

# Week 3: Causal Inference

Marcelo Coca Perrignon

University of Colorado  
Anschutz Medical Campus

Health Services Research Methods I  
HSMP 7607  
2019

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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:

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

- Or it could be

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

- 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-acting 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:
- $(Y_0, Y_1) \perp T | X_n$ , where  $Y_0, Y_1$  are the potential outcomes,  $T$  is treatment assignment and  $X_n$  are a set of  $n$  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:

$$Y_i = \beta_0 + \beta_1 X_{1i} + \dots + \beta_k X_{ki} + \epsilon_i$$

- 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