The Economics of European Regions: Theory, Empirics, and Policy

Dipartimento di Economia e Management

Co-funded by the Erasmus+ Programme of the European Union

Project funded by

European Commission Erasmus + Programme –Jean Monnet Action

Project number 553280-EPP-1-2015-1-IT-EPPJMO-MODULE

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November 11, 2019

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Introduction

Many empirical question in economics and other social science depend on causal effects of policies, as for example:

- **Medical therapies**: does aspirin reduce the headache? Do vaccines increased life expectancy?
- **Educational policies**: does college increase future earnings? Do students' loans increase college education and earnings of poor high school graduates?
- **Labor market programs**: does training increase the employment probability of unemployed? Does Employment Protection Legislation (EPL) increase unemployment?
- **Cohesion policy**: do EU Structural and Cohesion Funds boost regional economic growth in Europe?
- **Free movement policy**: does the implementation of the Schengen Agreement boost labour mobility?

Policy intervention

- A **policy** is an intervention targeted to a specific population with the purpose of inducing a change in a defined state and/or behaviour.
- A policy intervention is characterised by:
  - a *target population*: a well-defined set of units (e.g. persons, households, firms, geographic areas) upon which the intervention will operate at a particular time;
  - an *intervention*: an action, or a set of actions, whose effect on the outcome the analyst wishes to assess relative to non-intervention;
  - an *outcome variable*: an observable and measurable characteristic of the units of the population on which the intervention may have an effect.

Policy evaluations

- Public policies or interventions are implemented with the expectation of improving the situation of the individuals affected by them
  $\Rightarrow$ adequate **policy evaluation** exercise.

- Measuring the effect of publicly-provided training programmes on an individual's future employability it is not trivial:
  - Should the outcomes of the participants be compared to their pre-programme situations?
  - Or should the outcomes of the participants be compared with those of the non-participants?
  - What is the best approach to measure the effect of the programme on the outcomes of participants?

Policy evaluation applies evaluation principles and methods to examine the content, implementation or impact of a policy, to understand the merit, worth, and utility of a policy.

Why evaluate?

- To contribute to the design of interventions, including providing input for setting political priorities.
- To assist in an efficient allocation of resources.
- To improve the quality of the intervention.
- To report on the achievements of the intervention (i.e. accountability).

Conducting evaluation: causality

- The aim of policy evaluation is to measure the **causal effect** of a policy on outcomes of interest, on which it is expected to have an impact.
- The policy's causal effect is defined as the difference between the outcome of the units affected by the policy (the actual situation) and the outcome that these same units would experience had the policy not been implemented (the counterfactual).

Causality: the basic framework

Imbens and Rubin (2015)

"In everyday life, causal language is widely used in an informal way". One might say: "My headache went away because I took an aspirin," or "She got a good job last year because she went to college," or "She has long hair because she is a girl."

Imbens and Rubin (2015). *Causal Inference for Statistics, Social, and Biomedical Sciences*.

Causality: the basic framework (cont.)

- Such comments are typically informed by observations on **past exposures** of:
  - headache outcomes after taking aspirin or not;
  - characteristics of jobs of people with or without college educations;
  - the typical hair length of boys and girls.
- Therefore, these observations generally involve **informal statistical analyses**, drawing conclusions from **associations** between measurements of different quantities that vary from individual to individual (i.e., random variables).
- Correlation (or association) is not the same as causation!

Correlation vs causation

- A **causal statement** presumes that, although a *unit* was at a particular point in time exposed to a *particular action* (or **treatment**), the same unit could have been exposed to an *alternative* action (or treatment) *at the same point in time*.
- The same physical object or person at a different time is a *different* unit.

- For instance, we could have decided to take an aspirin to relieve the headache, or not to take the aspirin, or to take an alternative medicine.

Treatment and control

In most of the cases the possible actions are two and often:

- one of these actions corresponds to a more *active treatment* (e.g., taking an aspirin);
- in contrast to a more *passive action* (e.g., not taking the aspirin).

Formally the two treatments are viewed *symmetrically* although the literature usually labels:

- **treatment** the more active treatment; and,
- **control** the other.

