Introduction to Econometrics: Recitation 10

Seung-hun Lee[*]

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1 Big Data

Big data can mean so many things: it can simply mean data with large observations or large number of control variables. In general instances, atypical data such as text data, and satellite imagery are referred to as big data. In this class, we focus on the instance where there are many control variables, specifically on what to do if there are many control variables relative to the number of observations. Additionally, we focus on trying to get the best way to predict a result that is currently not in the dataset.

1.1 Prediction with many X's: Why we don't want too many X's

In this section, we will focus on the issue of **shrinkage**. I will introduce Ridge estimation, LASSO estimation, and principal component method. Before introducing these models, I will talk about the regression framework that will be used throughout the discussion

$$Y_i^* = \beta_1 X_{1i}^* + ... + \beta_k X_{ki}^* + u_i^*$$

where X_{1i}^* is the "standardized" version of X_{1i}'s, defined as

$$X_{1i}^* = \frac{X_{1i} - \bar{X}_1}{\sigma_{X_1}}$$

and we define Y_i^* as $Y_i - \bar{Y}$ - the "demeaned" version. Note that we CANNOT have a constant term β_0 here because if we demean β_0, which is the same for all i's, they vanish.

[*] Contact me at sl4436@columbia.edu if you spot any errors or have suggestions on improving this note.

We then define the mean squared prediction error, or MSPE as the expected value of the squared error made by predicting Y for an observation not in the dataset. In maths,

$$MSPE = E[Y^{OS} - \hat{Y}^{OS}]^2$$

where the $\hat{Y}^{OS}$ is obtained from the coefficients of β's made from the in-sample prediction

$$\hat{Y}_i^{OS} = \hat{\beta}_1 X_{1i}^{OS} + ... + \hat{\beta}_k X_{ki}^{OS}$$

and the Y^{OS} is the realized value of Y outside of the sample

$$Y_i^{OS} = \beta_1 X_{1i}^{OS} + ... + \beta_k X_{ki}^{OS} + u_i^{OS}$$

With these notations, we can define the prediction error as

$$Y_i^{OS} - \hat{Y}_i^{OS} = (\beta_1 - \hat{\beta}_1) X_{1i}^{OS} + ... + (\beta_k - \hat{\beta}_k) X_{ki}^{OS} + u_i^{OS}$$

Define $\sigma_u^2 = E[u_i^{OS}]^2$, then we can write MSPE as

$$MSPE = \sigma_u^2 + E[(\beta_1 - \hat{\beta}_1) X_{1i}^{OS} + ... + (\beta_k - \hat{\beta}_k) X_{ki}^{OS}]$$

Ideally, the smallest possible MSPE would be σ_u^2, referred to as the **oracle prediction**. Which happens if your predicted $\hat{\beta}$'s match the actual β. Unfortunately, we cannot predict β's perfectly. and the more predictors we have, we generally end up having larger MSPE. As such having a large set of X's will not be a good idea (Thus, the **problem of many predictors**). This is where Ridge, LASSO, and Principal Component method comes in.

1.2 Big data methods

The goal here is to find other estimators that does not increase the MSPE compared to the rate in which the same rises in OLS estimators. The idea here is to reduce the σ_u^2, the variance from the residual sums of squares, at the expense of introducing a small bit of bias. We do this by providing a penalty for having a model with large number of regressors (what we formally call 'shrinkage'). All of the methods I introduced above involve these tricks.

1.3 Ridge regression

The Ridge regression minimizes a 'penalized' sum squared of residuals in the following sense

$$\hat{\beta}_{Ridge} = \arg\min_{\beta_1,..,\beta_k} \left[\sum_{i=1}^{n} (Y_i - \beta_1 X_{1i} - .... - \beta_k X_{ki})^2 + \lambda_{Ridge} \sum_{j=1}^{k} \beta_j^2 \right]$$

Here, the last term on the right hand side is the penalty we are introducing. Note that for any term in which β_j is not zero, the penalty term is also nonzero. Also note that we are introducing some degree of bias (compared to OLS). However, the gain is that the variance term σ_u^2 will be reduced. Lastly, you need to capture that the variance and the bias that determines the MSPE moves in a trade-off relation - If you want to decrease one component, you increase the other (but not at a 1-for-1 rate). The Ridge estimator that comes from here minimizes MSPE by working through reducing the variance term to the extent that the bias term does not rise too drastically.

