Lecture I: Introduction to Impact Evaluation

Stanislao Maldonado

Universidad del Rosario
stanislao.maldonado@urosario.edu.co

Impact Evaluation
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1. Introduction to Program Evaluation

Definition (DiNardo and Lee 2011)

"Any systematic attempt to collect and analyze information about the implementation and outcomes of a program...A program is a set of interventions, actions and treatments, which are assigned to participants and are suspected of having some consequences on the outcomes experienced by participants."

- Examples of programs:

Why evaluate? (Gertler et al 2016)

- Need evidence on what works
  - Limited budget and bad policies could hurt
- Improve program/policy implementation
  - Design (eligibility, benefits)
  - Operations (efficiency, targeting)
- Information key to sustainability
  - Political support
  - Accountability

Types of evaluation

- Todd and Wolpin (2006): Ex-post and ex-ante
- Ex-post evaluation:
  - Selection of statistical model with a tight fit to the experiment actually happened.
  - Results are context-dependent.
  - Credibility depends on the credibility of statistical model of the experiment.
- Ex-ante evaluation:
  - Credibility depends on the validity of the statistical model of *behavior* of the individuals and the environment.

Comparison of ex-ante and ex-post (DiNardo et al 2011)

Ex post program evaluation	**Ex ante program evaluation**
What did the program do? Retrospective: what happened?	What do we think a program will do? Prospective/predictive: what would happen?
Focus on the program at hand	Focus on forecasting effects of different program
For what population *do* we identify causal effect?	For what population do we *want* to identify causal effect?
Desirable to have causal inferences not reliant on specific structural framework/model	Question ill-posed without structural framework/paradigm
No value judgments on "importance" of causal facts	Some facts will be more helpful than others
Inferences require assumptions	Predictions require assumptions
Desirable to test assumptions whenever possible	Desirable to test assumptions whenever possible
Ex Ante problem guides what programs to design/analyze	Would like predictions consistent with results of Ex Post evaluation
Inference most appropriate for situations that "resemble" the experiment and are similar to that which produce the observed data	Inferences intended for situations that are different than that which produced the observed data

IE is a growing business (Cameron et al 2016)

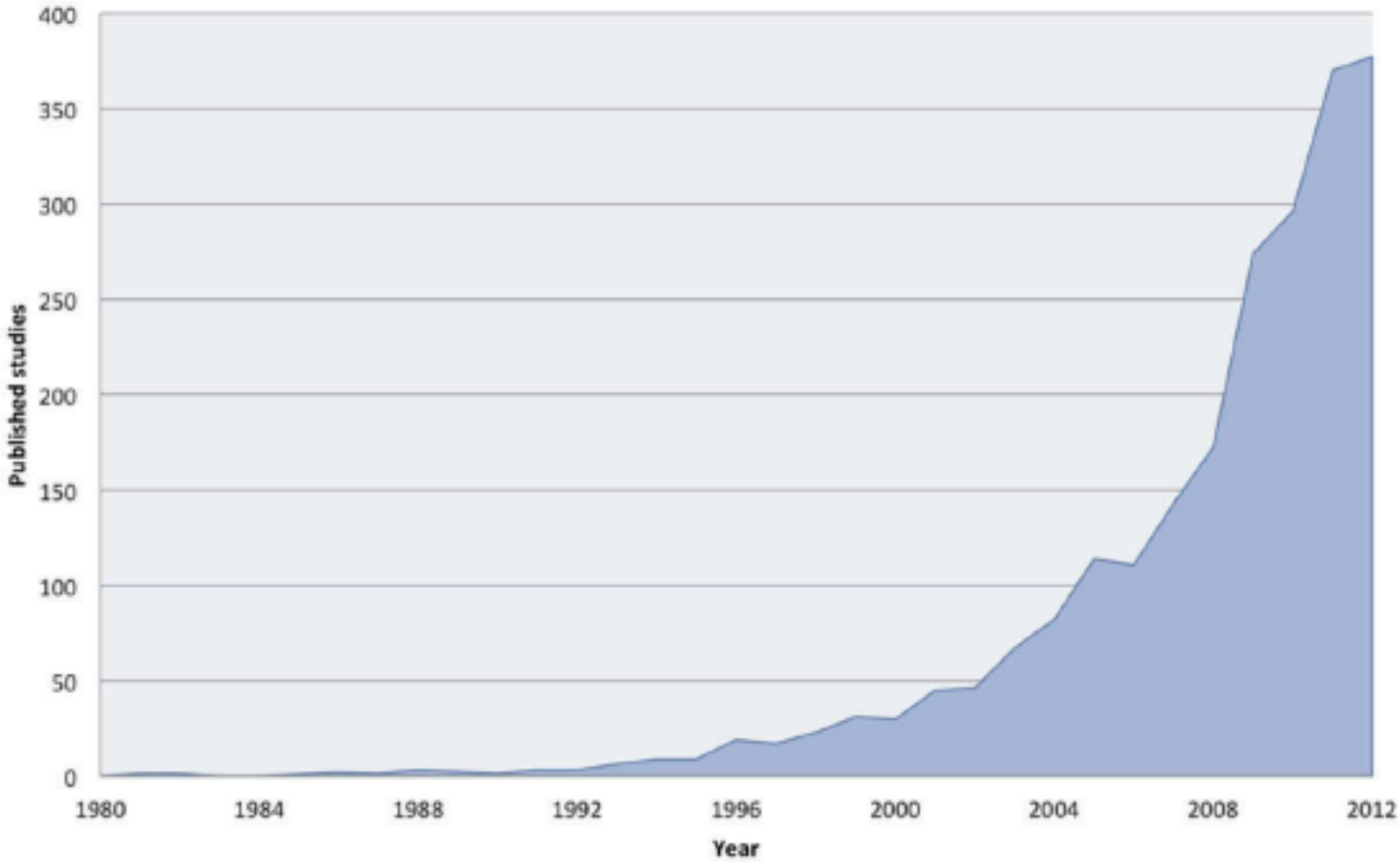


Figure 2. Impact evaluations published per year (1981–2012).

