Week 4 FAQ

What does 'conditioning on' a variable actually mean? Does it mean identifying that variable as the cause?

The answers to the second question is no. The answer to the first is:

Think about the linear regression of Y on X:

Y_hat = a + B_1 X

B_1 = the average unconditional (or marginal) difference in Y of a 1 unit difference in X.

Now add a control variable Z:

Y_hat = a + B_1 + B_2 Z

B_1 = the average conditional (conditional on Z) difference in Y of a 1 unit difference in X evaluated at any value of Z (that's the "control" bit).

Could you explain in more detail how conditioning on a collider unblocks a pathway?

What is it exactly that's "colliding" and why is it a problem?

I am not quite sure if I grasped collider variables and the effects of conditioning on them entirely.

See Research Design 2018 lecture 4 part 1 1.30-7.04. That was my best shot at explanation. If it didn't help then try Morgan & Winship pp 62-74. Another explanation can be found in Pearl, Glymour & Jewell (2016) *Causal Inference in Statistics: A Primer*, pp 40-45. It's also discussed In Elwert (2013) Graphical Causal Models – on the reading list.

Do qualitative methods have a role in clarifying the nature and direction of the relationships depicted in a DAG or is controlling for covariates and other statistical techniques sufficient?

It is hard to construct a structured DAG like the one in the lecture at the early and even middle stage. Could the 'close-the-back-door' technique help develop our causal inference models along the process rather than merely acting as an examination method (at the very end)?

The story you write down with the DAG has to come from somewhere. Remember it's your conjecture about how the world works. How you get there is entirely outside of the DAG. Once you've written it down the DAG just gives you are coherent way of figuring out which variables included in the DAG you need to condition on in order to make a causal inference. How you get to that point and how you then make the inference are entirely other matters. So qualitative insights, along with a lot of other sources of information, will inevitably go into writing the DAG down.

My question is - what are some more empirical uses of DAGs?

See answer above. If that doesn't answer your question, then I don't quite understand what you are asking.

My question about this week's reading is about how exactly we could use DAGs to visualise/settle the disagreement in the seminar readings?

That is what you are supposed to be thinking about.

Here is my question for this week: How do structural equation models work? Are they any different from just running a bunch of regressions in order to be more precise about multiple connections in a DAG (i.e. running a different regression for different focal relationships)?

There is a massive literature on SEMs. At a very high level of generality you can describe them as models that link together equations that "explain" outcomes. There are also measurement models for those outcomes. At their simplest they could just be a linked set of linear regressions. See for instance Kaplan, D. (2009) *Structural Equation Modeling: Foundations and Extensions*.

What are two and three staged least squares and when is it advised to use them?

They are just estimation techniques, commonly used when you have a system of equations. It is not necessary to know anything about them for this course. If you really want to know more there are standard references. For example Greene *Econometric Analysis* (1997) pp 740-742, 752-754.

Though you mentioned in your lecture videos that we really don't have time to go too far in-depth on instrumental variables, I was curious how one would go about finding a suitable instrumental variable. Would running various correlations to find what "fits" be an acceptable exercise?

Thinking. The IV has to be correlated with the treatment T, the stronger the correlation the better. That can be evaluated empirically. But the second requirement that the IV is uncorrelated with e_i is more tricky. This can't be tested when we only have one IV. In general it is difficult to come up with convincing IVs.

Could you please provide another example for an instrumental variable? I understand the reasons why or when to use it, but I did not understand where we would get an IV from/how we think of one.

Where to get an IV from – see answer above. There are 4 different examples in the lecture. For more examples than you can shake a stick at read Bollen (2012) 'Instrumental variables in sociology and the social sciences' *ARS*, on the reading list.

When you look for "naturally randomized" situations to observe (like birth dates in relation to grade school, as given in the lecture example), is there some protocol to check that the sample really is randomly sorted? Something may logically seem random, but there could be an unseen factor that systematically selects.

