Lab 8: Instrumental Variables

PHS2000B, Spring 2023

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March 23, 2023

Overview

1. **Moving from epidemiologic to econometric methods**
2. **Overview of endogeneity**
3. **Instrumental Variables**
4. **Assumptions for IV**
5. **Different types of individuals to consider in IV**
6. **IV by hand, examples and critiquing - group exercise**

Situating Ourselves

Still talking about causal inference

Rearview Mirror: Epidemiologic methods

- Propensity Score based methods
- Mediation Analysis
- Marginal Structural Models
- Sensitivity Analyses

Up Ahead: Econometric methods

- Instrumental Variables
- Regression Discontinuity
- Difference in Differences
- Interrupted Time Series
- Sensitivity analyses addressed throughout

What is common?

We are still trying to identify causal effects, so counterfactual / potential outcomes thinking is still helpful!

We will still be asking questions along the lines of: "What would have happened if a certain group of people were exposed versus not exposed?"

Epidemiologists and econometricians both rely on strong assumptions. Our work is shaped by our worldviews, beliefs and explicit or implicit theories.

What is different? Big Picture

Epidemiologists

- Measurement of confounders, DAGs, and various methods of quantifying unmeasured confounding, adjustment and bounding for confounding
- Often start with the question of interest, then identify or gather data to answer it

Econometricians

- Generally skeptical that confounding can be controlled such that no unmeasured confounding remains (DAGs only recently seeing more usage)
- May start identifying possible natural/quasi-experimental opportunities, or even with identifying data, and then asking what causal questions could be answered

What is different? Language

Concept	Economics	Epidemiology
Bias due to a back-door path involving common cause of exposure and outcome. Lack of exchangeability between treated and untreated groups.	**Selection bias**, or **selection on treatment**. Also **omitted variable bias**, but this is agnostic to the causal relationship between the omitted variable and the exposure/outcome.	**Confounding**, in case of common cause of exposure and outcome.
Conditioning on observed variables ensures exchangeability	**Conditional Independence Assumption**, or **Selection on Observables**.	**Conditional exchangeability**, or **No unmeasured confounding assumption**
Explanatory variable is correlated with error term in linear regression, creating bias	**Endogenous**. This can occur if there is (1) omitted variable bias, (2) measurement error in the exposure, (3) reverse causation/simultaneity; Y causes X.	Epi thinks in terms of causal structure, so no specific term. Would refer specifically to bias, differential measurement error, or reverse causation.
Absence of correlation between the error term and explanatory variables	**Exogenous**.	No specific term, but may coincide in usage when epi refers to absence of situations above.

Epidemiology and Econometrics - a synthesis

- This course is giving a bird's eye view of various methods in different disciplines
- Different methods from different fields will be suitable for different questions. And the same kind of questions can be answered using different methods, depending on context and data sources
- There is increasing work across disciplines - e.g. social epidemiologists using econometric methods, econometricians using DAGs and potential outcomes framework.[1]
- Ultimately, as always, it depends.
- Nothing can replace contextual knowledge, and explicit theories. 

[1] Imbens, Guido W. 2020. "Potential Outcome and Directed Acyclic Graph Approaches to Causality: Relevance for Empirical Practice in Economics." Journal of Economic Literature, 58 (4): 1129-79. DOI: 10.1257/jel.20191597

Endogeneity

The Problem of Endogeneity

Suppose we are trying to estimate the following equation:

$$y_i = \beta_0 + \beta_1 x_i + \epsilon_i$$

Endogeneity means that for some explanatory variable x_i, the variable is correlated with the error term ϵ_i. That is, if $cov(x_i, \epsilon_i) \neq 0$, then we have endogeneity in our model. We can also say that the error term is orthogonal to the predictors in the absence of endogeneity.

Endogeneity results in biased and inconsistent $\hat{\beta}_1$

Endogeneity and confounding are not perfectly synonymous, but they are related. Endogeneity can come from different sources, one of which bias due to unmeasured confounding.

Sources of Endogeneity

In general, there are three main sources of endogeneity that we are concerned with:

1. **Omitted Variable Bias**: An unmeasured variable U is correlated with A and is a derminant of Y (this is when endogeneity and confounding are the same)
2. **Measurement error in the independent variable**: Nondifferential measurement or misclassification error in the independent variable (recall: attenuation bias)
3. **Reverse causality or simultaneity**: One or more of the independent variables is jointly determined with the dependent variable (for example, through an equilibrium mechanism), or values of past time series data are influenced by expectations

How have we talked about addressing endogeneity so far?

