Week 3: Causal Inference

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Outline

- The big picture: statistics and causal inference
- Definition of causal effects with a single unit
- Learning about causal effects from multiple units
- Assignment mechanism
- Estimands
- Causal inference with models
Big picture

- Learning statistics is not the same as learning about **causal inference**, although causal inference is now a field in statistics.
- Statistics was developed within the framework of an experiment, in which causality is not a problem (we will see why briefly).
- As we saw last class, (frequentist) statistical theory is also based on the concept of **repeating an experiment many times**, which, of course, we can’t do in practice but we mimic this process using simulations.
- You have probably heard many, many times that **correlation is not causation**.
- But what we really want to learn is **under which circumstances correlation actually IS causation**.
Big picture II

- In econometrics, the notion of causality is often introduced in the context of the linear model.
- The purpose of today’s class is to separate the notion of causality from a particular method of estimating a relationship.
- Another way of putting it: today class is about research design versus analysis.
- See Rubin’s article *For Objective Causal Inference, Design Trumps Analysis*.
- **Research design**: You have a research question, then you think about the data you need to answer it, and the problems you could have establishing cause and effect. Research design is your strategy to answer the research question. It could be experimental or more likely in HSR, observational.
Basics concepts

- Causality is linked to a manipulation (treatment, intervention, action, strategy) that is applied to a unit.
- A unit could be a person, firm, an institution, country, a classroom, school and so on. Another way of saying it: an observation.
- Think of it as the “thing” that received the action or was manipulated.
- The unit could have been exposed to an alternative action.
- For simplicity, only two possibilities: receiving or not receiving the action or treatment (active versus control treatment in Imbens and Rubin, 2015).
- A unit (either receiving or not receiving a treatment) is linked to a potential outcome.
Potential outcomes

- The potential part refers to the idea that **only one outcome is realized** after the intervention; the other is, well, potential

  (Dictionary definition: Potential: having or showing the capacity to become or develop into something in the future)

- **Before** the intervention, there are **two potential outcomes**

- **Only one is realized** after the action is conducted

  Example: a person may or may not receive a job training program if unemployed

- One potential outcome is income one year after receiving training; the other is income one year later if not receiving treatment

- **Jargon alert**: economists like to use a priori, a posteriori, ex ante, ex post
Definition of causal effect

- The causal effect of receiving treatment for person $i$ is the **comparison of potential outcomes**
- We could denote outcome (income) as $Y$
- The causal effect of treatment for a person could be defined as:

$$Y(\text{Training}) - Y(\text{NoTraining})$$

- Or it could be

$$\frac{Y(\text{Training})}{Y(\text{NoTraining})}$$

- Note that the definition is independent from the **measurement** of the outcome
- The important parts are that a treatment effect is a) the **comparison of potential outcomes** and 2) it does not depend on which **action is actually taken**
The fundamental problem of causal inference

- The challenge in causal inference is that we do not observe both potential outcomes; we only observe one.
- Holland (1986) called this the “fundamental problem of causal inference.”
- In the economics literature, a similar version is called the fundamental problem of program evaluation.
- **Alert:** In this framework, the same unit receiving a treatment at a different time is a different unit.
- The non-observable or not-realized outcome is called the **counterfactual** (Dictionary: relating to or expressing what has not happened or is not the case).
- Also called the **Rubin causal model**
Finding causal effects

- We now have a definition of causal effects and a language.
- But **we also have a problem**: we can’t find causal effects because we only observe one potential outcome for each unit.
- What we need is a way of predicting what would happen to unit $i$ with or without treatment.
- Another way: *we want to predict the counterfactual*. Yet another way: we need a way to come up with the “what if” scenario.
- The solution to this problem involves using **multiple units**.
- Some will be exposed to the intervention and some will not; one group serves as the counterfactual for the other.
Why is this important?

- The importance of this framework is that it provides a **structure** to think more clearly about causality and it also provides a **language**
- That language, and its notation, has led to a deeper understanding of causality
- Example: The Colorado Family Planning Initiative: it provided free long-active contraceptives (LARCs) at Title X clinics
- Research question: What is the effect of providing free LARCs in terms of unwanted pregnancies?
  1. What is the action or intervention?
  2. What is the alternative action?
  3. What are the potential outcomes for a woman?
- See Imbens and Rubin (2015) for more examples
Multiple units

- We use multiple units to figure out causality in everyday reasoning
  1. Each of us at different times is a different unit. That’s how we figure out what works for us
  2. Different people getting different treatments are a source of comparisons

- We often compare different people doing different things (i.e. getting different “treatments”): crossfit or yoga or paleo? What on earth are Tom Brady and Halle Berry doing that they don’t seem to age at all?

- **BUT**... there is still something missing

- Intuitively, to correctly predict the counterfactual, we want to compare **similar units**
Ways to solve the fundamental problem of causal inference (a)

- **a) Randomization**: Randomly dividing units into treatment and control groups before the action or intervention takes place makes groups of units comparable
- (Don’t underestimate how deep (and mysterious) randomization truly is)
- After an action is applied to one group, the other group provides the counterfactual
- Another way of saying this is that the potential outcomes do not depend on the particular group assignment (we need another assumption; more on this in a bit)
- In the new epi causal inference literature they call this exchangeability: the groups are so similar that they could be exchanged; it does not matter which group receives the intervention
Ways to solve the fundamental problem of causal inference (b)

- **b) Statistical adjustment**: If randomization is not possible, then some characteristics of the units could be used to make comparisons.
  - In the training example, if it was more likely that younger people used the training opportunity, which was not randomly assigned, we could then compare the outcome by age.
  - Say, 18 to 25. This would make predicting the counterfactual more plausible.
  - In the homework example, we could compare drug treatment for each severity group. In other words, a **third factor induces conditional independence**.

