Is IV "Junk Science"?

(A Primer on Instrumental Variables and the Controversial Evidence linking Television Watching to Autism)

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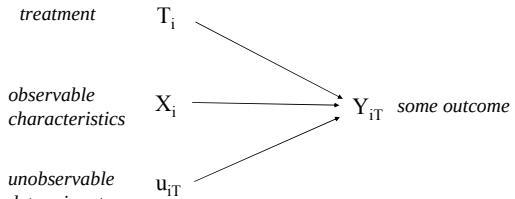
Outline

- The IV Primer
  - Review bias issues with OLS
  - How IV is similar to an imperfect RCT
  - Interpreting IV estimates
  - Sources of bias
  - Other issues (finding instruments, weak instruments, relevance of LATEs)
- Application: Does Early Television Cause Autism?
  - What do the reduced form models tell us?
  - What can we firmly conclude about the relationship between TV and autism?

The "Standard Research Question"

- What's the effect of treatment T?



Y_{i1} ~ outcome if T=1
Y_{i0} ~ outcome if T=0

The "Standard Research Question"

- What's the effect of treatment T?
- What kind of estimates might be interesting?
- Average Treatment Effect (ATE)
  - $E(Y_{i1}-Y_{i0})$
- ATE for specific types
  - $E(Y_{i1}-Y_{i0} \mid X_i=X^*)$ or $E(Y_{i1}-Y_{i0} \mid X_i<X^*)$
  - $E(Y_{i1}-Y_{i0} \mid Y_{i0}<Y^*)$
- Individual Expected Treatment Effect
  - $Y_{i1}-Y_{i0}$
  - usually impractical

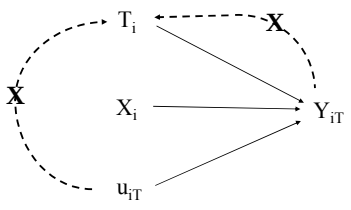
Problem: You only have observational data

- Straightforward estimation model (OLS):

  $Y_i = b_0 + b_1 T_i + b_2 X_i + u_i$
- Q: what does the estimate of b_1 tell us?
- Depends on what we're willing to assume…
  - If the "independence assumption" holds, provides an unbiased estimate of the ATE
    - IA: $Cov(T, u \mid X) = 0$
  - Can interact T∗X to determine how effect size varies across different types

Potential Bias Problems

- Omitted variable (selection) bias
- Reverse causality



Potential Bias Problems

- Omitted variable bias (selection bias)
  - T is correlated with other determinants of Y
  - ex1: effect of drug adherence on outcomes
  - ex2: effect of education on wages
  - ex3: effect of hospital care on mortality
  - ex4: effect of job training on wages
- Reverse causality
  - Y has a direct effect on T
  - ex: effect of income on health
- Misspecification bias
  - independence assumption might only hold if you control for the Xs appropriately

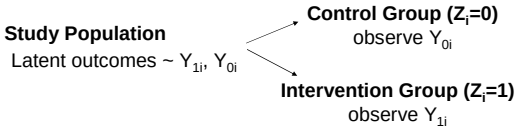
What does propensity matching buy you?

- Robustness to misspecification bias
  - Even if you're controlling for the "right Xs", you might fail to control for them the right way
  - PM is more robust to misspecification errors than OLS
- Can focus on treatment effect over a more relevant population
  - The population of "treated" includes some who would *always* receive treatment
  - The population of "untreated" includes some who would *never* receive treatment
  - Aggregate ATE probably less interesting than ATE over the population who potentially change treatments
  - Only an issue if Y_{i1}-Y_{i0} varies

What does propensity matching *doesn't* buy you?