Growing is more important in health (Cameron et al 2016)

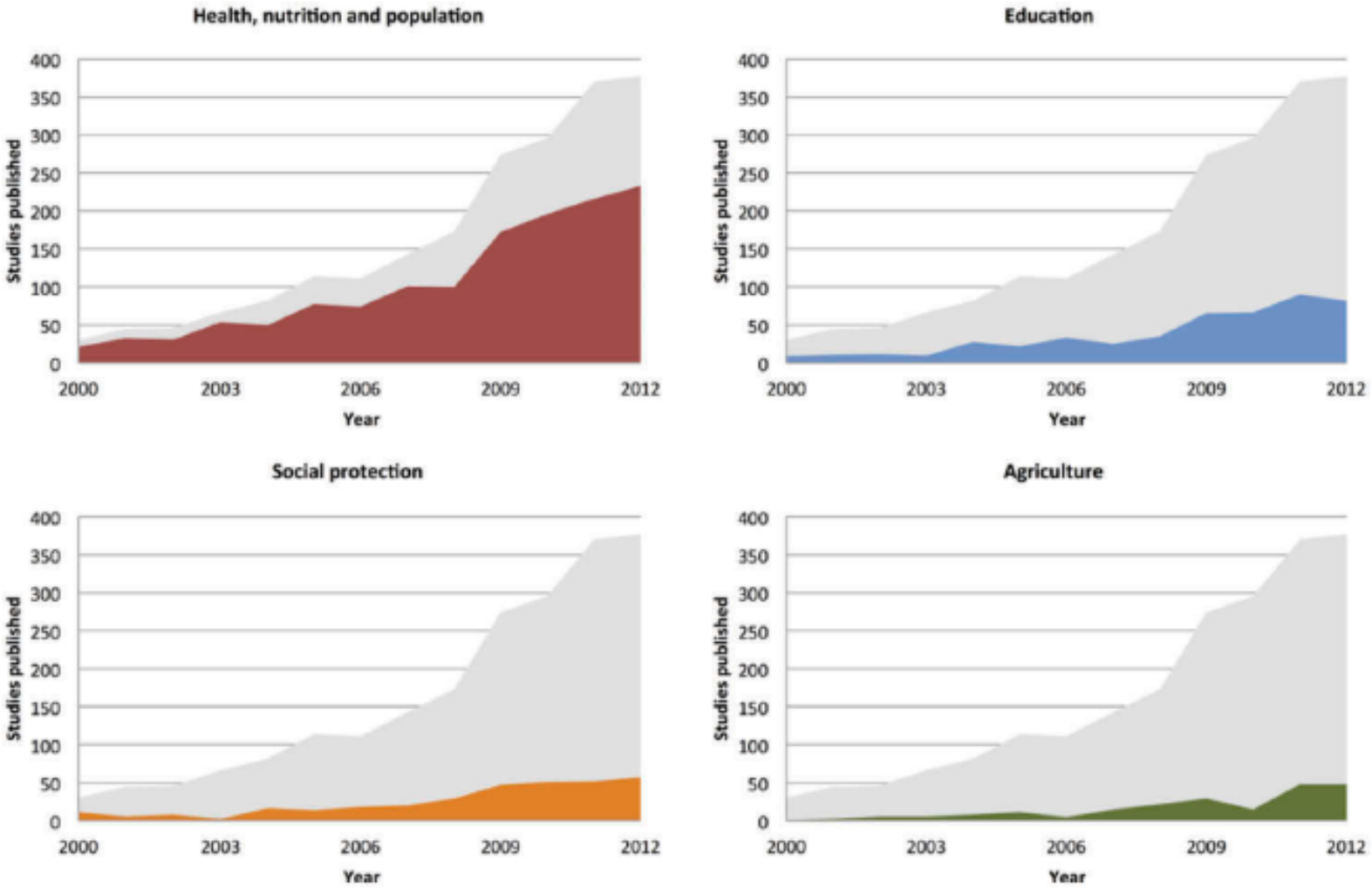


Figure 4. Impact evaluations published by major sector (2000–2012).

IE across the world (Cameron et al 2016)

