QUANTITATIVE POLICY EVALUATION IRREGULAR ASSIGNMENTS: INSTRUMENTAL VARIABLES.

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The critical premise underpinning randomised experiments is that of **unconfoundedness**.

Under this premise, the reception of an active or control treatment is independent of units' potential outcomes or characteristics.

In practice, however, this premise is difficult to justify beyond the confines of pure experimental settings.

Example 1: The effects of education.

In social sciences, for example, researchers are often interested in understanding the consequences of different levels of educational attainment on wealth and labour market outcomes (Angrist and Lavy (1999); Oreopoulos et al., 2008; Attanasio et al., 2015; Card, 2001).

Educational attainment is, however, not randomly allocated: It depends on various factors, among which household's socioeconomic status is of critical importance.

Example 1: The effects of education.

Socio-economic status is correlated with families' investment in children's education.

- ▶ Dahl and Lochner (2012) find that a \$1,000 increase in income raises achievement in maths and reading test scores by 6 percent of a standard deviation in the short run
- ▶ Similarly, Milligan and Stabile (2011) found that a child benefit program had significant positive effects on children's test scores (among other outcomes).
- ▶ A comprehensive review by Lochner and Monge-Naranjo (2012) presents further evidence that credit constraints have important implications for schooling (and other aspects of households' behaviour).

Example 1: The effects of education.

Socio-economic status is also correlated with the wealth of offspring.

▶ Black et al. (2015) use administrative data on the net wealth of a large sample of Swedish adoptees merged with similar information for their biological and adoptive parents.

We find that, even before any inheritance has occurred, wealth of adopted children is more closely related to the wealth of their adoptive parents than to that of their biological parents. This suggests that wealth transmission is primarily due to environmental factors rather than because children of wealthy parents are inherently more talented.

Example 1: The effects of education.

So wealth families invest more in children's education; children from better-off families also inherit greater wealth; so any positive effect of education on future wealth is intertwined with the wealth effect of inheritances and endowments.

Data reveal the result of this complex relationship; that is, the pure effect of education is **confounded**. Without further assumptions, there is no way data alone can separate these different effects.

Example 2: Epidemiological studies.

In epidemiology, researchers often want to understand the effects of smoking and life-style on a population's overall health.

Confounding when studying this kind of question arises because, again, people's decisions regarding specific health behaviours are likely to be driven by factors that are also correlated with health

These factors might include educational attainment, socio-economic status, environment and others e.g. -Frisell et al., 2012; Kubicka et al., 2001; Stautz et al., 2016).

Example 3: Randomized Control Experiments.

Finally, even in controlled clinical environments, who receives a specific treatment or medical innovation in a trial might be driven by factors beyond randomization.

Specifically, individuals assigned to the active treatment arm of an intervention might decide not to comply with the protocols or drop from the research.

Conversely, individuals assigned to the control arm of an intervention might be able to force their way into the active treatment group if they intuit that the medical innovation might lead to health benefits. This might occur, for example, through sharing of information among participants or spillovers due to the benefits of the active treatment (e.g. vaccines).

Though confounding arises in a varied range of disciplines and its sources are varied, it is possible to develop the analysis of irregular (confounded) mechanisms within the common framework of non-compliance with treatment assignment.

That is, we will distinguish between

- ▶ Assignment to the active/control treatment, which we will still assume to be unconfounded.
- **uptake** of treatment, which is confounded.

The unconfounded assignment can be used to estimate the **propensity** to uptake the active treatment.

Subsequently the estimated propensity to uptake treatment is used to identify the causal effect.

In this sense, the unconfounded assignment is an **instrumental variable** (Angrist, Imbens, and Rubin, 1996; Imbens and Angrist, 1994).

Consider the following hypothetical scenario

A completely randomized experiment with N = 10 patients, $N_t = 5$.

Subjects suffer from chronic obstructive lung disease (COLD).

The active treatment is to **encourage** the $N_t = 5$ patients to exercise.

