

EC402 - Problem Set 4

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18th of February 2009

Introduction

Today we will talk about what is a suitable **control group** in non-experimental settings.

Depending on the data and our level of comfort regarding unobservables we can then apply

- **difference-in-difference** estimation or
- (propensity-score) **matching methods**

and we will see how these can go wrong.

Introduction

Why a control group?

Suppose we are interested in the treatment effect on the treated (TOT). If $\tau_i = Y_{i1} - Y_{i0}$ is the treatment effect for individual i then TOT is

$$E[\tau_i | T_i = 1] = E[Y_{i1} | T_i = 1] - E[Y_{i0} | T_i = 1]$$

The trouble is that we never observe $Y_{i0} | T_i = 1$!

That is why we search for a group that *is not treated and behaves the same way as the treated group would have behaved in the absence of treatment*, a **control group**.

Introduction

Why a control group?

So, if we need a control group, why do we not just take experimental data?

- Often experiments might not be possible.
- Or collecting data for a control group is too expensive.

Question 1

The Difference-in-Difference idea

Suppose we have data on the outcome we are interested in for two groups both before and after some treatment occurred.

- *Group 1* was actually treated.
- For some untreated group, *group 0*, we believe that the **trend of its outcome data** is the same as the trend for *group 1* would have been if *group 1* had not been treated.

Question 1

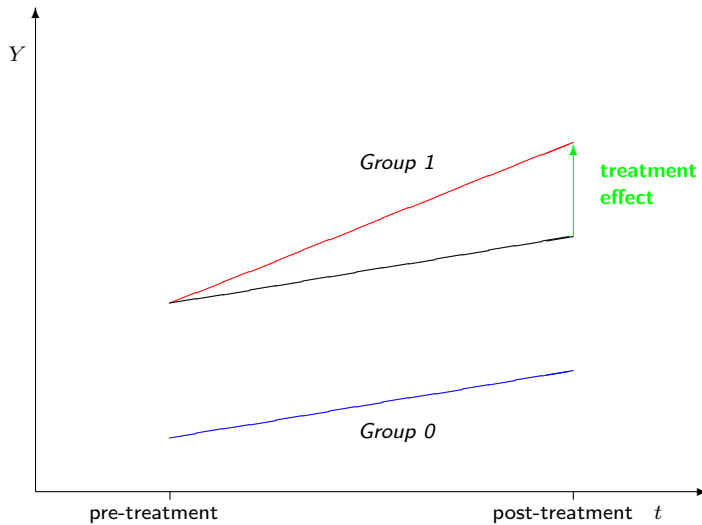
The Difference-in-Difference idea

Then we can take the actual development of the outcome for *group 0* and together with the initial value (pre-treatment) for *group 1* to calculate where *group 1* would have been, if it had not been treated, i.e. $E[Y_{i0}|T_i = 1]$.

The difference between what the outcome actually was and what it would have been in the absence of treatment is our **difference-in-difference estimate** of the treatment effect.

Question 1

The Difference-in-Difference idea



Question 1

B. Using experimental data

In our case we actually do have an experimental control group.¹ Using this we estimate the treatment effect to be

```
. reg re78 treatment
```

Source	SS	df	MS			
Model	137332501	1	137332501	Number of obs =	722	
Residual	2.8053e+10	720	38962866.3	F(1, 720) =	3.52	
Total	2.8191e+10	721	39099301.3	Prob > F =	0.0609	
				R-squared =	0.0049	
				Adj R-squared =	0.0035	
				Root MSE =	6242	

re78	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]
treatment	886.3037	472.0863	1.88	0.061	-40.52635 1813.134
_cons	5090.048	302.7826	16.81	0.000	4495.606 5684.491

886 dollars!

¹Question 1A, which I skip, shows how most observable characteristics which might influence the outcome as well are very similar in the control and treatment group. This is what we would expect under random treatment assignment. It is precisely the point of random assignment that all observable and - more importantly - unobservable characteristics are similar in both the treatment and control group! (Then, not surprisingly, including these controls does not change much in 1B.)

Question 1

D. Understanding diff-in-diff

So what went wrong when using the diff-in-diff method?

- Is the fact that many observable characteristics are different between the treatment group and the control group picked by LaLonde (1986) necessarily a problem for our diff-in-diff estimation?
- What is crucial for the diff-in-diff method to work?

Question 1

E. Understanding diff-in-diff

Obviously we never know how the treated would have developed in the absence of treatment, so we cannot test the crucial part of the **common-trend assumption**.