1.4 LASSO regression

LASSO regression also minimizes a penalized sum squared residuals. The difference is that the penalty term takes a different form:

$$\hat{\beta}_{LASSO} = \arg\min_{\beta_1,...,\beta_k} \left[\sum_{i=1}^{n} (Y_i - \beta_1 X_{1i} - .... - \beta_k X_{ki})^2 + \lambda_{LASSO} \sum_{j=1}^{k} |\beta_j| \right]$$

In essence, the idea is similar with the Ridge regression - you find an estimator that minimizes MSPE by reducing σ_u^2 at the expense of some amount of increase in the bias.

The difference between the two lies in the degree of shrinkage. The difference is stark when the OLS estimates are virtually close to zero. When the OLS estimates are small, the LASSO shrinks those estimates all the way to 0. While Ridge also shrinks those coefficients close to 0, they do not exactly set them to 0.

1.5 Principal component

Say that you have a giant k number of regressors. In the principal component method, you are using a linear combination of some subset of k variables. At the end, you end up using $p < k$ number of regressors and effectively 'collapsing' the model to a smaller dimension.

So how do you determine the right linear combinations? For a two-variable case, the linear combination looks something like

$$aX_1 + bX_2$$

where a, b are some real numbers. The optimal value of a and b is determined by

$$\max var(aX_1 + bX_2) \text{ s.t. } a^2 + b^2 = 1$$

Note that we want to solve the *maximization* problem in this case as we want the X's to explain more of the variation that we observe in the data. You can understand $a^2 + b^2 = 1$ as a regularization method.

So for a general case, you would be solving the following problem to get the j'th principal component PC_j

$$\max var\left(\sum_{i=1}^{K} a_{ji}X_i\right) \text{ s.t. } \sum_{i=1}^{k} a_{ji}^2 = 1$$

with another condition being that $corr(PC_j, PC_{j-1}) = 0$. This additional condition is here because we want to minimize the overlapping amount of information across different principal components.

So we still need to determine how many principal component to use. Scree plot gives you an idea about how much each new principal component adds to the variation that is explained by additional principal component. However, this does not give rigorous cutoff. For formal approach, m-fold cross validation approach can be used. You split the sample into m subsets of equal size. Then, one of them becomes your 'test' sample and the rest becomes an out-sample. You will derive a first estimate of MSPE. Repeat this for each of the m subsets until you get m estimates of MSPE. The right number of principal components minimizes the averages of these MSPEs. Note that determining the λ's in LASSO and Ridge can also be done by using the m-fold cross validation.

2 Economics of Experiments

2.1 Motivation

In previous chapters, we have learned that one way to control for many internal validity threats - such as selection bias, omitted variable bias - is an **ideal randomized controlled experiments**. This is where you categorize some individuals under treatment group and controlled group and run various tests as if you are in a laboratory experiment. While there are some degrees of randomized controlled experiments (not necessarily ideal, in the sense that subjects were not brought to the lab), this is difficult to conduct, due to logistical, financial, and ethical reasons. The alternative is to use a **quasi-experiment** or **natural experiments**. This is where there exists an exogenous event (like natural disasters, policy change etc) that affect some groups of the population differently than the others. To see the impact of the said event, we need to be able to compared the affected group (treatment) and the unaffected group (controlled) across different time periods.

So what is the value in studying experiments? For one thing, they provide a conceptual benchmark for assessing observational studies. In particular, experiment is one of the ways to see how economic theories can be applied in reality. More importantly, unlike surveys, they are free from various internal validity threats. Surveys suffer from selection bias - if people don't respond to surveys anymore and they are different from those who respond, we fail to obtain a representative sample. However, experiments suffer from their own validity threats.

Example of randomized controlled experiments

- *What are the barriers to technology adoption?* This is from a research of Prof. Eric Verhoogen of Columbia (with some co-authors)[a]. This team of researchers developed a new cutting technology that reduces waste of primary raw material involved in making soccer balls. Initially it gave a random subset of Pakistani producers an access to the technology. The take-up rate was very low, for surprising reasons. Then, they noticed that the employees get paid according to the number of soccer balls made, not taking into account the waste of resources involved. Since the new technology slowed them down initially, employees were reluctant to accept new technology. Then, the team conducted another experiment within the firms that had the access to the technology. Within each firm, one cutter and one printer received a lump-sum payment equivalent to monthly earnings. However, there is a string attached - each worker must be able to use the technology in presence of their owner. As a result, employees accepted the new technology more.