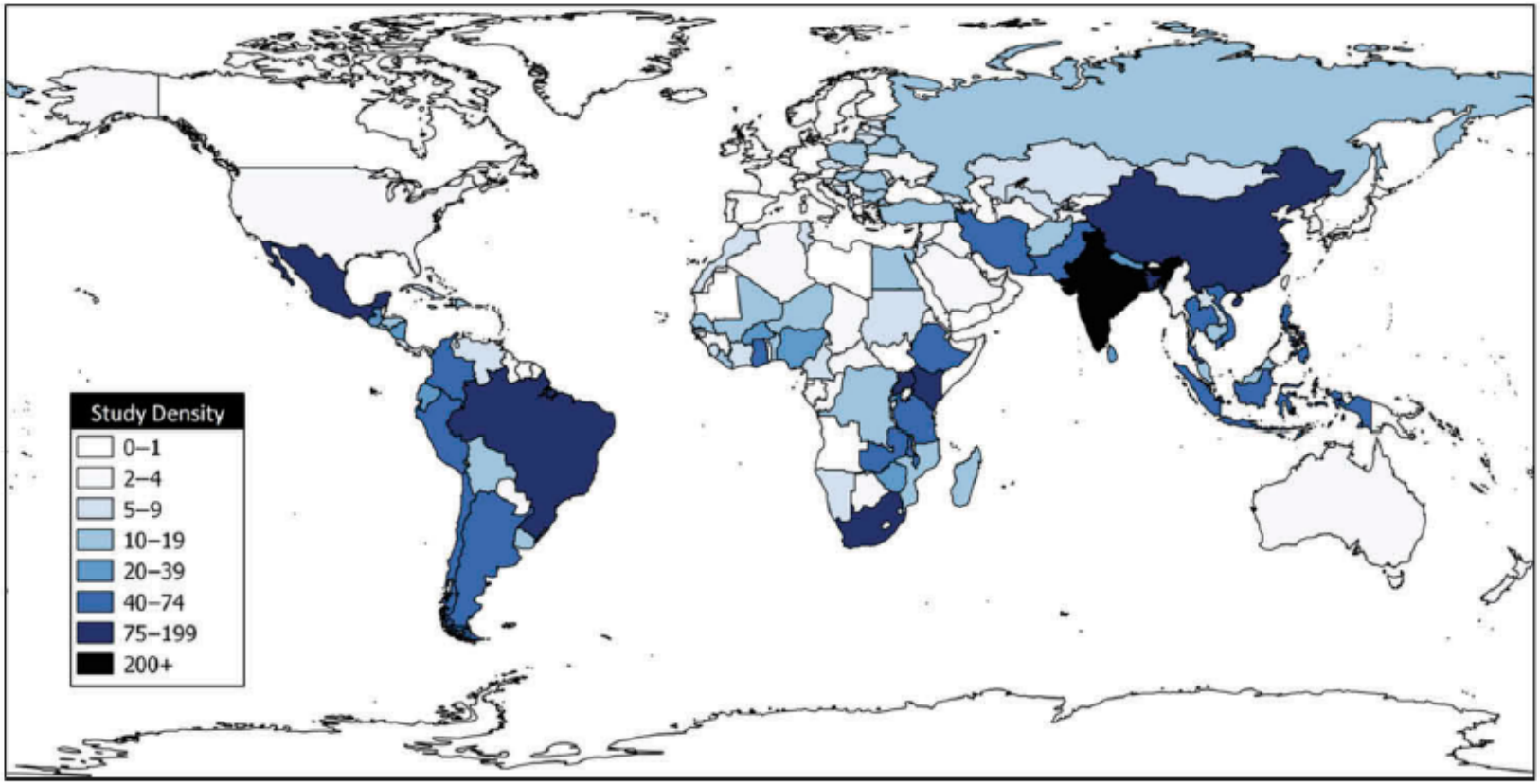


Figure 5. Heat map of low- and middle-income country impact evaluations (1981–2012).
Note: Map generated using shape files from Natural Earth in Quantum GIS version 2.2.0. Free vector and raster map data @ naturalearthdata.com.

2. Two approaches to causality in econometrics

- Two theories about causality (Pearl 2009):
  - Structural modeling (Havelmo 1943, Cowles Commission)
  - Potential outcomes framework (Neyman 1923, Rubin 1977, Holland 1986)
- But, what is causality?

- Structural modeling
  - Relies heavily in economic theory to guide empirical work
  - Interested in recovering the primitives of economic theory and seeks to estimate decision rules derived from economic models
- Potential outcomes or "experimentalist" or "design-based" approach
  - Uses economic theory to frame questions
  - The emphasis is on the problem of identifying the causal effects from specific events and situations
  - The ideal is to approximate a randomized experiment
- Modern econometrics is increasingly based on the experimentalist approach. However the research frontier is based on experimental work using structural models

Example: Structure vs. Potential Outcomes

- Imagine an economist with cross-section, plant level data on output (Q), labour inputs (L) and capital inputs (K). He wants to estimate:

$$lnQ_t = \theta_0 + \theta_1 lnL_t + \theta_2 lnK_t + \epsilon_t, \tag{1}$$

- What do we learn by estimating this regression?
- Potential answers depends on what it is assumed about the previous equation:
  - Descriptive tool
  - Causal parameter (structural and potential outcomes)

Descriptive Regression

- Best linear predictor of y given x (Goldberger 1991, Ch. 5):

$$y = lnQ_t, \tag{2}$$

$$x = (1, lnL_t, lnK_t), \tag{3}$$

$$BLP(y/x) = a + bx, \tag{4}$$

$$a = E(y) - bE(x), \tag{5}$$

$$b = cov(x, y)/var(x), \tag{6}$$

Structural Causal Regression

- Recall from basic micro the Cobb-Douglas Production Function (CDPF):

$$Q_t = AL_t^{\alpha} K_t^{\beta}, \tag{7}$$

- Equation (1) can be interpreted as a production function if we add an error term to the original deterministic relationship:

$$Q_t = AL_t^{\alpha} K_t^{\beta} exp(\epsilon_t), \tag{8}$$

- Where did the error term in (1) come from?
  - Key: affects how parameters estimated by OLS are interpreted (parameters of a CDPF or BLP)

