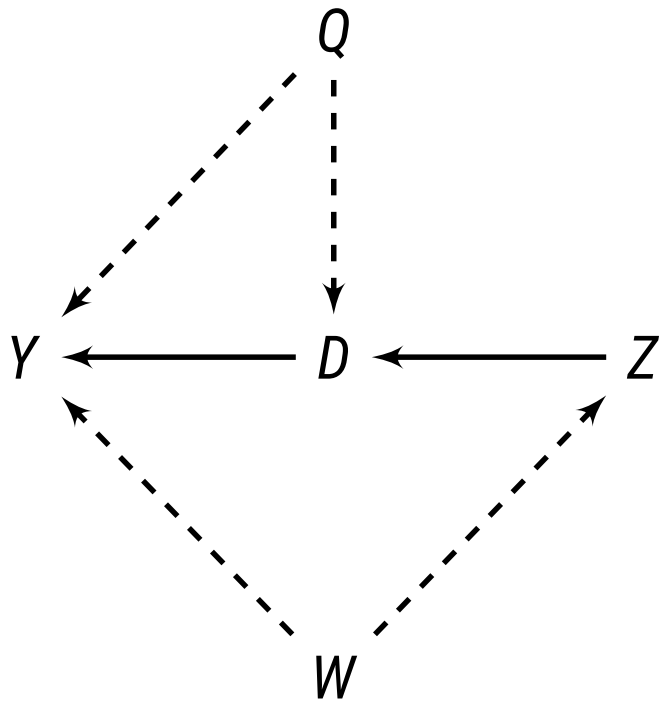

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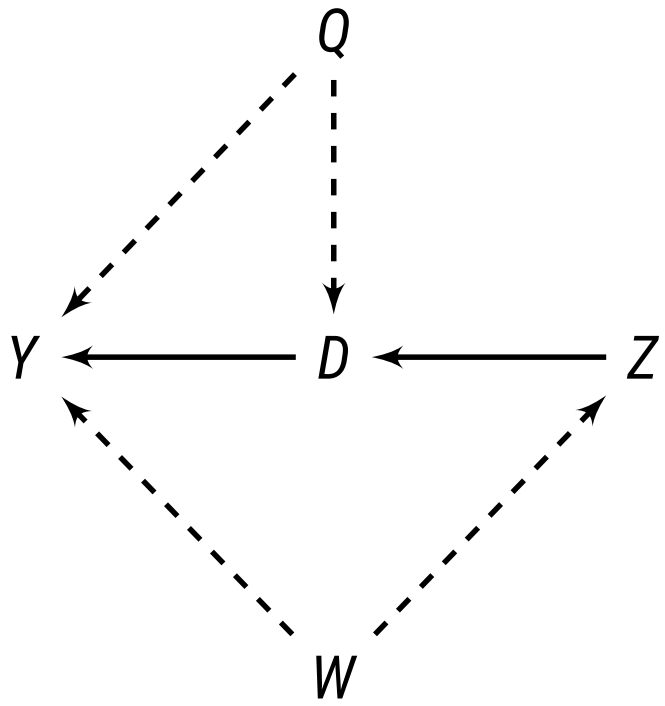

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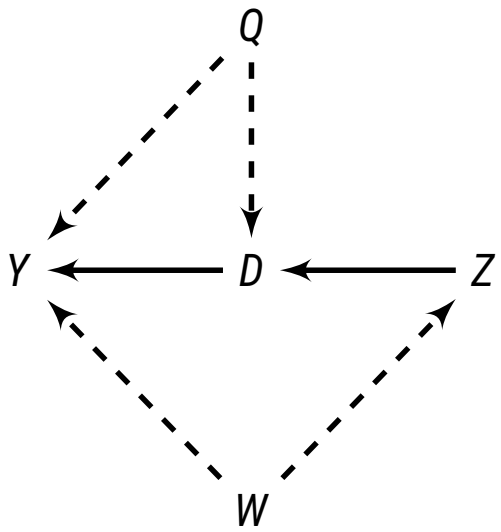
But only if our DAG is correct!