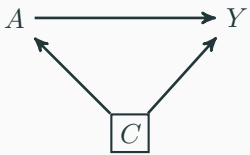
Randomized treatment assignment

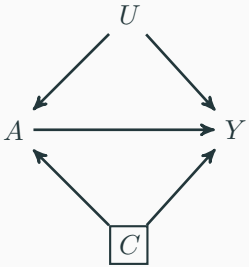
- An ideal RCT (with no loss-to-follow up, complete adherence, etc.) is the gold standard. In expectation, measured and unmeasured confounders are balanced across treatment and control groups
- Because treatment is assigned randomly, people cannot "select" into treatment. In the real world, people who take up a treatment may be different from those who don't take the treatment in ways that effect outcomes.

Control for confounding / close all backdoor paths

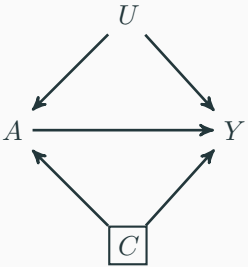
- We can also attempt to capture any sources of confounding, closing all backdoor paths (the epi way)
- In theory, if you can capture every source of confounding, you can estimate a causal effect. If we are wrong, we will have omitted variable bias, our model will have endogeneity, and our estimate of the causal effect will be biased.

Let's just see it on a DAG again for funsies





Where do we have conditional exchangeability / no omitted variable bias?

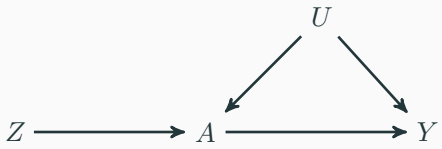

U
A
Y
C


(chuckles)
I'm in danger.

Instrumental Variables

Instrumental Variables

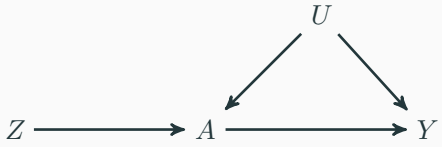
- With IV estimation, we can adjust for confounding even if we do not or cannot measure all the confounders.
- This can help in both experimental studies with imperfect adherence and in observation studies where people select into treatment.
- Let's start with an experimental study for a new drug, where treatment assignment, Z, is randomly assigned



- Z: assigned treatment (1: yes, 0: no)
- A: actually took the treatment (1: yes, 0: no)
- Y: outcome (alive at end of study period)
- U: unmeasured factors (healthy lifestyle)

Questions for you

1. Is the Z-A association a valid estimator of the causal effect of Z on A? (Is this association confounded?)
2. What about the association of Z-Y? Is this a valid estimator of the indirect effect of Z on Y?
3. Let's imagine that we estimate the effect of Z on Y – what type of effect is this capturing?
4. Will this effect tell us whether taking the treatment causes improvement in survival? Why / why not?



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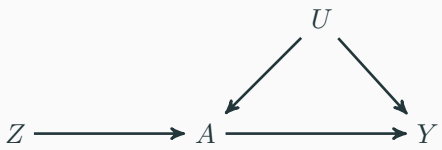
2. What about the association of Z-Y? Is this a valid estimator of the indirect effect of Z on Y? Yes
3. Let's imagine that we estimate the effect of Z on Y – what type of effect is this capturing? Intention-to-treat
4. Will this effect tell us whether taking the treatment causes improvement in survival? Why / why not? No, it will tell us the effect of being assigned treatment



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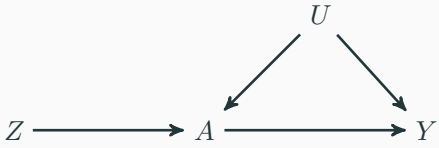
Intention-to-Treat vs. Per-protocol Effect

The effect of Z on Y is the **Intention-to-Treat (ITT) effect**.

The effect of A on Y that would have been observed under full adherence to assigned treatment is the **per-protocol effect.**

Can we just use the observed association between A and Y to estimate the per-protocol effect?



Intention-to-Treat vs. Per-protocol Effect

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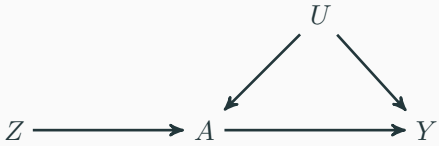
The effect of A on Y that would have been observed under full adherence to assigned treatment is the **per-protocol effect.**

Can we just use the observed association between A and Y to estimate the per-protocol effect?
No, there is endogeneity / confounding.



