Module 5: Randomized Promotion

Contents

1. INTRODUCTION

In the previous module we introduced the basics of experimental design and the benefits of conducting the gold standard, a Randomized Control Trial (RCT). Under ideal conditions, experimental designs provide a solution to the selection bias problem common in non-experimental empirical research. If we can find a proper control group such that selection bias is eliminated, then a comparison of means between treatment and control groups provides unbiased estimates of the causal effects.

In this module we will introduce another type of experimental method, randomized promotion, which is used when excluding some respondents from participating is not possible due to logistical or ethical reasons. We will then discuss the challenges faced by both experimental and non-experimental research, namely imperfect compliance, spillovers (externalities), and attrition.

At the end of this module, student should be able to:

- ✓ Understand methods to deal with imperfect compliance
- ✓ Estimate the causal effects in case of "externalities"
- ✓ Understand the effect of attrition as well as some analytical methods to nevertheless estimate unbiased coefficients
- ✓ In the next modules, we will dive deeper into the main quasi-experimental methods used in impact evaluations, mainly the Regression Discontinuity Design (RDD) and Difference-in-Differences (DID), followed by a module on how to properly conduct a power analysis and design a sample of interest.

2. RANDOMIZED PROMOTION

Randomized promotion, also known as randomized offering or an encouragement design, is a method of impact evaluation used when one is not able to choose who gets access to the program. In a sense, nobody can be excluded from receiving the treatment. Some examples of this would be a nationally funded project that is open to all, or a program that relies on participants volunteering to be part of the intervention. The fundamental problem with these kinds of program evaluations is that selection bias will be plaguing the internal validity of our estimates as the decision to enroll is correlated with both observed and unobserved covariates.

What, if any, is the difference between a Randomized Promotion and offer? A randomized offer is appropriate when you are in fact able to exclude some people from the treatment, but you cannot force anyone to participate. This means you would offer the program to a random sub-sample of respondents, then some will accept the offer while others will not.

If you cannot exclude anyone from the program, then you will likely use a randomized promotion to exogenously change the probability of participation. In this situation, there will be an additional incentive/encouragement/promotion to a random sub-sample of respondents in hopes of increasing

the chance of them signing up. These additional promotions, for example, could include supplemental information about the benefit of the program, a small incentive/gift for participation, or a way to lower the perceived costs of joining through subsidized transportation costs.

There are **three necessary conditions** that need to be in place before implementing a randomized offering/promotion:

- ✓ The groups need to be comparable! Those that are offered/promoted and not-offered/not-promoted need to be comparable. This means checking for successful randomization, and that those offered/promoted the program are not correlated with population characteristics.
- ✓ Offered/promoted group has higher enrollment in the program.
- ✓ Offering/promoting the program does not affect the outcomes directly.

We will discuss more of the math behind compliance in the subsequent section, but we can preemptively deduce that the analysis will come from comparing those on the margin of signing up; aka, all they needed was a promotion/nudge to engage. Those that always enroll and those that never enroll will be "differenced out" which means the change in enrollment will be entirely driven by those that are influenced by the encouragement/promotion/offer. This is what we refer to in section 3 as a "Local Average Treatment Effect (LATE)" as it is only valid for those people that complied with the assignment to treatment. The subsequent analysis provides us with a causal estimate called the "treatment on the treated", which will be covered in section 3.2.

Figure 1. Real World Example – Community Based School Management in Nepal

Before 2003, Nepal had a centralized method of managing schools. In 2003, the government of Nepal decided to allow local administers to handle the management of the schools in a decentralized manner. Since every school district in the country could technically be allowed to join, meaning that no one could be excluded from the process, this meant that the impact evaluators had to randomly encourage/promote the new policy change. This was done by offering a financial incentive of 1500 USD to interested schools.

In the end, 40 communities were offered the promotion/encouragement to participate, and 40 were not offered anything. Fifteen of the 40 schools that were given an incentive took up the program, while only five of the 40 schools in the control group participated.