Representing Interventions



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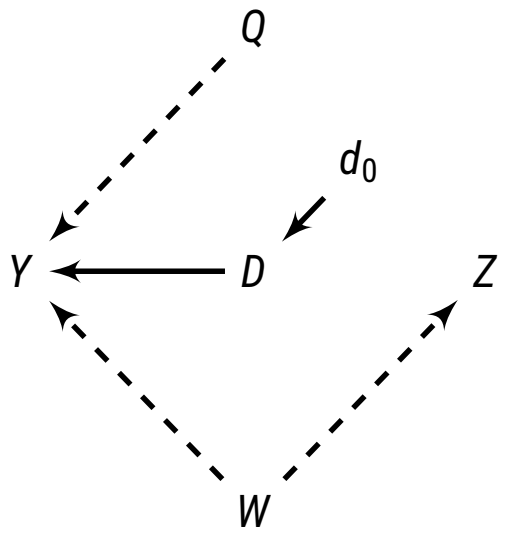
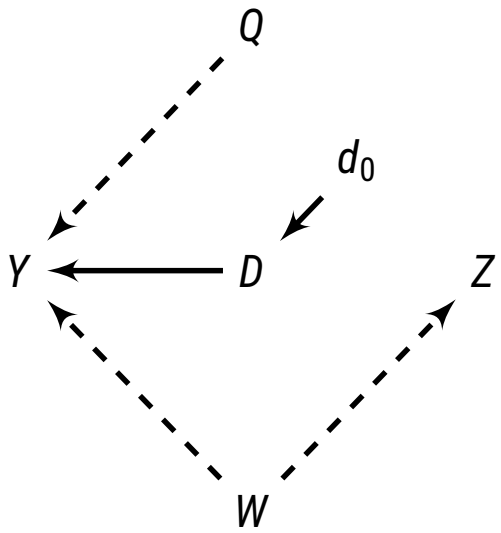


Treatments (interventions) are represented by the ***do***() operator.

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)?

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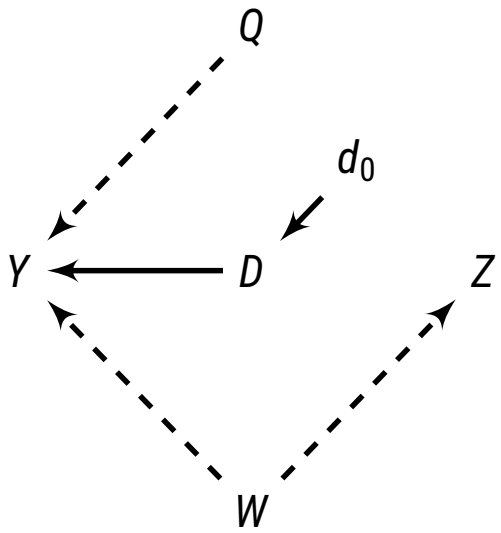

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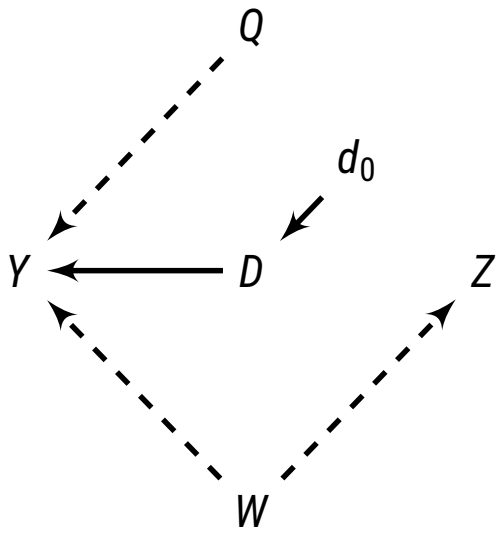

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Generally: We can identify the effect of D on Y if all back-door paths are blocked.

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3 Identification

4 Graphical Causal Framework

5 Assignment mechanisms

6 Summary

Assignment Mechanism

Definition (Assignment Mechanism)

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- 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)

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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
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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™

Least credible **Most credible**

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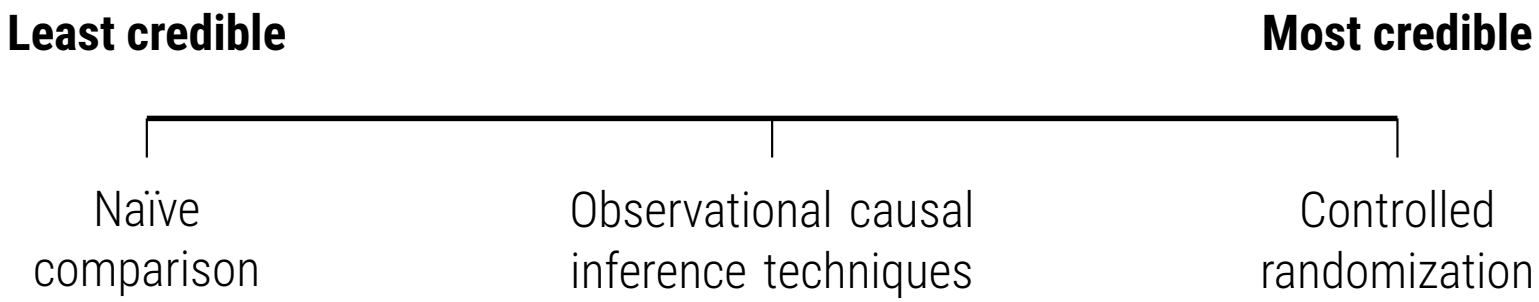