Example of natural experiments

- *What are the impacts of prenatal exposure to radioactive fallout?* This is from Prof. Douglas Almond and Prof. Lena Edlund's research[b]. They notice that different regions in Sweden were exposed to different levels of radioactive fallout as a result of Chernobyl disaster (the levels of fallout were generally considered physically harmless). Using this exogenous variation in radioactive fallout, they see whether exposure to such fallout has impacts on childhood cognitive abilities. The experiment reveals that those exposed more to the fallout performed worse in secondary school, mathematics in particular.

[a] Atkin, D., A. Chaudhry, S. Chaudry, A. K. Khandelwal, E. Verhoogen (2017), Organizational Barriers to Technology Adoption: Evidence from Soccer-Ball Producers in Pakistan, *The Quarterly Journal of Economics*, 132(3): 1101–1164

[b] D. Almond, L. Edlund, M. Palme (2009), Chernobyl's Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden, *The Quarterly Journal of Economics*, 124(4): 1729–1772

2.2 Potential outcomes framework

In class, we have discussed the possibility that the effect of treatment can differ depending on an individual (and hence the β_{1i}) notation. This is more realistic but also more difficult to address. So I will start by assuming that the treatment effect is identical for everyone (**constant treatment effect** assumption).

The framework, in its most simplest form, is as follows: Let $Y_{i,t}$ be the **observed** outcome variable for individual i at time t, X_{it} be the treatment variable: It is 1 if individual i is treated and 0 if otherwise. In equation form, we can write

$$Y_{it} = \beta_0 + \beta_1 X_{it} + u_{it}$$

Now we define a **potential outcome framework**, which is a key in understanding how to identify treatment effects in regressions. Let $Y_{it}(0)$ denote a potential outcome if subject i is not treated at time t and $Y_{it}(1)$ be the same if i is treated. Then Y_{it}, the observed outcome for individual i at time t, can be split into

$$\begin{aligned} Y_{it} &= Y_{it}(1)X_{it} + Y_{it}(0)(1 - X_{it}) \\ &= Y_{it}(0) + (Y_{it}(1) - Y_{it}(0))X_{it} \end{aligned}$$

This equation captures so many things. For one, we always see what Y_{it} is whether i is treated or not. However, we can only observe at most one of $Y_{it}(1)$ and $Y_{it}(0)$. This is because the very same individual i cannot be treated and untreated at the same time period t. This leads to what we call **fundamental problem of missing data**. For us to make any rigorous statements about the treatment effect, we need to be sure about what the missing outcome looks like. This is where perfect randomization, randomization conditional on observables, using instrumental variables on treatment variable X_{it} comes in.

Second, this framework (the second line in the equation above) gives hints as to how we can identify the treatment effect in a regression. β_1 itself would be enough to identify treatment effects. Even if we allow treatment effects to differ, we can obtain **average treatment effect**, which can be written as

$$ATE = E[Y_{it}(1) - Y_{it}(0)] = \frac{1}{N}\sum_{i=1}^{N}(Y_{it}(1) - Y_{it}(0))$$

which is related to the terms in front of the X_{it} variable. It gives us the hint that ATE can be obtained from estimating the right coefficient in the regression.

2.3 Average treatment effect under perfect randomization

We will now derive the average treatment effect in a regression. To start with a simple thought experiment, we will assume that the experiment is **perfectly randomized** - that the treated individuals and controlled individuals are identical except for treatment status, or that $E[u_{it}|X_{it}] = 0$ for all possible X_{it} values. In addition, let's assume constant treatment effect for now. Let's use this regression framework along with the potential outcome framework:

$$Y_{it} = \beta_0 + \beta_1 X_{it} + u_{it}$$

We now derive the expected value of Y_{it} separately for the treated and controlled individuals. For the controlled ($X_{it} = 0$, so that $Y_{it} = Y_{it}(0)$):