2.1 Things to Keep in Mind

- ✓ The promotion/offer itself should be an effective strategy to promote participation. This means you will need to pilot test the proposed idea before rolling it out to the rest of the sample.
- ✓ The use of a randomized promotion will not only allow you to estimate the impact of the program, but it will also shed light on what methods are most effective at increasing enrollment in said program.
- ✓ This is an ideal strategy when people cannot be excluded from accessing the treatment.
- ✓ The estimate calculated from a randomized promotion does not give us an average treatment effect that is applicable or expected for the average person in the population. Instead, it gives us a Localized ATE (Average Treatment Effect, which will be explained shortly) that shows us the expected impact for those who are on the margin between participating and not-participating; in this case, the promotion serves as the nudge to one group or another.

3. IMPERFECT COMPLIANCE

Simply put, imperfect compliance stems from choices made by study participants. In laboratory experiments, researchers may have better control over the experimental conditions, and tightly controlled studies can ensure high compliance with study design. However, most real interventions offer participants some choice in whether to adopt and adapt to various interventions. For example, researchers might open a school in a village, but school attendance decisions may be made by children and their parents, rather than by researchers.

Similarly, participants in the control group may demand or receive the intervention contrary to the intended study design. For example, political or ethical issues may require the implementing agency to provide the treatment to a part of the control group.

In summary, when the treatment group does not receive the treatment as intended, or when the control group receives some treatment despite the intention of the researchers, we have a problem of imperfect compliance.

Differences between **the original treatment assignment** and the **actual treatment status** can negate the advantage of initial treatment randomization. Randomization assumes that all measured and unmeasured confounders are balanced, then what factors explain the decision of some treatments not to adopt the treatment? If we compare people who actually receive the treatment to those who did not, then the two groups may differ in terms of some confounding factors that can explain such non-adherence to the study protocol (confounders that the initial randomization was designed to avoid). If we drop the people who did not adhere to the study protocol from our analysis, then the remaining sample may nevertheless be confounded by the non-random selection of analyzed participants. In summary, attempts to 'correct' imperfect compliance often results in losing the advantage of randomization.

The problem of control group contamination is also prevalent in evaluations of scaled up interventions. Treating the treatment group in such interventions may affect the outcome of interest in the control group, albeit not to the same extent, but the causal effect of the treatment would thereby be underestimated by our standard analysis discussed in the previous module.

In case of evaluations aimed at proving a concept, testing a new drug or advertisement plan, or efficacy trials, it may be important to evaluate the effect under conditions of perfect compliance, and thus these are evaluations performed under tightly controlled conditions (like laboratory experiments). In contrast, for large scale programs that are targeting large swaths of a population under real-world conditions, it may be important for policy makers to know the effect of the intervention taking into account that some people will not use it, a framework known as "intention to treat" analysis.

3.1 Intention to Treat Effect

The most straightforward approach to deal with compliance imperfection is to estimate **Intention-to-Treat (ITT)**. This estimate is robust because a) it is conservative (it underestimates the potential impact of an intervention in case of imperfect compliance); and b) it maintains randomization and is therefore less prone to selection bias. In ITT analysis, we evaluate the causal effect of *assignment* to the treatment group, whether or not the participant is actually treated. Recall that in practice, we evaluate causal effects as,

$$Impact = E[Y|T = 1]_{trt} - E[Y|T = 0]_{ctr}$$

where Y is the outcome of interest and T is the treatment status (1 or 0). In ITT analysis, T = 1 if the individual (or cluster) is assigned to treatment group, whether or not the actual treatment is received/adopted, and T=0 if an individual is assigned to the control group irrespective of her post-intervention behavior (e.g. Imagine we have an intervention that randomly distributed mosquito nets, but then someone decided to buy a mosquito net with her own money, instead of remaining "untreated" in the control group). Therefore, in the face of imperfect compliance, the ITT effect will underestimate the true causal effects: $E[Y|T = 1]_{trt}$ may be underestimated and $E[Y|T = 0]_{ctr}$ may be overestimated.

