Potential Outcomes and Directed Acylic Graphs

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Causality and counterfactuals

- Every economics research paper is *not* estimating a causal quantity
  - But, the implication or takeaway of papers is (almost) always a causal one

- Causality lies at the heart of every exercise

- Goal for today's class:
  1. Enumerate tools used to discuss causal questions
  2. Emphasize a *multimodal* approach
  3. Set terminology/definitions for future discussions

"We do not have knowledge of a thing until we have grasped its why, that is to say, its cause."

-Aristotle

Causality and counterfactuals - strong opinions

- The true underpinnings of causality are nearly philosphical in nature
  - If Aristotle didn't settle the question, neither will researchers in the 21t century

- I will avoid many of the discussions, but my biases will show up in one or two settings

- Key point: economics research is messy, and a careful discussion of causality entails two dimensions:
  1. A good framework to articulate your assumptions
  2. Readers that understand the framework

The problem of causal inference: a medical example

- Two variables:
  - $Y \in \{0, 1\}$: whether a person is immune to Covid-19
  - $D \in \{0, 1\}$: whether a person gets a vaccine

- Our question: does D cause Y?

- *Ignore the question of data for now* – this is purely a question of what is knowable.

- "The fundamental problem of causal inference" (Holland 1986) is that for a given individual, we can only observe one world – either they get the vaccine, or they do not

The problem of causal inference: a medical example

- What is knowable?
  - We need notation - begin with the Neyman-Rubin Causal model

- There is a population of n individuals, indexed by i.

- Let $Y_i(D_i)$ denote the outcome given a particular vaccine treatment
  - $Y_i(1)$: they receive the vaccine
  - $Y_i(0)$: they do not receive the vaccine

- **Key Assumption**?

The problem of causal inference: a medical example

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- Let $Y_i(D_i)$ denote the outcome given a particular vaccine treatment
  - $Y_i(1)$: they receive the vaccine
  - $Y_i(0)$: they do not receive the vaccine
- **Key Assumption**? person i's outcome is only affected by their own treatment. We will discuss relaxing this assumption later.
  - SUTVA - Stable Unit Treatment Variable Assignment

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0)$$

i	$Y_i(1)$	$Y_i(0)$	D_i	Y_i
1	1	0	1	1
2	0	0	1	0
3	1	0	0	0
		⋮		
n	0	1	0	1

Causal inference is a missing data problem

- In the potential outcomes framework, causal inference and missing data are tightly linked.
- Any causal answer uses assumptions to infer the “missing” counterfactual
- Goal of this course will be to discuss many ways to solve these types of problems
- Before diving into the many potential estimands, consider what the goal is.
  - A structural parameter? E.g. dInvestment/dTax Rate
  - Existence of an treatment effect?
  - A policy evaluation?

A brief aside: estimands, estimators and estimates

- Estimand: the quantity to be estimated
- Estimate: the approximation of the estimand using a finite data sample
- Estimator: the method or formula for arriving at the estimate for an estimand
- My way of remembering: `https://twitter.com/paulgp/status/1275135175966494721?s=20`

Causal estimands

- We will start with the Average Treatment Effect:
  - $\tau_{ATE} = \mathbb{E}(\tau_i) = \mathbb{E}(Y_i(1) - Y_i(0)) = \mathbb{E}(Y_i(1)) - \mathbb{E}(Y_i(0))$
- This expression is defined over the full population, and includes individuals who may never recieve the treatment.
  - Average Treatment Effect on the Treated $\tau_{ATT} = \mathbb{E}(\tau_i | D_i = 1) = \mathbb{E}(Y_i(1) - Y_i(0) | D_i = 1) = \mathbb{E}(Y_i(1) | D_i = 1) - \mathbb{E}(Y_i(0) | D_i = 1)$
  - Estimated effect for individuals who *received* the treatment.
  - Note that one piece of this measure is purely observed data: $\mathbb{E}(Y_i(1) | D_i = 1)$
- Conditional Average Treatment Effect: $\tau_{CATE}(x) = \mathbb{E}(\tau_i | X_i = x) = \mathbb{E}(Y_i(1) - Y_i(0) | X_i = x)$ where X_i is some additional characteristic.