- A solution to the potential problems of omitted variable bias and reverse causality
  - To the extent *unobservable* determinants of treatment are correlated with the outcome, propensity matching still produces biased estimates
  - Particularly concerning when you don't have an idea what causes people to "select into" treatment or can only imperfectly control for these factors
- In many cases, the independence assumption is hard to swallow even with the "best" Xs
  - E.g. effect of income on health, effect of job training on wages, peer effects

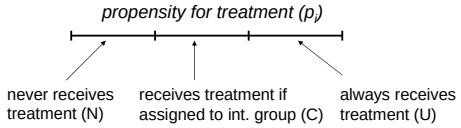
One Solution: The RCT



- Value of RCTs is that the source of variation in T is *known* and (by construction) not correlated with the outcome
  - the mean Y_0 observed for the control group is an unbiased estimate of the (unobserved) mean Y_0 for intervention group, and vice-versa
  - therefore, difference in means provides on unbiased estimate of the average treatment effect

Imperfect RCTs → the "slippage" issue

- In some RCTs, group assignment does not entirely determine treatment status
  - "no shows"
  - controls find another way to treatment
- Can think of the sample population in terms of their "propensity for treatment" distribution:



Imperfect RCTs → the "slippage" issue

- Q: What does difference in group means tell you?
  - expected mean outcome for int. group:
    $E(Y|Z=1) = Pr(N)E(Y_0|N) + Pr(C)E(Y_1|C) + Pr(U)E(Y_1|U)$
  - expected mean outcome for control group
    $E(Y|Z=0) = Pr(N)E(Y_0|N) + Pr(C)E(Y_0|C) + Pr(U)E(Y_1|U)$
- Therefore, expected difference given by
  $E(Y|Z=1) - E(Y|Z=0) = Pr(C)\,[E(Y_1-Y_0|C)]$
  = "intention to treat" effect
- If we "know" Pr(C), we can divide to produce
  $E(Y_1-Y_0|C)$ = effect of "treatment on the treated" (ETT)

Surprise: Estimating ETTs is really just an application of Instrumental Variables

- In economics lingo, the group assignment is an "instrument" for whether someone receives treatment
- What makes it a good instrument?
  - assignment affects treatment outcome
  - assignment is uncorrelated with determinants of the outcome
  - assignment only affects outcome through its effect on treatment*

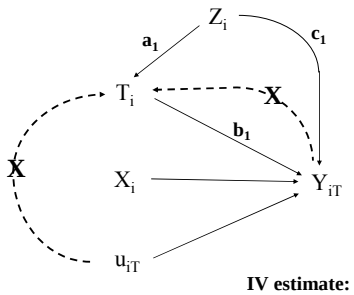
IV Estimation

- Roughly speaking, IV estimation is a regression-based approach to estimating the ETT…
- "First stage" model: $T_i = a_0 + a_1 Z_i + a_2 X_i + v_i$
  - Note: $E(\hat{a}_1) = Pr(C)$
- "Reduced form" model: $Y_i = c_0 + c_1 Z_i + c_2 X_i + w_i$
  - Note: $E(\hat{c}_1) = Pr(C)[E(Y_1 - Y_0 \mid C)]$
- The ETT can be estimated by $\hat{c}_1/\hat{a}_1$
- This is all IV estimation does!

IV Estimation

- Alternative approach…
- "First stage" model: $T_i = a_0 + a_1 Z_i + a_2 X_i + v_i$
  - Note: $E(\hat{a}_1) = Pr(C)$
- "Second stage" model: $Y_i = b_0 + b_1 \check{T}_i + b_2 X_i + e_i$
  - where $\check{T}_i = \hat{a}_0 + \hat{a}_1 Z_i + \hat{a}_2 X_1$
  - b_1 provides estimate of ETT
- But don't think of it this way…

IV Estimation



IV estimate: $b_1 = c_1/a_1$

IV beyond Imperfect RCTs

- Usually we don't have a "perfect instrument" like random assignment, and have to rely on "natural" sources of variation in treatment
  - Example: suppose the distance one lives from treatment center (D_i) affects probability one receives treatment
  - Same methodology applies

    "First stage" model: $T_i = a_0 + a_1 D_i + a_2 X_i + v_i$

    "Second stage" model: $Y_i = b_0 + b_1 \check{T}_i + b_2 X_i + e_i$
- Under what conditions is b_1 unbiased?
  - $Cov(D,v \mid X)=0$, i.e. a_1 must capture causal relationship
  - $Cov(D,e \mid X)=0$, i.e. D is uncorrelated with unobserved determinants of Y, and affects Y strictly thru its effect on T