No, but common-sense usually helps. It's difficult to know what you could do about some unknown or unseen factor. But there could be an unknown or unseen factor that would make whatever you propose to do in life unwise or unsafe. Very few though use that as a good reason to do nothing.

In the lecture, you talked about IV estimates and concluded by saying that IV estimate measure the local average treatment effects rather than the average treatment effects. What is the difference between LATE and ATE, and what is the significance of measuring LATE instead of ATE?

That is explicitly explained in the lecture. I suggest you go back and watch that part again.

When we use instrumental variable, how do we interpret the result of the regression? I mean, when we have I, T and Y as the lecture slides illustrated, and we replace T with I to evade error term, in the result, do we still mention T? If so, how do we interpret T's impact on Y in terms of how correlated T to I is?

The IV estimate is the LATE of T on Y.

You mentioned that the implicit exclusion from LATE is individuals who would always take the treatment no matter what - is there an empirical example of such exclusions and to what extent would such exclusion affects the results?

Imagine an experiment in which the treatment is to randomly encourage some subjects to watch Fox News at a particular time. Some of those randomized to the treatment group would never normally watch Fox. For others it is there normal source of news. The latter always take the treatment regardless of the treatment assignment. The consequence of this for the difference between the ATE & LATE depends, amongst other things, on the proportion of always takers.

Under what circumstances would it be appropriate to use IV estimates?

That is explained in the lecture, I suggest you watch it again. Alternatively or in addition read the references on the reading list that are explicitly about IV estimation.

Why does measurement error induce T to be correlated with the error term? (Slide 17)

Measurement error in the X becomes a component of the overall error in the equation for the observables $Y = a + BX + e\_1$

In the simplest case with independent measurement errors what we observe is $X = X^* + e\_2$ then $Y = a + BX^* + (e\_1 + e\_2)$

We want to know about BX^* but what we estimate BX.

My question for the seminar is in regards to the lecture: I don't quite grasp the diagram about instrumental variables presented on slide 3 of part 2 of the lecture. Could you clarify it as well as its relation to the formulas or the more simple diagrams on the first slide of that part of the lecture?

Not really. As I said explicitly in the lecture: "**some** people find the diagram helpful". If you don't that's OK. There are lots of different ways to understand the same basic concepts.

In Hans Zeisel's critique of the experiment, he states that payments only had the effect of "reducing the number of weeks worked by the ex-convicts... enabled them to spend these fewer work weeks on better paying jobs." (pg 388 & 389). He then goes on in point 4 to say there is a negative correlation between number of weeks worked and recidivism - leading to the need to test whether payments "that would not be a disincentive to work" would reduce recidivism. I don't really understand this point. How did he come to this conclusion from the original study? Is he

saying the authors missed the fact that PAYMENTS are not the important factor, rather, it's that the payments allow the ex-convicts time to look for a better job? How does that make the payments a "disincentive" to work?

Possibly of substantive interest but it's not important for the methodological points I want us to think about.

Is it bad to have a model that is counterbalanced, since the effects largely cancel off each other?

Bad in what sense? Unless you are a little more precise I can't really answer this question.

What is a censoring mechanism?

A censoring mechanism is a rule that specifies what is observed and what is not observed. You only observe the wages of people who actually have a job. Maybe you want to estimate the relationship between education and wages for the whole adult population. The censoring mechanism will then be an equation for something like (wage offer – reservation wage) with wage only being observed for those individuals where it is > 0 .

Expand on the notion of Mills ratio and is it only significant to Heckman's model?

It's not important for this course. If you really want to know the details there are plenty of standard treatments. You'll find a clear explanation in Breen (1996) *Regression Models: Censored, Sample Selected or Truncated Data*, pp 34-42. Also page 39 of Berk's article covers the same material but doesn't use the term "inverse Mills ratio". There are probably better ways to use your limited time.

Should an attempt be made to reduce 'sample selection bias' in qualitative research?

Depends on what you mean by "qualitative research" and depends what the question you are trying to answer is. The book by King et al. on the reading list has some discussion that is relevant. But you need to have a clear idea about my first two points before you can make sense of what they say.