Potential outcome

- Given a unit and a set of actions, we associate *each action-unit pair* with a **potential outcome**.
- These outcomes are **potential** outcomes because **only one will ultimately be realized** and therefore possibly **observed** (i.e., the one corresponding to the action actually taken).
- *Ex post*, the other potential outcomes cannot be observed because the corresponding actions were not taken.
- The **causal effect** of one action or treatment relative to another involves the **comparison of these potential outcomes**, one realized (and perhaps, though not necessarily, observed), and the others not realized and therefore not observable.
- Any treatment **must occur temporally before** the observation of any associated potential outcome is possible.

Example 1: headache

In the headache example the action is clear: "I took an aspirin, but at the time that I took the aspirin, I could have followed the alternate course of not taking an aspirin."

- Treatment applied: "aspirin taken";
- Counterfactual potential outcome is the state of the headache under "aspirin not taken".

$\Rightarrow$ The "because" statement is causal as it reflects the comparison of those two potential outcomes (unambiguous).

Example 2: college

In the college example it is less clear what the treatment and its alternative are: “she went to college, and at the point in time when she decided to go to college, she could have decided not to go to college”.

- Treatment applied: “she went to college”;
- Counterfactual potential outcome is her job a year ago had she decided “not to go to college”.

⇒ The implied causal statement compares the quality of the job she *actually* had then, to the quality of the job she would have had a year ago without going to college.

However, the potential outcome under the alternative action is somewhat murky: if not enrolled in college what would she have done?

Example 3: long hair

The informal statement is "she has long hair because she is a girl."

- *Implicit treatment*: "being a girl";
- Counterfactual potential outcome is the person's hair length if she were a boy rather than a girl.

But there is **no action articulated** that would have made her a boy and allowed us to observe the alternative potential outcome of hair length for this person as a boy!

$\Rightarrow$ This "because" statement is *ill-defined* as a causal statement.

Definition of causal effect

- Single unit at a particular point in time.
- Two treatment levels: taking an aspirin, and not taking an aspirin.
- If I take the aspirin, my headache may remain or not one hour later: we denote this outcome by Y(Aspirin), which can be either "Headache" or "No Headache".
- If I do not take the aspirin, my headache may remain or not one hour later: we denote this potential outcome by Y(No Aspirin), which also can be either "Headache", or "No Headache".
- There are therefore **two potential outcomes**, Y(Aspirin) and Y(No Aspirin), one for each level of the treatment.
- The **causal effect** of the treatment involves the **comparison of these two potential outcomes**.

Definition of causal effect (cont.)

Because in this example **each** potential outcome can take on **only two values**, the unit-level causal effect (i.e., the comparison of these two outcomes for the same unit) involves one of four (two-by-two) possibilities:

1. Headache gone only with aspirin (**Causal effect**):
   $Y(\text{Aspirin}) = \text{No Headache}, \; Y(\text{No Aspirin}) = \text{Headache}$
2. No effect of aspirin, with a headache in both cases (**Zero causal effect**):
   $Y(\text{Aspirin}) = \text{Headache}, \; Y(\text{No Aspirin}) = \text{Headache}$
3. No effect of aspirin, with the headache gone in both cases (**Zero causal effect**):
   $Y(\text{Aspirin}) = \text{No Headache}, \; Y(\text{No Aspirin}) = \text{No Headache}$
4. Headache gone only without aspirin (**Causal effect**):
   $Y(\text{Aspirin}) = \text{Headache}, \; Y(\text{No Aspirin}) = \text{No Headache}$

Definition of causal effect (cont.)

- The definition of the causal effect **depends** on the potential outcomes, but it **does not depend** on which outcome is actually observed.
  Whether I take an aspirin (and am therefore unable to observe the state of my headache with no aspirin) or do not take an aspirin (and am thus unable to observe the outcome with an aspirin) *does not affect* the definition of the causal effect.

- The causal effect is the comparison of potential outcomes, **for the same unit**, **at the same moment** in time post-treatment.
  The causal effect is *not* defined in terms of comparisons of outcomes at different times, as in a before-and-after comparison of my headache before and after deciding to take or not to take the aspirin.