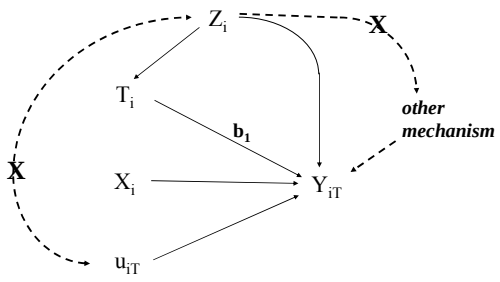
IV beyond Imperfect RCTs

- Assuming these conditions are met, how do we interpret estimate of b_1?
- If treatment effects are constant, IV provides an unbiased estimate of the ATE
- In general, we say IV estimates a "local area treatment effect"
  - Note that IV estimate is identified from the variation of T that is induced by variation in D
  - Analogous to the ETT, the estimate captures the mean effect for those affected by the instrument

IV beyond Imperfect RCTs

- T doesn't have to be binary (treatment can be in continuous units)
  - An instrument affecting one's income can be used to estimate causal effect of income
  - An instrument affecting one's amount of TV viewing…
- Z can consist of multiple instruments
  - Improves precision
  - Allows for additional specification tests

IV Estimation Bias



Other Potential Problems

- Finding instruments
  - what causes exogenous change in the covariate of interest?
- Relevance of LATE
  - if effect of T varies, IV estimates can vary depending on the instruments used
  - b_1 is identified off of the variance in T explained by Z
  - this could occur in an unimportant part of the T distribution
  - ex: if Bill Gates wins the lottery…

Other Potential Problems

- Loss of precision
  - OLS provides tighter confidence intervals than IV
  - OLS exploits the full variance in T (not explained by Xs)
  - IV only exploits variance in T explained by Z
- Weak Instruments (FS F-statistic <10)
  - IV estimate biased towards OLS
  - confidence intervals artificially tight
  - small violations of identifying assumption result in large biases

Application: Does TV Cause Autism?

- Waldman et al. 2007 (NBER WP 12632)
- Hypothesis:
  - Exposure to particular "environmental toxins" serve as triggers to autism
    - Vaccines: no empirical support
    - Air pollution: some empirical support
  - Waldman investigates whether TV watching might serve as a trigger
- Onset of autism occurs by age 3
  - Does "extra" TV watching through age 2 increase probability of developing autism?

Methodological Issues

- Waldman does not specifically measure the "effect" of TV watching on autism
  - only interested in whether there's evidence of causality
  - inferred from first stage and reduced form models
- Two instruments used in separate analyses:
  - county precipitation rates
  - county "cable TV" rates

Instrument 1: Precipitation

- First stage model (ATUS data):
  $TV_i = a_0 + a_1 PRCP_i + a_2 X_i + e_i$
  where
  TV_i = TV viewing time in day (inferred)
  $PRCP_i$ = precipitation in area on survey day
- Results:
  - level of precipitation has a strong but concave effect on TV viewing time
  - appears to peak at 2 inches
- Q: Is this evidence of a causal relationship?

Instrument 1: Precipitation

- Reduced form model:
  $AUT_k = b_0 + b_1 PRCP_k + a_2 X_k + e_k$
  where k ~ county/birthyear cohort
  AUT_k = cohort autism rate
  $PRCP_k$ = avg annual precip over…
- Results:
  - significantly positive b_1 for OR and WA
  - less consistent evidence for CA, but probably positive
- Q: Is this evidence of a causal relationship?

Instrument 1: Precipitation

- So what do we know?
  - Precipitation is positively correlated with TV viewing by 0-2 year olds
  - County precipitation rates over ages 0-2 are positively correlated with cohort autism rates
- Is this evidence that TV watching has a causal effect on autism?
  - Competing hypotheses?

Instrument 2: Cable Rates

- First stage model…
  - There is none!
- Do we believe that the percentage of households in county with cable influences amount of TV viewing among 0-2 olds in county?

Instrument 2: Cable Rates

- Reduce form model:

  $AUT_k = b_0 + b_1 CAB_c + a_2 X_c + e_k$

  where

  CAB_c = pct HHs with cable when cohort 0-2 y/o
- Results:
  - CAB positively correlated with cohort rate of autism in CA
  - Result insignificant for PA, but similar magnitude
- What does this relationship mean?

Is this "Junk Science"?

- What can we firmly conclude from these results?