- But in practice, there is likely more than one factor.

- Here is where **regression adjustment**, the topic of this semester, becomes a fundamental tool in causal inference. We include covariates in a model because we want to “control” for them (or hold them constant).
Estimands

- From trusty Wikipedia: **estimand** is the true value which we want to estimate in a statistical analysis (like $\mu$ and $\sigma$). So the population parameter.
- Since we use multiple units, treatment effects are often **average treatment** effects (ATE).
- But not the only type of effect. For example, average treatment effects on the treated (ATET).
- ATET is an example of LATE: Local Average Treatment Effect.
- Many of the statistical techniques to find causal effects are LATE: instrumental variables, regression discontinuity, propensity scores...
- An important consequence: **we CAN’T estimate unit-level effects**.
- We could make a prediction about the counterfactual for an individual $i$, but this prediction is based on the information from a group of units.
- Remember, **we cannot observe both (potential) outcomes**.
Assignment mechanism

- A key consideration when deciding if units are comparable is to determine the **assignment mechanism**
- Assignment mechanism: What determines which units receive (or not) the treatment?
- Imbens and Rubin (2015) define several classes of assignment mechanisms:
  1. **Classical randomized experiments**: Interventions under the control of the investigators. With good randomization, causality is not problematic (includes *conditional randomization*). There could be non-compliance, too
  2. **Regular assignment mechanisms**: Interventions not under the control or not known to the researcher – think observational studies. Several subtypes based on how much we know about assignment
- Most of health services research question are related to the second type
Stable Unit Treatment Value Assumption (SUTVA)

1. The **potential outcomes** for any unit do not vary with the treatment assigned to other units (no interference)
2. For each unit, there are no different versions of each treatment level (no hidden variation of treatments)

- These are examples of **exclusion restrictions**: assumptions that rely on theory or substantive knowledge to rule out (or in) the existence of a causal effect
- Assumptions about how the world works that allow us to “exclude” alternatives or define mechanisms
- In instrumental variables, for example, we need to rule out the possibility that the instrument is related to the outcome (conditional on other factors)
A fundamental assumption: The ignorable treatment assignment assumption

- This assumption says that conditional on observable covariates \( X_n \), the assignment of units to experimental groups is independent of potential outcomes:
  \[
  (Y_0, Y_1) \perp T|X_n, \text{ where } Y_0, Y_1 \text{ are the potential outcomes, } T \text{ is treatment assignment and } X_n \text{ are a set of } n \text{ observable covariates}
  \]
- This is exactly like the homework example. It means that once we control for \( S \), severity, treatment assignment is independent of potential outcomes (conditional independence)
- This assumption comes in many names, the most common perhaps is ‘no unmeasured confounders
- Other names: selection on observables, exogeneity, conditional independence, ignorability
- (Even more jargon: \( \perp \) is “perpendicular”, “orthogonal”)
Reminder: confounder

- From Wikipedia (with some edits):
  “In statistics, a confounding variable (also confounding factor, a confound, a lurking variable or a confounder) is a variable in a statistical model that correlates (directly or inversely) with both the dependent variable and an independent variable”

- Another way: a confounder predicts both a covariate and outcome

- More often than not we talk about a confounder in relationship to a treatment

- Antidepressants and the risk of suicide: severity of depression is a confounder. It’s correlated to both the probability of taking antidepressants (the treatment) and the probability of suicide (the outcome)

- The ignorability of treatment assignment says that if you can’t control for confounders, your statistical model is showing a correlation and not a causation
Assignment mechanism II

- If the units were not randomized, how some units ended up receiving treatment? What factors influenced that decision? **This is perhaps the most important consideration of a study design**

- Example: taking antidepressants and the risk of suicide. You have observational data; patients were not randomized to take antidepressants

- Why some took antidepressants? Severity of illness, access to care, family history...

- If you can’t control for these factors, you do not have conditional independence between antidepressant use (treatment) and suicide (outcome)

- In other words, your study is showing a correlation, not a causation

- Another example: Is telemedicine effective?
How economists talk (or not?) to each other

- The model is **endogenous**: They mean that the ignorability assumption is not satisfied. There are unobservable variables not controlled for.
- The model is not **identified**: It means that whatever model you are estimating does not represent the true model. You cannot learn the “true” causal value of the parameters from your model.
- **Identification strategy**: the method used for finding causal effects, as in “my identification strategy is to use XYZ as an instrument for…”
- **Selection on observables**: The economist version of no unmeasured confounders. Meaning, people (it’s usually people) selected into treatment based on factors that you can measure (and control for)
Big picture: So where are the models?

- In most stats regression books, causal inference is often not discussed.
- In econometrics, causal inference is discussed in the context of linear regression:
  \[ Y_i = \beta_0 + \beta_1 X_{1i} + \cdots + \beta_k X_{ki} + \epsilon_i \]
- We will see that causal inference problems can be expressed in terms of linear regression assumptions (for example, whether \(\epsilon_i\) is correlated with some of the \(X\) variables in the population). In the POPULATION!!
- But this obscures issues of causal inference more than illuminates them.
- Much better to disentangle thinking about causal effects from the estimation of a particular model; or study design versus estimation.
Summary

- The counterfactual framework offers a way of thinking about causal inference.
- It has resulted in a lot of progress in the field and has unified the causal inference approach in epi, stats, and economics.
- Get used to the language.
- Remember, we are trying to learn if our models are descriptive/correlational or if they can have a causal interpretation.
- Easy with experiments; more difficult with observational data.