Exercise 5.1: Open the `PanelPROGRESA_97_99year.csv`; this is the same dataset as that used previously. We assume that the villages (`villid`) were randomly assigned PROGRESA (`D`). However, the families in the study villages had a choice whether to participate in the program; we indicate participants with `D_HH`. In reality there was an eligibility criterion to participate in PROGRESA in the selected villages, but for this exercise we will assume that all households in treatment villages were eligible. Now, cross-tabulate the frequency of households assigned to the treatment groups and their actual participation. What is the level of compliance?

Now estimate the effect of D and D_HH on household income (IncomeLab_HH) in year 1998. Which effect is larger in magnitude (ignore their statistical significance)? Do you think that either effect is measured causally?

3.2 Average Treatment Effect on the Treated

In face of imperfect compliance, researcher may still be interested in knowing what would have been the causal effect had compliance been complete, which is referred to as the Average Treatment Effect on the Treated (ATET or TOT). In case of perfect compliance, ATET/TOT and ITT effects are the same. TOT effects can be policy relevant. For example, if we find much larger TOT effects than ITT effects, then we know that we can substantially magnify the impact by improving implementation of the programs to increase participation. If the TOT effect is low, then we can expect that the intervention will not be effective even when participation increases; the TOT should never be smaller than the ITT effect.

Let's decompose ITT effects to understand how we can estimate ATET or TOT effects from ITT effects as,

$$ITT = \delta_c . I_c + \delta_d . I_d + \delta_n . I_n + \delta_a . I_a$$

where I labels the causal effects and δ_i is the fraction of total sample size in group denoted by subscripts such that $\sum \delta_i = 1$. The subscript a represents the "always participating" group, n represents the "never-taker" group, c represents the "complier" group who adhere to the protocol, and d represents "defier" group who do opposite to their treatment assignment. If we can rule out the effect of defiers, never-takers and always-takers under assumptions, then the ATET or TOT effects (I_c) can be estimated as,

$$I_c = ITT / \delta_c$$

Another approach to estimate ATET or TOT effects is through **method of instrumental variables** (IV). It is useful to summarize the concept of endogenous and exogenous variables in economics to understand IV method; these terms are used slightly differently in other fields (such as epidemiology). An exogenous variable is a variable independent from the system we are studying. For example, in the short term the type of house construction, education level of the household head or occupation can be considered exogenous because they don't have time to change in response to the treatment or outcomes we are studying. On other hand, endogenous variables are determined within the studied system, changing in response to exogenous variables (whether measured or not) as well as the intervention and outcome itself. For example, participants may change their participation in the program in response to their expectations about the success of the program or based on their contemporaneous need. Therefore, the effect of their participation on the outcome will be biased (endogeneity bias) because their participation itself is affected by the outcome or expectation of the outcome). As we have argued under the potential outcomes framework, it is important for the intervention to be independent of (exogenous to) the outcome (restating the randomization assumption from the previous module). However, actual treatment status (participation) is endogenous. One interpretation of this endogeneity is that the treatment status and the regression model error term (that is, the regression residual, after differencing out the control variables) are correlated, and thus our inference will be biased. It is as if there are *confounders* in the error term (unmeasured or omitted variables) that could explain the outcomes in part but also correlated with actual treatment, such that the effect we estimate is not a pure effect of actual treatment.

To consistently estimate the causal effect of the endogenous variable (here, the actual participation in

the treatment which is a "choice") we can use instrumental variables. IVs are correlated with the actual treatment but not correlated with the measured or unmeasured confounders. In effect, we replace an endogenous treatment variable with exogenous IVs.

Figure 5 depicts the concept of instrumental variables. Part A shows that participation in the program is endogenous and determined on basis of randomized allocation to program (this can be purposive selection in the program as well, but identifying and measuring a valid instrument may be more challenging in that case), a few exogenous factors and confounders which can determine the participation as well as affect the outcome of interest.

As shown in Part B, the IV is strongly correlated with program participation but not the confounders, so that by including the IV instead of endogenous treatment (program participation) we can estimate unbiased TOT effects.