The fundamental problem of causal inference

As opposed to the *definition* of causal effects, for **estimation** and **inference**, we need to compare observed outcomes, that is, **observed realizations** of potential outcomes.

Rubin (1978, p. 38)

"The fundamental problem facing inference for causal effects is that if treatment t is assigned to the ith experimental unit (i.e., $W_i = t$), only values in Y^t can be observed, Y^j for $j \neq t$ being unobservable (or missing)."

Holland (1986, p. 947)

"It is impossible to *observe* the value of $Y_t(u)$ and $Y_c(u)$ on the same unit and, therefore, it is impossible to *observe* the effect of t on u" (emphasis in original).

Missing-data problem

- The focus is a population of units, indexed by $i = 1, ..., N$.
- The treatment indicator W_i takes on the values 0 (the control treatment, e.g., no aspirin) and 1 (the active treatment, e.g., aspirin).
- For each unit we have one realized (and possibly observed) potential outcome.
- For unit i, now $i \in 1, ..., N$, let Y_i^{obs} denote this realized (and possibly observed) outcome:

$$Y_i^{obs} = Y_i(W_i) = \begin{cases} Y_i(0) & \text{if } W_i = 0 \\ Y_i(1) & \text{if } W_i = 1 \end{cases}$$

- For each unit we also have one **missing potential outcome**, for unit i denoted by Y_i^{mis}:

$$Y_i^{mis} = Y_i(1 - W_i) = \begin{cases} Y_i(1) & \text{if } W_i = 0 \\ Y_i(0) & \text{if } W_i = 1 \end{cases}$$

Multiple units

- Because there is only one realized potential outcome per unit, we will need to consider **multiple units**.
- More specifically, we must observe multiple units, some exposed to the active treatment, some exposed to the alternative (control) treatment.
- One option is to observe the same physical object under different treatment levels at different points in time.

  Myself at different times, taking and not taking aspirin.
- Alternatively, one might observe different physical objects at approximately the same time.

  If both you and I have headaches, but only one of us takes an aspirin, we may attempt to infer the efficacy of taking aspirin by comparing our subsequent headaches.

The Stable Unit Treatment Value Assumption (SUTVA)

However, with more than one unit we might have problems that we need to restrict by assumption.

SUTVA, Rubin (1978)

The potential outcomes for any unit do not vary with the treatments assigned to other units, and, for each unit, there are no different forms or versions of each treatment level, which lead to different potential outcomes.

1. **No Interference**: units do not interfere with one another
   we could exclude the possibility that your taking or not taking aspirin has any effect on my headache;
2. **No Hidden Variations of Treatments**: for each unit there is only a single version of each treatment level
   we could exclude the possibility that the aspirin tablets available to me are of different strengths.

Exclusion restrictions

SUTVA is the first of a number of assumptions that are referred to generally as *exclusion restrictions*: assumptions that rely on external, substantive, information to rule out the existence of a causal effect of a particular treatment relative to an alternative.

Note, that these assumptions are not directly informed by observations **they are assumptions**!! Therefore, they rely on previously acquired knowledge for their *justification*.
Causal inference is generally impossible without such assumptions, and thus it is critical to be explicit about their content and their justifications.

SUTVA: No Interference

- In economics the general equilibrium effect might be a serious concern.
- A labour market program that affects the labour market outcomes for an individual potentially has an effect also on the labour market outcomes for others.
- In a world with a fixed number of jobs, a training program could only redistribute the jobs.
- In practice these general equilibrium effects may or not be a serious problems depending on the size of the indirect effect compared to the direct one.

Solutions:

- redefine the unit of interest;
- directly model the interactions (Manski, 1993).

The assignment mechanism

Even with SUTVA, inference for causal effects requires the specification of an **assignment mechanism**, i.e. a probabilistic model for how each individual came to receive the treatment level actually received.

In the headache example:

- Suppose that the person who chose not to take the aspirin did so because he had only a minor headache.
- Suppose then that an hour later both headaches have faded: the headache for the first person possibly faded because of the aspirin, and the headache of the second person faded simply because it was not a serious headache.
- When comparing these two observed potential outcomes, we might conclude that the aspirin had no effect, whereas in fact it may have been the cause of easing the more serious headache.