We want to see the effect of exercise on forced expiratory volume (FEV), with larger values indicating better health.



Unit	Encouraged	Exercised	$Y_i(0)$	Y_i
1	1	1	5	8
2	1	1	4	7
3	1	1	3	6
4	1	0	2	2
5	1	0	1	1
6	0	1	5	8
7	0	0	4	4
8	0	0	3	3
9	0	0	2	2
10	0	0	1	1

where $Y_i(0)$ is the potential level of FEV if the unit does not receive the treatment, and Y_i is the observed level of FEV at the end of the experiment.

Unit	Encouraged	Exercised	$Y_i(0)$	Y_i
1	1	1	5	8
2	1	1	4	7
3	1	1	3	6
4	1	0	2	2
5	1	0	1	1
6	0	1	5	8
7	0	0	4	4
8	0	0	3	3
9	0	0	2	2
10	0	0	1	1

Note that the effect of exercise equals 3 (this can be seen from the table for units 1, 2, 3, and 6).

The problem is that

- ▶ Patients 4 and 5 in the active treatment group were heavy smokers, and had so low FEV to being with that they couldn't bother exercising.
- ▶ Patient 6 in the control group happens to be an ex-marathon runner who decided to begin to exercise soon after being assigned to the control group.

The research team could only observe who adhered to the treatment/control groups, but they did not know why non-adherence happened.

Confounding arises because those with lowest FEV are not complying with the treatment and one of the fittest individuals in the study is not complying with the control treatment. This is an example of **noncompliance** due to **cross-over**.

So, although assignment was unconfounded (random) the uptake of treatment (exercise) is determined by factors that relate directly to the outcome of interest (FEV). So people **self-select** into treatment on the basis of potential outcomes.

In searching for a solution, a first attack to the problem would be compare FEV by **assignment** group; after all, <u>assignment</u> is unconfounded, and we now from our exploration of randomized experiments that a comparison of outcomes by unconfounded assignment has a causal interpretation.

This comparison will capture, however, the effect of **assignment** to treatment, that is the **Intention-to-Treat** (ITT) parameter, rather than the causal effect of exercise itself.

$$ITT = E(FEV|Encouraged) - E(FEV|Not Encouraged)$$
 (1)



The ITT can be estimated by a simple difference in means, which in our example yields

$$\bar{Y}_1 - \bar{Y}_0 = 1.2,$$
 (2)

which rightly (see the table) suggests that encouragement to exercise boosts FEV...

... but it grossly underestimates the effect of exercise (which we know, from the table, was equal to 3).

To understand why the ITT underestimates (in this example) the effect of exercise, note that

- ► E(FEV|Encouraged) is underestimated because some assigned units decided not to exercise -thus foregoing the beneficial effects of exercise.
- ▶ E(FEV|Not Encouraged) is over-estimated because one unit decided to exercise, even though this unit was assigned to the control group.

What can we do to circumvent this (in this example) underestimation?



Intuitively, the effect of exercise should manifest itself only through those units who adhered to their assigned treatment. These units are usually called **compliers**. So the I.V. solution is to try to attribute the ITT just to these compliers.

The question is, then, how can we figure out which proportion of the population are compliers.

In principle, we could look at the proportion P(Exercise|Encouraged). The problem is that this moment measures not only the proportion of compliers, but also the proportion of individuals who would take on the treatment even if there were not assigned to treatment¹ (we call these **always-takers**).

What if we then focus on the difference P(Exercise|Encouraged) - P(Exercise|Not Encouraged)?

The moment P(Exercise|Not Encouraged) measure the proportion of always-takers in the population. So, in principle the above difference would be capturing the proportion of compliers in the population

But if there were some hard-liners in the population who would always do the opposite of what they are asked to do, they would also be included in P(Exercise|Not Encouraged). Call the latter hard-liners **defiers**.

Though the existence of defiers sounds outlandish, we cannot rule it out from data alone. We need to **assume it out**. This assumption is one of the two essential pillars of IV analysis.