$$E[Y_{it}|X_{it} = 0] = E[Y_{it}(0)] = \beta_0$$

and for the treated, ($X_{it} = 1$, so that $Y_{it} = Y_{it}(1)$)

$$E[Y_{it}|X_{it} = 1] = E[Y_{it}(1)] = \beta_0 + \beta_1$$

Then, the average treatment effect can be characterized as

$$ATE = E[Y_{it}(1) - Y_{it}(0)] = (\beta_0 + \beta_1) - \beta_0 = \beta_1$$

where subtraction among $E[Y_{it}|X_{it} = 1]$ and $E[Y_{it}|X_{it} = 0]$ is possible since we are assuming perfect randomization. So under the current circumstances, identifying the average treatment effect is equivalent to obtaining the β_1 coefficient through an OLS process.

2.4 Validity threats: Unconfoundedness

However, having a perfectly randomized sample is very bold assumption and a rare circumstance. Like in general cases, there are many threats to the validity of our estimation results. To start with, we cannot automatically subtract $E[Y_{it}|X_{it} = 1]$ and $E[Y_{it}|X_{it} = 0]$ under **imperfect randomization**. Moreover, if the **attrition rate is nonrandom** - if it differs by a treatment status - we end up with a biased estimate of the treatment effect. In terms of experimental procedure, **failure to comply to experiment protocol** and other **experimental effects** that rises through peculiar behaviors of the experimental and the experiment subject could also serve as a validity threat. Additionally, constant treatment effect assumption that we have imposed on ourselves may not be accurate. There are external validity threats when the sample is not representative, is not replicable in other settings, and the results may have general equilibrium effects.

Some ways to address these problems are as follows: If the treated and the controlled differ in some observed characteristics, we can simply include control variables W_{it}. In that case, we would be observing the average treatment effect conditional on the combination of values observed through W_{it}. This would also allow us to identify treatment effects for people with different treatment effects as well.

For this, we need the following two conditions to hold

Unconfoundedness assumptions (or selection on observables in some books)

- Unconfoundedness: Conditional on W_i, the pair of counterfactuals $(Y_i(1), Y_i(0))$ is independent of X_i

  $$(Y_i(1), Y_i(0)) \perp X_i | W_i$$

  This applies to the case where probability of being treated is equal given some level of observables, although it may differ across observables.
  For random assignment, we have $(Y_i(1), Y_i(0)) \perp X_i$

- Overlap: For all values of w in the control variable W_i,

  $$0 < \Pr(X_i = 1 | W_i = w) = p(W_i = w) < 1$$

  or that each unit with w has a nonzero chance to be treated and non-treated. Here, $p(W_i = w)$ is commonly referred to as propensity score.

With the above assumptions, we can identify the average treatment effect as follows. The method we will use is called inverse probability weighting, and it will yield estimable form of ATE. The following is true based on unconfoundedness assumption

$$\begin{aligned}
E\left[\frac{X_i Y_i}{p(W_i)}\right] &= E\left[E\left[\frac{X_i Y_i(1)}{p(W_i)}|W_i\right]\right] \\
&= E\left[\frac{E[X_i|W_i]E[Y_i(1)|W_i]}{p(W_i)}\right] \\
&= E\left[\frac{p(W_i)E[Y_i(1)|W_i]}{p(W_i)}\right] = E[E[Y_i(1)|W_i]] = E[Y_i(1)]
\end{aligned}$$

Similarly,

$$E\left[\frac{(1 - X_i)Y_i}{1 - p(W_i)}\right] = E[Y_i(0)]$$

Thus, we can obtain the ATE by differencing the two, which gets us

$$E[Y_i(1) - Y_i(0)] = E\left[\frac{(X_i - p(W_i))Y_i}{p(W_i)(1 - p(W_i))}\right]$$

In terms of estimation, coming up with the sample analogue of this works. However, you need to separately estimate the propensity scores using logit/probit regression with X_i on the left and W_i on the right hand side.

We can do similarly for the ATT as well. The end result is that

$$E[Y_i(1) - Y_i(0)|X_i = 1] = E\left[\frac{(X_i - p(W_i))Y_i}{\rho(1 - p(W_i))}\right]$$

where $\rho = \Pr(X_i = 1)$ is the overall probability of treatment (not controlling for observables). The averaging is only done among those who are treated. Sample analogues could be used for estimation. For further results, consult Dehejia (1997).