Why do you assume that the candidate is charming?

It's an **example** of the sort of thing you might assume. Feel free to substitute anything else you think is preferable.

Hhow do you know what to assume with unobservables?

You have to assume something. There are no free lunches. Even not assuming anything is assuming something. Degree of plausibility based on existing subject level knowledge.

Berk (1983, p. 393) states the following:

The results on the far right derive from a probit model which rests on what we have been assuming so far: the two disturbances are bivariate normal. If one is prepared to assume that the disturbances are bivariate logistic, then the selection equation should be logistic (Ray et al., 1980). Finally, if one is prepared to assume that the disturbances in the selection equation follow a rectangular distribution and that the disturbances in the substantive

equation are a linear function of the disturbances in the selection equation, the linear probability model may be used to model selection (Olsen, 1980b).

My question is: What does it mean to be "prepared to assume" something? We've referenced it a lot in past seminar when discussing trade-offs, because no methodology can ever be perfect; however, we've never fully explored it. How would a researcher decide if s/he has prepared enough to be able to take on the assumptions, especially since the whole point of an assumption is that you can't verify it?

I take the general point, but it's not quite true to say that assumptions can't be tested. It's true though that not all assumptions can be tested simultaneously and that you are always going to have to assume something to get the show on the road. So how to assume: plausibility, common-sense, deep knowledge of the substance of the problem and the relevant context, a sense of magnitude for the consequences of assuming one thing rather than another. The answers won't fall out of the sky.

I very much like the idea of an interrupted time-series design. Could you speak more about which data is appropriate for modeling time-series effects? As in, would it only be an appropriate design for longitudinal panel studies, or could you incorporate lagged treatment effect estimates for other types of observational data?

Panels tend to have a small number of time points, time series studies many more – ie at least 30-40 to be effective. Lags can be includes in both. Time series analysis though is a way of life. Moreover it's a way of life that I've invested zero time in cultivating so I can't speak with any degree of credibility about it. If you are really interested in time series analysis you need to take a proper course in it and invest a significant amount of time – months rather than weeks - in learning about it. It isn't for the faint hearted or the overly optimistic.

My question for the week has to do with pre-testing; how do you determine how much pre-treatment in necessary for plausible relative to the time frame of the study?

As many as it takes to establish with certainty the time trend for a length of time that you care about.

How can we eliminate or reduce the threat of selection by maturation in cases where we don't have access to pre-test data?

Randomisation. Failing, that, with great difficulty or magic.

I want to know more about maturation effect and maturation threat in internal validity. In the PDM example that you gave in the lecture, is there any way to handle maturation effect? And how do we recognise/prove whether the research outcome is affected by maturation effect?

See previous answer.

Apart from pretesting observations and asking retrospective question are there any other ways of ruling out selection by maturation?

See previous answer.

The question is about the lecture (slide 40): If you can't control who is exposed to the treatment, why is your best option then to control when the observations are made? Are there any other options?

Well, you need a source of variation. The two obvious sources are time and space (ie geography)

My question is about how causal inferences can be complicated by the regression to the mean phenomenon. This is referring to slides 36-39 of the lecture. Is there a way to distinguish the treatment effect and regression to the mean? For example, on slide 36, can we claim the decrease in the slope for the project teams with PDM is caused by <u>both</u> treatment and regression to the mean? If this is the case, how can we then accurately identify/quantify the treatment effect?

You can't (without making a bunch of untestable assumptions).

Although unlikely, what if there was no treatment effect and the decrease in the slope was solely caused by the regression to the mean?

"What if" in what sense? If I believed it was a causal effect I would be wrong. Stranger things have happened.

My query is regarding - regression discontinuity design. And how this regression discontinuity design is better than pilot study. Or is pilot study a better approach than regression discontinuity design as both of them elicits the treatment effects by assigning the group to a certain level of treatment....

I don't really understand the question. The problem may be that you are using the term "pilot study" in a way that I'm not familiar with so that is preventing me from getting your point.