IV Analysis

We know:

- Effect of Z on Y (intention-to-treat effect) is unconfounded
- Effect of Z on A (adherence) is unconfounded

We can adjust the **ITT effect** by the degree of non-adherence to estimate the causal effect of A on Y using the standard or canonical IV estimator:

$$\text{Effect of } A \text{ on } Y = \frac{\text{Effect of } Z \text{ on } Y}{\text{Effect of } Z \text{ on } A} = \frac{ITT}{Adherence}$$

When the instrument is binary, this is $= \frac{E[Y|Z=1]-E[Y|Z=0]}{E[A|Z=1]-E[A|Z=0]}$

We can also estimate what is known as a **two-stage estimator** by first generating a regression for Z on A (first stage), then using predicted values of A to estimate A on Y (second stage) to reach the causal effect. This is what you will be doing in your Problem Set.

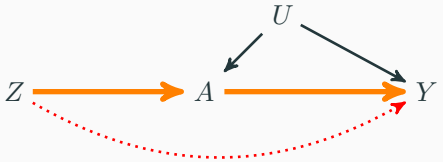
Assumptions for Instrumental Variables

Key Assumptions for IV

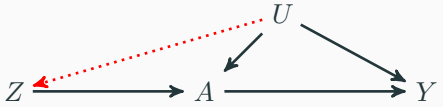
Random variable Z is an instrument if it satisfies all of the following criteria:

1. **Relevance**: Z is associated with A

2. **Exclusion Restriction**: Z affects the outcome Y only through A



3. **Independence**:There are no common causes of the Z-Y relationship



Relevance

1. **Relevance**: Z is associated with A

For a given candidate instrument Z and exposure A, relevance is defined as:

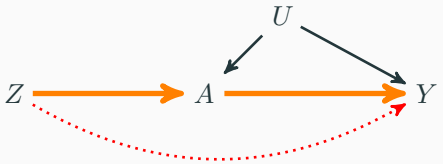
$$cov(z_i, a_i) \neq 0$$

This condition can be empirically verified (a common rule of thumb is an F-statistic > 10). If Z is strongly associated with A, we might consider the instrument Z to be a "strong" instrument.

What would happen to our estimator ($= \frac{\text{Effect of } Z \text{ on } Y}{\text{Effect of } Z \text{ on } A}$) of the instrument was "weak"?

Exclusion

2. **Exclusion Restriction**: Z affects the outcome Y only through A



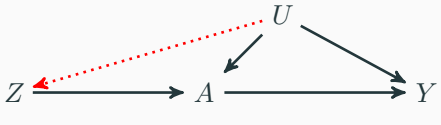
In potential outcomes notation, assuming a binary instrument:

$$Y_{z=1,a} = Y_{z=0,a} = Y_a$$

Going back to the RCT example, what would happen if blinding failed, and everyone knew their treatment assignment? Can you think of ways that simply knowing your treatment assignment might improve outcomes, separate from whether you actually take the treatment or not?

Independence

3. **Independence**:There are no common causes of the Z-Y relationship



Independence implies that there are no open back-door paths (e.g. no unmeasured confounders) from a candidate instrument Z to the outcome Y

In potential outcomes notation, assuming a binary instrument:

$$Z \perp\!\!\!\perp Y_a$$

Causal Effect Types from IV

Identifying Causal Effects

If we assume treatment effect is **homogenous**, and make the assumptions discussed above:

1. Relevance: Z is associated with the treatment A
2. Exclusion restriction: Z affects the outcome Y only through A for all individuals in the study population
3. Independence: There are no common causes of the Z - Y relationship

IV identifies the **Average Treatment Effect (ATE)**

Identifying Causal Effects

If we assume treatment effect is **heterogenous**, we have to make an additional assumption:

1. Relevance: Z is associated with the treatment A
2. Exclusion restriction: Z affects the outcome Y only through A for all individuals in the study population
3. Independence: There are no common causes of the Z - Y relationship
4. **Monotonicity**: The instrument affects treatment status for everyone in the same direction (i.e. no defiers)