2.5 Validity threats: Local average treatment effect

There is also an instrumental variable approach: If there exists a Z_{it} variable that influences the treatment status X_{it} and uncorrelated with u_{it}, we can use Z_{it} to instrument X_{it} and run 2SLS regression. The interpretation becomes slightly different, however. The predicted value of X_{it} used in the second stage represents the probability of being treated as according to Z_{it}. Therefore, 2SLS estimation estimates the causal effect for those whose value of X_{it} is influenced by Z_{it} and puts higher weight on those who is more likely to enter treatment. In effect, what we are doing is to measure the treatment effect 'localized' for those whose probability of treatment is most influenced by X_{it} with Z_{it} as an instrument. This is what **localized average treatment effect** (LATE) is about.

So how do you use IV in an experimental framework? Let Z_{it} be an instrument for X_{it}. Then the usual two stage least squares method can be applied in this manner

$$X_{it} = \pi_0 + \pi_{1i} Z_{it} + e_{it} \text{ (First stage)}$$
$$Y_{it} = \beta_0 + \beta_{1i} X_{it} + u_{it} \text{ (Equation of interst)}$$

When we are at the first stage, we can obtain predicted $\hat{X}_{it} = \hat{\pi}_0 + \hat{\pi}_{1i} X_{it}$. Given that X_{it} is binary, (recall from linear probabilities model), the first stage gets us the predicted probability of being treated. As we did with the typical two stage least squares, we put $\hat{X}_{it}$ in place of X_{it} in the equation of interest. Therefore, it puts more weight on those that are likely to be treated. Then it allows us to measure the causal effect for those whose predicted probability of treatment is heavily influenced by Z_{it} - this is called **local average treatment effect**. If

β_{1i}, π_{1i} are independent of $(u_{it}, v_{it}.Z_{it})$, $E(\pi_{1i}) \neq 0$, then we can get that

$$\hat{\beta}_1 \xrightarrow{p} \frac{E(\beta_{1i}\pi_{1i})}{E(\pi_{1i})}$$

Using the fact that $E(\beta_{1i}\pi_{1i}) = E(\beta_{1i})E(\pi_{1i}) + cov(\beta_{1i}, \pi_{1i})$, $\hat{\beta}_1$ can be written as

$$\hat{\beta}_1 \xrightarrow{p} E[\beta_{1i}] + \frac{cov(\beta_{1i}, \pi_{1i})}{E(\pi_{1i})} = \beta_1 + \frac{cov(\beta_{1i}, \pi_{1i})}{E(\pi_{1i})}$$

In this setting, treatment effect is large for individuals for whom the effect of the instrument is large.

2.6 Some words on Diff-in-Diffs and RD

The idea in using the **difference-in-difference**, (hereafter DD) estimator is to see the differences in the pre- and post-treatment outcomes for Y for treatment and control groups. Specifically, it estimates the treatment effect as

$$\hat{\beta}_1^{DD} = [\bar{Y}_{\text{treat,after}} - \bar{Y}_{\text{treat,before}}] - [\bar{Y}_{\text{control,after}} - \bar{Y}_{\text{control,before}}]$$

In a regression form, what we can get is as follows (Assume away W_i's for the moment). Suppose we have two time periods, one for before treatment and the other for after treatment. We can write

$$\Delta Y_i = (Y_{i,\text{after}} - Y_{i,\text{before}}) = \beta_0 + \beta_1 X_i + u_i$$