However, if we can show that for some periods prior to the treatment the control and treatment groups were developing similarly, this would be a convincing argument for our control group to be a good control group.

Question 1

E. Understanding diff-in-diff

In our case we have data for 1974 and 1975 (the treatment happened between 1975 and 1978).

```
. ttest bb_diff, by(treatment)
```

Two-sample t test with equal variances

Group	Obs	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]	
0	429	3152.752	280.3635	5806.977	2601.691	3703.813
1	185	563.5184	278.2071	3784.026	14.63228	1112.404
combined	614	2372.608	218.284	5408.865	1943.933	2801.284
diff		2589.234	464.4893		1677.047	3501.42

diff = mean(0) - mean(1)

Ho: diff = 0

t = 5.5744

degrees of freedom = 612

Ha: diff < 0

Pr(T < t) = 1.0000

Ha: diff != 0

Pr(|T| > |t|) = 0.0000

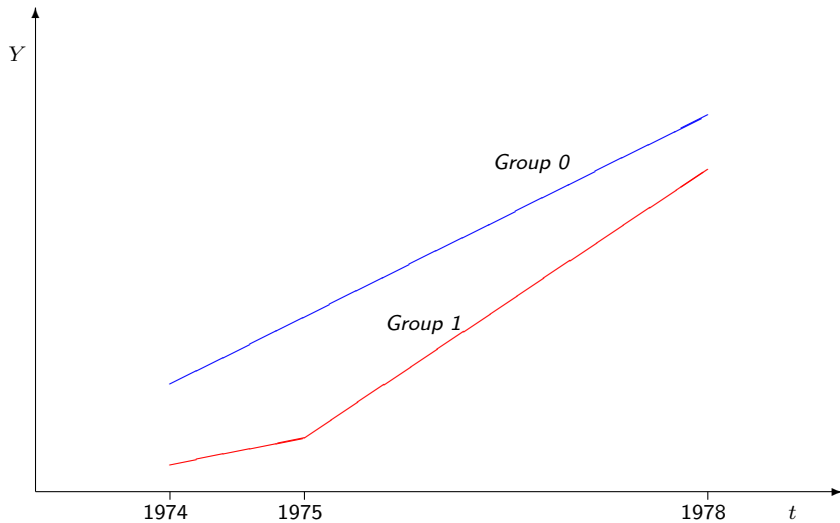
Ha: diff > 0

Pr(T > t) = 0.0000

The trends prior to treatment are in fact not at all similar!