A second brief aside: what is identification?

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- Intuitively, for an estimate of interest, τ_{ATE}, to be identified, it means that in a world with no uncertainty about data, can we always identify the value of τ from the data we observe?
  - In other words, it's an invertability condition

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- Why would something not be identified if we only observe (Y_i, D_i)?
  - Consider τ_{ATT}. $\mathbb{E}(Y_i(1)|D_i = 1)$ is identified, mechanically. What about $\mathbb{E}(Y_i(0)|D_i = 1)$?
  - Need an assumption on the relationship between D_i and $(Y_i(1), Y_i(0))$.

Under what conditions is the ATE identified?

Strong Ignorability: D_i is *strongly ignorable* conditional on a vector $\mathbf{X}_i$ if

1. $(Y_i(0), Y_i(1)) \perp\!\!\!\perp D_i | \mathbf{X}_i$
2. $\exists \epsilon > 0$ s.t. $\epsilon < \Pr(D_i = 1 | X_i) < 1 - \epsilon_i$

- The first condition asserts independence of the treatment from the "potential" outcomes
- The second condition asserts that there are both treated and untreated individuals
- N.B. The term "strong ignorability" is much more precise than exogeneous
  - But less commonly used in economics.
  - You might instead say "D_i is conditionally randomly assigned."
  - If you *might* even say D_i is exogeneous.

When could we not identify the ATE?

- Intuitively, we understand why we typically can't estimate a treatment effect
- Consider an unobservable variable, $U_i \in \{0, 1\}$ where $(Y_i(0), Y_i(1), D_i) \not\perp\!\!\!\perp U_i$
- Simple example: when $E(D_i|U_i = 1) > E(D_i|U_i = 0)$ and $E(\tau_i|U_i = 1) > E(\tau_i|U_i = 0)$.
- In other word, there is a variable that influences both the potential outcomes and the choice of treatment.
  - In this case, estimating the counterfactual is contaminated by the variable U_i
- Many of the goals in this class will be to address this

Theorem: Identification of the ATE

Theorem: If D_i is strongly ignorable conditional on $\mathbf{X}_i$, then

$$\mathbb{E}(\tau_i) = \sum_{x \in \text{Supp } X_i} (\mathbb{E}(Y_i|D_i = 1, \mathbf{X}_i = x) - \mathbb{E}(Y_i|D_i = 0, \mathbf{X}_i = x))Pr(\mathbf{X}_i = x)$$

Proof: Note that $\mathbb{E}(Y_i(0)|\mathbf{X}_i) = \mathbb{E}(Y_i(0)|D_i = 0, \mathbf{X}_i) = \mathbb{E}(Y_i|D_i = 0, \mathbf{X}_i)$ by strong ignorability. In essence, independence of D_i and $(Y_i(0), Y_i(1))$ lets us interchange counterfactuals and realized data in conditionals. The rest follows by the law of iterated expectations. □

- Key implication – counterfactual can be generated by using the averages.

Identification of the ATE - Intuition

i	$Y_i(1)$	$Y_i(0)$	D_i	Y_i
1	1	-	1	1
2	0	-	1	0
3	1	-	1	1
4	1	-	1	1
5	-	0	0	0
6	-	0	0	0
7	-	0	0	0
8	-	1	0	1

- We can estimate $\mathbb{E}(Y_i|D_i = 1) = 0.75$ and $\mathbb{E}(Y_i|D_i = 0) = 0.25$.
- We are defining our counterfactual in the missing data as 0.25, or 0.75, respectively.
- If we had covariates, we would condition within those groups.
- Note that this is all *non-parametric* identification – we have made no model restriction on the data-generating process

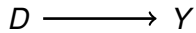
Identification through Directed Acylic Graphs (DAGs)

- Above, we encoded random variables' relationships functionally, using potential outcomes

- An alternative approach does this graphically (with similar modeling under the hood – to be continued...)

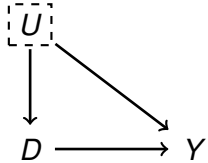
- We can encode the relationship between D and Y using an *arrow* in a graph. The direction emphasizes that D causes Y, and not vice versa.

