Instrumental Variables, and the Effect of Binary Treatments on Binary Outcomes

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Will Draw Upon Joint Work:

Will draw upon joint work from two projects:

1. Joint project with
   James Heckman, University of Chicago
2. Joint project with
   - Jay Bhattacharya, Stanford University
   - Cecilia Machado, Columbia University
   - Azeem Shaikh, University of Chicago
   - Nese Yildiz, Rochester University

Question of Interest:

How can we recover the effect of a binary, endogenous treatment on a binary outcome when we have access to an instrument?

For example, how to recover the effect of a medical intervention on later mortality?

Will conclude with empirical application to effect of Swanz-Ganz catheterization on mortality.

Question of Interest:

How can we recover the effect of a binary, endogenous treatment on a binary outcome when we have access to an instrument?

Issues:

1. If effect varies across people, what do we mean by "the effect"?
2. What sets of assumptions can coherently be imposed? plausibly be imposed?
3. How well do different sets of assumptions aid us in recovering different definitions of "the effect"?

Notation:

Let

- $D \in \{0, 1\}$ denote a binary treatment of interest (e.g., a medical intervention)
- $Y \in \{0, 1\}$ denote a binary outcome of interest (e..g., later mortality)
- Z denote an instrument for D (e.g., random assignment)

Examples

Example:

- Y mortality 12 months after start of study.
- D medical intervention.
- Z random assignment with imperfect compliance.

Example:

- Y dummy variable for contracting the flu
- D flu vaccination.
- Z random encouragement to take the vaccination

Example:

- Y employment at 12 months
- D receipt of job training at date 0.
- Z local labor market conditions at date 0.

Latent Outcomes:

Let

- Y_1 denote outcome if treated
- Y_0 denote outcome if not treated

And thus

$$Y = DY_1 + (1 - D)Y_0$$

$$Y_1 - Y_0 = \text{ individual treatment effect}$$

How to Define the Effect?

If effect of D on Y, $Y_1 - Y_0$, varies across individuals, how to define "the effect"?

- ATE, $E(Y_1 - Y_0)$
- Treatment on the treated, $E(Y_1 - Y_0|D = 1)$
- Instrument defined parameter (i.e., LATE)
- Policy relevant parameter?

Assumption and approach that aids in recovery of one parameter may not aid in recovery of another parameter.

What Assumptions Can We Not Impose?

Because Y is binary, we cannot coherently impose assumptions of:

- Classical instrumental variables
  - Y binary implies that structural equation for Y cannot be additive in error term
  - However, LATE assumptions of Imbens and Angrist (equivalently, selection model of Heckman-Vytlacil) still possible.
- Quantile IV
  - e.g., assumptions of Chernozhukov and Hansen, 2005
  - Y binary implies that Y cannot be strictly increasing in continuous error term

What Assumptions Can We Not Impose?

Because D is binary, we cannot coherently impose assumptions of:

- Control Variate Strategies
  - Classic reference: Rivers and Vuong (1988)
  - Semiparametric/Nonparametric generalizations: Blundell and Powell 2004, Imbens and Newey 2009
  - Key idea: Use regression of D on Z to recover unobservable that determines selection into treatment, control for it in second stage.
  - D binary implies D cannot be additive or strictly increasing function of continuous error term.
  - However, can follow Abrevaya, Khan and Hausman (2009) for direction of effect.

What Assumptions Can We Impose? BVP?

We can impose classic model:
Heckman (1978) Bivariate Probit model with Structural Shift

$$\begin{aligned} Y &= \mathbf{1}[X\beta + \alpha D - \epsilon \geq 0] \\ D &= \mathbf{1}[Z\gamma - \nu \geq 0] \end{aligned}$$

with

$$(\epsilon, \nu) \perp\!\!\!\perp (X, Z), \qquad (\epsilon, \nu) \sim BVN$$

Issues:

- Sensitivity to joint normality assumption?
- Sensitivity to linear index restriction?