- In other words:
  - *Economic assumptions* (Cobb Douglass PF) + *statistical assumptions* (conditions on the error term) = *Structural model*
- If a researcher assumes that error term is i.i.d., then OLS delivers consistent estimates of CDPF (perhaps too strong)
- Another researcher might consider a more realistic assumption:
  - Error term includes unobservable differences in productivity
  - OLS estimates are not longer consistent but a different econometric strategy can be used (IV)

Experimental Approach

- Imagine an economist with cross-section, plant level data on output (Q), labour inputs (L) and capital inputs (K)
- The data was collected from an experiment in which two different organization of labor schemes were randomly assigned. He wants to run the following regression:

$$lnQ_t = \varphi_0 + \varphi_1 lnL_t + \varphi_2 lnK_t + v_t, \tag{9}$$

- Are (1) and (9) equivalent? Which parameter is causal?
- Even if you are not able to run such experiment, thinking as if you were able to do it gives a causal framework to interpret (9)

- Again, what is causality?
- Depends on your theory of causality!
- Consider the structural approach:

Structural Definition

"A causal effect of X_j on Y is defined within the context of a well-specified economic model in which causes are the primitives of a relevant economic theory (such as L or K in a CDPF). The change of Y produced from the variation in X_j *holding all the other factors constant* is the causal effect of X_j" (Based on Heckman 2000)

- Consider the experimental approach:

Experimental Definition

"A causal effect is defined to be the effect on an outcome of a given action or a treatment, as measured in an *ideal randomized controlled experiment*. In such an experiment, the only systematic reason for differences in outcomes between the treatment and control groups is the treatment itself". (Stock and Watson 2007)

- Are these definitions equivalent?
- Pearl (2009) shows that both approaches are mathematically equivalent

- In fact, see this paragraph from Havelmo (1944), the founding father of the SME:

> A design of experiments (a prescription of what the physicists call a "crucial experiment") is an essential appendix to any quantitative theory. And we usually have some such experiments in mind when we construct the theories, although—unfortunately—most economists do not describe their designs of experiments explicitly. If they did, they would see that the experiments they have in mind may be grouped into two different classes, namely, (1) experiments that *we should like to make* to see if certain real economic phenomena—when *artificially isolated* from "other influences"—would verify certain hypotheses, and (2) the stream of experiments that Nature is steadily turning out from her own enormous laboratory, and which we merely watch as passive observers. In both cases the aim of theory is the same, namely, to become master of the happenings of real life. But our approach is a little different in the two cases.

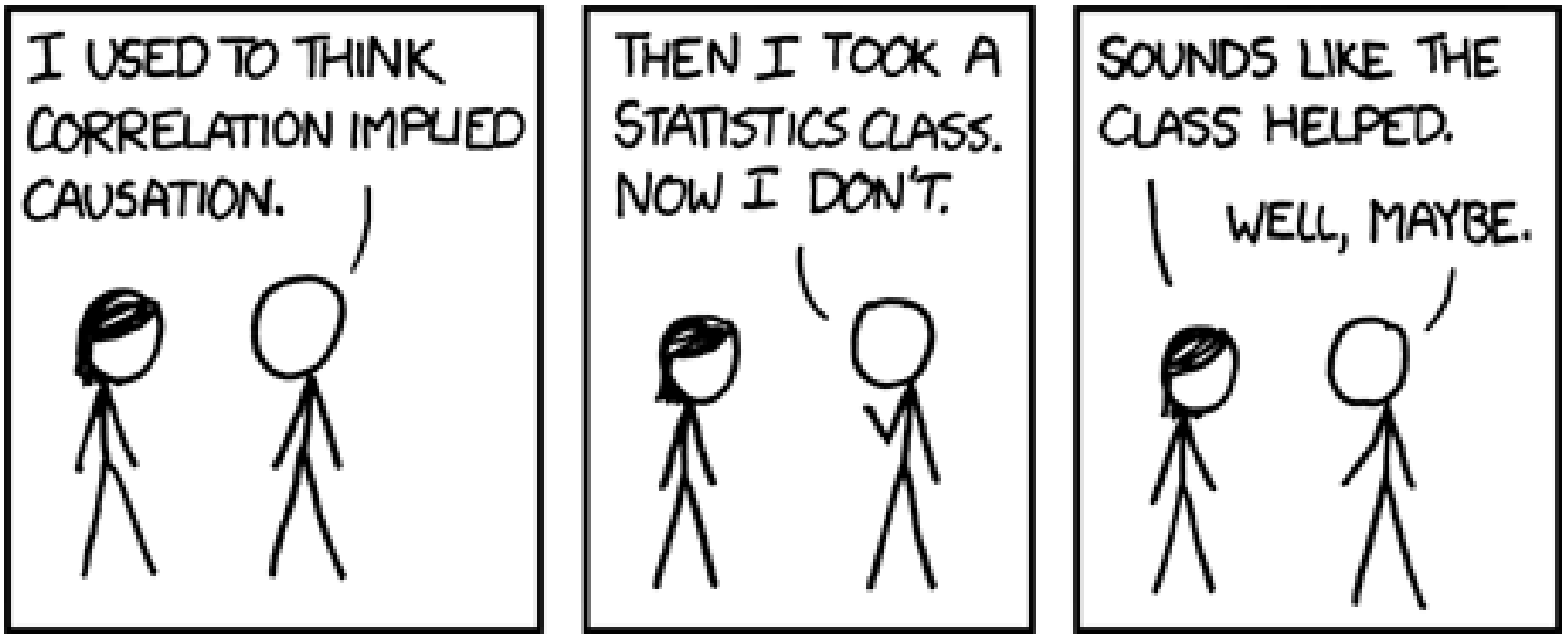
- Pearl (2009) defines an structural model using the analogy of an experiment in the spirit of Havelmo (1944):

Structural Model

"In an ideal experiment where we control X to x and any other set Z of variables (not containing X or Y) to z, the value of Y is given by $\beta x + \epsilon$, where ϵ is not a function of the setting x and z. Then, the equation $y = \beta x + \epsilon$ is said to be structural." (Pearl 2009, chapter 5)

3. Problems in estimating causal effects

- Correlation does not imply causation!!!