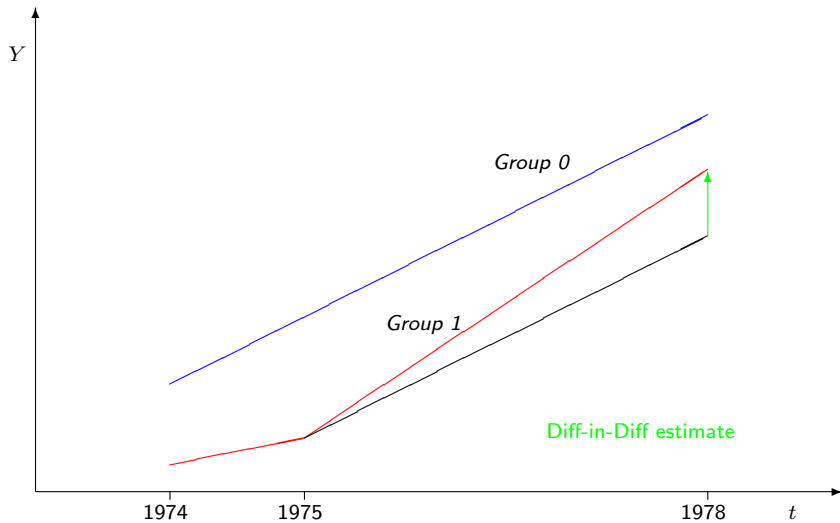
Question 1

E. Understanding diff-in-diff



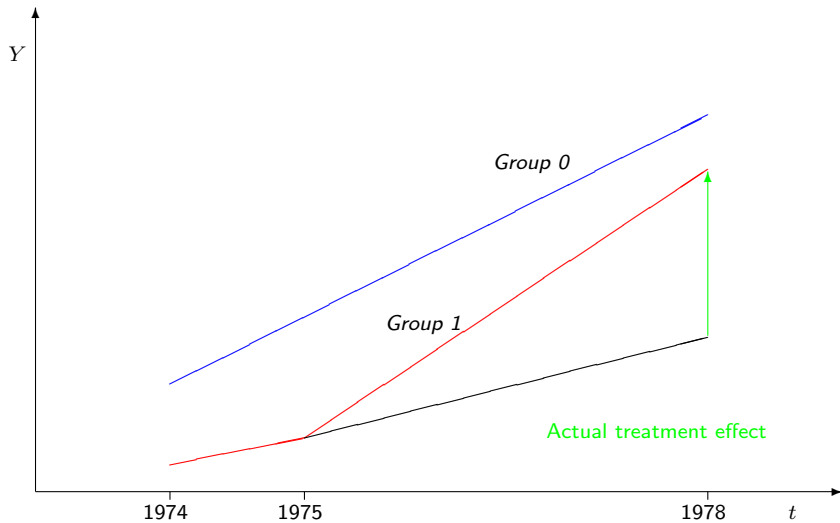
Question 1

E. Understanding diff-in-diff



Question 1

E. Understanding diff-in-diff



Question 1

Lessons

- If we have pre- and post-treatment data for treatment and control group, diff-in-diff might be a suitable method.
- Common trends assumption needs to be satisfied in the example at hand!
- We can gain some confidence about this by looking at pre- and post-treatment trends.

Question 2

Introduction

A different method to create a control group is a matching estimator. There we *match* treated observations to non-treated observations with similar observable characteristics X . **If** the treatment conditional on X_i is as good as random, comparing the outcomes for both groups gives an unbiased estimator. This assumption is called 'conditional independence assumption' (CIA).

However, if X is multidimensional this requires a lot of data - and is hence often not possible.

Question 2

Introduction

Propensity-score matching allows us to reduce the 'dimensionality of the problem': It has been proven that, rather than matching on all kind of combination of the multidimensional X , we can just match on $P(X_i) = E[T_i|X_i]$, which is one-dimensional.

If the CIA indeed holds (this is important), then this will give an unbiased estimate of the treatment effect.

Question 2

A. Implementation

Suppose we know the true propensity scores (which we never do). Then we can define

- a set of dummy variables p_m where $p_m = 1$ if an individual's propensity score $p(X)$ satisfies $m < p(X) \leq m + 0.1$ and $m \in M = 0, 0.1, 0.2, \dots, 0.9$.
- a set of interaction terms $T_i \cdot p_{m,i}$ which is 1 if the individual is in the treatment group **and** the propensity score is between m and $m + 0.1$.

We can then estimate

$$Y_i = \delta T_i + \sum_{m \in M} \gamma_m p_{m,i} + \sum_{m \in M} \beta_m (T_i \cdot p_{m,i}) + \epsilon_i$$

Intuition?

Question 2

B. Assumption

Note again:

This will only work if the Conditional Independence Assumptions (CIA) does hold!

Question 2

C. Implementation

Is the propensity score method flexible?

Question 3

A./B./C. How to estimate $P(X)$?

In practice the **propensity score** is unknown. We do know whether somebody was treated or not, but not what was the a priori probability that he will be treated.

We can try to estimate how this probability depends on X . We can use e.g.

- a **probit model** or
- a simple **linear probability model**.

Question 3

A. How to estimate $P(X)$?

If we use the **probit model** (or just **pscore** in stata) we have to drop the *nodegree* variable for the balancing property to be satisfied.

- What is the balancing property?
- When is it a problem to drop some variables, for which it is not satisfied?

Question 3

B. How to estimate $P(X)$?

How are the estimated propensity scores for the treated and untreated distributed?

p2	treatment		Total
	0	1	
0	14,333	20	14,353
.01	346	2	348
.02	215	4	219
.03	234	7	241
.04	189	7	196
.05	118	8	126
.06	54	5	59
.07	37	5	42
.08	16	0	16
.09	13	2	15
.1	21	0	21
.11	21	1	22
.12	13	2	15
.13	20	2	22
.14	20	3	23
.15	16	3	19
.16	12	3	15
.17	15	6	21
.18	18	4	22
.19	24	8	32
.2	30	13	43
.21	22	8	30
.22	40	13	53
.23	28	7	35
.24	9	6	15
.25	27	7	34
.26	12	2	14
.27	15	11	26
.28	10	6	16
.29	18	2	20
.3	14	6	20
.31	14	3	17
.32	9	4	13
.33	1	3	4
.34	1	0	1
.35	3	1	4
.36	1	1	2
.38	1	0	1
.39	1	0	1
.4	1	0	1
Total	15,992	185	16,177