What Assumptions Can We Impose?

What if relax to nonparametric threshold crossing model on each equation:

$$\begin{aligned} Y &= \mathbf{1}[\mu(X, D) - \epsilon \geq 0] \\ D &= \mathbf{1}[\theta(Z) - \nu \geq 0] \end{aligned}$$

with $(\epsilon, \nu) \perp\!\!\!\perp (X, Z)$, but

- no parametric restriction on functional form of $\mu(\cdot), \theta(\cdot)$
- no parametric restriction on distribution of (ϵ, ν)

What Assumptions Can We Impose?

Nonparametric threshold crossing model on each equation:

$$\begin{aligned} Y &= \mathbf{1}[\mu(X, D) - \epsilon \geq 0] \\ D &= \mathbf{1}[\theta(Z) - \nu \geq 0] \end{aligned}$$

- What is economic content of the model on D? On Y? How restrictive are these assumptions?
- Can show (Vytlacil 2002, 2006, Vytlacil and Yildiz 2007) that nonparametric threshold crossing model is equivalent to
  - LATE Monotonicity condition of Imbens-Angrist
  - weak monotonicity assumption between observed regressors and unobserved regressors

What Assumptions Can We Impose?

Nonparametric threshold crossing model on each equation:

$$\begin{aligned} Y &= \mathbf{1}[\mu(X, D) - \epsilon \geq 0] \\ D &= \mathbf{1}[\theta(Z) - \nu \geq 0] \end{aligned}$$

- How much identifying power is provided by nonparametric threshold crossing model on Y?
- How much identifying power is provided by nonparametric threshold crossing model on D?
- How much identifying power is provided by jointly imposing nonparametric threshold crossing models on both D and Y?

What Assumptions Can We Impose?

Consider imposing nonparametric threshold crossing model on:

1. D but not on Y
   - i.e., nonparametric selection model of Heckman-Vytlacil, equivalently LATE monotonicity assumption of Imbens-Angrist
2. Y but not on D
   - investigated by Machado, Shaikh, and Vytlacil, related work by Chiburis, and by Chesher
3. both Y and D
   - Vytlacil-Yildiz, Shaikh-Vytlacil, Bhattacharya-Shaikh-Vytlacil, and related work by Chiburis, and by Abrevaya-Hausman-Khan

Outline of Rest of Paper

We now consider how each alternative set of assumptions aids in recovering different definitions of "the effect" of treatment.

We conclude with brief empirical application to Swan-Ganz catheterization.

Selection Model

Consider imposing:

1. $D = \mathbf{1}[\theta(Z) - \nu \geq 0]$
2. $Z \perp\!\!\!\perp (\nu, Y_0, Y_1)$

while imposing no structure on the model for Y.

- Allowing arbitrary heterogeneity across people in $Y_1 - Y_0$
- Restricts heterogeneity across people in how Z affects D.
  - In Angrist-Imbens-Rubin terminology, rules out "defiers"
- Y need not be binary

Imposed by:

- Imbens-Angrist as LATE conditions
- Heckman-Vytlacil as Nonparametric Selection Model

Selection Model (cont'd)

Does this structure help in identification of $ATE = E(Y_1 - Y_0)$ or $TT = E(Y_1 - Y_0|D = 1)$?

- Answer: No

To see this, can compare bounds that exploit instrument and do or do not additionally impose selection model structure.

Selection Model (cont'd)

Sharp Bounds on ATE, TT exploiting instrument without additional structure

- Mean independence IV assumption (Manski, 1990).
- Full independence IV assumption
  - Balke-Pearl (1997) for binary Y, binary Z
  - Kitagawa (2009) for general case

Sharp Bounds on ATE, TT imposing selection model

- Balke and Pearl (1997) for case of binary Y, binary Z.
- Heckman and Vytlacil (2001) for general Y, general Z

Selection Model (cont'd)

Sharp bounds under selection model have simple form.