- Example: what is the impact of schooling (X) on earnings (Y)?
  - We want to estimate:
    - $X \Rightarrow Y$ (Causality)
  - However, evaluating the impact of X on Y can be affected by the following issues:
    - $Y \Rightarrow X$ (Reverse Causality)
    - $X \Leftrightarrow Y$ (Simultaneity)
    - $Z \Rightarrow X$ and $Z \Rightarrow Y$ (Omitted variables/confounding)
    - $X^* \Rightarrow Y$ (Measurement error)

Omitted variables

- Suppose we want to estimate $\mathbb{E}(y/X, W)$ assumed to be linear in (X, W) :

$$\mathbb{E}(y/X, W) = X\beta + W\gamma \tag{10}$$

- But you estimate: $y = X\beta + \mu$
- Therefore, $\mu = W\gamma + \epsilon$
- We will have:

$$b = (X'X)^{-1}X'y = \beta + (X'X)^{-1}X'W\gamma + (X'X)^{-1}X'\epsilon \tag{11}$$

- Asymptotically:

$$\text{plim}\, b = \beta + \text{plim}[(X'X)^{-1}X'W]\gamma \tag{12}$$

Measurement error

- Most of our data are measured with error
- Suppose the causal model is the following:

$$y_i = \alpha + \beta w_i + v_i \tag{13}$$

- However, we are not able to observe w. We can only observe x:

$$x_i = w_i + \mu_i \tag{14}$$

- Assuming classical measurement error:

$$\mathbb{E}(\mu_i / w_i) = 0 \tag{15}$$

- We can write the causal relationship as follows:

$$y_i = \alpha + \beta x_i + \epsilon_i = \alpha + \beta x_i + v_i - \beta \mu_i \tag{16}$$

- Note that x_i is correlated with composite error. This leads to bias/inconsistency in OLS estimator
- To illustrate the consistency of OLS estimator, we can write the estimator of β as follows:

$$\text{plim } b = \beta + \frac{\mathbb{E}[x_i \epsilon_i]}{\text{Var}[x_i]} = \beta + \frac{\mathbb{E}[(w_i + \mu_i)(v_i - \beta \mu_i)]}{\text{Var}[w_i + \mu_i]} \tag{17}$$

$$= \beta + \frac{-\beta \sigma_\mu^2}{\sigma_w^2 + \sigma_\mu^2} \tag{18}$$

$$= \beta \left(1 - \frac{\sigma_\mu^2}{\sigma_w^2 + \sigma_\mu^2} \right) \tag{19}$$

- b is asymptotically biased towards zero (attenuation bias). Therefore, the OLS estimator underestimates the true effect

Reverse Causality

- Correlation between y and x may be because it is y that causes x and not the other way around
- Suppose we are interested in the following causal model:

$$y = X\beta + \epsilon \tag{20}$$

- But the causal model can go in the other direction:

$$X = y\alpha + \mu \tag{21}$$

- We can estimate the "reduced form":

$$X = y\alpha + \mu = (X\beta + \epsilon)\alpha + \mu = \frac{\mu + \alpha\epsilon}{1 - \alpha\beta} \tag{22}$$

- x is correlated with ϵ. As we already know, this leads to bias in OLS estimates
- To sum up:
  - All problems have an expression in everyday language (omitted variables, reverse causality, etc)
  - All have an econometric form - the same one
  - A correlation of x with the error term

4. Potential outcomes framework

- The model was proposed originally by Neyman (1923) and further developed by Rubin (1974). We introduce here the basic terminology
- i is an index for individuals in a population
- D_i is the treatment or the potential cause of which we want to estimate the effect
  - $D_i = 1$ if individual has been exposed to treatment
  - $D_i = 0$ if individual has not been exposed to treatment
- $Y_i(D_i)$ is the outcome or the effect we want to attribute to the treatment
  - $Y_i(1)$ is the outcome in case of treatment
  - $Y_i(0)$ is the outcome in case of no treatment
- Note that:

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0) \tag{23}$$

The fundamental problem of causal inference

Definition 1: Causal effect

For every individual i, the causal effect of $D_i = 1$ is:
$\delta_i = Y_i(1) - Y_i(0)$

- Notice that the estimation of the causal effect is defined at an individual level

Proposition 1: Fundamental problem of causal inference (Holland 1986)

It is not possible to observe for the same individual the values $D_i = 1$ and $D_i = 0$ as well as the values $Y_i(1)$ and $Y_i(0)$.
Therefore, it is not possible to estimate the effect of D_i on Y_i for each individual i.

- Need to think in terms of counterfactuals!