By assuming that there are not defiers, the final step towards the solution of our problem is to attribute the ITT to the population of compliers; that is divide the ITT estimate of 1.2 by the mean difference in exercise across assignment groups:

$$P(\text{Exercise}|\text{Encouraged}) - P(\text{Exercise}|\text{Not Encouraged}) = \frac{3}{5} - \frac{1}{5} = \frac{2}{5} \tag{3}$$

and so our estimate of the effect of exercise is,

LATE =
$$\frac{E(\text{FEV}|\text{Encouraged}) - E(\text{FEV}|\text{Not Encouraged})}{P(\text{Exercise}|\text{Encouraged}) - P(\text{Exercise}|\text{Not Encouraged})}$$
$$= \frac{1.2}{0.4} = 3 \tag{4}$$

... spot on!



Example 1 (the classic): Angrist, 1990

Does serving in the military affect long term market outcomes?

We cannot just compare veterans and non-veterans, because those who serve in the military are likely to differ from the general population in non-trivial ways.

For instance, individuals with relatively few civilian opportunities might be more likely to sever in the military.

So the active treatment is confounded.

Example 1 (the classic): Angrist, 1990

Angrist (1990) notes, however, that eligibility to sever in the military during the Vietnam war was partially determined by five draft lotteries

Eligible individuals were allocated a random number from 1 to 365 (identifying birth day). Weeks after the lottery, when the Defence Department knew its manpower needs, a ceiling number between 1-365 was set and individuals with an allocated number below the ceiling would be given priority for induction.

The lottery serves as an unconfounded assignment from which the likelihood of serving in the military can be predicted.

Example 2: Random judges.

An important question in criminology is to what extent does punishment reduces the likelihood of recidivism.

In this debate, the effect of punishment on drug-related charges is of particular interest. In the US, for example, drug-related incarceration is particularly prominent, with punishment of this kind of crime increasing since the 1980s.

Some studies suggest that harsher punishment leads to lower levels of re-offending; other studies contend that the opposite is true.

The problem is that unobserved attributes of a defendant that lead to harsher sentences also might affect the defendants probability of rearrest. So data on their own cannot separate the effect of a hasher sentence on re-offending from the effect of an individuals' traits on re-offending.

Example 2: Random judges.

Green and Winik (2010) note that in some jurisdictions, judges are randomly allocated to court cases.

In so far different judges reveal different levels of leniency, we can use this natural experiment to determine whether **unconfounded** allocation to a harsh judge results in **confounded** hasher conviction.

Then, any variation in recidivism across defendants assigned to harsh/lenient judges can be attributed to the variation in the harshness of the conviction due to variation in the leniency of the judge.

Example 3: Pension eligibility.

People are living longer. At the same time, there has been a trend towards early retirement. When combined, these two trends are putting enormous pressure on welfare systems around the world.

A concern is that retirement might affect individual's health. On the one hand, retirement might signify the end of the hardships of a harmful or stressful job; on the other hand, however, retirement might lead to a loss of a social network and a sedentary life style.

If the latter effect is dominant, it can only exacerbate the negative effects of ageing on the financial viability of welfare systems.

Example 3: Pension eligibility.

However, health is a determinant of retirement decisions (Disney et al., 2006). As a result, data alone cannot disentangle the effect of health and retirement on each other.

Across the world, however, governments have imposed a number of rules determining the age at which one can retire or the age at which one can receive a State Pension.

State pensions tend to be significant sources of income (in the UK amounting to 25% of the average wage) and indeed, they seem cause about 15-20% of the retirement decisions.

Example 3: Pension eligibility.

The state pension age is determined by governments and there is a great deal of arbitrariness in the agreed exact state pension age. In other words, the state pension age does not seem to be related to people's health levels.

As a result, it was often been argued that, given the accident of birth, the moment when one individual reaches the stage pension age is allocated almost as if at random.

This suggests focusing on the variation in health of those individuals who cross the state pension age. Any observed variation can be attributed to the variation in retirement status caused by variations in state pension eligibility.

Other examples of instruments.