- Width of bounds depends on linearly on distance between largest value of $\Pr[D = 1|Z = z]$ and 1, and distance between smallest value of $\Pr[D = 1|Z = z]$ and 0.

Comparing to sharp bounds that exploit independence instrument but nothing more to sharp bounds that additionally impose selection model, B-P and H-V show that imposing selection model implies testable restrictions on observable data that simplifies the form of the sharp bounds but does *not* narrow sharp bounds.

Selection Model (cont'd)

Selection model does not aid in identifying ATE or TT, but it does critically allow the identification of other (local/marginal) parameters:

- LATE – average treatment effect defined by instrument (Imbens and Angrist, 1994)
- MTE – average treatment effect for someone at given quantile of latent desire to receive treatment (Heckman and Vytlacil, 2001, 2005)

Selection Model (cont'd)

Selection model does not aid in identifying ATE or TT, but it does critically allow the identification of other (local/marginal) parameters:

- Policy effects (given support conditions, Heckman and Vytlacil 2005)
- Marginal Policy Effects (Carneiro, Heckman and Vytlacil 2010)
- Average effect for those at the margin of indifference of whether to participate in treatment (Carneiro, Heckman and Vytlacil 2010)

Selection Model (cont'd)

Thus, imposing threshold crossing structure on the model for D (i.e., LATE assumptions of Imbens-Angrist, equivalently selection model assumptions of Heckman-Vytlacil), does not provide any identifying power for ATE or TT, but does provide identification of many marginal parameters that otherwise would not be identified.

Restriction on Outcome

Consider imposing:

1. $Y = \mathbf{1}[\mu(X, D) - \epsilon \geq 0]$
2. $Z \perp\!\!\!\perp (\{D_z\}, \epsilon)$

while imposing no structure on the model for D, where $\{D_z\}$ are latent choices.

- Allowing arbitrary heterogeneity across people in how Z affects D
- Restricts heterogeneity across people in how D affects Y.
- Special Case: Probit model with binary endogenous regressor

We follow analysis of Machado, Shaikh and Vytlacil (2010). Related to analysis by Chiburis (2009)

Restriction on Outcome (cont'd)

Machado, Shaikh and Vytlacil (2010) show that restriction does narrow sharp bounds on ATE and TT, though bounds are still frequently wide.

Show that:

- If effect of Z on D is strong enough (instrument is strong enough), and probability limit of IV is far enough from zero, then can identify sign of ATE, TT.
- If instrument is weak, or plim of IV is close to zero, cannot identify sign of ATE, TT.
- Counter-intuitive result: sometimes possible for IV to be so far positive that can infer ATE is negative. . . .

Comparison to imposing restriction on treatment: does not allow identification of local/marginal parameters, but does provide additional power for inference on ATE, TT.

Restriction on Outcome & Treatment

Consider imposing structure symmetrically on both equations:
Nonparametric threshold crossing model on each equation:

$$\begin{aligned} Y &= \mathbf{1}[\mu(X, D) - \epsilon \geq 0] \\ D &= \mathbf{1}[\theta(Z) - \nu \geq 0] \end{aligned}$$

with $(\epsilon, \nu) \perp\!\!\!\perp (X, Z)$.

- Nonparametric version of Heckman's bivariate probit model.
- Restricts heterogeneity across people in how Z affects D
- Restricts heterogeneity across people in how D affects Y

Restriction on Outcome & Treatment

Vytlacil and Yildiz (2007) show that if possible to vary X continuously holding $\Pr[D = 1|Z]$ constant, and in particular vary X sufficiently to exactly compensate for variation in D, then can identify and estimate ATE in this model.

Key Idea:

- Use modified version of IV that shifts Z and X simultaneously, in a particular controlled way, to uncover what shift in X exactly compensates for a shift in treatment.

Key Drawback:

- Require at least one component of X to be continuous, and to vary sufficiently conditional on $\Pr[D = 1|Z]$.
- What happens without such variation?