Table I: The fundamental problem of causal inference		
Group	$Y_i(1)$	$Y_i(0)$
Treatment ($D = 1$)	Observable as Y	Counterfactual
Control ($D = 0$)	Counterfactual	Observable as Y

Solutions to the fundamental problem of causal inference

- Two solutions:
  - Scientific solution
  - Statistical solution
- Scientific solution requires additional assumptions (see Holland 1986 for details)
- The statistical solution is based on estimating the average effect of the treatment instead of doing so at an individual level
  - Average treatment effect (ATE)
  - Average treatment effect on the treated (ATT)
  - Average treatment effect on the untreated (ATU)

- **Average treatment effect** (ATE)

$$\begin{aligned}\tau_{ATE} = \mathbb{E}[\delta_i] &= \mathbb{E}[Y_i(1) - Y_i(0)] \\ &= \mathbb{E}[Y_i(1)] - \mathbb{E}[Y_i(0)]\end{aligned}$$

- **Average treatment effect on the treated** (ATT)

$$\begin{aligned}\tau_{ATT} = \mathbb{E}[\delta_i / D = 1] &= \mathbb{E}[Y_i(1) - Y_i(0) / D = 1] \\ &= \mathbb{E}[Y_i(1) / D = 1] - \mathbb{E}[Y_i(0) / D = 1]\end{aligned}$$

- **Average treatment effect on the untreated** (ATU)

$$\begin{aligned}\tau_{ATU} = \mathbb{E}[\delta_i / D = 0] &= \mathbb{E}[Y_i(1) - Y_i(0) / D = 0] \\ &= \mathbb{E}[Y_i(1) / D = 0] - \mathbb{E}[Y_i(0) / D = 0]\end{aligned}$$

- Are these effects estimable?
- Recall Table 1

Table I: The fundamental problem of causal inference		
Group	$Y_i(1)$	$Y_i(0)$
Treatment ($D = 1$)	Observable as Y	Counterfactual
Control ($D = 0$)	Counterfactual	Observable as Y

- Estimating ATE, ATT or ATU is a missing data problem
  - What is missing for estimating ATT?
  - What is missing for estimating ATU?
  - What is missing for estimating ATE?

The selection problem

- So far, we have defined the parameters of interests. The question now is how to identify them using observational data
- Your intuition suggests you to compare those that receive the treatment versus those that do not receive it
- We call this difference *MDO*, the simple mean of difference in outcomes, also known as the naive estimator:

$$\begin{aligned}
\tau_{MDO} &= \mathbb{E}[Y_i/D=1] - \mathbb{E}[Y_i/D=0] \\
&= \mathbb{E}[Y_i(1)/D=1] - \mathbb{E}[Y_i(0)/D=0] \\
&= \mathbb{E}[Y_i(1)/D=1] - \mathbb{E}[Y_i(0)/D=1] + \\
&\mathbb{E}[Y_i(0)/D=1] - \mathbb{E}[Y_i(0)/D=0] \\
&= \tau_{ATT} + \mathbb{E}[Y_i(0)/D=1] - \mathbb{E}[Y_i(0)/D=0]
\end{aligned}$$

The experimental benchmark

- If the *MDO* does not help us to recover *ATE*, what else it can be done?
- Angrist and Pischke (2009): random assignment is the most credible and influential research design because solves the "selection problem"
- Key idea of the course: even if random assignment is not possible, a research design should seek to approximate a situation that resembles an experiment in which the treatment is randomly assigned
- From Table I:

$$\mathbb{E}[Y_i/D = 1] = \mathbb{E}[Y_i(1)/D = 1]$$

$$\mathbb{E}[Y_i/D = 0] = \mathbb{E}[Y_i(0)/D = 0]$$

- Computing treatment effects requires to know counterfactuals:
  - Computing ATT requires to know: $\mathbb{E}[Y_i(0)/D = 1]$
  - Computing ATU requires to know: $\mathbb{E}[Y_i(1)/D = 0]$
  - Computing ATE requires to know both counterfactuals
- The question is to estimate these counterfactuals from what we observe:

$$\mathbb{E}[Y_i(0)/D = 1] = \mathbb{E}[Y_i(0)/D = 0]$$

$$\mathbb{E}[Y_i(1)/D = 0] = \mathbb{E}[Y_i(1)/D = 1]$$

- Generally, none of these conditions hold with observational (non-experimental) data due to the existence of selection

- However, there is an important case in which these conditions are met. That is the case of randomized experiments
- In this case, the treatment D is independent to potential outcomes $Y_i(1)$ and $Y_i(0)$
- Therefore:

$$\mathbb{E}[Y_i/D=0] = \mathbb{E}[Y_i(0)/D=0] = \mathbb{E}[Y_i(0)/D=1] = \mathbb{E}[Y_i(0)]$$

$$\mathbb{E}[Y_i/D=1] = \mathbb{E}[Y_i(1)/D=1] = \mathbb{E}[Y_i(1)/D=0] = \mathbb{E}[Y_i(1)]$$

- Then, we can compute ATE:

$$\begin{aligned}\tau_{ATE} = \mathbb{E}[\delta_i] &= \mathbb{E}[Y_i(1) - Y_i(0)] = \mathbb{E}[Y_i(1)] - \mathbb{E}[Y_i(0)] \\ &= \mathbb{E}[Y_i(1)/D=1] - \mathbb{E}[Y_i(0)/D=0] \\ &= \mathbb{E}[Y_i/D=1] - \mathbb{E}[Y_i/D=0] = \tau_{MDO}\end{aligned}$$

- Notice that we random assignment:

$$ATE = ATT = ATU \tag{24}$$

- When there is no random assignment, we must assume that the treatment is "as good as randomly assigned". This assumption can be written as follows:

$$Y_i(0), Y_i(1) \perp\!\!\!\perp D_i \tag{25}$$

- Notice that estimating ATT and ATU requires a weaker version of (25)