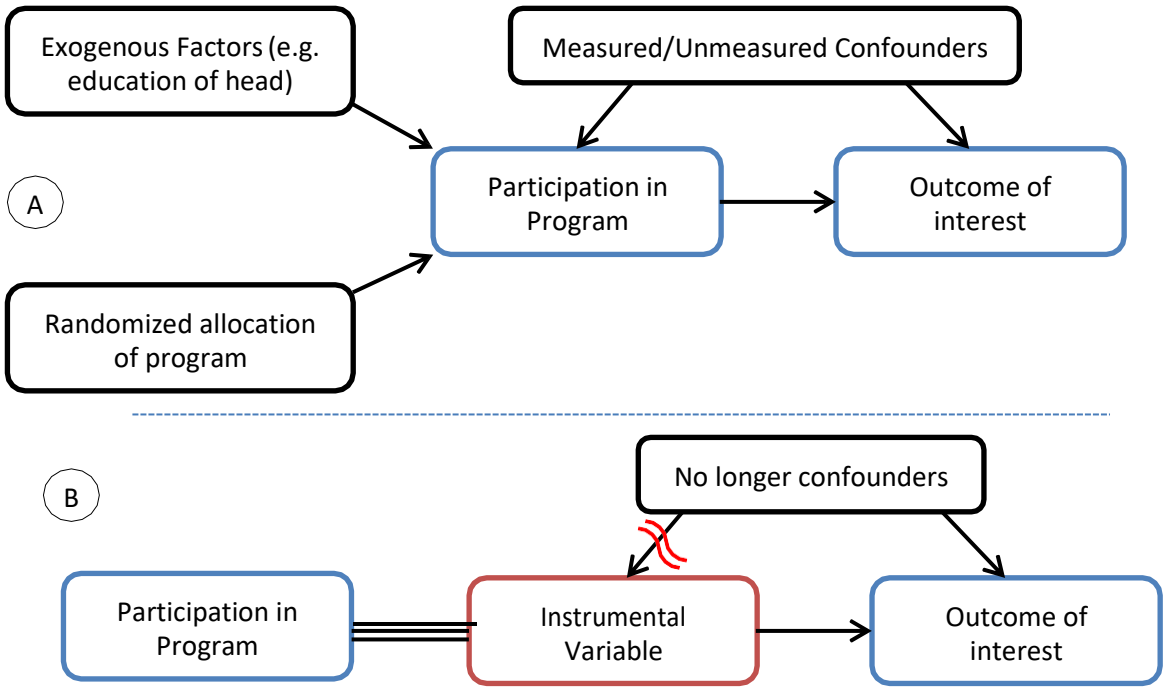

Figure 5. Use of IV to remove the confounding caused by endogenous program participation: (a) presence of confounding and (b) removing confounding bias.

Which is the best IV in the case of a randomized experiment with poor compliance or participation in the treatment group to estimate the TOT effects? We know that the assignment was random and participation in the actual intervention is highly dependent on such assignment, but the outcome is not dependent on the randomized allocation. Therefore, in our example dataset, the random assignment (`D`) itself can be used as an IV for the participation in intervention (`D_HH`). The IVs are typically used in a 2-stage least square models (hence the name IV/2SLS) stated as:

$$\textbf{\textit{Stage 1}}: D\_HH_{ij} = \beta_0 + \beta_1 . D_j + \varepsilon_{ij}$$

$$\textbf{\textit{Stage 2}}: Y_{ij} = \beta_0 + \beta_1 . \hat{D}\_HH_{ij} + \varepsilon_{ij}$$

where i indicates the individual or household and j indicates the unit of randomization (in this case the village).

The predicted `D_HH` participation estimates from stage one as used on the right-hand side in stage two to estimate the consistent causal effect of `D_HH` or participation in PROGRESA on the outcome of interest Y_{ij}. Stage 1 uses IVs to predict participation, and such predicted participation (the actual treatment of interest) is also exogenous. Stage 2 estimates the TOT effects.

Exercise 5.2: In R, specify a 2SLS model to estimate the ATET or TOT effects of PROGRESA participation on household income levels in 1999. The IV for `D_HH` should be `D`. Please read the help for command ivreg. Now use D and pov_HH as IVs for D_HH and estimate the TOT/ATET. Are the results same or different? Why?