▶ Weather shocks (e.g. drought). A case can be made that these shocks are unpredictable (unconfounded) but they are responsible for variations in food prices. This can assist to disentangle the effect of income/consumption on civil unrest, household welfare and other outcomes.

Other examples of instruments.

▶ Lotteries determine income to some extent, presumably independently of other factors, such as political attitudes. This provides us with the basis to examine the effect of income on people's views on, for instance, redistributive policies.

Other examples of instruments.

▶ **Historical institutions**. Did the British Colonial system cause the variation of development in India today?

British India

- Direct rule: British administrators collected taxes and administered local governments
- ▶ Indirect rule: Native princes collected revenue on behalf of the British, but retained some autonomy over local administration.
- ▶ Unfortunately, it seems that the British implemented direct rule depending of factors such as the region's agricultural potential.
- ▶ Therefore a comparison of current economic achievement by type of rule during colonisation does not have a causal interpretation.

Other examples of instruments.

- ▶ Enter the *Doctrine of Lapse*: if a local prince died without a heir, the region would be intervened and put under direct rule.
- ▶ 20 districts of present day India saw their prince die without heirs, of which 16 were put under direct rule.
- ▶ Thus implementation of the Doctrine of Lapse can be used as an instrumental variable to explain direct rule in certain regions.
- ▶ Any variation in current economic development across districts affected and unaffected by the death of the local ruler can be attributed to the variation in the type of rule caused by the Doctrine of Lapse.

To introduce the main results, we follow Angrist et al. (1996) and illustrate the development of the techniques from the perspective of a randomized experiment where assignment to treatment is unconfounded, but the actual reception of treatment is confounded (as in the COLD example earlier on). This will will manifest itself in non-compliance (with some units not adhering to their assigned treatment)

- ▶ We maintain Neyman's superpopulation approach.
- ► SUTVA is also maintained throughout.
- ► Three relevant variables:
 - ightharpoonup An outcome, Y
 - ▶ A binary indicator T which equals 1 if a unit receives the active treatment of interest (0 otherwise)
 - ▶ A binary indicator Z which equals 1 if a unit has been assigned to receive the active treatment (0 otherwise)
- \blacktriangleright We want to infer the effect of T on Y.



Assignment to the active treatment is random.

However, some units might decide not to adhere to the assigned treatment due to observed and unobserved factors².

Critically, Z is still assumed to determine treatment to a large extent. Specifically, received treatment is assumed to be a function of assignment. This allows us to define define the potential outcomes

$$T_i(Z_i) (5)$$

²In the COLD example, 2 units with very low initial FEV opted for not exercising, probably because of their lower level of pre-assignment fitness. Similarly, when studying the effect of retirement on health, ndividuals with a frail health might be prone to retire earlier; when looking at the effect of serving in the military on job market outcomes, individuals with relatively high lottery numbers might volunteer for military service in order to secure less exposed destination; participants in a clinical trial might not comply with treatment due to unforeseen side effects

Counfounded treatment manifests itself in non-compliance: some assigned units do not take the active treatment and some of the treated units were actually not assigned to receive this treatment.

This leads us to define 4 subpopulations:

- 1. Compliers: Those whose treatment is dictated by assignment, so that $Z_i = T_i$ or $(T_i(1) = 1, T_i(0) = 0)$
- 2. Always-takers: Those who take the active treatment regardless of their assignment, so that $(T_i(1) = 1, T_i(0) = 1)$
- 3. Never-takers: Those who never take the active treatment regardless of their assignment, so that $(T_i(1) = 0, T_i(0) = 0)$
- 4. **Defiers**: Those whose do the opposite of what their assignment dictated, $(T_i(1) = 0, T_i(0) = 1)$

The implications of confounding in this setting can be seen in the following table:

		Assignment		
		0	1	
Treatment	0	Never-takers Compliers	Never-takers Defiers	
	1	Always-takers Defiers	Always-takers Compliers	

Table: Supopulations defined by treatment and assignment

Data on treatment assignment and uptake of treatment cannot identify any of the four subpopulations. This is problematic: the identification strategy suggested by the COLD example relied on us attributing any variation in Y across assignment groups to those individuals whose treatment status was changed by assignment (the compliers).