IV identifies the **Local Average Treatment Effect (LATE)**

Interpreting Causal Effects

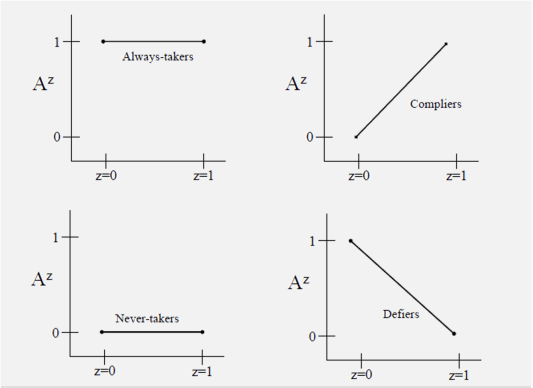
Let's imagine an RCT where participants are randomly assigned whether to take a daily aspirin. We are interested in the effects on the prevalence of cardiovascular disease (CVD).

- **ITT**: This is the effect of randomly being assigned to take aspirin on CVD
- **ATE**: This is the effect of actually taking aspirin on CVD. Even if we are only comfortable assuming monotonicity instead of homogeneity, we can't actually estimate this.
- **LATE**: This is the effect of actually taking aspirin on CVD among the group of people who would only have taken aspirin if assigned to do so (i.e., the compliers).

Different kinds of individuals in the population

We can think of 4 types of individuals in a population:

- **Always takers**: Individuals who take the treatment regardless of the value of the instrument. $A_{z=1} = A_{z=0} = 1$
- **Never takers**: Individuals who never take the treatment regardless of the value of the instrument. $A_{z=1} = A_{z=0} = 0$
- **Compliers**: Individuals who take the treatment when assigned to do so by the instrument, but don't when not assigned. $A_{z=1} = 1$ and $A_{z=0} = 0$
- **Defiers**: Individuals who take the treatment when assigned not to do so by the instrument but take the treatment when not assigned. $A_{z=1} = 0$ and $A_{z=0} = 1$



Different kinds of individuals in the population

These individual types describe counterfactual behavior, not observed behavior.

People who adhere to treatment assignments may be said to have complied with treatment assignment, but that does not automatically make them Compliers (i.e., they could be Always-takers depending on what they would have done if assigned to the control.)

	$Z_i = 1$	$Z_i = 0$
$A_i = 1$	Complier/Always Taker	Defier/Always Taker
$A_i = 0$	Defier/Never Taker	Complier/Never Taker

Local Average Treatment Effect

The monotonicity assumption implies that there are no defiers. This means that the local average treatment effect will only be the effect among compliers (i.e., folks whose exposure is changed by the instrument).

LATE is the treatment effect among compliers

$$\text{LATE} = E[Y_{a=1} - Y_{a=0} | A_{z=1} = 1, A_{z=0} = 0]$$

Better LATE than never?

Arguments against LATE:

- LATE does not equal ATE generally, which is really the quantity of interest
- Difficult to characterize the compliers, so difficult to think of what types of individuals the estimated treatment effect applies to
- This is especially true with continuous instruments, and non-causal instruments

Arguments for LATE:

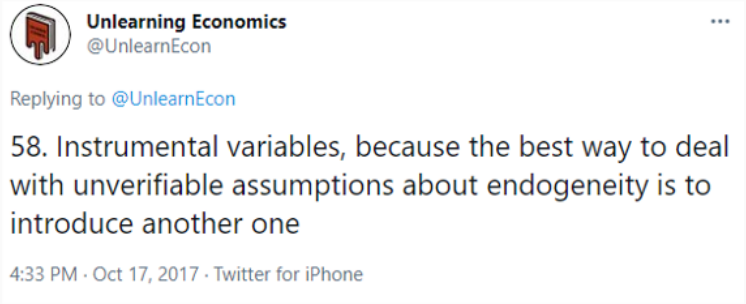
- Internally valid estimate
- RCTs with adherence adjustment are giving us the LATE
- ATE may not be available, whereas at least the LATE can be identified

Do you bel-IV?

It is one thing to have randomization as an instrument where exclusion is likely to hold.

However, the draw of IV historically has been the ability to find an instrument that can account for a host of various confounders.

The key argument is that there is no way the instrument affects the outcome except through the treatment of interest. How plausible is this?



It's Rainin' IV!

Rainfall is often used as an instrument for different questions. For example the effect of parental income on education in agricultural settings. Or TV viewing on child development (rain causes kids to watch TV instead of playing outside).

But if rainfall is affecting various exposures of interest.... then perhaps it is not a good instrument?