- Substantially more *intuitive*



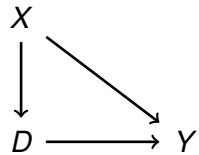
Identification through Directed Acylic Graphs (DAGs)

- We can also allow for the unobservable U, which drove identification concerns above
- In this case, U is termed a *confounder*. Why?
- Examine the paths by which D links to Y:
  - The standard direct effect $D \rightarrow Y$
  - The "Back-Door" path $D \leftarrow U \rightarrow Y$
- Note that the back-door is *not* causal
- Key point: effect of D on Y is not identified under this setup



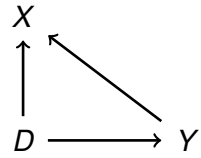
Identification through Directed Acylic Graphs (DAGs)

- We replace U with an observable X identification concerns above
- X is still a confounder, but we could condition on it and identify our effect. Why?
- Examine the paths by which D links to Y:
  - The standard direct effect $D \rightarrow Y$
  - The "Back-Door" path $D \leftarrow X \rightarrow Y$
- Now, conditioning on a variable along the path "blocks" the path
  - E.g. D is independent of Y *conditional* on X (strong ignorability)



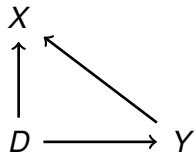
Identification through Directed Acylic Graphs (DAGs)

- One more example before formalizing the goal
  - X is now a "collider" (note direction of arrows
- Examine the paths by which D links to Y:
  - The standard direct effect $D \rightarrow Y$
  - The "Back-Door" path $D \rightarrow X \leftarrow Y$
- Key difference: a backdoor collider is automatically blocked (if it or upstream variables are not conditioned on)
  - If you condition on X, you open the path!
  - Example: conditioning on an outcome variable



Identification through Directed Acylic Graphs (DAGs)

- The graphs looked similar, but the order of true causal path mattered



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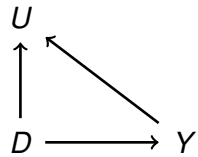
- Identifying colliders is a crucial aspect of identifying whether an effect is identified



Identification through Directed Acylic Graphs (DAGs)

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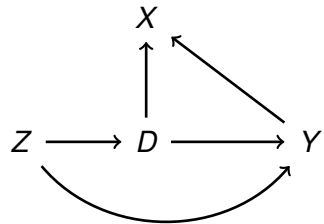
- Identifying colliders is a crucial aspect of identifying whether an effect is identified
- Key value in a DAG (to me) is laying out a model of causality, and clarifying what effects need to be restricted, even in a complicated setting
  - For example, how is the effect of D on Y identified here?



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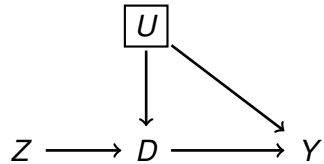
  - For example, how is the effect of D on Y identified here?
- What about now?



Key steps with a DAG

- Steps when using a DAG
  1. Write down the DAG, and identify what effect you want
  2. Write all paths between the two nodes
  3. What is the direct “causal” paths (e.g. the arrows all flow in the right direction)?
  4. How many backdoor paths are there? Are they blocked? Can they be?

- Crucial point: conditioning on colliders will cause more harm than good

- We will revisit this setup for some empirical settings
  - Let me know if you think there are good use cases!

Strutural equations and causal effects (Haile 2020)

- **Important**: do not lose sight of the fact that these should be estimates that inform our economic model (fully formalized or not)

- (Haile 2020) The reduced form equation is one where the inputs are i) *exogeneous* (ed note: we have not defined this) and ii) unobservable ("structural errors") and the outputs are endogeneous variables. [E.g. $Y_i = f(D_i, X_i, \epsilon_i)$]

- The PO framework's key insight was considering the sets of potential outcomes for the same individuals as a form of missing data. Note that these are effectively equivalent:

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0)$$
$$Y_i = \alpha + D_i(\tau + v_i) + u_i$$