The assignment mechanism (cont.)

Consider the example in Imbens and Rubin (2015):

- 4 patients (units).
- Two possible medical procedures (treatment) labelled 0 (Drug) and 1 (Surgery).

Table 1.4. ***Medical Example with Two Treatments, Four Units, and SUTVA: Surgery (S) and Drug Treatment (D)***

Unit	Potential Outcomes		Causal Effect
	$Y_i(0)$	$Y_i(1)$	$Y_i(1) - Y_i(0)$
Patient #1	1	7	6
Patient #2	6	5	−1
Patient #3	1	5	4
Patient #4	8	7	−1
Average	4	6	2

Years of post-treatment survival. Source: Imbens and Rubin (2015).

On average, Surgery is **better** than Drug by two years' life expectancy.

The assignment mechanism (cont.)

Suppose now that the doctor *knows* enough about these potential outcomes and so *assigns* each patient to the treatment that is *more beneficial* to that patient.

Table 1.5. ***Ideal Medical Practice: Patients Assigned to the Individually Optimal Treatment; Example from Table 1.4***

Unit i	Treatment W_i	Observed Outcome Y_i^{obs}
Patient #1	1	7
Patient #2	0	6
Patient #3	1	5
Patient #4	0	8

Assignment mechanism. Source: Imbens and Rubin (2015).

- Average observed outcome with surgery: $(7+5)/2=6$
- Average observed outcome with the drug treatment: $(6+8)/2=7$

The assignment mechanism (cont.)

- The average observed outcome with surgery (6) is one year **less** than the average observed outcome with the drug treatment (7).
- This might be led to believe that, on average, the drug treatment is superior to surgery.
- In fact, the opposite is true!
- If the drug treatment were *uniformly applied* to a population of these four patients, the average survival would be four years, as opposed to six years if all patients were treated with surgery!

$\Rightarrow$ we cannot simply look at the observed values of potential outcomes under different treatments and reach valid causal conclusions irrespective of the assignment mechanism!

Lord's paradox

To illustrate the clarity of the potential outcomes interpretation of causality consider:

The Lord's paradox:

A large university is interested in investigating the effects on the students of the diet provided in the university dining halls and any sex differences in these effects. Various types of data are gathered. In particular, the weight of each student at the time of his arrival in September and his weight the following June are recorded.

Results:

- For the males the average weight is identical at the end of the school year to what it was at the beginning (the whole distribution of weights is unchanged, although some males lost weight and some males gained weight).
- The same thing is true for the females.
- Females started and ended the year lighter on average than the males.

Lord's paradox (cont.)

The paradox is generated by considering the contradictory conclusions of two statisticians asked to comment on the data.

Statistician 1

There is no evidence of any interesting effect of diet (or of anything else) on student weight. In particular, there is no evidence of any differential effect on the two sexes, since neither group shows any systematic change.

Lord's paradox (cont.)

The paradox is generated by considering the contradictory conclusions of two statisticians asked to comment on the data.

Statistician 1

There is no evidence of any interesting effect of diet (or of anything else) on student weight. In particular, there is no evidence of any differential effect on the two sexes, since neither group shows any systematic change.

Statistician 2

After "controlling for" initial weight, the diet has a differential positive effect on males relative to females because for males and females with the same initial weight, on average the males gain more than the females.

Lord's paradox (cont.)

- Such gain scores are **not causal effects** because they do not compare potential outcomes at the same time post-treatment; rather, they compare changes over time.
- If both statisticians confined their comments to *describing* the data, both would be correct.
- For causal inference, both are wrong!

Lord's paradox (cont.)

- The units are the *students*.
- The time of application of active treatment (the university diet) is *September*.
- Time of the recording of the outcome Y is *June*.
- Let us accept the stability assumption.
- The potential outcomes are June weight under the university diet $Y_i(1)$ and under the "control" diet $Y_i(0)$.
- The covariates are sex of students, male versus female, and September weight.
- But the assignment mechanism has **assigned everyone to the new treatment**! There is **no one**, male or female, who is **assigned to the control** treatment.