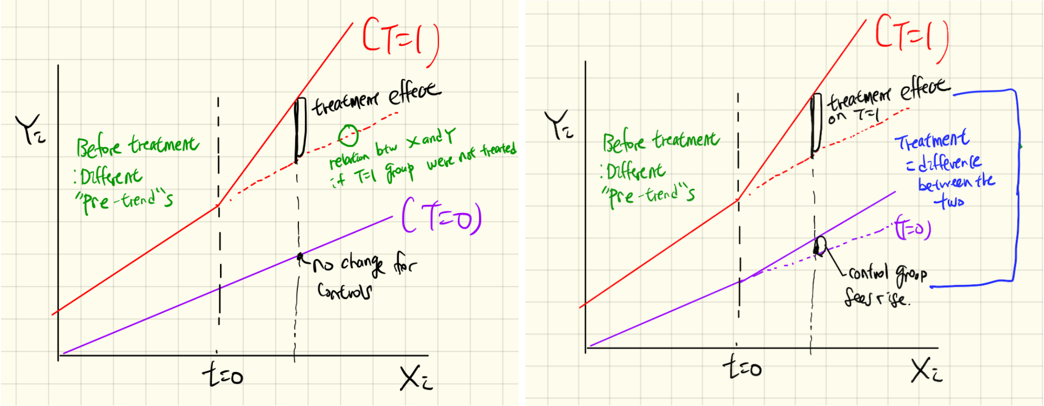
For those who are not treated, $E[\Delta Y_i|X_i = 0] = \beta_0$. For those treated, $E[\Delta Y_i|X_i = 1] = \beta_0 + \beta_1$. These two parameters represent the 'difference' within treatment and control groups. By taking the 'difference' between treatment and control groups, we are left with β_1. In other words

$$\beta_1 = E[\Delta Y_i|X_i = 1] - E[\Delta Y_i|X_i = 0]$$

So by taking the 'difference' (between treatment and control groups) of the 'difference' (changes over time within each group), we get the DD estimator (and this is where DD estimator gets its name).

The following picture should make things clear. In the following figure, $T = 1$ indicates the treatment group, $T = 0$ is the control group

Figure 1: DD estimator



(a) No change in control group

(b) Some change in control group

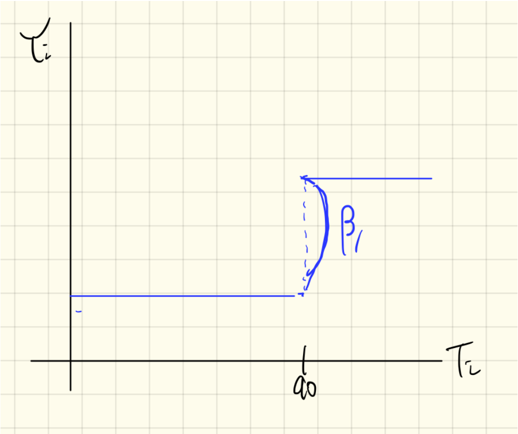
In words, the treatment effect measured by the DD estimator is the treatment effect on treated groups *on top of* effect on control groups.

Regression discontinuity design (or RD) applies to the case where the treatment status depends on whether individual i has passed a threshold. Suppose that there is a variable T_i, like a test score, for instance. Let's assume that there exists a program for gifted students that only accept those whose test score is higher than 90. In this case, $X_i = 1$ iff $T_i > 90$ and $X_i = 0$ if otherwise. In equation,

$$Y_i = \beta_0 + \beta_1 X_i + u_i$$

and the following figure captures the treatment effect in an RD setup.

Figure 2: RD treatment effects



For those not in the program, $E[Y_i|X_i = 0] = \beta_0$ and $E[Y_i|X_i = 1] = \beta_0 + \beta_1$. This implies that if the direct effect of T on Y is continuous in the absence of the treatment, the effect of treatment should show up as a jump of size β_1 at the threshold value of T_i, 90 in our case. Similar to what we have in our figure

To conduct RD rigorously, many things should be satisfied. Ideally, RD is most accurate in settings where subjects are not different in other traits (especially if we are including control variables W_i). In order to achieve this, we usually limit the subjects to those around certain bandwidth of the cutoff value of T_i. Therefore, it may induce a loss of sample issue. (This is actually an advanced topic)

For now, I assumed that everyone whose $T_i > 90$ is accepted. This is what is called **sharp RD**. However, this may not always be the case in reality. There might be exceptions - those whose test score are higher than 90 may not be part of the program and those whose score is under 90 might be part of the program. If this is the case and we carry out an RD here, we are conducting a **fuzzy RD**. The estimation in the case of fuzzy RD is tricky and quite advanced. So the takeaway here is that the accuracy of RD is susceptible to the compliance (of the program) issue. If the subjects are not fully complaint to the intentions of the program, accuracy of the RD is not fully guaranteed.