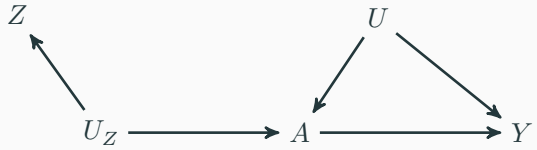
Group Work :)

Group work

1. Look through some of the DAGs in the following slides and discuss in small groups whether Z is a valid instrument.
2. Look through examples of instruments used in publish literature in the handout, and discuss whether you find it plausible that they meet the IV assumptions we have discussed (especially the relevance, exclusion and independence assumptions.)
3. Work through the table and questions in the handout.

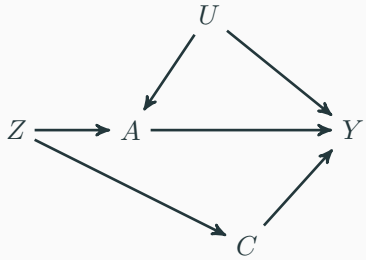
We will reconvene and discuss

DAG 1



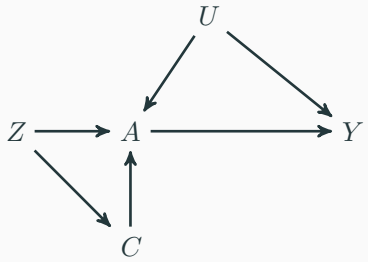
Yes, Z is a valid instrument. Z is associated with A through Uz. Z is associated with Y only through A. There are no open back-door paths from Z to Y .

DAG 2



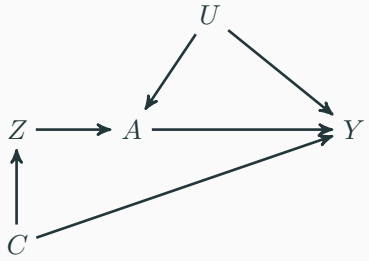
If we don't control for C, then Z is not a valid instrument. Z violates the exclusion restriction because Z $\rightarrow$ C $\rightarrow$ Y . By controlling for C, we can ensure that Z is a valid instrument. In practice, however, if there are common causes of both C and Y (which is likely), conditioning on C results in collider stratification and a violation of the exclusion restriction.

DAG 3



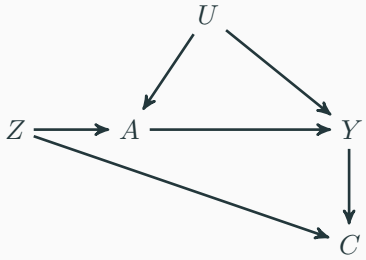
Yes, Z is a valid instrument. Z satisfied the relevance criterion. There is no direct path from Z to Y which does not go through A. There are no open back-door paths from Z to Y .

DAG 4



Yes, Z is a valid instrument as long as we control for C. This DAG shows that the independence criterion can be valid even if we have conditional independence of the Z — Y relationship.

DAG 5



Answer: Yes, Z is a valid instrument as long as we do not control for C. If we control for C, we will violate the independence assumption.

Critiquing Instrumental Variables

- What is the effect of economic growth on civil conflict?
- What is the effect of high-cost hospital care on mortality?
- What is the impact of Medicaid expansion on emergency department use?

Supplementary Slides

Indirect Least Squares

In lecture, we saw an estimator that used regression coefficients, using **Indirect Least Squares**. You have:

A model for the treatment (A_i) using the instrument as the predictor (Z_i) (first stage):

$$A_i = \gamma_0 + \gamma_1 Z_i + u_i$$

Here, γ_1 is the effect of a one unit change in the instrument on the treatment value.

Next, we can fit a model for the outcome (Y_i) with the instrument as the predictor (Z_i) (reduced form)

$$Y_i = \theta_0 + \theta_1 Z_i + v_i$$

Here, θ_1 is the effect of a one unit change in the instrument on the outcome value.

Indirect Least Squares

The model that we are interested in, that we cannot use because we have endogeneity / confounding is:

$$Y_i = \beta_0 + \beta_1 A_i + \epsilon_i$$

and β_1 is the coefficient of interest.

It can be shown that $\theta_1 = \beta_1 \gamma_1$, so $\hat{\beta}_1 = \frac{\widehat{\beta_1 \gamma_1}}{\hat{\gamma}} = \frac{\hat{\theta_1}}{\hat{\gamma_1}}$ **if the IV assumptions are met**

So, the IV estimate is the ratio of the coefficient from the reduced form model to the coefficient from the first stage model.