Restriction on Outcome & Treatment

Shaikh and Vytlacil (2010) :

- Impose restrictions without requiring X to contain a continuous component or any other support condition on X.
- Use modified-IV strategy that simultaneously shifts exogenous covariates for the Y equation and the instruments Z.
- Recover what shifts in exogenous covariates over- or under-compensates for shifts in D, use this information to construct sharp bounds on ATE.

Restriction on Outcome & Treatment

- Worst case, no exogenous covariates, still uncover with IV whether no shift in exogenous covariates over- or under-compensates for shifts in D.
  - Thus, can still uncover direction of effect in worst case
  - Related to Abrevaya, Hausman, and Khan (2009)
- Analysis extended by Chiburis (2009)

Application: Swan-Ganz Catheterization

Bhattacharya, Shaikh and Vytlacil (2010): Empirical Application to Swan-Ganz Catheterization

Question: How does placement of Swan-Ganz catheters upon admission to an ICU affect patient mortality?

More than 2 million patients catheterized each year.

Unclear whether catheterization increases or decreases mortality.

Swan-Ganz Catheterization

A Swan-Ganz catheter is a long slender tube with sensors that is placed by an ICU doctor in an artery near the heart.
The catheter measures several clinically important pressures inside the heart, which are then used by ICU doctors in determining treatments for patients.

Previous Research on Swan-Ganz Catheterization

Although several studies using non-experimental data have found that catheterization increases patient mortality, ICU doctors remain skeptial.

Most influential study is by Conners et al. (1996):

Detailed data on 5,735 patients from 5 major ICUs.

Used propensity score matching.

Found that catheterization increases 180-day mortality.

Remark: Major concern is that method assumes no endogeneity conditional on observed characteristics.

Swan-Ganz Catheterization

We re-analyze, applying Shaikh-Vytlacil bounds that allow for endogeneity while imposing nonparametric threshold crossing restriction on both equations. We implement their bounds in a way that is robust to the instrument not being relevant (robust to weak instrument or even irrelevant instrument).

How plausible are these assumptions?

Trade-off in assumptions?

Data

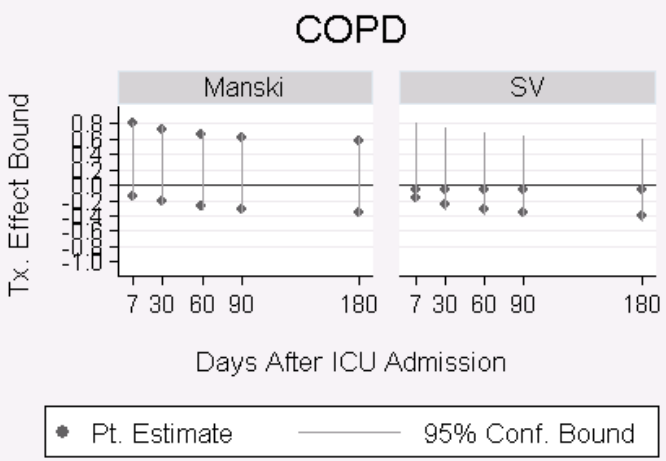
Same data used by Conners et al. (1996):

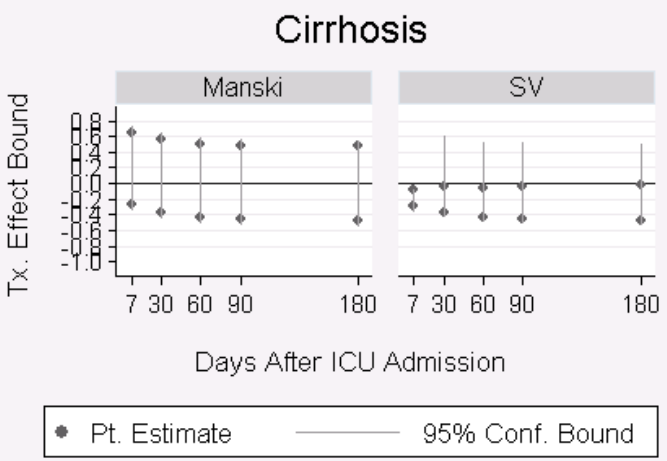
- 5,735 patients (3,551 of whom were catheterized within 24 hours of ICU admission).
- For each patient, observe 63 different characteristics, including demographic information, past medical history, and clinical variables.
- Mortality recorded at 7, 30, 60 and 180 days after admission.