$\Rightarrow$ there is absolutely no purely empirical basis on which to compare the effects, either raw or differential, of the university diet with the control diet!

The assignment mechanisms

The **assignment mechanisms** is defined as the conditional probability of receiving the treatment, as a function of potential outcome and observed covariates:

$$Pr\left(W|X, Y(0), Y(1)\right)$$

We can distinguish three classes of assignment mechanism:

1. Randomized experiments
2. Unconfounded assignments
3. Assignment mechanisms with some dependence

1. Randomized experiments

In **randomized experiments** the probability of assignment to treatment **does not depend** on potential outcomes and is a **known** function of covariate.

Consider a population of N units. In a *completely randomized experiment* $N_1 < N$ randomly chosen units are assigned to the treatment and the remaining $N_0 = N - N_1$ units are in the control group.

- There are *in practice* few experiment in economics, and most of them are completely randomized experiments.
- The use of formal randomization has become more widespread in the social science in recent years.
- In randomized experiments estimators for the average effect of the treatment are usually given by the difference in means by treatment status.

2. Unconfounded assignments

The **unconfounded assignments** mechanisms maintains the restrictions that the assignment probability **does not depend** on potential outcomes:

$$Pr\left(W|X, Y(0), Y(1)\right) = Pr\left(W|X\right)$$

- Unconfoundedness is also referred in the literature as selection on observables, exogeneity and conditional independence.
- All these labels refer to some for of the assumption that *adjusting treatment and control groups* for differences in observed covariates (or pre-treatment variables), remove all biases in comparisons between treated and control units.
- At each set of values of X_i (sex, age, etc.) that has a distinct probability of $W_i = 1$, there is effectively a complete randomize experiment.

Pre-treatment variables X_i

Examples:

- In the headache example pre-treatment variables could include the intensity of the headache before making the decision to take aspirin or not.
- In the evaluation of job training on future earnings pre-treatment variables could include age, previous educational achievement, family, and socio-economic status, or pre-training earnings.

Sometimes a covariate (e.g., pre-training earnings) differs from the potential outcome (post-training earnings) solely in the timing of measurement, in which case the covariates can be highly predictive of the potential outcomes.

The *key* characteristic of these covariates is that they are *a priori* known to be **unaffected by the treatment** assignment.

They are permanent characteristics of units, or they took on their values prior to the treatment being assigned

Pre-treatment variables X_i (cont.)

They have an effect on the assignment mechanism: assumptions about the assignment mechanism and its independence on potential outcomes are more plausible within subpopulations that are *homogeneous* with respect to some *covariates*, that is, **conditionally given the covariates**, rather than unconditionally.

Young unemployed individuals may be more interested in training programs aimed at acquiring new skills.
As a result, those taking the active treatment may differ in the values of their background characteristics from those taking the control treatment.
At the same time, these characteristics may be associated with the potential outcomes.

3. Assignment mechanisms with some dependence

The third class of assignment mechanisms contains all remaining assignment mechanisms with **some dependence** on potential outcome.

There are two general methods that relax the unconfoundedness without replace it with additional assumption:

- **Sensitivity analysis**: where robustness of estimates to specific limited departures from unconfoundedness are investigated (Rosenbaum and Runin, 1983; Rosenbaum, 1995).
- **Bounds on estimands**: where ranges of estimands consistent with the data and the limited assumptions the researcher is willing to make, are derived and estimated (Manski, 1990; 2003; 2007).

3. Assignment mechanisms with some dependence (cont.)

Many of the possible dependence create substantive problems for the analysis except in some special cases:

- **Instrumental variables**: it relies on the presence of additional treatments, the so-called instruments, that satisfy specific exogeneity and exclusion restrictions (Imbens and Angrist, 1994; Angrist, Imbens and Rubin, 1996).
- **Differences-in-differences**: it relies on the presence of additional data in the form of samples of treated and control units before and after the treatment (e.g., Ashenfelter and Card, 1985; Athey and Imbens, 2006).
- **Regression discontinuity designs**: it applies to settings where overlap is completely absent because the assignment is a deterministic function of covariates, but comparisons can be made exploiting continuity of average outcomes as a function of covariates (see Cook, 2007).