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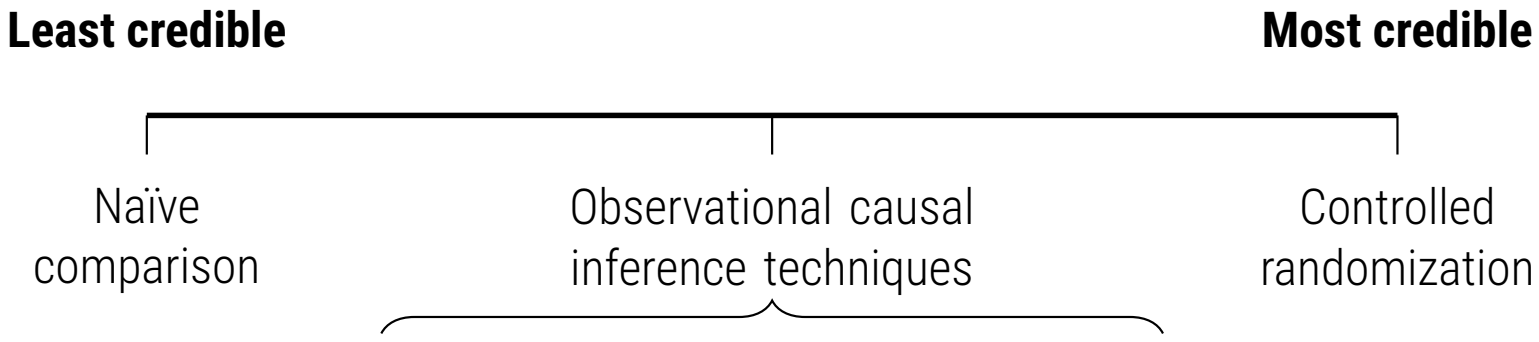

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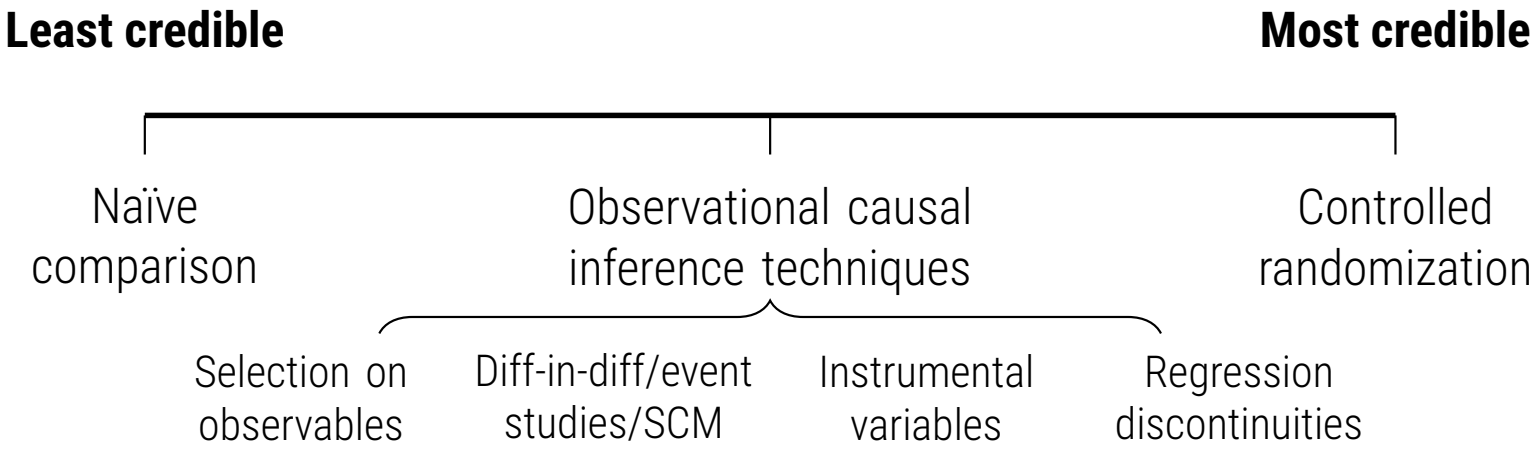