- With non-experimental data, we must assume some form of (25)
  - One way: argue that treatment is ignorable after conditioning by a set of covariates. This is known as selection on observables

$$Y_i(0), Y_i(1) \perp\!\!\!\perp D_i / X \tag{26}$$

  - Another way: exploiting some source of exogeneity in the data for arguing that treatment is ignorable. This is known as selection on unobservables

$$Y_i(0), Y_i(1) \perp\!\!\!\perp D_i / X, \epsilon \tag{27}$$

Potential outcomes and regression

- Re-write the observed outcome as follows:

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0) \tag{28}$$
$$= Y_i(0) + \{Y_i(1) - Y_i(0)\} D_i \tag{29}$$

- This is similar to:

$$Y_i = \alpha + \beta D_i + \epsilon \tag{30}$$

- Re-write (29) as follows:

$$Y_i = D_i Y_i(1) + (1 - D_i) Y_i(0) \tag{31}$$
$$= \underbrace{\mathbb{E}[Y_i(0)]}_{\alpha} + \underbrace{\{Y_i(1) - Y_i(0)\}}_{\beta = ATE} D_i + \underbrace{Y_i(0) - \mathbb{E}[Y_i(0)]}_{\epsilon_i} \tag{32}$$

- Taking expectations conditional on D:

$$\mathbb{E}[Y_i/D_i = 1] = \beta + \mathbb{E}[\epsilon_i/D_i = 1] \quad (33)$$

$$\mathbb{E}[Y_i/D_i = 0] = \mathbb{E}[\epsilon_i/D_i = 0] \quad (34)$$

- Estimating β using OLS gives:

$$\mathbb{E}[Y_i/D_i = 1] - \mathbb{E}[Y_i/D_i = 0] = \beta + \underbrace{\mathbb{E}[\epsilon_i/D_i = 1] - \mathbb{E}[\epsilon_i/D_i = 0]}_{\text{Selection bias}}$$

- When treatment is randomly assigned, the selection bias is zero:

$$\underbrace{\mathbb{E}[\epsilon_i/D_i = 1] - \mathbb{E}[\epsilon_i/D_i = 0]}_{\text{Selection bias}} = 0$$

- Therefore:

$$\mathbb{E}[Y_i/D_i = 1] - \mathbb{E}[Y_i/D_i = 0] = \beta \tag{35}$$

The stable treatment unit value assumption (SUTVA)

- SUTVA is a critical assumption in the potential outcomes framework

SUTVA (Morgan and Winship 2014)

"The potential outcomes of an individual is unaffected by potential changes in the treatment exposures of other individuals ."

- One way to understand SUTVA: no general equilibrium effects due to the treatment.
- Examples:

Table 2.2: A Hypothetical Example in Which SUTVA is Violated

Treatment assignment patterns			Potential outcomes	
$\begin{bmatrix} d_1 = 1 \\ d_2 = 0 \\ d_3 = 0 \end{bmatrix}$	or $\begin{bmatrix} d_1 = 0 \\ d_2 = 1 \\ d_3 = 0 \end{bmatrix}$	or $\begin{bmatrix} d_1 = 0 \\ d_2 = 0 \\ d_3 = 1 \end{bmatrix}$	$y_1^1 = 3$ $y_2^1 = 3$ $y_3^1 = 3$	$y_1^0 = 1$ $y_2^0 = 1$ $y_3^0 = 1$
$\begin{bmatrix} d_1 = 1 \\ d_2 = 1 \\ d_3 = 0 \end{bmatrix}$	or $\begin{bmatrix} d_1 = 0 \\ d_2 = 1 \\ d_3 = 1 \end{bmatrix}$	or $\begin{bmatrix} d_1 = 1 \\ d_2 = 0 \\ d_3 = 1 \end{bmatrix}$	$y_1^1 = 2$ $y_2^1 = 2$ $y_3^1 = 2$	$y_1^0 = 1$ $y_2^0 = 1$ $y_3^0 = 1$

"No causation without manipulation" (Holland 1986)

- Poorly defined treatments are those in which the treatment cannot be potentially manipulated
- Example:
  - She scored highly on the exam because she is female
  - She scored highly on the exam because she studied
  - She scored highly on the exam because her teacher tutored her
- In which case the potential outcomes are correctly defined?
- Some research at the frontier is looking to understand causal effects when manipulation is not possible

Advantages of the Potential Outcomes (Imbens et al 2008)

- Allows to define the causal effect before specifying the assignment mechanism without making functional or distributional assumptions
- Links the analysis of causal effects to explicit manipulations
- Separates the modeling of the potential outcomes from that of the assignment mechanism.
- Allows to formulate probabilistic assumptions in terms of potentially observable variables rather than in terms of unobserved components
- Clarifies where the uncertainty in the estimators come from