Before proceeding further, it remains to define the potential outcomes of Y.

In principle we cannot rule out that a unit's potential outcome is not affected by the assignment variable³, so that we could define $Y_i(Z_i, T(Z_i))$

If this were the case, however, any comparison of outcomes by treatment group (even under perfect compliance) would be capturing the two effects simultaneously: the effect of assignment on Y and the effect of treatment on Y. We would not be able to separate these two effects without considerably strong assumptions.

This leads us to introduce a critical assumption of the IV framework...

³For instance if Z_i were a function of age, and Y was a measure of age, \mathbb{R}

The exclusion restriction.

For all values of z, z' and t, Y(z;t) = Y(z';t).

Said differently, Z does not affect Y(.) directly; its effect is fully channelled through T. If Z violates this assumption, we say that Z is an **invalid instrument**⁴

Under the exclusion restriction, we can write $Y_i(Z_i, T(Z_i)) = Y_i(T_i(Z_i)) = Y_i(T_i)$

⁴Identification under invalid instruments is a very active area of current research.

Coming back to the attribution of the ITT to the compliers, you will recall that we need to rule out the existence of defiers.

Again, data alone cannot tell us which proportions of compliers, defiers, always-takers and never-takers there are in the population. So to rule out defiers, we need to assume their existence out.

Monotonicity.

$$T_i(1) \ge T_i(0)$$

Under this assumption, we rule out situations where a unit has $T_i(1) = 0$ and $T_i(0) = 1$. Again, this is not something we can test from data; we need to assume this holds.

And we are <u>almost</u> done. Given

- ▶ Unconfounded assignment
- ► SUTVA
- ► Exclusion
- ► Monotonicity

It follows that the ratio

LATE =
$$\frac{E(Y_i|Z_i=1) - E(Y_i|Z_i=0)}{P(T_i|Z_i=1) - P(T_i|Z_i=0)}$$
 (6)

equals the effect of T on Y for the subpopulation of compliers, where LATE stands for Local Average Treatment Effect.

LATE =
$$\frac{E(Y_i|Z_i=1) - E(Y_i|Z_i=0)}{P(T_i|Z_i=1) - P(T_i|Z_i=0)}$$
 (7)

- The above is 'Local' in the sense that it identifies the effect of T on Y for the compliers, not for the whole population.
- ► So, LATE≠ATE.

There is just one final issue: to successfully attribute the ITT to the compliers, the denominator of LATE must be positive. Once we have ruled out defiers by assumption, looking at Table 1, we can be 'sure' that $P(T_i|Z_i=1) - P(T_i|Z_i=0)$ is not negative.

However, $P(T_i|Z_i=1) - P(T_i|Z_i=0)$ could still be 0 (if there are actually no compliers in the population).

One principal reason why this might occur is if Z has no effect on T. Once again, we rule this out by assumption.

Non-zero causal effect of Z on T.

$$E\big[T_i(1) - T_i(0)\big] \neq 0$$

Estimation of LATE.

By virtue of the assumption of unconfounded assignment we can estimate LATE by

$$\hat{\tau}_{LATE} = \frac{\hat{\tau}_{ITT_Y}}{\hat{\tau}_{ITT_T}} = \frac{\bar{Y}_1 - \bar{Y}_0}{\bar{T}_1 - \bar{T}_0}$$
 (8)

where

$$\bar{Y}_1 = \frac{1}{N_1} \sum_{i=1}^{N} Z_i \cdot Y_i \text{ and } \bar{Y}_0 = \frac{1}{N_0} \sum_{i=1}^{N} (1 - Z_i) \cdot Y_i$$
 (9)

$$\bar{T}_1 = \frac{1}{N_1} \sum_{i=1}^{N} Z_i \cdot T_i \text{ and } \bar{T}_0 = \frac{1}{N_0} \sum_{i=1}^{N} (1 - Z_i) \cdot T_i$$
 (10)

and N_1 (N_0) is the number of units assigned to treatment (control) in the sample.