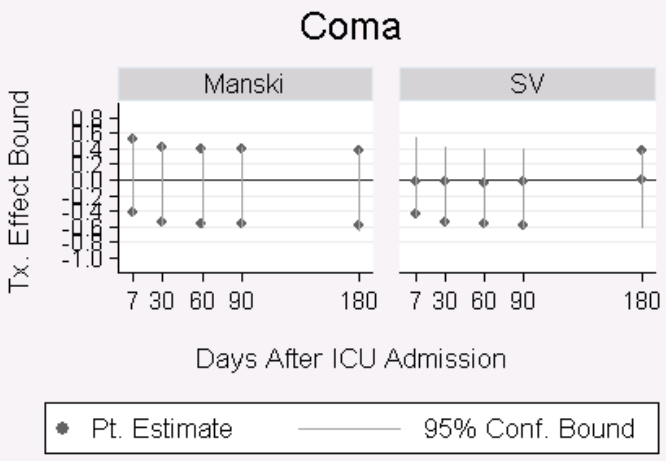
Instrument

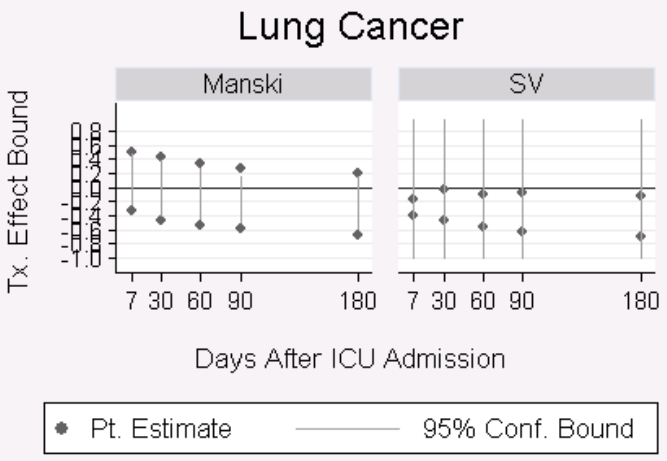
Use $Z = I\{\text{patient admitted on a weekday}\}$ as an instrument.

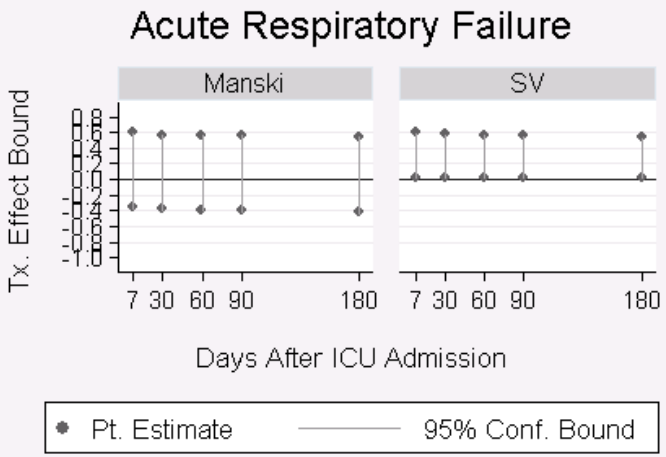
- Z predicts catheterization.
  Only doctors perform catheterizations and there are fewer doctors in the ICU on weekends.
- Patients admitted to ICU on weekends similar to patients admitted on weekdays.
  Comparing patients along 63 different characteristics, find only 4 statistically significant differences.
  After accounting for multiplicity of hypotheses under consideration, these differences disappear as well.
  Importantly, no difference in predicted 2-month mortality.