Critics to the Potential Outcomes Framework

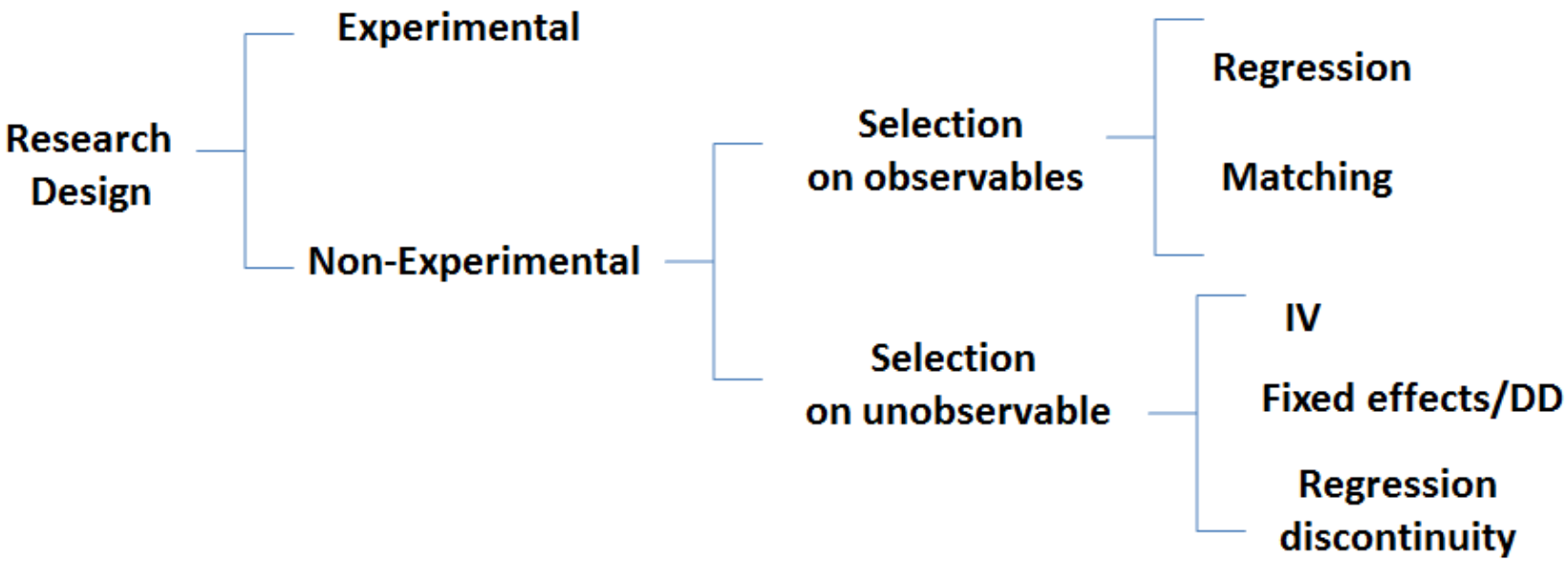
- Conflates the separate tasks of defining causality and identifying causal parameters from data (Heckman 2010)
- Abandonment of economic choice theory and lack of clarity about selection mechanisms
- Data cannot determine interesting economic relationships without a priori assumptions (Keane 2010)
- Difficulties for interpreting estimates of a econometric model

Potential outcomes versus structure (Heckman 2010)

TABLE 2
COMPARISON OF THE ASPECTS OF EVALUATING SOCIAL POLICIES THAT ARE COVERED BY THE NEYMAN–RUBIN APPROACH AND THE STRUCTURAL APPROACH

	Neyman–Rubin Framework	Structural Framework
Counterfactuals for objective outcomes (Y_0, Y_1)	Yes	Yes
Agent valuations of subjective outcomes (I_D)	No (choice-mechanism implicit)	Yes
Models for the causes of potential outcomes	No	Yes
Ex ante versus ex post counterfactuals	No	Yes
Treatment assignment rules that recognize the voluntary nature of participation	No	Yes
Social interactions, general equilibrium effects and contagion	No (assumed away)	Yes (modeled)
Internal validity (problem **P1**)	Yes	Yes
External validity (problem **P2**)	No	Yes
Forecasting effects of new policies (problem **P3**)	No	Yes
Distributional treatment effects	No[a]	Yes (for the general case)
Analyze relationship between outcomes and choice equations	No (implicit)	Yes (explicit)

Research design and causality



5. Validity issues

- "Validity" refers to the approximate truth of an inference (Shadish, Cook and Campbell 2002).
- Types:
  - Internal validity
  - External validity
  - Statistical conclusion validity
  - Construct validity
- We will focus on the first two, the ones more discussed in economics

- Internal versus external validity

Internal Validity

"Refers to the validity of inferences about whether observed covariation between X and Y reflects a causal relationship from X to Y in the form in which the variables were manipulated or measured."

External Validity

"Concerns to the validity of inferences about the extent to which a causal relationship holds over variation in persons, settings, treatment and outcomes."

Threats to internal validity

- Ambiguous temporal precedence: lack of clarity about which variable occurred first
- Selection: Systematic differences over conditions in respondent characteristics
- History: Events occurring concurrently with treatment
- Maturation: Naturally occurring changes over time could be attributed incorrectly to treatment
- Attrition: Loss of respondents to treatment or to measurement produce biased treatment effects

- Testing: Exposure to a test can affect scores on subsequent exposures to that test, fact that can be correlated with the treatment
- Instrumentation: The nature of a measure may change over time or condition in a way that can be confused with the treatment

Threats to external validity

- Interaction of the causal relationship with units: An effect found in certain kind of units might no hold if other kind of units had been studied
- Interaction of the causal relationship over treatment variations: An effect found with one treatment variation might not hold with other variations of that treatment
- Interaction of the causal relationship with outcomes: An effect found on one kind of outcome variation may not hold if other outcome observations were used

- Interaction of the causal relationship with settings: An effect found in one kind of setting may not hold if other kinds of settings were to be used
- Context-dependent mediation: An explanatory mediator of a causal relationship in one context may not mediate in another context