Estimation of LATE.

It can also be shown that the variance of $\hat{\tau}_{LATE}$,

$$V(\hat{\tau}_{LATE}) = \frac{1}{\hat{\tau}_{ITT_T}^2} V(\hat{\tau}_{ITT_Y})$$

$$-2\frac{\hat{\tau}_{ITT_Y}}{\hat{\tau}_{ITT_T}^3} \text{Cov}(\hat{\tau}_{ITT_Y}, \hat{\tau}_{ITT_T}) + \frac{\hat{\tau}_{ITT_Y}^2}{\hat{\tau}_{ITT_T}^4} V(\hat{\tau}_{ITT_T})$$
(11)

Where, $V(\hat{\tau}_{ITT_Y})$ is the sum of the sample variances of Y by assignment groups and $V(\hat{\tau}_{ITT_T})$ is the sum of the sample variances of T by assignment groups, and

$$Cov(\hat{\tau}_{ITT_Y}, \hat{\tau}_{ITT_T}) = \frac{1}{N_1 \cdot (N_1 - 1)} \sum_{i=1}^{N} Z_i \cdot (Y_i - \bar{Y}_1)(T_i - \bar{T}_1) \quad (12)$$

The traditional econometric approach

The starting point is:

$$Y_i = \alpha + \tau_{LATE} \cdot T_i + \varepsilon_i \tag{13}$$

Here τ_{LATE} is interpreted as the <u>constant</u> causal effect of the receipt of treatment on the outcome.

This relationship cannot be estimated by OLS because ε_i is potentially correlated with T_i . The classical approach is to <u>assume</u> that an instrumental variable Z exists, such that

$$E(\varepsilon_i|Z_i) = 0. (14)$$



The traditional econometric approach

Under $E(\varepsilon_i|Z_i) = 0$, we see that

$$E[Y_i|Z_i] = \alpha + \tau_{LATE} \cdot E[T_i|Z_i]. \tag{15}$$

It follows that we can write a new regression function for Y,

$$Y_i = \alpha + \tau_{IV} \cdot E[T_i|Z_i] + \eta_i \tag{16}$$

where $\eta_i = \varepsilon_i + (T_i - E[T_i|Z_i]) \cdot \tau_{IV}$ can be shown to be uncorrelated with Z_i or any function of Z_i , such as $E[T_i|Z_i]$.

The latter matters because if we knew $E[T_i|Z_i]$ then model (16) could be estimated by OLS.

The traditional econometric approach

In practice, we estimate $E[T_i|Z_i]$ by regressing T_i on Z_i , and obtain the predicted values of this regression, which are an estimate of $E[T_i|Z_i]$, say \hat{T}_i .

Then, we estimate the following model by OLS,

$$Y_i = \alpha + \tau_{IV} \cdot \hat{T}_i + \eta_i \tag{17}$$

This is known as **Two-Stage Least Squares** (2SLS).

In this case where not other variables (covariates) are considered it can be shown that the OLS estimator of τ_{IV} is <u>numerically</u> identical to the estimator of LATE, $\hat{\tau}_{LATE}$.

The traditional econometric approach

Although $\hat{\tau}_{IV} = \hat{\tau}_{LATE}$ when no other covariates are included in the analysis, there are significant <u>qualitative</u> differences between both estimators due to the underlying assumptions made. Specifically,

- ▶ The 2SLS assumed a constant treatment effect; however this is not required to estimate $\hat{\tau}_{LATE}$
- ▶ Second $E[\varepsilon_i|Z_i] = 0$ implies both the exclusion restriction and unconfoundedness. Thus $E[\varepsilon_i|Z_i] = 0$ mixes design-related assumptions (unconfoundedness) with substantive assumptions (exclusion). This makes it difficult to assess its overall plausibility.

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