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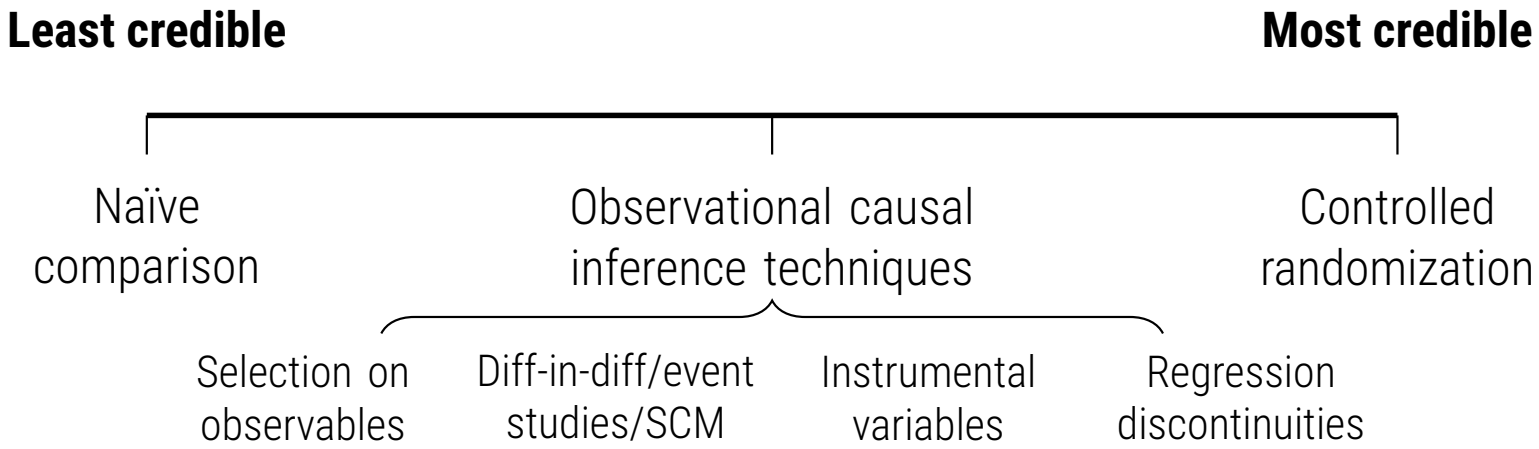

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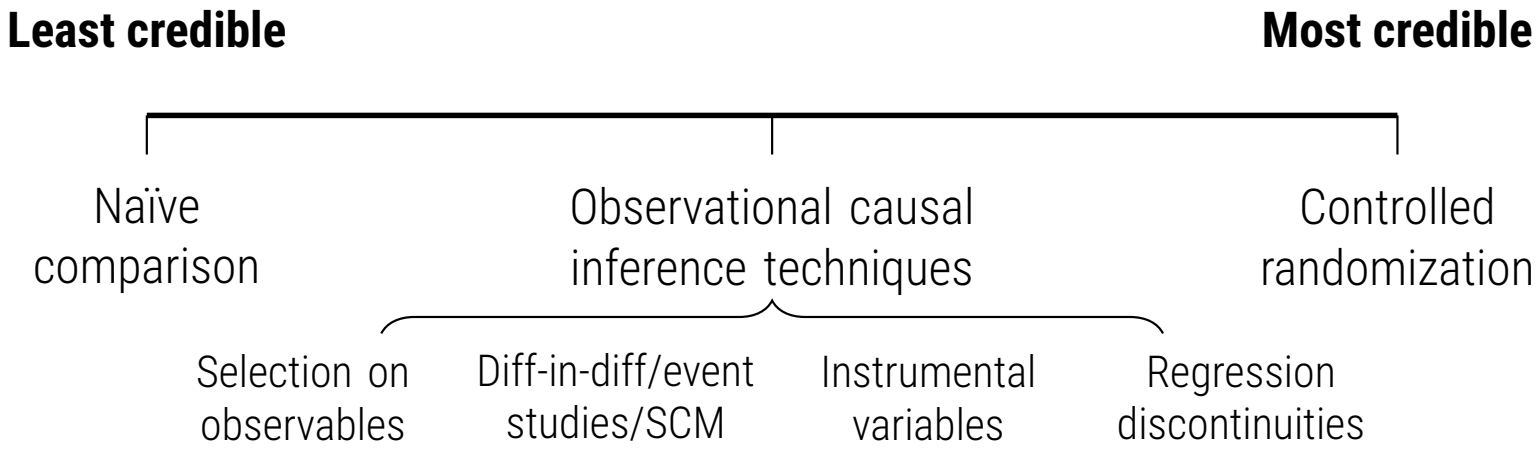

Key point: The art (and science) of applied causal inference is making defensible assumptions. There is no 'magic' solution to the fundamental problem of causal inference, only assumptions all the way down!

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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