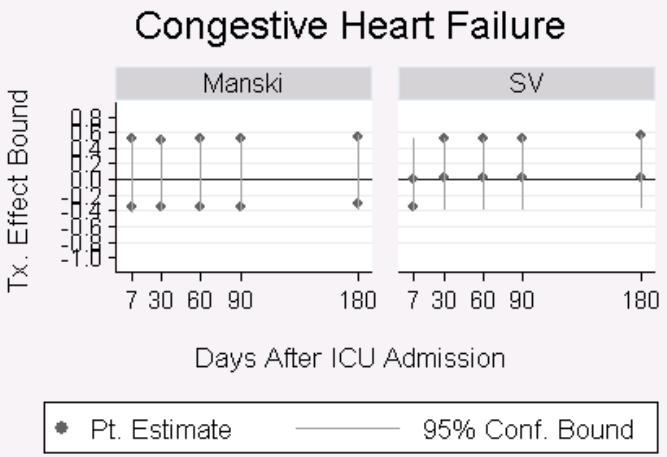

COPD
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound

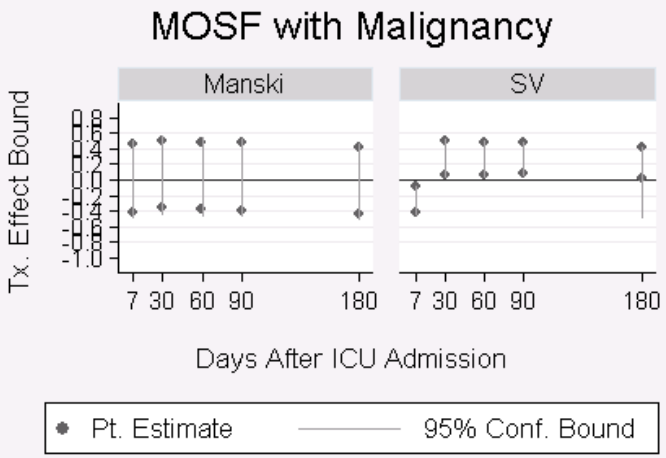

Cirrhosis
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound

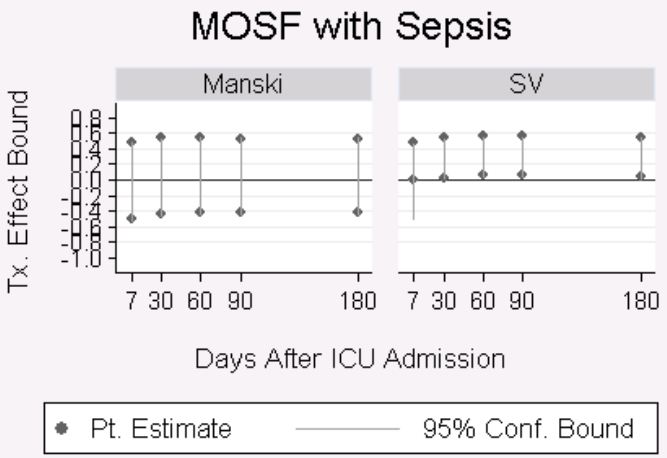

Coma
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound


Lung Cancer
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound


Acute Respiratory Failure
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound


Congestive Heart Failure
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound


MOSF with Malignancy
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound


MOSF with Sepsis
Manski
SV
Tx. Effect Bound
7 30 60 90 180
7 30 60 90 180
Days After ICU Admission
Pt. Estimate
95% Conf. Bound

Conclusion

Restricting heterogeneity in how treatment choices respond to the instrument does not aid in identification of ATE/TT, but does critically allow identification of local/marginal parameters.

Restricting heterogeneity in how outcomes respond to the treatment does aid in identification of ATE/TT, not for local/marginal parameters.

Restricting both has tremendous identifying power for ATE, especially in presence of other exogeneous covariates.