3.3 Local Average Treatment Effect (LATE)

So far, we have discussed a case in which there is imperfect compliance or participation in the intervention in the treatment group. We have shown that ATET or TOT effects can be estimated manually by dividing the ITT by the "participating" fraction of population in the treatment group. We also demonstrated an IV/2SLS method to estimate causal effects of the treatment. When the control group does not comply with the protocol—that is, when some control households also opt into the intervention—even then we can consistently estimate the ATET/TOT effect. However, there is a subtle difference in interpretation. The estimated causal effects cannot be interpreted at the population level but only among the sub-population – the compliers (those who will do exactly what they are supposed to as per the protocol for the treatment and control groups). This effect is thus distinguished as Local Average Treatment Effect (LATE). The LATE can be estimated theoretically as:

$$I_c = \frac{ITT}{\delta_{c,trt} - \delta_{n,ctr}}$$

where $\delta_{c,trt}$ indicates the proportion of people from the treatment group who were compliers (participated in the program) and $\delta_{c,trt}$ indicates the non-compliers from control group who participated in the program (when they were not supposed to).

4. SPILLOVER EFFECTS

Externality: Externality is a very useful economic concept, and is known by other names (like "spillover effect") in other fields. Externality is the cost or benefit of an event to an individual, household, or any other entity that did not directly or indirectly "chose" to experience the event. For example, a polluting factory may impose a negative externality on nearby villages by polluting their air and water.

The community may not even receive any benefit from the economic activities of the industrial plant. Externalities can be positive as well; for example, when an individual is immunized against a communicable disease, her community-members will thereby be less likely to receive the disease (since they won't get it from her). Similarly, an intervention can have "externalities or spillovers"

which are unintended effects (good or bad) of the intervention on the people/households not directly intervened upon.

SUTVA (the violation of stable unit value assumption; see above) is an assumption required for the unbiased estimation of causal effects using either the OLS or IV/2SLS methods. However, this assumption does not hold if the treatment affects the control group as well as the treatment group. For example, PROGRESA is a conditional cash transfer program that was randomly assigned to some villages. Within the treatment villages, all "eligible poor households" were offered the program. The PROGRESA households were required to keep their children in school to receive the second tranche of conditional cash transfers. Suppose we consider the non-participating households as the controls because they were from the same village and socio-economically similar. Arguably, they do differ from the participating households because of their choice not to participate in PROGRESA, but let's ignore this for sake of demonstration. It is possible that the 'control' households are influenced by the treatment of children through participating households and decide that their children also deserve better educations. The control households may thus decide to send their children to school, in part due to the treatment. That is, the program had within-village positive externalities. This logic can be extended to non-eligible households who we consider controls (again, we recognize that they are not theoretically strong controls, but it is useful to temporarily overlook this fact for the purpose of explaining externalities). Even these control households can decide to enroll their children in schools as a result of the treatment. We can extend this argument even further and say that the people from treatment villages may discuss PROGRESA with their relatives and friends from the control villages, and some of the households from the control villages might also enroll their children in school!

What will be the effect of this spillover on estimated ITT effects? Because we subtract the outcome in the control group form the outcome in the treatment group, we will find that the ITT estimates actually underestimate the true ITT.

It is best to deal with violations of SUTVA at the study design and implementation stages. For example, the use of placebos (so that the study participants do not know whether they are in the treatment or control group) is commonly used in epidemiological research (called blinded trials). We can also ensure some geographical separation between the treatment and control groups in order to reduce externalities. On the other hand, we may be interested in directly estimating the magnitude of the externalities.

In general, the following comparisons can be made in the presence of externalities:

- ✓ TOT or ATET effect: Compare the treated (participating) households/individuals with untreated but eligible households/individuals from the control villages. We need to ensure that these two groups are "exchangeable" to the extent feasible.
- ✓ Spillover effect: Compare the outcome in the ineligible households/individuals from the treatment villages with ineligible households/individuals from the control villages.

There are specific regression analysis methods to estimate the spillover effect or ATET/TOT effects when faced with spillover, and each of them essentially estimates these two steps. The class lectures may demonstrate some of these applications.