Question 3

C. How to estimate $P(X)$?

If we use a **linear probability model** instead, we get

```
. reg treatment age education black hispanic married
```

Source	SS	df	MS			
Model	18.1851766	5	3.63703532		Number of obs =	16177
Residual	164.699165	16171	.010184847		F(5, 16171) =	357.10
Total	182.884342	16176	.011305906		Prob > F =	0.0000
					R-squared =	0.0994
					Adj R-squared =	0.0992
					Root MSE =	.10092

treatment	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
age	-.0002708	.000081	-3.34	0.001	-.0004296	-.0001119
education	-.0010997	.0002873	-3.83	0.000	-.0016628	-.0005366
black	.1110172	.0029347	37.83	0.000	.1052648	.1167696
hispanic	.0057716	.0031484	1.83	0.067	-.0003996	.0119428
married	-.0195236	.0019524	-10.00	0.000	-.0233506	-.0156966
_cons	.037838	.0046064	8.21	0.000	.028809	.046867

- Why are the coefficient easier to interpret?
- What is the problem with the linear probability model?

Question 3

D. Using the Propensity-Score method

Finally, if we use the estimated propensity scores and match the treated and untreated individuals in 4 bins of propensity scores (0.0-0.1, 0.1-0.2,...), what are the **treatment effects in each bin of the propensity score**?

Question 3

D. Using the Propensity-Score method

```
. reg re78 p_1 p_2 p_3 treatment p_treat1 p_treat2 p_treat3
```

Source	SS	df	MS	Number of obs =	16177
Model	2.9769e+10	7	4.2527e+09	F(7, 16169) =	46.36
Residual	1.4831e+12	16169	91727540	Prob > F =	0.0000
				R-squared =	0.0197
				Adj R-squared =	0.0193
Total	1.5129e+12	16176	93528158.7	Root MSE =	9577.4

re78	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
p_1	-4101.933	700.8708	-5.85	0.000	-5475.717	-2728.148
p_2	-6940.625	690.1353	-10.06	0.000	-8293.367	-5587.884
p_3	-10853.74	1694.808	-6.40	0.000	-14175.75	-7531.728
treatment	-7105.638	1238.823	-5.74	0.000	-9533.868	-4677.409
p_treat1	4166.125	2014.548	2.07	0.039	217.3883	8114.861
p_treat2	3273.189	1831.397	1.79	0.074	-316.5515	6862.929
p_treat3	7550.119	3470.609	2.18	0.030	747.3407	14352.9
_cons	15001.49	76.74002	195.48	0.000	14851.07	15151.91

- For whom are the estimated treatment effects highest highest?
- Does this make sense?

Question 3

E. Using the Propensity-Score method

If we now include the covariates X , nothing should change if the CIA indeed holds.

```
. reg re78 p_1 p_2 p_3 treatment p_treat1 p_treat2 p_treat3 age education black hispanic married
```

Source	SS	df	MS		Number of obs =	16177
Model	1.4761e+11	12	1.2301e+10		F(12, 16164) =	145.63
Residual	1.3653e+12	16164	84465418.8		Prob > F =	0.0000
Total	1.5129e+12	16176	93528158.7		R-squared =	0.0976
					Adj R-squared =	0.0969
					Root MSE =	9190.5

re78	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]
p_1	1632.492	763.606	2.14	0.033	135.7398 3129.245
p_2	191.3484	755.1	0.25	0.800	-1288.731 1671.428
p_3	-2109.405	1670.47	-1.26	0.207	-5383.711 1164.902
treatment	-4191.879	1201.372	-3.49	0.000	-6546.701 -1837.058
p_treat1	1563.486	1940.636	0.81	0.420	-2240.376 5367.349
p_treat2	464.183	1765.967	0.26	0.793	-2997.308 3925.674
p_treat3	4733.499	3334.938	1.42	0.156	-1803.348 11270.35
age	64.86673	7.426033	8.74	0.000	50.31088 79.42258
education	446.6367	26.30531	16.98	0.000	395.0754 498.198
black	-2381.292	342.9629	-6.94	0.000	-3053.537 -1709.047
hispanic	-858.5486	286.9083	-2.99	0.003	-1420.921 -296.1765
married	4637.042	184.4558	25.14	0.000	4275.488 4998.596
_cons	4238.746	425.3136	9.97	0.000	3405.084 5072.407

- What changes, what not?
- Does this make you have doubts about whether the CIA holds?

Question 3

Conclusion

Are the results from propensity score matching **close to the experimental results**?