5. ATTRITION

Attrition is the loss of observations or study participants over time. Consider our typical study design: (a) we select participants for the study; (b) we randomize them into treatment groups; (c) we conduct a baseline survey; (d) we conduct endline or follow up survey of the study participants; and (e) we compare the outcomes in treatment and control groups. What happens when the study participants are not present in the follow-up survey?

The central assumption behind inferring causal effect is that the treatment and control groups are exchangeable except for the treatment itself. If after attrition exchangeability can be still maintained (which can be examined through comparisons of non-attrition baseline statistics), then we don't have a problem other than the unavoidable reduction in sample size, resulting in a loss of "statistical power" to detect impacts.

However, if attrition results in the groups being different at the baseline, then the advantages of baseline balance or exchangeability is lost in the follow-up. For example, if poorer households start migrating into the treatment villages in order to receive the conditional cash transfers under PROGRESA, then such immigration has changed to socio-economic composition of the treatment villages and they are no longer comparable to the control villages; the mean outcomes in the treatment village could appear worse due to an influx of lower-income poorer-health households, therefore making the results of the intervention appear worse than they actually were. Or, it may happen that because of PROGRESA, emigration in treatment villages is reduced among households who participate in PROGRESA while it continues in control villages, such that by the time of follow-up we do not have a comparable group of eligible yet controlled households in control villages.

Overall, when attrition is "**differential**" by the treatment status, the estimated causal effect can be biased; this is a form of selection bias, with certain participants selecting out of the sample. This attrition can be related to the outcome (such as children from treatment villages migrate out for higher education in the long term), but that does not necessarily bias the causal effects. We check the effect of attrition by comparing the measured confounders and covariates at the baseline between the treatment and control groups for the sample lost to follow-up and the sample that remains intact.

There is no perfect method, but below are some suggested options (we only provide example commands, but cannot actually estimate them because we don't have any attrition-related variable in our data).

- ✓ Regression of the attrition status of a household/individual on the treatment indicator. For example, `lm(attrition~D, data = PanelPROGRESA_97_99year)`. We can add other covariates we believe can explain attrition to this regression model as, `lm( attrition~D+Income_Baseline+pov_HH_baseline+HHedu_baseline)`

- ✓ Group mean test for several covariates to assess if the treatment and control group remain balanced in intact and lost panel. For example, `t.test(Income_Baseline pov_HH_baseline ... HHedu_baseline)` when attrition == 0, grouped by(D). Note, we always use baseline variables because covariates and confounders may differentially change as a result of treatment.

- ✓ A good field survey instrument will list the reasons for attrition. A simple frequency table of the reason for attrition (migration, death, locked house, etc.) by the treatment and control

group can be compelling, especially if the predominant reasons are not related to the treatment itself.

There are a series of "reweighting" methods to adjust estimation of causal effects by accounting for attrition. Basically, these algorithms give more weight to observations remaining in the sample that are similar to those who are lost in order to make up for their attrition. However, we will not cover these methods in detail in this course.

6. BIBLIOGRAPHY/FURTHER READINGS

1. Angrist, J. D. and A. Krueger (2001). "Instrumental Variables and the Search for Identification: From Supply and Demand to Natural Experiments", *Journal of Economic Perspectives, 15(4)*.
2. Angrist, J. D., G. W. Imbens and D. B. Rubin (1996). "Identification of Causal Effects Using Instrumental Variables", *Journal of the American Statistical Association,* Vol. 91, 434.
3. Bradlow, E., (1998). "Encouragement Designs: An Approach to Self-Selected Samples in an Experimental Design", *Marketing Letters,* 9(4)
4. Gertler, Paul J., Sebastian Martinez, Patrick Premand, Laura B. Rawlings, and Christel MJ Vermeersch. "Impact evaluation in practice." World Bank Publications, 2011.
5. Holland, Paul W. "Statistics and causal inference." *Journal of the American Statistical Association* 81.396 (1986): 945-960.
6. Imbens, G. W. and J. D. Angrist, (1994). "Identification and Estimation of Local Average Treatment Effects." *Econometrica,* 62(2).
7. Rubin, Donald B. "Estimating causal effects of treatments in randomized and nonrandomized studies." *Journal of Educational Psychology* 66.5 (1974): 688.